Diagnosing the cause of pulmonary infiltrates in an acutely unwell patient

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DESCRIPTION

A woman in her 60s who had been admitted with suspected sepsis and pulmonary infiltrates was referred for consideration of bronchoalveolar lavage for microbiological sampling.

Initial presentation occurred during the SARS-CoV-2 pandemic with exertional breathlessness, fever, night sweats and weight loss over 3 months. Her medical history included exposure to tuberculosis in early life, coeliac disease, diverticulosis and 22 pack-years of cigarette smoking. Initial investigations demonstrated C-reactive protein (CRP) of 120 mg/L, erythrocyte sedimentation rate (ESR) of 93 mm/hour, haemoglobin of 74 g/L, iron of 3 µmol/L, total white cell count of 11.95×10^9/L and platelets of 518×10^9/L. Microbiological investigations were unremarkable and nuclear cytoplasmic antibodies were reported to be 'indeterminate'. Thoracic CT imaging demonstrated patchy ground-glass changes in the upper lobes. Temporal artery Doppler ultrasound showed some features suggestive of arterial wall inflammation and ^{18}FDG PET CT demonstrated avidity of the intraparenchymal pulmonary infiltrates but no diagnostic features of vasculitis[1] (figure 1). A non-productive cough subsequently developed in conjunction with worsening breathlessness, fever and vomiting. Physical examination revealed a temperature of 38°C, oxygen saturation of 94% (FiO_2 of 28%), a non-blanching petechial rash and dependent pitting oedema, but normal thoracic auscultation. Serum creatinine concentration was 293 µmol/L, urine protein to creatinine ratio was 142.9 mg/mmol, haemoglobin was 52 g/L and an autoimmune profile revealed positive antineutrophil cytoplasmic antibody (perinuclear pattern) myeloperoxidase antibody (pANCA) with MPO of 31 kIU/L, consistent with MPO-positive vasculitis. Urinalysis showed white cell count $>100\times10^6$/L and red blood cell count $>100\times10^6$/L, with scanty epithelial cells and no growth (no comment had been sought on the presence of casts). Commencement of immunosuppressive therapy was desirable, but concern about coincident pulmonary sepsis remained.

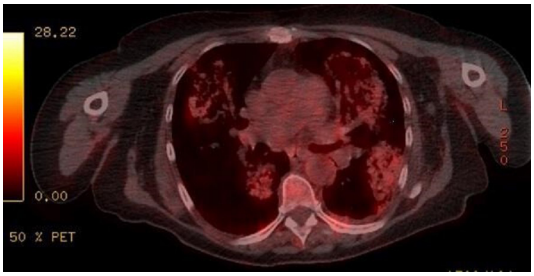

Figure 1 Axial fused ^{18}FDG PET CT (GE Healthcare Discovery 710) imaging showing bilateral multifocal patchy consolidation and ground-glass infiltrates with diffuse mild increased uptake, no significant lymph node abnormality and a small left pleural effusion. The aorta, main pulmonary arteries and their main branches do not show any metabolically active mural thickening.

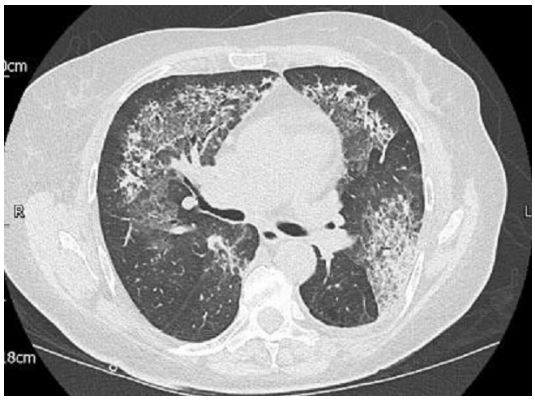

Figure 2 High-resolution CT (GE Healthcare Discovery CT 750 HD) showing a combination of bilateral peribronchial dense consolidative foci with reticulation and surrounding ground-glass shadowing, resulting in a crazy paving-type appearance.

Patient's perspective

A breathing test was a much more tolerable way to diagnose my lung problem than having an endoscopy, particularly when I felt so unwell.

Learning points

- Pulmonary parenchymal avidity on ^{18}FDG PET CT imaging may be present in vasculitis due to vasculitis-associated inflammation.
- Pulmonary infiltrates on thoracic imaging can result from intrapulmonary haemorrhage in the absence of haemoptysis, but can be expected to be associated with an increase rather than a decrease in gas diffusion, in contrast to other causes of infiltrates.
- Accurate interpretation of gas diffusion measurements requires knowledge of the haemoglobin concentration in order to perform appropriate adjustment.

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An opinion was sought on the utility of bronchoscopy to exclude pulmonary infection as the cause of the pulmonary infiltrates. Rather than perform an invasive investigation in an acutely unwell patient with respiratory failure, high-resolution CT imaging (figure 2) and urgent gas transfer measurement were advised as the initial investigations in the expectation that the cause of hypoxaemia and pulmonary infiltrates was intrapulmonary haemorrhage, rather than an infective process or interstitial lung disease. While a further reduction in haemoglobin concentration was suggestive of haemorrhage, it was not considered to be reliably predictive, particularly given the known high prevalence of anaemia in ANCA-associated vasculitis.[2] K_{CO} was initially reported as 1.62 mM/min/kPa/L (107% predicted), which was considered insufficiently raised to confidently diagnose intrapulmonary haemorrhage. However, following correction for haemoglobin concentration,[3] the value was revised to 3.32 mM/min/kPa/L (221% predicted). Plasma exchange, oral cyclophosphamide and pulsed intravenous methylprednisolone were commenced following renal biopsy, leading to resolution of symptoms and biochemical, imaging and gas transfer abnormalities. Crescentic glomerulonephritis was evident on subsequent histopathology.

Contributors All authors have made substantial contribution to the interpretation of data for the work and to the drafting and revising of the work. DP was responsible for clinical management of the patient, conception, design, data acquisition and interpretation, and revision of the manuscript. MUK was responsible for data acquisition and interpretation, and drafting of the manuscript. OA was responsible for data interpretation and contributed to the manuscript in relation to the imaging aspects. All authors approved the final version of the manuscript.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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