Case Reports

Intrapleural corticosteroid injection therapy for post-traumatic eosinophilic pleural effusion

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Introduction

Eosinophilic pleural effusion can be induced by a variety of causes: pneumonia, fungi, tuberculosis, hypersensitivity, malignant disease, pulmonary infarction, collagen vascular disease, etc. (1). Although post-traumatic pleural effusion was reported as a cause in 16–25% of cases (1,2), its precise aetiology and treatment remain unclear. This report describes a patient with post-traumatic eosinophilic pleural effusion which improved dramatically with a single intrapleural infusion of corticosteroid.

Case Report

The authors were unsuccessful in their attempt to insert a subclavian catheter into an 80-year-old man, hospitalized for cerebral infarction and receiving total parenteral nutrition. He had no history of allergy, and had received only heparin, an H₂-blocker and total parenteral nutrition. No foreign substances were injected or leaked into the pleural space during this procedure (e.g. anti-microbial agent or parenteral nutrition). Examination revealed an euphonic man with a resting blood pressure of 150/72 mmHg. There were neither cardiac murmurs nor carotid bruits. There was moderate left hemiparesis of the face, arm and leg. Hyper-reflexia with a positive Babinski sign was also present on the left, and speech was dysarthric. Physical examination was otherwise normal. Laboratory studies revealed: white cell count 9700 ml⁻¹ with 0% eosinophils; erythrocyte sedimentation rate 34 mm h⁻¹; C-reactive protein 0.2 mg dl⁻¹; aspartate aminotransferase 23 IU ml⁻¹; serum creatinine 1.2 mg dl⁻¹; immunoglobulin E (IgE) 123 IU ml⁻¹. Chest X-ray film was normal on admission and immediately after the attempted subclavian vein puncture. Marked left-sided pleural effusion, however, was seen 10 days later on chest film. Computed tomographic (CT) scan of the chest also showed left-sided massive pleural effusion with trivial pneumothorax [Plate 1(a)]. Thoracentesis yielded 50 ml of turbid yellow fluid. Gram-stained specimens of this fluid revealed 3300 leukocytes ml⁻¹, with 90% eosinophils and 2% lymphocytes. Numerous cultures and subsequent smears from pleural fluid, sputum and gastric washings were negative for fungi, tubercle bacilli, pyogens and malignant cells. Fluid analysis revealed protein concentration 3.5 g dl⁻¹ and glucose 60 mg dl⁻¹ and was negative for rheumatoid factor. Needle biopsied specimens revealed thickened pleura, consistent with non-specific chronic inflammation. Subsequent laboratory findings showed peripheral blood white cell count 9500 µl⁻¹, with 10% eosinophils and IgE 148 IU/ml. Serological tests for fungi, parasites and viruses were negative. Stool examinations were negative for ova and parasites.

The clinical course of this case is shown in Fig. 1. Drainages of the pleural effusion were performed repeatedly over a 2-month period. Although pleural eosinophilia decreased from 90% to 6%, no improvement was seen in the volume of pleural effusion and in peripheral blood eosinophilia. Therefore, a single dose of 24 mg dexamethasone was injected directly into the pleural space. The chest X-ray taken 7 days later showed a marked decrease in pleural effusion, confirmed by CT scan [Plate 1(b)]. Laboratory findings revealed white cell count 7870 µl⁻¹ with 0% eosinophils and IgE 114 IU/ml. No recurrence was reported over 9 months of follow-up.
Plate 1  (a) Computed tomographic (CT) scan of thorax showing massive pleural effusion and mild pneumothorax. (b) Following CT scan of thorax showing marked decrease in pleural effusion.

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Fig. 1  Clinical course. Eosinophilic pleural effusion improved dramatically after a single direct intrapleural infusion of dexamethasone. ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; WBC, white blood cell count; Eosino, eosinophils.

Discussion

Eosinophilic pleural effusion is relatively uncommon and an interesting phenomenon, as defined by the presence of 10% (or more) eosinophils among the cells in the pleural fluid, exclusive of erythrocytes (1). The etiology is diverse. Cambell and Webb summarized the various etiologies in 101 cases as
follows: idiopathic, 28; infection (pneumonia, tuberculosis, fungal, empyema), 24; trauma (pneumothorax, rib fracture, thoracotomy), 16; hypersensitivity (asthma, vascular allergy, dermatitis, transitory pulmonary infiltration), 14; malignancy, 6; pulmonary infarction, 5; heart failure, 4; rheumatoid arthritis, 2; cirrhosis, 1; sarcoidosis, 1 (1). Although the exact mechanism for eosinophilic pleural effusion is unclear, it is suggested that injection of crythrocytes into the peritoneal cavity of mice produces an effusion with a high eosinophil count and persistent peripheral eosinophilia (3). This study suggests that a foreign protein or its split products may be a stimulating factor (4). Some investigators indicate that eosinophilic chemotactic factors are associated with complex immune reactions, such as lymphokines, histamine, anaphylactic factors and tumour-associated eosinophilic chemotactic factors (5,6).

The diagnosis of post-traumatic eosinophilic pleural effusion in the present case was made based on his clinical course, cytological findings of the pleural effusion, and absence of evidence for other aetiologies including infection. Despite repeated drainage, an atypical massive pleural effusion with peripheral eosinophilia persisted for 2 months. Bando et al. and Beekman et al. previously reported similar cases with peripheral blood eosinophilia and a high level of serum IgE. They speculated that an immunologic reaction could be one of the aetiologies of persistent eosinophilic pleural effusion (5,7).

Repeated drainage of eosinophilic pleural effusion is worthwhile (8), because this condition has a good prognosis (6). Some problems, however, remain in cases with prolonged eosinophilic pleural effusion. Recent investigators have reported the clinical efficacy of systemic corticosteroid administration, and have proposed that an immune reaction may be responsible, at least in cases with peripheral eosinophilia (9). Peripheral eosinophilia and increased serum IgE observed in the present case suggested that the immunologic reaction may induce and/or prolong the eosinophilic pleural effusion. Therefore, the authors considered that it might be useful to administer corticosteroid directly into the pleural space.

Although the precise mechanism of corticosteroid therapy for eosinophilic pleural effusion is unclear, it is well known that corticosteroid therapy prevents the entry of eosinophils into the site of inflammation (10). Blackwell et al. have reported that both exudate volume and leukocyte number in pleural fluid are reduced significantly by local injection of dexamethasone in carrageenin-induced pleuritis in rat. They have also shown that both the anti-phospholipase and anti-inflammatory activities of the pleural proteins are increased by steroids (11). The significant decrease in IgE and the decrease in peripheral blood eosinophils in the present case may support this hypothesis.

The reported two cases with post-traumatic pleural effusion treated successfully with intrapleural steroids were not complicated with pneumothorax (7,12). The present case suggests that intrapleural corticosteroid injection therapy may be available not only for post-traumatic eosinophilic pleural effusion, but also for eosinophilic pleural effusion following pneumothorax. Further studies are required to determine the optimal dose, indication and mechanisms for its clinical efficacy.

References