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Isolated Right Ventricular Myocardial Infarction

A Patient with Chest Pain and Precordial ST-Segment Elevation

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Abstract

We present a challenging case of a patient with an isolated right ventricular myocardial infarction (iRVMI) caused by the spontaneous occlusion of a right ventricular branch mimicking an anterior myocardial infarction on the electrocardiogram. A high index of suspicion is required to diagnose an iRVMI because the electrocardiogram may be misleading.

Keywords: Coronary angiography; electrocardiography; magnetic resonance imaging; myocardial infarction; right ventricle; STsegment elevation

Case Presentation

A 54-year-old male smoker presented with 30 minutes of substernal chest pain. Vital signs were normal and the cardiopulmonary examination was otherwise unremarkable. The electrocardiogram (ECG) showed ≥0.1 mV ST-segment elevations (STEs) at the J point in leads V1-V4 (fig. 1A), subtle STEs in lead III, ST-segment coving in leads II and aVF, and subtle reciprocal ST-segment depression in lead aVL (fig. 1B). Emergency coronary angiography revealed patent major coronary arteries (suppl. video 1 and 2), whereas left ventriculography showed normal segmental and global systolic function (suppl. video 3). Scrutiny of the angiogram revealed occlusion of a right ventricular branch (RVB) with an underlying high-grade stenosis (fig. 1C), which was successfully recanalized with angioplasty (fig. 1D and suppl. fig. 2). This resulted in the resolution of the STEs, followed by the emergence of negative T waves in precordial leads with preserved R wave amplitude (suppl. fig. 3). Cardiac magnetic resonance imaging (CMR) showed subendocardial late gadolinium enhancement (LGE) of the right ventricular free wall consistent with myocardial infarction (MI) (fig. 1E and suppl. fig. 4). A two-month follow-up ECG showed resolution of the negative T waves. The patient remained asymptomatic at the six-month follow-up.

Discussion

Right ventricular myocardial infarction (RVMI) is associated with an increased risk of mortality and morbidity related to hemodynamic and electrophysiological complications, therefore warranting an early diagnosis [1, 2]. Typically, RVMI is associated with inferior MI and can be reliably diagnosed by a ≥0.1 mV STE in lead V4R. In such cases, a less dominant opposing inferior or inferior lateral left ventricular (LV) wall injury current or dilation of an ischemic right ventricle may help to unmask precordial STEs, which is easier recognized as a manifestation of RVMI [2–4]. In contrast, an inferior or inferior lateral LV wall injury current is absent or minimal in isolated RVMI manifesting precordial STEs; thus, it is easily misinterpreted as an anterior MI or even missed if not suspected, particularly when the culprit is a spontaneously occluded RVB, which may be more difficult to identify by angiography than an occluded non-dominant right coronary artery or stent-jailed RVB [5–7]. Failure to diagnose an isolated RVMI can result in therapeutic misadventures such as inappropriate administration of nitrates potentially leading to severe hypotension and precipitating cardiogenic shock or improper recognition of a concomitant lesion in the left anterior descending artery (LAD) as the culprit leading to unnecessary stent implantation across a stable and not necessarily ischemia-producing lesion [2, 3, 5].

Although there are distinguishing ECG features between anterior MI and RVMI that may facilitate differentiation, this is not always the case, as shown by the current presentation. In anterior MI, almost exclusively leads V2-V4 show the greatest STEs, and the STE in lead V3 is greater than in lead V1 [2, 3]. In the present case, the STEs progressed from lead V1 to V3, with the maximal STE observed in lead V3, and the frontal plane ischemic vector was directed inferiorly, producing subtle STEs or ST-coving in the inferior leads, suggesting an occlusion of the LAD distal to the first diagonal artery [8]. Nonetheless, the absence of angiographic evidence supporting an anterior MI, such as a suspicious lesion in the LAD and/or anterior LV wall motion abnormality on ventriculography, helped fuel the suspicion of an RVMI, which was eventually diagnosed despite the absence of an RVMI-related ECG pattern comprising STEs in lead V1 or V2≥V3 with absence of progression or decreased elevation towards more left-sided leads [1–4]. Consequently, this case highlights the exceptional association between an incremental pattern of STEs in leads V1-V4 and isolated RVMI. An incremental pattern of precordial STEs has rarely been described in cases of combined inferior and RVMI in which the ischemic right ventricle was dilated [2, 3]. Furthermore, as in our case, a culprit RVB with a large area of distribution causing an isolated RVMI has been linked with an incremental pattern of precordial STEs [9]. Although it was initially reported that precordial STE in RVMI does not result in decreased R wave amplitude or Q waves, subsequent cases suggested that it may evolve into a pseudo-anterior MI ECG pattern, often along with precordial T wave inversions attributed to a MI of the anterior right ventricular free wall [1–4]. As a result,

the absence of LGE and/or evidence of myocardial edema on CMR, implying an acute ischemic injury on the antero-apical LV wall, makes the evolution of transient precordial T wave inversions without Q waves and/or a decreased R wave amplitude another distinctive feature of this case. Finally, the frontal plane ischemic vector was directed at around +90° (isoelectric ST-segment in lead I), suggesting a small final RVMI, thereby explaining the absence of hemodynamic complications [10].

Conclusion

In conclusion, we have presented an exceptional case of isolated RVMI caused by spontaneous occlusion of an RVB, characterized by an incremental pattern of precordial STEs and the evolution of transient negative T waves in precordial leads with preserved R wave amplitude.

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Ethics Statement

The authors attest that they comply with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. Written informed consent was obtained.

Conflict of Interest Statement

The authors have no potential conflicts of interest to declare.

Author Contributions

Andreas Y. Andreou: Conceptualization: Lead; Data curation: Lead; Investigation: Lead; Writing – original draft: Lead; Writing – review & editing: Lead.
Elena Leonidou: Data curation: Supporting; Investigation: Supporting.
Andreas Tryfonos: Data curation: Supporting; Investigation: Supporting.

References

You will find the full list of references online at https://cvm.swisshealthweb.ch/en/article/doi/cvm.2024.1486643913/.

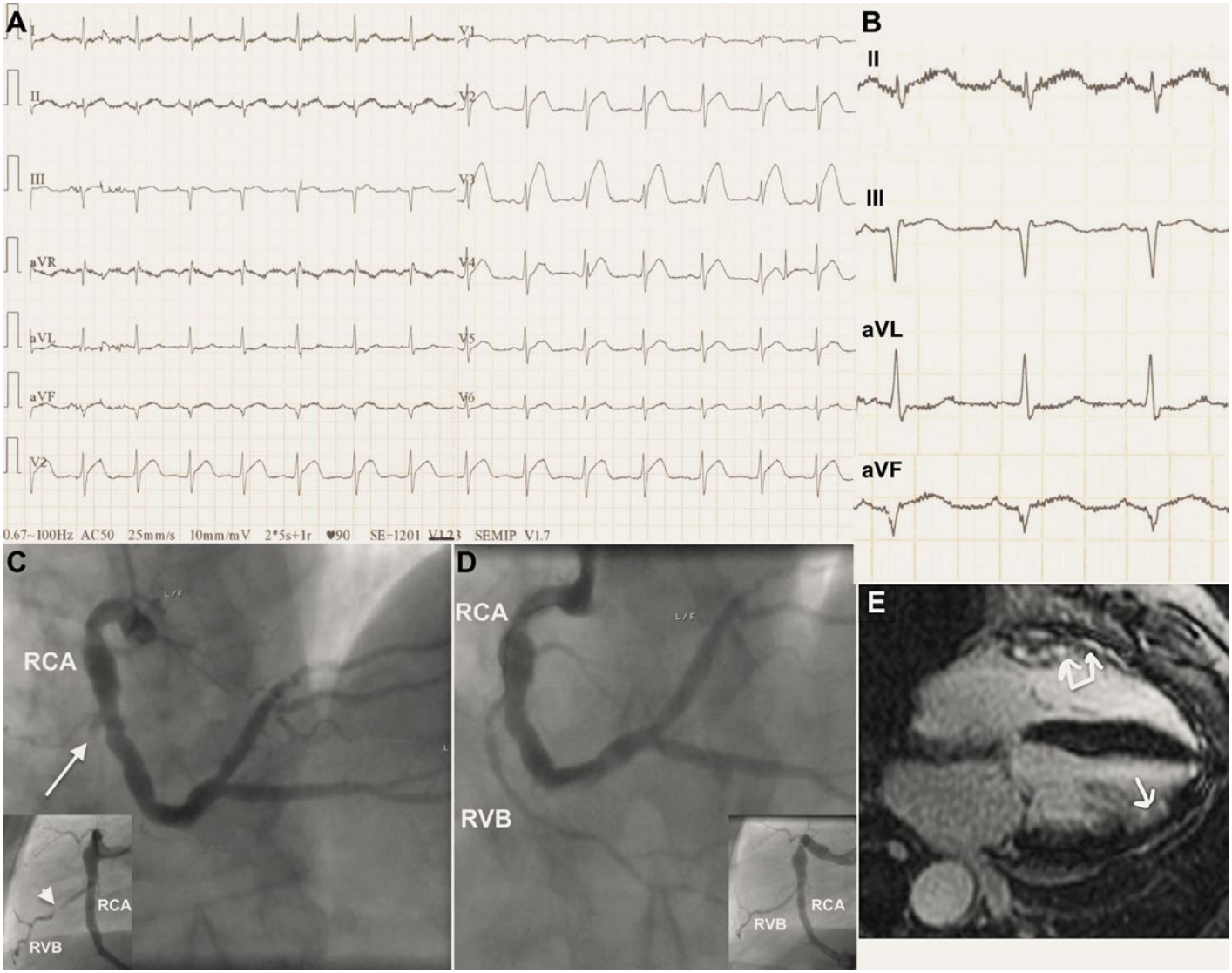

Figure 1: (**A**) Twelve-lead electrocardiogram (ECG) on admission showing ≥0.1 mV ST-segment elevation (STE) at the J point in leads V1-V4; STE progresses from lead V1 to V3 with the latter showing the maximum STE, suggesting an anterior rather than a right ventricular myocardial infarction. (**B**) Magnified ECG recording showing subtle STE in leads II, aVF and III and subtle ST-segment depression in lead aVL. (**C**) Conventional right coronary artery (RCA) angiographic image depicting an occluded right ventricular branch (RVB; arrow) with an underlying high-grade stenosis (embedded panel; arrowhead). (**D**) Conventional RCA angiographic image depicting a patent RVB following plain balloon angioplasty. (**E**) Cardiac magnetic resonance imaging performed one week after admission depicting subendocardial late gadolinium enhancement (LGE) of the basal and mid-inferior segments of the free wall (double arrow) of a normal-sized right ventricle. The subendocardial LGE noted on the apical lateral left ventricular wall (single arrow) was not related to the current presentation.