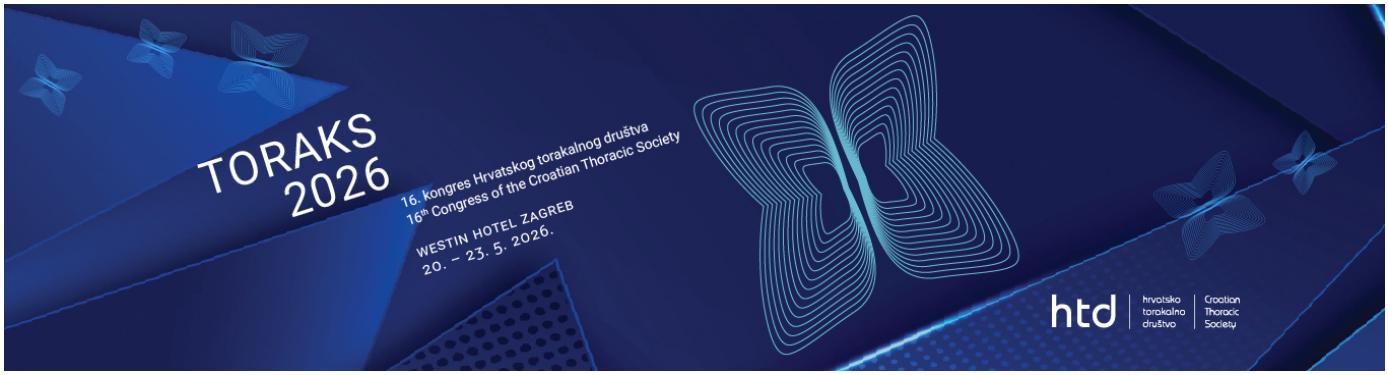


RHEUMATOID PLEURITIS IN THE DIFFERENTIAL DIAGNOSIS OF PLEURAL EFFUSION WITH ELEVATED ADA ACTIVITY: A CASE REPORT

KRIŠTO M.[1], Krolo A.[1], Hećimović A.[1], Rnjak D.[2], Mihelčić Korte D.[2], Đuzel M.[2]

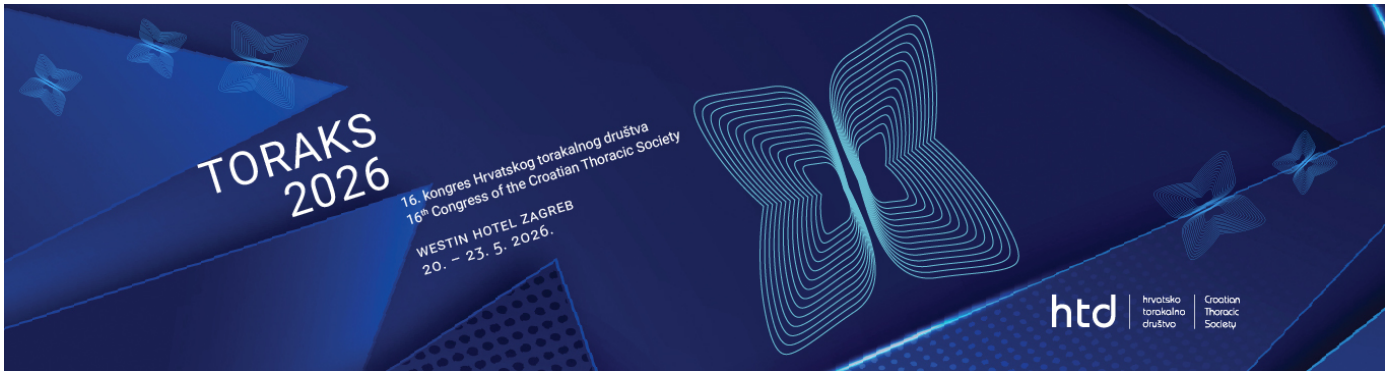
[1] University Hospital Center Zagreb, University of Zagreb, School of Medicine, Zagreb, Croatia
Clinic for Respiratory Disease

[2] University Hospital Center Zagreb, Zagreb, Croatia
Clinic for Respiratory Disease

Objective:

Introduction: Rheumatoid pleuritis is one of the rare extra-articular manifestations of rheumatoid arthritis (RA) and rarely presents as the first symptom of the disease. A diagnostic challenge is the similarity of the biochemical profile of rheumatoid effusion, which often mimics tuberculous (TB) pleuritis. Both are characterized by an exudate with high lactate dehydrogenase (LDH) levels, low glucose concentration, and elevated adenosine deaminase (ADA) activity.

Case presentation: A 51-year-old female patient was initially evaluated for a pulmonary infiltrate with cavitation in the right upper lobe. Initial workup raised suspicion of aspergilloma after Aspergillus fumigatus was isolated from a transthoracic aspirate. Because the infiltrate progressed



under therapy without a clear etiology, surgical biopsy was indicated; histopathology suggested an abscess. Postoperatively, a right-sided pleural effusion developed. Biochemical analysis of the pleural fluid showed markedly elevated LDH (>4500 U/L) and ADA (177 U/L) with cytologic lymphocyte predominance (91%). Laboratory tests showed mildly elevated inflammatory markers: WBC 11 × 10^9/L, CRP 11.3 mg/L; other results were unremarkable. Because of suspicion for tuberculosis, antituberculous (ATT) therapy was started. During follow-up there was no regression of the effusion and the patient developed symmetric polyarthritis of the small hand joints two months after treatment initiation. Immunologic testing confirmed seropositive rheumatoid arthritis: RF <20 IU/mL, anti-CCP >500 U/mL. Given the persistent effusion despite ATT and the clinical and immunologic findings, the effusion was concluded to be a manifestation of rheumatoid arthritis. Treatment with corticosteroids and subsequently methotrexate was initiated, leading to regression of the pleural effusion and articular symptoms.

Conclusion: Elevated ADA in pleural fluid is not pathognomonic only for tuberculosis and can also indicate rheumatoid pleuritis due to a strong immune response. Recognizing a rheumatoid etiology as a possible cause of such effusions allows timely adjustment of therapeutic strategy and initiation of appropriate immunomodulatory treatment.