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CASE REPORT

CARDIOLOGY // IMAGING

Acute Thrombotic Coronary Occlusion in a Patient with Coronary Artery Anomaly

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ABSTRACT

Patients with coronary artery anomalies are more susceptible to develop acute thrombotic coronary occlusions due to the abnormal anatomy of these arteries and the disturbance of the pathophysiological mechanisms that lead to an accelerated atherosclerosis development. The following article presents the case of a 64-year-old female patient diagnosed with anterior ST-segment elevation myocardial infarction. The patient underwent primary percutaneous coronary intervention, which revealed the absence of the right coronary artery and separated origins of the left anterior descending artery and the left circumflex artery from the aorta.

Keywords: coronary artery anomalies, acute thrombotic occlusion, STEMI, PCI

INTRODUCTION

It is difficult to define what is the normal anatomy of the coronary arteries.[1] In most individuals they originate as the right and left main coronary arteries, from the coronary ostia in the ascending aorta, just above the aortic valve.[2] Variations in coronary anatomy are often discovered in association with structural congenital cardiac disease. Coronary artery anomalies (CAAs) are a group of congenital disorders whose pathophysiological mechanisms and clinical signs are highly variable. CAAs are classified as major, severe, or important, with varying hemodynamic significance, according to the clinical impact on the patient.[3] Anomalies of the coronary arteries are rare, the incidence varying from 0.3% in necropsy studies to 1.6% in patients undergoing invasive coronary catheterization.[4] The most common coronary artery anomaly is a separate origin of the left anterior descending (LAD) and the left circumflex (LCX) artery, with an incidence of 0.41%.[1] Although these anomalies are rare, they may be seen with myocardial perfusion abnormalities and an accelerated atherosclerosis. Because of these abnormal mechanisms, these patients are highly exposed to myocardial infarction and ischemia.

Invasive coronary angiography is a highly specific method for evaluating the presence and characteristics of CAAs; however, it has the important disadvantage of being an invasive method. Today, CAAs can be evaluated using mod-

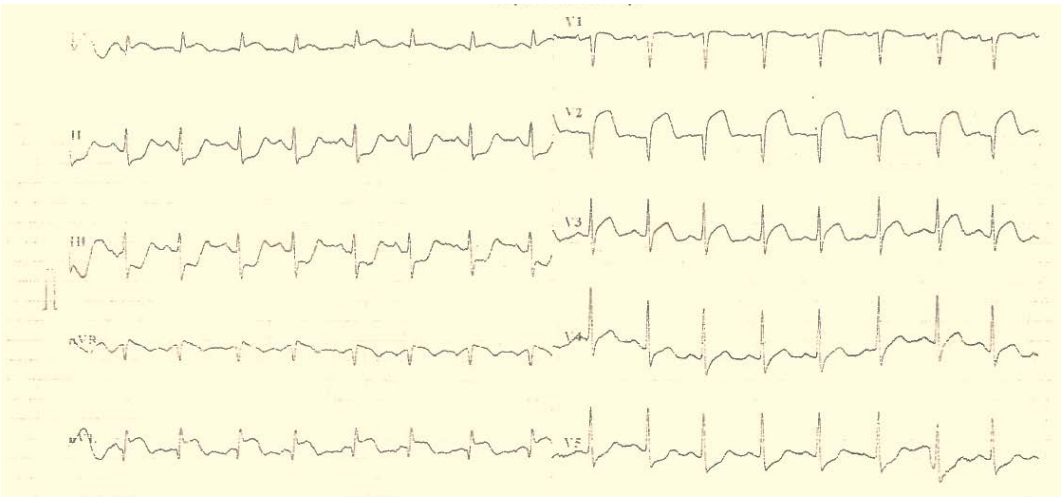

FIGURE 1. ECG-tracing in the Emergency Department showing sinus rhythm, heart rate of 100 beats per minute, ST segment elevation in leads V1–V3 and aVL, ST segment depression in leads V4–V6, DII, DIII, aVF — suggestive for acute myocardial infarction

ern, noninvasive imaging techniques. Coronary magnetic resonance (CMR) and multi-slice computed tomography (MSCT) broadened the clinical information by enabling visualization of the coronary anatomy.[5] The main disadvantage of these methods, compared to invasive coronary angiography, is that they are only diagnostic methods, while during the invasive procedure, an interventional treatment can also be applied.

The management of ST-elevation myocardial infarction (STEMI) depends on the time from the onset of symptoms, which should be less than 12 hours, although percutaneous coronary intervention (PCI) has better benefits if it is performed within the first 2–3 hours from the onset of symptoms.[6]

CASE PRESENTATION

A 64-year-old female patient, with known arterial hypertension controlled with anti-hypertensive treatment, presented in the emergency department complaining of the following symptoms: angina, dyspnea, and diaphoresis. The angina debuted in the last 12 hours. The patient declared that she was never a smoker, and she had no family history of cardiovascular disease. It was the first time in her life when she had a chest pain. The clinical examination did not reveal any particularity. The blood pressure upon presentation was 120/70 mmHg, and the heart rate was 80 beats per minute and regular. The patient's symptoms were suggestive for an acute coronary syndrome, and the electrocardiographic tracing (Figure 1) showed ST-segment elevation in the anterior leads.

Laboratory testing detected a major rise in troponin I levels, with a value of 23 ng/ml. Other enzymes that express myocardial cell necrosis (Table 1) were also highly increased, suggesting a massive cytolysis in the myocardium.[6]

The elevated levels of cardiac biomarkers, together with the ischemic symptoms and the ST-segment changes, confirmed the diagnosis of anterior ST-segment elevation myocardial infarction, Killip I class, for which the patient underwent emergency invasive coronary angiography.

After the administration of dual antiplatelet therapy (Aspirin and Clopidogrel) and unfractionated heparin, the inva-

TABLE 1. Laboratory tests in the Emergency Department

Analysis	Value	Reference Value
Troponin I (ng/mL)	23.8	0.000
Creatine kinase (IU/L)	5,051	192
Glutamic-oxaloacetic transaminase (IU/L)	636	0–40
Glutamic-pyruvic transaminase (IU/L)	100	10–50
Lactate dehydrogenase (IU/L)	1,199	220–460

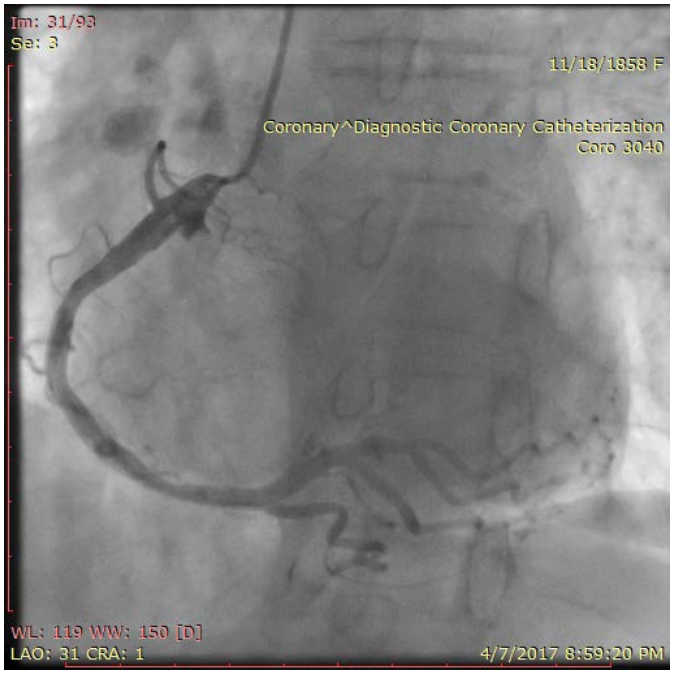

FIGURE 2. Total occlusion of the LCX in the proximal segment

FIGURE 3. Thrombectomy with thrombus aspiration

sive coronary angiography was performed; this showed the absence of the right coronary artery and separated origins of the LCX and LAD coronary arteries. The LCX presented an acute thrombotic occlusion at the origin (Figure 2), and the LAD presented a 90% stenosis in the third segment.

The interventional treatment included thrombectomy with thrombus aspiration (Figure 3) in the LCX, revealing the culprit lesion (Figure 4) for which a percutaneous coronary transluminal angioplasty with drug eluting stent was performed, with an optimum post-procedural result, TIMI III flow grade. Following primary PCI, the LAD lesion was approached with a guidewire, followed by a bare metal stent implantation, with a TIMI III flow grade (Figure 5).

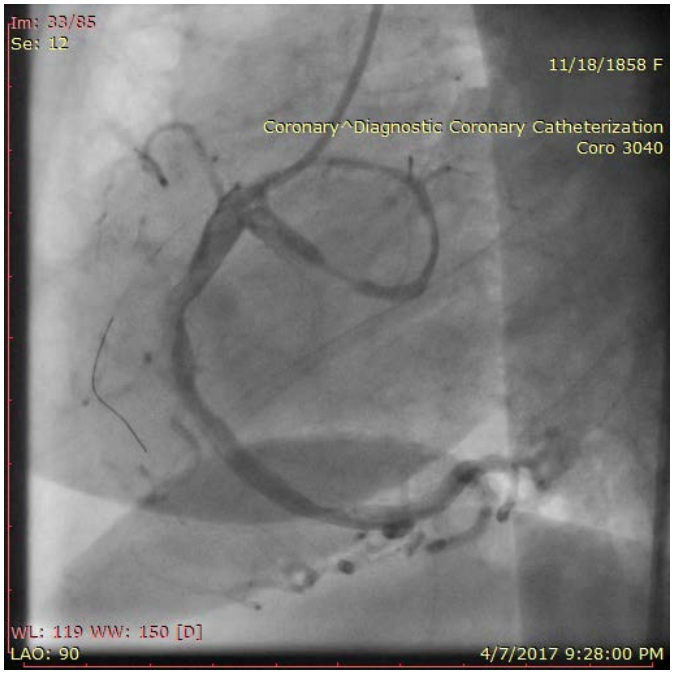

FIGURE 4. The culprit lesion in the LCX after the thrombectomy was performed

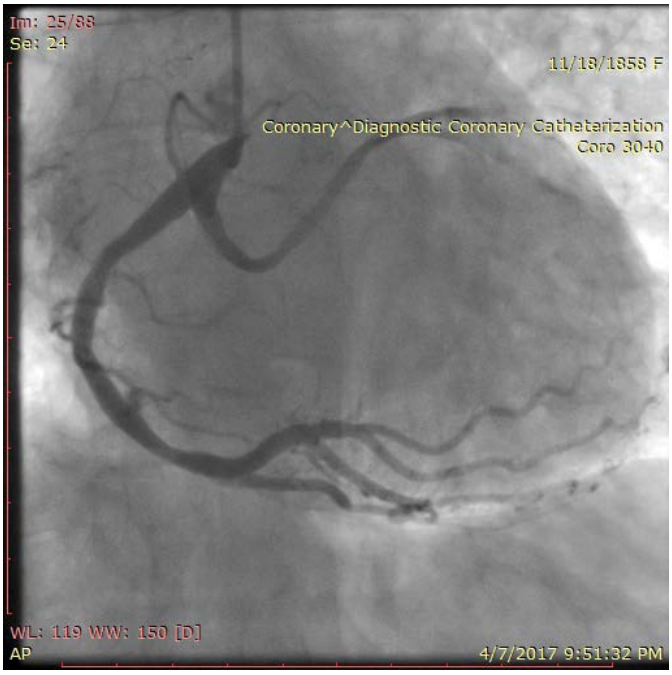

FIGURE 5. The final angiographic aspect after PCI

After the procedure, the patient presented improvement of the symptoms and >50% resolution of the ST-segment elevation. The patient received pharmacological treatment with heparin, dual antiplatelet therapy, beta-blockers, and statins and was discharged after 7 days of hospitalization.

The patient agreed to the publication of his data and the institution where the patient had been admitted, approved the publication of the case.

DISCUSSIONS

In a patient with a unique coronary artery anomaly, the impact of myocardial infarction by acute coronary thrombotic occlusion and the risk of sudden death are higher than in the general population. The management of a patient with coronary artery anomalies and myocardial infarction depends on the time from the onset of symptoms, which has to be as small as possible.[7] In this case, the time between symptom onset and PCI was less than 12 hours. The objectives of an invasive coronary angiography are to locate the culprit lesions that determined the myocardial infarction or ischemia and to treat these lesions by PCI with stent implantation or balloon angioplasty. In case of chronic total occlusion, a non-emergency high-risk PCI procedure — preferably on-site cardiovascular surgery — is required.[7]

Almost 20% of coronary artery anomalies are found while performing diagnostic procedures for a myocardial infarction, malignant arrhythmia, or Adam Stokes syncope, while 80% are benign and usually do not determine symptoms.[8]

CONCLUSIONS

Although the patient did not have major cardiovascular risk factors except for the high blood pressure (which was controlled with medication) and her gender, she presented a myocardial infarction by total coronary occlusion. Although coronary artery anomalies are a very rare group of disorders, such patients are highly exposed to a risk of extensive myocardial infarction with a higher rate of acute complications or sudden death. It is important for these patients that the anomalies are diagnosed when they are asymptomatic for a better therapeutic management.

CONFLICT OF INTEREST

Nothing to declare.

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