PULMONARY INFARCTION IN HEALTHY YOUNG MALES

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It is widely recognised that the elderly immobilised, sick, traumatised or postoperative patient is the most likely candidate to develop pulmonary embolism and infarction, and that the risk increases in direct proportion to the duration of the illness and to the age of the patient. Over the past few years, a number of autopsy studies have stressed the high incidence of deep venous thrombosis and pulmonary embolism in individuals who are otherwise normal. Unfortunately, many clinicians have been slow to appreciate this point and fail to recognise the condition unless well-established predisposing factors are present.

This paper reports the findings in four young male patients who developed pulmonary infarction in the absence of obvious precipitating causes.

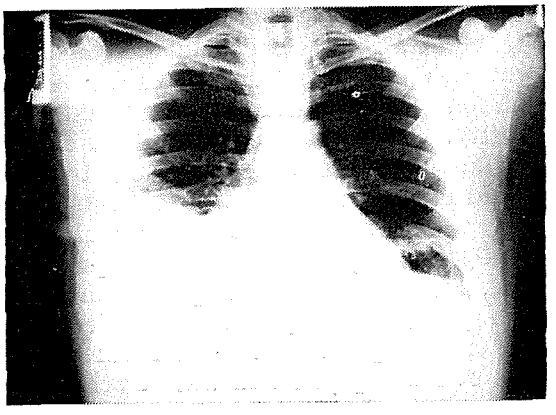
CASE 1

A.A., then a 28-year old Customs clerk, was initially referred to St. Luke's Hospital in December 1967. He gave a four-week history of pain in the left calf and ankle, which was relieved on walking. Four days prior to admission, he complained of left sided chest pain on breathing and about the same time he coughed up a small amount of bright red blood. Clinical examination showed a febrile patient (temp. 38.8°C), pulse 100/minute; J.V.P. not elevated; B.P. 130/100 mm Hg. The cardiovascular system was normal. There were clinical signs of consolidation in the lower zone of the left lung. Clinical examination of both lower limbs was entirely negative. A chest x-ray confirmed the clinical findings, besides consolidation it also showed elevation of the left hemi-diaphragm. The patient was anticoagulated, initially with heparin and then with phenindione. He showed some improvement on this regime; his temperature subsided as did his chest pain. He had no further attacks of haemoptysis and was discharged on a maintenance dose of phenindione. In September 1972 he was again referred to hospital with a 4-day history of pyrexia (38.3°C), right sided chest pain, which was made worse on deep breathing, cough and haemoptysis. Clinical examination showed diminished air entry in the right lung base. Chest x-ray showed a small right sided pleural effusion. The patient was given heparin and warfarin and his symptoms subsided within 5 days. He was discharged after three weeks in hospital on a maintenance dose of warfarin. The patient is being reviewed as an out-patient. and to date he has has remained well.

CASE 2

J.B., a 30-year old Customs Officer, was referred to the Department of Medicine in April 1975 with a two-week history of pyrexia. He subsequently coughed up some bright red blood. On the day prior to his admission to hospital he developed

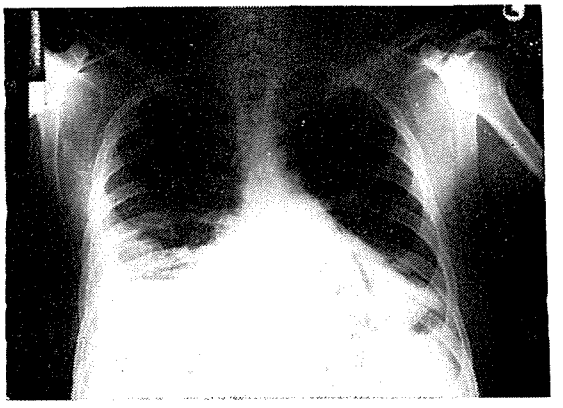
sharp pleuritic pain in the left side of his chest. He had been treated at home with ampicillin, cloxacillin and chloramphenicol, but there had been no improvement in his condition. On examination the patient was in considerable pain. His pulse was 100/min and his B.P. was 120/70 mm Hg. His temperature was 38.3°C. The cardiovascular system was normal. Examination of the lungs revealed diffuse coarse crepitations bilaterally. Four days after admission he developed pleuritic pain on his right side. A chest x-ray taken at this stage showed linear collapse in the left base, a small right sided pleural effusion as well as collapse consolidation in the right base (Fig.1). A diagnosis of pul-



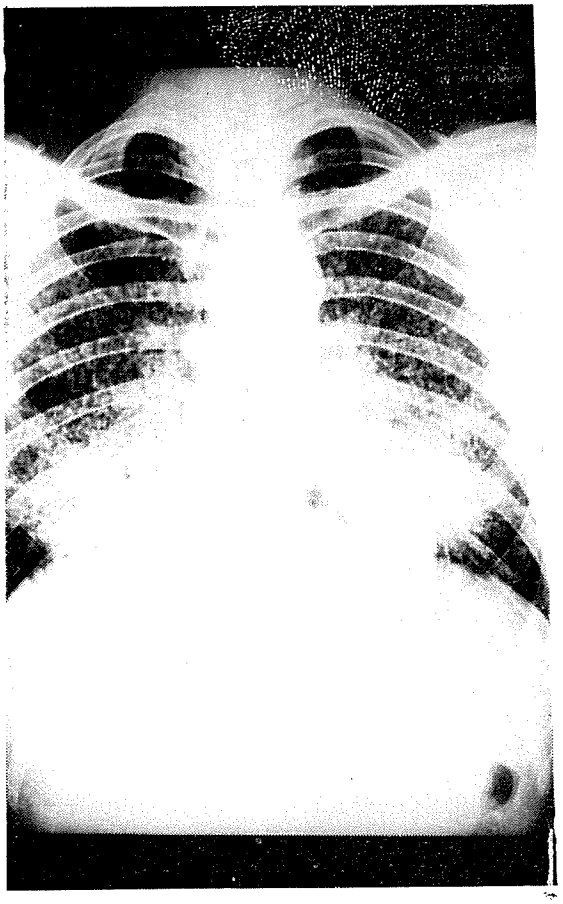
monary infarction was made and he was anticoagulated with heparin and warfarin. The temperature subsided after three days and the chest pain improved markedly. He continued to bring up blood tinged sputum for 10 days, after which all haemoptysis ceased. He was discharged on warfarin 5mg daily. A chest x-ray taken in June 1975, showed marked clearing of the effusion; there was still some collapse of the right base and elevation of the left hemidiaphragm (Fig.2). Clinically the patient has since remained well.

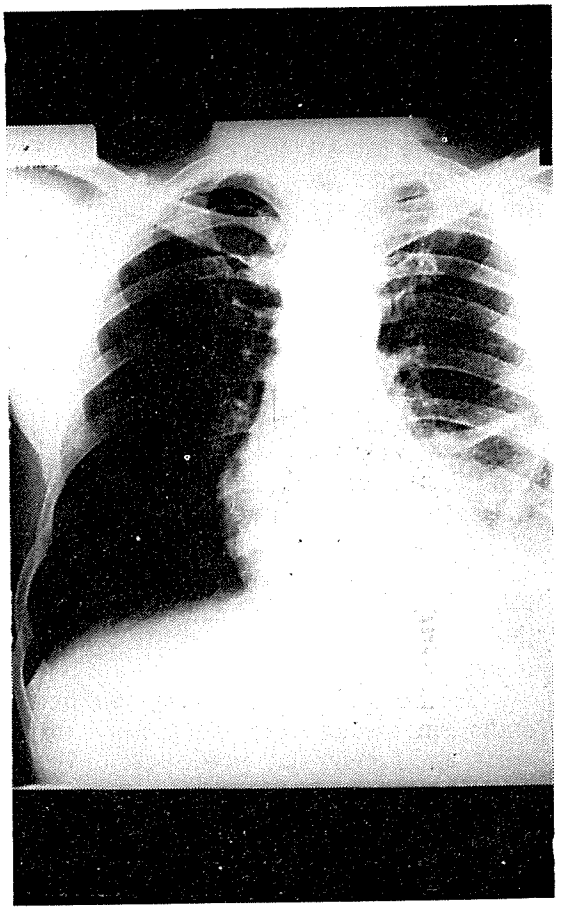
CASE 3

G.C., a 29-year old German travel agent, was referred to hospital in June 1975 with a three-day history of cough, dyspnoea and profuse haemoptysis. He had been previously healthy and enjoyed his



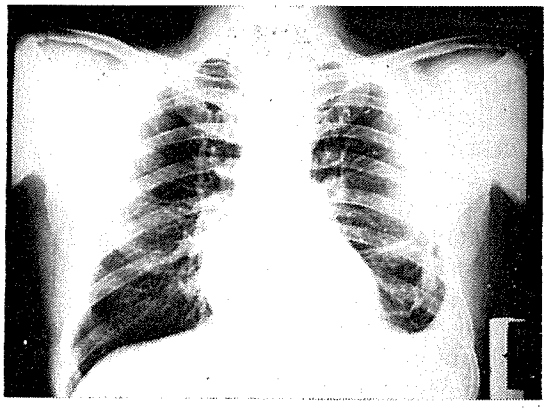
job, which involved quite long air and car journeys abroad, Clinical examination showed a pale, apprehensive patient with severe dyspnoea. He had marked finger clubbing — a feature which had been present ever since the patient could remember. He was running a temperature of 37.7°C. The clinical examination was otherwise normal. There was never any clinical evidence of deep vein thrombosis. A chest x-ray taken soon after admission was normal. He was started on ampicillin but his cough and haemoptysis became more severe and his temperature did not subside. Sputum culture showed a mixed flora sensitive to choramphenicol; no acid fast bacilli were present. In view of this, chloramphenicol was added to ampicillin, but no effect was noted. A chest x-ray, repeated 6 days after admission, showed diffuse coarse mottling of both lung fields (Fig.3). This was interpreted as being due to pulmonary haemosiderosis secondary to the profuse haemoptysis. At this stage, in view of the patient's lack of response to antibiotics, a provisional diagnosis of pulmonary infarction was made, and the patient was anticoagulated with heparin and warfarin. Within four days, he started feeling better: his dyspnoea diminished, the haemoptysis became much less marked and the temperature returned to normal. He maintained this improvement on anticoagulant therapy alone and was discharged on a maintenance dose of warfarin. A chest x-ray repeated three weeks later showed complete clearing of both lung fields.





CASE 4

J.G., a 35 year old male was admitted to our unit in July 1975 with a two-week history of cough, pleuritic pain in his left lower chest and dyspnoea on moderate exertion. He had no haemoptysis. He had been treated with antibiotics at home but failed to obtain any symptomatic relief. He had previously always been in good health, and had found no difficulty in coping with his job as a shipwright at the Drydocks, an occupation which he described as fairly strenuous. He volunteered the information that his job required him to stay in cramped places for hours on end. On examination the patient had a temperature of 37.6°C and there was diminished air entry over the lower zone of the left lung base. A chest x-ray showed a small left pleural effusion with some collapse/consolidation of the left lower lobe (Fig. 4). A provisional diagnosis of pulmonary infraction was made and the patient was anticoagulated. His condition improved and within two weeks he was discharged on warfarin. A chest x-ray, repeated just before he was discharged showed only pleural thickening in his left base, with linear atelectasis in the left lower lung zone (Fig. 5).



In all four patients, sputum revealed only a mixed bacterial flora; no acid fast bacilli were seen on direct microscopy or subsequently isolated on culture. The numerous other biochemical and electrocardiographic investigations carried out showed no abnomalities and did not contribute in any significant way to the diagnosis.

Discussion

Pulmonary embolism and infarction is a common disease; figures for the incidence of pulmonary embolism that are based on clinical findings almost certainly underestimate the true frequency of the condition (Miller 1973). Thus, Sevitt and Gallagher (1961) found auptopsy evidence of pulmonary embolism in 20% of patients admitted to the Birmingham Accident Hospital; in none of these had a diagnosis of pulmonary embolism been previously made.

In the majority of cases, pulmonary embolism results from deep vein thrombosis, and factors that predispose to venous thrombosis are therefore the same that predispose to pulmonary embolism. The diagnosis of deep vein thrombosis has traditionally depended on the presence of abnormal physical signs. However it has long been known that this condition can be clinically silent. Recent techniques using labelled fibrinogen and ultrasonic flow detectors have confirmed this view (Browse 1974). In fact it is not uncommon that the first clinical evidence of deep vein thrombosis may be the development of pulmonary embolism. (Homans 1943; Coon and Coller 1959).

There have been frequent reports of pulmonary embolism occurring in otherwise healthy individuals. In some it has followed long air journeys or after staying in a cramped position for several hours (as in two of our patients). In others no obvious predisposing factors were present. Homans 1943 described 11 cases of thrombus developing silently in deep veins of healthy people whose first symptoms were those of pulmonary infarction. Hampton (1945) described 10 cases of pulmonary embolism in people who were at work when they developed the embolus. None of these patients had been referred to hospital with the correct diagnosis. Short (1952) described three cases of pulmonary embolism in healthy persons who had been referred to hospital with a diagnosis of pneumonia. Cohen and Daly (1957) described 10 patients, whose ages ranged from 21 to 76 years, with recurrent pleurisy and haemoptysis and who responded to anticoagulant therapy. Petch (1958) described 11 such cases; their ages varied from 36 to 75 years. Kilburn and Sieker (1958) encountered 25 men under 40 years with recurrent pulmonary embolism in the absence of any other disease. Thomson and Hamilton (1962) described 6 patients, their ages varying between 35 and 80 years who presented with an illness resembling pneumonia but who responded to anticoagulants and not to antibiotics. All of these patients were ambulant until the onset of the respiratory illness rendered them unable to go to work, and none of them ever showed signs of venous thrombosis. Loehry (1966) reviewed the notes of all in-patients under the age of 40 at St. Thomas' Hospital who had been diagnosed as having pulmonary embolism and infarction. Out of 94 cases. 62 were associated with factors known to predispose to the condition — i.e. deep vein thrombosis, congestive cardiac failure, trauma, recent operation and pregnancy. The remaining 32 showed no evident preciptating factor; also none of the female patients in this group were taking oral contraceptives, which are now thought to increase the risk of thromboembolic disease.

Necropsy studies have also shown that pulmonary embolism does occur in otherwise healthy individuals. In 1962. Breckenridge and Ratnoff described 26 cases of pulmonary embolism — diagnosed at post mortem — in patients aged between 15 and 45 years. In none of these had a clinical diagnosis of the condition been made. In 1966, Fleming and Bailey reviewed autopsy reports at the University Department of Pathology, Cambridge, between 1945 and 1963. Patients over 70 years were excluded. In all 27 patients

were found to have had pulmonary embolism; their ages ranged between 23 and 69 years. Only 6 of these had clinical evidence of venous thrombosis before admission. A further two developed manifestations of deep vein thrombosis after admission to hospital. The remaining 19 patients showed no such evidence clinically.

In spite of improved methods of diagnosis available in several centres (e.g. radioisotope studies, pulmonary angiography) an early diagnosis of pulmonary embolism is only possible when the clinician has a high index of suspicion and a ready awareness of the disorder. Management of the condition depends on an early diagnosis, so that treatmet with anticoagulants can be started to prevent a fatal outcome or the development of thromboembolic pulmonary hypertension. The manifestations of pulmonary embolism may suggest pneumonia, but if symptoms persist, or if they recur, or if they change sides, or if the expected response to antibiotics fails to occur, an embolic episode must be considered — and should not be discarded, as so often it is, because of the absence of clinically obvious venous thrombosis.

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