

Figure 2. Perioperative. (a) Closure of the pseudoaneurysm using bovine pericardium. (b) Orifice of the left coronary artery with protruding implanted stent

dislocation of the stent in the left coronary artery. The operation and primary wound closure were performed successfully. The postoperative course was uncomplicated, and the patient was discharged after two weeks.

Discussion

The main question in this case is why the pseudoaneurysm occurred. The morphology of its orifice in the coronary sinus resembled a chronic aortic wall defect. The patient told us that she had undergone a cardiac catheterization several days after the closure of the ventricular septal defect because of a persistent murmur and a residual shunt. We assume that injury during this catheterization in the patient's childhood, together with the development of arterial hypertension and loss of aortic elasticity that occurred later in the patient's life led to the development of the pseudoaneurysm. The surrounding fibrous adhesions of the aorta as a result of the previous surgery may have limited the rupture of the aortic wall and prevented potentially catastrophic outcomes (5).

Conclusion

Occlusion of the coronary artery by a pseudoaneurysm can lead to myocardial infarction. In a patient with pericardial adhesions after cardiac surgery, percutaneous coronary intervention should be considered before surgical revascularization (1-3). The risk of delaying surgical treatment of the myocardial ischemia is higher than the risk of performing endovascular procedure.

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Informed consent: The patient has given informed consent to the publication of this case report, including the posting of images taken during the operation and the results of imaging methods.

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A proximal right coronary artery occlusion presenting with ST-segment depression in leads II, III, and aVF

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Introduction

The first electrocardiogram (ECG) finding during myocardial ischemia is the deviation of the ST-segment. The overall ST vector is usually shifted in the direction of the ischemic zone that can be localized by the 12-lead ECG. However, in some cases, it is subject to limitations and exceptions (1). We present a patient

with ST-segment elevation in leads aVR and V1 with diffuse ST-segment depression in other extremity leads whose right coronary artery (RCA) was occluded. The present case signifies that ECG could be limited in determining the infarct-related artery by individual variations in coronary anatomy and the presence of pre-existing conditions as collateral circulation.

Case Report

A previously healthy 45-year-old man presented to our emergency department complaining of typical cardiac chest pain, which started 1 hour before presentation. The patient was a smoker and had no family history of cardiovascular disease. His blood pressure was 110/70 mm Hg. Auscultation of the heart revealed normal heart sounds with no murmurs. The patient's initial ECG (Fig. 1)

showed normal sinus rhythm with ST-segment elevation in leads aVR, V1, and V3R through V5R with diffuse prominent ST-segment depression in other extremity leads, identifying left main or severe multivessel disease. He underwent coronary angiography that revealed a proximal total occlusion of the RCA. No significant lesions were found in the left anterior descending and the circumflex arteries. A drug-eluting stent was deployed in the RCA. The final angiographic outcome was excellent (Fig. 2). Subsequently, the patient showed symptomatic improvement, the ST changes resolved, and he was discharged 3 days later in a stable condition.

Discussion

Myocardial ischemia is a potentially life-threatening condition that occurs when myocardial blood flow decreases, which

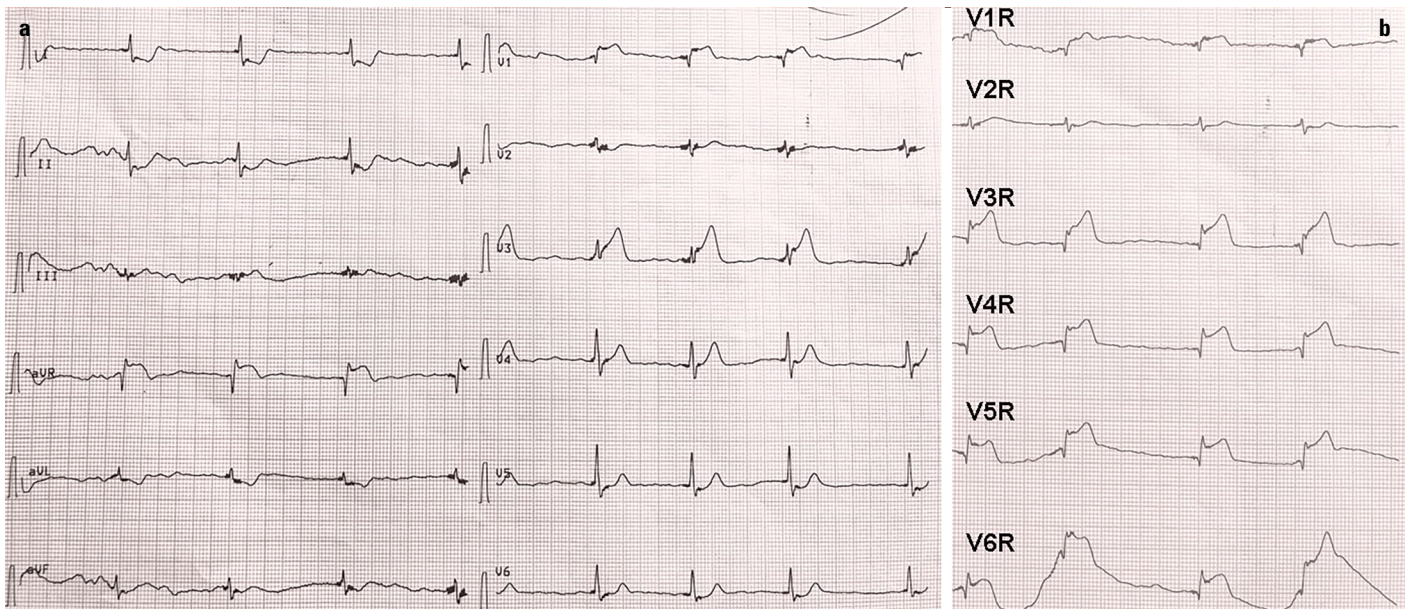


Figure 1. Patient's initial ECG. (a) 12-lead ECG, (b) Right-sided ECG

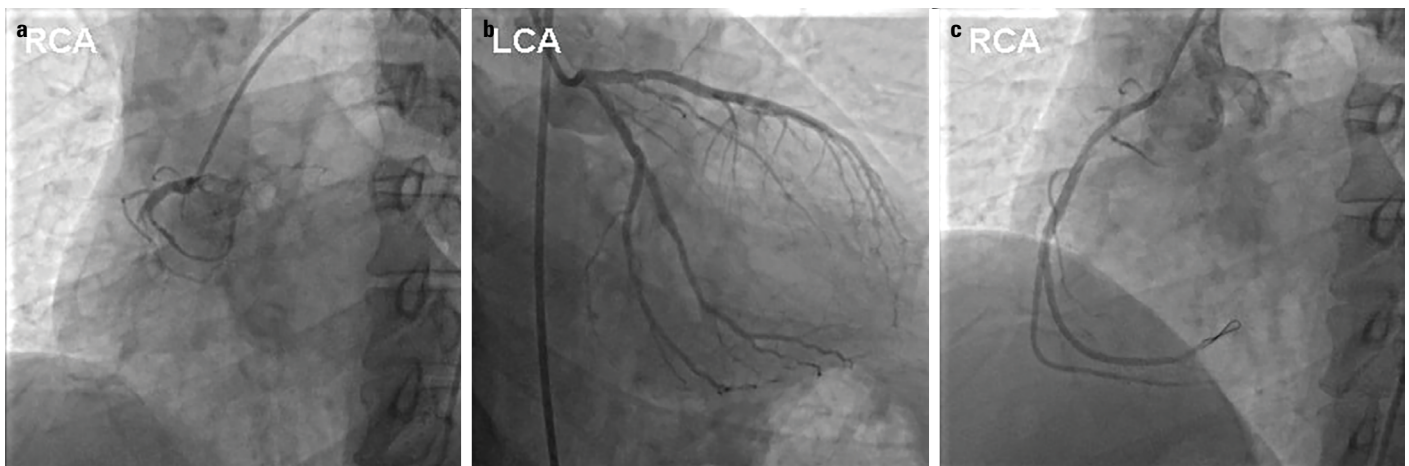


Figure 2. (a) Coronary angiography demonstrates the proximal total occlusion of the right coronary artery. (b) There are no significant lesions in the left anterior descending and the circumflex arteries. (c) The final angiographic view of the right coronary artery

leads to electrophysiological changes within the heart. Attenuated blood flow within ischemic tissues causes a reduction in adenosine triphosphate production, which leads to depressed Na^+/K^+ and Ca^{++} pump activity—resulting in altered local ionic concentrations, those produce subsequent changes in tissue electrophysiology, such as reduced action potential amplitude, decreased action potential duration, and less-negative resting membrane potential. The resulting extracellular potential differences between healthy and ischemic fields produce deviated ECG recordings that are used as markers for detecting myocardial ischemia (2, 3). The first and most consistent finding is the deviation of the ST-segment. When acute ischemia is transmural, the overall ST vector is usually shifted in the direction of the epicardial layers, and ST-segment elevation is produced over the ischemic zone that can be localized by evaluating the 12-lead ECG. For example, if ST-segment elevation is detected in two or more contiguous precordial leads (V1 through V6), anterior wall ischemia is diagnosed, while inferior wall ischemia induces ST-segment elevation in II, III, and aVF leads. Right-sided ST-segment elevation demonstrates the acute right ventricular infarction which also projects ST-segment elevation in leads V1 through V3 or V4, simulating anterior infarction. However, these criteria will always be subject to limitations and exceptions based on variances in coronary anatomy, collateral flow, the dynamic nature of acute ECG changes, and the presence of ventricular conduction delays (1).

On the other hand, ST-segment elevation in lead aVR may indicate a more complicated condition. Left main or severe multivessel disease should be considered when leads aVR and V1 display ST-segment elevation, particularly in concert with diffuse pronounced ST-segment depression in other leads. The 2017 European Society of Cardiology Guideline has emphasized ST-segment elevation in aVR as a potential ECG finding of left main or proximal left anterior descending artery occlusion (4). However, previous papers revealed that, although rare, ST-segment elevation in lead aVR could occur in unique clinical scenarios. Lin et al. reported a case of a 73-year-old man presenting with ST-segment elevation in lead aVR whose circumflex artery was occluded (5). Harhash et al. (6) described the likelihood of culprit lesion in RCA as 5% in acute coronary syndrome patients with ST-segment elevation in lead aVR. However, it was not clear whether RCA was occluded or there was a subocclusive disease with intact distal flow in those patients (6).

In the case of RCA occlusion, ECG generally displays ST-segment elevation in inferior wall leads (II, III, and aVF) with reciprocal ST-segment depression in V1–V3. When RCA is occluded proximal to right ventricular branches, an ST-segment elevation may be seen not only in inferior leads but also in leads from V1 to V3–V4, with greater ST-segment elevation in V1–V3; however, ST-segment elevation in V1 is usually greater than that in V3–V4 ($V1 > V3$ or $V4$). The lack of apparent ST-segment depression in V1–V3 may also be observed due to a very distal occlusion of a nondominant RCA (7). In those cases, because the myocardium

at risk of ischemia is small, the ST-segment elevation is not apparent in II, III, and aVF. In addition, rarely, atypical ECG presentations related to RCA occlusion have also been reported in the literature. As an example, one study reported a case where a proximal RCA occlusion produced not only anterior ST-segment elevation, but also anterior T-wave inversions and anterior Q waves (8).

Here, we present a case of a 45-year-old man whose RCA was occluded, however, ECG surprisingly revealed ST-segment elevation in leads aVR and V1 with ST-segment depression in I, II, aVF, and aVL. How should we interpret this finding?

After a careful evaluation of the patient's angiography, a poor retrograde flow was noted in the patent ductus arteriosus (PDA) on the anterior–posterior cranial view of the left coronary artery (Fig. 3). Thus, ischemic vector shifting could have been the underlying mechanism of the ECG findings. The retrograde flow, not reaching the right ventricular branch but supplying blood to the distal portion of the RCA, resulted in an ischemic vector that directed to the right upper quadrant (Fig. 4) (8).

Conclusion

The main learning point of this case is despite the fact that ECG has been the gold standard in localization of ischemic zone, there may be some exceptions as was the case in which RCA total occlusion was presented with ST-segment depression in leads II, aVF, and aVL. In such cases, the presence of collateral flow and individual variations in coronary anatomy should be carefully examined.

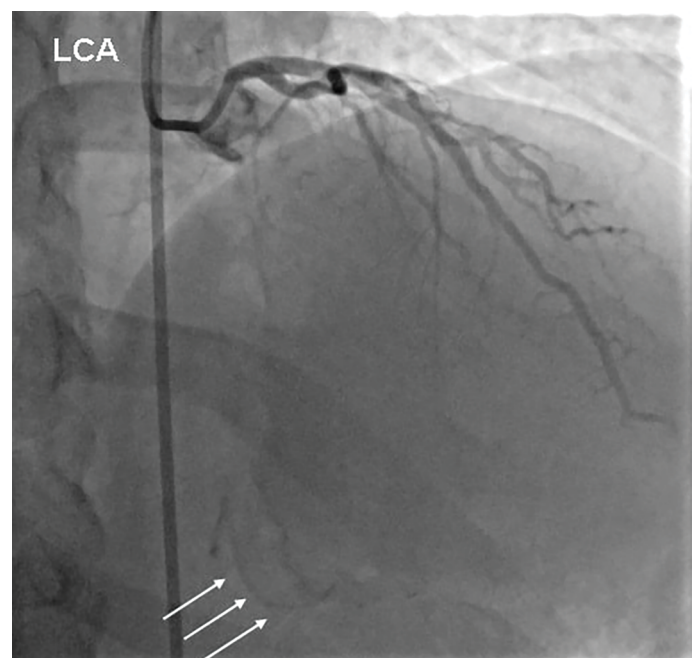


Figure 3. A poor retrograde flow (arrow) is noted in the PDA on the anterior–posterior cranial view

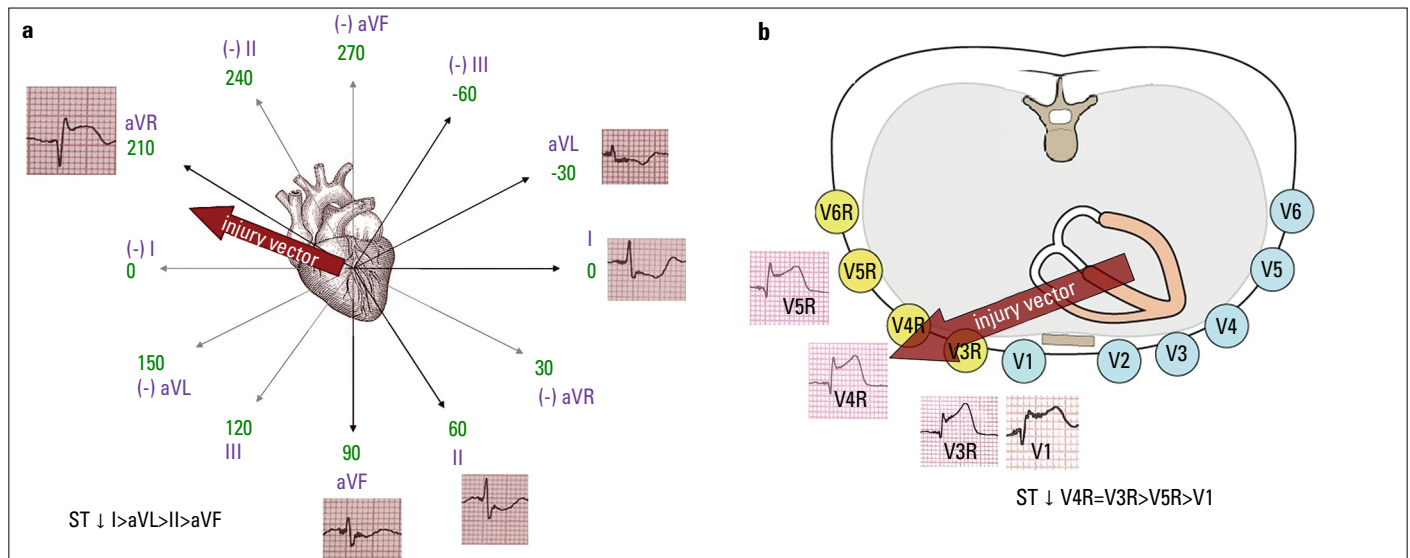


Figure 4. Schematic representation of the ischemic injury (a) in the frontal plane, and (b) in the horizontal plane. The arrow points toward the point of maximal ST elevation and away from the point of maximal ST depression

Informed consent: Written informed consent to publication was obtained from the patient.

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Atypical localization, atypical clinical course, unexpected age: Myxoma

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Introduction

The incidence of cardiac tumors in infants and the pediatric population is 0.08% (1). Rhabdomyoma is the most common tumor in the neonatal period (1, 2). However, myxoma is the most common primary tumor that is frequently localized in the left atrium in the adult population. The incidence of myxoma in the pediatric population is 20.2%. In fact, myxoma equally affects both atria during the pediatric ages (3).

Case Report

Our patient was born at the age of 32 weeks via elective cesarean section. She was followed-up in the neonatal intensive care unit for one month because of prematurity complications. After one month without any symptoms after discharge, she was