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CASE REPORT

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BIZZARE PATHWAY IN ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA

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Background: Atrioventricular nodal reentrant tachycardia (AVNRT) ablation has a high success rate. Fully understanding the substrate for AVNRT remains a chcallenge. Various inputs to the AV node occur in less than 5% of cases. Persistent VA dissociation during RV pacing is uncommon. Right sided inferior nodal extensions could be the critical slow pathway in some cases and may require targeting for successful treatment.
Case illustration: A 66 Y.O. female referred with diagnosis of Supra Ventricular Tachycardia (SVT) for EP study (EPS) and Ablation. EPS has been done with 2 quadripolar catheter to His and RV apex, 1 duo-decapolar catheter to CS and 1 ablation catheter. SVT consistently induced with extra stimuli. During SVT VA Interval 98 ms with earliest A in CS 1-2 (located in mid CS). RV pacing during SVT consistently showed AV dissociation and earliest A switched between CS 1-2 and 9-10 without SVT termination. AVRT was excluded due to AV dissociation during RV pacing. SVT consistently induced by extra-stimuli, sometimes followed with AV jump, which indicate AVNRT. Due to earliest A in mid septal which transitioned to CS 9-10 without terminating SVT, we hypothesized that the mechanism was atypical slow-slow AVNRT with Left Inferior Extension which sometimes switched to slow-fast AVNRT, especially during RV pacing which penetrate the slow pathway. Slow Pathway at Right Inferior Extension were ablated using 35 W power for 60 seconds. After ablation, no SVT was induced with burst and extra-stimuli pacing.
Conclusion: Differentiating AVNRT from other SVT can be complex. Specific pacing maneuvers often needed during EPS. The presence of VA block rules the diagnosis of AVRT. The presence of AV jump indicates a dual nodal pathway. Uniquely, gradually decreased VA interval and switching of the earliest A were observed during SVT, which could differentiate AT and shifting pathway from slow-slow to slow-fast AVNRT. Successful slow pathway ablation at RIE area can confirm atypical AVNRT with shifting pathways. Pacing maneuvers in conventional EPS of SVT can enhance the likelihood of identifying the pathway that should be ablated.

Keyword: AVNRT, shifting pathway, LIE, RIE

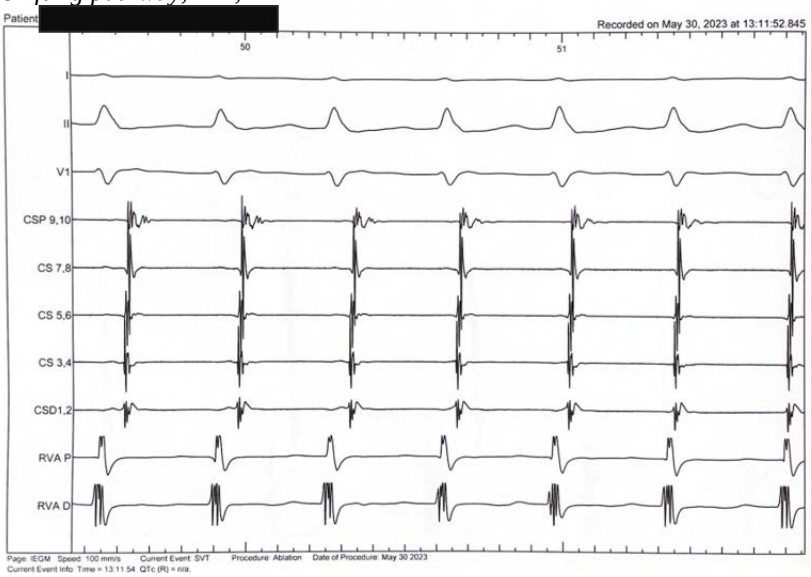

Figure 1. Decreased VA Interval with earliest A at CS 1-2

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BREAKING BOUNDARIES: CONVENTIONAL ABLATION USING TRANSJUGULAR APPROACH IN WOLFF-PARKINSON-WHITE SYNDROME WITH COR TRIATRIATUM

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Background: Cor triatriatum is a rare congenital anomaly where the left or right atrium is divided by a tissue pleat, membrane, or fibromuscular band. While typically diagnosed in children, its occurrence in adults is uncommon. Patients often remain asymptomatic, though some may develop symptoms resembling mitral or tricuspid stenosis, and occasionally, arrhythmias.

Case Illustration: A 45-year-old man presented at the outpatient clinic with palpitations. He had no family history of sudden cardiac death, hypertension, diabetes, or dyslipidemia. His vital signs were normal and physical examination revealed a wide fixed splitting S2. The twelve-lead electrocardiogram showed sinus rhythm with a positive delta wave in leads I, aVL, V2-V6, and negative in V1, indicative of a right-sided accessory pathway (AP). Transthoracic echocardiography revealed normal cardiac chambers with an atrial septal defect (ASD). Cardiac CT scan confirmed a 14.5mm secundum ASD and right cor triatriatum, with no patent ductus arteriosus or major aortopulmonary collateral artery observed. Right and left heart catheterization confirmed a secundum ASD with a left-to-right shunt and cor triatriatum. Despite being unable to obtain referral for 3D ablation at another hospital, the patient underwent conventional electrophysiology study and ablation. Antidromic AVRT with right-side AP was identified as clinical tachyarrhythmia induced by incremental pacing from the right ventricle with TCL 290 ms and VA 160 ms. A duodecapolar catheter was employed to localize the AP along the annulus of the tricuspid valve. The maximum VA fusion was observed in the posterior free wall of the tricuspid annulus area, as indicated by the electrograms. The radiofrequency ablation (RFA) was delivered via the right internal jugular vein using non irrigating ablation catheter during sinus rhythm. Elimination of delta wave and retrograde block were achieved after several seconds of RFA. One year follow-up confirmed sustained resolution of symptoms and absence of recurrence of WPW syndrome.

Conclusion: This case highlights the successful application of conventional EPS and ablation techniques for managing WPW syndrome in patients with cor triatriatum, showcasing both the feasibility and sustained clinical improvement of this approach in challenging anatomical conditions.

Keyword: Cor triatriatum, Wolff-parkison-white syndrome, ablation technique

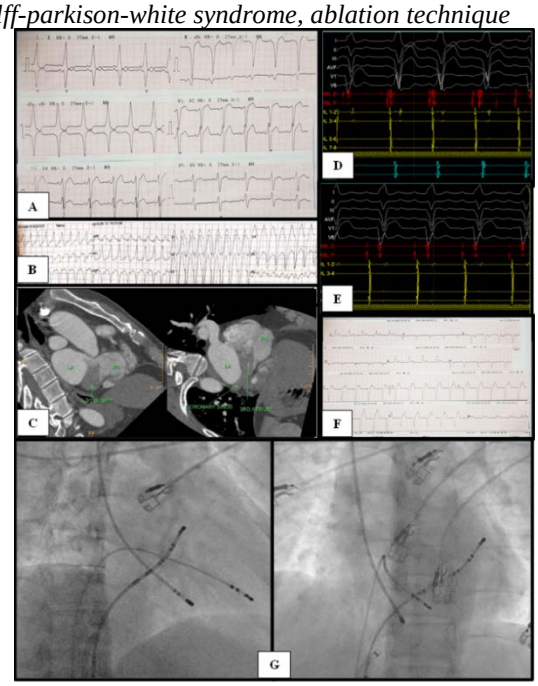

Figure 1. (A) Surface ECG demonstrating sinus rhythm with delta wave
(B) Surface ECG demonstrates SVT (C) Cardiac MSCT identifies the third atrium
(D) Accessory pathway ablation (E) Successful accessory pathway ablation
(F) Surface ECG after successful ablation (G) Fluoroscopic view during conventional ablation

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HYBRID SURGICAL THORACOSCOPIC RADIOFREQUENCY ABLATION AND ENDOCARDIAL CATHETER ABLATION IN LONG-STANDING PERSISTENT ATRIAL FIBRILLATION IN A PATIENT WITH HYPERTROPHIC CARDIOMYOPATHY

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Background: Atrial fibrillation (AF) is the most common arrhythmia in patients with hypertrophic cardiomyopathy (HCM). Ablation for atrial fibrillation is safe but less effective in patients with HCM compared to those without this condition. Hybrid ablation (HA), which combines thoracoscopic epicardial and transvenous endocardial approaches in a single procedure, has shown better outcomes in patients with persistent atrial fibrillation

Case Illustration: A 43-year-old male with long-standing persistent AF and hypertrophic cardiomyopathy underwent HA. Echocardiography revealed a left atrial diameter of 38 mm, an interventricular septum (IVS) thickness of 17 mm, a left ventricular ejection fraction of 59%, mild to moderate mitral regurgitation, and no left ventricular outflow tract obstruction. The patient received minimally invasive hybrid surgical thoracoscopic radiofrequency ablation (Maze procedure) and endocardial catheter ablation for AF, and with left atrial appendage resection. Surgical ablation lines were created along the bilateral pulmonary veins, left atrial roof, and between the superior and inferior vena cava (SVC and IVC).

Post-surgical ablation, the patient remained in AF. Left atrial mapping indicated scattered low-voltage areas on the anterior wall, mitral isthmus, and wide low-voltage areas in the bilateral pulmonary veins, posterior wall, and left atrial floor. (figure 1a) Right atrial mapping showed scattered low-voltage areas in the septal and posterior right atrium. (figure 1b) Additional endocardial catheter ablations targeted the roof line, mitral isthmus line, septal line, CTI line, and right atrial posterior line. During the procedure, the patient received ibutilide (1 mg), and during septal line ablation, the rhythm converted to sinus. (figure 1c) There were no complications related to the procedures.

Conclusion: This case demonstrates the successful use of hybrid surgical and endocardial catheter ablation in a patient with long-standing persistent AF and hypertrophic cardiomyopathy.

Keywords: Hybrid AF ablation, surgical radiofrequency ablation, catheter ablation, long-standing persistent AF, hypertrophic cardiomyopathy

Figure 1a.

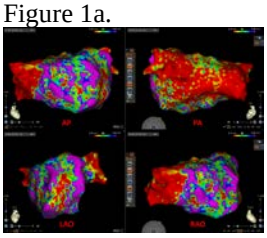


Figure 1b.

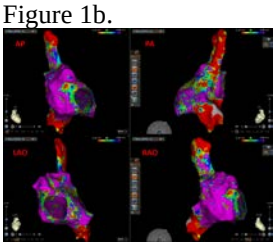
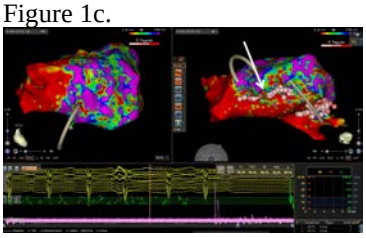


Figure 1c.



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RESYNCHRONIZATION THERAPY IN A HEART FAILURE PATIENT: IMPROVEMENTS AT ONE-MONTH FOLLOW UP

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Background: Cardiac Resynchronization Therapy (CRT) has been proven to be beneficial for selected fraction of heart failure patients. Left Bundle Branch Area Pacing (LBBAP) aimed to capture the physiologic conduction system has been reported to be a cost-effective alternative for CRT.
Case Illustration: A 73 year old male presented to the emergency department with shortness of breath that worsened 2 days before admission. There was history of significant limitation in doing daily activities. Patient had a history of percutaneous coronary intervention prior, resulted in three vessel and left main disease with 1 stent to LM-LCx and 1 stent to proximal RCA. Optimal medical therapy was taken routinely. Physical examination and chest x-ray were indicative of pulmonary congestion. Laboratory findings were unremarkable. The ECG showed sinus rhythm with complete LBBB. Basic echocardiography findings included dilatation of left atrium (LAVI 52.75 m^2) and ventricle (LVEDD 6.3cm), reduced EF of 20% and GLS of -6.1%. M-mode indicated presence of intraventricular dyssynchrony shown by delayed aortic pre-ejection interval of 168 ms, while septal-to-posterior wall motion delay (SPWMD) was around 230 msec. Patient was stabilized and subjected to LBBAP procedure. On 1-month follow up, patient presented with better functional status (NYHA class II) and overall quality of life. The QRS duration was narrowed. Echocardiography parameters were improved (LAVI 41.96 m^2 and LVEDD 5.8cm, EF 26%, GLS -7.7%), aortic pre-ejection interval reduced significantly, while SPWMD diminished.
Conclusion: LBBAP is a safe and effective option of CRT in patient with symptomatic heart failure and conduction delay. It showed the ability of improving clinical status, quality of life, as well as echocardiographic parameters of cardiac dyssynchrony after one month of procedure.

Keyword: Left Bundle Branch Area Pacing, Cardiac Resynchronization Theraphy

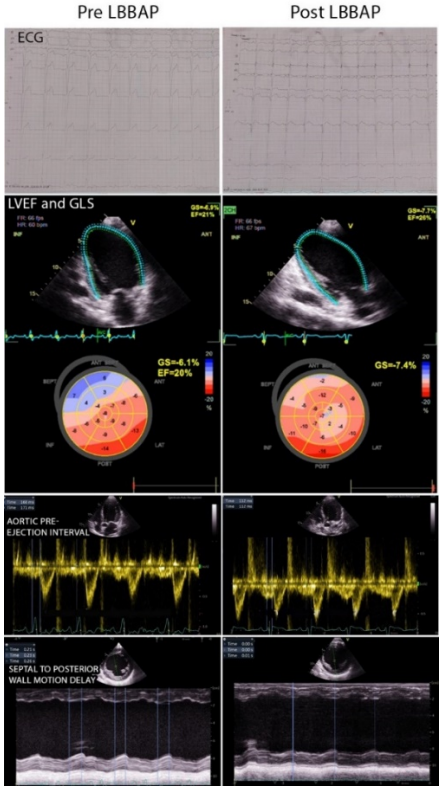

Figure 1. Pre and post LBBAP comparisons of ECG and Echocardiography Parameters

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INCESSANT SUPRAVENTRICULAR TACHYCARDIA IN WOLFF-PARKINSON-WHITE SYNDROME ESCALATING VENTRICULAR TACHYCARDIA AND VENTRICULAR FIBRILLATION: A CAUTIONARY TALE OF ANTIARRHYTHMIC THERAPY

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Background: This case report highlights the challenges in managing incessant supraventricular tachycardia (SVT) and subsequent ventricular tachycardia (VT) and ventricular fibrillation (VF) in a patient with Wolff-Parkinson-White (WPW) syndrome, emphasizing the complexities and potential complications of antiarrhythmic therapy.

Case illustration: A 54-year-old woman presented with palpitations, chest pain, and shortness of breath. Electrocardiography (ECG) revealed narrow complex tachycardia at 180 beats per minute (bpm), indicative of orthodromic atrioventricular reentrant tachycardia (AVRT). Hemodynamic instability was noted. Chest x-ray shows cardiomegaly and left pleural effusion. Electrical cardioversion, along with standard antiarrhythmic therapy was initiated, restoring a heart rate of 74 bpm, revealing WPW in sinus rhythm. Despite additional electrical cardioversion and medication adjustments, her SVT recurred. Electrical cardioversion up to 200 joules failed to terminate the SVT, necessitating the maximization of antiarrhythmic drug therapy including beta-blockers, calcium channel blockers, digoxin, and amiodarone. Additionally, it occasionally induced episodes of VT and VF, accompanied by several instances of seizures and loss of consciousness. Direct current (DC) shocks of 200 joules, combined with cycles of cardiopulmonary resuscitation (CPR), successfully restored sinus rhythm. The choice of therapy in this condition is crucial due to the antagonistic effects between antiarrhythmic drugs for rhythm control and vasoconstrictors and inotropic agents for stabilizing decreased blood pressure. This condition is life-threatening and requires unique considerations for acute management and ultimate ablation. An electrophysiological (EP) study and subsequent ablation of the right posterolateral accessory pathway were successfully performed to stabilize her rhythm, addressing the underlying arrhythmogenic substrate.

Conclusion: This case underscores the complexities of incessant SVT in WPW syndrome, escalating to VT and VF. Successful ablation of the accessory pathway in this case, promoting the importance of timely electrophysiological intervention. Moreover, it highlights the delicate balance between necessary therapeutic interventions for arrhythmia control and the potential for severe systemic complications. Therefore, clinicians should emphasize the importance of vigilant monitoring and comprehensive care.

Keywords: incessant supraventricular tachycardia, WPW syndrome, ventricular tachycardia, ventricular fibrillation, catheter ablation, antiarrhythmic therapy

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A FASCINATING RIDDLE: THE SUCCESSFUL MANAGEMENT OF RECURRENT ELECTRICAL STORMS USING CARDIAC CRUX ABLATION IN ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY - A CASE REPORT

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Background: Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a rare hereditary structural heart disease, with various phenotypes, resulting in fibrofatty replacement of the heart muscles and a proclivity to create spontaneous malignant cardiac arrhythmias arose from right ventricle that may lead to sudden death. Although most previous reports were treated by cardioverter defibrillator, hence preventing death, we report a case of its successful management of recurrent electrical storms using alternative method, cardiac crux ablation, thus making this case is the first report of using this treatment.

Case illustration: We reported a 55 year old male patient came to Emergency Room with worsening exertional palpitations and non-specific chest discomfort. Dynamic electrocardiogram recorded symmetric T wave inversions and possible epsilon waves in inferior leads. Subsequent work up showed right ventricular chamber enlargement and systolic dysfunction on echocardiography, fatty infiltration and many localized distensions at the edge of the right ventricular wall on cardiac magnetic resonance imaging. On following days the patient was clinically deteriorating with recurrent non-inducible LBBB-pattern ventricular tachycardia. Then prompt electrophysiologic study was planned with the result of inducible incessant ventricular tachycardia from the right ventricle. Those findings confirmed the diagnosis of ARVC and urgent ablation were selected to be performed on cardiac crux with satisfactory result.

Conclusion: This is the first reportable case wherein cardiac crux ablation is preferred as the initial treatment of ARVC, considering incessant ventricular tachycardia during electrophysiology study that threaten survival. Dramatic declining condition such as frequent episodes of electrical storms leads this case to be more challenging. With prompt recognition and life-saving action such as urgent ablation, those are important things to do with optimal management strategy as alternative therapy of ARVC.

Keywords: T wave inversion, epsilon waves, incessant ventricular tachycardia, arrhythmogenic right ventricular cardiomyopathy, cardiac crux ablation.

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PACEMAKER-INDUCED CARDIOMYOPATHY: OUTCOMES OF UPGRADING TO CONDUCTION SYSTEM PACING IN MULTIPLE CASES

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Background: The incidence of Pacemaker Induced Cardiomyopathy (PICM) depends on the definition used, but in general it occurs in 10-20% of patients after 3-4 years of implantation. In recent years, the use of conduction system pacing (CSP), whether through His bundle pacing (HBP) or left bundle branch area pacing (LBBAP), has shown promising results in the management of PICM as a resynchronization therapy. However, there is still limited data presenting the success of conduction system pacing in the management of PICM.

Case illustration: We report 8 cases of patients diagnosed with PICM who underwent conduction system pacing upgrade, mostly male (62.5%). The average age of the patients was 61.3 years, with an age range from 35 to 78 years. Patients underwent conduction system pacing upgrade due to the diagnosis of PICM and experienced heart failure symptoms such as fatigue, weakness, or dyspnea. Before undergoing conduction system pacing upgrade, patients underwent echocardiography with an average LVEF of 39.5%, with the lowest LVEF reaching 18% and the highest LVEF reaching 46%. About 3 patients (37.5%) had coronary artery disease, underwent percutaneous coronary intervention and completely revascularization. Six patients (75%) underwent LBBAP and two patients implanted His bundle pacing. All patient underwent echocardiography evaluation with an average follow-up of 6 months. Echocardiography showed an improvement in LVEF, with an average increase to 51.5%, representing an average increase in LVEF of 11.3% compared to before. Almost half patient had superresponder and normalization of LVEF. QRS duration before upgrade was 172 ms then become 131 ms after upgrade to conduction system pacing.

Conclusion: Conduction system pacing, whether with HBP or LBBAP, is a new physiological pacing modality that can be considered as one of the therapy options providing resynchronization in cases of PICM. HBP and LBBAP have been proven safe and effective with a physiological pacing strategy. Thus, HBP and LBBP can lead to improved LV function and reduced patient mortality. However, further research is still needed to determine whether CSP-based CRT is non inferior than conventional CRT.

Keyword: Pacemaker induced cardiomyopathy, conduction system pacing.

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DOUBLE TACHYCARDIA, VT WAS INDUCED BY SVT: A CASE REPORT IN 34 YEARS OLD WOMAN WITH HISTORY OF PALPITATION

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Background: Double tachycardia was defined as occurrence of two distinct tachyarrhythmias simultaneously or in alternating manner. AVNRT is the most common supraventricular arrhythmia. SVT faster than 250 beats per minute (bpm) may induce monomorphic ventricular tachycardia. We present case about a 34 years old woman without any structural heart disease suffering RVOT VT that was triggered by SVT.

Case Illustration: Woman, 34 years old age, with history of palpitation. She suffered palpitation during rest along with diaphoresis. There was history of VT at the ER in the last admission. In outpatient care, she was planned for ablation procedure. There was no relevant family history. Electrocardiograms (ECGs) showed a normal sinus rhythm with 76 bpm. Echocardiography and electrolytes revealed normal results. Holter monitoring showed multiple episodes of non-sustained monomorphic VT and infrequent PVCs. At EP lab, we found that VT was triggered by SVT with earliest activation -38 msec at mid septal RVOT region. Multiple radiofrequency ablation (RFA) were done at the area and the VT was terminated. Right ventricular overdrive pacing (RVOP) was performed and we found VAV response with ventricular post pacing interval greater than 115 msec longer than tachycardia cycle length. Multiple RFA were delivered in the slow pathway area, accelerated junctional rhythm was seen during RFA. The procedure was concluded after ensuring absence of AH jump and ventricular tachyarrhythmias by programmed atrial stimulation and burst ventricular pacing, respectively.

Conclusion: We showed case about successful VT and slow pathway ablation in 34 year old woman experiencing double tachycardia.

Keyword: Ablation, Palpitation, Double Tachycardia

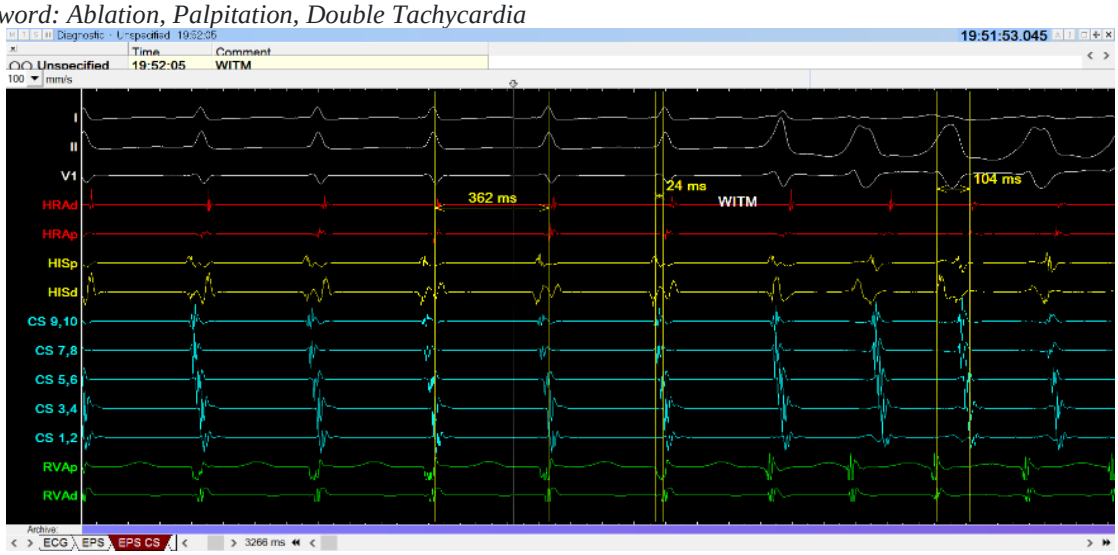

Figure 1 showed the episode of SVT induce VT

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FREQUENT PREMATURE VENTRICULAR COMPLEX ABLATION WITH ZERO FLUOROSCOPY IN A PREGNANT PATIENT AT SOETOMO GENERAL HOSPITAL IN SURABAYA, INDONESIA

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Background: Pregnancy complicated by arrhythmias pose a predicament for the treating physician due to radiation exposure and uncertain dangers for the mother and fetus. Hence, catheter ablation has been seldom performed and is often delayed until the postpartum period.

Case Illustration: A 34-year-old woman primigravida in the gestational age of 26th-27th week presented to our instituition with drug-refractory and poorly tolerated frequent premature ventricular complex (PVC). Cardiac examinations demonstrated structurally normal heart without any known history of cardiovascular disease. Her electrocardiography showed posteroseptal right ventricular outflow tract (RVOT) PVC origin and underwent catheter ablation. The patient successfully underwent zero-radiation ablation guided by the Ensite NavX system, a non-fluoroscopic navigation. Multiple radiofrequency ablations were accomplished using a 30-watt flexible ablation catheter at 45 degrees Celsius for 60-120 seconds in the posteroseptal RVOT. Accelerated ventricular tachycardia (VT) was observed and a further 30 minutes observation showed no PVCs. The patient was then discharged the following day and her antiarrhythmic medication was discontinued. No episode of PVC was observed during follow-up.

Conclusion: Catheter ablation of frequent PVC in pregnancy can be safely and effectively performed with a zero-fluoroscopy approach guided by the Ensite NavX system. In the cases of drug refractory, catheter ablation during pregnancy may be considered.

Keyword: premature ventricular complex, catheter ablation, pregnancy, zero-flouroscopy

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POSSIBILITIES IN THE UNEXPECTED: CONVENTIONAL ABLATION ACCESSORY PATHWAY IN COMPLEX CONGENITAL HEART DISEASE

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Background: Complex congenital heart disease is one of the predictors of arrhythmia related problems. The underlying condition deteriorates the disease, leading to worsen heart function and risk of sudden cardiac arrest. Ablation is the first recommendation in patients with tachyarrythmias. The unique anatomy makes conventional ablation not the main preference in congenital heart disease (CHD)

Case Illustration: A 3-year-old child with cc-TGA presented with frequent palpitations. Pulse oximetry measurements showed a pulse rate of 230-240 bpm. This condition often occurs intermittently with a duration of one hour, but currently the palpitations do not improve. The complaint was triggered by the patient's condition which was experiencing fever, cough and cold. The patient was previously admitted to another hospital with recurrent AVRT since the patient was 2-months year old. The condition improves with adenosine administration but three times had to undergo electrical cardioversion. Ablation was decided, considering the patient's age and weight, we performed conventional ablation. Mapping of the lateral AP retrogradely, earliest AV fusion was found in the left lateral area of mitral annulus (CS 1-2). Multiple RFAs (30 W, 60^0C,120 s) successfully separated the AV fusion within 3.10 seconds, retrograde ventricular pacing showed retrograde block. After 20 minutes of observation, delta wave did not reappear and programmed ventricular stimulation showed retrograde conduction with separated VA at all CS.

Conclusion: Arrhythmias remain one of the major causes of hospitalization and predictors of mortality in CHD patients. There are no randomized controlled trials currently to guide specific antiarrhythmic drug treatment due to the heterogeneity and continuing evolution of CHD. For this reason, catheter ablation guided by electro-anatomical 3D mapping is considered first-line therapy and is preferred over long-term pharmacological treatment in CHD-related arrhythmias, if there are no reversible hemodynamic or other factors that can be overcome. However, conventional ablation is feasible within the limitations of developing countries.

Keyword: AP ablation in congenital heart disease, conventional ablation in congenital heart disease, cc-TGA WPW ablation

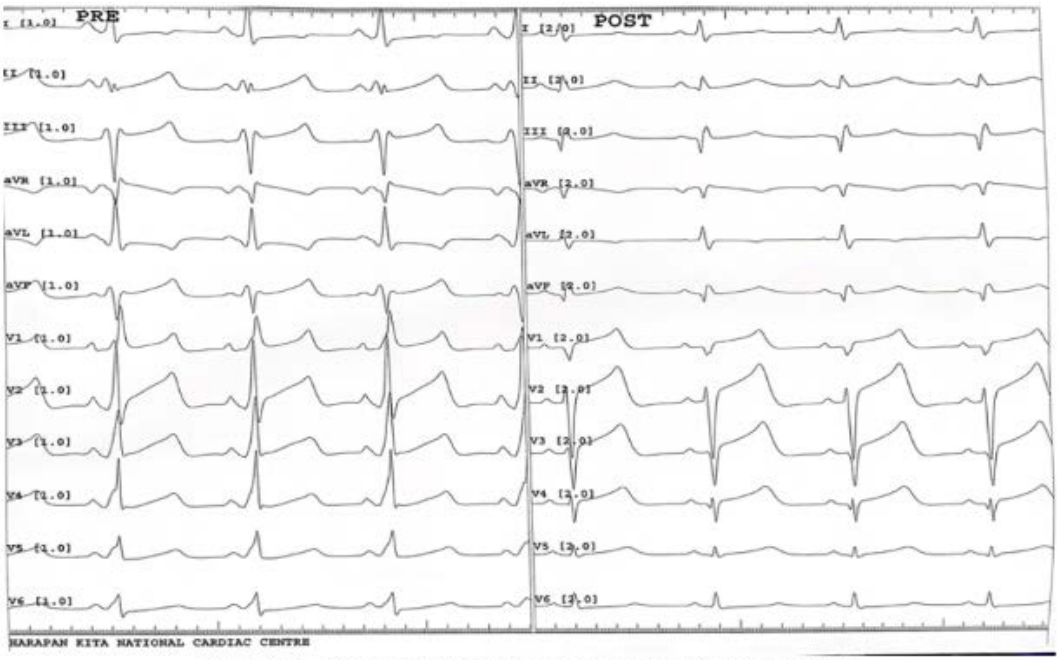

Figure 1. Electrocardiography pre-post procedural

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Optimization of AV and VV delay in patient HFrEF with IVCD implanted LOT CRT

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Background: LOT-CRT has shown encouraging results for QRS reduction and heart function improvement in IVCD patient. Optimal management of the AV and VV delay has become important method to improve left intraventricular synchrony of LOT-CRT therapy. We present a case of the greater electrical resynchronization through AV and VV delay optimization defined by shorten QRSd in male receiving LOT-CRT for severe HFrEF with IVCD.

Case Illustration:

A 40 yo male was hospitalized for intractable NYHA FC III symptoms of HF of a non-ischaemic aetiology. The ECG showed 1st degree AVB and IVCD with QRSd of 215 ms. He had reduced LVEF of 20%, dyskinetic base to mid IVS, hypokinetic base to mid anterior, anterolateral, inferolateral with akinetic other segments.

The patient underwent LOT-CRT. A Medtronic quadripolar LV lead was successfully implanted in a posterolateral branch of the CS. An 3830 SelectSecure^{R} lead was placed in LBBAP and an active atrial lead, Medtronic Capsurefix^{R} 53 MRI, was placed in the right atrial appendage. The atrial lead, LBBP lead and LV lead was connected to atrial port, RV port and LV port of SolaraQuad^{R} CRT-P generator respectively. Optimal LV quadripolar lead pacing configuration, AV delay and VV delay was determined during implantation in search of the narrowest QRSd. The LV1 to LV4 gave the satisfactory pacing parameter (QLV of 190ms; impedance of 758ohm) without resulting high capture threshold (0.75V at 0.5ms) and phrenic nerve stimulation. Due to the challenges in achieving LBB capture during the placement of the LBBP lead, LVSP was adopted as a bailout strategy for LBBP. The final best bipolar-LVSP mode alone paced QRSd and LVAT were 182ms and 120ms. The LVSP capture threshold at implant was 0.5V at 0.4ms and R-wave amplitude >20mV. Acute response was measured by QRSd immediately after implantation. Empirically program device to a fixed AV delay interval (Sensed AV of 120ms and Paced AV of 150ms) was used. The VV delay (LBBB pre-excitation) of 70ms, creating LV together with LVSP pace simultaneously, had shown to be the most effective resynchronization resulting narrowest QRSd of 120ms.

At 12-weeks post implant follow up, patient underwent optimization reprogramming. The patient was symptomatically much improved in NYHA FC I yet his LVEF remained 20%. Repeat echocardiography showed more synchronous activation of LV. The narrowest QRSd was still seen at a same value of programmed AV and VV delay during acute programming. His latest pacing check showed that both the threshold of the LV lead and LBBAP remained stable (0.75 at 0.4ms with impedance of 817ohm and 0.25V at 0.4ms with impedance of 646ohm, respectively). Overall, the percent of atrial pacing and LOT-CRT ventricular pacing rate was 13.0% and 99.6%, respectively.

Conclusion: Individualized optimizing AV timing and VV timing to be an integral part of the mechanism through which LOT-CRT delivers benefit to patient with IVCD. Adjustments of the AV and VV delay result in sequential LOT-CRT pacing and directly modify the sequence of atrioventricular and intraventricular activation, which may achieve narrower QRSd and better LV electrical synchrony.

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His Bundle Area Pacing Using Custom Stylet-Driven in Atrial Fibrillation: Revisited Old Technique Case Series

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Background: Over the past two decades, there have been significant advancements in cardiac pacing, including His bundle pacing (HBP). However, identifying the His bundle potential in atrial fibrillation (AF) patients may be challenging, especially in patients with structural heart problems. We present a custom stylet-driven pacing lead (C-SDL) that utilises a conventional pacemaker lead with high output in the His bundle area, making it feasible to identify His bundle area and make a non-selective HBP in our two AF patients.
Case illustration: All patients showed AF and underwent implantation using C-SDL from Biotronik Solia S-60 (Figure 1A). Patient 1 was a 69-year-old female who presented with dyspnea. Her echocardiogram showed severe mitral regurgitation (MR) and moderate tricuspid regurgitation (TR). Her electrocardiograms showed paroxysmal AF, with a QRS rate of 36 bpm. A temporary pacemaker (TPM) was inserted, and we didn't find other reversible causes of bradycardia, so she proceeded to VVIR permanent pacemaker (PPM) implantation in his bundle area. After 4 days post-implantation, her ECG showed QRSd of 80 ms. The His-lead parameters were R wave = 4,7 mV, impedance = 515 Ω, NS-HBP threshold 4V @ PW 0.75 ms, and RV myocardial capture threshold 0.9 V@ PW 0.5 ms (Figure 1B). Patient 2, a former navy officer, was a 48-year-old male with near syncope. He underwent Holter monitoring and showed persistent AF with a pause of 3.4 seconds during the daytime. He had VVIR PPM implantation in his bundle area. After 4 days, the post-implant follow-up ECG showed QRSd 100 ms with NS-HBP. The His-lead parameters were: R wave = 8,7 mV, impendence = 585 Ω, NS-HBP threshold = 0,8V @ 0,75 ms (Figure 1C).
Conclusion: His bundle area pacing, utilising C-SDL, is a viable technique for implantation pacing, especially beneficial for individuals with AF, regardless of whether they have had cardiac anatomy alterations. In addition, high output pacing in this technique can predict the tip of the pacemaker lead was near His bundle even when the operator could not see the His potential due to AF.

Keywords: His Bundle Area Pacing, Atrial Fibrillation, Case Series

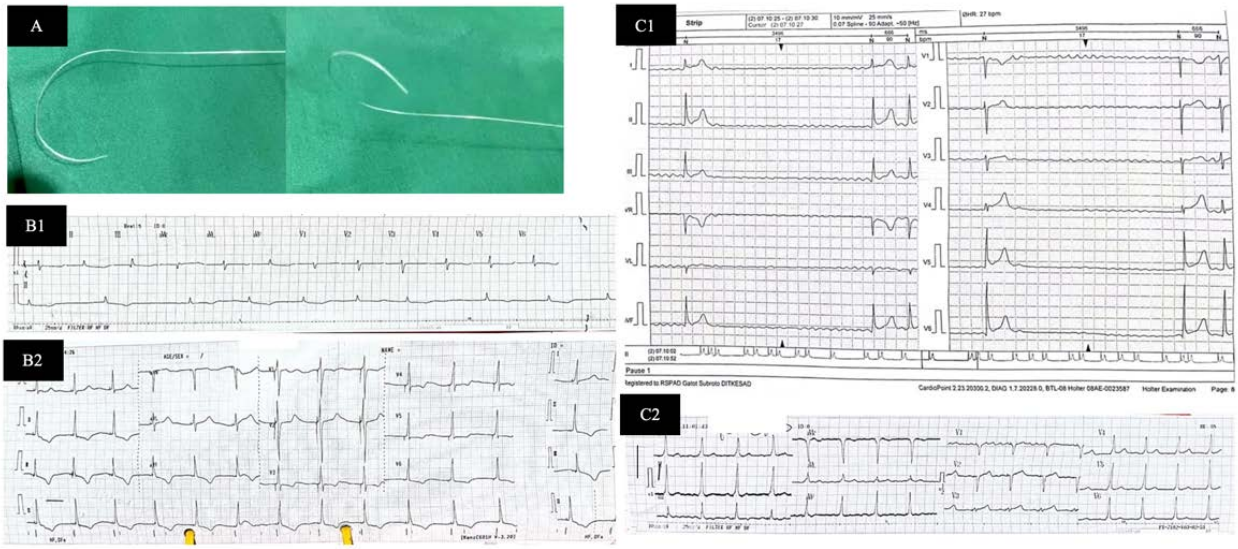

Figure 1. A: C-SDL which the operator created from conventional pacemaker lead with a curve shape and the stylet that was adjusted to His area by fluoroscopy guided during a pacemaker implantation procedure. B: Patient 1's ECG before (B1) and after (B2) implantation of his bundle area pacing. C: Patient 2's Holter and ECG before (C1) and after (C2) implantation of his bundle area pacing.

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Two Life on the Edge: Navigating Catecholaminergic Polymorphic Ventricular Tachycardia Through Pregnancy

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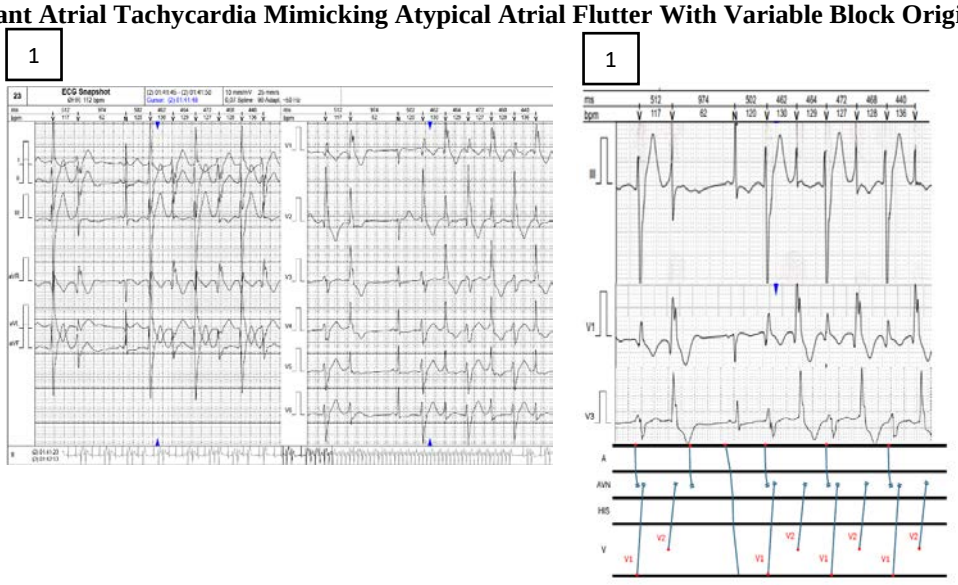
Background: Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a rare genetic disorder marked by episodes of bidirectional ventricular tachycardia triggered by stress, particularly catecholamines. Typically, patients have normal resting electrocardiogram and no structural heart disease. Pregnancy induces significant physiological changes, including heightened cardiovascular responses to catecholamines, and the peripartum period entails emotional and physical stress that can exacerbate arrhythmic events in CPVT. According to the WHO classification of maternal cardiovascular risk, CPVT falls into mWHO class III, with a maternal cardiac event rate ranging from 19% to 27%. Despite the need for comprehensive management in pregnant women with CPVT, there is limited literature available on CPVT during pregnancy and peripartum period.
Case Illustration: A 24-year-old G2P0A1 patient diagnosed with CPVT since 2018 came to clinic at 8 weeks gestational age (WGA). Her cardiovascular function was normal but holter monitor in the second trimester revealed recurrent asymptomatic bidirectional non-sustained ventricular tachycardia with premature ventricular contraction burden of 30%. The patient had been prescribed bisoprolol 2.5 mg once daily before pregnancy, which was continued throughout pregnancy and changed to propranolol 20 mg thrice daily peripartum. A multidisciplinary team including maternal-fetal medicine and anaesthesiology collaborated to plan the route and timing of delivery based on obstetric indication, with serial fetal ultrasounds through pregnancy to monitor growth. During delivery, continuous cardiovascular monitoring was implemented, sympathomimetic agents were avoided, and the newborn was closely observed in NICU for potential arrhythmias and hypoglycaemia risks. At 38 WGA, the patient underwent a caesarean section due to cephalopelvic disproportion with epidural anaesthesia, during which bigeminy premature ventricular contractions were noted. She delivered a healthy 2600 g female infant who remained asymptomatic during the NICU observation period. Propranolol was continued post-delivery.
Conclusion: Management of CPVT in pregnancy needs a multidisciplinary approach initiated preconception to mitigate mortality risks. Patients should receive comprehensive counselling regarding potential maternal and neonatal complications. Continuous beta-blocker therapy throughout pregnancy is crucial. Delivery mode and timing should be carefully determined based on obstetric indication, with vigilant cardiovascular monitoring, minimizing pain, and avoidance of sympathomimetic agents during labor. Neonates should undergo NICU monitoring for possible arrhythmias and hypoglycaemia.

Keyword: Catecholaminergic polymorphic ventricular tachycardia, pregnancy, delivery

Figure 1. Figure 1A shows non sustained bidirectional VT from 12 lead ECG. Ladder diagram shows AV dissociation with 2 sources of VT (V1 from inferior LV and

Recalcitrant Atrial Tachycardia Mimicking Atypical Atrial Flutter With Variable Block Originating From Left Superior Pulmonary Vein



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Background: Atrial Tachycardia (AT) have been demonstrated to arise from both atria and other surrounding structures. Its origin from left side atria, particularly in pulmonary vein (PV), is less frequent.
Case Illustration: A 41-year-old woman was referred to our emergency department with refractory and recurrent supraventricular tachycardia (SVT). ECG revealed narrow complex tachycardia with a rate of 166 beats per minutes, recognized as atrial flutter (AFL) with variable block (1:1-2:1). The patient was treated with amiodaron, beta blocker, and synchronized cardioversion. After recalcitrant SVT, she was implanted with a temporary pacemaker as atrial overdrive pacing. On physical examination, the patient was classified as overweight (BMI 25.3 kg/m2). The vital signs were normal, except for the increased heart rate. Laboratory and echocardiography results within normal ranges. The woman has had intermittent palpitations for the past 12 years, intensified over the last 3 years particularly during heavy activity. Before this referral, the patient underwent an Electrophysiology study (EPS) which revealed non-inducible tachyarrhythmia and normal function of the SA and AV nodes. However, complaints of palpitations still continue to recur. Hence, EPS with 3D activation mapping was performed. The surface ECG showed positive P-wave at V1 and bifid P-wave at lead II, suggestive of AT locating at left PV or left atrial appendage. Orion Cathether was placed in both atria for 3D activation mapping. The earliest activation was at Left Superior Pulmonary Vein (LSPV), thus radiofrequency ablation was applied there and the rhythm converted into sinus during ablation. AT often appeared with variable conduction. In such case, the variable conduction may occur because of the Wenckebach periodicity and the effect of anti-arrhythmic drugs that act as an AV Node blocker.
Conclusion: AT with variable conduction can be challenging to be differentiated from AFL. Besides, it is often refractory with anti-arrhythmic drugs. Cathether ablation with 3D activation mapping can effectively revealed origin and offers a high acute as well as long-term success rate.
Keywords: atrial tachycardia, variable conduction, left superior pulmonary vein

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Multifaceted of 2:1 AV Block: A Case of Infra-Hisian Block

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Background: Atrioventricular (AV) block was an AV conduction disorder that occured in various settings. Infra-hisian block was a condition that associated with blocked electrical conduction within or distal to the His bundle. This case emphasizes the importance of Electrocardiography (ECG) in the management of patients with infra-hisian block.
Case Illustration: A 60-year-old woman admitted to hospital with late onset of dizziness. The heart rate was low (<50x/min). The ECG recorded during symptomatic episode. We performed dual chamber Left Bundle Branch Area Pacing (LBBAP) pacemaker implantations.
We reported a case with multifaceted 2:1 AV block and alternating Bundle Branch Block (BBB), consisting of Right Bundle Branch Block (RBBB) and Left Bundle Branch Block (LBBB) who underwent EP study and was diagnosed with infra-hisian Block.
ECG properties were known to assist in the localization of conduction block, including width of QRS complex and PR interval length. ECG (1) showed sinus rhythm, 1st degree AV block, and bifascicular block that led to trifascicular block. It gave a hint on infra-hisian block that the patient had. Both ECG (2) and (3) showed 2:1 AV block. They showed an alternating BBB that also supported diagnosis of infra-hisian block. It mandated the patient to have pacemaker implantation.
The previous Ejection Fraction (EF) of the patient was 31%. Conduction System Pacing was performed due to her EF less than 55%. During pacemaker implantation, we noticed “A” signal was followed by “H” signal before the block, confirming infra-Hisian Block. Post pacemaker implantation showed non-selective LBBAP.
Conclusion: We described a 60-year-old woman who had alternating BBB combined with various 2:1 AV Block. EP study revealed infra-hisian block. In conclusion, It was rare presentation with multi-level conduction system disease. We had to think about the diagnosis of infra-hisian block earlier, if ECG showed multifaceted 2:1 AV block with alternating BBB.

Keywords: Alternating Bundle Branch Block, infra-hisian block

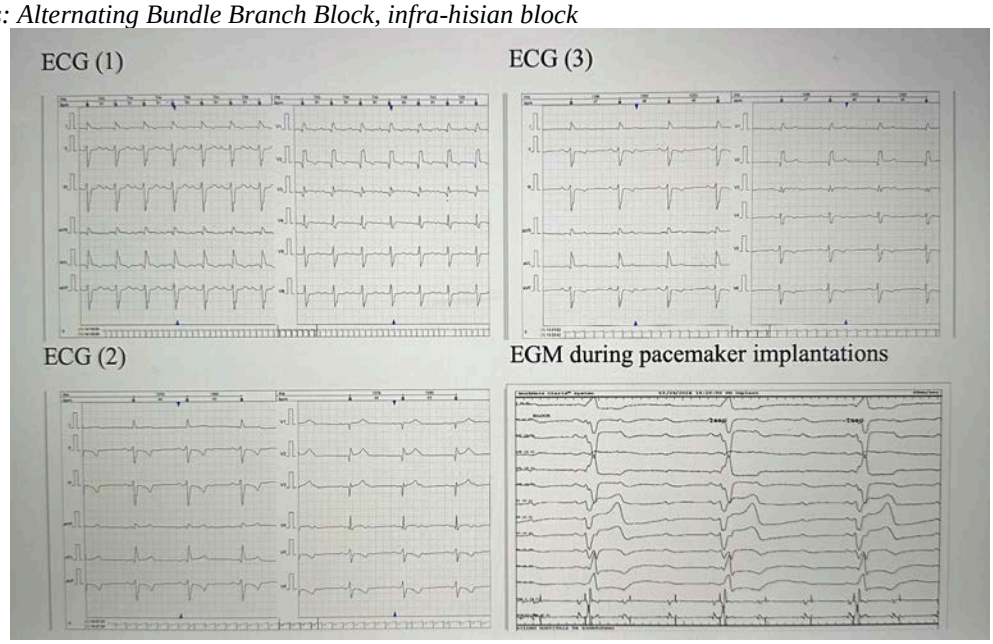

Figure 1. The ECG (1) showed Trifascicular block. Both ECG (2) and (3) showed 2:1 AV Block. ECG (2) showed RBBB, whereas ECG (3) showed LBBB. EGM during pacemaker implantations showed infra-hisian block.

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Electrical Storm in Long QT Syndrome: *beyond QTc shortening*

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Background: Long QT syndrome (LQTS) is an arrhythmia syndrome, characterized by prolonged QT interval, that may present with malignant arrhythmia and risk of SCD. The annual rate of SCD was estimated only around 0.5% in asymptomatic patients, but it was 10 times higher in those with history of syncope. Although it is already known that LQTS mainly caused by disturbance of ventricular repolarization, either due to channelopathy or acquired disturbance, till date, there is no definitive treatment of LQTS.
Case Illustration: A 24-year-old female was presenting to ED with shortness of breath and episodes of palpitation at rest, without any past illness. ECG showed ST, iRBBB, and long QT/QTc interval (400/534 mS; Figure 1). Vital signs were stable except for mildly increased HR. Although the symptoms were suspected due to LQTS and further evaluation was needed, she refused to be hospitalized.
The next day, she came to ED because of worsening symptoms and also having diarrhea and nausea. Vital signs remain stable except for mildly increased respiratory rate without any distress. ECG showed SR and bigeminy PVC (MB origin) with R-on-T Phenomenon. Suddenly, the patient was fainted without pulse, and TdP was recorded, prompting for resuscitation. After successful resuscitation, ECG recording showed SR with longer QT/QTc interval (560/659 mS; Figure 4). She was consulted to arrhythmia division and transported immediately to ICVCU. Laboratory evaluation only showed severe hypokalemia (3.09 mmol/L). Echocardiography showed LVEF 45,2%, dilatation of RA-RV with reduced RV function, and moderate TR. After kalium level was corrected, concomitant with beta-blocker administration, ECG showed shortening of QT interval (480/565 mS; Figure 5) but TdP episode occurred again multiple times. As ICD could not be implanted due to facility limitation, in attempt to supress VA episode, ranolazine was administered and had shorten QT/QTc interval (360/464 mS; Figure 6). Despite that, electrical storm remains, and after many resuscitation attempts, patient was pronounced deceased.
Conclusion: Despite Ranolazine can reverse action-potential prolongation and suppress the EADs and ventricular tachycardia induced by other agents, in this case, electrical storm still ensues. This indicates a complex mechanism of electrical storm in LQTS beyond QT interval prolongation.

Keywords: electrical storm, long QT syndrome, ranolazine

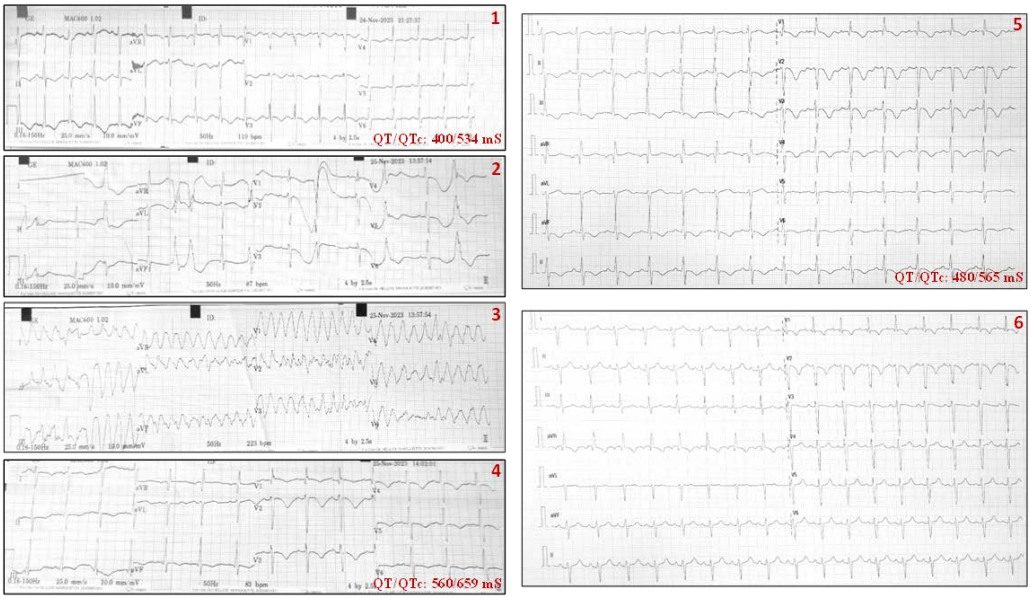

Figure 1. 1st ED admission; Figure 2. 2nd ED admission; Figure 3. During resuscitation;
Figure 4. Post-resuscitation; Figure 5. Post-kalium correction; Figure 6. After ranolazine administration

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Electrophysiologic Characteristic of Patient with Atrioventricular (AV) Block During AV Nodal Reentrant Tachycardia (AVNRT). It Has Been a Functional AV Block.

R. Chandara, H. Chhayroud, C. Sokha

Background: The lower common pathway is located at the distal junction of the slow, fast pathways and the His bundle in the AVN. The case aims to review and discuss about the electrophysiologic characteristic of LCP block during AVNRT.

Case Illustration: A 55-year-old female was admitted for recurrent palpitation. The EKG during admission showed narrow complex tachycardia with HR 190bpm. The day after that, the patient was undergoing for ablation. The baseline electrocardiogram was normal. Procedure was done under local anesthesia. One 7F non-irrigated ablation catheter and two quadripolar electrode catheters were inserted via right femoral veins and placed at HRA, His and RVA respectively. One right jugular vein puncture was performed. One 6F sheath was inserted. One decapolar catheter was inserted and secured at the coronary sinus. While placing the CS catheter, the SVT induced 2:1 AVNRT and spontaneously converted to 1:1 narrow complex tachycardia with TCL 320 ms. During tachycardia showed AV dissociation (Figure 1). SVT occurs with 2:1 AV conduction and the AVRT was ruled out.

AVNRT with 2:1 block to the atrium with resumption of 1:1 conduction and acceleration of the AVNRT is showed in Figure 2. Block is also initiated at the onset of tachycardia/ begin as 2:1 block and when PVC/HSPVB was given, it becomes 1:1 conduction in the lower common pathway. During tachycardia maneuver was demonstrated, RVOP maneuver showed VAV response, PPI-TCL > 115 ms, VA interval 37 ms. HRVPB showed no reset. AH is more than HA during tachycardia. Therefore the diagnosis favors slow-fast AVNRT with LCP block. The slow pathway ablation was mapped at the mid-posterior septal region of triangle of Koch. Accelerated junctional rhythm was seen during RFA. After 20 minutes of observation, programmed atrial stimulation showed no AH jump, no echo, the FP ERP becomes shorten after ablation and did not induce AVNRT.

SVT with 2:1 AVB is less common, but the most commonly occurs below the His in either a 2:1 or Wenckebach fashion at the onset of the tachycardia. The observation of block below the His has no implication as to the site of turn around. Although A-V block has most commonly been observed during typical AVNRT. However, one must prove that this rhythm is not atrial tachycardia by demonstrating its initiation with ventricular programmed stimulation(VPS) and the ability to reset or entrain the tachycardia with VPS, while maintaining the exact same retrograde sequence as the tachycardia and demonstrating a VAV response after the tachycardia convert to 1:1 conduction. Evidently termination of the tachycardia by VPS which do not reach the atrium, excludes AT and AVRT.

We considered 3 reasons suggesting that 2:1 AVB occurs during episode of AVNRT wasn't caused by pathology of AV block: (1)phenomen of 2:1 AVB during AVNRT was independent and wasn't associated with HV interval prolongation (2)mode of initiation of episode of AVNRT associated with 2:1 AVB usually were compatible with exposure of HB to relatively long cycle length , consistent with functional bloc (3) AVNRT with 2:1 AVB also had several episode of AVNRT at same CL associate with 1:1 AV conduction.

There are some constraints of this case: (1) Atropine doesn't use to clarify the AVB still persist after atropine suggesting that site of block isn't in the AVN. (2) The exact mechanism of UCP couldn't determine.

Conclusion: In this case, the AVNRT with AV dissociation base on electrophysiology properties lead to variation in the demonstration of lower common pathway. It's important to give VPS/do some maneuver to exclude the other form of SVT to match a diagnosis.

Key words: Atrioventricular nodal reentrant tachycardia, lower common pathway, AV block.

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Successful conventional radiofrequency catheter ablation of Wolf-Parkinson-White Syndrome at the His bundle: a case report

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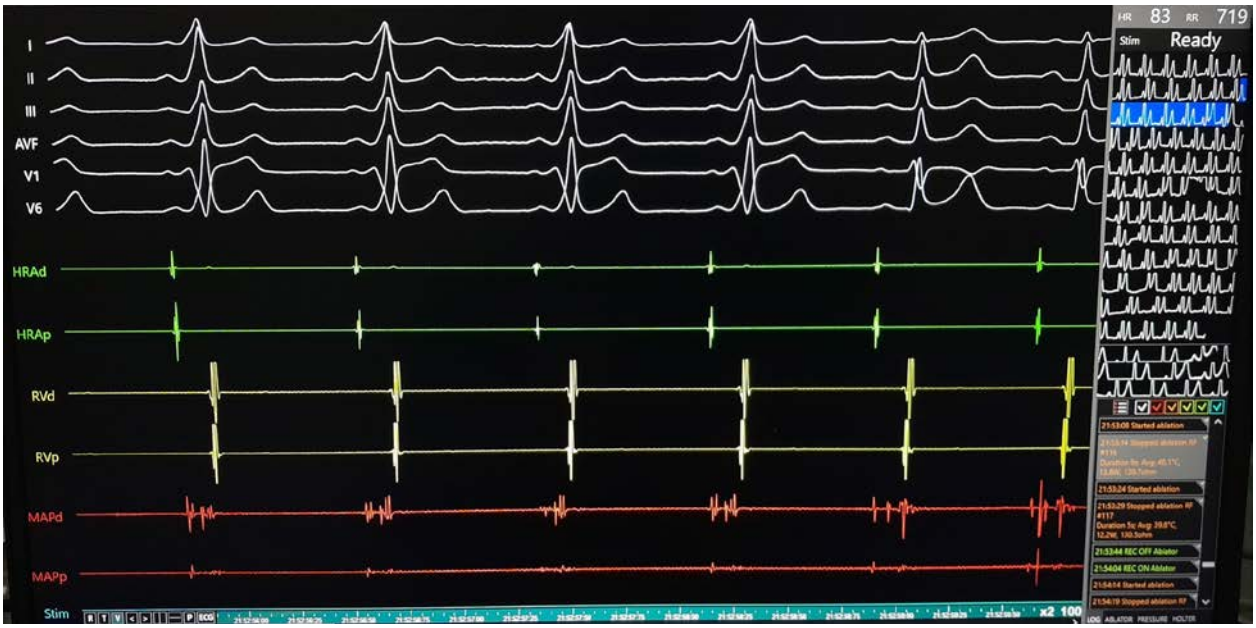
Background: Wolf-Parkinson-White (WPW) syndrome is a rare cardiac arrhythmia characterized by an accessory pathway connecting the atria and ventricles, leading to abnormal electrical conduction and palpitations. Radiofrequency catheter ablation (RFCA) is a widely accepted treatment for WPW syndrome, offering a high success rate and minimal complications.
Case Illustration: A 31-year-old Indonesian male presented with a chief complaint of palpitations lasting one year, with no abnormal physical findings, laboratory results, or radiology examinations. His medical history was clear of any significant cardiac or systemic conditions. Electrocardiography (ECG) revealed a short PR interval and delta wave, consistent with WPW syndrome. The patient underwent RFCA at the His bundle under local anaesthesia, guided by fluoroscopy. Before ablation, atrioventricular (AV) signal fusion was more profound in the His area, indicating the presence of His signal. Owing to its rural location, conventional 2D ablation was performed using an ablation energy of 20 watts for 30 s (low power, short duration). The fusion signal was successfully eliminated, and no evidence of junctional rhythm was observed during ablation. After the procedure, the patient's palpitations resolved, and his ECG showed normalization of the PR interval and the absence of the delta wave.
Conclusion: This case highlights the successful treatment of WPW syndrome using RFCA in the His bundle in a patient with no significant medical history or physical findings. The patient's symptoms resolved and his ECG normalized following the procedure. The use of low-power, short-duration ablation in a rural setting, where 3D equipment is unavailable, demonstrates the adaptability and effectiveness of RFCA in treating WPW syndrome.

Keywords: Wolff-Parkinson-White Syndrome, Electrophysiological Study, Radiofrequency Catheter Ablation, His Bundle



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Familial Hypertrophic Cardiomyopathy: An Exploration of Varied Clinical Presentations Arising from Genetic Mutations

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Background: Familial hypertrophic cardiomyopathy (HCM) is an autosomal dominant disease that could affect as many as 1 in 500 individuals, showcasing variability among patients regarding the timing of onset, phenotype, and clinical progression. The two genes in which most mutations have been described are the B-myosin heavy chain (*MYH7*) and the myosin binding protein C (*MYBPC3*).

Case illustration: The patient a 60-year-old mother whose genetic status was unknown, electrophysiology study revealed sick sinus syndrome included atrial fibrillation and bradyarrhythmia. Echocardiography revealed a dilated LA and concentric LVH with a borderline LV ejection fraction (EF) (53%). Late gadolinium enhancement (LGE) indicated transmural enhancement at the apicoinferoseptal region, patchy enhancement at the mid inferoseptal and anteroseptal areas. She passed away suddenly with an undocumented malignant arrhythmia suspected as the cause of death. Her 39-year-old son was found to have the MYH7 mutation. Echocardiography showed biatrial dilatation with a reduced EF (40%). LGE revealed prominent focal patchy mid-wall enhancement at both right ventricular (RV), particularly at the anterior extending to the basal anterior, basal to mid anteroseptal, and apical anterior RV. The QRS complex initially appeared narrow but gradually widened over time, indicating significant progression compared to his mother. After recurrent episode of ventricular tachycardia (VT), an implantable cardioverter-defibrillator (ICD) was implanted. However, he did not survive thereafter due to a VT storm followed by pulseless electrical activity. Both patients were diagnosed with HCM.

Conclusion: The life-long process of LV remodeling and progressive dysfunction that occur in a substantial proportion of HCM patients and culminates in the rare but dramatic clinical evolution termed as *end-stage* or *burned-out* phase characterized by severe functional deterioration of the LV. It is clinically important to distinguish between the various stages of HCM. A key mechanism for adverse outcomes is believed to be myocardial fibrosis, a pathological hallmark of the condition.

Keyword: Familial hypertrophic cardiomyopathy, genetic mutation.

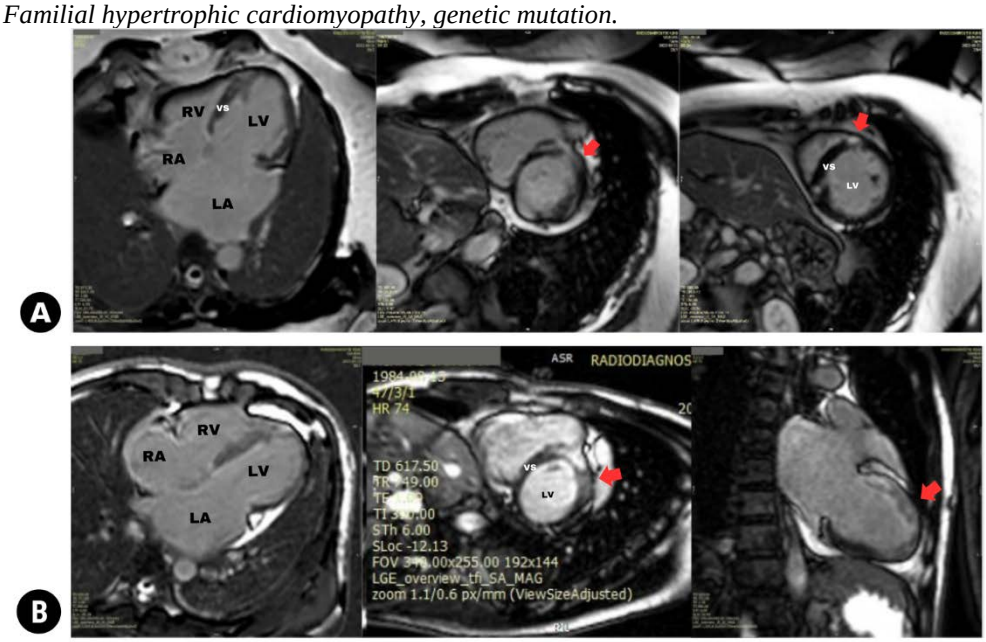

Figure 1) Four-chamber and short-axis cardiac magnetic resonance views, from **A)** 60-year-old female HCM patient with unknown genetic status, and **B)** 39-year-old male HCM patient with the MYH7 mutation.

Recurrent torsade de pointes of acquired long QT syndrome: catastrophic arrythmia behind subclinical hyperthyroid

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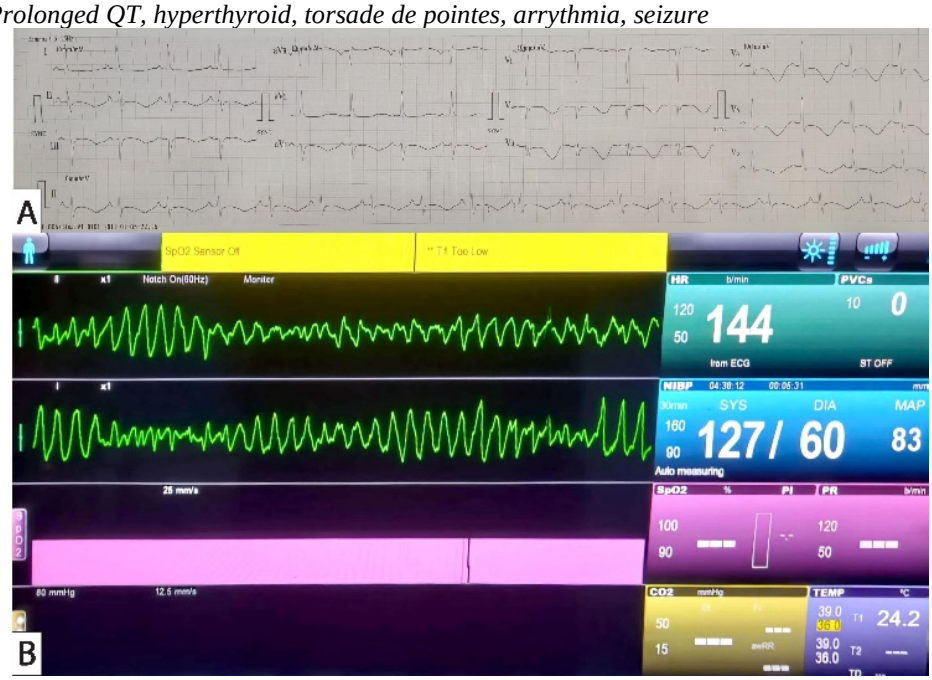
Background: Corrected QT (QTc) prolongation is well-documented in hypothyroidism, but its occurrence in hyperthyroidism remains controversial due to conflicting reports. This case report describes a patient with hyperthyroidism who exhibited QTc prolongation, resulting in severe arrhythmia.
Case illustration: This case report presents a 70-year-old female patient who was referred to our hospital with severe epigastric pain, a history of syncope, and a brief generalized tonic-clonic seizure occurring the previous day, initially suspected to be due to acute coronary syndrome. Further history taking revealed a 13-year history of recurrent seizures preceded by headaches. Electrocardiography (ECG) reexamination showed prolonged QTc, inverted T waves in leads V1-V6, II, III, and aVF, and ST depression in leads V4-V6. Laboratory tests indicated slight hypokalemia, hypocalcemia, and normal troponin I levels. During her hospital stay, the patient experienced three seizure episodes, each lasting approximately one minute and resolving spontaneously, with monitoring ECG indicating a distinct torsades de pointes pattern. Magnesium sulfate and a lidocaine drip were administered to prevent further seizures. Subsequent investigations revealed subclinical hyperthyroidism, characterized by mildly elevated free T4 and very low thyroid-stimulating hormone levels. Hyperthyroidism therapy was initiated with propylthiouracil and a beta-blocker.
Conclusion: Screening for thyroid hormone levels should not be overlooked in patients presenting with arrhythmias of unclear etiology. The administration of antiarrhythmic drugs must be selective in hyperthyroid patients with prolonged QTc; therefore, the use of an implantable cardioverter-defibrillator (ICD) becomes crucial, including and especially for patients in rural areas.

Keywords: Prolonged QT, hyperthyroid, torsade de pointes, arrythmia, seizure



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Rare Yet Possible - Ventricle Induced Atrial Tachycardia

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Background: Focal atrial tachycardia (AT) is defined by atrial activation originating from a discrete focus with centrifugal spread, often caused by automaticity, triggered activity, or micro-reentry. Despite its relative rarity, AT can lead to significant clinical symptoms and requires precise diagnosis and intervention, often through electrophysiology (EP) studies and radiofrequency ablation (RFA).

Case Illustration: We report a case of a 38-year-old female civil servant, Mrs. AH, presenting with dizziness, palpitations, and episodic non-sustained ventricular tachycardia (VT). Initial evaluations of Holter monitoring indicated infrequent multifocal PVC with episode of non-sustained VT. Patient was given Bisoprolol since then and exercise stress test was done for treatment evaluation, showed no arrhythmia during test and negative ischemic response. Despite medical management with Bisoprolol, symptoms persisted, necessitating an EP study. During the EP study, incremental ventricular pacing showed retrograde block at 375 ms. PVC was non-inducible so isoproterenol was given. With 300 ms ventricular pacing, supraventricular tachycardia was induced within Wenckebach periodic. RV pacing manoeuver showed no reset, long PPI-TCL, VAAV sequence and episode of warming up on atrial rate, suggesting Atrial Tachycardia. Mapping was performed using non irrigating catheter at Crista terminale, Bachman Bundle Area, Septal Area of RA, Lateral Area of RA and Inferior Area near CS ostial (all RA was mapped). Earliest A recorded at inferior septal RA near CS ostial and successfully ablated using multiple radiofrequency.Post-ablation, the patient's symptoms significantly improved, and follow-up ECG showed no recurrence of AT.

Conclusion: This case highlights the importance of comprehensive evaluation and targeted ablation in managing focal AT. Successful ablation can significantly alleviate symptoms and improve quality of life in patients with AT. Further structural heart evaluations may be warranted to ensure long-term outcomes.

Keywords: Focal Atrial Tachycardia, Electrophysiology, Radiofrequency Ablation, 2D Mapping, Non-sustained Ventricular Tachycardia

Figures :

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The whimsical heart block: A case series of a rare manifestation of myocarditis

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Background: In a rare occurrence, acute myocarditis can manifest as a high-degree atrioventricular conduction block (HDAVB). Previous data suggests only 1.14% of myocarditis manifests as an HDAVB. Therefore, diagnosing myocarditis as the etiology of HDAVB needs an extensive process of excluding other possible reversible causes. Despite the risk of hemodynamic compromise, this condition is mostly reversible.
Case Illustration: Case 1: A 24-year-old female presented with weakness and right upper quadrant pain, blood pressure was 68/50 mmHg, and the heart rate was 35 bpm. The ECG showed a third-degree AV block with an episode of ventricular standstill. A transvenous pacemaker (TVP) was immediately implanted. Echocardiography showed a reduced left ventricular ejection fraction (LVEF) of 33% with global hypokinetic and increased Troponin T level. Due to no chronicity and other reversible causes, myocarditis was considered. Cardiac magnetic resonance imaging (CMR) showed mid-wall fibrosis at the basal and septal aspects of the right and left ventricle (LV), suggesting acute myocarditis. On day 7, the patient's condition improved with ECG showing intrinsic sinus rhythm. The patient was discharged and evaluation in outpatient visits showed improved LVEF and no symptom recurrence.
Case 2: A 30-year-old female presented with fever and weakness. Fever was observed 7 days prior and was diagnosed as typhoid fever. On the day of admission, the patient felt weak with a heart rate of 35 bpm. ECG showed a third-degree AV block and TVP was implanted. Due to high suspicion of myocarditis from symptoms, CMR was considered. TVP was removed despite intrinsic ECG still showing a second-degree AV block. The CMR shows a thin mid-wall fibrosis at inferoseptal LV with myocardial edema at basal, septal, and anterior LV, suggesting myocarditis. Steroid treatment was given, and the patient's condition improved. The patient was discharged with no symptom recurrence. ECG evaluation in outpatient visits showed sinus rhythm.
Conclusion: Patients presenting with HDAVB need scrutiny of the possible etiologies. Myocarditis should be considered after all other causes are excluded. In addition, since most conditions are reversible, cardiologists need to be mindful before planning invasive and expensive options such as permanent pacemaker implantation.

Keywords: Myocarditis, atrioventricular block, HDAVB, CMR, TVP

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Simultaneous AF Ablation and Renal Denervation: A Great Clinical Outcome for AF and Uncontrolled Hypertension

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Background: Atrial fibrillation (AF) ablation procedures alone in patients with AF and uncontrolled hypertension show high rates of atrial arrhythmia recurrence. Uncontrolled hypertension is one of the factors causing post-ablation AF recurrence. Renal denervation (RDN), apart from controlling blood pressure, is known to reduce the recurrence rate of AF by reducing afferent signals to the kidneys, which also reduces sympathetic signals to other organs, including the cardiac conduction system.

Case illustration: A 53-year-old man with palpitation and a documented AF and atrial flutter (AFL) during a three-day Holter monitoring. The total AF episode was 51 minutes. In addition, the patient also has uncontrolled hypertension and obesity. He underwent simultaneous radiofrequency catheter ablation for his AF—AFL and RDN for his uncontrolled hypertension. After one year of follow-up, there was no episode of AF recurrence during a seven-day Holter monitoring. Furthermore, the patient showed normal blood pressure during his follow-up without taking anti-hypertension medication.

Conclusion: The combination of radiofrequency catheter ablation for atrial fibrillation (AF) and renal denervation (RDN) in a patient with uncontrolled hypertension highlights the potential benefits of a combined therapeutic strategy in select patients.

Keywords: atrial fibrillation, hypertension, catheter ablation, renal denervation

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Arrhythmic Mitral Valve Prolapse with Features of Mitral Annular Disjunction and Myocardial Fibrosis

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Background: Mitral valve prolapse (MVP) is superior displacement ≥2 mm of any part of the mitral leaflet beyond the mitral annulus, measured echocardiographically in the parasternal or apical long axis views. In rare condition, It is associated with malignant ventricular arrhythmias (VA) and sudden cardiac death (SCD). Mitral annulus disjunction (MAD) is defined as mitral annular detachment from the basal left ventricular (LV) myocardium with an abnormal systolic excursion of the leaflet hinge point into the left atrium. MAD have been linked to increased arrhythmic risk. MAD >8.5 mm is a strong predictor of VA.
Case Ilustration: 36-years-old female came with complaints palpitation, dyspnea on effort, and episode of near syncope. From electrocardiogram (ECG) revealed sinus rhythm, QRS rate 75-85 bpm, multifocal PVC with RBBB patern. From holter ECG revealed sinus rhythm, good AV conduction, good chronotropic competence, multifocal benign infrequent PVC from posteromedial papillary muscle, and with 2 episodes of non-sustained VT. From echocardiography, mitral regurgitation ec prolapse AML-PML (A2-P2) ec degenerative with mitral annular disjunction (MAD) 11 mm, tricuspid regurgitation mild with low probability of PH. From Cardiac Magnetic Resonance (CMR), moderate MR ec AML-PML prolapse with mitral annular disjunction at PML (P1, P2, P3), moderate TR ec anterior tricuspid leaflet prolapse, with myocardial fibrosis.
Conclusion: We present case report, a young woman diagnosed with PVC predominant from posteromedial papillary muscle and MVP AML-PML with features of MAD and myocardial fibrosis assessed by electrocardiography, holter electrocardiography, echocardiography, and cardiac magnetic resonance (CMR). The current understanding of arrhythmogenesis in MVP involves the development of a substrate for arrhythmias (cardiac fibrosis) combined with a trigger for arrhythmias. Future prospective research is needed to further delineate optimal methods for SCD risk stratification and treatment

Keywords: Arrhythmic Mitral Valve Prolapse, Mitral Annular Disjunction

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Unveiling Cardiovascular Enigma: The Rare Coexistence of Wollf-Parkinson-White Syndrome and Left ventricular Non-Compacted Cardiomyopathy in Remote of Riau

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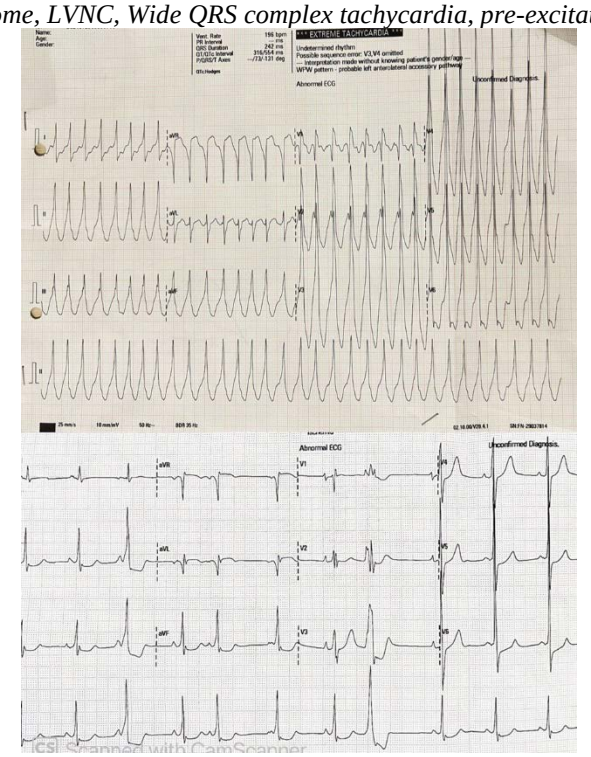
Background: Wide QRS complex tachyrcardia can arise from several conditions, including atrial fibrillation (AF) with pre-excitation or Wolff-Parkinson-White (WPW) syndrome. The challenge intensifies when the irregularity are hard to differentiate, making meticulous measurement vital for accurate diagnosis and appropriate initial management. Left ventricular non-compaction (LVNC) cardiomyopathy is a rare myocardial disorder characterized by prominent trabeculations and deep intertrabecular recesses. The coexistence of LVNC cardiomyopathy and WPW syndrome can complicate the clinical presentation and management.
Case Illustration: A 54-year-old man came to the emergency room with episodes of palpitations and near-syncope that had been occurring over the past six months. His physical examination revealed no abnormalities. The initial ECG indicated a slightly irregular wide QRS complex with a heart rate of 180-200 beats per minute. Despite administering amiodarone, the tachyarrhythmia did not resolve. Electrical cardioversion at 120 J successfully converted the ECG to a sinus rhythm, which showed increased QRS voltage and intermittent pre-excitation, indicative of a WPW type-A pattern. These findings suggest that the tachyarrhythmia was atrial fibrillation (AF) with pre-excitation. A transthoracic echocardiogram showed mild left ventricular dysfunction with an ejection fraction of 45% and left ventricular enlargement. Additionally, there was significant trabeculation and a non-compacted to compacted (NC:C) ratio of greater than 2, meeting the criteria for left ventricular non-compaction (LVNC) cardiomyopathy.
Conclusion: Accurate assessment and measurement of ECG irregularities are crucial to manage malignant arrhythmias early, potentially preventing sudden cardiac death due to more advanced arrhythmias. The combination of LVNC cardiomyopathy and AF with pre-excitation in this patient is an extremely rare situation. Electrophysiological studies at more sub-specialized centers are recommended to determine the causality of these conditions and guide appropriate treatment strategies.
Keywords: AF, WPW syndrome, LVNC, Wide QRS complex tachycardia, pre-excitation



Picture (above) shows Wide QRS complex tachycardia with HR 180-200 beats per minutes. Picture (below) show the ECG after converted to sinus rhythm, depicted increased QRS voltage and intermiten pre-excitation.

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Atrial Fibrilation Turned Malignant

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Background: Atrial fibrilation is the most commonly seen arrythmia. It does not directly cause haemodynamic upset, but in our case it turned malignant via an accessory pathway.

Case Illustration: A 71-year-old female was referred to our facility with symptoms of repeated dizziness, syncope and vomiting. During hospitalization the serial electrocardiogram (ECG) showed patterns of tachycardia-bradycardia syndrome, where bradycardia alternates with tachycardia, in this case, an atrial fibrilation (AF), indicating sick sinus syndrome (SSS). For AF patients with SSS, catheter ablation should be considered as it is associated with lower risks of AF recurrence, AF development and adverse outcomes including all-cause mortality, stroke and hospitalization of heart failure.(1) Therefor cryoablation was planned and done with no complication. On the next day, drowsiness, vomiting and pre-syncope was felt by the patient with an ECG showing ventricular tachycardia (VT). On the serial ecg (Figure 1.) we could see that there was a delta wave with a wide QRS and a short PR interval, indicating Wolff-Parkinsos-White (WPW) Syndrome. With this finding, radiofrequency ablation to terminate the accessory pathway was planned and followed by permanent pacemaker (PPM) implantation. The procedure was done without any complication and the goal to ablate the accessory pathway was achieved. After the PPM implantation, the ECG showed sinus rhythm with pacing. All the complaints were gone and the patient was able to be discharged from inpatient care.
Conclusion: Our case emphasizes the importance of precise diagnosis and a comprehensive treatment. Where a case of sick sinus syndrome with atrial fibrilation turned malignant due to an accessory pathway. Multiple ablation was done with different targets (cryoablation for pulmonary vein isolation and radiofrequency ablation for accessory pathway) coupled with PPM implantation for a thorough treatment of this complex case.

Keyword: Arrythmia, Wolff-Parkinsos-White (WPW) Syndrome, Sick Sinus Syndrome, Ablation.

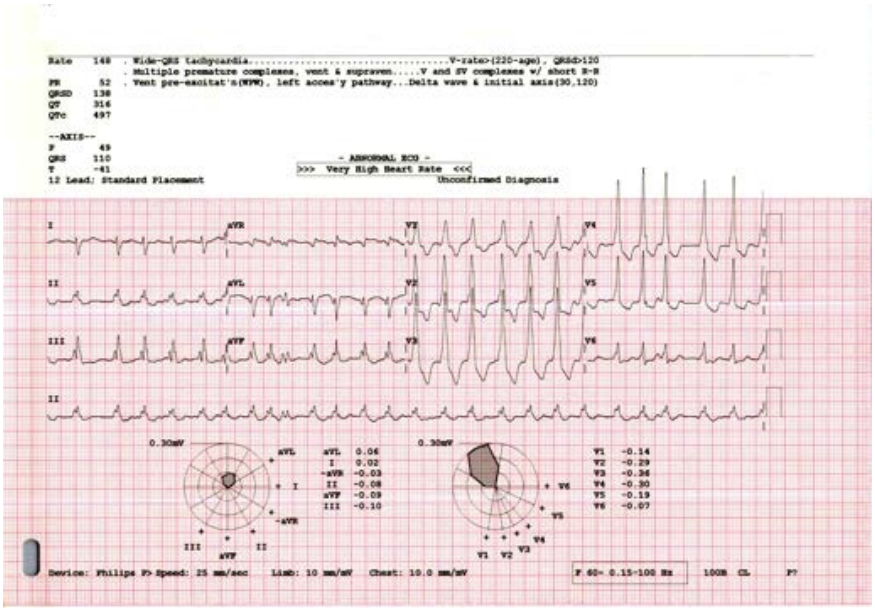

Figure 1. ECG showing tachycardia with Delta wave.

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Unveiling the Intersection: Cardiocerebral Infarction (CCI) When Acute Ischemic Stroke Meets Wellens Syndrome – A Rare Case Study

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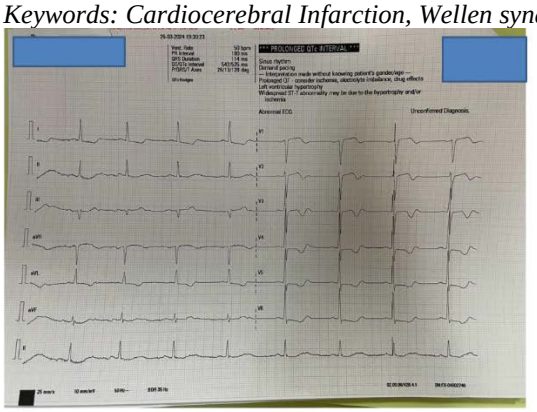
Background: Concurrent Cardio-Cerebral-Infarction(CCI) is a-rare-condition for which limited information is available in medical literature. Moreover, the ominous ECG findings of Wellens Syndrome (WS) and the sudden neurological deficits of acute stroke create urgent puzzles. We aim to present a challenging situation where the acute Ischemic-Stroke(IS) concurrent with ECG of-WS.

Case Illustration: A 47-year-old-male was brought to the Emergency-Department after experiencing sudden left-sided-weakness for 3.5 hours before admission. Physical examination revealed dysarthria, drooping on the left-side of his face, and left conjugate eye deviation (NIH-Stroke-Scale-score19). Examination of the-heart-revealed-regular-heart-sounds(S1S2) without-murmurs. However, the ECG showed Wellens-syndrome-type-2. Surprisingly, after further questioning, he had experienced typical chest pain lasting +/-10 minutes just before the onset of stroke. Serial hsTrop-T showed an increase from 37 to 693 ng/L. Non-contrast-head-CT-scan revealed a large-vessel-occlusion. The patient was diagnosed with simultaneous CCI due to WS-type-2 and IS. He received IV-TPA at a dose of 0.9mg/kgBB and was later intubated due to difficulty breathing and decreased consciousness. Our patient's characteristic is younger to those described in existing literature, which states that the average age-specific rates are 59 years old, with a predominantly male population (90%). Wellens syndrome is marked by specific ECG changes indicating critical stenosis of the left anterior descending (LAD) coronary artery. Patients typically present without pain, and cardiac enzymes may be normal or slightly elevated. However, these ECG patterns signify a high risk for impending large anterior wall acute myocardial infarction. Although representing a pre-infarction state, patients are at risk for extensive myocardial infarction and even death due to unstable coronary perfusion.

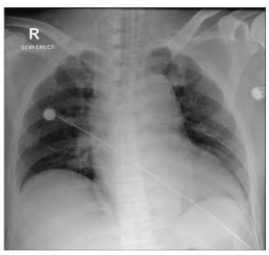
Our patient's presentation aligns with this chronology, but our facility's limitations prevent evaluation of LAD stenosis with coronary CT-angiography. Ibekwe et al, stated 4 major pathophysiologic mechanisms of CCI and the most likely underlying mechanism in our patient is due to cardio-embolic. While myocardial infarction and stroke share similar pathophysiology, their treatment options vary greatly. From several previous studies, proposed to administer IV-tPA(0.9mg/kgBB) then proceed to Percutaneous-Transluminal-Coronary-Angioplasty-and-Stenting (PTCAS) if indicated for ACS.

Conclusion: Cardio-cerebral-infarction is uncommon-and-rare with devastating-clinical-scenario. In addition, clinicians need to pay attention to—and neither-underestimate nor-ignore—the presence of ECG findings even-in-stroke.

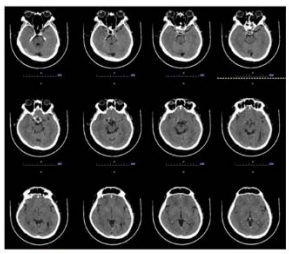
Keywords: Cardiocerebral Infarction, Wellen syndrome, Ischemic Stroke



Rhythm	Sinus
Heart rate	51x/m
axis	Normoaxis
P wave	Lead II (+) aVR (-), amp 1mV, 0.08s
PR segment	0.08s
QRS	0.08s
T wave	biphasic T waves (with initial positivity and terminal negativity) in V2 and V3; biphasic T waves in V4-6, lead I aVL
ST segment	No depression nor elevation
QTc	516ms
Wellens Syndrome type 2	



X-ray Thorax



Head CT Scan Non Contrast. Acute infarct of right frontal and temporal lobes with Hyperdense MCA sign

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Brugada Phenocopy (BrP) in a Patient with Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)

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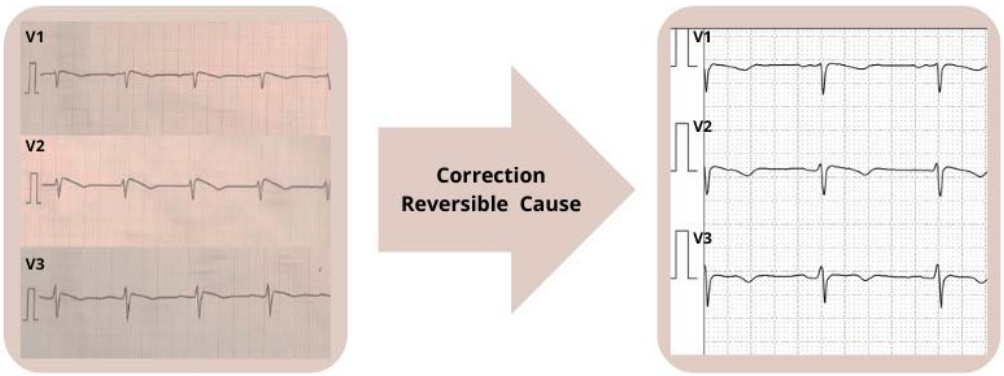
Background: Brugada phenocopies (BrP) are clinical conditions that have different causes from real congenital Brugada syndrome with one of the signs being obtained from the electrocardiogram (ECG). The presence of Brugada ECG pattern in patients with Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) who suffer syncope symptoms may suggest the presence of cardiac abnormalities. Anamnesis, physical examination, and further diagnostic tests may be performed to determine the underlying cause of syncope.
Case Illustration: A 69-year-old male with a medical history of hypertension and smoking arrived with a syncope. The patient's vital signs were within the normal range and there were not any signs of any neurological impairment. The patient's diagnosis of SIADH was established based on the presence of hyponatremia (Na 118 mmol/L), a hyperosmolar state, and normal blood volume. Upon arrival, a twelve-lead electrocardiogram revealed ST-segment abnormalities indicative of a Brugada ECG pattern. No instances of ventricular arrhythmia were seen over the whole 24-hour holter monitoring period. The coronary angiography showed no anomalies in the coronary arteries. The electrocardiogram (ECG) demonstrated the restoration of normal ST-elevations and the removal of the Brugada ECG pattern following the correction of hyponatremia (Na 130 mmol/L). Following a three-month period of observation, the patient, who exhibited a normal sodium level, did not experience any symptoms of syncope.
Conclusion: Severe hyponatremia in patients with Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) can lead to syncope and a Brugada phenocopy. Syncope presenting with a Brugada-like ECG pattern may be indicative of both cardiac and non-cardiac etiologies. A thorough diagnostic approach is essential to exclude cardiac causes. The management of syncope in SIADH patients exhibiting a Brugada pattern ECG should be based on the underlying cause.

Keyword: Brugada phenocopies, Syncope, Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)

FIGURE 1. Electrocardigram (ECG) changes in ST Segment



ECG at admission, revealing ST-segment abnormalities that resemble a Brugada ECG pattern, with a serum sodium level of 118 mmol/L.

ECG reveals the resolution of ST-segment elevations when the serum sodium level content reaches 130 mmol/L.

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Triple atrial tachyarrhythmias in a patient with atrial septal defect post closure

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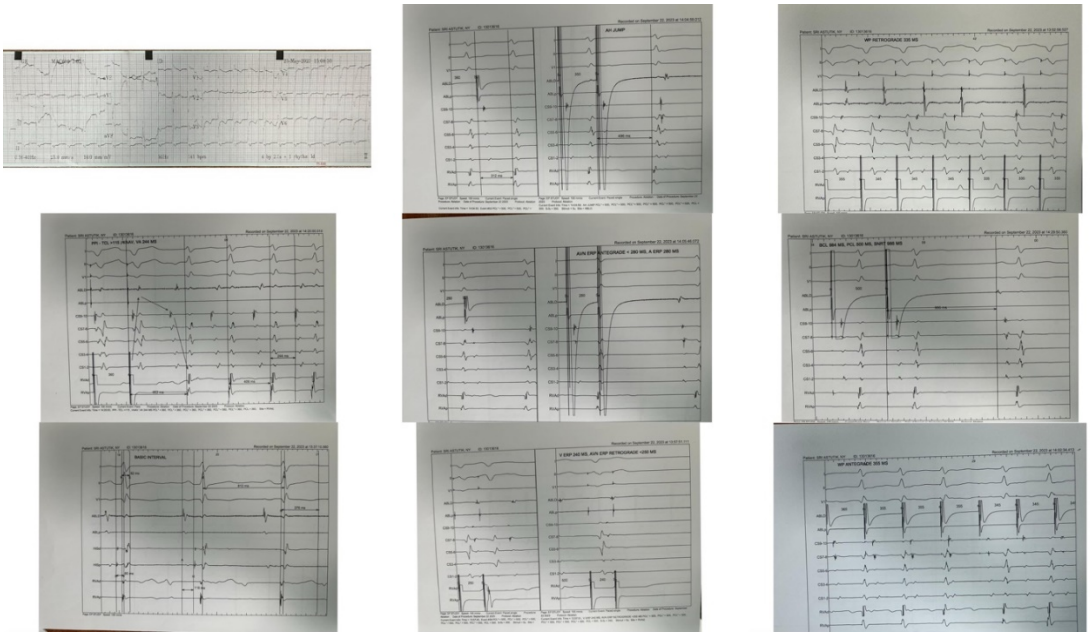
Background: Atrial Septal Defect (ASD) is the most common congenital heart disease in adults. Atrial arrhythmias are common in ASD patient, either before or after ASD closure, and they may occur concomitantly.

Case Illustration: A 51-year old woman with a history of transcatheter ASD closure by Amplatzer Septal Occluder (ASO) procedure in 2005 was referred with intermittent palpitations. Previously, she was admitted to the emergency room with a diagnosis of supraventricular tachycardia (SVT) and was given bisoprolol 1x2.5 mg. The patient denied several cardiac risk factors, but she was classified as overweight (BMI 29.4 kg/m2). The vital signs were normal, except for the irregular and tends to be high heart rate (97 bpm). Holter monitoring showed several episodes of SVT. In September 2023, Electrophysiology study (EPS) procedure revealed atrial tachycardia (AT) and dual AV Node physiology. Thus, EPS with 3D activation mapping ablation was carried out in February 2024. At first, reverse typical atrial flutter was acquired because entrainment pacing revealed PPI-TCL -2ms at the CTI area, then ablation was performed and converted into sinus rhythm. During observation period, isoproterenol was injected and atrial burst pacing was given. Another atrial tachyarrhythmia was induced with varying VA interval and earliest activation at CS 9-10, suggesting an AT thus ablation was performed at the CS ostium. Atrial entrainment pacing was given and induced the third atrial tachyarrhythmia. Diagnostic maneuvers are resulting no reset, VAV pattern, also VA interval 18ms, indicating a typical AVNRT, hence ablation at slow pathway was performed. Supraventricular arrhythmia may occur in ASD patients due to structural and electrical remodeling of the atrium. The risk of SVT also increases in patients who undergo transcatheter ASD closure procedures such as ASO.

Conclusion: Diagnosing and treating arrhythmia in a patient with ASD is critical, especially when multiple mechanisms of tachyarrhythmia are coexist. 3D activation mapping catheter ablation is a rational and effective strategy to meliorate symptoms, reduce morbidity, also reduce the need for long-term anti-arrhythmic therapy and their associated side-effects.

Keywords: Atrial Septal Defect, Multiple Atrial Tachyarrhythmias, Catheter ablation



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Clinically Suspected Acute Right Ventricular Fulminant Dengue Myocarditis Complicating with Dual Lethal Arrhythmias: A Rare Case Report

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Background: Acute right ventricular (RV) myocarditis is rare, comprising only 18% of myocarditis cases. Despite being relatively infrequent at 12.4%, dengue-induced myocarditis has a high mortality risk of 26.4%. This report presents a novel case of acute fulminant RV myocarditis due to severe dengue infection, complicated by dual electrical disturbances: complete heart block and ventricular tachycardia.

Case Illustration: A 49-year-old male patient was referred to our hospital with a temporary pacemaker due to a complete heart block. He had a history of recurrent syncope over three days and a fever five days before admission. Initial electrocardiography showed a total atrioventricular (AV) nodal block progressing to a high-degree AV block with a left bundle branch block, indicating an infra-Hisian block. Laboratory findings included thrombocytopenia, elevated troponin, high creatinine, increased liver transaminases, and a positive dengue non-structural protein 1 test, confirming a diagnosis of dengue infection. Echocardiography showed normal all chamber dimensions, reduced RV systolic function (TAPSE: 11 mm, FAC: 22%), normal left ventricular (LV) systolic function (EF: 50%), hypokinetic RV free wall, dyskinetic intraventricular septum, normal anatomy and function of all heart valves, and low probability of pulmonary hypertension, indicating an acute RV systolic dysfunction without other possible etiologies. Coronary angiography revealed normal coronary anatomy. An endomyocardial biopsy was deferred due to severe thrombocytopenia. On the third day, the patient's condition worsened, developing cardiogenic shock and LV systolic dysfunction (EF: 35%). He subsequently experienced a seizure and slow ventricular tachycardia originating from the right coronary cusp, followed by cardiac arrest. The patient's family claimed not to resuscitate the patient. Furthermore, the patient died shortly after.

Conclusion: This case underscores the critical need for prompt diagnosis and aggressive management of clinically suspected acute fulminant RV myocarditis because complications can rapidly progress to LV systolic dysfunction, leading to cardiogenic shock and sudden cardiac death.

Keywords: myocarditis, right ventricle, complete heart block, ventricular tachycardia

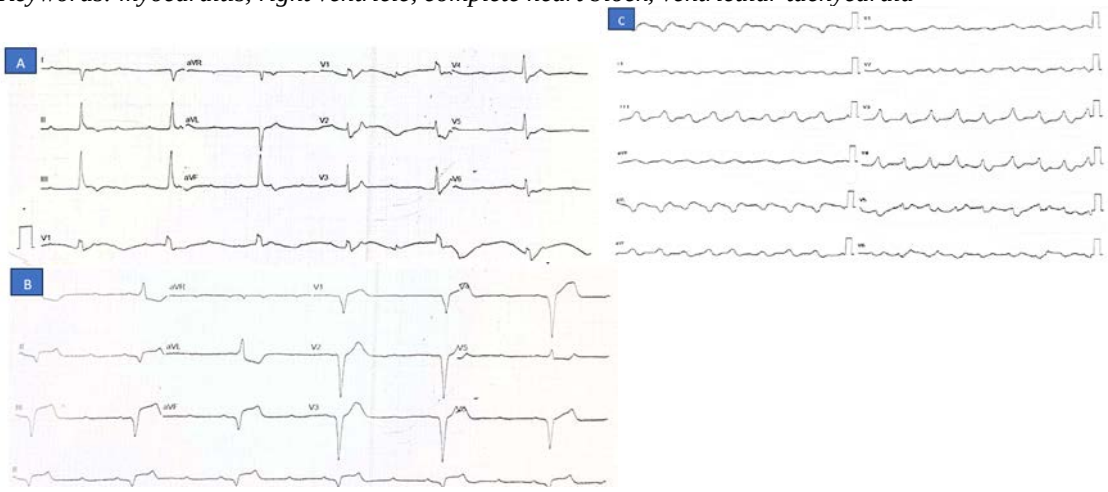

Figure 1. The electrocardiography showed a total atrioventricular (AV) nodal block **(A),** high degree AV nodal block 3:1 with left bundle branch block **(B),** and slow ventricular tachycardia **(C).**

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Symptomatic Bradycardia in Young Adult: A concomitant BrS, AVND & SND case?

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[2]*Heartology Cardiovascular Hospital, Jakarta, Indonesia*

Background: Brugada Syndrome (BrS) is an autosomal-dominant inherited arrhythmic disorder caused by *SCN5A* mutations which is characterized by coved-type ST elevation with inverted T wave in the right precordial leads without any structural cardiac abnormalities. Patients with BrS are at risk for sudden cardiac death (SCD) due to ventricular fibrillation (VF) or ventricular tachycardia (VT). However, *SCN5A* mutations are also implied in long-QT-syndrome (LQTS) type 3, progressive cardiac conduction disease (first degree to complete AV block), sick sinus syndrome, or combinations of these.
Case Illustration: A female, 25 years old, complained slow heart pulse and several episodes of presyncope since 2 months before admission. Family history of sinus node dysfunction (SND) and pacemaker implantation was confirmed. Physical examination and echocardiography showed normal cardiac findings. ECG tracing through *Apple iWatch* showed sinus bradycardia with episode of sinus pause. ECG showed sinus bradycardia with junctional escape beats. Patient was then referred for Holter ECG dan pacemaker implantation. Holter ECG showed a Brugada pattern type 1, sinus bradycardia with episode of sinus pauses and junctional escape beats. EP study was then planned to look for possibility of ventricular arrhythmia. EP study showed sinus node dysfunction (SND), AV node dysfunction (AVND) and several episodes of non-sustained VT. Patient was planned for dual chamber pacemaker or ICD implantation.
Conclusion: *SCN5A* mutations are associated with BrS, AV node dysfunction and sick sinus syndrome. The diagnosis of BrS can be confirmed by ECG, Ajmaline test and genetic testing. Holter ECG and EP study are needed for evaluating AV node dan SA node function in BrS patient. Device implantation, such as pacemaker and ICD, is a therapeutic option for this patient.

Keywords: Brugada Syndrome, Sinus Node Dysfunction, Pacemaker, ICD

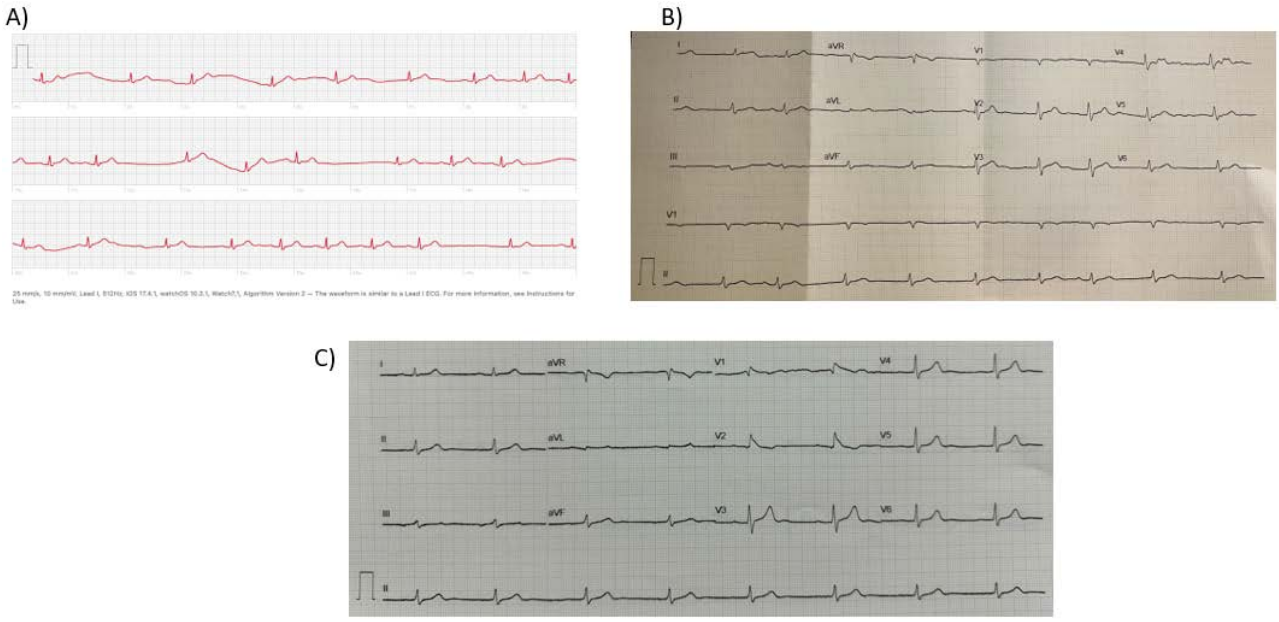

Figure 1. Patient ECGs. A) *Apple iWatch* ECG tracing showed sinus bradycardia with episode of sinus pause, B) First ECG showed sinus bradycardia with junctional escape beats, C) Secondary ECG (V1-V2 leads located in 2nd ICS) showed sinus bradycardia and Brugada pattern type 1

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Promising Result of The 2D Mapping Radiofrequency Cathether Ablation for Sustained Ventricular Tachycardia with History of Cardiac Arrest and Congestive Heart Failure : A Case Report

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Background : Sustained Ventricular Tachycardia (VT) with structural heart disease such as congestive heart failure ideally undergoes radiofrequency ablation with 3D mapping and placement of implantable cardioverter-defibrillator (ICD). However, due to healthcare financial constraints and limited 3D mapping device in several hospital, initial ablation management with 2D mapping is considered to improve the patient condition.
Case Presentation : A 45-year-old woman suddenly experienced cardiac arrest at outpatient clinic. She had previously complained chest pain on left side radiating to her back, palpitations, and cold sweats. The patient was administered intravenous amiodarone 150 mg bolus, followed by two cardioversions. Sinus rhythm was restored and maintaint with intravenous lidocaine pump at 0.5 mg/hour and oral bisoprolol 5 mg. Considering the patient's history of frequent symptomatic episodes leading to cardiac arrest and echocardiography finding show left ventricular dilatation and reduced ejection fraction (31%), she was scheduled for catheter ablation using 2D mapping device. The VT location found in the right ventricular outflow tract (RVOT) with earliest activation at 35 ms. Ablation was performed with 40 watts of electrical activity and temperature at 60°C for 60 seconds, terminating the VT episode. Induction attempts to provoke VT with atropine sulfate 0.5 mg and adrenaline 5 micrograms did not re-induce VT. Observation after 10 minutes also did not show VT recurrence. Two months post-ablation, the patient reported no palpitations or cardiac arrest episodes. However, dyspnea sometimes persisted which was related to heart failure symptom.
Conclusion : Radiofrequency ablation with 2D mapping in patients with sustained VT and heart failure can be considered as adjunctive therapy in condition of device limitation and healthcare cost expenditure. However, this management don’t replace the primary management, which is radiofrequency with 3D mapping and ICD placement

Keywords : Sustained VT, RVOT, Congestive Heart Failure, Radiofrequency Ablation, 2D Mapping, Sudden Cardiac Arrest

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Left bundle branch area pacing in patient with reduced left ventricular ejection fraction: a dazzling alternative for cardiac resynchronization therapy

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Background: In patients with impaired LV function and wide QRS, Cardiac Resynchronization Therapy via biventricular pacing (BiVP) has shown significant benefit. However, BiVP has several shortcomings, including high pacing thresholds, phrenic nerve stimulation, difficulties in device implantation, and potential nonresponders. Left bundle branch area pacing (LBBAP) has recently developed as a novel technique for conduction system pacing that has lower pacing threshold, shorter QRS duration, and is easier technically. In this report, we present a case of concurrent symptomatic sinus node dysfunction and heart failure with reduced ejection fraction with wide QRS undergoing LBBAP.

Case Illustration: A 81-year-old male presented to the emergency room with worsening dyspnea and near syncope since 1 day ago. He had experienced these complaints for 6 months. Dyspnea was still aggravated by exertion and while lying supine despite optimal guideline-directed medical therapy. Vital signs on admissIon were: blood pressure 168/79 mmHg, heart rate 57 bpm, respiratory rate 26 times per minute, O2 saturation was 96%. Rales was found on auscultation. Electrocardiogram revealed junctional rhythm with RBBB morphology and QRS duration of 160 ms. Laboratory results were unremarkable. Echocardiography showed eccentric LV hypertrophy with regional wall motion abnormalities, reduced LV ejection fraction (32%), and grade 1 diastolic dysfunction. He was scheduled for temporary pacemaker implantation followed by DDDR pacemaker with LBBAP several days after. During procedure, LVAT was 84 ms, pacing threshold was 0.75 V/0.4 ms. Electrocardiogram after procedure showed pacing rhythm with QRS shortening to 100 ms. The patient was then discharged, and on one-month follow-up, no complications were found and he reported improvement in physical capacity and no episodes of near syncope.

Conclusion : Although BiVP is a well-established method of cardiac resynchronization therapy, LBBAP is emerging as a reliable and safe way to achieve physiologic conduction system pacing with shorter QRS duration, simpler technique, lower cost, and particularly, symptom improvement. LBBAP criteria will keep evolving as more research is conducted and long-term safety and efficacy data are gathered.

Keywords: cardiac resynchronization therapy, heart failure, left bundle branch area pacing.

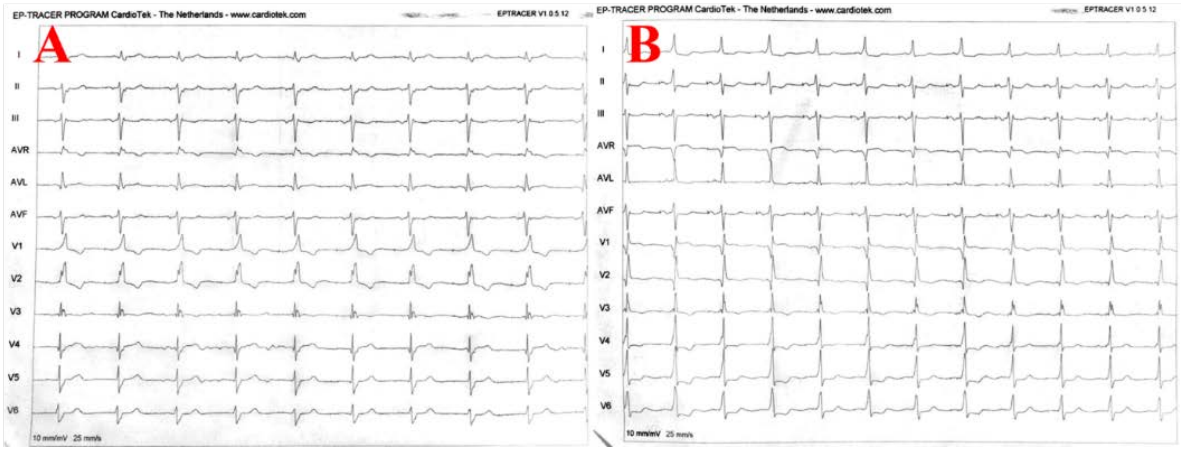

Figure 1. Patient's electrocardiogram before (A) and after DDDR LBBAP implantation (B).

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"Case Series Analysis: Atrial Fibrillation Ablation in Normal vs. Enlarged Left Atrium"

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Background: Atrial fibrillation (AF) is a prevalent cardiac rhythm disorder, with rising global incidence. AF can lead to serious complications such as congestive heart failure, myocardial infarction, and thromboembolism. Catheter ablation, a minimally invasive procedure, aims to eliminate AF triggers but its success varies based on factors like left atrium (LA) size. Enlarged LA (LAE) is linked to poorer ablation outcomes, yet the mechanisms behind this relationship remain unclear.

Case Illustration: This study presents a case series analysis of two patients undergoing catheter ablation for AF, comparing outcomes and complications between those with normal LA size and those with LAE. The LA geometry and pulmonary veins were mapped using the ENSITE 3D mapping system. Pulmonary vein isolation (PVI) was performed, and isolation was confirmed through entrance and exit block tests.

Conclusion: The outcomes of AF ablation can significantly differ based on LA size. Patients with LAE face more complex procedures and higher recurrence risks. However, with meticulous management and follow-up, favorable outcomes are achievable. Further research is essential to understand the LA size-AF ablation outcome relationship and improve procedural success rates.

Keywords : Atrial Fibrillation (AF), Left Atrial Enlargement (LAE), Catheter Ablation, Pulmonary Vein Isolation (PVI)

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Lidocaine and Amiodarone as a Challenging Treatment for Patient 69 Years Old with Persistent Ventricular Tachycardia, Renal acute Failure and Hyponatremia: A Case Report

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Background: Significant in-hospital and 6-month mortality is linked to persistent VT, requiring immediate stabilization and critical secondary prevention. While the cornerstones of emergency care are ACLS and antiarrhythmic medications (AAD), early referral is recommended for the implantation of an AICD, catheter ablation, and/or sympathetic cardiac denervation. Amiodarone and/or lidocaine can be given as boluses or as an infusion in the case of persistent or recurring VT (including VT storm). This approach can prevent the requirement for admission to intensive care by stabilizing the patient for transfer (if necessary) and preventing the need for escalation to intubation and sedation for central sympathetic blocking.

Case Illustrations: A 69 year old man with complaints of chest pain and shortness of breath came to the emergency department. 8 hours before coming to the hospital, the patient felt tightness and chest palpitations. During triage, the patient had a heart rate of 172 bpm with an ECG showing monomorphic ventricular tachycardia (Fig. 1). Examination showed hyponatremia and negative troponin electrolyte levels. After administering lidocaine bolus 1 mg/minute slow bolus followed by lidocaine 0.5 mg/kg slow bolus, CPG 1x75 mg, nitrocaf 2x2.5 mg, Furosemide 1x60 mg IV, spironolactone 1x25 mg, the patient still had monomorphic VT again and then the patient was pro-ICU. After that, a slow bolus of amiodarone infusion of 1 amp is given in 2 ampoules 10 minutes apart. Sustained monomorphic VT was observed, prompting administration of a 3rd bolus of amiodarone 150 mg IV followed by drip amiodarone 300 mg in 6 hours. 60 minutes passed the patient's sinus rhythm returned to normal. On the 4th day of treatment, the ECG showed VT again, 1 ampoule of lidocaine was given, slow bolus 0.5 mg IV digoxin, and 50 mg PO metoprolol, then maintenance lidocaine drip was given 1 mg/minute – 60 mg/hour until the 7th day. On day 8, the EKG began to show sinus rhythm.

Conclusions: Amiodarone and lidocaine may be administered as infusions in addition to boluses to achieve patient stabilization in the emergency setting

Keyword: Persistent Ventricular Tachycardia, Amiodarone, Lidocaine, Metoprolol, Acure Renal Failure

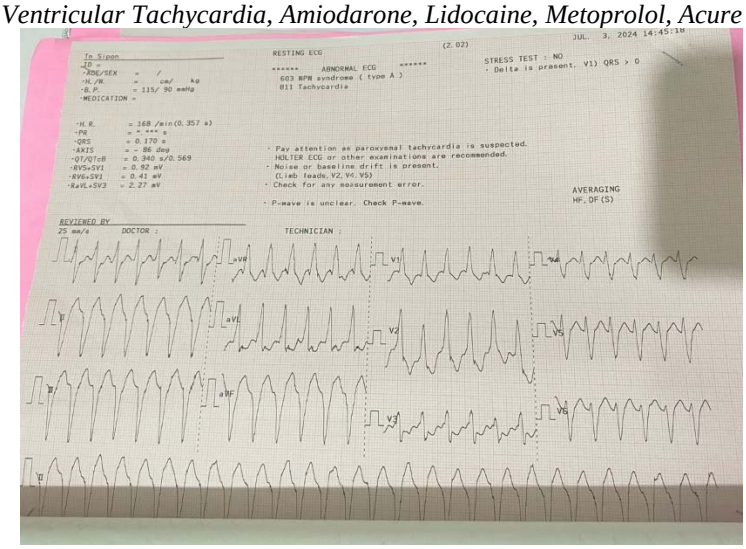

Figure 1

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Impostor of Wide Complex Tachycardia: The Importance of Sine Wave Morphology

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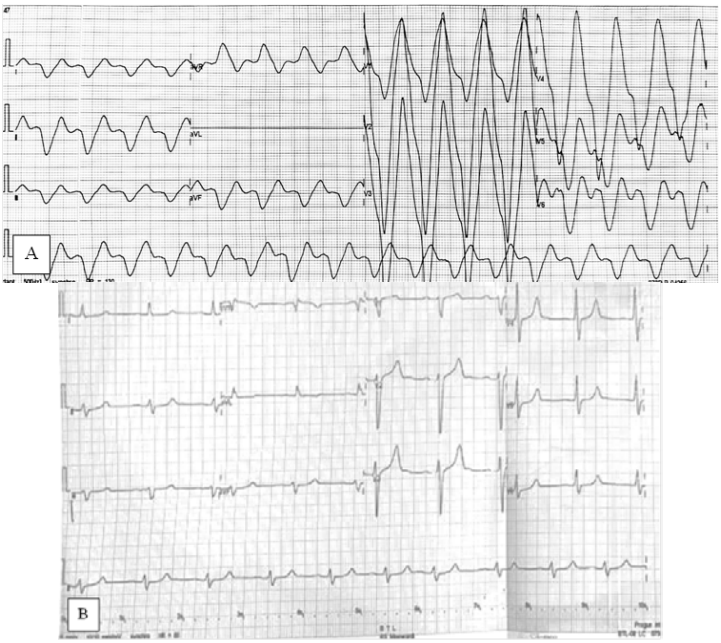
Background: Wide Complex Tachycardia (WCT) is an abnormal heart rhythm with numerous possible causes, including different types of arrhythmias, structural heart diseases, electrolyte imbalances, and metabolic disturbances. Hyperkalemia is one of the conditions that cause WCT and frequently leads to cardiac emergencies involving life threatening arrhythmias.
Case Illustration: A 60-year-old man with history of hepatic cirrhosis, chronic kidney disease, chronic hepatitis B, and type-2 DM, who routinely underwent hemodialysis, came to the ER with a chief complaint of altered consciousness for last three days. Patient had no previous cardiac diseases and family history of sudden cardiac death. Physical examination showed GCS E3V2M4, BP 90/60 mmHg, HR 110 bpm, RR 22 x/min, SpO2 98%, and dilated heart borders. The patient was diagnosed with hepatic encephalopathy, decompensated hepatic cirrhosis, chronic hepatitis B, diabetic kidney disease, and type-2 DM. ECG showed WCT (QRSd 400 ms) with HR 110 bpm, in which Slow Ventricular Tachycardia (VT) was suspected, several synchronized cardioversion was performed but failed to convert the WCT. The ER team quickly made discussion with Cardiovascular Departement and sine wave ECG morphology was established. Electrolyte laboratory test confirmed hyperkalemia condition (7.2 mEq/L), then patient directly treated to lower serum potassium level. The ECG then converted to sinus rhythm. This case showed sine wave morphology that mimics slow VT by affecting SA node and causing a junctional rhythm with widening QRS complex. Higher levels of extracellular potassium decrease heart muscle's excitability, affecting both pacemaker cells and conducting tissues. As hyperkalemia worsens, it increasingly inhibits impulse by SA node and diminishes conduction through AV node and his-purkinje system.
Conclusion: Hyperkalemia is one of possible causes leading to severe cardiac arrhythmias. Visual recognition of sine wave morphology will often be confused with slow VT in the emergency setting, thus this case highlights the importance of accuracy and foresight in distinguishing slow VT and sine wave ECG of hyperkalemia by correlating the ECG with patient's clinical conditions and medical history.
Keywords: *Hyperkalemia, Sine Wave, Slow VT, Wide Complex Tachycardia*



Convert Rhythm in BRASH Syndrome: Breaking The Bradycardia Vicious Cycle Beyond ACLS Algorithm

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Background: Bradycardia, renal failure, atrioventricular nodal blockade, shock, and hyperkalemia (BRASH) syndrome remains a rare case with a prevalence of 0.04% in the present study carrying a mortality of 5.7%. This case tries to report bradycardia as a result of CCB routine use and hyperkalemia due to renal failure and how to treat it not as bradycardia in common.
Case Illustration: An 84-year-old woman came to ED with shortness of breath and general weakness. She is a patient with polypharmacy for her comorbidities. On arrival, her respiratory rate was 22 breaths/minute with SpO2 was 99% on room air, heart rate was 34 bpm, and blood pressure was 84/53 mmHg. Her laboratory examination showed hyperkalemia, azotemia, hypercoagulopathy. She had pulmonary edema and minimal bilateral pleural effusion with concentric LVH, diastolic dysfunction grade I, and normal EF (81%). A temporary pacemaker could not be inserted because the facilities do not yet support it.
For routine medication, she consumed diltiazem, candesartan, spironolactone, clopidogrel, flunarizine, and other medications for diabetic mellitus, dyspepsia, dyslipidemia, and uremic nephropathy. All of these medications were stopped when the patient was admitted. Atropine was given as emergency medication. This patient got glucose-insulin protocol, Ca polystyrene sulfonate, and furosemide also norepinephrine and dopamine. After two days in the intensive unit, her laboratory results were getting better and it was followed with converted rhythm to AFSVR and on the fourth day it became a normal sinus rhythm with normotension. Treating bradycardia with atropine and cardiac pacing only might fail in BRASH syndrome with severe shock.
Conclusion: Management of BRASH syndrome is not only related to treating bradycardia but also includes lowering potassium levels, improving kidney function, and treating the underlying cause.

Keywords: bradycardia, BRASH syndrome, cardiogenic shock, hyperkalemia, renal failure

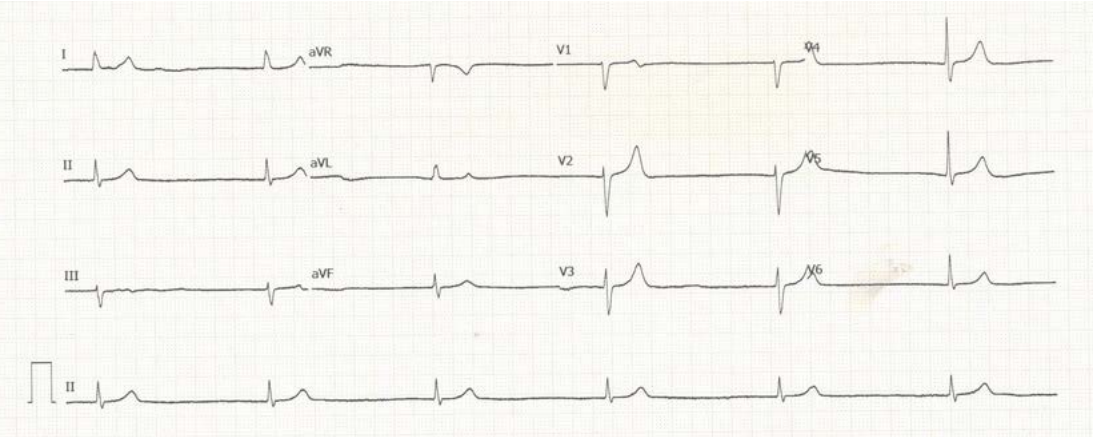

Figure 1. Junctional rhythm appeared in ECG of the patient at admission

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Combination Therapy for AF Ablation: PVI and CFAE Ablation

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Background: Ablation strategies targeting complex fractionated atrial electrograms (CFAE) in atrial fibrillation (AF) remain controversial. We present serial ablation cases involving persistent AF patients treated with a combination of Pulmonary Vein Isolation (PVI) and CFAE ablation.
Case Illustration:
1.**Patient 1:** A 65-year-old man with persistent AF underwent 3D ablation. He has a history of coronary artery bypass graft and mitral valve repair. Despite PVI and posterior box isolation, cardioversion failed. Subsequent CFAE ablation at specific sites in the left atrium successfully restored sinus rhythm.
2.**Patient 2:** A 53-year-old woman with heart failure (EF 40%) and persistent AF received PVI. Induced atrial flutter was terminated by cavotricuspid isthmus (CTI) ablation. CFAE mapping and ablation at the left atrium (LA) prevented AF recurrence. Furthermore, the LV ejection fraction was normalized during one year of follow-up.
Conclusion: Combining CFAE ablation with PVI shows short and long-term benefits, including successful AF termination and sustained sinus rhythm.

Keyword : Atrial Fibrillation, Complex Fractionated Atrial Electrograms, Pulmonary Vein Isolation

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Beyond Coronary Arteries: Painful Left Bundle Branch Block Syndrome, an Uncommon Cause of Chest Pain

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Background: Chest pain syndrome associated with intermittent left bundle branch block (LBBB) in the absence of myocardial ischemia has been described and later labeled “painful LBBB syndrome.” The mechanisms and treatment options of painful LBBB syndrome are unclear. With faster heart rates, an impulse may reach left ventricular myocytes during the refractory period, allowing proper ventricular contraction to start in the right before the left ventricle inducing transient dyssynchrony, typical of LBBB.
Case Illustration: A 54-year-old man was referred to Kariadi Hospital due to severe chest pain that he experienced during an activity four hours prior to admission. Chest pain felt like heavy pressure with a duration of more than 30 minutes and didn't resolve with rest. An ECG from the previous hospital during a chest pain episode showed sinus rhythm, a heart rate of 95 bpm, a leftward axis with LBBB, and an anterior S/T ratio of 1.8. The physical examination was within normal limits. During an episode of chest pain, we performed an ECG, which revealed sinus rhythm, a heart rate of 80 bpm, a leftward axis with a LBBB, and an anterior S/T ratio of 1.4. We decided to perform coronary angiography, and the result was a normal coronary artery. We treated the patient with 2.5 mg of bisoprolol and moved him to the ward for monitoring. A repeat ECG after resolution of the chest pain showed resolution of LBBB morphology, sinus rhythm with a heart rate of 70 bpm, a normal axis, and an inverted T wave present in the precordial lead. Chest pain improved, and the patient was discharged with bisoprolol therapy.
Conclusion: Painful left bundle branch block syndrome is an uncommon cause of angina and should always be considered without any evidence of coronary artery disease or cardiomyopathies that can explain the symptoms. There are no defined management guidelines, however several therapy strategies have shown unsatisfactory results.

Keywords: chest pain, LBBB, normal coronary artery

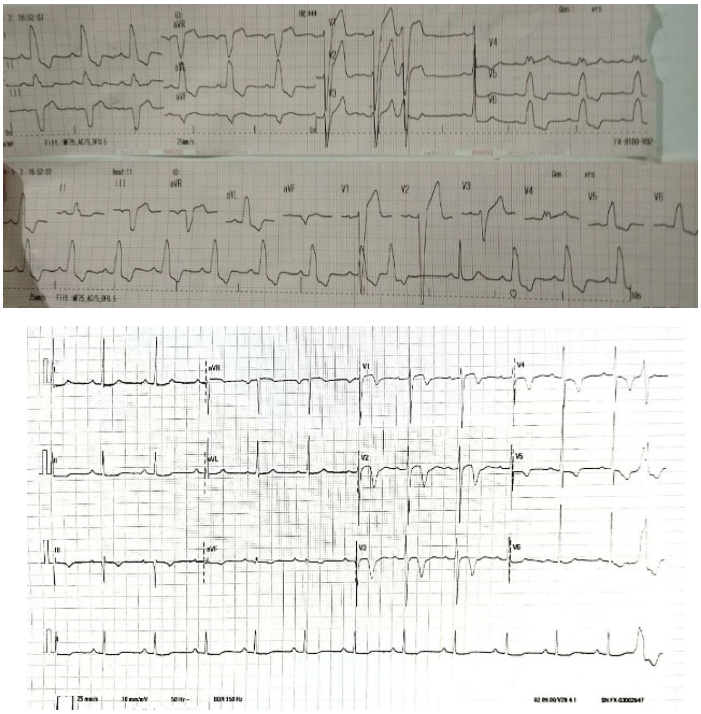

Figure 1. ECG in ER Kariadi Hospital during an episode of chest pain and Repeat ECG after resolution of the chest pain

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Role of Electrophysiology Study in Unexplained Syncope with Bradycardia Episode in Brugada Type I ECG Pattern : A Case Report

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Background: Brugada syndrome had a significant challenges for risk stratification and management despite in symptomatic or asymptomatic patients.
Case Illustration: A 51 years old woman was admitted to emergency department due to sudden collapse during her routine exercise. There was no chest pain,seizure, nor palpitation before syncope. She reported this as first episode ever. There was no sudden death in familial history. Her vital sign showed normal blood pressure with low heart rate of 50 beat per minutes. Physical examination was normal.
Electrocardiography showed sinus bradycardia, QRS rate 44bpm, normoaxis, with coved ST-T elevation in V1-V3 suggesting Type I Brugada ECG pattern and the holter monitoring showed chronotropic incompetence with sinus pause (2530ms) with infrequent PAC. Thyroid function was normal.
Despite lack of genetic testing modality, the patient underwent electrophysiology study. Result showed abnormal SA node intrinsic function, abnormal atrial conduction, abnormal AV conduction infra His with HV interval of 84 ms, and paroxysmal AF. She was planned later for double chamber permanent pacemaker implantation.
Conclusion: Spontaneous type I ECG Brugada pattern had associated with higher arrythmia event. Genetic testing of SCN5A gene remain as class Ic recommendation due to latest guideline from ESC. Electrophysiology studies were controversial at the moment (class IIb), but multicentre and some prospective study showed that induction of sustained VA during EPS associate with higher risk of future VA events.

Keyword : Brugada ECG pattern,Electrophysiology study,Syncope

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The first implantation of Subcutaneous Implantable Cardioverter Defibrillator (S-ICD) in Indonesia in a patient with brugada syndrome

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Background: Brugada syndrome (BrS) is an inherited cardiac disorder characterized by specific electrocardiographic (ECG) patterns and an increased risk of ventricular fibrillation and sudden cardiac death (SCD). In this patient population, an implantable cardioverter defibrillator (ICD) is implanted mainly for the defibrillator function but not for pacing purposes. The subcutaneous implantable cardioverter defibrillator (S-ICD) represents a major advance in the care of patients who have an indication for an internal cardiac defibrillator without pacing indications.

Case illustration: We present a case of 47-year-old male presented with no symptoms but had an irregular heart rhythm noted during a workplace medical check-up. The patient had no history of palpitations or syncope. A family history revealed a sibling's sudden unexplained death. Baseline ECG showed a type-2 Brugada pattern. Therefore, a flecainide provocation test was performed and converted the ECG to a type-1 Brugada pattern. An electrophysiology study was performed to better stratify the arrhythmic risk, and two extra stimuli could easily induce ventricular fibrillation (VF). For this reason, we successfully implanted a S-ICD.

Conclusion: Early diagnosis and appropriate intervention are critical in managing Brugada syndrome to prevent sudden cardiac death. In young patients with defibrillator indication but no pacing indication, a S-ICD is an effective method of preventing sudden cardiac death.

Keywords: Brugada syndrome, ventricular fibrillation, subcutaneous implantable cardioverter-defibrillator

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Contemporary approach for typical atrial flutter radiofrequency ablation to reduce radiation dose

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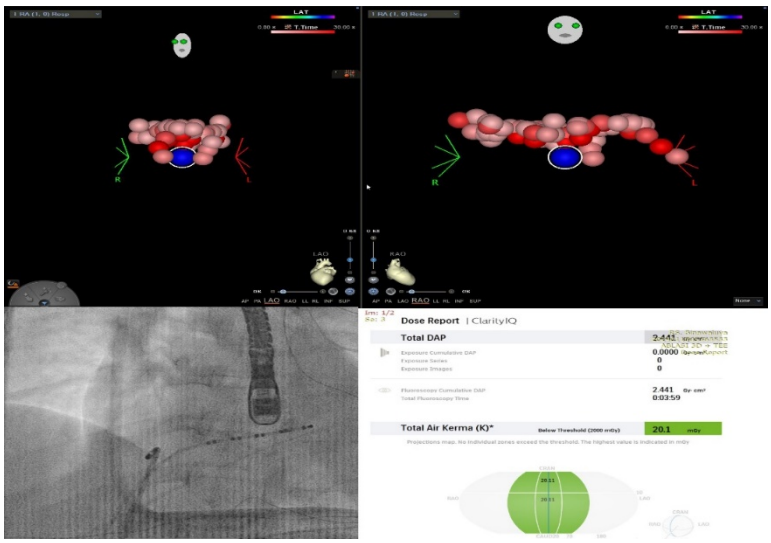
Background: Catheter ablation was a cornerstone in modern arrhythmia management. Fluoroscopy enabled interventional electrophysiologist to gain profound catheter movement and develop specific ablation approaches. However, the application of X-ray technologies imposes serious health risks to patients and operators. We present a case of atrial flutter ablation to offer valuable insights of very low dose radiation in ablation procedure.
Case Illustration: A 37-year-old man with history of hypertension, dyslipidemia, and insomnia presented to the outpatient clinic at Binawaluya Cardiovascular Center with intermittent palpitation and lightheadedness for a month. 12-lead ECG showed typical CTI-dependent atrial flutter with variable block 2:1-4:1 (atrial rate 215 bpm, mean HR 90 bpm), RAD, RVH. The CXR was normal. Transthoracic echocardiogram showed normal biventricular function without structural abnormality. CT coronary and LA-graphy showed no LA/LAA thrombus and normal coronary artery. Hematology, basic metabolic, liver, and renal function panel were normal. Edoxaban 60mg qD, amiodaron 200mg TID, and rosuvastatin 20mg qD were prescribed and planned for elective cardioversion followed by radiofrequency catheter ablation (RFA) on the next 3 weeks. After converted to sinus rhythm by cardioversion, the 3D RFA procedure was done on the next day. Decapolar CS catheter was inserted and THERMOCOOL SMARTTOUCH™ 8Fr RFA catheter was placed at cavotricuspid isthmus (CTI) guided with CARTO™ 3 3D tracing system and intermittent low frame-rate fluoroscopy. Continuous RFA (35Watt) was performed at the central CTI resulting in a permanent bidirectional block. RV pacing showed concentric atrial activation. RA programmed pacing revealed AV jump with non-inducible AVNRT. Burst RA pacing showed no inducible atrial flutter/fibrillation. RFA procedure was done with total air kerma 20 mGy.
Conclusion: CTI-dependent atrial flutter is a common atrial arrhythmia, associated with atrial fibrillation, rapid ventricular rate, embolic stroke, and rarely tachycardia-induced cardiomyopathy. Because of well-defined substrate and typically pharmacologic resistance, RFA is a safe and effective first-line treatment. As there are risks associated with radiation exposure during procedure, it is essential to keep the ALARA (as low as reasonably achievable) principle. Large studies[1,2] showed mean fluoroscopy time and dose were 13 (7-28) min and 13.13 Gy.cm^2 compared with 3.58 min fluoroscopy time and 2.44 Gy.cm^2 radiation dose in our procedure. Fluoroscopy integration with three-dimensional electroanatomical mapping system may allow radiation reduction during RFA procedure.
Keywords: Radiation dosage reduction, very low-dose radiation, atrial flutter 3D radiofrequency catheter ablation



Multiple Atrial Arrhythmias Following Triple Valve Surgery

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Background: Post operative arrhythmias (POAs) are commonly found complications following cardiac surgery associated with higher morbidity and mortality. The most frequent form are atrial tachyarrhythmias. Multiple perioperative factors account for the susceptibility of POA, from patient-related factors, surgery-related factors,

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and electrophysiologic abnormalities. Patients with valvular heart disease often have the substrate of atrial enlargement and elevated atrial pressures that promotes propagation of re-entry circuits. Hemodynamic stressors can also trigger focal arrhythmias. Management depends on the specific type of arrhythmia and clinical presentation.

Case Illustration: A 16-years-old boy presented to the emergency room with recurrent episodes of worsening heart failure caused by severe mitral regurgitation, aortic regurgitation, and tricuspid regurgitation due to rheumatic heart disease (left ventricular ejection fraction 63%). Baseline electrocardiogram (ECG) showed sinus rhythm with left ventricular hypertension. The patient underwent urgent surgery with replacement of mitral and aortic valve into mechanical prosthetic valve, and repair of tricuspid valve. A day later, the patient became hemodynamically unstable, with heart rate 178-205 bpm, mean arterial pressure (MAP) <50 mmHg, and his ECG changed into atrial tachycardia suggested from coronary sinus ostium. Synchronized cardioversion was performed several times, but there was no improvement. Bedside echocardiography revealed cardiac tamponade. The patient immediately underwent redo operation and tamponade evacuation. After the tamponade subsided, the patient's ECG became typical counterclockwise atrial flutter with 2:1 conduction, with heart rate 150-155 bpm, MAP >65 mmHg, and low diastolic pressure. He was given intravenous amiodarone infusion and the heart rate decreased to 110-120 bpm. Bedside echocardiography revealed severe central leakage of prosthetic aortic valve. Redo replacement of aortic prosthetic valve was planned for the patient.

Conclusion: Patients with multiple heart valve disease carry increased risk of perioperative arrhythmias due to pre-surgery structural heart abnormalities and the added burden of hemodynamic stress after surgery. Prompt management of unstable arrhythmia and correction of transient causes is important to minimize further worsening of patient's condition.

Keyword: *multiple atrial arrhythmias, atrial tachycardia, atrial flutter, valvular heart disease, post cardiac valve surgery complications*

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Dual Drug Approach: Oral Aminophylline and Oral Salbutamol in Treating Total Atrioventricular Block - A Case Study in Rural Area

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Background: Total atrioventricular block (TAVB) is a critical condition characterized by complete dissociation between the atria and ventricles, resulting in severe bradycardia and potential hemodynamic collapse. Aminophylline and salbutamol are known to have the effect of increasing heart rate and can be utilized to manage heart rate in patients with TAVB, especially in rural areas where immediate transcutaneous pacing is not readily available.
Case Illustration: A 62-year-old female with a history of inferior myocardial infarction came to the ER with sudden onset dizziness, headache, and weakness. Vital signs revealed HR of 30 bpm, blood pressure 90/70 mmHg, and CRT >2 seconds with cold lower extremities. The ECG showed TAVB with ventricular rate 43 bpm. In the ER, the patient received atropine sulfate 0.5 mg intravenously every 5 minutes, three times, but the HR remained at 44 bpm. Then, dopamine 0.5-10 mcg/kg/minute was administered. The HR remained <50 bpm with the maximum dose of dopamine, so an adrenaline drip at 0.1 mcg/kg/minute was added to the therapy, and her HR increased to 52 bpm. She was admitted to the ICU and received salbutamol 3x4 mg, aminophylline 1x200 mg, followed by adrenaline drip at 0.1 mcg/kg/minute, and other oral medications such as CPG, betahistine, dimenhydrinate, sucralfate, atorvastatin, and paracetamol. The following day, her HR increased to 52 bpm with no symptoms. The ECG showed TAVB with infrequent PVC, and the adrenaline drip was stopped with aminophylline dosed up to 3x200 mg. The patient was discharged after 3 days in the ICU with HR of 62 bpm.
Conclusion: This case underscores the challenges in managing TAVB in patients with a history of myocardial infarction in rural areas where access to immediate transcutaneous pacing is unavailable, highlighting the ability of aminophylline and salbutamol to modulate cardiac conduction as an adjunctive treatment for TAVB. However, further research and clinical studies are warranted to establish their efficacy and safety in emergency settings.

Keywords: TAVB, rural area, aminophylline, salbutamol

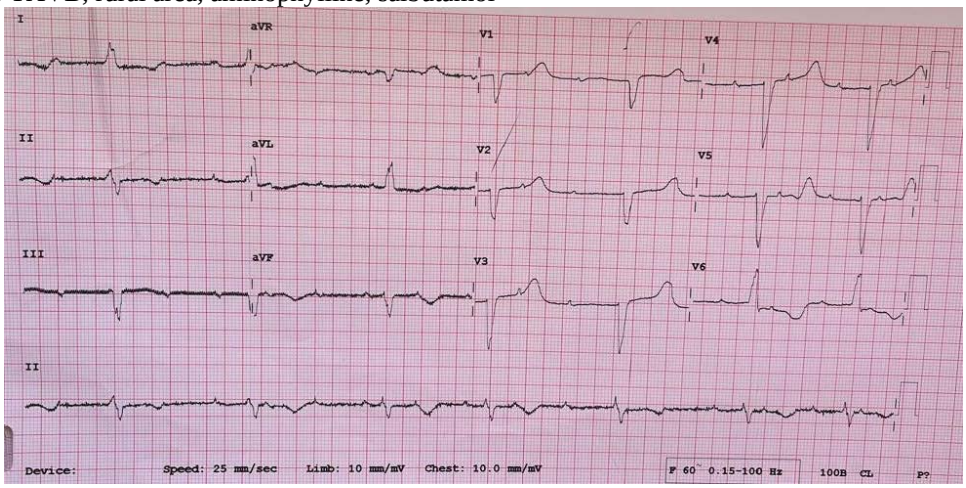

Figure 1. ECG showed Total AV Block with Ventricular rate 43 bpm.

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Clinical Worsening due to Misleading Treatment in Pre-excited Atrial Fibrillation: A Case Report

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Background: Atrial Fibrillation (AF) is the most common cardiac arrhythmia, but identifying the underlying causative mechanism and other coexistences is challenging in daily practice. Wolff-Parkinson-White (WPW) syndrome is a pre-excitation syndrome characterized by the presence of an accessory pathway responsible for ventricular pre-excitation that can lead to severe arrhythmias. Coexistence with AF exposing to potential degenerescence into ventricular arrhythmias when atrial impulses are transmitted along the accessory pathway. The choice of drug in this special presentation must be noteworthy to avoid potentially dangerous treatments, such as digoxin, which is the rate control therapy for AF based on recent algorithms and the availability at hospital.

Case Illustration: We report a case of a 54-year-old female with atrial pre-excitation who routinely consumed digoxin and presented with palpitations, general weakness, and lightheadedness. The symptoms worsened from three years ago when the patient was initially diagnosed solely with AF and began her medication regimen at a different hospital as part of her routine care. After switching therapy based on the clinical, electrocardiography, and echocardiography findings, better symptoms and clinical outcomes were observed.

Conclusion: Pre-excited AF can be a cause of sudden cardiac death, especially when therapy errors occur. Therefore, a holistic diagnosis and appropriate therapy lead to better clinical outcomes for patients.

Keyword: Atrial Fibrillation, WPW syndrome, Digoxin

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A Series of Unfortunate Events: Long QT Syndrome in Post-Partum Period and Emerging Ventricular Tachycardia

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Background: Long QT Syndrome (LQTS) represents a spectrum of disorders characterized by a prolonged corrected QT (QTc) interval, encompassing both congenital and acquired forms. Congenital LQTS, although uncommon, presents a substantial risk of life-threatening cardiac events. Diagnosis hinges on identifying prolonged QTc intervals on resting electrocardiograms (ECGs), with particular attention to medications that inadvertently prolong QT duration, necessitating vigilant management.
Case Illustration:
Case 1: A 33-year-old woman with a history of epilepsy and post-partum ventricular tachycardia (VT), managed with phenytoin, exhibited sudden aggressive behavior due to delirium upon hospital admission. She experienced ventricular tachycardia necessitating defibrillation, with a consistently prolonged QTc interval averaging 520 ms. Despite discontinuation of phenytoin, her QTc persisted at 500 ms, suggestive of congenital LQTS exacerbated by medication. Implantation of an intracardiac defibrillator (ICD) was performed to mitigate the risk of sudden cardiac death.
Case 2: A 28-year-old post-partum woman with massive vaginal bleeding underwent planned hysterectomy without prior history of arrhythmia or cardiac issues. During hospitalization, she experienced unstable ventricular tachycardia, with a prolonged QTc interval averaging 600 ms. Following identification of prolonged QTc, she underwent ICD implantation as a preventive measure against sudden cardiac death.
Conclusion: These cases underscore the critical importance of ECG screening for LQTS in pregnant women and individuals scheduled for medications that prolong QTc intervals. Prolonged QTc intervals in LQTS significantly elevate the risk of ventricular tachycardia and sudden cardiac death, highlighting ICD implantation as a crucial intervention to forestall adverse outcomes in affected patients.

Keywords: Long QT Syndrome, QTc Prolongation, Sudden Cardiac Death

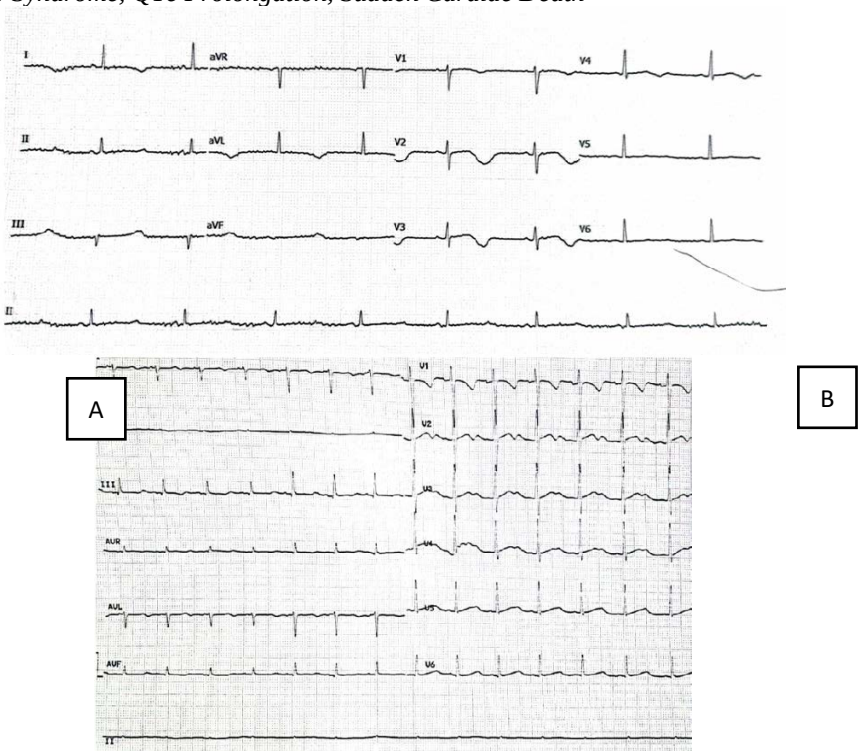

Figure 1. (A) ECG of case I with QTc interval averaging 520 ms; **(B).)** ECG of case I with QTc interval of 615 ms

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A Teen's Heart Race: Uncovering a Hidden Cardiac Condition During a Fever

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Background: Arrhythmias in children are rare, constituting only 5% of emergency admissions. Supraventricular tachycardia (SVT) is the most common, with an incidence of 13 per 100,000 and a prevalence of 2.25 per 1,000. Although typically sporadic, SVT has a familial link, with a 5.5% to 7% frequency in first-degree relatives.[1,2] This case report details a rare instance of SVT associated with Wolff-Parkinson-White (WPW) syndrome in a 14-year-old female.

Case Illustration: A 14-year-old female presented with palpitations and shortness of breath (SOB) for two hours, following a fever and sore throat the previous day. Episodes of palpitations and SOB had occurred after physical activity over the past year. Vital signs showed a heart rate of 225 beats per minute, blood pressure of 126/86 mmHg, temperature of 39.8°C, and respiratory rate of 27 breaths per minute. Physical examination revealed pharyngeal hyperemia and acute tonsillitis. An electrocardiogram (ECG) indicated SVT with a heart rate of 198 beats per minute. The patient received oxygen, monitoring, and an intravenous line. Her heart rate stabilized at 146 beats per minute, transitioning to sinus tachycardia prior to the administration of any maneuvers or antiarrhythmic drugs. The cardiologist recommended treating the fever and infection rather than antiarrhythmic medication. The pediatrician suggested crystalloid infusion, paracetamol, and cefotaxime due to elevated leukocyte count (17,450/mm³). After 24 hours, the patient's condition stabilized, and a follow-up ECG showed sinus rhythm with a shortened PR interval (110ms) and delta wave, indicating a WPW pattern. After three days, she was discharged in stable condition with a scheduled electrophysiology study to investigate accessory pathways. WPW syndrome is suspected to cause SVT during fever and infection. Studies indicate 20% of children with SVT experience recurrence, with higher rates in those with WPW syndrome.[3]

Conclusion: This case report highlights a rare instance of SVT in a child with WPW syndrome. Managing the underlying fever and infection stabilized the patient's condition. An electrophysiology study is planned to investigate the accessory pathways further. This case underscores the importance of recognizing and addressing potential SVT recurrence in pediatric patients, particularly those with WPW syndrome.

Keyword: SVT, Wolf-Parkinson-White, Pediatric, Arryhtmia, Accessories Pathway

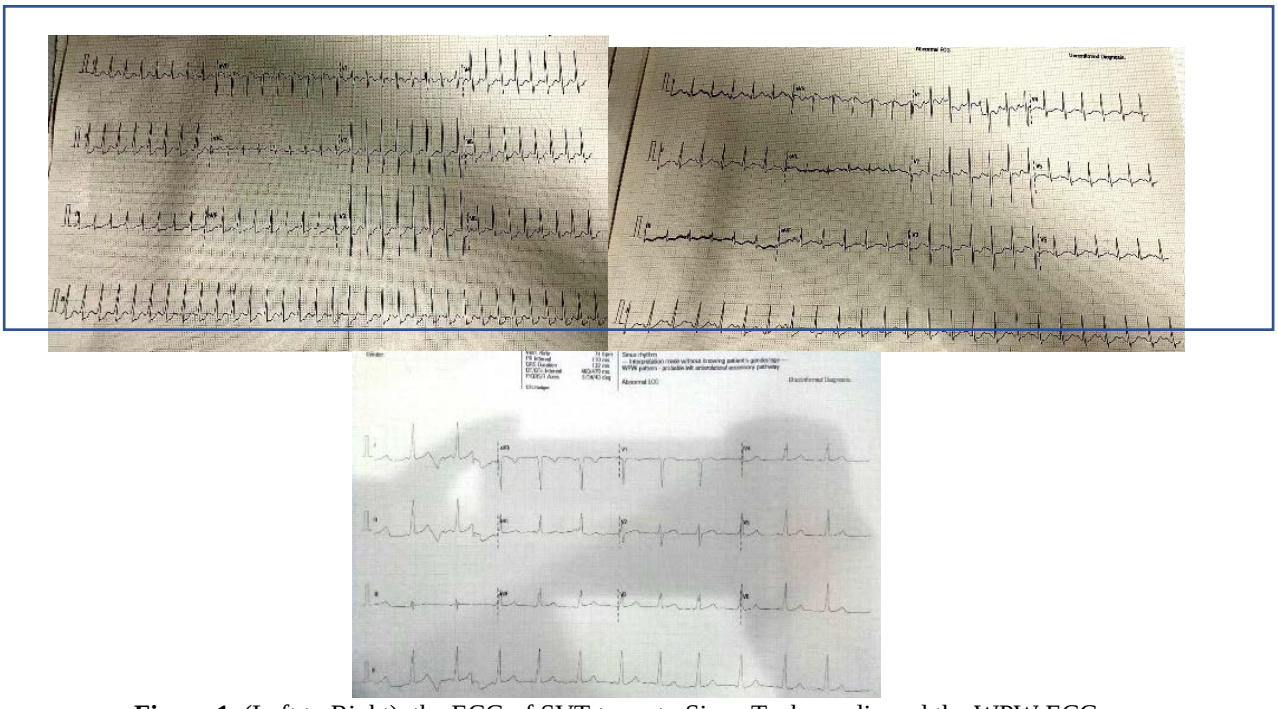

Figure 1. (Left to Right), the ECG of SVT turns to Sinus Tachycardia and the WPW ECG

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Persistent atrioventricular nodal reentrant tachycardia leading to tachycardia induced cardiomyopathy and conduction system dysfunction

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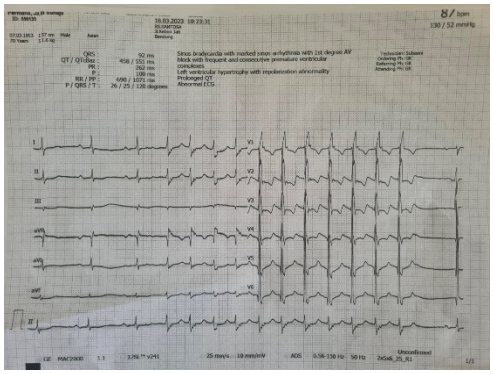
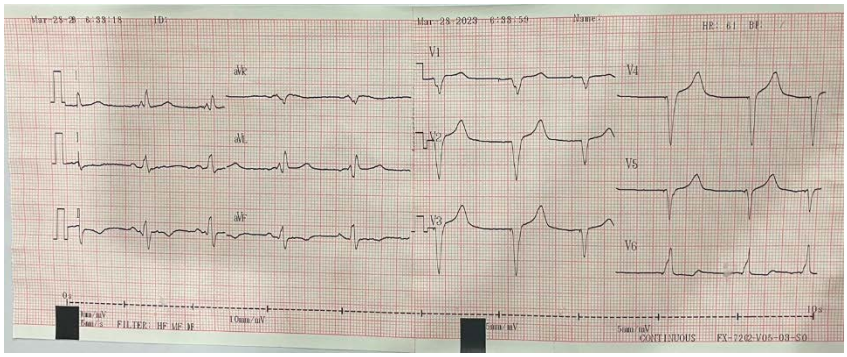
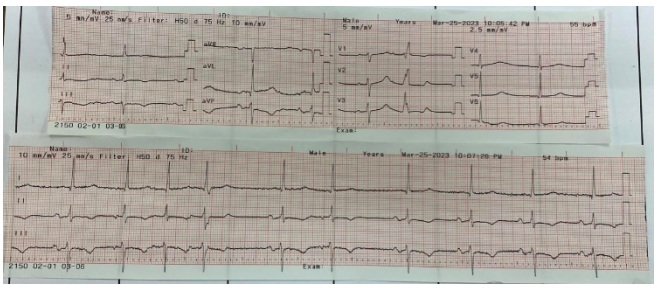
Background: Atrioventricular nodal reentrant tachycardia (AVNRT) is a common paroxysmal supraventricular arrhythmia, can rarely lead to cardiomyopathy and post-AVNRT ablation abnormalities, including sinus and AV nodal dysfunction.
Case illustration: A 70-year-old male was referred for syncope lasting less than 30 seconds, followed by full recovery of consciousness, preceded by sudden onset of palpitations without identifiable triggers. These palpitations had been occurring intermittently for 5 months, lasting up to 1 hour with 2-3 episodes daily, with no chest pain or shortness of breath. A 12-lead ECG showed sinus bradycardia with first-degree AV block and SVT with RBBB Abberancy. Echocardiography showed dilated chambers and reduced left ventricular systolic function (LVEF Biplane's 38%). The Electrophysiology (EP) Study, showed sinus rhythm, prolonged PR interval, intermittent SVT with RBBB abberancy. V pacing demonstrated VA decremental conduction. Atrial extrastimulus testing revealed AH jump then induced SVT with VA interval 0 ms. Atrial pacing demonstrated SA and AV nodal dysfunction. Slow pathway ablation was performed, and no more tachycardia afterward. Post-ablation ECG showed sinus AV nodal dysfunction, leading to a decision for Dual Chamber Left bundle Pacing implantation. Follow up after 1 month of ablation and pacemaker implantation, patient was asymptomatic and left ventricular systolic function was improved to 50 %.
Conclusion: This case emphasizes the rarity of AVNRT-induced cardiomyopathy and conduction system dysfunction, highlighting the diagnostic challenges and the importance of comprehensive treatment in managing incessant AVNRT complications.
Keywords: Incessant AVNRT, cardiomyopathy, sinus AV nodal dysfunction, Left bundle Pacing







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Masquerade: Atypical accessory pathway uncovering antidromic atrioventricular re-entrant tachycardia mimics ventricular tachycardia – electrocardiography insight

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Background: Ventricular tachycardia (VT) is frequently identified in cases of severe palpitation and hemodynamic instability. Despite the availability of several algorithms to differentiate SVT and VT, none easily distinguish between antidromic AVRT and VT, as both present with wide QRS. An accessory pathway (AP) can have various placements of insertion which can generate such a "hidden" or atypical pattern in ECG, potentially obscuring the existence and mimicking other arrhythmia.
Case Illustration: A 43-year-old female with 7-years-ago-cardiac disease history presented with severe palpitations and chest pain for 3 hours. She was fully alert but gradually lethargy, with BP 140/90 mmHg and HR 220 bpm. ECG revealed regular monomorphic WCT with AV dissociation suggestive of VT with LBBB-morphology and left superior axis possible origin from posteromedial septum. A 200 J synchronized cardioversion was converting the rhythm to sinus with multivocal PVCs, short PR interval, normal QRS duration, rS complex in V1, and slight delta wave likely to WPW via right-sided AP possibly from anterolateral tricuspid annulus, which made this tachyarrhythmia tended to be antidromic AVRT. Repeat ECGs in sinus rhythm come with minimal even without pre-excitation showed by near normal PR interval, normal QRS duration, and barely visible delta wave demonstrated atypical AP with 'Mahaim-like' properties. No significant abnormalities finding in laboratory examination and echocardiography. She got antiarrhythmic therapy with amiodarone continuous infusion then discharged in stable condition following 11-day ICCU hospitalization. No further cardiac imaging nor electrophysiology study (EPS) were performed.
Conclusion: Minimal ventricular pre-excitation may not be easily noticed, resulting from various AP placement, only small amount of myocardium activated by AP due to impulses being efficiently conducted via AV node, or antegrade decremental AP conduction. To clearly differentiate the arrythmia from VT and unmasking non-manifest AP, further investigation using EPS is needed. This case highlights it's necessary to be aware that WCT can stem from various etiologies, even in individuals with prior cardiac disease. Through analyzing the ECG carefully, clinicians can uncover subtle differences in the electrical patterns, aiding in accurate diagnosis and management.
Keywords: accessory pathway, antidromic AVRT, VT, Mahaim

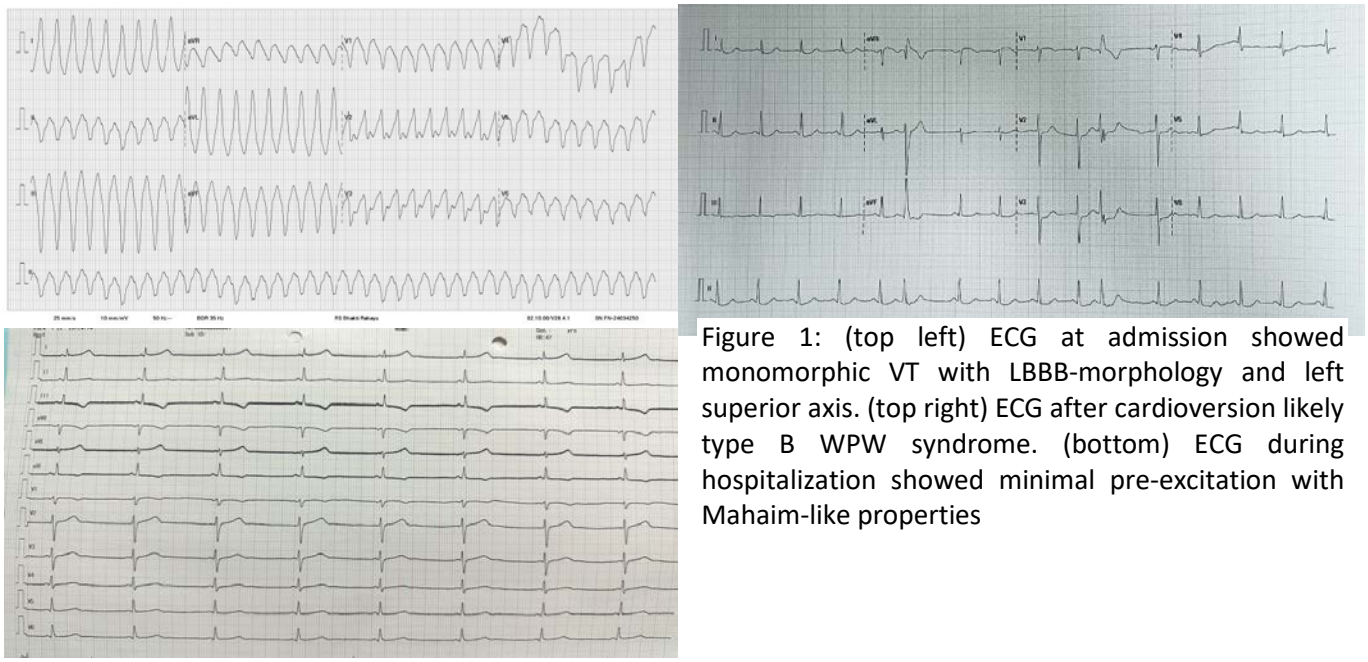

Figure 1: (top left) ECG at admission showed monomorphic VT with LBBB-morphology and left superior axis. (top right) ECG after cardioversion likely type B WPW syndrome. (bottom) ECG during hospitalization showed minimal pre-excitation with Mahaim-like properties

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A VERY RARE CASE OF INTER-ATRIAL CYST RESULTING TOTAL ATRIOVENTRICULAR BLOCK: A DILEMMA OF PACING OR SURGERY FIRST

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Background: Cystic tumors of the AV node are very rare and can disrupt the conduction system. Until now, due to the small number of cases, treatments for patients with cystic tumors in the AV node which cause atrioventricular block not yet established.

Case Illustration: A 68 year old male with syncope and fatigue admitted to emergency unit diagnosed with permanent total AV block planned for installation of permanent percutaneous pacemaker. During observation in the ICCU, hemodynamic echocardiography was carried out and a suspicion of an inter-atrial cystic mass was found. A full echocardiography examination revealed a cystic mass 1.97 x 2.11 centimeters of size in the inter-atrial septum close to the non-coronary cusp. Consultation was carried out with thoracic and cardiovascular surgeon to discuss the disease due to its rarity and no specific treatment for this case in the literature. From various cases that have been reported both antemortem and postmortem, although the mass is cystic in shape, its nature tends to infiltrate and damage the conduction system as obtained from histological examination. From several reported cases, the risk of TAVB due to surgical complications is also very high. We decided to install a PPM first because it was hypothesized that permanent damage had occurred to the conduction system and the nature of the cyst in this patient did not affect haemodynamics.

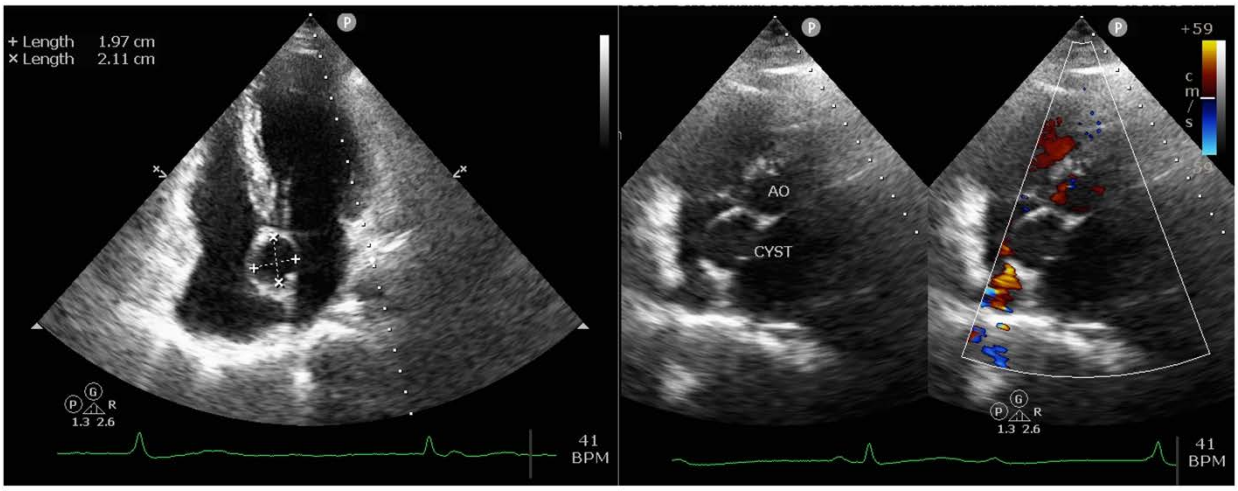

Figure 1. Echocardiography showed a cystic mass 1.97 x 2.11 centimeters of size in the interatrial septum close to the non-coronary cusp.

Conclusion: Total AV block caused by interatrial cysts from several case reports tends to be permanent even after surgery. PPM installation in this case where damage to the conduction system is suspected cannot be avoided.

Keywords: Interatrial cyst, TAVB, Permanent pacemaker, Surgery

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Supraventricular Tachycardia with Aberrancy Due to Beta-Blocker Abrupt Cessation in Paroxysmal Atrial Fibrillation Patient: A Case Report

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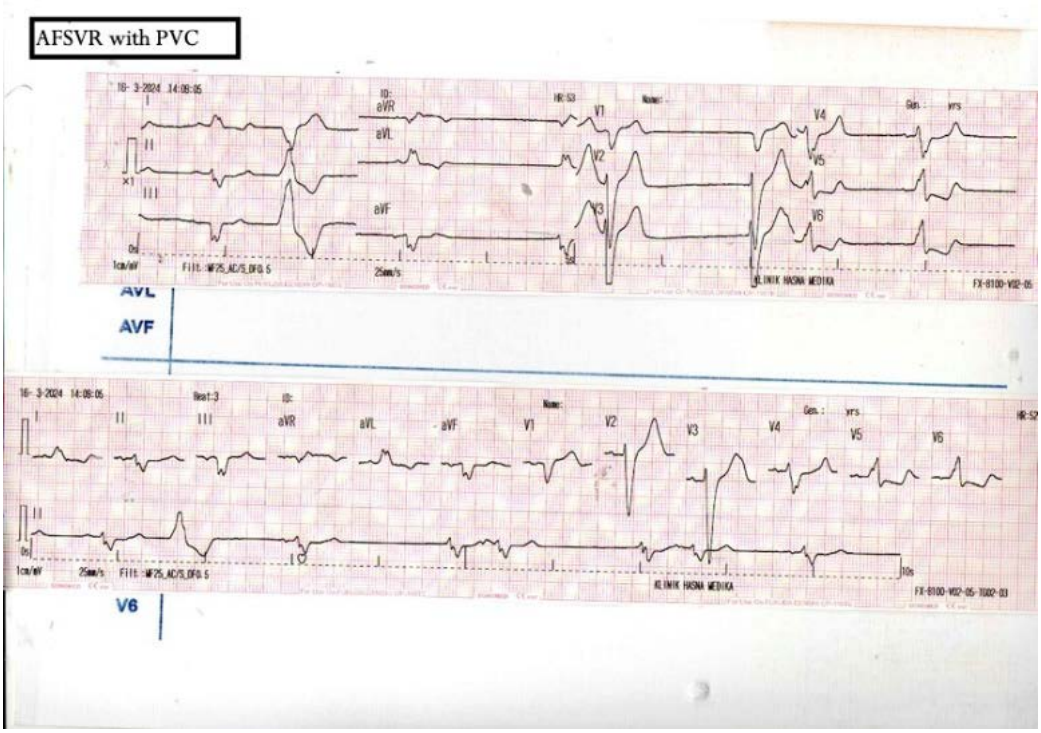
Background: Supraventricular tachycardia (SVT) with aberrancy is a dysrhythmia originating at or above the atrioventricular (AV) node with wide QRS complex. Beta-blockers, such as bisoprolol, are commonly used to control heart rate and maintain rhythm stability in atrial fibrillation (AF) patients.

Case Illustration: A 64-year-old male patient presented to the emergency room (ER) due to irregular heartbeat and agitation. Upon examination, the patient was alert with heart rate (HR) of 50bpm and blood pressure (BP) of 120/67mmHg. Electrocardiography (ECG) revealed AF with slow ventricular response (AFSVR) and premature ventricular contraction (PVCs). The patient had previous history of percutaneous coronary intervention with stent placement on right coronary artery in December 2023. His prescribed medications included bisoprolol 1.25mg q.d, furosemide 40mg q.d, ramipril 5mg q.d, dual antiplatelet therapy (DAPT), and statin. However, the patient refused to be admitted. Due to AFSVR, bisoprolol was temporarily stopped. Four days later, the patient returned to the ER with palpitation and agitation that had persisted since the previous night. Upon examination, he was alert with HR of 156bpm and BP of 144/81mmHg. ECG revealed regular wide QRS complexes tachycardia, RBBB-like morphology, and no AV dissocation; indicative of SVT with aberrancy. After administering 150mg intravenous bolus of amiodarone, the ECG converted to sinus rhythm. During inpatient care, the patient was given amiodarone maintenance dosage and reinitiated on bisoprolol 1.25mg q.d., along with his previous medications. No further episodes of SVT were observed.

Conclusion: This case underscores the risks associated with the abrupt cessation of beta-blocker therapy in patients with paroxysmal AF, as evidenced by the development of SVT with aberrancy. The initial decision to withhold bisoprolol due to AFSVR led to significant tachyarrhythmia, necessitating acute intervention with amiodarone to restore sinus rhythm. The patient's subsequent stabilization with a maintenance dose of amiodarone and reintroduction of bisoprolol highlights the need for careful and continuous management of antiarrhythmic medications.

Keywords: Supraventricular Tachycardia, Beta-Blockers, Atrial Fibrillation



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Effective Rate Control in Post-Operative Atrial Fibrillation Following Non-Cardiac Surgery: A Clinical Case Report

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Background: Postoperative atrial fibrillation (POAF), characterized by new-onset atrial fibrillation (AF) following surgery, is a common complication affecting 0.4% to 15% of patients after non-cardiac surgeries[1,2]. It is associated with prolonged hospitalizations, increased healthcare costs, and has been linked to adverse cardiovascular events (including stroke, heart failure, and mortality)[1,2,4]. Recent research has explored the pathophysiology of POAF and proposed therapeutic strategies, with inconclusive evidence favouring either rate or rhythm control[3,5].

Case Illustration: An 80-year-old male developed new-onset AF two hours post-laparotomy. The patient's clinical assessment was initially challenging due to effects of anesthesia. His medical history included controlled hypertension, coronary artery disease 3 vessels disease with complete revascularization, and a medication regimen comprising acetylsalicylic acid, clopidogrel, ramipril, and bisoprolol. Due to the occurrence of melena, dual antiplatelet therapy was discontinued, and ramipril was withheld one day prior to surgery; simvastatin was added to his regimen.
Laboratory investigations revealed hypokalemia, hypernatremia, and decreased e-GFR. Echocardiography indicated a LVEF of 62%, grade I diastolic dysfunction, mild mitral regurgitation, and mild tricuspid regurgitation with low probability of pulmonary hypertension. The electrocardiogram confirmed new-onset AF with a rapid ventricular response. Patient was successfully managed with intravenous digoxin, achieving sinus arrhytmia within six hours and maintained a steady sinus rhythm throughout the hospitalization and was discharged stable after seven days.

Conclusion: POAF represents a significant clinical challenge, increasing healthcare burden and cardiovascular risks post-surgery. In this patient, POAF may have been precipitated by an electrolyte imbalance. While strategies such as digoxin for rate control can achieve sinus rhythm restoration, uncertainties remain regarding its long-term efficacy and the underlying causal mechanisms for recurrent atrial fibrillation. Further research into POAF's pathophysiology and comparative therapeutic effectiveness is imperative to optimize management strategies and improve patient outcomes.

Keyword: Postoperative atrial fibrillation, rate control, digoxin, non-cardiac surgery

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Electrolyte Dysregulation and Cardiac Chaos: A Case of Hypokalemia-Induced Polymorphic Ventricular Tachycardia in a Postpartum Woman with Hyperthyroid State

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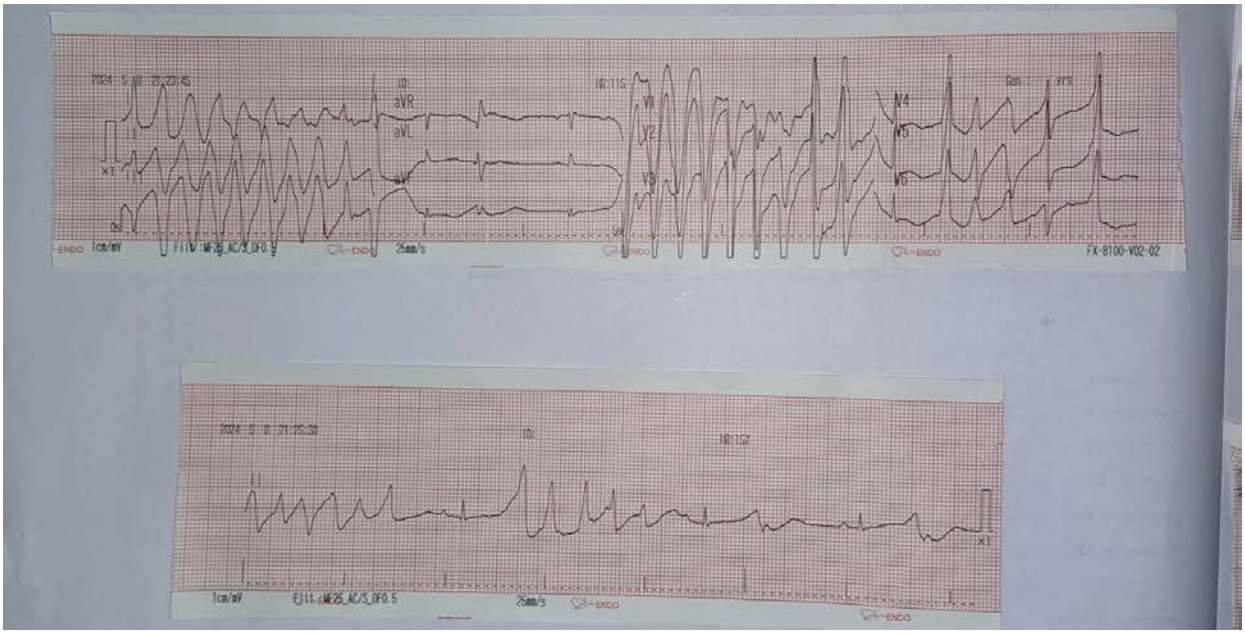
Background: Hypokalemia is a potentially fatal condition that could be triggered by hyperthyroid state, which increases potassium excretion leading to increased heart electrophysiological properties, thereby increasing the likelihood of arrhythmias.
Case Illustration: A 29-year-old woman with a history of postpartum 1 month ago, presented to our emergency department with refractory ventricular tachycardia (VT). Four days prior she experienced a seizure and was brought to the nearby hospital and diagnosed with refractory VT due to hypokalemia and hyperthyroidism. She was referred due to her frequent VT episodes, occurring 5–6 times per day. She reported no chest pain or dyspnea; upon arrival, she was conscious with normal blood pressure, physical examination revealed no abnormalities. She experienced seizures with Polymorphic VT, as observed on the bedside monitor followed by a period of pulseless VT required defibrillation, and she regained Return of Spontaneous Circulation (ROSC). The laboratory findings indicate a potassium level of 2.5mmol/L, a TSH of 0.25uI/U/mL, FT4 of 21.6pmol/L. The echocardiogram reveals a left ventricular ejection fraction (LVEF) of 56%, while chest X-ray within normal limits. Lidocaine infusion was given to control cardiac rhythm and potassium chloride through the central line to correct the electrolyte imbalance. She was admitted to the Intensive Care Unit (ICCU) and consulted with the Endocrine Department regarding her hyperthyroid status. In ICCU she encountered repeated occurrences of premature ventricular contractions (PVC), which ultimately resulted in NSVT. Following the correction of her electrolyte imbalance and the initiation of therapy for her hyperthyroid condition, we successfully terminated her period of VT. She was transferred to the ward 24 hours after the last VT episode and discharged 2 days later.
Conclusion: Managing hypokalemia and hyperthyroidism is crucial to prevent arrhythmias because both conditions can significantly increase the risk of ventricular tachycardia (VT).

Keywords: Polymorphic Ventricular Tachycardia, Hypokalemia, Hyperthyroid.



ECG showing R on T

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Undelivered Shock in Unstable Hemodynamic VT: the Hurdles of Discrimination of ICD Morphology Criterion

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Background: An adverse consequence of an ICD is the occurrence of inappropriate therapy. Two potential scenarios are probable: an unnecessary shock, which arises when shock therapy is applied for rhythms other than VT or VF, and instead an undelivered shock, which happens when no shock treatment is administered. The device often delivers inaccurate treatment due to its misclassification of sinus tachycardia or atrial fibrillation/flutter with rapid atrioventricular (AV) conduction. To address this issue, modern ICD mechanisms integrate multiple discriminators into their algorithms to distinguish between SVT and VT. This case illustrated a situation in which the single chamber discriminator ICD (morphology criterion) incorrectly categorized VT as SVT.
Case Illustration: A 40-year-old man suffered an abrupt onset of seizures following cardiac arrest without any preceding reports of chest pain. The patient had previously had an ICD placement due to recurrent VT and a prior episode of cardiac arrest. Patient with medical background of coronary artery disease and underwent complete revascularization, but hadn't previously experienced another episode of supraventricular tachycardia. We immediately commenced the interrogation of the ICD. The electrogram revealed the presence of NSVT and SVT episodes, but no VT episode or shock therapy was recorded. At the following period, patients performed electrophysiology studies which the result was no SVT was induced with normal function of AV and SA node. Coronary angiography was conducted simultaneously, confirming patent stents in three vessels.

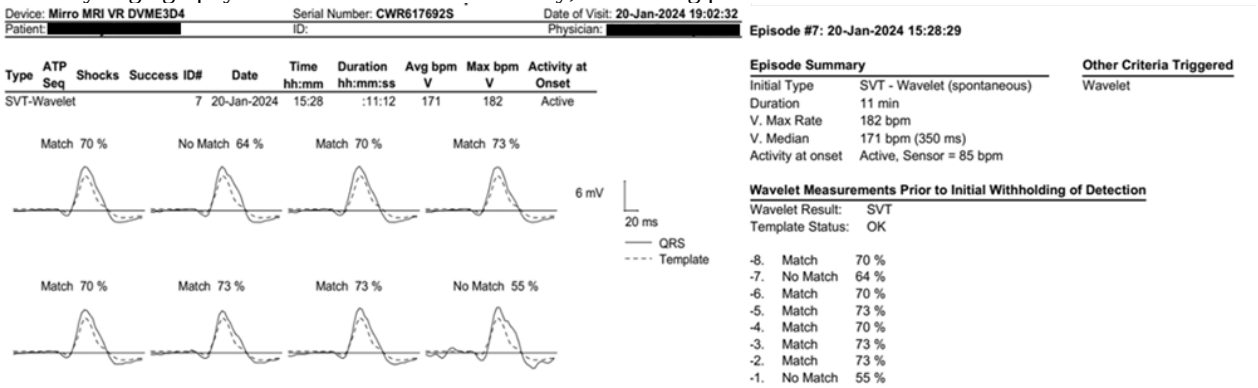

Figure 1. Wavelet result device electrograms (EGMs) at SVT episode.

Conclusion: In this case, the ICD misidentified ventricular tachycardia episodes as SVT, leading to the administration of undelivered shock therapy. Morphology discriminating criterion errors in single-chamber ICDs might happen due to some possible causes. It was highly recommended to increase the duration of the detection period to avoid misclassifying ICDs.

Keyword: Wavelet discrimination, Supraventricular tachycardia, ventricular tachycardia, ICD misclassification

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Ventricular Tachycardia as a Sole Manifestation of Pulmonary Stenosis in Adulthood: a Case Report

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BACKGROUND: Congenital heart disease (CHD) stands as the most prevalent major birth defect, impacting over 1% of newborns. Pulmonary stenosis (PS) comprises approximately 8% of congenital heart disease (CHD) cases. Adults living with PS face enduring complications, including arrhythmias, and ultimately sudden cardiac death (SCD) and 80% of documented arrhythmias preceding SCD events in CHD patients involve ventricular arrhythmias.

CASES PRESENTATION: A 46-year-old female referred to our emergency department reporting intermittent palpitations, lasting for 30 minutes for the last 5 days. She denied any history of dyspnea, syncope, or chronic diseases. In the previous hospital, angiography showed a non-limiting flow coronary artery disease (CAD) complicated with hemodynamically stable monomorphic VT. Upon physical examination, vital signs were stable and a grade 4/6 pansystolic murmur at the left lower sternal border with a positive Carvallo sign was observed. ECG showed normal sinus rhythm. Laboratory tests showed elevated Troponin I levels (265 ng/mL) and mild hypokalemia (3.1 mEq/L). Transthoracal echocardiography showed situs solitus, AV-VA concordance, with severe infundibular PS (peak gradient 69-85 mmHg) mild PR, moderate TR. Furthermore, CMR was performed and revealing extensive RV fibrosis at base to apical free wall (Scar Burden 47.6%), and prominent focal fibrosis at both RV insertion points. ICD implantation and intervention for PS was planned for our patient as a secondary prevention. Unfortunately, the patient was lost to follow up

CONCLUSION:
In adult patients with PS, VT can emerge as a potential complication. This arrhythmia may arise due to RV dilation and dysfunction, leading to myocardial stretching and fibrosis, thereby creating an arrhythmogenic focus. Scar burden by CMR could stratify the risk of VA in the future and used for consideration of the next management.

Keywords: Arrhythmia; Pulmonary stenosis; Right ventricle; Ventricular tachycardia

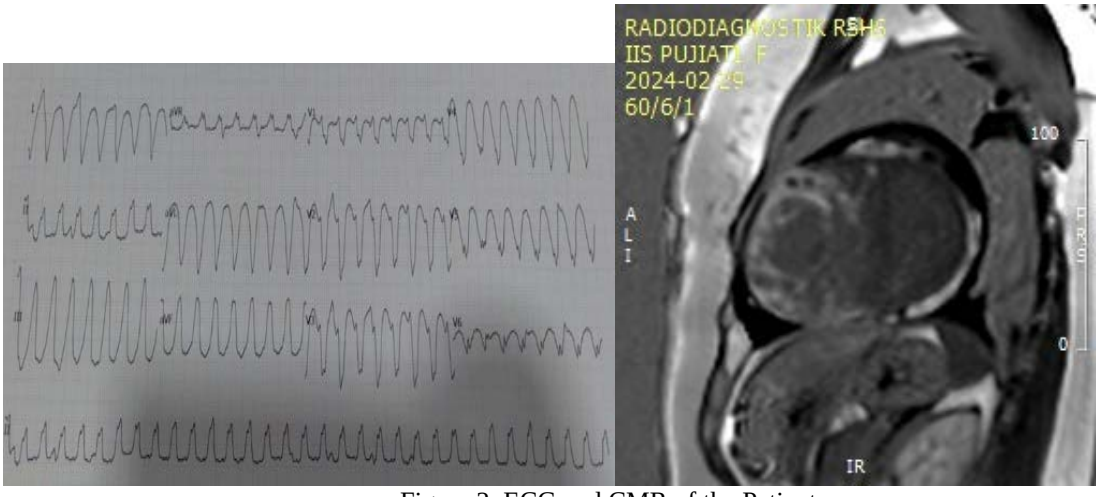

Figure 2. ECG and CMR of the Patient

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Improved high degree atrioventricular conduction block in myocarditis patients: A case series of a rare condition

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Background: High-degree atrioventricular conduction block (HDAVB) is a rare manifestation of myocarditis, with previous data suggesting an incidence of 1.14% out of all myocarditis patients. Diagnosing myocarditis as the etiology of HDAVB needs an extensive process of excluding other possible reversible causes. Despite the risk of hemodynamic compromise, this condition is mostly reversible.

Case Illustration:
Case 1: A 24-year-old female presented with weakness and right upper quadrant pain, blood pressure was 68/50 mmHg, and the heart rate was 35 bpm. The ECG showed a third-degree AV block with an episode of ventricular standstill. A transvenous pacemaker (TVP) was immediately implanted. Echocardiography showed a reduced left ventricular ejection fraction (LVEF) of 33% with global hypokinetic and increased Troponin T level. On day 7, the patient's condition improved with ECG showing intrinsic sinus rhythm. Due to no chronicity and other reversible causes, myocarditis was considered. CMR showed mid-wall fibrosis at the basal and septal aspects of the right and left ventricle (LV), suggesting acute myocarditis. The patient was discharged and evaluation in outpatient visits showed improved LVEF and no symptom recurrence.

Case 2: A 30-year-old female presented with fever and weakness. Fever was observed 7 days prior and was diagnosed as typhoid fever. On the day of admission, the patient felt weak with a heart rate of 35 bpm. ECG showed a third-degree AV block and TVP was implanted. Due to high suspicion of myocarditis from symptoms, CMR was considered. TVP was removed despite intrinsic ECG still showing a second-degree AV block. The CMR shows a thin mid-wall fibrosis at inferoseptal LV with myocardial edema at basal, septal, and anterior LV, suggesting myocarditis. Steroid treatment was given, and the patient's condition improved. The patient was discharged with no symptom recurrence.

Conclusion: Patients presenting with HDAVB need scrutiny of the possible etiologies. Myocarditis should be considered after all other causes are excluded. In addition, since most conditions are reversible, cardiologists need to be mindful before planning invasive and expensive options such as permanent pacemaker implantation.

Keywords: Myocarditis, atrioventricular block, HDAVB, CMR, TVP

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A Case Series of Ventricular Tachycardia in Preschoolers: How Should the Management Be Strategically Timed?

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Background: Ventricular arrhythmia is uncommon among pediatric patients, with an incidence of approximately 1.1 episodes per 100,000 childhood years, often appearing unexpectedly in those with structurally normal hearts. Nevertheless, prompt consideration of specific etiologies is crucial when managing ventricular arrhythmia in children, particularly those with underlying cardiac conditions, necessitating tailored diagnostic and therapeutic approaches.

Case Illustrations:
Case 1: A 4-year-old boy presented with palpitations. His ECG revealed monomorphic ventricular tachycardia (VT) at 243 bpm originating from the left superior aspect. Intravenous amiodarone successfully restored sinus rhythm with complete right bundle branch block morphology. Holter revealed premature ventricular contractions (PVCs) burden of 2.3% and sustained VT. Echocardiography demonstrated decreased right ventricular function alongside a well-contracting left ventricle. Arrhythmogenic right ventricular dysplasia was suspected, prompting confirmation with cardiac magnetic resonance (CMR) imaging revealing right atrial and ventricular dilation without fibrosis, suggestive of an idiopathic etiology. The patient was discharged on propranolol, with subsequent outpatient evaluations showing no symptom recurrence at the 6-month follow-up, though intermittent non-sustained VT persisted on Holter.
Case 2: Another 4-year-old boy presented with a subfebrile fever persisting for 7 days following a prior hospitalization for pneumonia. He was found to have non-sustained monomorphic VT during hospitalization, prompting referral. Continuous rhythm monitoring demonstrated sinus rhythm interspersed with non-sustained VT episodes, with Holter revealing a 18.9% burden of PVCs. Echocardiography revealed a structurally normal heart, while CMR exhibited right atrial dilation without evidence of fibrosis, suggesting an idiopathic origin of the VT. Intravenous amiodarone was administered, and the patient was discharged on propranolol. At the 3-month follow-up, outpatient evaluations showed no recurrence of VT, with ongoing Holter planned.

Conclusion: Ventricular tachycardia in young children with structurally normal hearts generally carries a favorable long-term prognosis, often resolving spontaneously. Therefore, a cautious approach to invasive interventions is warranted. For those with preserved left ventricular dimensions and function, conservative antiarrhythmic therapy may suffice. However, in refractory cases, catheter ablation may be considered.

Keywords: Ventricular tachycardia, pediatric, idiopathic

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Implantable Cardioverter Defibrillator Implantation in Hypertropic Cardiomyopathy Patient : Do clinical features need to be considered?

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Background: Hypertrophic cardiomyopathy (HCM) is a common genetic condition with various complications such as SCD, atrial fibrillation (AF) and heart failure (HF). Early detection and ICD intervention has reduced mortality rate. Risk stratification and targeting candidates ICD can be complex due to clinical scenarios, arrhythmogenic substrate and a risk factor.

Case Illustration: A young man with HF was visited to clinic with history of cardioversion from VA. ECG showed atrial flutter and poor R wave progression. Echocardiography showed suggestive HCM and reduced EF. An MRI examination showed prominent focal patcy and fibrosis consistent of HCM. Scar burden 44,5%. Genetic testing revealed positive pathogenic variant identified in MYH7. The ICD was implanted. During procedur, patient suffered VA, ICD overdrive pacing successfully convert into sinus. Amiodarone drip was given. During hospitalization, he got a shock. Interogate showed episodic VA that recognize as ventricular fibrillation. A shock was delivered without prior ATP. Tram tracking sign was observed. The day after, he was urgently transferred to Intensive Care because unstable hemodinamik. Norephinephrine was administered. Patient experienced PEA and was declared dead. The earlier onset,and worse outcomes common in sarcomeric mutation patients. HCM with reduced EF will develop into refractory HF and myocardial scar. Fibrosis greater than 15% was associated with risk of SCD. Patient with AF also had a higher risk of mortality.

Conclusion: Patient selection, clinical features, arrhytmia, high electrical voltage to be considered in ICD management.

Keyword: Genetic, Implantable Cardioverter Defibrillator, Hypertropic cardiomyopathy

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Intractable Ventricular Arrhythmias Management in Limited Modality Area: What can I do?

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Background: Intractable ventricular arrhythmia was a critical condition that needs some circumstances modalities, such as specific antiarrhythmic drugs (AAD), electrical cardioversion and defibrillation, emergent PCI, even catheter ablation. In certain situations, except radiofrequency ablation was done, tachyarrhythmia could not be terminated. In Indonesia, not all AAD were available. Sometimes overdrive pacing with a temporary pacemaker and old AAD (quinine or propranolol) could be used to treat intractable ventricular arrhythmia.

Case Illustration: A 13-years-old boy, 38 kg, was consulted to our division with stable sustained SVT, heart failure ROSS IV, and suspected tuberculosis. He was already given amiodarone infusion for 48 hours and cardioversion, but arrhythmia did not terminate. Electrocardiography revealed wide-complex QRS tachycardia, RBBB morphology, QRS 180 bpm, and right axis deviation. Echocardiography showed low ejection function without structural heart disease. Electrolytes were normal. Our hospital has no verapamil, neither intravenous beta blocker. We performed a temporary pacemaker in the cath lab and overdrive pacing until 200 bpm three times, and ECG was converted to sinus rhythm with rate 70 bpm, without AAD maintenance, and ventricular arrhythmia was not induced. Temporary pacemaker was maintained for 72 hours. Patient was discharged in stable condition without AAD. A 50-years-old man with anterior STEMI already performed primary PCI with complete revascularization in LAD and incomplete lesion in another vessel. He was consulted with VT electrical storm. Defibrillation and CPR were done five times a day before. Electrocardiography showed polymorphic VT, Torsade de Pointes, and sometimes VF. He was intubated and sedated for 72 hours. We gave oral propranolol until 4 x 40 mg due to metoprolol not available, and no further attacks of ventricular arrhythmia. We planned to refer him to National Cardiovascular Center Harapan Kita for emergent 3D VT ablation, but not transportable. Patient was discharged after seven days and got propranolol 4 x 40 mg.

Conclusion: In limited area, some ventricular arrhythmias are best managed with an overdrive pacing and antiarrhythmic drugs to provide effective arrhythmia termination and control recurrent arrhythmias.

Keywords: ventricular arrhythmias, idiopathic left fascicular tachycardia, overdrive pacing, antiarrhythmic drugs

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Right Sided Pacemaker Implantation in Persistent Left Superior Vena Cava (PLSVC): a Case Report

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Background: Persistent left sided superior vena cava (PLSVC) is a congenital anomaly of the venous, often asymptomatic and discovered during cardiac procedures. Placement of a pacemaker in cases of PLSVC can be challenging, requiring precise techniques to ensure that the leads can reach the target organ.

Case illustration: a 64-year-old woman was referred to the hospital for permanent pacemaker (PPM) implantation. She was diagnosed with sick sinus syndrome and frequently experienced recurrent syncope. Venography with fluoroscopy revealed contrast filling the left-sided superior vena cava extending to the coronary sinus, indicating the presence of persistent left sided superior vena cava (PLSVC). When attempting the 7 Fr sheath and PPM's lead through the coronary sinus, the device could not enter the right atrium due to a sharp exit angle. Therefore, the PPM implantation was performed in the right hemithorax via the right subclavian vein, and the lead was successfully positioned to the RV septum. Chest X-ray evaluation confirmed successful placement of the PPM below the right subclavian vein without any complications.

Conclusion: Placement of a PPM through the right subclavian vein can be considered when the stylet and PPM's lead cannot pass the right atrium due to PLSVC (Persistent Left Superior Vena Cava).

Keyword: Permanent pace maker, persistant left vena cava superior syndrome, sick sinus syndrome

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Hidden P in the Mountain of T : Electrocardiographic Manifestation of Hyperthyroidism

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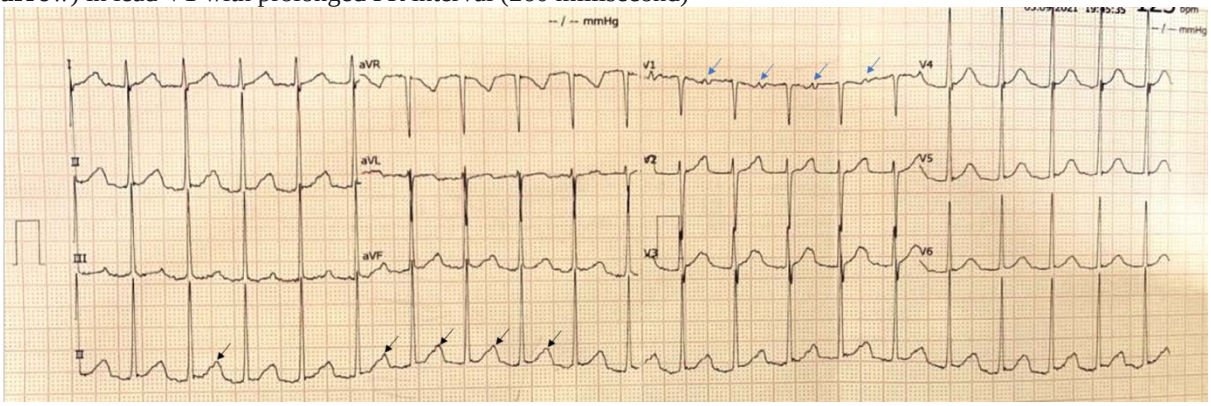
Background: Hyperthyroidism can cause various ECG manifestation mimicking other conditions. Therefore, scrutinization of the ECG is indispensable to avoid unnecessary treatment. Here we present a case of hyperthyroidism from underlying Graves Disease that produce unusual ECG characteristic.

Case Illustration: A-13 year old girl came with chief complaint of palpitation since 2 months ago. The patient also mentioned that she was experiencing menstrual irregularity and insomnia for the past 3 months. History of chest pain, syncope, weight loss , increased bowel movement, increased perspiration, irritability were denied by the patient. The girl's mother however, had a history of Graves Disease that was under treatment process. Physical examination showed that she was tachycardic (130 x/ min), tachypneic (22 x/min), and her blood pressure was 132/116 mmHg. There was a palpable, diffuse, enlarged mass in front of the patient's neck that moved when the patient swallowed something. Initially, the patient's ECG was interpreted as junctional tachycardia with regular rate of 125 x/min as no visible atrial activity could be seen. However, after closer inspection, there was superimposed p-waves on the preceeding T wave that are upright in leads II (**black arrow**) and biphasic (**blue arrow**) in lead V1 with prolonged PR interval (260 millisecond)



Therefore, we concluded that the rhythm was most likely sinus tachycardia with first degree AV Block. Although atrial tachycardia originating from the crista may produce similar appearance on the surface ECG, the prevalence of atrial tachycardia in hyperthyroidism is less common. The patient did, in fact, have hyperthyroidism (TSH : 0.01 uIU/mL FT4 : 7.19 ng/dL) with high titer of thyroid receptor stimulating antibody (TRAb : 5.6 IU/L) and was referred to pediatric endocrinologist.

Conclusion: Hyperthyroidism can produce uncommon ECG manifestations, one of them is varying degree of atrioventricular block. Awareness of such cases is important to ensure optimal and necessary treatment was given to the patient.

Keyword: Hyperthyroidism, Tachycardia , P wave, T wave, AV-Block

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Hyperkalemia, The Great Imitator, Mimicking de Winter T-Wave and ST-Segment Elevation in pseudo-STEMI Patient: Which Treatment Strategy to Use?

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Background: De Winter ECG pattern is considered an equivalent risk to ST-elevation myocardial infarction, requiring immediate revascularization. The peaked T-waves can be confused with hyperkalemia, an electrolyte abnormality necessitating urgent intervention.
Eventhough infrequent, untreated hyperkalemia may present as ST-segment elevation on an ECG. The proposed mechanism is that elevated levels of potassium shorten the action potential in phase three repolarization resulting in ST-segment elevation.
This study aims to raise awareness of symptomatic acute hyperkalemia with ECG findings mimicking myocardial infarction in order to administer correct therapy and prevent unnecessary coronary interventions.
Case Illustration: A 52-year-old male was admitted to our emergency department with a complaint of crushing chest pain persisting for 3 days worsening an hour prior, accompanied by nausea, cold sweats, and vomiting.
The ECG upon admission showed ST-segment elevation in V1-V3. Upsloping ST-depression and peaked T-waves were present in V4-V6 which resembled de Winter pattern, potentially concealed hyperkalemia. Downsloping ST-depression and T-inversion were seen in the inferior leads. Cardiac enzyme examinations were not carried out due to inadequate facilities, thus we could not confirm if it was a true STEMI.
Abnormal laboratory findings were hyperkalemia (7.72 mmol/L) and increased creatinine (8.98 mg/dl). Primary PCI or angiography cannot be performed because of elevated creatinine levels. We made approaches to treat this patient with unstable angina and hyperkalemia. He was given dual antiplatelet therapy of aspirin and clopidogrel, intravenous anticoagulant, nitroglycerin, and statin. We admitted him to our ICU and consulted the internist. Serum potassium levels was being corrected and creatinine levels returned back to normal (0.79 mg/dl). On serial ECG, ST-elevation and peaked T-waves were resolved. Echocardiography showed no sign of wall motion abnormalities. The patient covered 504 meters on 6 minutes walking test. We carefully discharged him after another serial ECG disclosed no worrying pattern.
Conclusion: Quick evaluation of serum potassium levels in this scenario saved the need for unnecessary catheterization lab activation and more importantly, averted the patient from malignant arrhytmia in case of treatment delay. Aggressive treatment of hyperkalemia resulted in successful reduction of serum potassium level. Notably, ECG pattern reverted back to baseline without any evidence of pseudoinfarction pattern.

Keywords: Hyperkalemia. Pseudo STEMI, ST-segment elevation, de Winter, peaked T-wave.

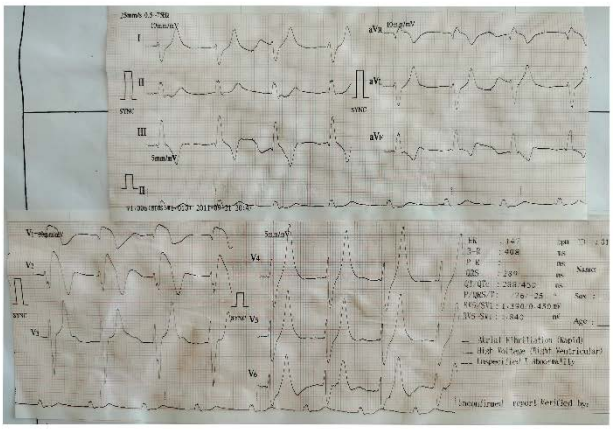

Fig 1. ECG upon Admission

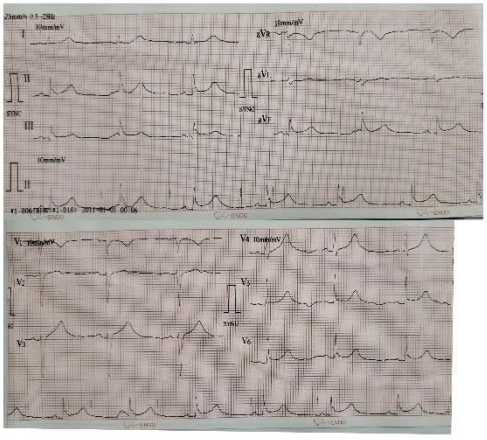

Fig 2. ECG after Potassium Correction

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Isorhytmic AV Dissociation Resembles Accelerated Junctional Rhytm in WPW Syndrome: a Case Report

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Background: Wolff-Parkinson-White (WPW) syndrome is a preexcitation syndrome that arises from abnormal electrical conduction of the heart through the accessory pathway. Atrioventricular (AV) dissociation is a cardiac conduction disorder with the atria and ventricles beating independently of each other. Detailed electrocardiogram (ECG) review is necessary to identify the source and type ranging from benign to lethal AV dissociation. In this case, we reported an unusual change in the ECG picture in a patient with WPW syndrome, namely isorhythmic AV dissociation with junctional rhythm.

Case Illustration: A man came to the emergency room with the main complaint of weakness since 2 days ago. A 12 lead ECG examination showed SVT with a rate of 214 bpm. After injection of diltiazem, the SVT changed to a junctional rhythm with a rate of 100x/minute. However, the P waves after the QRS complex in this junctional rhythm do not look like retrograde P waves which should be inversions, but positive P waves are seen in leads II,III,AvF and V2-V6 which indicates that the P waves are very likely to originate from the sinus, with a P-P wave rate of 96x/minute and R-R 100x/minute. It is difficult to distinguish whether the P wave after the QRS complex in V1-V3 is a competed sinus P wave or an epsilon wave in Leads V1-V3. Echocardiography examination showed no anatomical and functional abnormalities in the right ventricle. On the 3rd day of treatment the ECG changed to Sinus Rhythm with Preexcitation.

Conclusion: This patient with an AVRT (Atrioventricular Reentrant Tachycardia). After diltiazem injection, the ECG changed to Accelerated Junctional Rhythm with the appearance of P waves originating from the sinus. The P wave V1-V3 and deep inversion of T wave return to normal and disappear when the rhythm changes to sinus with preexcitation. Echocardiography was normal, so this is not epsilon caused by ARVD (Arrhythmogenic Right Ventricular Dysplasia) but an isorhythmic AV dissociation. Eventually, the slight difference in rates between the two pacemaker site allows one site to discharge early enough to capture the other site, breaking the cycle and resulting in a return to sinus rhythm.

Keyword: Accelerated Junctional Rhytm, Epsilon, Isorhytmic AV Dissociation, Retrogade P wave, WPW Syndrome

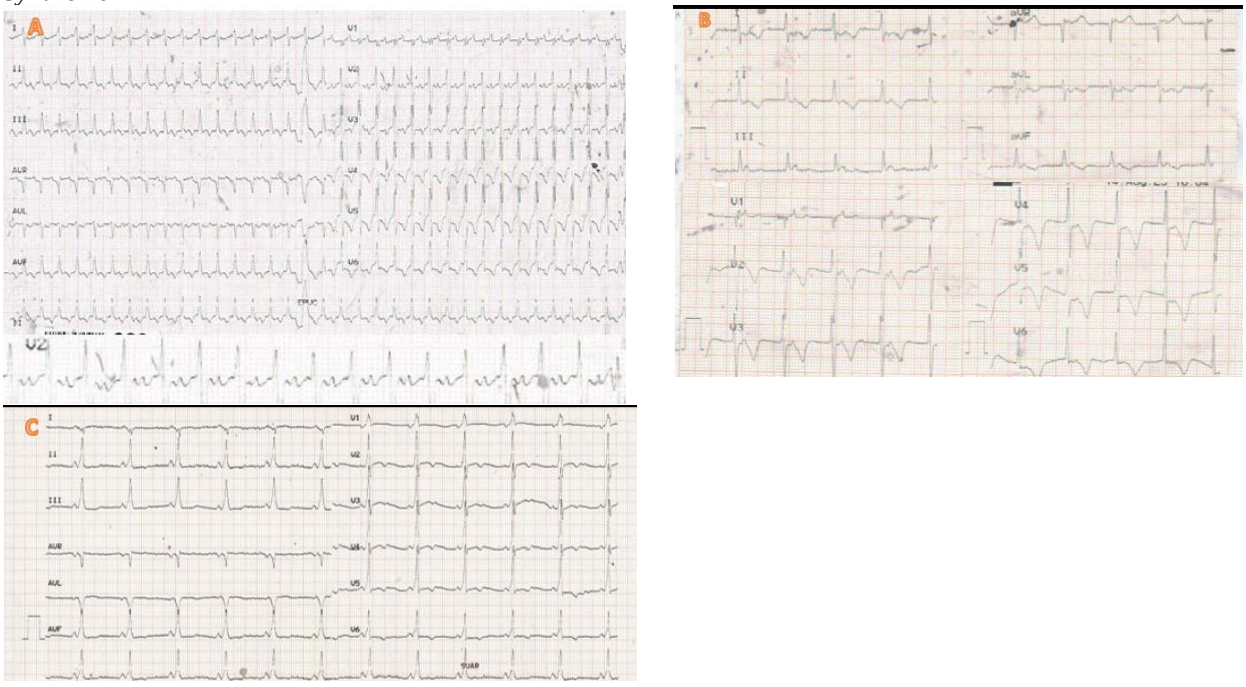

Figure 1 (A) ECG in the emergency room shows narrow QRS tachycardia rhythm, rate 214x/minute, normo axis, RP interval >80 ms (RP interval 140ms). **(B)** ECG Post injection of diltiazem 20 mg, rhythm changes to Accelerated Junctional rhythm with Positive P Wave after QRS complex, with ST Depression V4-V6 and I, AvL and T inversion in V2-V6, I, II, AvL. **(C)** The ECG on the 3rd day of treatment shows a return to the Sinus Rhythm, shortening of the PR Interval accompanied by a “delta wave” appearance.

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Wolff-Parkinson-White Syndrome with Multiple Accessory Pathways: A Case Report

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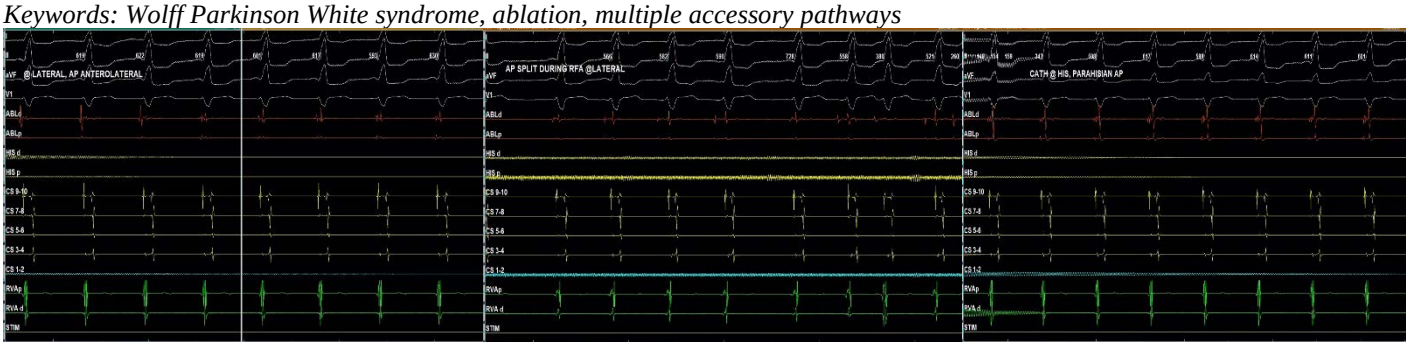
Background: Wolff-Parkinson-White syndrome is a condition that occurs due to an abnormal extra electrical pathway between atria and ventricles. The abnormal pathway connects atria and ventricles at a site other than the AV node. Patients with Wolff-Parkinson-White syndrome rarely have multiple accessory pathways. Parahisian is one of unusual locations of accessory pathways that are associated with a His bundle at its atrial or ventricular insertion site. Radiofrequency ablation (RFA) is first-line treatment of choice for people with symptomatic and accessory pathways in Wolff-Parkinson-White syndrome.

Case Illustration: A 58-year-old woman with recurrent palpitations. No SVT was documented. ECG showed sinus rhythm with short PR interval, positive delta waves at lead II, III, aVF, and isoelectric delta waves at lead V1 which suggest Wolff-Parkinson-White syndrome with right anterolateral pathway. Electrophysiology study showed earliest ventricular activation at anterolateral tricuspid annulus. Atrial stimulation induced narrow complex tachycardia with TCL 480 ms. Right ventricular overdrive pacing showed VAV response with VA interval of 133 ms. His synchronized pacing showed no reset. RFA was done in that area, the AV fusion was separated but tachycardia of the same TCL was still induced. Re-mapping the accessories pathway showed most AV fusion at the Parahisian area. It was decided not to perform ablation at that area due to proximity with His bundle. Mapping at the anterolateral TV annulus showed intermittent AV fusion suggesting multiple accessory pathways in this patient. Unfortunately, the ablation was stopped due to patient preference.

Conclusion: Multiple accessory pathways in patients with Wolff-Parkinson-White syndrome are rare and one of the criteria for symptomatic patients to get treatment with radiofrequency ablation. Parahisian ablation is associated with high risk of AV block. Alternative approaches may be considered such as superior venous approach and/or reverse loop within the right ventricular inflow.

Keywords: Wolff Parkinson White syndrome, ablation, multiple accessory pathways



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Syncope with Premature Ventricular Complexes, How to Approach it?

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Background Premature ventricular complexes are cardiac electrical conduction abnormalities originating in the ventricles that cause ventricular contractions to be separated from their original rhythm. PVCs are common in the population, but their symptoms and prognosis vary widely. The clinical presentation can be benign such as asymptomatic or symptoms such as palpitations, lightheadedness, chest discomfort or skip beats. The malignant presentation may include syncope or sudden cardiac death.
Case Illustration A 58-year-old woman came to the emergency room with complaints of palpitation and shortness of breath. Two hours earlier she was witnessed by the family suddenly becoming unconscious while everyone was at home. The patient has no history of hypertension and diabetes. There are no sudden deaths history in the family. She has a history of arrhythmia, but in the last 6 months she had not received regular treatment. She experienced syncope for the first time. On physical examination was unremarkable with pulse 110 bpm and pulse deficit was noted. When an EKG was performed, the rhythm was sinus rhythm with a rate of 130 bpm regularly irregular, some PVCs couplet with a coupling interval of 340ms, an interpolated PVC at the 7th PVC and possibly originating from the RVOT. Laboratory results and chest x-ray were unremarkable. She was given bisoprolol 2.5mg o.d. Palpitations were reduced after administering bisoprolol. We suspect that the syncope experienced by the patient was due to arrhythmia.

Conclusion A history of syncope with evidence of PVC couplets led to the ideas that the patient's syncope could be caused by an arrhythmia. Further examinations such as tilt table testing, holter monitoring to determine the PVC burden, echocardiography and electrophysiological studies are required because of the patient's complaints of syncope. Even so, the syncope that occurs must still be distinguished whether it really originates from the cardiac or non-cardiac.

Keywords: PVC, syncope, palpitation, ablation, interpolated PVC

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Exercise-induced atrial ectopic rhythm as a predictor for myocardial infarction

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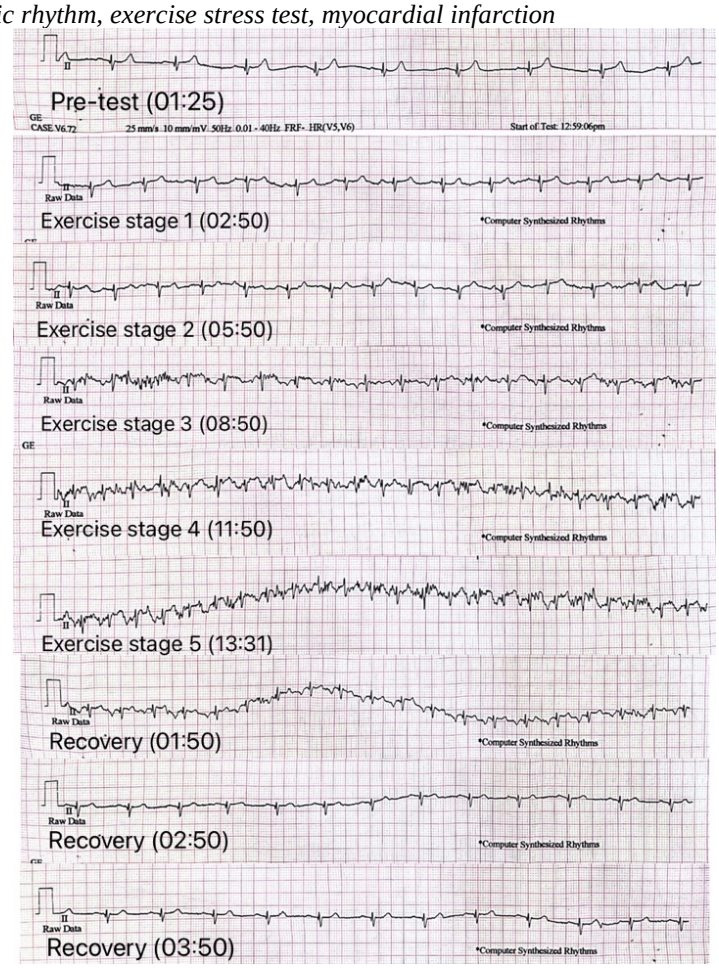
Background: Exercise stress test is one of the most commonly used methods by clinicians to identify myocardial ischemia and evaluate its response to activity. Atrial or ventricular arrhythmias during treadmill tests were frequently found in people over 50 with documented cardiovascular disease, which were linked to an abnormal resting ECG. According to a large retrospective study by Bunch et al., atrial ectopy may indicate a higher risk of myocardial infarction. Currently, there are very few studies that address exercise-induced atrial ectopy. Therefore, the purpose of this case is to support the theory that atrial ectopy discovered during stress test may be a predictor of a higher risk of myocardial infarction.

Case Illustration: A 47-year-old man visited the hospital for a routine medical check-up. He didn't have any previous stable angina, near syncope, or syncope histories. The patient was subsequently put through a Bruce Protocol exercise stress test. The pre-test ECG showed a sinus rhythm with small P waves, left anterior fascicular block, left axis deviation, and a heart rate of 61 beats per minute. As the stress test progressed, the atrial ectopic beat was discovered. The ECG reverted to sinus rhythm during recovery. Symptoms, hypertension, and other arrhythmias were not detected during exercise.

Conclusion: The patient in this case report did not have any myocardial infarction symptoms or risk factors. However, atrial ectopy was observed during the stress test. Other symptoms and examinations should be closely followed in this patient. Exercise-induced atrial ectopic rhythm is a novel finding that must be further investigated, as this case is one of the rare outcomes in stress tests.

Keyword: atrial ectopic rhythm, exercise stress test, myocardial infarction



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Brugada Syndrome Presenting with Monomorphic Ventricular Tachycardia: a Rare Case in Rural Setting Area

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Background: Brugada Syndrome (BrS) is inherited cardiac channelopathy characterised by a typical electrocardiogram (ECG) pattern and predisposition to develop malignant ventricular tachyarrhythmias that can lead to syncope and sudden cardiac arrest. Risk stratification in patients with BrS remains challenging, it is vital for clinicians to be able to accurately identify and manage patients suspected of having BrS. We present a case of a young patient with no significant past medical history who presented to the emergency department (ED) with first-time signs and symptoms consistent with Brugada syndrome.

Case Illustration: A 29-year-old male was brought to ED for history of syncope. He complained of chest pain and palpitation approximately 9 hours before being admitted to the hospital. The patient had a positive family history of sudden cardiac death. He appeared weak and fully conscious, with blood pressure 80/50 mmHg, pulse 190 x/minute, breathing frequency 26x/minute with O2 saturation of 96%, and axillar temperature of 36,5^{o}C. ECG showed monomorphic ventricular tachycardia. The patient was then given electrical cardioversion to treat hemodynamically unstable wide complex tachycardia. The ECG returned to normal sinus rhythm with coved ST-segment elevation in V1-V3 after being converted with 100 Joules on the first attempt. There was no significant laboratory result and troponin was negative. The patient's presenting symptoms and findings found on ECG ultimately corroborated a diagnosis of Brugada syndrome with a type 1 pattern.

Conclusion: Brugada syndrome is a rare genetic ion channelopathy predisposing patients to ventricular arrhythmias. Assessment of clinical and the 12-lead ECG plays a pivotal role in the diagnosis. Management in BrS requires an understanding of the pharmacologic and interventional treatment modalities.

Keyword: Brugada Syndrome; Ventricular Tachycardia; Electrocardiography

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Epicardial Pacing Lead Implentation in Complete Heart Block as a Manifestation of Cardiac Mass

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Background: Patients with atrioventricular (AV) conduction may range from being asymptomatic to severe symptoms due to bradycardia. Pacemaker is essential, especially in a complete AV block. While transvenous lead pacemakers are common due to their minimal invasiveness and effectiveness, they may be infeasible in cases with comorbidities or anatomical changes. This report presents a rare case of a cardiac mass obstructing the AV conducting pathway, where the standard transvenous approach was not suitable. In such complex scenarios, epicardial pacemaker implantation emerges as the most suitable and effective option.

Case Illustration: A 48-year-old male presented with complaints of weight loss and dyspnea that had persisted for six months, with symptoms worsening over the past week. Initial transthoracic echocardiography revealed the presence of an intracardiac mass involving the right atrium, interatrial septum, and left atrium, along with a suspicion of cor triatriatum. Further diagnostic evaluation using a CT scan revealed a malignant, enhancing solid mass filling the interatrial septum, which extended to the right atrium, left atrium, right pulmonary vein, and inferior vena cava. The mass was irregular in shape and measured approximately 6.0 x 6.0 x 8.2 cm. This mass disrupted the continuity of the AV conducting pathway, resulting in a complete heart block and hemodynamic instability.
We deemed immediate pacemaker implantation necessary due to the urgency of the situation. Considering the complications posed by the large tumor in the right atrium, which could impede transvenous lead delivery, we opted for surgical epicardial lead implantation. The pacemaker implantation was successfully completed without any complications, leading to a significant improvement in the patient's hemodynamic status. The patient's condition stabilized postoperatively, and he was discharged from the hospital 5 days after surgery, demonstrating marked clinical improvement. This case highlights the efficacy of epicardial pacemaker implantation in managing complex cases where standard transvenous lead placement is not suitable.

Conclusion: Epicardial lead placement should be considered when primary transvenous lead placement cannot be performed in challenging cases, such as in the case where a cardiac mass obstructs the AV conducting pathway.

Keywords: epicardial pacemaker; complete atrioventricular block; intracardiac mass; cardiac mass obstruction.

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Lost in the Coronary Sinus: Unveiling the Mystery of Misplaced Pacemaker Leads

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Background: Incorrect placement of cardiac pacing leads can result in suboptimal device performance and adverse clinical outcomes. This case report highlights the challenges and resolution associated with the misplacement of a temporary pacemaker lead into the coronary sinus, and its subsequent correction.

Case Illustration: A 64-year-old retired male presented with symptoms of profound weakness, nausea, and cold sweats. He had a history of coronary artery disease, type 2 diabetes mellitus, dyslipidaemia, and chronic kidney disease. He was admitted to the emergency department with severe fatigue and dizziness, particularly upon changing positions. Initial assessment revealed symptomatic bradycardia with a heart rate of 46 bpm, blood pressure of 95/54 mmHg, and signs of heart failure. ECG showed a junctional rhythm rate of 42 bpm and left anterior descending, and Q waves in leads V2-V5.
The patient underwent temporary pacemaker insertion due to symptomatic bradycardia. Post-procedure ECG revealed inferior axis with RBBB and unsatisfactory pacing results, prompting further investigation. Imaging and subsequent review identified that the pacing lead had been inadvertently placed into the coronary sinus. This misplacement explained the suboptimal pacing and persistent bradycardia.
Corrective intervention was undertaken, involving the repositioning of the lead to the right ventricular outflow tract. The procedure was challenging due to the lead's fixation within the coronary sinus, necessitating careful manipulation and re-advancement. Following successful repositioning, post-procedural ECG confirmed effective pacing with inferior axis, venctricular pacing, LBBB, and improved clinical symptoms.

Conclusion: This case underscores the importance of accurate lead placement in cardiac pacing to ensure effective device function and patient outcomes. Misplacement of the pacemaker lead into the coronary sinus is a rare but significant complication that can be rectified with prompt recognition and appropriate corrective procedures. This highlights the need for vigilant intraoperative monitoring and post-procedural verification to avoid similar occurrences.

Keywords: Pacemaker lead misplacement, coronary sinus, symptomatic bradycardia, elderly patient, lead repositioning, cardiac device complications

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Arrhythmia Worsened by Aripiprazole in Elderly Patient in Rural Mental Hospital : A Case Report

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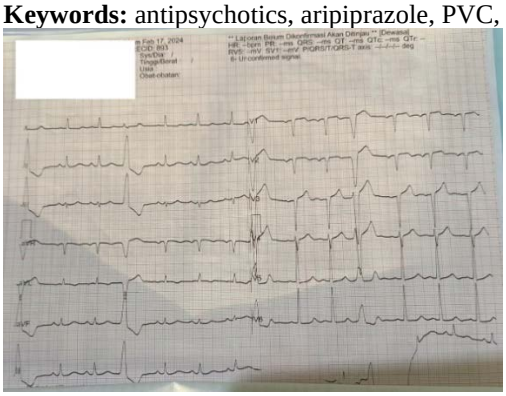
Background: Aripiprazole has been known as the antipsychotic drug that has the least cardiovascular effects in addition to having a low incidence of QT prolongation. Still, here we reported the effects of aripiprazole in elderly patient with PVCs.

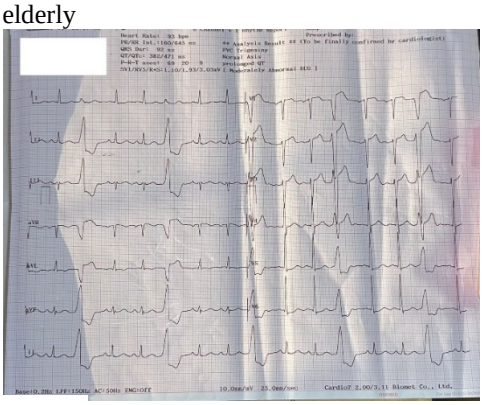
Case illustration: Mr. S, 66 years old, was admitted with unspecified schizophrenia. He had no physical complaints and no recorded morbidities; however, his ECG showed PVCs (shortest CI: 440 msec) and prolonged QT (QTc: 470 msec), and his chest x-ray presented with cardiomegaly. He was given the treatment of aripiprazole 2,5 mg twice a day and lorazepam 0,5 mg once a day. Due to an adverse event, Mr. S was given an incorrect dose of aripiprazole, as much as 10 mg, for two doses. After the wrong dose, Mr. S presented with nausea and lightheadedness. An ECG showed a trigeminy PVCs (shortest CI: 380 msec) and prolonged QT (QTc: 471 msec). As soon as the aripiprazole was stopped, an ECG was retaken, and it showed occasional PVCs (shortest CI: 520 msec) and QTc of 465 msec. The psychiatrist then decided to give a 2.5 mg dose of aripiprazole and monitor the ECG, which later showed trigeminy PVCs (shortest CI: 400 msec) with a QTc 470 msec. The patient was then stopped from receiving aripiprazole.

Conclusion: Typical antipsychotics, through sodium and potassium channel blockade, cause QT interval prolongation, which can lead to arrhythmia. There was no evidence to suggest that aripiprazole directly blocks sodium or potassium channels and is considered to have a lower risk of cardiac arrhythmias. Aripiprazole's risk for arrhythmia would likely be caused by its effect on dopamine and serotonin receptors, which modulate ion channels indirectly and cause sympathetic nervous system activation. Because of changes in organ function, metabolism, and other age-related factors, elderly people are more vulnerable to side effects; therefore, it's critical to evaluate the patient's cardiovascular risk factors and closely monitor any side effects when prescribing antipsychotics, including aripiprazole.

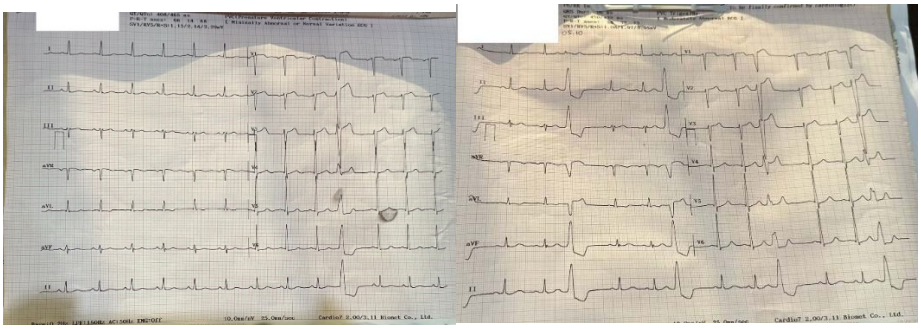
Keywords: antipsychotics, aripiprazole, PVC, arrhythmia, elderly



ECG on Admission



ECC after 10 mg aripiprazole



ECG after Aripiprazole was stopped ECG after 2,5 mg Aripiprazole

Figure 1

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Dual-Chamber Pacemaker Implantation for Bradycardia-Induced Polymorphic Ventricular Tachycardia in a Patient with Recurrent Syncope: A Case Report

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Background: Polymorphic ventricular tachycardia (PVT) is a life-threatening form of ventricular tachyarrhythmia characterized by a varying QRS pattern. This condition can resolve itself on its own, potentially causing syncope. Identifying the underlying cause of PVT is crucial since different arrhythmias require specific treatments. Bradycardia is a well-recognized risk factor for PVT, primarily because it can lead to a prolonged QT interval. PVT has been documented in various conditions associated with bradycardia, such as atrioventricular (AV) block, and is marked by episodes of syncope and seizures.

Case Illustration: A 76-year-old woman with a history of recurrent syncope that has worsened over the past two months was brought to the emergency department following seizure episodes. The seizures lasted for 1 minute, after which she regained consciousness but remained lethargic. The resting ECG showed TAVB at 44 bpm with junctional escape rhythm and frequent PVCs. During a seizure, ECGs showed PVT with 224 bpm. Laboratory tests indicated a potassium level of 2.7. During a seizure, the patient was treated with 150 mg of amiodarone intravenously over 30 minutes, followed by 2 mg of midazolam IV. The patient was transferred to the High Care Unit, where seizures persisted alongside PVT and treated with 1 g MgSO4 IV and 2 mg midazolam IV. Maintenance therapy included diazepam 5 mg orally twice daily and potassium correction with KCL drip 50 meq/hour. The patient was scheduled for implantation of a temporary pacemaker, followed by successful implantation of a dual chamber pacemaker with initial settings of DDDR, base rate 60 bpm, and output 3.5 mV. Ventricular threshold 0.4 V, impedance 778 ohms. Atrial threshold 1.1 V, impedance 936 ohms.

Conclusion: Bradycardia, particularly in atrioventricular block, significantly increases the risk of PVT by prolonging the QT interval, potentially causing syncope or seizures. Given the rarity of this condition, dual-chamber pacemaker implantation and intravenous therapy underscores the importance of tailored medical care in managing bradycardia-induced PVT.

Keywords: Bradycardia, Polymorphic VT, Pacemaker

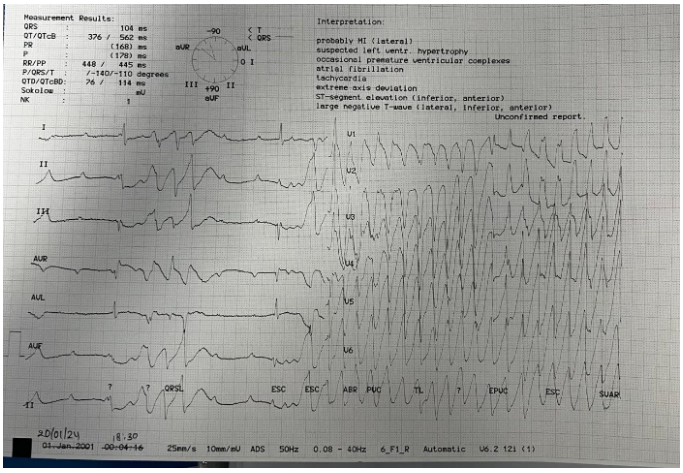

Fig 1. ECG : Polymorphic Tachycardia during Seizure Episode

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Approach for Atrial Fibrillation with Slow Ventricular Response in Limited-Resource Setting: A Case Report

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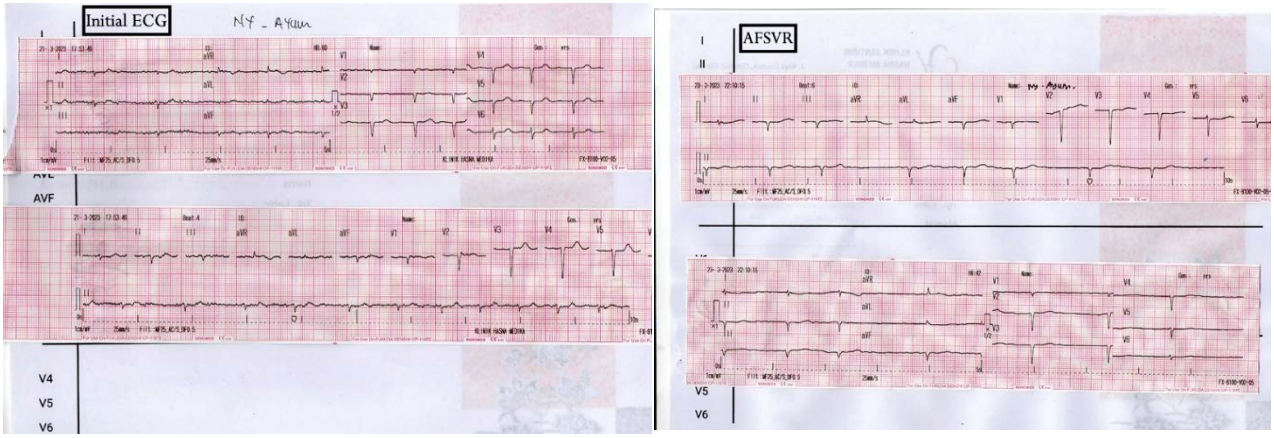
Background: Atrial fibrillation with slow ventricular response(AFSVR) is an arrhythmia with uncoordinated atrial electrical activation with ventricular rate <60 bpm. AFSVR may lead to intracardiac hemodynamic changes and thrombus formation. AFSVR presents unique challenges in management, especially in settings with limited resources.

Case Illustration: A 63-year-old female patient presented to the emergency room with a chief complaint of left-sided chest pain that started 10 hours prior. Upon examination, blood pressure(BP) was 181/77mmHg. Initial ECG revealed AF and an anterior old myocardial infarction(OMI). The troponin T level was <50. The patient was diagnosed with unstable angina pectoris and urgent hypertension. During inpatient care; subcutaneous fondaparinux, intravenous(IV) nitroglycerin drip, dual antiplatelet therapy(DAPT), IV furosemide, ramipril, and statin were administered. On the second day, the patient reported hematuria. Consequently; fondaparinux, DAPT, and nitroglycerine were discontinued. By the third day, she complained of continued hematuria and feeling agitated. Upon examination, the BP was 84/52mmHg with a heart rate(HR) of 42bpm. An ECG revealed atrial AFSVR, a superior axis, and an anteroseptal OMI. A crystalloid bolus of 200cc was administered, resulting in an increase in BP to 106/60mmHg and HR to 49bpm. An IV dobutamine drip was initiated to raise BP and HR. With dobutamine at 6 mcg/kg/min, BP was 81/47mmHg with HR of 42bpm. Subsequently, IV dobutamine, IV atropine sulfate, IV tranexamic acid, and IV vitamin K were administered. Despite these interventions, BP and HR remained low at 82/60mmHg and 61bpm. Because of that, the patient was referred to a tertiary hospital for pacemaker placement.

Conclusion: This case underscores the challenges in managing patients with complex cardiovascular conditions. The progression to significant bradycardia and hypotension required aggressive intervention with the initial use of crystalloids, followed by inotropic support with dobutamine and atropine. Despite our efforts, the patient's persistent hemodynamic instability necessitated transfer to a tertiary centre for pacemaker placement.

Keywords: Atrial Fibrillation Slow Ventricular Response, Pacemaker, Myocardial Infarction



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Salbutamol-Induced Supraventricular Tachycardia in Severe Asthma: Case Report

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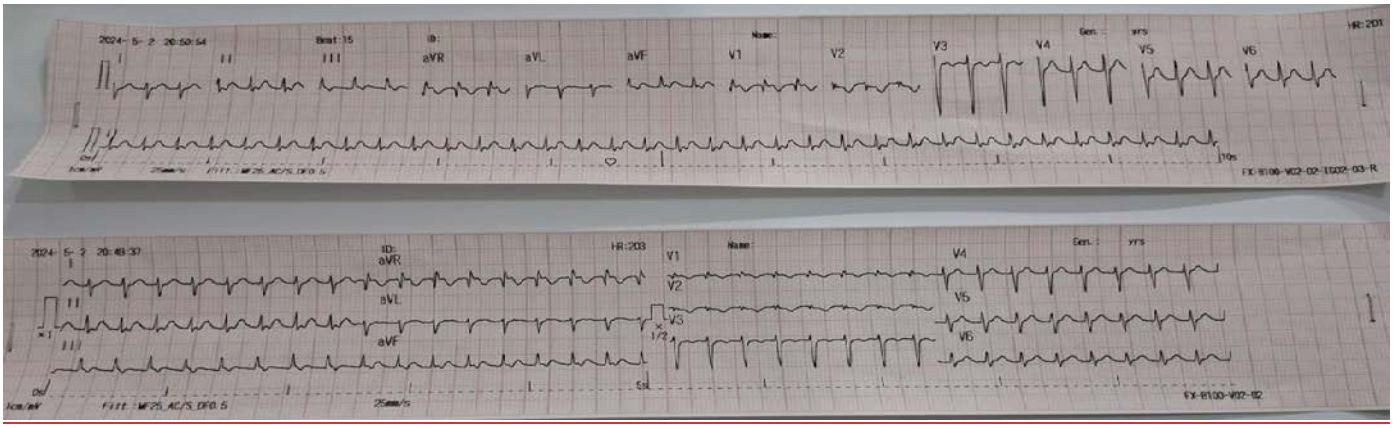
Background: Acute asthma exacerbation is a common case in the emergency department (ED). According to Riskesdas 2018, around 2.4% people in Indonesia have asthma. Combination of Short-Acting Beta$_2$ Agonist (SABA) and Inhaled Corticosteroid (ICS) often use to treat acute asthma exacerbation. Using salbutamol can cause cardiovascular side effects such as tachycardia even serious arrhythmias, although this is rare. The incidence and risk factor of salbutamol induced supraventricular tachycardia remain unknown.

Case Illustration: A 14-year-old male admitted to ED after 1 day of dyspnea and cough. He had a history of asthma before. His vitals were unstable with heart rate (HR) 130/m, respiratory rate (RR) 40/m, SaO2 90%. On physical examination found wheezing in right and left lungs. On laboratory examination found leucocytosis 12.600 and severe respiratory distress with P/F ratio 65, other routine blood count and electrolyte were normal. He was intubated and sedated with Midazolam, inhaled SABA and ICS were given. After 5 days nebulized with SABA and ICS, his HR was 206/m, blood pressure 146/73, ECG showed SVT, wheezing slightly audible, rhonchi and cold extremity were not found. He was given an amiodarone loading dose and continuous infusion for 24 hours. Electrolyte and transaminase enzymes were normal after re-evaluation. Inhaled SABA stopped and only treated with ICS. Amiodarone oral given for rhythm control. He had no further episodes of SVT. He was discharged 3 weeks later.

Conclusion: SVT is a rare cardiovascular side effect from usage of inhaled SABA. Early diagnosis and prompt treatment can give a good outcome to the patient. Further research is needed to know about the risk factor SABA induced SVT and how to minimize the risk.

Keyword: Short-Acting Beta$_2$ Agonist, Supraventricular Tachycardia



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Unseen Complexity: Atrial Flutter and Multilevel Atrioventricular Block – Closing the Recognition Gap

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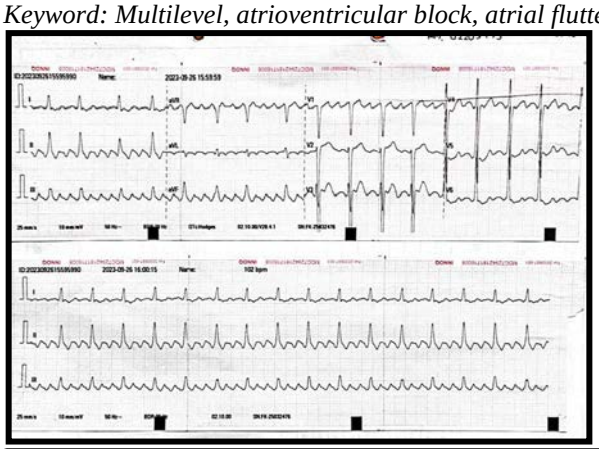
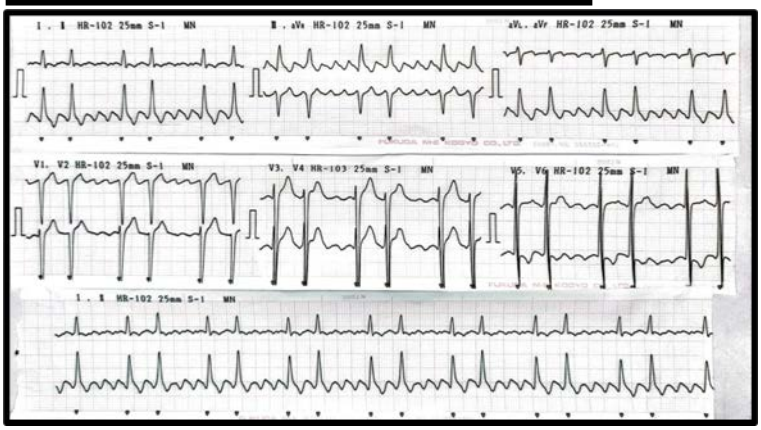
Background: Multilevel atrioventricular block frequently manifests but is frequently overlooked in cases of atrial flutter. The variable atrioventricular conduction ratios observed during atrial flutter and other atrial tachyarrhythmias are commonly attributed to delays or blockages occurring at multiple levels within the conduction system.

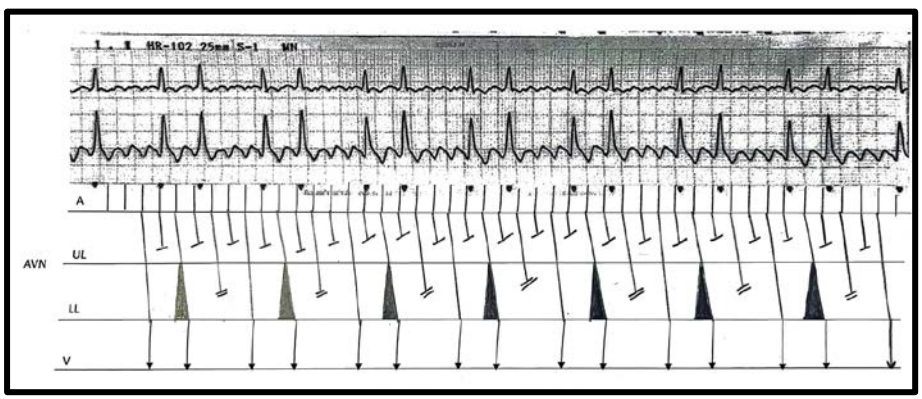
Case Illustration: 66 year-old-male came to Emergency Department due to sudden hemiparesis and was found with ischemic stroke from Head CT Scan finding. The patient was consulted from Neurology to Cardiology department due to irregularity of ECG findings and near syncope complaints during hospitalization. First ECG showed typical atrial flutter with 3:1 conduction and regular ventricular responses. Second ECG depicted typical atrial flutter with variable conduction with 2:1 and 4:1 pattern conducted in the rate of 100 bpm. Ladder diagram demonstrates that this finding can be caused by conduction delay or block at two or more levels of the conduction system. A 2:1 conduction ratio at the upper level of the AV node and 2:1 conduction of flutter impulses at the second level of block result in net 4:1 conduction at the ventricular level. The 4:1 conduction ratio creates R-R intervals of sufficient length to allow full recovery of the distal conduction system. Proximal conduction through the His-Purkinje system is not recorded on a surface electrocardiogram. This phenomenon was associated with concealed conduction and occurs under a variety of clinical circumstances.

Conclusion: Multilevel atrioventricular block should be considered in the finding of group beating with variant R-R intervals during Atrial Flutter.

Keyword: Multilevel, atrioventricular block, atrial flutter







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Dopamine Effect on Sinus Node Dysfunction Patient at a Secondary Hospital : A Case Report

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Background: Sinus node dysfunction, also known as sick sinus syndrome, is a condition characterized by the inability of the sinoatrial node to generate and propagate electrical impulses effectively, leading to irregular heart rhythms. This condition can result in significant clinical symptoms and often necessitates the implantation of pacemakers.

Case Illustration: A 55-year-old male with a history of vertigo, ischemic stroke, and type 2 diabetes mellitus presented to the emergency department of a secondary hospital with lightheadedness, syncope, dyspnea on exercise, and atypical chest pain. Vital signs showed bradycardia, while manual heart rate examination revealed a slow and irregular pulse. ECG showed type II second-degree sinoatrial exit block, and the laboratory results showed a slightly increased CK-MB. The patient was treated with atropine sulfate injection and fixed-dose dopamine infusion while waiting for referral to a tertiary hospital for pacemaker implantation. Sixteen hours later, the ECG showed a conversion into sinus rhythm, and the patient was referred for angiography at a tertiary hospital where insignificant lesions in the coronary arteries were found.

Conclusion: Sinus node dysfunction is a multifaceted condition with various intrinsic and extrinsic causes. Intrinsic factors like degenerative fibrosis are more common in older individuals, while extrinsic factors such as medications and metabolic abnormalities are often reversible. Understanding these diverse mechanisms is essential for developing targeted treatments and improving patient outcomes. Dopamine plays a significant role in managing sinus node dysfunction. Its regulatory mechanisms, including retrograde transfer and DAT inhibition, highlight its complex influence on cardiovascular health. Further research is needed to fully understand these mechanisms and their implications for treating sinus node dysfunction.

Keyword : Sinus node dysfunction, Sinoatrial exit block, Dopamine

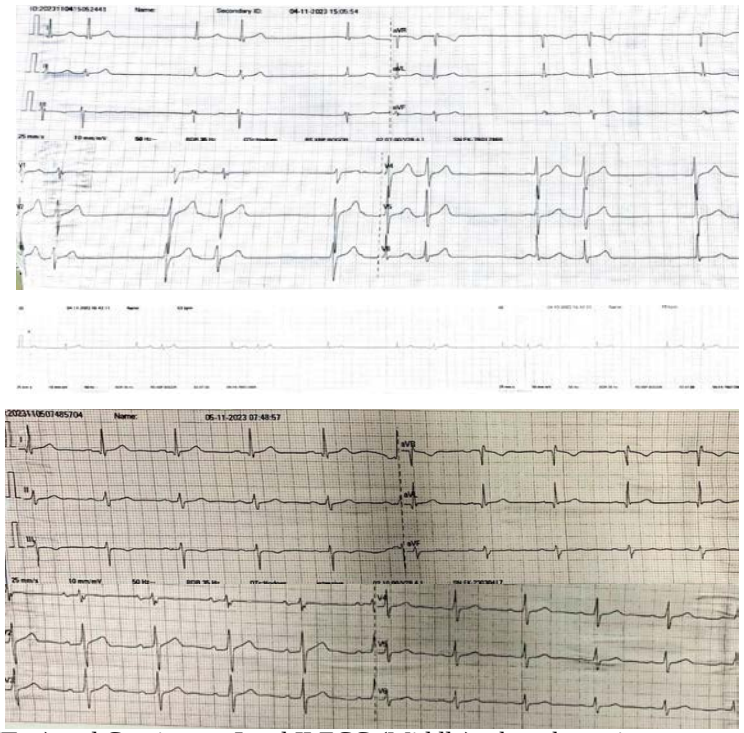

Figure: 12-Lead ECG (Top) and Continuous Lead II ECG (Middle) when the patient presented to the emergency department, and 12-Lead ECG 16 hours post-dopamine infusion (Bottom)

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Anterior and Inferior ST Elevation, yet it wasn't a Coronary Artery Disease: A Case Report

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Background: ST-Elevation Myocardial Infarction (STEMI) is a type of acute coronary syndrome (ACS) that occurs when the coronary artery is completely blocked. This can become an emergency phenomenon if not recognized and treated quickly. However, some conditions like myocarditis can also be represented with ST Elevation in ECG Examination. So an appropriate diagnostic algorithm is also needed to differentiate between these several conditions.

Case Illustration: A 40 years old male came to the emergency room with shortness of breath. On examination, BP 110/70, HR 120 bpm, rhonchi (+), and wheezing (-). The ECG examination results showed there was ST elevation on Lead III, V1 - V4 and aVF. The Chest XRay result showed cardiomegaly signs. Due to the suspicion of an infarction, Diagnostic Coronary Angiography was performed and the results were normal vascular without occlusion. Even so, the patient still experienced hypotension and tachycardia until that time. Then the Echocardiography was performed and the result was EF 20% (LV IDd : 5,01 cm) with diastolic restriction. Patient's laboratory tests show leukocytosis. Patient received treatment in intensive care rooms with close monitoring and optimal care according to guidelines. On the 17th day of treatment, the patient's EF increased exceeding 40%, and the hemodynamic was stable. The patient was sent home because his condition had stabilized and the diagnosis was highly suspected as myocarditis.
Myocarditis refers to inflammation of the heart muscle. Normally, the ST segment on ECG represents the period between ventricular depolarization and repolarization. When the heart muscle becomes inflamed, it can cause a current of injury that was reflected as an ST segment elevation on the ECG results. These ST elevation seen in myocarditis was diffuse and could be seen in multiple leads, reflecting the widespread inflammation. This is different from the ST elevation seen in myocardial infarction, which was localized to specific leads that correspond to the area of the heart supplied by the blocked coronary artery.

Conclusions: Even though ST elevation often indicates an infarction due to coronary artery occlusion, the diagnosis algorithm for heart disease still must be implemented correctly so the therapy could be appropriate.

Keyword: ST-Elevation Myocardial Infarction, Myocarditis, Acute Coronary Syndrome

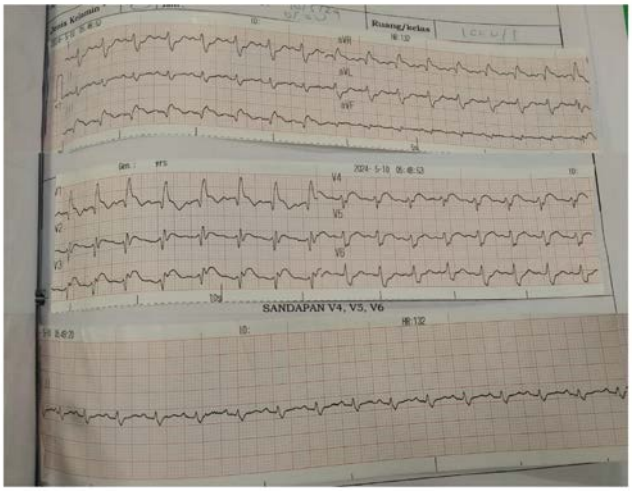

Figure I. ECG Examination result when patient entering emergency room

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Unusual 2:1 block: to pace or not to pace?

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Background: Defining the site of block in AV block is important to define whether pacemaker implantation was necessary or not. Sometimes, surface ECG can not define the site of block such as in 2:1 block. In this case report, we present 2 cases of 2:1 block with unusual EGM data and need several manuvers and treadmill data to confirm the site of block

Case Illustration: First case is a-69-year-old female referred due to near syncope and bradycardia. No chest pain, fever, or SCD history. Her surface ECG was unremarkable with only prolonged PR interval (300 ms) and wide QRS (304 ms). Sinus node function was normal EP study showed 2:1 block with drop beat showed A not followed by H, with A-H 106 ms, HBE 28 ms, HV 178 ms, suggesting suprahisian block. However, during programmed atrial pacing PCL 500 ms, there is worsen block from 2:1 to 3:1 with morphology changes of drop beat from A not followed by H to A-H not followed by V during 3:1 block suggesting intermittent infra-hisian block. Treadmill showed the block was worsen during exercise supporting diagnosis of infrahisian block.

Second case is a-41-year-old female referred with dizziness. Her surface ECG was normal but holter monitoring showed intermittent 2:1 block with episode of second-degree type II block. EP study showed AH 106 ms, HV 46 ms, HBE 20 ms. Sinus node function was normal. Programmed atrial pacing showed 2:1 block with suprahisian block pattern (A not followed by H-V). However, after isoproterenol and atropine, the conduction was strangely worsened from 1:1 to 2:1 eventhough EGM data suggesting suprahisian block. Treadmill showed worsening conduction from 1:1 conduction to Wenckebach block during exercise, favoring diagnosis of suprahisian block/proximal his block.

Conclusions: Defining the site of block is important. Sometimes EP study was needed to confirm the site of block. Sometimes, basic EGM data alone can not define the site of block. Here, we present several manuvers and test to confirm the site of block so that we can provide the best management for the patient.

2:1 block with AH interval 106 ms, HBE 28 ms, HV interval 178 ms. Drop beat showed A not followed by H interval suggesting suprahisian block

Figure 1. Baseline EGM in first patient showed 2:1 conduction with suggestive suprahisian block

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Taming the Electrical Storm: A Follow-up Approach to Managing Arrhythmias in Ebstein's Anomaly

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Background: Ebstein's anomaly (EA) is a rare heart defect that occurs when the tricuspid valve (TV) is shifted downward toward the right atrium (RA). This can cause a number of problems, including arrhythmias. They exhibit a greater rate of recurrence compared to those with normal hearts. Although surgery is a complex procedure, it can correct EA. Catheter ablation is a newer treatment option that is less invasive to overcome arrhythmia, but it has a higher recurrence rate. High-definition (HD) mapping and three-dimensional (3D) ablation are new techniques that may be suboptimal, attributed to anatomical characteristics. The management tactics used may need to be modified in accordance with personal traits.

Case Illustration: A 37-year-old male presented with intermittent palpitations over the years. The electrocardiogram (ECG) is supraventricular tachycardia (SVT). Echocardiography shows typical features of EA. During ablation, multiple arrhythmias were induced, such as multiple atrial tachycardia and ventricular tachycardia. Throughout the observation period, the patient exhibited complications related to several episodes of recurrent arrhythmia and he experienced complete atrioventricular block, leading to the decision to undergo a permanent pacemaker procedure. We postulated that specific characteristics of the right atrioventricular groove structure observed in pathological samples of EA in general could explain less than ideal results in ablation procedures.

Conclusion: Managing arrhythmia in EA could be challenging. A prominent ridge alongside the lower atrioventricular groove is a typical characteristic of EA and is associated with the clinical background of accessory pathways (AP). Electrophysiologists working with this group of patients need to understand this anatomical aspect, as it may require adjustments to their management approaches.

Keyword: Ebstein's Anomaly, Arrhythmia, Accessory Pathway, Catheter Ablation, Personalized Management

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Successful Low Voltage External Cardioversion in Patients with Pulsed Unstable VT

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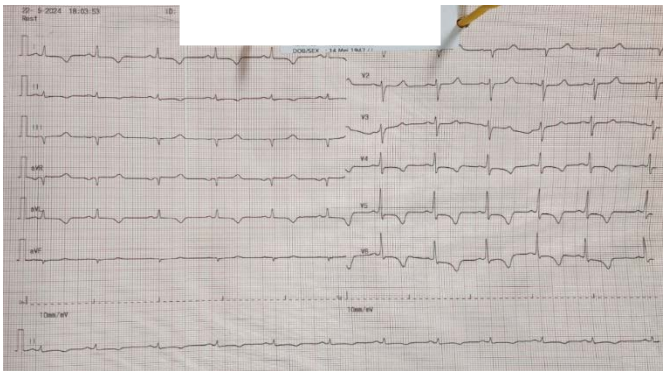
Background: Ventricular tachycardia (VT) is one form of malignant arrhythmia that requires prompt treatment. Advanced Cardiac Life Support (ACLS) algorithm for treating unstable VT recommends synchronized cardioversion starting from 100 joule. In this case we present a case of VT convert to sinus rhythm using low joule external cardioversion.
Case Illustration: A 77-year-old male presented to ER with weakness since morning before admission. His past history revealed that the patient was once admitted to hospital with NSTEMI very high risk and Incessant VT (unresponsive to 7 times of cardioversion). This patient was then referred for PCI. This time, monitor was placed and showed hypotension of 82/53mmHg and rapid heart rate of 178 bpm. ECG monitor showed regular wide complex QRS tachycardia. Laboratory results showed normal troponin I, leukocytosis, thrombocytopenia, mild hypokalemia, elevated ureum and creatinine. Ideally in this case, cardioversion should be performed immediately but there was full ICU capacity. This patient was then given IV bolus of amiodarone but failed to convert. After communicating the risk to the patient's family, they agreed to proceed to cardioversion with no ICU and ventilator backup. Cardioversion was successfully performed once with 50 J using biphasic defibrillator due to suspicion of SVT aberrancy. Later on, we used Brugada and Verneckei algorithm to identify that the patient actually had VT rather than SVT aberrancy. The patient was stabilized, given antibiotics, inotropes, vasopressors, TC transfusion, correction of potassium levels, and 2 sessions of hemodialysis. After 10 days of care, the patient was discharged with final diagnosis of Unstable-Pulsed-VT convert with cardioversion, UAP, Corrected Hypokalemia, AKI due to ADHF (HFrEF 35%) on ACS, Pneumonia with Sepsis.
Conclusion: Low-energy cardioversion is rarely used in treating VT. Several factors affect successful cardioversion. Proper diagnosis using various algorithms to differentiate VT and SVT aberrancy on ECG is required to prevent unnecessary or possibly harmful treatment.

Keyword: Ventricular Tachycardia, Cardioversion

ECG obtained Pre Cardioversion showed Wide QRS Complex Tachycardia (suggesting VT using Brugada Algorithm: signs of AV Dissociation and Marriott's Sign and Verneckei Algorithm: initial q wave in aVR >40ms)



ECG obtained Post Cardioversion showed successful conversion to Sinus Bradycardia with inverted T in lead I, aVL, V4, V5, and V6

Figure 1. ECG Pre and Post Cardioversion

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Diaphragmatic breathing practice for acute management of paroxysmal supraventricular tachycardia with wolff-parkinson-white syndrome: a case report

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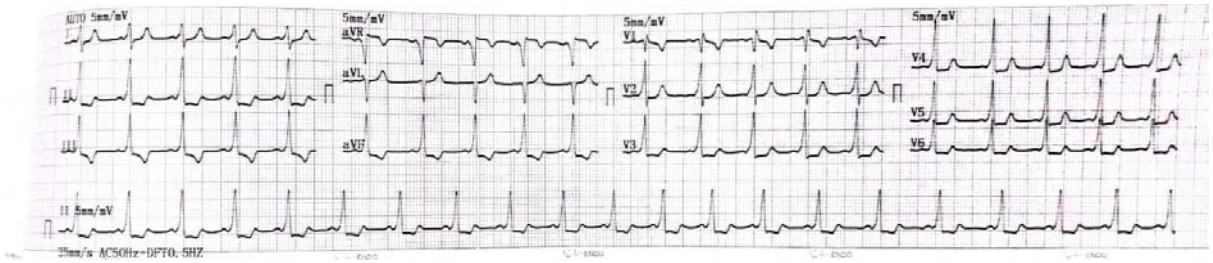
RSUD Prof. Dr. Margono Soekarjo, Banyumas, Indonesia

Background: Supraventricular tachycardia (SVT) is a narrow QRS-complex tachyarrhythmia due to re-entrant circuit, enhanced automatization or triggered activity. Wolff-Parkinson-White (WPW) syndrome is a pre-excitation disease with anterograde atrio-ventricular bypass tract that may cause paroxysmal SVT. Acute managements of SVT are vagal maneuvers, anti-arrhythmic agents and electrical synchronized cardioversion. Most of primary health facilities in Indonesia have limited resources, hence the important of vagal stimulation for SVT management before emergency referral. Therefore, we presented a case report of paroxysmal SVT conversion in WPW syndrome patient after diaphragmatic breathing practice.
Case Illustration: A female 10-years old patient came to Emergency Department with palpitation, shortness of breath and anxious feeling that lasted for 4 hours before admission. She had WPW syndrome that underwent two ablation therapies in 2022 and 2023. The vital sign showed blood pressure of 112/88 mmHg with heart rate of 220 bpm, respiratory rate of 24 times per minute and oxygen saturation of 95% in room air. Her radial pulses were regular with normal amplitude. She had cannon jugular vein pulsations with no sign of pulmonary oedema, acute heart failure or peripheral hypoperfusion. The monitor electrocardiogram showed regular narrow QRS-complex tachyarrhythmia. The patient was positioned lying down, given oxygen therapy by nasal canula and instructed to practice diaphragmatic breathing technique that consist of low respiratory rate with long exhalation to reduce anxiety. The monitor showed heart rate reduction, at a rate of 104 bpm, without carotid massage. The patient felt better without palpitation. Surface electrocardiogram showed sinus tachycardia at a rate of 107 bpm with short PR intervals and delta waves (Picture 1). The patient refused to be hospitalized and discharged against medical advice.



Picture 1. Surface ECG after paroxysmal SVT conversion

Conclusion: Deep-breathing practice could help in the acute management of paroxysmal SVT for limited-resource health facilities. Deep-breathings produce respiratory vagal nerve stimulation that inhibits AV node conduction and cut-offs the re-entrant circuit.

Keywords: Supraventricular tachycardia, Wolff-Parkinson-White syndrome, Diaphragmatic breathing

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Brugada Phenocopy Caused by Intracranial Hemorrhage; A Case Report

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Background: Brugada syndrome (BrS) is a congenital channelopathy associated with increased risk of malignant ventricular arrhythmias and sudden cardiac death in individuals without any structural cardiopathy. Brugada phenocopies (BrPs) are clinical entities that present electrocardiographic patterns similar to BrS that are elicited only under transitory pathophysiological conditions, with normalization of the ECG pattern after the resolution of those conditions. We present a rare case of BrP due to intracranial hemorrhage.

Case Illustration: We hereby present a 56-year-old man with no significant clinical history was admitted to the Emergency room (ER) due to fell from the second floor of his house. The accident resulted in Polytrauma coincide serious head trauma. Reported onsite Glasgow Coma Scale score were 8. A head CT performed Subdural Hemmorhage and Intracranial Hemmorhage which indicate to Craniectomy. Before the operation, ECG showed type 1 Brugada pattern. After craniectomy performed, patient were sedated, ventilated, and transported to the intensive care unit (ICU). On the 2nd day after craniectomy, the patient's condition improved and we found resolution to the initial normal ECG without Brugada pattern was documented. On the 3rd day after craniectomy, the ECG showed normal ECG with no further changes.

Conclusion: True BrPs related to intracranial hemorrhage are very rare. We present the case of a middle aged man who experienced head trauma resulting in subdural hemmorhage and intracranial hemmorhage associated with elevated intracranial pressure. We observed a type 1 BrS ECG pattern that present at the admission, which resolved after the craniectomy procedure. During this period, no other conditions or agents known to modulate BrP were present, and the patient presented a negative provocative test.

Keywords: Brugada ecg pattern, brugada phenocopy, subdural hemmorhage, intracranial hemmorhage

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Chaotic Arrhythmia in Peripartum Cardiomyopathy: A Case Report

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Background: Peripartum cardiomyopathy (PPCM) is a dilated cardiomyopathy defined as systolic cardiac heart failure in the last month of pregnancy or within five months of delivery. In this peripartum period, there is an increased incidence of arrhythmias, which may range from 50% to 90%, even in women without structural heart disease. This case report examines the arrhythmic variations in a patient diagnosed with PPCM two weeks postpartum, emphasizing the need for timely diagnosis and comprehensive management.

Case Illustration: A 34-year-old woman arrived at the ED with severe dyspnea lasting two days. She couldn't lie flat due to orthopnea and found some relief by leaning forward. She had undergone a caesarean section two weeks earlier, had no complications during pregnancy or delivery, and had no known cardiovascular risk factors.
On examination, the patient had a blood pressure of 177/122 mmHg, a pulse rate of 116 beats per minute, a respiratory rate of 42 breaths per minute, and an oxygen saturation of 87% on room air. Lung examination revealed bilateral fine basal crackles. An ECG indicated sinus tachycardia. A chest radiograph showed cardiomegaly and pulmonary edema. The patient has a normal magnesium count and an increased prolactin level. Echocardiography revealed a significantly reduced LVEF of 32% with a dilated LV, indicative of heart failure due to PPCM.
Initial treatment with diuretics provided temporary stabilization. However, within 12 hours, the patient experienced a seizure with polymorphic VT, which was observed on the monitor. Prompt defibrillation restored sinus rhythm and consciousness. Within 24 hours, she developed SVT, which reverted to sinus rhythm following amiodarone administration. Bromocriptine was then initiated, and her condition gradually improved during hospitalization.

Conclusion: This case highlights the complexity of PPCM and the need for vigilant monitoring for arrhythmic complications. The range of arrhythmic presentations—from sinus tachycardia to life-threatening VT and SVT—demonstrates the need for a comprehensive approach to managing PPCM. Early recognition and diagnosis are crucial, particularly for postpartum women with unexplained dyspnea. The treatment regimen for PPCM typically includes standard heart failure therapies, and bromocriptine, as a prolactin inhibitor, is fundamental to improving outcomes.

Keyword: arrhythmia variation, peripartum cardiomyopathy, heart failure, postpartum cardiac complications, maternal morbidity

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Painful Left Bundle Branch Block Syndrome: A Case of Rate-Dependent Cardiac Dyssynchrony Mimicking ST-Elevation Myocardial Infarction (STEMI)

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Background: Painful left bundle branch block (LBBB) syndrome is characterized by chest pain associated with new-onset LBBB in the absence of coronary artery disease, which resolves alongside the disappearance of LBBB. The exact mechanism of chest pain remains unknown, but acute cardiac dyssynchrony is thought to be involved. This syndrome typically occurs when the cycle length is equal to or less than the refractory period of the left bundle, mainly during physical exertion.

Case Illustration: A 52-year-old male with uncontrolled hypertension presented with a 2-hour onset of left-sided chest pain, described as a pressing sensation during activity without radiation. He denied prior cardiac history or family history of syncope and sudden cardiac death. Physical examination was unremarkable. An initial ECG at the referring hospital showed sinus rhythm at 88 bpm with new-onset LBBB. A repeated ECG at our hospital showed sinus rhythm at 75 bpm with LBBB and supraventricular extrasystole with a compensatory pause followed by a single normal conduction QRS complex. Due to ongoing chest pain, primary percutaneous coronary intervention (PPCI) was performed, but coronary angiography revealed normal coronary arteries, and echocardiography showed normal cardiac function. Laboratory examinations, including myocardial enzymes, were normal. The patient was treated with optimal medical therapy. During hospitalization, clinical evaluation and ECG showed sinus rhythm at 60 bpm and resolution of LBBB, with the disappearance of chest pain at the same time.

Conclusion: This case highlights a patient with painful LBBB syndrome presenting with symptoms mimicking acute STEMI. Despite initial ECG findings and ongoing chest pain, coronary angiography and echocardiography revealed normal coronary arteries and cardiac function. The patient's chest pain and LBBB were rate-dependent, resolving at a lower heart rate. This supports the hypothesis that acute cardiac dyssynchrony due to LBBB is the underlying mechanism for the chest pain. Optimal medical therapy led to symptom improvement and normalization of the ECG. This case underscores the importance of considering painful LBBB syndrome in differential diagnosis when evaluating patients with chest pain and new-onset LBBB in the absence of coronary artery disease.

Keywords: *Chest Pain, Rate-Dependent, Left Bundle Branch Block, Normal Coronary Arteries*

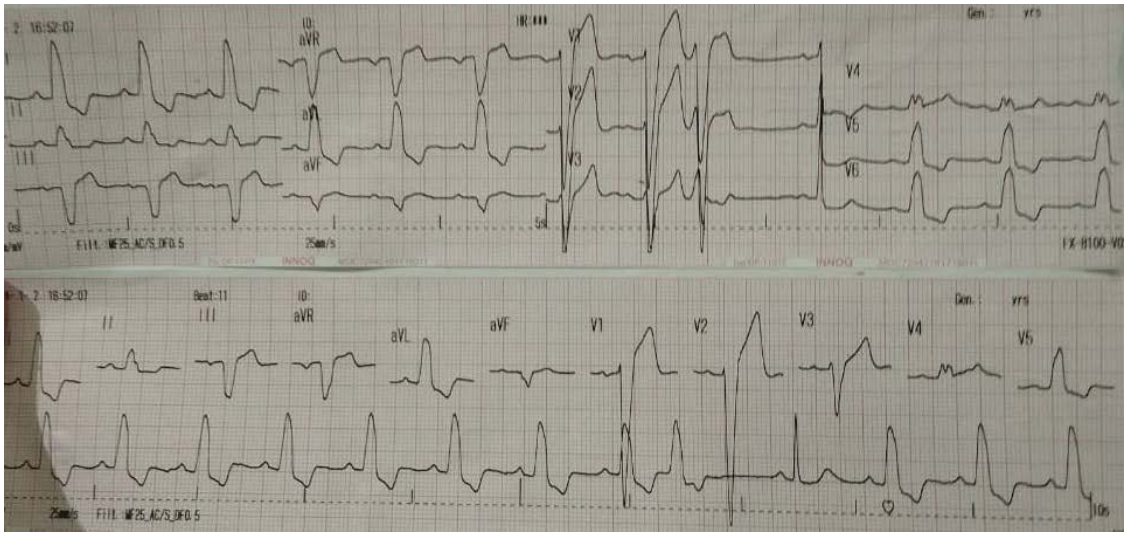

Figure 1. ECG showed sinus rhythm at 75 bpm with LBBB pattern and supraventricular extrasystole with compensatory pause followed by single normal conduction QRS complex

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Non-sustained Ventricular Tachycardia and Premature Ventricular Complexes with Heart Failure Reduced Ejection Fraction as Cardiac Manifestations of Systemic Lupus Erythematosus. A Case Report.

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Background: Systemic lupus erythematosus (SLE) is a multi-organ autoimmune disease that affects the heart and can cause lupus cardiomyopathy and also arrhythmias. Arrhythmias and conduction systems disorders are among the cardiovascular disturbances related to SLE. Sinus tachycardia, premature atrial complex, and AF are the most common arrhythmia in SLE.[1] However, ventricular tachycardia (VT) is a rare manifestation.[2]

Case Illustration: A 25-year-old male presented with dyspnoea on exertion, alopecia, weight loss, chronic fever, and arthritis. Laboratory findings revealed ANA IF (+) 1:320, hemoglobin of 14,7 g/dL, leucocyte of 24.370/mm^3, thrombocyte of 236.000/mm^3, hs-Tropnonin I 0,05 ng/ml (normal < 0,04,), albumin 2,8 g/dL, sodium 146 mmol/L, potassium 3,5 mmol/L, and normal thyroid function. This case has fulfilled the criteria of SLE.[3] Echocardiography showed normal heart chambers with global hypokinetic and ejection fraction of 31%. (figure 1a)

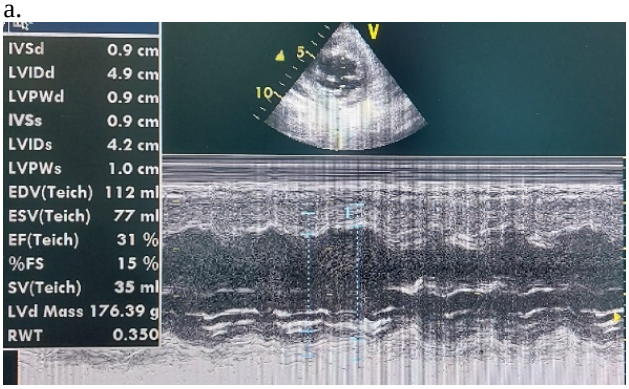
During hospitalization, the patient experienced palpitation and worsening dyspnea, necessitating intubation and mechanical ventilation. ECG findings were non-sustained VT with RBB pattern with superior axis and PVCs. (figure 1b and 1c) Hemodynamic remained stable during non-sustained VT. The QTc interval was 477ms. The patient did not receive drugs that prolonged QT interval such as hydroxychloroquine for SLE.

The patient was treated with amiodarone IV infusion for 24 h followed by oral amiodarone, but the non-sustained VT persisted. Additional treatments include lidocaine 1 mg/min for 24 h and bisoprolol 2.5 mg once daily. To manage the inflammation, methylprednisolone 250 mg twice daily was given. Heart failure management included ACE inhibitors, aldosterone antagonists, and diuretics, according to current guidelines.

Conclusion: Non-sustained VT is a rare arrhythmia in SLE. Effective management involves not only anti-arrhythmic drugs but also aggressive immunosuppressive therapy and comprehensive heart failure treatment to address underlying lupus cardiomyopathy.

Keywords: Non-sustained VT, Multifocal PVC, Heart Failure Reduce Ejection Fraction, Systemic Lupus Erythematosus

a.



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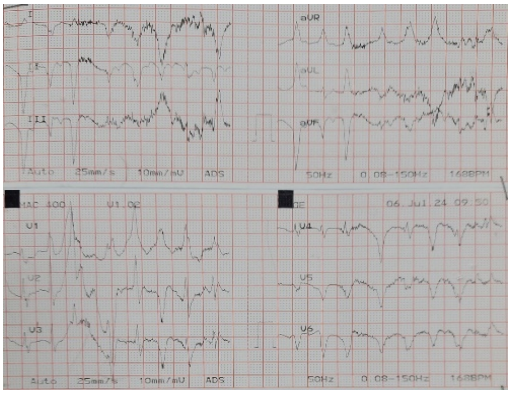

c.

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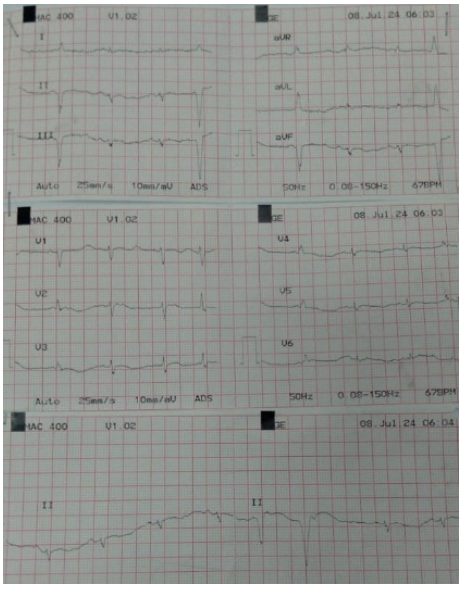

Figure 1

ıary Artery Fistula presenting as Recurrent Ventricular Tachycardia in Elderly patient: A Case Report

[redacted]. Saerang[1], A. Rizal[1], W. Karolina[1], A. Prastya[1]

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Background: Coronary artery fistulae (CAF) is a rare anomaly of the coronary anatomy, especially in the elderly, particularly in the elderly, they can occasionally induce functional myocardial ischemia and manifest as angina, dyspnea, and arrhythmia when hemodynamically significant. The clinical presentation of CAFs can vary from asymptomatic to sudden cardiac death. Cases of coronary artery fistula to the pulmonary artery with recurrent ventricular tachycardia (VT) are rare.

Case Illustration: An 81-year-old female patient presented to hospital with chest pain, palpitations and syncope, with an ECG showing monomorphic VT. The patient was cardioverted and given amiodarone with reversion to sinus rhythm with multifocal PVCs from LV origin. From physical examination, there was a continuous 'machinery' murmur at the left upper sternal border. During amiodarone treatment, the patient experienced recurrent VT which was terminated with lidocaine and at a rate of 60-70 bpm. Coronary angiography showed a left main coronary fistula leading to the pulmonary artery through a large tortuous vessel. Coronary CT was performed for procedural planning and confirmed a coronary artery fistula leading through a large tortuous left main coronary artery to the pulmonary artery.

Conclusion: Coronary artery fistula in elderly patients are rare, especially when symptomatic. The mechanism of ventricular tachycardia in coronary artery to pulmonary artery fistulae is functional due to the 'coronary steal phenomenon'. Adults with haemodynamically significant coronary fistulae often present with symptoms and life-threatening complications such as myocardial ischaemia or infarction, aneurysm formation and rupture, arrhythmias and congestive heart failure. An adequately performed CT scan with 3D reconstruction is invaluable in visualising the coronary anatomy for further treatment planning and correction of coronary artery fistulae. Closure is indicated for large or symptomatic fistula, either surgically or percutaneously, to prevent patient complications and sudden death.

Keywords: Ventricular tachycardia; Coronary artery to pulmonary artery fistula; Sudden death, coronary steal.

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Case report : Complete Atrioventricular Block in Rheumatic Heart Disease

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Background: Bradycardia in patients with rheumatic heart disease usually occurs as first degree atrioventricular block, while complete atrioventricular(AV) block is very rare event and sometimes recovers within several days. Symptomatic permanent complete heart block requires intervention by implanting a pacemaker.

Case Illustration: A 31 year old woman came to emergency department with chief complaints of left chest pain since 2 days ago, she felt the pain continuously like being stabbed, radiating to the back and left arm, accompanied with shortness of breath especially when activity, nausea, vomiting three times, she also easily tired when doing activities, coughed for a month. She had been diagnosed with rheumatic heart disease since 1 year ago in another hospital. Physical examination findings the patient was alert, blood pressure 112/59mmHg, pulse 40 beat per minute, no tachypnea, normal temperature and no hypoxia. Complete blood count within normal limit, creatinine slightly elevated (0.99), hyponatremia (132), slightly elevated troponin T. Chest x ray showed cardiomegaly. ECG showed complete av block with junctional rhythm. Echocardiography showed dilated left atrium, right atrium, right ventricle, eccentric left ventricular hyperthrophy, reduced ejection fraction (30%), global hypokinetic with diastolic dysfunction grade I, high probability of pulmonary hypertension, severe mitral stenosis, mild mitral regurgitation suggestive consequences of Rheumatic Heart Disease(RHD). The patient was given therapy with dopamine 5mcg/kg/m titration,and therapy for symptomatic. After six days of treatment, the patient still had complete heart block and we refer the patient for pacemaker implantation but she refused and discharge against medical advice.

Conclusion: We must find out and eliminate all the possibilities causes of atrioventricular block especially complete av block in a patient. In patients with acute rheumatic fever, rapid therapy can reverse the complete AV block, while delayed therapy can make complete AV block become permanent.

Keywords: Complete heart block, rheumatic heart disease, pacemaker

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IS PACEMAKER ALWAYS THE ANSWERS IN LATE PRESENTING HIGH DEGREE HEART BLOCK AFTER MITRAL VALVE REPLACEMENT

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Background: Atrioventricular (AV) block is one of the complications that occur after Mitral Valve Replacement (MVR) surgery. Late-presenting AV block is very rare after this procedure. Pacemaker placement is the definitive therapy for this kind of case.

Case Illustration: A 44-year-old woman presents with complaints of weakness and fever for the past week. The patient underwent MVR surgery 6 months ago with good results. Post-operative ECG before discharge showed first degree AV block. Transesophageal echocardiography (TEE) examination 3 weeks ago revealed thrombus on the prosthetic mitral valve. ECG examination on the following days revealed intermittent High degree AV block. The patient received antibiotic therapy and underwent an electrophysiology (EP) study, revealing normal HV interval (50ms), prolonged AH interval (420ms) and suprahisian block at S1 500ms. High degree AV block is suspected to be caused by inflamation (myocarditis/pericarditis). ECG during the remainder of the treatment revealed first degree AV block after 1 week of antibiotic therapy. She had an uneventful recovery afterwards. Follow up ECG in cardiology clinic 6 month after discharge still showed first degree AV block without symtomps.

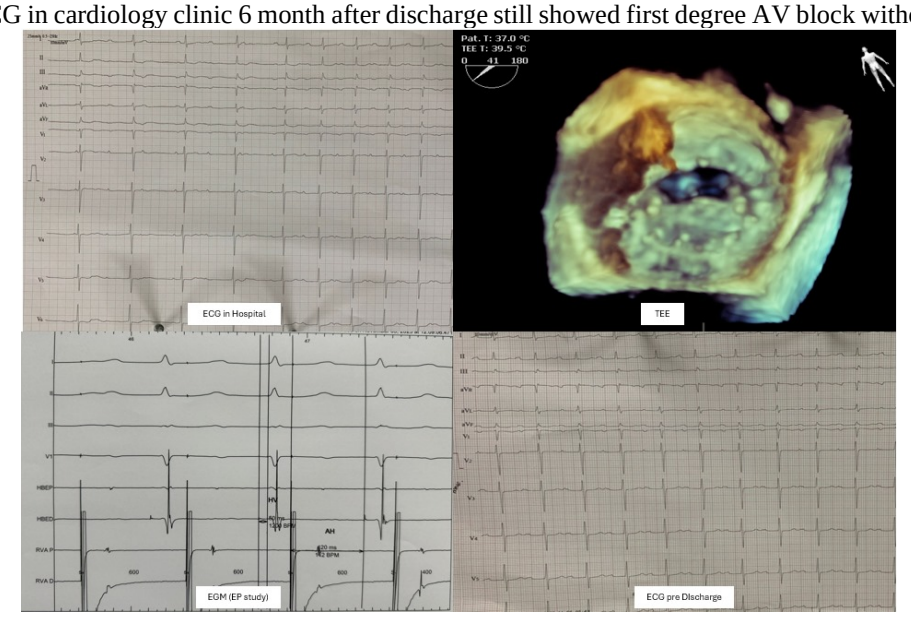

Figure 1. Patient's ECG, TEE and EGM

Conclusions: Late-presenting AV block is rare occurrence in post-MVR patients, but not all High degree AV block require pacemaker placement as definitive therapy. An EP study can be one of the procedures to assess the source of block (suprahisian, intrahisian or infrahisian), then determine whether pacemaker implantation is necessary or not.

Keywords: High degree AV block, post MVR, Myocarditis, EP study, Late presenting

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Is it ventricular tachycardia?

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Background: Implantable cardioverter defibrillator (ICD) has been used as prevention of sudden cardiac death in patients at high risk for ventricular arrhythmias. Multiple consecutive discharges (MCDs) might be appropriate due to resistant ventricular tachyarrhythmia (VT) and is associated with higher mortality risk; but it might also be inappropriate due to supraventricular arrhythmia, oversensing, or improper device setting. This report aimed to present MCDs in a patient and the exploration of its cause.

Case illustration: A 48-year-old-man with ischemic HF (EF 25%) and history of MI underwent ICD implantation as primary prevention of sudden cardiac death. Following PCI and optimized medications, the patient showed improvement (NYHA class II). Three years later, he developed right-sided face swelling that persisted despite diuretics. Thorax CT-scan showed thyroid mass and total obstruction of the right jugular vein. He underwent thyroidectomy and bypass grafting of the right jugular vein to right atrium (RA); postoperative drain was placed in the pericardium near RA. He developed postoperative rapid AF and received MCDs from his ICD. Interrogation revealed anti-tachycardia pacing during high-rate episode of AF (Figure 1a) and after initial shock was delivered, the QRS turned wide. Waveform analysis did not match for supraventricular tachyarrhythmias (Figure 1b) and was considered ventricular tachyarrhythmia – 27 MCDs were delivered.
Post-MCDs and amiodarone administration, rhythm converted to sinus. One week post-drain removal, follow-up interrogation revealed no tachyarrhythmia events and changes were made in the threshold rate program (Figure 1c). Although VT and SVT share the same rate threshold, shocks will be deferred and treated as SVT first. Several mitigations for this patient included magnet placement for acute ICD inappropriate shock prevention while monitoring 12-lead-ECG, and considering dual-chamber ICD for long-term plan.

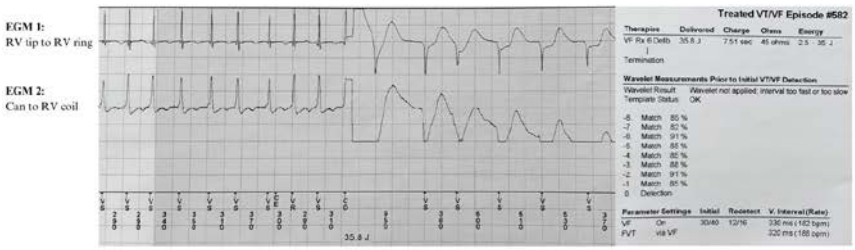

Figure 1a. Antitachycardia pacing during high rate episode of AF and its waveform analysis

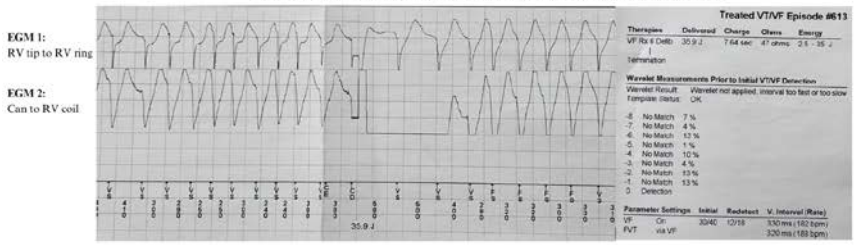

Figure 1b. Shock delivered during wide QRS episode and its waveform analysis

	Before	After
VT	330 ms	300 ms
FVT	320 ms	340 ms
VT	350 ms	350 ms
SVT limit	320 ms	300 ms

Figure 1c. Lead re-programming after MCDs

Conclusion: We have presented a case of ICD MCDs in a patient with ischemic HF and history of MI who had postoperative drain placed near RA. Two possibilities remain for this case in consideration to the patient's extensive LV scar: appropriate shocks due to VT, or inappropriate shocks due to AF with aberrancy. Several strategies have been planned to mitigate this condition.

Keyword: Implantable cardioverter defibrillator; multiple consecutive discharges

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Symptomatic sinus bradycardia as the result of cholecystitis: recollecting Cope's sign

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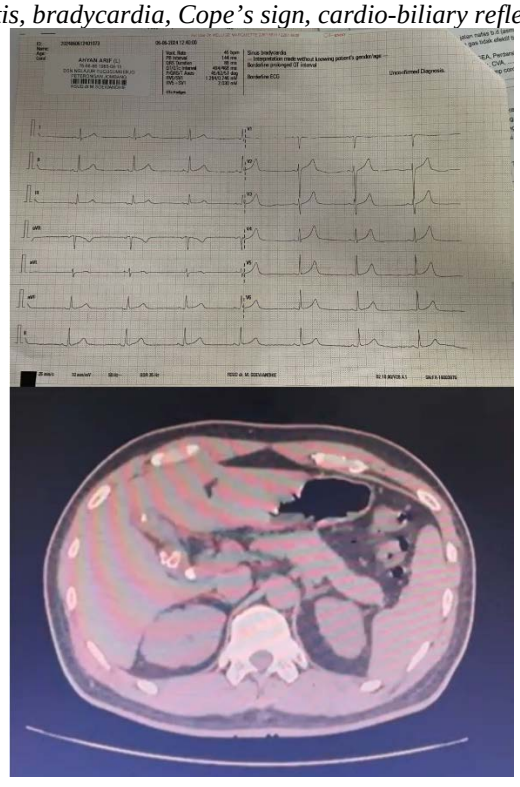
Background: "Cope's sign" was named after Sir Zachary Cope who first discerned an association between calculous cholecystitis with bradycardia, terming it the cardio-biliary reflex, likely due to increased vagal tone. Few cases have been reported since, but the terms remain relatively unfamiliar.
Case illustration: A 38-year-old male with a two-month history of cholelithiasis came to the emergency department (ED) complaining of sudden, severe upper central abdominal pain radiating to his back. He also reported malaise, nausea without vomiting, a fever since the previous day, and jaundice for the past three months. He denied chest pain, headache, and confusion. He was not on any regular medication and had no other prior medical history.
On arrival, he was bradycardic at 45 bpm, hypotensive at 86/49 mmHg, and afebrile. Physical examination showed epigastric tenderness, but Murphy's sign was negative. Blood tests revealed an elevated neutrophil-to-lymphocyte ratio (5.74), and elevated levels of alanine aminotransferase (88 U/L), aspartate aminotransferase (87 U/L), total bilirubin (11.37 mg/dL), and direct bilirubin (8.42 mg/dL). The ECG showed sinus bradycardia, and the chest X-ray was normal. An abdominal MSCT performed three weeks earlier showed multiple calculi in the gallbladder, cystic duct, extrahepatic duct, and common bile duct (CBD), causing obstruction at the ampulla of Vater.
The patient was given 1 mg of atropine sulfate in the ED, which improved his heart rate to 100 bpm and resolved the hypotension. He underwent a cholecystectomy and CBD exploration on the sixth day of admission, then the bradycardia was fully resolved.
The cardio-biliary reflex involves disorders where afferent and efferent parasympathetic pathways cause inappropriate vasodilation, bradycardia, hypotension, and syncope. The afferent signal, originating in the gallbladder, responds to mechanical stimulation such as increased pain and inflammation in the gallbladder, leading to vagally mediated bradyarrhythmia.
Conclusion: Cardio-biliary reflex does exist and can results in serious consequences. Early recognition of the sign can minimize unnecessary cardiac workups and guide appropriate treatment.

Keywords: calculous cholecystitis, bradycardia, Cope's sign, cardio-biliary reflex



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Brugada Syndrome: A Crucial Diagnosis Often Overlooked

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Backgrounds: Brugada Syndrome is a rare, genetically inherited cardiac disease derived from abnormalities in cardiac ion channels. It presents a spectrum of symptoms, from asymptomatic to potentially fatal arrhythmias resulting in sudden cardiac death. Its hallmark lies in the distinctive ST elevation morphology observed in precordial leads on electrocardiograms. This syndrome is more prevalent in males and exhibits a higher incidence among Southeast Asia population. Despite the diagnostic challenges it poses, it is important for physicians to accurately identify this critical disease. Early recognition of this condition facilitates appropriate management and, crucially, mitigates adverse outcomes.

Case Illustration: A 57-year-old Asian male, without remarkable risk factors, was admitted due bacterial intestinal infection complicated by electrolyte imbalance, he received intravenous fluids, antibiotics, antipyretics, and gastric medications. However, several hours post-admission, the patient was discovered unconscious with no palpable pulse. Ventricular tachycardia was detected on the monitor. Emergency cardiac resuscitation and defibrillation successfully restored the patient's rhythm and pulse. The patient was intubated and transferred to the intensive care unit for further monitoring and stabilization. Regrettably, the patient's condition deteriorated, leading to cardiac arrest. Despite the hard effort to resuscitate the patient, he could not be revived and was, later, pronounced deceased. Subsequent analysis of the initial electrocardiogram revealed coved ST elevation with T-wave inversion in the V1-V2 precordial leads, indicative of Brugada type 1 morphology.

Conclusion: Brugada syndrome, a rare channelopathy, poses a significant risk of life-threatening cardiac arrhythmias. Despite its nonspecific clinical presentation, it is imperative for all physicians, particularly those in the emergency room, to recognize this condition. Identification may be aided by the presence of coved ST-elevation in precordial leads, particularly when accompanied by a history of familial sudden cardiac death. Prompt diagnosis is essential for implementing supportive therapies and considering implantable cardiac defibrillation to prevent the risk of sudden cardiac death.

Keywords: Brugada Syndrome, ST-elevation, Sudden Cardiac Death

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New-onset atrial fibrillation in ST-segment elevation myocardial infarction after primary PCI: exploring the causes and strategies for management

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Background: About 9% of patients with ST-elevation myocardial infarction (STEMI) experience atrial fibrillation (AF) during or shortly after PCI. New-onset atrial fibrillation (NOAF) during MI is associated with higher risk of stroke, major cardiac and cerebrovascular events, and mortality, even if converted to sinus rhythm before discharge. Treatment guidelines for long-term anticoagulation therapy in transient NOAF during MI after PCI remain unclear.

Case Illustration: A 54-year-old male with typical chest pain was diagnosed with anteroseptal STEMI 16 hours after onset. He had history of smoking, hypertension, and infarct stroke. Angiography revealed single-vessel CAD and primary PCI was performed with implantation of 1 DES on LAD. He received DAPT, fondaparinux, statin, nitrate, ACE inhibitor, trimetazidine, and lumbrokinase. The next day, he developed asymptomatic AF with rapid ventricular response. Amiodarone IV was administered and his ECG converted to sinus rhythm 3 hours later. Unfortunately, he left the hospital against medical advice after being hospitalized for 3 days. Two subtypes of NOAF have been identified: early NOAF (AF within 24 hours from MI) and late NOAF (AF developing after 24 hours). While early NOAF is associated with atrial ischemia, late NOAF is a complication of ventricular dysfunction, hemodynamic overload, and atrial enlargement, suggesting that AF may develop despite successful reperfusion therapy. Achieving adequate rate control and prompt sinus rhythm conversion is crucial due to associated mortality risks. Patients with late AF following acute MI had higher stroke rates compared to no AF and early paroxysmal AF. Chronic oral anticoagulation, guided by CHA2DS2-VASc and HAS-BLED scores, is considered in this patient with careful monitoring for bleeding risks by adjustment of drug combination, dosing, and duration of therapy.

Conclusion: This case highlights challenges in managing a 54-year-old male with anteroseptal STEMI complicated by late NOAF. Appropriate management of acute events and cardiovascular risk factors with thorough follow-up is important and may differ depending on individual clinical conditions. Further studies are needed to understand the mechanisms, investigate prognoses, and explore treatments of NOAF subtypes in STEMI patients to reduce adverse events and mortality rates.

Keyword: new-onset atrial fibrillation; ST-elevation myocardial infarction; primary percutaneous coronary intervention

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Transient Bradycardia Related to Hepatitis-A and Choledocholithiasis

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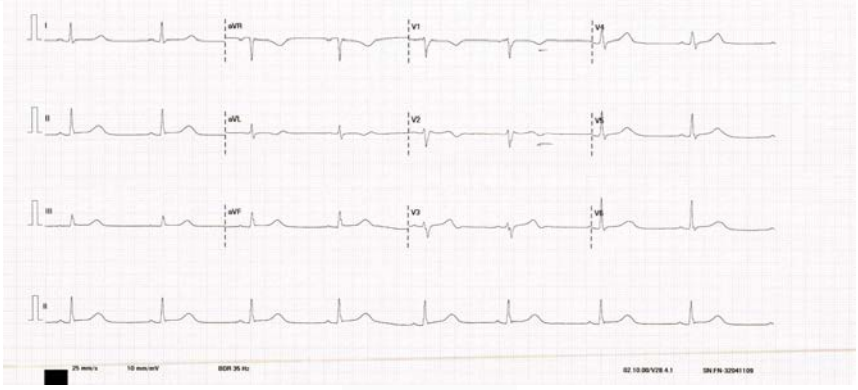
Background: Hepatitis A virus (HAV) is one of the well-known viruses that cause hepatitis all around the world. HAV is most commonly transmitted through the oral-fecal route via exposure to contaminated food, water, or close physical contact with an infectious person. A high level of serum bilirubin is associated with cardiac dysfunction, obstructive jaundice is a known cause of sinus bradycardia. Bile acid has been strongly suggestive of the predominant factor that induces sinus bradycardia by inhibitory impact on the SA node and that achieved by direct vagal stimulation. The higher the bilirubin levels are, the lower the heart rate.

Case illustration: A 42-year-old male presented with the complaint of chest discomfort, itching, and gradually increasing yellow coloring of the body and eyes that started 12 days ago. There was no fever, diarrhea, or abdominal pain. Blood pressure was 90/50, temperature 36.7C, and the heart rate was 49bpm. Cardiological examination and tests were within normal limits. The patient's total bilirubin was 12.05 mg/dL (normal: 0.1–1.0 mg/dL), direct bilirubin was 9.11 mg/dL (normal: 0.1–0.25 mg/dL) and the troponin-I level was in the normal range. Anti-HAV-IgM/IgG was positive in the patient. ECG showed sinus bradycardia with a rate of 49 bpm and there was no pathological finding in echocardiography. An abdominal CT scan with contrast was performed, it showed multiple impacted stones in the Ampulla of Vater. He was given several doses of atropine and then he was started on dopamine drip. Symptomatic treatment was administered to the patient, and no antiviral or antibiotherapy was required. ERCP is performed to evacuate the stone, it has been shown that HR returned to normal, and bradycardia disappeared as bilirubin levels decreased.



(Figure 1: ECG)

Conclusion: We concluded that obstructive jaundice and viral hepatitis can induce sinus bradycardia. Sinus bradycardia is associated with circulating bile salts and the severity of jaundice by the mechanism of bilirubin depresses the sinoatrial node via vagal centers. A case of HAV-induced bradycardia reminded us that patients with acute HAV infection should be closely monitored for cardiovascular effects.

Keyword: Bradycardia; hiperbilirubinemia; jaundice; viral; hepatitis

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Case Report: When the Eyes Speak to the Heart: Timolol-Associated Complete Atrioventricular Block in a Glaucoma Patient

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Background: An increase in intraocular pressure (IOP) potentially damages optic nerve is known as glaucoma. Its prevalence rises with age. Topical eye drops lowering intraocular pressure are the most widely used medical treatment for glaucoma. For it effectively lowers intraocular pressure, timolol maleate, a potent non-selective beta-adrenergic blocker, is the most commonly prescribed glaucoma medicine. Although administered topically, it can reach systemic circulation and have detrimental effects on cardiovascular system. We report a case of long-term timolol use in a glaucoma patient that revealed and exacerbated an underlying cardiac conduction abnormality, manifested as a complete Atrioventricular (AV) block.

Case illustration: A 55-year-old woman presented to Emergency Room (ER) with dizziness followed by a seizure and a syncopal episode while waiting at our hospital Eye Polyclinic for routine control. She had a past medical history of glaucoma the past two years including having trabeculectomy last year and timolol eye drops as routine medication. She was immediately transferred to ER and the electrocardiography indicated a heart rate of 30 bpm with a complete AV block. A temporary pacemaker was installed on patient then we transferred the patient to Cardiovascular Care Unit (CVCU) for next monitoring and evaluation. After consulting with an ophthalmologist, due to elevation of IOP required glaucoma medication, we decided to install a permanent pacemaker.

Conclusion: Long-term topical timolol use can worsen cardiac conduction abnormalities, leading to an AV block. For the length of therapy, medical professionals should be aware of any potential cardiovascular side effects of timolol and monitor the patients regularly to reduce adverse events and hospitalizations. Moreover, before treating any patients who present with an AV block, cardiologists should carefully assess their medication regimen.

Keywords: atrioventricular block, timolol, glaucoma

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TRANSIENT SECOND DEGREE A-V BLOCK DUE TO ANEMIC CONDITION IN PREOPERATIVE PATIENT

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Background: Second degree A-V Block occured due to failure of conduction of atrial impulse to ventricle, with dropped (nonconducted) P waves on ECG. One of the hallmark of Mobitz type II is a stable P-P interval without any prolongation of the PR interval before a conduction failure. In some cases, a transient atrioventricular blocks occurred due to anemic condition. Anemia have different causes of etiology, in this case we suspected moderate anemia due to Myelodysplastic syndrome.

Case illustrations: A 75-year-old man with an elective cystoscopy procedure to evacuate clots obstruction due to prostate hiperplasia conditions. He had haematuria, hypertensive states and history such of Benign Prostatic Hiperplasia, first degree atrioventricular block, chronic kidney disease, and pansitopenia due to Myelodysplastic syndrome. Laboratory examination showed anemia with haemoglobin levels was 6.5 mg/dl and thrombocytopenia of 94.000 u/mm^3.ECG showed second degree A-V Block Mobitz type II. Echocardiography showed decreased LV ejection fraction of 40% (Simpson's) TAPSE 1.7cm TR mild. The procedure was postponed and planned for transfusion. Post transfusion hemoglobin returned normal to 10.1mg/dl, his ECG returned to first degree a-v block. Cystoscopy was perfomed with minimal haematuria , stable haemoglobin and there was no failed atrioventricular conduction in the ECG monitor. Afterward the patient was referred for pacemaker implantation.

Anaemia is a condition in which the number of red blood cells or the haemoglobin concentration within them, is lower than normal. Anaemia caused 50 million years of healthy life lost due to disability in 2019. The largest causes were dietary iron deficiency, thalassemia, malaria, and many more which are preventable or treatable. Complication of anemia can affect inadequate oxygen delivery to tissue, can cause myocte dysfunction in the heart. Cardiac Output should increase to compensate and make adequate oxygen delivery. LV dysfuction condition became from a high output (compensated) cardiac state when Haemoglobin level reach 7g/dl. in many cases patients with anemia may be asymptomatic and obtain no management.

Myelodysplastic syndrome have clinical manifestations such as decrease Red Blood Cells, platelets dan white blood cells. Reccurent lower RBC in patient with MDS , which have symptom, require frequent blood transfusions. However, still no studies show direct relation between arrhythmia and MDS, But anemia condition will usually occurred in MDS.

This case report showed a **rare condition** of a stable patient who developed second degree type 2 atrioventricular block during his worsening anemic condition. AV node have a significant role to regulate heart rhythm. Decrease of hemoglobin level might also impact vascularization to AV nodal artery, which 90% got perforating branch from right coronary artery. Low blood supply and low hemoglobin content to heart muscle could reduce the flow of oxygen and nutrients to heart muscle. His medical condition, which suspected MDS will lead him to have reccurent anemia. When the condition of anemia worsened, the AV node will have ischemic condition. Coronary angiography was not performed because the patient remained asymptomatic. No history of medication that impaired atrioventricular conduction. In this case, his heart rhythm returned normal after we did transfusion. Due to myelodysplastic syndrome will lead him to recurrent high degree AV block, we performed pacemaker implantation. We presume that hypoxemia due to anemic condition could be associated with transient atrioventricular block. However this will need more studies to prove the relation.

Conclusion: Catastrophic condition such as syncope, presyncope or sudden cardiac aresst due to A-V Block. Anemic condition should be explored when transient second degree A-V Block occurred. The management is to stabilize haemoglobin levels with transfusion, showed better atrioventricular conduction. When the anemia condition become recurrent in addition to treat the anemic problem, placement of pacemaker maybe better solution to prevent unexpected complication

Keywords: A-V Block, Anemia, MDS

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Case Study: Heart Failure with Interventricular Dyssynchrony and Narrow QRS - Exploring Resynchronization Potential

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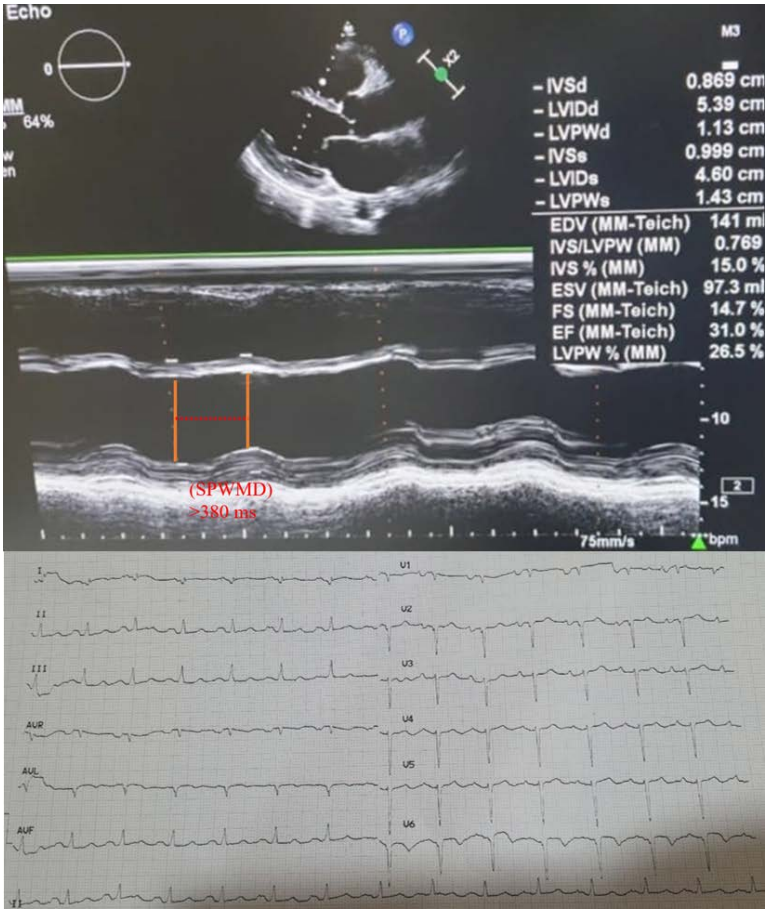
Background: Conduction delay affecting 30-50% of heart failure (HF) patients leads to decreased cardiac contractility due to intra- and interventricular dyssynchrony. Cardiac resynchronization therapy (CRT) is recommended as Class I therapy for HF patients with left ventricular ejection fraction ≤35% and QRS duration ≥120 ms. The use of CRT in HF patients with EF <35% and QRS <120 ms is still a Class IIb recommendation. Nevertheless, several studies have shown that systolic asynchrony is present in 27-43% of HF patients with narrow QRS complexes, and CRT shows benefits in these patients.
Case Illustration: The patient is a 56-year-old male presenting with complaints of fatigue, dyspnea, and abdominal distension persisting for >5 months. He has a history of cardiogenic shock due to myocardial infarction and underwent PCI 2 years ago. The patient is classified as NYHA II. The ECG shows sinus rhythm with Q waves in leads I, AVL, V2, and V6, P mitrale, and QRS duration of 0.98 seconds. Transthoracic echocardiography reveals dilatation of the left atrium (LA) and left ventricle (LV), moderate to severe tricuspid regurgitation (TR), mild pulmonary regurgitation (PR), and mitral regurgitation (MR). There is global systolic dysfunction of the LV, septal-to-posterior wall motion delay (SPWMD) >380 ms, with an ejection fraction of 31%. The patient has been receiving medical therapy with furosemide, ARNI, bisoprolol, spironolactone, dapagliflozin, atorvastatin, and clopidogrel. Despite optimal medical therapy, there has been no improvement in ejection fraction. The patient is scheduled for resynchronization therapy using CRT-P.
Conclusion: Interventricular dyssynchrony can occur even with a narrow QRS complex. Optimal medical therapy does not always result in significant clinical improvement. Consideration for resynchronization therapy still requires further investigation, both in terms of indications and the type of resynchronization modality used.

Keyword: Interventricular Dyssynchrony, Heart Failure, CRT Therapy, Narrow QRS.

Appendix



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Unexpected pacemaker implantation for sinus node dysfunction after atrial arrhythmia ablation in an adult with repaired tetralogy of fallot

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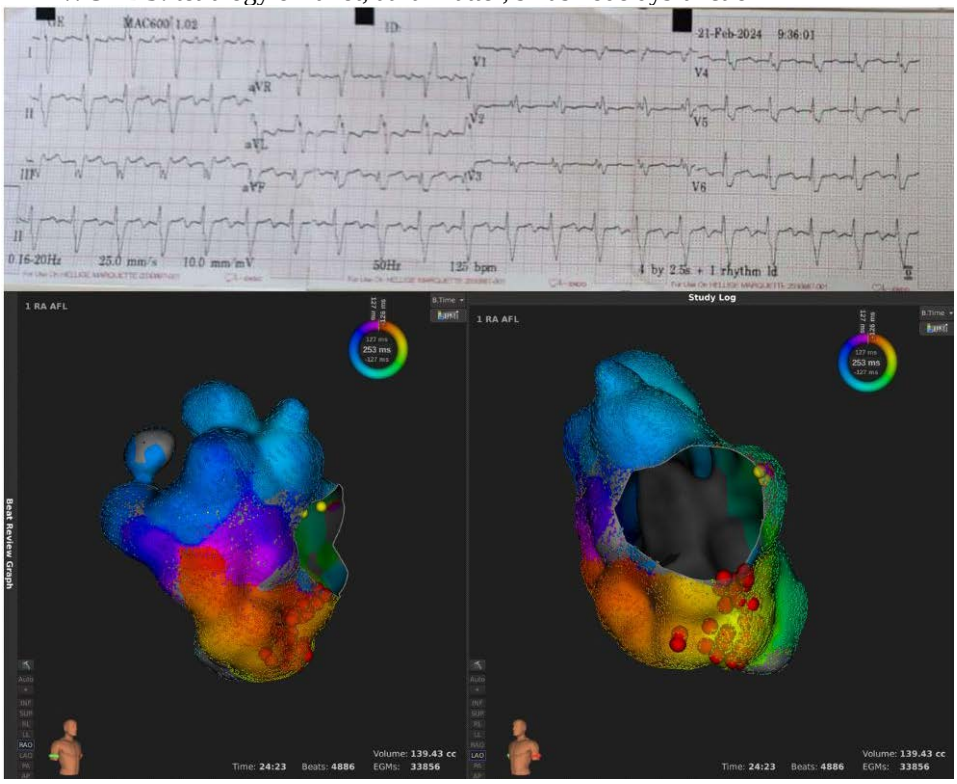
BACKGROUND: Arrhythmias remain a major cause of morbidity and mortality for adults with repaired Tetralogy of Fallot (TOF). Atrial arrhythmias (AA) were presented in 34% of those cases. Sinus node dysfunction (SND) and AA frequently occur together and may perpetuate each other.

CASE ILLUSTRATION: A 30-year-old female was referred with increasing shortness of breath over the past week and also palpitations, which both was exacerbated by physical activity. The patient had a history of TOF that was surgically repaired when she was 3.5 years old and there had been no significant complaints thenceforward. On physical examination and vital sign, all findings were within normal limits, except for grade 1 obesity (BMI 33.33) and elevated heart rate of 136 beats per minute. ECG revealed a sinus tachycardia rhythm with complete RBBB. Echocardiography revealed dilation of the right atrium and right ventricle, with normal ejection fraction. Supraventricular tachycardia were the most notable finding on Holter monitoring, specifically atrial tachycardia, or possibly atrial flutter (AFL) as a differential diagnosis. Electrophysiology study (EPS) with 3D activation mapping was performed. Atrial burst pacing was given, then AA was induced with 3:1 conduction and the P-wave showed negative at inferior lead but positive at V1, suggesting for typical AFL. Radiofrequency ablation was applied at Cavo Tricuspid Isthmus (CTI) area and the rhythm converted into sinus. A short time later, junctional rhythm suddenly observed, hence SND were suspected and single-chamber atrial permanent pacemaker was implanted. AA and cardiac conduction disorder system may occur attributable to atrial scarring, structural and electrophysiological remodelling, or surgical injury from the TOF repair.

CONCLUSION: In such case, recognition of the underlying pathology is important to enhance our ability to design better therapeutic strategies. Catheter ablation for AFL in adult patients with surgically repaired TOF had a high success rate, besides using atrial pacemakers to treat SND is the key recommendation, henceforth could improve symptoms.

KEYWORDS: tetralogy of fallot, atrial flutter, sinus node dysfunction



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Diagnostic Evaluation of Atypical Chest Pain with Stepwise Supportive Examination : A Case Report

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Background: Atypical chest pain is a frequent complaint in a clinical setting. This condition is characterized by discomfort on the chest without a distinct reason. The possible underlying cardiac issues make this presentation a substantial source of ambiguity. To choose the best course of action, a diagnosis must be made accurately. The usefulness of a stepwise supportive examination in intermediate risk cardiac issues is investigated in this study.
Case Illustration: A female patient 56 years old with atypical chest pain was admitted to Soebandi General Hospital after 4 days of hospitalization in primary care. Normal vital signs and a non-contributory medical history were found. ECG examination also show normal presentation. A non invasive echocardiography revealed 48% of ejection fraction and no abnormalities in heart structure. Nonetheless, Treadmill Test (TMT) reveals ST depression at V3-V6 lead within Electrocardiography (ECG). Diagnostic coronary angiography (DCA), a more invasive procedure, was used to confirm this diagnosis. DCA shows a proximal stenosis in Left Anterior Descending (LAD) artery and non significant 80% lesion in distal Right Coronary Artery (RCA). Based on this result, the patient was treated with Percutaneous Coronary Intervention (PCI).
This case study demonstrates the potential advantages of a stepwise supportive examination. In this instance, the first tests used were non-invasive and less priced ones like treadmill testing and echocardiography. This method helped narrow the differential diagnosis by ruling out heart failure, structural abnormalities, and spotting ischemia in the ECG. This information then guided to the more definitive DCA, which injects contrast dye to the coronary artery. This is more costly and invasive in order to confirm the diagnosis. This approach guaranteed that the patient received a precise diagnosis, which in turn facilitated the appropriate course of treatment, in this case is PCI. Additionally, it facilitates efficient resource allocation by preventing superfluous operations.
Conclusion: This example illustrates the benefit clinically and cost-effectively in using a stepwise supportive examination method as a diagnostic tool. This strategy lowers the possibility of needless treatments, allowing the effective use of resources and patient care.

Keyword: Atypical chest pain, Stepwise diagnosis, Non-invasive tests, PCI procedure, Resource allocation

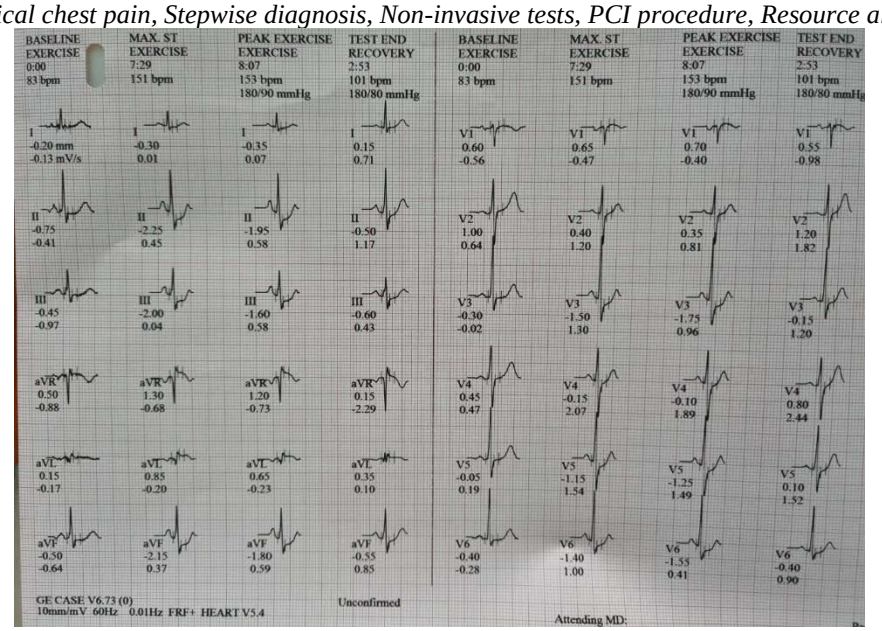

Figure 1. The Treadmill Test (TMT) examination shows positive stress test ST depression horizontal in V3-V6 lead.

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PEDIATRIC DILATATED CARDIOMYOPATHY AND CONGESTIVE HEART FAILURE: A Case Report

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Background: Cardiomyopathies are diseases of the heart affecting the systole, diastole, or cardiac cycle. Cardiomyopathy can be divided into primary (genetic, mixed, or acquired) and secondary categories, which ultimately led to varied phenotypes including dilated, hypertrophic, and restrictive patterns. Dilated cardiomyopathy (DCM) in the pediatric age group is characterized by unobstructed, dilated, and contracting left ventricular chamber defects and is associated with heart failure (HF). Clinical manifestations of DCM are mainly the symptoms of heart failure; it is diagnosed by EKG, chest x-ray and echocardiography. Pediatric HF is an important clinical condition with high morbidity and mortality. Management includes decongestive therapy, treatment of underlying causes, preventing progression, and managing pulmonary or systemic obstructions.
Case Illustration: An 11-year-old boy present with short breathlessness and cough, worsen by lying flat, alleviated by sitting upright, and peripheral edema for 4 days, he has no other history of illness prior to hospitalization. He was tachypneic and cyanotic. Vital sign: BP120/70mmHg, HR120x/m, RR30x/m, Temp37^{o}C, Sp$O_2$88%. Physical examination: Bibasilar rhonchi and wheezing, peripherals edema. Laboratory examination: Leukocyte 24.500/µL, ASTO and RF examinations were negative, other examinations are normal. Chest X-ray showed cardiomegaly. Echocardiography showed LA and LV dilatation, global hypokinesia, 21% ejection fraction, normal heart valves, and no structural heart defects. The patient was admitted for congestive heart failure and dilatated cardiomyopathy, and treated with intravenous Furosemide and Ceftriaxone, and oral medication Digoxin, Spironolactone, and Ramipril. After 3 days of hospitalization, the heart failure symptoms improved, and he is no longer tachypneic. The patient's parents requested for outpatient treatment and regular follow-up visits.

Conclusion: Pediatric DCM is associated with HF, and is an important clinical condition with high morbidity and mortality. Early identification and treatment can reduce symptom severity and improve outcomes.
Keywords: *Dilatative cardiomyopathy, pediatric heart failure*

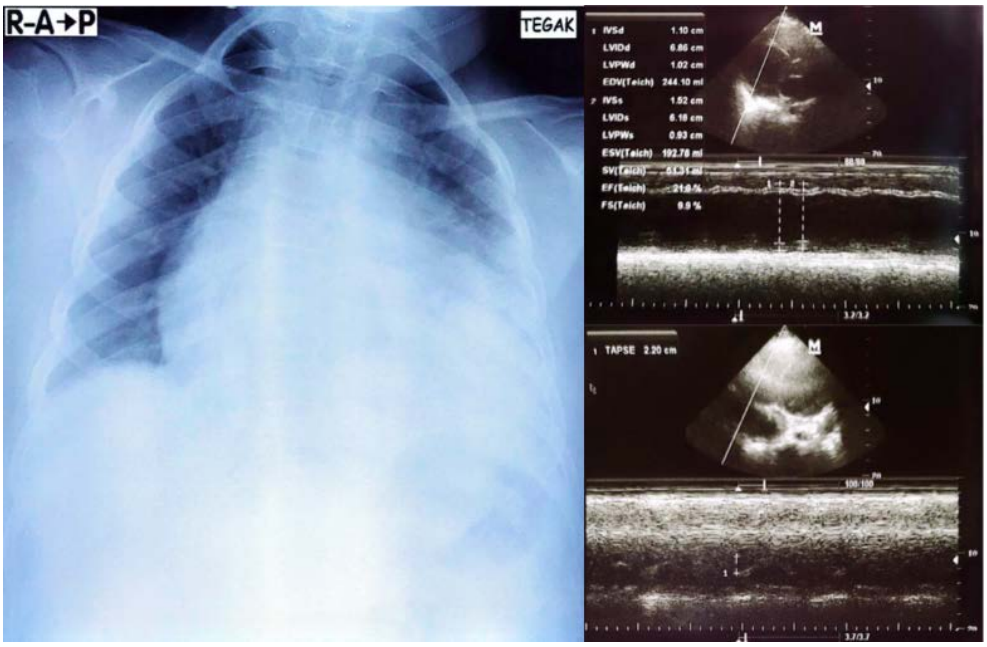

Figure 1. Chest X-ray showing cardiomegaly and echocardiography showing LA and LV dilatation, with global hypokinesia and 21% ejection fraction.

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Amiodarone-Induced R-on-T Phenomenon of Ventricular Premature Complex Bigeminy in a Acute Kidney Injury Patient with Paroxysmal Atrial Fibrillation

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Background: R-on-T phenomenon is the superimposition of ectopic R wave on T wave. It can be associated with or without QT prolongation. R on T associated with QTc (corrected) leading to ventricular arrhytmia (VA) which can lead to cardiac arrest. We are reporting interest case of R-on-T phenomenon in bigeminy premature ventricular response leading to sudden cardiac arrest in patient with atrial fibrillation after receiving drip IV amiodaron.
Case Illustration: A 70-year old female with acute kidney injury, type II diabetes mellitus and sinus tachycardia presented with complaints of fever of 38.8ºC, shortness of breath and anorexia. Her physical examination showed oxygen saturation of 74% room air, with respiratory rate of 28 times/minute. She had moderate lung congestion. Her blood glucose was 814 mg/dL. Her initial ECG showed sinus tachycardia, narrow ventricular response with normal QT and QTc interval. She received ambulatory treatment for hyperglicaemia and was directed to undergo haemodialysis. At the second day of hospitalization, she experienced a decrease in consciousness with a GCS of 12, and her temperature was 39.5°C, her ECG developed atrial fibrillation with rapid ventricular response with normal QT and QTc. She received antipyretics, and a drip IV dose of 150 mg amiodarone was begun for 30 minutes. After 10 minutes of infusion, she developed a sudden atrial fibrillation with slow ventricular response with narrow ventricular complex, intermittent bundle branch block, and ventricular premature complex bigeminy with R on T phenomenon. The amiodarone drip was immediately stopped. But not long afterward, she had a cardiac arrest. The cardiopulmonary resuscitation was performed but no signs of sppontaneous circulation.
Conclussion: Amiodarone is routinely administered to patients with atrial fibrillation without increasing mortality. However, intravenous amiodarone may cause serious conduction system abnormalities in certain patient.

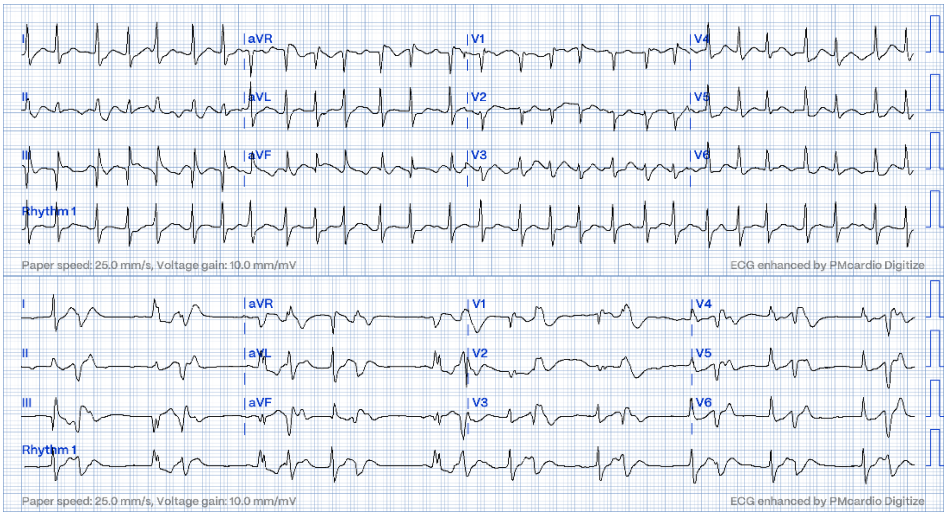

Figure 1. (A) Atrial fibrillation with rapid ventricular response normal QT and QTc interval of 460 ms. (B) ECG after 10-minutes administration of drip IV amiodarone

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Ivabradine Use for Slow Atrial Tachycardia Treatment in Patient with Cardiogenic Shock: A Challenging Case Report

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[2]*Intensive Care Unit, Gatot Soebroto Central Army Hospital, Jakarta, Indonesia*

Background: Ivabradine use in atrial tachycardia (AT) has already shown a benefit in some case reports. However, in patients with cardiogenic shock (CS) with slow AT, the condition it's more complicated because the use of inotropic makes AT more challenging to treat.
Case illustration: A -77-year-old male came to our ER with DOE accompanied by bilateral limb edema. He has a previous history of pulmonary tuberculosis and is suspected of COPD. On the second day, he deteriorated and moved into ICU due to respiratory distress and hypotension along with narrow complex tachycardia. The patient was lethargic with BP 88/53 mmHg, HR variate between 75 to 120 bpm, RR 23x/min, SpO2 100% with nasal cannula 4 lpm. The work of breathing was increased, accompanied by crackles on pulmonary auscultation with no sign of congestion. There were RHF signs like distended JVP, slight hepatomegaly, non-pitting lower limb edema, and cold extremities with CRT>2 seconds. The ECG showed slow AT with a CLBBB. The bedside echocardiogram revealed a mildly reduced LV and RV function with an intermediate probability of PH. We initiated dobutamine 3 to 5 mcg/kg/min for the CS but his HR increased up to 140 bpm. Amiodaron couldn't be used due to an increase in the liver enzyme so the digoxin was given, unfortunately, it didn't work so ivabradine was added. Following ivabradine administration, the patient's condition improved, AT resolved to sinus rhythm, his symptoms were relieved, and hemodynamics were stable so we could wean the dobutamine. Two days later the patient could step down to the ward.
Dobutamine use in the initial therapy of CS is needed for hemodynamic stabilization and to improve tissue perfusion. However, it can paradoxically worsen tachycardia through its arrhythmogenic effect. About 64% of AT was proved sensitive to ivabradine whose mechanism of work didn't directly affect hemodynamics.[1] This gives it more benefit as an anti-arrhythmia drug in this condition. Furthermore, studies have shown that combining ivabradine with dobutamine in advanced heart failure patients effectively blunts the dobutamine-induced heart rate increase along with an improvement in LV diastolic function and right ventricular contractile parameters on echocardiography. [2,3]
Conclusion: Ivabradine may be used to treat AT in CS patients receiving inotropic support when anti-arhythmic drugs cannot be given.

Keywords: *slow AT; ivabradine; dobutamine; cardiogenic shock; inotropic*

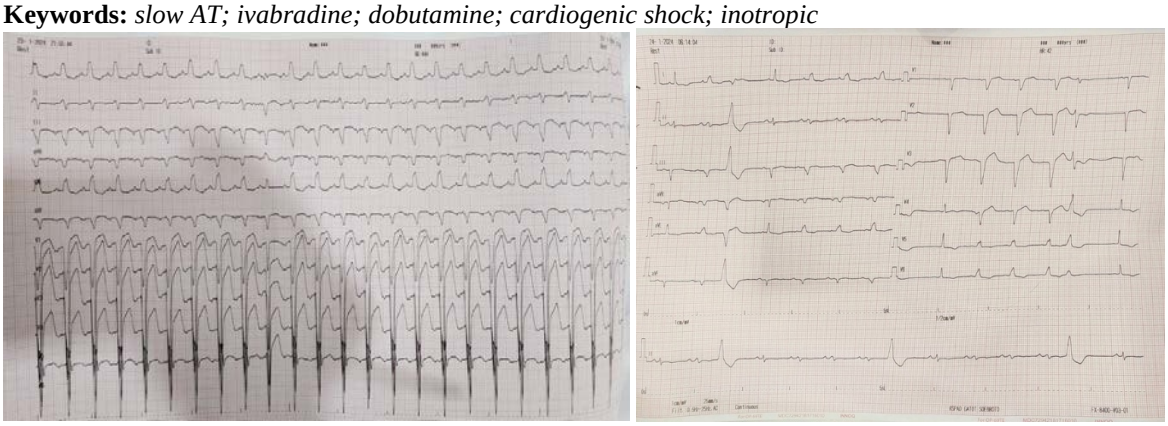

Figure 1. ECG result showed an atrial tachycardia suspected of right pulmonary vein origin which converted to sinus rhythm after ivabradine was given

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UNEXPECTED PACEMAKER IMPLANTATION FOR SINUS NODE DYSFUNCTION AFTER ATRIAL ARRHYTHMIA ABLATION IN AN ADULT WITH REPAIRED TETRALOGY OF FALLOT

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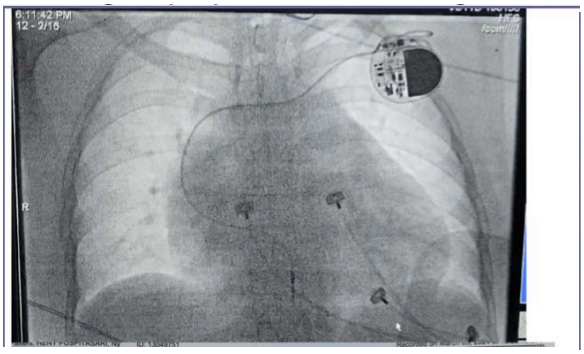
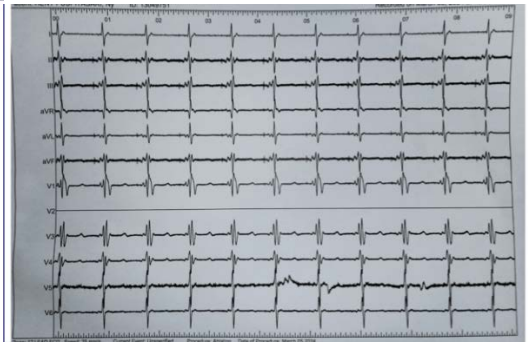
Background: Arrhythmias remain a major cause of morbidity and mortality for adults with repaired Tetralogy of Fallot (TOF). Atrial arrhythmias (AA) were presented in 34% of those cases. Sinus node dysfunction (SND) and AA frequently occur together and may perpetuate each other.

Case Illustration: A 30-year-old female was referred with increasing shortness of breath over the past week and palpitations, which both was exacerbated by physical activity. The patient had a history of TOF that was surgically repaired when she was 3.5 years old and there had been no significant complaints thenceforward. On physical examination and vital sign, all findings were within normal limits, except for grade 1 obesity (BMI 33.3 kg/m2) and elevated heart rate of 136 beats per minute. ECG revealed a sinus tachycardia rhythm with complete RBBB. Echocardiography revealed dilation of the right atrium and right ventricle, with normal ejection fraction. Supraventricular tachycardia were the most notable finding on Holter monitoring, specifically atrial tachycardia, or possibly atrial flutter (AFL) as a differential diagnosis. Electrophysiology study (EPS) with 3D activation mapping was performed. Atrial burst pacing was given, then AA was induced with 3:1 conduction and the P-wave showed negative at inferior lead but positive at V1, suggesting for typical AFL. Radiofrequency ablation was applied at Cavo Tricuspid Isthmus (CTI) area and the rhythm converted into sinus. A short time later, junctional rhythm suddenly observed, hence SND were suspected and single-chamber atrial permanent pacemaker was implanted. AA and cardiac conduction disorder system may occur attributable to atrial scarring, structural and electrophysiological remodelling, or surgical injury from the TOF repair.

Conclusion: In such case, recognition of the underlying pathology is important to enhance our ability to design better therapeutic strategies. Catheter ablation for atrial flutter in adult patients with surgically repaired TOF had a high success rate, besides using atrial pacemakers to treat SND is the key recommendation, henceforth could improve symptoms.

Keywords: Atrial Flutter, Sinus Node Dysfunction, Tetralogy of Fallot





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Atrial fibrillation and hyperthyroidism in infants with normal cardiac structure

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Background: Cardiac arrhythmias in pediatrics is very rare with 1% prevalence. Common arrhythmias occurring in neonates to infants include sinus tachycardia, atrioventricular reentrant tachycardia, and atrial flutter. Atrial fibrillation is quite rare in infants and children. The possibility of atrial fibrillation occurring in pediatric can be attributed to severe rheumatic heart disease, cardiomyopathy and hyperthyroidism. Uncontrolled atrial fibrillation can increase the risk of heart failure and thromboembolic events, leading to prolonged hospital stays.

Case Illustration: A 2-months-old boy weight 5.9 kg came to emergency department with history of dyspneu for 1 day prior to admission. The baby was born vaginally without any complications. There were no signs of cyanosis. On physical examination, the pulse was 216/min, blood pressure was 100/55 mmHg, respiratory rate was 35/min, temperature was 36.7°C, and SpO2 was 99-100%. An electrocardiogram revealed atrial fibrillation with a rapid ventricular response of 203 bpm. Chest X-ray showed slight left lung markings. Subsequent echocardiography revealed mild dilation of the left ventricle, 2mm patent foramen ovale with left-to-right shunt, and left ventricular ejection fraction 57%. The patient admitted to Cardio Vascular Critical Care Unit for stabilization. Blood tests showed elevated FT4 (1.65 ng/dL), TSH (6.73 ul/ml), and FT3 (5.3 pg/mL), suggesting subclinical hyperthyroidism. During CVCU treatment, the patient received amiodarone drip at initial dose 25 mcg/kg/min within 4 hours and continue with maintenance dose, and additional propranolol tablets. Then ECG monitor showed sinus rhythm with 146-160 bpm, with systolic 66-110 mmHg and diastolic 40-55 mmHg, SpO2 98-100%. The patient was managed in CVCU for 2 days and then transferred to ward without any symptoms with a stable heart rate <170 bpm.

Conclusion: The importance of understanding the relationship between disorders of the endocrine system and cardiovascular events cannot be overstated. Thyroid hormones serve both physiological functions and can act as arrhythmogenic agents. Patients with subclinical hyperthyroidism are at significant risk for developing atrial fibrillation. Although rare in pediatric cases, pediatricians should be cautious regarding AF in cases of hyperthyroidism in children with normal cardiac structure and function, as it can be a complication.

Keywords : atrial fibrillation, arrhytmia, hyperthyroidsm, pediatric.

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A Case Report of Supraventricular Tachycardia (SVT) : a Challenge in Acute Management in Very Old Patients

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Background: Supraventricular Tachycardia (SVT) is commonly a reason for emergency department visits. It is caused by the mechanism which involves tissue from the His bundle or above characterized by the presence of rates excess of 100 bpm at rest, narrow QRS complex, and frequent absence of P waves on ECG. Women have twice the risk and individuals more than 65 years of age have 5 times the risk of younger peoples. It happened because of frail conditions and comorbidities that patients have

Case Illustration: A 90-year-old woman came to the emergency department with near-syncope 1 hour before arriving at the hospital accompanied by palpitations and dizziness. The patient didn't have cardiovascular disease history but has a history of diabetes for 5 years. Vital sign was stable with 112/70mmHg blood pressure at the first measurement, without any chest pain nor signs of shock. An ECG showed rates 200 bpm, narrow QRS complex, and absence of P waves. Troponin T and electrolyte serum was normal. Vagal maneuvers are performed and bisoprolol 2.5mg was given to the patient under consideration for very old patients. There is no significant response because the patient is in a dementia condition and not cooperative to following the instruction. Diltiazem with an initial dose of 10mg bolus was given to the patient over 10 minutes followed by a maintenance dose of 2.5mg/h. After that, the patients heart rate decreasing gradually to 86 bpm. The patient was then being observed for 24 hour in the ICU and there were no signs of danger from cardiac condition

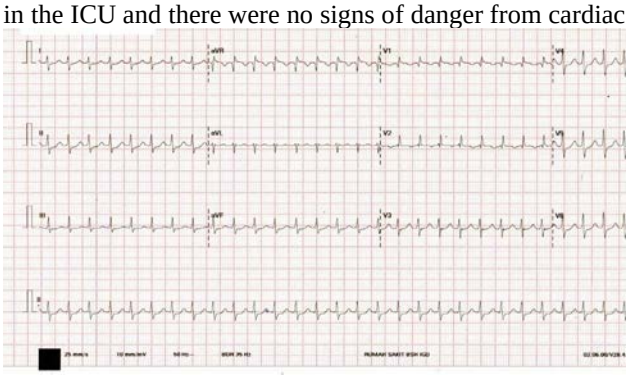

Figure 1. ECG upon arrival

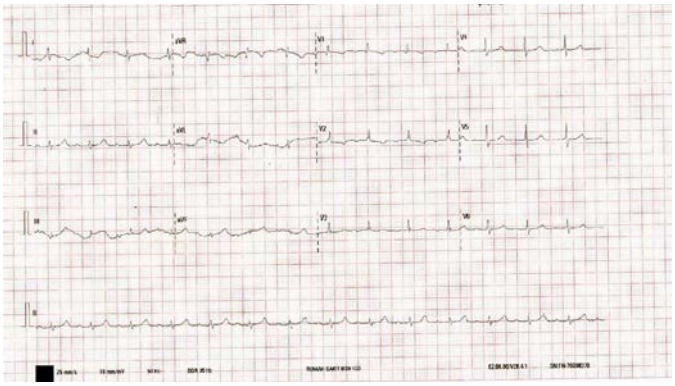

Figure 2. ECG after diltiazem adminitration

Conclusion: Lots of considerations in acute management SVT for Very Old patients included risk factor and side effects from therapy according to the guidelines. Intense observation of hemodynamic and cardiac electricity should be performed

Keyword: Supraventricular Tachycardia, Very Old, Dementia

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Case Report: Left Atrial Cardiac Myxoma Presenting with Atrial Fibrillation

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Background: Cardiac myxoma is the most common primary benign tumor of the heart, predominantly located in the left atrium. Patients may present with various clinical manifestations; some are asymptomatic, while others exhibit constitutional symptoms such as dyspnea, palpitations, and chest pain. In some cases, cardiac myxoma causes valve or outflow tract obstruction, resulting in atrial fibrillation (AF) that can lead to life-threatening events. Diagnosing these cases is often challenging due to the variety of presenting symptoms.

Case Illustration: A 50-year-old female was admitted to the emergency department with chest pain that began one week prior and worsened over the past two days. The pain radiated to her left arm and penetrated to her back. She also has cold sweats and shortness of breath. The patient was fully alert, and a blood pressure of 116/88 mmHg, a heart rate of 142 beats per minute with irregular rhythm, respiration rate of 22 breaths per minute, and an oxygen saturation of 98% on room air. A murmur in the mitral area was heard during auscultation. An electrocardiogram showed atrial fibrillation with a rapid ventricular response. Laboratory results showed normal cardiac Troponin T levels, and chest x-ray demonstrated cardiomegaly with early manifestations of pulmonary edema and left pleural effusion. The patient was administered 150 mg of Amiodarone over 30 minutes, followed by maintenance doses. The patient was then transferred to the Intensive Care Unit. A transthoracic echocardiogram revealed a left atrial myxoma measuring 4.8 cm x 6 cm, attached to the medial wall of the left atrium and covering part of the inflow tract. After treatment for one week, she had no complaints and her vital signs are stable.

Conclusion: The broad range of symptoms of cardiac myxoma that mimic many cardiovascular diseases may result in the condition being overlooked, potentially leading to critical incidents. Early diagnosis is crucial, and echocardiography is highly effective in detecting intracardiac tumors.

KEYWORDS: Cardiac myxoma; Atrial Fibrilation; Case report

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Brugada Syndrome: case report

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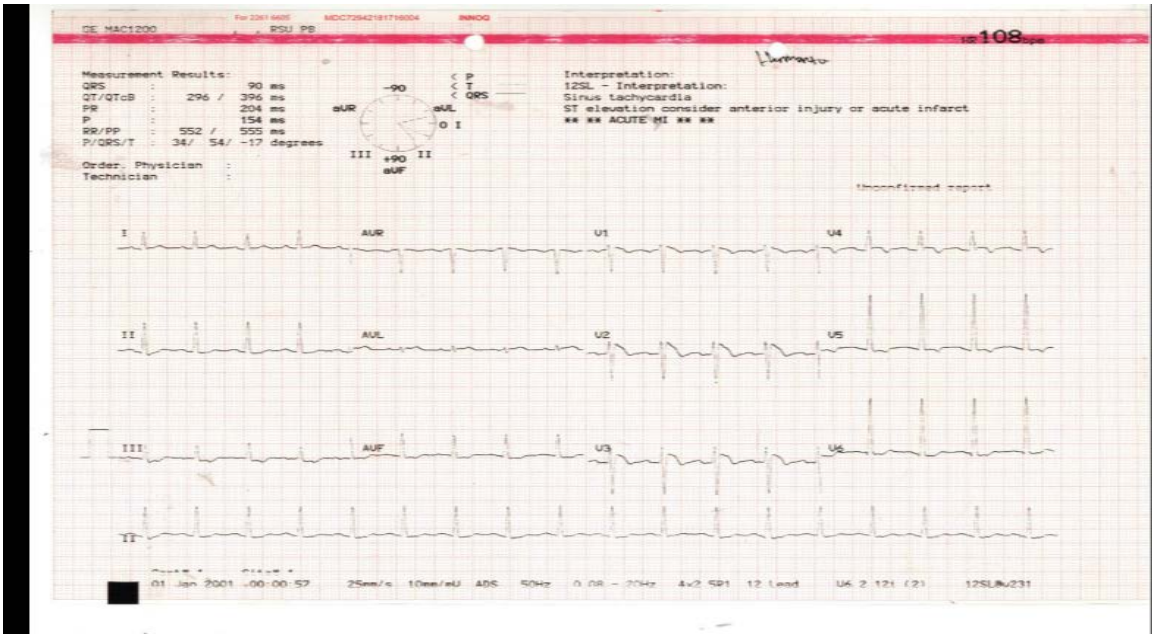
Background: Brugada syndrome (BS) is an arrhythmogenic disease characterized by coved ST segment elevation and J point elevation of at least 2 mm in at least two of the right precordial ECG leads (V1-V3). Brugada syndrome is a dangerous condition because it predisposes to malignant arrhythmias and sudden cardiac death. Fever is a trigger for malignant arrhythmias, syncope, or sudden death in BS. However, patients with Brugada syndrome show normal cardiac anatomy and no evidence of ischemia or electrolyte imbalance.

Case illustration: A 51 year old man was admitted to the Emergency Unit due to febris, nausea, vomitting, and abdominal discomfort. The patient did not have related palpitations, chest pain or shortness of breath. Laboratory investigations including hemogram, lipids, electrolytes, liver and renal profile as well as cardiac biomarkers were normal. Electrocardiogram showed a coved ST-segment elevation of ≥2 mm in leads V2 and V3, inverted T wave in V1-V4. Echocardiography (EF 60%) and X-ray chest revealed no abnormality. Based on these results, the patient was diagnosed with type 1 BS.

Conclusion: The pathophysiological mechanisms of BS are not fully understood yet, and even though many patients are asymptomatic. Brugada syndrome should be suspected in patients showing characteristic ECG changes in the form of ST segment elevation followed by inverted T wave in V1 and V2 leads along with clinical history of the patient as well as of his family members. Early recognition and intervention can save the life of the patient.

Keywords: Brugada syndrome, sudden cardiac death, arrhythmia.



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The Role of Chronotropic Effect of Hormonal Changes to Arrhytmia : A Case Report

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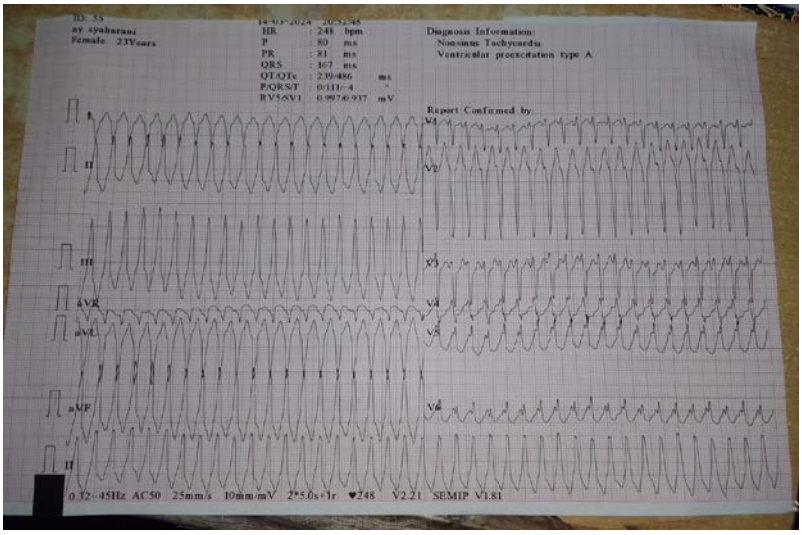
Background: An increased incidence of arrhythmia during and after pregnancy resulted in higher burden disease compared with pre pregnancy. Moreover, new onset VT may occur in normal structural heart. An increased plasma catecholamine, chronotropic effect of relaxin, an increased end diastolic volume, and hormonal changes contribute to pro-arrhythmia effect.

Case Illustration: A young female, 23 years old, came to the emergency room in March 2024 with the symptoms of palpitation along with chest discomfort 5 days before admission. This symptom deteriorated at that moment during evening along with shortness of breath. However, chest pain has not been reported. The patient also stated nausea, vomiting, bloating, and epigastric pain. Physical examination revealed hypotension 80/60, rate 240, respiration 24, and normal saturation. ECG examination showed supraventricular tachycardia with aberrant. The patient was administered amiodaron 200 mg within 100 cc/hour in the Intensive Care Unit. After the symptoms resolved, she explained that she had been to the Cardiologist 2 times in December 2023 and diagnosed with supraventricular tachycardia paroxysmal post pregnancy. Bisoprolol ¼ tab was administered in the morning at that time. She stated that the symptoms revealed 5 months after delivery. This patient had also been to the hospital with VES bigemini in January 2024 but she did not want to be admitted to the hospital. Eventually, the patient was discharged after 3 days inpatient with amiodarone.

Conclusions: Close monitoring of arrhythmia during and after pregnancy is essential to prevent mortality especially because of lethal arrhythmia. However, ventricular tachycardia with nonstructural heart disease is usually associated with good prognosis.

Keywords: Arrhythmia, Post Pregnancy, Chronotropic



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Acute Inferior Myocardial Infarction in Young Woman with Type 2 Diabetes Melitus at Primary Health Care Setting: A Case Report

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Background: Base on data of global burden of disease 2019, mortality of cardiovascular event on woman has higher amount, 35%. Woman has high risk of coronary disease when they get > 40 years old. Diabetes melitus is one of risk factor that increase cardiovascular event on young age.

Case illustration: A 35 years old woman complained epigastrium pain since 3 hours prior and nausea. Pain was radiated to the chest, back and left hand. Patient had diabetes melitus since 3 years ago, and took glimepirid 2 mg routinely. There was no history of hypertension, cardiovascular disease or stroke. There was no family history of cardiovascular disease and sudden death. Patient was a housewife and no routine exercise. Physical examination and vital sign was within normal limit. Electrocardiograph found that elevation ST-segment at inferior lead and depression ST-Segment at lateral lead (figure 1). Troponin I was negative and random blood glucose was 253 mg/dl. Patient was diagnosed inferior STEMI with type 2 diabetes melitus. Patient was given normal salin infusion, dual antiplatelet aspilet 160 mg chewed, clopidogrel 300 mg, isosorbit dinitrate 5 mg sublingual and injected fondaparinux 2,5 mg/24 hour subcutis for 3 days. Patient was also given injected novorapid 3 x 6 unit for controlling blood glucose. Reperfusion therapy was not done to this patient because lack of facility. Patient hospitalized for 3 days until the condition stable and planned refer to the cardiologist.

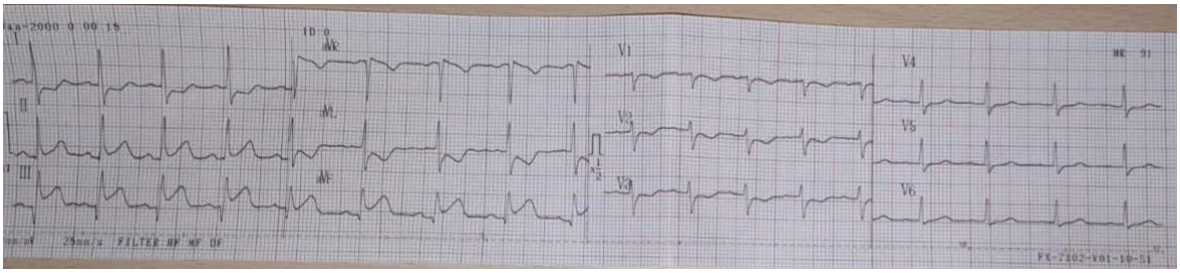

Figure 1. ECG result

Conclusion: The cardiovascular event of women has increased because of the high rate of diabetes melitus at young age. Patient had good condition after hospitalized for 3 days without reperfusion therapy because of the lack facility. Patient was not showed any symptom of complication until she left the clinic. Patient was suggested to the cardiologist for coronary angiography.

Key word: ACS, STEMI, diabetes melitus, young women

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Sustained Ventricular Tachycardia in a Rural Hospital: What to do?

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Background: Ventricular tachycardia is a type of abnormal heart rhythm, or arrhythmia, that requires emergency treatment to prevent sudden cardiac arrest. Acute treatments following guideline recommendations provide effective management during critical situations but should not replace clinical judgment.
Case illustration: A 46-year-old female came to the emergency room complaining of weakness and fluttering for three days. The patient also complained of chest pain for the last few months and had been under the care of the cardiology department. The patient was fully aware and had never experienced a syncope episode before. Clinically, the patient's appearance was stable with a BP of 101/70 mmHg, no chest pain, warm extremities, and no shortness of breath. Electrocardiography showed sustained ventricular tachycardia (VT). A vagal maneuver was recommended as the first step to manage the wide QRS tachyarrhythmia, but the EKG failed to convert. According to ACLS guidelines, anti-arrhythmic drugs such as Amiodarone 150 mg bolus IV were administered over 30 minutes, followed by a maintenance dosage of 1 mg/min for 6 hours. Unfortunately, there was no improvement in the EKG. Therefore, Amiodarone was terminated. Due to the malfunction of the defibrillator, Lidocaine became an option in this case. A Lidocaine 1 mg/kg bolus IV was administered, followed by a maintenance dosage of 2 mg/min, after which the EKG showed a sinus rhythm. The patient was transferred to the HCU for further examination and electrolyte correction if needed.
Conclusion: In this case, we demonstrated that Lidocaine can be used as an effective antiarrhythmic drug, even though it is not the first-line choice according to ACLS guidelines. This highlights the importance of clinicians being able to quickly identify and implement alternative treatments to address emergency situations and prevent sudden cardiac death.
Keywords: sustained VT, Amiodarone, Lidocaine, Rural hospital

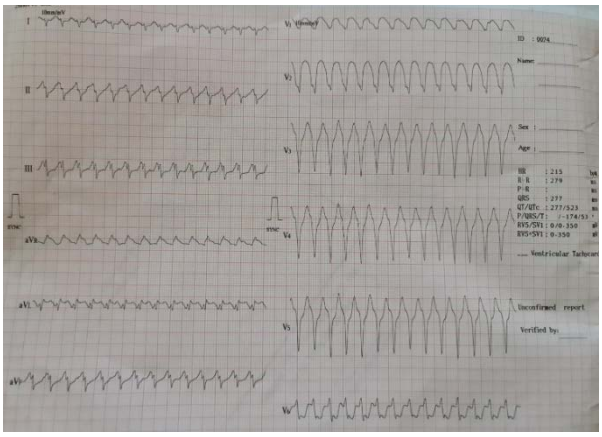

Figure 1: EKG showed sustained VT

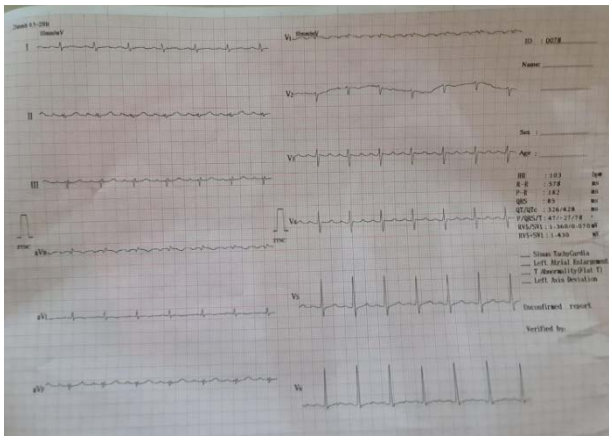

Figure 2: EKG showed sinus rhythm (converted by Lidocaine)

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Case Report: Recurrent Supraventricular Tachycardia in Children

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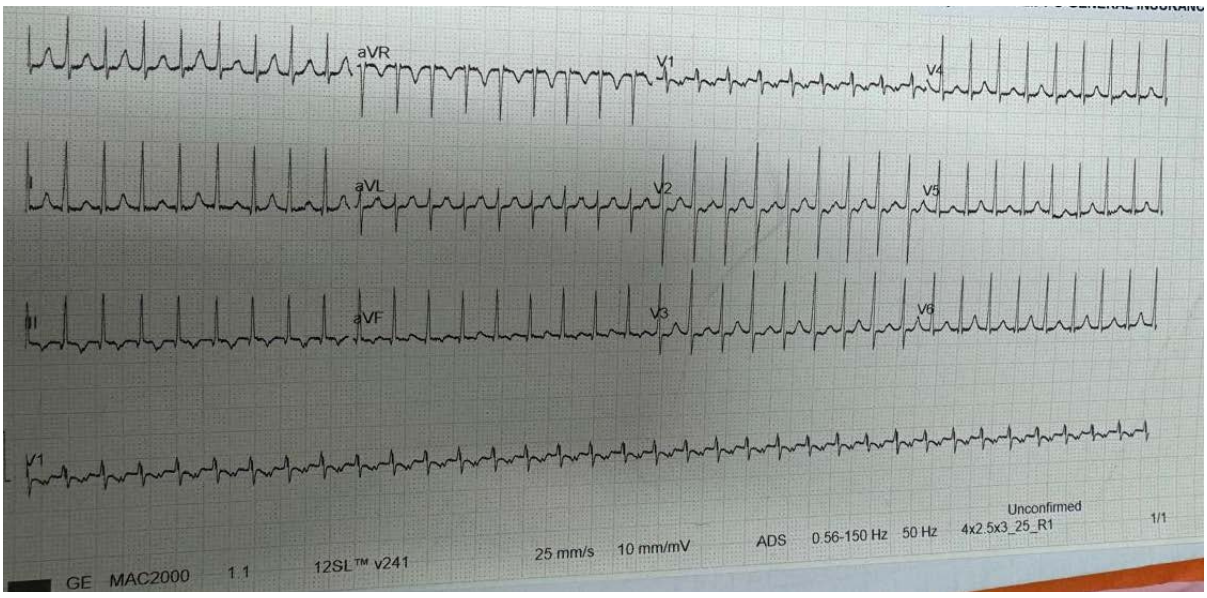
Background : Supraventricular tachycardia (SVT) is a common arrhythmia in children, characterized by a fast heart rate originating above the ventricles. Recurrent SVT poses significant challenges in pediatric care, requiring long-term management strategies for optimal outcomes. This case report provides insight into the presentation and management of recurrent SVT in a pediatric patient, emphasizing the need for comprehensive and individualized treatment approaches.

Case illustration : A 9-year-old boy arrived at the emergency room complaining of palpitations, weakness, decreased appetite, and a continuous cough lasting for one day. Upon examination, it was found that the pulse frequency had increased to 240 beats per minute. Electrocardiography showed a narrow QRS wave indicating SVT. This was the patient's third episode of SVT within the last 3 months despite taking routine propanolol. Chest x-ray and echocardiography results were normal, with a left ventricular ejection fraction (LVEF) of 64%. Laboratory results revealed leukocytosis. The patient underwent vagal maneuvers three times, but did not show improvement. Subsequently, the patient was administered intravenous therapy with digoxin 0.25 mg, adenosine 3 mg, and oral therapy with propranolol 10 mg. The patient was then treated in the Pediatric Intensive Care Unit for 24 hours and exhibited clinical improvement along with positive changes in ECG results. The patient was discharged with a plan for ablation at the Harapan Kita National Heart Center Hospital.

Conclusion : Recurrent supraventricular tachycardia (SVT) in children presents management challenges. Acute treatment with vagal maneuvers and adenosine can terminate SVT episodes, but long-term management may require medications or catheter ablation. The condition can impact emotional well-being, emphasizing the need for education and support. Ongoing research is crucial to improve patient outcomes in pediatric SVT.

Keywords : Recurrent, Supraventricular, Tachycardia, Children



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Malignant arrhythmia in Hyperthyroid patient

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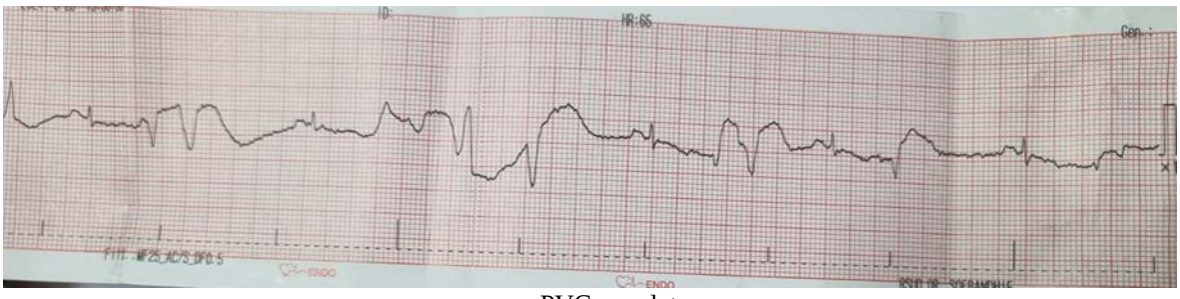
Background:
Increasing of thyroid hormones in the blood stream confer significant risk for MACE, including malignant arrhythmia. Thyroid hormones play a significant role in modulating heart rate, so that in condition with increased thyroxine hormones may also potentiate ectopic beat and atrial fibrillation. In the ventricles, it promotes triggered activity and formation of reentry circuit that giving rise to ventricular tachycardia, flutter and fibrillation[1].

Case Illustration:
In this case report, a 64 year-old women admitted to emergency room due to shortness of breath. Physical examination found ronchi at both lung and CXR shows pulmonary edema and bilateral pleural effusion. The Initial ECG showed the Atrial Fibrillation with rapid ventricular response that convert into sinus rhytm after initial management. The day after, the patient presenting cardiac arrest and got the spontaneous circulation after cardiac resuscitation and shock. ECG monitor at ICCU reveal PVC bigeminy, multivocal PVC, often cauplet PVC and rarely run of VT despite of continuous antiarrhythmia agent. The ECG also present polimorphyc VT (torsa de pointes). Laboratory finding shows normal electrolyte & blood glucose level, leukocytosis (14.700), increase of creatinine (1,7 mg/dl), elevated SGOT & SGPT (1713 & 1203 U/L), low TSH level (<0.05 uUI/ml) and high Free T4 (43,49).

Conclusion:
Usually we found atrial fibrillation as the manifesting arrhythmia in hyperthyroid patient. In this patient, several condition can cause the manifestation of ventricular arrhytmia. Increased SGOT and SGPT may indicate congestive hepatopathy that correlate with tachymyopathy since the duration of atrial fibrillation is unknown in this patient. Leukocytosis with increased vascular markings of lung signified the presence of infection/inflammation. Given the risk of malignant arrhytmia in deviation of thyroid hormones, it is important to achieved euthyroid state and maintain optimum clinical condition of the patient.

Keyword: hyperthyroid, arrhythmia, PVC, Torsa de pointes

Reference:
[1] Johannes W. Dietrich, Patrick Müller, and Melvin Khee Shing Leow. Editorial: Thyroid hormones and cardiac arrhythmia. *Front Endocrinol (Lausanne).*13: 1024476.Sept 2022



PVC couplet

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Acute lupus myocarditis with severe bradyarrhythmia: chaotic complications of a young man with systemic lupus erythematosus.

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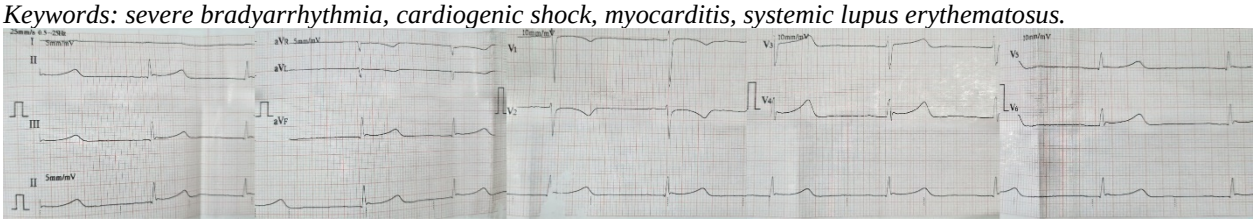
Background: Systemic lupus erythematosus (SLE), one of the most common systemic autoimmune diseases, is more frequently observed in females, particularly at a young age. Our case report highlights an uncommon and severe cardiac manifestation of bradyarrhythmia leading to cardiogenic shock in a male patient with newly diagnosed SLE. Additionally, the nature of this "great imitator" disease makes diagnosis and treatment challenging, especially in areas with limited resources.

Case Illustration: A 21-year-old male patient was admitted to the emergency room with chronic fever, vomiting, weakness, and weight loss. Physical examination showed low blood pressure and a slow, regular heart rate with no other abnormalities. The electrocardiogram (ECG) revealed a junctional rhythm with a rate of 40 bpm. Severe pancytopenia was also found from the blood sampling. The patient rapidly developed severe hypotension and cardiogenic shock. We administered atropine sulfate followed by continuous dopamine infusion and adequate fluid therapy, but the vital signs continued to deteriorate. We were unable to utilize a cardiac pacemaker due to the limited resources available at our facility. Echocardiography revealed severe hypokinesis of the left ventricle, with a reduced ejection fraction (EF) of 36%, pericardial effusion, and grade III diastolic dysfunction. Subsequent laboratory examinations showed positive ANA-IF, elevated CRP, and elevated Troponin I. A diagnosis of SLE was made and confirmed using the 2019 EULAR/ACR criteria. The patient showed improvement only after the administration of corticosteroids concomitantly with inotropic and vasoactive agents to support his failing heart. After a week of hospitalization, he was discharged and continued his medication as an outpatient.

Conclusion: In young patients with unexplained bradyarrhythmia accompanied by acute heart failure, autoimmune myocarditis must be considered as a differential diagnosis. The complexity of SLE diagnosis was simplified using the 2019 ACR/EULAR criteria. Uncontrolled hemodynamics despite drug administration underscores the importance of pacemaker availability, especially in areas with limited resources and remote locations.

Keywords: severe bradyarrhythmia, cardiogenic shock, myocarditis, systemic lupus erythematosus.



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TEMPORARY PACEMAKER TROUBLESHOOTING ON DAILY PRACTICE : A CASE REPORT OF FAILURE TO SENSE WARRANTS FOR REPOSITION

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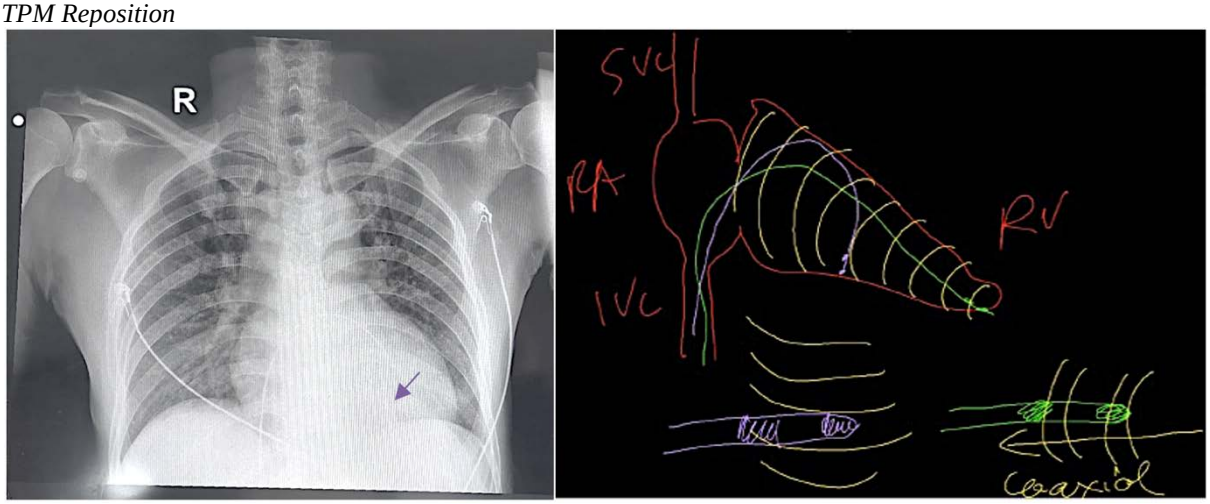
Background. Temporary pacemaker problem is common and often neglected. Failure to sense mainly happens because of inappropriate generator setting. However sometimes the problem can be caused by the lead inside. Sensing is a process of impulse detection between anode and cathode of the pacemaker. Bipolar sensing detects the intrinsic electrical activity occurring between the tip electrode and the ring electrode of the lead. Unipolar sensing detects electrical activity occurring between the tip of the lead, and the metal shell of a permanent pulse generator.

Case Illustration. A 59 years old man came to emergency room with total AV block in acute inferior STEMI that occured 1 day before admission. Primary PCI and temporary pacemaker placement was performed. Failure to sense was found 1 day after TPM placement, without evidence of failure to capture. Sensitivity setting was increased, and generator was replaced but the problem persisted. Echocardiogram showed that the lead was inserted properly up to right atrium, however there was no clear view of lead position in the ventricle. Chest X-ray revealed that the TPM was inserted into right ventricle, but not properly placed in the apex. We suspected the failure to sense to be caused by inappropriate angulation of the lead hindering the ability to sense while keeping its ability to pace. TPM reposition was performed and the TPM function return to normal.

Conclusion. In order for a pacemaker to sense an impulse, a time difference of incoming impulse between anode and cathode was required. Incorrect angulation of the lead resulted in impulse being detected at the same time resulting failure of detection. This situation warrants for TPM reposition.

Keywords: Transvenous Temporary Pacemaker Troubleshooting, Failure to Sense, Total AV Block in STEMI, TPM Reposition



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Recurrent Nocturnal Chest Pain in Patient with Brugada Syndrome and Myocardial Bridge: A Case Report

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Background: Brugada syndrome (BrS) is an autosomal-dominant inherited disorder without significant structural heart abnormalities, characterized by coved ST-segment elevation ≥2 mm with T-wave inversion at precordial leads (V1-V3). Brugada Syndrome is associated with sudden cardiac death (SCD) and ventricular tachyarrhythmias, such as sustained ventricular tachycardia (VT) and ventricular fibrillation (VF).[1-3,9] Patients with BrS often complain chest pain that must be differentiated from coronary artery disease (CAD). In this case report, we present a unique case of BrS patient with recurrent nocturnal chest pain.

Case Illustration: A 37-year-old male complaining recurrent chest pain during night time with type 1 Brugada Syndrome electrocardiogram (ECG) pattern. Angiography was done to exclude coronary artery disease. Insignificant coronary artery occlusion and myocardial bridging in mid left anterior descending (LAD) artery was found during angiography. Patient then underwent electrophysiology study (EPS), inducible polymorphic VT with normal sinoatrial (SA) and atrioventricular (AV) nodes function was found. Patient was diagnosed with Brugada Syndrome and implantable cardioverter defibrillator (ICD) was implanted after risk stratification. Despite this, he continues to experience occasional nighttime chest pain. Implantable cardioverter defibrillator reprogramming was done revealing no inducible VT after implantation, thereby we suggest recurrent chest pain is caused by myocardial bridging.

Conclusion: Recurrent nocturnal chest pain in BrS patients might be related to myocardial ischemia. In this case report, it might be associated with myocardial bridging found in mid LAD artery. This condition requires further treatment in order to terminate chest pain symptom.

Keywords: Recurrent nocturnal chest pain, Brugada syndrome, Myocardial bridging, Case report

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Sinus Arrest with Junctional Escape in Patient with Hyperkalemia due to Diabetic Nephropathy: A Case Report

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[1]Medical Faculty, University of Jember, Jember, Indonesia; [2]Department of Cardiology, Soebandi General Hospital, Jember, Indonesia

Background: Hyperkalemia is a potentially life-threatening metabolic problem that can cause arrhythmias, leading to cardiac arrest. One of the main causes of hyperkalemia is the kidneys' inability to excrete potassium. Diabetes mellitus (DM), one of the most common etiologies of kidney damage, is a risk factor for hyperkalemia.
Case Illustration: A 64-years-old man came to primary health care with the chief complaint of general fatigue. The patient had a history of hypertension and type-2 DM under treatment. Electrocardiographic (ECG) examination showed sinus arrest rhythm with junctional escape which was symptomatic bradycardia **(Figure 1)**. The patient's laboratory examination results showed hyperkalemia with a value of 9.14 mmEq/ml and a high creatinine of 2.17 g/dl. The potassium level was still high, 7.11 mmEq/ml, after initial correction. Thus, patient was referred for temporary pacemaker (TPM) installation. At the referral hospital, ECG showed normal heart rate. Laboratory examination still showed hyperkalemia with a value of 7.08 mmEq/ml and an increase in creatinine levels to 3.7 g/dl. Due to the normal heart rate, TPM installation was canceled. Instead, the patient underwent hemodialysis, and the post-hemodialysis evaluation showed an improvement in his heart rhythm.
Hyperkalemia, due to decreased glomerular filtration rate (GFR), reduces potassium excretion. This condition is likely to occur in patients with a history of hypertension and DM, which can lead to nephropathy. Upon presentation, the patient was in a state of acute kidney injury, indicated by elevated creatinine levels. The hyperkalemia caused disturbances in cardiac electrical activity during the ventricular repolarization phase. The ventricular repolarization became prolonged, potentially leading to symptomatic bradycardia. This condition indicated the need for TPM installation; however, heart rate became normal shortly. Interestingly, post-hemodialysis evaluation showed an increase in heart rate without use of pacemaker, along with a reduction in potassium and creatinine levels.

Conclusion: Diabetic nephropathy is one of the causes of reduced kidney ability to excrete potassium which can cause hyperkalemia. If hyperkalemia is not resolved, it will cause sinus arrest which can lead to cardiac arrest.

Keywords: diabetic nephropathy, hyperkalemia, sinus arrest

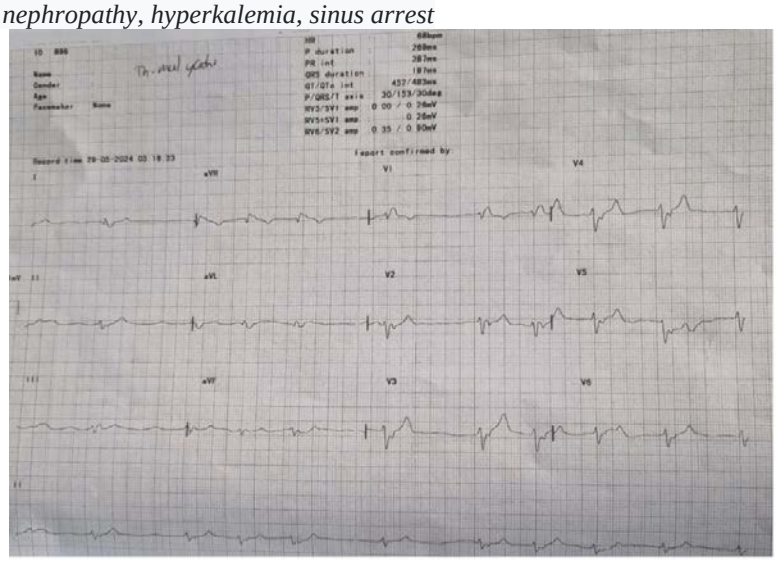

Figure 1. Electrocardiography (ECG) examination shows sinus arrest rhythm with junctional escape.

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From Chaos to Clarity: Identifying Atrioventricular Reentrant Tachycardia in a Busy Emergency Department

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Background: Paroxysmal Supraventricular Tachycardia cases are very common in the emergency department, but regardless of atrioventricular reentry tachycardia (AVRT), atrioventricular nodal reentry tachycardia (AVNRT) or atrial tachycardia cases become difficult when an ER doctor is dealing with a large number of patients or fatigue factors during night duty. We present a unique case that can help general practitioners to detect AVRT so that they can educate patients on further treatment.

Case Illustration: A 46-year-old man presented to Emergency Department with complaints of sudden chest palpitations for 1 hour. The frequency is continuous and does not improve with rest. The complaint was accompanied by tightness, which did not improve with change of position, and was particularly felt during light physical activity. The patient complained of cold sweats accompanied by abdominal discomfort. The patient had never had such complaints before. A family history of the same complaint was denied. Physical examination revealed blood pressure 120/67 mmHg, pulse 214 bpm, respiration 30 bpm, SpO2 95% room air. ECG examination revealed supraventricular rhythm, retrograde P wave buried before T wave with RP interval 120 ms, positive deflection P wave in avR, HR 214 bpm, inverted T in leads II, III, avF, poor R wave progression, so orthodromic AVRT was diagnosed. After pharmacological cardioversion with amiodarone 150 mg drip in 15 minutes, the ECG showed sinus rhythm, HR 89 bpm, normoaxis, PR interval 80 ms, delta wave, R/S ratio V1 >0.5, R/S ratio avF >1, so that the definitive diagnosis of WPW syndrome with LA/LL accessory pathway was made.

Conclusion: AVRT can be diagnosed by assessing the presence of a bump (P wave) after the QRS wave, then calculating the P to R interval > 70 ms in patients with supraventricular rhythms. Although the initial management for all paroxysmal SVT therapies is the same, and the diagnosis will be confirmed after rhythm conversion, the ED doctor who can differentiate AVNRT or AVRT will have a lot of fun and increase confidence during hospital duty.

Keywords: Atrioventricular Reentry Tachycardia, Emergency Department, Diagnosis

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PREGNANCY WITH TOTAL ATRIOVENTRICULAR BLOCK IN A REMOTE AREA

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Background: Total atrioventricular block is a very rare condition that can cause serious complications during pregnancy. The prevalence is estimated to be one in every 15,000 to 20,000 pregnancies. The cause can be either congenital or acquired. Total atrioventricular block can be asymptomatic and not recognized until adulthood. Pacemaker implantation is recommended in symptomatic pregnant patients.

Case Illustration: A 41-year-old female, G5P4A0 gravid 34 weeks, was referred by the primary health center for bradycardia, pre eclampsia and a history of heart block dating back a year. An electrocardiogram revealed total atrioventricular block with junctional rhythm. The prior echocardiography examination revealed LVEF 47.7%, mild global hypokinesia at rest, diastolic function grade I, trivial MR, low probability of PH, mild TR, and normal LV contractility. The patient's pregnancy was terminated via caesarean section due to eclampsia. Due to limited resources, pacemaker implantation cannot be performed.

Conclusions: Total atrioventricular block in pregnancy is a very rare condition, with congenital causes being the most common. The electrocardiogram is a very reliable and easily accessible tool for diagnosing this condition. Complications that can occur in the mother include cardiac arrest, while complications in the fetus include impaired fetal growth and development, oligohydramnios and premature birth. For patients with total atrioventricular block, a multidisciplinary approach and close monitoring of symptoms and cardiac function are required.
Keywords: Total atrioventricular block; Pregnancy; Pacemaker; Bradycardia.

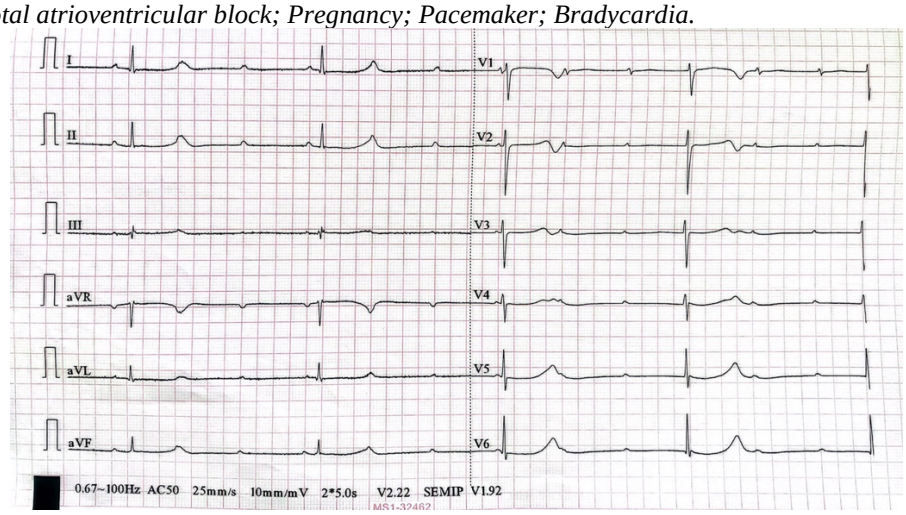

Figure 1. Patient's electrocardiogram

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Brugada syndrome In Rural Setting : A Case Series Seeing the Unseen

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Background: Brugada syndrome (BrS) is a type of arrhythmia disorder, which is characterized by abnormal electrocardiogram (ECG) findings and an increased risk of sudden cardiac death (SCD). We describe the two cases with brugada pattern that came to our emergency room.

Cases illustration: The first patient was 61 years old male came with chest pain and palpitation. There was neither previous history of any diseases, but he admitted there's family history of SCD. His blood pressure was 130/90, pulse rate 150 beats/min, Temp was 37.8ºC. the ECG revealed sinus tachycardia with Brugada pattern in V1 and V2. The laboratory result Ur/Cr 99/5,06 mg/dl AST/ALT 426/207 mg/dl. The patient was treated by oxygen & ISDN 5mg SL. Unfortunately, the family refused to be referred. The patient died several hours after diagnose established. The second patient was 54 years old male came with fever and chest pain. He admitted there was history of SCD in his sister. His temperature was 39'3 ºc, other vital sign and laboratory revealed no abnormality. The ECG revealed Type I Brugada pattern in V1 and V2. The patient was treated with antipyretic and ISDN 5mg SL and then was referred to another center for further evaluation.

Conclusion: Clinical presentation and ECG is the most important parameter in risk stratification and diagnosis in patient with Brugada syndrome. Fever are trigger that unmask Type I BrS.

Keyword: Brugada syndrome, Sudden cardiac death, Arrhythmia

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R-on-T Phenomenon Triggered Torsades de Pointes during Placement of Permanent Pacemaker in Complete Heart Block Patient with QTc prolongation

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Background: Torsades-de-pointes (TdP) is malignant tachyarrhythmia that can causing cardiac arrest. R on T is likely to initiate TdP and can be associated with QT-prolongation. Association between complete heart block (CHB) and QT-prolongation in literature is rare. Therefore we presented our case.
Case-Illustration: A-55-year-old female referred to our hospital to undergo elective permanent pacemaker (PPM) placement. She had history of myocardial infarction three years prior and was hospitalized for syncope with CHB two weeks prior. During procedure she was alert with BP of 103/62 mmHg, HR of 55 bpm, RR of 18 per-minutes with no significant laboratories finding. We noticed R-on-T at the beginning of the procedure, then suddenly the rhythm changed to TdP (figure-1). We performed cardiopulmonary resuscitation for 30-second before she regained consciousness. Overdrive pacing with temporary transvenous pacemaker was done as we continued the implantation of the PPM. Single chamber PPM was placed with VVIR mode and showed ventricular pacing at HR 60 bpm. She was observed for three days and discharged with no complaints. Later we inspected the ECG before the implantation and showed CHB with prolonged QTc by 533 ms. Bradycardia with prolonged QT interval is known to be one of the major factors predisposing to TdP. TdP preceded by QT interval prolongation are caused by congenital or acquired long QT syndrome (LQTS). Existing QT prolongation prolongs the duration of repolarization, making the heart more susceptible to premature impulses. When R-on-T occurs in this context, the risk for triggering TdP is increased because it occurs during ventricular repolarization period, exacerbating electrical instability and triggering TdP. Therefore identification of the cause is important. Studies suggested that patients with TdP in the setting of atrioventricular block represent a phenotypic manifestation of latent congenital LQTS. Implantation of PPM in this case have reduced QTc to 480ms and this suppressed occurrence of TdP.

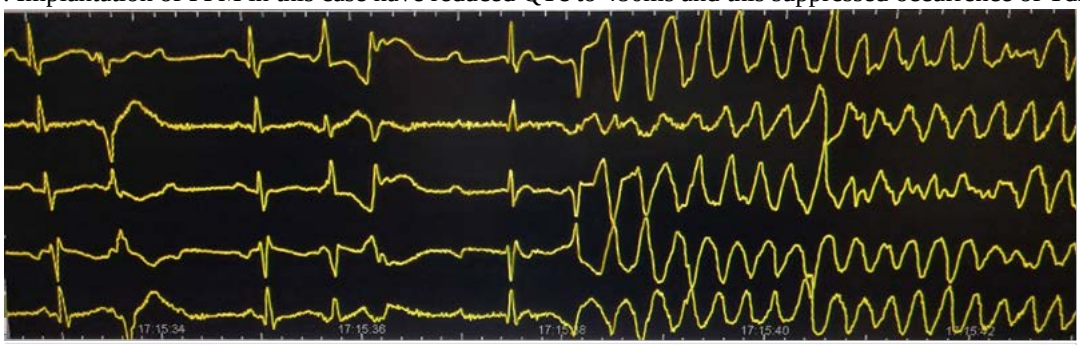

Figure 1. Torsade de Pointes triggered by R-on-T Phenomenon in Patient with Complete AV-Block

Conclusion: R-on-T is a critical condition that requires medical attention. Treatment and prevention of QT prolongation are essential to reduce the risk of TdP, in this case implantation of PPM has reduced the QTc and suppressed occurrence of TdP.

Keyword: R-on-T, Complete Heart Block, Torsades-de-Pointes

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SUCCESSFUL DIGOXIN TREATMENT TO TERMINATING ATRIAL FLUTTER IN PULMONARY ARTERIAL HYPERTENSION RELATED TO EBSTEIN ANOMALY

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Background: Ebstein anomaly (EA) is a rare congenital heart anomaly affecting 1 per 200,000 live births. Tricuspid valve abnormality in EA is the cause of pulmonary arterial hypertension associated with congenital heart disease. Atrial tachycardia can occur with this condition, with an incidence of 25%-65% for atrial fibrillation, atrial flutter (AFL), and ectopic tachycardia. There are limited studies concerning pulse and rhythm control treatment in this condition. A meta-analysis on sinus conversion in patient AFL stated that digoxin is not better than beta-blockers, calcium channel blockers, and amiodarone. AFL management is challenging for EA and pulmonary hypertension (PH) patients.

Case Illustration: A 28-year-old male patient with EA and PH was admitted due to complaints of dyspnea, palpitations, and cyanosis. The patient's electrocardiogram revealed AFL with 1:1 conduction and rapid ventricular response. Administration of digoxin i.v. 0.5 mg and oral 0.25 mg/day as maintenance to control pulse rate. Sinus rhythm was achieved four hours after digoxin administration. The patient gradually improved during clinical treatment and sinus rhythm was maintained.

Conclusion: AFL in EA and PH patients is caused by rapid atrium remodeling due to chronicity of right ventricular fluid. Digoxin is known as one of the therapies for pulse control in AFL with rapid ventricular rate. Digoxin administration can convert AFL to sinus rhythm and maintain it, especially in AFL patients with PH and EA.

Keywords: Atrial Flutter, Ebstein Anomaly, Pulmonary Hypertension, Digoxin

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Transurethral Resection of The Prostate Syndrome (TURP Syndrome) with Torsades de Pointes Case Report

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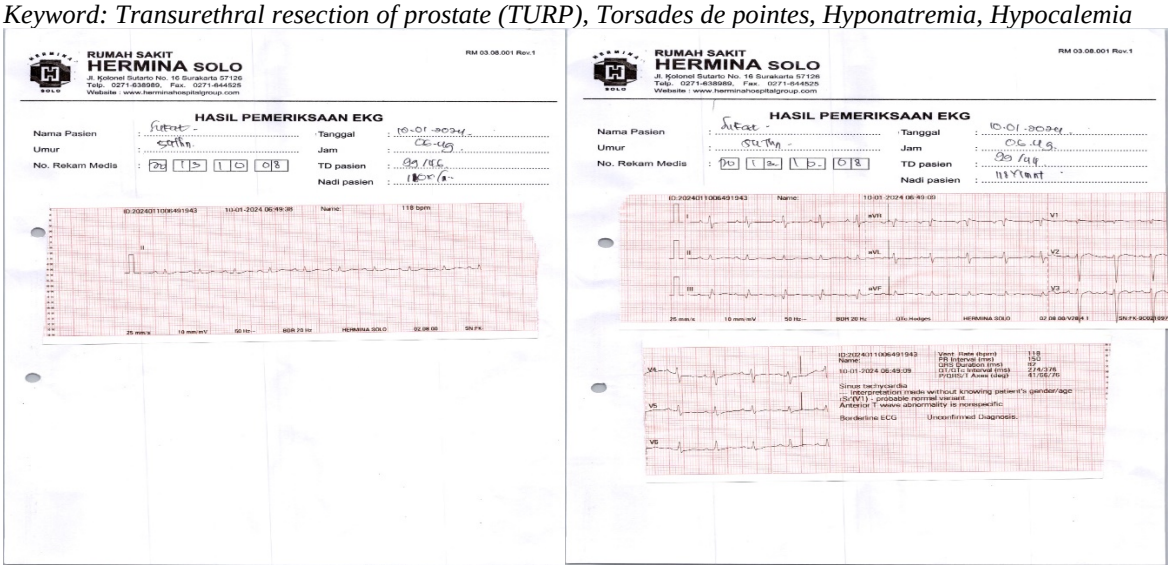
Background: Transurethral resection of prostate (TURP) syndrome is a systemic complication of transurethral resection of the prostate, caused by excessive absorption of electrolyte-free irrigation fluids. This syndrome may potentially hyponatremia and hypocalemia causes prolongation of QT interval. Torsa de pointes is one of the life threatening arrhythmias that associated with prolonged QT interval. Here we present a rare case of TURP syndrome causing hyponatremia and hypocalemia leading to Torsa de Pointes.
Case Illustration: A 54th years old male patient was complained of blood in urine and difficulty urination. Patient with medical history Non Hemmoragic Stroke and Hypertension, prostate volume 71.09 ml on ultrasonography. The heart rate at the start of the operation was 75 bpm, blood pressure 125/70 mmHg, normal electrocardiogram and SpO2 was 98%. Preoperative electrolytes were not checked. The patient was positioned in lithotomy posture and TURP surgery was started. Cystoscopy bladder neck was steep, kissing lobe prostate 2cm, and resection 30gr prostate. The irrigation fluid used by urologist was steril water and patient have been given 10-15L steril water. The total intraoperative bleeding was estimated at 250ml. Patient presented a severe TURP syndrome 5 minutes after surgery completed. Patient experienced altered mental status, hypoxemia, hypotension, seizure with Torsades de pointes heart's rhythm. Electrolyte analysis revealed an acute hyponatremia (sodium concentration 130.4 mmol/L) and hypocalemia (potassium concentration 3 mmol/L). Patient transferred to ICU after cardiopulmonary resuscitation, intubation and deffibrilation. In the ICU, the condition getting worse such as metabolic acidosis, intestinal bleeding, anemia, hypoalbumin and cardiac arrest. Patient died after four days in Intensive Care Unit.
Conclusion: Transurethral resection of prostate (TURP) syndrome can cause a wide variety of asymptomatic hyponatremia, hypocalemia, life threatening arrhythmia such as Torsades de pointes, and death.
Electrolyte examination should be performed before TURP operation especially those who have high cardiovascular rick facts to prevent electrolyte abnormalities that induce prolong QT interval in Torsades de pointes.

Keyword: Transurethral resection of prostate (TURP), Torsades de pointes, Hyponatremia, Hypocalemia



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Deceptive Pre-existing Right Bundle Branch Block: A Case Report on STEMI Mimicry

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Background: Acute Myocardial Infarction (AMI) remains a prominent cause of death globally. The outcomes depend on patient factors, the efficiency of Emergency Medical System, and system delays. Several rare or seemingly normal Electrocardiography (ECG) findings in ST-Elevated Myocardial Infarction (STEMI) equivalent patients can delay diagnosis and early therapy, thereby worsening the outcomes. Unlike new-onset Right Bundle Branch Block (RBBB), in patients with pre-existing RBBB, examining the morphology and changes in prior ECGs is crucial for the diagnostic process. Therefore, a better understanding of these mimicking ECG patterns would lead to early diagnosis and therapy for the patient and increase prognosis, respectively.

Case Illustration: We Report a case of a 39-year-old smoker male presenting with typical chest pain that began four hours prior, with a Visual Analogue Scale (VAS) rating of 8-9 out of 10. His ECG revealed a Bi-fascicular Block (RBBB with Left Anterior Fascicular Block) that differs in morphology and exhibits progressive ST-segment changes compared to his previously documented ECG, leading to suspicion of Left Anterior Descending (LAD) Artery occlusion. The patient was referred to nearest Percutaneous Coronary Intervention (PCI) center hospital, where the diagnosis of critical occlusion of proximal LAD Artery was confirmed, and PCI was conducted.

Conclusion: Pre-existing RBBB doesn't hinder the diagnosis of STEMI, but recognizing STEMI equivalents can be challenging. Early diagnosis and urgent therapy are crucial for improving outcomes, particularly given the association with increased mortality in AMI.

Keyword: RBBB, STEMI, PCI

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Antidromic Atrioventricular Reentrant Tachycardia due to Wolff–Parkinson–White Syndrome Mimicking Ventricular Tachycardia

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Background: Atrioventricular Re-entry Tachycardia (AVRT) associated with syncope as manifestation of an underlying, life-threatening arrhythmia might potentially be the harbinger of sudden cardiac death. Identifying the aetiology is imperative to provide appropriate treatment and prevent recurrence.
Case illustration: A 20-year-old male was admitted to the emergency department with sudden onset of palpitations, dyspnea and syncope. He didn't give any medical history or family history of Sudden cardiac death. His HR was 200 bpm, BP 71/46 mmHg, and was awake but mildly lethargic. ECG at presentation demonstrated a wide-complex tachycardia (WCT). We diagnosed the patient with antidromic AVRT with hemodynamically unstable. Multiple accessory pathways (APs) can develop in patients with Wolff Parkinson White (WPW) syndrome. WPW Syndrome describes manifest ventricular pre-excitation in combination with SVT and can be associated with syncope or sudden death. In 90–95% of patients, these SVTs are orthodromic AVRT, while approximately 5% are antidromic AVRT. Rarely, these APs can participate in antidromic AVRT which can be life-threatening and requires unique considerations for acute management and ultimate ablation. In this case, medical cardioversion was started based on Adult Cardiac Life Support (ACLS) algorithm. Within 1 hour ECG was converted to broad QRS complexes (120 ms) followed by P waves, short PR interval (<120 ms), and delta wave was positive. ECG findings are suggestive of WPW syndrome. After 3 days of hospitalization, his signs and symptoms decreased. Then the patient was given bisoprolol and transferred to EPS Center for further treatment. Many conditions can cause malignant arrhythmia including WPW syndrome.
Conclusion: The differential diagnosis of WCT might be challenging and includes both ventricular and supraventricular tachycardias. In young patients without structural heart disease experiencing WCT, an EP study should be offered to make a final diagnosis with the potential to provide definitive treatment.

Keywords: AVRT, WPW, SVT

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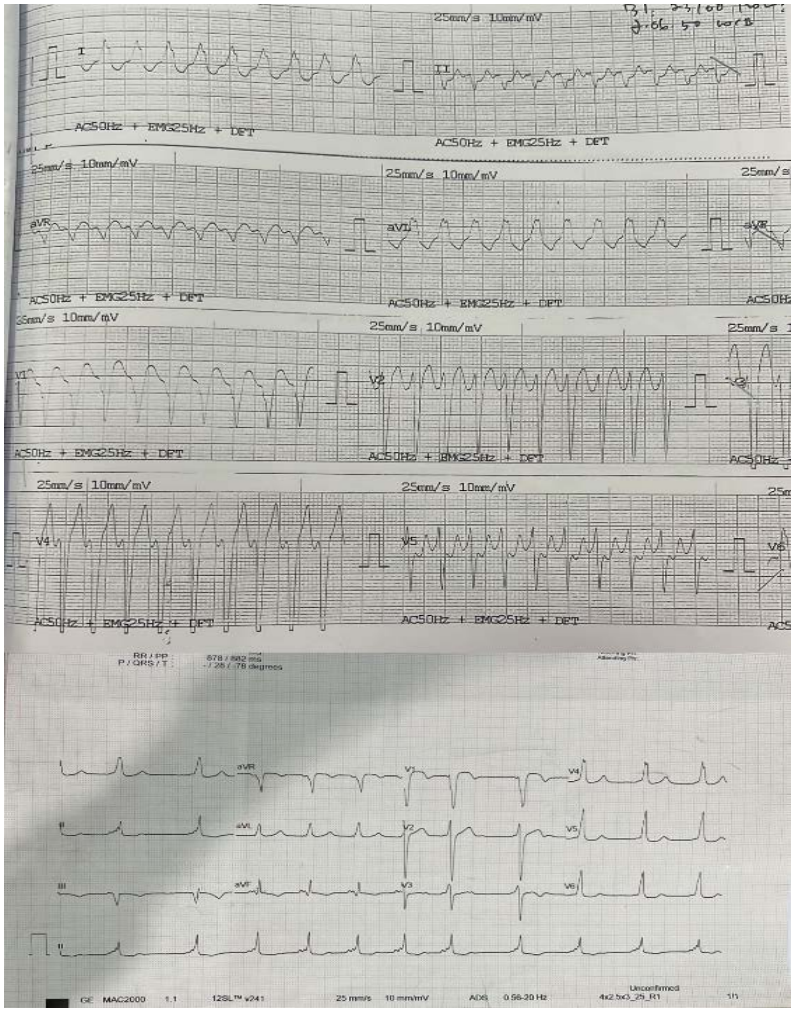

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Timing of atrial septal defect closure in the presence of atrial tachycardia

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Background: Atrial septal defects (ASDs) are the most common form of congenital heart disease, and account for approximately 6–10% of congenital heart defects, with approximate population incidence of 1–3 per 1,000. ASD typically results in a left-to-right shunt, resulting in right atrial enlargement, right ventricular dilation and, to a lesser extent, left atrial enlargement. Atrial tachyarrhythmias are commonly seen in patients with ASDs, regardless of ASD type. The left-to-right shunt enabled by the presence of an ASD results in cardiac remodelling secondary to long-standing hemodynamic overload, which plays an important role in the pathogenesis of atrial arrhythmias. NKX2-5 gene mutation may also explain the occurrence of familial ASD and conduction disorders.

Case illustration: A 58 years-old man was referred to our hospital complaining palpitation and chest discomfort. Patient also has decreased activity tolerance during last 2 years. He objected any history of heart complaints. Admission ECG from referring hospital showed a narrow complex tachycardia, 216 bpm, with long RP duration. Patient was already hospitalized for 3 days before being referred, receiving IV amiodarone and IV furosemide. Repeat ECG in our hospital showed atrial rhythm, 97 bpm, incomplete RBBB, RVH, with Crochetage sign. Transthoracic and subsequent transesophageal echocardiography confirmed the suspicion of Secundum ASD with L to R shunt, 18-24 mm in diameter and sufficient rims, with enlarged right atrium and right ventricle, and high probability of pulmonary hypertension. The patient was treated with sildenafil, bisoprolol, and ramipril, and scheduled for future electrophysiological (EP) study and radiofrequency ablation (RFA), before closing the ASD.

Conclusion: Atrial arrhythmia is a common occurrence in ASD that resulted from either triggers that generate ectopic activity or modifiers of substrate that promote reentry. Device closure of the ASD alone in patients with persistent atrial arrhythmia is not likely to restore sinus rhythm, regardless of the degree of reverse remodelling. Access to the left atrium may be restricted after device closure, therefore prior EP study and RFA instead of closing the ASD first is the preferred step.

Key words: Atrial tachycardia, ASD closure, reverse remodelling

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Supratherapeutic International Normalised Ratio (INR) In Warfarin Use Among Atrial Fibrillation Patient In Emergency Laparotomy, What's A Dilemma, A Case Report

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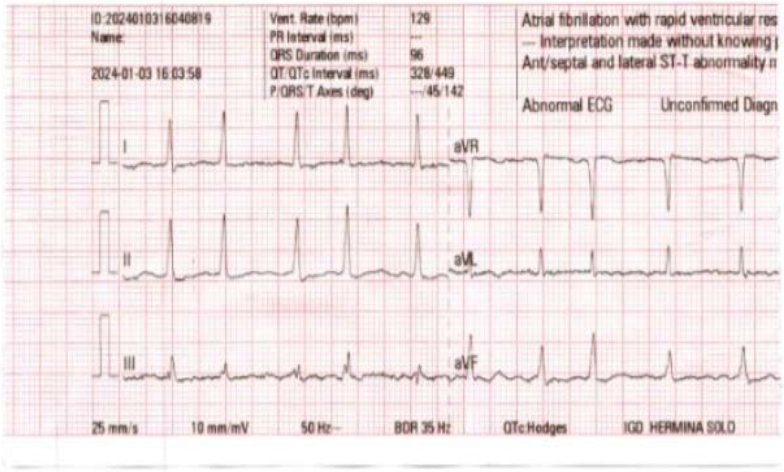
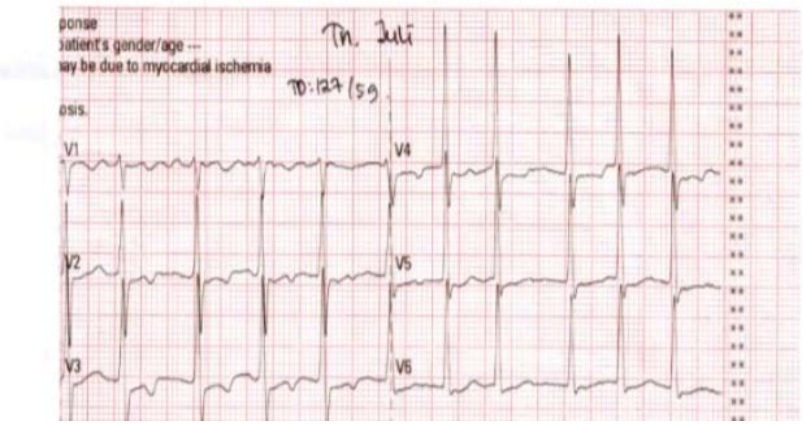
Background: Atrial Fibrillation (AF) is the most common cardiac arrhythmia, it characterised by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. Warfarin is recommended for most AF patients to reduce AF related deaths. Warfarin is proven to be an effective anti coagulant but it has a narrow therapeutic range which poses a challenge in the treatment of AF. International Normalised Ratio (INR) derived from the ratio between actual prothrombin time and that of a standardised control serum, it used as a guide to adjust the doses of warfarin. Supratherapeutic INR in AF is defined as an INR greater than the target range. One of the most common adverse reactions of supratherapeutic INR is haemorrhagic incidents.
Case Illustration: A referred patient from rural hospital diagnosed of intestinal obstruction suspect caused by bowel necrosis. Patient was treated for 3 days complained pain throughout the abdomen, obstipation and haematemesis. Patient has medical history of hypertension, heart failure and atrial fibrillation. Patient regularly takes warfarin but never checks the INR. INR was checked before surgery and the result revealed 9.48. Furthermore, using consideration from the surgeon, cardiologist and anesthesiologist, it decided for an emergency laparotomy with high risk condition. After surgery, patient experienced bleeding at the suture marks, drain and nasogastric tube. Routine hematology released an anemia (Hb 7.2 g/dl) encourage atrial fibrillation with rapid ventricular respons. Atrial fibrillation wasn't improved well and cardiac arrest happened. Patient transferred to Intensive Care Unit, cardiopulmonary resuscitation was performed but patient died.
Conclusion: Emergency surgery with supratherapeutic INR is a dilemma. INR monitoring is very important to patient who are on warfarin.

Keywords: Supratherapeutic International Normalised Ratio, Warfarin, Atrial fibrillation, Emergency laparotomy





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Unique ECG in Elderly Octogenarians: Sinus Node Dysfunction or Atrial fibrillation Only or "Tremor" effect

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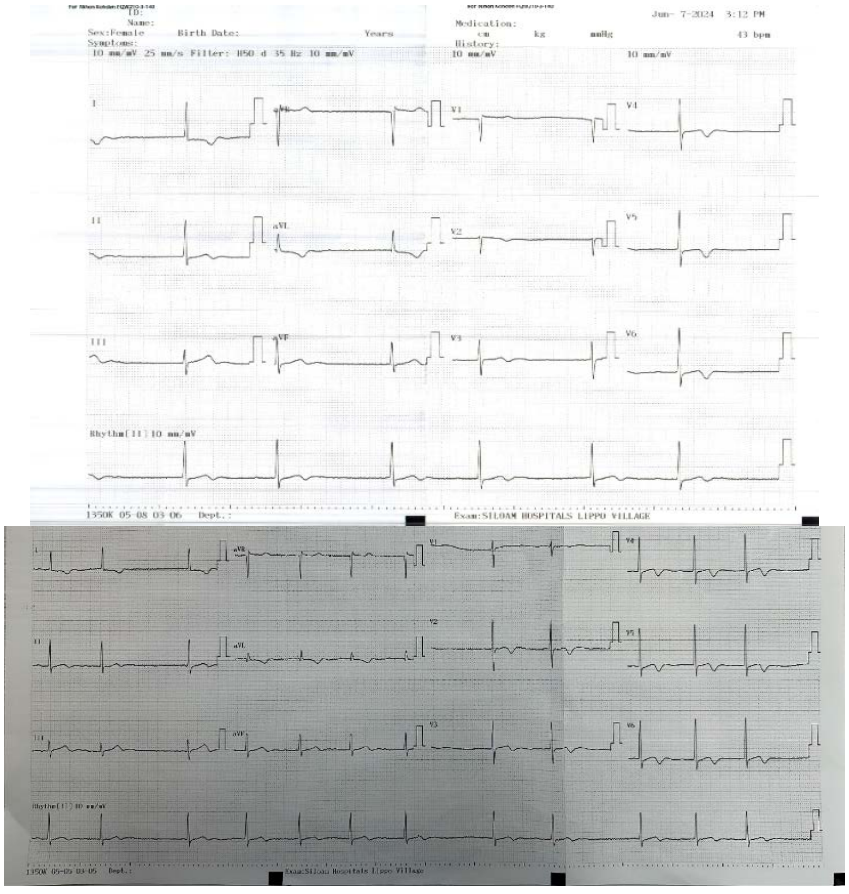
Background: SND (Sinus Node Dysfunction) is an umbrella term for conditions that either affect the automaticity of the sinoatrial node or blocks the impulse from reaching the atria. Disturbed automaticity and blocked impulses lead to arrhythmias that characterize sinus node dysfunction such as sinus bradycardia, chronotropic incompetence, sinus arrest and pause, sinoatrial block, also associated with a high risk of supraventricular tachyarrhythmias especially atrial fibrillation (AF) and atrial flutter.
Case illustration: A 99-year-old women presented with unstable blood pressure, and headache. No chest pain or dyspnea. History of hypertension since young, controlled with candesartan. On physical examination heart rate was 45 bpm, blood pressure was 154/54 mmHg, no murmur or gallop, good capillary refill time.
ECG examination showed junctional rhythm 42 bpm, with ST depression I aVL, bifasic T at V3-6. Echocardiography good LVEF 67%, global normokinetic. Next plan was laboratory check and Xray thorax.
After few days, the patient back with laboratory result, Xray thorax. The ECG was confused, between junctional or atrial fibrillation because the patient was "shacking" due to tremor. Therefore, after repeat ECG, and the result was same. The lab and Xray thorax were within normal. The plan was observation the blood pressure and pulse.
Conclusion: SND is a condition brady-tachycardia syndrome, it is commonly affect to elderly because degeneration. In this case, because the patient elderly came with bradycardia we need to established the SND. First, we determine the ECG of SND, in the limitation of "shacking" due to tremor to differentiate it with atrial fibrillation.
Also, we need to know if the patient had atrial fibrillation only or SND, because the treatment also different between them. In this case, we concluded that the patient had SND, not atrial fibrillation.

Keyword: SND, elderly, octogenarians



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Sinus Node Dysfunction Associated with Cerebral Ischemic Stroke in a 64-Year-Old Woman: A Case Report

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Background: Cardiac arrhythmias are commonly regarded as predisposing factors for ischemic stroke. Sinus node dysfunction (SND), previously known as sick sinus syndrome, is one of the arrhythmias that may cause ischemic stroke through hemodynamic or thrombo-embolic mechanisms, often potentiating each other. Patients with SND exhibit non-specific symptoms like dizziness, palpitations, fatigue, and confusion, which are associated with decreased cardiac output. Rarely, SND can lead to syncope, congestive heart failure, thromboembolism, pulmonary edema, cardiac arrest, or sudden death. This report presents a case of sinus node dysfunction with sinus arrest followed by the clinical manifestation of acute stroke.

Case Illustration: A 64-year-old woman with a history of uncontrolled hypertension presented with episodes of dizziness, nausea, and vomiting. Her blood pressure was 187/93 mmHg, and her heart rate was 93 bpm. A couple of hours later, the patient showed a decline in consciousness and left-sided body weakness. Vital signs showed blood pressure of 135/71 mmHg and a heart rate of 30 bpm, with normal blood test results, and an electrocardiogram (ECG) showed sinus arrest. A CT scan was performed, revealing an infarction in the right parietal cortex. She was sent to the intensive care unit and treated with intravenous fluids, an up-titration dose of dopamine, citicoline, anticoagulants, and a temporary transvenous pacemaker (TTPM) was immediately planned. Following TPM placement, her vital signs and clinical condition improved, with an ECG showing a return to sinus rhythm. However, due to persistent weakness and ongoing symptoms, the patient was referred to a cardiac center for permanent pacemaker (PPM) implantation.

Conclusion: Patients with SND should be considered at high risk of stroke, although such cases remain rare. Future research is needed to investigate the association between SND and ischemic stroke, for the prevention and management of SND patients.

Keyword: Sinus Node Dysfunction, Sinus Arrest, Ischemic Stroke, Pacemaker.

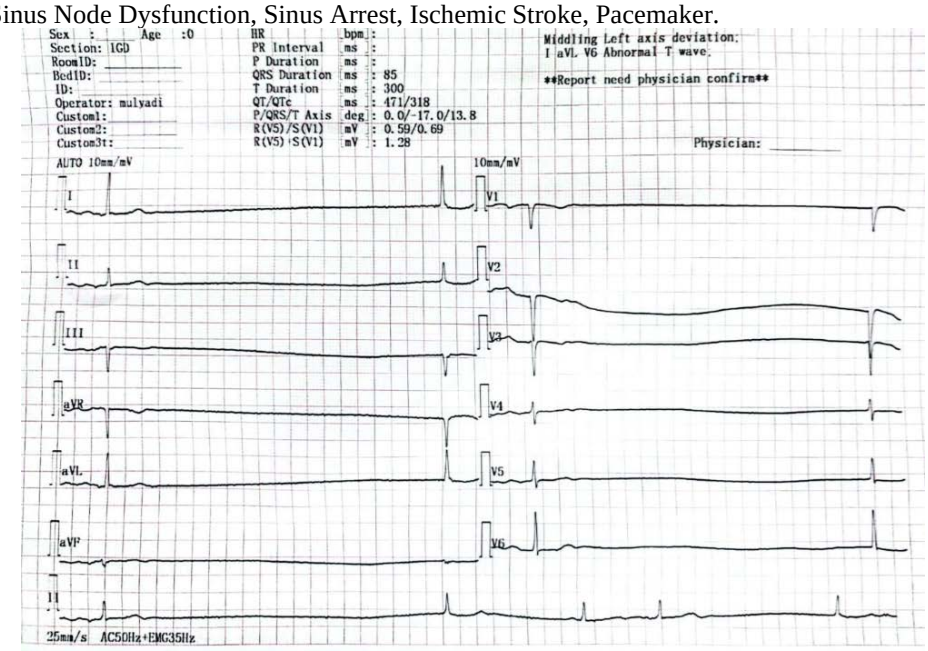

Fig 1. Prolonged absence of sinus node activity > 3 seconds.

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Sinus arrest related subarachnoid hemorrhage: a case report from rural area

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Background: Subarachnoid hemorrhage (SAH) is a life-threatening condition that result from the accumulation of blood between the arachnoid and pia mater. About 85% of nontraumatic SAH cases are secondary to aneurysmal rupture. Cardiac abnormalities following intracranial haemorrhage are well described. Here we presented a case of collapse, turned out to be SAH, and the ECG showed junctional bradycardia with episodes of sinus arrest and atrial fibrillation.

Case illustration: A 63 year-old male presented with headache then collapsed in the bathroom an hour prior to admission to emergency room. He had uncontrolled hypertension. Upon admission, Glasgow Coma Scale (GCS) showed E3V3M5, along with the following vital signs: blood pressure (BP) 180/100 mmHg, pulse per minute at 30 beats, respiratory rate per minute at 22 breaths, oxygen saturation at 98% in room air, as well as 37.7°C body temperature. Neurological examinations showed central nervous VII paresis with left lateralization. An initial electrocardiogram (ECG) revealed junctional bradycardia (28 beats per minute) with episodes of sinus arrest and atrial fibrillation. Cranial computed tomography (CT) scan was performed and revealed diffuse subarachnoid haemorrhage (SAH) in right cerebellum. SAH was treated using haemostatic drug and intracranial pressure lowering therapy. Initial therapies for bradycardia were administered. The patient then referred to advanced cardiocerebrovascular centre.

Conclusion: Abnormalities of ECG were commonly seen in patients who happened to SAH. According to literatures review, significant arrhythmia happened to about 5% of SAH patients. Physicians should keep in mind of junctional bradycardia not certainly as a cardiac problem, it may appear in a cranial emergency.

Keywords: Bradycardia; cerebral-cardiac event; cerebral-cardiac event; subarachnoid haemorrhage

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Suppression of Premature Ventricular Contractions by One-Month Pacing: A Case Report from Single Regional General Hospital

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Background: Premature ventricular contractions (PVCs) that comprise more than 15% of total heartbeats can induce cardiomyopathy in patients with systolic dysfunction. Cardiac ablation is commonly employed to decrease PVCs in this specific patients group, but it may not always easily accessible. We report a case demonstrating an elderly patient experienced ventricular tachycardia (VT) and high degree atrioventricular block accompanied by bigeminy PVCs, which managed with the implantation of a permanent pacemaker (PPM).

Case illustration: A 86-year-old man presented to the ER with worsening dyspnea and an episode of palpitation lasting for one hour. On physical examination, the pulse rate was 180 beats per minute, the blood pressure was 90/60 mmHg, and the other vital signs were within normal limits. The chest was clear on auscultation, and the general examination was normal except for pallor. The electrocardiogram (ECG) in the ER showed monomorphic VT of right-bundle branch morphology. The echocardiography revealed impaired LV function with a 29% ejection fraction (EF), global hypokinesis, a TAPSE of 2.1 cm, mild MR, and mild TR. As he was hemodynamically stable, an intravenous infusion of amiodarone was started, and sinus bradycardia was reverted within an hour. On the second day of hospitalisation, the ambulatory ECG showed AV block grade II type II with bigeminy PVCs. Thus, a temporary pacemaker (TPM) was decided to be implemented. Following TPM placement, her vital signs and clinical condition improved, but PVCs were still detected. We didn't find other reversible causes of bradycardia, so she proceeded to PPM implantation septum pacing (single chamber Medtronic). After one month of follow-up, the patient wasn't feeling any complaints, and his ECG showed normal pacemaker rhythms without any PVCs.

Conclusion: This suppression of PVC activity following one month PPM might be caused by ventricular remodelling of the original ectopic focus. However, further electrophysiology study is needed to approved this hypothesis while the access very difficult.

Keywords: Premature Ventricular Contractions, Atrioventricular Block, Ventricular Tachycardia

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A Case Report: Cardiac Arrest Survivor due to Complete Atrioventricular Block and Cardiogenic Shock In a Young Patient with Hypertrophic Obstructive Cardiomyopathy

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BACKGROUND: Hypertrophic Obstructive Cardiomyopathy (HOCM) is a genetic condition characterized by the thickening of the heart muscle, particularly affecting the interventricular septum and ventricles. The most alarming complication associated with HOCM is sudden cardiac death (SCD), caused by life-threatening arrhythmias or mechanical obstruction of blood flow. This report highlights a distinctive case of a young adult with HOCM and Complete Atrioventricular Block (CAVB), who presented with clinical weakness, dyspnea, severe bradycardia and recurrent cardiac arrests. Interestingly, the condition worsened after vasopressor and inotropic drugs were administered. However, successful resuscitation followed by the insertion of a temporary pacemaker (TPM) and subsequent placement of a dual-chamber permanent pacemaker (PPM) resulted in clinical improvement and his eventual discharge.

CASE ILLUSTRATION: A 22-year-old Asian man with no significant medical history presented to our emergency department due to weakness and shortness of breath. On examination, he showed severe bradycardia with HR of 33 beats/m, BP at 67/32 mmHg, a systolic murmur at the cardiac apex and cardiomegaly on chest X-ray. The ECG revealed atrial flutter with 3:1 conduction, right bundle branch block and signs of left ventricular hypertrophy. The rhythm monitor later indicated a third-degree atrioventricular block (TAVB). His condition deteriorated, leading to respiratory distress, desaturation, and hypotension, even after being given vasopressor and inotropic drugs. He experienced recurrent cardiac arrest but was successfully resuscitated. Then, TPM was carried out quickly and his condition improved. Echocardiography showed concentric LVH and ejection fraction was 64%. An LV outflow tract obstruction was noted, with a pressure gradient of 35 mmHg at rest. A dual-chamber-permanent-pacemaker (DDDR) was implanted. Following pacemaker placement, the patient showed significant improvement, was weaned off support, extubated, and discharged.

CONCLUSION: Our case underscores the importance of recognizing and managing HOCM with CAVB promptly. Early intervention, including temporary pacing and subsequent permanent pacemaker placement, can lead to favorable outcome.

Keywords: cardiomyopathy, cardiac arrest, permanent pacemaker

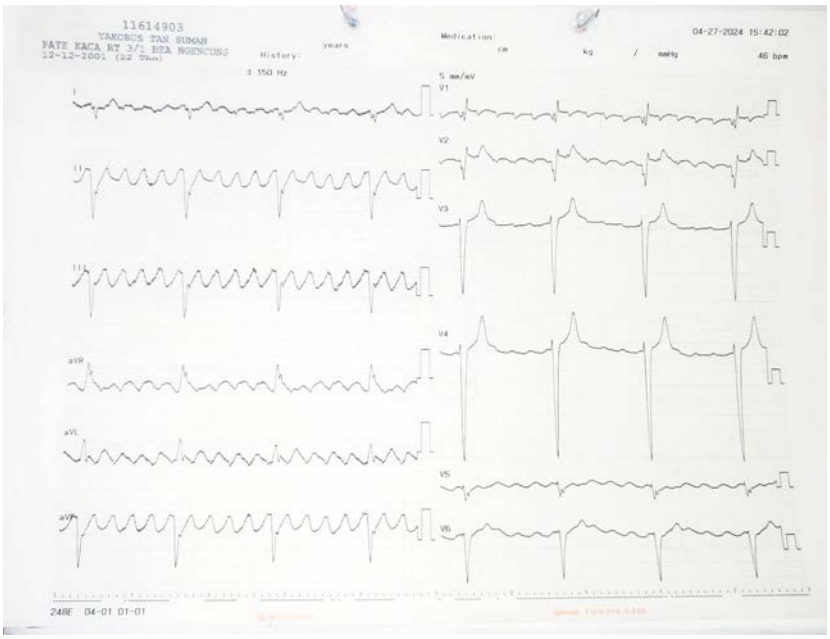

Figure 1: ECG

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Pharmacological Cardioversion on Unstable Atrioventricular Nodal Reentrant Tachycardia Patient at a Secondary Hospital : A Case Report

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Background: Atrioventricular Nodal Reentrant Tachycardia (AVNRT) is the most common form of paroxysmal supraventricular tachycardia. It is characterized by a reentry circuit within the atrioventricular node, which can lead to significant hemodynamic instability. Effective management is crucial to restoring normal sinus rhythm and stabilizing the patient through immediate electrical cardioversion with sedation.

Case Illustration: A 50-year-old male with a history of hypertension and heavy smoking presented to the emergency department of a secondary hospital with palpitation, lightheadedness, and syncope. Vital signs showed low blood pressure at 70/50 and extreme tachycardia at 220 x/min. The ECG showed AVNRT, and the patient was planned for emergency electrical cardioversion, but the patient refused. The patient was treated with an amiodarone drip infusion backed by a dobutamine drip infusion for the treatment of unstable AVNRT. The subsequent ECG converted to sinus rhythm, and improvements in vital signs were found in the patient.

Conclusion: Treatment options for the patient were limited due to the secondary hospital settings. Amiodarone works to terminate AVNRT by prolonging refractoriness and slowing conduction through the AV node and ventricles. However, its use is limited by a significant incidence of adverse effects, including symptomatic bradycardia and a high rate of serious adverse events. While dobutamine can improve cardiac output and stroke volume in certain conditions, its tendency to increase heart rate and induce arrhythmias necessitates careful consideration and monitoring. Electrical cardioversion remains the treatment of choice for immediate rhythm control in unstable patients.

Keyword : AVNRT, Hemodynamic Instability, Amiodarone

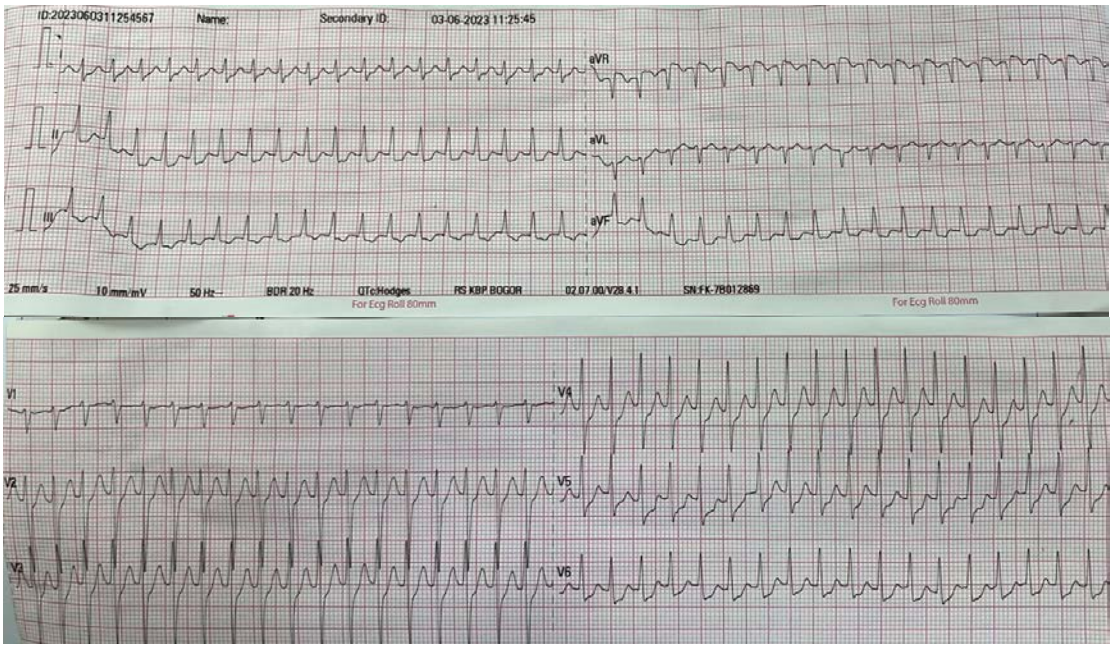

Figure: 12-Lead ECG when the patient presented to the emergency department

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Fever-Induced Brugada Type 1 Pattern with Transient Inferior T Wave Inversion in Exacerbated Chronic Obstructive Pulmonary Disease (COPD): A Case Report

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Background: Brugada syndrome (BrS) is inherited cardiac channelopathy characterized by coved or saddle-back ST segment elevation in V1-V3 terminating in an inverted T wave, but typically not in the inferior leads. It is associated with a high risk of ventricular arrhythmias (VA) and sudden cardiac death (SCD) in patients with structurally normal hearts. These ECG changes can be unmasked by many factors, including fever.

Case Illustration: A 68-year-old male with COPD presented with acute exacerbation, including worsening dyspnea, productive cough, atypical chest pain, and a fever of 40.5°C. His SpO2 was 85% with a respiratory rate of 28 breaths/min. Physical examination revealed wheezing and coarse rhonchi. The initial ECG showed a rightward QRS and P-wave axis (inverted in aVL), P pulmonale, clockwise rotation (transitional lead = V4), and sagging PR and ST segments, which are typical ECG findings for COPD. Additionally, there was coved ST segment elevation >2 mm in V1-V3 terminating in an inverted T wave, indicative of a Brugada Type 1 pattern, and T wave inversion in the inferior leads. Laboratory results, including serial troponin, were normal. The patient denied any history of syncope or SCD in his family. He received antipyretics, IV antibiotics, bronchodilators, and corticosteroids. Following the resolution of fever and improvement in vital signs and respiratory symptoms, the Brugada pattern and T wave inversion on the ECG disappeared.

Conclusion: Febrile patients with BrS are at high risk for VA and SCD. Recognizing the ECG pattern and its triggers are crucial for appropriate treatment. Although the chest pain in this patient was likely due to COPD exacerbation, it is also considered as one of the atypical symptoms of BrS. Brugada pattern typically does not feature T-wave inversion in the inferior leads; therefore, considering the patient's condition, these could have been caused by hyperventilation, hypoxia, respiratory alkalosis, anxiety, or tachycardia, rather than myocardial ischemia.

Keyword: Brugada Syndrome, T Wave Inversion, COPD

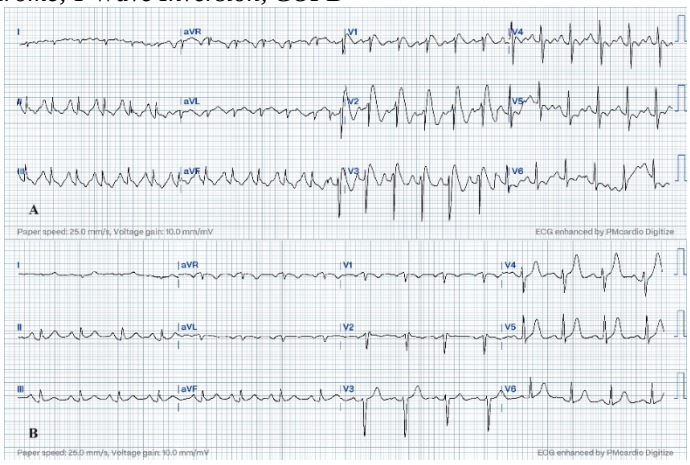

Figure 1. (A) Initial ECG: Brugada Type 1 pattern, right axis deviation (both QRS & P-wave axis), P pulmonale, sagging PR and ST segment, and inferior T wave inversion. (B) Follow-up ECG after resolution of fever and respiratory symptoms

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Wolff Parkinson White Pattern: Can It Limit the Patient's Future?

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Background: Wolff Parkinson White Pattern, is one of the unusual ECG patterns with many chances of sudden cardiac event and might increase the risk of sudden cardiac death, especially in some vigorous or challenging jobs. This study contains three cases of WPW patterns which have been investigated by exercise testing to determine their prognostic

Case Illustration: Three cases of patients with two asymptomatic young men and one woman in middle age with palpitation are all investigated on a treadmill test. In the first two cases, males without any history of familial or symptomatic palpitation, are on general medical check up for job entry. From all three cases we define one patient with a high risk of WPW pattern from a treadmill test, The patient are recommend for an ablation test and some limitation warning for daily activity

Conclusions: Three cases of patients with WPW patterns have been investigated by exercise tests. The result from this test might be significant in characterizing this disease and defining the patient's future job, daily limitations, and quality of life. Advance reference to arrhytmiologist may be considered as the risk calculated from the exercise test

Keywords: WPW pattern, SCD, general medical check-up, police entry requirement

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ECG FEATURES IN HYPERKALEMIA INDUCED HEART FAILURE: A CASE REPORT

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Background: Hyperkalemia is defined as serum potassium level greater than 5.0 mmol/L and leads to electrophysiological changes. Severe hyperkalemia can lead to lethal cardiac dysrhythmias. The earliest ECG changes in hyperkalemia is T-tall. Hyperkalemia induced Acute Decompensated Heart Failure (ADHF) is an infrequent case, but over time the incidence can be large accounting for 26-48 % of hyperkalemia within the first year of heart failure, especially in chronic kidney disease (CKD) patients. Diabetes mellitus with CKD is one of the risk factors of hyperkalemia induced ADHF. The treatment for ADHF induced hyperkalemia can be done by immediately decreasing the potassium level and controlling the fluid overload conservatively by RAAS inhibitor.

Case Illustration: In this case report, we reported a 55-year-old male patient with a history of uncontrolled diabetes mellitus with stage 4 CKD and chronic hypertension. The patient was taken to the emergency department in progressive dyspnea nine hours before hospital admission, with a history of dyspnea for three weeks before. The physical examination findings were rales in lung sound and bilateral lower extremity pitting oedema. ECG showed junctional rhythm and T-tall with laboratory results presenting hyperkalemia (Potassium: 7.8 mmol/L), severely decreased kidney function (eGFR: 24.79 mL/minutes/1.73 m^2), and hyperglycemia (Random blood glucose: 216 mg/dL). ECG of the following days showed the ECG rhythm returned back to sinus rhythm and the transformation of the T wave consequent to decreasing serum potassium level. The return of sinus rhythm appeared first, meanwhile the T-tall had just completely disappeared in three days similarly with the reestablish serum potassium level. The patient was treated for seven days and was discharged with clinical improvement.

Conclusion: One of the reasons for ADHF is hyperkalemia. Hyperkalemia leads to electrophysiological changes such as the peaked T wave in the earliest manifestation. ECG transformation of T waves showed as consequent from decreasing the serum potassium level in this patient.

Keywords: Hyperkalemia, T-tall, Acute Decompensated Heart Failure

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Effectiveness Of The Modified Valsava Manuver For Reversion Of Atrioventricular Nodal Re-Entrant Tachycardia In Emergency Departement: A Case Report

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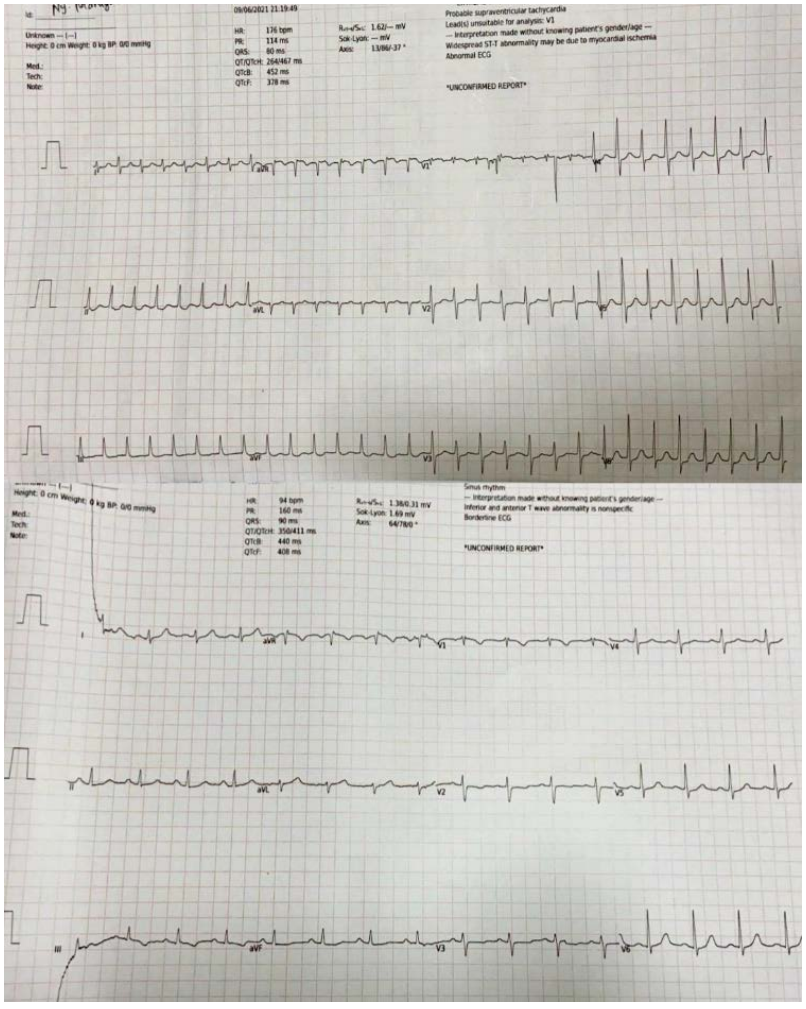
Background: The Modified Valsalva Maneuver (MVM) has never before been performed in the Sultan Suriansyah General Hospital. Currently, we prescribe MVM using a 10 cc syringe as therapy for patients with symptomatic Atrioventricular Nodal Re-Entrant Tachycardia (AVNRT).
Case illustration: A generally healthy, 47-year-old female, presented to the emergency department with sudden onset of palpitations. Vitals at presentation were within normal limits except for pulse rate of 176 bpm. Electrocardiogram showed a regular narrow complex tachycardia with Pseudo r' wave in V1 with a short-RP interval, measured at 110 milliseconds, suggestive of slow fast AVNRT of the typical form. The MVM is a postural technique performed by initially placing the patient in a semirecumbent position. The patient is then encouraged to blow into a manometer to achieve a 40 mmHg intrathoracic pressure for 15 seconds. Once the 40 mmHg intrathoracic pressure is achieved, the patient is repositioned supine, and their legs are raised passively to 45 degrees for 15 seconds. The patient is then returned to the semirecumbent position for 45 seconds before cardiac rhythm reassessment. The passive leg rise will maximize the venous return which will increase the preload resulting in an increase in the cardiac output causing higher vagal tone response. The MVM has shown to have an increased termination rate of AVNRT with no documented serious adverse events. The MVM can be performed in a time-effective manner and is cost effective as intravenous (IV) cannulation is not required.
Conclusion: MVM has shown to have an increased termination rate of AVNRT with no serious adverse events. The MVM is a simple procedure and cost effective. It is recommended that hospital consider the MVM in their clinical pathway for the treatment of new-onset AVNRT.

Keywords: MVM, AVNRT



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Ventricular Arrhythmia in Acute Myocardial Infarction in a Rural Hospital

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Background: Ventricular tachycardia (VT) is one of the life-threatening malignant ventricular arrhythmias. Ventricular tachycardia is one of the causes of death in patients with acute myocardial infarction. Ventricular tachycardia is a rare early finding in acute myocardial infarction and is one of the complications of coronary artery disease. Early revascularization may improve the prognosis in acute myocardial infarction.

Case Illustration: A male 78 years old, came to our emergency department with a chief complaint of palpitations since 2 hours ago. Patient presented with symptom of chest pain since 2 hours ago. The clinical examination showed pulse rate of 115/min with ECG showing presence of VT and the hemodynamic BP 120/80 mmHg, RR 24/ min SpO2 97%. A diagnosis of VT with hemodynamically unstable was made. Synchronized cardioversion was performed on the patient. The Heart rhythm was changed to sinus at a rate of 76 beats per minute. On the ECG, ST elevation was found on leads V2-V6. The patient was given maintenance amiodarone and recommended for primary PCI. Percutaneous coronary intervention (PCI) was performed with stent placement in the intermediate coronary artery due to anterior STEMI. Coronary angigraphy showed intermediate critical stenosis, diffuse critical stenosis in the distal osteal of the right coronary artery, and 70-90% stenosis in the distal left coronary artery. However, 1 month after PCI the patient returned with complaints of chest pain. From the current ECG compared to the previous ECG there is no evolution with persistent ST elevation in v2-v6. Echocardiography showed left ventricular ejection fraction of 37%.

Conclusion: In this case report, we present a case of VT as the initial presentation of STEMI in a previously healthy individual. VT with a pulse accompanied by unstable hemodynamics, the treatment option is electrical cardioversion. In this case report, ischemia is thought to be the cause of the arrhythmia, so early reperfusion is required.

Keyword: Ventricular tachycardia (VT), ST elevation myocardial infraction (STEMI)

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Supraventricular Tachycardia in Pregnancy and Successful Management in the Rural Hospital: A Case Report

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Background: Arrhythmias during pregnancy are extremely concerning for the health of the maternal and fetal. One of the arrhythmias that becomes a complication during pregnancy is Supraventricular Tachycardia (SVT). Management of Supraventricular Tachycardia during pregnancy is challenging. Most antiarrhythmic drugs are classified as category C in the Food and Drug Administration (FDA) labeling system in pregnancy.

Case illustration: A 31-year-old woman in her second trimester (G2P1A0) came to the emergency room in a conscious state with complaints of palpitations, shortness of breath, and cold sweat. The patient is in her second pregnancy with her first pregnancy born alive. On examination, the heart rate was 217 beats per minute. ECG showed supraventricular tachycardia with Atrioventricular Nodal Reentry Tachycardia (AVNRT). Vital sign examination showed blood pressure of 107/92 mmHg, heart rate of 217 bpm, respiratory rate of 28 bpm, and oxygen saturation of 91% on room air. The acral was cold, and besides that, there were no other signs of abnormality. The first management was with vagal maneuver such as carotid massage but failed to correct the pulse. Due to the unavailability of adenosine and beta blocker intravenous in our hospital, intravenous diltiazem 0,25 mg/kgBB was administered in 2 minutes. The heart rate can convert to sinus rhythm. Subsequently, the patient was observed in the Intensive Care Unit. After the patient's condition stabilized, the patient underwent an echocardiography examination showed no structural abnormalities in the heart and ultrasoundnography examination, the fetus was in good condition with a 155 beats per minute fetal heart rate.

Conclusion: Supraventricular tachycardia during pregnancy requires therapeutic management based on hemodynamic conditions and the trimester of pregnancy by considering the safety of the maternal and fetal. Vagal maneuver is the first line of management for stable supraventricular tachycardia. If there is no improvement, antiarrhythmic drugs can be given with consideration of various factors and drug availability in the hospital.

Keywords: Pregnancy, Supraventricular Tachycardia, Antiarrhythmic Drugs, Diltiazem

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Total Atrioventricular Block Resolved After Reperfusion of Right Coronary Artery: a Classical Case

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Background: Arrhythmia is broadly categorized into bradyarrhythmias and tachyarrhythmia based on the heart rate. Bradyarrythmia is a condition when conduction rate of the heart is below 60, which can be caused by either physiological or pathological process. Some pathological process includes atrioventicular blocks and sinus node disorder.

Case Illustration: We present the case of a 58 years old male who was referred to the ER with chief complaint of typical chest pain with 24 hours onset, without history of fever, syncope, seizure, and palpitation. The patient was an active heavy smoker, and have a history of uncontrolled diabetes type 2, and no other known significant medical history. From physical findings, the patient is normotensive without any sign of failure. On ECG finding, we found inferior ST Elevation with total AV block, which doesn't respond to dopamine. Laboratory finding shows mildly elevated leukocyte, normal serum electrolyte, elevated liver enzymes, elevated creatinine, and elevated troponin T. Angiograph was performed, and shows 90-95% stenosis in porximal lcx, 50% stenosis in LAD, and critical stenosis in RCA. PCI was performed to RCA, followed by 48 hrs of heparinization. After reperfusion was performed, the total AV block resolves, and converted to low grade av block with rate of 92x/mins. After 24 hours observation without any arrhythmia and chest pain, the patient was discharged.

Conclusion: In this patient, Total AV Block occurs because inadequate perfusion to the SA node, which is caused by total occlusion of RCA. After reperfusion of RCA, the TAVB converted into 1st degree AV block without any symptoms, which according doesn't need any further intervention.

Keywords: Coronary artery disease, PCI, ECG, arrhytmia, Total AV Block,

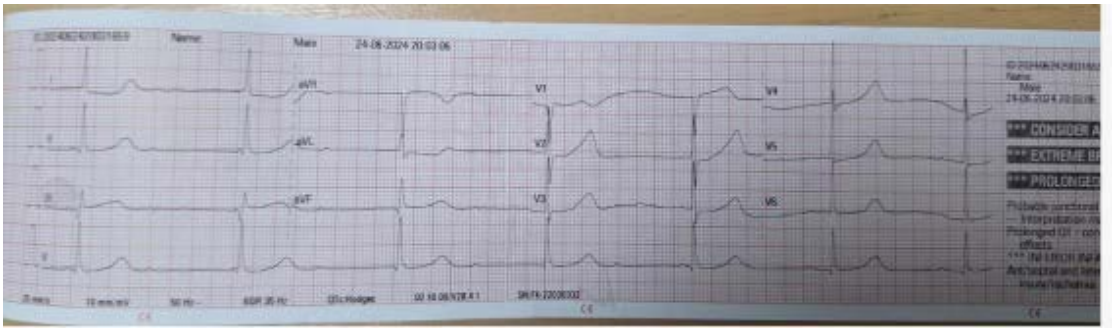

Figure 1. Patient's initial ECG, showing AV dissociation

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"Ventricular Extrasystole Trigeminy in Stroke Patient with Electrolyte Imbalance A Case Report"

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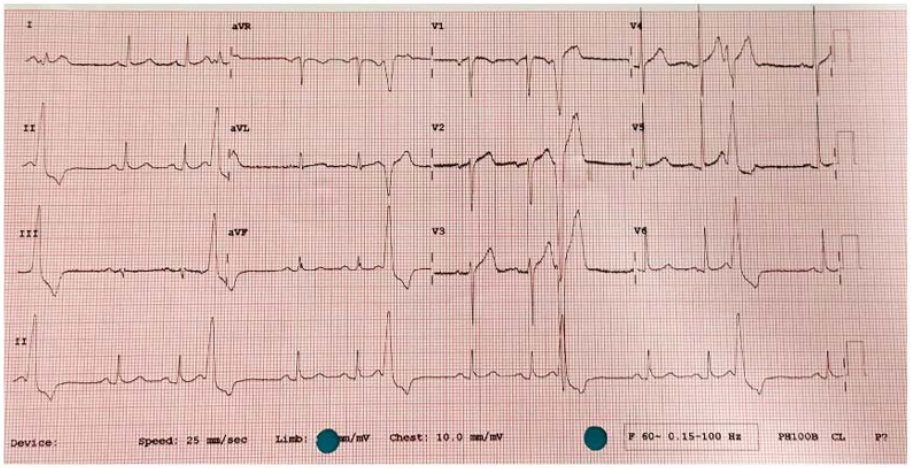
Background : Ventricular trigeminy is a cardiac arrhythmia characterized by alternating normal beats and premature beats. It can have significant implications for patients, especially those with underlying health issues such as stroke. Stroke patients are particularly vulnerable to arrhythmias due to factors like nervous system dysregulation and electrolyte imbalances. Understanding this interplay is crucial for effective management and improved patient outcomes.

Case illustration : A 53-year-old man with uncontrolled hypertension presented with weakness on the right side of his body, followed by the left side, and headaches two weeks prior to admission. He had high blood pressure and an echocardiogram showed diastolic dysfunction with an ejection fraction of 65%. Electrocardiography found Sinus Rhytmn, 85 times per minutes, QTc 427, trigeminal VES. Brain MRI revealed bleeding in the thalamus and right cerebellum, as well as multiple stenoses of the carotid arteries and cerebral arteries. Laboratory results indicated leukocytosis, hyperkalemia, hyponatremia, hypercalcemia, and increased creatinine. During treatment the patient was given intravenous therapy of calcium gluconate, mecobalamin, citicoline, vitamin K, tranexamic acid, ceftriaxone, mannitol. As well as oral therapy candesartan, amlodipine, hydrochlorthiazide, ketoacids, folic acid. The patient spent 8 days in the critical unit and then transferred to a normal room for further treatment.
This case underscores the complex relationship between stroke, electrolyte imbalance, and cardiac arrhythmias. Patients with stroke are at risk of arrhythmias due to disrupted autonomic heart regulation and electrolyte imbalances. Comprehensive cardiac monitoring and a multidisciplinary approach are essential for diagnosis and management, including correcting electrolyte imbalances and continuous cardiac monitoring. Preventive measures such as maintaining optimal electrolyte levels and regular cardiovascular evaluations are crucial for these patients.

Conclusion : This case report highlights the importance of closely monitoring and managing the cardiac health of stroke patients, especially those with electrolyte imbalances. Ventricular extrasystole trigeminy can be risky, requiring a multidisciplinary approach for diagnosis and treatment. Correcting electrolyte imbalances and continuous cardiac evaluation are crucial in reducing arrhythmia risks and improving patient outcomes in stroke management.

Keywords : Ventricular Extrasystole, Trigeminy, Electrolyte Imbalance, Stroke



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Modified Valsava Manuver in Supraventricular tachycardia with hemodynamic stable Patients with a History Cardioversion in Emergency Unit Rural Hospital Is Really Effective?

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Background: Supraventricular arrhythmias are common cardiac arrhythmia encountered in emergency departments, the patient came with complaints of palpitations and several other symptoms. In this condition treatment is required with drugs and electrophysiological procedures. Supraventricular tachycardia (SVT) prevalence is 2.25/1000 persons and the incidence is 35/100.000 person-years. The Valsalva manoeuvre should be the first line of treatment for the management of narrow complex supraventricular tachycardia in the hemodynamically stable patient. Immediate direct-current cardioversion is the first choice in haemodynamically unstable patients with narrow QRS tachycardia.

Case Illustration: A female 59 years old, admitted to the hospital due to recurrent episode of narrow complex tachycardia. Women came with complaints palpitation with dyspnea, there was no chest pain. She has history of supraventricular tachycardia with hemodynamically unstable and received electrical cardioversion therapy 4 times at 1 month ago. At this time the patient presented with stable hemodynamics. The clinical examination showed pulse rate of 191/min with ECG showing presence of SVT and the hemodynamic BP 100/60 mmHg, RR 26/ min SpO2 96%. A diagnosis of hemodynamically stable SVT was made. Carotid Sinus Massage (CSM) was carried out twice. However, his heart rate remained at 185–201 beats per minute. Modified Valsava Maneuver (MVM) was then performed, and his heart rhythm was changed to sinus at a rate of 105 beats per minute. Diltiazem drip is carried out for maintenance during treatment.

Conclusion: In this case report modified valsava maneuver is effective for the treatment of supraventricular tachycardia with hemodynamically stable with history of electrical cardioversion. The modified Valsalva maneuver procedure is carried out by positioning the patient semi-recumbent and blowing into syringes, then raise both legs 45-degree in passively. This can reduce heart rate through activation of the vagus nerve and changing the rhythm to sinus.

Keyword: Supraventricular tachycardia (SVT), Modified Valsava Maneuver (MVM)

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Atrioventricular Block Induced Adam – Stokes Syndrome In A Rural Hospital

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Background: Atrioventricular block is a heart rhythm disorder that causes heart to beat more slowly than it should. It is caused by communication problems within the heart's electrical conduction system. For some people it doesn't cause symptoms, but for others, it may be life-threatening.

Case Illustration: A 63-year-old man arrived at the emergency room complaining of seizures he had experienced a day before being admitted to the hospital. He had seized 13 times at home and 3 times in the emergency room. The seizures lasted for 5 seconds with his eyes looking upwards and his teeth clenched. After the seizure, the patient regained consciousness. His systolic blood pressure was 70mmHg and his pulse was 30 beats per minute. There were no murmurs or gallops sounds. An ECG showed a complete atrioventricular block, and no cardiomegaly was found on the X-ray. After receiving a dopamine infusion, the patient's blood pressure increased to 96/67 mmHg, his pulse rate rose to 68 beats per minute, and the seizures stopped.

Conclusion: Atrioventricular block can be symptomatic like seizure called Adam-Stokes syndrome especially in malignant atrioventricular block.

Keywords : atrioventricular block, Adam-Stokes syndrome

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UNEXPLAINED TYPE OF SHOCK AND ECG CHANGING ON DIABETES MELLITUS AND HEART FAILURE PATIENT IN TYPE D-HOSPITAL: A CASE REPORT

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Background: Diabetes mellitus is a risk factor for heart failure and increases mortality in patients with those diseases. Diabetes mellitus can lead to septic shock due to severe infection and heart failure can lead to cardiogenic shock. Data shows Diabetes Mellitus contribute to heart failure and increases the risk of developing cardiogenic shock by incidence of acute myocardial infarction. Without the evidence of acute myocardial infarction and the presence of sepsis, we must be careful to diagnose the type of shock on Diabetic Patient.

Case Illustration: 60-year-old female patient in inpatient department was admitted because of her hyperglycemic state. Her blood glucose was >500mg/dl. Appropriate therapy was given in emergency department. She has hypertension and been taking bisoprolol and aspirin tablets for five years. Before admitting the patient to the ward, ECG was taken and shows sinus tachycardia.

Her laboratory results were not significant for sepsis, WBC 10.400/mm3, platelet count 204.000/mm3. However, physical examination showed heart failure symptoms including hypertension, shortness of breath and bilateral pulmonary rales on auscultation. The vital signs showed tachycardia, blood pressure 148/112mmHg, tachypnea (39x/m), and desaturation (85% room air). Furosemide was injected. Fifteen minutes later, her vital signs dropped. She looks drowsy and lethargic. Her blood pressure dropped to 80/50mmHg. Her extremities were cold, pulses were weak and no urine output. ECG shows neither sign of acute myocardial infarction nor ischemia pattern (figure 1).

Unfortunately, we didn't check the ketone level and we cannot search the etiology of shock any further. Patient was diagnosed as cardiogenic shock with suspected Diabetic Ketoacidosis and referred to another hospital for more management.

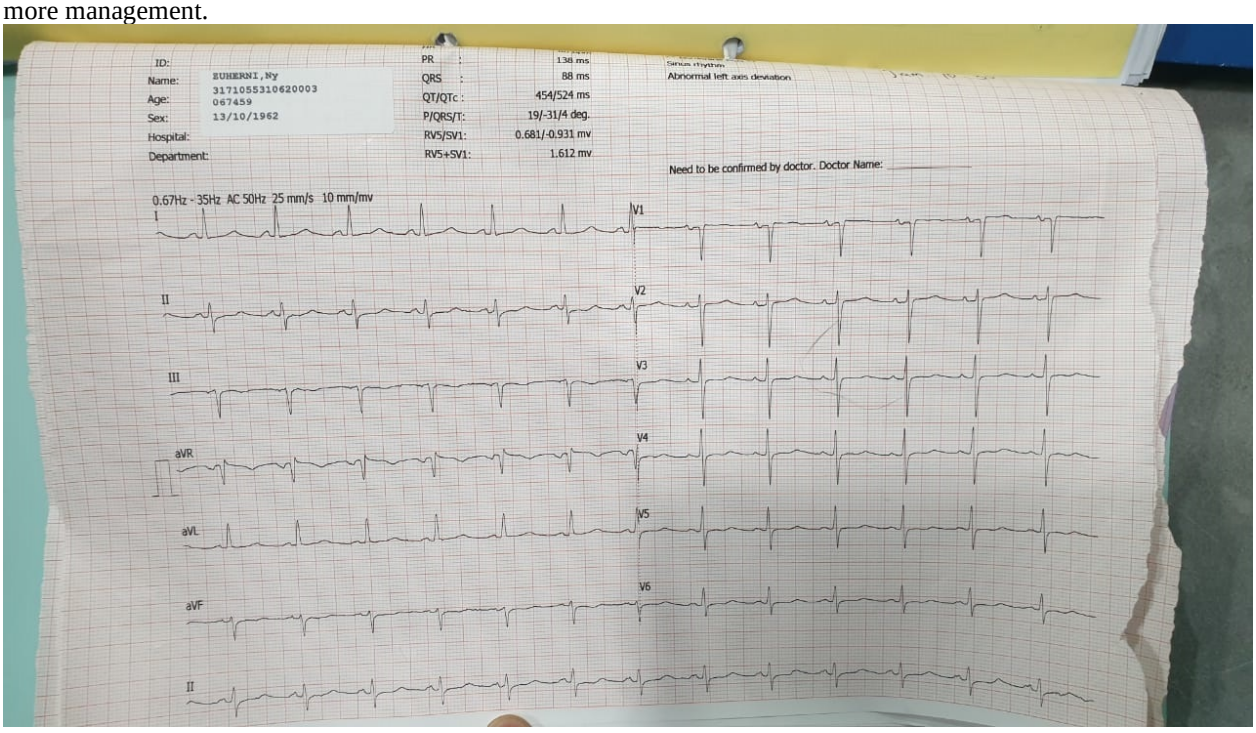

Figure 1: Patient's ECG while having shock state

Conclusion: Septic shock and cardiogenic shock are common in diabetes mellitus. They contribute to higher morbidity and higher mortality in hospital. Physicians should be careful to distinguish those type of shock to improve the patient condition. However, it is quiet challenging to distinguish those form of shock in limited resources hospital. More data and examination should be done for a precise diagnosis.

Key Words: Diabetes Mellitus, cardiogenic Shock, Septic Shock, Sepsis

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SUCCESSFUL PERMANENT PACEMAKER THERAPY IN A PATIENT WITH TOTAL AV BLOCK, ANTEROLATERAL MYOCARDIAL INFARCTION, TYPE II DIABETES MELLITUS, ACUTE RENAL FAILURE, AND HYPOALBUMINEMIA: A CASE REPORT

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Background: Patients with total AV block (TAVB) and anterior myocardial infarction have a 37% higher mortality rate compared to TAVB patients with inferior/posterior infarction. Permanent pacemaker (PPM) is the most effective therapeutic option to manage severe bradyarrhythmias. However, mortality increases in patients with type II diabetes mellitus, heart failure, coronary artery disease, older age, and male gender.

Case Illustration: A 78-year-old male was referred with complaints of a slow heartbeat (44 bpm). The patient had no other complaints. He had type II diabetes mellitus on metformin 1x500 mg, hypertension on amlodipine 1x10 mg, and a history of a heart attack in 2022 but did not follow up. The patient appeared seriously ill, was conscious (Glasgow Coma Scale (GCS) 15), with a blood pressure (BP) of 167/56 mmHg, a heart rate (HR) of 45 beats per minute (bpm), a respiration rate of 20 breaths per minute, a temperature of 36.6°C, an oxygen saturation of 98% on room air, a body weight of 55 kg, and a height of 160 cm, with normal nutritional status. General physical examination was within normal limits. Electrocardiography (ECG) showed TAVB with an accelerated junctional escape rhythm and anterolateral myocardial infarction. Laboratory results showed albumin 2.55 g/dL, LDL 197 mg/dL, fasting glucose 120 mg/dL, 2-hour postprandial glucose 143 mg/dL, urea 42.5 mg/dL, creatinine 1.85 mg/dL, eGFR 32.11 mL/min/1.73 m². The patient was diagnosed with TAVB, anterolateral myocardial infarction, type II diabetes mellitus, acute renal failure dd/ acute on chronic renal failure, and hypoalbuminemia. A temporary pacemaker (TPM) was initially placed, then replaced with a PPM set to a rate of 60-130 bpm. Coronary angiography (CAG) revealed three-vessel disease (CAD 3 VD). After PPM placement, the patient's condition improved, with no complaints, BP 153/80, HR 60 bpm, normal physical examination, ECG showing ventricular pacing, and no cardiac abnormalities on radiology.

Conclusion: This case report demonstrates a good outcome of PPM in a 78-year-old male patient with total AV block, anterolateral myocardial infarction, type II diabetes mellitus, and acute renal failure.

Keywords: TAVB, CAD, PPM

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Challenges In Management of ST Elevation Myocardial Infarction in Rural Hospital. Can It Be Resolved?

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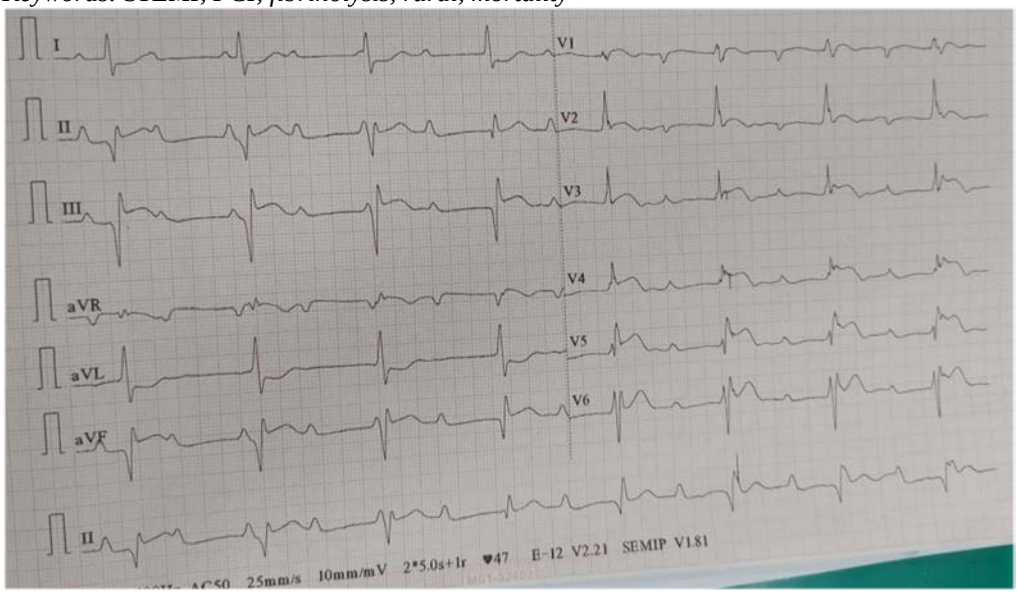
Background: Rapid reperfusion therapy is the primary treatment for acute ST-Elevation Myocardial Infarction (STEMI). Rural area patients with STEMI may be less likely to receive reperfusion because cardiac catheterization laboratories are far and too few to serve the large numbers of patients with STEMI.

Case Illustration: A 56 years old male came to emergency unit with chest pain that radiated to the neck and felt four hours before admission with more than 30 minutes duration. He was a smoker and had previous history of hypertension. On examination normotensive 105/70 mmHg, bradycardic heart rate 48 beats per minutes, tachypnea 26 breath per minutes, oxygen saturation 98%. The initial ECG showed inferior STEMI with third-degree atrioventricular (AV) block. The patient could not be tested for troponin due to limited facilities.
The patient was given 320 mg aspirin, 300 mg clopidogrel, and 40 mg atorvastatin and atropine sulfate 0.5 mg repeated with a total dose of 3 mg, but there was no improvement in the patient's heart rate. In our hospital at that time there was no epinephrine and dopamine. It would be ideal for patients to undergo primary PCI. But sadly, in West Pasaman Regency there was no hospital with PCI or fibrinolytic.
Coronary reperfusion therapy either in the form of PCI or fibrinolysis should be administered as quickly as possible in all eligible acute STEMI patients. Fibrinolysis should be recommended to patients with acute STEMI presenting within 12 hours of onset of symptoms when primary PCI cannot be delivered within 120 minutes of presentation. But, the big problem is management of STEMI patients can't be successfully implemented in many rural district.

Conclusion: STEMI is a life-threatening condition. In this patient there was indications for PCI, but due to limitations the patient ultimately could not be treated immediately. This condition often encountered in the rural area which is associated with the increasing of mortality because every minute delay in reperfusion is associated with increased mortality.

Keywords: STEMI, PCI, fibrinolysis, rural, mortality



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Antiplatelet and Anticoagulant Strategies in Chronic Kidney Disease Patients with Acute Coronary Syndrome at Non-PCI Centre Hospitals

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Background: Patients with CKD are at a higher risk of ACS, significantly higher mortality, and increased bleeding problems. Multiple registries also reveal that medical therapy alone does not enhance the outcome of ACS patients with severe CKD. Antiplatelet and antithrombotic drugs are the cornerstone of ACS management but should be used with caution in patients with CKD.

Case Illustration: A 69-year-old man with CKD reported left chest pain for three weeks, which was exacerbated within six hours before admission. Vital signs and cardiac examination were normal. A 12-lead ECG revealed a sinus rhythm of 85 BPM, normoaxis, and ST depression leads I, V4, V5, and V6, with pathological Q waves in leads III and aVF. Echocardiography revealed mild mitral and aortic regurgitation, with reduced ejection fraction (44.68%). Renal ultrasonography also detected contractions in the left kidney. The patient was given ISDN 5mg, Aspilet 80mg, Clopidogrel 300mg, Carvedilol 6.25mg, and Atorvastatin 40mg orally, as well as Furosemide 20mg and Heparin 4000IU intravenously followed by 750 IU/hour during 48 hours. Then, the patient was observed in the ICU for three days. After being stabilized, the patient got hemodialysis and was discharged after three days on the ward.

Conclusion: According to the recent guidelines, there is no different medication and dose selection of antiplatelet and anticoagulant between CKD stage V and normal renal function. In this case, the selected management strategy involved categorizing patients based on the degree of CKD. However, there is insufficient evidence regarding the administration of clopidogrel and high-intensity statin in CKD stage V.

Keywords: Acute Coronary Syndrome, Anticoagulant, Antiplatelet, Chronic Kidney Disease

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Modified Valsalva Maneuver for Cardioversion of Supraventricular Tachycardia in Rural Settings: a Safe, Non-Invasive, and Cost-Effective Early Treatment

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Background: Supraventricular tachycardia is a tachyarrhythmia and is accompanied by palpitations, shortness of breath, or even without symptoms. The Valsalva maneuver is an internationally recommended first-line emergency treatment for supraventricular tachycardia (SVT). Despite empirical data demonstrating that the conventional method is less effective (5%-20%) than the Modified Valsalva maneuver for the first therapy of stable SVT, some physicians continue to use it. However, a study by Wang et al revealed a higher success rate of return to sinus rhythm with the modified Valsalva maneuver compared to the conventional procedure (47.78% vs 15.38%).

Case illustration: A 54-year-old female and a 59-year-old male arrived at the ED with complaints of sudden palpitations. No previous vigorous activity had triggered the beginning. Both patients' vital signs only revealed tachycardia without hemodynamic instability. Both patients' ECGs showed supraventricular tachycardia. A modified Valsalva maneuver was performed. The patient is in a semi-recumbent position, attached to a cardiac monitor. Then, both patients were told to vigorously exhale for 15 seconds while blowing into a 10 mL syringe to move the plunger. Following straining, the patient had to lie down with their legs lifted at 45 degrees for 15 seconds. Then, both patients were placed in a semi-recumbent position for reassessment. After 45-60 seconds of observation, both patients' rhythms returned to sinus.

Conclusion: The modified Valsalva maneuver was proven to have good cardioversion rates in the emergency management of SVT, especially in rural areas. It’s more effective, safer, and less costly than conventional procedures. The procedure may also be performed in primary healthcare settings and by the patients themselves.

Keywords: Modified Valsalva Maneuver; Palpitations; Supraventricular Tachycardia

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Early Repolarization Pattern a Case Report: Higher Risk for Ventricular Arrhythmias?

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Background: We describe the case of a 35 years old male that come to EMD with chief complaint chest discomfort and early repolarization pattern on ECG.

Case Illustration: A 35 years old male came to EMD with chest discomfort for at least 1 hour. There was no history of any other disease. He never had experienced the same complaint before. He looked ill, His blood pressure was 120/70, pulse rate 59 beats/min, oxygen saturation 98%. Physical examination show no abnormality. Series laboratory test shows normal cardiac enzyme, First high sensitive cardiac troponin test (hs-cTnT) 10,57 ng/L, second hs-cTnT 13,01 ng/L. Other laboratory test shows no abnormality. Echocardiography shows Normal systolic function with EF 59% with global normokinetic, Normal RV contractility (TAPSE 23mm). The chest x ray shows no abnormality. The ECG shows QRS notching at V5-V6 (*Fish hook sign*) with slightly horizontal ST segment elevation. Patient then admitted for hospitalization to be evaluated related to chest discomfort and Holter monitoring. At hospitalization patient was treated with bisoprolol 5mg once a day and Nitrate IV 5mcg/min At first day hospitalization patient had complaint palpitation and chest discomfort for several times. From holter monitoring it showed period of PVC bigeminy and multiple PAC (Triplet PAC). Patient chest discomfort starting to subside after 2 days hospitalization. Patient then recharged home with Nitrate 5mg three times daily and bisoprolol 5mg once daily.

Conclusion: Early repolarization pattern in the inferior and/or lateral leads of the standard 12-lead ECG with an increased risk for life threatening arrhythmias.

Keyword: Early repolarization pattern, Arrhythmias, Sudden cardiac death.

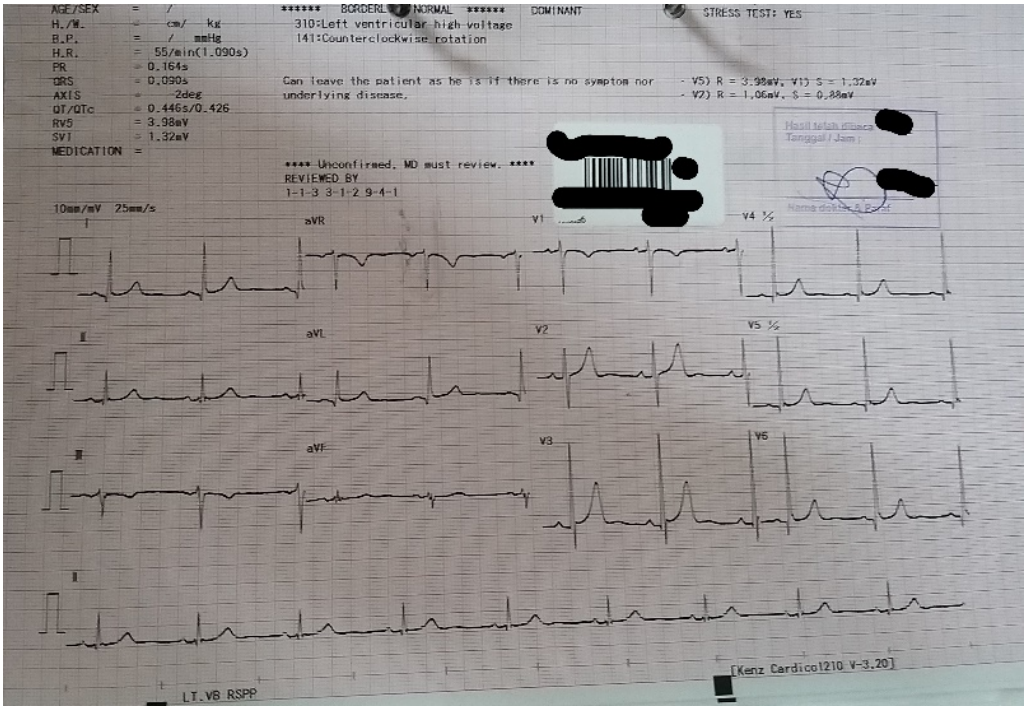

Fig 1. Patient standart 12-lead ECG showed early repolarization pattern

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Antiarrhythmic Effect of SGLT-2 Inhibitors in High-Degree AV Block Caused by Heart Failure with Mid-Range Ejection Fraction: A Case Report in Rural Setting

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Background: According to WHO's Top 10 Global Health Threats, non-communicable diseases such as heart failure contribute to more than 70% of all deaths worldwide. Several studies have shown that SGLT-2 inhibitors can considerably reduce heart failure. As research progresses, SGLT-2 inhibitors may lessen the risk of arrhythmias in heart failure patients.

Case Illustration: A 57-year-old female with uncontrolled hypertension came to the ED after collapsing at home. The vital signs and physical examination are normal. The preliminary ECG showed a high degree AV Block with multiple multifocal PVC. Early laboratory revealed mild hypokalemia. The patient experienced a seizure the next day, and an ECG showed total AV block with a non-sustained VT episode with PVC R on T. Following critical cardiac care and stabilization, the patient underwent echocardiography, which showed mild MR and AR with a reduced ejection fraction (47.7%). Once the patient's condition has stabilized, the patient is given 1x10mg Empagliflozin PO for 4 days in the ward. Following the treatment, the patient no longer complained of breathlessness, no seizures, and felt better. The latest ECG results revealed sinus rhythm with 50 BPM. The patient's condition improved, and she was discharged.

Conclusion: As research progresses, it has been discovered that SGLT-2 inhibitors may significantly reduce the risk of arrhythmia and heart failure by inhibiting sodium-hydrogen exchange in myocardial cells and the sympathetic nerve system.

Keywords: heart failure, high-degree AV block, sodium-glucose co-transporter-2 inhibitors, syncope

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Left Ventricular Thrombus Formation in an Untreated Acute Myocardial Infarction Patient with Uncontrolled Diabetes: A Case Report

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Background: Myocardial infarction is one of the main health problems in Indonesia and the world because of its high mortality and morbidity rates. According to WHO, the prevalence reaches 17.3 million sufferers every year. Left ventricular thrombus is the most common complication of acute and chronic myocardial infarction. Thrombus in the left ventricle is found in 7-46% of cases, especially in anterior or apical myocardial infarction. The patient's diabetes mellitus condition causes prolonged stress injury which will cause endothelial dysfunction. Ventricular akinesia and dyskinesia due to myocardial infarction causing blood stasis and hypercoagulability conditions may also occur in myocardial infarction sufferers through increased concentrations of prothrombin, von Willebrand factor, and decreased concentrations of the ADAMTS enzyme. Virchow's triad causes left ventricular thrombus formation.

Case Illustration: A 68 year old woman came with complaints of worsening shortness of breath since a day. history of uncontrolled diabetes mellitus and stroke, infarction known 1 month before admission to hospital. From electrocardiography examination, ST elevation was found in leads II, III, IV. Transthoracic echocardiography examination showed a thrombus at the left ventricular apex. Chest x-ray examination showed cardiomegaly with a CTR >55%.

Conclusion: Untreated acute myocardial infarction has a high risk of left ventricular thrombus.