Myocardial infarction with contemporary bivasal occlusion: one case, two fates

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Abstract

A 42-year-old man was admitted to our department with a diagnosis of myocardial infarction with ST-elevation. The coronary angiography showed the thrombotic occlusion of both circumflex and right coronary arteries. Only the dependent territory of circumflex artery, treated before recanalization with adenosine, showed a total recovery.

Keywords: myocardial infarction; adenosine; cardioprotection; microcirculation

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Case Report

History

A 42-year-old man was referred to our department for chest pain lasting for three and a half hours. The electrocardiogram (ECG), obtained at the Emergency Department upon arrival, showed a ST-elevation in the inferior leadsand in V6 (Fig. 1).

The patient had been asymptomatic until that afternoon. His cardiovascular risk profile included: a family history for coronary artery disease (his mother suffered from a large myocardial infarction); smoking on 20 cigarettes/day; arterial hypertension treated with ACE-inhibitors and β-blockers.

The physical examination showed the patientwas suffering, the blood pressure was 160/90 mmHg, the pulse was 90/min, oxygen saturation was 96%. A grade II olosystolic murmur was heard together with a third heart sound. The chest and the abdomen examinations were normal; no peripheral edema was found.

The echocardiogram revealed akinesia of the mid-basal segments of the infero-lateral and inferior walls andhypokinesia of the antero-lateral wall with a mild left ventricular systolic dysfunction (EF 46%). In addition, there was a moderate mitral regurgitation.

The patient was treated with oxygen, aspirin, andglycoprotein IIbIIIa inhibitors, and he was immediately referred to the cardiac catheterization laboratory.

The patient underwent a left ventriculography confirmed the akinesia of the mid-basal inferior wall with normal left ventricular volume and mildly depressed ejection fraction.

On the coronary angiography, the left main coronary artery was free from critical stenosis; the left anterior descending coronary artery (LAD) had a focal stenosis in the proximal segment followed by minor irregularities in the middle tract and a tight stenosis in the distal segment. The circumflex coronary artery (Circ) was occluded at the origin of a large second marginal

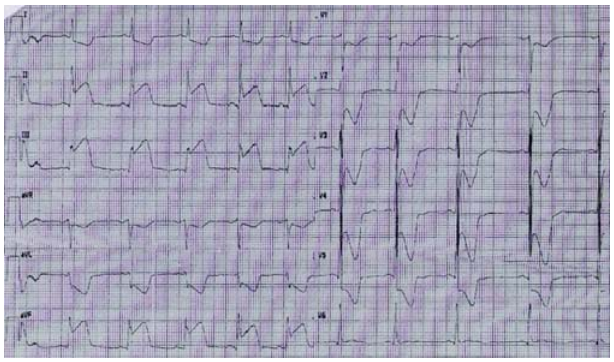

Fig. 1 *ECG in the Emergency Department.*

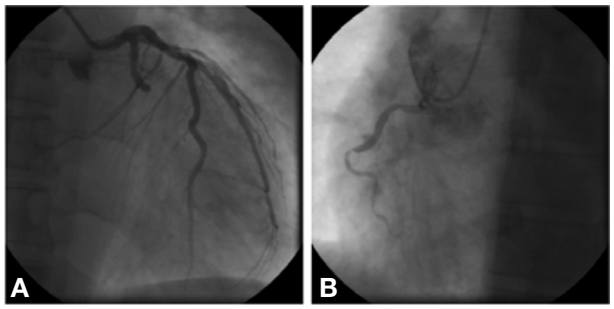

Fig. 2 *These coronary frames show the total Circ (panel A) and RCA (panel B) occlusion.*

branch (Fig. 2, panel A); another large marginal branch was free of stenosis. The right coronary artery (RCA) was dominant and was occluded in its middle tract (Fig. 2, panel B), immediately after the origin of the marginal branch. So, we found two vessels, the circumflex coronary artery and the right coronary artery, simultaneously occluded; that being so, both arteries could have been considered responsible for the clinical set. Given the technical difficulties of putting the catheter into the right coronary artery, first we decided to treat the Circ; so, in order to prevent the myocardial ischemia/reperfusion injury, a bolus of adenosine was directly injected into the marginal branch (4 mg in 2 ml saline) before performing the percutaneous coronary intervention (PCI) [1]. Then, a bare metal stent was implanted in the second marginal branch. After all we finally performed PCI and stenting of the medium tract of the right coronary artery (RCA).

The patient, returned to the Intensive Coronary Unit, was asymptomatic for chest pain. The ECG revealed a mild ST elevation in the inferior leads and ST depression in V1–V6.

Immediately after the PCI, we repeated the echo examination and we confirmed both the akinesia of the mid-basal segments of infero-lateral and inferior walls and the hypokinesia of the antero-lateral wall. Abciximab was administrated for 12 hours after the stenting procedure; the patients also received aspirin, enalapril, metoprololp.o., high dose of atorvastatin and lansoprazol.

From the second day after the PCI, the echo exam revealed a complete recovery of the prominent hypokinesia at the antero-lateral wall, while the alterations in the infero-lateral and inferior wall continued to be. Besides, we found a moderate to severe mitral regurgitation (eccentric jet, vena contracta width, got in long-axis parasternal view: 0.7 cm; mild dilated left atrial) and a restrictive diastolic pattern (E/A 1.6; DT 120 ms; average lateral and septal Tissue Doppler E': mm; E/E' 18).

Discussion

In animal models, infarct size and subsequent impairment of ventricular function are determined in a non-linear way after coronary occlusion [2]. In dogs, if reperfusion is activated after 5–15 minutes of vessel closure, there is almost complete recovery of the myocardium at risk. A significant recovery is still possible after occlusion lasting up three hours. When the "occlusion time" is extended beyond three hours, reperfusion does not result in recovery of contractile function [3].

Attempts to transfer these concepts on the clinical level have led to conflicting results (4–6). A relatively recent study about the effects of "ischemia time" on some clinical variables demonstrated that the favorable effects of thrombolysis on infarct size and ejection fraction are exclusive to patients who receive the optimal treatment within two hours after onset of symptoms [7]. Nevertheless, numerous studies have shown that status of coronary microcirculation plays a fundamental role in the prognosis of patients after an acute myocardial infarction. Failure to restore myocardial perfusion after recanalization of the artery responsible for infarction, documented by contrast echocardiography, is a strong negative prognostic factor and it is related to worse recovery of contractile function [8].

In a study of 31 patients with myocardial infarction, coronary velocity-flow after angioplasty and stenting

was demonstrated being an important predictor for the recovery of global and regional left ventricular function [9].

In recent times it has been shown that the presence of antegrade flow in the infarct before reperfusion, results in myocardial salvage and a good early functional recovery of stunned myocardium [10]. In the long-term follow-up was observed that patients with evidence of reperfusion before performing primary percutaneous transluminal coronary angioplasty (PTCA) had a more favorable course, with a significantly reduction of cardiogenic shock, with a better result of the procedure, with small size of the myocardial infarct area, and reduction in mortality [11].

Our group has shown that the intracoronary administration of adenosine immediately before reopening the culpit-lesion vessel dramatically improves myocardial perfusion. This reperfusion strategy is associated with an early recovery of contractile function [12]. More recently, Claeys et al. obtained similar results [13]. However, the administration extra-venously of lower adenosine doses did not alter significantly the clinical outcomes of patients with STEMI enrolled nell'AMISTAD-II trial [14], while achieving a reduction in size of the myocardial infarct area.

Several mechanisms contribute to the ischemia-reperfusioninjury, including production of oxygen-free radicals, neutrophilactivation, endothelial and myocyte edema, loss of antioxidant enzymes, and cardiomyocyte apoptosis [15]. Given this complex pathogenesis, several strategies are currentlyunder investigation to prevent or lessen myocardial damage. Adenosine, an endogenous purine nucleoside, antagonizes many of the biochemical and physiological mechanisms implicated in ischemia-reperfusion injury and has been shown to reduce post-ischemic ventricular dysfunction and myocyte necrosis and apoptosis [16,17]. The exact mechanism of the cardioprotective effect of adenosine is not fully understood, although inhibition of neutrophil activation and prevention of endothelial damage seem to play a major role. Encouraged by this theoretical and experimental framework, we investigated the effects of adenosine as an adjunct to PTCA in acute myocardial infarction (AMI). Given the disappointing results of intravenous adenosine administration [18], we developed a strategy for the selective treatment of the ischemic territory right before the onset of reperfusion.

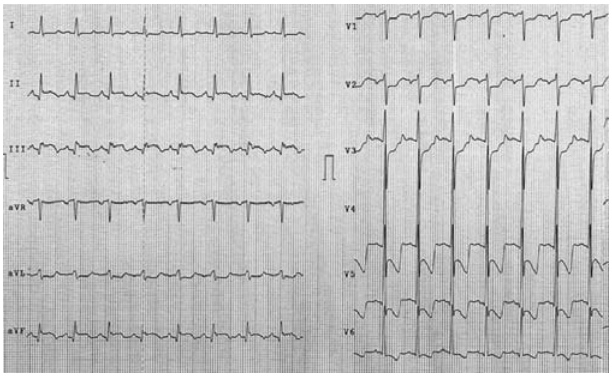

Fig. 3 *Post-procedural ECG.*

Conclusion

In this case report, we documented the relatively rare occurrence of two coronary arteries both responsible of myocardial infarction, but only one territory was protected with adenosine.

Ischemia and reperfusion both contribute to myocardial damage in AMI. Adenosine has been shown to limit ischemia-reperfusion injury in animal models. In this study, we have shown that adenosine administration in the infarct-related artery is feasible in the setting of primary angioplasty and that this treatment is safe and well tolerated and does not prolong procedural time. In this pilot study, intracoronary adenosine administration was associated with beneficial effects on coronary flow, on ventricular function, and on clinical course (Fig. 3). These observations are consistent with the hypothesis that a component of the ischemia-reperfusion injury can be prevented in humans by adenosine adjunct to primary PTCA. •

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