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An Interesting Uncanny Clinical Presentation of Acute Myocardial Infarction

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ABSTRACT: Myocardial infarction can present to the emergency room in the form of various complications. Anterior wall MI is notorious for producing mechanical complications like ventricular free wall rupture, ventricular septum rupture, and papillary muscle rupture. Inferior wall MI is usually associated with conduction abnormalities like heart blocks. Here, reperfusion of the myocardium and concurrent management of the complication should go hand in hand. We had a patient presenting to the ER with an ECG that was confusing between a PSVT and VT which when reverted revealed an anterior wall MI. The patient was salvaged with prompt revascularization.

KEYWORDS: Myocardial Infarction, PSVT, Ventricular Tachycardia.

INTRODUCTION: The definition of MI, whether it be STEMI or NSTEMI, according to the 2018 joint task force of the European Society of Cardiology (ESC), American College of Cardiology Foundation (ACCF), American Heart Association (AHA), and World Health Federation (WHF) is the presence of acute myocardial injury detected by abnormal cardiac biomarkers in the context of acute myocardial ischemia evidence [1]. Acute myocardial infarction is known to cause electrical conduction abnormalities. They could be brought on by ischemia/infarction impacting the conduction system or autonomic instability. Bradycardia is the most frequent arrhythmic outcome; it may or may not be symptomatic. In this situation, if complete heart block with a delayed escape rhythm is not identified and treated right away, it could be fatal [2]. As far as tachyarrhythmias are concerned, other than atrial fibrillation (AF) or atrial flutter, supraventricular arrhythmias are generally rare during the periinfarction phase. Any tachydysrhythmia that originates from above the level of the Bundle of His is referred to as a supraventricular tachycardia (SVT), which includes regular atrial, irregular atrial, and regular atrioventricular tachycardias. It is frequently used interchangeably with an SVT variant known as AV nodal re-entry tachycardia (AVNRT). The ECG will show a narrow complex tachycardia if there is no abnormal conduction (such as bundle branch block) [3]. But in PSVT with an abnormal conduction, the QRS complex is often broad and hence becomes difficult to distinguish from a VT. Less than 10% of individuals with an acute MI develop paroxysmal supraventricular tachycardia (PSVT), yet it may call for immediate treatment because of a high ventricular rate [4].

CASE REPORT

A 69-year-old male, known case of systemic hypertension on irregular medications and coronary artery disease on treatment with aspirin, atorvastatin and trimetazidine for 5 years presented to the emergency room with history of chest pain for 2-3 days. He was complaining of diffuse heaviness of chest associated with dyspnoea and palpitations. On arrival, patient was sweating profusely and was restless. Vitals were within normal limits except for pulse rate of 220/min, which was regular on palpation. Chest examination revealed bilateral fine basal crepitations. ECG was done that was suggestive of a regular broad complex tachyarrhythmia with left axis deviation, absent P wave and with qRBBB pattern (Figure 1). On initial look, the ECG appeared like a PSVT with qRBBB. But, on a second look, it appeared more like a ventricular tachycardia.

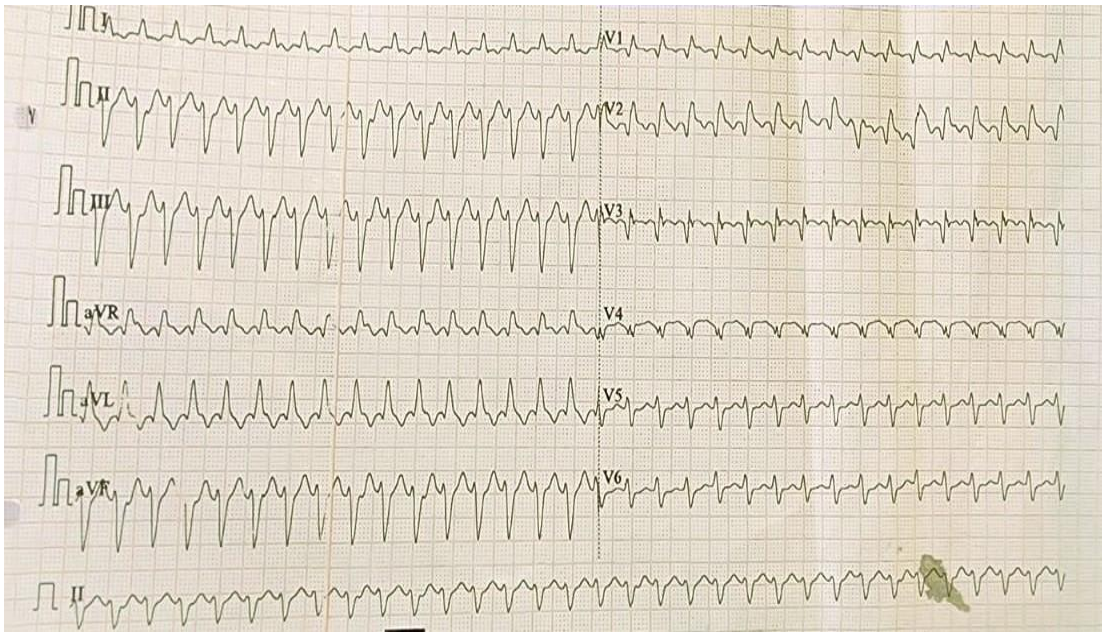

FIGURE 1: Broad complex regular tachyarrhythmia with qRBBB pattern

In the background of a confusing picture between VT and PSVT and the patient being a case of tachyarrhythmia with heart failure (unstable tachycardia), patient was decided to manage with electrical cardioversion. ECG done after cardioversion revealed an ECG with regular rhythm, normal axis, absent P wave and narrow QRS (Junctional Rhythm). Q waves were identified in leads V1-V3 and T wave inversions in inferior leads and V4-V6 (Figure2). This ECG was enlightening as it showed a change in axis from the previous ECG, which was more indicative of the previous ECG being a VT. The patient was symptomatically better but was maintaining blood pressure only on ionotropic support and oxygen saturation on 10 L oxygen via facemask.

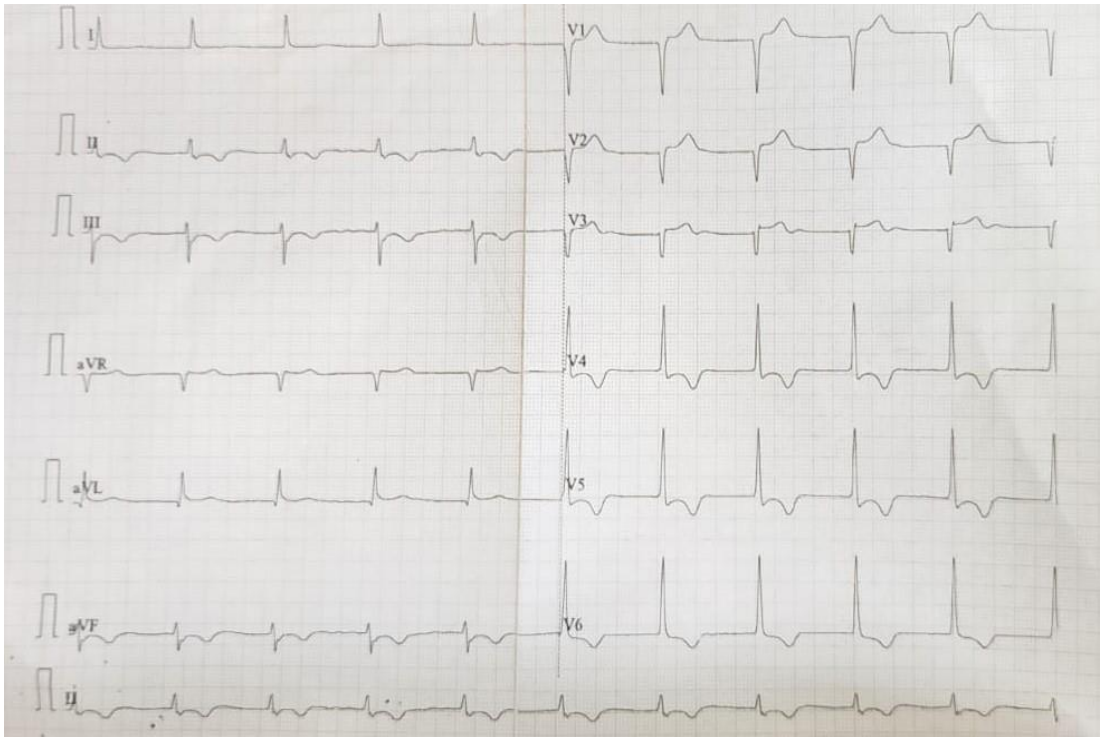

FIGURE 2: Narrow complex tracing with regular rhythm and absent P waves

Patient was admitted in the ICU and ECG was repeated in ICU after an hour of cardioversion which now revealed ST segment elevations and q waves in precordial leads with reciprocal changes in inferior leads (Figure3). Now things started falling in place. We now realised this was a case of ANTERIOR WALL MYOCARDIAL INFARCTION THAT DECIDED TO WEAR THE MASK OF VT TO COME TO THE ER!

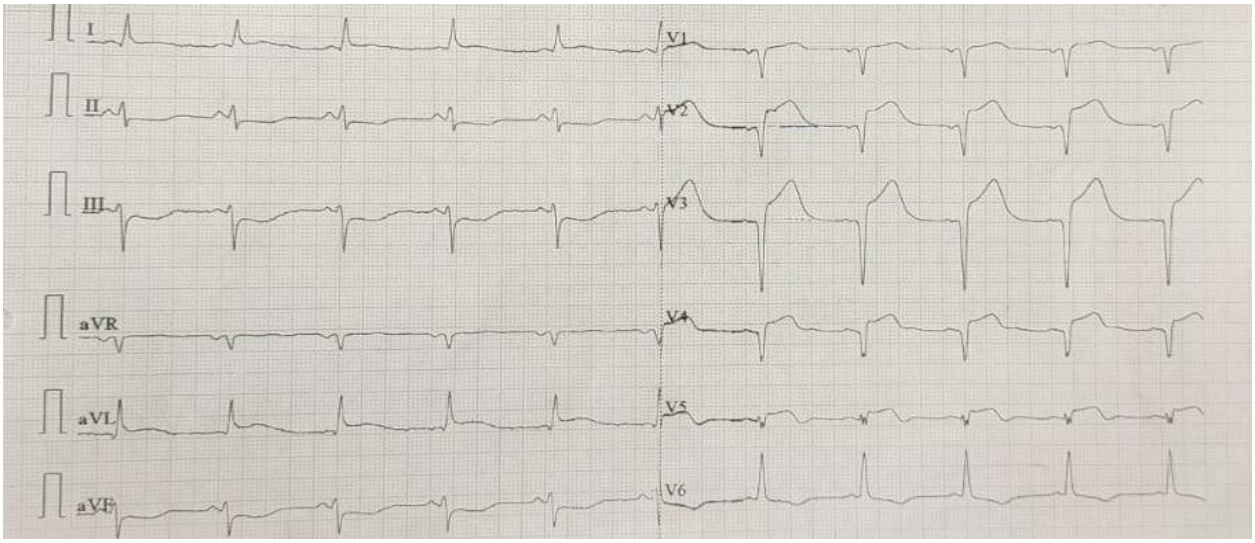

Figure 3: Anterior wall ST segment elevation with reciprocal changes

In view of unavailability of facility for PCI, he was thrombolysed with streptokinase (1.5 million IU) and a repeat ECG was done after 90 minutes which revealed more than 70 % ST resolution (Figure4).

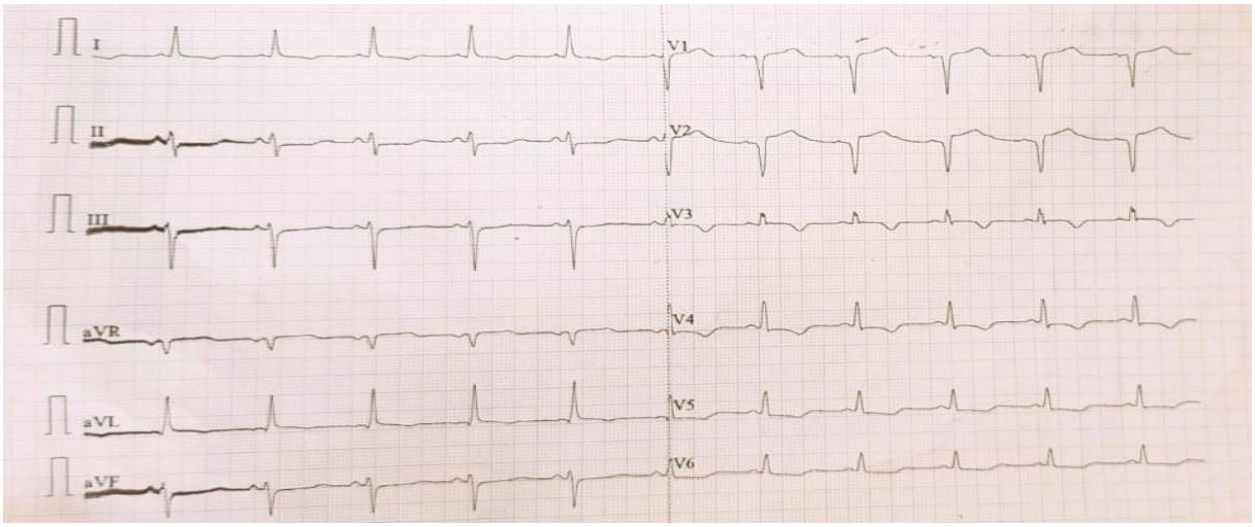

Figure 4: ECG 90 minutes post thrombolysis

Following thrombolysis, shock improved and inotropes were tapered and stopped over the next 24 hours. Further investigation reports were suggestive of acute kidney injury and ischemic liver injury (Table 1). Initial serum creatinine was 6.22 mg/dl and urea were 176 mg/dl. Liver function test was suggestive of markedly elevated liver enzymes (SGOT=4779 and SGPT= 2007). High sensitivity troponin I was also markedly elevated to 9.5 nanogram/ml (Normal= less than 0.034 nanogram/dl). BNP levels were also raised to 2029 picogram/ml (Normal= less than 300 picogram/ml). Thyroid function test was also sent to rule of PSVT due to thyrotoxicosis, but came out to be normal. Conservative management was then initiated with titrated fluid therapy, dual antiplatelets, statins, betablockers and angiotensin converting enzyme inhibitors in appropriate doses with gradual tapering of inotropes. Echocardiography was done which was suggestive of concentric left ventricular hypertrophy and a LV ejection fraction of 45% with no significant RWMA. Over the course of hospital stay, shock improved and patient was discharged on oral antiplatelets, statins, beta-blockers and angiotensin converting enzyme inhibitors on appropriate doses.

Table 1: Serial investigation results

	SGOT (U/L)	SGPT (U/L)	Urea (mg/dl)	Creatinine (mg/dl)	Troponin I (ng/ml)
Day 1	4779	2007	176	6.2	9.5
Day 2	2080	1467	181	5.3	
Day 3	871	944	126	4.2	
Day 4	126	327	67	1.9	1.7

DISCUSSION

Histopathologically, coagulative necrosis of the myocardium is what defines acute MI. Within 30 to 40 minutes of prolonged ischaemia in a non-reperfused MI, this is observed; the alterations are only evident at a resolution of electron microscopy. The scar starts to form at two weeks and moves from the edges to the core by the second month. Reperfusion attempts have the ability to change this process [5]. Various arrythmias are associated with myocardial infarctions. Since more than half of the population receives their sinus node's supply through the RCA, sinus bradycardia is commonly observed in inferior wall infractions [4]. Between 1994 and 1997, 297,832 patients with acute MI were hospitalised to hospitals in the United States, and the National Registry of Myocardial Infarction 2 (NRMI-2) assessed the incidence of bundle branch block in these patients: On the first ECG, 6.7% of patients had an LBBB and 6.2% had an RBBB [6]. Reviewing almost 76,000 patients with ST-elevation MI (STEMI) who took part in four large randomised trials yielded the biggest experience with high degree AV block in the fibrinolytic period, with an overall incidence of 6.9 percent (9.8 percent with inferior MI and 3.2 percent with anterior MI) [7]. In about 40% of cases of acute MI, sinus tachycardia will also manifest; this is typically indicative of sympathetic activation. A considerable region of infarction and reduction in left ventricular function are typically linked to persistent sinus tachycardia [8]. Atrial fibrillations, the most prevalent type of atrial tachycardia, have a prevalence of 6-20% during the periinfarction phase and are also linked to ischemia and infarctions. In myocardial infarction, ventricular arrhythmias ranging from VPCs to VF are also frequent. The incidence of different ventricular arrhythmias in MI was determined to be as follows prior to the advent of fibrinolytics: 3–39% VT, 4–20% VF, and 10–93% VPCs [9-11]. Paroxysmal Supraventricular Tachycardias (PSVT) are one of the least common arrythmias that an ischemia or infarction can present with accounting for a maximum incidence of 10% [4]. 20% of supraventricular tachycardias respond to initial measures to increase the vagal tone like vagal massage and Valsalva manoeuvre. 66.67% of cases are reverted by a first dose of adenosine (6 mg). If not reverted, adenosine is repeated again to a maximum of 30 mg, conventionally given as 12mg twice. Other second line medications include beta blockers, calcium channel blockers and amiodarone. As per the AHA algorithm for tachyarrhythmias, any unstable tachycardias are to be managed by synchronised DC cardioversion.

In our case the initial ECG was confusing between PSVT with qRBBB and VT. In real life scenarios this could be differentiated using the BRUGADA CRITERIA. In our case, the ECG was fulfilling all the initial 3 criteria for PSVT, but not the last one and hence it was identified as a case of VT.

CONCLUSION

In this case, our patient was a known case of coronary artery disease who developed VT which on reverting with electrical cardioversion revealed a junctional rhythm and then progressed to anterior wall MI. This kind of electrical and conduction abnormalities are more common in inferior wall MI and mechanical complication like free wall rupture are more common in anterior wall MI. But in this case, patient developed a tachyarrhythmia in spite of having anterior wall MI. Also, very often it becomes difficult to differentiate between a PSVT with bundle branch block and a VT due to the presence of broad QRS in both. Algorithms like the Brugada's algorithm and Vereckei Algorithm can be of use in such situations. In situations of confusion between an SVT and VT, it's always recommended to proceed with electrical cardioversion rather than pharmacological cardioversion.

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