Case Reports and Reviews

Case Report

Complex Consolidation: the importance of the white cell differential

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A 38-year old male international pilot presented to accident and emergency with an eight-week history of dry cough, breathlessness, fevers, weight loss and night sweats. His only medical history was a diagnosis of asthma three years previously for which he took a regular fluticasone inhaler and salbutamol as required. Optimisation of inhaled therapy by his general practitioner had not provided any symptomatic relief. He was a non-smoker and denied previous exposure to tuberculosis (TB). On examination, he was febrile at 37.5°, tachycardic at 110 and desaturating to 93% on air. Chest auscultation revealed right lower and mid zone inspiratory crepitations.

Admission blood tests demonstrated a raised white cell count (WCC) of 12.0 x 10^9/L (n=4.0-11.0), an eosinophilia of 3.8 x 10^9/L (n=<0.4), a C-reactive protein (CRP) of 114.5 mg/L (n=<3.0) and platelet count of 426 x 10^9/L (n=150-400). A chest X-ray revealed bilateral, multi-lobar, patchy consolidation (Figure 1). Empirical intravenous antibiotic therapy with benzylpenicillin and clarithromycin was started to treat community-acquired pneumonia. Additional laboratory testing was negative for HIV, atypical pneumonia antigens, autoimmune disease and viral infection.

Further imaging with a CT thorax confirmed patchy bilateral consolidation but predominantly affecting the right upper and middle lobes (Figure 2). Mediastinal, hilar, subcarinal and precarinal lymphadenopathy was also noted (Figure 3). Fibreoptic bronchoscopy uncovered mild mucosal inflammation and right upper/lower lobe washings cultured Haemophilus Influenzae, sensitive to amoxicillin and tetracycline, but resistant to clarithromycin. Staining for acid-fast bacilli was negative.

His antibiotic therapy was escalated to tazocin on day four of admission in view of increasing inflammatory markers (WCC 13, CRP 253) and persistent pyrexia. Following a week's course of tazocin, he improved clinically and biochemically (WCC 11.5, CRP 47.1) but continued to spike low grade fevers. He was discharged on oral amoxicillin with a plan for early ambulatory clinic follow up.

At his first clinic appointment, he continued to report ongoing night sweats and fevers, preventing him from returning to work. A repeat blood test demonstrated an increasing eosinophilia of 9.6. The mycobacterial culture from bronchoscopy was found to be negative at this stage. A second plain film of the chest showed worsening right sided multi-lobar consolidation (Figure 1). The patient was subsequently referred for a second diagnostic bronchoscopy which collected a focused bronchoalveolar lavage from the right middle lobe bronchus. A cell differential demonstrated 75% eosinophils, 15% histiocytes, 7% neutrophils and 2% lymphocytes.

In view of the eosinophil differential of over 25% and radiological consolidation, a diagnosis of chronic idiopathic eosinophilic pneumonia was confirmed. Following three days of pulsed intravenous methylprednisolone infusions, the patient reported a marked improvement in his symptoms. A plain chest film performed a week after treatment revealed complete resolution of the radiological findings (Figure 1). He continued on a tapering dose of oral steroids with regular outpatient follow up as the risk of relapse is known to be high.

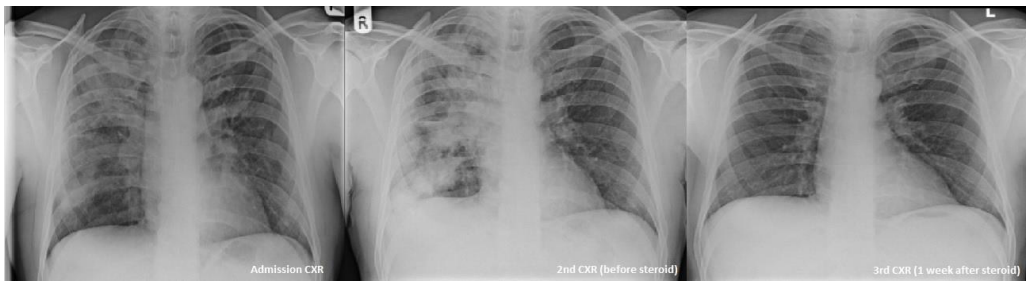

Figure 1: Chest X-Ray Series

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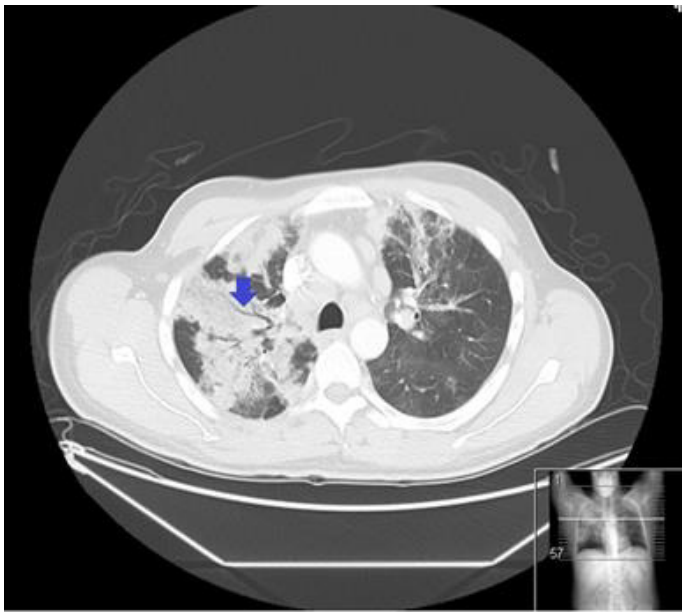

Figure 2: CT Thorax - apical lung slice. *Blue arrow: air bronchogram within consolidation.

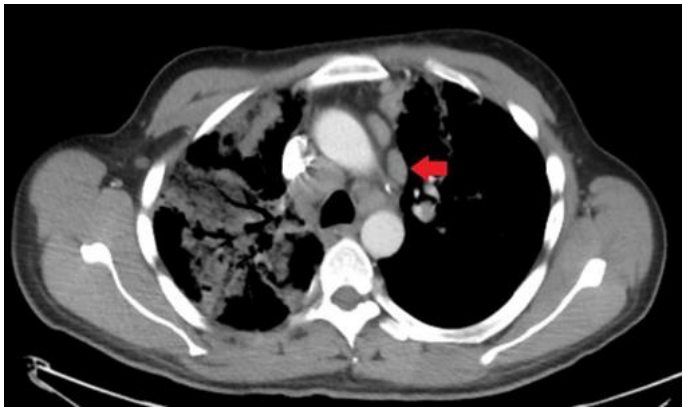

Figure 3: CT Thorax – apical lung slice. *Red arrow: thoracic lymphadenopathy.

Discussion

The clinical syndrome of pulmonary eosinophilia can be attributed to several triggers. These include helminthic infection [1], drugs, allergic bronchopulmonary aspergillosis [1,2], vasculitis and idiopathic eosinophilic pneumonia, which can manifest in an acute or chronic form. Chronic eosinophilic pneumonia (CEP) is categorised as an interstitial lung disease (ILD), typified by a large accumulation of eosinophils in the alveolar and interstitial spaces [3]. It represents only 0 - 2.5% of ILD cases in Europe per year [4]. Women are affected twice as often as men, with a peak incidence of 30-40 years of age [5]. Interestingly, the condition has a preponderance for those who are non-smokers, asthmatic and of Caucasian ethnicity [1,6].

As this case demonstrates, the presentation of CEP is subacute, characterised by systemic symptoms of night sweats, weight loss and fever5, which lends itself to a wide differential, all of which must be excluded. Diagnosis should be based on the clinical presentation, serum eosinophilia [5,8], classical radiological findings of bilateral, pleural or peripherally-based consolidative opacities [1,7], and a bronchoalveolar lavage (BAL) differential indicative of >25% eosinophils [5,9]. Biopsy of lung tissue is only required in cases of poor treatment response, atypical imaging findings or an equivocal BAL [5].

Following initiation of systemic glucocorticoid therapy, the expected response includes resolution of blood eosinophilia within hours, clinical improvement after 48 hours [5] and radiographic improvement at 2 weeks [8]. The optimal duration of steroid treatment is unknown and the majority of patients unfortunately require prolonged tapering courses with associated severe side effects [5,8,10]. Studies looking at the close relationship between CEP and atopy [6] have suggested that asthmatics treated with inhaled steroids have a lower rate of relapse, but could not control the disease as a monotherapy [10,11]. Should inhaled corticosteroids be used concurrently in asthmatic patients therefore, to reduce the maintenance dose of oral steroid following a diagnosis of CEP?

Learning points

- In a case of pulmonary eosinophilia, one must always exclude the alternative causes to idiopathic eosinophilic pneumonia, such as parasitic infection, drugs, vasculitis, malignancy and allergic bronchopulmonary aspergillosis
- Always check the cell differential in the setting of a raised white cell count as this can provide early clues to the aetiology of the condition
- When a patient presents with a subacute, systemic illness, always consider a more serious underlying disease process
- Consider lung biopsy in the setting of a poor treatment response to steroids, atypical imaging findings or an equivocal bronchoalveolar lavage
- Monitor closely for the side effects of systemic glucocorticoid therapy and initiate preventative therapy sooner rather than later

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