Cardiac Imaging

Sequential Spontaneous Coronary Dissection/ Mural Haematoma Within All Three Coronary Arteries over a Nine-Year Period

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Spontaneous coronary artery dissection (SCAD) is an unusual but increasingly recognised cause of an acute coronary syndrome (ACS). It presents in younger patients, without conventional risk factors for coronary artery disease. Previous reports of SCAD involving all three coronary arteries within a period of time are lacking and are limited to a few case reports with contemporaneous three-vessel SCAD.[1]

We present a case of sequential SCAD in all three coronary arteries over a period of nine years and we illustrate the characteristic and specific aspects of the angiographic presentation and treatment. A 53-year-old lady presented with an ACS. There was no significant past medical history. Physical examination was unremarkable. Initial markers of myocardial necrosis were negative, but the ECG revealed sinus rhythm with inverted T-waves in leads V_5 and V_6. Transthoracic echocardiography showed a structurally and functionally normal heart. Coronary angiography (CA) demonstrated an unobstructed left coronary system (Figure 1A) with a long tubular abnormality within the RCA (Figure 1B), which did not respond to intracoronary nitroglycerine and was not felt to be flow limiting. She received medical treatment with aspirin, statin, and beta-blockers.

Two years later she was readmitted with a further ACS. CA showed a completely normal right coronary artery (RCA) with resolution of the tubular abnormality (Figure 2A), a normal left anterior descending artery (LAD), but a new long segment of abnormality within the left circumflex (LCX) that was felt to be the culprit lesion and angiographically flow limiting (Figure 2B). In view of the patient's ongoing pain, this was treated with percutaneous coronary intervention (PCI) and stenting with a 2.25/20 TAXUS to obtuse marginal OM1, enlarged to 2.4 mm, and a 2.5/16 TAXUS to the proximal LCX, enlarged to 3 mm, with an excellent angiographic result (Figure 2C). Recovery was unremarkable, with a plan for treatment with clopidogrel for 1 year and aspirin lifelong.

Seven years later the patient presented once again. CA demonstrated a new angiographically moderate long tubular segment of abnormality within the LAD, which did not respond to intracoronary nitroglycerine (Figure 3A). The LCX stent was patent (Figure 3A) and the RCA remained normal (Figure 3B). She was treated medically and is currently asymptomatic 9 months later.

This case highlights a condition that is a rare cause of ACS. Previous studies show that SCAD is commonest in the fifth decade, with a striking female predomi-

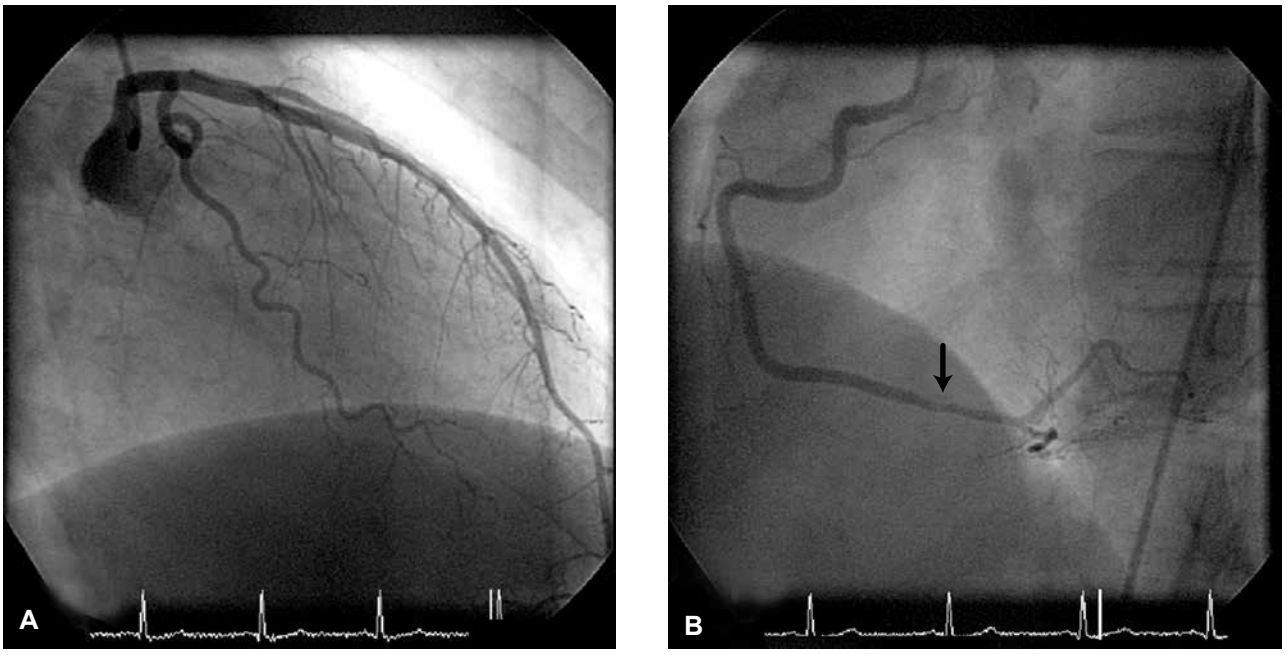

Figure 1. At the initial presentation of the patient: (A) Right anterior oblique (RAO) caudal projection of the left coronary artery (LCA) with no apparent coronary artery disease. (B) Left anterior oblique (LAO) view showing long tubular stenosis of the distal right coronary artery (RCA), with abrupt demarcation from normal proximal segments, which did not respond to intracoronary nitroglycerine, caused by mural haematoma (black arrow).

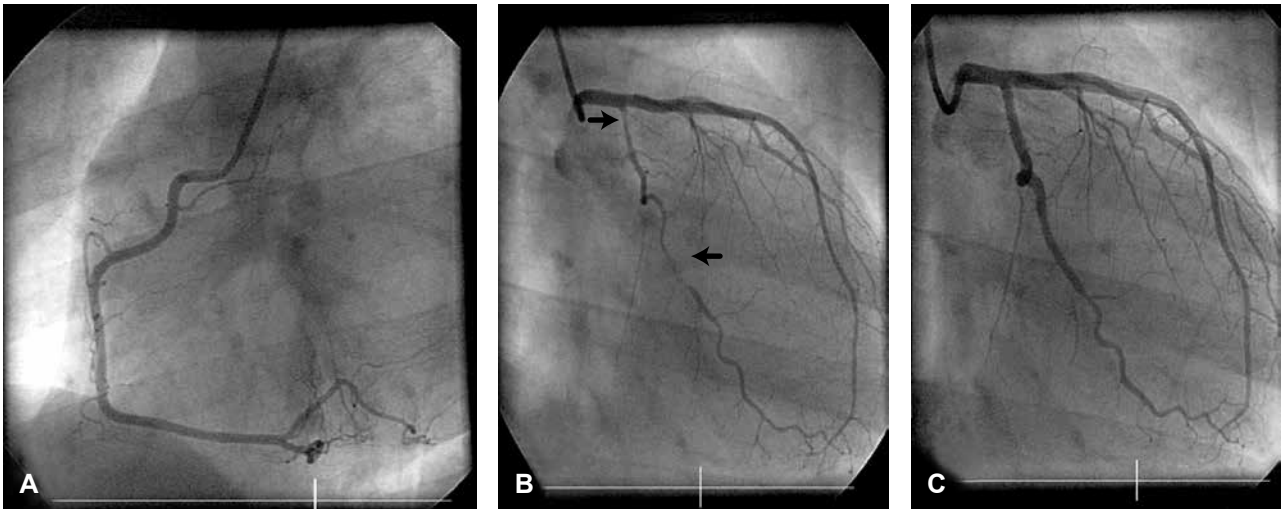

Figure 2. Two years later: (A) LAO view of the RCA showing complete resolution of the angiographic appearance. (B) RAO caudal projection with normal left anterior descending (LAD), diffuse proximal disease in the left circumflex (LCX), and a long critical obtuse marginal (OM1) stenosis (black arrows). (C) RAO caudal projection showing an excellent post-PCI angiographic result (2.25/20 TAXUS stent to OM1 enlarged to 2.4 mm and 2.5/16 TAXUS to proximal LCX enlarged to 3 mm).

nance, particularly in the peripartum period, with 25-31% of reported cases occurring during this time. Other predisposing factors include connective-tissue and vasculitic disorders, cocaine abuse, and in some cases it has been described in association with oral contraceptive use in previously healthy individuals. Urgent CA is indicated if SCAD is suspected and the definitive diagnosis of an intimal flap can be confirmed by intravascular ultrasound or optical coherence tomography. However, this procedure is not always feasible, because of significant luminal compression by the extraluminal haematoma, and needs

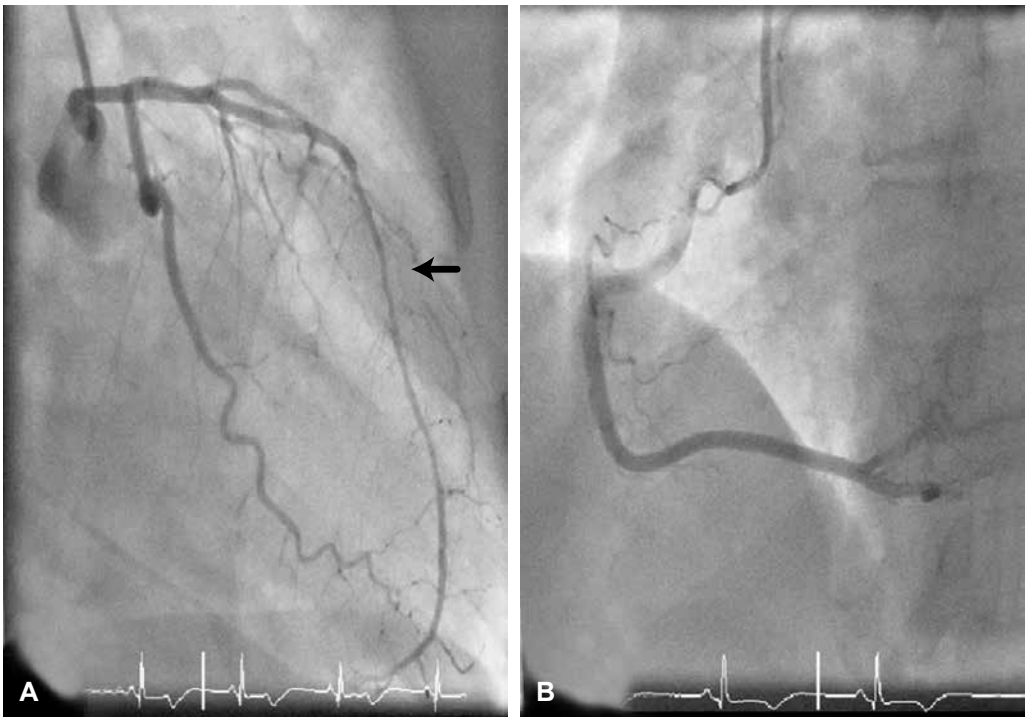

Figure 3. At 9 years: (A) RAO caudal projection with patent stents in the LCX and new long tubular stenosis within the mid and distal LAD, with abrupt demarcation from normal proximal segments, which did not respond to intracoronary nitroglycerine, caused by mural haematoma (black arrow). (B) LAO view showing a favourable angiographic appearance of the RCA.

special care, as it can exacerbate the disruption of the vessel and cause vessel occlusion. Its pathogenesis is partially understood and the therapeutic strategies include conservative medical therapy, coronary artery bypass graft surgery or PCI.[2,3]

The principal abnormality observed in SCAD is the development of haematoma in the outer third of the vessel media, causing luminal encroachment. Importantly, an intimal tear is not always identifiable and the abnormality may appear to represent a long segment of atheroma. Typically, in the presented case this mural haematoma will usually resolve with time.[2]

Considerable controversy surrounds the aetiology of SCAD. Although atherosclerotic plaque rupture has been suggested as a possible mechanism, this seems unlikely given the frequent absence of atherosclerotic risk factors and the relatively early age of onset. It is more likely that this process represents spontaneous rupture of *vasa vasorum* in the vessel media, occurring as a result of vascular shear stress or abnormal connective tissue structure. The eosinophilic infiltrates observed in some cases may damage collagen and lead to cystic medial necrosis, and progesterone-induced microstructural changes may be important in peripartum and contraceptive-associated SCAD. A frequent association with underlying coronary fibromuscular dysplasia has been suspected, although this requires proof from histology.[4]

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