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Vertebral Osteomyelitis Caused by Group B Streptococci (*Streptococcus agalactiae*) Secondary to Urinary Tract Infection

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Infections due to group B streptococci usually occur in the peri- and neonatal setting or in adults with chronic underlying diseases. A case of pyogenic vertebral osteomyelitis caused by *Streptococcus agalactiae* in a 54-year-old man suffering from phimosis with urinary retention and urinary tract infection is reported. This case adds to the few existing reports of vertebral osteomyelitis caused by group B streptococci.

During recent decades, the spectrum of microorganisms causing vertebral osteomyelitis has shifted from mycobacteria to pyogenic bacteria. *Staphylococcus aureus* is responsible for up to 85% of cases of pyogenic vertebral osteomyelitis (1), but streptococci and gram-negative rods may also be involved (2). The disease is notorious for its insidious onset, and serious neurological complications and fatalities may occur when the diagnosis is unduly delayed.

Group B streptococcus (*Streptococcus agalactiae*), usually associated with perinatal infections, has recently been recognized as an important infectious agent of invasive disease in nonpregnant adults. The majority of cases reported thus far have occurred in elderly patients or those with significant underlying conditions such as diabetes mellitus, malignancy, or liver disease (3). We report the case of a 54-year-old man suffering from phimosis and urinary tract infection who developed group B streptococcal vertebral osteomyelitis with epidural involvement.

Case Report. A 54-year-old man was admitted to our hospital for treatment of low back pain. He had suffered from general malaise for several months, occasional chills for one week, and low back pain radiating down the lateral aspect of the left leg for three days. His medical history was unremarkable except for a phimosis that had existed since his childhood, associated with occasional self-limited dysuria. On admission the patient's oral temperature was 37.7°C. A tight phimosis was confirmed. Neurological examination revealed tenderness and hyperextension of the lower lumbar spine with bilateral muscular rigidity. There was no sensory or motor deficit of the lower extremities, and reflexes were normal.

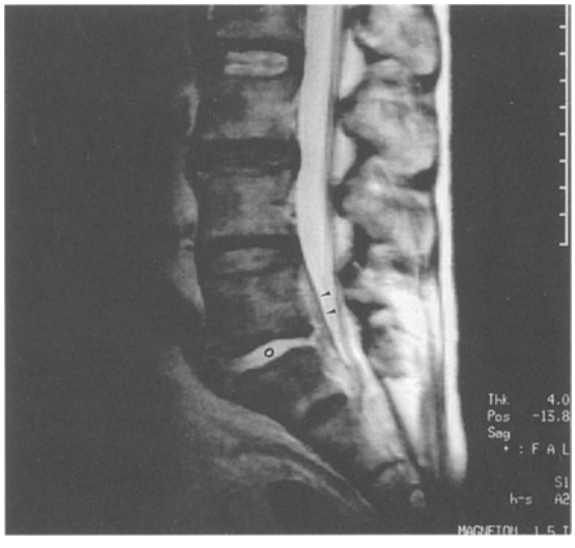

Figure 1: Midsagital T2-weighted magnetic resonance image on admission. The hyperintense L5/S1 disk (circle) indicates diskitis. Note the posterior displacement of the posterior longitudinal ligament by a hyperintense mass at the same level (arrows).

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Laboratory investigations revealed haemoglobin of 14.9 g/dl, a leukocyte count of 12.6×10^9/l (58% polymorphonuclear neutrophils, 15% band forms), and C-reactive protein of 267 mg/l (normal range, < 5 mg/l). Urine microscopy revealed 40 leukocytes/high power field (hpf) and four erythrocytes/hpf. Urinary cultures grew group B streptococci, whereas three sets of blood cultures remained without growth. Lumbar spine plain film radiographs showed a slight narrowing of the L5/S1 disk space but normal vertebral end plates. Magnetic resonance imaging revealed an extensive inflammatory process with abscess formation, associated with a hyperintense disk and bone marrow edema (Figure 1). A needle biopsy at the L5/S1 level was performed under guidance by computed tomography. Cultures of the aspirate grew group B streptococci, and histopathological examination showed signs of osteomyelitis and diskitis.

Initial treatment including bed rest, diazepam, ibubrufen, and low molecular weight heparin resulted in prompt defervescence. Benzylpenicillin (5×10^6 U i.v. every 6 h) was added upon receipt of the microbiological results. After two weeks the leukocyte count was 6.42×10^9/l and the C-reactive protein 17 mg/l. Urological investigations revealed a residual urinary volume of 115 ml and multiple urethral strictures. The patient underwent circumcision and urethral dilatation. After four weeks, ceftriaxone (2 g/day i.v.) was substituted for benzylpenicillin because of drug-induced liver injury. After a six-week course of antimicrobial treatment, the patient was mobile without backache, and the C-reactive protein was 9 mg/l. Magnetic resonance imaging performed prior to discharge showed complete resolution of the epidural abscess and narrowing of the L5/S1 intervertebral disk space. Twelve months after discharge the patient remained free of symptoms.

Discussion. Our case illustrates several typical features of pyogenic vertebral osteomyelitis, such as the presence of a genitourinary source of infection, a considerable delay in presentation, and a serious complication in terms of epidural involvement. The patient's chronic phimosis, as the cause of urinary retention and recurrent urinary tract infection, is likely to have been the initiating event for the vertebral osteomyelitis.

Primary infections elsewhere are identified in two-thirds of patients with vertebral osteomyelitis (4). The genitourinary tract accounts for the majority of cases, possibly because of direct venous spread through intercommunications between the veins draining the pelvic organs and the valveless external and internal venous plexus of the spinal canal (Batson's plexus) (4). Although group B streptococci are well-known pathogens affecting the genitourinary tract, they have rarely been reported to cause vertebral osteomyelitis. We are aware of only eight cases, five of which occurred in patients with chronic underlying diseases such as diabetes mellitus (5, 6), systemic lupus erythematosus (7), alcoholism (8), or intravenous drug abuse (9). One case occurred in an otherwise healthy patient with soft tissue infection (10), and in only two patients was no predisposing condition reported (11, 12). To our knowledge, no case with bacteriological evidence of a causal relationship between urinary tract infection with group B streptococci and vertebral osteomyelitis in the absence of any underlying disease has been reported thus far. The failure to grow streptococci from blood is compatible with the concept of local spread through venous intercommunications. However, intermittent bacteraemia with seeding of the intervertebral disk space cannot be excluded.

The diagnosis of vertebral osteomyelitis remains difficult because of vague symptoms and unspecific findings. Diagnosis is delayed for more than three months in up to 50% of cases (4). Plain film radiographs may be entirely normal for several weeks, and subtle signs such as disk space narrowing may be mistaken as degenerative changes. Computed tomography scans may also be normal early in the course of infection, showing loss of intervertebral disk height, cortical bone erosions, and soft tissue swelling in advanced cases only. Radionuclide bone scans are sensitive indicators of early disease, but their specificity of 65% is low (13). The imaging modality of choice is magnetic resonance imaging, offering a sensitivity and specificity of approximately 100% and visualisation of morphologic changes in the vertebral body, disk space, paraspinous soft tissue, and epidural space (14). In view of the large variety of potential pathogens, bacteriologic diagnosis must be obtained in all cases without positive blood cultures. Computed tomography-guided diskovertebral needle biopsy has proven to be a safe and effective tool for identifying the causative agent.

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Pleural and Peritoneal Leishmaniasis in an AIDS Patient

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The case of an AIDS patient who developed pleuritis and peritonitis in the course of relapsing visceral leishmaniasis is reported. Visceral leishmaniasis, considered an opportunistic infection in patients infected with the human immunodeficiency virus (HIV) who live in endemic areas, has a chronic relapsing course. Typical manifestations such as fever, hepatosplenomegaly, lymphadenopathy, weight loss, or pancytopenia are not specific in advanced HIV infection. Atypical clinical presentations are becoming more frequent. This is believed to be the first report of peritoneal involvement by *Leishmania* in an AIDS patient.

Visceral leishmaniasis caused by *Leishmania donovani* has been described as an opportunistic infection in immunocompromised patients living in endemic areas (1). Patients infected with the human immunodeficiency virus (HIV) develop an impairment in cellular immunity that triggers reactivation of a latent infection and development of visceral leishmaniasis (1, 2). Typical features of visceral leishmaniasis, such as fever, hepatosplenomegaly, lymphadenopathy, weight loss or pancytopenia, can be present but are not specific to advanced HIV infection, and atypical clinical presentations are becoming more frequent. Pleuropulmonary (3–5), laryngeal (6), and digestive involvement (7, 8) in HIV-infected patients have already been described. We report the case of an HIV-infected patient who developed pleuritis and peritonitis in the course of chronic relapsing visceral leishmaniasis. *Leishmania donovani* was isolated from ascitic fluid.

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