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Case Report

Spontaneous Coronary Pseudoaneurysm Presenting As Non-St-Segment Elevation Acute Coronary Syndrome

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Abstract

Coronary pseudoaneurysm is an extremely rare condition. We present the case of a 77-year-old patient with myocardial ischemia caused by extrinsic compression of left main coronary artery due to a pseudoaneurysm of the circumflex artery. In our case, pseudoaneurysm was successfully treated with cardiac surgery.

Keywords: Acute Myocardial Infarction; Coronary Pseudoaneurysm; Cardiac Surgery

Case presentation

We present the case of a 79-year-old male who is former smoker, with a medical history of dyslipidemia and past treatment for paroxysmal atrial fibrillation by pulmonary vein isolation nine years earlier. As a part of the pre-ablation assessment, a cardiac MRI was conducted, revealing an oval structure measuring 27 by 20 mm, in proximity to the left atrium, left atrial appendage and circumflex artery. However, its precise characterization was not possible at that time.

No other cardiac conditions were noted in the patient's clinical history. The current treatment was bisoprolol and acenocoumarol maintaining INT within target range.

The patient presented to the emergency department complaining of oppressive chest pain, which lasted for one hour and subsided upon arrival at the emergency department. The pain did not radiate and was not associated with vegetative symptoms. He reported experiencing recurrent and transient episodes of retrosternal oppressive chest pain over the past ten days, occurring both during physical exertion and at rest, along with exertional dyspnea. He denied having fever or any other cardiovascular or infectious symptoms.

On arrival at the emergency department, the patient was hemodynamically stable, he had normal vital signs and his physical examination was unremarkable with no signs of overt heart failure. He was eupneic, with no need for oxygen therapy.

The initial electrocardiogram showed sinus rhythm with normal PR segment, narrow QRS with incomplete right bundle branch block and no signs of acute ischemia. Sequential ECG did

not change (Figure 1). Laboratory findings included an elevation of high-sensibility cardiac troponin I up to 498 ng/L (N<45 ng/L). The rest of the blood tests (hemogram, renal function, ionogram) were normal.

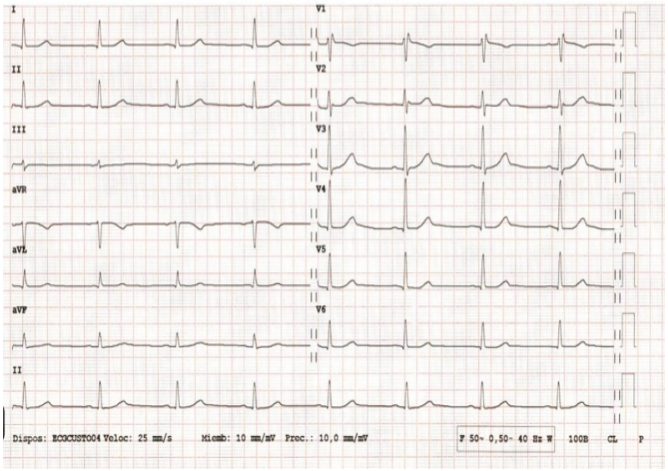

Figure 1: ECG: sinus rhythm at 50 bpm with normal PR, narrow QRS with incomplete right bundle branch block and no repolarization alterations.

An echocardiogram was performed which showed a non-dilated and mildly hypertrophic left ventricle, with a normal ejection fraction despite lateral hypokinesia (LVEF 55%) and no pericardial effusion.

The patient was classified as Killip I class Non-ST-Elevation Myocardial Infarction (NSTEMI), monoantiplatelet therapy with ASA and anticoagulation with enoxaparin was started, and coronary angiography was requested.

Coronary angiography (Image 2) showed right dominance with mild non-obstructive coronary artery disease in the main epicardial arteries. However, a contrast leakage was seen from the proximal Left Circumflex Artery (LCx) to an adjacent round structure causing displacement and compression of the left main and proximal LCx artery (Figure 2, panel A and B). Subsequently, a multi-slice Coronary Computed Tomography Angiography (CCTA) (Figure 2, panel C and D) was ordered to better characterize the nature of the lesion and to describe its location related to other cardiac structures. CCTA confirmed the leakage of contrast iodine from a calcified plaque lesion in the LCx artery into the periphery of a rounded, well-defined and encapsulated extravascular cavity measuring 45 by 40 by 38 mm.

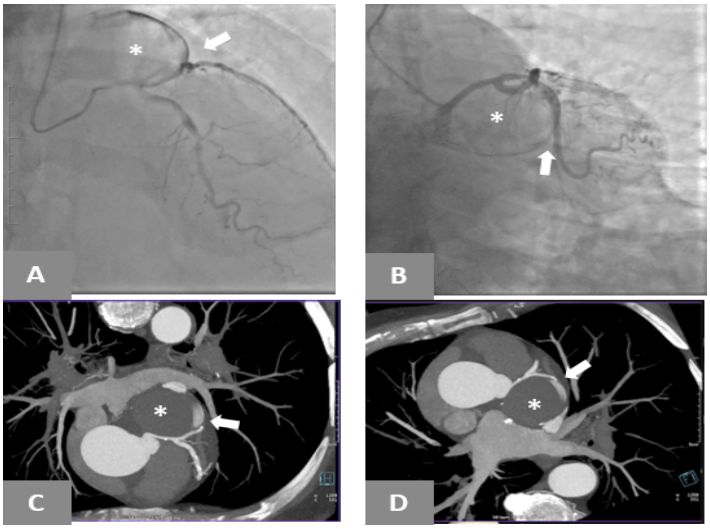

Figure 2: Multimodality imaging. Cranial and right-oblique frame projections from the coronary catheterization (panels A and B) and corresponding Maximal Intensity Projection Frames from the CCTA (panels C and D). The location of the pseudoaneurysm is labeled with an Asterisk (*) and the origin of the ruptured calcified plaque in the proximal LCx artery causing contrast leakage is labeled with a white arrow. CCTA: Coronary Computed Tomography Angiography. LCX: Left Circumflex.

The signal intensity in the lesion was slightly heterogenous, showing an attenuation of 75±31 Hounsfield Units, consistent with a thrombus. No signs of malignancy were observed. A tentative diagnosis of coronary pseudoaneurysm arising from the LCx causing extrinsic compression of the left main and proximal segment of the LCx as the cause of myocardial infarction was made.

Comparing the images with the cardiac MRI performed before the pulmonary vein isolation confirmed the presence of the structure at that time, so it was ruled out its relationship with the procedure. However, its previous size was notably smaller (27 by 20 mm).

Based on the above, given the suspicion that compression of the pseudoaneurysm on the left coronary artery tree was the cause of NSTEMI, it was decided to undergo cardiac surgery. The EUROSCORE 2 score predicted low operative risk. A cavitated lesion arising from a calcified plaque in the proximal LCx was confirmed during surgery (Figure 3), requiring an aneurysmectomy and saphenous vein graft to the obtuse marginal branch (Video 1). Surgical findings matched those on the CCTA.

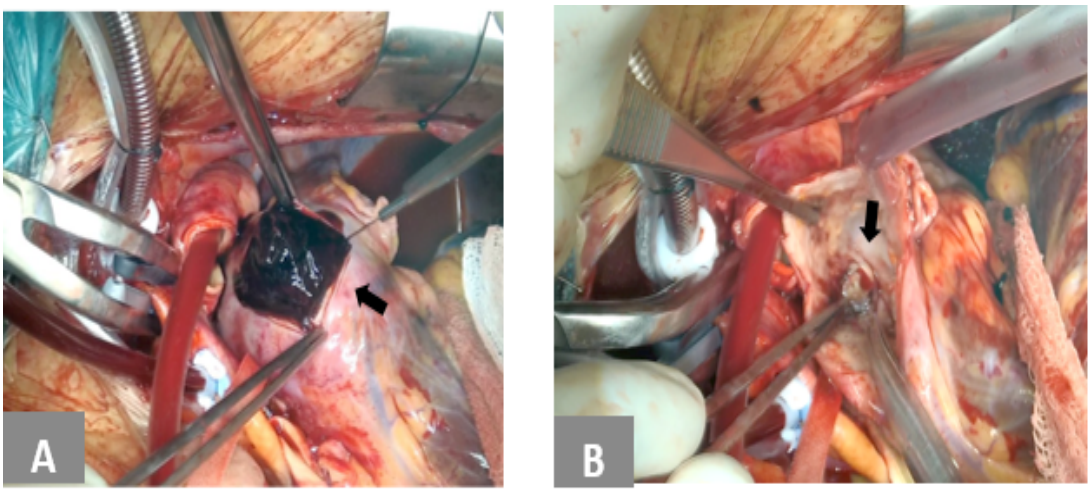

Figure 3: The pseudoaneurysm was opened, fresh thrombus removed (A, black arrow). A ruptured calcified plaque in the LCx artery was identified in the posterior sac (B, black arrow).

The intraprocedural transesophageal echocardiogram (Figure 4) confirmed the correct resection of the pseudoaneurysm.

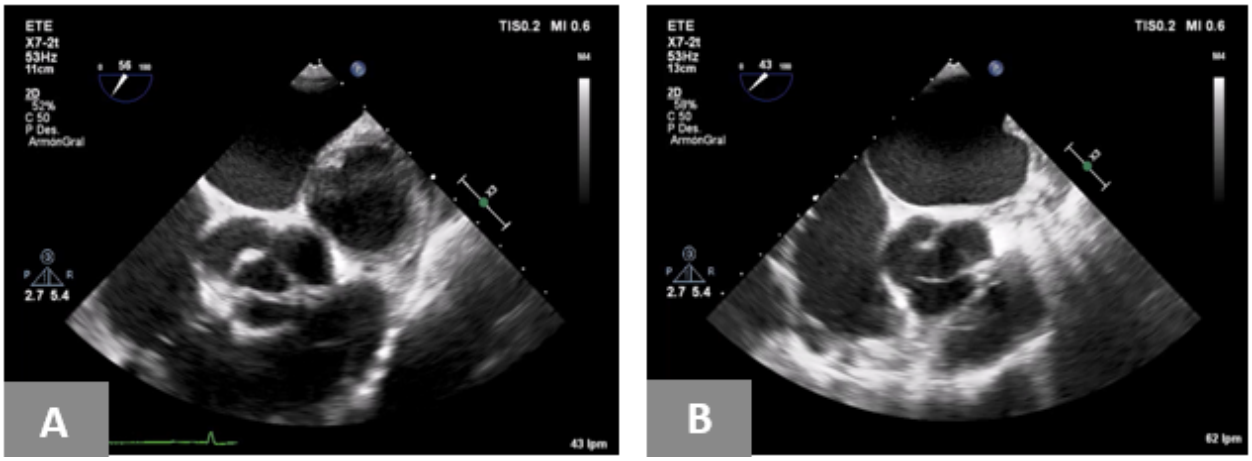

Figure 4: Transesophageal echocardiogram in high esophageal plane at 43-56º: A. Giant pseudoaneurysm B. It shows the absence of the pseudoaneurysm with postresection cicatricial changes.

The sample referred to pathological anatomy showed the existence of a fibromuscular wall with reparative changes and little inflammatory component, consistent with the diagnosis made of coronary pseudoaneurysm.

After the intervention, the patient had a good evolution in the ICU with early extubation and withdrawal of vasoactive agents. Forty-eight hours after the intervention, the patient was transferred to the hospital ward, where the drainages were removed. The pre-discharge echocardiogram showed preserved biventricular function without pericardial effusion and he was discharged home on the eighth day after the intervention.

At 2 months follow-up, the patient had no new episodes of angina or cardiovascular events.

Discussion

Coronary aneurysm is a rare condition defined as a dilatation in a specific section of a coronary artery, that is more than 1.5 times the adjacent normal coronary artery. Coronary ectasia is also defined as a dilatation of more than 1.5 times larger with respect to the adjacent normal segments, but it is a diffuse dilatation, as opposed to a coronary aneurysm which is a short and focal expansion [1].

True coronary aneurysm is characterized by having all three layers of the arterial wall (intima, media and adventitia), unlike pseudoaneurysm which lacks of at least one of the layers and consists of a single or double layer that extends outward, and which develops from loss of vessel wall integrity [2-4]. Aneurysms are classified as small (x1.5-2), large (x2-5) or giant (>x5) according to the diameter over the reference vessel [5] and our case could be classified as giant.

The most frequent cause of aneurysms is atherosclerosis. Other causes include inflammatory diseases (Kawasaki, Behcet's disease), infections (septic, syphilis), connective tissue diseases (Marfan syndrome, Ehlers-Danlos syndrome), drug-related, traumatic or iatrogenic [2-5]. On the other hand, pseudoaneurysms most commonly occurs from arterial dissection or perforation of the coronary artery after a catheter intervention. In our case, no iatrogenic cause was found, given that the presence of the pseudoaneurysm preceded the interventional treatment of pulmonary vein ablation, and the patient had not undergone any other procedures.

Clinically, most coronary pseudoaneurysms are asymptomatic and a casual finding on coronary angiography. However, they can cause heart failure or acute coronary syndrome due to compression of adjacent structures or even hemodynamic instability due to cardiac tamponade caused by its rupture [6].

Diagnosis can be made by noninvasive techniques such as cardiac CT angiography or cardiac magnetic resonance imaging, whose main advantage is the low risk associated with them, or by invasive techniques such as coronary angiography or Intracoronary Ultrasound (IVUS), which are considered the "gold standard" and can even be therapeutic [3].

Treatment depends on the clinical condition and the size of the pseudoaneurysm and can be either percutaneous with stents or coils or surgical with exclusion of the pseudoaneurysm. In asymptomatic patients with small pseudoaneurysms, follow-up with periodic imaging may be considered. However, in the case of large or symptomatic pseudoaneurysms, such as ours, percutaneous or surgical treatment may be recommended to alleviate symptoms or prevent the risk of progression or rupture [6].

Conclusions

We have presented a rare case of acute coronary syndrome due to left coronary artery compression by a coronary pseudoaneurysm originating from the circumflex artery. Despite initial hemodynamic stability, the patient experienced recurrent chest pain episodes. Our case highlights the role of multimodality imaging in identifying this complication of atherosclerotic coronary artery disease requiring specific surgical repair.

Disclosure

Author Contributions: Conceptualization, J.P., I.A., S.B and J.T.O.; writing—original draft preparation, J.P. and I.A; writing—review and editing, J.P., I.A., B.R., B.D., S.B., J.A and J.T.O; supervision, S.B., J.A and J.T.O.

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Conflicts of Interest: None.

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