

SVT with Aberrancy, a Deceitful Doppelganger of VT: A Need for Cautious Introspection for Faultless Elucidation

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Abstract

Patient with SVT with aberrancy should be carefully delineated from VT as management of both arrhythmias are quite different. Inability to accurately identify VT can be life threatening as it can degenerate into ventricular fibrillation, cardiac arrest and sudden death. Moreover, improper diagnosis of SVT as VT and subsequent treatment with Verapamil can be detrimental as it can precipitate acute severe hypotension, cardiac arrest and sudden cardiac death. SVT with aberrancy being a wide complex tachycardia needs careful interpretation looking into aspects including AV dissociation, QRS morphology, fusion complexes, and presence of RBBB or LBBB. That being said efforts should be made to identify these subtle EKG changes so that this wide complex tachycardia is clearly discerned for effective management. Therapeutic options for SVT with aberrancy in a patient who is hemodynamically stable includes beta-blockers, calcium-channel blockers, class II-a & II-b anti-arrhythmic drugs and catheter ablation. Our patient is young age (24 years), with no history of coronary artery disease and absence of myocardial scarring making SVT with aberrancy more likely. Since our patient has paroxysmal SVT, he was prescribed intranasal calcium channel blocker (Ertapamil) and scheduled for elective catheter ablation for his paroxysmal SVT.

Keywords

Supraventricular tachycardia, Re-entry tachycardia, Ventricular tachycardia, Beta blockers, Calcium channel blockers, Re-entry tachycardia, Catheter ablation.

Clinical Case

24-year-old male with no significant past medical history came to ED for evaluation of increased heart rate, shortness of breath and chest pain. Patient mentioned that he went for a morning run at 7.30 AM and his heart rate reached as high as 190. After 1hr his heart rate reached to 140 and did not return to baseline. Patient stated that this happened frequently in the past, notably starting after ACL (Anterior cruciate surgery) surgery that he had several months ago. After the ACL surgery, he was noted to have SVT with aberrancy, but eventually converted into normal sinus rhythm. Patient has noticed that since then after work outs, during

times of fatigue, dehydration, exhaustion and during sickness, he experienced increased heart rate. He denied syncope, bit endorses fatigue, lightheadedness and chest pain. EMS reports that in route to the emergency department, patient received 2 doses of adenosine and patient did not convert to the sinus rhythm. Upon arrival to the ED, patient heart rate is still in between 140-150's. Patient is started on metoprolol 25 mg BID orally. Cardiology was consulted. Work including complete blood count, comprehensive metabolic profile and Chest X ray were unremarkable. Troponin was mildly elevated 28.3. Renal function and electrolytes were within normal limits. The patient's SVT is evaluated by the electrophysiologist in our institution and they recommended intranasal calcium channel blocker for his paroxysmal SVT and advised him to undergo ablation which will be scheduled in the outpatient basis.

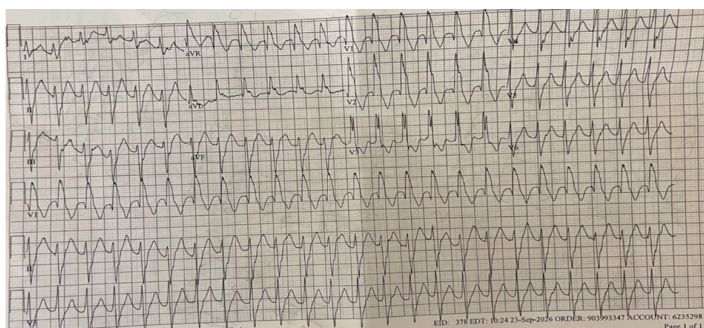


Figure 1: Examination of electrocardiogram in this patient shown below suggests the diagnosis of supraventricular tachycardia (SVT) rather than VT. Despite being a wide complex tachycardia (160 ms), factors that favors SVT include lack of AV dissociation, absence of fusion complexes, and lack of concordance in QRS morphology. Careful interpretation and delineation of SVT from VT is essential for optimum management of SVT as well as preventing deleterious complications of VT. Improper management of VT can be result in ventricular fibrillation, acute severe hypotension and cardiac arrest.

Discussion

Examination of electrocardiogram in this patient shown below suggests the diagnosis of supraventricular tachycardia (SVT) rather than VT. Despite being a wide complex tachycardia (160 ms), factors that favors SVT include lack of AV dissociation, absence of fusion complexes, lack of concordance in QRS (presence of positive and negative waves), QRS duration of 160 ms in the presence of LBBB (Left Bundle Branch Block), absence of initial R wave in aVR, young patient, lack of significant cardiac history including coronary artery disease.

The differential diagnosis of wide complex tachycardia (QRS duration > 120 ms) is very wide. The possibilities include Ventricular tachycardia, supraventricular tachycardia (SVT) with pre-existing bundle-branch block or intraventricular conduction defect, SVT with aberrant conduction due to tachycardia, SVT with wide QRS due to metabolic or electrolyte abnormality, paced rhythm, and artifact [1].

The main mechanism for occurrence of SVT include re-entry tachycardia that travels through AV conduction system [2]. Specifically, the main dilemma is to differentiate between SVT with aberrancy from ventricular tachycardia (VT). It is very critical to differentiate between the two because the management differs between the two arrhythmias.

Presence of dissociated P waves, and A/V ratio < 1 (Slower atrial rate than ventricular rate, thus resulting in fewer P waves as compared to QRS waves) are more likely to be VT as compared to SVT [1-3]. Next QRS duration > 140 ms in RBBB [Right Bundle Branch Block] as well as QRS > 160 ms in LBBB [Left Bundle Branch Block] is more suggestive of VT [3]. Furthermore, as we look at the morphology of QRS complex, synchronous concordance either uniformly positive or negative is more indicative of VT rather than SVT where there will be a combination of positive and negative

QRS complex [1-3]. Also, right axis deviation in LBBB and left axis RBBB suggests VT [3]. Presence of fusion complexes which represent hybrid QRS complex caused by hybrid stimulation of ventricle by supraventricular complex as well as ventricular impulse denotes the presence of VT [1]. Absence of RS complex in the precordial leads is highly indicative of VT rather than SVT where RS complexes are present [4].

According to Vereckei et al., presence of initial R wave in the aVR complex indicates the presence of VT [5]. Calculation of ratio of initial (Vi) and terminal (Vt) ventricular activation velocity can provide information for differentiating between VT and SVT [5]. Additionally, the absence of dominant R waves in V1 and Q waves in V6 suggests the presence of SVT rather than VT [1-6].

The patients with VT tend to older population with past medical history of coronary artery disease, stenting, myocardial fibrosis or healed myocardial scar tissue [3]. In the contrary, patients with SVT are younger population with relatively no cardiac risk factors. It is important to accurately identify and separate VT from SVT as improper diagnosis as management differs between clinical entities [1]. A wrongly assumed VT and subsequent treatment with Verapamil or Diltiazem can be detrimental as it precipitate acute severe hypotension, loss of consciousness and degeneration into ventricular fibrillation (VF) [7]. Studies indicate verapamil induces shortened re-entry cycle due to increased catecholamine secretion from its vasodilatory effect, thus increasing the risk of (VF) [8]. However, idiopathic fascicular VT (IFVT) is a rare form of narrow complex VT which has RBBB with either right or left axis deviation is very sensitive to Verapamil with no further recurrences [9].

The management of SVT depends on whether patient is hemodynamically stable or unstable [1]. Hemodynamic unstable patient needs synchronized cardioversion [1]. The treatment options in hemodynamically stable patients including medical and surgical options [1]. If there is pre-excitation in the sinus rhythm them EP study and catheter ablation would a first line choice for management [1]. If there is no preexcitation in the sinus rhythm, then medical management can be pursued [1]. In an acute setting, SVT of unknown mechanism can be controlled by IV beta-blockers (Esmolol & Metoprolol tartarate) or IV calcium channel blockers (Diltiazem or Verapamil) [1]. Options for long-term medical management of SVT of unknown mechanism include beta-blockers, calcium channel blockers (Verapamil, & Diltiazem), class IIa anti-arrhythmics (Flecainide & propafenone), class IIb anti-arrhythmics (Amiodarone, dofetilide and sotalol) and digoxin [1].

Conclusions

Younger age of presentation, lack of significant cardiac history including coronary artery disease and absence of myocardial fibrosis will increase the probability for SVT. Familiarizing with subtle EKG findings for precise identification of SVT is mandatory and should be emphasized for medical students, residents and clinicians in the clinical settings. That being said, EKG findings

including QRS duration less than 140 ms, absence of AV dissociation, shortfall of fusion complexes, lack of concordance of QRS morphology in precordial leads will increase the plausibility of SVT rather than VT. High clinical suspicion, and accurate EKG interpretation is mandatory efficient management of this re-entry tachycardia.

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