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Cardiology

The Posterior Trap: Circumflex Artery Occlusion in Disguise: About Two Cases

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Abstract **Case Report**

Left circumflex artery occlusion often escapes early diagnosis due to atypical or silent ECG findings. We report two cases of posterior myocardial infarction caused by LCx occlusion, initially misdiagnosed due to non-specific ECG changes. Coronary angiography confirmed the culprit lesion, and timely revascularization led to good outcomes. These cases highlight the need for high suspicion and extended ECG analysis in posterior infarctions.

Keywords: Left circumflex artery, posterior myocardial infarction, atypical ECG, coronary angiography.

INTRODUCTION

Myocardial infarction due to occlusion of the left circumflex artery presents a diagnostic challenge as it can produce subtle or atypical electrocardiographic patterns. These patterns does not always suggest critical occlusion despite its presence [1]. In fact, ECG findings are often non-diagnostic for ST-elevation when the culprit lesion is located in the left circumflex artery and poorly represented on the standard 12-lead ECG [2]. This under-recognition may lead to a delay in coronary reperfusion.

In this report, we present two cases of inferobasal myocardial infarction caused by an occlusion of the left circumflex artery and successfully treated by percutaneous coronary intervention. Through these cases, we aim to highlight the kind of blind spot of the circumflex occlusion and the need for an early coronarography when clinical suspicion is high despite inconclusive ECG findings.

OBSERVATION 1

A 44-year-old male, chronic smoker with no personal or family history of cardiovascular disease and engaged in regular athletic activity, presented to the emergency department with typical chest pain suggestive of angina. The chest pain had persisted for approximately 8 hours prior to admission. It was retrosternal, constrictive, and occurred at rest, with no associated symptoms such as dyspnea, palpitations, syncope. On physical examination, the patient was hemodynamically stable, afebrile, and without signs of heart failure. Cardiovascular and pulmonary auscultations were unremarkable. The admission 12-lead ECG revealed a greater than 2 mm ST-segment elevation in lead V1 (figure 1). The inferior and lateral leads (DII, DIII, aVF, V5–V6) were isoelectric but showed negative T waves, suggestive of underlying myocardial ischemia. Posterior derivations revealed an isolated 1 mm ST-segment elevation in lead V1R (figure 2) and negative T waves. The rhythm analysis noted frequent ventricular extrasystoles occurring in a trigeminy pattern, characterized by a right bundle branch block morphology with negative deflection in the inferior leads, suggesting a probable ectopic focus originating from the left posteroseptal region (figure 1). Cardiac troponin I was significantly elevated at 2.3 ng/mL (normal < 0,031 ng/mL). Transthoracic echocardiography revealed regional wall motion abnormalities involving the basal and mid-lateral segments. Urgent coronary angiography demonstrated a thrombotic stenosis classified as type B1 located in the mid-segment of the left circumflex artery (figure 3). The right coronary artery was dominant and free of significant stenosis. The left main coronary artery is of normal length with no stenosis. The left anterior descending artery is of normal caliber with no stenosis. A successful percutaneous coronary intervention (PCI) was performed with a stent placement on the culprit lesion. The patient remained hemodynamically stable

post-procedure, and no complications were observed during the hospital stay.

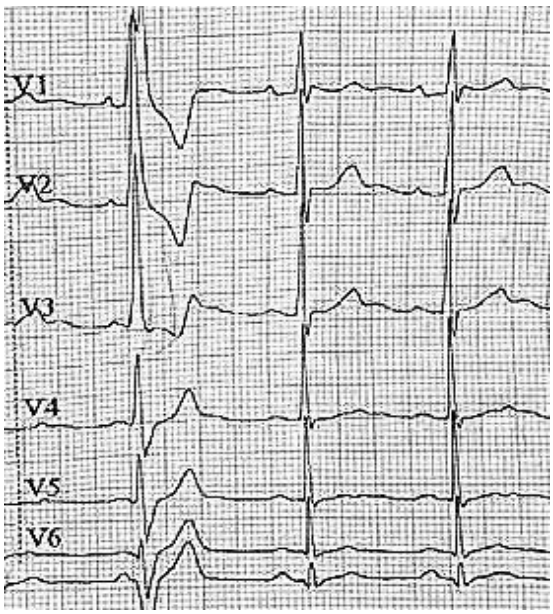

Figure 1: ST-segment elevation in V1 and ventricular extrasystoles

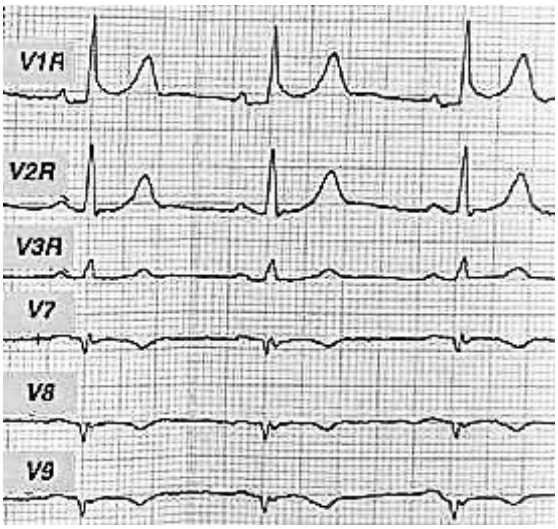

Figure 2: Posterior derivations showing an ST-segment elevation (1mm) in V1R

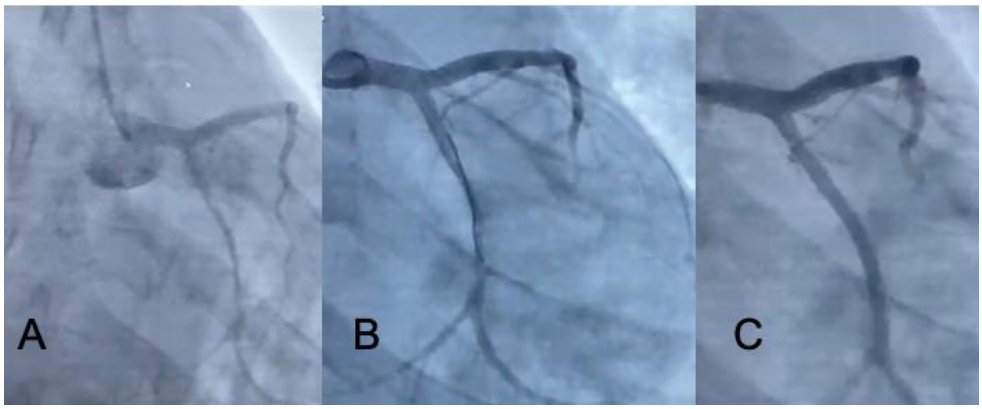

Figure 3: Significant stenosis of the mid-segment of the left circumflex artery before (A) and after stenting (B) with restarting normal coronary perfusion (C)

OBSERVATION 2

A postmenopausal woman with a medical history of type 2 diabetes mellitus presented to the emergency department with epigastric discomfort and marked fatigue that had persisted for approximately 6 hours. She did not report typical chest pain, dyspnea, or palpitations. On initial evaluation, the patient was hemodynamically stable and afebrile. Physical examination was unremarkable, with no signs of pulmonary congestion or peripheral hypoperfusion. The 12-lead electrocardiogram at admission revealed STsegment depression across the septal, anterior and lateral leads, accompanied by repolarization abnormalities including flat and inverted T waves in the same territories. Notably, a significant ST-segment elevation (>2 mm) was present in the inferior leads (DII, DIII, aVF) (Figure 4). In addition, posterior leads showed an ST elevation (1mm) in V1R and negative T waves in V7-V8V9 (Figure 5). Transthoracic echocardiography revealed regional wall motion abnormalities involving the basal and mid-lateral segments. Angiography demonstrated a tight stenosis in the mid-segment of the left circumflex artery. A drug-eluting stent was successfully implanted, with restoration of adequate distal flow. The patient remained clinically stable following percutaneous coronary intervention and showed favorable recovery without early complications.

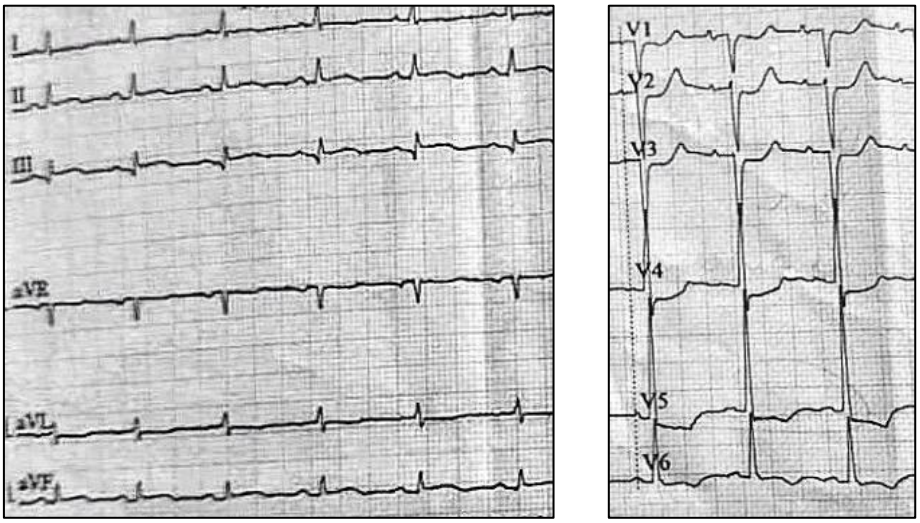

Figure 4: ST-segment depression across the septal, anterior and lateral leads and ST-segment elevation (>2 mm) was present in the inferior leads (DII, DIII, aVF)

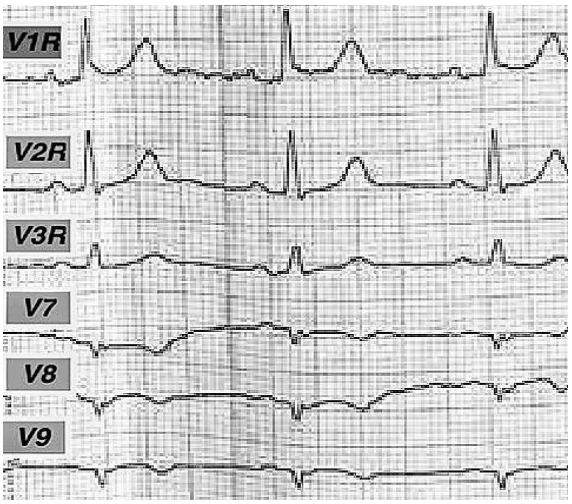

Figure 5: Posterior leads showing an ST elevation in V1R and T negative waves in V7-V8-V9

DISCUSSION

Circumflex artery occlusion remains one of the most under diagnosed causes of acute myocardial infarction due to the 12-lead ECG silent presentation [1]. Unlike anterior or right coronary occlusions, which typically present a clear ST-segment elevation, circumflex artery manifest with misleading ECG changes [2].

Multiple studies have shown that up to one-third of patients with acute circumflex occlusion may not present with ST-segment elevation in conventional leads [3]. The circumflex artery typically supplies the lateral

and posterior walls of the left ventricle, which are poorly visualized on standard leads. Huey and al. reported that significant ST-segment elevation in the lateral leads was present in only 50% of enzyme-confirmed infarctions [4]. Infarctions in these territories may manifest indirectly as: ST-segment depression in V1-V2-V3, representing a mirror image of a posterior infarct; subtle or flat/inverted T waves in lateral leads (V5V6, I, aVL); ST-segment elevation in posterior leads (V7-V9), if recorded [5].

In our first case, the absence of ST-segment elevation in the inferior or lateral leads, despite typical chest pain and elevated troponin levels, is consistent with the literature describing "electrically silent" infarctions involving the posterior myocardium. The presence of ST elevation in lead V1 and V1R, associated with negative T waves in inferolateral leads, supports the hypothesis of inferobasal ischemia. Moreover, the pattern of premature ventricular contraction suggests a left posteroseptal origin, aligning with the circumflex territory.

The second case was also misleading, with diffuse ST-segment depression in the septal, anterior and lateral leads, with a concurrent ST-segment elevation greater the 2 mm in the inferior leads. This pattern is often associated with multi-vessel disease or anterior ischemia. However, in the absence of left anterior descending artery involvement, the findings strongly suggest a posterior myocardial infarction, typically resulting from circumflex artery occlusion. The flat and inverted T waves in the lateral and apical leads support this diagnosis. The basal myocardial infarction can be associated to lateral or/and inferior myocardial infarction, as it seems to be in our two cases.

Myocardial infarction involving an occlusion of the circumflex artery is associated with wall motion abnormalities in the basal and mid-lateral segments in echocardiography [6].

This constellation of findings highlights the importance of maintaining a high index of suspicion and considering early angiographic assessment in similar clinical scenarios. In fact, compared to patients with right coronary artery occlusion, those with circumflex artery occlusion exhibited higher levels of enzymatic leakage, lower post-infarction ejection fraction, and increased short- and long-term mortality, despite a less pronounced ST-segment changes [5].

Various methods have been proposed to improve the diagnosis of circumflex artery occlusions.

The first one is the reversed lead concept [7]. Transmural ischemia generates an epicardial injury current directed toward the ischemic region. On the standard ECG, this appears as ST-segment elevation when the injury vector points toward the exploring electrode, and ST-segment depression when it points away. The reversed lead concept (used in the Cabrera presentation, – aVR instead of aVR) builds on this principle. It reorients the electrical vectors to better visualize ischemia. In a balloon inflation study in patients with stable coronary disease, Perron *et al*. demonstrated that using 7 additional reversed leads (–V1, –V2, –V3, –aVL, –I, aVR, and –III) increased the sensitivity for detecting acute coronary occlusion from 61% to 78%, while maintaining high specificity [8]. The improvement was especially marked for circumflex occlusions, with sensitivity rising from 32% to 64% [8]. This method doesn't need any physical manipulation on the patient. However, it requires a specific digital ECG system that is not yet available in all settings and accurate interpretation requires adequate clinician training.

The posterior lead concept is more practical: (V7–V9) are placed on the back to directly record electrical activity from the posterior wall of the left ventricle [9]. In a balloon-induced occlusion of the circumflex artery, Aqel and colleagues observed greater ST-segment elevation in V7–V9 compared to other leads, with an increased sensitivity for significant ST-segment elevation (74% vs. 38%) [10]. Amplitude criteria play a crucial role in diagnostic sensitivity. In a study of balloon-induced circumflex occlusions, sensitivity increased from 58% to 94% when the ST elevation threshold in leads V7–V9 was adjusted from 1 mm to 0.5 mm [7].

CONCLUSION

Electrocardiographic presentation of circumflex artery occlusion is frequently subtle and may escape recognition on the standard 12-lead ECG.

These two cases highlight the importance of maintaining a high index of suspicion when clinical symptoms are suggestive, even in the absence of classical ST-segment elevation in the standard 12-lead ECG. Incorporating posterior or reversed leads, as well as other electrocardiography indicators of ischemic involvement of the circumflex territory and early echocardiography can significantly improve diagnostic accuracy and accelerate the culprit vessel reperfusion.

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