UNUSUAL PRESENTATION OF PULMONARY EMBOLUS

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CASE REPORT

We report an atypical presentation of multiple pulmonary emboli in an otherwise fit 39-year-old lady. The patient presented to a gynaecologist with signs of peritonitis, having a gynaecological history of microprolactinoma in 1998 which had been managed by prolactin screening.

She was admitted to hospital with sudden onset severe left upper quadrant abdominal pain, with associated nausea and vomiting. Clinically, she had a rigid abdomen but no evidence of an ectopic pregnancy. Fluid resuscitation, intravenous antibiotics and opiates settled the patient overnight. Anti-embolism stockings were applied and 20mg subcutaneous enoxaparin given on admission as per ward protocol. In total, she had five litres crystalloid over 24 hours. An abdominal ultrasound scan (figure 1) carried out 12 hours post admission revealed a thick walled cystic lesion posterior to the uterus (8x6x8cm); this was most likely to be a ruptured ovarian cyst. The patient suddenly deteriorated 24 hours post admission and was admitted to the intensive care unit (ICU).

Figure 1 Ultrasound scan showing a large ovarian cyst with hyperechogenic contents

On examination she was pale, pyrexia 38.2°C, tachycardic 125 bpm, BP 110/80, tachypnoeic with a respiratory rate of 25 and unable to complete full sentences (she was, however, warm and well perfused). There was no clinical evidence of a deep venous thrombosis (DVT). Air entry in the chest was poor in the midzones and bases but clear in the apices. Arterial blood gas (ABG) on air revealed a type 1 respiratory failure – hypoxia without concomitant hypercarbia. (See table 1)

	pH	P CO ₂ (kPa)	P O ₂ (kPa)
At presentation	7.37	5.3	7.5
24 hours post op	7.38	5.19	7.9

Table 1 Blood gases showing type 1 respiratory failure

A chest X-ray (figure 2) showed collapsed lower lobes and a bilaterally raised diaphragm. Her abdomen was rigid and tender with scanty bowel sounds. Abdominal X-ray showed faecal loading in the ascending colon. In view of her obvious shock she was resuscitated with a further 1000mls colloid, high flow oxygen and taken to theatre. A ruptured right-sided endometrial 'chocolate' cyst was found at laparotomy. The cyst was shelled out of the pelvis and a right salpingo-oophorectomy performed, with minimal blood loss. She required 100% inspired oxygen during the operation to maintain saturations of 96% or more. In theatre, a central venous pressure of 14cmH₂O was maintained and mean arterial pressure remained approximately 80 and heart rate of 90 beats per minute.

Figure 2 Portable chest X-ray just prior to laparotomy indicating bilaterally elevated hemidiaphragm and gas in the splenic flexure of the large colon (more likely to indicate significant abdominal pathology than a PE)

After surgery she was transferred to the ICU and sedated. Her oxygen requirements quickly reduced to 35%. Nine hours later, her cardiorespiratory parameters had normalised and her endotracheal tube was removed. She returned to the ward with regular intravenous paracetamol and a patient-controlled analgesia device provided intravenous morphine on demand.

She remained comfortable 24 hours after ICU discharge, but continued to complain of breathlessness with a slightly raised heart rate. Because she had bilateral bronchial breathing and reduced air entry further investigations were instituted. There was no evidence of right heart strain and T wave inversion in lead III was the only ECG abnormality.

Repeat chest X-rays showed marked basal atelectasis and bilateral lower lobe collapse. Despite low concentration oxygen therapy she continued to display features of type 1 respiratory failure.

In view of her persisting type 1 respiratory failure a computed tomography (CT) pulmonary angiogram was urgently arranged. This revealed multiple pulmonary emboli, bilateral basal consolidation and bilateral pleural effusions. She was anticoagulated with heparin and made a good recovery. The patient's routine thrombophilia screen was negative and there was no positive family history of thromboembolism.

DISCUSSION

In the United States of America (USA), pulmonary embolism (PE) is present in 70% of patients with DVT, even though more than half the patients are asymptomatic. PE is the third most common cause of death in hospitalised patients, with at least 650,000 cases occurring annually. Autopsy studies have shown that approximately 60% of patients who died in the hospital had PE, and the diagnosis was missed in up to 70% of the cases.

Most patients with PE have no obvious symptoms at presentation. In contrast, patients with symptomatic DVT commonly have PE confirmed on diagnostic studies in the absence of pulmonary symptoms.

This lady's surgery was delayed because her signs on examination and investigation and her initial progress suggested that it was safe to leave her overnight. Indeed, at surgery, the surgeons were surprised that a ruptured cyst alone with minimal pelvic bleeding could have caused such a rapid clinical deterioration in her status necessitating emergency laparotomy. However, a combination of systemic inflammatory response syndrome (SIRS) due to pelvic pathology with acute lung injury and diaphragmatic splinting was our initial explanation for her deterioration. In retrospect, we should have considered the differential diagnosis of type 1 respiratory failure in more detail.

It is most likely that her PE occurred prior to theatre in view of her sudden hypoxia and signs of shock. Although tachycardia, hypoxia and reduced air entry in the chest were non-specific pointers to a thromboembolic cause, a proven pelvic abnormality on ultrasound scan alone could easily present in this fashion.

The risk of DVT after hospitalisation

- 11% of all medical patients placed on bed rest for one week
- 31% of all patients in medical intensive care units
- 23% of patients with pulmonary diseases who are given bed rest for three or more days
- 30% of those admitted to a critical care unit after a myocardial infarction
- 48% of patients who are asymptomatic after a coronary artery bypass graft

Respiratory failure

There are many alternative and historical definitions:

1. Respiratory failure occurs when pulmonary gas exchange is sufficiently impaired to cause hypoxaemia with or without hypercarbia, when the PaO_2 is less than 8kPa (60mmHg).
2. Respiratory failure is the inability of the lungs to perform their basic task of gas exchange, the transfer of oxygen from inhaled air into the blood and the transfer of carbon dioxide from the blood into exhaled air. The basis of respiratory failure may be failure of the exchange of oxygen and carbon dioxide within the alveoli; failure of the muscles required to expand the lungs; or failure of the central control of respiration.
3. In the 1925 edition of Osler's text⁽¹⁾, respiratory failure was described as 'frothy pulmonary edema that resembles serum, not the sanguinous transudative edema fluid seen in dropsy or congestive heart failure'.

Respiratory failure is sub-classified into one of two types of respiratory failure based on the level of PaCO_2 :

- type 1 respiratory failure: PaCO_2 is less than 6.5kPa and PaO_2 less than 8kPa
- type 2 respiratory failure: PaCO_2 is greater than 6.5kPa and PaO_2 is less than 8kPa

Common causes of type 1 (hypoxemic) respiratory failure are:

Chronic bronchitis and emphysema (COPD); pneumonia; pulmonary oedema; pulmonary fibrosis; asthma; pneumothorax; **pulmonary embolism**; pulmonary arterial hypertension; pneumoconiosis; granulomatous lung diseases; cyanotic congenital heart disease; bronchiectasis; adult respiratory distress

Clinical presentation

The presentation of PE may vary from a sudden onset of catastrophic haemodynamic collapse to gradually progressive dyspnoea. The diagnosis of PE should be sought in patients with respiratory symptoms unexplained by an alternate diagnosis. The symptoms of PE are often non-specific; consequently a high index of suspicion is required, particularly when a patient has risk factors, which include recent surgery, immobility or a hypercoagulable state.

Pathophysiology

The pathophysiology of patients with PE can be categorised into four classes, based on the speed and severity of pulmonary arterial occlusion:

- massive pulmonary embolism
- acute pulmonary infarction
- acute embolism without infarction
- multiple pulmonary emboli

Massive pulmonary embolism

Large emboli compromise sufficient pulmonary circulation to produce circulatory collapse and shock. The patient is hypotensive, pale, sweaty, oliguric and with depressed mental state.

Acute pulmonary infarction

Approximately 10% of patients have peripheral occlusion of a pulmonary artery causing lung tissue infarction. These patients often present with acute onset of pleuritic chest pain, breathlessness and haemoptysis. Although the chest pain may be indistinguishable from ischemic myocardial pain, right heart strain findings on ECG and no response to glyceryl trinitrate (GTN) make this diagnosis unlikely.

Acute embolism without infarction

Patients have non-specific symptoms of unexplained dyspnoea and/or sternal discomfort.

Multiple pulmonary emboli

This class consists of two groups of patients. Both groups present with pulmonary hypertension and, ultimately, *cor pulmonale*.

The first group has repeated documented episodes of pulmonary emboli over years, eventually presenting with signs and symptoms of pulmonary hypertension and *cor pulmonale*.

The second group has no previously documented pulmonary emboli but has widespread obstruction of the pulmonary circulation with clot. Patients present with gradually progressive dyspnoea, intermittent exertional chest pain and, eventually, features of pulmonary hypertension and *cor pulmonale*.

The signs and symptoms of pulmonary hypertension and the development of *cor pulmonale* are subtle in the early

stages of the disease and may not be apparent for months or even years. As the disease progresses, signs and symptoms become more noticeable. They include:

- dyspnoea on exercise initially and latterly at rest
- fatigue
- dizziness and syncope
- abnormal heart sounds
- chest pain
- ankle oedema, abdominal ascites and hepatomegally
- engorged neck veins
- peripheral and central cyanosis
- tachycardias and palpitations

Outcome

Eighty per cent of unexpected deaths in hospital are associated with PE. Ten per cent of patients who suffer PE die within the first hour, a further 30% die of recurrent emboli. Anticoagulant treatment can halve the mortality rate. It is said that 100,000 of the estimated 400,000 deaths a year in the USA could be prevented by proper diagnosis and treatment. The risk of PE is increased in pregnancy and during the postpartum period.

CONCLUSION

Pulmonary embolus detection is notoriously difficult to establish clinically. CT pulmonary angiography has practically become the first line modality for imaging of pulmonary circulation in patients suspected of having PE. We should maintain a high index of suspicion for pulmonary embolus in an unresolved hypoxia patient with signs of cardiovascular compromise, especially in the young and physically fit group.

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FURTHER READING

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