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Case Report

Simultaneous total occlusion of the 3 major coronary arteries in a patient with inferior ST-segment elevation myocardial infarction complicated by cardiogenic shock and sinus bradycardia ☆,☆☆

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ABSTRACT

Simultaneous occlusion of more than one coronary artery is uncommon and associated with poor prognosis. We reported a particular case of a 62-year-old patient, who presented with an inferior ST-segment elevation myocardial infarction with right ventricular involvement complicated by cardiogenic shock, sinus bradycardia, and an extensive echocardiographic ischemia with severe left ventricular systolic dysfunction. Coronary angiography revealed occlusion of 3 major coronary arteries. Primary percutaneous coronary intervention of the right coronary artery was performed with hemodynamic recovery, chest pain, and ST-segment resolution. Treatment for heart failure with reduced ejection fraction was initiated, with a good outcome.

Abbreviations: ECG, electrocardiogram; LAD, left anterior descending coronary artery; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; RCA, right coronary artery; STEMI, ST-segment elevation myocardial infarction; TAPSE, tricuspid annular systolic excursion; TIMI, the thrombolysis in myocardial infarction.

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Introduction

Simultaneous occlusion of multiple coronary arteries is a rare presentation in patients with ST-segment elevation myocardial infarction (STEMI) [1] and is associated with patient fatality [2]. The most suggested mechanism when there is no other identifiable etiology is multiple plaque rupture [3]. It is facilitated by hypotension and hemodynamic instability due to occlusion of a major artery resulting in blood stasis and destabilization of vulnerable plaques [2].

We reported a case of a 62-year-old patient, presented with an inferior STEMI complicated by cardiogenic shock and sinus bradycardia, whose coronary angiography revealed simultaneous occlusion of the three major coronary arteries that improved spectacularly after the percutaneous coronary intervention (PCI) of the right coronary artery (RCA).

It is clinically crucial to correctly diagnose this condition in order to start an efficient and early course of treatment.

Case report

This is the clinical case of a 62-year-old patient with diabetes for 15 years on Metformin 850 mg daily, hypertension for 5 years poorly controlled on Amlodipine 5 mg daily, a chronic smoking stopped 1 year ago, with no evidence of illicit drug abuse or coagulopathy. He reported exertional angina grade II of the Canadian classification and dyspnea class II of the New York Heart Association classification, evolving for 2 years, neglected by the patient. He presented to the emergency department with resting chest pain starting 11 hours before presentation, associated with a feeling of dizziness and vomiting. On arrival, the patient was conscious, hemodynamically unstable with a blood pressure of 76/45 mmHg, bradycardic at 45 beats/min, with a respiratory rate of 17 cycles/min, an oxygen saturation of 99% on room air, and a capillary blood glucose level of 2.6 g/L with no acetonuria on urine dipstick. The cardiac examination showed slowed heart sounds, no murmur on auscultation, and no signs of right or left heart failures. The electrocardiogram on admission showed sinus bradycardia at 49 beats/min, with ST-segment elevation in the inferior and right leads, necrotic Q waves, and negative T waves in the same territories (Fig. 1a). An urgent echocardiographic evaluation showed akinesis in the inferior, lateral and anterior territories of the left ventricle with severe systolic dysfunction (ejection fraction at 26%), without mechanical complications, a dilated right ventricle with systolic dysfunction (tricuspid annular systolic excursion at 12 mm, S wave velocity at 0.9 m/s), without visible intracardiac thrombus or valvular abnormalities. The diagnosis of acute inferior STEMI with right ventricle involvement complicated by cardiogenic shock and sinus bradycardia was retained; the patient received then a loading dose of 600 mg of clopidogrel, 300 mg of aspirin, and the curative dose of enoxaparin. He also received intravenous fluid therapy with 0.09% saline and was started on 0.3 μg/kg/min of norepinephrine and 6 μg/kg/min of dobutamine with hemodynamic status improvement. Laboratory analyses were carried out, the main results of which are shown on Table 1.

Table 1 – Significant laboratory findings.

Examen	Results	Normal values
Troponin level (ng/mL)	6500	<26
B-Type Natriuretic Peptide (pg/mL)	796	<100
Albumin (g/L)	37	34-54
C-reactive protein (mg/L)	1.86	6-12
Urea (g/L)	0.62	<0.45
Creatinine (mg/L)	18.78	6-12
Potassium (mmol/L)	4.7	3-5
Natremia (mmol/L)	138	135-140
Prothrombin	55	70-100
White blood cells (E/mm^3)	18470	4000-10,000
Hemoglobin (g/dL)	12.9	>13
Hematocrit	37.6	40-52
Platelets	170,000	150,000-400,000

Coronary angiography performed at 26 hours of pain onset showed a 40% long distal left main stenosis, occlusion of the proximal RCA, ostial occlusion of the left circumflex artery, and occlusion of the distal left anterior descending artery (Figs. 2a-c). As the culprit artery, the RCA benefited from primary PCI. After balloon dilatation, a Promus ELITE 2.75/28 mm drug-eluting stent was deployed over the middle to distal part of the RCA, and another Promus ELITE 3.0/24 mm stent over the proximal to middle part of the RCA. On final opacification, coronary flow was improved to "the thrombolysis in myocardial infarction" III with good coronary brushing (Fig. 2d).

After PCI, the hemodynamic status recovered with vasoactive drugs' withdrawing and the heart rate increased to around 70 beats/min, with chest pain resolution and ST-segment elevation decrease on electrocardiogram (Fig. 1b). The patient was discharged 9 days later with medical treatment of ischemic heart disease and of heart failure with reduced ejection fraction. The patient was asymptomatic at its discharge. Viability assessment of the akinetic anterior and lateral territories was performed later by cardiac MRI, with no significant residual viability in these territories.

Discussion

The most prevalent cause of mortality globally is acute myocardial infarction, which is responsible for over 75% of sudden cardiac death cases [4]. Acute STEMI often occurs as a result of atheromatous plaque rupture or erosion followed by a thrombotic occlusion of a one coronary artery [5]. Only 4.8% of STEMI patients also had thrombosis affecting more than one artery simultaneously. However, it comes with a substantial risk of cardiogenic shock and a life-threatening arrhythmia [6].

In a systematic review [6] of 29 articles that included 56 patients with simultaneous occlusion of multiple coronary

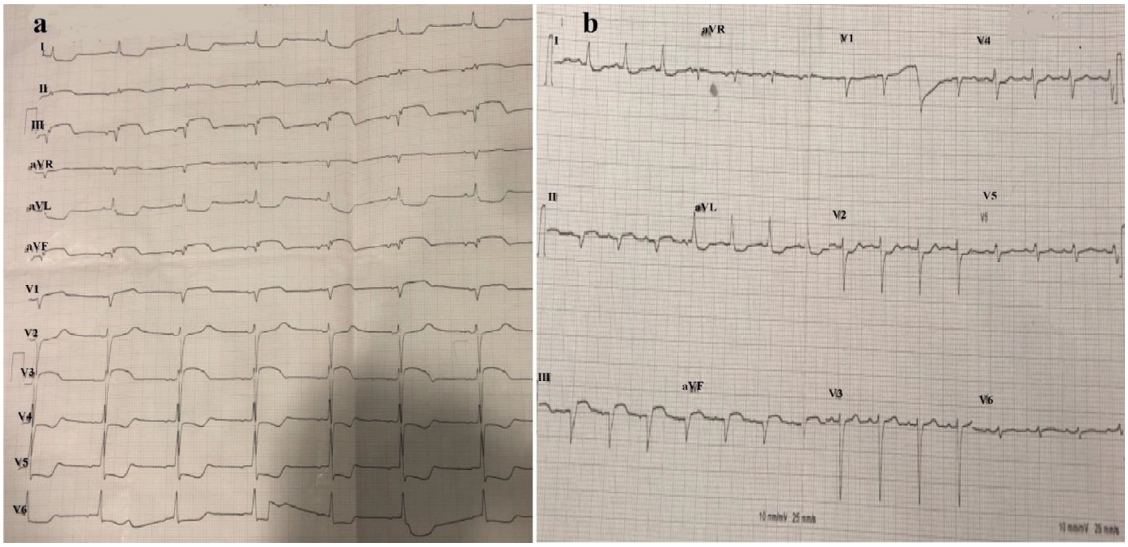

Fig. 1 – Admission electrocardiogram (ECG) showing acute inferior ST-segment elevation myocardial infarction (a), ECG after percutaneous coronary intervention of the right coronary artery (RCA) showing ST-segment resolution (b).

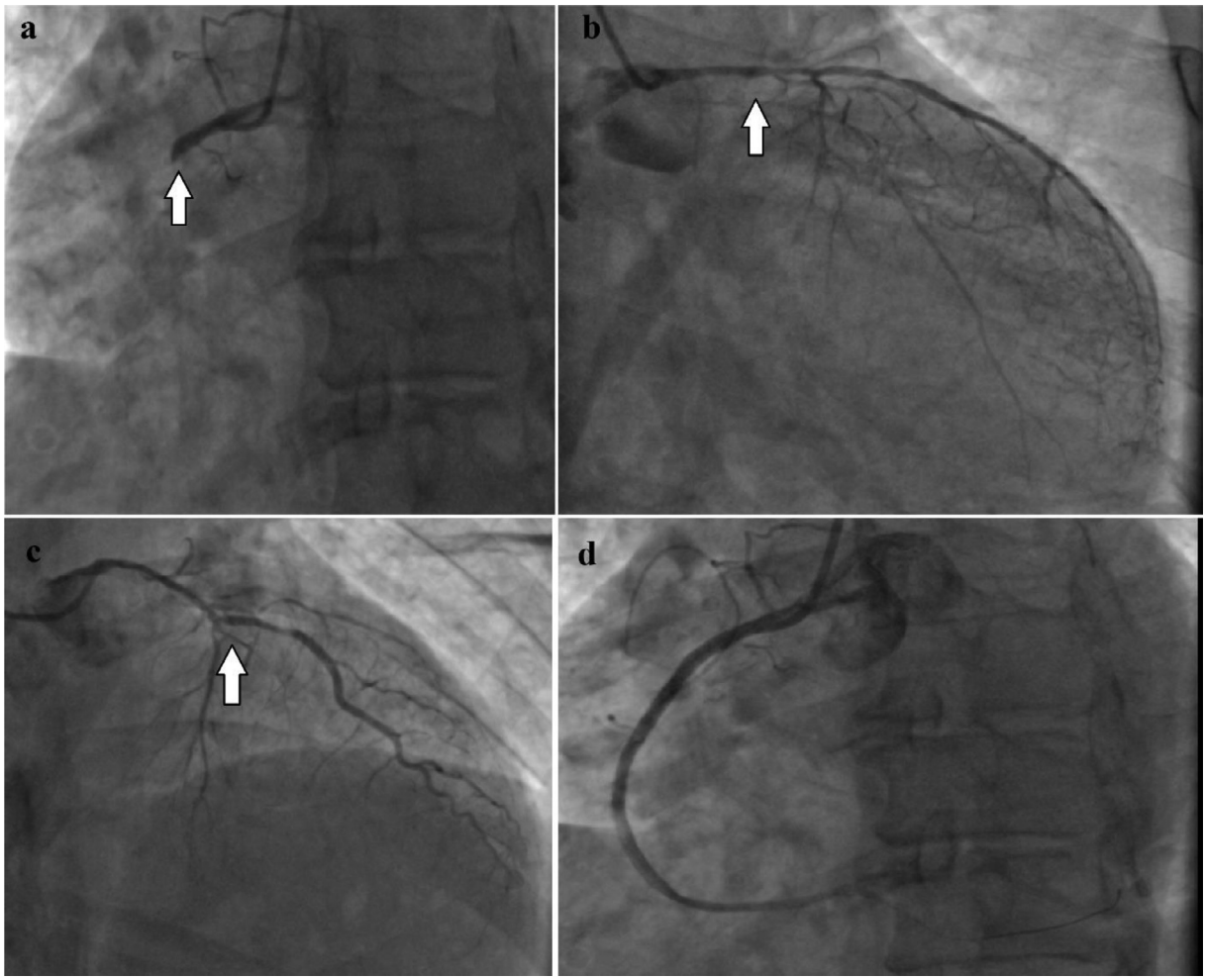

Fig. 2 – Coronary angiography showing occlusions of the proximal right coronary artery (RCA), the ostial left circumflex artery, and the distal left anterior descending artery (a, b, and c, respectively). White arrows show lesions site; the final angiographic result of the RCA after percutaneous coronary intervention (PCI) (d).

arteries, patients had a mean age of 59 years and were predominantly males (88%). The most common presentation was cardiogenic shock (41% of cases) and bradyarrhythmia (18% of cases). Both observed in our patient. The most frequent angiographic finding was simultaneous occlusion of the left anterior descending and RCA (50% of patients). A triple occlusion of the 3 major coronary arteries is extremely rare. This condition may usually result from coronary vasospasm, cocaine abuse, endocarditis of the aortic, or mitral valves or systemic thrombocytosis [3,7,8]. None of these etiologies were found in our case. Multiple coronary plaques' rupture is one proposed explanation of the multiple coronary occlusions in presence of the acute coronary syndrome [9]. It mostly results from the extensive inflammatory process known as "panarteritis," or from a catecholamine surge that activates platelets and induces thrombotic occlusion of many coronaries. The initial ischemic event that impairs blood flow to other arteries due to the cardiogenic shock, and results in acute secondary thrombosis is another potential explanation [10].

Given that myocardial revascularization is the only evidence-based therapy that has been shown to improve survival in patients with cardiogenic shock caused by acute STEMI [11], we performed a PCI of the RCA, which was the culprit lesion, with rapid hemodynamic recovery and heart rate normalization. Therefore, early diagnosis and appropriate PCI of the culprit lesions are keys of effective treatment.

In order to properly study outcomes and guide practice, cases of STEMI with simultaneous coronary arteries' occlusions should continue to be reported, and prospective studies should be conducted.

Conclusion

STEMI with simultaneous occlusion of multiple coronary arteries is a rare condition. Early diagnosis should be done and appropriate PCI of the culprit lesions should be performed in order to improve the clinical prognosis. However, more data are required to develop prompt therapeutic strategies in front of such a situation.

Patient consent

Written informed consent was obtained for the submission of the case report to the journal.

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