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Case Report

Radiofrequency Ablation of Focal Atrial Tachycardia from Coronary Sinus

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Focal atrial tachycardia (AT) is defined as atrial activation originating from a discrete focus with centrifugal spread. Available information suggests that focal activity can be caused by automaticity, triggered activity, or microreentry. Generally, AT response poorly to medication but can be treated by radiofrequency ablation with high long-term success. Focal AT represents approximately 3% to 17% of the patients referred for supraventricular tachycardia (SVT) radiofrequency ablation (RFA). Electrophysiology study is important to correctly diagnose the mechanism of the SVT before RFA is performed. Observation and several pacing maneuver could be done to identify the mechanism of SVT.

A 54 year old female came with chief complaint of palpitation. During palpitation her ECG showed narrow complex regular tachycardia with the P-wave that was difficult to ascertain clearly. Electrophysiology study showed VA interval 130 ms, differences between VA interval during tachycardia and VA interval during RV pacing was 55 ms, no advanced in atrial activation, difference between ventricular post pacing interval (PPI) and tachycardia cycle length (TCL) was 130 ms, ventricular pacing during tachycardia results in V-A-A-V response before tachycardia resumes, and showed concentric atrial activation with earliest point at CS 9-10, indicating an AT from coronary sinus origin. AT was terminated during the RFA.

Electrophysiology study is important to correctly diagnose AT, especially when P-wave during tachycardia in the surface ECG is uncertain. Several pacing maneuver during electrophysiology study can be very helpful to verify the diagnosis of AT.

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Keywords: focal atrial tachycardia (AT), electrophysiology study, pacing maneuver, radiofrequency ablation (RFA)

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Laporan Kasus

Ablasi Frekuensi-Radio pada Takikardia Atrium Fokal di Sinus Koronarius

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Takikardia atrium (TA) fokal dapat didefinisikan sebagai aktivitas atrium yang berasal dari suatu fokus dengan penyebaran yang sentrifugal. Berdasarkan informasi yang tersedia saat ini, aktivitas fokal dapat disebabkan oleh otomatisitas, aktivitas pemicu, ataupun mikro *reentry*. Secara umum, TA memiliki respons yang buruk terhadap obat-obatan namun dapat ditangani oleh ablasi frekuensi-radio dengan keberhasilan jangka panjang yang tinggi. TA fokal diperkirakan mencakup 3% sampai 17% pasien dengan takikardia supra ventrikel (TSV) yang mendapatkan ablasi frekuensi-radio (AFR). Studi elektrofisiologi penting untuk mengetahui mekanisme dari TSV sebelum dilakukan AFR. Pengamatan dan manuver pacu dapat dilakukan untuk mengindentifikasi mekanisme TSV.

Seorang pasien wanita berusia 54 tahun memiliki keluhan utama jantung berdebar. Saat berdebar, EKG pasien menunjukkan kompleks takikardia reguler sempit dengan gelombang P yang sulit ditentukan dengan tepat. Studi elektrofisiologi menunjukkan interval VA 130 mdetik, perbedaan antara interval VA saat takikardia dan saat dilakukan pacu ventrikel kanan adalah 55 mdetik, tidak terdapat pemanjangan pada aktivasi atrial, perbedaan interval setelah picu ventrikel dan saat takikardia adalah 130 mdetik, dan picu ventrikel saat takikardia menghasilkan respons V-A-A-V sebelum takikardia berlanjut, dan menunjukkan aktivitas atrium yang konsentrik dan paling awal dari CS 9-10, mengindikasikan TA berasal dari sinus koronarius. TA berhasil dihilangkan saat AFR.

Studi elektrofisiologi penting untuk mendiagnosis TA, terutama bila gelombang P dari EKG saat takikardia tidak jelas. Beberapa manuver pacu saat studi elektrofisiologi sangat membantu untuk menegakkan diagnosis TA.

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Kata kunci: takikardia atrium (TA) fokal, studi elektrofisiologi, manuver pacu, ablasi frekuensi-radio (AFR)

Introduction

Focal atrial tachycardia (AT) is relatively uncommon arrythmia. It accounts approximately 10% of supraventricular tachycardia (SVT). Although there is a greater absolute number of women with AT, the proportion of AT in both genders is similar. AT comprise a progressively greater proportion of those with paroxysmal SVT with

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increasing age, accounting for 23% of patients older than 70 years old. Generally, AT response poorly to medication and may be associated with atrial fibrillation and atrial flutter. With the advent of radiofrequency ablation (RFA), this form of tachycardia can be treated with high long-term success.[1, 2]

Focal AT represents approximately 3% to 17% of the patients referred for SVT radiofrequency ablation. An electrophysiology study should be done before radiofrequency ablation to correctly diagnose the mechanism of the SVT. It is important to recognize that as with most diagnostic tests, no single observation or maneuver is 100% sensitive or specific. Therefore, it is important to obtain data from multiple observations and maneuvers to verify the diagnosis before proceeding with ablation.[3, 4]

Case Illustration

A 54-year-old female presented to the outpatient clinic of National Cardiovascular Center Harapan Kita (NCCHK) with chief complaint of palpitation, reporting that she had suffered recurrent palpitation since five years ago. There was sudden onset of palpitation and sustained, sometimes relieved at rest. She also had three times of hospitalization in the previous five years because of palpitation. No history of syncope or lightheadedness. Slow release oral verapamil 240 mg once daily failed to attenuate the reccurences of arrythmia. She denied any risk factors for coronary artery disease nor other chronic diseases.

During examination the patient was afebrile. The pulse was 89 beats per minute and regular, the blood pressure was 142/85 mmHg, the respiratory rate was 16 breaths per minute, and the oxygen saturation was 99%. Physical examination was within normal limit. The chest X-ray and laboratory findings were also unremarkable.

During palpitation from the last hospitalization, the ECG showed narrow complex regular tachycardia with QRS rate 193 times/minutes. The P-wave was difficult to ascertain clearly (**Figure 1**).

The patient was assessed with working diagnosis of symptomatic supraventricular tachycardia (SVT). She was planned to go an electrophysiology evaluation and ablation. Slow release oral verapamil was discontinued five days before the procedure.

During electrophysiology study, three quadripolar electrode catheters were inserted via two 6F sheaths and one 7F sheath into right femoral vein and placed in right ventricle apex, His bundle area, and High Right Atrium (HRA). Jugular vein puncture performed and decapolar catheter was inserted and placed in coronary sinus.

By catheter maneuver, narrow QRS complex supraventricular tachycardia (SVT) with VA interval 130 ms could be induced (**Figure 2**). Diagnostic pacing maneuver then performed. The difference between VA interval during tachycardia and VA interval during

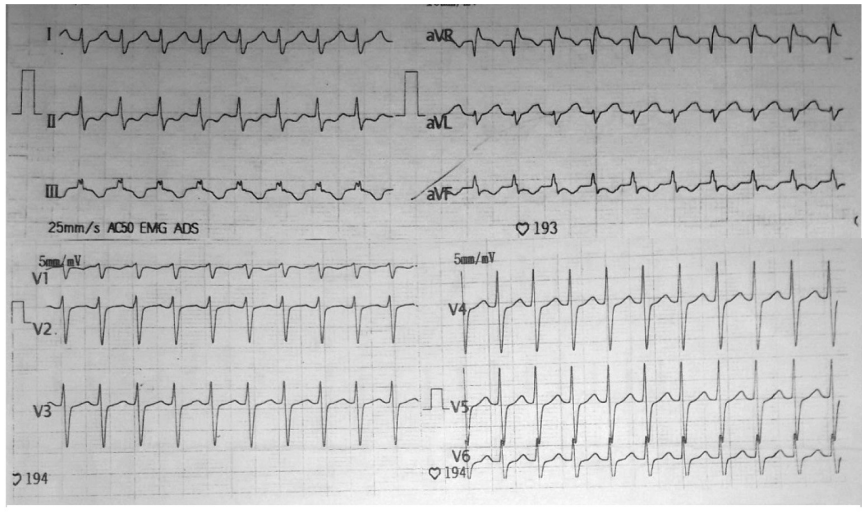

Figure 1. ECG during Tachyarrythmia. Regular narrow complex QRS tachycardia with P-wave that was difficult to ascertain

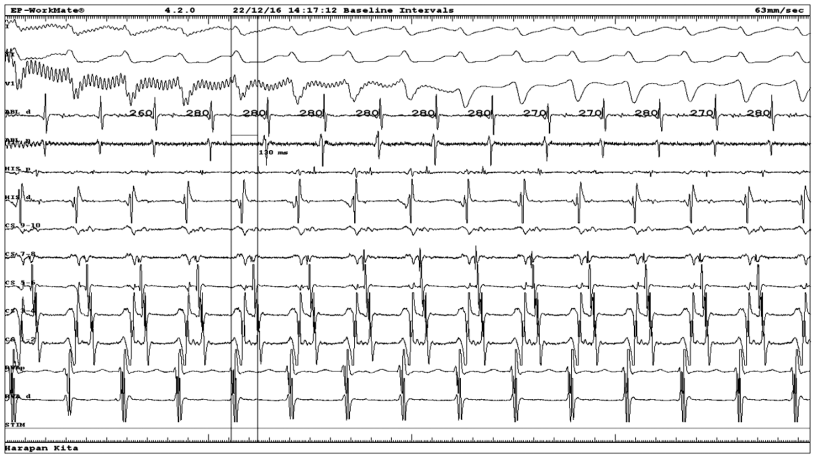

Figure 2. VA interval during tachycardia (VA interval 130 ms).

RV pacing was 55 ms (**Figure 3**). It was showed no advanced in atrial activation on PVC (**Figure 4**) and difference between ventricular post pacing interval (PPI) and tachycardia cycle length (TCL) was 130 ms.

Ventricular pacing during tachycardia results in V-A-A-V response before tachycardia resumes and showed concentric atrial activation with earliest point at CS 9-10 (**Figure 5**), indicating an AT from coronary sinus origin.

Multiple RFA at coronary sinus (CS) ostium then performed (20 watt, 50°C, 60 second) at the earliest activation area (**Figure 6**), AT was terminated during the RFA. After 15 minutes evaluation, AT was not inducible. Then patient was sent home without any medication. At follow-up, patient came to the outpatient clinic and had no palpitation anymore.

Discussion

Focal AT is characterized as a fast rhythm from a discrete origin, discharging at a rate that is generally

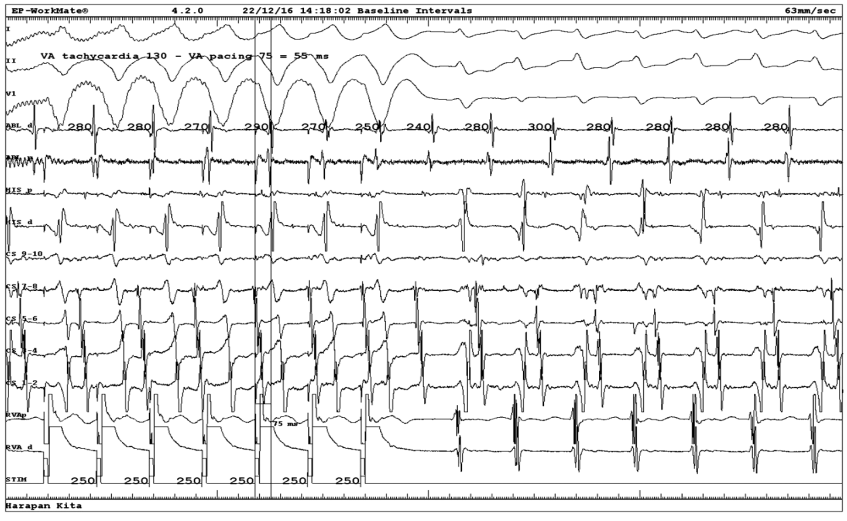

Figure 3. Differences between VA interval during tachycardia–VA interval during RV pacing was 55 ms.

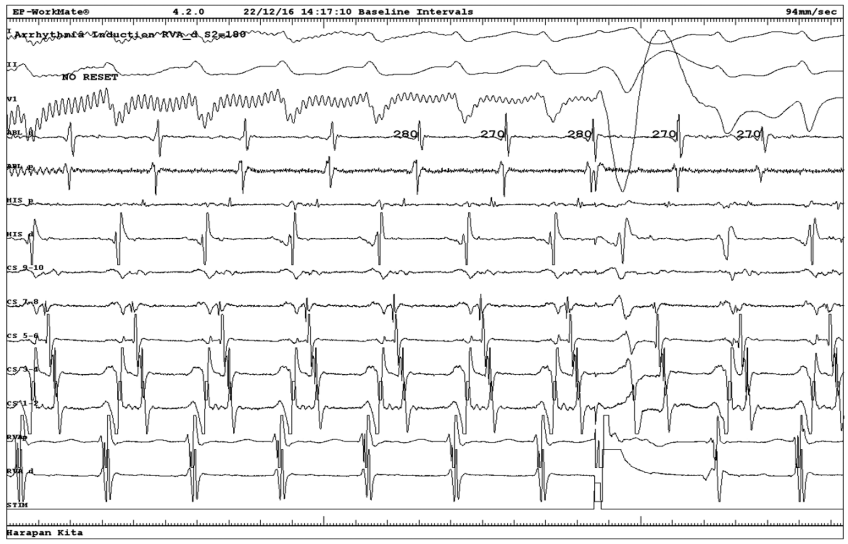

Figure 4. Ventricular extrastimuli showed no advanced in atrial activation (no reset).

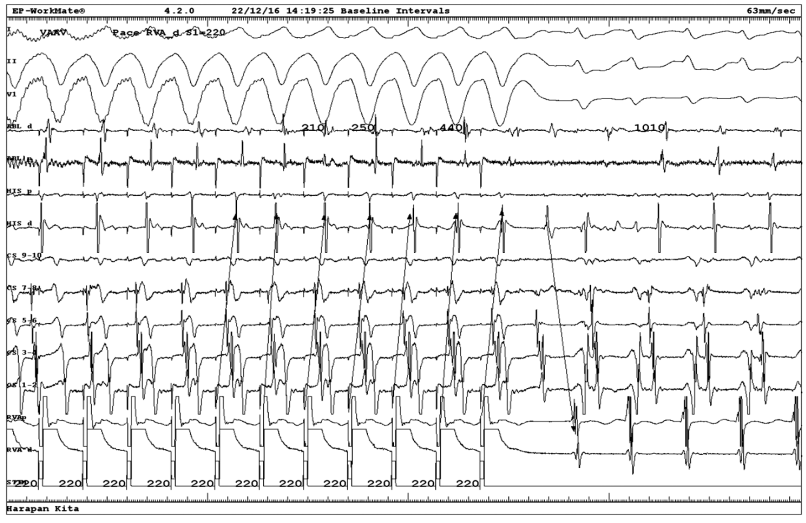

Figure 5. Pacing maneuver from RV apex showed response to ventricular over drive pacing was VAAV.

regular, and conducting in a centrifugal manner throughout the atrial tissue. The demographics of focal AT in the adult population will continue to change as SVTs are increasingly ablated at a younger age.[3]

Definition

AT is defined as atrial activation originating from a discrete focus with centrifugal spread. Typically, activation is not continuous throughout the cardiac cycle length, the hallmark of re-entry. The focus originates from an area of atrial myocardium arbitrarily defined as<2 cm in diameter. However, focal AT can generally be defined by a slower P-wave rate (<200/min) and an isoelectric interval between P-waves. Clinically, AT is suspected if we found patient with narrow complex QRS regular tachycardia with long RP interval.[1,3]

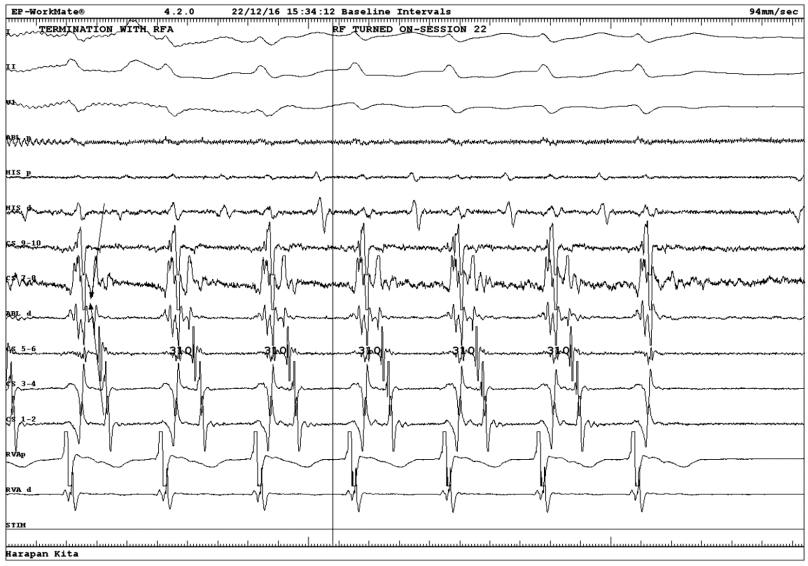

Figure 6. Ablation at earliest atrial activation point, termination after 4 beats.

Mechanism

Focal AT can be sustained or non-sustained. The atrial rate during focal AT is usually between 100 and 250 bpm. Presence and severity of symptoms during focal AT are variable among patients. Focal AT in the adult population is usually associated with a benign prognosis, although AT-mediated cardiomyopathy has been reported in up to 10% of patients referred for ablation of incessant SVT. Non-sustained focal AT is common and often does not require treatment.[3]

Available information suggests that focal activity can be caused by automaticity, triggered activity, or microreentry. There are some overlaps based on the pharmacological characterization of these different AT mechanisms.[2,5] Automatic AT is suggested by the following characteristics:[6]

- The onset of AT is followed by a gradual acceleration or "warm-up", and termination follows slowing or "cool-down"
- AT is initiated spontaneously or with the administration of isoproterenol
- AT cannot be initiated, entrained, or terminated by programmed electrical stimulation
- AT can be transiently suppressed with overdrive atrial pacing, but subsequently resumes, with a gradual increase in the atrial rate

Triggered activity is suggested by the following characteristics:[6]

- AT is initiated or terminated by programmed electrical stimulation, and its initiation is cycle length–dependent.
- Pacing does not entrain tachycardia, but can produce suppression or termination

A *reentry mechanism* is suggested by the following characteristics:[6]

- AT is reproducibly initiated and terminated by programmed electrical stimulation.
- AT can be entrained by pacing

Location

Focal AT tends to originate in characteristic locations associated with anatomic structures. Sixty three percent of AT arose in the right atrium and 37 percent in the left atrium. The distribution of sites of origin among the right atrial tachycardias include tricuspid annulus (35%), crista terminalis (34%), coronary sinus ostium (17%), perinodal tissues (9%), and RA appendage (4%). Left atrial tachycardias were predominantly located around the pulmonary veins (67%). Less common sites of origin include the mitral annulus (17%), coronary sinus body (6%), left intraatrial septum (6%), and the LA appendage (4%).[6,7] The electrophysiology study

revealed that the earliest activation of focal AT was from coronary sinus ostium.

Diagnosis

For a patient presenting in SVT, the 12-lead ECG can potentially identify the arrhythmia mechanism (**Figure 7**). The tachycardia should first be classified according to whether there is a regular or irregular ventricular rate. An irregular ventricular rate suggests atrial fibrillation (AF), multifocal atrial tachycardia (MAT) or atrial flutter with variable AV conduction. If the SVT is regular, this may represent AT with 1:1 conduction or an SVT that involves the AV node. SVTs that involve the AV node as a required component of the tachycardia reentrant circuit include AVNRT and AVRT. A long RP interval is typical of AT because the rhythm is driven by the atrium and conducts normally to the ventricles. In AT, the ECG will typically show a P-wave with a morphology that differs from sinus that is usually seen near the end of or shortly after the T-wave.[3] During palpitation, ECG of our patient showed narrow regular complex tachycardia with P-wave that was difficult to ascertain clearly. Therefore it could be an AVNRT or other mechanism.

An electrophysiology study should be done before radiofrequency ablation. It can be performed to correctly diagnose the mechanism of the SVT. It is important to recognize that as with most diagnostic tests, no single observation or maneuver is 100% sensitive or specific. Therefore it is important to obtain data from multiple observations and maneuvers to verify the diagnosis before proceeding with ablation.[4]

Once the tachycardia is initiated in the electrophysiology laboratory, or if the patient presents to the laboratory in tachycardia, certain tachycardia characteristics may provide important clues to the mechanism of the tachycardia even before attempting diagnostic maneuvers.[4]

- Rate: There is substantial overlap in the rates of tachycardias and different mechanisms but an SVT cycle length of over 500 ms (<120 bpm) has a relatively high positive predictive value for AVNRT (approximately 83%)
- VA interval: The VA interval is similar to the RP interval measured on a surface ECG. A septal VA interval of<70 ms was found in 47% of patients with SVT and had a 99% PPV for AVNRT. Generally, AVRT and AT have VA intervals of>70 ms

Heart rate of our patient was 190-220 bpm, and the VA interval was 130 ms. Therefore, our patient had a possibility of AVRT or AT.

An eccentric atrial activation favors the diagnosis of AVRT and AT. This has a positive predictive value of 76% and 24% for AVRT and AT respectively. However, eccentric activation cannot rule out AVNRT as the mechanism of tachycardia.[4] Our patient's electrophysiology study showed an eccentric atrial activation.

Pacing maneuver can be very helpful in differentiating AT from AVNRT and AVRT (**Table 1**). During atrial extrastimulus testing, dual AV nodal physiology is typically manifested by a jump of 50 milliseconds or longer in the A2H2 interval following a shortening in the A1A2 interval by 10 milliseconds. When two atrial extrastimuli are delivered, a jump from fast to slow pathway conduction is defined as an increase in the A3H3 interval of 10 milliseconds in the A2A3 interval (A1A2 being constant).[7]

Delivery of His-synchronous premature ventricular contractions extrasystole, whether spontaneous or induced, can often help identify the mechanism of arrhythmia. A commonly used maneuver is to the deliver a His-synchronous premature ventricular contraction (PVC), delivered on time or within 40 ms of the His potential. Once this PVC is delivered, careful measurements should be made to assess whether the subsequent atrial signal has been advanced. In AVRT, the atrial activation may be advanced or delayed or the tachycardia terminated without conduction to the atria by premature ventricular stimuli during His refractoriness. Meanwhile fast-slow AVNRT and AT are not affected by appropriately timed ventricular extrastimuli unless retrograde His bundle activation is advanced.[4,7,8] Patient showed no advanced of atrial activation by PVC during His refractory period.

Overdrive pacing from the right ventricle (RV) at a cycle length (CL) that is 10–40 ms shorter than the tachycardia CL provides a rapid tool to rule AT in or out. If, during overdrive ventricular pacing, the atrial CL is accelerated to the pacing CL, and the tachycardia continues after pacing is stopped, then a post ventricle overdrive pacing (VOP) response that is atrial-atrial-ventricular (A-A-V) rules in AT (VAAV response) while a post-VOP response that is atrial ventricular (AV) effectively ruling in AVRT or AVNRT (VAVA response).[4,9]

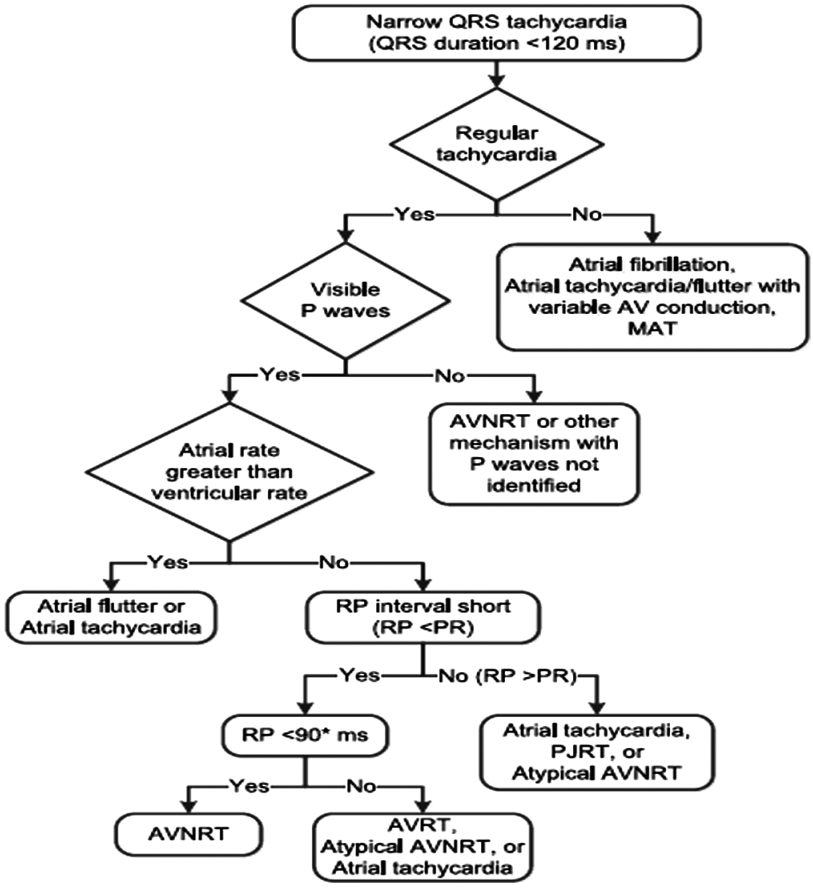

Figure 7. Differential Diagnosis for Adult Narrow QRS Tachycardia

Table 1. Pacing Maneuver to differentiate Atrial Tachycardia from Atrioventricular Reentrant Tachycardia and Atrioventricular Nodal Reentrant Tachycardia.[4,7]

Maneuver	AVRT	AVNRT	AT
Late ventricular extrastimulus delivered during tachycardia	Advances (or delays) atrial activation or terminates tachycardia without conduction to atrium	Unable to advance atrial activation unless retrograde His bundle activation is advanced	Unable to advance atrial activation unless His is advanced
Post ventricular overdrive pacing response	V-A-V-A, unless decrementally conducting APs	V-A-V-A, unless fast-slow or slow-slow AVNRT	V-A-A-V
Difference between VA during ventricular pacing at TCL and VA during tachycardia	<85 msec	>85 msec	Variable, VA conduction may be absent

In a VAAV response, which is consistent with a diagnosis of AT, the last paced ventricular beat (V) goes up the AV node and suppresses the atrial automatic focus with an early atrial electrogram (A). The atrial event following last paced V cannot descend down the AV node as it will be still refractory. If the tachycardia continues the next beat will initiate an atrial depolarization (A) which conducts to the ventricle (V) yielding the VAAV response.[4]

In a VAVA response which is consistent with either AVNRT or AVRT, the last ventricular paced beat (V) travels up to the atrium (A), either through the AV node or accessory pathway depending on whether the SVT is AVNRT or AVRT. Subsequently the ventricle and then the atrium are again activated by continuation through the re-entrant loop (VA). In some situations, a pseudo-VAAV response can occur especially when the retrograde conduction is slow either in an atypical AVNRT or a retrogradely conducting slow accessory pathway. In this situation recognizing the last entrained atrial beat is important as that is the first postventricular atrial beat.[4]

Ventricular pacing during tachycardia results in VAAV response before tachycardia resumes in our patient, indicating that the narrow regular complex tachycardia was an AT.

In response to ventricular overdrive pacing, the differences in VA times between pacing and tachycardia are less than 85 milliseconds suggesting AVRT. On the other hand, differences in VA times between pacing and tachycardia are more than 85 milliseconds suggesting AVRT. Meanwhile AT has variable differences in VA times between pacing and tachycardia, sometimes VA conduction may be absent.[7] The differences between VA interval during tachycardia and VA interval during RV pacing in our patient was 55 ms. It was shown that the possibility of the mechanism in this patient was AVRT or AT.

Management

The management of focal AT is divided into acute treatment and chronic suppressive therapy. The acute management of a patient with an AT is guided by the hemodynamic stability of the patient.

Intravenous beta-blockers or nondihydropyridine calcium channel blockers (i.e., diltiazem or verapamil) may be given for acute treatment of focal AT to the hemodynamically stable patient. If intravenous beta-blockers or nondihydropyridine calcium channel blockers are ineffective or if patient is intolerant, or likely to be intolerant, intravenous amiodarone or ibutilide may be reasonable in the acute setting to either restore sinus rhythm or slow the ventricular rate in hemodynamically stable patients with focal AT. Intravenous adenosine can be useful in the acute setting to either restore sinus rhythm or diagnose the tachycardia mechanism in patients with suspected focal AT. In hemodynamically unstable patient that suspected as a focal AT synchronized cardioversion is recommended.[3]

Chronic therapy of repetitive focal AT is designed to prevent arrhythmia recurrence and to control the ventricular rate if the arrhythmia recurs. Patients with relatively rare and brief arrhythmias and few or no symptoms do not require chronic therapy. Oral beta-blockers, diltiazem, or verapamil are reasonable for ongoing management in patients with symptomatic. Patients who do not respond to one of these agents may have successful suppression with another. Catheter ablation is recommended in patients with symptomatic focal AT as an alternative to pharmacological therapy focal AT. More aggressive antiarrhythmic drugs may be considered for patients who fail beta-blockers, diltiazem and verapamil who do not want or are not good candidates for ablation therapy. Flecainide or propafenone can be effective for ongoing management in patients without structural heart disease or ischemic heart disease who have focal AT. In patients with significant comorbidities and structural heart disease, oral amiodarone may be reasonable.[3]

Catheter ablation is a well-established therapeutic option for patients with atrial tachyarrhythmia. Mapping, characterization and ablation of AT is often challenging due to a complex underlying substrate and a great variability of AT mechanism and localization throughout the atria. Conventional mapping techniques such as activation mapping or entrainment maneuvers can be challenging to interpret and may be misleading, inconsistent or involve the risk of AT-termination or degeneration in other, not clinically relevant, arrhythmias. The use of 3D electro-anatomical mapping systems can facilitate mapping and guide ablation. Further, high-density mapping of AT can reveal even more complex activation patterns that may extend beyond the common understanding and categorizing of atrial tachycardia as focal, localized reentry or macroreentry.[10,11]

3D Electro-anatomical mapping has better acute success rate than conventional mapping (90% vs. 60%). Six-month outcome is also better in 3D electro-

anatomical mapping than conventional mapping (85% vs. 75%). Although based on data of prior publications, conventional mapping can still be a beneficial choice to guide catheter ablation based on the high success rate and lower cost.[11]

Our patient had a chronic recurrent focal AT. Slow release oral verapamil 240 mg once daily failed to attenuate recurrences of arrythmia. This patient was recommended to go an ablation, and agreed to do so. Although the origin of focal AT was originated from a rare area, conventional mapping successfully revealed that focal AT was from CS ostium. Multiple RFA at CS ostium then performed with the termination of AT.

Conclusion

A 54-year-old female presented to the outpatient clinic of NCCHK with chief complaint of palpitation. During palpitation her ECG showed narrow complex regular tachycardia with the P-wave that was difficult to ascertain clearly. Electrophysiology study using conventional mapping showed that the narrow complex reguler tachycardia was AT from CS ostium origin. Multiple RFA at CS ostium then performed with the termination of AT, and was not inducible after evaluation. Electrophysiology study has an important role to diagnose focal AT and to find location of the earliest activation signal. RFA is recommended for patient with symptomatic focal AT.

Abbreviations

AF: atrial fibrillation
AV: atrioventricular
AT: atrial tachycardia, *takikardia atrium*/TA
AVNRT: atrioventricular nodal reentrant tachycardia
AVRT: atrioventricular reentrant tachycardia
CL: cycle length
ECG: electrocardiogram, *elektrokardiogram*/EKG
HRA: high right atrium
LA: left atrial
MAT: multifocal atrial tachycardia
NCCHK: National Cardiovascular Center Harapan Kita
PPI: post pacing interval
PVC: premature ventricular contraction
RA: right atrial
RFA: radiofrequency ablation, *ablasi frekuensi-radio*/AFR
RV: right ventricular
SVT: supraventricular tachycardia, *takikardia supra-ventrikel*/TSV
TCL: tachycardia cycle length
VA: ventriculoatrial
VOP: ventricle overdrive pacing

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