Iatrogenic spondylodiscitis

Case report and review of literature

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Iatrogenic intervertebral disc space infection is encountered following microsurgical discectomy, percutaneous laser disc decompression, automated percutaneous lumbar nucleotomy operations, and discography. The purpose of this paper is to present a case report and review the literature on the uncommon origins of pyogenic spondylodiscitis and to emphasize the significance of prophylactic antibiotic therapy following transrectal ultrasonography-guided needle biopsy of the prostate (TUGNBP). According to the authors, this is the first reported case of pyogenic spondylodiscitis as a complication of TUGNBP in the English language literature.

KEY WORDS • pyogenic spondylodiscitis • vertebral osteomyelitis • complication

Hematogenous infections of the spine have been described as discitis, spondylodiscitis, spondylitis, vertebral pyogenic osteomyelitis, and epidural abscess. This varied nomenclature creates confusion in those who read the literature.²³ In general, hematogenous pyogenic infection of the spine has been referred to as either “spondylodiscitis” or “pyogenic osteomyelitis.” Pure discitis has been encountered rarely. Pyogenic spondylodiscitis is an uncommon primary infection of the nucleus pulposus with secondary involvement of the cartilaginous endplate and VB. Its primary incidence represents only 2 to 4% of all cases of bone infection and 8 to 16% of cases of hematogenously spread osteomyelitis.⁴² Most cases occur in the lumbar spine and, in fact, may occur spontaneously or following some procedures, as outlined in Table 1. Iatrogenic intervertebral disc space infection is encountered following microsurgical discectomy, percutaneous laser disc decompression,¹⁷ automated percutaneous lumbar nucleotomy,¹⁴ and discography.^{22,38}

The purpose of this report is to review the literature for uncommon origins of pyogenic spondylodiscitis.

CASE REPORT

History and Examination. This 53-year-old man was admitted to the Neurosurgery Service on January 10, 2001 suffering from severe back and leg pain and bilateral lower-extremity weakness. He had been experiencing

lower-back and leg pain for 4 weeks. Since January 5, 2001, he had been unable to walk because of progressive lower-extremity weakness. His medical and surgical histories were unremarkable, except for the presence of poorly controlled DM for 4 years and high serum PSA levels for 3 years. Because of the high serum PSA levels, he had undergone an uncomplicated TUGNBP in October 1998. The histopathological features were consistent with chronic prostatitis and adenomyomatous hyperplasia. Because the high serum PSA levels persisted he underwent a second biopsy on December 5, 2000. At the time of the biopsy procedure the patient was fasting because of Ramadan, the holy month for Muslims. After this second biopsy, he did not take the prophylactic antibiotic agents. He had, however, taken the prophylactic antibiotics following the first biopsy procedure. The day after the second biopsy, the patient experienced intermittent high fever and fatigue. A urine analysis and hemogram showed hematuria and an increased white blood cell count (14,000 cells/ml), respectively. A week following the biopsy, his back pain started, became severe, and progressed to an unbearable level in just a few days. Two weeks after the biopsy he experienced bilateral groin pain and muscle cramps in the lower extremities. During this period, the intermittent high fever persisted and he was treated for his symptoms.

Four days before admission to our service, he began to experience bilateral progressive lower-extremity weakness. On neurological deterioration, he underwent MR imaging of the lumbar spine with contrast enhancement. The T₁-weighted contrast-enhanced sagittal and axial MR images (Fig. 1) revealed spondylodiscitis with epidural abscess at the L2–3 level together with L2–3 listhesis and osteomyelitis of the L-2 and L-3 VBs. The epidural ab-

Abbreviations used in this paper: DM = diabetes mellitus; MR = magnetic resonance; PSA = prostate specific antigen; TUGNBP = transrectal ultrasonography-guided needle biopsy of the prostate; VB = vertebral body.

TABLE 1

*Literature review of cases involving pyogenic spondylodiscitis**

Authors & Year	Incident
Kerdiles, et al., 1975	following translumbar aortography
Bouvenot, et al., 1978	following spinal injury (L-1 fracture)
Herbiere, et al., 1981	after insertion of Mobin-Uddin caval umbrella filter
Baudrillard, et al., 1985	following embolization of extramedullary intraspinal arteriovenous fistula
Fieve, et al., 1986	following translumbar aortography
Griffet, et al., 1986	following lumbar puncture
Brunet, et al., 1989	following tracheal intubation
Durance, 1989	following urinary catheter placement
Cabezudo, et al., 1990	following placement of percutaneous lumbo-peritoneal shunt for benign intracranial hypertension
Cailleux, et al., 1991	following genital prolapse op (fixing uterus to promontory of sacrum)
MGH Case Records, 1991	following pulmonary op
Bogaert, et al., 1992	following infection of abdominal aortic graft
Hummel, et al., 1993	after heart transplantation
Marlier, et al., 1993	following colonoscopy
Schulze & Mayer, 1995	following stab wound & vertebral fracture
Fonga-Djimi, et al., 1996	following perforation due to swallowed radiolucent foreign body
Hamilton & Stambough, 1996	following transpedicular screw fixation
Mascalchi, et al., 1996	following percutaneous femoral artery catheterization
Eisenberger, et al., 1998	following renal transplantation
Ortiz, et al., 1998	following allogenic bone marrow transplantation
Plubel, et al., 1998	after peridural infiltration w/ prednisolone
Kikuchi, et al., 1999	following femoral vein catheterization for hemodialysis
van Ooij, et al., 1999	following removal of fishbone
Chiapparini, et al., 2000	following lumbar epidural anesthesia
Junquera Crespo, et al., 2000	following prostatic biopsy
Porter & Wray, 2000	following long-term antibiotic (tetracycline) therapy
Coapes & Roysam, 2001	caused by epidural catheter use

* MGH = Massachusetts General Hospital.

scsis at the L2–3 level was compressing the dural sac and the L-3 nerve roots bilaterally. In addition, the infectious process extended into the paravertebral muscles at the same level, and multiple microabscesses developed in both psoas muscles. Because of chronic back pain, the patient underwent additional MR imaging studies of the lumbar spine 6 months before the second biopsy procedure, which demonstrated only degenerative changes (Fig. 2).

Operation and Postoperative Course. Because his lower-extremity weakness progressively worsened, a right L2–3 hemilaminectomy, drainage of epidural and intradiscal abscesses, and an L2–3 discectomy were performed. The disc material was soft and liquefied. Following surgery, he was able to walk without a cane. The culture and antibiogram of the infected disc material was positive for *Escherichia coli*. Intravenous ceftriaxone sodium (1 g twice daily) and gentamycin (160 mg/day) were administered immediately following sensitivity testing. On postoperative Day 7, the patient's back and leg pain improved,

Fig. 1. Axial (A) and sagittal (B) T₁-weighted MR images of the lumbar spine obtained with gadopentetate, revealing spondylodiscitis with epidural abscess at the L2–3 level along with L2–3 listhesis and osteomyelitis of both the L-2 and L-3 VBs. The epidural abscess at the L2–3 level caused compression of the dural sac and L-3 nerve roots bilaterally. In addition, the infectious process extended into the paravertebral muscles at the same level and multiple microabscesses developed in both psoas muscles.

and he was discharged home with a thoracolumbosacral orthosis on Day 8 postsurgery. At the 3-year follow-up examination, he was neurologically intact and free of pain.

DISCUSSION

We reported on a case of pyogenic spondylodiscitis that occurred following a TUGNBP complication. This is the first reported case of discitis following a TUGNBP in the English language literature. Genitourinary infections are the most common coexisting infection in patients with spinal osteomyelitis.⁵⁰ In this report, pyogenic spondylo-

Fig. 2. Magnetic resonance image of the lumbar spine obtained 6 months prior to biopsy, demonstrating only degenerative changes.

discitis occurred following iatrogenic inoculation of the bacteria into the prostate gland. Although genitourinary infections are the most common coexisting infections in patients with pyogenic spondylodiscitis,^{4,40,47} the coexistence of spondylodiscitis and endocarditis³⁶ and other infections⁴⁶ have also been reported.

Pyogenic discitis has occurred due to urinary tract infection,⁴ and acute bacterial prostatitis⁴⁷ has been reported. Although pyogenic spondylodiscitis in the present case did not originate from primary prostate infection, it did occur after an iatrogenic bacterial inoculation into the prostate gland by a needle passing through the highly contaminated rectum. Thompson, et al.,⁴⁸ reported that bacteremia develops in every patient after transrectal needle biopsy: 27% of their patients demonstrated symptoms and 87% had a urinary tract infection. A prophylactic antibiotic regimen reduced the bacteremia rate to 53% and prevented urinary tract infection. Although minor complications following TUGNBP appear to be extremely frequent, major complications are rare.⁴³ Most attention has been given to the incidence of infectious complications. Rodriguez and Terris⁴³ reported an infectious complication rate of 2.5%, and all patients were treated on an outpatient basis with no significant sequelae or they required no treatment. Analysis of the literature indicates that a more prolonged course of antibiotic therapy decreases the rate of infection following transrectal prostate biopsy. In different series, by extending the prophylaxis for at least 4 days, the infection rate dropped to between 0 and 0.8%.^{2,12,45} In our case, however, the patient refused to take his prophylactic antibiotic drugs during the daytime following the second TUGNBP because he was fasting during the holy month of Ramadan. Following the first TUGNBP he had taken the prophylactic antibiotics and did not experience any major or minor complication from the biopsy.

Identified risk factors for the development of pyogenic spondylodiscitis include increased patient age, liver disease, DM, ankylosing spondylitis, rheumatoid arthritis, trauma, ethanol abuse, intravenous drug abuse,³² other initial sites of infection,^{36,40,46} immunosuppression due to steroid use, organ transplantation^{16,26} or infection with human immunodeficiency virus, and malignancy. Rheumatoid arthritis, DM, advanced patient age, and steroid use also increase the risk of developing paralysis caused by spinal osteomyelitis. The patient in our case had poorly controlled DM.

In diagnosis, the laboratory findings most predictive of pyogenic spondylodiscitis include an elevated erythrocyte sedimentation rate and C-reactive protein level. White blood cell counts were elevated to more than 10,000 in only 8% of cases. Blood cultures may be positive in 50% of cases. Conventional x-ray studies may not demonstrate characteristic diagnostic changes until 2 to 6 months after the onset of symptoms, and thus long delays can occur in obtaining the correct diagnosis.³³

The MR imaging study has demonstrated very high sensitivity and specificity for the evaluation of pyogenic spondylodiscitis. Characteristic MR imaging findings of discitis include decreased signal from the disc and adjacent portion of VBs on T₁-weighted sequences and an increased signal from these structures on T₂-weighted se-

quences. Characteristic findings may occur 3 to 5 days after the onset of symptoms. Following Gd injection adjacent vertebral bone marrow, disc space, and posterior annulus fibrosus enhance. The MR imaging examinations of spondylodiscitis include T₁-weighted sequences with and without paramagnetic contrast enhancement and T₂-weighted sequences. The T₁-weighted image demonstrates an extension of soft tissue into the spinal canal, but does not differentiate between granulation tissue and purulent material. Nonetheless, T₁-weighted images with Gd contrast reveal infection or granulation tissue as high-intensity areas or a high intensity halo surrounding a center of low intensity (abscess).³³

Staphylococcus aureus has been the most commonly isolated pathogen in association with spondylodiscitis, followed by *Streptococcus* spp. and Gram-negative bacilli.^{23,27} In one series multiple organisms involved 25% of the patients; however, polymicrobial infections did not alter patient outcome. In our case, isolation of *E. coli* as an originating agent is not surprising, because this bacteria is present in the gastrointestinal flora. Irrespective of the originating organisms, the histological features of pyogenic spondylodiscitis include vascular proliferation associated with granulation tissue, myxoid degeneration, and variable acute and chronic inflammation.³³

Antibiotic treatment usually involves 6 weeks of intravenous administration followed by 6 weeks of oral administration. Therapy lasting less than 4 weeks in duration is associated with a 25% relapse rate. Therapeutic failure was defined as persistent or worsening symptoms plus elevated serum erythrocyte sedimentation rate and/or C-reactive protein levels, or worsening imaging findings such as increased uptake of ⁶⁷Ga on radionuclide scanning after a long course of antibiotic therapy. With appropriate treatment, relapse rates vary from 0 to 4%.^{23,27}

The rate of sequelae in pyogenic spondylodiscitis varies from 25 to 45% of cases. Jimenez-Mejias, et al.,²⁷ reported that only 45% of patients returned to work and daily normal activities. Most patients with pyogenic discitis, even those with associated epidural abscess, can be successfully treated using intravenous antibiotics, bedrest, and external immobilization. Nonetheless, a small subset of patients (25%) will require surgery. This latter category is characterized by uncertain diagnosis with suspicion of neoplasm, decompression of neural structures due to epidural abscess, and compressed granulation tissue. Rarely, the patients require fusion for unstable spine as a complication of nonsurgical management.

At the postoperative 4th week in our case, the patient was neurologically intact and pain free, although his control MR image of the lumbar spine revealed L-3 compression and progressed L2–3 listhesis along with dural sac compression. His lateral plain flexion–extension x-ray studies of the lumbosacral spine demonstrated solid fusion with no instability at the L2–3 level. Because he had solid fusion and no neurological abnormality, the fusion operation was cancelled.

CONCLUSIONS

Acute pyogenic spondylodiscitis should be considered among the major complications of TUGNBP. Following

the procedure, prophylactic antibiotic therapy should always be given to the patient. Persistent back pain and fever following TUGNBP, despite the administration of antibiotic agents prophylactically, indicate that the patient should be evaluated by performing lumbar MR imaging studies for early diagnosis of this serious complication.

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Manuscript received April 16, 2004.

Accepted in final form May 17, 2004.

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