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CASE REPORT OF A PATIENT WITH NEUTROPHIL-PREDOMINANT TUBERCULOUS PLEURAL EFFUSION

ŠKORO M.[1], Badovinac S.[2], Banić M.[1], Džubur F.[1], Kovačić H.[1], Moćan A.[1], Pavliša G.[1], Puretić H.[1], Samaržija M.[1], Žmak Lj.[3], Janković Makek M.[1]

[1] Klinika za plućne bolesti Jordanovac, Zagreb, Croatia
Odjel intenzivne i postintenzivne skrbi

[2] Klinika za plućne bolesti Jordanovac, Zagreb, Croatia
Odjel za bronhoskopiju i interventnu pulmologiju

[3] Hrvatski zavod za javno zdravstvo, Zagreb, Croatia
Nacionalni laboratorij SZO za tuberkulozu

Objective:

INTRODUCTION

Tuberculous pleural effusion (TPE) is the second most common form of extrapulmonary tuberculosis. Although the definitive diagnosis is made by positive pleural fluid culture, in clinical practice, the diagnosis is generally made by the combination of biochemical and cytological pleural fluid analysis. Tuberculosis is typically characterized by lymphocyte-predominant exudate (lymphocyte to neutrophil ratio >75%) and by adenosine deaminase (ADA) values > 40 U/L. However, in 7% of the cases, TPE

presents as neutrophil-predominant fluid, hence implying the parapneumonic effusion.

CASE REPORT

35-year-old S.M., with a history of recurrent alcohol-induced pancreatitis, was admitted due to prolonged dry cough, progressive exertional dyspnea, malaise, and weight loss over the course of two months. Aside from an initial fever of 38,8°C, he was afebrile. The initial imaging revealed a large left-sided pleural effusion, along with elevated inflammatory markers and high levels of liver function tests. Left hemithorax drainage and empiric antibiotic therapy (meropenem and vancomycin) were initiated. The pleural fluid analysis revealed neutrophil-predominated exudate (60% neutrophil, 33% lymphocyte), ADA level of 78 U/L, and sterile initial microbiological analysis. CT pulmonary angiography excluded pulmonary embolism and affirmed left-sided circumferential pleural thickening alongside with parenchymal consolidation of the lower left lobe with cavities. Bronchoscopy showed normal findings, bronchoalveolar lavage (BAL) samples were smear- and PCR-negative on *Mycobacterium tuberculosis*. Opportunistic pathogens, *Lactobacillus species*, and *Rothia mucilafinosa* were isolated, and initial antibiotic therapy was continued.

The additional examination confirmed liver cirrhosis with portal hypertension and ascites (probably due to alcoholic liver disease, with negative acute viral hepatitis serologic markers) alongside with previously diagnosed chronic pancreatitis. The course of hospitalization was complicated with worsening of hepatic function and new-onset thrombocytopenia (min Trc $3x10^9$/L), without signs of hemorrhagic diathesis. After broad hematologic workup, the diagnosis of immunotrombocytopenia was made, with a good response to steroid therapy. After one month of treatment, significant, but incomplete regression of infiltrate and pleural effusion was observed, with normalization of acute-

phase reactants. The patient was discharged until the microbiological culture results. Finally, three weeks after discharge, BAL fluid culture confirmed tuberculosis and the patient was hospitalized for initiation of ATL therapy.

CONCLUSION

The diagnosis of pleural tuberculosis can be a challenge. Although lymphocyte-predominant exudate with high ADA level is considered characteristic for TBC infection, the diagnosis is not excluded by neutrophilic predominance on cytological analysis. Neutrophil-predominant TPE is associated with a more intense inflammatory response and higher positive rates in microbiological testing.