Dehiscence of an atheromatous plaque at an aortic valve commissure: an unusual cause of acute aortic regurgitation

C K MOK, JOHN W L TSO, M AUNG KHIN
From the Division of Cardiothoracic Surgery, Department of Surgery, University of Hong Kong, Grantham Hospital, Hong Kong

SUMMARY A 78 year old man underwent aortic valve replacement because of acute aortic regurgitation and intractable heart failure. At operation the intact commissure between the left and right coronary cusps of a grossly normal aortic valve was found to have separated from the aortic wall. Histopathological examination of the surgical specimen showed that the site of separation was an atheromatous plaque. This is believed to be the first report of this feature.

Severe aortic regurgitation of acute onset is usually caused by aortic dissections or infective endocarditis. Rarely, blunt chest injury and straining can lead to avulsion of a "normal" aortic valve resulting in acute aortic valve incompetence.1-3 We describe an unusual cause of acute aortic regurgitation precipitated by dehiscence of an atheromatous plaque at one of the commissures of a previously normal aortic valve. We believe that this feature has not been reported before.

Case report

During a routine visit to his family doctor, a 78 year old man with mild diabetes mellitus was found to have a cardiac murmur suggestive of aortic regurgitation. The murmur had not been noted by his family physician before. There was no history of chest injury or straining. Three weeks later exertional dyspnoea developed and he was admitted to a general hospital. Severe aortic regurgitation and pulmonary oedema were diagnosed. He was managed with diuretics and digoxin for six days without improvement and was referred to us for consideration of aortic valve replacement.

On admission, he was found to be in a poor general condition with features of left heart failure and severe aortic regurgitation. His electrocardiogram showed sinus rhythm and a pattern suggestive of left ventricular hypertrophy. His chest radiograph showed features of pulmonary oedema. Cross sectional echocardiography showed a mildly dilated left ventricle and a normal sized right ventricle with normal contractions. The anatomy of the aortic and mitral valves was normal. In view of his poor condition, he was intubated and ventilated mechanically. Emergency cardiac catheterisation and cineangiography were planned to exclude aortic dissections. During cardiac catheterisation, he went into a low output state and the procedure was abandoned after an aortogram showed a normal ascending aorta and severe aortic regurgitation. He was transferred directly to the operating theatre and put on cardiopulmonary bypass.

At operation, the ascending aorta was normal externally. The aortic wall was thin and appeared to be normal. The aortic valve was tricuspid with normal looking cusps. There was no macroscopic calcification of the aortic valve or at the aortic sinotubular junction. The intact commissure between the left and right coronary cusps was separated from the aortic wall (fig 1). There was no evidence of infective endocarditis or aortic dissections. The aortic valve was replaced with a 27 mm Medtronic-Hall aortic prosthesis. He withstood the operation well and was returned to the intensive care unit in a satisfactory condition. He had one tarry stool on the first postoperative day and endoscopy of the upper

Requests for reprints to Professor C K Mok, The Grantham Hospital, 125 Wong Chuk Hang Road, Hong Kong.
Fig 1 Photograph at operation showing a normal appearance of aortic root. The intact commissure between the right and left coronary cusps had dehisced from the aortic wall (black arrow). There was no calcification at the aortic sinotubular junction.

Fig 2 (a) Macroscopic appearance of the surgically excised aortic valve cusps and the dehisced commissure showing their normal appearance. (b) Microscopic appearance of a section cut perpendicular to the detached commissure. This shows a hyalinised fibrotic mass with an irregular area of lipid deposition, cholesterol clefts, foamy macrophages, and fibroblasts at the ragged edge of the site of separation. Fibrinous masses and small blood clots are also present on the surface. (Haematoxylin and eosin. Original magnification × 100.)
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gastrointestinal tract failed to show the source of bleeding. During the subsequent two days he remained haemodynamically stable with a satisfactory urinary output. Oliguria developed during the subsequent two days. He remained haemodynamically stable with a satisfactory urinary output. Oliguria developed on the fourth postoperative day and peritoneal dialysis had to be started on the fifth postoperative day. He ran a downhill course with features of adult respiratory distress syndrome, hepatic failure, and fulminating septicemia. He died eight days after aortic valve replacement. Necropsy was refused by his family.

Histopathological examination of the excised aortic valve (fig 2) showed a hyalinised fibrotic mass underneath the commissure between the left and right coronary cusps with an irregular area of lipid deposition, cholesterol clefts, foamy macrophages, and fibroblasts. At the site where the commissure separated from the aortic wall, fibrinous masses and small blood clots were also present on the surface. Calcification or degenerative aortic disease was not seen. Elastic van Gieson stain showed absence of medial elements at the site of dehiscence and evidence that might indicate a localised aortic dissection. Apart from moderate fibrotic thickening and mild patchy mucinous change, the valve cusps showed no abnormality. These features were in keeping with ulceration and rupture of an atheromatous plaque at that site.

Discussion

It is commonly known that atheromatous plaques on the aorta may lead to thinning of the media beneath the plaques; weakening of the aortic wall, and aneurysm formation; or formation of mural thrombus over ulcerated areas, with embolisation to more distal radicles of the arterial system. Detachment of an atheromatous plaque with distal embolisation is also well documented. To the best of our knowledge, however, detachment of an atheromatous plaque leading to aortic regurgitation has not been reported before. The present case shows that this can occur.

References