

Morbidity and Mortality: University of Arkansas for Medical Sciences, Department of Emergency Medicine

A case of low back pain

**Barry Brenner MD,
PhD[^], C Robinson
MD, W Duda MD,
L Kass MD**

[^]Department of Emergency
Medicine, University of
Arkansas for Medical
Sciences

Dr. Duda (chief resident): Today's case is a 56-year-old white female who presented to our emergency room with the chief complaint of lower back pain, leg weakness, and urinary retention. She had been seen three times over the last 3 weeks in various emergency departments for back pain with developing unilateral leg weakness, and was scheduled for an outpatient MRI, but presented again with bilateral leg weakness and the new, additional symptom of urinary retention.

Dr. Kass (residency director): Before you go on, can you elaborate on the history of her previous ED visits?

Dr. Duda: She reported being admitted approximately 1 month previously for 6-7 days and being treated medically for a case of diverticulitis. A CT scan of her abdomen was done during that admission and was reported as otherwise unremarkable. Approximately 1 week after discharge she noted the gradual onset of back pain and so presented to her local ER. She was given prescriptions for a pain medication and muscle relaxant, and was discharged home. The pain progressed and she developed some associated weakness in her right leg. She returned to the same ER a week later, was given more pain medication, a steroid shot, and was again discharged home. One week later, she came to our ER. At this point the pain and weakness necessitated the use of a cane for ambulation. She was diagnosed with lumbago, treated with diazepam 5mg orally, scheduled for an outpatient MRI in one week with follow up thereafter, and discharged with advice to use an over-the-counter NSAID as needed.

Dr. Brenner (Chairman): Can you describe her examination at the time of this first visit to our ED?

Dr. Duda: At this visit, her physical examination revealed an obese white female in moderate distress from back pain. She had a blood pressure of 120/89, pulse of 118, temperature of 98.6 F, and respirations of 20. There was point tenderness on her midline back in the low lumbar region, but no other abdominal, back, flank, or costovertebral angle tenderness. The right lower extremity was diffusely mildly weak (4/5), with normal strength elsewhere. Extremity sensation was intact to light touch and pinprick. Reflexes were 2+ and symmetric. Toes were down-going to Babinski testing bilaterally. Pulses were full and symmetric with normal extremity color, warmth, and capillary refill. Rectal sphincter tone was normal, with soft brown guaiac stool in the vault, and perianal sensation was intact.

She returned 8 days later with acute loss of motor strength in the left lower extremity and urinary retention, both having started within the previous 24 hours.

She reports her pain as gradual in onset over the past 30-45 days, and is now constant and intense. She describes it as a sharp and aching pain at rest felt in the mid to lower back and radiating to the buttocks and legs (right worse than left). It is exacerbated by any motion of the trunk or lower extremities and only marginally relieved by rest and sitting.

She also notes urinary retention – hesitation with efforts at voiding, and then only being able to dribble. On review of systems she denies fevers, chills, malaise, headaches, stiff neck, chest pain, palpitations, shortness of breath, wheezing, abdominal pain, or skin rashes.

Dr. Robinson (resident): Does she have a medical history of anything such as malignancy, osteoporosis, vascular disease, diabetes, recent trauma, surgery, or intravenous drug use that would put her at increased risk for any of the serious causes of back pain?

Dr. Duda: Her past medical history is only significant for the previously mentioned episode of diverticulitis, and lower back pain experienced around 20 years ago after lifting. Her only past surgery is a hysterectomy. She denies taking any medications other than those prescribed for her back pain. She is allergic to levofloxacin and metronidazole. Her family history is significant for a father with remote history of back pain. She used to smoke but quit 3 months previously. She denies alcohol and drug use. She works as a security guard.

Dr. Kass: Can you describe her physical examination on this visit?

Dr. Duda: She was a morbidly obese white female 5'6" tall, wt 295 lbs who was brought into the ER in a wheel chair due to her inability to ambulate. She was mildly anxious and mildly in distress. Her vital signs were P 120, BP 103/73, R 18, Temp 97.8, and Pulse Ox 97%. Her neck was supple and non-tender. Her cardiac, lung, and abdominal examinations were normal. There was tenderness to palpation over the lower thoracic to lumbar spine. She had a positive straight leg raise test bilaterally (40 degrees on right and 10 degrees on left), markedly decreased motor strength in lower extremities (1/5 hip flexion and 3/5 ankle flexion and extension on right, 2/5 hip flexion and 5/5 ankle flexion and extension on left). Patellar and ankle reflexes were absent bilaterally. Perineal examination revealed normal rectal tone, normal perineal sensation, and no inguinal lymphadenopathy.

Dr. Brenner: At this point what are your immediate management concerns and differential diagnosis?

Dr. Duda: The paraplegia and urinary retention point to impingement of the spinal cord or the cauda equina. The rapidity of its development suggests diagnoses such as disc herniation, vertebral fracture, a bony metastasis with a resultant vertebral fracture, an epidural hematoma, or an epidural abscess. Other possibilities include an intrinsic tumor or a syrinx of the cord, but these are more often insidious in their presentation.

The management goals at this point were: a. to make a specific diagnosis, b. to minimize further cord impingement, and c. to obtain an immediate neurosurgical evaluation for emergent stabilization and decompression surgery.

Therefore the patient was sent for an MRI of her back, started on steroids and broad-spectrum antibiotics, and had a neurosurgery consultation in the ER.

Dr. Kass: I agree with those goals. I think it is important to point out the relative uselessness of other laboratory

data at this point. A PT/PTT may be useful if she has any other signs of bleeding or bruising and would raise the suspicion for an epidural hematoma if abnormal. A white count, erythrocyte sedimentation rate, or C-reactive protein would be fairly non-specific markers of inflammation. Blood and urine cultures should certainly be obtained as part of the evaluation for a possible epidural abscess. But none of these should delay or replace the MRI. What did the MRI reveal?

Dr. Duda: The MRI revealed discitis and osteomyelitis at the T12-L1 level with an epidural abscess that demonstrated a severe degree of impression on the spinal cord. Upon returning from MRI, the patient reported an inability to feel her legs. Repeat examination revealed 0/5 strength in her lower extremities bilaterally, absent sphincter tone, and an L1 sensory level to light touch. She was taken urgently to the operating room for decompression and drainage. Cultures of her blood, urine, and her abscess all ultimately grew *Staphylococcus aureus*. She was discharged, 5 weeks later, still on antibiotics, wheelchair-bound.

Dr. Brenner: Dr. Duda, can you review epidural abscesses for us?

Dr. Duda: Spinal epidural abscess is a rare condition where an infection develops between the dura matter and the surrounding tissue. It has an incidence of 0.2-2.8 cases per 10,000 admissions with a peak incidence in the age range of 60-70 years (1,2,3). The diagnosis can be challenging and it is frequently not considered in the initial differential for patients who present with back pain. However, if left untreated, spinal epidural abscesses become an expanding suppurative infection causing spinal cord impingement with sensory symptoms, motor dysfunction, and ultimately paralysis and death. Therefore, early diagnosis and intervention is imperative to improve outcome.

These abscesses most commonly occur in the posterior epidural space that contains adipose tissue, small arteries and a venous plexus, and they usually spread over several vertebral levels. They can, however, spread along the whole length of the spinal cord (4). Most are believed to occur from hematogenous spread from a localized focus (5) such as cellulitis, endocarditis, catheters, UTI's, peritoneal or retroperitoneal infections, and retropharyngeal abscesses (6). Risk factors include diabetes (noted in 50% of cases), intravenous drug use, renal failure, alcoholism, and immune compromise, as well as epidural catheter placement (7,8) and minor back trauma (7). However, in up to 20% of cases no predisposing condition is found (9). Direct extension can occur from osteomyelitis. *Staphylococcus aureus* is the most common causative organism (3,4).

The exact mechanism with which most abscesses damage the spinal cord is not known, but three have been demonstrated on post mortem examinations: compression of the cord with preservation of the arterial supply, compression and thrombosis of the veins, and edema/infarction of the cord (11). Compromised circulation to the cord seems to be the most commonly accepted cause (4,10), but it likely is a combination of compression and ischemia that cause the damaging effects on the spinal cord (12).

The most common presentation is a complaint of back pain, with localized tenderness noted 90% of the time (5). Fever is noted in only 33% of presentations. Associated neurologic symptoms range from radicular pain and paresthesias, seen early in the course of disease, to muscular weakness, sensory loss, rectal sphincter dysfunction and ultimately paralysis. Reflexes may be absent, normal, or hyperreflexic. Absent reflexes may indicate spinal shock with transient inhibition of spinal reflexes. Clonus and pathologic Babinski responses have been reported.

The rapidity of progression to weakness and paralysis is unpredictable (8); therefore epidural abscess should be high on the differential diagnosis for anyone with fever and back pain/tenderness (12). If epidural abscess is suspected, immediate imaging of the cord is imperative along with a prompt neurosurgical consultation.

Although CT myelography has been traditionally used, MRI is now the imaging study of choice. It is as sensitive (approximately 92%) as CT myelography (4,13) and can also detect other causes of back pain such as acute transverse myelitis or herniated disc (12,13).

Laboratory studies should be obtained, but they are typically nonspecific. There is, however, an association between white blood cell and C-reactive protein levels with eventual outcome (13). The ESR is usually elevated but not always (5,13).

Emergent surgical decompression is the treatment of choice, but there have been successful cases with



Fig. 1: Sagittal MRI demonstrating discitis and cord compression

non-surgical management in patients who are poor surgical candidates or have no neurologic deficits (4,7). Surgical treatment also allows for wound cultures, which aids specific antibiotic therapy. However empirical antibiotics with antistaphylococcal drugs should be started pending culture and sensitivity results.

Mortality rates up to 30% and more have been reported (3,4,7). Poor outcomes have been shown to be associated with increased age, degree of thecal sac compression, abscess location, surgical findings, sepsis on presentation, and duration of symptoms (14).

Early detection can be a key to reduction in morbidity and mortality; therefore physicians should keep the diagnosis of spinal epidural abscess high on their differential when evaluating high risk patients (such as diabetics, intravenous drug users, other infectious risk factors [e.g., diverticulitis]) who present with back pain, especially if associated with fever.

References

- Martin RJ, Yuan HA. Neurosurgical care of spinal epidural, subdural, and intermedullary abscesses and arachnoiditis. *Orthoped Clin North Am* 1996;27:125-136
- Mackenzie AR, Laing RB, Smith CC, Kaar GF, Smith FW. Spinal epidural abscess: the importance of early diagnosis and treatment. *J Neurol Neurosurg Psychiatr* 1998;65:209-212
- Soehle M, Wallenfang T. Spinal epidural abscess: clinical manifestations, prognostic factors, and outcomes. *Neurosurgery* 51;1:79-85
- Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: a ten year perspective. *Neurosurgery* 1990;27:177-184
- Tang HJ, Lin HJ, Liu YC, Li CM. Spinal Epidural Abscess-experience with 46 patients and evaluation of prognostic factors. *J Infect* 2002;45:76-80
- Jang YJ, Rhee CK. Retropharyngeal abscess associated with vertebral osteomyelitis and spinal epidural abscess. *Otolaryngol Head Neck Surg* 1998;19:705-708
- Nussbaum ES, Rigamonti D, Standiford H, Numaguchi Y, Wolf AL, Robinson WL. Spinal epidural abscess: A report of 40 cases and review. *Surgical Neurol* 1992;38:225-231
- Danner RL, Hartman BJ. Update of spinal epidural abscess: 35 cases and review of the literature. *Rev Infect Dis* 1987;9(2):265-274
- Vike GM, Honingford EA. Cervical spine epidural abscess in a patient with no predisposing risk factors. *Ann Emerg Med* 1996;27:777-780
- Siao P, Yagnik P. Spinal epidural abscess. *J Emerg Med* 1988;6:391-396
- Russel NA, Vaughan R, Morley TP. Spinal epidural infection. *Can J Neurol Sci* 1979;15:158-160
- Chao D, Nanda, A. Spinal epidural abscess: A diagnostic challenge. *Am Fam Phys* 2002;65(7):1341-1346
- Lee, S. Spinal epidural abscesses a case report and review of the literature. *Del Med J* 2000;72(10):433-438
- Khanna RK, Malik GM, Rock JP, Rosenblum ML. Spinal epidural abscess: evaluation of factors influencing outcome. *Neurosurgery* 1996;39:958-964