Lesson of the Week

Pulmonary thromboembolism presenting as abdominal pain

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Pulmonary thromboembolism has protean manifestations. In 1957 Israel and Goldstein described six patients who presented with abdominal symptoms.¹ Despite this, abdominal pain is not commonly listed as a presenting feature of the condition—for example, it is not mentioned in recent major textbooks of general² or respiratory³ medicine. We report two patients in whom abdominal pain was the sole presenting feature of pulmonary thromboembolism.

Case 1

A 55 year old man with a history of rheumatoid arthritis, ischaemic heart disease, and peripheral vascular disease was admitted after the acute onset of severe breathlessness. On admission he was dyspnoeic and distressed with widespread inspiratory crackles in his chest. Radiography confirmed the clinical impression of pulmonary oedema and he responded rapidly to intravenous diuretics. A few hours after admission he became acutely breathless again and clinical examination once more disclosed the signs of pulmonary oedema. This deterioration coincided with his cardiac rhythm changing from sinus on admission to atrial fibrillation with a ventricular rate of 156 min. His symptoms once more responded to intravenous diuretics and his heart rate was controlled with digoxin. The electrocardiogram showed atrial fibrillation and incomplete left bundle branch block. His creatinine kinase activity was considerably increased on admission (4800 u/l), falling to normal over the next few days. Activities of other cardiac enzymes remained normal over this period.

On the fourth day after admission he complained of pain in the left hypochondrium. Nothing was found on examination and he was given analgesics. A diagnosis of mesenteric ischaemia was considered in view of the history of severe widespread vascular disease and atrial fibrillation, but there was no confirmatory evidence such as rectal bleeding. The next day the pain had become more severe and was worsened by respiration. He coughed up a small amount of fresh blood. On examination a loud pleural rub was heard at the left base laterally. There was no change in the chest radiograph or electrocardiogram. A clinical diagnosis of pulmonary thromboembolism was made and he was anticoagulated with heparin initially and later warfarin. Lactate dehydrogenase activity rose transiently to 1180 u/l after this episode. A ventilation-perfusion lung scan showed a matched ventilation-perfusion abnormality at the right apex and an unmatched perfusion defect at the left base suggestive of the condition. The diagnosis was proved conclusively by a pulmonary angiogram, which showed clear evidence of pulmonary thromboembolism in the left lower lobe. His symptoms subsided over the next few days and he was discharged home taking oral anticoagulants.

Case 2

A 63 year old engineer presented with general malaise, herpes labialis, and a persistent productive cough. The white cell count was $176 \times 10^9/l$, the film showed mature lymphocytes, and bone marrow examination con-

Patients with pulmonary thromboembolism may present with acute abdominal pain and have no other features suggesting the diagnosis.

firmed the diagnosis of chronic lymphatic leukaemia. Clinical examination and a chest radiograph showed right upper lobe pneumonia. No causative organism was identified in the sputum. Transbronchial lung biopsy disclosed changes consistent with an organising pneumonia; no granulomas or leukaemic infiltrates were seen. Varicella zoster titre was raised at 1/64. He was treated with acyclovir, prednisolone, and chlorambucil. Although there was no clear evidence of tuberculosis he was given chemoprophylaxis with isoniazid. There was a good clinical response; his white cell count fell to $120 \times 10^9/l$ and the shadowing in his right upper lobe diminished substantially.

The next month he represented with left hypochondrial pain radiating through to the back, which was worsened by respiration and percussion over the splenic region. The spleen was not palpable and there were no other abnormal physical signs. A radiograph showed resolving changes at the right apex but no abnormalities at the left base. The electrocardiogram was normal. A diagnosis of splenic infarction was considered in view of the history of chronic lymphatic leukaemia, but an isotope scan of liver and spleen was normal. In the light of our experience in case 1 a diagnosis of pulmonary thromboembolism was thought possible and a ventilation-perfusion lung scan performed, which showed an unmatched perfusion defect at the left base. Phlebography disclosed deep venous thrombosis in the left leg. The day after admission he developed a pleural rub at the left base and the radiograph showed elevation of the left hemidiaphragm. He was treated with heparin and later with warfarin with complete resolution of symptoms.

Comment

In both cases the principal and indeed only presenting feature was abdominal pain. In the first case there was initially no pleuritic element to the pain and no pointer to an abnormality above the diaphragm. In the second case exacerbation of the abdominal pain by respiration was a prominent feature but there were no clinical or radiological signs to suggest a thoracic cause. In both cases the diagnosis was in considerable doubt and in each entirely different diagnoses were considered initially.

The mechanism of the abdominal pain in these patients is not entirely clear. The most likely cause is diaphragmatic pleurisy as a result of infarction of a basal part of the lung. This, however, does not explain why abdominal pain so clearly preceded other features of pulmonary infarction such as haemoptysis, nor the fact that in case 1 there was initially no pleuritic element to the pain. In the six patients reported by Israel and Goldstein the abdominal pain was predominantly right sided and they suggested that hepatic congestion secondary to acute right heart failure was the cause in at least some patients.¹ In our patients there was no evidence of right heart failure.

Other mechanisms which have been advocated as the cause of pain in pulmonary thromboembolism include tension on sensory nerve endings in the parietal pleura and hyperalgesia of intercostal muscles.⁴ Either or both of these mechanisms might explain some

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of the features of our patients, such as exacerbation by movement (case 1) or percussion (case 2).

The tyro surgeon is warned to beware of right basal pneumonia mimicking an acute abdomen⁵ but pulmonary thromboembolism is not listed as a diagnosis of which to be wary, although it is recorded that cholecystectomy has erroneously been performed in patients with the condition.¹

It is not uncommon at necropsy to find pulmonary thromboembolism that was unsuspected during life. One reason for this is the varied presentation of the condition.^{6,7} We suggest that abdominal pain as a presenting feature deserves to be more widely known.

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(Accepted 31 July 1984)

Clinical Algorithms

Urticaria and angio-oedema

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The urticarias are characterised by transient, localised, itchy swellings of the skin. The lesions begin suddenly as erythematous patches which become raised with central paling as oedema develops in the dermis; they last for a few hours but rarely for longer than 48 hours. Angio-oedema is the name given to the same process when it occurs in the subcutaneous tissues. The resulting deep swellings, which often affect the eyelids, lips, and tongue, cause a burning sensation or pain. Angio-oedema and urticaria often occur together, although each may occur on its own.

These conditions affect 15% to 20% of people at some time in their lives.¹ In most cases urticaria is short lived and requires only antihistamines without the need for detailed investigation. Acute urticaria is that which lasts less than two months. It often has an allergic cause and is often associated with atopy. Possible allergens include drugs, injections, food, drinks, and inhaled particles, but in 75% of cases no causal agent is found.¹ Treatment of patients with chronic urticaria is unsatisfactory, and as 20% are still affected after 10 years this can mean years of frustration for the patient and doctor.¹

Urticaria is only rarely due to general medical causes and very rarely due to malignancy. For this reason a thorough history and examination are required, but only patients with relevant symptoms or signs warrant further investigation. Routine investigation of all patients is rarely of value² but is sometimes carried out to reassure the patient and doctor that no causes have been missed.

Physical urticarias

A different approach is required for the physical urticarias,³ in which physical factors, such as friction or cold, trigger the rash, and for contact urticaria,⁴ in which contact with an allergen produces localised weals. A physical cause is more likely if individual lesions disappear within 30 minutes. The patient may not have noticed the physical cause, and it is worth using the

physical provocation tests (described in the flow chart) in all cases of uncertain cause. The commonest physical urticarias are symptomatic dermographism and cholinergic and cold induced urticaria. The morphology of the lesions provides few clues, but 2-3 mm papules suggest cholinergic urticaria while linear lesions occur in symptomatic dermographism. Persistent lesions which resolve with scaling or pigmentation suggest urticarial vasculitis.⁵ Lesions in areas exposed to light are a feature of solar urticaria while disease of the hand and mouth favours contact urticaria.

Preventing urticaria

Avoidance of the factors which commonly exacerbate urticaria will often lead to improvement. The most important is aspirin, which causes an exacerbation in 30% to 50% of patients. All patients should be warned not to take aspirin. Other substances which exacerbate urticaria in fewer patients are the food colouring tartrazine and the food preservative sodium benzoate. A challenge test battery has been used to identify patients in whom these factors are implicated.⁶ Using this, Warin and Smith achieved considerable improvement in 75% of patients with positive reactions by appropriate dietary manipulation; patients were able to relax the diet after five to six months. Provocation tests may be difficult to interpret, they are time consuming, and particularly when aspirin is used severe reactions are possible, making it desirable for the test to be done in an inpatient. An alternative is to administer a low salicylate, low benzoate, and tartrazine free diet for a trial period and continue it only in those showing benefit. Diagnosis of specific food allergy is helped by the patient keeping a diary recording diet and attacks. This is most useful in patients with well defined intermittent attacks. An exclusion diet with reintroduction of foods on successive days need be used only for severely affected patients who have not benefited from other measures.

Particular attention should be paid to any infection, including dental sepsis and chronic sinusitis. Eosinophilia should alert the clinician to the possibility of parasitic infection. Treatment of these infections sometimes leads to improvement in the urticaria. More commonly, the patient's drug treatment is responsible. Penicillin and other antibiotics cause urticaria by an allergic mechanism