Chronic spontaneous coronary dissection: evaluation by cardiac magnetic resonance

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ABSTRACT

This paper reports the clinical case of a patient with a history of polycythaemia vera and suspicion of pulmonary arterial hypertension with a spontaneous coronary dissection of uncertain chronology of the left anterior descending artery and its first diagonal branch detected during haemodynamic study. A stress cardiac magnetic resonance showed akinesia in the apical septum and medium anterior segment at rest as well as perfusion defect with dipyridamole and late gadolinium enhancement with transmural necrosis pattern exclusively in akinetic segments. Conservative management was decided upon due to the absence of inducible ischaemia, and the patient has remained asymptomatic at clinical follow-up. © 2012 Biomedical Imaging and Intervention Journal. All rights reserved.

Keywords: Chronic spontaneous dissection; coronary artery; cardiac magnetic resonance; polycythaemia vera.

INTRODUCTION

Spontaneous coronary dissection (SCD) is a rare cause of acute coronary syndrome (ACS) (<1% in different angiographic series) [1]. It usually implies the existence of a thrombus or blood in a false lumen that normally originates from the outer third of the tunica media. SCD may occasionally progress circumferentially or spirally, compromising blood flow through the true lumen and causing coronary ischaemia. SCD commonly occurs in the left anterior descending (LAD) artery and remains an underdiagnosed condition that may go undetected on angiography without the use of intracoronary imaging techniques [2].

The pathogenesis of SCD remains controversial. It has been traditionally associated with young, predominantly female, patients without cardiovascular risk factors, especially in the peripartum period. In recent years, SCD has also been associated with connective tissue diseases, some vasculopathies and chronic inflammatory processes [3].

CASE PRESENTATION

A 51-year-old man with a history of polycythaemia vera (PV) and portal hypertension secondary to liver cirrhosis was referred for left and right heart catheterisation due to suspicion of pulmonary arterial hypertension (PAH). The patient was being studied for dyspnea on moderate exertion, which began in previous months, without angina, palpitations or any other sign to suspect an underlying coronary disease.
Haemodynamic right heart study confirmed the diagnosis of severe postcapillary PAH (mean pulmonary arterial pressure: 40mmHg and mean pulmonary capillary wedge pressure: 25mmHg). Left ventriculography demonstrated elevated diastolic pressures but also revealed an apical akinesia. A coronary angiography was subsequently performed and showed the presence of SCD, of uncertain chronology, in the LAD and first diagonal branch (1D), which extended broadly by both vessels (Figure 1; DA: black arrows, 1D: dotted arrows). In-depth history revealed an episode of typical chest pain approximately two years ago for which the patient did not seek medical attention due to the self-limiting nature of the pain. Furthermore, the patient had been completely asymptomatic for angina during this period.

Based on these findings, a stress cardiac magnetic resonance (CMR) with contrast was performed to evaluate inducible ischaemia and myocardial viability. CMR confirmed the presence of akinesia in the apical septum and medium anterior segment (Figure 2 and Video 1; A and B: cine MRI longitudinal four chambers view; C and D: cine MRI short axis; arrows indicate akinetic regions; comparative views in telediastole -A and C- and telesystole -B and D-). Perfusion defect (Figure 2E and Video 2; arrows) and late gadolinium enhancement (LGE) with transmural necrosis pattern was also observed exclusively in akinetic segments.
ised artery dissection in the primary aorta thickness [9], endothelial atheroma. It is an incidental finding in a patient's history of PV, which was successfully treated percutaneously sixteen years after a previous admission for unstable angina [6]. Finally, Ghosh et al. reported a 36-year-old man who was admitted with ventricular tachycardia during chemotherapy. Twelve years before, an ACS secondary to SCD had been managed conservatively after a nuclear stress test showed up negative for ischaemia [7]. These cases reflect how SCD can become chronic lesions and remain asymptomatic for years.

The patient described in this paper underwent coronary angiography due to apical akinesia observed in ventriculography. Moreover, it is known that vascular complications represent the most common cause of mortality in PV, usually associated with thrombosis. In a recent cohort of patients with JAK2-positive myeloproliferative neoplasms, ACS was the most frequent vascular complication [8].

Although the relationship between blood rheological variables (eg, haematocrit and blood viscosity) and raised intima media thickness [9], endothelial dysfunction [10] and altered arterial compliance [11] has been demonstrated, only the previously referenced case report [6] had described the association between PV and SCD. Therefore, the need for more evidence to establish the causal link between both diseases is acknowledged.

With all this, the present case becomes unusual because it is an incidental finding in a patient without recent ischaemic symptoms. Moreover, the case illustrates the diagnostic performance of CMR in the functional impact of SCD, allowing for an appropriate therapeutic approach. These findings could help extend the indications for stress CMR in the presence of such pathology.

REFERENCES