

# Chronic spontaneous coronary dissection: evaluation by cardiac magnetic resonance

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Received 10 April 2012; received in revised form 31 August 2012; accepted 8 September 2012.

## 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

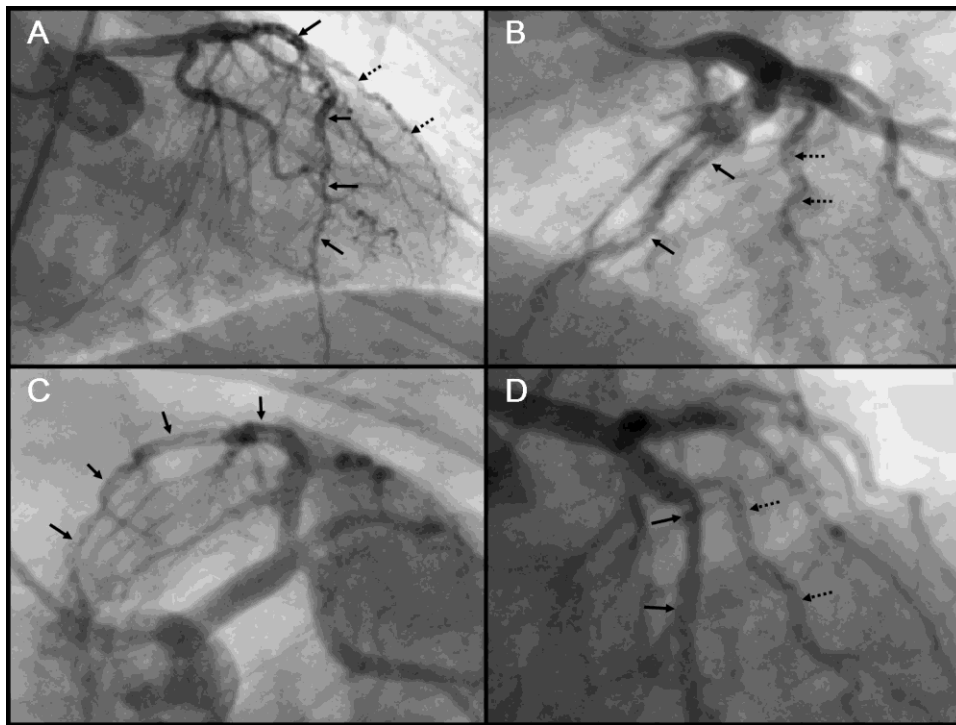
Spontaneous coronary dissection (SCD) is a rare cause of acute coronary syndrome (ACS) (<1% in different angiographic series) [1]. It usually implies the existence of a thrombus or blood in a false lumen that normally originates from the outer third of the tunica media. SCD may occasionally progress 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].

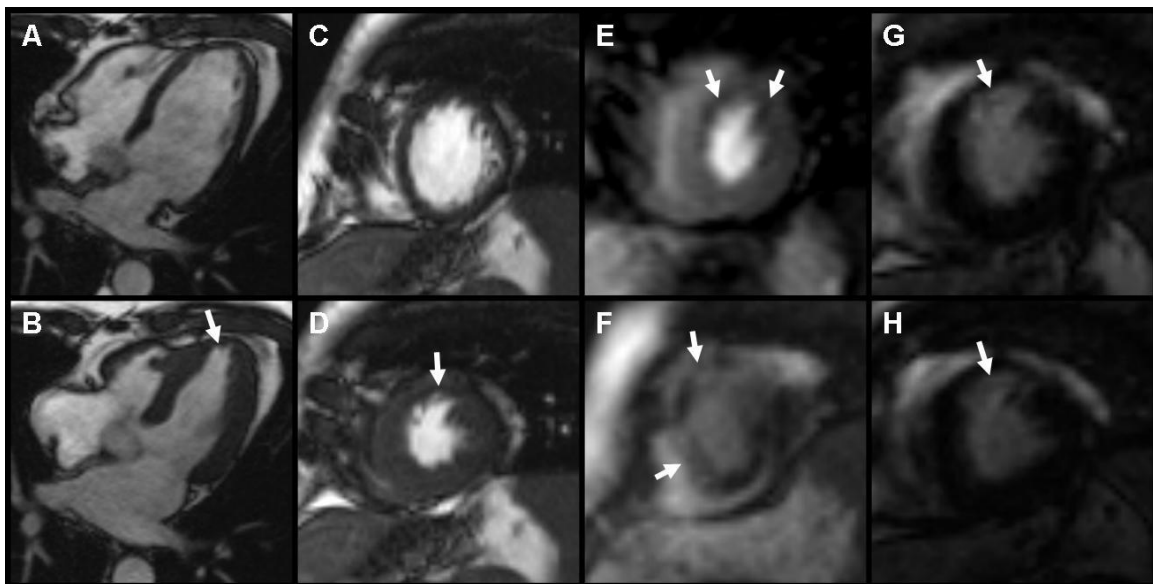
## CASE PRESENTATION

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.

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**Figure 1** Coronary angiography: coronary artery dissection of left anterior descending artery and first diagonal branch.

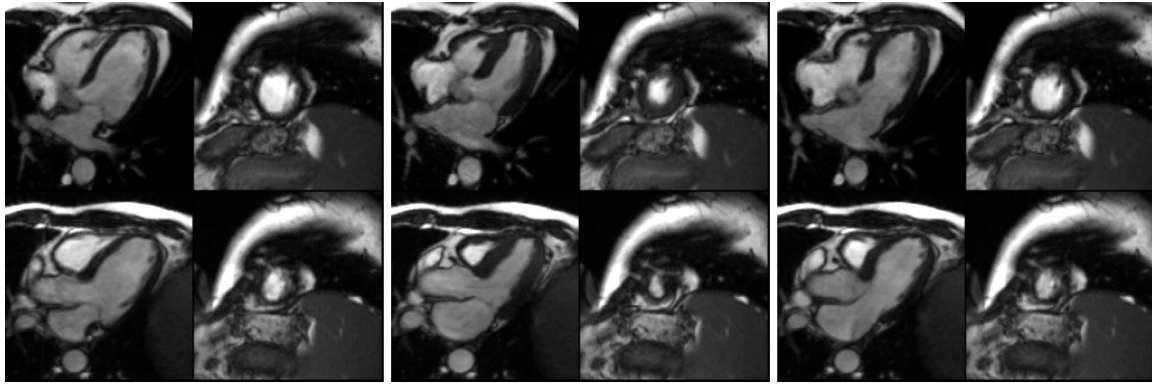


**Figure 2** Cardiac magnetic resonance imaging: cine MRI, dipyrindamole stress first-pass myocardial perfusion and late gadolinium enhancement.

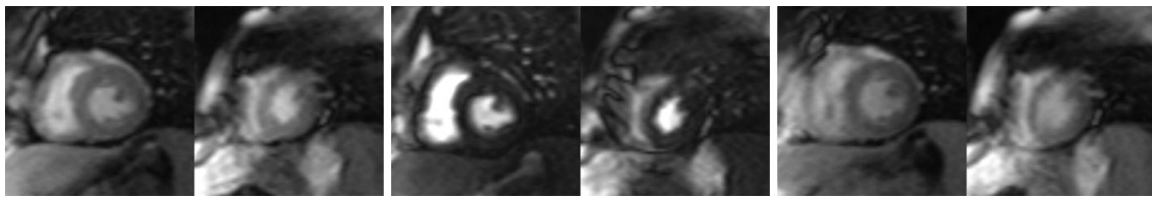
Haemodynamic right heart study confirmed the diagnosis of severe postcapillary PAH (mean pulmonary arterial pressure: 40mmHg and mean pulmonary capillary wedge pressure: 25mmHg). Left ventriculography demonstrated elevated diastolic pressures but also revealed an apical akinesia. A coronary angiography 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 aknetic 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 aknetic segments



**Movie 1** Cine MRI: Longitudinal view (four and five chambers) and short axis view (mid and apical sequences) showing an akinesia restricted to the apical septum and medium anterior segment.



**Movie 2** Dipyridamole stress first-pass myocardial perfusion: Short axis view (mid and apical sequences) showing a perfusion defect exclusively in akinetic segments.

(Figure 2F-H: ECG-gated single-shot short-axis; arrows indicate LGE). In the absence of inducible ischaemia, conservative management was decided upon and the patient remained asymptomatic at one year follow-up.

## DISCUSSION

Optimal treatment of the SCD is unclear. In the context of ACS, management depends on the location, accessibility and extent of dissection, and the patient's clinical status [4]. Percutaneous angioplasty or coronary bypass surgery is recommended in multivessel or proximal lesions, or in the case of persistent ischaemia, because these are considered the cases with the worst prognosis. Conservative treatment is preferred when SCD involves side branches or small vessels with TIMI 3 flow [1, 4].

The finding of a chronic SCD in coronary angiography is extremely rare. Only three cases in the literature have described the persistence of coronary dissection after an ACS in previous years. All cases are presented in the context of ischaemic clinical recurrence. Osaki *et al.* were the first to report a 58-year-old patient with inferior myocardial infarction by SCD in the right coronary artery, who was managed conservatively, and showed persistence of SCD ten years later [5]. Kay and Williams described the case of a 48-year-old man with a 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.

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