Cool-down Phenomenon in Differentiating Supraventricular Tachycardias

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January 30, 2024

Abstract

A 63-year-old male patient with a history of hypertension, type 2 diabetes mellitus, prostate cancer and class two obesity was admitted for altered mental status and hypertensive emergency. During his hospital stay he developed narrow complex tachycardia and it was difficult to definitively diagnose the underlying arrhythmia. Observation of the cool down phenomenon on electrocardiogram allowed us to make the diagnosis of atrial tachycardia and elegantly ruled out other causes on the differential. We report this interesting case of narrow complex tachycardia.

Introduction

Atrial tachycardia (AT) is a regular supraventricular tachycardia originating in the atria and away from the sinus node.[*1*] Focal ATs arise from a single site within the left or right atrium, in contrast to macro reentrant atrial arrhythmias such as atrial flutter and atrial fibrillation, which involve multiple sites or larger circuits. AT is relatively uncommon, accounting for between 5 and 15 percent of arrhythmias in adults undergoing an electrophysiology study for paroxysmal supraventricular tachycardia.[2] The incidence of AT is similar among men and women.

Warm-up and cool-down phenomena refer to an observable acceleration when transition from normal sinus rhythm to tachycardia and deceleration, when transitioning from tachycardia to a normal sinus rhythm. The presence of this suggests that enhanced automaticity is the underlying mechanism of the tachyarrhythmia.[1] We observed the cool down phenomenon and report the application of this to diagnose a case of narrow complex tachycardia.

Case presentation

A 63-year-old man with a past medical history significant for hypertension, type 2 diabetes mellitus, prostate cancer and class two obesity (BMI 38.9) was admitted from an outside facility for altered mental status. The patient was nonverbal, tachycardic and hypertensive at the outside facility. Brain MRI did not reveal any acute intracranial finding and a CT angiogram of the head and neck showed no vascular occlusion or significant stenosis. Shortly after arrival at our hospital the patient had a tonic-clonic seizure and was noted to have a pronounced facial droop. On physical exam the patient was tachycardic at 139 bpm, tachypneic to 35 per minute, febrile with a temperature of 100.6^{o}F and hypertensive with a blood pressure of 184/115 mmHg. ECG at this time showed sinus tachycardia. Initial lab work was unremarkable. Permissive hypertension was allowed due to possible stroke. Patient was emergently intubated for decreased responsiveness and transferred to the medical ICU.

On day three of admission the patient developed a narrow complex tachycardia that was initially noted on cardiac telemetry. Electrolytes and troponin levels at that time were unremarkable. ECG obtained at this time is shown in Figure 1. The differential diagnosis of this long RP point included atrial flutter, atypical fast-slow AVNRT, orthodromic AVRT and supraventricular tachycardia. The patient was subsequently started on 25ug/min/kg Esmolol drip and the dose was later increased to 50ug/min/kg due to consistent tachycardia of 80-120 bpm. He was later transitioned to 50 mg oral Metoprolol every 6 hours and 30 mg oral Diltiazem every 6 hours. Adequate rate control of 70-80 bpm was eventually achieved. The tachycardia was felt to be related to his acute illness. The patient improved clinically over the following ten days and no further episodes of SVT occurred.

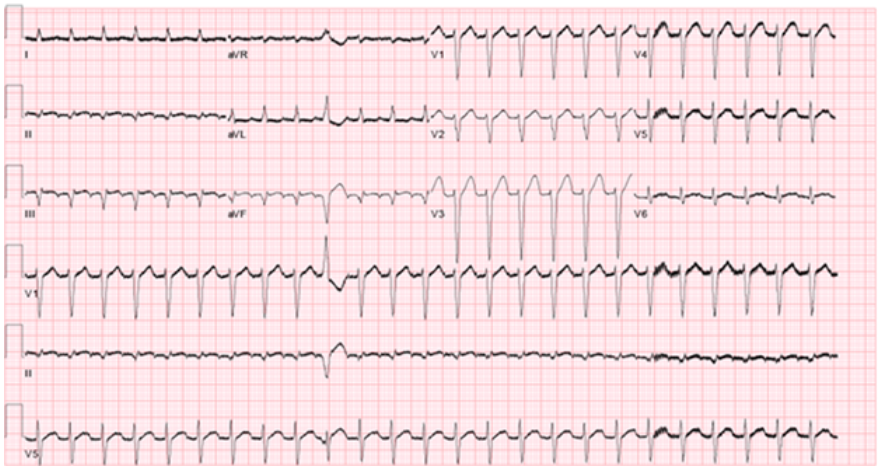

Figure 1. An ECG was obtained while the patient was in long RP supraventricular tachycardia. The differential diagnosis included atrial flutter with a 2:1 AV block, atypical fast-slow AVNRT, orthodromic AVRT, and atrial tachycardia.

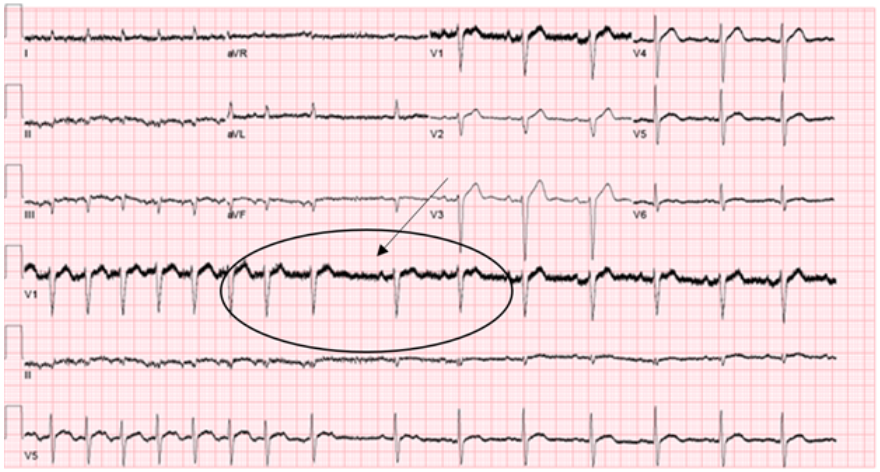

Figure 2. This ECG obtained the same day shows the transition from a supraventricular tachycardia to normal sinus rhythm. The circle and arrow draw attention to the cool down phenomenon before conversion to normal sinus rhythm.

Discussion

The differential diagnosis for this case includes atrial flutter with 2:1 AV block, atrio-ventricular nodal reentry tachycardia (AVNRT), atrio-ventricular reentry tachycardia (AVRT) and atrial tachycardia. Making the correct diagnosis is important because it implicates patient management. We identify the disease process and showcase the rationale used to make the diagnosis.

The diagnosis of typical atrial flutter with 2:1 AV block was initially considered. Typical atrial flutter is caused by a macro-reentry circuit bound by the Cavo-tricuspid isthmus inferiorly and the right atrial roof or the supero-posterior right atrium.[4] This produces an atrial rate of 240-350 bpm and is usually accompanied by a 2:1 AV block with a ventricular response of 120-150 bpm.[4] In the case presented, the patient had a regular supra-ventricular tachycardia with a ventricular rate of 150 bpm during admission. This ventricular rate, so characteristic of typical atrial flutter with a 2:1 AV block, made us feel that this diagnosis was most likely and that the atrial flutter waves were simply masked by the rapid ventricular repolarization/depolarization.

With that said, it was difficult to rule out atypical AVNRT and AVRT from the differential. Atypical AVNRT is caused by a re-entry circuit localized to the AV node and produces a narrow complex tachycardia with long RP P-waves.[5] Orthodromic AVRT is a macro-reentry circuit passing antegrade through the AV node and His-Purkinje system and retrograde through an accessory pathway, and this also produces a narrow complex tachycardia with retrograde long RP P-waves.[3]. This made exclusion of AVNRT and AVRT challenging.

Atrial tachycardia was also considered. Atrial tachycardia is caused by one of several etiologies, including enhanced automaticity, triggered potential or micro-reentry circuit.[1] The presence of discernable P-waves, a long R-P interval and an isoelectric baseline between atrial deflections differentiates atrial tachycardia from other causes of supraventricular tachycardia in most cases.[3]

Ultimately, observation of the cool down phenomenon elegantly confirms the diagnosis of focal atrial tachycardia due to enhanced automaticity. Cool down and warm up describe a more gradual deceleration/acceleration with changes in heart rate, typically lasting a few seconds. Cool down and warm up are only seen in cases where enhanced automaticity is the underlying cause of the arrhythmia. [1,6] The EKG during admission shows the cool down phenomenon as pointed out in Figure 2. Observing this essentially ruled out arrythmias caused by reentry circuits, including atrial flutter with 2:1 AV block, atypical AVNRT and orthodromic AVRT, and allowed us to make the diagnosis of focal atrial tachycardia.

Conclusion

Differentiating causes of narrow complex tachycardia can be challenging. Evaluation usually involves two primary components: assessment of the patient for symptoms and signs of hemodynamic stability (or instability), and assessment of the patient's ECG for clues to the type of tachycardia present. Observation of the warmup or cool down phenomenon on ECG can help clinicians correctly identify the cause of narrow complex tachycardia when the arrhythmia is difficult to define.

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