### Brief Reports

## Septic Discitis Resulting from Escherichia coli Urosepsis

Charles D. Ponte, PharmD, and Michael McDonald, MD

Morgantown, West Virginia

Septic discitis refers to a primary suppurative process involving the intervertebral disc space and occurs as a result of hematogenous invasion or contamination by pyogenic organisms. A case of septic discitis is described in a 77-year-old woman following an episode of *Escherichia coli* urosepsis. Despite bed rest, an orthosis, and appropriate antibiotics, the patient ultimately had to undergo surgical disc removal. The diagnosis of

septic discitis is often made in the context of other diseases that share common clinical and laboratory findings. Magnetic resonance imaging appears well suited for diagnostic confirmation of septic discitis. Needle biopsy and aspiration results should be used to determine the appropriate choice of antibiotic for this disease process. Key words. Escherichia coli; lumbar vertebrae.

J Fam Pract 1992; 34:767-771.

Septic discitis may be simply defined as an inflammation of the intervertebral disc space resulting from invasion by suppurative organisms. This disease entity should be differentiated from osteomyelitis since the insult involves a primary infection of the disc space rather than the surrounding bony structures. The following is a description of a case of septic discitis in an elderly woman resulting from an episode of *Escherichia coli* urosepsis. A brief review of septic discitis and its management are described.

#### Case Report

T.B. was a 77-year-old white woman who initially presented to the family practice center with a 3-day history of nighttime rigors, mental status changes, and urinary frequency. Her past medical history was significant for chronic renal insufficiency, osteoarthritis, a seizure disorder, and hypertension. Her vital signs were normal. Laboratory test results were as follows: white blood cell count,  $16.2 \times 10^9/L(16,200/mm^3)$ ; differential, 78% polymorphonuclear leukocytes, 11% bands; blood urea nitrogen, 19.3 mmol/L (54 mg/dL); and serum creatinine, 353.6 mmol/L (4 mg/dL). Microscopic urinalysis revealed too numerous WBC per high power field to count, WBC clumps, and numerous bacteria.

The patient was admitted to the hospital. A computed tomography (CT) scan of the head was normal, and cerebral spinal fluid studies were unremarkable. The patient was treated for presumed urosepsis with intravenous cefazolin, 1 g every 12 hours. Blood and urine cultures subsequently grew  $E\ coli$  sensitive to cefazolin, confirming the diagnosis of urosepsis. The patient exhibited marked improvement following 48 hours of the antibiotic, which was subsequently continued for a total of 7 days. The patient was discharged, and oral cephalexin, 500 mg four times daily, was prescribed to complete 2 weeks of total therapy. The WBC count was  $9.9 \times 10^9/L\ (9900/mm^3\ [normal\ differential])$ .

The patient was seen in the clinic for a follow-up examination 1 week later. She complained of severe low back pain and mild lower abdominal pain. A repeat urine culture grew <10,000 gram-negative rods. A subsequent CT scan of her abdomen was negative, and a barium enema revealed only multiple sigmoid diverticuli. A white blood cell count at that time was  $13.3 \times 10^9/L$  (13,300 mm<sup>3</sup> [normal differential]). Erythrocyte sedimentation rate was 126 mm/h.

Three weeks after discharge, the patient returned to the clinic with increasing abdominal and back pain and intermittent confusion. Her vital signs were normal. Physical examination revealed a nonacute abdomen with moderately severe right lower quadrant pain and diffuse moderate low back pain. Neurologically, the patient was intact except for impaired short-term memory and cognitive function. Laboratory testing revealed a white blood cell count of  $11.9 \times 10^9 / L$  ( $11,900 / mm^3$  [normal differential]) and an erythrocyte sedimentation rate of 124 mm/h. She was again admitted to the hospital. Urine

Submitted, revised, February 18, 1992.

From the Department of Clinical Pharmacy (Dr Ponte), and Family Medicine (Drs Ponte and McDonald), West Virginia University Health Sciences Center, Morgantown, West Virginia. Requests for reprints should be addressed to Charles D. Ponte, PharmD, West Virginia University Health Sciences Center, School of Pharmacy, Morgantown, WV 26506.

© 1992 Appleton & Lange

ISSN 0094-3509



Figure 1. A computed tomography scan shows spinal infection with discitis in the L2-3 disc space, and osteomyelitis of the inferior margin of the L-2 body and superior margin of the L-3 body; also, likely small epidural abscess posterior to L2-3 disc and along posterior margin of L-2 vertebral body, and air in the anterior epidural space at L-2. Arrow indicates erosion of the lateral aspect of L-2 vertebral body.

and blood cultures were obtained, and after 24 hours, the blood cultures grew E coli and the urine culture grew >100,000 Proteus mirabilis. Again she was started on intravenous cefazolin, 1 g every 12 hours, to which both organisms were susceptible. Lumbar spine roentgenograms showed degenerative changes in the lower spine and no lytic lesions. A CT scan and an MRI both showed the degeneration of the L2-3 intervetebral disc (Figure 1) and an MRI both showed the degeneration of the L2-3 intervertebral disc (Figure 2). The disc was subsequently aspirated and a biopsy of the L-2 vertebral body was obtained. Cultures ultimately grew E coli with the same antibiotic sensitivity pattern noted during her prior hospitalization for urosepsis. Cefazolin was continued for 10 days in the hospital, and there was moderate improvement in her pain and mental status. The decision was made to manage her conservatively with a 6-week course of antibiotics and weekly roentgenograms of her lumbosacral spine. Arrangements were made for her to be cared for at home. Intravenous ceftriaxone, 2 g every 24 hours through a left subclavian Hickman catheter, was prescribed; the patient was placed in a thoracic lumbar



Figure 2. Magnetic resonance imaging shows narrowing at L2-3; other changes secondary to discitis and osteomyelitis or inferior spondylitis. The associated epidural abscess and associated changes of the psoas muscle are also visualized. Arrow indicates obliteration of L2-3 disc space and interior impression of thecal sac.

sacral orthosis, and she was discharged. Erythrocyte sedimentation rate at the time of discharge was 133 mm/h.

One week following discharge, the patient was seen in the clinic, and she reported continued back pain. Lumbar spine roentgenograms were unchanged. Five days later, an MRI revealed increased destruction of the intervertebral disc and an epidural abscess. Her erythrocyte sedimentation rate was 150 mm/h at this time. She was admitted to the hospital for the third time and underwent surgical disc removal and iliac bone grafting. Ceftriaxone was prescribed for an additional 6 weeks following surgery. She was subsequently admitted to the hospital's skilled nursing unit for continued physical therapy and rehabilitation. Her erythrocyte sedimentation rate 1 month after surgery was 38 mm/h.

#### Discussion

Discitis or spondylodiscitis is an inflammatory condition that involves the intervertebral disc space and predominately affects the lumbar spine.<sup>2,3</sup> It is more commonly a

continued on page 770

continued from page 768

disease of childhood and results from either trauma or the invasion of blood-borne bacteria. Staphylococcus aureus and Hemophilus influenzae are common bacterial isolates.3 Symptoms are nonspecific and may mimic such diseases as septic arthritis, meningitis, vertebral osteomyelitis, and appendicitis. Fever and an elevated erythrocyte sedimentation rate are associated findings. Diagnosis is confirmed using radiographic or radionuclide imaging techniques or both. Treatment involves bed rest and immobilization. Body casts may also be helpful in the management of this condition. The use of antibiotics is controversial, and when used, should be directed toward the most likely organisms. The duration of antibiotic treatment is highly variable and no consensus has yet been determined. The prognosis is usually favorable, although the disease process may take 2 months to re-

Septic discitis implies a primary suppurative process involving the intervertebral disc space resulting from hematogenous invasion or contamination by pathogenic organisms. McCain et al1 reviewed 16 cases of septic discitis in an adult population. The disease is insidious in nature: the average interval between symptoms and diagnosis is 14 weeks. Low grade fever and chills were noted in less than half of the cases. The most useful clinical finding was point tenderness over the affected area with associated paravertebral tenderness. The erythrocyte sedimentation rate was commonly elevated, a finding corroborated by other investigators.4 The complete blood cell count was usually normal, and occasionally serum alkaline phosphatase was elevated. The most common bacteria associated with septic discitis in the cases studied by McCain et al were S aureus and E coli. Bone scans were valuable in making the diagnosis of septic discitis in cases where plain radiographs and CT scans were negative. Bed rest and antibiotics represented the treatment standard, but the choice of antibiotics should reflect the results of culture and sensitivity studies. McCain et al treated all their patients with 2 to 4 weeks of parenteral antibiotics followed by orally administered antibiotics for an additional 8 weeks. Back pain, point tenderness, and the erythrocyte sedimentation rate were used to determine the length of parenteral therapy. Surgery was reserved for potential spinal chord compression and epidural abscess formation.

In our patient, septic discitis developed following an episode of *E coli* urosepsis. This seeding of organisms probably occurred before or during her first hospitalization. It has been suggested that such seeding occurs through the posterior venous plexus and Batson's veins.<sup>5</sup>

The antibiotics originally chosen were appropriate, and the dosage was consistent with the patient's degree of chronic renal insufficiency. Her complaints of back and

abdominal pain in addition to her elevated erythrocyte sedimentation rates were, in retrospect, consistent with the clinical presentation of septic discitis. It must be remembered, however, that these associated laboratory and physical findings are not specific for septic discitis. A positive bone scan suggested a suppurative or inflammatory process in the L2-3 area. Importantly, the MRI findings confirmed the diagnosis of discitis in the lumbar spine. The value of magnetic resonance imaging in the diagnosis of spondylodiscitis has recently been studied.<sup>6</sup> The positive biopsy and aspiration cultures confirmed the infectious nature of the discitis in our patient.

This patient was managed conservatively following the diagnosis. Bed rest, an orthosis, and appropriate antibiotics were initiated. Home parenteral antibiotic therapy was provided, and weekly lumbosacral spine roentgenograms were made following hospitalization. Although both cefazolin and ceftriaxone have been used successfully in the management of bone and joint infections, it could be argued that the antibiotic doses employed in our patient were not appropriate for the disease under treatment.<sup>7,8</sup> Whether both drugs penetrate the avascular intervertebral disc is unclear, which may explain the failure of the antibiotic regimen and the need for her discectomy. Appropriate antibiotic therapy usually results in a fall in the erythrocyte sedimentation rate, yet this was not seen in our patient, further evidence of the lack of an antibiotic response.1 Interestingly, animal research has suggested that cefazolin can penetrate the intervertebral disc through the anulus fibrosus, yet may be unable to affect the course of the discitis.9 This finding may have resulted from the rapid multiplication of bacteria within the disc and the inability of the relatively small concentration of antibiotic in the disc to effectively eradicate the organism. Other unknown factors may have been responsible for the lack of therapeutic success.

#### Summary

Septic discitis is a clinical entity that poses problems for diagnosis and management.<sup>1,10–13</sup> Whether septic discitis more accurately represents hematogenous vertebral osteomyelitis remains controversial.<sup>14</sup> Its insidious nature makes diagnosis problematic since it must be evaluated in the context of other diseases that manifest with similar subjective complaints and physical and laboratory findings. Therefore, any septic episode or violation of the intervertebral disc with attendant back pain or abdominal pain, or both, should raise the suspicion of septic discitis. Diagnostic confirmation requires the implementation of radiographic and/or radionuclide studies, and MRI seems well suited for this purpose. Needle biopsy and

aspiration can identify pathogenic organisms as causative agents and aid in the selection of antimicrobial therapy. Despite the lack of guidelines, it seems prudent to administer antibiotics for 4 to 6 weeks. Whether antibiotics will alter the course of the disease appears somewhat controversial. Prophylactic antibiotics may prove more valuable if used before surgical invasion of the intervertebral disc space, although further investigation is warranted.<sup>9</sup>

#### Acknowledgments

The authors wish to acknowledge Drs Dorian Williams and Cindy Lee Martinec for their assistance in the preparation of this manuscript.

#### References

- McCain GA, Harth M, Bell DA, Disney TF, Austin T, Ralph E. Septic discitis. J Rheumatol 1981; 8:100-9.
- Hensey OJ, Coad N, Carty HM, Sills JM. Juvenile discitis. Arch Dis Child 1983; 58:983-7.
- 3. Amir N, Hurvitzh H, Korn-Lubetzki I, Shalev RS. Gower's sign in discitis in childhood. Clin Pediatr 1986; 25:459-61.
- Schulak DJ, Rayhack JM, Lippert FG, Convery FR. The erythrocyte sedimentation rate in orthopaedic patients. Clin Orthop 1982; 167:197-202.
- Norden CW. Osteomyelitis. In: Principles and practice of infectious diseases. New York: Churchill Livingstone, 1990:922-30.
- Frank AM, Trappe AE. The role of magnetic resonance imaging (MRI) in the diagnosis of spondylodiscitis. Neurosurg Rev 1990; 13:279-83.
- Kucers A, Bennett NM. Cefazolin, cephacetrile and cephapirin. In: The use of antibiotics. Philadelphia: JB Lippincott, 1987:381-97.
- 8. Kucers A, Bennett NM. Ceftriaxone. In: The use of antibiotics. Philadelphia: JB Lippincott, 1987:510-24.
- Fraser RD, Osti OL, Vernon-Roberts B. Iatrogenic discitis: the role of intravenous antibiotics in prevention and treatment. Spine 1989; 14:1025-32.
- Durance JP. Lumbar discitis in a patient with quadriplegia: Case report. Arch Phys Med Rehabil 1989; 70:233-5.
- DeSouza LJ. Disc space infection in children, late adolescents, and adults. Minn Med 1980; 63:314-20.
- 12. Myles PS. Septic discitis as a presentation of endocarditis [Letter]. Med J Aust 1987; 146:656-7.
- Short DJ, Webley M, Hadfield J. Septic discitis presenting as a psoas abscess. J R Soc Med 1983; 76:1066-8.
- Ryan MD, Taylor TK. Septic discitis—a misnomer [Letter]. Med J Aust 1987; 147:415.

# Important news for sufferers of intestinal gas!

A new double-acting anti-gas tablet called Charcoal Plus is now available to fight the pain, bloating and diarrhea caused by stomach or intestinal gas. Charcoal Plus is double-acting because it fights gas in both the stomach and intestines with harcoa two recognized anti-gas agents: Simethincone (for stomach gas) and Activated

is released first in the stomach. Then, after an intermediate coating dissolves, the inner core of activated charcoal is released in the intestines.

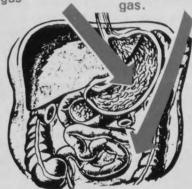
SIMETHICONE fights stomach gas

Charcoal (for

intestinal gas).

Simethicone

ACTIVATED CHARCOAL for intestinal



Charcoal Plus is available in bottles of 120 tablets. Each dosage of two tablets contains Activated Charcoal USP (400 mg.) and Simethicone (80 mg.).

Use Coupon For Free Samples!

# Complete And Mail Today! Kramer Laboratories 8778 S.W. 8th St. Miami, FL 33174 Please send FREE samples and literature

Please send FREE samples and literature on new CHARCOAL PLUS. Name

Address\_\_\_\_\_\_City/State/Zip \_\_\_\_\_\_\_Telephone

For Immediate Action Call 1-800-824-4894 or 305/223-1287