

Short Communication

Spinal Epidural Abscess Caused by Group B *Streptococcus* in a Diabetic Woman Presenting with Febrile Low Back Pain

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SUMMARY: Because spinal epidural abscess is usually ignored in the preliminary differential diagnosis of low back pain, appropriate treatment may be delayed. Adult spinal epidural abscess is sparsely caused by the pathogen known as group B *Streptococcus*. In this paper, we report the case of a diabetic woman with lumbar epidural abscess and vertebral osteomyelitis caused by group B *Streptococcus*. Owing to the main manifestations of fever, pyuria and low back pain, which originally led us to suspect acute pyelonephritis, empirical antibiotics were applied. When the symptoms and signs persisted, other focal infections were considered. Magnetic resonance imaging led to the correct diagnosis. Group B *Streptococcus* was isolated from the blood but not from the abscess itself, probably due to the prior antibiotic treatment. The patient recovered well after surgical debridement followed by prolonged intravenous penicillin therapy. Therefore, despite the potential for fatality, our results suggest that epidural abscess can be successfully treated with surgery and antibiotic therapy provided that it is detected early enough.

Low back pain is an unspecific symptom caused by many diseases, such as neuralgia, inflammation, malignancy and infection, which may involve nerve, muscle, bone or organs in the retroperitoneal space (1,2). When low back pain is accompanied with fever, acute pyelonephritis (APN) is common. If the symptoms and signs persist after apparently adequate antibiotic treatment, the possibility of other focal infections, such as spinal epidural abscess, should be investigated. The spinal epidural abscess, although uncommon, can cause permanent neurologic dysfunction and fatality if not treated immediately (3-5). In most such cases, spinal epidural abscess is not suspected initially. The clinician should thus have a high index of suspicion for spinal epidural abscess in order to ensure an early and accurate diagnosis.

Group B streptococcal spinal epidural abscess is rarely reported (6-9), even though there is increasing recognition of the correlation between group B streptococcal infections and diabetes (10-12). Accordingly, we here report the case of a diabetic woman with clinical manifestations mimicking APN unresponsive to apparently appropriate antimicrobial therapy. A spinal epidural abscess, which also involved the vertebral body, was diagnosed with the aid of magnetic resonance imaging (MRI) of the spine. Group B streptococcal bacteremia was concomitantly identified. To our knowledge, there are only four other cases of group B streptococcal spinal epidural abscess in the English literature.

A 64-year-old woman with a medical history of diabetes mellitus, hypertension, and gouty arthritis presented in January, 2004 with a complaint of decreased appetite, dysuria, severe low back pain and fever over the previous 3 days. The patient had visited a regional hospital, but no diagnosis had been made. Upon arrival at our hospital, she was well

oriented with stable vital signs, but mildly febrile. Physical examination revealed a blood pressure of 136/82 mmHg, pulse rate of 106 beats/min, respiratory rate of 18 beats/min and temperature of 38.2°C. The breathing sound was clear and the heart rate was regular without murmur. The extremities were warm and freely movable without cyanosis or pitting edema. There was a remarkable knocking pain over the lower back area.

The primary laboratory data denoted leukocytosis (a white blood cell count of 22,630/mm³ with 87% neutrophils and 7% lymphocytes), and numerous white blood cells were present in a urine analysis. The Gram's stain of the urine specimen did not reveal any organism. On the first day of hospitalization, after the urine and blood cultures were collected, the patient was given intravenous cefazolin (1.0 g every 8 h) and gentamicin (60 mg every 12 h). On the 4th day of hospitalization, the urine culture showed no growth of any organism. Nevertheless, two sets of blood culture yielded group B *Streptococcus* susceptible to penicillin and vancomycin, but resistant to erythromycin (only 3 drugs were tested). On the 7th day of hospitalization, the antimicrobial therapy was shifted to intravenous crystal penicillin G (3 million units every 6 h). Nonetheless, both the fever and the low back pain remained after 3 days of penicillin G treatment. Therefore, a Tc-99m MDP whole body bone scan and an MRI of the spine were performed. The former revealed vertebral osteomyelitis in the lower lumbar spine region (Fig. 1), the latter, epidural abscess and osteomyelitis at the L5 level (Fig. 2). We consulted a surgeon for further therapy, and surgical decompression by laminectomy was performed on the 14th day of hospitalization. The patient became afebrile, and the low back pain was significantly reduced at about 2 days postoperatively. The pus culture using pus obtained at surgery revealed no growth of any organism. The Gram's stain and acid-fast stain of the epidural pus did not reveal any organism. A vaginal culture was not performed because no leucorrhoea or local symptoms were noticed. The echocardiogram revealed good

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Fig. 1. Osteomyelitis scan indicating increased uptake over the lumbar region.

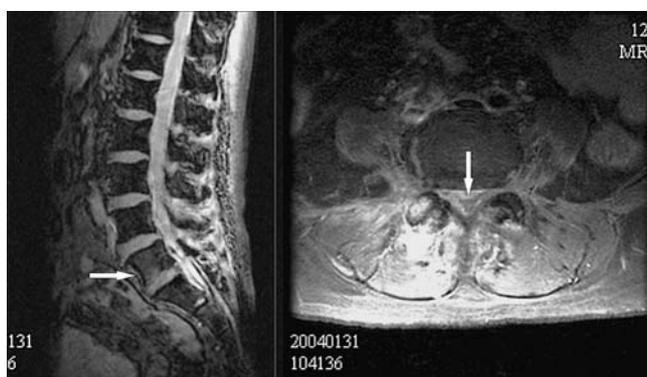


Fig. 2. Left: T₂-weighted sagittal MRI of the spine showing osteomyelitis at L5 (white arrow). Right: T1 enhancement evincing an occupying lesion with high intensity over epidural space at L5 (white arrow), compatible with epidural abscess.

cardiac performance, and normal cardiac valves without evidence of vegetations. The patient was discharged uneventfully after a total of 40 days of intravenous crystal penicillin G therapy and continued to take oral antibiotics with amoxicillin (500 mg four times per day) for another 4 months. The epidural pus culture for *Mycobacterium tuberculosis* revealed no growth of any organism.

Back pain is one of the most common complaints among individuals who come to emergency departments. Although emergency rooms encounter and treat both non-urgent conditions (e.g., back strain or acute disc herniation) and life-threatening conditions (e.g., rupture of abdominal aortic aneurysm), spinal epidural abscess has an estimated incidence of less than one case per million in the general population, or 0.2 to 2 cases per 10,000 hospital admissions (4), and thus is rarely included in the differential diagnosis (3-5). The symptoms and signs of epidural abscess consist of fever, malaise, back pain, and neurological signs such as paresis, genital-urinary dysfunction, and mental change (3-5). Consequently, it is important to consider this condition in cases of infection where the use of apparently appropriate antibiotics is ineffective.

An epidural abscess usually occurs due to the spread of infection either directly from the nearby infections or throughout the bloodstream. *Staphylococcus aureus*, the most commonly cultured germ, accounts for 57-75% of reported abscesses

(3), and other Gram-positive cocci, such as *Staphylococcus epidermidis*, *Streptococcus pneumoniae*, and viridans group streptococci, account for an additional 10% of cases, but a range of different causative organisms, including fungus, may be responsible (4). In the present case, the organism was not isolated directly from the epidural abscess, probably because it was suppressed by the prior antibiotic treatments, which consisted of 1 week of cefazolin plus gentamicin and another week of penicillin G therapy. The reason why the primary culture of the urine was negative remains unknown, although it is likely that the patient had received some antibiotics in the regional hospital prior to admission to our institution. Thus, together with negative results of the echocardiogram, the concomitant group B streptococcal bacteremia was presumed to be the microbiological etiology of the spinal epidural abscess.

Group B *Streptococcus* may colonize the vaginal and gastrointestinal tracts in healthy women and commonly cause infections in neonates and pregnant women. Invasive group B streptococcal infections in nonpregnant adults have been observed in recent decades (10-12). Nevertheless, it is extremely rare for group B *Streptococcus* to cause epidural abscess (6-9). Three of the four previously reported cases were related to acupuncture injury (6), vesicouretral reflux (7), and pregnancy (9), and the other occurred in an otherwise healthy elderly individual (8). For group B streptococcal bacteremia in nonpregnant adults, the most common underlying disease is diabetes mellitus (49%), and the most common infectious sources are urinary tract infections, pneumonia and soft tissue infections (12). Hence it is not surprising that group B *Streptococcus*, although seldom reported in the literature, caused the lumbar epidural abscess in our patient with underlying diabetes mellitus. Whether it entered through the vaginal or gastrointestinal tract remained uncertain, because we did not perform stool and vaginal cultures to survey for group B *Streptococcus*. Other conditions predisposing to invasive group B *Streptococcus* infections in patients other than neonates and pregnant women include underlying malignancy, liver cirrhosis and arteriosclerotic disease (10,11).

The risk factors for spinal epidural abscess include trauma, immunodeficiency (such as AIDS), alcoholism, diabetes mellitus, distal site infection, spine surgery, blood stream infection and intravenous drug abuse (4,13). Yet, in up to one-third of patients, there is no proved source of infection elsewhere (4). A spinal epidural abscess may be seen in patients with infections of the vertebral column (14,15). The only risk of our case responsible for group B streptococcal epidural abscess and concomitant vertebral osteomyelitis was attributed to diabetes mellitus.

With the aid of MRI, spinal epidural abscess is routinely detected much earlier and more easily (16-18). MRI is the cornerstone for establishing the diagnosis (4), and its sensitivity and specificity are 95 and 92% when vertebral osteomyelitis is present (16-18). CT scanning with intravenous contrast may also reveal fluid collections in the epidural space (16,17).

Once the diagnosis is made, empiric antibiotic therapy should be started instantly, and should consist of coverage of Gram-positive cocci, in particular staphylococci, as well as Gram-negative bacilli. Third-generation cephalosporins offer excellent Gram-positive and Gram-negative coverage, as well as penetration into the epidural space (19). The unresponsiveness of the first-line therapy (cefazolin plus

gentamicin) in our case may be explained partly by poor penetration into the epidural space. Although the efficacy of cefazolin against group B *Streptococcus* was not tested in our laboratory, it may be an alternative for the treatment of otherwise group B *Streptococcus* infections in Taiwan, based on a previous report of its effectiveness (10). Previous studies have reported that an increase of resistance to erythromycin was found in Taiwan group B *Streptococcus* isolates (20,21), and such increased resistance was also seen in our patient. Penicillin remains the drug of choice for the treatment of infections caused by group B *Streptococcus*, and resistance to this antibiotic was rarely found in Taiwan (20,21). Conversely, there have been few case reports of successful treatment of spinal epidural abscess with antibiotics alone (19,22). Antibiotics therapy alone is used only in individuals who cannot tolerate surgical intervention with an unacceptably high rate of surgical morbidity. If a patient's neurologic deficit progresses despite adequate medical treatment, emergency surgical decompression and drainage of purulent material should be performed as soon as possible, and prolonged intravenous antibiotics should be implemented for 4-6 weeks (4,23).

In conclusion, group B *Streptococcus* should be included in the differential microbiological etiology of the spinal epidural abscess in diabetic patients. Prompt surgical intervention and adequate penicillin therapy, if susceptible, may achieve a good clinical outcome.

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