

## Case reports

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# Dissecting aneurysm of the thoracic aorta presenting as right atrial obstruction

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**SUMMARY** A patient with a dissecting thoracic aortic aneurysm presented with acute low output cardiac failure due to right atrial obstruction. Five years earlier she had had her aortic valve replaced because of aortic regurgitation. Obstruction of the superior vena cava and pulmonary artery has been reported elsewhere as a complication of compression by thoracic aortic dissecting aneurysm. This case was unusual in presenting as right atrial obstruction.

Chronic non-dissecting thoracic aortic aneurysms may be complicated by rupture into or compression of adjacent vascular structures. Recorded vascular complications include aortocardiac fistula formation and fistula formation with or without compression of the superior vena cava and pulmonary artery.<sup>1</sup> Such complications with dissecting thoracic aneurysms, however, have been infrequent; this may be partly because most dissecting aneurysms are acute lesions.<sup>2</sup> We describe a case of dissecting thoracic aortic aneurysm that presented as compression of the right atrium.

### Case report

A 50 year old woman first presented in July 1979 with exertional dyspnoea. She had been treated for hypertension for the past 20 years. Examination showed signs of aortic incompetence, and her blood pressure was 200/75 mm Hg. Investigations confirmed severe aortic incompetence; there was no aortic valve pressure gradient, no evidence of mitral valve disease, and coronary angiography was normal. She underwent aortic valve replacement with a

size 27 Björk-Shiley prosthesis. A standard cardiopulmonary bypass procedure was used with cross clamping of the aorta. The native valve had three cusps and histology showed dense collagenous fibrosis. Recovery after operation was uneventful.

During follow up she remained well, although antihypertensive medication had to be increased. Four years after valve replacement her chest radiograph, which had been satisfactory after operation, showed that the ascending aorta was dilated. She remained symptom free, the prosthetic valve sounds were satisfactory, and no cardiac murmurs were present.

In April 1985 she was admitted to hospital because for four days she had been breathless, weak, and tired but she did not have chest pain. She was pale, cold, and clammy with a respiratory rate of 50 per minute. Her pulse was 120 beats per minute and regular, systolic blood pressure was 50 mm Hg, and the jugular venous pressure was discernible 8 cm above the sternal angle. The prosthetic valve sounds were normal and there were no cardiac murmurs. The chest radiograph again showed a widened mediastinum and the lung fields were clear. Acute pulmonary embolism was suspected and the patient was transferred for further investigation.

On arrival at this hospital she had a profound metabolic acidosis (arterial pH 7.06). Echocardiography

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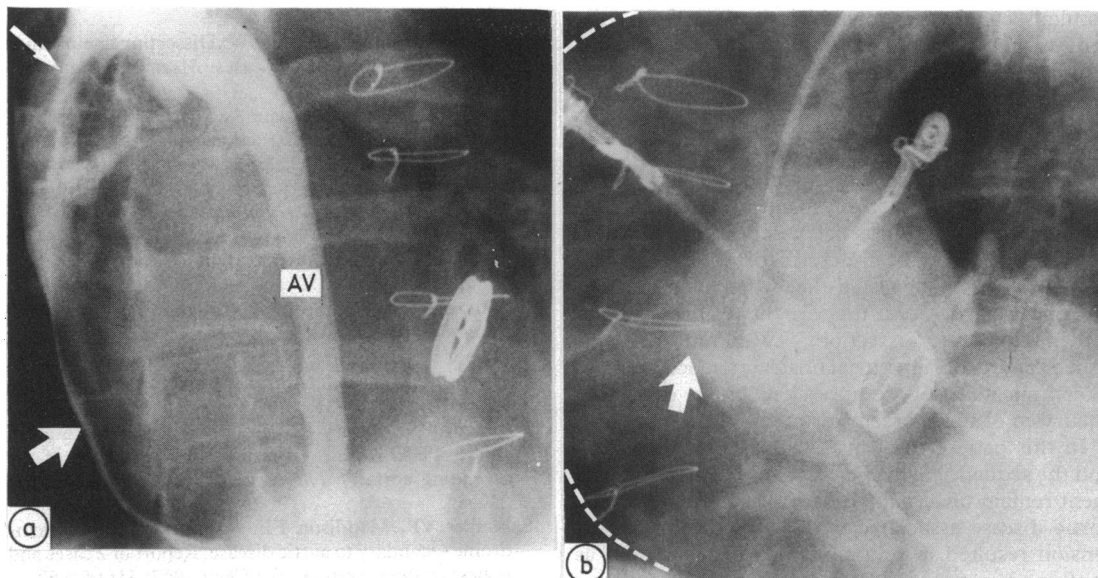


Fig. 1 (a) Injection via catheter in superior vena cava (small arrow) with contrast in the right atrium (large arrow) and azygous vein (AV). There is a huge filling defect in the right atrium. An aortogram (b) shows a dilated ascending aorta and the entry point into the false lumen (arrow) whose limit is indicated (dotted line).

was technically difficult and unhelpful. During right heart catheterisation it was difficult to manoeuvre the catheter in the right atrium. Injection of contrast into the superior vena cava showed a huge filling defect in the right atrium. Contrast flowed to the inferior vena cava via the azygous system and thence to the right atrium and right ventricle (Fig. 1a). Simultaneous recording of pressure in the superior vena cava and right ventricle showed a mean end diastolic pressure gradient of 10 mm Hg (Fig. 2).

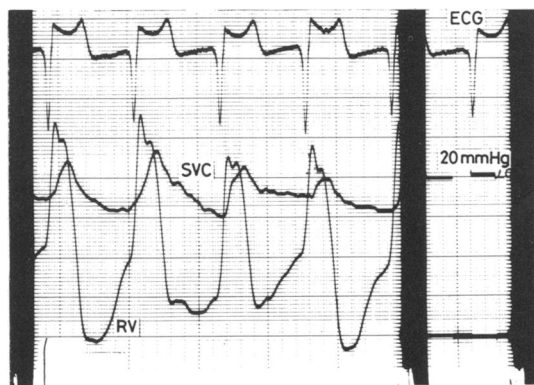


Fig. 2 Simultaneous recording of superior vena cava (SVC) and right ventricular (RV) pressures showing an end diastolic gradient. ECG, electrocardiogram.

Pulmonary arterial oxygen saturation was 59%, right atrial saturation was 57%, and aortic saturation was 93%. An aortogram showed gross dilatation of the ascending aorta. There was a dissection with the entry point 3–4 cm above the aortic valve. The false lumen was compressing the right atrium (Fig. 1b). Operation offered this patient the only possible chance of survival, and her clinical condition was poor.

At operation a massive ascending aortic aneurysm was found. The aneurysm was incised and the false lumen was entered. The old and new thrombus that it contained was removed. There was a tear in the right side of the ascending aorta 4 cm above and parallel to the previous valve suture line. The aneurysm had eroded the superior and medial walls of the right atrium; these were repaired and the ascending aorta was replaced by a woven Dacron graft. The patient required inotropic agents after operation and an intra-aortic balloon was subsequently inserted. She deteriorated progressively and died two days after operation.

## Discussion

The vascular complications of chronic non-dissecting thoracic aortic aneurysms include obstruction of the superior vena cava and pulmonary artery together with fistula formation with these structures and also aortocardiac fistulas. Reports of

fistulous communications or compression of adjacent vascular structures by dissecting aneurysms, however, have been infrequent. One case of aorto-left atrial fistula<sup>3</sup> and four cases of aorto-right atrial fistula formation<sup>4-7</sup> associated with aortic dissection have been reported. Dissections may obstruct the superior vena cava and pulmonary artery as a result of extension of an intrapericardial haematoma; direct aneurysmal compression of these structures has also been described.<sup>8-10</sup> Morris and Barwinsky have suggested that when such vascular complications do occur they are the result of chronic rather than acute dissection.<sup>11</sup> We found no published reports of right atrial obstruction occurring as a complication of compression by a dissecting aneurysm of the thoracic aorta.

In this patient the cause of the aortic dissection and the sequence of events after aortic valve replacement remain uncertain. It seems likely that chronic aortic disease associated with longstanding hypertension resulted in progressive aortic dilatation, as confirmed on serial chest radiographs, and that this was eventually complicated by acute aortic dissection. Both aortic cannulation and cross clamping during cardiopulmonary bypass, however, may occasionally produce acute aortic dissection.<sup>12,13</sup> It is therefore possible that aortic dissection, which was not initially apparent radiologically, occurred after operation. A slow progressive enlargement of the false lumen could then explain the subsequent clinical and radiological course in this patient.

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