CASE REPORT

Application of a paraffin-embedded pleural effusion cell block to detect mycobacteria : A case of *Mycobacterium goodii* pleuritis

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Abstract : The diagnosis of pleuritis caused by mycobacteria is not always easy. Ziehl-Nielsen staining utilizing cell blocks (CBs) of lower respiratory samples was reported to be useful in the diagnosis of pulmonary tuberculosis. However, CBs of pleural effusion (PE) are not commonly used in the differential diagnosis of benign pleuritis. A 100-year-old woman was transferred to our emergency department due to respiratory failure. The patient had massive right PE and an elevated serum carbohydrate antigen 125 level, but no tumor lesions were identified. Mycobacterial examinations using the usual procedures for PE did not lead to a definitive diagnosis. However, Ziehl-Nielsen staining detected several accumulations of acid-fast bacilli in paraffin-embedded PE-CB sections. Finally, sequence analysis of the 16S rRNA gene using the remaining PE-CB showed high homology (99.79%) to that of *Mycobacterium goodii*. This case suggests that analysis of PE-CBs may be useful for diagnosing suspected cases of mycobacteria-induced pleuritis with negative acid-fast bacilli smears for PE. J. Med. Invest. 72 : 202-206, February, 2025

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INTRODUCTION

Nontuberculous mycobacteria (NTM), especially *Mycobacterium avium-intracellulare* (MAI), occasionally causes pleuritis, probably due to direct spread from pulmonary lesions (1). However, several cases of pleural involvement without distinct pulmonary disease have been reported, including pleuritis due to rapidly growing mycobacteria (2-5).

The diagnosis of NTM pleuritis is challenging because there are no diagnostic criteria. Other than the detection of mycobacteria in pleural effusion (PE) or pleural biopsy specimens, no specific or sensitive laboratory findings for NTM pleuritis have been established. On the other hand, mycobacterial cultures of PEs are often negative in patients with clinically suspected NTM pleuritis (6-9).

We herein report a case of mycobacterial culture-negative pleuritis in which Ziehl-Neelsen staining detected several accumulations of acid-fast bacilli in a paraffin-embedded PE-cell block (CB) section ; sequence analysis of the16S ribosomal RNA gene utilizing the remaining PE-CB identified *Mycobacterium goodii*.

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CASE REPORT

A 100-year-old woman was transferred to our emergency department due to respiratory failure. The patient had been bedridden at home for a long time due to heart failure caused by chronic atrial fibrillation and spinal canal stenosis. On physical examination, her temperature was 36.0 ℃, pulse 89/min, blood pressure 126/90 mmHg, respiratory rate 28/min, and SpO2 86%. Chest imaging examinations showed a large amount of right PE, cardiac enlargement, mediastinal lymphadenopathy, and a small amount of left pleural effusion (Fig. 1A-C).

Laboratory data were as follows : white blood cell count 13100/μL (neutrophils 66.70%, lymphocytes 25.9%, eosinophils 0.9%, monocytes 6.1%, basophils, 0.4%) ; total protein 6.3 g/dL ; lactate dehydrogenase 295 IU/L ; C-reactive protein 3.17 (< 0.3) mg/dL ; carcinoembryonic antigen 1.485 (< 6.19) U/mL ; cytokeratin 19 fragments (CYFRA 21-1) 1.1 (< 3.5) ng/mL ; progastrin-releasing-peptide 54 (< 81) pg/mL ; carbohydrate antigen 125 (CA125) 681.5 (< 35) U/mL ; B-type natriuretic peptide 235.9 pg/mL. An interferon-gamma release assay (T-SPOT.TB) was negative and the anti-MAI glycopeptidolipid-core IgA antibody (anti-MAI Ab) level was 0.64 (< 0.6) U/mL. Two thoracentesis procedures were performed three days apart, draining 670 ml and 650 ml of pale yellowish pleural fluid, respectively. Results of PE analysis at first thoracentesis were as follows : pH 7.645 ; cell count 575/μL (lymphocytes 73%, macrophages 7%, neutrophils 20%) ; protein 3.6 g/dL ; lactate dehydrogenase 421 IU/l ; glucose 110.0 mg/dL, indicating exudative effusion by Light's criteria. Cytological examination of PE showed no malignant cells by Papanicolaou staining. Lymphocytic pleurisy with an elevated serum CA125 level suggested tuberculous pleurisy, but the pleural adenosine deaminase (ADA) level (22.5 IU/L) was not high. Morover, PE acid-fast bacilli

smears and real-time PCR tests for *M. tuberculosis* (MTB) and MAI were negative, as was a general bacterial culture. No tumor cells were detected in a specimen of paraffin-embedded CB prepared from 200 ml of PE at second thoracentesis using hematoxylin-eosin staining, but Ziehl-Neelsen staining detected several accumulations of acid-fast bacilli in other sections (Fig. 2A-B). Although the patient was treated with isoniazid, rifampicin, and ethambutol for suspected tuberculosis pleurisy complicated with heart failure, she died two weeks after admission due to exacerbation of heart failure. Subsequently, two PEs and three sputum mycobacterial cultures were all found to be negative. Therefore, we attempted to identify the mycobacteria using the remaining PE-CB. IS6110 and RD1 gene amplification of MTB and Xpert® MTB/Rifampicin were negative (10, 11). Finally, sequence analysis of the 16S ribosomal RNA gene showed high homology (99.79%) with that of *Mycobacterium goodii* (12-14).

DISCUSSION AND CONCLUSIONS

Ziehl-Nielsen staining using CBs of sputum, aspirate samples, and bronchial washing has been reported to be useful in the diagnosis of pulmonary tuberculosis (PTB) (15, 16). The smear-positive and CB-positive rate of the above samples from 64 PTB cases were 91.7% and 91.8%, respectively, and four and five cases were positive for smear only or CB only, respectively (15). However, to the best of our knowledge, there are no reports of Ziehl-Nielsen staining using PE-CBs, even though the diagnosis of pleuritis caused by mycobacteria is not always easy (6-9). This case suggests that analysis of PE-CBs may be useful in diagnosing suspected cases of mycobacterium-induced pleuritis. Mycobacteria are intracellular parasites, and in PE with low bacteria counts, most may be present in phagocytes such as macrophages. CB analysis is characterized by its ability to enrich nucleated cells, to remove mucus components, and to eliminate cellular stratification by preparing thin sections ; it is therefore

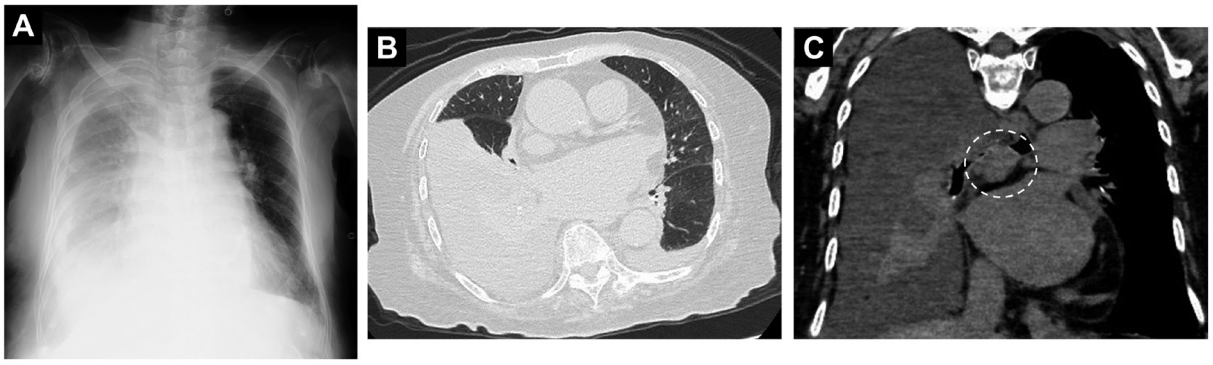

Fig. 1. Radiological findings at the initial visit (A : chest X-ray, B-C : CT, B : lung parenchyma window, C : mediastinal window). (A-C) A large amount of right PE, cardiac enlargement, mediastinal lymphadenopathy (circled), and a small amount of left PE.

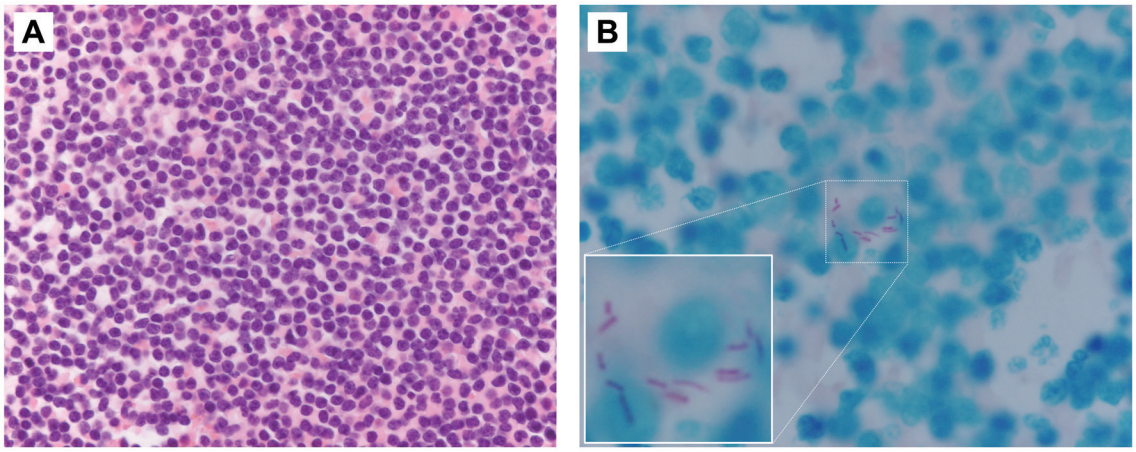

Fig. 2. Analysis of a pleural effusion cell block (A : hematoxylin and eosin staining, B : Ziehl-Neelsen staining, A : ×40 objective, B : ×100 objective). (A) Lymphocyte-dominated pleural effusion cells without tumor cells. (B) Accumulations of acid-fast bacilli.

suitable for detection of mycobacteria in PE.

CA125, a high molecular weight glycoprotein, is known as a tumor marker for epithelial ovarian tumors. On the other hand, CA125 is also produced by mesothelial cells, and its elevation in sera of patients with non-malignant serosal fluid has been reported (17). We could not find any reports of serum CA125 measurements in NTM pleuritis, but the level in this case was extremely high at 681.5 U/ml. In cases of PTB without PE and tuberculous pleurisy, high serum CA125 levels and their diagnostic value have been demonstrated, although the mechanism for the elevation is not fully understood (18-21). There was no clear correlation between serum CA125 levels and pleural fluid CA125 levels in cases of tuberculous pleurisy (20). These findings suggest that elevated serum CA125 levels do not simply reflect a mesothelial reaction associated with pleural effusion. Hirose *et al*. reported a case of tuberculous pleuroperitonitis with a serum CA125 level of 1100 U/ml at diagnosis (21). Serum CA125 has also been reported to be increased in NTM pulmonary diseases (NTMPD) as in PTB (59.0 ± 58.1 U/ml versus 59.3 ± 56.8 U/ml, respectively) (22). Therefore, the elevated serum CA125 in the present case was also considered to be due in part to pleuritis caused by *M. goodii*. However, in recent years, CA125 has emerged as a reliable surrogate of congestion in patients with heart failure and has been reported to be elevated in various acute heart failure scenarios and in chronic heart failure with pleural effusion (23, 24). The pleural effusion in this case was not transudative, but chronic heart failure may have also contributed to the accumulation of pleural effusion and the elevation of serum CA125.

Although NTM pleuritis commonly results in high pleural ADA levels as in tuberculous pleurisy, it was not elevated in the present case (1, 6). However, in suspected NTM pleuritis cases in which the pleural fluid mycobacterial culture was negative, but chemotherapy was successful, the pleural ADA levels were reported to be less than 25 IU/L in three of five cases, suggesting insufficient sensitivity (6). In addition, an NTM pleuritis case with a positive mycobacterial culture on pleural biopsy without an elevated pleural ADA level has been reported (25).

M. goodii is a newly classified rapidly growing mycobacteria that is an independent species from *Mycobacterium smegmatis* (14). *M. goodii*, an emerging pathogen in nosocomial infections, generally infects the skin, soft tissues, respiratory organs, bone marrow, and surgical sites. To the best of our knowledge, a total of 53 cases with clinical data have already been reported in the English literature ; 14 (26%) cases were community-acquired wound/bone infections due to trauma (14, 26-29) and 26 (49%) cases were due to prosthetic material, a device or catheter-related infections (14, 26, 29-46). Six (11%) cases were infections associated with hospital-based other invasive procedures (cardiac bypass, surgical wound, skin graft, breast and bursal infections) (14, 26, 28, 47, 48). 11 (21%) cases were pulmonary disease (pneumonia including lipoid or granulomatous pneumonia, and vasculitis) (14, 26, 49-53). Four cases had overlapping conditions (14, 28, 29). To date, only one case in which *M. goodii* was detected in PE has been reported ; this was from Zambia with no description of serum CA125 or pleural ADA (50).

M. goodie is generally resistant to rifampin and clarithromycin because overexpression of the *wag31* gene thickens the peptidoglycan layer and the presence of the *erm* gene contributes to macrolide resistance (54, 55). The pathogen is difficult to treat due to its unique resistance pattern, requiring long-term treatment, and delayed identification is associated with a worse prognosis. Since no standard treatment for *M. goodii* infections has been established, more emphasis is being placed on antimicrobial susceptibility testing. *M. goodii* is usually susceptible to amikacin, sulfamethoxazole, imipenem, tetracycline, and ethambutol (14). Sulfamethoxazole-trimethoprim and doxycycline are the most used oral medications, although monotherapy often leads to treatment failure. In severe cases, parenteral amikacin or imipenem is additionally administered (56).

In this case, the anti-MAI antibody level was slightly elevated. Anti-MAI antibodies can be positive in some patients with NTMPD due to rapidly growing mycobacteria, but there have been no reports on anti-MAC antibodies in patients with NTMPD due to *M. goodii*, so the positivity rate is unknown (57, 58).

In summary, this case suggests that analysis of PE-CBs may be useful in diagnosing suspected cases of mycobacteria-induced pleuritis with negative acid-fast bacilli smears for PE. In addition, *M. goodii* may cause pleuritis with a high serum carbohydrate antigen 125 level.

CONFLICT OF INTEREST

The authors declare no competing interests.

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None.

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