Case Report

Spondylodiscitis secondary to *Mycobacterium chelonae*: a case report

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Background: Spondylodiscitis secondary to *Mycobacterium chelonae* (*M. chelonae*) is a rare primary infection of the spine, with a few case reports highlighted. Treatment of this infection is not well established but here we discuss a case where a patient recovered well following early aggressive surgical intervention and antibiotic treatment.

Case Description: A 32-year-old male presented with a 3-month history of worsening low back pain, Horner's syndrome, dysphagia, lower extremity weakness, and a 5-day history of bowel and bladder incontinence. The patient had an extensive orthopedic history but no recent trauma or history of spinal surgery. He had no known prior medical conditions that would suggest immunocompromise. Magnetic resonance imaging (MRI) scan showed lumbar spondylodiscitis, and blood cultures did not show any growth. The patient underwent L4-S1 decompression and fusion with iliac crest bone grafting, and intraoperative biopsy. Intraoperative tissue cultures grew *M. chelonae*. Repeat computerized tomography (CT)-guided biopsy confirmed the pathogen. The patient was initially treated with vancomycin and piperacillin-tazobactam. Numerous alterations in antibiotic regimen occurred secondary to medication adverse effects and noncompliance, and he was ultimately treated with azithromycin and tigecycline. Interval follow-up demonstrated gradual improvement of bilateral lower extremity strength and return of bowel and bladder function. Follow-up at 16 months post-operatively demonstrated significant improvement in pain and neurological symptoms, with no signs of infection recurrence.

Conclusions: This case demonstrates the importance of aggressive surgical management of *M. chelonae* spondylodiscitis. Early aggressive surgical management in combination with antibiotics may improve clinical outcomes for these patients.

Keywords: *Mycobacterium chelonae* (*M. chelonae*); discitis; spine; case report

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Introduction

Spondylodiscitis is an infection of the intervertebral disc that accompanies secondary osteomyelitis of the endplates of the vertebrae (1). It is an infection of the spine with a previously noted incidence of 0.4–2.4/100,000 per year although newer studies have claimed rates as high as 12/100,000 due to improved diagnostic methods (2,3). Common pathogens associated with spondylodiscitis include *Staphylococcus species*,

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Escherichia coli, *Proteus*, *Klebsiella*, *Pseudomonas aeruginosa*, *and Mycobacterium tuberculosis* with *Staph aureus* being the most common culprit (1). Gram negative bacteria are often seen in immunocompromised patients, diabetics, intravenous (IV) drug use, and procedures or infections of the genitourinary of gastrointestinal tracts. Endemic regions have higher presentations of spondylodiscitis due to tuberculosis (TB) which is also typical of patients who are immunosuppressed, prisoners, IV drug users, or homeless (3). Sequelae of spondylodiscitis include reduced mobility, instability, and compression of neural structures, often necessitating surgical intervention. Severity of this disease is primarily dependent on the infectious components of the disease as well as the origins in the viscera, such as cardiac or gastrointestinal organs. It is of vital importance to correctly identify the causative pathogen in the initial stages of infection to determine appropriate and prompt management. The most common manifestation of this condition is back and neck pain. Pain and dissemination of the neurological structures are principal effects of the degeneration of the vertebral motion segments, a phenomenon known as segmental instability. Abscesses may accompany this infection and are typically localized to either the spinal canal or track distally into the musculature of the pelvis.

Blood cultures and magnetic resonance imaging (MRI) remain the gold standard in diagnosing spondylodiscitis. However, blood cultures are often negative. Therefore, management of this disease has certainly posed difficulties, especially when caused by uncommon microbes. Spondylodiscitis secondary to non-tuberculous mycobacteria (NTM) is exceptionally rare, and no consensus exists regarding the optimum antibiotic regimen or treatment duration for spinal infections secondary to NTM. The patient of interest in this case is a 32-year-old male presenting with spondylodiscitis due to *Mycobacterium chelonae* (*M. chelonae*). We present the following case in accordance with the CARE reporting checklist (available at https://jss.amegroups.com/article/view/10.21037/jss-22-3/rc).

Case presentation

A 32-year-old male presented to the emergency department with a 3-month history of gradually progressing low back pain. Pain initially radiated down his posterolateral right leg with associated numbness and tingling. Five days prior to presentation to the emergency room, he began experiencing gradually progressive diffuse weakness, loss of bowel and bladder control, complete left-sided Bell's palsy, and dysphagia. Patient had no underlying medical diseases beyond developmental delay. Vital signs were stable on presentation. Physical examination showed a thin, chronically malnourished patient. Musculoskeletal examination was significant for severe atrophy of bilateral lower extremities with the right side greater than left, limited lumbosacral extension, lumbar pain with extension, inability to ambulate secondary to pain, and diminished strength and sensation in the bilateral lower extremities. Rectal examination demonstrated significant loss of rectal tone, concerning of cauda equina. Examination was also significant for a newly developed sacral ulcer.

The patient was admitted to the hospital for further evaluation. Advanced imaging of the lumbosacral spine including computerized tomography (CT) and MRI, as seen in *Figures 1–4*, showed bone destruction and disc enhancement at the level of L5-S1, consistent with chronic spondylodiscitis. Imaging also showed severe compression of the spinal canal and bilateral neural foramina, adjacent phlegmonous changes in the epidural space with reactive enhancement surrounding the posterior elements of L5, increasing fluid within the intervertebral disc space, decreased size of epidural fluid collection superficial to the L5-S1 level, and a right psoas abscess measuring 2.3 cm × 1.2 cm. C-reactive protein (CRP) and sedimentation rate were elevated at 10.7 mg/L and 60 mm/h, respectively. Complete blood count (CBC) with differential was significant for a hemoglobin of 11.7 g/dL, hematocrit of 38.5%, 72.5% neutrophils, and 18.1% lymphocytes. Comprehensive metabolic panel (CMP) was within normal limits.

Within 24 h, the patient underwent L4-S1 decompression and fusion with left-sided iliac crest bone grafting, and intraoperative biopsy. Blood culture obtained on admission showed no growth after 42 days. Intraoperative tissue cultures grew *Mycobacterium chelonae* resistant to cefoxitin, imipenem, trimethoprim-sulfamethoxazole, and ciprofloxacin, with susceptibility to amikacin, doxycycline, linezolid, tobramycin, and clarithromycin. Microscopic imaging with acid fast staining of *M. chelonae* can be seen in *Figure 5*. The patient was initially treated with IV vancomycin and piperacillin-tazobactam. Per infectious disease recommendations, the patient was transitioned to a 6-week course of ceftriaxone, doxycycline, and rifampin. This provided empiric coverage for brucellosis, which was continued despite negative antigen due to the chronicity of his condition. The psoas abscess was drained percutaneously under computed tomography guidance, and cultures showed no growth. His immediate post-operative course showed

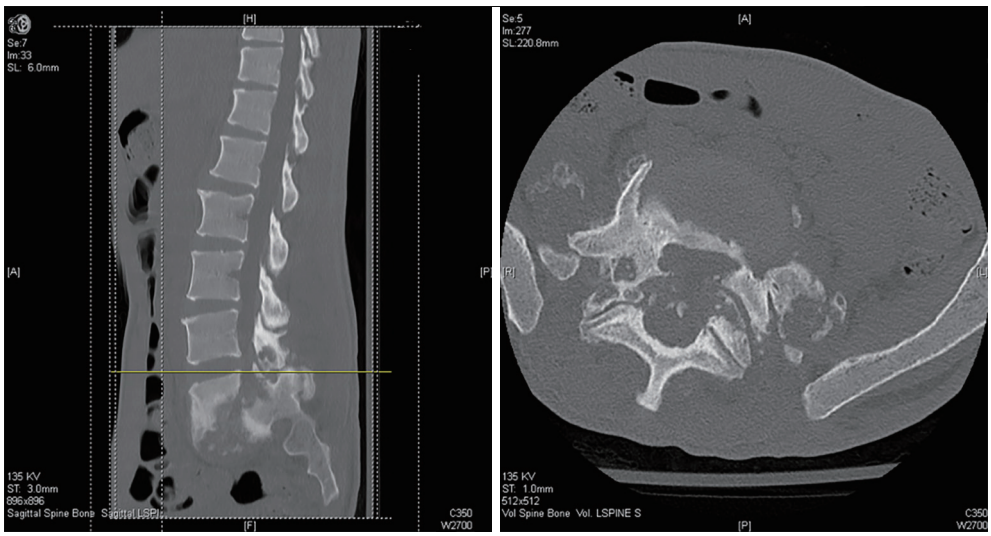

Figure 1 CT of lumbar spine (L4-5) on presentation. CT, computerized tomography.

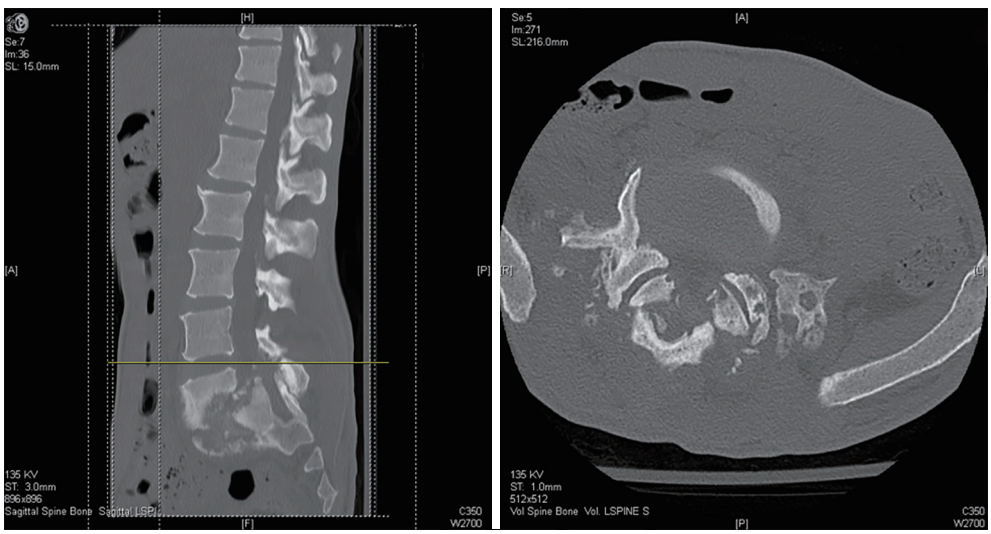

Figure 2 CT of lumbar spine (L5-S1) on presentation. CT, computerized tomography.

gradual improvement with respect to his lower extremity symptoms. However, he continued to have severe dysphagia requiring percutaneous endoscopic gastrostomy (PEG) tube placement 1 week after surgery. The patient was discharged to a rehabilitation facility 9 days after admission.

Approximately 1 week after discharge, he developed significant fevers and a cough, and was found to have aspiration pneumonia. He was subsequently hospitalized for 3 weeks. During this time, he underwent irrigation and debridement of his sacral wound. His dysphagia improved during his hospitalization and he was transitioned to oral feeds. Following resolution of his symptoms, he was discharged to a rehabilitation facility on a 6-week course of amikacin, doxycycline, and clarithromycin.

Repeat CT-guided biopsy 8 months post-operatively showed no culture growth. MRI of his lumbosacral spine was repeated at 15 months post-operative which can be seen in *Figures 6*,7. It showed a considerable decrease in enhancement, edema and inflammatory changes of the disc and endplates. In addition, it showed multilevel disc

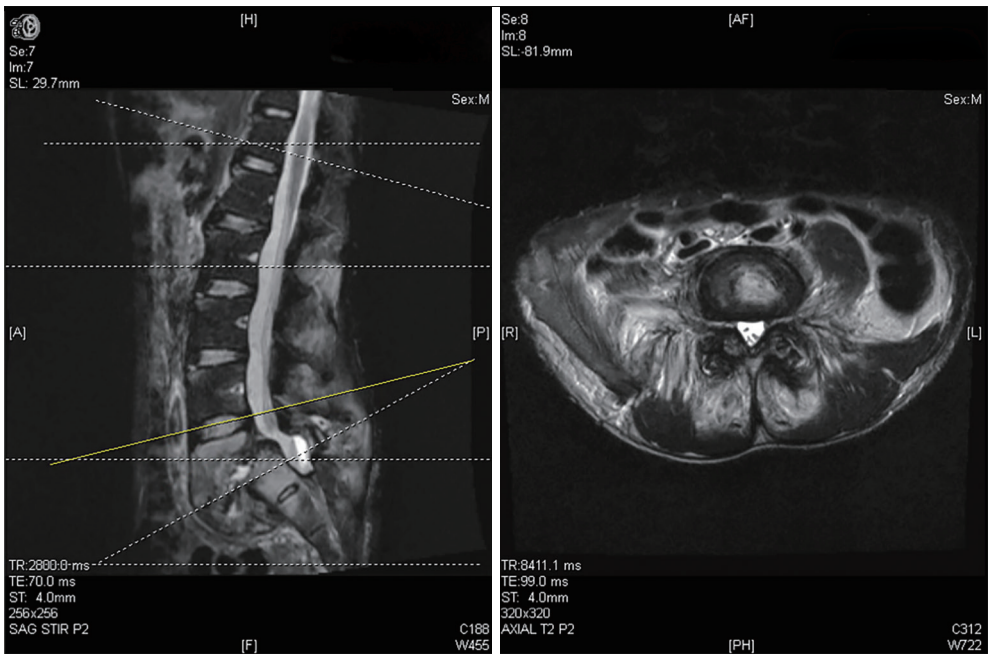

Figure 3 T2-weighted MRI imaging of lumbar spine (L4-5) on presentation. MRI, magnetic resonance imaging; TR, repetition time; TE, echo time.

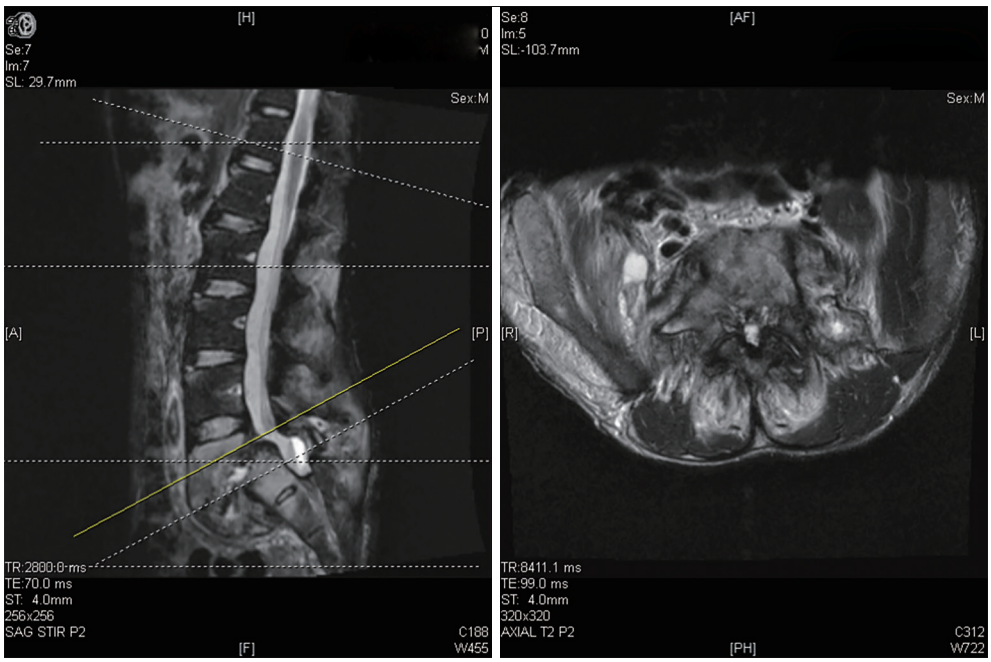

Figure 4 T2-weighted MRI imaging of lumbar spine (L5-S1) on presentation. MRI, magnetic resonance imaging; TR, repetition time; TE, echo time.

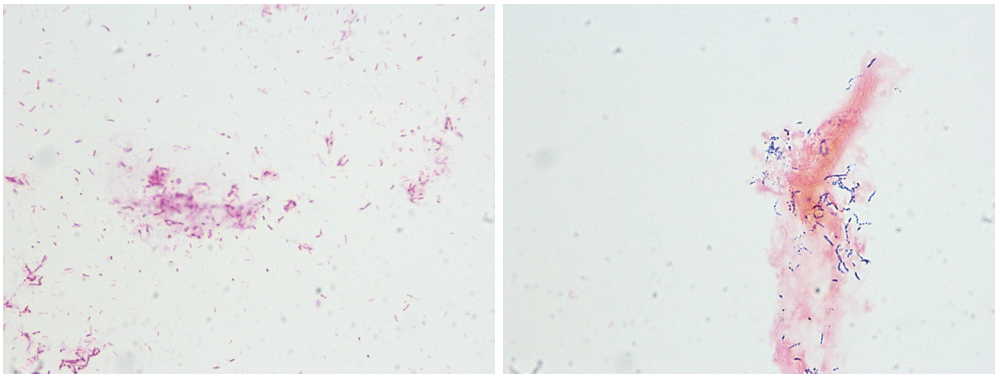

Figure 5 Acid fast stain of *Mycobacterium chelonae* (magnification =100×).

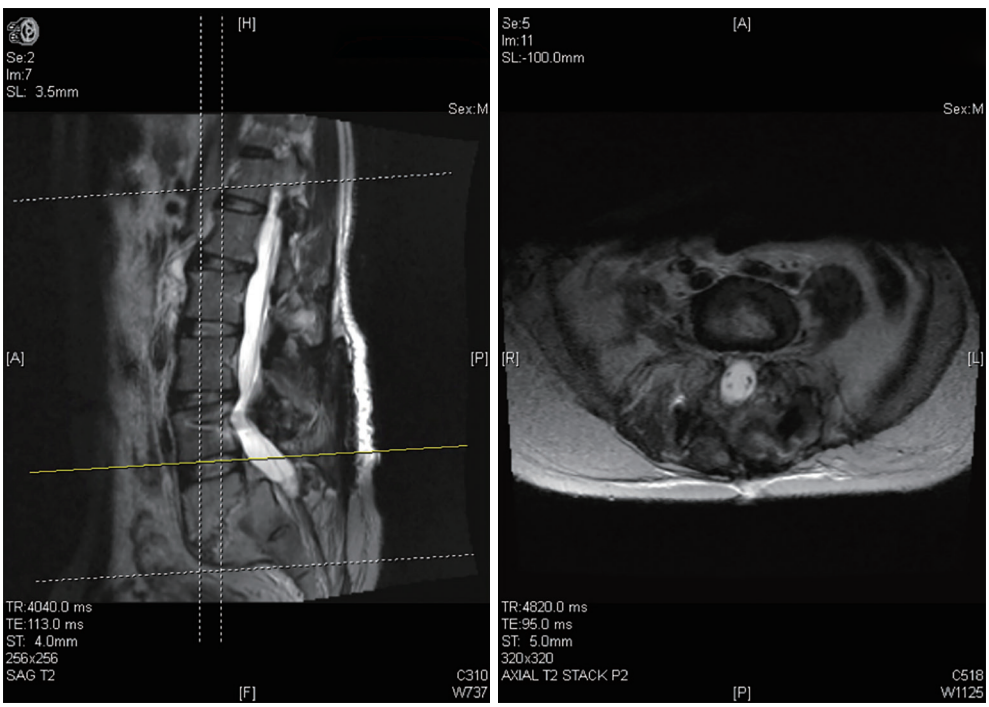

Figure 6 Repeat T2-weighted MRI imaging of lumbar spine (L4-5) at 15 months post-operation. MRI, magnetic resonance imaging; TR, repetition time; TE, echo time.

desiccation, L3-L4 disc bulge with progressive central canal narrowing, as well as no epidural or compressing fluid collection. His 16-month post-operative visit showed significant improvement in overall strength, ability to ambulate at home with minimal assistance, full control over bowel and bladder function, and resolution of his lower back pain. He had been followed by infectious disease and underwent numerous transitions in antibiotic regimen secondary to adverse medication effects and non-compliance with oral agents. His antibiotic regimen at present consists of IV azithromycin and tigecycline, which is to be continued for a total course of 18 months. No evidence of neurological sequelae has been observed or reported during his 16 months follow-up period.

Patient was pleased with the outcome of both his surgery and final antibiotic regimen. Side effects of some antibiotics

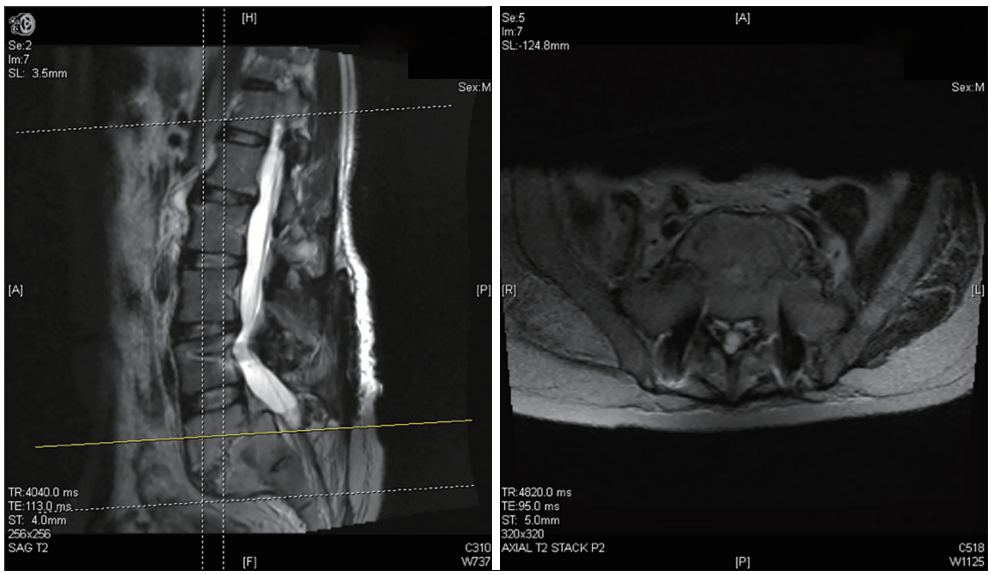

Figure 7 Repeat T2-weighted MRI imaging of lumbar spine (L5-S1) at 15 months post-operation. MRI, magnetic resonance imaging; TR, repetition time; TE, echo time.

used were not pleasant, but he is happy with the end results. He returned to pre-infection level of activity and is caring for himself independently at home. His bowel and bladder control improved, and he endorsed improved global strength.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

NTM infections are most commonly localized to the lungs, lymph nodes, subcutaneous tissues, and the skin. Disease due to *M. chelonae* usually occurs in immunosuppressed patients (4). Vertebral infection due to *M. chelonae* is extremely rare.

Diagnosis of spondylodiscitis is challenging as the presenting symptoms are usually non-specific, which often results in delayed diagnosis. In general, diagnosis is based on radiological, clinical, laboratory, and microbiological findings (1). While plain radiographs are the first-line imaging for patients with non-specific symptoms, MRI remains the gold standard for detecting spondylodiscitis, and CT scans are reserved for those with contraindications to MRI. Laboratory testing includes blood testing for leukocyte count, CRP, and sedimentation rate. While acute infection often demonstrates elevated inflammatory markers, they may be within normal limits in chronic cases. CRP count and sedimentation rate are non-specific tests, although they may be used to follow response to antibiotic therapy (1). Microbiological testing is often initiated in the form of blood cultures. However, this is not reliable, as the pathogen is only identified in 25–59% of blood cultures (5). Further histopathological investigation can be achieved via CT-guided fine needle biopsy or surgical biopsy. Given the complexity involved in diagnosing spondylodiscitis and identifying the causative pathogen, delay of 2–12 weeks between diagnosis and treatment initiation is not uncommon (5).

The primary goals of treatment in spondylodiscitis include elimination of the infection, restoration of spinal functionality, and improvement of pain or other neurological symptoms (1). Identification of the causative pathogen and susceptibility testing guide the initiation of antibiotic treatment. Of note, first line *M. tuberculosis* drugs have no role in the treatment of *M. chelonae*. Clarithromycin appears to be the mainstay of therapy for rapidly growing mycobacterium (RGM) such as *M. chelonae*, though combination antimicrobial therapy is recommended due

to inducible resistance to macrolides with monotherapy. Additional agents shown to have good *in vitro* activity against *M. chelonae* include linezolid, amikacin, tobramycin, imipenem, and tigecycline (6). Of note, limited clinical data exists regarding the use of tigecycline, and it is only recommended in the absence of other options (7). Several of these recommended agents were used in the treatment of our patient but due to several medication adverse effects and patient noncompliance, the final treatment regimen consisted of tigecycline and azithromycin. There is no consensus on the recommended duration of pharmacologic treatment for *M. chelonae* osteomyelitis, though it is recommended that patients receive at least 6–12 months of combination antimicrobial therapy (6).

Literature review identified only two other cases of localized vertebral osteomyelitis due to *M. chelonae*. One case report demonstrated a successful treatment with antimicrobial therapy and spinal instrumentation and fusion, similar to the operative management seen in our case. However, the patient's treatment course was complicated by recurrent psoas abscesses and extensive antibiotic resistance requiring multiple surgical drainage procedures and antibiotic alterations over a 33-month period (8). Resolutions of symptoms occurred while using ciprofloxacin, clarithromycin, and amikacin which was eventually discontinued and replaced with doxycycline. Another study reported a case of *M. chelonae* vertebral osteomyelitis in a patient with a history of IV drug abuse and hepatitis C infection (6). Initial clinical improvement was seen following decompressive laminectomy of T7-T8, though this was later complicated by regression of neurologic status, which required fusion of T7-T10. Following this second operation, the patient only saw mild improvement in lower extremity strength. Specific antibiotic therapy used in this case is not stated. Final antibiotic therapy for our patient was azithromycin and tigecycline after tobramycin was discontinued due to ototoxicity. This differed from one of the cases mentioned above, which used a macrolide, tetracycline, and a fluroquinolone.

Our case demonstrates the importance of aggressive surgical management of *M. chelonae* spondylodiscitis. Although current literature indicates surgical treatment for spondylodiscitis, prior reports differ either in post-operative complications or type of initial surgical intervention (6,8). In our case, initial aggressive operative treatment resulted in restoration of vertebral column stability and significant improvement in neurological symptoms. Furthermore, early surgical intervention provided us with accurate pathological diagnosis and antimicrobial susceptibility testing to guide appropriate pharmacologic management.

Conclusions

We describe a case of spondylodiscitis secondary to *Mycobacterium chelonae* that was treated with initial aggressive surgical management and antibiotics that resulted in significant recovery. Initial aggressive surgical intervention may provide better outcomes for patients than conservative management.

Acknowledgments

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://jss.amegroups.com/article/view/10.21037/jss-22-3/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jss.amegroups.com/article/view/10.21037/jss-22-3/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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