



Case report

Acute eosinophilic pneumonia following recent cigarette smoking



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ARTICLE INFO

Article history:

Received 3 August 2016
 Received in revised form
 17 August 2016
 Accepted 21 August 2016

Keywords:

Acute eosinophilic pneumonia
 ARDS
 Smoking
 Bronchoscopy
 Eosinophilia

ABSTRACT

In this report we describe the case of an 18 year old female who presented with fever, shortness of breath, and chest pain. Chest X-ray revealed diffuse bilateral infiltrates and eosinophilia was reported from her bronchoalveolar lavage (BAL) fluid. She started smoking 3 weeks prior to the onset of symptoms and based on her clinical presentation, BAL findings and dramatic improvement, acute eosinophilic pneumonia (AEP) was diagnosed.

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1. Introduction

Acute eosinophilic pneumonia (AEP) is a rare syndrome characterized by abrupt onset of fever, dyspnea and cough along with a characteristic radiographic pattern which includes diffuse bilateral pulmonary infiltrates. AEP is a serious cause of acute hypoxemic respiratory failure in young adults and is potentially life threatening. The etiology of AEP is not well understood. Some articles have reported smoking as a trigger for AEP.

1.1. Case presentation

We present the case of an 18 year old female who was admitted to the local hospital with an abrupt onset of fever (101.4 °F), rapidly progressing shortness of breath for 1 day, non-productive cough, chest pain and syncope. She was admitted to the ICU with a diagnosis of acute respiratory distress syndrome (ARDS). Her WBC was 18×10^3 /dL, with 26% bands and a lactic acid level of 5.6 mEq/L. The patient was allergic to sulfa drugs and firework smoke. She reported no alcohol or illicit drug use, however, she had started smoking within the past 3 weeks to the amount of 2–3 cigarettes per day. The patient had been camping 3 weeks prior to admission, however, no tick bite was noted. She received Piperacillin-

Tazobactam and Vancomycin for the diagnosis of pneumonia but failed to improve. Due to the rapid deterioration of her respiratory status she was intubated and transferred to tertiary care on the 2nd day of admission.

The patient arrived to the tertiary care center intubated, deeply sedated and dependent on ventilator support with temp 101.1 °F, BP 93/53 mmHg, HR 130 bpm, RR 15 bpm, and SPO₂ of 95% on 100% FIO₂. Her pupils were equal and reactive to light, auscultation of her chest revealed diffuse bilateral crackles. Chest radiography demonstrated diffuse bilateral haziness and air bronchogram (Fig. 1).

CT scan of the chest revealed scattered Tree-in-bud opacities in both lungs, with peribronchial thickening (Figs. 2 and 3).

The patient developed profound hypoxemia and hypotension which required vasopressor initiation. ABG analysis showed a pH of 7.20 and PaO₂ of 102 mmHg while on 100% FIO₂. Additionally, she was noted to have significant dyssynchrony with the ventilator so the ventilator was set to deliver low tidal volume at 100% Fio₂ and PEEP of 15 cmH₂O.

Further tests revealed negative ANA, RF, ANCA, urine legionella antigen, and blood culture. Serology for leptospirosis, anaplasmosis, and tularemia were all negative. Flexible bronchoscopy with BAL was performed and turbid, non-bloody fluid was noted. Antigen testing and culture of this fluid yielded no positive results. The differential cell count on BAL fluid revealed 38% neutrophils, 23% lymphocytes, 8% monocytes, and 31% eosinophils.

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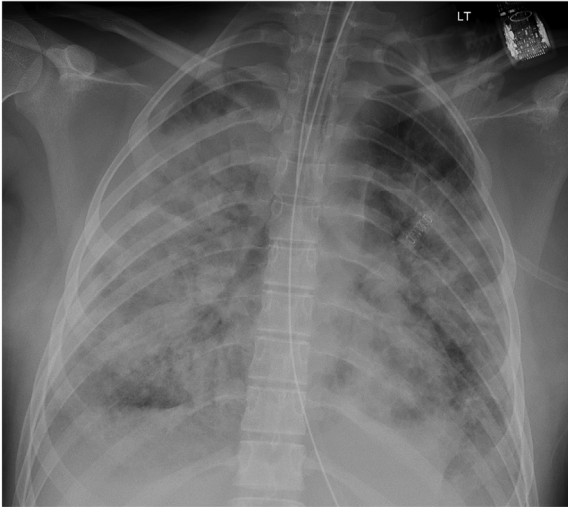


Fig. 1. Chest X-ray on the day of admission showing diffuse bilateral haziness and air bronchogram.



Fig. 3. CT scan of chest revealing scattered Tree-in-bud opacities in both lungs with peribronchial thickening.

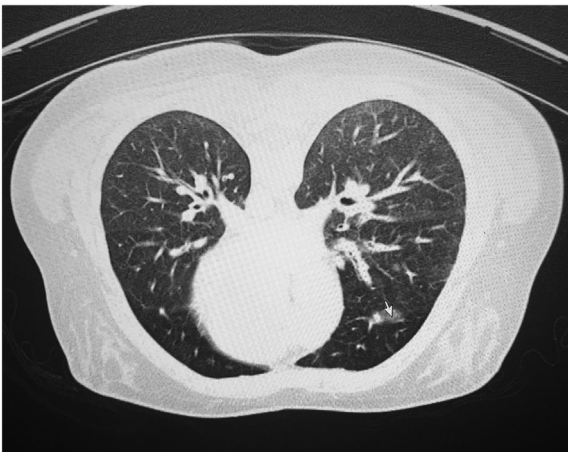


Fig. 2. CT scan of chest revealing scattered Tree-in-bud opacities in both lungs, with peribronchial thickening.

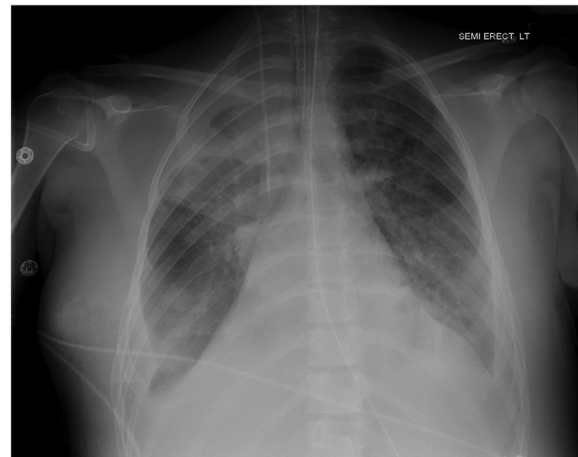


Fig. 4. Chest X-ray after 24 hours of admission.

1.2. Treatment

Upon arrival her antibiotic was changed to Ceftriaxone, Azithromycin and Doxycycline. She was treated with methylprednisolone 60 mg IV Q6H, neuromuscular blocking drug and inhalation Epoprostenol. Repeated ABG analysis revealed a low P/F ratio of 96 despite maximum ventilator setting. Prone position ventilation (PPV) was initiated as a salvage therapy. The patient remained in PPV for 16 consecutive hours before returning to supine position ventilation (SPV), however, she was not able to tolerate SPV and thus PPV protocol was resumed. The patient was weaned off from mechanical ventilation to non-invasive ventilation (NIV) within 4 days and then transitioned to nasal cannula (Fig. 3). She was discharged on a tapering dose of prednisone, 40 mg OD, which was gradually decreased to 10 mg within the next 4 weeks and eventually stopped. She was asked to avoid active and second hand smoking, firework smoke and sulfur containing materials. On her follow-up, 3 weeks post-discharge, she reported a significant improvement in symptoms and her chest radiograph showed marked improvement.

2. Discussion

AEP is characterized by the abrupt onset of fever, dyspnea and cough along with a characteristic radiographic pattern which includes diffuse bilateral pulmonary infiltrates [1]. Eosinophilia of more than 25% in BAL cytology, in the absence of infectious etiologies (including HIV, PCP and aspergillosis), is required for diagnosis.

The pathophysiology of AEP is not fully understood, however, a cytokine-mediated inflammatory response can stimulate lung macrophages leading to AEP [2]. Indeed, IL5, which is prominent in BAL fluid and peripheral blood, is a potent chemotaxin that triggers the release of eosinophils and inhibits apoptosis, prolonging eosinophil survival [3]. Thereby cytokines have been implicated in the role of disease process. Furthermore, Venlafaxine, Sertraline, NSAIDs, Gemcitabine and cocaine-use have been shown to cause AEP [4–6], as well as environmental exposure such as the inhalation of gasoline or high dust and smoke content [7,8]. Smoking has also been reported as a trigger for AEP, with most cases involving young adults, aged between 18 and 37 years old, who had recently started smoking [9]. Bok et al. have reported symptoms of AEP, eosinophilia on sputum analysis and worsening of pulmonary

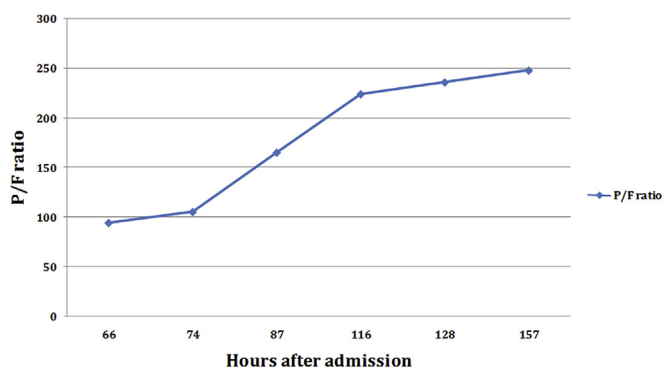


Fig. 5. Graph demonstrating P/F ratio improvement after admission.

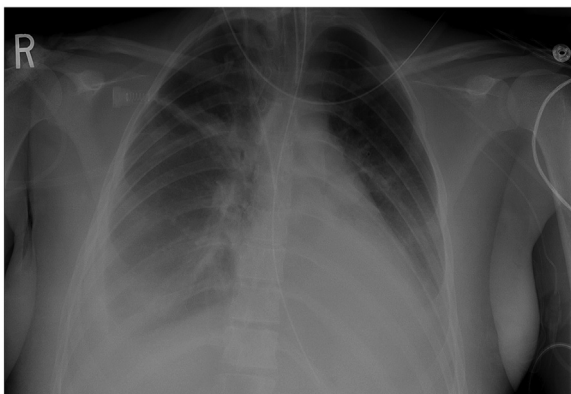


Fig. 6. Chest X-ray on the day of discharge.

function test, during a provocation test with smoking [10]. While those patients' symptoms were mild, they were almost identical to those that occurred with our patient at admission.

Our patient was treated with methylprednisolone, 60 mg IV Q6H, resulting in a dramatic improvement within 24 hours, exemplified by improved aeration on chest radiography (Fig. 4), decreased FIO₂ on ABG (Fig. 5) and toleration of the supine ventilation. After improvement in her respiratory symptoms and her chest x-ray (Fig. 6).

She was weaned from neuromuscular blocking agents and Epoprostenol within 48 hours of steroid treatment. The patient was weaned off from mechanical ventilation to NIV within 4 days, and then transitioned to nasal cannula support with good tolerance.

Corticosteroids are the drug-of-choice for the treatment of AEP. IV Methylprednisolone 60–125 mg IV Q6H should be prescribed to all patients with AEP needing admission to the ICU, followed by oral prednisone 40–60 mg tapered over 2–6 weeks [11]. Rapid response to steroid treatment is characteristic of AEP. If no improvement is noted, an alternative diagnosis should be considered.

3. Conclusion

AEP should be investigated in patients new to smoking or those who have changed smoking habits that present with acute respiratory failure and diffuse parenchymal lung disease. The clinical feature of AEP is similar to ARDS and acute interstitial pneumonitis, so it is easily misdiagnosed. Therefore BAL should be performed as early as possible and if corticosteroid is instituted promptly AEP has an excellent prognosis. In the absence of infection it is important to consider AEP in patients with acute febrile respiratory failure. With proper investigations and timely prescription of medications we can avert morbidity and mortality associated with AEP.

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