

Recurrent Spontaneous Coronary Dissection: When the Conservative Strategy Fails

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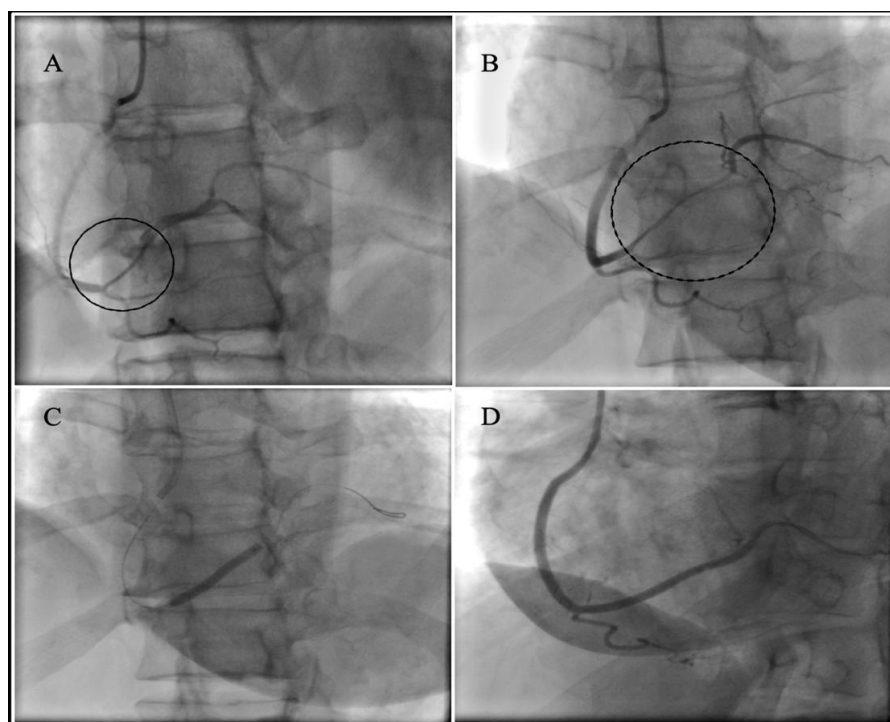


Figure 1: Coronary angiography. A. spontaneous coronary artery dissection of the right coronary artery (arrow). B. Extension of the dissection, type 2 (arrow). C. Percutaneous coronary intervention, D. Final result.

direct implantation of two drug-eluting stents. D. Restoration of TIMI III flows post-angioplasty. E. Sinus rhythm, Fc: 75bpm, normal electrical axis, ST-segment elevation in the inferior and low lateral walls, anteroseptal depression, atrioventricular block I degree.

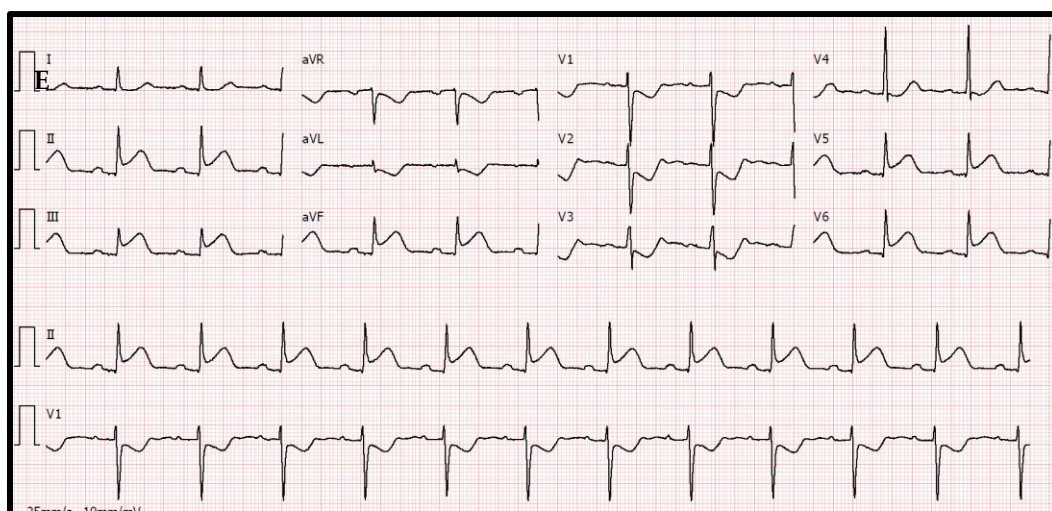


Figure 1: (Continued).

A 45-year-old woman with dyslipidaemia and no other known cardiovascular risk factors, who was autonomous in her daily activities, presented to the emergency department with moderate chest pain, which started suddenly and had been worsening for the last two hours. The pain did not radiate and increased with exertion, accompanied by a feeling of breathlessness. The electrocardiogram showed no enhancement changes, and the ultrasensitive troponin showed a slight alteration, so as the pain worsened, a coronary angiogram was carried out which revealed spontaneous coronary dissection (SCD) of the right coronary artery (Figure 1A).

Initially, a conservative approach was adopted, and he was discharged 2 days later. However, on the 21st day after discharge, he returned to the emergency department with chest pains that were more intense than those that had motivated his previous hospitalisation for coronary dissection. She also had anxiety, difficulty breathing and a feeling of fainting. The electrocardiogram showed sinus rhythm, Fc: 75bpm, normal electrical axis, ST-segment elevation in the lower and lower lateral walls, anteroseptal depression, I degree atrioventricular block (Figure 1E), and ultra-sensitive troponin I enzyme elevation: 218.3 ng/mL (reference value: 0.0-0.056). Given the electrocardiographic changes, persistent pain and elevated troponin I, coronary angiography was performed, which demonstrated extension of the previous type 3 coronary dissection to type 2 dissection, with extension to the posterolateral branch, resulting in 90% stenosis (Figure 1B).

Angioplasty was performed using intracoronary ultrasound. During the procedure, a drug-eluting stent (Xience Alphine 2.5 mm x 38 mm) was implanted and overdilated with a 3.0 mm x 30 mm balloon (Figures 1C to 1D). Due to poor expansion of the proximal stent, in a tortuous area and reduced TIMI, it was necessary to implant a new stent (Xience Alphine 2.75 mm x 12 mm) in a more proximal position, with overlap, successfully reestablishing TIMI 3 flow. The patient was discharged five days later, with dual antiplatelet therapy consisting of Aspirin 100 mg and Ticagrelor 90 mg, as well as Atorvastatin 40 mg, Ezetimibe 10 mg and Bisoprolol 2.5 mg. When she returned for the follow-up consultation, one month later, no new events or suggestive symptoms were recorded, so the patient will continue to be followed in coronary outpatient consultations, as well as in an outpatient study to investigate possible underlying causes.

Spontaneous coronary dissection, described in 1931 by Harold Pretty1, has a recurrence rate of around 27% in the first year when associated with hypertension, coronary tortuosity, fibromuscular dysplasia, predisposition to valsalva, a history of migraines and

previously compromised coronary beds.² It is an infrequent non-atherosclerotic and non-traumatic cause of acute myocardial infarction type 2 and sudden death, frequently reported among women at low cardiovascular risk in the reproductive phase, mainly under the age of 50, with a reported prevalence of 87 to 95% [3, 4].

Two hypotheses elucidate the pathophysiology of coronary dissection: the first, called “inside-out dissection”, postulates that the penetration of blood into the subintimal space of the true lumen occurs after rupture of the endothelial-intimal layer, and the second, characterized as “outside-in dissection”, suggests that the formation of the hematoma originates in the middle layer as a result of the rupture of transverse microvessels. The fact is that both cause variable obstruction of the true lumen and can occur in a new coronary territory or extension of a pre-existing coronary dissection, with an impact on the imbalance between the supply and demand of nutrients for cardiomyocytes [2-4].

Advances in diagnostic imaging, such as coronary angiography, optical coherence tomography, intracoronary ultrasound and magnetic resonance imaging, have had an impact on increasing ante-mortem detection [2, 5]. The conservative strategy is recommended in hemodynamically stable patients, as 95% of dissections heal spontaneously within 30 days. On the other hand, percutaneous coronary intervention (PCI) and coronary artery bypass grafting are essential in cases of cardiogenic shock, ventricular arrhythmias, persistent ischemia, coronary trunk obstruction, complex coronary anatomy and recurrent chest pain [2, 4, 5].

Currently, there are no randomized clinical trials on pharmacological management after SCAD. A recent study showed that beta-blockers reduced the recurrence of SCAD by 64%. Anticoagulants were associated with the risk of dissection extension and stains were only recommended if dyslipidemia was present [2, 3, 5, 6]. The patient, who was initially treated conservatively, had a recurrence of the dissection and hemodynamic instability, probably due to the extent of the previous dissection. During hospitalization, she remained stable with no complications.

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Conflicts of Interest: None.

Supplementary Materials: None.

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