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CASE REPORT

Vertebral osteomyelitis caused by non-tuberculous mycobacteria: case reports and review

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Abstract There are currently few reports of vertebral osteomyelitis caused by non-tuberculous mycobacteria. To date, only 38 cases, excluding human immunodeficiency virus patients, have been reported. We describe 3 patients with vertebral osteomyelitis caused by *Mycobacterium avium-intracellulare* complex or *Mycobacterium kansasii*, and review previous reports of vertebral osteomyelitis caused by non-tuberculous mycobacteria. *Case 1* is a 50-year-old man who presented with lower back pain. Radiologic examination revealed L1–L5 enhancement and paravertebral abscess. The surgical specimen was positive for *Mycobacterium avium-intracellulare* complex. The patient was successfully treated by surgical excision and antibiotic administration. *Case 2* is a 68-year-old woman who presented with upper back pain. Spine MRI revealed multiple lesions at T9–T12, L2, L4, and L5. Her back pain worsened, and repeated MRI revealed extensive bone lesions. *Mycobacterium kansasii* was isolated from a T5 vertebral body specimen. Surgery was not performed. *Case 3* is a 38-year-old woman who had been taking prednisolone for systemic lupus erythematosus. We diagnosed her condition as suppurative knee arthritis caused by *M. avium-intracellulare* complex. Vertebral MRI revealed T9 vertebral body enhancement and a paravertebral abscess at T8–T9. Tissue culture of a T9 specimen yielded *M. avium-intracellulare* complex. Her clinical condition improved following posterior thoracic spinal fusion. In conclusion, vertebral osteomyelitis caused by non-tuberculous mycobacteria should be included in the differential diagnosis, even in immunocompetent patients.

Keywords Atypical mycobacteria · Non-tuberculous mycobacteria · Osteomyelitis

Introduction

Non-tuberculous mycobacteria (NTM) are slow-growing bacteria that commonly cause invasive infections in immunocompromised hosts, such as in patients infected with human immunodeficiency virus (HIV). The most common infection site in the immunocompetent host is the respiratory system; other sites include the lymph nodes, skin, soft tissues, and bones.

Vertebral osteomyelitis caused by NTM is rare, particularly in immunocompetent patients. Furthermore, its clinical features are often indistinguishable from those of pyogenic osteomyelitis or tuberculous osteomyelitis. Early precise diagnosis of these infections is important because the patient's neurological deficits would progress without immediate and adequate treatment.

To the best of our knowledge, only 38 cases, excluding patients with HIV, have been reported in the literature. Here, we describe 3 cases and review vertebral osteomyelitis caused by NTM.

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Case reports

Case 1

A 50-year-old man presented with a history of lower back pain for several years. He had visited an orthopedic surgeon regularly and had been treated with analgesics. The patient had a history of bipolar disorder. He had not taken immunosuppressive medications in the past and had no underlying immunodeficiency problems. His back pain intensified 3 months before admission, and he was unable to walk without assistance on the day of admission. He was afebrile and did not have any respiratory symptoms. Tenderness over the lumbar spine (L2–L4) was found on physical examination at the initial visit.

Vertebral magnetic resonance imaging (MRI) revealed enhancement of the vertebral bodies at L1–L5 and intervertebral disks between L1–L2, L2–L3, L3–L4, and L4–L5 (Fig. 1a). A paravertebral abscess and right psoas abscess were found at the level of L4 (Fig. 1b). A specimen taken from the disk between L3 and L4 by transcutaneous biopsy was tuberculous bacteria–polymerase chain reaction (TB-PCR) negative. We therefore considered the possibility of pyogenic osteomyelitis. As the tissue culture yielded no viable bacteria, a wait-and-see approach was taken. Five weeks after the biopsy, the tissue culture yielded a positive result for *Mycobacterium avium-intracellulare* complex.

The patient underwent surgical excision of the inflamed tissue, followed by drainage of the paravertebral abscess and psoas abscess. His back pain gradually subsided with treatment with clarithromycin (800 mg/day), ethambutol (1,000 mg/day), and rifampin (600 mg/day). The patient was pain free at the 12-month follow-up and was able to walk unaided. Treatment will be discontinued depending on the patient's condition.

Case 2

A 68-year-old woman presented with a 10-month history of progressive upper back pain. The patient had no medical history and had taken no immunosuppressive medications. At the initial visit, the patient was afebrile and did not have any respiratory symptoms. Laboratory examination revealed a white blood cell count of 11,400/μl and an erythrocyte sedimentation rate (ESR) of 130 mm/h.

MRI of the spine revealed multiple lesions at T9–T12, L2, L4, and L5 (Fig. 2a). We initially considered these lesions to be multiple bone metastases from a distant cancer. We attempted the first biopsy from the vertebral body. Diffused fibrosing lesions were observed between the trabecular bone with invasion of small lymphocytes and phagocytes. However, no malignant cells were detected from a pathological specimen taken from a vertebral body,

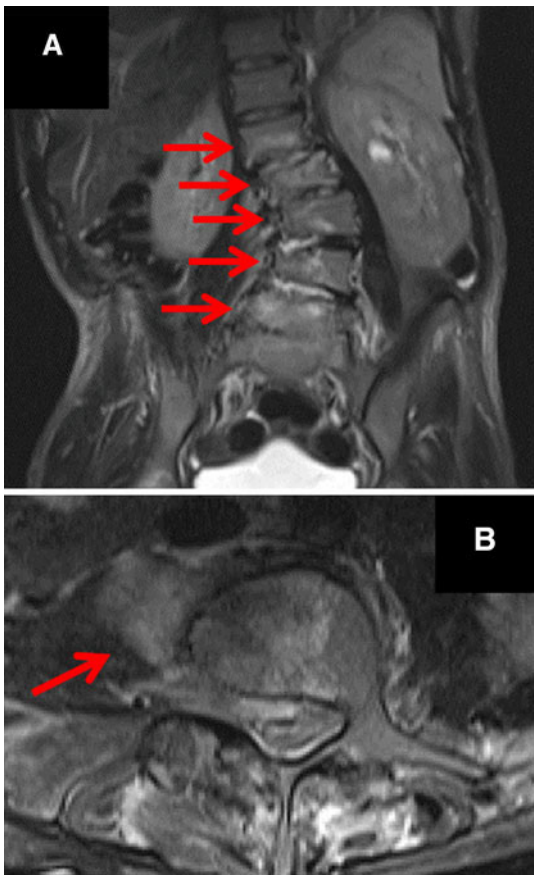

Fig. 1 Magnetic resonance imaging (MRI) scan (T_2-weighted image) of case 1. **a** Coronal MRI shows enhancement of vertebrae at L1–L5 and intervertebral disks between L1–L2, L2–L3, L3–L4, and L4–L5 (*arrows*). **b** Transverse MRI shows a right paravertebral abscess and a right psoas abscess (*arrow*)

and we could not identify any primary tumor. Moreover, tissue culture of the specimen from the vertebral body yielded no organisms. Analgesics were used pending the definitive identification of the cause of the back pain.

The patient's back pain gradually worsened, and repeated MRI revealed extensive bone lesions compared with the previous MRI findings, collapse of the T5 vertebral body, and paravertebral abscesses at T4–T6 and at T9 (Fig. 2b). Fortunately, no neurological deficits were observed. Two months after the first biopsy, we attempted a second biopsy to pinpoint the exact cause of the vertebral lesions.

After several weeks of tissue culture of a specimen from the T5 vertebral body obtained at the second biopsy, *Mycobacterium kansasii* was isolated and identified. Although no granulomatous lesions from the specimen obtained from the vertebral body were detected, and the histopathological findings were only inflammatory cell invasion and fibrotic changes as shown by hematoxylin and eosin staining, acid-fast organisms were detected by Ziehl–Neelsen staining (Fig. 3).