

Case Report

“Isolated” Right Ventricular Myocardial Infarction, Mimicking Anterior Myocardial Infarction - Diagnosis and Potential Mechanisms for the Electrocardiographic Changes

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ABSTRACT

The association of right ventricular infarction (RVI) with inferior myocardial infarction is well known and not uncommon, but “isolated” RVI is rare. Its presentation on electrocardiography (ECG) can further confuse the diagnosis as it can look like an anterior myocardial

infarction on the ECG and a wrong diagnosis can be harmful as the management is different in the two conditions. The ECG features in such a presentation and the possible mechanisms underlying these changes are discussed.

KEYWORDS: anterior myocardial infarction, mimic, “Isolated”, right ventricular infarction

INTRODUCTION

Coronary Care Unit (CCU) is the mainstay of any full-fledged modern general hospital. The awareness of unusual occurrences is as important as the detailed knowledge of common conditions. Electrocardiography (ECG) still remains the pivotal investigation in the diagnosis of coronary artery disease (CAD). The site of a myocardial infarction remains a subject of discussion in a substantial number of patients admitted to any CCU necessitating further investigations to determine the exact location. One such situation is the presence of ST-segment elevation in left precordial leads along with ST segment elevation in the right precordial leads particularly V_{4R}. Is this an anterior myocardial infarction (AMI) with a right ventricular infarction (RVI) that is not a known association, or is it an “isolated” right ventricular infarction mimicking an AMI? We are reporting one such case. It is important to recognize the difference as the management protocols vary with the site of the infarction. Discussion is based on the clinical suspicion for the diagnosis and the potential mechanisms underlying the ECG changes encountered.

CASE REPORT

A 54-year-old-gentleman was admitted from the emergency department with sudden, severe, retro-sternal chest pain of more than three hours duration that was radiating to the back and to both arms. The pain was associated with sweating and vomiting. There was no past history of any exertional chest pain. He was a known Type II diabetic for the past four years and has been

smoking around 10 cigarettes per day for the past 40 years. On clinical examination, the patient was a medium-built person, fully conscious and oriented. The heart rate was 60/minute and the blood pressure was 140/80 mmHg. The jugular venous pressure was not elevated and the respiratory rate was 20/minute. There was no pedal edema. Examination of the heart was unremarkable and the lungs were clear. There was no organomegaly on abdominal examination. Central nervous system was normal and there were no signs of peripheral neuropathy. Fundus examination showed no retinopathy.

An ECG done in the CCU immediately upon admission showed ST elevation with inverted T waves in the precordial leads V₁ – V₄. A routine right sided precordial lead (V_{4R}), done along with the usual 12 lead ECG as part of our CCU protocol, showed a ST elevation of more than 3 mV (Fig. 1). The next serial ECG done 6 hours later showed the precordial lead changes more clearly with more ST segment elevation in leads V₁ to V₃ and sagging of ST segment and T wave inversion in leads II, III, and aVF (Fig. 2). Cardiac markers (CK-MB mass and Cardiac Troponin I) were significantly elevated, thus confirming an acute MI.

A transthoracic echocardiography showed normal movements of the interventricular septum, left ventricular posterior wall and the inferior wall with a left ventricular ejection fraction of 65%. The right ventricular free wall had a distorted shape with hypokinesia and hyperechogenicity best seen in the sub-sternal view (Fig. 3). There was no tricuspid or mitral regurgitation but a physiological

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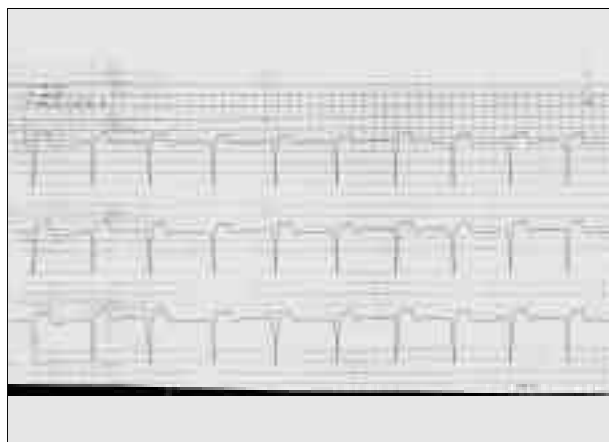


Fig. 1: The ECG taken from the right precordial V_1 position shows the marked ST elevation of more than 3mV, suggestive of a right ventricular infarction.



Fig. 2: The 12 lead ECG shows the presence of ST elevation in leads V_1 - V_3 and is strongly suggestive of an acute anterior myocardial infarction.

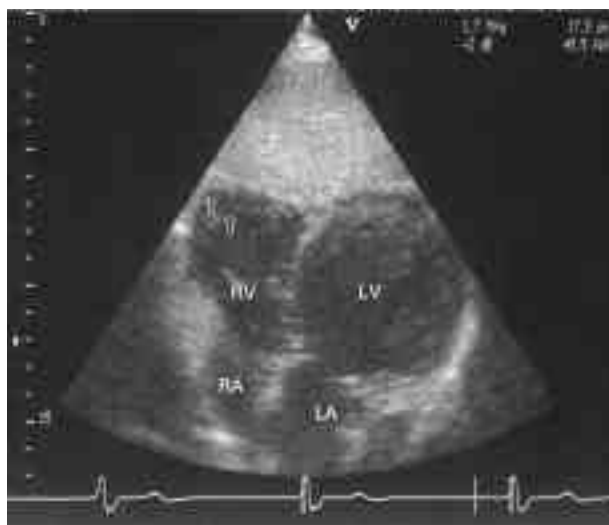


Fig. 3: The 2-D Echocardiography seen from the sub-sternal view shows a dilated and distorted right ventricular cavity with a hyperechoic RV wall.

pulmonary regurgitation was seen. The rest of the colour doppler flows were normal.

A Tc99m Tetrofosmin rest myocardial perfusion study and a Tc99m pyrophosphate infarct avid cardiac study were inconclusive and could not confirm or rule out a right ventricular infarction. There was no evidence of any other site of MI in the left ventricular wall.

The patient was thrombolysed with tissue plasminogen activator followed by heparin infusion, aspirin, and betablockers. With the possibility of an associated additional RVI, no nitrates were used. The patient had no recurrence of chest pain. He had an uncomplicated and uneventful hospital stay. He was discharged on the 10th day as per our hospital protocol for acute myocardial infarction.

DISCUSSION

Right ventricular infarction occurs in 40-50% of patients with acute inferior MI and its presence can

have important clinical, haemodynamic, and prognostic implications^[1-3]. It is almost always due to occlusion of the right coronary artery at or proximal to its right ventricular marginal branch^[4]. The extent of the infarction depends on, whether the occlusion occurred before the RV free-wall and RV marginal branches, whether collateral flow from LAD is present, and on the extent of blood flow through the Thebesian veins^[5].

The classic constellation of hypotension, elevated jugular venous pressure (JVP) with clear lung fields and no dyspnea^[1] is highly specific but insensitive for RV infarction. The finding on examination of an elevated JVP > 8 cm of water and Kussmaul's sign are sensitive and specific for haemodynamically significant RV infarction^[6].

The first ECG diagnosis of RVI was reported in 1976 by Erhardt et al who showed that ST segment elevation in the right precordial leads V_3R and V_4R was a reliable sign of RVI as verified by autopsy^[7]. The sensitivity and the specificity of ST elevation in the right-sided precordial leads, especially lead V_4R , for the diagnosis of RVI are well over 90%. These ST changes tend to be most prominent in the early hours of acute MI and then dissipate over the subsequent 24 hours or so^[4]. An ST segment elevation of > 0.5 mm in lead V_4R is highly sensitive for RVI^[8].

In 1985 ST segment elevation in lead V_1 was reported in patients with acute inferior wall infarction associated with RVI^[9]. These precordial ST changes could be easily mistaken for signs of acute anterior MI, especially when the ST elevation extends as far as lead V_5 and V_6 . These findings were noted only in a minority of patients (< 10%) despite the 40 % to 50% prevalence of RVI in patients with inferior infarction^[4].

Based on the principles of reciprocal ECG changes in ischaemia and infarction, the ST

segment changes in the anterior leads can be understood. The direction of precordial ST segments changes in acute inferior MI is subjected to two opposing influences: RVI tends to elevate the ST segment, and inferior and posterolateral infarctions tends to produce reciprocal ST depression. Thus, in patients with acute inferior MI with concomitant RVI, the tendency for RV current of injury to elevate the precordial ST segment would be opposed by the reciprocal effects of inferoposterior current of injury and vice versa. The left-precordial ST segment elevation would occur only when RV involvement predominates and the inferoposterior injury is small^[4]. These problematic ECGs have been studied along with necropsy^[10], echocardiography, cardiac nuclear imaging^[2], coronary angiographies and during angioplasty procedures^[11-14].

The following initial and evolving ECG patterns to guide the differential diagnosis between RVI and AMI have been suggested. When the magnitude of ST segment elevation in $V_1 - V_5$ decreases from right to left and when the progressive regression of ST segment occurs without the development of Q waves the ECG is suggestive of RVI^[2,10]. However these changes are not consistent in all the reports and evolutionary QS patterns have been observed. In all of these reports there was a consistency that there was no anterior wall motion abnormality on echocardiography^[2]. Another criterion used is to compare the ST elevation of V_4R to V_3 . It is seen that in RVI the ST elevation in V_4R is more than the ST elevation in V_3 . Also by contrast the ST elevation in an anterior MI is always more than the ST elevation in V_4R , if any, and is less than the $V_3 - V_4$ elevations^[8].

In our patient, the ST segment elevation from V_1 to V_4 progressively decreased and the V_4R showed a greater ST elevation than the V_3 . There was a reduction in the ST elevation in the serial ECGs and no QS pattern developed. The R wave, although dwarfed, remained in all the precordial leads, with a minor leftward shift of the R wave to S wave transition in the precordial leads.

In conclusion, acute occlusions of small, nondominant right coronary arteries (RCA), occlusions of proximal RCA before the outlet of ventricular branches, particularly in patients with previous inferior MI and/or with left dominance or co-dominance, can cause RV lesions that prevail over the posteroinferior acute ischaemia. Such lesions may also occur with the loss of ventricular branches during angioplasty of the proximal or middle RCA and in patients with severe lesions preventing collateral circulation^[15]. All these lesions may give rise to "isolated" RVI and infarction prevailing in the RV causing ST segment elevation in the anterior chest leads.

In summary, a diagnosis of RVI has to be considered when such an ECG pattern is observed because of the specific in-hospital complications, high in-hospital mortality rates, and the therapeutic consequences^[16].

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