Haemorrhagic pulmonary oedema: post-pulmonary embolectomy

JULIUS W. GARVEY, GEORGE WISOFF,
CHOUDARY VOLETTI, and MARVIN HARTSTEIN

*Cardiothoracic Service, Division of Surgery, Long Island Jewish Hospital,
New Hyde Park, New York, USA*

Garvey, J. W., Wisoff, G., Voletti, C., and Hartstein, M. (1976). Thorax, 31, 605-609. Haemorrhagic pulmonary oedema: post-pulmonary embolectomy. A case of haemorrhagic pulmonary oedema after successful pulmonary embolectomy is presented. The relevant literature is reviewed. Thirteen cases are analysed as well as the four survivors. The aetiology appears to be ischaemic damage of the capillary bed. This has previously been called incomplete infarction by Castleman. The incidence is low after acute pulmonary embolectomies but appears to be much higher after chronic endarterectomies, especially with severe pulmonary hypertension. Therapy is outlined.

The purpose of this report is to document the complication of 'haemorrhagic pulmonary oedema' subsequent to successful pulmonary embolectomy, a complication not stressed in the literature.

CASE REPORT

On 12 November, a 37-year-old man was brought to the emergency room at Queens Hospital Center complaining of severe back pain as a result of a car accident. His blood pressure was 40 mmHg systolic and he had a laceration of the left cheek.

The abdomen was rigid and paracentesis was positive for blood. Radiographs revealed fractures of the left 8th and 9th ribs and left superior pubic ramus. A splenectomy was done for a ruptured spleen, and a lacerated kidney was repaired. His subsequent course was one of deterioration of pulmonary status and on the second postoperative day the Po_2 was 34 mmHg breathing room air. An initial chest radiograph showed an ill-defined opacity in the right upper lobe, and on 14 November there was extensive alveolar filling of the left lung. He had a minor transfusion reaction probably secondary to leukoagglutinins. The possibilities of blood transfusion reaction, fat embolism, and aspirations were entertained. He was started on steroids because of the possibility of fat embolism and on heparin because of the necessity for prolonged bed rest due to the pelvic

fractures. His initial studies showed a 20% pulmonary shunt. He was placed on 100% oxygen by face mask with an arterial Po_2 of 53.5 mmHg. Over the next 48 hours the Fio_2 was gradually reduced to 0.45 with a Po_2 in the high sixties. On 20 November on room air he had a Po_2 of 66 mmHg.

The patient's progress was gradual. He continued to run a low-grade fever, and radiographs showed a raised left diaphragm and some left basilar atelectasis; the possibility of a subphrenic abscess was considered. He was started on clindamycin on 26 November.

On 28 November he complained of dizziness and became diaphoretic and pale. His blood pressure was 50 mmHg systolic. A chest radiograph still showed an atelectatic area of the left lower lobe and a raised left diaphragm. An electrocardiogram was without significant change. His haematocrit was 29%, Po_2 73 mmHg on room air, pH 7.5, and Pco_2 25 mmHg. With intravenous infusion of saline his blood pressure was 100/70 mmHg. He was transfused two units of packed cells overnight. However, his blood pressure remained below 100 mmHg. His central venous pressure rose to 20 cmH₂O and he became oliguric and then anuric the next day. By the next morning he was complaining of tight chest pain over the precordium. He had a loud P_2 and had both tachycardia (120/min) and tachypnoea

(28/min). His ECG showed inverted T waves over the right precordium. A diagnosis of pulmonary embolus was made and a lung scan was performed. It showed decreased perfusion in the right upper lobe, the left apical region, and the left mid lung field. We were consulted and advised right heart catheterization and a pulmonary angiogram. The right atrial pressure was 14 mmHg, right ventricle systolic pressure 50 mmHg, pulmonary artery mean pressure 35 mmHg, and pulmonary capillary wedge mean 6 mmHg. The pulmonary angiogram showed obstruction to flow in the right upper lobe and left upper lobe arteries and diminished flow in the left lower lobe arteries and a negative shadow straddling the bifurcation of the pulmonary artery (Figure). The estimated pulmonary vascular bed obstruction was 60–70%. Because of the high pressures in the right ventricle, and the presence of low output for about 26 hours, embolectomy and inferior vena cava plication was advised.

Through a median sternotomy incision the pericardium was opened and about 300 ml of cloudy, purulent fluid was found within. A fibrinous pericarditis was present. Cultures of the fluid were negative for organisms.

Under total cardiopulmonary bypass the pulmonary artery was opened and multiple clots were removed from both sides using forceps, a Fogarty

catheter, flushing with saline, strong suctioning and finally opening both pleural cavities and squeezing the lungs. All lobar arteries were felt to be patent, the pulmonary artery was closed and the patient was put on partial bypass. It was noted that there was very little ventilation of the lungs and when the endotracheal tube was suctioned, large amounts of bloody, frothy fluid were aspirated continuously. Approximately 1200 ml was suctioned in a few minutes. Blood gases at this time showed P_{CO_2} of 60 mmHg, pH 7.19, P_{O_2} 160 mmHg, and O_2 saturation of 98%. The lungs were extremely difficult to ventilate and this had to be achieved by hand. Frusemide (Lasix), 40 mg, was added to the pump and suctioning was continued. There seemed to be a slight decrease of bloody fluid in response to the Lasix. The patient was taken off bypass and the heparin was neutralized rapidly with protamine, 4 mg/kg. An epinephrine drip was started to maintain an adequate blood pressure. Another 40 mg of frusemide was given and digoxin, 0.25 mg, intravenously. The haemorrhagic bronchorrhoea gradually subsided. Methylprednisolone sodium succinate (Solumedrol), 100 mg, was then given.

The volume of bloody fluid suctioned from the trachea gradually subsided and the lungs became easier to ventilate. A total of 1500 ml was aspirated in 30 minutes. The lungs appeared pink

FIGURE Pulmonary angiogram showing obstruction of right upper lobe and left main pulmonary arteries with clot straddling the pulmonary artery bifurcation.

whereas before embolectomy they had been white. No areas of haemorrhage were noted.

Subsequent blood gas estimation showed improvement within one hour. The inferior vena cava was plicated using a right flank retroperitoneal approach and a DeWeese clip.

In the recovery room the patient required an epinephrine drip for 24 hours. He was kept on the Bennett respirator MAI overnight with the FIO_2 gradually reduced to 0.4. The next morning he was taken off the respirator and extubated in the afternoon. He continued to cough up bloody sputum for 10 days after operation.

His chest films showed atelectasis or pleural effusion in the area of the lower left lobe. He was on heparin postoperatively and then was switched to warfarin sodium (Coumadin).

The patient was discharged three weeks after operation with an improved chest radiograph and still taking warfarin sodium.

DISCUSSION

It is known that pulmonary infarction after embolism can produce an haemorrhagic effusion and haemoptysis. The pathology shows destruction of alveolar walls and haemorrhage into the interstitium and alveoli. The haemoptysis is usually not significant and is not known to produce pulmonary insufficiency or death.

The occurrence of pulmonary oedema with pulmonary embolism, however, is more controversial. The classical teaching is that pulmonary embolism does not cause pulmonary oedema. However, there is some experimental and clinical evidence to the contrary (Dexter, 1965; Daley, 1973; Windebank and Moran, 1973).

While no mechanism has been proven, pulmonary oedema can occur in man secondary to pulmonary embolism. Haemorrhagic pulmonary oedema has been reported 12 times (Table I). An analysis was made of the cases for common aetiological factors (Table II). No generalized haemorrhagic diathesis was noted in any of the patients. Several patients were on steroids, including our own. This association, suggested by Makey *et al.* (1971), was not consistent as nine had no known steroid abnormality.

A raised left atrial pressure did not seem to be a factor as many patients were still on partial cardiopulmonary bypass when the complication occurred.

Pulmonary artery hypertension and a 'two-compartment' pulmonary vascular bed was suggested by Castleman and Scannell (1964) and

TABLE I
PREVIOUS REPORTS OF HAEMORRHAGIC
PULMONARY OEDEMA

Author	No. of Cases	No. of Survivors
Couves <i>et al.</i> (1973)	5	1
Castleman <i>et al.</i> (1964)	1	0
Makey <i>et al.</i> (1971)	2	0
Bonnabeau <i>et al.</i> (1972)	1	1
Moser and Braunwald (1973)	1	1
Stirling <i>et al.</i> (1968)	2	0
Total	12	3

elaborated by Moser and Braunwald (1973). Both of their cases had pulmonary artery pressures in excess of 100 mmHg.

In the report of Stirling *et al.* (1968), one patient had a pulmonary artery systolic pressure of 95 mmHg. The other was described as having pulmonary hypertension; no figures were given. Our case had a systolic pulmonary artery pressure of 50 mmHg. All other eight cases had no recorded pressures.

Chronicity in itself was not universal. Eight cases appeared after relief of massive acute pulmonary emboli. We reviewed all the published technically successful chronic embolectomies in the English literature. We found 11; four of these developed haemorrhagic pulmonary oedema, a 36% incidence (Table III). Five of the chronic embolectomies were done without cardiopulmonary bypass. Six were done with bypass and four developed the syndrome, a 66% incidence. None of the patients treated without cardiopulmonary bypass developed the syndrome. Therefore, chronicity and the use of cardiopulmonary bypass for chronic thromboendarterectomy may be important precipitating factors.

Of the 12 patients who are reported with this syndrome in the literature, only one was treated without cardiopulmonary bypass. However, it is obvious that cardiopulmonary bypass of itself cannot be incriminated. There must be other operative factors. It is felt that the association with cardiopulmonary bypass is relative to the fact that the patients are heparinized at the time of re-establishment of the pulmonary circulation. Our patient was a young man and had no known pre-existing cardiac or pulmonary disease. Only one had this previous history. All of the acute cases were hypotensive and anoxic; this was not true of the chronic cases.

The aetiology at present is obscure. Most experimental and clinical studies show that if the pulmonary artery flow is blocked, the integrity of

TABLE II
SALIENT FEATURES OF ALL PATIENTS HAVING HPE AFTER PULMONARY EMBOLECTOMY

Author	Patient (Age/Sex)	HPE		CP	PA Pressure	Previous Cardiopulm. disease	Significant Associated Conditions	Significant Medication	Outcome
		Acute	Chronic						
Couves <i>et al.</i> (1973)	35 F	4 hr	An episode 1 wk before admission An episode 2 wk before admission	No		No			Died
	40 F	1 hr		Yes		No			Died
	44 M	40 hr		Yes		No			Died
	25 F	4 hr		Yes		No			Lived
Makey <i>et al.</i> (1971)	72 M	12 hr	6 wk	Yes	100 30 M = 55 mmHg	Chronic airways obstruction No	Pregnancy Chest Trauma	Steroids Steroids	Lived
	55 F	34 hr		Yes		No	Adrenal haemorrhage Myelofibrosis		Died
Castleman <i>et al.</i> (1964)	53 M	4 hr	6 wk	Yes	113 35 M = 56 mmHg 95 mmHg systolic	No	Obesity		Died
	39 M	4 hr		Yes		No			Died
Bonnabeau <i>et al.</i> (1972)	30 F	24 hr	An episode 48 hr previously	Yes	10 yr of progressive dyspnoea	No	Birth control pills		Lived
Moser and Braunwald (1973)	69 M		10 yr	Yes	1 yr of progressive dyspnoea	10 yr of progressive dyspnoea			Lived
Stirling <i>et al.</i> (1968)	34 F		6 mth-1 yr	Yes	Pulmonary hypertension 50 systolic M = 35 mmHg	No	Chest and abdominal trauma	Steroids	Lived
			6 mth	Yes					Died
Present study	37 M	26 hr		Yes					Lived

CP = cardiopulmonary bypass HPE = haemorrhagic pulmonary oedema PA = pulmonary artery M = mean

TABLE III
PREVIOUS TECHNICALLY SUCCESSFUL CHRONIC
PULMONARY EMBOLECTOMIES

Author	No. of Cases	HPE	CP
Snyder <i>et al.</i> (1963)	1	No	No
Moser <i>et al.</i> (1965)	2	No	No
Nash <i>et al.</i> (1968)	1	No	Yes
Moor and Sabiston (1970)	1	No	Yes
Frater <i>et al.</i> (1965)	1	No	No
Makey <i>et al.</i> (1971)	1	No	No
Moser and Braunwald (1973)	1	Yes	Yes
Castleman <i>et al.</i> (1964)	1	Yes	Yes
Stirling <i>et al.</i> (1968)	2	Yes	Yes

HPE = haemorrhagic pulmonary oedema;
CP = cardiopulmonary bypass

the lung parenchyma is maintained by precapillary collaterals from the bronchial arteries. Vidone and Liebow (1957) showed that it is only when these are ligated or the pulmonary venous outflow is obstructed or compromised that pulmonary infarction develops. Less dramatic pathology with simple pulmonary embolism has been observed but usually neglected. Roach and Laufman (1955), Karsner and Ash (1912-13), and Chapman, Guggle, and Wheeler (1949) recorded interstitial and alveolar oedema and red blood cells in the alveoli

subsequent to pulmonary embolism. Hampton and Castleman (1940), in a detailed necropsy study noted 'incomplete infarction' among other changes. Their concept of incomplete infarction was oedema and haemorrhage without the loss of alveolar septa. This was therefore a reversible condition. This finding has not been popularized nor accepted in the medical literature. However, it does seem to fit clinical circumstances of patients suffering from this syndrome. Patients without clinical evidence of infarction, such as pleuritic chest pain, haemoptysis or appropriate radiographic changes, may develop this problem after their pulmonary circulation has been restored.

In reviewing the pathology of cases with necropsy reports, it is noted that haemorrhagic congestion is the predominant finding.

Patients with massive embolism because of either the quantity of embolus, poor collateral circulation, hypoxia, or hypotension may have 'incomplete infarction' which, when pulmonary artery flow is restored, allows proteinaceous fluid and red blood cells into the interstitium and alveoli through a damaged capillary endothelium. It is reasonable to assume that this problem would

be worsened by chronicity, pulmonary artery hypertension, and heparinization. Some of these cases may have been early true infarcts in which the full pathological picture had not had time to develop.

With the clinical circumstances of severe hypoxia, hypotension, chronicity, and pulmonary artery hypertension, one might be alerted to the development of this syndrome.

In analysing the data of the survivors, certain factors could be of importance in the treatment which led to survival. Moser and Braunwald (1973) emphasized prolonged respiratory support with PEEP if necessary, and postural changes that kept the good lung dependent, maximizing flow to this area and minimizing shunting through the damaged lung. Our patient was treated almost identically with the case of Couves *et al.* (1973). The anaesthetist was asked to suction the patient as frequently as possible. Frusemide was given to reduce the volume of fluid in the lungs. Digoxin was given and an epinephrine drip was started. It was felt that this cardiotonic regimen would lower the left atrial pressure if any element of failure was present. Steroids were given largely in desperation, but they are reported to stabilize cellular membranes.

As a final alternative, resection of a lobe or operative occlusion of a bronchus may be attempted if gross evidence of haemorrhagic infarction can be localized.

We are grateful to Dr. M. Marter who cared for this patient medically.

REFERENCES

- Bonnabeau, R. C. Jr., Mossburg, W. L., Armanious, A. W., Cuono, C. B., and Lancaster, J. R. (1972). Pulmonary embolectomy utilizing cardiopulmonary bypass. *West Virginia Medical Journal*, **68**, 331.
- Chapman, D. W., Gule, L. J., and Wheeler, P. W. (1949). Experimental pulmonary infarction—abnormal pulmonary circulation as a prerequisite for pulmonary infarction following an embolus. *Archives of Internal Medicine*, **83**, 158.
- Couves, C. M., Nakai, S. S., Sterns, L. P., Callaghan, J. C., and Sproule, B. J. (1973). Hemorrhagic lung syndrome. Hemorrhagic lung infarction following pulmonary embolectomy. *Annals of Thoracic Surgery*, **15**, 187.
- Daley, R. (1973). Pulmonary oedema in pulmonary thromboembolism. (Letter.) *British Medical Journal*, **2**, 55.
- Dexter, L. (1965). Thromboemboli as a cause of cor pulmonale. *Bulletin of the New York Academy of Medicine*, **41**, 981.
- Frater, R. W. M., Beck, W., and Schrire, V. (1965). The syndrome of pulmonary artery aneurysms, pulmonary artery thrombi, and peripheral venous thrombi. *Journal of Thoracic and Cardiovascular Surgery*, **49**, 330.
- Hampton, A. O. and Castleman, B. (1940). Correlation of postmortem chest teleroentgenograms with autopsy findings: with special reference to pulmonary embolism and infarction. *American Journal of Roentgenology*, **43**, 305.
- Karsner, H. T. and Ash, J. E. (1912–13). Studies in infarction: II. Experimental bland infarction of the lung. *Journal of Medical Research*, **27**, 205.
- Makey, A. R., Bliss, B. P., Ikram, H., Sutcliffe, M. M. L., and Emery, E. R. J. (1971). Fatal intra-alveolar pulmonary bleeding complicating pulmonary embolectomy. *Thorax*, **26**, 466.
- Moor, G. F. and Sabiston, D. C. Jr. (1970). Embolectomy for chronic pulmonary embolism and hypertension. Case report and review of the problem. *Circulation*, **41**, 701.
- Moser, K. M. and Braunwald, N. S. (1973). Successful surgical intervention in severe chronic thromboembolic pulmonary hypertension. *Chest*, **64**, 29.
- Moser, K. M., Houk, V. N., Jones, R. C., and Hufnagel, C. C. (1965). Chronic, massive thrombotic obstruction of the pulmonary arteries. Analysis of four operated cases. *Circulation*, **32**, 377.
- Nash, E. S., Shapiro, S., Landau, A., and Barnard, C. N. (1968). Successful thrombo-embolectomy in long-standing thromboembolic pulmonary hypertension. *Thorax*, **23**, 121.
- Roach, H. D. and Laufman, H. (1955a). Relationship between pulmonary embolism and pulmonary infarction: an experimental study. *Annals of Surgery*, **142**, 82.
- Snyder, W. A., Kent, D. C., and Baisch, B. F. (1963). Successful endarterectomy of chronically occluded pulmonary artery. Clinical report and physiologic studies. *Journal of Thoracic and Cardiovascular Surgery*, **45**, 482.
- Stirling, G. R., Morris, K. N., McLean, K. H., Mirams, J. A., Rubinstein, D., and Wagner, G. R. (1968). Surgical aspects of pulmonary embolism. *Medical Journal of Australia*, **1**, 571.
- Vidone, R. A. and Liebow, A. A. (1957). Anatomical and functional studies of the lung deprived of pulmonary arteries and veins, with an application in the therapy of transposition of the great vessels. *American Journal of Pathology*, **33**, 539.
- Windebank, W. J. and Moran, F. (1973). Pulmonary oedema in pulmonary thromboembolism. (Letter.) *British Medical Journal*, **1**, 485.

Requests for reprints to: Dr. J. W. Garvey, The Cardiothoracic Service, Division of Surgery, Long Island Jewish Hospital, New Hyde Park, New York.