

Magnetic resonance imaging findings of eosinophilic pleural effusion: A case report

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Abstract. We report a case of idiopathic eosinophilic pleural effusion, the diagnosis of which was troublesome due to a considerable difference in properties between right and left pleural effusions. A 58-year-old woman was admitted with a 4-week history of right-sided chest pain. Bilateral pleural effusions were detected on computed tomography. EPE was not identified by initial aspiration from left-sided pleural effusion with scant eosinophils, but was diagnosed by secondary aspiration from right-sided pleural effusion rich in eosinophils. We searched for a means to distinguish between bilateral pleural effusions. As a result, diffusion-weighted imaging was found to distinguish right- from left-sided effusion. The patient was subsequently treated successfully with corticosteroid treatment.

Key words: Magnetic resonance imaging, diffusion-weighted imaging, eosinophilic pleural effusion

1. Introduction

Diffusion-weighted imaging (DWI) is a functional magnetic resonance imaging (MRI) method that depicts differences in the diffusion of molecules, typically water, in the tissues. The diffusion of water molecules can be quantified by a parameter known as the apparent diffusion coefficient (ADC), which is determined using DWI with two different b factors (1,2). The use of DWI has been limited to the central nervous system, although its range of applications has been increasing (3-5). In particular, it has been reported that DWI is capable of differentiating transudative effusion from exudative effusion (1,2). We report herein a case of idiopathic eosinophilic pleural effusion (EPE), the diagnosis of which was troublesome due to a considerable difference in properties between right and left pleural effusions. Only DWI findings such as ADC map and ADC were able to distinguish right from left pleural effusion.

2. Case report

A 58-year-old woman was admitted to our hospital with a 4-week history of right-sided chest pain. She was a non-smoker, had not been taking any medication, and had no history of allergic or pulmonary diseases. Physical examination revealed diminished breath sounds with dullness to percussion at bilateral lung bases. Neither articular swelling nor skin rash suggestive of collagen disease or allergic disease was present. Laboratory findings were as follows: white blood cell count, 5,560/ μ l (neutrophils, 57.0%; eosinophils, 9.0%; monocytes, 7.2%; lymphocytes, 25.2%); red blood cell count, 375 $\times 10^4$ / mm^3 ; hemoglobin, 11.7 g/dl; platelets, 13.7 $\times 10^4$ / mm^3 ; total protein, 5.9 g/dl; albumin, 3.2 g/dl; and C-reactive protein, 0.25 mg/dl. Serological tests showed the following: immunoglobulin (Ig)G, 947 mg/dl; IgM, 55 g/dl; IgA, 226 mg/dl; C₃, 120 mg/dl; and C₄, 35 mg/dl. Immune complex (IC) was not detected by C_{1q}-binding assay. Total serum IgE level was 134 IU/ml, and IgE specific to multiple inhaled or ingested allergens was not detected by the CAP-RAST system. Neither antinuclear antibody nor rheumatoid factor was detected. Negative results were obtained for myeloperoxidase and proteinase-3 antineutrophil cytoplasmic antibodies. Quantiferon test also yielded negative results. Urinalysis revealed neither proteinuria

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nor hematuria. Chest roentgenography confirmed the presence of bilateral pleural effusions. Chest computed tomography (CT) revealed bilateral pleural effusions without pulmonary infiltration shadows. Abdominal CT revealed neither enlarged lymph nodes nor solid tumors. Right chest pain was severe; therefore, aspiration from the left-sided pleural effusion was carried out. Left pleural effusion was translucent, showing: total protein (TP), 3.9 g/dl; lactate dehydrogenase (LDH), 65 U/l; sugar, 123 mg/dl; adenosine deaminase (ADA) level, normal; and no IC. Pleural effusion was thus considered exudative. Smear tests for *Mycobacterium tuberculosis* and bacterial culture yielded negative results. No malignant cells could be detected in the pleural effusion, but mesothelial cells, histiocytes, erythrocytes and eosinophils were scantily present. At this stage, we were unable to reach a diagnosis. Aspiration from right-sided pleural effusion was therefore carried out. Right pleural effusion was also translucent, showing: TP, 4.2 g/dl; LDH, 60 U/l; sugar, 122 mg/dl; ADA level, normal; and no IC. Pleural effusion was thus again considered exudative. Smear tests for *Mycobacterium tuberculosis* and bacterial culture yielded negative results. No malignant cells could be detected in the pleural effusion, but lymphocytes, neutrophils, histiocytes, erythrocytes and eosinophils were plentifully present (Figure 1). Esophagogastroduodenoscopy and colonoscopy showed no abnormal lesions associated with malignancy. Moreover, no

malignant gynecological lesions were found. Based on these findings, idiopathic EPE was diagnosed. In an attempt to find other means to distinguish right- from left-sided pleural effusion, MRI was performed using a 1.5 T scanner (Achieva A-series, Philips, Amsterdam, Netherlands). MRI revealed hypointensity in bilateral pleural effusions on T1-weighted imaging (Figure 2a) and moderate hyperintensity on T2-weighted imaging (Figure 2b). DWI with a b factor of 1000 sec/mm² revealed moderate hyperintensity and prominent hyperintensity in bilateral lower pleural effusions (Figure 2c) and bilateral upper pleural effusions (Figure 2d).

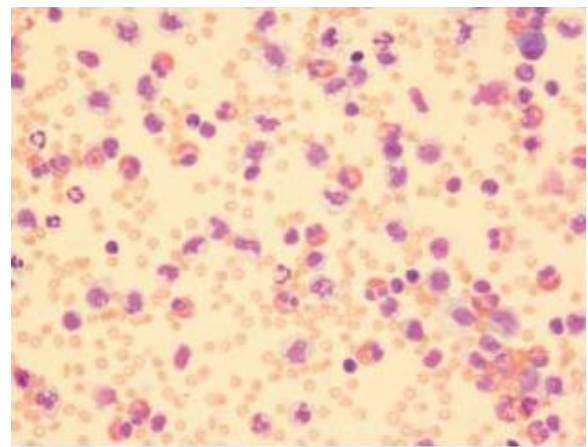


Fig. 1. Right-sided pleural effusion aspiration specimen showing lymphocytes, neutrophils, histiocytes, erythrocytes and eosinophils.

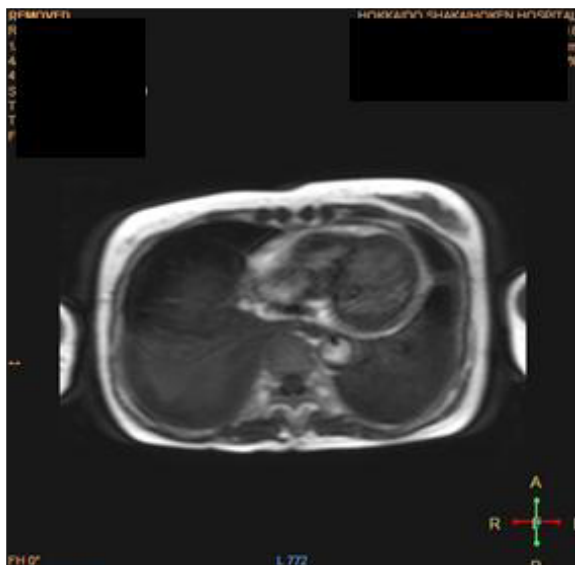


Fig. 2a) Magnetic resonance imaging (MRI) showing hypointensity in bilateral pleural effusions on T1-weighted imaging.

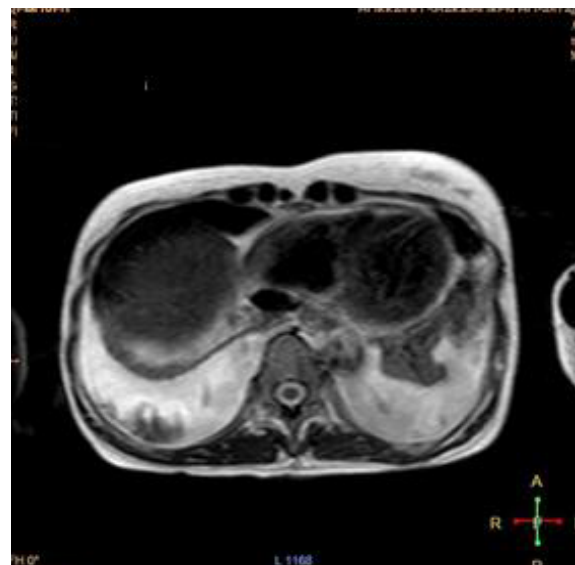


Fig. 2b) MRI showing moderate hyperintensity in bilateral pleural effusions on T2-weighted imaging.

Based on DWI with b factors of 0 and 1000 sec/mm², we created ADC map and calculated ADC. The ADC map revealed prominent hyperintensity accompanied by moderate hyperintensity in the right lower pleural effusion and prominent hyperintensity in the left lower pleural effusion (Figure 2e). And the ADC map revealed moderate hyperintensity in the right upper pleural effusion and prominent hyperintensity in the left upper pleural effusion (Figure 2f). ADC was 3.22×10^{-3} mm²/sec for right upper pleural effusion and 3.96×10^{-3} mm²/sec for left upper pleural effusion. As a result, both the ADC map and ADC values were able to distinguish between bilateral pleural effusions.

The patient was treated with prednisolone (PSL) at 30 mg/day for 2 weeks. Right chest pain and eosinophil counts in peripheral blood improved immediately. At the same time, bilateral pleural effusions also gradually improved. The PSL dosage was tapered, and by 6 months after starting administration of PSL, when PSL dosage was 5 mg/day, pleural effusions had resolved.

3. Discussion

EPE is usually defined as a pleural effusion containing at least 10% eosinophils among the total white blood cells in the effusion (6). The relative incidence of EPE has been estimated at

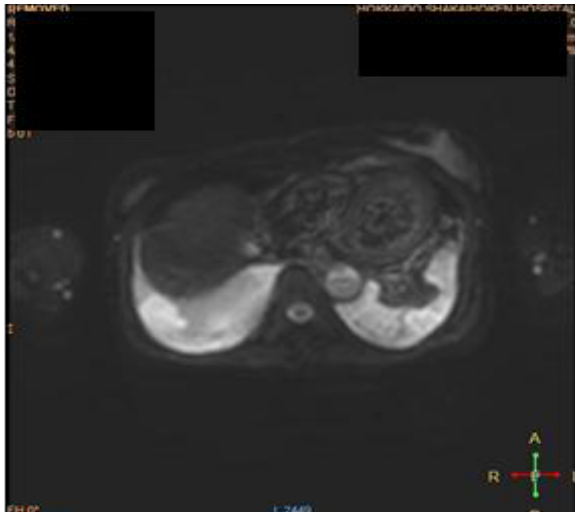


Fig. 2c) Diffusion-weighted imaging (DWI) with a b factor 1000 sec/mm² showing moderate hyperintensity and prominent hyperintensity in bilateral lower pleural effusions.

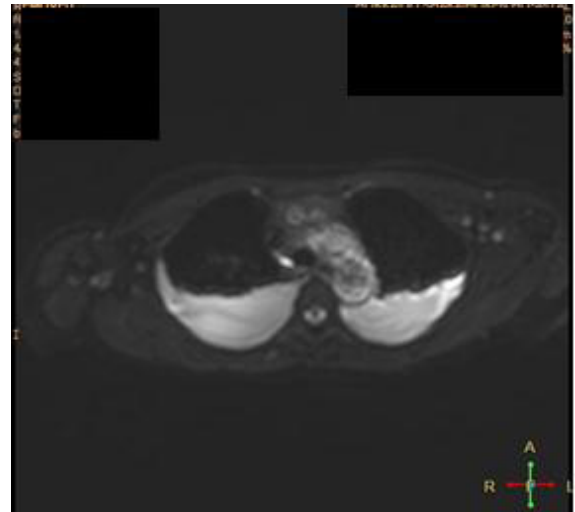


Fig. 2d) DWI with a b factor of 1000 sec/mm² showing moderate hyperintensity and prominent hyperintensity in bilateral upper pleural effusions.

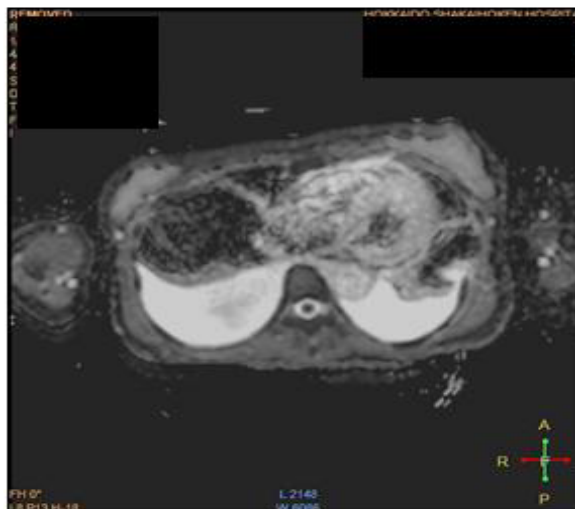


Fig. 2e) Apparent diffusion coefficient (ADC) map showing prominent hyperintensity accompanied by moderate hyperintensity in the right lower pleural effusion and prominent hyperintensity in the left lower pleural effusion.

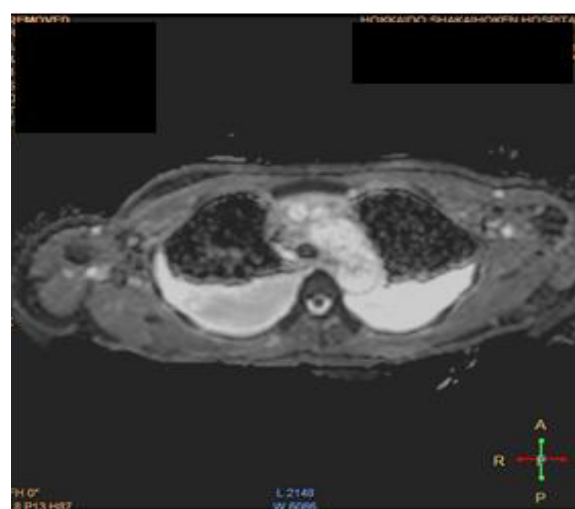


Fig. 2f) ADC map showing moderate hyperintensity in the right upper pleural effusion and prominent hyperintensity in the left upper pleural effusion.

between 5% and 16% of all pleural effusions (6-8). The most common causes associated with EPE were cancerous (36.7%). Benign causes were found in 43.3% of cases. Benign causes included tuberculosis, nonspecific pleuritis, pulmonary thromboembolism, uncomplicated parapneumonic effusion, systemic lupus erythematosus and so on. Despite thorough investigations, the cause of EPE remains undiagnosed and the condition is labeled as idiopathic in 20.0% of cases (9), as in the present case.

In this case, the patient only complained of severe right chest pain, not left chest pain. Interestingly, a considerable difference in percentage of eosinophils existed between right and left pleural effusions; right-sided pleural effusion showed high cell counts including 55% eosinophils and 4.2 g/dl protein, whereas left-sided pleural effusion showed scant cells with only 2% eosinophils and 3.9 g/dl protein. In accordance with this difference in components between right and left pleural effusions, the ADC map and ADC values determined by DWI also revealed a difference between right and left pleural effusions.

In this case, the ADC map in the right lower pleural effusion was composed of a mixture of prominent hyperintensity and moderate hyperintensity. On the other hand, the ADC map in the right upper pleural effusion was composed of moderate hyperintensity without mixture. The difference in the ADC maps between right upper and lower pleural effusions might be due to motion artifact. To be concrete, the fluid in lower pleural effusion might be susceptible to the influence of respiratory motion of diaphragm and cardiac motion. As a result, the lower pleural fluid, namely water protons might move more easily, leading to prominent hyperintensity and higher ADC value. So we adopted ADC values in the bilateral upper pleural effusions, not in the bilateral lower pleural effusions.

Baysal et al. (1) indicated the effectiveness of ADC for differentiating between transudative and exudative pleural effusions. They indicated that ADC was significantly lower with exudative pleural effusion than with transudative pleural effusion. Because pleural fluid is extracellular and normally contains only a few cells besides some protein, protein content is seen as the main factor influencing the ADC. Accordingly, ADC values revealed that increases in the protein content corresponded to decreases in diffusion levels. Exudative effusion was thus considered to

contain a higher protein than transudative effusion.

On the other hand, Inan et al. (2) indicated that parapneumonic effusions, malignant effusions, and tuberculous pleuritis have proteinaceous fluid and rich cell counts (inflammatory cells, tumor cells, and lymphocytes, respectively); therefore, these fluid collections would display a decreased ADC (2).

In the present case, ADCs of right- and left-sided pleural effusions were 3.22 and 3.96×10^{-3} mm²/sec, respectively. The lower ADC for right-sided pleural effusion compared with the left was thus thought to indicate a higher protein content and possibly rich cell content, including eosinophils.

For these reasons, an ADC map and ADC determined from DWI may be helpful either to analyze the properties of pleural effusion in a non-invasive manner or to plan for effusion aspiration.

More extensive research by the use of DWIs of various types of pleural fluids is required to substantiate our findings in the future.

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