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Case Report
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ST Segment Elevation during Supraventricular Tachycardia (SVT) – An Enigma!

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Abstract

Atrioventricular nodal reentry tachycardia (AVNRT) represents one of the most common arrhythmias in emergency cardiology. It can occur in patients of all age groups and may occur in presence or absence of heart disease including coronary artery disease. During the periods of AVNRT, it is not uncommon to see ST segment depression and T wave changes. These, generally, persist transiently in the post conversion period and do not represent ischemia. We present a unique case report of ST segment elevation in the Left anterior descending artery (LAD) territory with T wave inversion during AVNRT. Coved ST segment and T wave inversion persisted till the patient was discharged from the hospital. CT angiography of coronaries done revealed 99% block of obtuse marginal (OM) after its origin.

Keywords: ST segment elevation; Atrioventricular nodal re-entry tachycardia; Supraventricular tachycardia

Introduction

Atrioventricular nodal re-entry tachycardia (AVNRT) is the most common type of reentrant supraventricular tachycardia (SVT). During AVNRT, it is not uncommon to see ST segment depression and T wave changes. However, ST elevation is a very uncommon feature and does not occur except due to underlying myocardial infarction. We report a rare case of ST elevation in the LAD territory with T wave inversion during AVNRT.

Case Presentation

A 77-year-old male patient was admitted to ICU with history of palpitation, breathlessness and chest discomfort of 1 h duration. He was hypertensive on regular medication (amlodipine 5 mg OD) and a non smoker. On admission to ICU, he was coherent, dyspnoeic, afebrile, had mild pallor, pulse was 140/minute, regular, BP was 140/70 mmHg, heart sounds were heard, chest was clear. Examination of other systems was normal. His ECG showed narrow complex supraventricular tachycardia with rate of 150/minute and absent P waves, suggestive of AVNRT. Also there was coved ST segment from V1 to V4 with symmetric T inversion from V2 to V6 (Figure 1). He was given carotid sinus massage (CSM), after which his arrythmia reverted to sinus rhythm. He felt comfortable and denied history of any chest pain. He was then started on tablet verapamil 80 mg tid. His ECG in sinus rhythm continued to show coved ST segment elevation from V1 to V4 and T inversion from V2 to V6 (Figure 2). Considering his age, risk factors for coronary artery disease (CAD) and ST elevation, he was also treated for acute coronary syndrome with aspirin, clopidogrel, LMWH, statin and sorbitrate. His blood investigations revealed normal reports except hemoglobin of 8.5 g/dL. CKMB on admission was 16 U/L and after 6 h was 132 U/L. His chest X-ray was normal. Echocardiography showed normal cardiac valves, LVEF of 64%, no regional wall motion abnormality (RWMA), concentric LVH and grade 1 diastolic dysfunction. His ECGs done subsequently on 5th day showed sinus rhythm, intermittent junctional rhythm, incomplete RBBB and persistence of ST-T changes as described earlier (Figure 3). Verapamil was stopped and ECG repeated on day seven showed restoration of sinus rhythm with incomplete RBBB and persistence of ST-T changes (Figure 4). He did not have old ECG record, but an ECG done in 2012, as a part of routine health check-up was reported as normal in his medical book. As the serial ECGs were suggestive of proximal left anterior descending artery (LAD) lesion, CT angiography of coronaries was done after 8 days of admission, to rule out the possibility of involvement of LAD. It showed normal LAD, RCA and 99% blockage of obtuse marginal (OM1) after its origin from the left circumflex (LCX).

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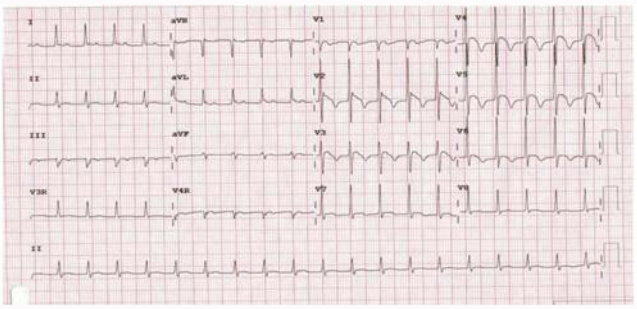

Figure 1: ECG on admission showing supraventricular tachycardia- AVNRT.

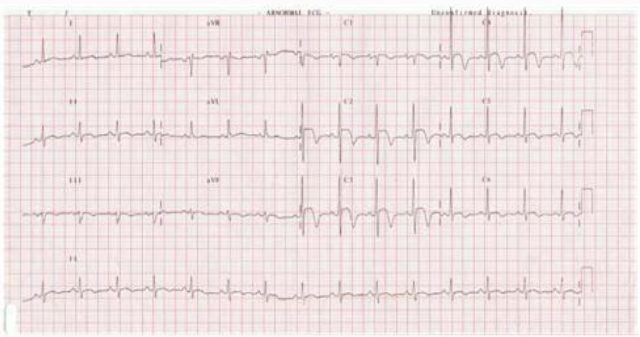

Figure 2: ECG on day 2 showing coved ST segment, prominent R wave with T inversion from V2 to V6.

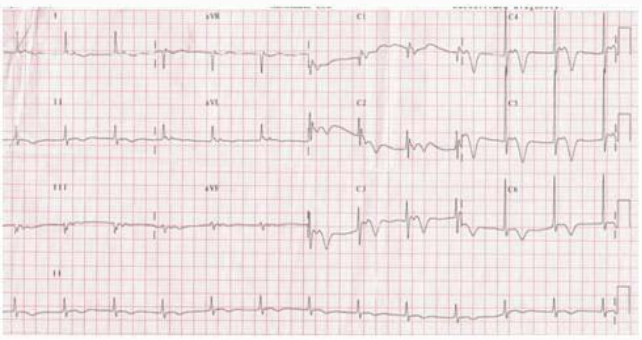

Figure 3: ECG on day 5 showing junctional rhythm and persistence of T inversion fromV2 to V6.

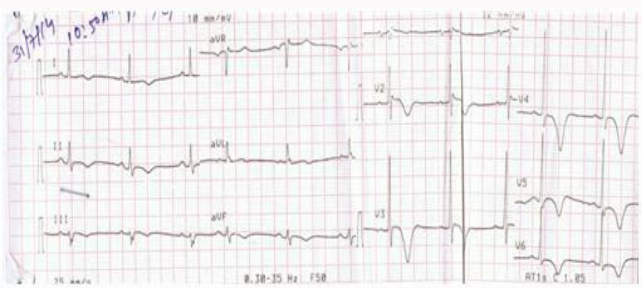

Figure 4: ECG on day 7 showing incomplete RBBB with symmetric T inversion from V 2 to V6. Shallow T wave inversions also noted in L I & II.

Discussion

AVNRT is a common arrhythmia presenting to the emergency department. It can occur in patients of all age groups and may occur in the presence or absence of heart disease, including coronary artery disease [1]. During the episodes of AVNRT and just after conversion, it is not uncommon to see ST segment and T wave changes. These may persist transiently in the post conversion period. These changes are nonspecific and seem to be a poor predictor of flow-limiting coronary stenosis [2,3]. There was also no observation of any relationship between the presence of significant ST depression with age, sex, mechanism or rate of tachycardia [4]. There have been many postulations as to why ST-T changes are observed. Slavich et al. [2] suggested it was related to coronary artery spasm and went on to propose that echocardiographic examination during these ST-T changes would be helpful [2].

However, in our case and cases published elsewhere in literature, no abnormality in wall motion or myocardial contraction was noted, just upon conversion. Nelson et al. [3] measured lactate levels in the coronary sinus during SVT episodes associated with ST depression. There was no significant increase in lactate production in these patients compared to control group (persons with SVT who were known to have underlying CAD), where increased levels were noted. Troponin is now heavily relied upon in acute coronary syndrome and risk stratification techniques for assessment of chest pain [5-7]. Redfearn et al. [5] demonstrated that fast paroxysmal arrhythmias can cause elevation of troponin levels, despite patients having a normal coronary angiogram. The exact mechanism of injury, however, remains unclear, but it may point to some degree of myocardial injury during the episode of tachycardia [7,8]. It has also been postulated to increased demand during a relatively long period of tachycardia, combined with a reduction of oxygen supply to myocardium due to shortened diastole during the tachycardia. No correlation has been found between the extent of marker increase and the duration and rate of tachycardia. In our patient, elevated CKMB may be due to myocardial ischemia because of the rate-limiting flow during tachycardia (99% stenosis of OM at its origin).

Another study found that repolarisation changes during SVT initiation were caused by concurrent haemodynamic changes. Patients with shorter tachycardia cycle length, elevated systolic blood pressure (SBP) before tachycardia induction and greater reduction of SBP, had a higher incidence of repolarisation changes [9]. Lee et al. [10] observed that ST segment depression of ≥ 1 mm was more common in AVRT than AVNRT (79.9% vs. 27.8%, P<0.001) and T inversion occurred more frequently in AVRT than AVNRT (30.3% vs. 7.4%, P<0.01 %) can thus be used complementally to the classic ECG parameters in the differential diagnosis of SVT. ST segment elevation during the narrow QRS tachycardia has been described in a VR and it favors the atrioventricular reentry through an accessory pathway as the mechanism of tachycardia [11]. In a study by Ho et al. [12] the prevalence of a VR ST segment elevation was 71% for AVRT, 31% for AVNRT and 16% for atrial tachycardia and thus, ST segment elevation in a VR is used to identify the mechanism of tachycardia. Obtuse marginal (OM1) is the branch of left circumflex artery (LCX), which is given off by the proximal or initial portion of the LCX at the posterior interventricular sulcus, travelling along the left margin of the heart towards the apex and supplies the lateral wall of the left ventricle along with LAD. Occlusion of OM can remain electrocardiographically silent or produce ST-T changes in leads L1, a VL, V5 and V6. Our patient had 99% occlusion of OM but had ST elevation (from V2 to V6) and T wave changes during tachycardia suggestive of LAD occlusion and not OM occlusion. His LAD was, however, normal in CT angiogram of coronaries. ST elevation regressed to near normal after 48 h after the termination of thear

rhythmia. However, coved ST and symmetrical T wave inversion persisted till he was discharged (10 days). Subsequently, patient was lost to follow up.

There are case reports of ST segment depression and T wave changes during SVT and normalization of these changes after reversal of SVT to sinus rhythm [1]. A thorough search of literature did not reveal any case report describing ST elevation during the SVT and persistence of ST-T changes after conversion to sinus rhythm.

To conclude, ST-T alterations are commonly seen during SVT, ST depression being more common than ST elevation. ST depression is not an evidence for the presence of underlying ischemia and thus does not warrant treatment. It must be interpreted in the clinical context. ST elevation, however, may reflect rate related critical flow due to underlying coronary artery disease but it poorly correlates with the site of the culprit vessel as in this case. Hence, ST elevation during SVT should make one suspect underlying coronary artery disease and needs further evaluation. This, however, being an isolated case report, further studies are required to substantiate the observation.

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