CASE REPORT

Eosinophilic pneumonia presenting as life-threatening ARDS

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SUMMARY

We present a case of a 25-year-old woman with sudden onset of shortness of breath, cough and malaise, 24 h after discharge from a psychiatric hospital. She had been there for 2 weeks after a suicide attempt with lye, and started treatment with paroxetine, alprazolam and valproic acid. She also started smoking 20 cigarettes/day during that hospital admission. Brought to the emergency department, she evolved in the first 24 h with respiratory failure and shock needing intensive care unit (ICU) admission, with mechanical ventilation and vasopressor support. Empiric antibiotic therapy was started (piperacillin-tazobactam and azithromycin) suspecting healthcare-associated pneumonia. The patient's chest radiography progressed with bilateral infiltrates. Peripheral blood eosinophilia was seen on the second day. A bronchoalveolar lavage was performed and had 50% of eosinophils. She was started on treatment with steroids and the next day no longer needed vasopressors; 4 days later she was extubated.

BACKGROUND

Acute eosinophilic pneumonia (AEP) is a severe acute illness, frequently misdiagnosed as community-acquired pneumonia and often indistinguishable from acute respiratory distress syndrome (ARDS).

Despite infection as the first cause of ARDS, we should always keep in mind other inflammatory lung diseases that prompt different therapy, and for which an early and accurate diagnosis, and early institution of therapy, can obviate unnecessary morbidity and mortality.

CASE PRESENTATION

After a suicide attempt with lye, a 25-year-old woman was admitted to a psychiatric hospital for 2 weeks. She started treatment with paroxetine, alprazolam and valproic acid. She also started smoking 20 cigarettes/day.

After 24 h of discharge, she developed shortness of breath, dry cough and malaise. On Emergency Department admission she was conscious, afebrile, with a blood pressure 108/61 mm Hg, pulse rate 125/min and respiratory rate 30/min. Cardiac examination was normal. Auscultation of the lungs showed decreased breath sounds. The abdomen appeared normal with no organomegaly.

Arterial blood gas with high-flow oxygen mask (FiO₂ 0.85) revealed a pH 7.39, pO₂ 104 mm Hg, pCO₂ 42 mm Hg, HCO₃ 24.9 mmol/L. The patient's peripheral white cell count was 23 470/μL with 91.6% neutrophils, 4.6% lymphocytes and 2.3% eosinophils. C reactive protein was 41.7 mg/dL.

Figure 1 Bilateral interstitial thickening of the bases.

Renal and hepatic functions were normal. Serum IgE was 40K U/L (<100). Chest X-ray showed interstitial thickening of the bases (figure 1). Echocardiogram was normal. Angio-thorax CT excluded pulmonary embolism and there was no alterations in the parenchyma. She was negative for rheumatoid factor, anti-nuclear antibody and antineutrophil cytoplasmic antibody. HIV was negative. Blood cultures were negative. Urine tests were negative for antigens of *Legionella* and *Streptococcus pneumoniae*. The patient was started empirically on antibiotics with piperacillin-tazobactam and azithromycin, on suspicion of healthcare-associated pneumonia and atypical

Figure 2 Bilateral, diffuse, alveolar infiltrates.

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Figure 3 Normal X-ray.

pathogens. In the first 24 h in the emergency department, she evolved with respiratory failure ($\text{PaO}_2/\text{FiO}_2 < 100$) and need of mechanical ventilation (FiO_2 : 100%, positive end expiratory pressure: 10 (PEEP)), and was admitted in the ICU. In the first 24 h on the ICU, she developed shock requiring norepinephrine; her chest X-ray worsened (figure 2). Peripheral blood eosinophils of 6.5% ($620/\mu\text{L}$) appeared after 48 h in ICU and had a maximum value of $3390/\mu\text{L}$ (10.3%). A bronchoalveolar lavage (BAL) was performed and showed $5.79 \times 10^5/\text{mL}$ cells with 50% eosinophils. BAL cultures were negative for bacteria and fungi. The patient was started on methylprednisolone 125 mg 6/6 h. The next day, vasopressors were suspended and 4 days later she was extubated. A right pneumothorax, interpreted as secondary to the high PEEP and post-bronchoscopy, prompted a chest drain, which was removed after 48 h. She was then transferred to a high dependency unit for continuation of care.

DIFFERENTIAL DIAGNOSIS

- ▶ Healthcare-associated pneumonia
- ▶ Eosinophilic granulomatosis with polyangiitis
- ▶ Fungal infection
- ▶ Eosinophilic pneumonia due to drugs
- ▶ Cardiogenic pulmonary oedema
- ▶ Pulmonary embolism

The differentials were excluded during the investigation process.

TREATMENT

The patient was kept on steroid therapy for 10 weeks with a tapering plan.

OUTCOME AND FOLLOW-UP

Three months later, the patient was evaluated in the outpatient clinic. She had stopped smoking and was perfectly asymptomatic. She continued taking her medication. AEP could also be secondary to drugs such as SSRI's (paroxetine); but in this case, the fact that the patient kept taking them and there was no relapse is proof against this aetiology. Lung function tests were performed and were normal as well a chest X-ray (figure 3).

DISCUSSION

A temporal relation has been reported between the development of AEP and several inciting factors such as heavy dust exposure, drugs and several occupational exposures. However, cigarette smoke is the most frequently implicated trigger in susceptible individuals. There are several data regarding recent initiation of tobacco smoking and its relation with AEP,^{1–3} a recent change in smoking habits and resuming smoking.⁴ AEP can occur within days after the initiation of smoking large quantities of cigarettes.⁴

Learning points

- ▶ Severe acute illness.
- ▶ Misdiagnosed as community-acquired pneumonia.
- ▶ Indistinguishable from acute respiratory distress syndrome.
- ▶ Cigarette smoke is the most frequently implicated trigger in susceptible individuals.
- ▶ Steroids are the gold standard of treatment and improve outcome.

Competing interests None declared.

Patient consent Obtained.

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