

A Case of Unusual Electrocardiographic Presentation of Right Ventricular Myocardial Infarction

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Abstract

This is a case of an unusual electrocardiographic manifestation of a patient with right ventricular (RV) myocardial infarction occurring in association with left ventricular inferoposterior myocardial infarction. There was massive ST-segment elevation in the precordial leads resembling that of an anterior myocardial infarct in addition to the ST-segment elevation seen normally in right ventricular leads. Two-dimensional echocardiography confirmed right ventricular hypokinesia and coronary angiography revealed single-vessel coronary artery disease involving the right coronary artery. It served to remind us that the presence of diffuse and massive ST-segment elevation in the precordial leads in a patient with inferior myocardial infarction may indicate simultaneous RV infarction and warrants further confirmatory tests.

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Introduction

The diagnosis of right ventricular (RV) myocardial infarction has both prognostic and therapeutic importance. RV infarct occurs in variable proportion (19% to 51%) of patients with acute inferior myocardial infarction¹ and carries a higher risk of in-hospital mortality rate and complications.⁴ Among the various diagnostic tools, electrocardiography is the most readily available and inexpensive technique. First described by Erhardt, the presence of ST-segment elevation, Q waves, or both, in right precordial leads (V3R to V6R), is found to be highly sensitive and specific in the diagnosis of RV infarction.^{5,6} However, RV infarct can also cause changes in the precordial leads in the form of ST-segment elevation. We report a case of such an unusual electrocardiographic presentation in our hospital.

Case Report

NCK, a 50-year-old Chinese man, was admitted with a 2-hour history of severe chest discomfort associated with diaphoresis and nausea. He had cardiovascular risk factors of hypertension, chronic smoking and hyperlipidaemia. He had antecedent anginal symptoms for 3 years prior to his present attack. Physical examination on admission revealed the patient to be in distress

with cool and clammy skin. He was hypotensive with systolic blood pressure of 78 mmHg and bradycardic with pulse rate of 50 per minute. His resting 12-lead electrocardiogram (ECG) at the Accident and Emergency Department showed massive ST-segment elevation in precordial leads of V1 to V4 and inferior leads of II, III and aVF (Fig. 1). The maximum magnitude of ST-segment elevation in precordial leads was 19 mm in V4 (measured 60 msec from the J-point) and that in the inferior leads was 2 mm in lead II. Within minutes of transfer to the Coronary Care unit, his ECG showed disappearance of the ST-segment elevation in the precordial leads. There was instead ST-segment depression, more in leads V2 and V3 than in V1. The inferior leads now showed greater ST-segment elevation of maximal magnitude of 5 mm in lead III (Fig. 2). Simultaneous RV precordial lead recordings showed ST-segment elevation of more than 1 mm (Fig. 3). The ECG diagnosis was that of an acute inferoposterior and right ventricular myocardial infarction. Bedside two-dimensional echocardiogram showed hypokinesia of the left ventricular (LV) inferior and posterior wall segments. The RV was also mildly hypokinetic. The LV anterior wall was normal. Upon successful thrombolysis, the depressed ST-segment in the precordial leads normalised. Six hours

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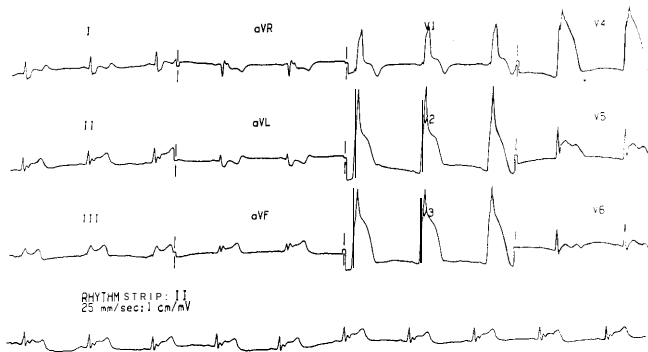


Fig. 1. 12-lead electrocardiogram in the Emergency Department showing marked ST-segment elevation in the precordial and inferior leads

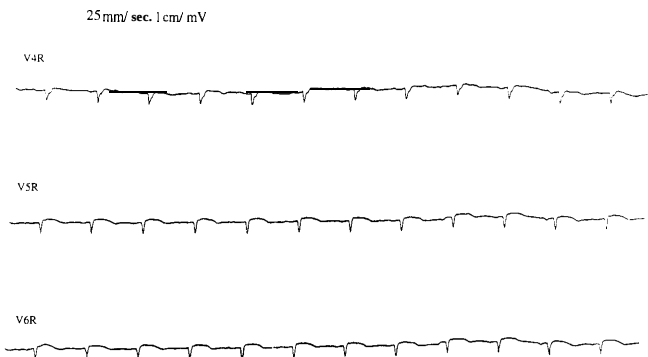


Fig. 3. Right ventricular leads showing acute right ventricular myocardial infarction.

later, there were fully evolved inferoposterior transmural myocardial infarction changes seen on the ECG (Fig. 4). There was no evolving Q-wave seen in the anterior leads with progression of the myocardial infarction. The peak creatine kinase assayed was 339 U/L and the MB fraction was 18 U/L.

The patient underwent cardiac catheterisation on day 6 which showed a significant tubular stenosis in the proximal right coronary artery, at the site of a branching medium-sized anterior right ventricular vessel (Fig. 5). The left coronary arteries were normal (Fig. 6). The LV inferior and posterobasal wall segments were moderately hypokinetic. Ad hoc coronary angioplasty with elective placement of a 3.5 mm Johnson & Johnson Palmaz-Schatz coronary stent was successfully performed (Fig. 7). Following which, the patient was put on post-stent medical therapy of warfarin and aspirin. His convalescence was uneventful and he was discharged 3 days later.

Discussion

Electrocardiographic changes accompanying RV infarction often occurs transiently and most would have resolved within 10 hours of the onset of the symptom. The use of 1-mm ST-segment elevation in V4R remains the most predictive ECG changes for RV infarct. How-

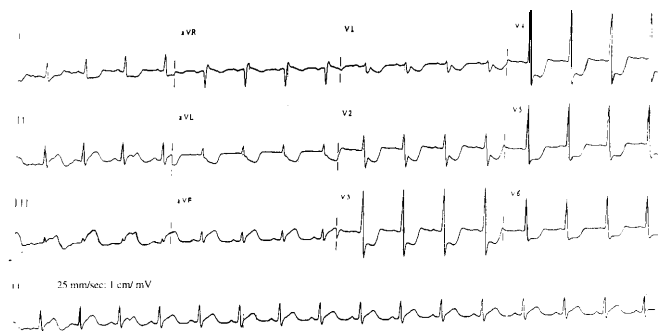


Fig. 2. 11-lead electrocardiogram in Coronary Care Unit showing acute inferoposterior myocardial infarction.



Fig. 4. 12-lead electrocardiogram showing fully evolved inferoposterior transmural myocardial infarction.

ever, it is well known that this loses specificity in the presence of any heart disease that induce ST-segment elevation in lead V1, for example, pericardial diseases, acute pulmonary embolism, left anterior fascicular block, and acute anterior myocardial infarction. Ten per cent of anterior myocardial infarction has concomitant right ventricular involvement.

The classically adopted ECG criteria for RV infarct is that described by Erhardt which is ST-segment elevation in the right precordial leads. The incidence of such ST-segment elevation in RV infarct varies between 26.3% reported by Geft et al⁷ and 72.7% by Chou et al.⁸ RV infarct can also cause ST-segment elevation in the precordial leads although this is a more common electrocardiographic finding in LV anterior wall injury involving the left anterior descending artery.^{7,9,10} In our reported case, the ECG recordings corresponded with the diagnosis of RV infarct that was confirmed on echocardiography and coronary angiography.

The association between ST-segment elevation in leads V1 and V2 and RV infarction was first noted by Meyers et al in 1949¹¹ and by Levy and Hyman in 1950.¹² The characteristic ECG precordial lead ST-segment changes associated with RV infarction are (a) progressive reduction in amplitude of ST-segment elevation from V2 to V5, (b) progressive regression of the ST-segment changes

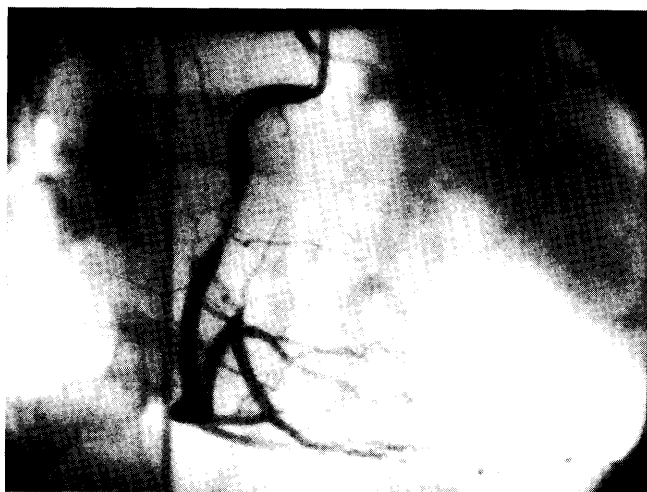


Fig. 5. Coronary angiography showing a significant discrete proximal RCA stenosis near a branching right ventricular artery.



Fig. 7. Coronary angiography showing a successfully stented right coronary artery.

without the appearance of Q wave. Because of concomitant inferior infarct, there is accompanying reciprocal ST-segment depression in lead I and aVL, more marked in the latter. The morphology of the elevated ST-segment is not specific for RV infarct. In the presence of RV infarct-associated precordial ST-segment elevation, it is noted that the inferior leads often manifest ST-segment elevation, though of smaller amplitude.¹⁰

In this reported case, there was rapid resolution of the initial massive ST-segment elevation in the precordial leads within minutes of the patient's admission. This probably reflected the dynamic flow state of the electrical injury current accompanying both the LV inferoposterior and RV myocardial infarction. Another possibility that has to be considered is that the precordial ST-segment elevation is not due to RV infarction but is a reflection of acute anterior transmural myocardial ischaemia. Since the left coronary arteries were shown to be normal in the subsequent coronary angiogram, acute



Fig. 6. Coronary angiography showing normal left coronary arteries.

anterior myocardial ischaemia, if it had occurred, most likely would have been a result of transient spasm of the left anterior descending artery. However, to have two pathological events, one involving thrombosis of the right coronary artery and the other involving spasm of the left anterior descending artery, occurring simultaneously and resulting in the presenting abnormal ECG (Fig. 1) is indeed exceedingly rare.

Secondly, the ST-segment elevation was diffuse, involving 9 leads on the resting ECG, much like a large anterior myocardial infarct. This indicated that a relatively small RV myocardial mass was also capable of generating significant electrical voltage changes on ECG. The relative infrequency of precordial ST-segment elevation associated with RV infarct is attributed to suppression of the RV injury current by the dominant and directly opposing electrical forces of simultaneous LV inferoposterior injury. In a series of 24 patients collected by Kataoka et al,¹⁰ it was shown that lead V1 had the highest prevalence of maximum ST-segment elevation (66%) while lead V5 showed the least prevalence.

The third feature in this patient was that the amplitude of the ST-segment elevation was unusually high. There has so far been only 1 reported case of ST-segment elevation associated with RV infarct exceeding 10 mm in the literature.¹⁰ It is difficult to explain the reason for the marked ST-segment changes although the presence of partial right bundle branch block may be a contributory factor.

This case illustrates the unusual manner in which RV myocardial infarct can manifest itself electrocardiographically. It serves to remind physicians that in the event of an acute inferior myocardial infarction accom-

panied by precordial ST-segment elevation, coexistent RV infarct should be considered and that further tests need to be carried out for confirmation.

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