



CASE REPORT

Severe coronary vasospasm[☆]

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PALAVRAS-CHAVE

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Abstract A 50-year-old man, with chronic kidney disease and on dialysis, underwent coronary angiography in the context of acute coronary syndrome, which revealed focal lesions (type A) in the proximal left anterior descending and mid circumflex arteries. Ad-hoc angioplasty was performed on both lesions with direct stenting. An immediate drop in arterial blood pressure was observed and the angiogram showed new lesions with reduced flow throughout the coronary tree, progressing to cardiogenic shock and electromechanical dissociation. During cardiopulmonary resuscitation maneuvers, intracoronary verapamil was administered and TIMI 3 flow, sinus rhythm and a rise in blood pressure were obtained. Clinical stability was progressively restored. The patient was discharged medicated with calcium channel blockers and nitrates. During follow-up, he was twice readmitted for unstable angina. Coronary angiography revealed findings that could easily have been interpreted as new obstructive lesions, but these resolved after administration of intracoronary nitrates.

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Grave vasoespasm coronário

Resumo Homem de 50 anos, insuficiente renal crónico em hemodiálise, submetido a cateterismo cardíaco no contexto de síndrome coronária aguda, que revelou lesões estenóticas focais (tipo A) localizadas na descendente anterior proximal e circunflexa média. Efetuou-se angioplastia *ad-hoc* com implantação direta de *stent*. Imediatamente depois, observa-se abrupta queda tensional e o angiograma revela «novas» lesões com redução do fluxo em toda a árvore coronária e instalação de choque cardiogénico. Durante as manobras de reanimação, em dissociação eletromecânica, é administrado verapamil intracoronário com obtenção fluxo coronário TIMI 3, ritmo sinusal e pressão arterial. Assistiu-se progressivamente à melhoria e estabilização clínica. Foi implementado tratamento com antagonistas do cálcio e nitratos, registando-se no

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seguimento mais 2 admissões hospitalares por angor instável. A coronariografia voltou a revelar locais na árvore coronária que poderiam ser facilmente interpretados como novas lesões, mas as «estenoses» angiográficas foram solucionadas após administração de vasodilatadores intracoronários.

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Introduction

Spontaneous coronary vasospasm is associated with changes in autonomic nervous system activity¹ (sympathovagal imbalance) and endothelial dysfunction^{2,3} (reduced nitric oxide bioavailability). It is considered to be the pathophysiological mechanism behind variant or Prinzmetal angina and occurs in 1–5% of diagnostic procedures and percutaneous interventions as these can trigger or aggravate coronary spasm through endothelial injury or trauma.⁴ Its location, distribution and severity determine the seriousness of the clinical setting, which can include myocardial infarction, severe arrhythmias, cardiogenic shock and even death.⁵ Treatment in the acute phase is based on coronary artery vasodilators.

Case report

A 50-year-old man, a smoker, hypertensive, with chronic kidney disease on regular dialysis, but no history of heart disease, cocaine use, syncope or cardiovascular events, was admitted for chest pain at rest of short duration that occurred during an evening dialysis session.

He had already been referred for invasive coronary angiography for similar episodes in the previous few months, although no evidence of significant ischemia had been found on myocardial perfusion scintigraphy.

On arrival in the emergency department, he was almost completely asymptomatic (only residual pain) and was admitted for unstable angina (no significant ECG changes and negative markers of myocardial necrosis). Global and segmental left ventricular systolic function was preserved.

Risk stratification for myocardial infarction/death was: (a) GRACE score: 5% in-hospital and 13% at 6 months; (b) TIMI score: 5% at 14 days.

Cardiac catheterization was performed the following day, which revealed focal type A lesions in the proximal left anterior descending artery (LAD) and mid circumflex artery (Fig. 1).

Ad-hoc angioplasty was performed on both vessels with direct stenting (Fig. 2), using standard intracoronary devices (3.0 mm × 12 mm in the proximal LAD and 3.0 mm × 15 mm in the circumflex artery).

Angiographic control of the last lesion treated (proximal LAD) showed a good angiographic result at the stent implantation site, but a new pre-stent lesion was now visible in the proximal circumflex artery (Fig. 3).

Immediately afterwards there was a drop in arterial blood pressure and the angiogram revealed other new lesions and reduced flow throughout the coronary tree. The patient went into cardiogenic shock and the coronary angiogram

showed retention of contrast at various points of the left coronary artery, principally intrastent, and TIMI 0 distal flow (Fig. 4A). Basic and advanced life support maneuvers were begun, a temporary pacing lead was implanted (due to complete atrioventricular block followed by ventricular asystole) and an intra-aortic balloon pump was inserted.

There were various possible causes for this situation including coronary dissection and no-reflow phenomenon, but the most likely in view of the clinical and angiographic context was severe diffuse vasospasm of the coronary tree; intracoronary verapamil was accordingly administered while the patient was still undergoing resuscitation maneuvers and in electromechanical dissociation. This, together with the rest of the pharmacological arsenal and resuscitation efforts, was successful in obtaining TIMI 3 flow (Fig. 4B), sinus rhythm and a rise in blood pressure.

Invasive ventilation and hemodynamic support with intra-aortic balloon pump were required for 48 hours, and resulted in progressive improvement and clinical stabilization.

During hospital stay, there were various episodes of angina associated with transient ST-segment elevation on continuous ECG monitoring (Fig. 5), which resolved after administration of coronary vasodilators.

The patient was discharged home, asymptomatic, medicated with calcium channel blockers and nitrates. He has since been readmitted twice for unstable angina, with a similar clinical setting to the first admission. In both cases, the patient had completely or partially discontinued the recommended therapy.

During the latest hospitalization, invasive coronary angiography was repeated, which once again revealed coronary findings that could easily have been interpreted as new lesions, but these resolved after aggressive treatment with intracoronary nitrates (Fig. 6).

Discussion

Patients with variant angina present typical chest pain at rest associated with transient ST-segment elevation, mainly at night or in the early morning.⁶

Diagnostic coronary angiography, with administration of intracoronary nitrates and if necessary injection of contrast for non-selective catheterization, excludes the possibility of coronary vasospasm as opposed to obstructive atherosclerotic disease in most cases.⁷

Not all the typical clinical and imaging features of vasospastic coronary artery disease were present in our patient at admission. While he had experienced several anginal episodes at rest, almost all had occurred during evening dialysis sessions, and there was no evidence of significant ischemia on myocardial perfusion

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