Pyogenic Spondylodiscitis Presenting As A Spontaneous Ruptured Flank Abscess

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Abstract
Spondylodiscitis is an inflammatory state of intervertebral discs and adjacent vertebral bodies caused by an infecting organism. We report a 47-year-old patient suffering from pain in the lumbar-sacral spine presenting as a ruptured flank abscess after treating conservatively for two months with nonsteroidal anti-inflammatory drugs. The disease course with fever, leucocytosis and ESR as well as the results of diagnostic investigations, particularly CT of the spine, confirmed preliminary diagnosis. Positive bacterial isolation and response to antibiotic treatment, also suggested that the diagnosis of spondylodiscitis was correct. This case is an example of rarely considered and frequently missed cause of lower back pain, pyogenic spondylodiscitis, which occurs sporadically among frequent diseases of these structures. Prompt and accurate diagnosis, the cornerstone of treatment of spinal infections, requires high index of suspicion followed by an appropriate evaluation and treatment prevents severe morbidity.

CASE REPORT
A 47-year-old housewife was admitted to the Department of Surgery, General Hospital, Port-of-Spain, Trinidad due to a ruptured left flank abscess and severe pain in the lower back. She was first seen for two months at another institution because of history of discomfort in the lower back that did not respond to nonsteroidal anti-inflammatory drugs. On examination there was a large ruptured abscess draining non-foul smelling pus in the left flank region (Figure 1). Patient's body temperature remained at 37-39 degree centigrade with mild dehydration and tachycardia. Lumbar spine examination revealed stiffness with considerable tenderness over lower lumbar spines. Because of spasms of lower back muscles, bilateral Lasegue test was inconclusive but, bilateral lower limb muscles power, and bilateral knee and ankle reflexes were symmetrically retained. No dysesthesia was present. There was normal bladder and bowel functions.

Laboratory investigation revealed mild microcytic hypochromic anemia with a hemoglobin of 10.2 g/dL; white blood cell (WBC) count of 18.8 X 10^3 µL (Neutrophils 81.4%); and platelet count of 599 X 10^3/µL. The erythrocyte sedimentation rate (ESR) by Westergren method was 120mm/hour. The results of urinalysis, liver function tests and serum chemistries including blood glucose were normal. Tuberculin test (CO985AA), urine and blood cultures yielded negative results. No lesions were detected on the chest x-ray.
Roentgenograms (Figure 2) of lumbosacral spine revealed massive collapse of L4 and L5 vertebral bodies with loss of height, more so anteriorly than posteriorly, and near complete destruction of the intervertebral disc space. There was significant osteolysis of inferior half of L4 and superior half of L5 vertebral bodies, mild scoliosis and abnormal psoas shadows. There was also narrowing of intervertebral foramen with flattening and sclerosing of facet joints of L4 and L5 vertebrae. These are the typical radiographic findings of an established pyogenic spondylodiscitis. Computed tomography (CT) scans (Figure 3a,b,c) of the lumbar region revealed a large irregular wedge shaped myocutaneous tissue defect in the left flank abdominal wall with apex at the junction of erector spinae and external oblique muscles. Markedly enlarged, irregular poorly defined margins of left psoas and erector spinae muscles with hypodense inflammatory changes. Psoas muscle changes extend distally along its length into the ileac fossa. The L4/L5 intervertebral disc and adjacent half of vertebral bodies are not defined due to infectious destruction and collapse of these structures. The remaining hyperdense L4 and L5 vertebral bodies show irregular margins with subperiosteal osteoblastic reaction. Additionally, the spinal canal as well as intervertebral foramen at L4-L5 is narrowed with poorly defined facet joints. Following intravenous contrast there was a ring enhancement of these hypodense areas indicating marked inflammatory changes involving left erector spinae and bilateral psoas muscles with residual collection of pus and granulation tissue.

Figure 2
Figure 2: Roentgenograms of lumbosacral spine: Loss of L4 and L5 vertebral height with destruction of L4-L5 end plates and intervertebral disc.

Figure 3
Figure 3a: Pre-contrast computed tomographic scans of lumbar region
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**Figure 4**
Figure 3b: Post-contrast axial computed tomographic scans of the lumbar region.

**Figure 5**
Figure 3c: Post-contrast computed tomographic scans of lumbar region.

Microscopic analysis of discharge and granulation tissue showed acute inflammatory as well as dead and degenerated cells along with gram-positive cocci. Microscopy was negative for malignant cells. Ziehl Neelsen's stain for acid-fast bacilli was negative. Gram's stain showed Gram-positive cocci and Gram positive bacilli. Bacterial culture yielded negative for acid-fast bacilli and fungal agents, but positive for Staphylococcus aureus, Group B Streptococci and Acinetobacter species sensitive to cloxacillin and co-trimoxazole. At week four, repeat cultures yielded positive for Staphylococcus epidermidis and Pseudomonas aeruginosa sensitive to ceftazidime and ciprofloxacin.

**CLINICAL COURSE**
The treatment included cloxacillin 1g IV (intravenous) four times daily and oral co-trimoxazole 960 mg every 12 hours for first four weeks, nonsteroidal anti-inflammatory drugs, lumbar spinal support and bed rest. The abscess cavity was debrided three times during this period. At the end of week four, antibiotics switched to IV ceftazidime 2 g every 12 hours for another four weeks, then oral ciprofloxacin 500 mg twice daily was started. First, the white blood cells returned normal followed by, weeks later, ESR with a gradual alleviation of pain. The abscess cavity gradually closed over twelve to fourteen weeks and the wound healed by second intention (Figure 4). Ciprofloxacin was continued until the wound healed. A check-up CT scan of lumbar spine performed six months later, indicated resolved inflammatory process and healing scar tissue (Figure 5). The L4-L5 intervertebral disc was destroyed with adjacent vertebral osteoblastic response and sclerotic new bone formation. Roentgenograms, 14 months after first presentation, showed progressive collapse of the L4 and L5 vertebrae with partial fusion (Figure 6). There is normal lumbar lordosis and no worsening of mild scoliosis. She remains under constant outpatient supervision, continues to be well without any significant symptoms apart from some low back stiffness and loss of three inches of body height. At 8 and 14 months after presentation, a repeat ESR and white blood cell count remained normal.
DISCUSSION

Before the introduction of modern antibiotic therapy, mortality in patients with spondylodiscitis was as high as 25% (1). Antibiotic therapy combined with surgical debridement and stabilization has decreased mortality to less than 5-15% (2-4). Pyogenic spondylodiscitis is a bacterial infection that can arise from a number of sources – direct inoculation, contiguous spread from an adjacent infection, or hematogenous seeding (5). Hematogenous seeding infection is still by far the most common mechanism of spinal infection with Staphylococcus aureus accounting for >50% (5). Infections with gram-negative organisms may occur following genitourinary infections or procedures. The two major theories for hematogenous dissemination are venous theory and the arteriolar theory. Batson (6) developed the venous theory using both live animal and human cadaveric models and demonstrated retrograde flow from the pelvic venous plexus to the perivertebral venous plexus via the valveless meningorrhachidian veins. In the arteriolar theory, Wiley and Trueta (10) proposed that bacteria could become lodged in the end-arteriolar network near the vertebral end plate. Both these mechanisms are significant in the establishment of an infectious focus in the spinal column.

Pyogenic spinal infection affects the lumbar spine more commonly than the thoracic or cervical spine (11). In adults, once the infection is established adjacent to the end plate of one vertebral body, direct local spread can occur into the
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Infections. Technetium 99m bone scintigraphy is sensitive much more sensitive than radiographs in detecting early especially in thoracic spine. Radionuclide studies can be also destroyed and a kyphotic deformity may be present, preservation of the disc spaces. At later stages, the disc is often demonstrate vertebral destruction with relative normalization of the ESR and CRP level (5). Once the organism has been identified, intravenous antibiotic therapy should be initiated according to the culture results and sensitivities. A course of 2 to 6 weeks of parenteral antibiotics is usually recommended followed by a course oral antibiotic, depending on the virulence of the organism, susceptibility of the host, and other factors, such as retained hardware (5). Conversion to oral antibiotics should be made only with clinical improvement, normalization of the ESR and CRP level (5), or resolution of infection as demonstrated in imaging studies. In addition, immobilization of the affected area aids in pain relief and helps prevent deformity.

Computed tomography (CT) is useful in delineating the extent of bony destruction and soft tissue extension and in preoperative planning. Although the CT scan with intravenous contrast also can demonstrate soft-tissue extension, distinction between abscess and granulation tissue may be difficult. Magnetic resonance imaging (MRI) has an extremely high sensitivity (96%) and specificity (93%) in detecting infections of the vertebral column (5). It allows detection of paravertebral and epidural extension, and clearly visualizes neurologic structures. T1-weighted sequences demonstrate decreased signal intensity in both the vertebral body and disc edema. T2-weighted images show increased signal intensity in both the vertebral body and disc with loss of the normal intranuclear cleft. The administration of gadolinium in combination with MRI improves resolution and allows an infectious process to be distinguished from degenerative changes of the end plate and intervertebral disc. Even with MRI, however, granulomatous infections can be difficult to distinguish from tumors of the spine. Thus, a biopsy is often required to make a definitive diagnosis (5).

The goals for treatment of spinal infections should be to establish a diagnosis and identify the pathogen, eradicate the infection, prevent or minimize neurologic involvement, maintain spinal stability, prevent progressive deformity and provide an adequate nutritional state to combat infection. Once the organism has been identified, intravenous antibiotic therapy should be initiated according to the culture results and sensitivities. A course of 2 to 6 weeks of parenteral antibiotics is usually recommended followed by a course oral antibiotic, depending on the virulence of the organism, susceptibility of the host, and other factors, such as retained hardware (5). Conversion to oral antibiotics should be made only with clinical improvement, normalization of the ESR and CRP level (5), or resolution of infection as demonstrated in imaging studies. In addition, immobilization of the affected area aids in pain relief and helps prevent deformity.

Surgery is indicated in five circumstances (5): to obtain a tissue diagnosis after a failed closed needle biopsy or from a location inaccessible by closed methods; for drainage of an abscess; to treat neurologic deficit secondary to compression

Pyogenic spondylodiscitis is more common in elderly males than females. However, the incidence of infection is increasing in younger population with intravenous drug abuse or immunocompromise after organ transplantation, chemotherapy or HIV/AIDS (5). Back pain is the most consistent symptom in >90% of patients (5), is often quite severe and is associated with paraspinal muscle spasm. The pain is present regardless of activity level and may occur at night with or without radiation to legs. Fever and weight loss are seen in approximately 50% of the affected population (5).

Imaging studies are crucial to localize the infection, assess the extent of involvement, and determine the response to treatment. Radiographs may demonstrate osteolysis and end plate destruction. As the disease progresses, the disc space narrows and eventually collapses. Soft tissue extension must be suspected in the presence of an abnormal psoas shadow; however, plain radiographs may take several weeks to demonstrate abnormal findings. In contrast with pyogenic infections, skeletal radiographs in a tuberculosis infection often demonstrate vertebral destruction with relative preservation of the disc spaces. At later stages, the disc is also destroyed and a kyphotic deformity may be present, especially in thoracic spine. Radionuclide studies can be much more sensitive than radiographs in detecting early infections. Technetium 99m bone scintigraphy is sensitive (~90%) but nonspecific, especially in adults with degenerative joint disease (5). When used in conjunction with technetium 99m scans, gallium 67 citrate scans have high sensitivity and specificity in detecting foci of infection (5).

adjointing disk and vertebral body. The disc is relatively avascular and is rapidly destroyed by bacterial enzymes. From the lumbar spine, abscess formation can track along the psoas muscle and into buttock (piriformis fossa), perianal region, the groin, or even the popliteal fossa (5). Our case is unique in that there were no reported cases in the literature where the abscess cavity had ruptured through the flank abdominal wall. The extension of infection into the spinal canal may result an epidural abscess or even bacterial meningitis. Destruction of the vertebral body and disc can potentially lead to instability and collapse.

Neurologic deterioration can occur if an epidural abscess compresses the neural elements or cause thrombosis or infarction of the regional vascular supply to the spinal cord. Pathologic fracture and / or spinal instability resulting from destruction of the disk, vertebral bone, and posterior stabilizing structures can cause neural impingement. Other risk factors (5) that predispose to neurologic impairment include diabetes, rheumatoid arthritis, steroid use, advanced age, a more cephaloid level of infection and infection with staphylococcus species.
either by the infection (abscess or granulation tissue) or structural destruction; for structural instability or deformity; or failure of medical management to reduce persistent instability or deformity; or failure of medical management to reduce persistent symptoms or elevated laboratory measurements.

Hematogenous spondylodiscitis is a common spinal infection; successful diagnosis and treatment depend on an appropriate index of suspicion. Early detection and antibiotic treatment may obviate the complications and the need for surgical intervention. When surgical debridement is indicated, its prompt initiation appears to result in good clinical outcomes.

References

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