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CASE REPORT
ΕΝΔΙΑΦΕΡΟΥΣΑ ΠΕΡΙΠΤΩΣΗ

A case of acute eosinophilic pneumonia

Acute eosinophilic pneumonia (AEP) is a very rare disease. It was described for the first time in 1989, and so far about very few cases have been described worldwide. We describe a case of acute respiratory failure in a young female patient exhibiting peripheral eosinophilia that was eventually attributed to acute eosinophilic pneumonia. The patient was successfully treated with corticosteroids with good clinical compliance. A possible trigger was the inhalation of mint-flavored cigarette smoke which is a very uncommon cause of AEP.

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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2025, 42(6):842–844

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Περίπτωση οξείας ηωσινοφιλικής πνευμονίας

Περίληψη στο τέλος του άρθρου

Key words

Crazy paving pattern
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Eosinophilic pneumonia
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Acute eosinophilic pneumonia (AEP) is a rare disease characterized by acute respiratory failure and infiltration of the pulmonary parenchyma by eosinophils. Peripheral eosinophilia, although uncommon, may also be present. The clinical picture consists of fever, cough, and the recent appearance of dyspnea.[1] It is treated with glucocorticoids, and in some cases it may require mechanical ventilation. The usual outcome is recovery. The disease has been linked to inhaled agents such as tobacco smoke, desert sand, fire smoke, cocaine use, drugs, fungal and parasitic infections, and more recently SARS-CoV-2 infection. AEP has been reported in Gulf War veterans and firefighters involved in the World Trade Center attack in New York, and after exposure to smoke of flavored cigarettes and vaping.[2–6] Our patient was a smoker of electronic cigarettes, but the trigger may have been the inhalation of flavored cigarette smoke. Only three cases of AEP secondary to flavored cigarette smoking have so far been described in the literature.[4,7]

CASE PRESENTATION

A 26-year-old female patient was referred to the emergency department of our hospital due to fever with concomitant dyspnea that started three days prior to her presentation. She had a history of childhood asthma, she had been infected with SARS-CoV-2 a year earlier, was a user of electronic cigarettes, and prior to the onset of symptoms she reported inhalation of mint-flavored cigarette smoke. She reported no chronic medication regimen and no illicit drug use. Clinically, the patient was tachypneic, with tachycardia and crackles were audible over all pulmonary fields. She was in severe respiratory distress, with no use of accessory muscles. Her oxygen saturation was 95% on a 100% non-rebreather mask. Initial vital signs were: Blood pressure: 125/78 mmHg, heart rate: 120 bpm, temperature at presentation 36.8 °C, and later 38 °C. The chest computed tomography (CT) scan revealed extended converging intermediate pulmonary consolidations and ground glass opacities, a finding described as "crazy paving" (fig. 1). The patient's chest X-ray exhibited diffuse opacities (fig. 2). A noteworthy finding from the laboratory tests was the presence of leukocytosis

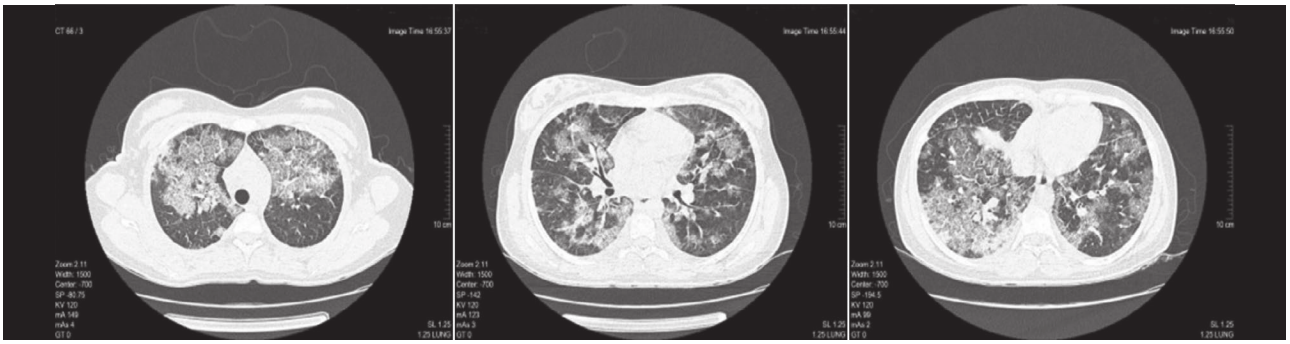

Figure 1. Patient's computed tomography (CT) of chest sections showing a "crazy paving" pattern.

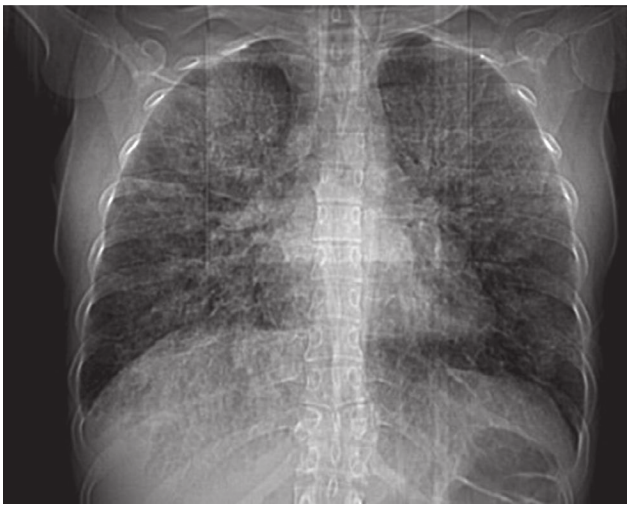

Figure 2. Patient's chest X-ray.

Table 1. Arterial blood gases of the patient.

pH	pO_2	pCO_2	Lactate	HCO_3^-
7.42	73.0 mmHg	36.0 mmHg	1.0 mmol/L	23.4 mEq/L

Table 2. Patient's laboratory findings upon presentation.

Laboratory results	Value	Normal range
White blood cells/µL	25,000	4,000–11,000
Granulocytes (%)	81	35–72
Eosinophils (%)	10.8	0–6
Eosinophils # per µL	2,700	0–600
Procalcitonin (ng/mL)	0.05	<0.05
D-dimers (ng/mL)	1,155	<250
C-reactive protein (mg/L)	162	<5
Erythrocyte sedimentation rate (mm/hour)	37	0–20

with peripheral eosinophilia (tables 1, 2). Urinalysis did not reveal white blood cells or erythrocytes in the urine. The blood cultures obtained were negative. Also, the tests for HBV, HCV, HIV, RSV, FLU-A, FLU-B, SARS-CoV-2 were all negative. The patient initially received a regimen of intravenous moxifloxacin 400 mg once daily, *per os* doxycycline 100 mg twice daily, along with bronchodilators. Based on the findings of peripheral eosinophilia and the chest CT scan results mentioned above, the patient was administered intravenous methylprednisolone 125 mg twice daily with the presumptive diagnosis of AEP. The result was an improved clinical picture and a decrease of the percentage and absolute number of eosinophils in the peripheral blood. This supported our hypothesis for the diagnosis of acute eosinophilic pneumonia. The patient was subsequently transferred to a central hospital where bronchoscopy was performed and the bronco-alveolar lavage (BAL) revealed 2% lymphocytes, 1% granulocytes, 19% alveolar macrophages, and 76% eosinophils. Also, BAL culture was negative for *B. Koch*, *P. carinii*, and other pathogens. Tests for anti-dsDNA, anti-CCP, anti-ENA, c-ANCA, p-ANCA were all negative. The patient was continued on a de-escalating regiment of intravenous prednisolone 40 mg once daily with further improvement and was eventually discharged.

COMMENT

Acute eosinophilic pneumonia is a rare clinical entity, manifested by acute respiratory distress, coughing, and fever of an onset of less than seven days and up to four weeks. Findings from the chest CT scan may include ground glass opacities with reticular pattern, a picture also referred to as "crazy paving" pattern, and possibly pleural fluid.[8] As far as the blood count is concerned, the number of white blood cells is elevated with granulocytosis, but later the number of eosinophils may rise significantly. The diagnostic criteria for acute eosinophilic pneumonia include four parameters; namely, a fever of less than four weeks, hypoxemic respiratory distress with SpO_2 <90% on ambient air, ground glass opacities and converging intermediate pulmonary consolidations from the CT scan, as well as a broncho-alveolar lavage (BAL) with presence of eosinophils >25%.[1] The dif-

ferential diagnosis includes Churg-Strauss polyangiitis (with BAL eosinophilia), and without BAL eosinophilia: Acute respiratory distress syndrome, acute interstitial pneumonia, fulminant cryptogenic pneumonia, diffuse alveolar hemorrhage and granulomatosis with polyangiitis.

The mechanism that triggers eosinophilic pneumonia possibly consists of the activation of an inflammation cascade that involves the action of cytokines such as interleukin-5, interleukin-6, interleukin-7, and tumor necrosis factor, that may trigger the presence of eosinophils in the alveoli.[9] The therapeutic approach consists of respiratory support and glucocorticoids iv or *per os* in milder cases.[10]

Only a small number of acute eosinophilic pneumonia cases have been described. Moreover, we were able to find 98 case reports on PubMed concerning acute eosinophilic pneumonia caused by vaping or cigarette smoking, and only two publications concerning three cases of AEP caused by the inhalation of smoke of a flavored cigarette.[4,7] We have described a case of AEP with possible trigger the inhalation of flavored cigarette smoke on a ground of long-term vaping by the patient. Our case is a good illustration of the successful diagnosis and treatment of this very rare disease. It is also an addition to the three previously known cases of AEP due to inhalation of flavored cigarette smoke.

ΠΕΡΙΛΗΨΗ

Περίπτωση οξείας ηωσινοφιλικής πνευμονίας

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Η οξεία ηωσινοφιλική πνευμονία είναι μια σπάνια νόσος. Περιγράφηκε για πρώτη φορά το 1989 και μέχρι σήμερα έχουν δημοσιευτεί πολύ λίγα περιστατικά παγκοσμίως. Παρουσιάζεται η περίπτωση οξείας αναπνευστικής ανεπάρκειας σε μια ασθενή στην οποία διαπιστώθηκε αρχικά περιφερική ηωσινοφιλία και στη συνέχεια τέθηκε η διάγνωση της οξείας ηωσινοφιλικής πνευμονίας. Η ασθενής αντιμετωπίστηκε με ενδοφλέβια χορήγηση κορτικοστεροειδών, με καλή κλινική ανταπόκριση. Πιθανός πυροδοτικός παράγοντας ήταν η εισπνοή από την ασθενή καπνού αρωματικού τσιγάρου, που συνιστά μια σπάνια αιτία οξείας ηωσινοφιλικής πνευμονίας.

Λέξεις ευρετηρίου: Ηλεκτρονικό τσιγάρο, Ηωσινοφιλική πνευμονία, Μοτίβο άναρχης πλακόστρωσης, Περιφερική ηωσινοφιλία, Πρεδνιζολόνη

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