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Case Report

Acute Anterior Reinfarction Complicating with Transient Symptomatic Total Atrioventricular Block

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Abstract

Background: Total atrioventricular block (TAVB) is frequently become the conductive disturbance complication of acute reinfarction. Inferior MI has low long-term mortality and greater reversibility than anterior MI which has higher in-hospital and long-term mortality.

Case Illustration: 44 old years man underwent PPCI stenting at proximal LAD of his acute anterior MI 2 days before presentation at the previous hospital. Patient had acute stent thrombosis then underwent urgent PCI and developed TAVB with idioventricular escape. In emergency department Cardiovascular Centre Harapan Kita, patient develop loss of consciousness with blood pressure 57/30 mmHg, heart rate 30 bpm and TAVB rhythm. Laboratory showed increased serum lactate level 5.2. Patient was diagnosed with Total AV block caused by anterior MI. Patient was planned for emergency temporary pacemaker (TPM) implantation. After 24 hours close monitoring, the patient intrinsic rhythm resolved with spontaneous recovery. Patient was hemodynamically stable until discharge.

Discussion: Stent thrombosis of proximal stent of LAD will cause TAVB because of the source of the distal portion of the AV node is originating from septal branch of LAD. Cardiogenic shock could be a manifestation of TAVB with idioventricular escape. It is caused by extensive necrosis with higher in-hospital and long-term mortality, often culminated in permanent pacemaker. However, spontaneous recovery of TAVB into sinus rhythm take place. This could be caused by transient reversible ischemia of infra nodal region of AV node which supplied by septal perforator branch in anterior infarction.

Conclusion: Stent thrombosis of proximal stent of LAD will cause TAVB because of the source of the distal portion of the AV node is originating from septal branch of LAD. Cardiogenic shock could be a manifestation of TAVB with idioventricular escape. It is caused by extensive necrosis with higher in-hospital and long-term mortality, often culminated in permanent pacemaker. However, spontaneous recovery of TAVB into sinus rhythm take place. This could be caused by transient reversible ischemia of infra nodal region of AV node which supplied by septal perforator branch in anterior infarction

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Keywords: Reinfarct, Stent Thrombosis, Total AV Block.

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Introduction

Acute stent thrombosis (ST) is a frequent cause of myocardial infarct (MI) after stent placement. Stent thrombosis is an uncommon but life-threatening complication after percutaneous coronary intervention (PCI), frequently manifesting as acute coronary syndrome (ACS) or even cardiac death.[1] Total atrioventricular (AV) block is frequently become the conductive disturbance complication of acute reinfarction. This complication could develop from either inferior MI or anterior MI. There is differing mechanisms leading to TAVB between patients who developed MI at different anatomic locations. Two thirds of patients (66 %) with TAVB complicating an anterior MI died during hospitalization compared to only 37% of patients with an inferior/ posterior MI and TAVB.[2,3] Total AV block complicating the anterior MI has high mortality and poorer outcome, generally propagated by extensive infarction involving the conduction system distal to the AV node,[4,5] lead to extensive acute left ventricular damage and hemodynamic instability.[7,8,9]

Case illustration

A 44 years old man, in the previous referrer hospital, patient had history of anterior extensive STEMI at 2 days before admission and underwent primary percutaneous coronary intervention (PPCI) with stent implantation DES at proximal LAD. No residual stenosis was seen. In the following day after PPCI, patient had clinical symptom of ischemic chest pain, ST elevation > 1 mm, and increased cardiac troponin. In the previous hospital, patient was diagnosed with acute anterior reinfarction and immediate catheterization evaluation. The coronary angiography found total occlusion thrombosis proximal stent LAD with no distal flow and thrombus was visualized at proximal stent location. Stent was deployed in previous stent (intra-stent) with TIMI 3 Flow. Patient has risk factor for coronary artery disease: obese, diabetes mellitus, and smoker.

Several hours after second PCI, patient develop altered mental status, with seizure episode and TAVB rhythm on vital sign monitor. The blood pressure was 50/30 mmHg, rate 30 bpm. Patient was stabilized with transcutaneous pacing at the prior hospital and referred to NCCHK. Medication at prior hospital was ASA 80 mg o.d, ticagrelor 90 mg b.d, rosuvastatin 20 mg o.d, and fondaparinux 2.5 mg SC o.d.

Patient came to emergency department National Cardiovascular Centre Harapan Kita (NCCHK) with chief complaint altered mental status 12 hours before admission. This was accompanied by several episode of seizure (not more than 20 seconds). patient present with blood pressure 57/30, heart rate 20 -30 with TAVB rhythm on monitor. Patient was alert only with pain stimuli. Laboratory finding showed increased serum lactate level 5.2. Hb 15.4 / Ht 45 / leucocyte 20.500 / creatinine 2.42 / GFR 29. Patient was diagnosed with total AV block with wide QRS morphology ec acute anterior reinfarction, cardiogenic shock, DM hyperglycemia, pre-renal acute kidney injury, metabolic acidosis.

Patient performed bedside echocardiography examination with result: end-diastolic dimension 55 mm, end-systolic dimension 44 mm, normal valves, LVEF 33 % (Simpson), TAPSE 12 mm, regional wall motion abnormality akinetic (RWMA) at anterior segment, LV thrombus appear at LV apex. Patient then planned for emergency temporary transvenous pacemaker (TPM) implantation and procedure was successful with setting rate 80, threshold 0.5, output 2 mA. In addition to pacing therapy, patient was given with i.v fluid challenge and dopamine infusion 10 mcg/kg/min while waiting for TPM placement. After TPM placement, patient was observed at Intensive Cardiovascular Care Unit (ICVCU).

Observation at Intensive Cardiovascular Care Unit (ICVCU) at second day of admission showed recovery of the patient's intrinsic rhythm. Patient was also heparinized with unfractionated heparin (UFH) due to renal insufficiency. In the following day, patient has no symptom, with normal blood pressure and heart rate. Patient's ECG showed sinus rhythm with rate 110 bpm. The TPM was switched into standby off mode and close observation of patient rhythm was done. After 24 hours of standby off TPM, there was no re-appearance of TAVB rhythm on patient monitor, and then the TPM was removed on the third day of admission. On the 4th day of admission, there was no chest pain and dyspnea, with stable hemodynamic, then patient planned for transferred to general ward, then two days later patient was discharged from general ward

with stable hemodynamic and sinus rhythm on 12-lead ECG. During patient hospitalization, the medication was ASA 80 mg o.d, ticagrelor 90 mg b.d, simvastatin 20 mg o.d, candesartan 8 mg o.d.

Discussion

Acute stent thrombosis is a frequent cause of acute reinfarction after stent placement and extensive myocardial necrosis could occur due to proximal occlusion of large coronary territory. Stent thrombosis (ST) is an uncommon but life-threatening complication after percutaneous coronary intervention (PCI), frequently manifesting as acute coronary syndrome (ACS) or even cardiac death.[1] Traditional classification categorizes this complication into early (including acute and subacute ST, within 24 h and from 24 h to 30 days, respectively), late (from 30 days to 1 year), and very late (after 1 year). The patient in this report develops acute stent thrombosis because of the immediate event of acute reinfarction after PCI less than 24 hours later.

The broad spectrum of risk factor categories is related to the patient (include clinical presentation), lesion, stent, and antiplatelet therapy. Among them, premature cessation of dual antiplatelet therapy (DAPT) seems to be the strongest single risk factor for ST.[1] Multivariable predictors of subacute ST included insulin-treated diabetes mellitus, history of congestive heart failure, baseline platelet count, baseline and final TIMI 0/1 flow, and non-use of a loading dose of clopidogrel (600 mg). Patient in this report develop stent thrombosis on hospital admission after PCI, hence premature cessation of antiplatelet therapy is less likely become the risk. For other risk factor, patient had history of diabetes mellitus.

Occurrence of irreversible or delayed TAVB following occlusion of first septal perforator (SPB) after PCI of LAD is an extremely rare complication. Various mechanisms have been proposed, including plaque shift, the snow-plow effect, and stent jailing of the side branch.[4] First septal perforator supplies the superior and anterior portion of the interventricular septum. Conversely, the right bundle branch is supplied by SPB from LAD. The left anterior fascicle is also supplied by SPB from LAD. The left posterior fascicle has a dual supply by RCA and SPB from LAD; its occlusion following PCI of LAD can cause infarction, conduction disturbances arising to symptoms of angina, arrhythmia, and heart failure.[8,9] Development of new onset bifascicular block (RBBB associated with LAFB) in this patient following first PCI was a warning sign that the blood supply of the conduction system compromised. These kinds of patients are one step away from TAVB.[3] Early sign of septal perforator branch occlusion/ "jailed" after PCI LAD will showed complete RBBB with left anterior fascicular block (LAFB), this is typical sign that develops into a delayed TAVB. In this patient report, ECG after acute stent thrombosis showed the same morphology.

TAVB is an important clinical complication in patients hospitalized with acute myocardial infarction (AMI).[2,7,8,10] Overall results are consistent with these findings, suggesting that the odds of dying in hospital for patients who develop TAVB is approximately 3 times higher than for those who did not develop TAVB.[7,10] Two thirds of patients with TAVB complicating an anterior MI died during hospitalization compared to only 37% of patients with an inferior/posterior MI and TAVB.[8] Spencer, et al. reported 1 of 3 patient with TAVB complicating by anterior MI survived to hospital discharge.

Inferior MI prone to develop TAVB 2-4 times greater than anterior MI. Gang et al said, the incidence of complete heart block from STEMI with RCA culprit lesion is 7 %, in the other hand, the incidence of TAVB in anterior STEMI is 1%.[6,10] Likewise, Rosa et al reported the similar result, inferior STEMI being the most frequent diagnosis in TAVB group (79.1%).[8] Development of TAVB in STEMI patient have several predictors. These predictors are RCA culprit lesion (OR 5.95), age > 65 years (2.45), female (1.92), hypertension (1.77), and diabetes mellitus (2.15).[6,10] When patient develop TAVB in MI, it has greater 30 day-mortality than patient does not develop TAVB, otherwise after 30 day, the mortality is similar in TAVB and non-TAVB group. MI patient who developed TAVB has greater mortality with hazard ratio 4.06 compared with non-TAVB group. Cardiogenic shock was about seven times more frequent in TAVB patient (33.0 vs. 4.5%).[6,8] The only clinical predictor for TAVB which present in this case is diabetes mellitus. The patient in this report also develops symptomatic bradycardia and cardiogenic shock due to TAVB.

There is differing mechanisms leading to TAVB between patients who developed AMI at different anatomic locations. In patients with an anterior MI,

TAVB is generally propagated by extensive infarction involving the conduction system distal to the AV node. While heart block may contribute to increased mortality in this setting, much of the poorer outcomes observed in these patients is primarily related to the extent of acute left ventricular damage. Transient Total AV block in anterior MI can caused by occlusion of first septal perforator branch. Reversible ischemia is most reasonable explanation of transient TAVB in anterior wall MI. In patients with an inferior MI, TAVB is generally caused by compromised supply to the AV node – in many patients, a stable escape rhythm is established and pacing is not always required. The patient presentation at NCCHK Emergency Ward showed hemodynamic instability and wide QRS morphology.

This patient presentation of cardiogenic shock with cerebral hypoperfusion was believed mainly induced by extreme bradycardia caused by TAVB. Hence, in this clinical setting we tried bridging medication for increase the heart rate (HR). The pharmacological approach by dopamine was not successful to increase HR, then transcutaneous pacing (TCP) approach taken

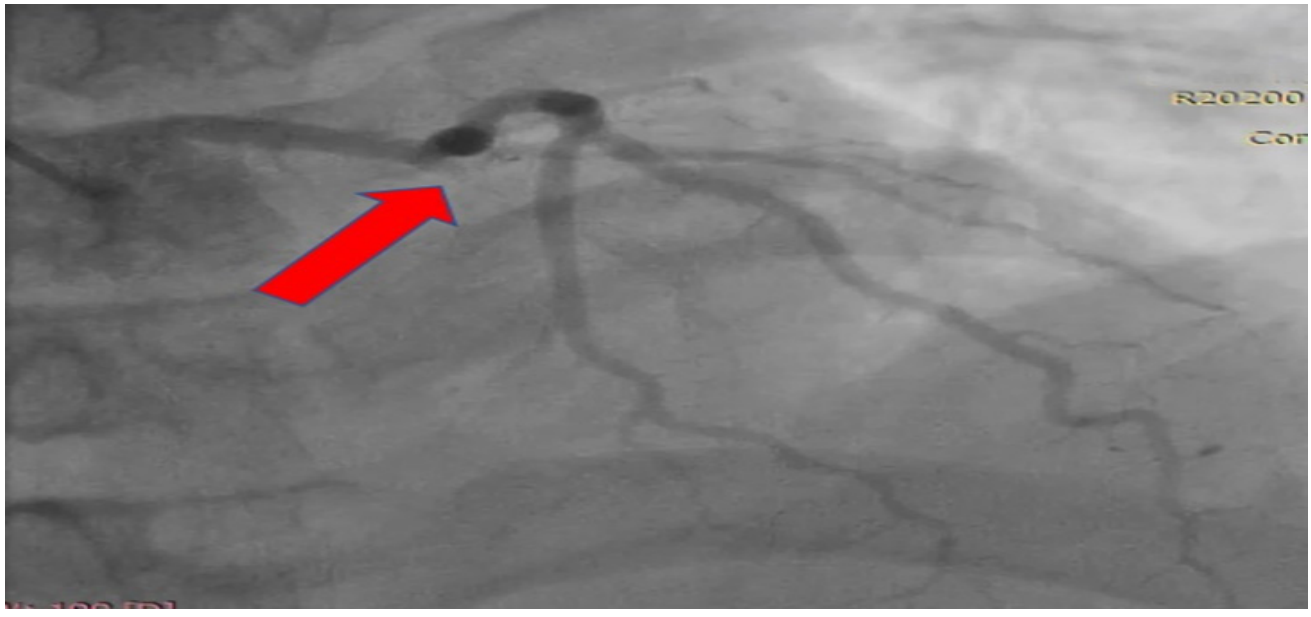


Figure 1. Acute Total Occlusion of Proximal LAD Stent after Primary Percutaneous Coronary Intervention.

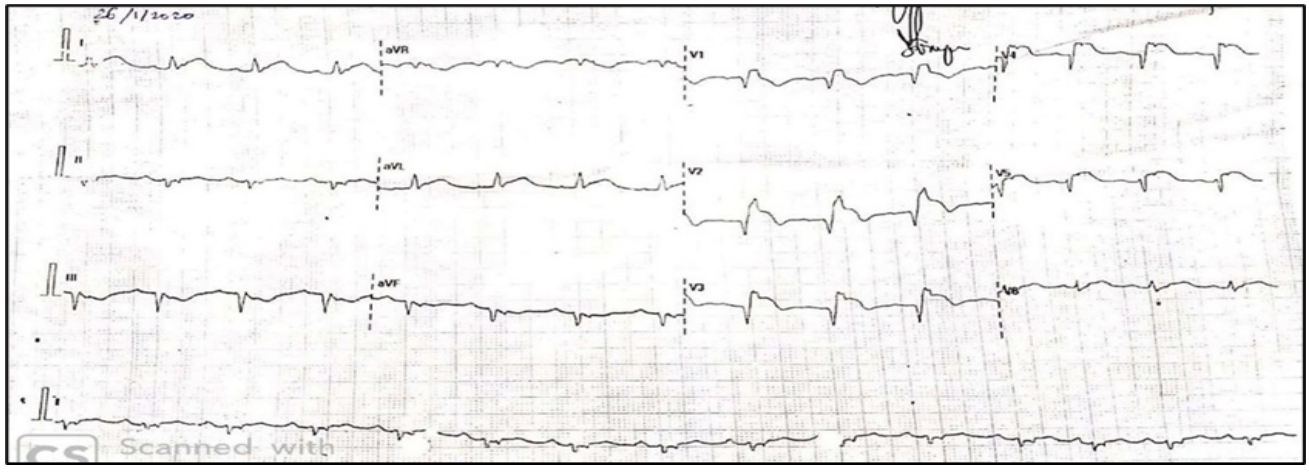


Figure 2. Patient Electrocardiography (ECG) at Onset of Acute Reinfarction After PCI at Referrer Hospital.

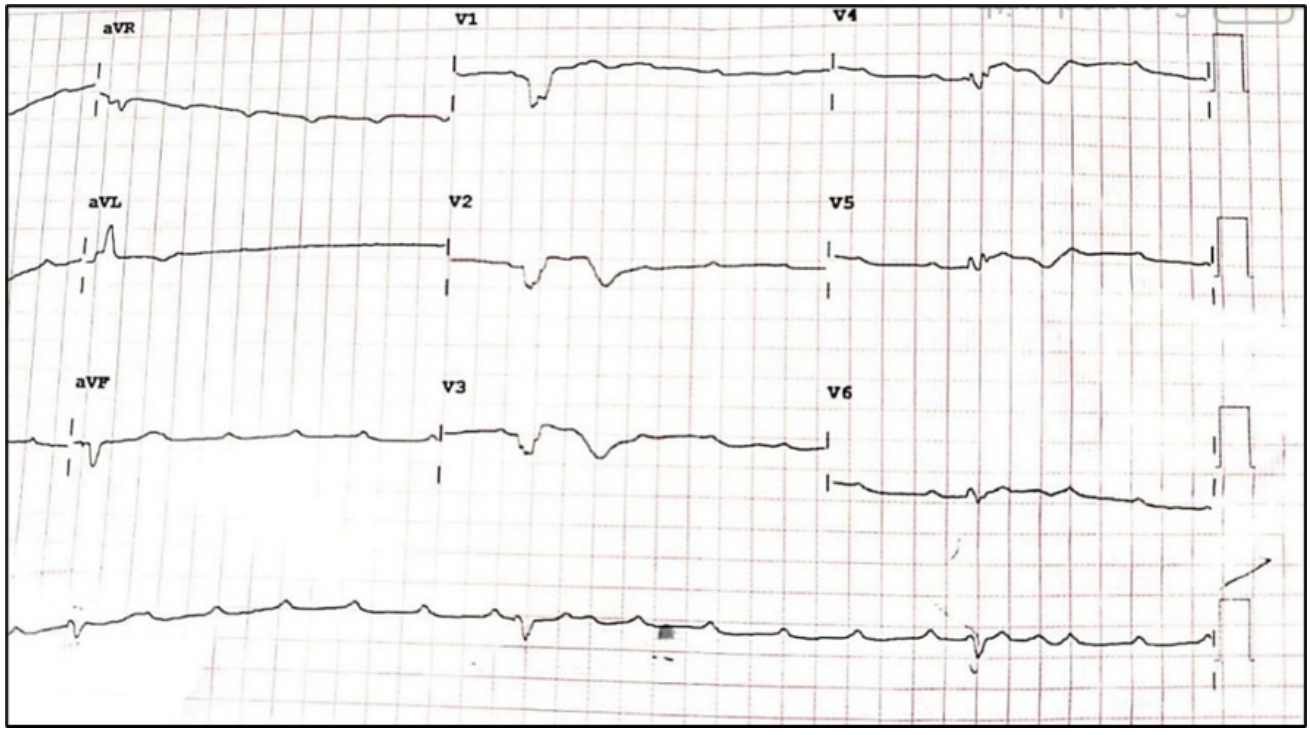


Figure 3. Patient Electrocardiography (ECG) at Presentation in Emergency Ward National Cardiovascular Centre Harapan Kita. The ECG showed Total AV Block with Wide Complex Escape Rhythm.

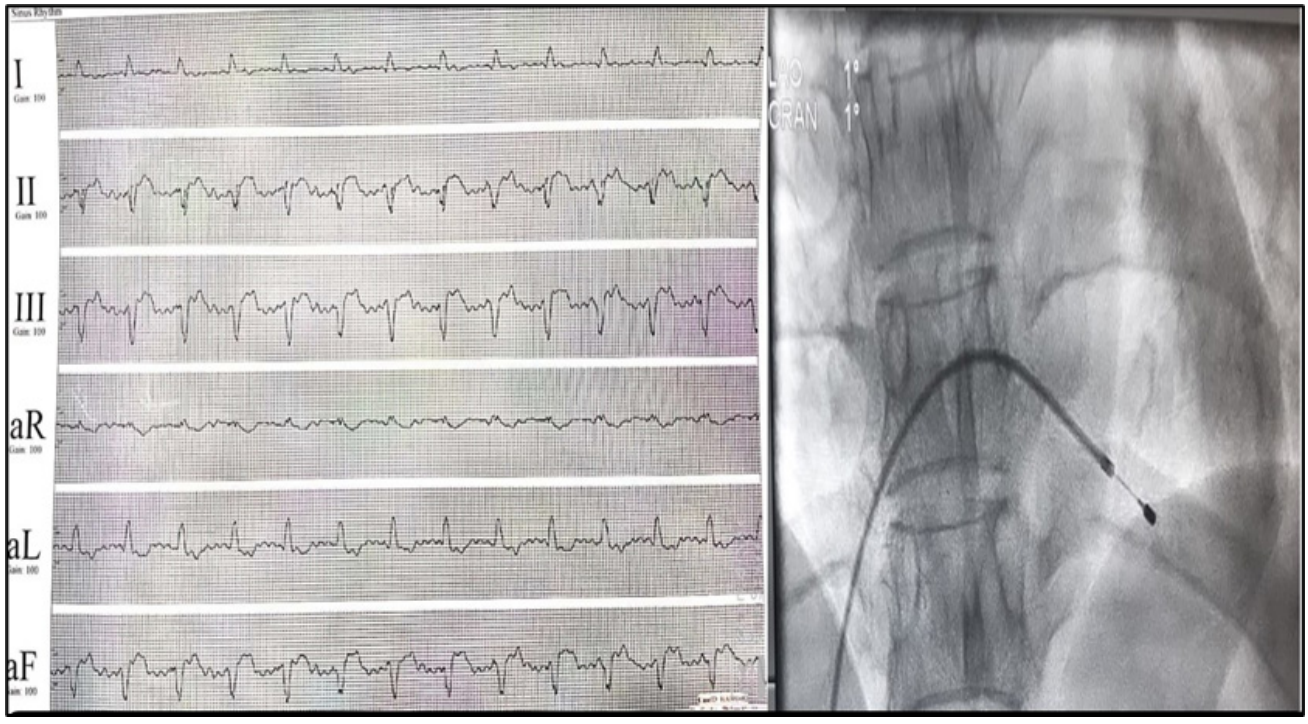
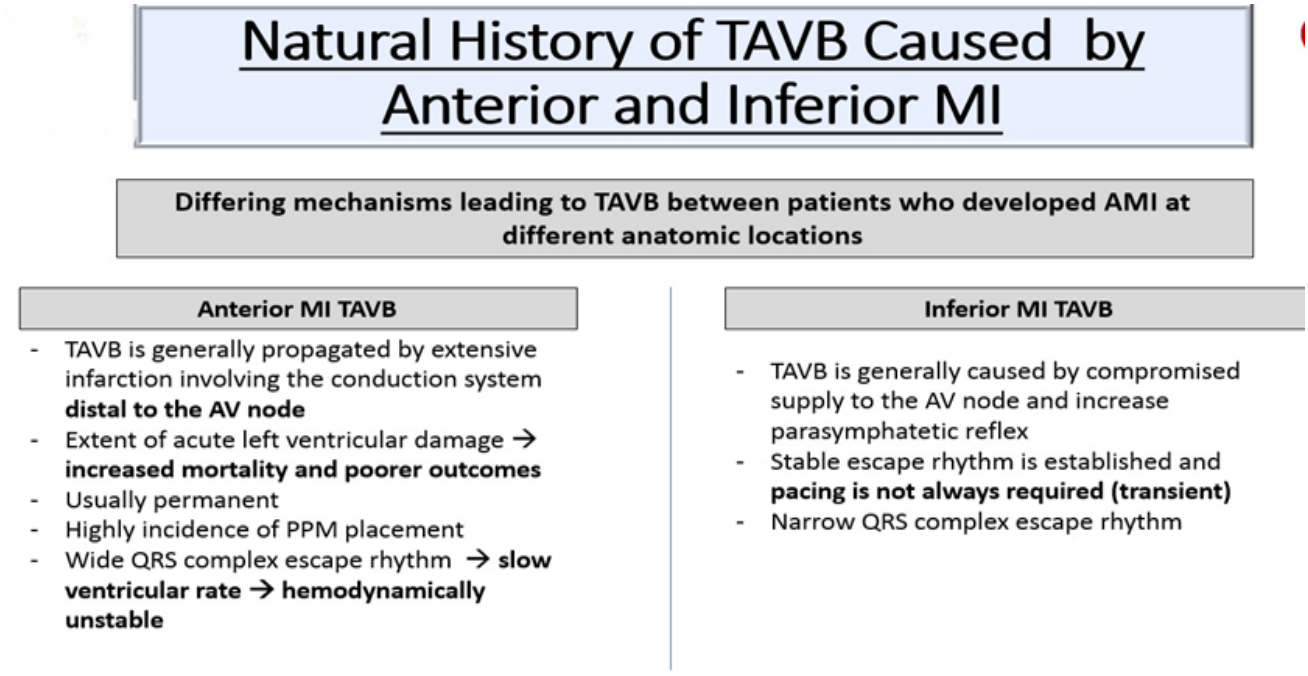


Figure 4. Transvenous Temporary Pacemaker was Successfully Implanted with Setting rate 80, threshold 0.5, output 2 mA. ECG Pacing Rhythm.



for bridging therapy to TPM placement.

Aplin et al reported similar result. Incidence of TAVB in inferior MI is 9.4 %, compared with anterior MI 2.5 %, whereas from all patient with TAVB, 9 % required permanent pacemaker placement prior to discharge.[7] Whereas Rosa et al reported 8.8 % patient who developed TAVB underwent definitive pacemaker implantation.[8] Spencer, et al. reported that TAVB developed in 3.3% of patients with anterior AMI and 6.3% of patients with inferior AMI. In 35 % TAVB patient underwent TPM and 5.9 % had PPM placement. This case presents an unusual course of TAVB caused by anterior wall MI, where the patient had spontaneous recovery of sinus rhythm after 2 days of acute anterior reinfarction event and TPM placement.

Rosa et al reported, the main symptom of TAVB was syncope in 13.6% compared with 1.5% in non-TAVB group. Clinical prognosis is worse when an MI result in RBBB with LAFB due to large amount of myocardium involved. Electrocardiography which showed Complete RBBB with LAFB, this might be a typical sign that develops into a delayed TAVB. Notably, five of the eight published cases required permanent pacemaker implantation.[4]

There are theoretical mechanisms of AV block reversibility in coronary artery disease. Firstly, vagal hypothesis which caused activation of non-myelinated afferent C-fibers from the vagus nerve, which in turn result in increased vagal tone and bradyarrhythmia. This mechanism is known as the Bezold-Jarisch reflex. Secondly, Ischemia hypothesis where the AV node blood supply is provided by the AV nodal branch, which most commonly arises from the RCA. Infranodal conduction system structures are supplied almost entirely by the septal perforator branches of the LAD artery. Decreased flow to the septal branches or RCA is therefore associated with a variety of conduction disturbances.[13]

Conclusion

It has been presented case of a 44 years old man who developed Total AV block during the course of acute anterior STEMI, and had spontaneous resolution where it was only happen in 1/3 patient population from previous study. Mechanisms of spontaneous resolution of total AV block in the setting of acute anterior MI is associated with loss of collateral circulation which supplied AV node. Attention is drawn to a subgroup of patients, albeit a minority, who have a better prognosis owing to reversible causes than classically expected and seen.

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