

CASE REPORT

## Concealed Myocardial Infarction Revealed by High Lateral and Posterior Electrocardiographic Leads

Sofia Metaxa, MD, Spyridon Koulouris, MD, Dimitrios Stalikas, MD,  
Georgios Velissariou, MD, Antonis S. Manolis, MD

*First Department of Cardiology,  
Evangelismos General Hospital of  
Athens, Athens, Greece*

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coronary angioplasty; coronary stenting*

**ABBREVIATIONS**

CK = creatine kinase  
ECG = electrocardiogram  
LAD = left anterior descending (coronary  
artery)  
LCx = left circumflex (coronary artery)  
MI = myocardial infarction  
NSTEMI = non-ST segment elevation  
myocardial infarction  
RCA = right coronary artery

*Correspondence to:*

Spyridon Koulouris, MD  
First Department of Cardiology  
Evangelismos Hospital  
Athens, Greece  
E-mail: [spyridon.koulouris@gmail.com](mailto:spyridon.koulouris@gmail.com)

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ABSTRACT

We report the case of a 50-year-old man with history of hypertension who presented with chest pain and subtle electrocardiographic (ECG) abnormalities. Only with the aid of additional high lateral and posterior ECG leads (V<sub>7-9</sub>) was a posterolateral acute myocardial infarction identified and subsequently confirmed with positive cardiac enzyme serology. Coronary angiography showed total occlusion of the left circumflex coronary artery, which was successfully recanalized via coronary angioplasty and stenting. This case highlights the use of high lateral and posterior ECG leads (15-lead ECG) in enhancing the diagnostic yield of a conventional ECG in patients presenting with symptoms suspicious for an acute coronary syndrome by disclosing a posterior myocardial infarction and guiding reperfusion therapy.

INTRODUCTION

The electrocardiographic (ECG) finding of ST-segment elevation remains the hallmark for the accurate diagnosis of acute myocardial infarction (MI), facilitates appropriate decision making and allows for early interventional approach to these patients. We report an unusual case of acute MI with ST-segment elevation disclosed only by high lateral and posterior ECG leads, in a patient with chest pain and subtle changes in the conventional 12-lead ECG, who was finally diagnosed to have an acute occlusion of the left circumflex coronary artery, which was reperfused successfully via percutaneous coronary intervention.

CASE REPORT

A 50-year-old gentleman with a history of arterial hypertension under medical therapy was admitted to the emergency department due to severe chest discomfort. He complained of retrosternal heaviness which started at rest 15 hours earlier, lasted for about 15 minutes, and had no radiation or other accompanying symptoms. The discomfort recurred when the patient went for a walk, lasted for another 30 minutes, when he chose to chew a 500 mg tablet of aspirin at the advice of his relatives.

On physical examination, his blood pressure was 170/120 mmHg and his pulse

rate was recorded at 60 bpm. On auscultation normal heart sounds were heard and the rest of the examination revealed no other abnormalities. The 12-lead ECG showed sinus rhythm and minimal (<1 mm) ST-segment elevation in the lateral precordial leads V5 and V6 (Figure 1). While the patient was being examined in the emergency room, he experienced chest discomfort again and the 12-lead ECG showed subtle, if any, differences in the lateral leads (Figure 2) with no ST-segment depression in the anterior precordial or inferior limb leads. Subsequently, additional leads were obtained in high lateral and posterior positions (V7, V8, V9), which demonstrated clear ST-segment elevation of 1 mm (Figure 3) suggesting acute myocardial injury of the posterolateral wall. The patient was given intravenous heparin and nitrates, which produced partial relief of the discomfort, while the echocardiographic examination showed normal systolic function of the left ventricle with an ejection fraction estimated at 70% and no wall motion abnormalities.

With the working diagnosis of an acute coronary syndrome the patient was informed about the necessity of urgent coronary angiography but he initially refused until a few hours later when the laboratory results confirmed the diagnosis of acute MI with a measured peak creatine kinase (CK) of 1149 U/l, CK-MB of 119 U/l and a cardiac troponin-T level of 2.48 µg/l.

He finally consented and was taken to the cardiac catheterization laboratory with minor symptoms, almost 6 hours after his admission. Coronary angiography disclosed a complete occlusion of the left circumflex coronary artery (LCx) right after the origin of the first obtuse marginal branch (arrow, Figure 4). The culprit lesion was successfully crossed with an angioplasty wire, dilated and stented and thus the artery was fully recanalized (arrow, Figure 5). Regarding the rest of the angiographic findings, the left main coronary artery was patent, the left anterior descending (LAD) and the right coronary (RCA) arteries were atheromatic but with no significant stenoses. The subsequent hospital course of the patient was uneventful; he received a platelet glycoprotein IIb/IIIa inhibitor for 24 hours. He was discharged home 2 days later and received instructions for medical therapy which included metoprolol, nitrates, aspirin, clopidogrel and simvastatin. He was also advised to have an exercise testing scheduled at 4-6 weeks later and to have a regular clinical follow-up with his cardiologist.

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## DISCUSSION

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Apart from significant progress which has taken place in

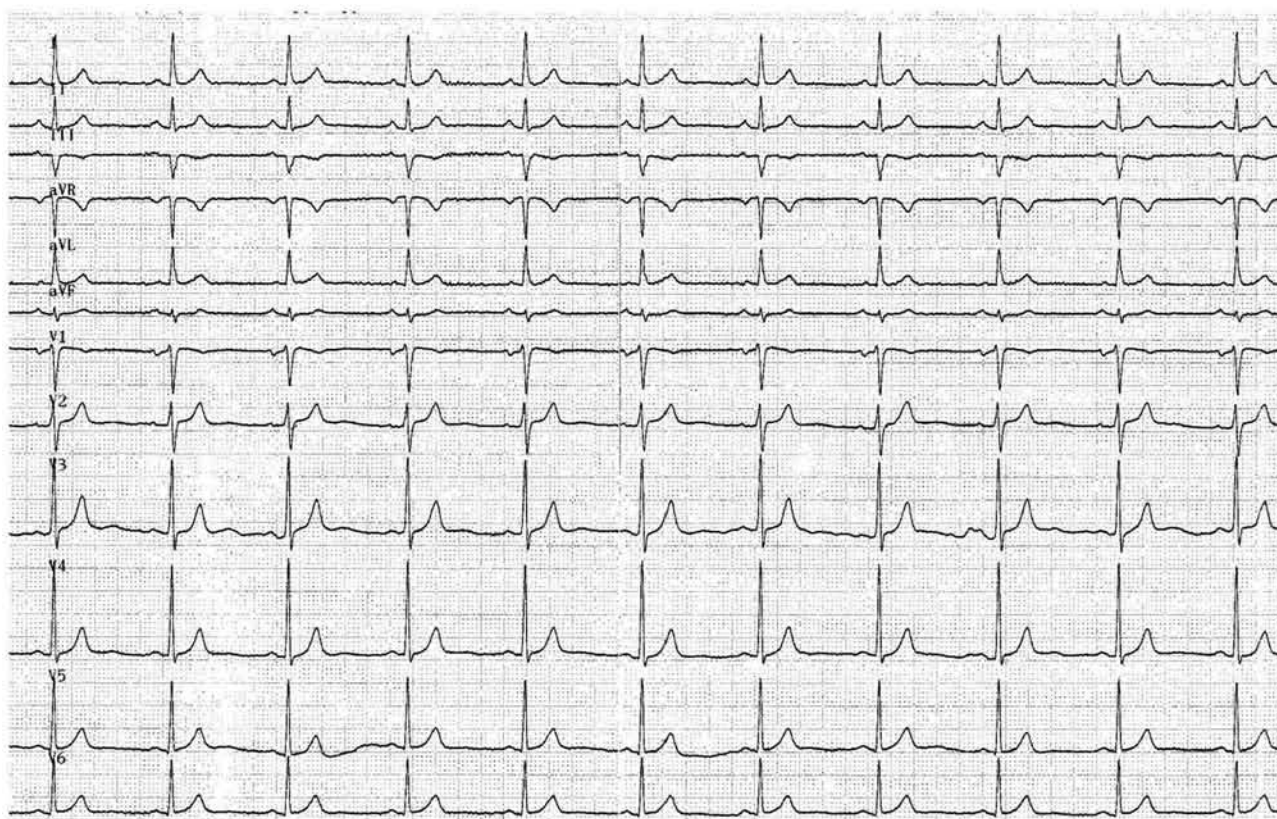


FIGURE 1. 12-lead ECG on admission displaying subtle, if any, albeit non-diagnostic, ST abnormalities.

## CONCEALED MI



FIGURE 2. 12-lead ECG during episode of recurring chest discomfort with no additional diagnostic changes.

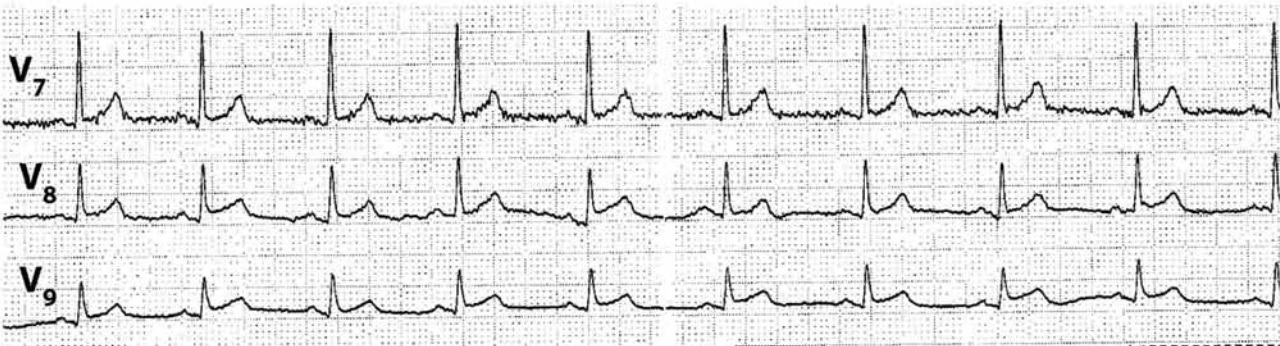
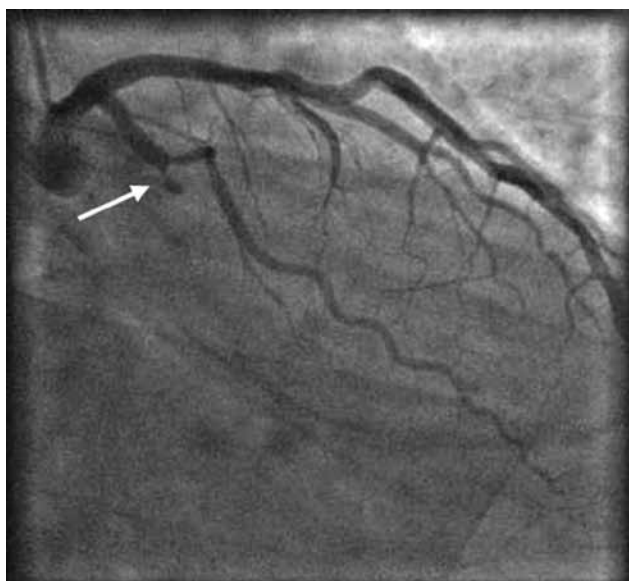


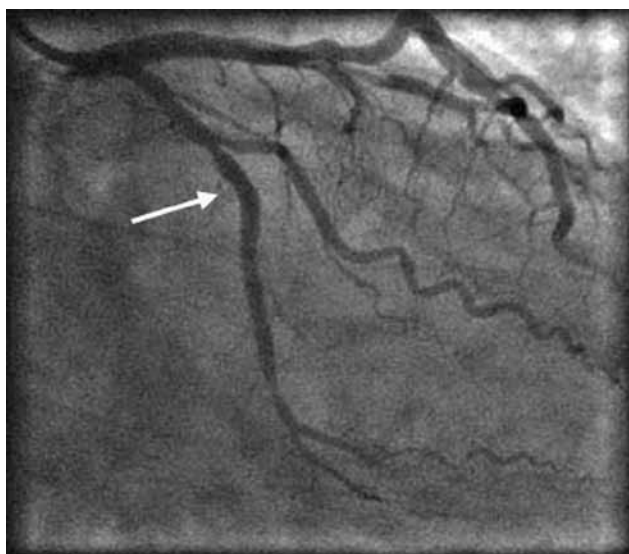
FIGURE 3. High lateral and posterior ECG leads V<sub>7</sub>, V<sub>8</sub>, V<sub>9</sub> during chest discomfort documenting at least a 1-mm ST-segment elevation confirming the suspicion of a posterolateral acute MI.

cardiology over the last several decades, in the case of acute MI, the initial diagnosis is still based on the surface ECG. Urgent reperfusion therapy, attained with thrombolysis or more effectively with primary percutaneous coronary intervention, has greatly improved the prognosis of patients with acute MI. It is indicated in patients with chest pain or discomfort of <12-hour duration who have persistent ST-segment elevation or new left bundle-branch block,<sup>1</sup> meaning that the role of ECG is fundamental in guiding reperfusion therapy in this setting.

An earlier retrospective study of 100 consecutive cases of autopsy-proven acute MIs demonstrated that only 53% of them were correctly diagnosed ante-mortem.<sup>2</sup> In many patients ST-segment elevation is not seen on routine 12-lead ECG as some patients have subendocardial or non-Q-wave myocardial infarction (currently termed non-ST elevation MI - NSTEMI), while others show ST depression in the anterior leads suggestive of posterior myocardial infarction<sup>3</sup> which is misinterpreted as unstable angina or anterior NSTEMI attributed to ischemia



**FIGURE 4.** Coronary angiogram shows complete occlusion (white arrow) of the left circumflex coronary artery immediately after the origin of the obtuse marginal branch.



**FIGURE 5.** Post-percutaneous coronary intervention coronary angiogram shows the restoration of flow in the circumflex coronary artery (white arrow).

in the LAD territory. An erroneous or a belated diagnosis mostly plagues acute posterior-wall MI, since the ST-elevation of the posterior wall is displayed as ST-depression in the anterior ECG leads and only when a concurrent ST elevation is noted in the inferior leads and/or a tall R wave is recorded in the right precordial leads ( $V_{1,2}$ ), a correct diagnosis may be

derived. Heuy et al<sup>4</sup> observed acute ST-segment elevation in only 48% of patients with LCx occlusion (versus 71% in RCA and 72% in LAD occlusion) and found that 38% of patients with a LCx-related infarct had no significant ST changes on admission. Similarly, Blanke et al found that 56% of patients with LCx occlusion had 'non-classic electrocardiographic abnormalities'.<sup>5</sup> They supported the concept that the ECG pattern of true posterior and isolated lateral wall acute MI in the absence of classic changes in the inferior leads was highly specific and predictive of LCx disease.

In patients suspected of having an acute MI, particularly if the affected area is the far postero-lateral portion of the left ventricle, three additional ECG chest leads (the high lateral and posterior  $V_7$ ,  $V_8$ , and  $V_9$  leads) are useful in detecting and localizing myocardial injury or necrosis. The posterolateral wall of the left ventricle is not directly represented by any standard ECG lead. This is the reason why posterolateral MIs are characterized as 'dead angle infarctions' of the ECG,<sup>6</sup> they are often misdiagnosed and as a result the patients are undertreated. It has been reported that 5% of non-Q-wave MIs are retrospectively diagnosed as posterior MIs in which reperfusion therapy would have been justified if additional ECG chest leads had been recorded.<sup>7</sup>

A posterolateral MI is suspected in the 12-lead ECG when there is ST-segment depression in leads  $V_1$ - $V_3$ , ST-segment elevation in leads I, aVL,  $V_5$  and  $V_6$  and can be confirmed by placing extra leads in the high lateral  $V_7$ ,  $V_8$  and  $V_9$  positions. For these three additional leads the electrode is placed over the posterior axillary line ( $V_7$ ), over the midscapular line ( $V_8$ ) and halfway between the midscapular line and the spine ( $V_9$ ), all at the same level as  $V_6$ , providing a 15-lead ECG. ST-segment elevation of >1mm in these high lateral and posterior leads is suggestive of posterior MI,<sup>8</sup> but because of the greater distance between the infarcted area and the leads in the posterior-wall MI, it is suggested that an elevation of 0.5 mm is sufficient to justify the diagnosis of posterior MI and the decision for aggressive treatment.<sup>6</sup>

The majority of patients with the ECG abnormalities suggestive of true posterior or posterolateral MI have critical stenosis or occlusion of the LCx coronary artery, while significant RCA disease is less prevalent.<sup>9</sup> Moreover, in isolated posterior MIs the infarct-related artery is found to be the LCx coronary artery,<sup>3,10,11</sup> suggesting that in the appropriate clinical setting posterior leads may help to discern LCx from RCA occlusion. Patients with inferior or lateral MIs with concomitant posterior wall involvement, have a larger-sized infarct with an increased risk of complications and are in dire need for reperfusion therapy. In keeping with this notion, in a group of patients with ST-segment elevation recorded only in leads  $V_7$ - $V_9$ , isolated posterior MI was found to be complicated up to 69% with mitral regurgitation which was moderate to severe in one third of patients.<sup>11</sup>

The high percentage of electrocardiographically undi-

agnosed acute MI, especially due to LCx occlusion, remains an issue that cannot be satisfactorily addressed by additional posterior ECG leads. Yet in a cohort of patients the percentage of ECG-undiagnosed LCx occlusion was reduced from 50 to 39% when the criterion of ST-segment elevation of 2 mm in V<sub>1</sub> through V<sub>6</sub> and 1 mm in V<sub>7</sub> through V<sub>9</sub> was used.<sup>12</sup> Zalenski et al<sup>13</sup> studied posterior (V<sub>7</sub> to V<sub>9</sub>) and right ventricular (V<sub>4R</sub> to V<sub>6R</sub>) leads to assess their accuracy compared with standard 12-lead ECG and found that these leads increased sensitivity for acute MI by 8.4% but decreased specificity by 7%. A total of 149 patients admitted with suspected acute MI or unstable angina were studied on admission with 12-lead versus 15-lead ECG showing increased sensitivity of ST elevation for acute MI from 47.1% to 58.8% respectively, with no decrease in specificity. It was also found that 22% of patients negative for ST-segment elevation on 12-lead ECG were positive on 15-lead ECG and there was a 6-fold increase in the odds of meeting the ECG criteria for thrombolytic therapy.<sup>14</sup> Nevertheless, in a study investigating the use of a 15-lead ECG in comparison with a 12-lead ECG in every patient presenting at the emergency room with chest pain, no change in diagnosis or management was proved although physicians had a more complete anatomic picture of the acute coronary syndrome.<sup>15</sup>

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