Case Report

Eosinophilic pleural effusion and peripheral eosinophilia—an uncommon complication of thoracoscopic parathyroidectomy

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Introduction

Eosinophilic pleural effusion was first described by Harmsen in 1894 [1], defined as a pleural effusion with the presence of 10% or more eosinophils [2]. Adelman et al. [1] analysed 343 cases reported since 1959 until 1983 and summarized that this disorder was caused by air in the pleural space in 28% of all the cases. Pneumothorax, repeated thoracocentesis, thoracic surgery, and trauma were the causes of air in the pleural space, and Chapman et al. [3] considered that intrapleural erythrocytes could be a cause of eosinophilic pleural effusion. Thirty-five per cent of the cases were considered to be idiopathic [1]. The reported contribution of eosinophils in the pleural effusion was 4–98% [2,4,5], or up to 16 125/μl [2]. Prognosis of the disease has been reported to be benign in most of the cases, although malignancies including lung carcinoma, metastatic cancer, or mesothelioma have also been documented in a minority of patients [1,2,5]. Peripheral eosinophilia is associated in some but not all the patients.

Here we report a case with eosinophilic pleural effusion associated with peripheral eosinophilia following thoracoscopic parathyroidectomy.

Case

A 64-year-old male with a history of end-stage renal failure on maintenance haemodialysis was admitted for evaluation of secondary hyperparathyroidism on 14 August 1997. Twenty-five years before admission, he had undergone left nephrectomy for renal stone. He was started on regular haemodialysis 15 years prior to admission. Seven years before admission, he was diagnosed to have secondary hyperparathyroidism and underwent total parathyroidectomy with autotransplantation of the parathyroid tissue into the forearm muscle. One year before admission, the autotransplanted parathyroid tissue was removed for recurrence of hyperparathyroidism. However, parathyroid hormone (HS-PTH) concentration remained markedly elevated to 135 000 pg/ml (normal, 150–500) even after the second surgery. Magnetic resonance imaging revealed a golf-ball sized (35 × 20 mm) ectopic mediastinal parathyroid gland with a central necrosis, located at the anterior aspect of the aortic arch.

The patient was admitted to the First Department of Surgery where thoracoscopic resection of the mediastinal mass was performed on 3 September 1997. On the third post-operative day, severe left-sided pleural effusion was found, and the drainage yielded a daily production of 300-ml serosanguinous pleural fluid. Thoracic drainage tube was removed 9 days after the surgery. The patient was transferred to the Medical ward on 17 October.

On transfer, the patient was alert with blood pressure of 100/56 mmHg, with respiration rate of 24/min, and pulse rate of 66/min. Moderate anaemia was noted. There was a subcutaneous swelling and tenderness of the drainage wound at the left thoracic wall. There was no lymphadenopathy. Respiratory sound was decreased in the left lower lung field with percussive dullness. The abdomen was unremarkable. Neurological examinations were negative.

Laboratory data revealed white blood cell of 7500/μl (segmented neutrophils 19%, lymphocytes 12%, monocytes 5%, eosinophils 61%, basophils 3% respectively), red blood cell 2.66 × 1012/μl, haemoglobin 8.0 g/dl, platelets 121 × 109/μl, total protein 5.5 g/dl (albumin 54.5%, γ-globulin 25.5%), blood urea nitrogen 19 mg/dl, serum creatinine 6.2 mg/dl, uric acid 5.1 mg/dl, total bilirubin 0.4 mg/dl, calcium 8.2 mg/dl, phosphate 2.5 mg/dl, and alkaline phosphatase 1008 IU/l. HS-PTH was 3100 pg/ml.
been continued three times a week. Thoracocentesis on 23 September revealed exudative pleural effusion with 80% eosinophils. To exclude allergy as a cause, all the medications were discontinued except calcitriol. Materials sterilized with ethylene oxide were exchanged for autoclaved materials. Repeated stool examinations for parasitic ova were negative. Cultures of the pleural effusion yielded no bacterial growth. Bone marrow aspiration showed normocellular marrow with 47.6% eosinophils but no atypical cells. Intrathoracic oozing continued and multiple blood transfusions were required. Peripheral eosinophils reached 25,092/μl (84% of the total leukocytes) on 4 October. On 11 October, sudden hypotension developed due to massive intrathoracic haemorrhage. Thoracoscopic haemostasis of the bleeding site at the parietal pleura was performed on October 14. After the procedure, a parallel decline of peripheral as well as pleural eosinophils was observed, together with improvement in general malaise and pleural effusion. The postoperative course was uneventful. The pleural effusion finally became minimal and the blood eosinophils decreased to 300/μl on 8 December 1997. The patient’s clinical course is depicted in Figure 2.

Discussion

This patient with pleural and peripheral eosinophilia did not meet the criteria of a hypereosinophilic syndrome, i.e. blood eosinophils of at least 1500/μl for...
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more than 6 months [6]. Other common disorders causing eosinophilia, such as parasitic disease, drug allergy, or haematological malignancies, were excluded by appropriate investigations. The final diagnosis was eosinophilic pleural effusion, because eosinophils in the pleural effusion exceeded 10% after thoracic surgery. This is the first record of eosinophilic pleural effusion developing after thoracoscopic parathyroidectomy.

Adelman et al. [1] summarized 343 cases of pleural fluid eosinophilia between 1959 and 1983. They reported that 28% of the patients were associated with air in the pleural cavity. In the present case eosinophilic pleural effusion developed after thoracoscopic surgery and insertion of a thoracic drainage tube. Furthermore, repeated thoracocentesis as well as re-insertion of thoracic tube could have exacerbated eosinophilic pleural effusion.

The mechanisms of how pleural air and/or blood causes eosinophilic pleural effusion remain unclear. Thoracic trauma with erythrocytes in the pleural space is known to be a cause of eosinophilic pleural effusion. Chapman and Reynolds [3] reported that an experimental intraperitoneal infusion of erythrocytes caused severe increase in peritoneal eosinophil count in mice. Eosinophilic chemotactic factors such as leukotrienes or immune complexes have been considered to be implicated in the development of eosinophilic pleural effusion [1,5]. Intraperitoneal administration of interleukin (IL)-2 has been reported to induce marked pleural eosinophilia and moderate peripheral eosinophilia in patients either with lung cancer or malignant mesothelioma [7]. These effects were accompanied by enhanced eosinophil colony-stimulating factor activity in the pleural fluid, which was considered to be mediated by IL-5, IL-3, and granulocyte/macrophage colony-stimulating factor. The authors also found that IL-2 per se locally induced chemotactic factors for eosinophils [7]. Thus, various kinds of interleukins may be involved in the pathogenesis of eosinophilic pleural effusion.

Our case showed extraordinarily elevated blood eosinophils with the highest count of 25092/μl. Although Adelman et al. [1] reported that ‘striking’ blood eosinophilia was uncommon in patients with eosinophilic pleural effusion, other reports indicated that peripheral eosinophilia was found in 25–75% of the cases, averaging 52% [2,4,5].

Treatment with corticosteroids for eosinophilic pleural effusion has been successful in some cases [8]. Oral prednisone of 30 mg is reported to have improved chest pain, the amount of pleural effusion, and peripheral eosinophilia in a 56-year-old male with idiopathic eosinophilic pleural effusion [8]. On the other hand, spontaneous remissions have been observed in most of the reported cases. In our patient, corticosteroids were not used because of the high risk of infection in a compromised host. Maltais et al.[9] emphasized that normalization of blood eosinophilia was observed only when the pleural effusion completely cleared and that persistent peripheral eosinophilia suggested non-resolution of the pleural effusion. In our case, spontaneous regression of the pleural effusion was seen promptly after the haemostasis, and was in parallel with the improvement in blood eosinophils.

References


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