Pleuritic chest pain and hypoxia – a diagnostic dilemma

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Introduction

Pulmonary thromboembolism (PE) is notoriously difficult to diagnose since it commonly presents in a non-specific manner. Only 15–30% of the patients identified at post-mortem as having a massive PE have been diagnosed correctly prior to death.[1,2] However, large studies have shown that certain clinical symptoms and features such as dyspnoea, tachypnoea, pleuritic chest pain with a normal chest radiograph and a low Pa_{O2} are present in more than 90% of patients with PE.[2] Clinicians in a district hospital setting have to rely on these features, especially when facilities for detailed imaging such as computerised tomography (CT) or pulmonary angiography are not available.

Occasionally, certain other diseases can mimic the clinical picture of PE and lead to delay in instituting appropriate treatment. We present two patients with symptoms and clinical investigations which were highly suggestive of acute PE but who turned out to have very different diagnoses in the end.

Case report

Case 1

A previously fit 39-year-old woman was admitted to our hospital in mid-October 1998 with a three day history of right pleuritic chest pain, dry cough and dyspnoea. There were no major risk factors for PE. On admission, she was pyrexial (temperature 39.1°C), tachypnoeic and hypoxic (92% oxygen saturation on room air). Physical examination revealed a heart rate of 110 bpm, a blood pressure 109/65 mmHg, no features of right ventricular strain but reduced air entry on auscultation of her right lung base.

Routine blood investigations were normal apart from a leucocytosis (18.5x10/L) and thrombocytopenia (83x10/L). The arterial blood gas confirmed hypoxia with respiratory alkalosis (pH 7.49, P_{O2} 7.3 kPa, P_{CO2} 3.7 kPa). The electrocardiogram (ECG) on admission was unremarkable, as was her presenting chest X-ray. The differential diagnosis at this stage was either acute pneumonia or PE. She was commenced on antibiotics and low molecular weight heparin. A ventilation/perfusion (VQ) scan was requested.

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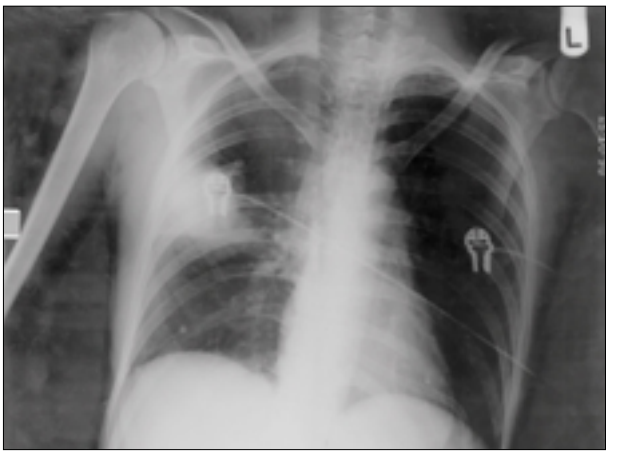
Figure 1. Chest X-ray of case 1 one day after admission, showing segmental consolidation



The following day, she suddenly collapsed on the ward with transient loss of consciousness. On examination, she was pale, clammy but apyrexial (temperature 37°C). Her blood pressure had dropped significantly (80/40 mmHg), she was tachycardic and chest auscultation again revealed poor air entry in the right lung. The ECG had not changed but a repeat chest film now showed a well demarcated segmental consolidation (figure 1). Intravenous colloids were administered and she was transferred to the Coronary Care Unit for thrombolysis.

The picture was suggestive of an extensive PE with haemodynamic compromise. However, an urgent echocardiogram failed to show any evidence of right ventricular strain or pulmonary hypertension, which cast doubt over the diagnosis. An urgent spiral CT scan of her chest (figure 2) revealed an area of consolidation in the right lobe consistent with pneumonia rather than infarction, with no evidence of thrombus in the major pulmonary vessels.

Thrombolysis was discontinued and further antibiotics were added. Her condition improved over the following days and she was discharged after a week.

Case 2

A previously well 39-year-old woman presented as an acute medical admission in early April 1999 with a two-week history of a dry

Figure 2. CT scan of case 1, showing an area of consolidation in the right lobe consistent with pneumonia

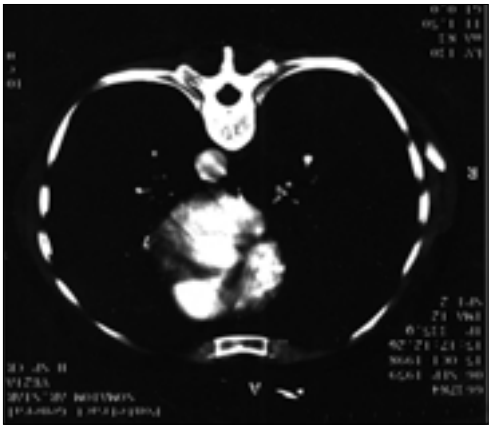
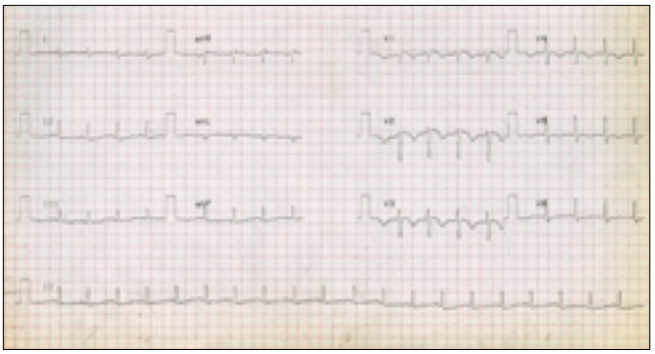


Figure 3. ECG of case 2, showing right ventricular strain

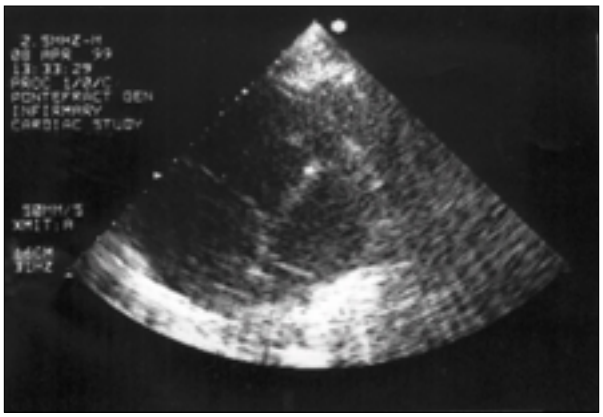


cough and, more recently, left pleuritic chest pain with increasing dyspnoea. Prior to the onset of her presenting symptoms, she had felt lethargic for two months. A course of antibiotics prescribed to her had had no effect. The only risk factor for thromboembolic disease was her usage of the oral contraceptive pill.

On admission she was clearly tachypnoeic, with an oxygen saturation of 91% on room air. Her heart rate was 120 bpm and regular, her blood pressure 137/83 mmHg, there was no clinical evidence of right heart failure and some soft crackles were heard in the left lung base. Abdominal examination was normal. Routine haematology, biochemistry and coagulation profile were normal, apart from a raised C-reactive protein (CRP) of 61.9 mg/dL. The arterial blood gas on air revealed marked hypoxia with respiratory alkalosis (pH 7.51, P_{CO2} 2.7 kPa, P_{O2} 7.9 kPa, base excess –5). Her ECG showed features of right ventricular strain (figure 3) and the plain chest film was reported as normal. At this point, the most probable diagnosis was acute PE and she was commenced on low molecular weight heparin.

The following day, a VQ scan was reported as showing a

Figure 4. Echocardiogram of case 2 showing dilated right ventricle and atrium



low probability of PE. An urgent echocardiogram showed significant right ventricular dilatation, (figure 4) severe tricuspid regurgitation and high right ventricular and pulmonary arterial pressures, all in the absence of any obvious cardiac cause. It was felt that PE was still the likeliest diagnosis, despite the negative VQ scan, and she was commenced on intravenous heparin and warfarinised.

On the fourth day of her admission, the patient began to deteriorate, with increasing hypoxia (despite high-flow oxygen). She looked pale and clammy and her blood pressure dropped to 100/60 mmHg. With a view to considering thrombolysis, an urgent spiral CT of the chest was performed which failed to reveal any thrombi in the pulmonary vessels or any other significant abnormality. Further differential diagnoses of primary pulmonary hypertension and pulmonary vasculitis were considered, but her clinical picture did not fit these.

Plans were made for the patient to be transferred for pulmonary angiography. Unfortunately, her condition deteriorated further that same day and she died from circulatory failure despite aggressive inotrope support and assisted respiratory ventilation. A post-mortem later revealed the unexpected findings of metastatic adenocarcinoma with multiple pulmonary tumour emboli. The source of cancer was uncertain, but it appeared to arise from the intra-abdominal cavity.

Discussion

The two cases described here were unusual as they mimicked PE. In our first case, the initial pyrexia and leucocytosis obviously favoured an infective aetiology. However, the subsequent hypotensive episode with no fever suggested otherwise. The sudden extension of pneumonic consolidation might have resulted in a temporary 'vaso-vagal' phenomenon.

In our second case, the diagnosis of pulmonary tumour embolism (PTE) was quite unexpected. This phenomenon has been recognised in known cancer patients since 1937.[3] Only 11 cases have been reported in the English literature in which pul-

Key messages

- Pulmonary thromboembolism remains difficult to diagnose with certainty; its mimics are many
- Transthoracic echocardiography can be recommended as a standard investigative tool in the diagnosis of acute PE
- Routine abdominopelvic ultrasound in all patients with PE who have no obvious risk factors can increase diagnostic yield

monary hypertension, due to PTE, was an initial symptom of occult cancers.[4] A variety of malignancies can cause PTE, including hepatoma, choriocarcinoma, and adenocarcinomas of breast, stomach, pancreas, gall bladder and prostate. The incidence has been reported to be between 2.4% to 26% in patients with cancer and clinically PTE is extremely difficult to differentiate from PE.[5] Both conditions can lead to acute pulmonary hypertension with cor pulmonale. It is important to differentiate between the two as the treatment modalities are quite different – PE requires anticoagulation/thrombolysis whereas PTE (if detected early enough) can be managed with aggressive chemotherapy.[6]

Several other conditions that mimicked PE at clinical presentation have been mentioned in the literature. The more recent reports include an ascending aortic pseudoaneurysm,[7] myocarditis and pleuritis.[8] In both cases, the eventual diagnosis was established with the help of echocardiography and computed tomography.

Pulmonary angiography is the gold standard for detailed imaging in PE but there are associated difficulties and it is not easily accessible.[2] Spiral CT is less invasive but does not reveal haemodynamic information.[9] By comparison, echocardiography is more readily available, cheaper, mobile and will provide cardiovascular measurements.[10]

The echocardiographic features of right ventricular and atrial dilatation, abnormal motion of the interventricular septum and tricuspid regurgitation were seen in approximately 70–75% of patients with acute PE in several studies.[11] Although such findings can be seen with any cause of pulmonary hypertension, these usually present less acutely. More recently, transoesophageal echocardiography has been used to identify thrombus within central pulmonary arteries,[12] a feature which is diagnostic of PE. Many experts now recommend echocardiography as a standard investigative tool in the diagnosis of PE.

Routine abdominopelvic ultrasonography may have a role in the assessment of some patients with PE. It might have established the diagnosis in our second patient. It is well recognised that patients with all malignancies have an increased risk of thromboembolic disorders. The frequency of PE in patients with cancer at necropsy is particularly high in the elderly. A recent study has shown that intra-abdominal and pelvic malignancies have the highest frequency of PE.[13] Unfortunately, several studies from Europe have examined the usefulness of routine abdominopelvic ultrasonography in patients with a diagnosis of deep vein thrombosis (DVT) or PE and have showed no advantage in the majority of cases.[14] However, the fact that these were mixed cases of PE and DVT may have affected the results.

In conclusion, we have presented two separate conditions which can mimic PE, and highlighted the difficulties in differentiating between them. There is a significant risk associated with anticoagulation in PE, which increases with age and comorbidity. Thrombolysis is usually reserved for extensive PE,[2] but there is a 2–4% risk of intracranial haemorrhage associated with it.[15]

We recommend performing echocardiography routinely to detect those patients with acute pulmonary hypertension. Furthermore, we suggest routine abdominopelvic ultrasonography in all patients with PE who have no obvious risk factors, as this can detect occult intra-abdominal malignancies. Further studies will have to be conducted to evaluate this common diagnostic tool with respect to PE.

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