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CASE REPORT

A Needle in a Haystack: uncovering RV branch compromise after primary PCI of RCA via ECG.

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Abstract

Background: ECG remains the mainstay of diagnosis of STEMI in clinical practice and proper interpretation helps to identify the culprit artery. We present a rare case in which ST elevation in ECG proved to be quite misleading.

Case Presentation: We present the case of an elderly male patient who presented to emergency department with acute onset of severe chest pain. Ecg done in emergency department which showed inferior wall myocardial infarction (MI) complicated by complete heart block. He underwent angioplasty of right coronary artery. Patient in CCU developed ST elevation in anterior chest leads which was attributed to RV branch occlusion.

Management and Results: Patient was managed medically as he was pain free and hemodynamically stable. Serial ECG's done. He was strictly monitored in CCU for any hemodynamic or electrical instability. Patient remained pain free throughout his stay in CCU. After 6 to 8 hours ST segment elevation completely settled down with no Q wave formation.

Conclusion: ST segment elevation in anterior leads is hallmark of anterior wall MI. Interestingly, isolated right ventricle MI can also produce similar changes in ECG. Very few case reports found on management of isolated RV branch occlusion and patient can present with a wide spectrum of clinical presentation from being completely asymptomatic to suffering from cardiac arrest. Management of isolated RV branch occlusion is another dilemma and it can vary from case to case.

Keywords

Right Ventricle MI, Anterior Wall MI, RV Branch.

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Introduction

St segment elevation MI has been one of the leading causes associated with mortality. St segment elevation in leads V1to V6 is greatly associated anterior wall STEMI. However, there are few case reports where ST segment elevation in anterior leads is associated with right ventricle myocardial infarction. Discrimination between the two is of vital importance, because both of them may be associated with different complication. Even if presenting with same complication (e.g., shock), different management may be required. However, due to minimal data, no consensus has been made to differentiate ECG changes of the two different types of STEMI. In continuity with previous case reports we present a case of profound anterior ST segment elevation after primary PCI of RCA.

Case Presentation

75 years old male patient, known hypertensive and active smoker presented with excruciating chest pain and listlessness since last 2 hours before arrival to emergency department. ECG on arrival showed ST segment elevation in inferior leads with complete heart block (Figure1). Blood pressure was 130/80 and heart rate was 55 beats per minute. After informed consent, he was shifted to cath lab. Angiography proceeded with tiger catheter from right radial route which showed complete obstruction of the right coronary artery (RCA) at the level of RV branch and minimal to moderate disease in left anterior descending artery (LAD) and critical disease in left circumflex artery (LCX) (Figure 2). Primary PCI proceeded. RCA engaged with JR4 guide. As soon as the BMW wire passed, flow restored in RCA and sinus rhythm resumed. Due to diffuse nature of the disease three stents implanted in the RCA. TIMI III flow restored achieved (Figure 3). Patient shifted to CCU. On arrival to CCU, first post PCI ECG done which showed new ST segment elevation in precordial (V1 to V6) leads and ST segment elevation in inferior leads settled down (Figure 4a). Another ECG 30 minute later showed similar ST elevation in anterior leads (Figure 4b). Despite new ST elevation patient remained pain free. Echocardiographic examination revealed inferior wall hypokinesia. And no segmental wall motion abnormality in anterior territory. Another 30 minutes later, third ECG done showed which showed profound ST elevation in anterior leads (Figure 4c).

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Diagnostic Assessment

Due to suspicion of LAD plaque rupture, patient shifted back to cath lab. Angiography repeated from right femoral route which revealed similar status of coronary arteries. However, it was found that RV branch of RCA was completely occluded from the ostium, which went unnoticed during index procedure.

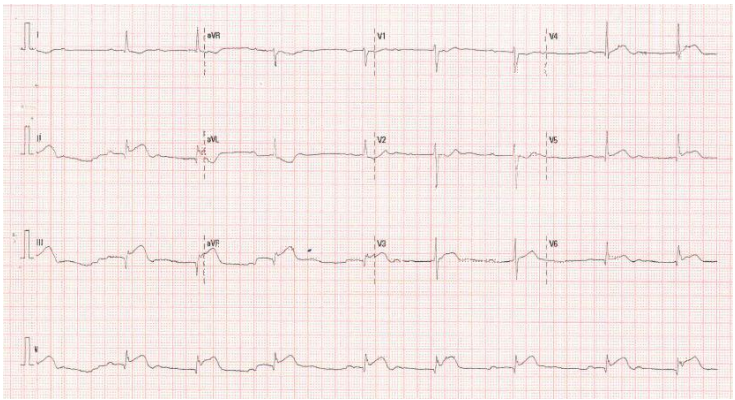

Figure 1: ECG showing ST elevation in inferior leads

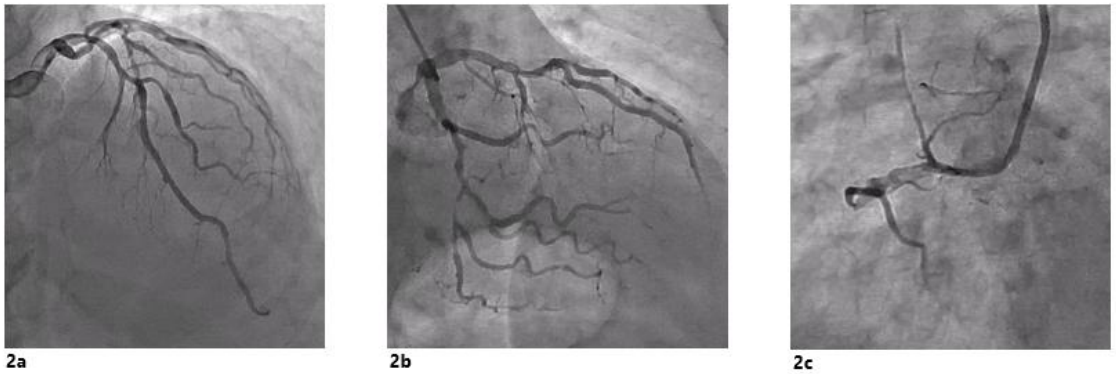

Figure 2a: Angiogram of LAD showing mild plaque in proximal to mid LAD
Figure 2b: Angiogram showing critical disease in LCX
Figure 2c: Angiogram showing total thrombotic occlusion of RCA

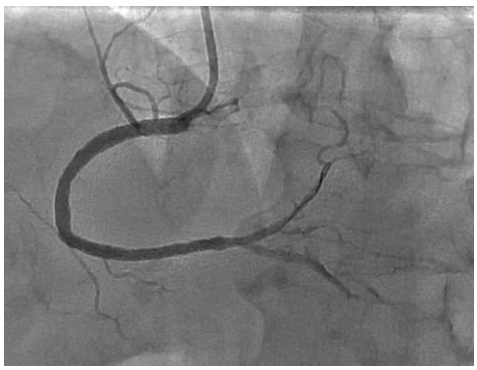

Figure 3: Angiogram showing patent RCA after stenting with loss of RV branch.

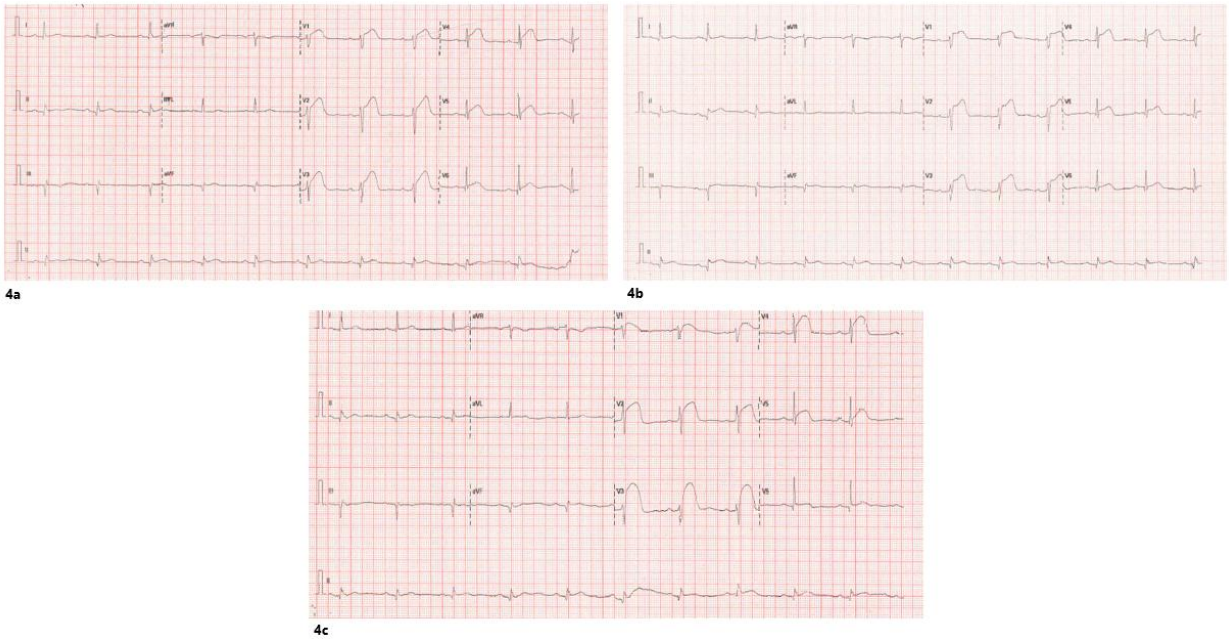

Figure 4a: ECG soon after angioplasty
Figure 4b: ECG 30 minutes after procedure
Figure 4c: ECG 1 hour after procedure

Therapeutic Intervention

As the patient was pain free and hemodynamically stable, no attempt was made to open the artery. Patient started on infusion tirofiban and shifted back to CCU. His ECG repeated few hours later which showed complete resolution of ST segment resolution in anterior territory with no q wave formation (Figure 5). His echocardiography repeated next morning which showed preserved ejection fraction and normal LAD territory but some hypokinesia in inferior wall.

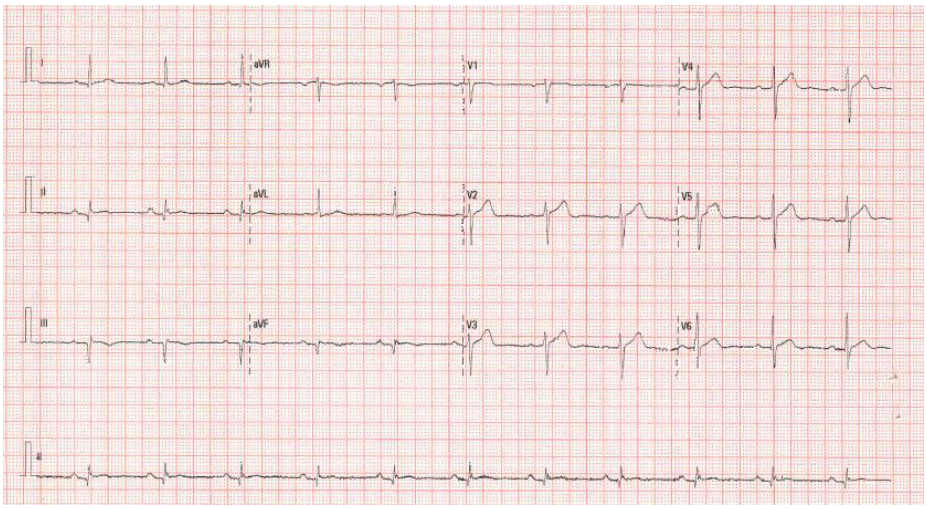

Figure 5: Final ECG 6 hours after procedure

Follow-up and Outcomes

Patient discharged thereafter and followed through opd visit showing remarkable recovery and no complication developed.

Discussion

RV infarction usually occurs in association with inferior wall MI. However, there are few case reports in which isolated RV MI has occurred and presented like anterior STEMI on ECG. Identifying RV MI in such situation is imperative because both types of MI are potential causes of shock, but requiring different management. While, shock with anterior wall MI requires ionotropic support, shock precipitated by RV infarction requires fluid resuscitation.

RV MI in the presence of inferior MI is usually recognized by ST elevation in II, III, aVF with elevation in lead III greater than lead II, accompanied by slight ST elevation in V1 which will be greater than ST elevation in V2. Utilization of right sided leads for diagnosing RV MI is of paramount importance. The presence of ≥1mm ST elevation in V4R is 100% sensitive and 87% specific for RV MI[1].

Isolated RV MI manifesting as anterior STEMI usually occurs in the context of non-dominant RCA. In this setting RCA supplies only right ventricle with LCX supplying inferior wall of left ventricle. The anatomical position of RV is responsible for these ecg changes as it overlies the LV depicting ST elevation in especially first two precordial leads[2,3]. There is one small study showing 5 out of 69 (7%) patients having ST elevation in precordial leads V1 to V5 had isolated right ventricle involvement, which later on confirmed by technetium uptake[4]. In those patients ST elevation in cases of RV MI were greater in leads V1 and V2 and started diminishing towards V1 to V5. In cases of anterior STEMI involving left ventricle, ST elevation started increasing in amplitude from V1 to V5. Unlike anterior STEMI. RV MI is not associated with decrease in amplitude of R wave and development of Q waves in precordial leads. Contrary to these expected ECG findings described for RV MI, our

case demonstrated greater ST elevations in lead V2 & 3 than V1.

In our case it was initially inferior wall MI associated with obstruction of dominant RCA immediately after the origin of RV branch. Subsequently, there was resolution of ST elevation in inferior leads after PCI of RCA. However, patient developed ST elevation in anterior leads because of RV branch occlusion. Kocaman et al. reported similar case in which patient developed ST elevation in leads V1 to V4 after primary PCI of RCA[4,5]. They did report slight PR depression in same leads, probably secondary to atrial injury. In our case no PR depression was noted. Notably, similar to our case, their case also demonstrated higher ST elevations in lead V3 & 4 than V1 & 2, which they attributed to a well-developed RV branch.

Isolated RV MI is not only a diagnostic dilemma, but its management is also challenging. Previously only conus branch occlusion is thought to be associated with arrythmia. However, one case report has clearly shown that occlusion of RV MI can also cause fatal arrythmia[2]. In our case patient was clinically stable so, we consider medical management for our patient.

Taking into consideration, such frank changes in ECG, absence of any chest pain and hemodynamic instability in our patient are notable findings. Right ventricle being thin-walled structure is relatively resistant to ischemia. Another explanation comes from the blood supply of the right ventricle. RV derives nutrients from blood in the cavity via diffusion. Also, RV is the site of extensive collateralization of right and left coronary artery[6].
We presented a rare case of RV MI occurring after primary PCI of RCA. While ST elevation in anterior leads is highly suggestive of anterior MI. Nonetheless, one should be mindful that that such extensive changes can also be found in isolated RV MI in the absence of inferior MI.

Conclusion

This case highlights the diagnostic challenge presented by ST-segment elevation in the anterior leads following primary PCI of the right coronary artery (RCA), where the culprit was an isolated right ventricular (RV) branch occlusion rather than a classic anterior wall myocardial infarction (MI). The clinical presentation and ECG findings initially suggested a more common anterior STEMI, but careful evaluation revealed that the RV branch occlusion was the cause of the ST-segment elevation. The patient remained pain-free and hemodynamically stable throughout, and medical management proved successful with complete resolution of the ST elevation without any significant complications. This case underscores the importance of considering isolated RV MI as a potential diagnosis in patients with atypical ECG changes, even in the absence of inferior MI. Early recognition and appropriate management are critical, as isolated RV MI can manifest with a broad spectrum of clinical presentations.

Acknowledgment

None.

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