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# Idiopathic acute eosinophilic pneumonia: A rare cause of hypoxic respiratory failure<sup>☆</sup>

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## ABSTRACT

Idiopathic Acute Eosinophilic Pneumonia (IAEP) is a life-threatening cause of hypoxic respiratory failure. IAEP is challenging to diagnose as it may mimic infectious pneumonia or acute respiratory distress syndrome. Distinguishing IAEP from these alternatives is important; the mainstay of treatment for IAEP is corticosteroids, a therapy which might not otherwise be indicated. Patients treated appropriately usually experience a full recovery. In this case report we describe the presentation, evaluation, and management of a 19-year old male who presented to the emergency department (ED) in respiratory failure from IAEP. The patient was a military trainee who recently moved to the United States from Saudi Arabia. He also recently began smoking cigarettes for the first time, a known risk factor for IAEP. Upon initial presentation, the patient was in respiratory distress and had an oxygen saturation of 82% on room air. His ED diagnostic workup included chest X-ray showing diffuse interstitial thickening and chest computed tomography that demonstrated diffuse nodular opacification of pulmonary parenchyma. The patient was admitted to the intensive care unit (ICU) where bronchoscopy yielded cytology with 30% eosinophilia. The patient ultimately required 3 days of extra corporeal membrane oxygenation (ECMO) due to worsening hypoxic respiratory failure. After both intravenous and outpatient oral steroid treatments, the patient went on to have a full recovery with no ongoing respiratory issues. To our knowledge, this is the first case of IAEP requiring ECMO reported in the emergency medicine literature.

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## 1. Introduction

Idiopathic Acute Eosinophilic Pneumonia (IAEP) is a life-threatening cause of hypoxic respiratory failure characterized by fever, bilateral pulmonary infiltrates, and pulmonary eosinophilia. It is difficult to diagnose because it mimics infectious pneumonia or acute respiratory distress syndrome in previously healthy individuals [1]. It differs from idiopathic chronic eosinophilic pneumonia (ICEP) by its acute onset, more severe hypoxemia, and usual lack of blood eosinophils at the onset of disease [1]. IAEP often begins acutely in previously healthy young adults, with mean age of 30 years, and with a male predominance [1]. Two-thirds of patients are smokers and usually there is no history of asthma. There are many triggers for the disease including medications,

recent initiation or re-initiation of smoking, smoking large quantities, or change in smoking habits [1,3,6]. Additionally, short-term secondary exposure to cigarette smoke and myriad non-specific inhaled contaminants can induce IAEP [1,4]. While most patients experience a full recovery following treatment with steroids, we present a case of an otherwise healthy male who went on to require extra corporeal membrane oxygenation (ECMO) as a result of IAEP. To our knowledge, this is the first case of IAEP requiring ECMO reported in the emergency medicine literature.

## 2. Case report

A 19 year-old Saudi Arabian male, who had moved to the United States one week earlier, who presented to an urgent care clinic with shortness of breath, fever, and pleuritic chest pain. He was transferred to our Emergency Department (ED) via ambulance and upon arrival was found to be in respiratory distress and hypoxic to 82% on room air. He denied any sick contacts or lower extremity swelling or pain. He denied any previous medical history to include asthma or lung disease. Notably, the patient reported having started smoking cigarettes for the first time in his life upon arrival to the United States one week ago.

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In the ED, his initial vital signs were BP 174/87 mm Hg, heart rate 137 beats per minute, respiratory rate 54 per minute, temperature 100.7 degrees Fahrenheit, and oxygen saturation 94% on 4 l by nasal cannula. He was speaking in 1–2 word sentences. His physical exam revealed decreased lung sounds most pronounced in the right basilar lung field and mild diffuse wheezing in the left lung field. The remainder of the physical examination was non-contributory.

The ED treatment team started 15 l of oxygen by non-rebreather and placed the patient in a negative isolation room given concern for possible infectious etiologies. A chest X-ray revealed diffuse interstitial thickening (Fig. 1). Computed tomography of the chest showed diffuse nodular opacification of the pulmonary parenchyma with right lower lobe predominance (Fig. 2). Laboratory values were notable for a leukocytosis of 32,600 cells with 83.6% neutrophils, 6.4% bands, 1.0% lymphocytes, 8.2% monocytes, and 0.9% eosinophils. His other laboratory studies were unremarkable including metabolic panel, lactate, and urine studies.

He received 1 g of ceftriaxone intravenous for possible pneumonia. He then received three doses of nebulized albuterol with ipratropium. The patient experienced significant improvement in his respiratory status following these interventions. His pulmonary examination demonstrated improved left-sided wheezing. The patient's respiratory rate improved to 32 per minute and his oxygen saturation was 93% on 12 l by non-rebreather mask. The patient was then admitted to the medical intensive care unit (ICU).

Shortly following arrival to the ICU, the patient's respiratory status deteriorated. An arterial blood gas on 15 l of supplemental oxygen yielded a PaO<sub>2</sub> of 35 mm Hg. The ICU team intubated the patient for hypoxic respiratory failure and then performed bronchoscopy which was notable for purulent secretions most pronounced in the basilar and superior segments of the right lower lobe. By hospital day 3, bronchial alveolar lavage samples including culture, and viral studies remained negative for any infectious etiology. Cytology was notable for 30% eosinophils, which was consistent with idiopathic acute eosinophilic pneumonia.

The ICU team initiated methylprednisolone 125 mg IV every 6 h. This resulted in marked improvement in his oxygenation with his arterial blood gas PaO<sub>2</sub> values increasing from 73 mm Hg with FiO<sub>2</sub> of 95% prior to starting treatment to 73 mm Hg with FiO<sub>2</sub> of 70% the following day. However, the patient then experienced an undulating course with recurrent episodes of hypoxemia despite maximal ventilator settings and runs of supraventricular tachycardia to the 200 s with a trace pericardial effusion. On hospital day 9, in the setting of worsening hypoxemia and hypercarbia, the patient was transferred to another facility for ECMO. He was decannulated and extubated after 3 days on ECMO. He continued to improve and shortly after, was discharged. He completed a four-week course of prednisone with taper as an outpatient.

### 3. Discussion

IAEP is a very rare disease, and it is rarer still for these patients to progress to requiring ECMO to temporize their respiratory failure. To our knowledge, this is one of only a handful of cases of ECMO being utilized in a patient presenting with hypoxic respiratory failure from IAEP, and the first such case reported in the emergency medicine literature [2,5,6]. Though the presentation of IAEP is similar to infectious pneumonia and ARDS. It is important to distinguish IAEP from these alternatives due to the fact that the mainstay of treatment for IAEP is corticosteroids [2], a therapy which might not otherwise be indicated. Though this patient's presentation could have easily been mistaken for an infectious pneumonia, antibiotics would have done little to nothing to improve this patient's symptoms.

This case report highlights two important issues: first, though IAEP is a rare cause of acute dyspnea and respiratory failure, it should be on the differential diagnosis for emergency physicians when evaluating patients with acute dyspnea; when index of suspicion for this disease process is high, there should be consideration of steroid therapy. Secondly, though few cases of IAEP will require ECMO, physicians should consider transfer to a center

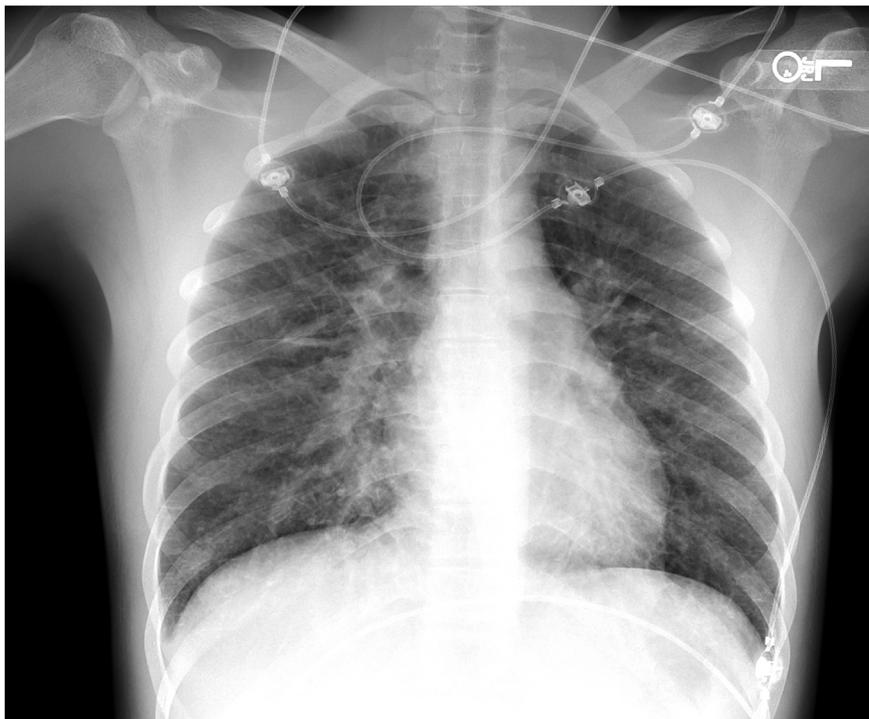
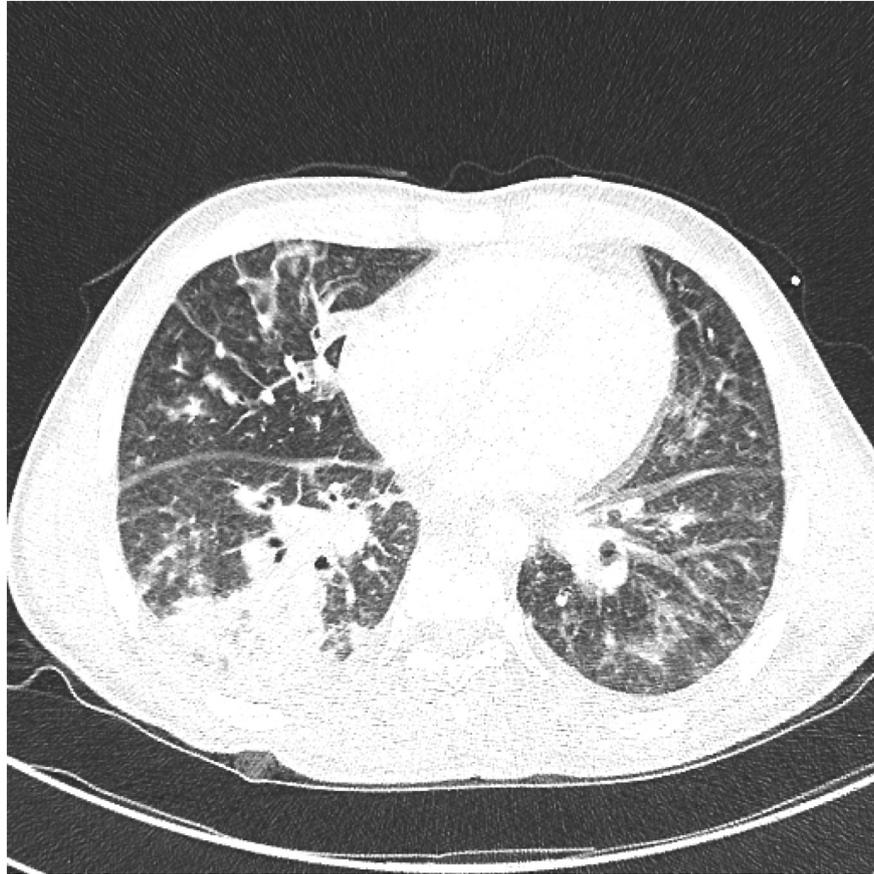


Fig. 1. A portable chest radiograph demonstrating diffuse interstitial thickening.



**Fig. 2.** CT pulmonary angiography notable for diffuse nodular opacification of the pulmonary parenchyma with right lower lobe predominance.

capable of delivering this intervention early when treating patients with severe disease.

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#### Declaration of competing interest

None.

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