Acute Eosinophilic Pneumonia with Eosinophilic Leukemoid Reaction: A Rare Hematologic Presentation

Sourya Acharya · Samarth Shukla ·
Pradyumna Gadewar · Manoj Gupta ·
S. N. Mahajan

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Sir,

A 26-year-old man presented to us with chief complaints of fever, cough, and dyspnea since 15 days. There was no history of hemoptysis, chest pain, orthopnea, palpitations, or edema feet. A more detailed history was negative for environmental or medication exposure. Past history was insignificant for any prolonged illness. There was no history of any addiction.

On examination, pulse was 100/min, regular. Respiratory rate was 18/min, and the temperature was 38.6 °C. JVP was not raised. Auscultation of the lungs revealed bilateral, diffuse rales. Cardiovascular system examination was normal.

Investigations revealed, Hb of 11 g %, Total leucocyte count (TLC) was 52,000, DLC revealed polymorphs of 15 %, eosinophils 78 %, lymphocytes 6 %, monocyte 1 % (Fig. 1). Peripheral smear was devoid of any blasts. Absolute eosinophil count was 38,000/mm³ (normal 52–250 mm³). Platelet count was 250,000/mm³. The bone marrow aspiration revealed eosinophilic proliferation without any blasts (Fig. 2). The blood biochemistry was normal. CXR revealed bilateral haziness. HRCT of thorax revealed bilateral ground glass opacities (GGOs) (Figs. 3, 4). Two dimensional (2D) echo was normal. ABG revealed partial pressure of oxygen, 68 mmHg; partial pressure of carbon dioxide, 29 mmHg; and pH, 7.50. SpO₂ was 84 % while patient was breathing ambient air. Cultures of

sputum, blood, and urine were negative for bacteria, mycobacteria, fungi. ESR was 24 mm in first hour. Peripheral smear examination for parasites and microfilaria was negative.

Patient was treated with empirical antibiotics, NIPPV, oral corticosteroids (Tab. prednisolone 60 mg/day). Patient's breathlessness decreased dramatically on the third day. NIPPV was discontinued. He was maintaining O₂ saturation of 98 % in ambient air. ABG became normal. A bronchoalveolar lavage was done, which revealed, total cell count of 132×10^3 with 22 % eosinophils. In the first week after treatment TLC came down to 15,000/mm³, with 25 % eosinophils. AEC was 4,000/mm³. Repeat HRCT showed partial clearing of the GGOs. Fever, cough and dyspnea subsided. A diagnosis of acute eosinophilic pneumonia with eosinophilic leukemoid reaction was made. By the end of second week, TLC was 10,000/mm³. DLC revealed 12 % eosinophils. AEC was 1,100/mm³. Patient was discharged with a tapering dose of prednisolone, and was advised follow up after two weeks.

Discussion

Acute eosinophilic pneumonia (AEP) is a recently described syndrome characterized by a febrile illness, diffuse infiltrates on chest radiograph, and pulmonary eosinophilia [1]. Patients with AEP may also present with respiratory failure requiring mechanical ventilation. Our patient also presented with type 1 respiratory failure which required NIPPV.

A number of conditions constitute peripheral blood eosinophilia with pulmonary infiltrates [2]. An exhaustive diagnostic workup is required to pinpoint towards a particular etiology, but subtle clues are helpful in coming to a

S. Acharya (✉) · P. Gadewar · M. Gupta · S. N. Mahajan
Department of Medicine, JNMC, DMIMS University, Sawangi
(Meghe), Wardha 442004, Maharashtra, India
e-mail: souryaacharya@yahoo.co.in

S. Shukla
Department of Pathology, JNMC, DMIMS University, Sawangi
(Meghe), Wardha 442004, Maharashtra, India

Fig. 1 Peripheral smear (Leishman stained $\times 10$ view) showing eosinophilia

Fig. 2 Bone marrow slide (Leishman stained $\times 40$ view) showing eosinophilic predominance

conclusive diagnosis. Acute eosinophilic pneumonia in our case was a diagnosis of exclusion. Analysing the clinical, laboratory and imaging parameters we could rule out some of the common differential possibilities in our case (table 1), though all the other causes require extensive investigations for a confident diagnosis and cannot be excluded completely.

Ground-glass opacification is a hazy area of increased attenuation in the lung with preserved bronchial and vascular markings. Ground-glass opacification can be seen in many of the eosinophilic pneumonias, like simple pulmonary eosinophilia (SPE), idiopathic hypereosinophilic syndrome (IHS), AEP and in eosinophilic drug reactions. In AEP the usual pattern in HRCT thorax is bilateral patchy areas of GGO with septal thickening like in our case. Histopathologically, lung biopsies in patients with AEP reveal both acute and organizing diffuse alveolar damage

Fig. 3 Coronal CT image shows bilateral, diffuse and patchy ground glass opacity (arrows)

Fig. 4 Axial CT image showing bilateral ground glass opacity

with eosinophils filling both the alveolar and interstitial spaces. Peripheral eosinophilia may be noted in AEP; however, it is often absent at the time of presentation, unlike in our case where the patient presented with eosinophilic leukemoid reaction. Corticosteroids remain the mainstay of therapy for AEP, and relapses have not been reported. Dramatic response to steroid therapy is the hallmark of AEP as in our case.

Eosinophilic leukemoid reactions are often seen in parasitic, allergic, neoplastic, collagen vascular diseases

Table 1 Characteristic features of some lung disorders associated with peripheral blood eosinophilia

Chronic eosinophilic pneumonia	Chronic eosinophilic pneumonia presents with significant systemic symptoms including fever, chills, night sweats, cough, anorexia, and weight loss of several weeks–months duration. The chest X-ray classically shows peripheral infiltrates. Onset and duration in our case was acute less than 2 weeks
Allergic bronchopulmonary aspergillosis	Absence of history of Bronchial asthma, brownish plugs in sputum. HRCT characteristically shows Central bronchiectasis. Our patient didn't have these features
Allergic granulomatosis of Churg and Strauss	No evidence of multisystem involvement and vasculitis present in this case
Hypereosinophilic syndrome	Though our patient had several features suggestive of IHS, there was no evidence of multiple organ involvement inform of CNS (Strokes, encephalitis, peripheral neuropathy), Cardiovascular (CCF, evidence of myocardial fibrosis), thromboembolic phenomena. Dramatic response to steroids rules out IHS
Loeffler's syndrome	Acute eosinophilic pneumonia of unknown cause characterized by migrating pulmonary infiltrates and minimal clinical manifestations, usually by parasites and drugs. No history of intestinal parasitosis, no history of recent travel was present in this case. Peripheral smear was negative for microfilaria. Hetrazan challenge test was negative. No history of any drug intake known to cause eosinophilia with pulmonary infiltrates like nitrofurantoin, sulfonamides, penicillin, chlorpropamide, thiazides, tricyclic antidepressants, hydralazine, gold salts, isoniazid, or indomethacin was present
Acute eosinophilic pneumonia	Acute eosinophilic pneumonia is an idiopathic acute febrile illness of <7 days' duration with severe hypoxemia, pulmonary infiltrates, and no history of asthma, and there is usually a prompt response to steroid therapy. Our case fulfilled these features

and drug reactions [3]. A leukemoid reaction is usually differentiated from leukaemia by the absence of premature blasts suggesting it to be benign proliferations. There is no absolute criteria for differentiation between leucocytosis and leukemoid reactions. We considered this patient as having eosinophilic leukemoid reaction primarily because of leucocytosis, very high eosinophil count (absolute eosinophil count $3,000/\text{mm}^3$) and bone marrow aspiration showing eosinophilic proliferation. Leukemoid reactions are always a secondary phenomenon and as the cause is removed or cured, the reactions subside as in our patient.

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