Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Critical Care Medicine

Acute Respiratory Distress Syndrome Secondary to Eosinophilic Pneumonia: A Case Report

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DOI: 10.36347/sjmcr.2022.v10i06.012 | **Received:** 28.04.2022 | **Accepted:** 04.06.2022 | **Published:** 09.06.2022

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Abstract **Case Report**

We report a case of acute respiratory distress syndrome (ARDS) in a 26-year-old women. Hypoxemia was refractory despite conventional treatment of ARDS, but the evolution was satisfactory after corticosteroid therapy. Boiological data, results on bronchoalveolar lavage and the clinical evolution led us to the diagnosis of acute eosinophilic pneumonia. This case demonstrates that acute eosinophilic pneumonia should be included in rigorous etiological research before any ARDS, in order to start the appropriate treatment as soon as possible.

Keywords: Eosinophilc pneumonia, acute respiratory distress syndrome, bronchoalveolar lavage, corticosteroid therapy.

INTRODUCTION

Acute idiopathic eosinophilic pneumonia (AEP) is one of the eosinophilic pneumonia that includes various disorders characterized by eosinophilic infiltration of lung tissue, with or without the presence of eosinophils in peripheral blood [1, 2]. Patients with AEP present with febrile dyspnea that can lead to acute respiratory distress in more than 50% of patients, often initially confused with infectious pneumonia. Ventilation support is often necessary [3]. We describe a case of the occurrence of a respiratory distress syndrome of non-infectious cause, linked to idiopathic eosinophilic pneumonia. High-dose corticosteroid therapy, started in the critical period, has allowed to obtain an improvement rapid ventilatory function.

CASE PRESENTATION

A 26-year-old woman without relevant medical history was admitted at the emergency room of our hospital with acute respiratory distress. According to the patient, she had dyspnea, having started with fever 6 days before. She did not smoke and denied any history of drug use, insect bite, or travel history recently. On presentation, she was febrile with temperature on 38,5 ºC, tachycardic with 190 beat/min, respiratory rate was 25 breaths/min, and oxygen saturation was 88% while on 6 L oxygen via nasal cannula. Arterial blood gas analysis showed PaO2 of 52 mmHg. Bilateral pulmonary auscultation showed bilateral crackles. A chest x-ray showed bilateral peripheral opacities mor marked on the right side (Fig 1), completed with Chest CT that confirmed bilateral condensation diffuse, central ground glass images with a bilateral small pleural effusion (Fig 2).The patient's laboratory investigations revealed a white blood cell (WBC) count of 16800 cells/mm^3, neutrophils of 10700 cells/mm^3, and eosinophils of 1800 cells/mm^3. C-reactive protein (CRP), and procalcitonin were 176 mg/l and 0,06 ng/ml respectively. Intravenous antibiotics with ceftriaxon and ciprofloxacin were given immediately for suspected community acquired pneumonia, but the fever was uncontrolled and the dyspnea rapidly progressed, after which she was transfered to the medical intensive care unit. She had worsening hypoxia necessitating use of a high flow of oxygen under high concentration mask and subsequently request mechanical ventilation after tracheal intubation. Results of a transthoracic echocardiogram were normal. Further detection revealed normal antineutrophil cytoplasmic antibodies and anti-nuclear antibody. Antigen-specific IgM against Epstein–Barr virus, cytomegalovirus, coxsackie virus were all negative. Bronchoalveolar lavage (BAL) revealed approximately 42% eosinophils, 24% lymphocytes, 16% neutrophils. We did not find malignant cells and no bacteria, mycobacterium, or fungus were cultured in the BAL fluid. Based on her

clinical history, BAL, and typical radiologic findings, she was diagnosed with AEP. Systemic methylprednisolone therapy 80 mg/8h was immediately used for treatment. Her clinical condition improved favorably. After 5 days, there was a gradual temperature normalization, rapid improvement in hematosis with reduced oxygen requirements and improved blood gases, very rapid normalization of WBC and inflammatory syndrome and radiological improvement (Fig 3). On the 10 day, complete radiological resolution were observed (Fig 4), the patient weaned from the respirator and extubated after 07 days of tracheal intubation. Then after, she was discharged with oral low dose of steroids.

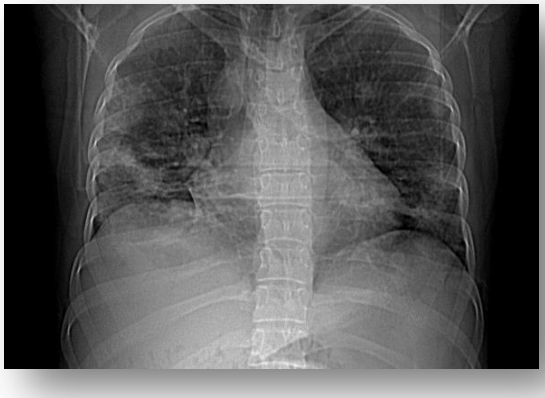

Figure 1: Chest x-ray showed bilateral peripheral opacities more marked on the right side

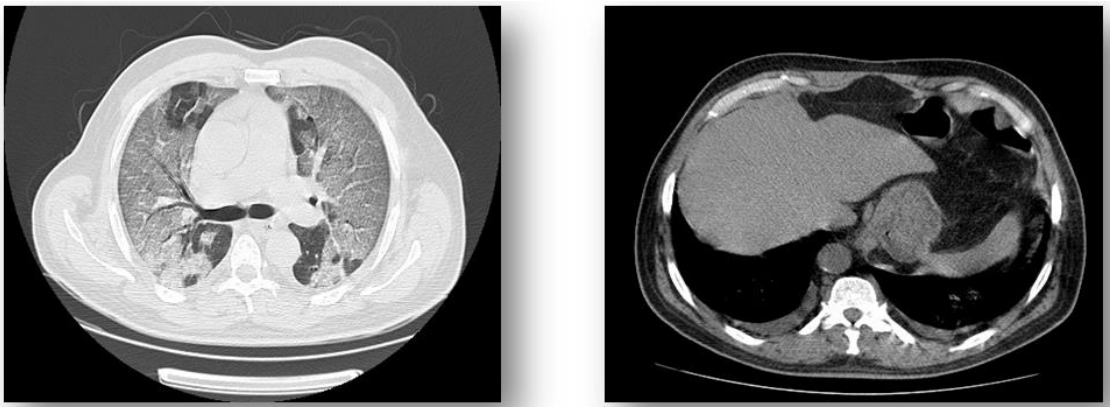

Figure 2: Chest CT schowing bilateral scattered ground-glass opacities and peripheral consolidation with a bilateral small pleural effusion

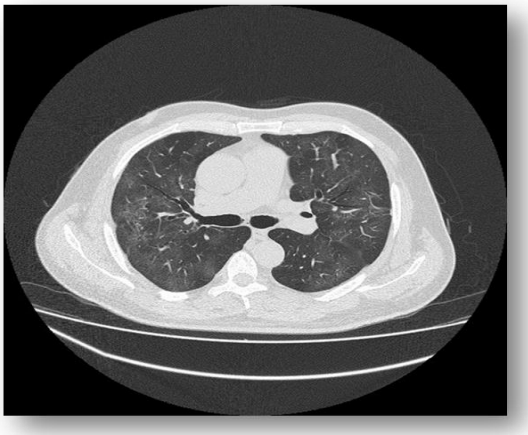

Figure 3: Chest CT scan showing partial radiological resolution of previous lesions after 5 days of corticosteroid therapy

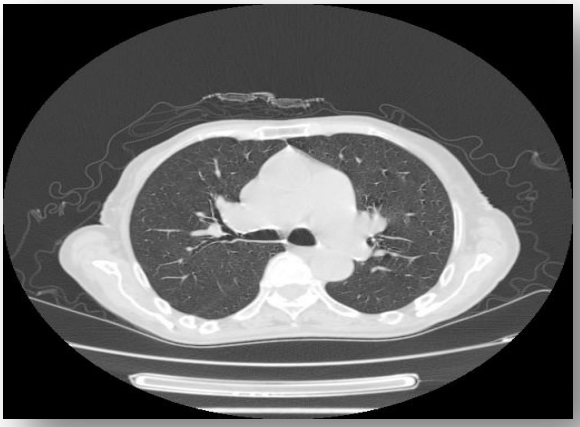

Figure 4: Chest CT scan showing complete radiological resolution of the initial lesions after 10 days of corticosteroid therapy

DISCUSSION

This case reminds us of the need to undertake rigorous etiological research before any ARDS .For this patient without any pathological history (asthma or atopy), the sudden evolution symptoms (<7jours), the level of blood and alveolar PNE has directed research towards eosinophilic disease. After having eliminated a drug or infectious cause, several nosological groups had to be mentioned in this context. Thus, the diagnosis of acute idiopathic eosinophilic pneumonia (AEP) was made. This is a vast nosological framework including acute forms and chronic forms, but cases of ARDS are rare although a few observations have been described. Furthermore, the satisfactory evolution after corticosteroid therapy supported this diagnosis.

First described in 1989 by Allen *et al.*, as a cause of acute respiratory failure [4, 5], the pahogenesis of AEP is not fully known, but several studies have proposed that cigarette smoke is potentially related to the onset of AEP [6]. Philit *et al.*, reviewed twenty two patients with AEP, including eight current smokers, and found that six of the eight current smokers had started smoking within three months before the onset of AEP [7]. More recently, an epidemiologic study11 of this disease identified 18 patients with AEP among 183,000 US military personnel deployed in or near Iraq, indicating that all of the patients were smokers, with 78% of them recently beginning to smoke [8].

The criteria for diagnosis of AEP are defined by the presence of : acute onset of febrile respiratory disease (<1 month) ; bilateral diffuse infiltrates on chest radiography; hypoxemia; lung eosinophilia (either >25% eosinophils in BAL, or marked eosinophilic pneumonia on lung biopsy); no other causes of eosinophilic pneumonia [1-5].

All patients present with some degree of acute respiratory failure and up to 50% require mechanical ventilation. AEP usually occurs bilaterally, and the most common features identifiable on a chest radiograph of patients with AEP are bilateral ground glass attenuation mixed with consolidation and bilateral pleural effusion in more than 90% of the cases [11]. Radiological images of our patient clearly illustrate these findings.

Acute eosinophilic pneumonia is very responsive to corticosteroids therapy and the prognosis is excellent. Another distinguishing feature of acute eosinophilic pneumonia is the complete recovery of both symptoms and x-ray abnormalities without recurrence or residual sequelae, within weeks of treatment [1, 2, 7].

CONCLUSION

Acute eosinophilic pneumonia (AEP) and infectious pneumonia may have a similar clinical presentation, but it is very important to distinguish between the two in patients with respiratory distress syndrome, because the management differs for these conditions. Bronchoalveolar lavage analysis and the exclusion of other causes of alveolar eosinophilia confirm the diagnosis of AEP. Treatment with corticosteroid therapy, initiated early, leads to rapid and often spectacular improvement.

Conflicts of Inerests: All authors of this article have not competing interests.

Sources of Funding: The building and writing of this article has not fundings sources.

Consent: Written informed consent was obtained from the patient for the publication of this case report and its accompanying images.

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