



Acute Myocardial Infarction Presented with Isolated Precordial Lead Elevation in V2

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Atrial fibrillation (Af) with acute myocardial infarction (AMI) is a life-threatening disease, and electrocardiogram is indispensable for the initial diagnosis of high-risk patterns in the emergency department. Here we report a case of a 63-year-old male of Af with AMI, presented with isolated elevation of the precordial V2 lead ST-segment, mostly arising from major occlusion of the left main coronary artery and right coronary artery.

Key words: Atrial fibrillation, isolated precordial V2 lead, ST-segment elevated myocardial infarction

INTRODUCTION

Atrial fibrillation (Af) is the most prevalent arrhythmia and is associated with thromboembolism, stroke, congestive heart failure, acute myocardial infarction (AMI), and all-cause mortality.^{1,2} Af with AMI is a critical condition, and electrocardiogram (ECG) is indispensable for the initial diagnosis of high-risk patterns in the emergency department (ED). Usually, transmural infarction will cause ST elevation in the precordial leads and could prompt the ED to inform cardiologists to perform percutaneous transluminal coronary angioplasty as soon as possible. Here we report a case of Af with AMI originating from left main coronary artery (LMCA) and right coronary artery (RCA) occlusion, presented with an unusual finding of prominent ST-segment elevation in a single precordial lead (V2) on 12-lead ECG.

CASE REPORT

A 63-year-old male smoker with a history of hypertension presented to the ED with an 8-h duration of chest pain and palpitation. Physical examination revealed a body temperature of 36°C, a blood pressure of 77/65 mmHg, a heart rate of 123 beats/min, and a respiratory rate of

18 breaths/min. Laboratory data included potassium (4.2 mmol/L), creatine phosphokinases (171 U/L), creatine phosphokinases-MB [32 U/L (<24 U/L)], and troponin I (0.61 ng/mL [<0.04 ng/mL]). A 12-lead ECG showed Af; ST-segment elevation (STE) in leads V2, I, aVL, and aVR; and ST-segment depression (STD) in leads II, III, aVF, V5, and V6 [Figure 1]. Coronary angiography showed total occlusions of the LMCA [Figure 2] and the distal segment of the RCA [Figure 3]. Subsequently, the patient developed bradycardia and cardiac arrest despite treatment with coronary intervention, despite intra-aortic balloon pump and temporary pacemaker support. We were unable to save his life despite attempting cardiopulmonary resuscitation.

DISCUSSION

By searching the Medline or PubMed databases using keywords “acute myocardial infarction and isolated precordial lead ST-segment elevation (STE),” we identified no other case noting this kind of finding. AMI with prominent STE in lead V2, but not in other precordial leads with concomitant STE in leads I, aVL, aVR, and reciprocal changes in inferior

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How to cite this article: Chen LH, Wu LW, Huang SC. Acute myocardial infarction presented with isolated precordial lead elevation in V2. J Med Sci 2020;40:194-6.

Received: January 01, 2020; Revised: January 20, 2020; Accepted: April 02, 2020; Published: April 22, 2020

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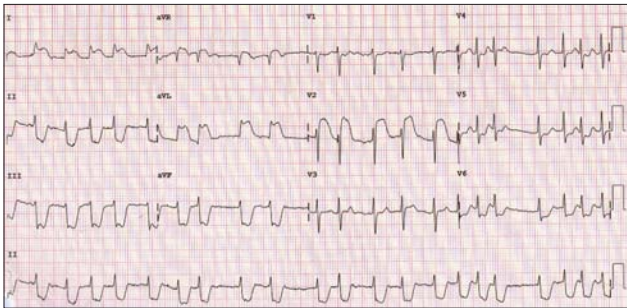


Figure 1: Emergency electrocardiogram showed a rhythm of atrial fibrillation at a rate of 120 BPM, with leftward axis and ST-segment elevated at isolated V2, aVR, I, aVL and depressive ST-segment at lead II, III, and aVF

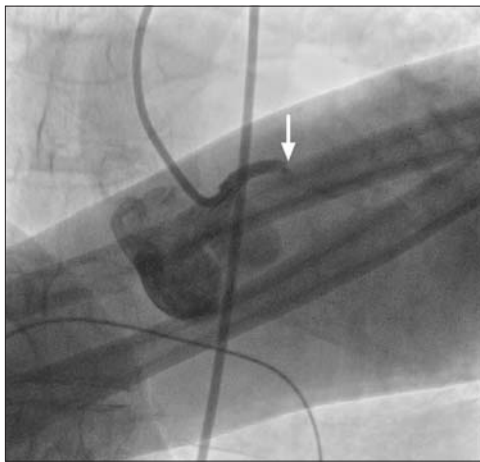


Figure 2: Coronary angiography demonstrating total occlusion of the distal segment of the left main coronary artery (white arrow)

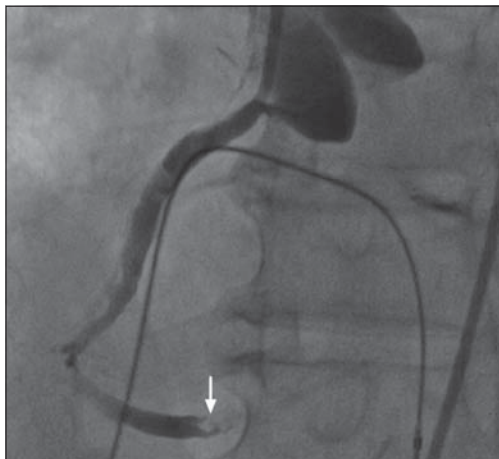


Figure 3: Coronary angiography demonstrating total occlusion of the mid and distal segments of the right coronary artery (white arrow)

leads II, III, and aVF in the ECG, can mislead us to believe that only relatively limited area of transmural ischemia in the anterior wall is involved, which reduces our awareness to have

a prompt approach slows the response in this kind of case.³⁻⁵ The presentation of ECG due to LMCA usually shows STE at aVR (67%), V2–V6 (35%), or depressive ST-segment in more than six leads (31%).⁶ However, the cancellation of the vector of the ST elevated segment in precordial leads can accompany RCA occlusion due to near equilibration of electrical forces from the apical and the basal parts of the left ventricles.⁷ However, the ischemia in the apical segments is somehow not counterbalanced by the basal part and thus, can be exposed in lead V2. This case also confused the emergency room staff, leading to a delay in informing the cardiologist and slowing the therapeutic schedule.

Both STD and STE with direction and the magnitude of the ST vector in certain leads can be informative about which lesion is involved and the severity. When the ST vector points in the direction of the leads, it will cause ST elevation. Otherwise, the ST vector will be less proportionally elevated if the vector is perpendicular to leads. In our case, we should not ignore “the elephant in the room” by only focusing on isolated ST elevation at V2, neglecting the finding of ST depression in the inferior leads (II, III, and aVF) and elevation in I, aVL, and aVR.

Fujii *et al.*⁸ reported ECG findings of STE in lead V2 with larger magnitude STD in inferior leads, and STD in the lead V5 suggests AMI owing to LMCA lesions. Moreover, widespread STD in inferior leads with STE in lead aVR in the ECG displayed occlusions of LMCA and multiple vessels, which led to ischemia of large portions of the left ventricle and high risk of cardiac death.⁴ Prolonged ischemia contributes to the delayed conduction of Purkinje fibers, resulting in irreversible necrosis of myocardium and poor prognosis.³

Dysfunction of sinus and atrioventricular nodes usually indicates the occlusions of RCA or left circumflex coronary artery, which increase the risk of Af and atrial complications.⁷ Af is the most prevalent arrhythmia and is associated with thromboembolism, stroke, congestive heart failure, AMI, and all-cause mortality.^{1,2} Identifications of ST-segment elevated myocardial infarction (STEMI) are based on clinical symptoms (chest pain), ECG morphologies of STE at the J point in at least two contiguous leads of ≥ 2 mm (0.2 mV) in men or ≥ 1.5 mm (0.15 mV) in women in leads V2–V3 and/or of at least 1 mm (0.1 mV) in other contiguous chest or limb leads in the absence of left ventricular hypertrophy or left bundle branch block, and elevated cardiac biomarkers.⁹ The recommendations of therapy – antithrombotic agents (anticoagulant and antiplatelet medications) and primary percutaneous coronary intervention (PCI) – should be provided in patients with STEMI and ischemic symptoms of <12-h duration.^{2,9} The delayed performance of PCI after admission to hospital is associated with a high risk of in-hospital mortality.⁹

In conclusion, Af with AMI, particularly involving occlusions of LMCA and RCA, is a life-threatening condition. Analyses of the ECG data detected Af and ST elevation in isolated precordial lead V2, with widespread STD in II, III, and aVF and STE in I, aVL, and aVR, resulting from acute LMCA and distal RCA occlusion. Clinicians should be aware of these critical ECG morphologies to enable early detection and timely coronary revascularization.

Declaration of patient consent

This article does not include studies with human or animal subjects performed by any of the authors. For this type of study, formal consent is waived due to the death of the patient. The authors certify that the patient's name and initials will not be published, and all reasonable efforts will be made to conceal their identity. The images and other clinical information will only be published in this journal article.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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