Acute coronary syndrome complicated by cardiogenic shock in a young adult: a case report from Dakar, Senegal

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Abstract
Coronary artery disease is the leading cause of cardiovascular deaths worldwide. It is becoming a major concern in developing countries, partly due to the adoption of Western lifestyles. It affects young adults as well as older patients over 45 years of age. In this report, we present a case of cardiogenic shock related to myocardial infarction in a young adult. He completed cardiac rehabilitation after the surgery. The outcome was favourable at the six-month follow up.

Keywords: acute coronary syndrome, cardiogenic shock, young adult

Case Report
A 39-year-old man with cardiovascular risk factors of hypertension, which was treated with amlodipine (taken irregularly), active smoking and occasional alcohol consumption was admitted to our intensive care unit for cardiogenic shock. There was no family history of sudden cardiac death or cardiovascular disease.

He had reported 10 days earlier with a severe constrictive chest pain lasting for more than 30 minutes, radiating to the back, which was associated with shortness of breath, one episode of haemoptysis and two episodes of vomiting. He was managed initially in his native country (located in the subregion) six days after the onset of symptoms.

On examination, the pulse was 108 beats per minute and systolic blood pressure was 70 mmHg with cold and clammy peripheries. There was mild respiratory distress with oxygen saturation of 82% in room air. The jugular venous pressure was elevated with a displaced apex beat. Pansystolic murmur was heard at the tricuspid and mitral valve foci. The rest of the clinical examination were unremarkable. The electrocardiogram (ECG) showed a sinus rhythm with negative T waves in the inferior leads, septal Q waves, ST-segment elevation in V1 and V2 and a Q3T3S1 pattern (Fig. 1).

Laboratory testing identified abnormalities, including elevated troponin I level, decreased estimated glomerular filtration rate of 31 ml/min/1.73, blood urea nitrogen level of 161.3 mg/dl and elevated white cell count of 18.67 cells/µl. The D-dimer level was within the normal range and the haemoglobin was 14.3 g/dl. Transthoracic echocardiogram (TTE) highlighted left ventricular (LV) dilatation with an ejection fraction (EF) of 30%, and anteroseptal and anterolateral wall motion abnormalities. It also showed severe mitral and tricuspid regurgitation and pulmonary hypertension (right ventricular systolic pressure 65 mmHg).

A chest computed tomography scan was advised to exclude pulmonary embolism and aortic dissection. This was not performed due to renal insufficiency. His medication included high-flow oxygen, enoxaparin, warfarin, atorvastatin and dobutamine infusion.

The patient was transferred to our hospital for further management five days later. At admission, his pulse rate was 125 beats/min, blood pressure was 140/90 mmHg on dobutamine infusion, oxygen saturation was 99% while under high-flow oxygen therapy. The physical examination revealed similar findings to the previous one.
The ECG on admission was similar to the first one, apart from more deep negative T waves in the inferior leads, with the addition to V6 (Fig. 2). TTE showed a 28% EF (Simpson biplane) with anterior and lateral wall abnormalities, reduced cardiac index (1.8 ml/min/m²), moderate ischaemic mitral regurgitation and minor tricuspid regurgitation. The coronary angiogram showed a double total occlusion of the left circumflex coronary artery (LCX) and the left anterior descending coronary artery (LAD) with thrombolysis in myocardial infarction (TIMI) flow 0 in both arteries (Fig. 3).

We decided after a discussion with the heart team to perform a double percutaneous coronary intervention (PCI) of both occluded coronary arteries, the LCX first. After predilatation with a small balloon, the LCX was opened and a drug-eluting stent was implanted, with good results (Fig. 4).

The LAD occlusion was approached with a standard guidewire...
but it did not cross the occlusion. Most of the attempts engaged with the collateral diagonal artery. A CTO guidewire, PROVIA, finally crossed the occlusion through a microcatheter (Fig. 5A). We took the precaution to ensure that it was the true coronary artery lumen by gently injecting through the microcatheter into the distal LAD (Fig. 5B). After small balloon inflations, the flow towards the LAD was restored with a Medina 0-1-0 bifurcation lesion (Fig. 5C).

We then proceeded to a provisional single-stent technique. Unfortunately, it was complicated by flow deterioration towards the diagonal branch (Fig. 5D), causing us to end with the proximal optimising technique with a non-compliant balloon (Fig. 5E) and side branch ostium post-dilatation (Fig. 5F), with a good final result (Fig. 6).

The patient’s haemodynamic parameters improved and he was gradually weaned off dobutamine. Follow-up echocardiography 72 hours later showed a better LV function with EF of 35%, but with the persistence of wall motion abnormalities. His medical treatment comprised aspirin, clopidogrel, statin, ACE inhibitor and beta-blocker.

The patient was assessed six months after he had completed a cardiac rehabilitation programme. The clinical examination was normal and LV function had improved significantly, with an EF of 45%.

**Discussion**

Patients under 40 years of age represent 4% of all patients who present with acute myocardial infarction (AMI). Acute coronary syndrome may be very brutal in young patients due to lack of the myocardial pre-conditioning phenomenon. Cardiogenic shock complicates nearly five to 10% of AMI with 30 to 40% of cardiogenic shock following AMI occurring at admission and 60 to 70% during the course of hospitalisation. It is the leading cause of death after myocardial infarction, with an unchanged in-hospital mortality rate of around 40 to 50% during the last decade.

Our patient presented initially with cardiogenic shock, which was stabilised with dobutamine infusion. Due to his age, a diagnosis of myocarditis was raised but rapidly excluded after coronary angiography. Coronary embolism could be discussed as a potential cause, given the double coronary artery occlusion. Coronary embolism is known to cause AMI in the setting of atrioseptal defect with or without aneurysm or atrial fibrillation.
A transoesophageal echocardiogram, which we did not perform in our patient, can identify the probable source in 76.2% of patients, and it is 92% sensitive and 98% accurate for detecting thrombi in the left atrial appendage, which is the most common area for thrombus formation, particularly in the setting of AF.

However, in our patient, premature atherosclerosis, initiated and promoted by conventional cardiovascular risk factors such as smoking and hypertension, is the most plausible underlying mechanism explaining the coronary artery disease. He denied other drug use, including marijuana, cocaine or methamphetamines, which are classical in this group of the population, as shown in the Partners Young-MI registry.

When it comes to practical considerations, we learned in the CULPRIT-SHOCK one-year follow-up study that culprit-lesion-only PCI rather than immediate multivessel PCI leads to fewer drawbacks, especially in terms of renal replacement therapy. However, the mortality rate did not differ significantly between the two groups.

In our patient, identification of the culprit vessel was quite challenging. Neither ECG nor echocardiography was very helpful. For this reason, PCI of both vessels in the same procedure was decided on in order to improve the haemodynamics and prognosis.
LAD recanalisation was surprisingly difficult, highlighting the severity of the coronary artery disease in this young patient. The LAD recanalisation might be considered a CTO-like procedure, since it necessitated utilisation of a microcatheter and guidewire escalation to cross the occlusion. We ended up with a provisional stenting technique, as recommended by the current European Bifurcation Club. Provisional stenting reduces complication rates driven by multi-stenting strategies and offers additional ‘bailout’ side-branch stenting if good results are not achieved (> 70% stenosis, TIMI flow < III or dissection > B).

Conclusion
Cardiogenic shock is a major complication of myocardial infarction, particularly in young adults, if not addressed early. Prompt management with rapid diagnosis and revascularisation may help to prevent it and to improve outcomes. However, absolute smoking cessation as well as the avoidance of illicit drug use may be the best prevention in this group of young people.

References