

Case reports

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Rupture of an aortic aneurysm into the pulmonary artery

*An unusual case with antemortem diagnosis*¹

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An acquired aorta-to-pulmonary artery shunt from rupture of a thoracic aortic aneurysm in an elderly patient is reported. The diagnosis was suspected clinically and was confirmed by special cardiac studies. Surgical intervention was refused by the patient who died shortly after admission to the hospital.

Rupture of an aneurysm of the thoracic aorta into the pulmonary arterial tree is an uncommon event (Boyd, 1924; Giacobine and Cooley, 1960). The diagnosis is usually established at necropsy; only rarely has an antemortem clinical diagnosis been made (Buttross and Salatich, 1955; Mark *et al.*, 1958; Schattenberg and Harris, 1943). The lesion is potentially surgically correctable, though surgery has rarely been performed (Schattenberg and Harris, 1943). An elderly patient is described in whom the diagnosis of rupture of the thoracic aorta into the pulmonary artery was suspected on clinical grounds and confirmed at cardiac catheterization. The patient refused surgical correction of her lesion.

Case report

A 72-year-old woman was admitted to the Medical Service of Grady Memorial Hospital on 15 January 1969, because of weakness and chest, back, and shoulder pain which progressed over a four-day period. She had never been seen by a physician until 1968, at which time she had a blood pressure of 200/130 mm. Hg. Clinical investigation revealed no cause for the hypertension. She was satisfactorily treated with alpha methyl-dopa and thiazide diuretics. The chest radiograph showed a large 'egg-shaped' aneurysm of the terminal aortic arch and proximal descending aorta; cardiomegaly was present (Fig. 1A). The electrocardiogram showed regular sinus rhythm and left axis deviation. There was no history of chest trauma, syphilis, or previous heart disease. The VDRL (Venereal Disease Research Laboratory) and FTA (fluorescent treponema antibody absorption) tests were negative.

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The patient did well until four days before admission when she developed severe chest, back, and shoulder pain. The pain was sharp and was aggravated by the supine position. The pain continued until admission and was associated with anorexia, weakness, and severe dyspnoea.

Physical examination revealed an acutely ill dyspnoeic, tachypnoeic, sweating woman. The blood pressure was 106/60 mm. Hg. The pulse rate was 120/min. The pulses were bounding, symmetrical, and of good volume. There was no pulsus paradoxus or alternans. The jugular venous pulse was palpable and showed both inspiratory accentuation and rapid 'y' collapse. Breath sounds were diminished over the entire left hemithorax. A mediastinal shift was not detectable. The entire left anterior chest was hyperdynamic, with the cardiac impulse in the sixth interspace in the left anterior axillary line. Both components of the second heart sound were accentuated and splitting seemed fixed during respiration. A loud ventricular diastolic gallop was present. The most striking auscultatory finding was the presence of a grade 2/6 continuous murmur which peaked at the second heart sound and was well localized to the second and third interspaces at the left sternal border. The liver was slightly enlarged and tender. The remainder of the physical examination was normal. Admission haematocrit was 27 per cent and the blood urea nitrogen was 70 mg./100 ml. Chest radiograph showed an obvious increase in the size of the aneurysm and a further increase in heart size (Fig. 1B). The electrocardiogram showed sinus tachycardia, left axis deviation, and ST and T wave changes compatible with myocardial ischaemia.

Shortly after admission, emergency right heart catheterization and supravulvar aortography were performed. A left-to-right shunt was demonstrated by hydrogen curve analysis at the level of the pulmonary artery. There was a moderate increase

TABLE Catheterization data

	Pressures (mm. Hg)
Aorta	110/60 (82)
Pulmonary artery	60/33 (35)
Right ventricle	60/22
Right atrium	(18→22)
'a' 20; 'x' 13; 'v' 20; 'y' 9	

Figures in parentheses are means.

of the pulmonary artery pressure and evidence of acute right heart failure (Table). Supravalvar aortography showed the large aortic aneurysm with aorta-to-pulmonary artery shunt. Aortic regurgitation was not seen (Fig. 2). The patient was offered immediate operation, but refused. She died four hours later of unrelenting heart failure. Permission for necropsy was not granted.

Discussion

Aneurysms of the ascending aorta are most commonly due to cardiovascular syphilis (Boyd, 1924). They are now less frequently seen than in previous years but remain a challenge to the clinician, because dangerous complications may occur from the lesion and because the lesion is surgically correctable (Buttross and Salatich, 1955). The aneurysm may rupture externally or into the oesophagus or tracheobronchial tree and cause prompt death from exsanguination (Buttross and Salatich, 1955). Rupture into the pericardium, mediastinum, or pleural spaces is also rapidly fatal (Buttross and Salatich, 1955). Other complications are compatible with a less rapidly deteriorating clinical course. Many of these complications are remediable by operation, including superior vena cava syndrome, bronchial compression and obstruction, compression of the pulmonary artery with resultant right ventricular failure, obstruction of the coronary ostia, and aortic regurgitation. An acquired left-to-right shunt may occur from rupture of the aneurysm into the superior vena cava or the pulmonary arterial tree. Our patient represents the latter.

In 1924 Boyd reported 4000 aneurysms of the thoracic aorta. Approximately 25 per cent of the aneurysms had ruptured, but only 3 per cent had ruptured into the pulmonary arterial tree. It is also noteworthy that rupture into the tracheobronchial tree occurs five times more frequently than into the pulmonary artery (Giacobine and Cooley, 1960).

Rupture of an aortic aneurysm into the pulmonary artery is more common when the aneurysm is in the ascending aorta; the frequency of rupture into the pulmonary artery decreases with aneurysm location in the arch

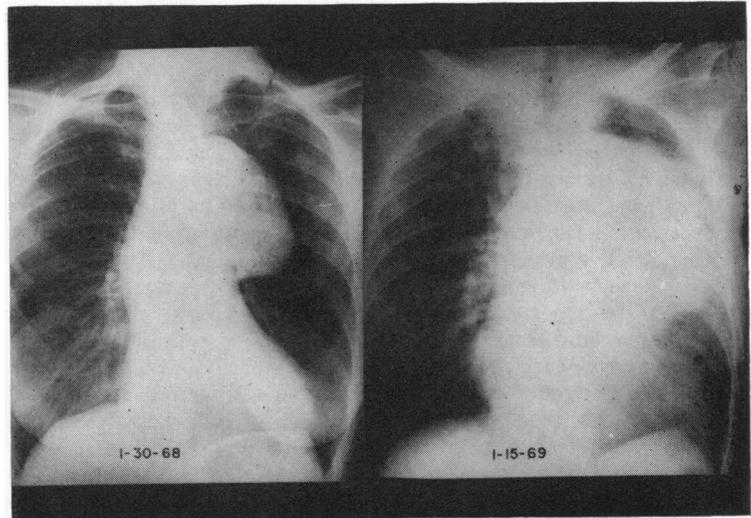
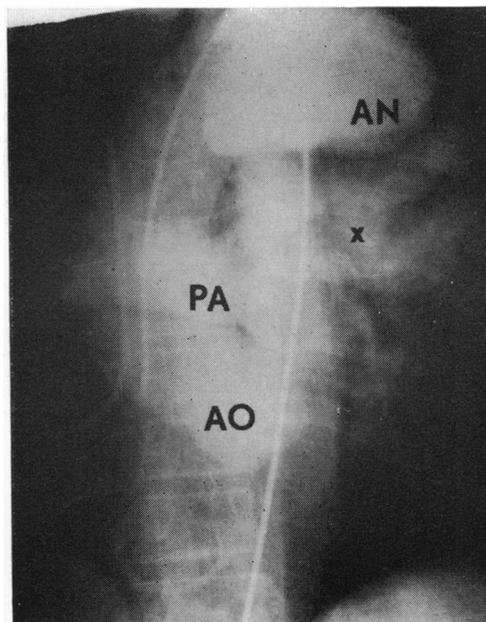


FIG. 1 (A) Large 'egg-shaped' aneurysm of aorta with cardiomegaly. Note flecks of calcium in aneurysm. (B) Chest x-ray at time of admission showing increase in size of aneurysm and heart.

FIG. 2 Supravalvar aortogram with catheter just above root of aorta (AO). Injection reveals large aneurysm (AN) with filling of the pulmonary artery (PA) from the aorta. 'X' indicates the suspected area of rupture. Note absence of aortic regurgitation.



and descending thoracic aorta. The aetiology of the aneurysm in our patient is not clear. The position of the aneurysm (Fig. 1) is more typical of an aneurysm secondary to trauma. The patient's history was negative for severe chest trauma, but previous, apparently trivial, trauma could not be ruled out.

Until 1955 only two patients were proven by antemortem cardiac catheterization to have rupture of an aortic aneurysm into the pulmonary artery (Klein and Porter, 1950). Isolated case reports have since appeared (Schattenberg and Harris, 1943; Buttross and Salatch, 1955; Mark *et al.*, 1958), with only rare surgical correction of the lesion (Schattenberg and Harris, 1943).

The clinical course after rupture of the aorta into the pulmonary artery is variable and depends upon the size of the communication and the general cardiovascular status of the patient. Death may be rapid (Schrire, Beck, and Barnard, 1963), though survival for over four years without surgical intervention has been reported (Scott, 1924). The clinical picture is usually of congestive heart failure with evidence of a left-to-right extracardiac shunt, mimicking a large persistent ductus arteriosus. The presenting complaints include dyspnoea, fatigue, abdominal pain from visceral congestion, ascites, peripheral oedema, and shock. The onset of symptoms may be insidious or may be immediate and dramatic. Often earlier history of cardiovascular syphilis is lacking. Sudden chest pain may represent the actual rupture of the aortic aneurysm. In the patient who survives long enough to seek medical attention, sinus tachycardia and bounding peripheral pulses are usually present. The jugular venous pulse is full, with a prominent 'a' wave. A thrill may be palpable over the upper anterior thorax, and a continuous murmur is usually heard in the second and third left intercostal spaces. The murmur has been described as 'machinery', 'to-and-fro', and 'humming top' (Schrire *et al.*, 1963; White, Chamberlain, and Kelson, 1941). Tender hepatomegaly, ascites, and peripheral oedema are variable. The electrocardiogram often shows acute right ventricular overload, and right ventricular hypertrophy if the clinical course has been prolonged; however, the electrocardiogram may be normal. The chest x-ray often shows

pulmonary plethora; however, evidence of aortic aneurysm may be lacking (Giacobine and Cooley, 1960; Schrire *et al.*, 1963; White *et al.*, 1941). The diagnosis is established by cardiac catheterization and angiographic studies. Cardiac catheterization reveals a left-to-right shunt at the pulmonary artery level. The pulmonary artery, right ventricular end-diastolic and right atrial pressures are usually raised. An ascending aortogram will delineate the extent and location of the aneurysm, the shunt, and the presence or absence of aortic regurgitation.

The clinical course appears to be either one of rapid death or of a progressive downhill course, with death occurring in a period of weeks to years.

Because of the universally unfavourable outlook, once the diagnosis is suspected, it should be confirmed by cardiac catheterization and angiography; operation should be recommended to the patient at the earliest possible time.

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