

Fatal intra-alveolar pulmonary bleeding complicating pulmonary embolectomy

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Two patients are reported in whom fatal alveolar pulmonary haemorrhage occurred after pulmonary embolectomy. Possible causes and methods of prevention are discussed.

Pulmonary haemorrhage after embolic occlusion of a pulmonary artery usually follows the infarction of lung parenchyma. This complication is particularly prevalent in patients suffering from chronic congestive cardiac failure. The usual finding in such cases is obstruction of a segmental or smaller artery with infarction of the parenchyma supplied by it. The haemorrhage itself is usually slight and unimportant as regards eventual prognosis.

Parenchymal bleeding following obstruction of the main branches of the pulmonary artery, without overt evidence of infarction, and severe enough to cause death at a time when the obstructing embolus has been removed, is a rare event.

The purpose of this communication is to describe two patients in whom fatal pulmonary haemorrhage occurred following the removal of the obstructing clot. This complication of pulmonary embolectomy is relevant, as several non-operative methods of treating acute pulmonary embolism have recently been introduced.

CASE HISTORIES

CASE 1 Mrs. R. S., aged 55 years, had a right partial nephrectomy for renal calculus. There was no previous history of cardiac disease. On the fifth post-operative day thrombophlebitis occurred in the left leg. A few hours later chest pain, dyspnoea, and a fall in blood pressure occurred. Heparin therapy was started, but 22 hours later there was a further episode with dyspnoea, cyanosis, and hypotension. An electrocardiogram showed acute right heart strain (Fig. 1). A pulmonary angiogram (Fig. 2) showed a complete blockage of the right main pulmonary artery and a further block in the artery to the left lower lobe.

In view of continued deterioration despite routine medical treatment, emergency pulmonary embolec-

tomy with cardiopulmonary bypass was carried out 34 hours after the initial embolus. A large coiled clot was removed from the right main pulmonary artery, and smaller fragments from the left lower lobar artery. The total time on full bypass was 20 minutes. While weaning off bypass there was a massive exudation of haemorrhagic pulmonary oedema fluid from the endotracheal tube. This rapidly resembled pure blood and appeared to come from the right lung, which looked congested and infarcted. The left lung became flooded, despite attempts to exclude the right with a Carlen's tube. It proved impossible to oxygenate the arterial blood, and the patient died from cardiac arrest.

Necropsy showed a healthy heart and coronary vessels. There was antemortem thrombus in the left femoral vein. Fluid blood was present in the right bronchial tree. The right lung was soft and haemorrhagic with antemortem thrombus in the smallest arteries, but the major pulmonary vessels were normal. Histological examination confirmed infarction of the parenchyma (Fig. 3). An incidental finding was a recent right adrenal haemorrhage (Fox, 1969).

CASE 2 Mr. J. W., aged 53 years, had suffered from myelofibrosis for many years. A splenectomy was performed for massive splenomegaly. He had been on steroid medication for the haematological condition, and the operation was performed under steroid cover. This operation was performed with no untoward effects or unusual bleeding.

On the fourth postoperative day he collapsed on his way to the lavatory. A clinical diagnosis of acute pulmonary embolism was made, and in view of the inability to sustain the circulation despite vasopressors, emergency embolectomy was undertaken without further investigation.

Cardiopulmonary bypass was begun four hours after the collapse. Fragmented clot was present in the right pulmonary arterial tree. This was removed and the artery was closed. A few seconds after the heart was restarted haemorrhagic fluid poured from the trachea. This became almost pure blood. A

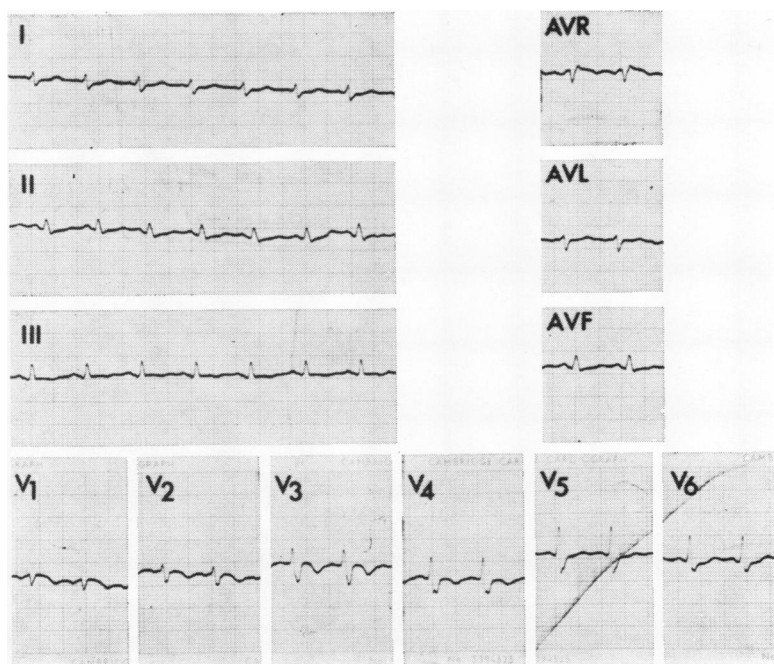


FIG. 1. Case 1. ECG shows right axis deviation and T-wave inversion in the right chest leads indicative of acute right heart strain.

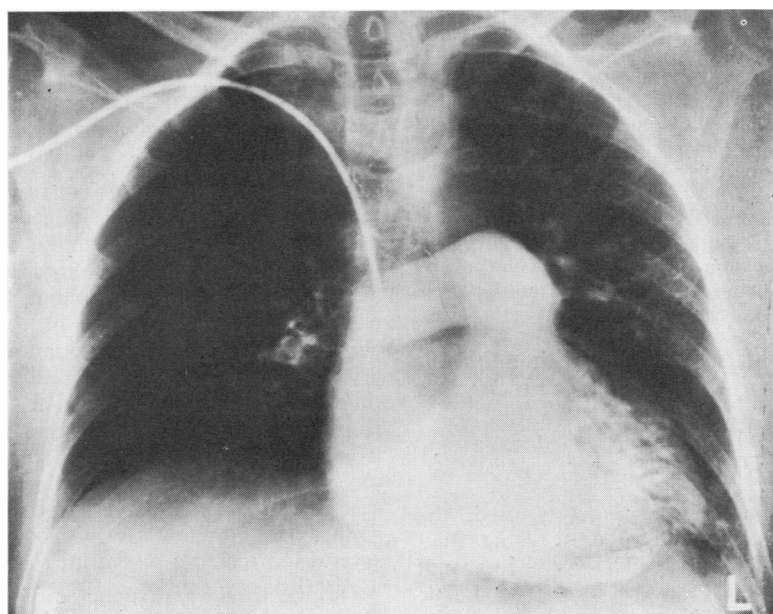


FIG. 2. Case 1. Angiocardiogram with injection of contrast into right atrium shows absence of filling of the pulmonary arterial tree on the right.

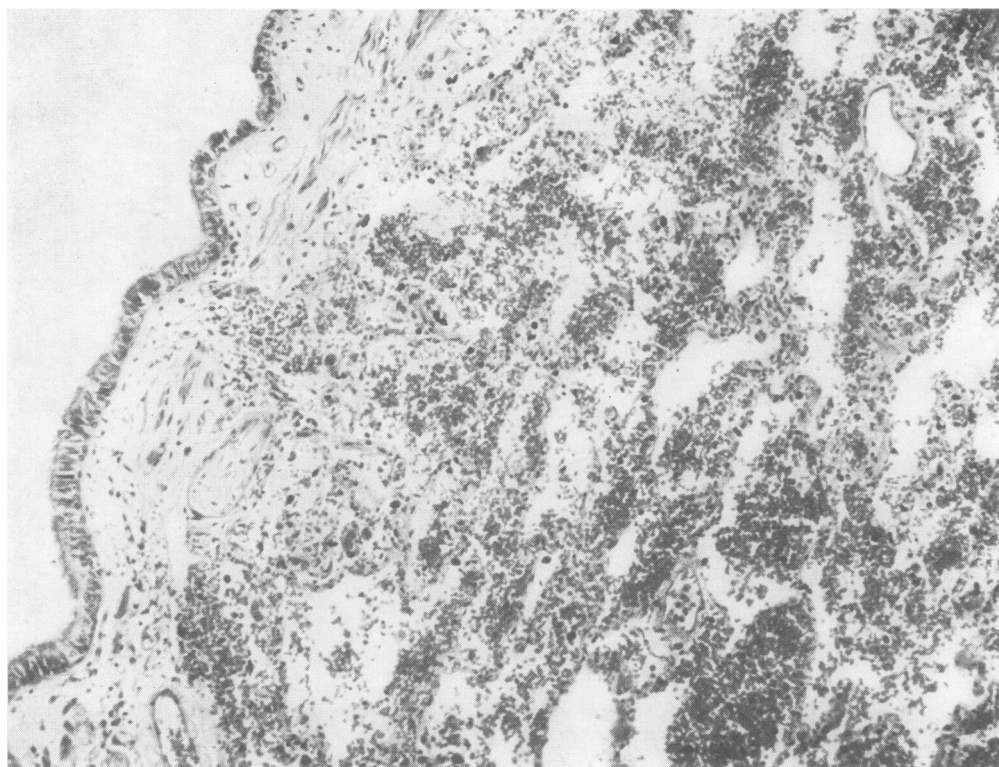


FIG. 3. Case 1. Section of lung shows severe intra-alveolar haemorrhage. ($\times 100$).

Carlen's tube was passed, and this enabled the source of the bleeding to be identified as from the right main bronchus. The haemorrhage proved uncontrollable, and the patient died in asystolic cardiac arrest.

Gross anatomical examination after death showed a congested right lung, which appeared infarcted. Histological examination showed the alveoli to be filled with blood but no established infarction. There was disruption of the alveoli in some parts of the lung, and others showed areas of thickening and giant-cell infiltration. Intravascular thrombosis was present in the lung (Fig. 4). The adrenals were normal.

DISCUSSION

Although the term 'haemorrhage' is used, the exudate begins as a bloodstained oedema fluid and may not be pure blood. (No haematocrit values are available.) Thus the picture is similar to the haemorrhagic pulmonary oedema of the newborn (Adamson, Boyd, Normand, Reynolds, and Shaw, 1969), which occurs in conditions in

which asphyxia is present, or likely to be present, as well as having an association with raised left atrial pressure.

The incidence of this complication of pulmonary embolectomy as judged from a survey of the literature appears to be small. Only one identical case was discovered in the English language literature (Castleman, 1964). In this patient, successful removal of the embolus had been carried out and cardiopulmonary bypass was discontinued before the intrapulmonary haemorrhage began. Pulmonary oedema fluid welled up from the tracheostomy tube and the patient died from hypoxia despite ventilation with 100% oxygen. Another point of similarity to our second case was that this patient had been on steroid medication preoperatively. Necropsy showed several infarcts and severe intra-alveolar bleeding.

Despite the lack of detailed reports fatal intra-pulmonary bleeding in patients suffering from pulmonary embolism is mentioned. Stirling *et al.* (1968) mention three patients who died with

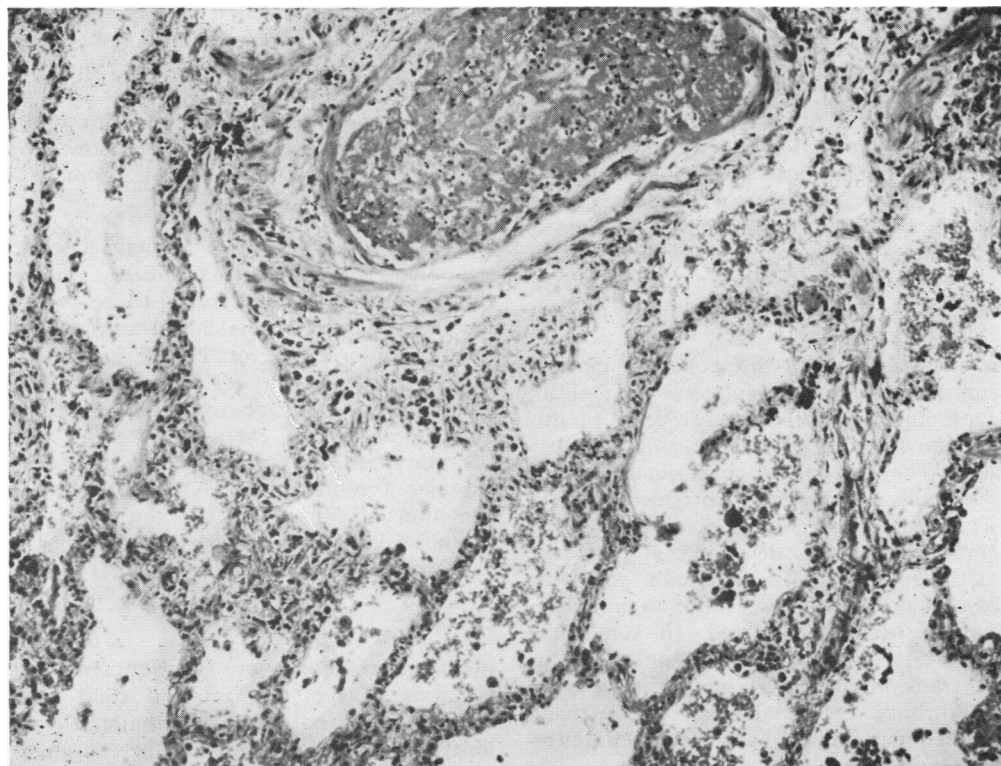


FIG. 4. Case 2. Section of lung shows some intra-alveolar haemorrhage and antemortem thrombus. ($\times 160$).

three days after embolectomy from extensive intrapulmonary bleeding. Lam, Taber, and Gahagan (1967) report a patient who died of uncontrollable haemorrhage after embolectomy, but the site of bleeding is not mentioned. Hirsh *et al.* (1968), discussing streptokinase therapy for acute pulmonary embolism, attribute a falling haematocrit in some patients to intrapulmonary haemorrhage. However, Cross and Mowlem (1967) did not record this complication in a review of 137 pulmonary embolectomies, and hence its true incidence remains uncertain.

In our patients the clinical course and necropsy findings suggest that extensive damage to the lung vessels at capillary level was present, but any tendency to haemorrhage was prevented by the obstructing embolus. When this was removed, and pulmonary arterial blood flow was restored, the weakness in the capillary walls was revealed. In short, subclinical infarction had occurred and the restoration of blood flow converted it into a 'haemorrhagic' pulmonary infarction. Several studies have supported these observations (Agrest,

Lanari, and Roncoroni, 1953; Roach and Laufman, 1955).

HAEMODYNAMIC CONSIDERATION The infarction of 'lung tissue' in patients without previous cardiac or pulmonary pathology is difficult to explain. Virchow (1856) observed that obstruction of the branches of the pulmonary artery was not in itself sufficient to cause pulmonary infarction. Karsner and Ash (1912) demonstrated that infarction following experimental pulmonary embolism occurred only if congestion of the lung by ligation of the lobar veins or compression by pleural effusions had been produced previously. Other workers (Roach and Laufman, 1955; Chapman, Gugle, and Wheeler, 1949) have generally confirmed these findings. The only exception to the thesis that pre-existing derangement of lung circulation has to be present for pulmonary embolism to result in infarction is in the work of Steinberg and Mundy (1936), who produced infarcts in dogs using multiple lead shots as emboli.

Sufficient nutrition to maintain the integrity of

the pulmonary tissues when massive embolic obstruction of lobar branches of the pulmonary arteries occurs would appear to come from anastomoses with the bronchial arteries. These anastomoses are normally present, and have been shown to increase following experimental ligation of a major pulmonary artery. In dogs, this collateral flow reaches $1 \text{ l./m}^2/\text{min}$, roughly a third of the total right ventricular output (Bloomer, Harrison, Lindskog, and Liebow (1949). Good bronchial flow to infarcted areas in dogs is present within six days, reaching a maximum at four weeks (Steinberg and Mundy, 1936).

When interference with this collateral circulation occurs concomitant with pulmonary embolism, necrosis of pulmonary tissue may occur. In this connexion the work of Chapman *et al.* (1949) is interesting. They produced experimental pulmonary embolism using clots produced in the veins of normal dogs. Neither large nor small emboli produced infarcts detectable at necropsy. However, when pulmonary congestion was produced by alphanaphthylthiourea prior to embolism then alveolar necrosis occurred. In some this consisted of alveolar haemorrhage only, and in others of frank infarction. Hampton and Castleman (1940) have called alveolar haemorrhage without other pathological evidence of infarction 'incomplete infarction'.

Systemic hypotension, when present, is dangerous for two reasons. First, the impairment of coronary flow may cause left ventricular failure and, secondly, reduction of bronchial flow may exacerbate the existing pulmonary ischaemia. Systemic hypotension requires treatment with pressor agents or with partial bypass to assist the circulation.

Elevation of pressure or distension of the left side of the heart due to 'heart failure' or to increased pulmonary venous return following relief of obstruction might result in capillary damage. This could be prevented by venting the left side.

Steroid function A feature common to our cases and to the one from the Massachusetts General Hospital (Castleman, 1964) is some abnormality of factors governing corticosteroid production. Case 1 had an undiscovered haemorrhage in the right adrenal; case 2 had been on chronic steroid medication, as had the case reported by Castleman. We are unable to implicate a definite mechanism. Steroids have an influence on thrombogenesis, and in Castleman's case every attempt to discontinue steroids was associated

with a recurrence of chest pain. It is possible that steroid medication resulted in an increased propensity to thrombose the smaller lung vessels. Patients who are not on treatment with steroids but whose acute pulmonary embolus was otherwise managed identically have been free from this complication (Hampton and Castleman, 1940; Makey and Bliss, 1966).

Coagulation defects All patients on cardiopulmonary bypass are heparinized, yet haemorrhage from the lungs appears to be rare, and it would seem unlikely that heparinization can be a major aetiological factor. Indeed, it is known that in case 1 all heparin was neutralized by the end of the operation, as judged by protamine titration, and there was no excessive bleeding from the operation site. It is possible that extensive intravascular thrombosis (Fig. 4) may have led to depletion of fibrinogen, but the normal wound haemostasis and the clotting in laboratory investigations does not support this.

CONCLUSIONS

Pulmonary embolectomy is an uncommon operation, and out of our total of eight cases two showed this apparently rare complication. As is hinted at in the literature, other surgeons may have experienced but not recorded it. A rise in left atrial pressure was not considered to be responsible for the bleeding, as both patients were still on partial bypass at the time of its occurrence. Left atrial pressure monitoring is not regularly performed in this emergency.

Apart from steroid medication or adrenal pathology, there does not appear to be any common factor by which these patients may be identified. The occurrence of this complication unexpectedly at the end of an otherwise successful and uncomplicated pulmonary embolectomy, and the fatal outcome in both cases, influence opinion in the direction of non-operative management. Several methods are now available, including the use of streptokinase, urokinase and the venom of the Malayan pit viper, Ancrod. Since 1968 almost all our cases have been treated with streptokinase through the pulmonary artery by catheter used for angiography. Our results have been so satisfactory that the practical conclusion to be drawn from our experience is that the management of major pulmonary embolism should be by fibrinolytics, provided the patient is likely to live long enough for these to take effect. If inadequate improvement occurs, embolectomy can be performed later.

If operation is unavoidable, then adequate steroid replacement therapy should be instituted, systemic hypotension treated with vasopressors or supportive perfusion, and the distension of the left side of the heart avoided. If pulmonary haemorrhage occurs, a trial of cross-clamping the pulmonary artery and bronchus at the hilum on the more affected side may be performed as a desperate measure to prevent the other lung being swamped with the exudate. Should this succeed in controlling the haemorrhage, then pneumonectomy should be considered.

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