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Case Series

Surface Electrocardiogram Clues to Differentiate Spontaneous AV Nodal Reentrant Tachycardia from Junctional Tachycardia

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ABSTRACT

Supraventricular Tachycardia (SVT) is a very commonly encountered arrhythmia in clinical practice. It is defined as an arrhythmia originating above the level of the ventricles and resulting in a heart rate greater than 100 beats per minute. SVT usually presents as a narrow complex tachycardia, but can occasionally present as a wide complex rhythm as well. The differential diagnosis of SVT consists of Atrioventricular Nodal Reentrant Tachycardia (AVNRT), Atrioventricular Reentrant Tachycardia (AVRT), Atrial Tachycardia (AT) and Junctional Tachycardia (JT), albeit rare in adults. Although a definitive diagnosis of the arrhythmia requires an invasive electrophysiological study, certain clues on the surface 12 lead ECG as a result of spontaneously occurring electrical events help make an accurate diagnosis. Short RP SVT is most of the time due to typical AVNRT; however, albeit rare in adults, it would be very difficult to differentiate it from JT. Here, we present three such scenarios and discuss various electrophysiological principles that underlie these findings.

Keywords: *Junctional Tachycardia; AVNRT; ECG; SVT.*

INTRODUCTION

Supraventricular Tachycardia (SVT) is a very commonly encountered arrhythmia in clinical practice. It is defined as an arrhythmia originating above the level of the ventricles and resulting in a heart rate greater than 100 beats per minute. SVT usually presents as a narrow complex tachycardia, but can occasionally present as a wide complex rhythm as well. The differential diagnosis of SVT consists of Atrioventricular Nodal Reentrant Tachycardia (AVNRT), Atrioventricular Reentrant Tachycardia (AVRT), Atrial Tachycardia (AT) and Junctional Tachycardia (JT), albeit rare in adults. Although a definitive diagnosis of the arrhythmia requires an invasive electrophysiological study, certain clues on the surface 12 lead ECG as a result of spontaneously occurring electrical events help make an accurate diagnosis. Short RP SVT is most of the time due to typical AVNRT; however, albeit rare in adults, it would be very difficult to differentiate it from JT. Here, we present three such scenarios and discuss various electrophysiological principles that underlie these findings.

CASE PRESENTATION

Case 1

A 75-year-old man with no significant past medical history presented with recent palpitations, light headedness, and a narrow complex rhythm of around 100 beats per minute. There were no clear P waves evident on the surface ECG (Figure 1). Vagal maneuver with valsalva led to termination of the tachycardia. Review of the termination revealed the loss of a terminal positive deflection of the QRS in lead II while the patient was in sinus rhythm. This was suggestive of a very short RP tachycardia, consistent with typical slow-fast AVNRT. (Figure 2) below shows the rest of the rhythm strip from Figure 1 with termination of the rhythm after performance of the Valsalva maneuver as shown by the black arrow.

Figure 1: There were no clear P waves evident on the surface ECG

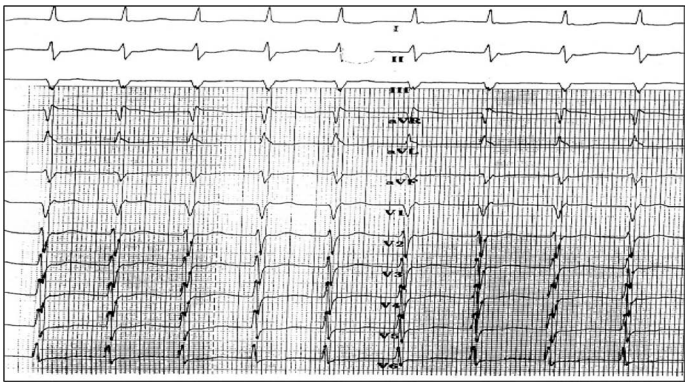
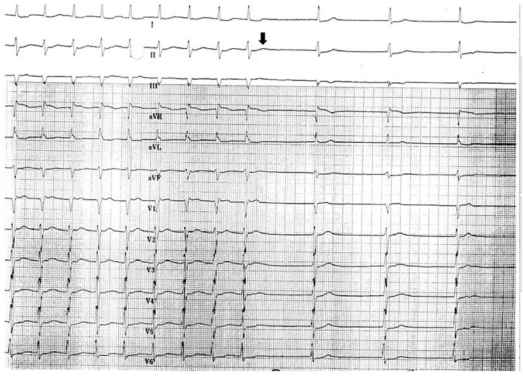


Figure 2: The rest of the rhythm strip from Figure 1 with termination of the rhythm after performance of the Valsalva maneuver as shown by the black arrow



Case 2

An 84-year-old female presented with the arrhythmia shown in Figure 3 below. Her ECG showed a narrow complex tachycardia at a rate of 125 bpm. There was evidence of a r' in lead V1 suggestive of typical AVNRT (Figure 3). A repeat EKG later showed a narrow complex tachycardia at a rate of 154 bpm shown in Figure 4. As shown by the black arrows in the figure, the patient had premature atrial complexes occurring in a pattern of trigeminy spontaneously. A closer review of the effect of these premature atrial complexes on the tachycardia revealed a consistent pattern of tachycardia reset with these appropriately timed premature atrial complexes (Figure 5). This again is very suggestive of AVNRT as the mechanism of the tachycardia.

Figure 3: An 84-year-old female presented with the arrhythmia

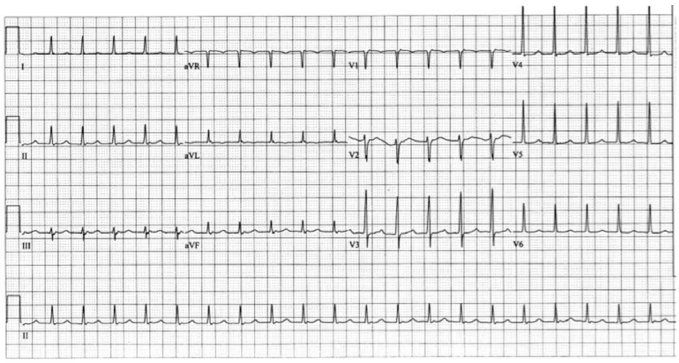


Figure 4: A repeat EKG later showed a narrow complex tachycardia at a rate of 154 bpm

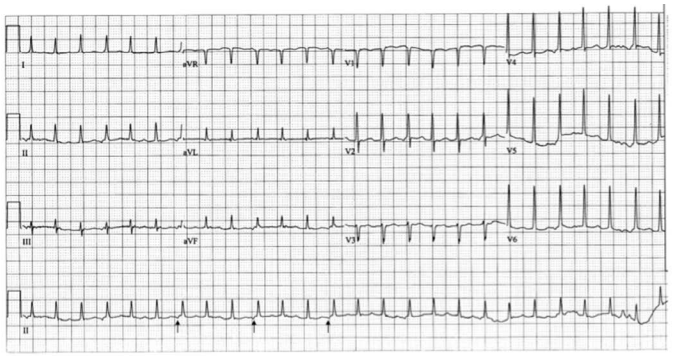
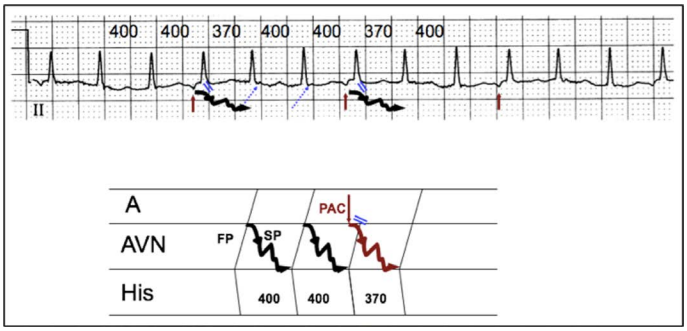


Figure 5: A closer review of the effect of these premature atrial complexes on the tachycardia revealed a consistent pattern of tachycardia reset with these appropriately timed premature atrial complexes



Case 3

A 46-year-old male who was in pre-op for liver transplant developed the narrow complex slow tachycardia with a rate of 109 bpm. ECG showed a short to mid RP tachycardia with an RP interval of 90 ms (Figure 6). Previous ECGs had shown normal PR interval and P wave morphology. Repeat EKG shown in Figure 7 below revealed two spontaneously occurring PVCs (black arrows) with termination of the tachycardia. The patient was then treated with beta blockers and underwent his liver transplant. The observation of tachycardia termination in response to PVCs supports a diagnosis of a reentrant tachycardia mechanism rather than junctional tachycardia. A non-reentrant junctional tachycardia would not be expected to terminate in response to PVCs.

Figure 6: ECG showed a short to mid RP tachycardia with an RP interval of 90 ms

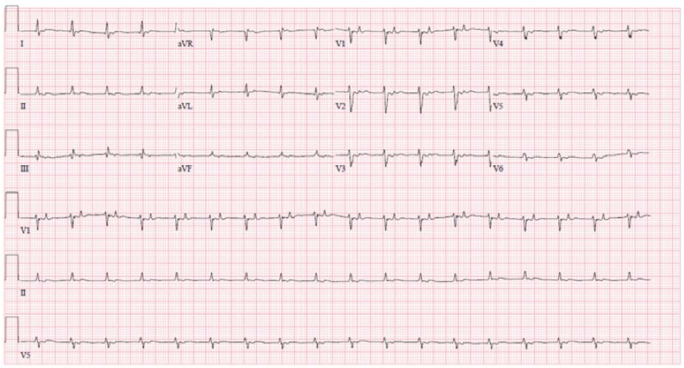
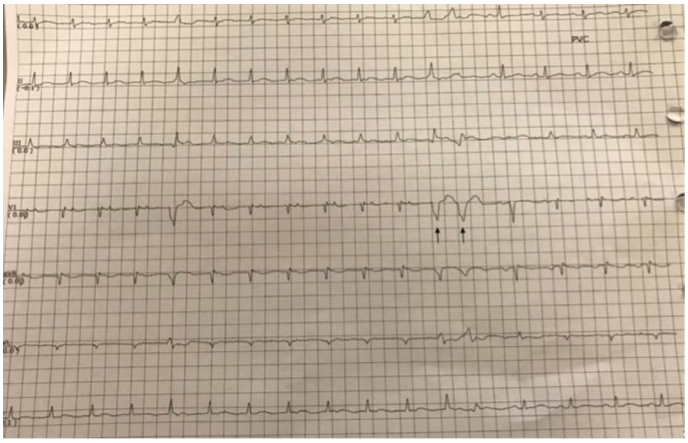


Figure 7: Previous ECGs had shown normal PR interval and P wave morphology. Repeat EKG



DISCUSSION

Patients with AVNRT often have symptoms including palpitations, light headedness, and neck pounding.[1] In these cases, patients can be offered multiple options for treatment. In the acute setting, Valsalva maneuvers, when performed correctly, can terminate paroxysmal SVT such as AVNRT approximately 54% of the time.[2] When vagal maneuvers are not successful for AVNRT, medications including intravenous adenosine, diltiazem, verapamil or beta blockers can be used and are effective 80-98% of the time.[1] In rare instances, when treatment with medications is not successful, synchronized cardioversion can be performed if the patient is unstable[1] For ongoing management of AVNRT, oral beta blockers, diltiazem, or verapamil can be useful, although an Electrophysiologic (EP) study with ablation often provides a permanent solution to the problem.[1] If the arrhythmia was indeed a junctional rhythm, ablation has very low success rates with higher incidence of heart block when compared with patients who are in AVNRT.[3] Therefore, differentiation of these two arrhythmias becomes important when deciding further treatment options for a patient.

Electrophysiologic studies can be used to differentiate AVNRT and junctional tachycardia, although often difficult since both are often very near each other in a small anatomic area around the AV node. A common maneuver used is the introduction of appropriately timed premature atrial complexes. To further explain this technique, if there is any disruption including advancement, delay, or termination of the tachycardia after a PAC, which has been timed to His refractoriness, it proves the rhythm is AVNRT. This will rule out junctional tachycardia as the PAC would not be able to penetrate a spontaneously firing junctional focus. Alternatively, when the early PAC advances the His potential immediately after it without terminating arrhythmia, this proves the rhythm is junctional tachycardia.[3] The downside of this technique is the requirement to do an EP study in order to differentiate the two abnormal rhythms. This raises the question of whether there are more noninvasive techniques to determine whether an arrhythmia is AVRNT or JT. The reason they are difficult to differentiate is due to the fact that they often have similar characteristics including clinical features and intracardiac activation pattern. However, they have different mechanisms. The mechanism of AVNRT is a reentry circuit while the mechanism of JT is most often enhanced automaticity. Given these differing mechanisms, they respond to certain stimuli in different ways. In the remainder of this section, with help from the cases above, we will discuss some of those stimuli and how they can contribute to making a clinical diagnosis of AVNRT versus JT. The cases above provide examples of the utilization of vagal maneuvers, spontaneous PACs, and spontaneous PVCs in differentiating AVNRT from JT, all of which are more noninvasive techniques.

In case 1 above, the initial differential diagnosis includes junctional tachycardia versus slow AV Nodal Reentrant Tachycardia (AVNRT). In this case, after performing vagal maneuvers, the arrhythmia terminated, as shown in both Figure 2 above. Vagal maneuvers will cause a block in the AV node and in this case terminated the tachycardia by blocking the slow pathway, which proves that this arrhythmia was dependent on the AV node. Alternatively, junctional rhythms originate from a focus near (usually below) the AV node. Therefore, Valsalva maneuvers will only slow junctional rhythms rather than terminate them. Of note, termination of the fast pathway by performance of a vagal maneuver is controversial given there is evidence suggesting that vagal tone does not affect the fast pathway.[4] Once diagnosed with AVNRT, the patient was able to be properly treated with medication by increasing the dose of metoprolol to 25 mg po bid without the need for invasive procedures. Also, as demonstrated in the Figure 2 above, the slow pathway was blocked with Valsalva maneuver.

In case 2 above, the initial differential diagnosis would once again include junctional tachycardia and slow AV nodal reentrant tachycardia. A repeat EKG revealed spontaneous PACs that reset the tachycardia by conducting down the slow pathway. As discussed previously, there is evidence showing that timed PACs can be used in electrophysiologic studies to differentiate between AVNRT and JT with 100% specificity and high sensitivity.[3] Therefore, spontaneous PACs occurring when a patient is in a supraventricular tachycardia will also help to confirm the diagnosis of either AVNRT or JT. In this case, since the tachycardia was reset after spontaneous PACs, this patient was in slow AVNRT. If the patient had been in JT, the arrhythmia would not have been reset.

In the final case, a patient presented with slow supraventricular tachycardia. As in the previous two cases, the differential included slow AVNRT versus JT. In a subsequent EKG, the patient exhibited two spontaneous PVCs, after which the abnormal rhythm was terminated. In prior studies, PVCs have been used in EP studies to determine whether a supraventricular tachycardia was JT or AVNRT.[5] During an EP study, a His refractory PVC can be inserted close to the atrial insertion site. If there is advancement of the atrium, then the arrhythmia is AVNRT. If late-coupled His refractory PVCs advance or delay atrial activation, then the arrhythmia is JT.[5]

CONCLUSION

In conclusion, in a clinical practice setting vagal maneuvers, spontaneous PACs, and spontaneous PVCs can be used to differentiate between slow AVNRT and JT. These noninvasive techniques are very helpful due to the fact that they will determine whether further more invasive treatment techniques need to be explored. In the case of AVNRT, there is much evidence to show that ablation can lead to successful treatment and prevention of future events. Alternatively, if a clinician determines that a patient has JT, they will know that further treatment with ablation is of limited success with risk for adverse events. This may lead the clini-

cian to decide against further invasive treatments.

CONFLICTS OF INTEREST

None.

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