Mycobacterium mantenii is a slow growing, nontuberculous NTM species, most closely related to Mycobacterium scrofulaceum that was described firstly in 2009 [1]. Little is known about its clinical significance, natural habitat and virulence. We report for the first time a case of chronic osteomyelitis caused by M. mantenii in an immunocompromised patient with sarcoidosis and diabetes mellitus.

**Case Presentation**

A 59-year-old woman was referred to the outpatient clinic of Infectious Disease Division, for the management of a chronic unsecured inflammation on left forefinger nail, over the last eight months.

The patient had a history of sarcoidosis and liver failure caused from extensive hepatic granulomatus foci, with a record of hepatic encephalopathy one year ago. She also had mild renal failure as a complication of diabetes mellitus. Her medications included insulin, corticosteroids and thyroxin. She worked as housewife. She had travelled outside Greece during the last three years and hadn’t owned pets. No contact with swimming pool water was mentioned.

On physical examination, the patient appeared well. There was no fever, jaundice or lymphadenopathy. The cardiovascular and respiratory systems were intact. The hand radiograph revealed a destructive process at the final phalanx of the left forefinger (sequestra).

**RESULTS**

A 439-bp fragment of the 65-kDa heat shock protein (hsp65) gene was amplified using the protocol and the primers TB11 and TB12 [3]. The PCR product digested with HaeIII and BstEII (New England Biolabs) produced at least three fragments of 145, 130 and 60 bp.

For Mycobacterium strain GR5098 the BstEII digestion produced two fragments of 235 and 200 bp and the HaeIII digestion produced four fragments of 130, 185, 70 and 50 bp while for M. intracellulare BstEII digestion produced three fragments 234, 117, 100 and HaeIII digestion produced at least three fragments of 145, 130 and 60 bp.

According to results of PCR-RFLP strain GR5098 was different than M. intracellulare.

The strain was identified as M. intracellulare by the Genotype Mycobacterium CM (Hain, Lifescience).

According to sequencing results the strain was identified as M. mantenii.

The isolate identified as M. intracellulare from the Geneotype Mycobacterium CM (Hain, Lifescience)

![Fig 4. Results of the Geneotype Mycobacterium CM assay for the recovered strain](image)

The isolate was considered: susceptible to clarithromycin, rifabutin, rifampin, ethambutol, amikacin, linezolid and moxifloxacin.

Resistant to ciprofloxacin, isoniazid, and trimethoprim-sulfamethoxazole

The minimum inhibitory concentration (MICs) in mg/L were determined using commercially available microtiter plates (SLMYCO/TREK Diagnostic systems) following the Clinical and Laboratory Standards Institute (CLSI) recommendations [7].

**REFERENCES**


**Fig. 3. Ziehl-Nielsen stain of Mycobacterium mantenii strain grown in the MGIT960 tubes (600x)**

**Fig. 4. Results of the Geneotype Mycobacterium CM assay for the recovered strain**

**Fig. 5. Agarose (3%) gel electrophoresis of products produced by digestion of PCR amplification products of the hsp65 gene of M. intracellulare (lane 1, 2) and Mycobacterium strain GR-5098 (lane 3-4) with BstEII (lane 2, 3) and HaeIII (lane 1, 4). The restriction mixture was run at 100 V. M1: GeneRuler 50 bp DNA ladder (Fermentas) M2: GeneRuler 25 bp DNA ladder (Fermentas).**

**Fig. 6. Phylogenetic relationship of M. mantenii strains GR-5098 and related species of the genus Mycobacterium based on 16S rDNA (open triangle) and rpoB (open square) gene sequences. Neighbor-joining tree was constructed, bootstrapped 1000 times and visualized with MEGA4.1. Bootstrap values are indicated at the nodes. The tree was rooted with the use of M. abscessus subsp. abscessus as the outgroup. The scale bar presents a 0.25 divergence in nucleotide sequences.**

**Fig. 7. Picture and radiograph of the left forefinger after six months of therapy.**

A triple therapy with clarithromycin, moxifloxacin and ethambol for 5 weeks was initiated and moxifloxacin plus ethambol followed for six months. The patient experienced considerable clinical improvement (Fig.7) and a follow-up fine needle specimen, three months after the initiation of therapy was negative in both liquid and solid culture media.

**CONCLUSIONS**

Our findings suggest that the osteomyelitis case in the immunocompromised patient was caused by the novel species M. mantenii. Sequencing analysis of the genes hsp65, 16S rDNA and rpoB allowed the identification of this uncommon mycobacterial species, which was misidentified by commercial probes.