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SEVERE NONTUBERCULOUSSIS MYCOBACTERIAL LUNG DISEASE CAUSED BY M. MALMOENSE IN A YOUNG PATIENT WITH DIABETES

ĆUK B.[1], Anamarija Š.[2], Krpina K.[2], Marušić A.[3], Janković Makek M.[2], Strelec D.[4], Popović-Grle S.[2]

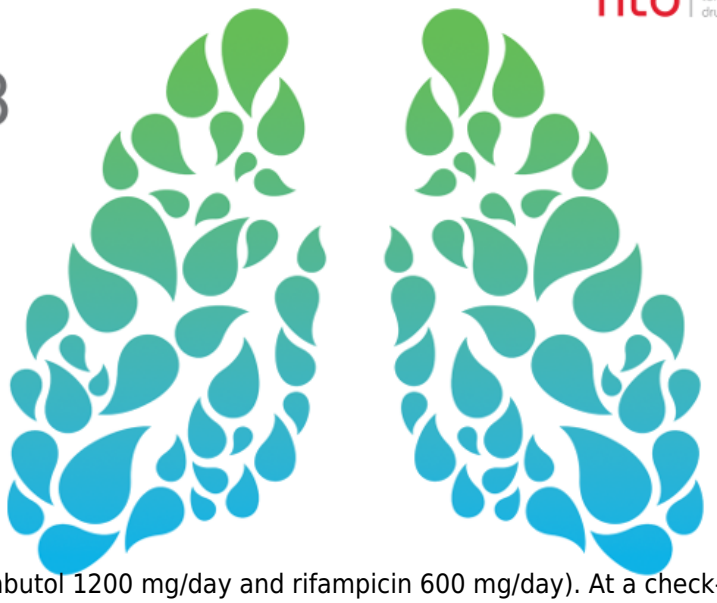
[1] General hospital "Dr Ivo Pedišić", Sisak, Croatia
Department of respiratory disease

[2] University Hospital Centre Zagreb, Zagreb, Croatia
Clinical Center for pulmonary Disease Jordanovac

[3] University Hospital Centre Zagreb, Zagreb, Croatia
Department of radiology

[4] Hospital for Pulmonary Diseases and TBC Klenovnik, Klenovnik, Croatia
Department for respiratory disease

Objective: Introduction: The prevalence of nontuberculous mycobacterial lung disease (NTM-LD) has been increasing worldwide, even in immunocompetent individuals. It is caused by ubiquitous enviromental pathogen and clinical, microbiologic, and radiographic criteria should be met to confirm NTM-LD.
Case report: We present a 39-years old male patient with NTM-LD caused by M. malmoense The patient is an outdoor chef, an ex-smoker (40P/Y), uses wood as a fuel and is an insulin-dependent diabetic. He first presented 10/2014 with weight loss, haemoptysis, fever. Radiological finding of destructive infiltrates in both upper lung lobes. Inflammatory parameters were slightly elevated. Due to a high suspicion of tuberculosis initial treatment regime included isoniazid, rifampicin, pyrazinamide, and ethambutol. Short course of clarithromycin was added when M. malmoense was identified as causative agent. After 2.5 months of treatment, some clinical improvement



occurred and dual therapy was continued (ethambutol 1200 mg/day and rifampicin 600 mg/day). At a check-up in September 2015. he was still directly positive, and ciprofloksacin was added to the regimen. Still he remained sputum and culture positive. In June 2016. clarithromycin replaced ciprofloksacin and sputum and culture conversion was achieved for the first time in almost two years of treatment. Therapy continued, the patient clinically improved (gained 30kg) and has remained culture negative until July 2017.At that point radiological work-up revealed progression of the infiltrate in the upper right lobe (25x13x31mm in diameter) and a newly developed cavity in the left lower lobe while bronchoscopy showed narrowed orifices in the apico-posterior segment of the left upper lobe. Bronchial catheter aspirate was M. malmoense positive and cytological examination detected granulomatous inflammation. Humoral and cellular immunity showed no abnormality, and pharmacogenomic examination was conducted. Since addition of levofloksacin yielded no improvement. In 12/2017 a new regime was initiated (rifampicin 600 mg/day, ethambutol 1600 mg/day, parenteral: amikacin 1000 mg/day and azithromycin 500 mg/day). Control check-up clinical improvement, culture conversion, while radiological examination revealed stationary polymorphic changes in pulmonary parenchyma with a slight progression of confluent nodules. The treatment continues with rifampicin 600 mg/day, ethambutol 1600 mg/day, and azithromycin 500 mg/day.
Conclusion: We aimed to show how challenging it is to determine an active NTM-LD. It is important to include this disease in the differential diagnosis even in the immunocompetent individuals. Currently recommended treatment regimens and management are a lengthy, complicated process and should be species directed.