# CASE REPORT



# > THE PATIENT

23-year-old woman

# SIGNS & SYMPTOMS

- Radiating low back pain
- History of urinary symptoms
- Past surgery for scoliosis

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### **THE CASE**

A 23-year-old immunocompetent woman was referred to our spinal clinic with a 6-month history of low back pain that radiated to her right flank, buttock, and groin. She'd had intermittent urinary problems, including mild dysuria and frequency, and had been treated with antibiotics for a presumed urinary tract infection on 3 previous occasions, but her pain gradually increased and eventually became constant.

The patient had no history of fever, malaise, or weight loss. She denied consuming unpasteurized milk or undercooked poultry, and hadn't recently experienced diarrhea or vomiting.

Eight years earlier, she had undergone anterior fusion of her spine for idiopathic scoliosis. At that time, she was at Risser grade 1, and her Cobb angle was 50°; metallic instrumentation was implanted at T10 to L3 to prevent progression of the scoliosis. Her recovery had been uneventful.

During examination, her temperature, pulse, respiratory rate, blood pressure, and nervous system were all normal. Her hips appeared normal, as well, and a straight leg raise was negative bilaterally. The patient had mild midline lumbar tenderness. Spinal range of movement revealed decreased flexion and mild pain.

X-rays (FIGURE 1) showed no changes in the previous metalwork in her spine. There

#### FIGURE 1 X-rays showed no change in implanted instrumentation



Eight years earlier, the patient had undergone anterior fusion of the spine for idiopathic scoliosis. X-rays showed no change in the implanted metal instrumentation.

was decreased disk height at the L3/4 level, but no significant bony erosion or soft-tissue shadows. Laboratory testing revealed a C-reactive protein (CRP) level of 240 mg/dL (normal, <1 mg/dL) and her erythrocyte sedimentation rate (ESR) was 102 mm/h—more than 5 times higher than it should have been.<sup>1</sup> The patient had a normal peripheral white cell count (WCC). Midstream urine cultures were negative.

The patient was admitted to the hospital for further work-up. Magnetic resonance imaging (MRI) of the lumbar spine showed gross abnormality at the L3-L4 disk level with erosion of the end plates, fluid in the disk space, marked enhancing edema, and mild surrounding soft-tissue edematous changes, but no evidence of any epidural abscess (FIGURE 2). The patient had a fluoroscopy-

guided needle biopsy of the disk on the same day and received intravenous (IV) ceftriaxone 2 g twice a day. Blood and urine cultures were negative.

#### **THE DIAGNOSIS**

We suspected our patient had spondylodiscitis, an infection of the spine that includes spondylitis (inflammation of the vertebrae) and discitis (inflammation of the vertebral disk space). After 48 hours, the biopsy sample grew *Salmonella typhimurium* and confirmed the diagnosis. The organism was sensitive to ceftriaxone and ciprofloxacin; parenteral ceftriaxone was continued and the patient wore a thoracolumbar brace for immobilization. For 3 days, her inflammatory marker levels were checked daily, then every other day for the rest of that first week, and then 2 more times in the following week.

#### DISCUSSION

Thoracic and lumbar vertebrae are the most common sites of spondylodiscitis.<sup>2</sup> Spondylodiscitis accounts for 3% to 5% of pyogenic osteomyelitis in patients in developed countries.<sup>3</sup> The incidence of pyogenic spondylodiscitis may be rising due to an increase in the number of elderly and immunocompromised patients, as well as a rise in invasive medical procedures.<sup>4-6</sup>

If left untreated, spondylodiscitis can spread longitudinally (involving the adjacent levels), posteriorly (causing bacterial meningitis, abscess formation, and cord compromise), or anteriorly (causing paravertebral abscess). Untreated spondylodiscitis can also send distant infective emboli and cause endocarditis<sup>7.9</sup> or mycotic abdominal aneurysm.<sup>10</sup>

Historically, mortality in patients with vertebral osteomyelitis has been as high as 25%.<sup>11</sup> The combination of earlier diagnosis, antibiotics, and surgical debridement and stabilization has decreased mortality to less than 15%.<sup>12-14</sup>

**Risk factors for spondylodiscitis** include male sex, IV drug abuse, diabetes, morbid obesity, having had a genitourinary or spinal procedure, and being immunocompromised (eg, from alcohol abuse, malignancy, organ transplantation, chemotherapy, or corticosteroid use).<sup>12,15,16</sup>

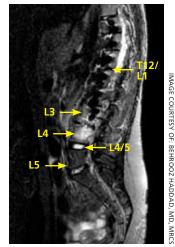
Gram-positive organisms cause most spine infections in both adults and children, with 40% to 90% caused by *Staphylococcus aureus*.<sup>17</sup> Gram-negative organisms (*Escherichia coli, Pseudomonas*, and *Proteus*), which can also cause spondylodiscitis, typically occur after genitourinary infections or procedures. IV drug abusers are prone to *Pseudomonas* infections.<sup>18</sup> Anaerobic infections may be seen in patients with diabetes or after penetrating trauma.<sup>15</sup> Salmonella species can cause spondylodiscitis, especially in patients with sickle cell disease from an intestinal source.<sup>19</sup>

*Mycobacterium tuberculosis* is the most common nonpyogenic infecting agent that also can cause spondylodiscitis. Infection caused by tuberculosis (TB) has had a recent resurgence with resistant strains, especially in patients with human immunodeficiency virus.<sup>15</sup> Although less than 10% of patients with TB have skeletal involvement, 50% of the skeletal involvement oc-

curs in the spine.  $^{\scriptscriptstyle 15}$ 

The clinical presentation of spondylodiscitis depends on the location of the infection. the virulence of the organism, and the immune status of the patient. Discitis can present as pain in the back, hip, abdomen (especially in children<sup>20</sup>) and, occasionally, with meningeal involvement.<sup>11</sup> Patients with discitis often have a normal temperature.<sup>15,21</sup> In patients with discitis, the patient's WCC will be normal, but the ESR is almost always elevated.15,22 Suspect spondylodiscitis in patients who present with persistent or increasing pain 3 to 4 weeks after back surgery. For such patients,

#### FIGURE 2 MRI told another story



Imaging revealed abnormalities at L3-L4 disk, with destruction of the end plates and intervertebral disc, fluid in the disk space, marked enhancing edema, and mild surrounding soft-tissue edematous changes. L4-L5 disk was normal with an intact end plate. T12-L1 had satisfactory fusion. measure inflammatory markers and order imaging of the spine.

X-ray findings for patients with spondylodiscitis will include osteolysis and end plate erosions (early) and narrowing and collapse of the disk space (late). (In TB, relative preservation of the disk spaces is seen, with significant vertebral destruction.)

MRI is the modality of choice for diagnosis and assessment of suspected spondylodiscitis because it can provide imaging of the soft tissue, neural elements, and bony changes with a high sensitivity and specificity.<sup>23</sup> Once infection is suspected, the diagnosis should be confirmed by fluoroscopic- or computed tomography-guided biopsy before starting antibiotic treatment.

#### Long-term antibiotics are required to prevent recurrence

IV antibiotics are the mainstay of treatment for spondylodiscitis;<sup>24</sup> the specific drug used will depend upon the organism identified. Patients typically receive 2 to 6 weeks of IV therapy. Then, once the patient improves and inflammatory markers return to normal levels, the patient receives a course of oral antibiotics for 2 to 6 more weeks. Grados et al<sup>19</sup> found recurrence rates of 10% to 15% for patients who were treated 4 to 8 weeks compared to 3.9% in those treated for 12 weeks or longer; therefore, a total duration of 12 weeks is commonly chosen.<sup>25-28</sup>

To minimize the risk of spondylolisthesis, kyphosis, and fractures of the infected bone, patients are advised to rest and the spine is often immobilized with a spinal brace. Surgery may be needed if antibiotics are not effective, or for patients who develop complications such as fluid collection, neurologic deficits, or deformity.

**I Our patient's pain improved** after 2 weeks and she became more comfortable wearing the thoracolumbar brace. Her CRP and ESR also improved and there was no radiologic evidence of fluid collection. The patient was discharged with a peripherally inserted central catheter in place and received IV ceftriaxone for 6 more weeks at home. This was followed by 4 weeks of oral ciprofloxacin 750 mg twice daily, thereby

completing a 12-week course of antibiotics.

Our patient's response to treatment was monitored clinically and the inflammatory markers were checked weekly after discharge until the end of treatment and at 6 and 12 months after start of treatment. At 12 months, our patient's CRP was <1 mg/dL and ESR was 22 mm/h. One year later, our patient remained asymptomatic with normal inflammatory marker levels and no evidence of recurrence.

#### THE TAKEAWAY

Spondylodiscitis is an important differential diagnosis of lower back, flank, groin, and buttock pain. It's important to be aware of this diagnosis, especially in patients who have risk factors such as IV drug abuse, diabetes, and morbid obesity. Although previous spinal surgery is a risk factor, spondylodiscitis should be considered in patients with persistent back pain even if they haven't had spinal surgery. It can be present even when there is no tenderness over the spinous process or any fever.

Checking inflammatory markers is a reasonable next step if a patient's pain does not resolve after at least 4 weeks. If levels of inflammatory markers such as CRP and ESR are elevated and symptoms continue, MRI can confirm or rule out the presence of spondylodiscitis. Treatments include orthotic support, antibiotics, and surgical intervention when complications arise.

#### References

- 1. Miller A, Green M, Robinson D. Simple rule for calculating normal erythrocyte sedimentation rate. *Br Med J*. 1983;286:266.
- Calhoun JH, Manring MM. Adult osteomyelitis. Infect Dis Clin North Am. 2005;19:765-786.
- Sobottke R, Seifert H, Fätkenheuer G, et al. Current diagnosis and treatment of spondylodiscitis. *Dtsch Arztebl Int.* 2008;105:181-187.
- Beronius M, Bergman B, Andersson R. Vertebral osteomyelitis in Göteborg, Sweden: a retrospective study of patients during 1990-95. Scand J Infect Dis. 2001;33:527-532.
- Digby JM, Kersley JB. Pyogenic non-tuberculous spinal infection: an analysis of thirty cases. J Bone Joint Surg Br. 1979;61: 47-55.
- Gouliouris T, Aliyu SH, Brown NM. Spondylodiscitis: update on diagnosis and management. J Antimicrob Chemother. 2010;65 suppl 3:iii11-iii24.
- Aoki K, Watanabe M, Ohzeki H. Successful surgical treatment of tricuspid valve endocarditis associated with vertebral osteomyelitis. *Ann Thorac Cardiovasc Surg.* 2010;16:207-209.
- 8. Gonzalez-Juanatey C, Testa-Fernandez A, Gonzalez-Gay MA.

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, Risk factors for spondylodiscitis include IV drug abuse, diabetes, morbid obesity, and having had a genitourinary or spinal procedure.

#### CONTINUED FROM PAGE 642

Septic discitis as initial manifestation of streptococcus bovis endocarditis. *Int J Cardiol*. 2006;108:128-129.

- Morelli S, Carmenini E, Caporossi AP, et al. Spondylodiscitis and infective endocarditis: case studies and review of the literature. *Spine (Phila Pa 1976)*. 2001;26:499-500.
- Learch TJ, Sakamoto B, Ling AC, et al. Salmonella spondylodiscitis associated with a mycotic abdominal aortic aneurysm and paravertebral abscess. *Emerg Radiol*. 2009;16:147-150.
- Guri JP. Pyogenic osteomyelitis of the spine. J Bone Joint Surg Am. 1946;28:29-39.
- Carragee EJ. Pyogenic vertebral osteomyelitis. J Bone Joint Surg Am. 1997;79:874-880.
- Garcia A Jr, Grantham SA. Hematogenous pyogenic vertebral osteomyelitis. J Bone Joint Surg Am. 1960;42-A:429-436.
- Eismont FJ, Bohlman HH, Soni PL, et al. Pyogenic and fungal vertebral osteomyelitis with paralysis. J Bone Joint Surg Am. 1983;65:19-29.
- Tay BK, Deckey J, Hu SS. Spinal infections. J Am Acad Orthop Surg. 2002;10:188-197.
- Krogsgaard MR, Wagn P, Bengtsson J. Epidemiology of acute vertebral osteomyelitis in Denmark: 137 cases in Denmark 1978-1982, compared to cases reported to the National Patient Register 1991-1993. Acta Orthop Scand. 1998;69:513-517.
- Francis X. Infections of spine. In: Canale ST, Beaty JH, eds. *Campbell's Operative Orthopaedics*. 11th ed. New York, NY: Mosby; 2007:2241.
- 18. Roca RP, Yoshikawa TT. Primary skeletal infections in heroin

users: a clinical characterization, diagnosis and therapy. *Clin* Orthop Relat Res. 1979;(144):238-248.

- Grados F, Lescure FX, Senneville E, et al. Suggestions for managing pyogenic (non-tuberculous) discitis in adults. *Joint Bone Spine*. 2007;74:133-139.
- Cheyne G, Runau F, Lloyd DM. Right upper quadrant pain and raised alkaline phosphatase is not always a hepatobiliary problem. Ann R Coll Surg Engl. 2014;96:118E-120E.
- Varma R, Lander P, Assaf A. Imaging of pyogenic infectious spondylodiskitis. *Radiol Clin North Am.* 2001;39: 203-213.
- Lehovsky J. Pyogenic vertebral osteomyelitis/disc infection. Baillieres Best Pract Res Clin Rheumatol. 1999;13:59-75.
- Modic MT, Feiglin DH, Piraino DW, et al. Vertebral osteomyelitis: assessment using MR. *Radiology*. 1985;157:157-166.
- Amritanand R, Venkatesh K, Sundararaj GD. Salmonella spondylodiscitis in the immunocompetent: our experience with eleven patients. *Spine (Phila Pa 1976)*. 2010;35:E1317-E1321.
- Govender S. Spinal infections. J Bone Joint Surg Br. 2005;87:1454-1458.
- 26. Lam KS, Webb JK. Discitis. Hosp Med. 2004;65:280-286.
- Gasbarrini AL, Bertoldi E, Mazzetti M, et al. Clinical features, diagnostic and therapeutic approaches to haematogenous vertebral osteomyelitis. *Eur Rev Med Pharmacol Sci.* 2005;9: 53-66.
- Cottle L, Riordan T. Infectious spondylodiscitis. J Infect. 2008;56:401-412.

