

A curious case of idiopathic eosinophilic pleural effusion post swimming pool diving

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Abstract

Eosinophilic pleural effusion is defined as pleural effusion with >10% eosinophil count. Even though the mechanism underlying eosinophilic pleural effusion is poorly understood, it is considered to be caused by pleural fluid sequestration of bone marrow-derived eosinophils. Even though there is a wide spectrum of diseases recognized to be associated with eosinophilic pleural effusion, including pleural irritation, trauma (hemothorax, pneumothorax, thoracic surgery), malignancy, parasitic infection, drug/toxin-induced and pulmonary embolism; almost 25% of cases remain idiopathic. We herein report a curious case of

eosinophilic pleural effusion, which has developed post blunt trauma to the chest, sustained after diving into a swimming pool. The patient was extensively evaluated for possible etiologies but was found negative for any underlying known pathology that could lead to eosinophilic pleural effusion.

Introduction

Eosinophilic pleural effusion is defined as pleural effusion with >10% eosinophil count. Even though the mechanism underlying eosinophilic pleural effusion is poorly understood, it is considered to be caused by pleural fluid sequestration of bone marrow-derived eosinophils.¹ Even though there is a wide spectrum of diseases recognized to be associated with eosinophilic pleural effusion, including pleural irritation, trauma (hemothorax, pneumothorax, thoracic surgery), malignancy, parasitic infection, drug/toxin-induced and pulmonary embolism; almost 25% of cases remain idiopathic.²

Case Report

We herein report a curious case of eosinophilic pleural effusion, which has developed post blunt trauma to the chest, sustained after diving into a swimming pool. The patient was extensively evaluated for possible etiologies but was found negative for any underlying known pathology that could lead to eosinophilic pleural effusion.

29-year-old young male, resident of Jammu, vegetarian by diet and with no known comorbidities, developed right-sided chest pain after a 6-meter dive into a swimming pool. The pain was acute in onset, sharp pricking type, localized to the right infra-axillary region, non-radiating, aggravated on deep breathing, and relieved on lying in the right lateral position. He was managed conservatively at a peripheral hospital with oral analgesics. The patient had symptomatic improvement in chest pain with medical management but developed breathlessness on exertion (mMRC II) over the following week, along with a non-productive cough. On evaluation at the peripheral hospital, his chest radiography revealed homogenous opacity in the right lower zone, blunting the costo-phrenic angle. Chest ultrasonography demonstrated effusion in the right pleural cavity with no internal loculations. Diagnostic tap of the pleural effusion resulted in yellowish serous fluid which on evaluation was found to be exudative (pleural fluid LDH: 698 IU/L, serum LDH: 316, pleural fluid protein: 5.1 g/dL, serum protein: 6.9 g/dL, pleural fluid glucose: 98 mg/dL, serum glucose: 84 mg/dL) with more than 10% eosinophils. In view of exudative eosinophilic pleural effusion, he was referred to our center for further evaluation. On reporting to our center, the patient was asymptomatic. He was afebrile with a heart rate of 78/min, blood pressure of 114/80 mm of Hg, respiratory rate of 18/min, and was maintaining 98% saturation on room air. Auscultation revealed absent air entry in the right infra-axillary and infrascapular region. On evaluation, he had a hemoglobin of 13.8 g/dL, a total leuko-

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cyte count: 8000/cumm (neutrophil: 60.8%, lymphocyte: 24.9%, eosinophil: 7.7%, monocyte: 5.9%), platelets: 394000/cumm, urea: 29 mg/dL, creatinine: 0.83 mg/dl), alanine transaminase: 26 U/L, aspartate transaminase: 23 U/L, alkaline phosphatase: 114 U/L, D dimer: 1650 ng/mL. An Electrocardiogram (ECG) demonstrated normal sinus rhythm. Chest radiography taken at our center showed persistence of right-sided pleural effusion with no evidence of hydro-pneumothorax (Figure 1a). Repeat pleural tap done at this center yielded free-flowing, yellowish serous fluid (Figure 2), which was exudative on evaluation (pleural fluid LDH: 724 IU/L, serum LDH: 326 IU/L, pleural fluid protein: 5.3 g/dL, serum

protein: 7.1 g/dL, pleural fluid glucose: 105 mg/dL, serum glucose: 88 mg/dL, pleural fluid adenosine deaminase: 19 IU/L). Pleural fluid cell block evaluation revealed greater than 90% eosinophils with no malignant cells. Gram stain and Ziehl-Neelson stain of pleural fluid were negative, and there was no growth in aerobic and anaerobic cultures. In view of eosinophilic pleural effusion, he was worked up for malignancy. Positron Emission Tomography (PET) revealed non-Fluorodeoxyglucose (FDG) avid right-sided pleural effusion (Figure 3). He was worked up for pulmonary embolism in view of eosinophilic pleural effusion with a raised D-dimer level. Computed tomographic pulmonary angiography was negative for

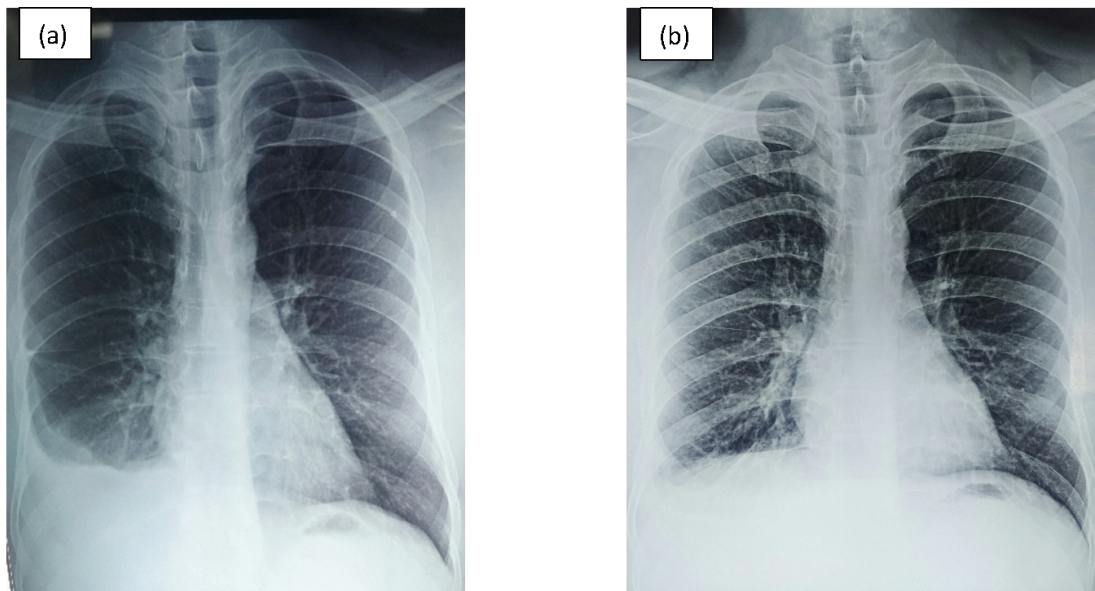


Figure 1. a) Chest radiography Posteroanterior (PA) view showing a homogenous opacity in the right lower zone blunting the right costophrenic angle. b) Follow-up chest radiography of the patient was taken after 4 weeks, and it showed significant resolution of pleural effusion.



Figure 2. Yellowish serous pleural fluid on diagnostic pleurocentesis.

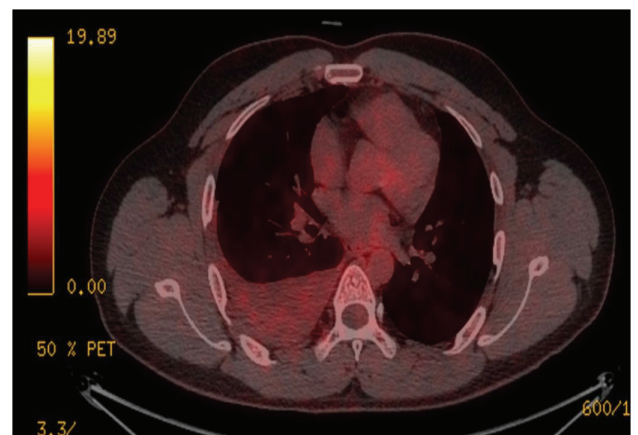


Figure 3. Positron Emission Tomography (PET) of the patient demonstrating non-Fluorodeoxyglucose (FDG) avid right-sided pleural effusion.

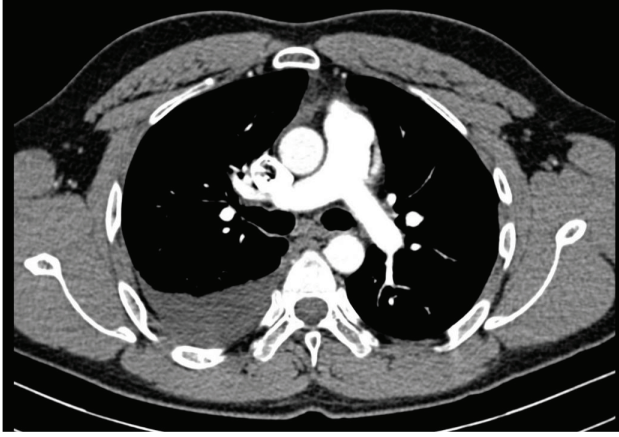


Figure 4. Computed tomographic pulmonary angiography shows no evidence of pulmonary thromboembolism.

pulmonary thromboembolism (Figure 4). He was diagnosed to have idiopathic eosinophilic pleural effusion post blunt trauma to the chest sustained during diving and was managed conservatively. On follow-up after four weeks, the patient had symptomatic resolution with significant improvement in chest radiography (Figure 1b). Eosinophilic pleural effusion is a rare pathology accounting for only 10% of diagnosed exudative pleural effusions.³ A meta-analysis of 687 cases of eosinophilic pleural effusion revealed 25% of cases of eosinophilic pleural effusion to be idiopathic, which is second only to malignancy, which comprised 26% of cases.² A relatively huge portion of eosinophilic pleural effusion being idiopathic reflects the current void in our understanding of this disease.

Blunt trauma to the chest wall is an established etiology for eosinophilic pleural effusion, but the underlying pathology is hemothorax or pneumothorax. Our patient had no evidence of hemothorax even on repeated thoracentesis, and there was no hydro-pneumothorax on chest imaging. The patient is a vegetarian who has never consumed crabs or crawfish, which could transmit paragonimiasis. He was afebrile, and there was no evidence of pleural inflammation on the PET scan. He was clinically and radiologically negative for malignancy. Even though he had high D-dimer levels, he had no clinical, ECG, or radiographic evidence suggestive of pulmonary embolism.

The cause of his eosinophilic pleural effusion remains idiopathic. This case demonstrates the current lacunae in the medical literature regarding the etiologies of eosinophilic pleural effusion and the possibility of eosinophilic pleural effusions developing post-blunt trauma to the chest without an obvious hemothorax or pneumothorax.

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