Pseudoanterior wall myocardial infarction: a diagnostic pitfall

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DESCRIPTION

A man in his 50s presented to the emergency department (ED) with 6 hours of severe, acute anterior chest discomfort radiating to the left shoulder. There was no associated history of diaphoresis, loss of consciousness, nausea or vomiting. He had no known comorbidities and was not on any medication. On examination, he was alert with the following vitals: pulse rate: 92/min, blood pressure: 146/98 mm Hg, respiratory rate: 18/min and SpO2 of 98% on room air. There were no added breath sounds or murmurs on auscultation of the chest and precordium, respectively. The initial 12-lead ECG (figure 1) revealed normal sinus rhythm at 94 bpm, with a right bundle branch block (RBBB) morphology, and a QRS complex width of around 160 ms. There was a convex upwards ST elevation (STE) of 5 mm in lead V1. STE was also noted in leads V2–V5 after recognising the J-point carefully (figure 2). Minimal STE was also noted in inferior leads II and III. Initial high-sensitivity troponin I was positive. Cardiac contractility was adequate on bedside echocardiography, and no regional wall motion abnormality (RWMA) was noted. The right ventricle (RV) diameter was more than the left ventricular (LV) diameter on eye balling. RV-free wall appeared hypokinetic.

The ECG was initially interpreted as anterior wall ST-elevation myocardial infarction (STEMI). A qRBBB pattern on the ECG in the setting of anterior STEMI is suggestive of proximal left anterior descending (LAD) artery occlusion.[1 2] However, considering the prominent STE in V1, right precordial leads were obtained which revealed around 1 mm STE in V4R (figure 3). A diagnosis of RV myocardial infarction (RVMI) was, thus, reached. The patient was administered antithrombotics and intravenous opioids for chest pain. The patient was thrombolysed in the ED with intravenous streptokinase with complete resolution of his chest pain. A repeat ECG revealed ≥50% resolution of STE (figure 4). The coronary angiography performed the next day revealed 95%–99% stenosis of the right coronary artery (RCA) and calcific LAD artery with 60%–70% stenosis (figure 5). Revascularisation was not performed due to monetary constraints. A formal 2D-echocardiography showed no evidence of RWMA. LV ejection fraction was 60%, E>A, all valves and chambers were normal. No clots, vegetations or pericardial effusion were noted. Interventricular septum (IVSd)-9 mm; LV internal diameter in systole and diastole (LVIDd and LVIDs respectively)-36mm and 28mm; Left

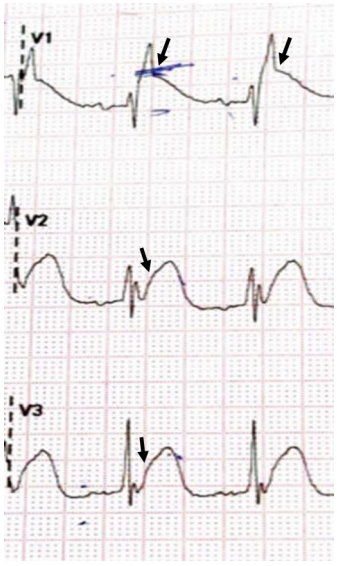

Figure 2 Zoomed image of the initial ECG obtained in emergency department (ED). Arrows point towards J-point in various precordial leads. ST-elevation is maximum in lead V1 (5 mm). Lesser degrees of ST elevations present in other precordial leads.

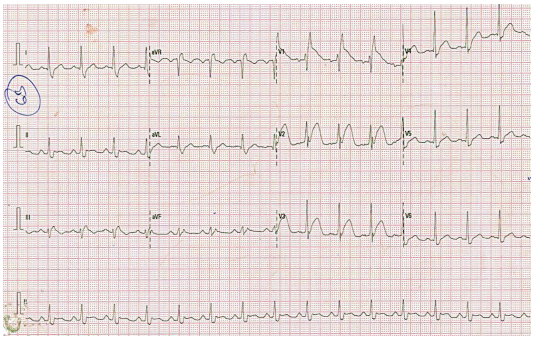

Figure 1 Initial 12-lead ECG obtained in the emergency department.

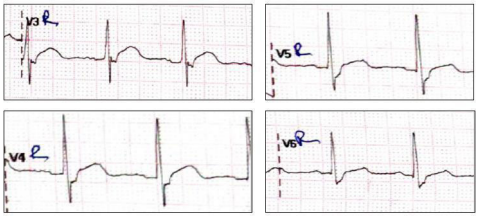

Figure 3 Right precordial leads obtained on arrival in the emergency department (ED). Note convex upwards ST elevations present in V3R and V4R.

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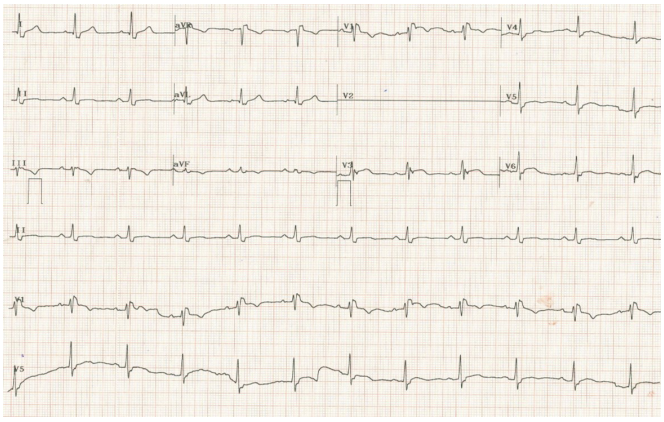

Figure 4 ECG obtained after thrombolysis with streptokinase in emergency department (ED). The ECG reveals a significant resolution of initial ST elevations after thrombolysis. Lead V2 was recorded as isoelectric due to loose lead placement.

atrium (LA-30mm; Aortic valve (AO)-26mm. The patient was discharged symptom free after 5 days of ICU stay.

RVMI is associated with higher prevalence of complications along with increased morbidity and mortality.[3 4] RCA supplies the LV (inferior wall) as well as the RV. Hence, RVMI is usually seen with inferior wall STEMI. When RVMI is suspected, an STE of ≥1 mm in V4R is diagnostic of RVMI.[4 5] However, isolated RVMI may be seen with RCA occlusion. RVMI may manifest with STE in anterior leads V1–V5,[3 6 7] thereby mimicking anterior STEMI ('pseudoanterior wall MI' pattern). Leads V1 and V2 are placed close to the sternal border and overlie the RV. Hence, these leads pick the STE of RVMI better than others.[8] Thus, STE is more in V1–V2 than in other precordial leads. This is in contrast to the anterior STEMI, where STE is more prominent in leads V3–V5.[3 6] This is a subtle difference which may be easily missed, and recognising it may help in making the correct diagnosis of RVMI in the ED.

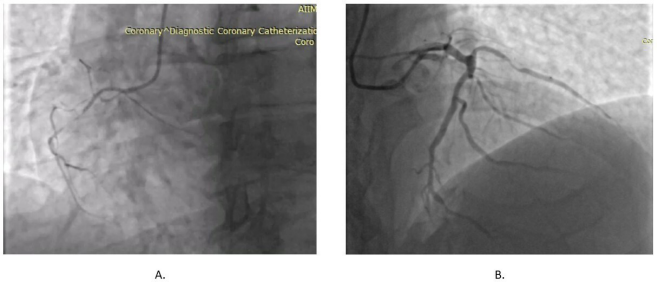

Figure 5 Cardiac catheterisation image showing 95%–99% stenosis of right coronary artery (A) and a calcific left anterior descending artery with 60%–70% stenosis (B).

Learning points

- Isolated right ventricular myocardial infarction (RVMI) is an uncommon entity.
- RVMI may present with ST elevations in anterior precordial leads, referred to as pseudoanterior wall MI pattern.
- Anterior wall MI has more prominent ST elevations in leads V3–V5 as compared with leads V1 or V2. However, when the ST elevations in V1/V2 are more than in V3–V5, emergency physicians must consider the diagnosis of RVMI.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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