Acute idiopathic eosinophilic pneumonia

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ABSTRACT

Introduction: Acute eosinophilic pneumonia (AEP) is reported to be caused by a variety of etiological agents most of which are drugs. The objective of this case report is to describe the presenting features and the clinical course of AEP in a patient with no obvious etiological cause for an acute pulmonary process. Case Report: This case report details the clinical course of a patient who presented with respiratory distress and was found to have radiological changes. His clinical presentation did not fit that of an infectious cause or that of an anaphylactic etiology. He initially received antibiotics and steroids but these were discontinued after fluid from bronchoalveolar lavage was found to have a high content of eosinophils. His symptoms resolved with continued steroid treatment and corresponded with resolution of his radiological changes. Conclusion: This case report highlights the importance of promptly differentiating the possible etiology of pneumonic process. Treatment with antibiotics alone will not affect the clinical course of eosinophilic pneumonia and treatment with steroids alone will exacerbate pneumonia with infectious etiology.

An early diagnosis significantly changes the management and the eventual outcome. Acute idiopathic eosinophilic pneumonia should always be considered as a viable alternative in patients who do not seem to have an obvious etiology. Timely treatment with steroids may significantly reduce the morbidity associated with this condition.

Keywords: Eosinophilic, Pneumonia, Idiopathic, Acute

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INTRODUCTION

Acute eosinophilic pneumonia is an entity which comes under the larger family of hypereosinophilic syndromes. These syndromes are characterized by persistent eosinophilia, evidence of eosinophil mediated end organ damage and the absence of evidence of allergic, parasitic or other etiologies which are known to evoke eosinophilic response [1]. Disorders such as Loffler’s syndrome, Churg Strauss syndrome, allergic bronchopulmonary aspergillosis and eosinophilic bronchitis share a similar clinical presentation to that of acute eosinophilic pneumonia. The diagnosis of eosinophilic pneumonia is aided by the detection of eosinophils by bronchoalveolar lavage. Notably, there may or may not be an increase in the eosinophil count in peripheral blood. The radiographic presentation of eosinophilic pneumonia is similar regardless of the etiology and therefore eliciting correct history from the
patient is important in determining the underlying possible etiology. Several agents cause acute eosinophilic pneumonia, notable among them are antibiotics and anti-inflammatory drugs. Environmental factors such as dust and personal habits such as smoking tobacco have also been associated with eosinophilic pneumonia. However, there are peculiar situations where there is no obvious underlying etiological factor. This case report highlights such a scenario where the patient had some possible predisposing factors but on closer examination did not have any significant association with them. Hence, the possibility of an acute idiopathic eosinophilic pneumonia was considered and he was managed accordingly.

CASE REPORT

A 44-year-old male presented with a two day history of low grade fever, dyspnea, chest pain, dry cough and generalized myalgia. He had a history of hypertension, hyperlipidemia and was a chronic active smoker. He has been working as a bicycle mechanic but denied significant exposure to metal fumes at his work place. He admitted working in a dusty environment but denied any history of dust-related respiratory problems. There was no history of sick contact, asthma, allergic rhinitis or recent travel. He did not take any home medications. He was allergic to seafood and had a history marijuana, usage in the remote past. He specifically denied any recent changes in his smoking habits. Notable findings on physical examination during his initial presentation were elevated blood pressure (144/90), tachycardia (108/min), fever (101.2°F), usage of accessory respiratory muscles with bilateral crepitations and expiratory wheezes.

Laboratory studies showed acute respiratory alkalosis with hypoxemia, an elevated A-a gradient, leukocytosis (WBC count - 24.8x10⁹/mm³) with neutrophilia (93%) and elevated LDH (265 IU/L). The blood eosinophil count was 2% on day-1. Chest X-ray showed increased interstitial markings with bilateral pleural effusions (Figure 1A). Computed Tomography (CT) of the chest showed bilateral ground glass opacities and pleural effusions (Figure 1B). He tested negative for HIV, influenza, legionella, toxocara and strongyloid and all cultures were negative. An extensive testing of possible allergic reactions was not considered as the patient denied any history of allergies.

Initial treatment included antibiotics and steroids. Patient symptomatically improved by the third day when bronchoalveolar lavage showed 15% eosinophils. A diagnosis of AEP was considered and antibiotics were discontinued. By day-7 his blood eosinophil count reached 10% and his chest X-ray showed remarkable attenuation of the interstitial markings noted earlier (Figure 1C). A follow up chest X-ray done on day-17 showed complete resolution of all earlier noted abnormalities.

DISCUSSION

A variety of airway disorders (asthma, eosinophilic bronchitis, allergic bronchopulmonary aspergillosis, bronchogenic granulomatosis etc.) and interstitial diseases may mimic AEP. The idiopathic form was first described in 1989 as an acute and reversible respiratory failure [2]. Hypersensitivity to common allergens such as firework smoke and sand dust has been reported to cause AEP. It is characterized by acute febrile illness (< 3 weeks), dyspnea, nonproductive cough, pleuritic chest pain, malaise and myalgia. The patient often presents with hypoxemia (PaO₂ <60, PaO₂/FiO₂ <300, sO₂ <90%), tachypnea, tachycardia, crackles, wheezes and distributive shock [3]. Initial laboratory studies may be non-specific with leukocytosis and elevated ESR. Detection of a high percentage of eosinophils (>25%) on bronchoalveolar lavage confirms the diagnosis. Recently a biomarker with high sensitivity and specificity for AEP; thymus- and activation-regulated chemokine (TARC)/CCL17 has been described [4]. The response to steroids is dramatic but spontaneous resolution has also

Figure 1: A) Chest X-ray of patient at the time of presentation; B) Chest computed tomogram (CT) at the time of presentation, C) Chest X-ray demonstrating the resolution of eosinophilic pneumonia.
been observed [5]. Clinical resolution precedes radiographic resolution. It is recommended that steroids be continued for 2–4 weeks after clinical resolution. AEP has not been documented to relapse. Infectious and auto-immune disorders were ruled out in this patient and his condition was considered idiopathic due to the absence of underlying etiological factors. It is possible that a combination of environmental factors (dust) and personal habits (smoking tobacco) may have played a role in the initiation of his pulmonary pathology, but after closely examining the circumstances preceding his illness, it became apparent that they were unlikely to be contributory. He specifically denied any changes in his work habits and personal habits. The patient, upon discharge continued with his original occupation with no further health complaints. He was advised to quit smoking tobacco and marijuana. No further testing modalities such as spirometry were considered as the patient completely recovered in terms of his clinical symptoms and radiological changes.

CONCLUSION

This case illustrates the necessity of actively considering AEP in a patient who does not have any obvious etiological factors. It may be difficult to rule out infectious causes at the initial course of the disease but a timely diagnosis with the help of bronchoalveolar lavage will help to determine appropriate clinical management and reduce prolonged hospitalization.

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Author Contributions
Creticus Petrov Marak – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published
Achuta Kumar Guddati – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of submission.

Conflict of Interest
Authors declare no conflict of interest.

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