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Outcomes Following Conservative Management of Spontaneous Coronary Artery Dissection.

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Spontaneous coronary artery dissection (SCAD) is a rare but a serious cause of myocardial ischaemia and infarction that occurs most frequently in younger female patients. The management of this rare condition remains controversial. In this case series we describe the spectrum of outcomes observed following conservative management.

Keywords Spontaneous coronary dissection • Conservative management • Intravascular ultrasound • Percutaneous coronary intervention • Fibromuscular dysplasia

Background

Spontaneous coronary artery dissection (SCAD) is a rare but a serious cause of myocardial ischaemia and infarction that occurs most frequently in younger female patients. The management of this rare condition remains controversial. In this case series we describe the spectrum of outcomes observed following conservative management.

Case 1

A 33 year-old multiparous female, with no recent history of pregnancy, presented to a peripheral hospital with one-hour history of central heavy chest pain. She was a current smoker and was taking an oral combined contraceptive. No other risk factors for coronary artery disease were identified. Physical examination was unremarkable. Anterior ST-segment-elevation was demonstrated on an electrocardiogram and fibrinolysis was successfully administered. The peak serum high sensitivity troponin T was 3134 ng/L (ULN <13 ng/L) the day following presentation. Standard medical therapy was continued for the acute coronary syndrome (ACS) and coronary angiography was undertaken, following transfer to our centre, two days later. This revealed a coronary artery dissection involving the origin of the left anterior descending artery (LAD) and extending to the distal vessel with thrombolysis in myocardial infarction (TIMI) 3 flow in the vessel [Fig. 1A]. The other coronary arteries appeared angiographically normal but the left ventricular function was moderately reduced with anterior akinesis demonstrated. Medical management was continued and she was discharged five days later with no ongoing symptoms.

A cardiac MRI performed two weeks post presentation demonstrated 50-75% thickness scar in the LAD territory with overall moderately reduced left ventricular ejection fraction (LVEF). The patient remained asymptomatic and coronary angiography performed five weeks later demonstrated a persisting dissection with a large false lumen extending from the distal left main coronary artery (LMCA) to the distal LAD with compression of the true lumen of the LAD. [Fig. 1B]. There was concern regarding involvement of the distal LMCA. A consensus opinion was sought from the Cardiosurgical (Heart) team and continued medical management was suggested with close observation and repeat coronary angiography to exclude progressive involvement of the LMCA. The third coronary angiogram was undertaken

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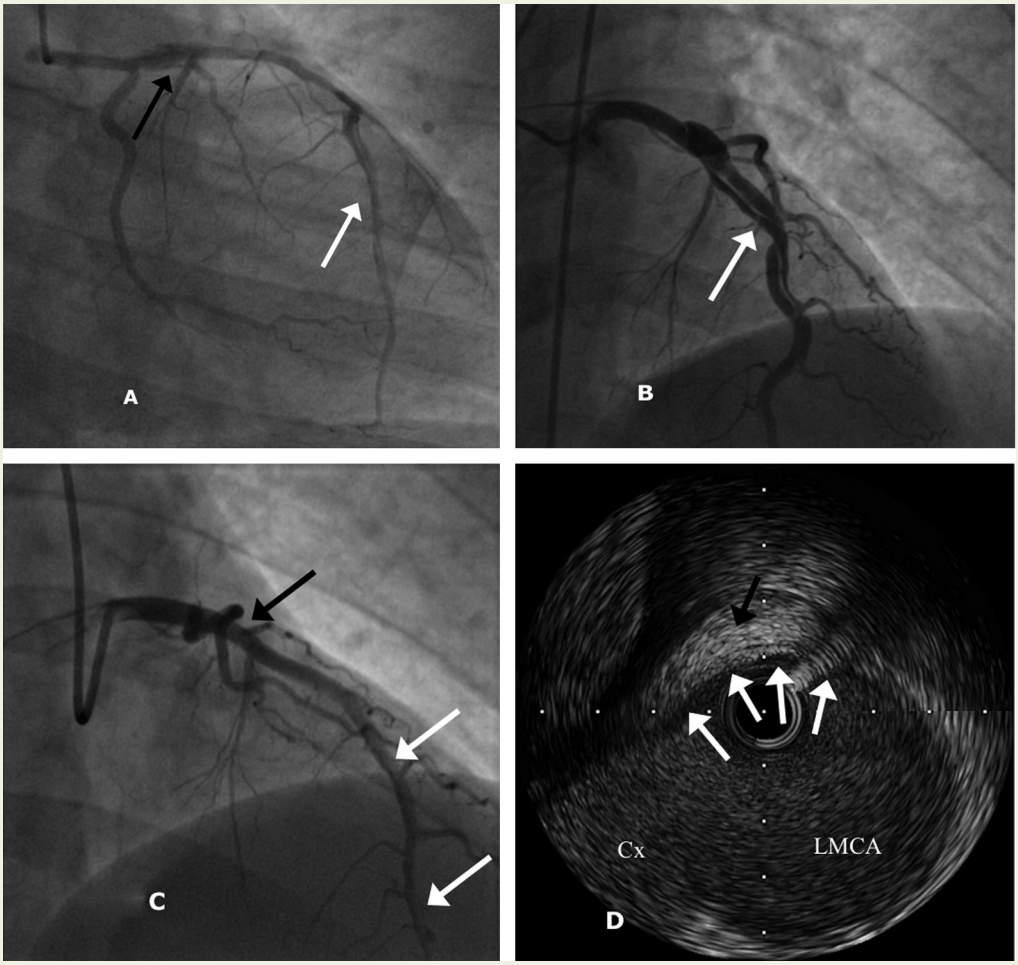

Figure 1 (A) - Case 1: Initial angiographic findings. Dissection is seen in the proximal LAD (black arrow) and involves the length of the vessel with a visible luminal flap in the mid and distal LAD (white arrow). (B) - Case 1: Follow up angiographic findings at five weeks. A luminal flap is seen in the proximal and mid LAD (white arrow). (C) - Case 1: Follow up angiographic findings at 29 weeks. Resolution of the proximal LAD angiographic abnormality (black arrow) with residual compromise of the true lumen in the mid and distal LAD (white arrows). (D) - Case 1: Intravascular ultrasound demonstrating resolving haematoma in the sub intimal space (false lumen) in the distal LMCA (black arrow). Cx = Circumflex coronary artery, LMCA = Left main coronary artery.

24 weeks later and demonstrated improvement in the angiographic appearances with resolution of the dissection in the distal LMCA and proximal LAD and improvement in the appearance of the mid and distal LAD with no flow in the false lumen and less compression of the true lumen. [Fig. 1C]. Intravascular ultrasound interrogation confirmed that the false lumen involving the distal LMCA as well as the LAD had thrombosed [Fig. 1D]. The thrombosed false lumen was still compressing the true lumen in the mid and distal LAD.

Case 2

A 41 year-old primiparous female, two weeks post-partum, presented to a peripheral hospital with two hours of dull central chest and back pain, radiating to the arms and neck, whilst breastfeeding. Her past medical history was unremarkable and her only known risk factor for coronary artery disease was a positive family history of premature ischaemic heart disease. The physical examination was unremarkable apart from mild hypertension. An electrocardiogram demonstrated evolving anterolateral T wave inversion with serum troponin I measurements rising to 29.6 mcg/L (ULN <0.03 mcg/L). Standard medical therapy for an ACS was commenced. Coronary angiography, performed four days later, identified long mid-vessel dissection of both LAD and obtuse marginal arteries, with angiographically normal vessels elsewhere [Fig. 2A]. The LVEF was mildly impaired with apical akinesis. The patient experienced no further symptoms during hospital admission and was discharged on medical therapy. The patient remained asymptomatic on subsequent review. Follow up coronary