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A Case Report: *Mycobacterium fortuitum* Spondylodiscitis

Bir Olgu Sunumu: *Mycobacterium fortuitum* Spondilodiskiti

Sinan MERMER[1], Cengiz ÇAVUŞOĞLU[2], Selin BARDAK ÖZCEM[3], Meltem TAŞBAKAN[4], Hüsnü PULLUKÇU[4], Bilgin ARDA[4], Tansu YAMAZHAN[4], Sercan ULUSOY[4], Oğuz Reşat SİPAHİ[4]

[1]Çine State Hospital, Clinic of Infectious Diseases and Clinical Microbiology, Aydın, Turkey
[2]Ege University Faculty of Medicine, Department of Medical Microbiology, İzmir, Turkey
[3]Dr. Burhan Nalbantoğlu State Hospital, Clinic of Infectious Diseases and Clinical Microbiology; Near East University Hospital, Clinic of Infectious Diseases and Clinical Microbiology, Lefkosia, Cypru
[4]Ege University Faculty of Medicine, Department of Infectious Diseases and Clinical Microbiology, İzmir, Turkey

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Dear Editor,

Spondylodiscitis refers to infection of both the intervertebral disc and vertebrae. It may develop primarily or as a healthcare-associated infection following spinal surgeries[1].

Tuberculosis (TB) is still endemic in Turkey, and can cause extrapulmonary involvement in various systems. One of the common sites of extrapulmonary TB is bone, with 10% of cases having vertebral involvement. Nontuberculous mycobacterial (NTM) infections are usually sporadic. Of the NTM, *Mycobacterium fortuitum*, is rarely an infectious agent, but can cause infections after trauma or surgery, especially infections associated with orthopedic implants[2]. Herein, we present a case of spondylodiscitis due to *M. fortuitum*.

A 44-year-old female patient with no known chronic disease underwent surgery at another center in 2000 and 2007 due to a herniated disc at L4-L5. Approximately one month after the second surgery, she presented to the neurosurgery clinic of our hospital due to increased pain in her lower back and legs. Examination and imaging revealed lumbar stenosis at L4-L5, and the patient underwent L4-L5 laminectomy and L4-L5-S1 posterior fixation. Five months after discharge, the patient presented to a private medical center for persistent leg and lower back pain and underwent a fourth operation. Some of the screws at L4 appeared infected and were removed during the procedure. Afterwards, she was given an antibiotic for over three weeks, but she did not know the name of it. The patient's complaints of pain in the legs and lower back persisted during the first postoperative month. Lumbar magnetic resonance imaging (MRI) showed an abscess formation with peripheral enhancement, over 5 cm in diameter, extending to the L5-S1 pedicular screws and under the skin posterior to L4-L5. The patient was readmitted to the neurosurgery clinic of our hospital and underwent abscess drainage and debridement. Rose Bengal test performed at this time was negative. A specimen taken from the abscess was analyzed with bacterial culture, mycological culture, TB culture, acid-fast bacillus (AFB) staining, and pathological examination. Bacterial and mycological cultures did not yield any pathogen and AFB staining was negative. Inflammatory granulation tissue was observed in pathological examination. Consultation was requested due to the patient's negative cultures; the patient was started on empirical ceftriaxone and teicoplanin therapy and transferred to Infectious Diseases Clinic postoperatively. After nearly one month of antibiotherapy with ceftriaxone and teicoplanin and follow-up in our clinic, recurrent abscess was observed on control lumbar MRI. The patient was transferred

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Address for Correspondence/Yazışma Adresi: Sinan Mermer MD
Çine State Hospital, Clinic of Infectious Diseases and Clinical Microbiology, Aydın, Turkey
E-mail: sinanmermer@hotmail.com **ORCID ID:** orcid.org/0000-0001-9937-6267
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back to neurosurgery for abscess drainage and removal of the L4-L5 instrumentation. A specimen obtained intraoperatively from the abscess on day 27 of antibiotherapy was cultured on MGIT960 medium (Becton Dickinson, USA) and yielded *M. fortuitum*. The patient was transferred back to our clinic and her antimicrobial therapy was changed to amikacin 1 g once daily, ceftazidime 2 g three times daily, and moxifloxacin 400 mg once daily. Antibiotic susceptibility test results indicated sensitivity to amikacin, ciprofloxacin, and imipenem; moderate sensitivity to clarithromycin; and resistance to doxycycline and cotrimoxazole. Therefore, ceftazidime was discontinued and replaced with imipenem 500 mg four times daily and treatment was continued for 45 days. Serology for human immunodeficiency virus (HIV) was negative. Laboratory results before antimycobacterial treatment indicated white blood cell (WBC) count: 5710/mm^3, C-reactive protein (CRP): 3.29 mg/dL, and sedimentation rate: 78 mm/h. After antimycobacterial treatment, these values were WBC: 4840/mm^3, CRP: 0.35 mg/dL, and sedimentation: 14 mm/h. Oral moxifloxacin and clarithromycin were prescribed at discharge as maintenance therapy and continued for a total of six months. After treatment, no relapse occurred during one year of follow-up.

Spondylodiscitis, or infections affecting both the intervertebral discs and the vertebrae, may arise due to pyogenic infections, brucellosis, or TB[1]. The differential diagnosis of primary spondylodiscitis includes mycobacteria; pyogenic and fungal infections due to *Staphylococcus aureus*, *Cryptococcus* spp., and *Actinomyces* spp.; other granulomatous diseases such as sarcoidosis and brucellosis; and tumors (metastasis, myeloma, lymphoma). Because of imaging techniques are insufficient for differential diagnosis, bacteriological and histological confirmation is required[3]. Secondary spondylodiscitis usually occurs secondary to resistant Gram-negative and Gram-positive bacteria. Isolation of the agent from tissue biopsy culture is considered to be the gold standard in diagnosis and the specificity of this method is nearly 100%. Molecular biology techniques are crucial for early detection, particularly in mycobacterial infections. In spondylodiscitis, MRI findings are nonspecific and preclude a definitive interpretation regarding etiology [4]. Conducting a computed tomography-guided biopsy is essential due to the difficulty of determining the etiology of spondylodiscitis. This method allows identification of the causative agent in 30-70% of cases[5]. About 50% of pyogenic spondylodiscitis and 75% of tuberculous spondylodiscitis can be detected in this way. Spinal fluid culture has a diagnostic value of 52%[6]. In the present case, MRI revealed an abscess, sampling was performed intraoperatively during abscess drainage and debridement, and inflammatory granulation tissue was observed in pathological examination of the material. Acid-fast bacilli were not detected in microscopic examination of a smear prepared from a specimen obtained during surgery. The sample was cultured on MGIT960 medium and bacterial growth detected after 12 days was identified as *M. fortuitum* using a GenoType Mycobacterium kit (Hain Lifesciences, Germany). Drug susceptibility testing in OADC-supplemented Mueller Hinton Broth using the E-test method showed that the isolate was susceptible to amikacin, ciprofloxacin, and imipenem, and had intermediate susceptibility to clarithromycin. The patient was diagnosed with NTM spondylodiscitis based on clinical, imaging, and mycobacterial culture findings and treatment was initiated with amikacin, imipenem and moxifloxacin. After 45 days of treatment, the patient's CRP and sedimentation values returned to normal levels and she was discharged with oral therapy as maintenance. There was no relapse during 1 year of follow-up.

Although mycobacterial spondylodiscitis is generally caused by *Mycobacterium tuberculosis*, in rare cases it can be caused by NTM. In a review by Petitjean et al.[7], it was reported that of 31 patients with NTM-associated vertebral osteomyelitis between 1965 and 2013, only 5 cases were caused by *M. fortuitum*. The etiological agent in our case was also the rare *Mycobacterium fortuitum* is among the rapidly proliferating mycobacteria, able to proliferate in culture in less than a week. It usually causes infections after trauma or surgery, especially infections associated with orthopedic implants[2]. Combination therapy is often used in patients with extensive and deep involvement, chronic infections, or HIV infection. Antimycobacterial susceptibility testing should be performed. Based on the results of in vitro susceptibility testing, treatment may include amikacin, erythromycin, doxycycline, cefoxitin, ciprofloxacin, trimethoprim/sulfamethoxazole, clarithromycin, rifampicin, and/or imipenem. Longardner et al.[8] reported a patient with history of intravenous drug use and spinal osteomyelitis whose abscess culture grew *M. fortuitum* and who was treated with a combination of cefoxitin and amikacin for two weeks, followed by oral treatment with trimethoprim/sulfamethoxazole + doxycycline. In another case of *M. fortuitum* vertebral osteomyelitis presented by Duttaroy et al.[9], combination of ciprofloxacin + gentamicin elicited a treatment response, but the patient died during follow-up. Although the optimal duration of treatment for NTM-associated spondylodiscitis has not been defined, it is suggested that treatment should continue for a minimum of 6-12 months[10]. Our case was treated with dual therapy for six months.

This case highlights yet again the importance of biopsy culture in identifying uncommon etiological agents during the treatment of spondylodiscitis.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: C.Ç., Analysis or Interpretation: S.M., O.R.S., S.U., T.Y., B.A., M.T., H.P., S.B.O., Literature Search: S.M., O.R.S., S.U., T.Y., B.A., M.T., H.P., S.B.O., Writing: S.M., O.R.S., S.U., T.Y., B.A., M.T., H.P., S.B.O.

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