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Letters to the Editor

Serial spontaneous coronary artery dissections: A transient predisposition affecting multiple coronary arteries?

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Spontaneous coronary artery dissection (SCAD) is rare. In an analysis of a coronary angiographic series of 11605 patients, only 23 cases of SCAD were identified [1]. In another angiographic series, only 22 cases of SCAD were identified among 32869 angiogram studies [2]. More than 70 per cent of SCAD cases involve women in whom it is an important and under-recognised cause of acute coronary syndrome and sudden cardiac death [3]. Here, we describe two cases of recurrent multi-vessel SCAD managed successfully with medical therapy and percutaneous coronary intervention (PCI), respectively. These two cases of temporal clustering of SCAD may be due to a previously unrecognised transient predisposition to coronary artery dissection in these patients.

Case one: A 64-year-old female presented to a local hospital with sudden onset severe left sided chest pain with radiation to her back. Her other medical history included diet controlled type 2 diabetes mellitus, hypertension, dyslipidaemia and obesity. She had no prior history of ischaemic heart disease. Electrocardiogram (ECG) demonstrated sinus rhythm with 1 mm elevation in the ST segment in leads V1 to V3. Serum troponin peaked at more than 50 ug/L (normal <0.04 ug/L), and serum creatine kinase peaked at 2589 units/L (normal: 20–180 units/L). On a computed tomography (CT) aortogram which excluded aortic dissection, a single pulmonary embolus (PE) was found incidentally in the right upper lobe. An initial coronary angiogram was interpreted as demonstrating mild to moderate mid to distal tapering in the left main coronary artery (LMCA). The left anterior descending artery (LAD) was reported as having mild proximal disease with severe hazy lesion in the mid-segment. The left circumflex artery (LCX) was small with no significant coronary artery disease. The right coronary artery (RCA) was a large, dominant vessel with normal angiographic appearance. Given the concurrent finding of the right upper lobe PE, it was initially postulated that the patient's presentation resulted from embolic obstruction of her LMCA and LAD through a patent foramen ovale (PFO). The patient was managed medically with intravenous tirofiban and heparin infusions for 48 h, and ongoing aspirin, atorvastatin and carvedilol. Warfarin was subsequently commenced for PE. The patient became pain free and remained stable after day 1 of hospital admission.

Transthoracic and transoesophageal echocardiography (TTE and TOE) confirmed moderate segmental left ventricular dysfunction in the LAD territory, but failed to show any evidence of PFO to support the initial postulate of coronary artery embolus. Screening for other systemic thrombotic disease demonstrated a right below knee deep vein thrombosis (DVT), but the patient's thrombophilic and vasculitic screens were negative. A repeat coronary angiogram was performed 6 days later. In the LMCA, there was staining of vessel consistent with a dissection flap, extending into the LAD. The LCX and RCA were normal. At this stage, the patient was transferred to our institution for consideration of coronary artery bypass graft (CABG) surgery in a haemodynamically stable, pain free state.

Her coronary angiograms were reviewed. The initial coronary angiogram demonstrated a dissection of the left main trunk which extended down the proximal and mid-LAD. RCA was dominant and normal (Fig. 1). Review of the subsequent coronary angiogram demonstrated staining in the mid-distal LMCA, with a clear cap in the mid-LAD consistent with dissection (Fig. 2). TOE excluded dissection of the aortic root. Therefore, the patient had spontaneous dissections of the LMCA and LAD, with an incidental finding of PE. Given the patient has remained stable and pain free, the decision was made to manage her conservatively. Medical therapy was up-titrated with the addition of perindopril. The patient was discharged after a 2-week period of intensive monitoring in hospital, with regular follow up arranged.

Since discharge, the patient had been well. She was monitoring her blood pressure through a home blood pressure device. 28 days after hospital discharge, the patient presented to a regional hospital with sudden onset epigastric as well as interscapular pain at rest. ECG demonstrated 2 mm ST segment elevation in leads II, III and aVF. At time of presentation, she was hypertensive with a systolic blood pressure of 220 mmHg. Serum troponin peaked at 18.7 ug/L (normal <0.04 ug/L). Given her recent spontaneous dissection of the LMCA and LAD, and the potential need for urgent CABG surgery, the decision was made not to perform coronary angiography at the regional centre. She was urgently transferred to our institution. The patient was stabilised

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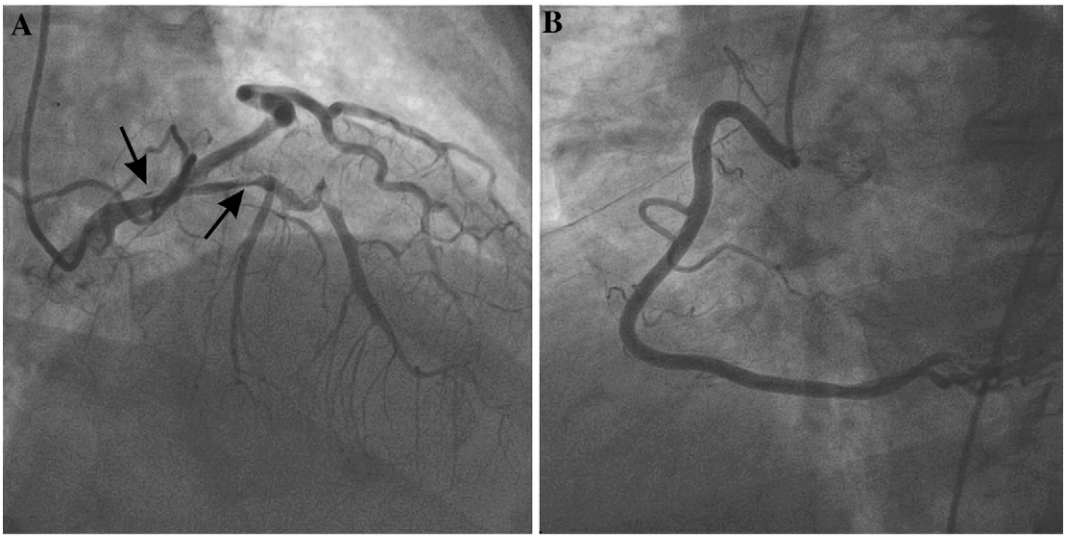

Fig. 1. Case one's initial coronary angiogram in the right anterior oblique caudal projection, demonstrating a small area of spontaneous dissection in the LMCA, and spontaneous dissection of the mid-portion of the LAD (arrows), with compression of the proximal segment (A). The RCA was dominant and normal as demonstrated in the left anterior oblique cranial projection.

with intravenous heparin infusion. Her blood pressure was controlled to a systolic target of 130–140 mmHg with intravenous glyceryl trinitrate infusion. Repeat coronary angiography was performed. This demonstrated that the LMCA and LAD were now normal, with no evidence of the previous dissections. There was some minor irregularity in the proximal LAD but no obvious residual flap, and the lumen of the vessel was much improved compared with previous angiograms, with good distal flow to distal vessels. The LCX was normal. The RCA, which was previously a normal large calibre vessel, was found to have a new abrupt calibre change in its mid-portion. This reduced diameter extended from its mid-portion down to the bifurcation, and then extended into the left ventricular branch. In the mid-portion of the left ventricular branch, there was complex subtotal occlusion extending peripherally. This appearance was consistent with spontaneous dissection of the RCA (Fig. 3). An autoimmune screen for underlying connective tissue disease was negative. On this occasion, the patient also became pain free and remained stable after day 1 of admission. It was decided to continue conservative management. Her carvedilol and perindopril were increased to ensure that her pulse rate and blood pressure were optimally controlled. The patient was discharged after intensive monitoring, with ongoing follow up. To date, three months after the spontaneous dissections, she has not experienced any further episodes of chest pain.

Case two: A 47-year-old female without prior symptoms of coronary artery disease presented to a rural hospital with an acute coronary syndrome consisting of acute chest pain associated with anterior T wave changes on ECG and elevated serum troponin. Her other medical history included treated localised breast carcinoma. She had a family history of ischaemic heart disease. She was initially managed as a case of ischaemic myocardial infarction, with anti-platelet therapy, beta-blockade, a statin and anticoagulation. She was transferred to our institution. Coronary angiography was performed. It demonstrated a normal LMCA. The proximal and mid-LAD, and the first diagonal vessel were normal. Distal to the large first diagonal vessel, there was a segment in the LAD which had the appearance consistent with a spontaneous coronary artery dissection (Fig. 4). The LAD was then subtotally occluded. The distal vessels filled by

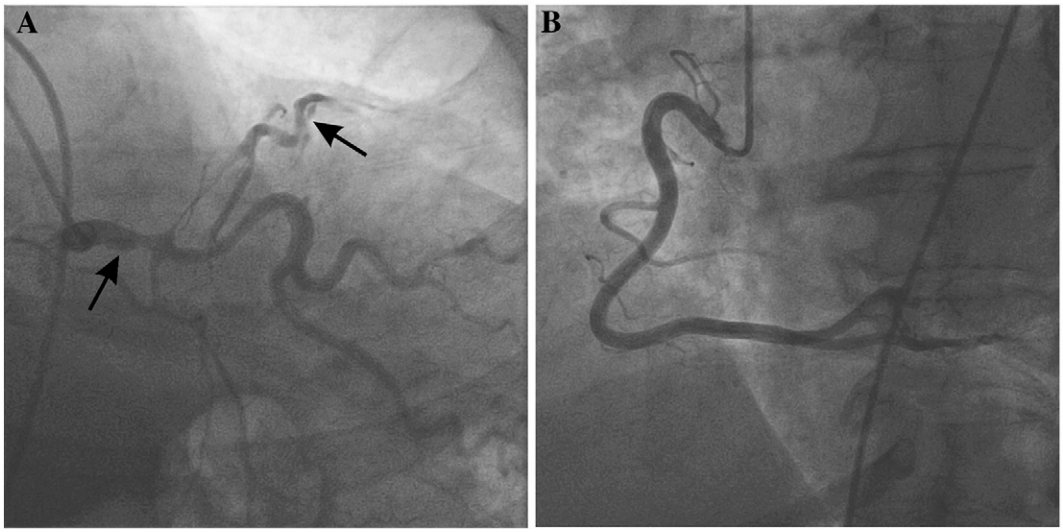

Fig. 2. Case one's subsequent coronary angiogram in right anterior oblique caudal projection (A), demonstrating staining in the mid-distal LMCA, with a clear cap in the mid-LAD consistent with dissection (arrows). Note that the RCA still had a normal angiographic appearance throughout, as demonstrated in the left anterior oblique cranial projection (B).