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CASE REPORT

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Diffuse precordial ST-segment elevation in inferior-right myocardial infarction

Andreas Y. Andreou, Marios A. Ioannides,
George M. Georgiou, Panayiotis C. Avraamides

Nicosia General Hospital, Nicosia, Cyprus

Abstract

A right ventricular (RV) myocardial infarction (MI) may yield precordial ST-segment elevation (STE). Accordingly, combined inferior and precordial STE may be produced during an inferior-RV MI. Such an electrocardiographic picture may be mistakenly regarded as showing wrapped left anterior descending artery (LADA) occlusion or double vessel occlusion. We present a patient with inferior-RV MI and STE in the inferior, all precordial and right chest leads, in whom the diffuse precordial STE was probably mistakenly regarded as showing anterior MI. However, the STE resolution in V1–V2 and late R' wave in V1, which were combined with a recanalized RV branch, favored the RV origin of this STE. Furthermore, the LADA was patent when V3–V6 showed severe ischemia, while its lesion was angiographically stable. Thus its simultaneous occlusion was unlikely. The late R' wave in V1 indicates RV transmural conduction delay;as highlighted herein, it is diagnostic of a RV myocardial infarction. (Cardiol J 2010; 17, 6: 628–631)

Key words: myocardial infarction, ST elevation, transmural conduction delay

Introduction

Right ventricular (RV) myocardial infarction (MI) usually occurs in association with inferior MI. Beyond ST-segment elevation (STE) in V4R, which is its most predictive electrocardiographic (ECG) feature, it may yield STE in the precordial leads [1–3]. In such cases, differentiation from anterior MI is important for proper management, particularly when a concomitant left anterior descending artery (LADA) lesion is revealed on coronary angiography. We present a patient who suffered an inferior-RV MI in whom the diffuse precordial STE on the presenting ECG was probably misinterpreted as a sign of anterior MI. We discuss the ECG features that could have pointed to the correct diagnosis, highlighting the ECG sign of RV transmural conduction delay.

Case presentation

A 50 year-old male patient presented for a routine examination in the outpatients section of our department. Review of his medical notes revealed a history of hyperlipidemia, cigarette smoking, balloon angioplasty at proximal LADA because of unstable angina and acute MI. The latter manifested transient complete atrioventricular block with a narrow QRS escape rhythm at a rate of 58/min along with STE in the inferior, all precordial and right chest leads on the admission ECG (Fig. 1). Thrombolysis resulted in > 50% STE resolution only in V1–V2 (Fig. 2); V1 showed a prolonged late R' wave (arrows) and an apparent STE of ~1 mm amplitude. Urgent coronary angiography revealed total proximal occlusion of a dominant right coro-

Address for correspondence: Andreas Y. Andreou, MD, Cardiology Department, New Nicosia General Hospital, Old Road Nicosia-Limassol 213, P.O. Box 2029, Strovolos, Nicosia, Cyprus, tel: 00357 22603490, fax: 00357 22604322, e-mail: y.andreas@yahoo.com

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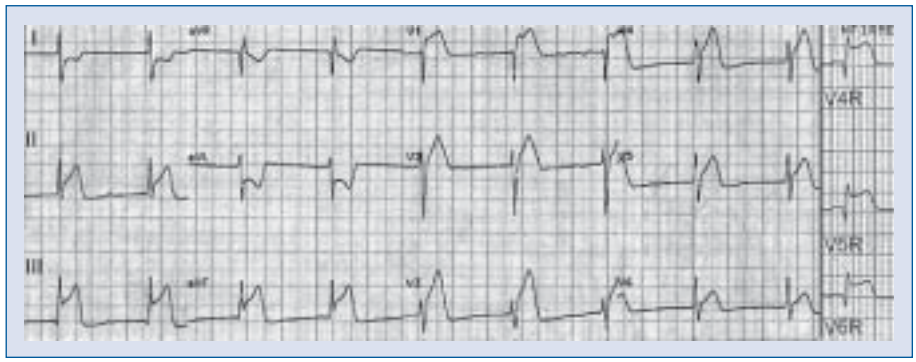

Figure 1. Electrocardiogram on admission displaying complete atrioventricular block and ST-segment elevation over the inferior, all precordial and right chest leads.

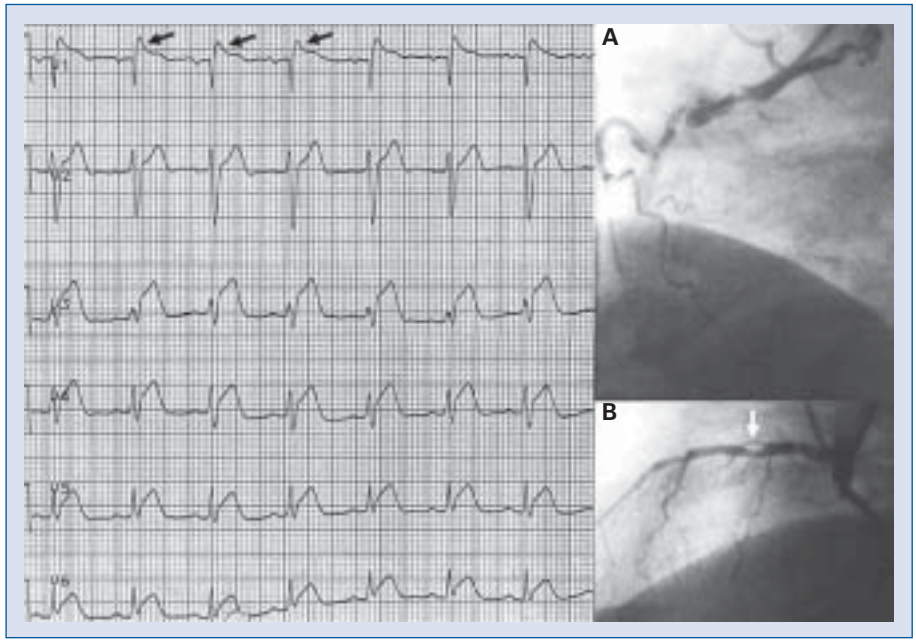

Figure 2. Electrocardiogram obtained following thrombolysis displaying ST-segment elevation (STE) resolution only in V1–V2. Note the late R' wave in V1 (arrows) that leads to an apparent STE. Leads V3–V6 show an evolving ischemic process provided the decreased QRS forces, increased STE in V6 and decrease in the amplitude and disappearance of the S wave in V5 and V6 respectively. **A**. Left anterior oblique view of the proximally occluded right coronary artery. Note the recanalized first right ventricular branch; **B**. Lateral view displaying a patent left anterior descending artery with an angiographically stable lesion distal to the first diagonal branch.

nary artery (RCA) with a recanalized first RV branch and a patent LADA with a lesion located distal to the first diagonal branch. The LADA lesion was successfully tackled with stent angioplasty but the attempt to recanalize the RCA failed. Creatine kinase and creatine kinase-MB peaked at 3533 IU/L (reference value: 26–171 IU/L) and 254 IU/L (reference value: 0–25 IU/L) respectively. The patient was discharged home in a clinically stable condition following a seven day uneventful hospital course.

Discussion

It is well-known that RV MI may show precordial STE up to lead V5, with an incidence of 7% [1–3]. The development and extent of such STE depends on the ratio of the magnitude of the coexisting electrical forces produced by the concomitant inferior, posterior-lateral and RV MI, degree of clockwise rotation of the heart in the horizontal plane and body geometry [1, 2]. An isolated RV MI

presenting precordial STE may be mistakenly regarded as anterior MI. An ECG pattern consisting of inferior and precordial STE due to an inferior-RV MI may be erroneously interpreted as showing a combined inferior and anterior MI due to a wrapped LADA lesion or double-vessel occlusion. Such misinterpretations may complicate decisions in the catheterization laboratory regarding the culprit vessel and lead to unnecessary and potentially harmful treatment.

A distinct RV MI-related precordial STE pattern has been described; i.e. V1 or V2 show the highest STE which decreases towards more left-sided leads or STE in V1 ≥ V3 with absence of progression of STE from V1 to V3 that may facilitate differentiation from anterior MI where almost exclusively V2–V4 show the greatest STE and STE in V3 is higher than in V1 [1, 2]. However, such a differentiation is better served when using vector concepts to interpret ECGs [3]. Both an inferior--RV MI with precordial STE and anterior MI produce an anteriorly directed mean ST vector, i.e. 10–40° and 10–70°, respectively. However, the mean ST vector in the frontal plane is different and facilitates their distinction, i.e. more than 90° to the right (this produces ST-segment depression in lead I) in the former and usually –30° to –90° in the latter. Furthermore, STE > 2 mm in V5–V6 during inferior MI has been reported to predict a 'mega' RCA or circumflex artery featuring a large posterolateral and posterior descending branch with 94% sensitivity and 98% specificity, while a large RCA that extends to the apex has been advocated in cases manifesting STE in V3–V6 [1, 4].

In our case, we found STE in lead III > II, ST--segment depression in lead I and STE in lead V4R which favored occlusion of a dominant RCA proximal to the RV branch. Coronary angiography confirmed such a culprit anatomy, albeit with a recanalized RV branch; the latter probably accounted for STE resolution in V1–V2 and supported the notion that this STE was due to a RV-derived anteriorly directed injury current. Simultaneous RCA and LADA occlusion producing combined STE in the inferior leads and V3–V6 is possible. However, such a scenario was unlikely in the present case provided that the LADA was patent when the ECG inscribed STE in V3–V6. Moreover, leads V3–V6 signified an evolving ischemic process provided the reduction of the QRS forces, the increased STE in V6 relative to admission and the marked decrease in the amplitude and disappearance of the S wave in V5 and V6, respectively.

Furthermore, the LADA lesion appeared stable; to be precise, it showed a smooth contour and no evidence of thrombosis. Patients with double vessel occlusion usually manifest cardiogenic shock and malignant ventricular tachyarrhythmias. The absence of such adverse repercussions in our patient also goes against a diagnosis of simultaneous LADA occlusion. Consequently, STE in V3–V6 which was > 2 mm in V5–V6 favored the occlusion of a 'mega' RCA, probably extending to the apex.

Resolution of STE in V1 following thrombolysis unmasked a late R' wave that led to STE of ~1 mm amplitude. This ECG sign is not uncommon in patients with RV MI manifesting precordial STE, is unmasked upon amelioration of ischemia and STE resolution, and has been reported to indicate RV conduction delay [5, 6]. Kataoka et al. [6] did not observe it in 42 consecutive patients with first acute anterior MI.They concluded that it is diagnostic of RV MI with 100% specificity and 50% sensitivity. RV transmural wedge preparations exhibiting a prominent epicardial action, potential outward K+ current (Ito)-mediated spike, and dome configuration have been shown to present a true STE following exposure to global ischemia at a time when conduction was not greatly affected [7]. The epicardial action potential dome is lost, while the plateau phase of the endocardial action potential is relatively maintained, leading to a transmural voltage gradient and STE. In preparations with a less prominent Ito, ischemia resulted in an apparent coved STE secondary to a prolongation of the R' wave that was due to transmural conduction delay. Slowing of the conduction is attributed to inactivation of the sodium channels secondary to membrane depolarization from the ischemia-induced increased extracellular potassium concentration.

In addition, the latter reduces Ito which also contributes to QRS prolongation. Indeed, in the cases reported by Kataoka et al. [6] in which a late R' wave was documented upon amelioration of ischemia, there was such a coved STE during the acute phase of ischemia [4]. Apparently our patient manifested this ECG sign upon amelioration of ischemia and STE resolution, indicating the transmural conduction delay that is invariably present during acute ischemia. Furthermore, this ECG sign may give the false impression of the presence of right bundle branch block; in our case QRS duration was < 110 ms in leads other than V1 thus excluding such a scenario. Consequently a pattern of precordial STE due to RV MI where lead V1 manifest an apparent STE which is actually the prolonged R' may be mistakenly regarded as showing anterior MI due to a LADA lesion proximal to the first septal branch.

Conclusions

In conclusion, the case of a RCA-related inferior-RV MI presented herein illustrates an interesting presenting ECG, namely combined STE in the inferior, all precordial and right chest leads. STE resolution only in V1–V2, the late R' in V1 and the ECG changes of worsening ischemia in V3–V6, along with the patent LADA revealed, pointed against an anterior MI-related precordial STE. A late R' wave in V1 indicates RV transmural conduction delay, and it is diagnostic of a RV MI.

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