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Case Report

A rare discovery of acute eosinophilic pneumonia associated with intensive cannabis use: Case report ☆

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ABSTRACT

This article explores the case of acute eosinophilic pneumonia (AEP) linked to heavy cannabis inhalation, amidst the rising prevalence of cannabis use globally. AEP, characterized by eosinophilic pulmonary infiltration, poses unique challenges due to its unclear pathogenesis. This case study involves a 20-year-old with recent intense cannabis use, presenting with acute chest pain, cough, and dyspnea. Diagnostic evaluation revealed bilateral interstitial syndrome on thoracic imaging and elevated blood eosinophilia. Additional investigation through bronchoalveolar lavage confirmed the diagnosis of AEP. The patient's condition rapidly improved with glucocorticoids, highlighting the significance of prompt treatment. This article underscores the importance of raising awareness among clinicians of the possibility of AEP following cannabis exposure as a diagnosis to consider, as timely diagnosis and intervention are paramount in averting potentially fatal outcomes.

Introduction

The consumption of cannabis has become increasingly widespread in many regions of the world with varied formulations and modes of consumption. While its psychoactive and therapeutic effects are well-documented, the impact on respiratory health is garnering more and more interest. From this perspective, we examine a rare case of acute eosinophilic pneumonia (AEP) attributed to heavy cannabis inhalation.

AEP is a rare clinical entity characterized by eosinophilic pulmonary infiltration, the pathogenic mechanisms of which remain largely unknown. While less common than other types of pneumonia, AEP presents distinctive features that make it worthy of interest and thorough understanding.

Case report

We present the case of a 20-year-old male, computer science student with no notable medical history and minimal

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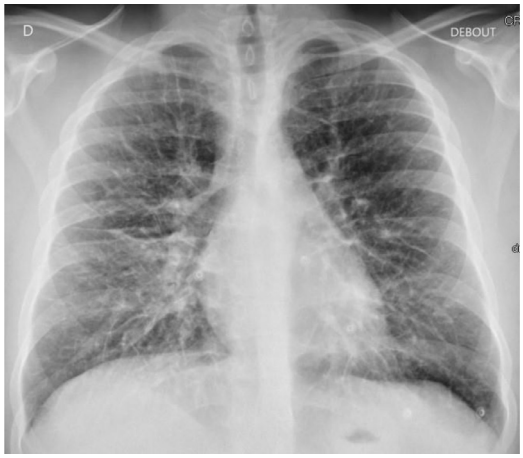


Fig. 1 – Initial chest X-ray showing bilateral diffuse reticular opacities suggestive of an interstitial syndrome.

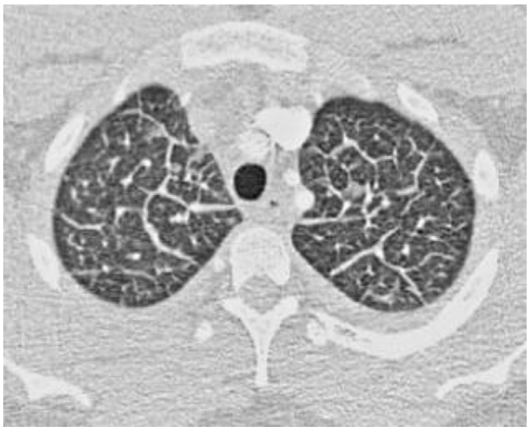


Fig. 2 – Thoracic CT presenting regular septal thickening and ground-glass opacities.

exposure risks, aside from an estimated active smoking habit of 1 pack-year (equivalent to smoking 1 pack per day for 1 year). Prior to hospitalization, he engaged in a 3-day period of heavy cannabis consumption, exceeding 10 joints per day, during social gatherings with friends. Notably, there was no alcohol consumption, nor any history of medication use or exposure to other toxins.

The patient was initially admitted to the emergency department with acute chest pain associated with cough and dyspnea.

Cardiac assessment, including electrocardiography, serum levels of NT pro-BNP and troponin, and transthoracic echocardiography, showed no abnormalities.

Chest X-ray revealed a bilateral interstitial syndrome (Fig. 1). Further evaluation with thoracic computed tomography (Figs. 2-6) showed acute interstitial pneumopathy characterized by regular thickening of septal lines and diffuse ground-glass opacities without air trapping, associated with bilateral moderate pleural effusion, suggestive of acute eosinophilic pneumonia.

The complete blood count (CBC) revealed a swift and significant increase in blood eosinophilia over a span of 3 days,

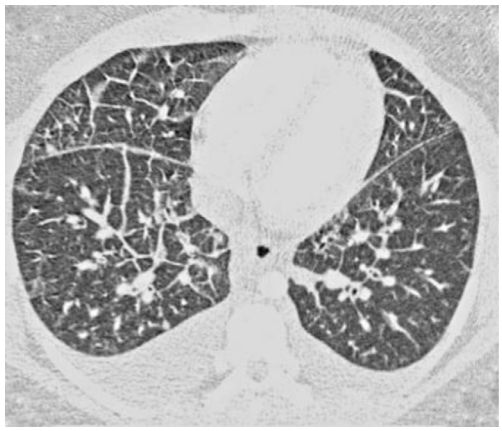


Fig. 3 – Thoracic CT revealing regular septal thickening, ground-glass opacities, and bilateral pleural effusion.

peaking at 5559 elements/mm^3, markedly surpassing the normal threshold of 500 elements/mm^3. This was accompanied by a biological inflammatory syndrome characterized by neutrophilic leukocytosis and an elevated C-reactive protein (CRP) level of 116 mg/L, far exceeding the normal limit of 5 mg/L.

The patient was urgently placed on dual antibiotic therapy due to the hypothesis of intracellular germ pneumonia, and bronchoscopy was indicated due to strong suspicion of AEP.

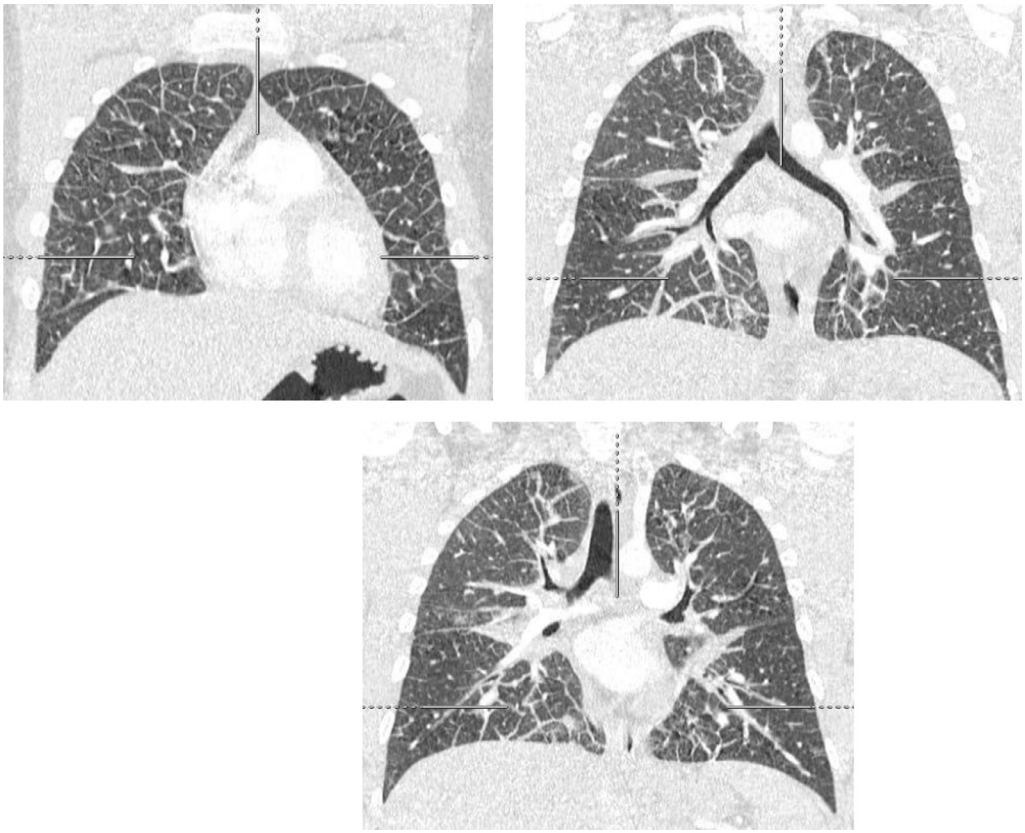
In our case, bronchoscopy showed diffuse inflammatory changes with no other anomalies. Bronchoalveolar lavage (BAL) with bacteriological, mycological, and parasitic study favored eosinophilic leukocytosis of 46% without any pathogens or suspicious elements.

Our comprehensive assessment aimed to definitively exclude differential diagnoses. Parasitic infections were ruled out through negative BAL tests and serological assays for aspergillosis, hydatidosis, trichinosis, toxocariasis, and schistosomiasis, along with a 3-day stool parasite examination yielding negative results. Respiratory viral PCR, including COVID testing, returned negative results. The patient's lack of a history of atopy, rhinitis, or asthma minimized consideration of allergic etiologies. Furthermore, normal IgE levels and a negative autoimmune panel, which included tests for antinuclear antibodies (ANA) and antineutrophil cytoplasmic antibodies (ANCA), ruled out allergic bronchopulmonary aspergillosis (ABPA) and eosinophilic granulomatosis with polyangiitis (EGPA). Absence of hematologic abnormalities such as anemia and thrombocytopenia in the blood count made acute eosinophilic leukemia unlikely.

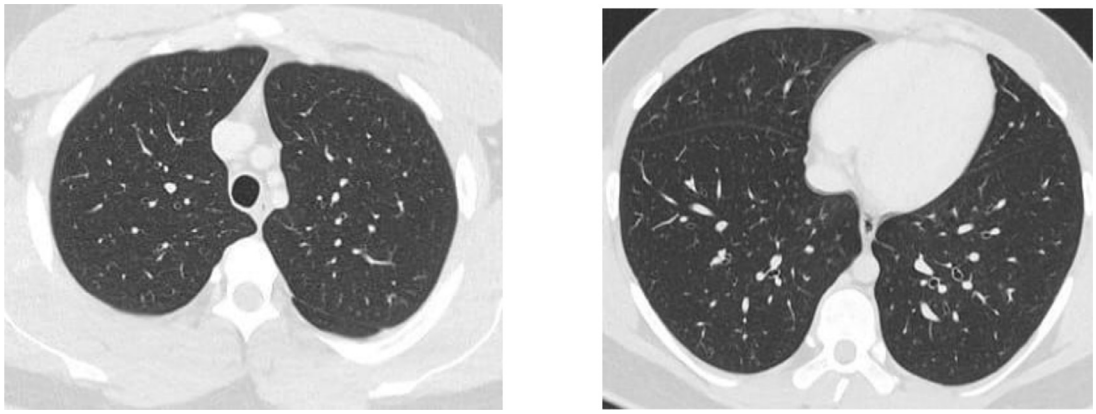
The patient's condition rapidly improved with oral corticosteroid therapy initiated at an initial dose of 60 mg per day, then gradually reduced over 15 days, leading to clinical improvement, radiological lesion reversibility (Figs. 7 and 8), and disappearance of blood eosinophilia.

Discussion

AEP is often associated with parasitic infections, drug reactions, or autoimmune diseases. However, in recent years, sev-



Figs. 4, 5, 6 – Coronal CT scan images showing the distribution of abnormalities.



Figs. 7 and 8 – Resolution of abnormalities on follow-up thoracic CT scan postcorticosteroid treatment after 1 month.

eral cases have been reported in cannabis and psychoactive substance users [1,2].

Although the exact mechanisms remain incompletely understood, with even less clarity surrounding drug-induced cases, research indicates that in individuals with a predisposition, exposure to triggers such as cigarette smoke, drugs, or other offending agents initiates a cascade of immune responses, predominantly characterized by a Th2-polarized immune response [3]. This leads to the production of cytokines that facilitate a substantial recruitment and activation of eosinophils within the lungs. While eosinophils play a central role in AEP, the precise mechanisms through which they cause tissue injury are not fully elucidated. It is hypothesized that eosinophilic inflammation contributes to lung damage through the release of eosinophil granules into alveolar spaces and the interstitium, causing direct injury manifested by protein exudation and the induction of surfactant-associated proteins. Although other immune cells including macrophages, pulmonary neutrophils and lymphocytes are also implicated in the pathogenesis of AEP, the exact pathways through which they induce lung injury are not entirely understood [4].

AEP predominantly affects individuals under 50 years old [5], presenting with acute dyspnea and characteristic radiographic images. There is no consensus on AEP diagnosis, but diagnostic criteria for idiopathic AEP are described and usually used: 1) Abrupt onset of respiratory symptoms (rapidly progressive dyspnea, cough) associated with fever (duration <1 month and especially <7 days); 2) Diffuse bilateral lung infiltrates on imaging; 3) PaO_2 at room air <60 mmHg or PaO_2/FiO_2 <300 mmHg or oxygen saturation at room air <90%; 4) Pulmonary eosinophilia with >25% eosinophils in BAL; 5) Absence of specific lung involvement including eosinophilic granulomatosis with polyangiitis, hypereosinophilic syndrome, and allergic bronchopulmonary aspergillosis [5,6].

In our case, all diagnostic criteria were met. However, blood eosinophilia, usually minimal or absent, rapidly progressed in our patient to a moderately elevated value, a phenomenon also observed in other cases [7].

The radiographic presentation described in our case is consistent with other cases reported in the same context, presenting regular septal thickening, ground-glass opacities, and bilateral pleural effusions without cardiomegaly or air trapping [7–9].

Rapid improvement under corticosteroids is typical, with clinical and radiological improvement visible within 7 days and thoracic imaging normalization within 3 to 4 weeks [4].

The management of AEP depends on its etiology. In the cases where exposure to irritant agents is implicated, the treatment is focused on administration of glucocorticoids and elimination of the causative agent [4]. Currently, there is no established consensus on the optimal dosage and duration of glucocorticoid therapy for AEP. In clinical practice, the initial glucocorticoid dose is generally tailored to the severity of the disease. For hospitalized patients with severe hypoxemic respiratory failure necessitating mechanical ventilation, intravenous methylprednisolone is typically administered at doses ranging from 60 to 125 mg every 6 hours or an equivalent regimen. Once respiratory failure resolves, intravenous glucocorticoid therapy may transition to oral prednisone, administered for a treatment duration of up to 4 weeks. In cases without respiratory failure, oral prednisone is initiated at a dose of 40 to 60 mg per day and is gradually tapered over 2 to 6 weeks, depending on symptom improvement and objective clinical findings [4,10].

In our patient's case, and considering the strong implication of cannabis inhalation as the cause of acute eosinophilic pneumonia (AEP), and to prevent long-term complications, cessation of cannabis exposure is imperative. Patient education on abstaining from the use of illicit substances is our most effective strategy [2–4].

Conclusion

AEP associated with cannabis consumption is a rare but increasingly reported complication in the literature. This case once again highlights the dangers of cannabis and raises important questions regarding its respiratory effects, especially with the emergence of more potent extraction and concentration methods.

Clinicians should be aware of the possibility of AEP following cannabis exposure as a diagnosis to consider in a typically young patient presenting with unexplained respiratory symptoms and suggestive pulmonary involvement on thoracic imaging. Prompt initiation of systemic corticosteroid therapy may be crucial in emergency situations where life prognosis is at stake.

Further research is needed to elucidate the underlying mechanisms of this association and its clinical implications.

Patient consent

Consent for publication of this case was obtained from the patient. This consent ensures that the patient have been fully informed about the nature of the publication and have agreed to the use of the case details.

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