

CARDIOGENIC SHOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION: SIMULTANEOUS THROMBOSIS IN TWO VESSELS IN A PATIENT WITH CORONARY ANOMALY

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Date Received: March 12, 2019

Date Revised: May 03, 2019

Date Accepted: May 19, 2019

Contribution

GEPB, XERA and SPGC conceived the idea of the case report. FJVA, JOMV, LI and PC collected the pictures. DAIM and AECE finalized case report. RP did final review. All authors contributed equally to the submitted case report.

All authors declare no conflict of interest.

This article may be cited as: Baztarrica GE, Aguilar XER, Cantos SPG, Aguirre FJV, Vega JOM, Cendeno LIP, Montesdeoca DAI, Espinoza AEC, Porcile R. Cardiogenic shock complicating acute myocardial infarction: simultaneous thrombosis in two vessels in a Patient with coronary anomaly. Pak Heart J 2019; 52 (03):274-6

ABSTRACT

Simultaneous thrombosis of more than one coronary artery is an uncommon coronary angiographic finding in acute ST-segment elevation myocardial infarction, and usually associated to cardiogenic shock or even sudden cardiac death. The right coronary artery (RCA) originating from the left anterior descending artery (LAD) is a very rare variant. We presented a 48-year-old male who had a blood hypertension, smoking, and dyslipidemia history was hospitalized due to acute myocardial infarct with cardiogenic shock. Coronary angiography revealed an aberrant RCA originating from the mid-LAD with thrombosis in both arteries. A successful percutaneous coronary intervention was performed and he was discharged from the hospital tenth days after admission.

Key Words: Coronary anomaly; Acute myocardial infarction; Acute coronary syndrome; Percutaneous coronary intervention, Cardiogenic shock.

INTRODUCTION

The incidence of coronary artery anomaly is about 0,2- 1.4% in the largest reported series.¹ Anomalous origin of the right coronary artery (RCA) arising from the left anterior descending artery (LAD) is a very rare coronary anomaly and discovered incidentally during a coronary angiography. It is known that coronary artery anomalies can be related to coronary ischemia and sudden cardiac death. We present here a rare presentation of a patient with an acute anterior and inferior myocardial infarction who developed cardiogenic shock by multi-vessel thrombosis. Coronary angiography revealed an aberrant RCA originating from the mid-LAD with thrombosis in both arteries.

CASE REPORT

This is the case of a 48-year-old male with history of hypertension, smoking, and dyslipidemia. Upon admission, the patient had angina pectoris symptoms. HR: 80'. BP: 70/40 mmHg. Hypoperfusion signs were manifested clinically by cool extremities, decreased urine output, and alteration in mental status. Electrocardiogram indicated ST-segment elevation in DII-DIII-aVF and V1-V4 derivations (Figure 1A). As regards the enzyme curve, troponin T=834 ng/L, peak creatine phosphokinase (CPK) was 2180 U/L and CPK Isoenzymes MB = 226 U/L (upon admission).

Figure 1A: Electrocardiogram on admission and before discharge.

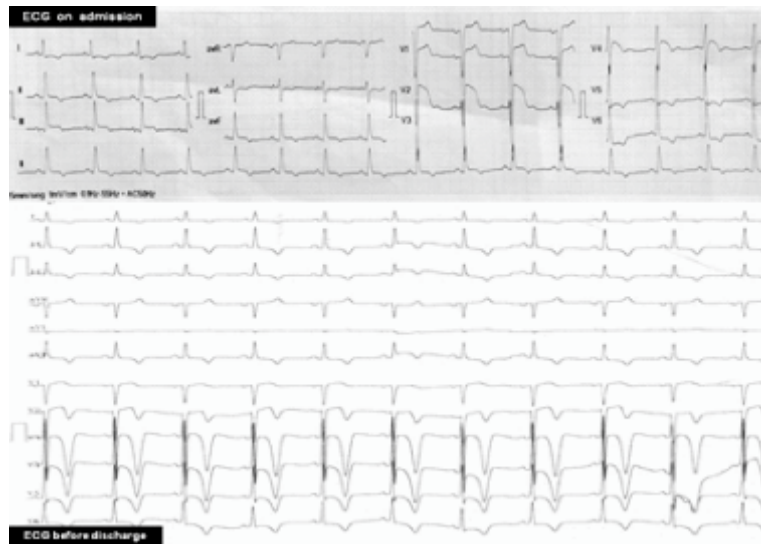


Figure 2: A) Coronary angiography projection showing the origin of the anomalous RCA (white arrow) and thrombosis total of the mid-LAD artery (black arrow). B) Coronary angioplasty: stent (black arrow) was implanted successfully at the stenosis of the LAD coronary artery beyond the origin of the RCA (white arrow). Proximal RCA was treated with balloon but the coronary angiography showed total occlusion of the distal third of the RCA (asterisk).

LMA: left main artery; LAD: left anterior descending; CXA: circumflex artery; RCA: right coronary artery.

Figure 2a:

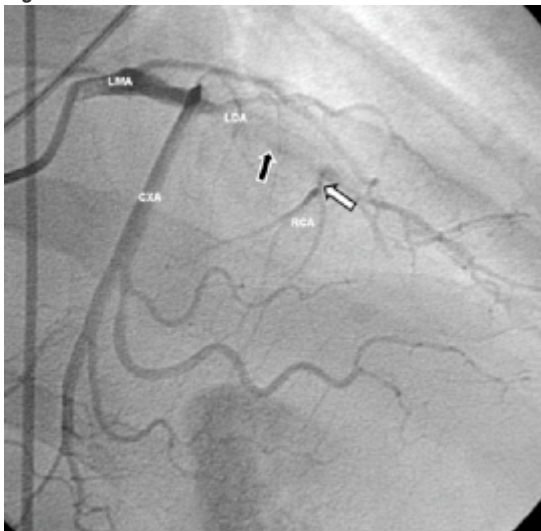


Figure 2b:



According to the clinical presentation, the patient required inotropic agents (dobutamine $10\mu\text{g}/\text{kg}/\text{min}$) and intra-aortic balloon pumping. Coronary angiography was performed and showed occlusion with a large thrombus load of the LAD coronary artery and of RCA, originating from the mid-LAD (Figure 1).

Considering the patient's clinical condition and the coronary angiography results, after abciximab, angioplasty and stenting of the LAD was performed. The thrombus containing lesion of RCA was treated with balloon angioplasty. This artery was had a path ahead of the pulmonary artery until it reaches the atrioventricular groove on the right side, where it assumes its usual path, but the coronary angiography showed total occlusion of the distal third of the RCA (Figure 2 A). Electrocardiogram before discharge indicated QS DI-aVL and T negative in V1-V6 derivations (Figure 2 B). The transthoracic echocardiogram showed hypokinesis of the inferior and anterior wall with severe dysfunction left ventricular.

The patient progressed with no symptoms during hospitalization and had no others complications and the discharge from hospital occurred on the tenth day. The patient is currently asymptomatic (echocardiography control showed hypokinesis septum anterior and inferior wall with mild dysfunction left ventricular).

DISCUSSION

The mechanisms suggested for ischemia in coronary anomalies include external compression, acute angulation, direction, torsion, spasm, and atherosclerotic obstruction (higher incidence of atherosclerosis in patients with coronary anomalies than no anomaly, due to an uncertain mechanism). To these are added the classic cardiovascular risk factors of each patient (in our patient, hypertension, smoking and dyslipidemia).^{2,3}

Although most of coronary anomaly patients are asymptomatic, it is well known that coronary artery anomalies can be associated with chest pain, dyspnea, palpitations, syncope, ventricular fibrillation, myocardial ischemia and sudden cardiac death.¹⁻⁴

The form of presentation of cardiac events depends on several factors: the course of the aberrant artery, location and type of affection. We described the case of a patient presented with acute inferior and anterior myocardial infarction (ischemic involvement of two vessels, LAD and RCA) associated cardiogenic shock that was found to have a very rare congenital abnormality consisting of an origin of the RCA from the mid-segment of the LAD. The extensive ischaemia induces profound depression of myocardial contractility, which initiates a vicious spiral of reduced cardiac index and low blood pressure which in combination impair cardiac power index and further promote coronary ischaemia. Irrespective of early revascularization, the basic treatment measures include initial stabilization with volume expansion to obtain euvolaemia, inotropic therapy, preferably with dobutamine plus additional therapy with active percutaneous left-ventricular assist devices (in our case, we were used dobutamine plus intra-aortic balloon pump).

Timely and complete restoration of flow in infarct-related artery helps to reduce infarct size, preserve left ventricular function, and improve survival rates in patients with acute myocardial infarction in patients presenting with ST-segment elevation and coronary anomalies. The angioplasty is more difficult to perform in

anomaly patients than in patients without coronary anomaly.^{4,5} Although our patient presented an acute myocardial infarction with cardiogenic shock (left ventricular with severe dysfunction), the rapid reperfusion prevented the evolution to myocardial necrosis and reduced the morbidity and mortality in-hospital and at six months.

CONCLUSION

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 by multi-vessel thrombosis (in our case presented LAD and RCA thrombosis with cardiogenic shock) or sudden death. The therapeutic management of acute myocardial infarction in patients presenting with ST-segment elevation is similar in anomaly patients than in patients without coronary anomaly, but percutaneous coronary intervention is more difficult to perform.

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