

Fatal massive haemoptysis after embolectomy for chronic pulmonary embolism

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Since the first report of successful "late" embolectomy for recurrent pulmonary embolism,¹ a number of patients have been described with significant pulmonary hypertension where clinical improvement associated with lowering of pulmonary artery pressure has followed removal of extensive, well-organised thrombo-embolic material from major pulmonary vessels.² The following case history illustrates an unfavourable and highly unusual outcome in a patient with chronic pulmonary embolism complicated by severe, sustained pulmonary hypertension. Embolectomy was followed, five months later, by sudden death with massive haemoptysis from a fistula between a pulmonary artery and main bronchus, a complication not previously reported.

Case report

A previously healthy 22-year-old housewife presented to an outlying hospital in May 1969 with symptoms and signs suggesting acute pulmonary embolism. Her oral contraceptive was withdrawn but she did not receive anticoagulant treatment. A second episode followed in March 1971 when the patient was admitted to Glasgow Western Infirmary. Treatment with oral anticoagulants was discontinued six months later before laparoscopic sterilisation. However, in March 1972 she was readmitted with her third clinical episode of acute pulmonary embolism. On this occasion bilateral lower limb phlebography disclosed the presence of a persistent filling defect in the left iliac vein. When her clinical condition permitted the inferior vena cava was plicated.

Between 1972 and 1975, despite good control of oral anticoagulant therapy, effort dyspnoea noticeable on moderate exertion became a feature. Treatment with Warfarin was finally stopped in June 1976 because of severe menorrhagia, at a time when the patient's clinical condition was deemed stable. Increasing effort dyspnoea over a period of four months culminated in her emergency admission in April 1977 after sudden onset of dyspnoea at rest. She was severely hypoxic, hypotensive, and oliguric. After clinical improvement with supportive treatment and intravenous heparin, pulmonary angiography (fig 1) showed a large filling defect in the right main pulmonary artery with virtual absence of perfusion throughout the right lung and lesser perfusion defects in the left middle and lower zones.

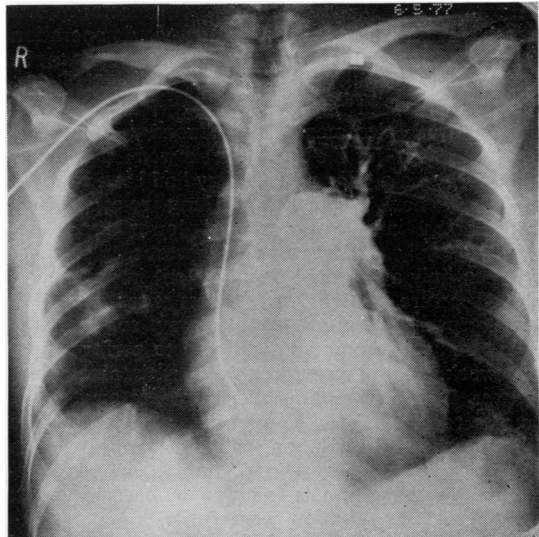


Fig 1 Preoperative pulmonary angiogram.

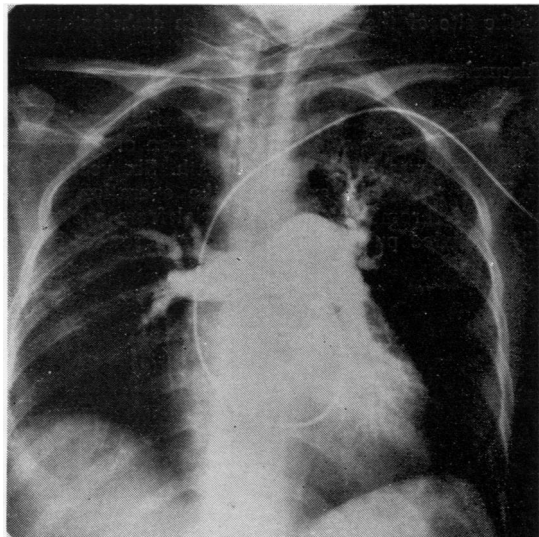


Fig 2 Pulmonary angiogram after right pulmonary embolectomy.

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Table Pressure recordings (mmHg) before and after pulmonary embolectomy

	Site			
	Main pulmonary artery (mean)	Right ventricle	Right atrium—"a"	Right atrium—"v"
Before operation	100/35 (60)	80/0	12	8
After operation	90/30 (48)	90/0	14	8

Pulmonary hypertension was severe (table). Two weeks after admission pulmonary embolectomy was undertaken on cardiopulmonary bypass. At operation the right main pulmonary artery was found to be occluded proximal to its bifurcation with old, friable, partially organised and adherent thrombus which was removed piecemeal. A pulmonary angiogram (fig 2) 10 days after the operation showed that patency of the right main pulmonary artery had been restored and blood flow to the right lung re-established. Large areas in the upper and lower zones remained grossly underperfused, however, and there had been only a marginal improvement in pulmonary artery pressure (table). Nevertheless there was clinical improvement after embolectomy so that the patient returned home and was able to perform light housework. This improvement was maintained until October 1977, five months after embolectomy, when she was readmitted for routine reassessment. Before right heart catheterisation could be repeated the patient experienced a sudden, unheralded, and exsanguinating haemoptysis followed by cardiac arrest. Attempts at resuscitation were futile.

Postmortem examination showed that death had resulted from rupture of the right main pulmonary artery into the adjacent right main bronchus. Histology demonstrated severe changes of pulmonary hypertension and confirmed the presence of a fistula at the site of the patient's previous embolectomy.

Discussion

In 1950 Carroll³ reported on the first patient in whom a diagnosis of chronic occlusion of a pulmonary artery was made before death. Six years later Hollister and Cull⁴ suggested the operation of removal of thrombo-embolic material from a chronically occluded pulmonary artery. In a review of the

literature up to 1977 Sabiston *et al*² described 18 patients with chronic pulmonary embolism treated by embolectomy: four deaths all occurred during or shortly after operation. Continued clinical improvement in those surviving operation was associated with lowering of mean pulmonary artery pressure and long-term patency of the main pulmonary arteries up to eight years after the operation. A successful result has been reported even when embolic obstruction was likely to have been present for 10 years and was associated with severe pulmonary hypertension.⁵

Emphasis has been given to the relative ease with which thrombotic material of some chronicity may be removed from major pulmonary vessels, even when no obvious plane of cleavage has existed between clot and intima. Despite claims of angiographic evidence of healing after delayed embolectomy, it is surprising that the inevitable trauma to the vascular endothelium has not been reported as a cause of significant morbidity. In the present case it is likely that the vascular trauma inflicted by embolectomy in association with severe, established pulmonary hypertension persisting after the operation was responsible for the fatal outcome. Clearly, the removal of organised thrombus from a chronically occluded main pulmonary artery may be associated with significant vascular injury and this may contribute to mortality.

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