CASE REPORT

Aortic root abscess presenting as unstable angina due to extrinsic compression of the left coronary artery

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Coronary ischaemia in acute endocarditis is usually due to pre-existing coronary disease or occasionally as a result of embolism from vegetations. A 68 year old man with known mixed aortic valve disease presented with a four week history of progressive exertional angina, which became unstable. He was afebrile with no peripheral signs of endocarditis. Three sets of blood cultures were negative. Transthoracic echocardiography with suboptimal windows confirmed moderate mixed aortic valve disease. Marked reversible ST segment depression with angina recurred at rest. Aortography showed severe aortic regurgitation with a distorted aortic root. Coronary angiography showed severe proximal narrowing of the left anterior descending and circumflex arteries with an unusual long and tapering contour. Emergency surgery revealed a large anterior aortic root abscess which had destroyed the left and right coronary cusps. Aortic root abscess and other rare causes of extrinsic coronary compression are discussed.

CASE REPORT

A 68 year old man with known mixed aortic valve disease presented with a four week history of progressive exertional angina, which became unstable. A month previously he experienced rigors but spontaneously improved without treatment. He had no risk factors for coronary disease. Examination revealed moderate mixed aortic valve disease and blood pressure 140/70 mm Hg.

He was afebrile with no peripheral signs of endocarditis. The resting electrocardiogram showed left ventricular hypertrophy, left axis deviation, and 1 mm of ST depression in V6. The chest radiograph showed mild cardiomegaly and upper lobe venous diversion. Haemoglobin concentration was 93 g/l, mean cell volume 70 fl, serum iron 0.9 µmol/l, total iron binding capacity 55 µmol/l (45–75), white cell count $10.8 \times 10^9/l$, platelets $316 \times 10^9/l$, erythrocyte sedimentation rate (ESR) 100 mm/hour, and C-reactive protein 60 mg/dl. Three sets of blood cultures and serial cardiac enzymes were negative. Transthoracic echocardiography with suboptimal windows revealed overall moderate left ventricular systolic function, posterioapical hypokinesis and a calcified, immobile aortic valve. Doppler predicted a peak aortic valve gradient of 60 mm Hg with moderate aortic regurgitation. Despite oral metoprolol, nitrates, intravenous unfractionated heparin and a three unit blood transfusion, marked reversible ST segment depression with angina recurred at rest accompanied by hypotension. Emergency coronary angiography showed severe proximal narrowing of the left anterior descending (LAD) and circumflex arteries with an unusual long and tapering contour (fig 1). Aortography showed severe aortic regurgitation with a distorted aortic root and a peak to peak withdrawal gradient of 45 mm Hg across the aortic valve.

Emergency surgery revealed a large anterior aortic root abscess which had destroyed the left and right coronary cusps

Figure 1 Right lateral coronary angiogram showing severe proximal narrowing of the circumflex and left anterior descending arteries with a smooth tapering contour. The distal vessels are normal.

with penetration into but not through the interventricular septum. The aortic root was replaced with a 25 mm Carbomedics valved conduit with reimplantation of the coronary arteries and saphenous vein grafts to the LAD artery and circumflex marginal. Histological examination of the aortic valve and abscess material showed a dense polymorph infiltrate with some giant cells but no organisms and culture yielded no growth. Serology for Q fever, mycoplasma, legionella, chlamydia, brucella, and syphilis was negative. Antinuclear and antineutrophil cytoplasmic antibody were negative. Colonoscopy and upper gastrointestinal endoscopy were normal. He received six weeks of benzylpenicillin 1.2 g four hourly and gentamicin 60 mg twice a day for culture negative endocarditis and remained afebrile.

The ESR fell to 40 mm/hour and the C-reactive protein to 20 mg/dl at the end of antimicrobial treatment. He remained well with a good exercise tolerance and a competent normally functioning aortic valve prosthesis for 16 months. Unfortunately he represented as an emergency with acute central chest pain followed by angina on mild exertion and a new aortic diastolic murmur. Transoesophageal echocardiography demonstrated partial dehiscence posteriorly of the proximal and distal suture lines of the Dacron graft. In systole blood entered the perigraft space while in diastole blood passed from a small defect in the distal suture line via the perigraft space into the left ventricle. At surgery the right coronary button was found detached and lying in the perigraft space between the Dacron and a wall of dense adhesions. The suture lines were repaired and the right coronary revascularised with a vein graft. Mediastinitis and breakdown of the sternal wound

Abbreviations: ESR, erythrocyte sedimentation rate; LAD, left anterior descending (artery)

Learning points

- Acute coronary syndromes can occur without atherosclerotic disease.
- Unstable angina in addition to heart failure in a patient with valve disease may be an unusual presentation of endocarditis.

led to a prolonged hospitalisation. Fifteen months after his second operation he died with congestive cardiac failure.

DISCUSSION

Coronary ischaemia in acute endocarditis is usually due to pre-existing coronary disease or occasionally as a result of embolism from vegetations. The mechanism of refractory unstable angina in this patient was likely secondary to extrinsic compression of the proximal LAD and circumflex arteries by the aortic root abscess. Only four single case reports of extrinsic coronary compression complicating aortic root abscess have been described despite the common occurrence of the latter, which has an incidence up to 40% in large autopsy series with endocarditis.¹⁻⁴ Abscesses involving the non-coronary cusp extend towards the atrioventricular node and mitral valve annulus whereas those involving the right or left coronary cusps extend beneath the main pulmonary artery or into the interventricular septum.⁵ Aortic root abscess complicating aortic valve endocarditis commonly develops posteriorly in the region of the mitral aortic intervalvular fibrosa.⁶ Pseudoaneurysm arising from a defect in the prosthesis suture line occasionally complicates aortic root surgery, particularly in the presence of aortic root abscess and may cause extrinsic coronary compression and angina.^{7,8}

Although there are several documented causes of compromised coronary flow, fewer than 5% of patients with acute myocardial infarction do not have atherosclerotic coronary artery disease. Rare causes of extrinsic coronary compression leading to acute coronary syndromes include sinus of Valsalva aneurysms and metastatic deposits.^{9,10} Coronary artery aneurysms generally do not cause extrinsic compression but may cause acute coronary syndromes after in situ thrombosis and embolisation.

This was clearly a difficult and high risk case. In retrospect the outcome may have been better if an aortic homograft had been used for the aortic valve and root replacement. Several surgical series have shown significantly reduced mortality rates and lower incidence of recurrent infection when aortic homografts are used in preference to mechanical prostheses in the context of aortic valve endocarditis with root abscess

formation.^{11,12} Preoperative transoesophageal echocardiography at the initial presentation would have clearly defined the extent of the aortic root abscess. However, in view of the refractory angina and haemodynamic instability emergency surgery was undertaken. The clinical suspicion of endocarditis was not high at presentation because of the absence of fever and the presentation with unstable angina. Transoesophageal echocardiography remains the optimum imaging technique to define the mechanism of aortic reflux and the complications of aortic valve endocarditis, including formation of root abscess and fistulae. Extrinsic coronary compression may be overlooked as a mechanism of acute coronary syndrome and may represent a complication of a more sinister problem.

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