

CASE REPORT

A young woman with chest pain

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Abstract

A case of a previously fit young woman admitted with chest pain, who was found at coronary angiography to have dissection of the left main stem which extended to the left anterior descending and circumflex coronary arteries, is presented. Emergency coronary artery bypass grafting was performed (vein grafts to the left anterior descending, the diagonal, and circumflex arteries). The patient made an uneventful recovery, and three years after initial presentation she remains free of cardiac symptoms.

(Heart 2000;84:e12)

Keywords: spontaneous coronary artery dissection

A previously fit 38 year old white woman was admitted to the coronary care unit of our hospital with sudden onset of faintness, and a one hour history of central chest discomfort. She was a non-smoker and had no known risk factors for coronary artery disease. She denied use of oral contraceptives, or illicit drugs. On examination, her supine heart rate was 114 beats/min and blood pressure was 118/84 mm Hg. She had no clinical signs of Marfan syndrome or heart failure.

On admission, serum creatinine was 85 $\mu\text{mol/l}$ and creatinine phosphokinase (CPK) was 98 U/l (normal range 30–250 U/l). Total fasting cholesterol was 4.7 mmol/l (high density lipoprotein 1.7 mmol/l, low density lipoprotein 2.8 mmol/l, triglycerides 0.47 mmol/l) and blood glucose was 10 mmol/l. The resting 12 lead ECG showed right bundle branch block, left axis deviation, and 1–2 mm ST elevation in the precordial leads V1–V3. The chest radiograph was normal.

The patient was treated with aspirin, and intravenous heparin and nitrates for acute coronary syndrome. Eight hours later she complained of further chest discomfort. The ECG showed further ST elevation in precordial leads V1–V3, and inferolateral ST depression. This was associated with a rise in CPK to 1892 U/l. Urgent coronary angiography was undertaken. This showed dissection of the left main stem which extended to the left anterior descending and circumflex coronary arteries (fig 1). The lumen of the left anterior descending coronary appeared flattened and there was reduced distal flow. The dominant right coronary artery was normal. Emergency coronary artery by-

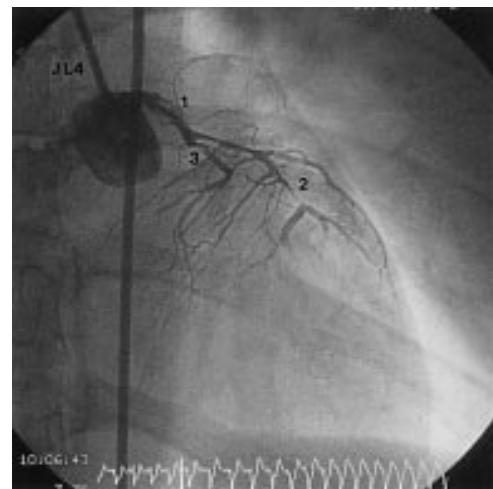


Figure 1 Coronary angiography in the right anterior oblique position using a left Judkins (JL4) catheter. This shows a thin radiolucent line corresponding to a dissection of the left main stem (1) extending to the left anterior descending (2) and circumflex (3) coronary arteries. In addition there is flattening of the lumen of the left anterior descending coronary artery (2) with reduced distal flow.

pass grafting was performed (vein grafts to the left anterior descending, the diagonal, and circumflex arteries). The patient made an uneventful recovery. Three years after the initial presentation she remains free of cardiac symptoms.

Spontaneous coronary artery dissection was first described at postmortem examination in a 42 year old woman almost 70 years ago.¹ Since then it has been reported as a rare cause of myocardial ischaemia and infarction, predominantly in young women without classical coronary risk factors, and is associated with a high mortality.² Approximately one in four female patients with spontaneous coronary artery dissection are in the peripartum period. This condition has also been described in patients with Marfan syndrome, women taking the contraceptive pill, and after intense physical exercise. The diagnosis is made at postmortem examination in over 25% of cases.

The pathogenesis of spontaneous coronary artery dissection is poorly understood. Spontaneous haemorrhage in the vasa vasorum has been suggested as a possible cause. The histological findings include periadventitial inflammatory infiltrate, comprising eosinophils, lymphocytes, and histiocytes. There is no necrosis of the media and no evidence of antigen/antibody complexes. Release of lytic substances (proteases) from eosinophils has

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Accepted 26 July 2000

been proposed as a mechanism leading to coronary dissection. However, it is now known that spontaneous coronary artery dissection may occur even in the absence of inflammatory infiltrates.³

In patients surviving the initial episode and who subsequently remain asymptomatic, medical management is safe and compatible with good long term survival. Thrombolytic treatment may be effective in lysing the clot in the false lumen, and is probably beneficial in those arteries where intramural haematoma is compressing the true lumen, allowing the latter to re-expand. Intracoronary stenting may be deployed to achieve vessel patency in selected cases, other than in those involving the left main stem.⁴ However, coronary artery bypass grafting is recommended in patients with dissection involving the left main stem, and in those patients with ongoing myocardial ischaemia, refractory to medical treatment.⁵

Our case shows that spontaneous coronary artery dissection is an important cause of myocardial ischaemia and infarction in young women without classical coronary risk factors.

This report also illustrates some important aspects of spontaneous coronary artery dissection. Firstly, sudden death without myocardial ischaemia is a frequent mode of presentation. Secondly, the overall mortality in those who present with myocardial infarction is in excess of 70%. Finally, our case clearly demonstrates that prompt diagnosis with coronary angiography, and treatment with coronary artery revascularisation, especially in patients with ongoing myocardial ischaemia, provides the only means of improving survival in this usually fatal condition.

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