Acute eosinophilic pneumonia is a rare disorder first described in 1899. This disease can occur at any age but typically presents in patients 30 to 60 years of age and is more common in males than in females. It is often associated with a variety of underlying conditions, most commonly respiratory infections, with marked respiratory insufficiency often leading to hypoxemia and alveolar flooding. The most common symptoms are fever, cough, shortness of breath, and hemoptysis. Other signs and symptoms include hypoxemia, dyspnea, nonproductive cough, fever, tachycardia, tachypnea, low blood pressure, and pneumonia-like signs (2).

On physical exam, patients typically have a very high fever, tachypnea, and tachycardia. Laboratory test are non-specific. Previously reported lab findings include elevated white blood cell counts, increased neutrophils, increased eosinophils, increased LDH, and increased serum ferritin. Additionally, eosinophilic pneumonia may present with coagulopathy, disseminated intravascular coagulation, and increased neutrophilic infiltrates.

Acute eosinophilic pneumonia (AEP) is a disease that primarily affects the lungs and is characterized by the accumulation of eosinophils in the alveoli. It is a rare condition that can be challenging to diagnose and treat. The clinical presentation of AEP can vary widely, and symptoms may include fever, cough, shortness of breath, and hemoptysis. The diagnosis of AEP is typically made through a combination of clinical, radiographic, and histopathologic findings. Treatment options for AEP are limited, and management often includes supportive care and immunosuppressive therapy. Recovery is variable, and outcomes can range from complete resolution to chronic lung disease.

Case Report

A 49-year-old male presented to emergency department with a chief complaint of shortness of breath and not feeling well for the past 3 days. Additional symptoms included cough, fever, and right-sided pleuritic chest pain. He also reported a recent sick contact with “viral” symptoms. His past medical history was significant for rheumatoid arthritis and previous biopsy-proven non-necrotizing eosinophilic pneumonitis. He had a procedure involving prolonged callout but was not noted to have any respiratory symptoms. He reported occasional alcohol use and denied drug use, specifically substances with a risk of lung injury. On physical exam, the patient was tachypneic with a respiratory rate of 30 breaths/minute. He had a temperature of 100.4°F (38.0°C), heart rate of 110 beats/minute, blood pressure of 120/80 mm Hg, and oxygen saturation of 94% on 4 liters of oxygen. He received 10 mg of prednisone, followed by 30 mg of prednisone, and was admitted to the hospital. He was treated with supportive care and supportive measures under the impression of eosinophilic pneumonia.

Lungs performed in the emergency department included a normal complete blood count with differential, performed elevated ESR count and random non-eosinophilic leukocytes. The patient required 4 liters of oxygen to maintain a satisfactory oxygen saturation. A CT scan showed a number of bilateral infiltrates. The patient admitted to the intensive care unit where he was intubated on the 2nd day. He continued to have persistent fever and symptoms, and his oxygenation and ventilatory support was stabilized on admission. He continued to have persistent hypoxemia which required vasopressors to maintain adequate oxygenation. Despite improvement in clinical status, the patient continued to have fever. He remained intubated, however, he failed to appropriately ventilate on his own and was promptly reintubated. There was no response to current therapy with current fever, respiratory rate, and diuresis. A repeat blood culture and blood culture with BAL and bronchial wash revealed negative cultures and only acute respiratory distress syndrome (ARDS) and eosinophilic pneumonia were noted.

Discussion

The clinical diagnosis of AEP is a diagnosis of exclusion. Current literature suggests that the following findings should be present: acute febrile illness (1-5 days), hypoxic respiratory failure, diffuse pulmonary infiltrate, and eosinophilia in BAL fluid, absence of parasitic, fungal, or other infections, lung biopsy evidence of eosinophilic infiltrate and rapid clinical resolution of symptoms and findings (1-6). It should be noted that there is some disagreement about the necessity of a lung biopsy. The etiology of AEP has been incompletely described, however current evidence suggests an acute hypersensitivity reaction to an unidentified antigen in otherwise healthy individuals. Commonly there is a history of smoking. Usually AEP occurs following the first smoking event or in former smokers at the time they quit the habit. There are also cases associated with weekend “binge” smoking (8-9). Other commonly associated agents include inhaled drugs or chemicals. Typically this involves crack cocaine or heroin abuse (10-13). In the best of our knowledge this case is the first report linking AEP to inhaled methamphetamines.

The use of inhaled methamphetamines has become a major drug of abuse in the United States and is associated with numerous social and psychological consequences. According to the National Survey on Drug Use and Health over 1.4 million Americans tried methamphetamines during 2006 (15). If the true extent of the problem continues then it is possible that the number of cases of AEP in association may also increase. Since AEP responds dramatically to the appropriate care it is important that it be identified, both on the clinical and laboratory levels. Further study is needed to fully understand the relationship and consequences of AEP with inhaled methamphetamines.

![Image 1: Eosinophilic pneumonitis](https://example.com/image1.png)

![Image 2: Intersitial eosinophil infiltration and COX2 upregulation](https://example.com/image2.png)