

Spinal Epidural Abscess Potentially Misdiagnosed as Multiple Myeloma

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A spinal epidural abscess must be treated promptly because it can quickly lead to frank spinal cord impairment, manifested by weakness, paraparesis, paralysis, or sphincter-control disturbances.¹⁻³ Clinical features suggestive of a spinal epidural abscess include fever, back pain, and reproducible spinal tenderness. Such an abscess may be caused by the contiguous or hematogenous spread of bacteria from a local or remote primary site of infection to the spinal epidural space.⁴ Commonly recognized predisposing conditions include infectious endocarditis, pneumonia, pharyngitis, sinusitis, and dental, cutaneous, and urinary tract infections. This report describes a patient with a spinal epidural abscess that was nearly misdiagnosed as multiple myeloma. The article discusses the etiology, clinical features, diagnosis, and treatment of spinal epidural abscesses.

CASE PRESENTATION

Initial Presentation

A 79-year-old woman presented to the emergency department (ED) with the chief complaint of increasing back pain. She stated that she had had intermittent episodes of back pain radiating to her left leg for several years. She presented to the ED because the current episode, which had begun the previous evening, involved a greater degree of pain than had previous episodes. None of her previous episodes had been evaluated.

She localized the pain to the midline lower back with radiation down the posterior aspect of her left leg. The pain was continuous and sharp in nature. In general, the patient had had no difficulty walking but had felt a degree of weakness in her left leg. She reported that she had experienced an episode of rigors with fever the previous evening; her temperature had been as high as 102°F. However, the rigors and fever had resolved within 1 hour of their onset and had not returned. The patient denied experiencing nausea, vomiting, diarrhea, dizziness, chest pain, and abdominal

pain. She also denied having any respiratory, urinary, and gastrointestinal complaints.

Medical History

The patient had no history of urinary or fecal retention or incontinence. One month previously, she had had chicken pox (varicella-zoster virus infection) that was limited to her skin and that resolved without consequence. Her medical history was significant only for mild gastritis for which she had been taking 150-mg ranitidine twice daily. She had undergone a colorectal polypectomy 20 years ago but reported no disease recurrence. She had not undergone any recent surgical procedures and denied any recent trauma. She was on no other medications, and she denied abusing drugs, using tobacco products, and consuming alcoholic beverages.

Physical Examination

On physical examination, the patient was in substantial pain but was able to walk. Her rectal temperature was 100.8°F. Her blood pressure was 110/70 mm Hg. She had a heart rate of 108 bpm and a respiratory rate of 18 breaths/min. She had a supple neck with no meningismus and no evidence of lymphadenopathy. She had no heart murmurs, rubs, or gallops. An abdominal examination showed no palpable masses, tenderness, or bruits. All of her pulses were symmetrical and strong, and she had normal rectal tone with guaiac-negative stool.

There was reproducible midline tenderness on the patient's back, from lumbar vertebra 1 to the top of

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the sacrum. A straight-leg raise test on the left side was positive at 40 degrees. However, motor function was notably weaker in the left lower extremity, with grade 3 to 4+ strength in all muscle groups. Some of the observed weakness may have been attributable to the pain. No sensory deficits were noted.

Laboratory Studies

Initial laboratory studies yielded a leukocyte count of $29 \times 10^3/\mu\text{L}$, with 56% bands and 35% segmented neutrophils. The erythrocyte sedimentation rate (ESR) was 75 mm/h. Her serum electrolyte, calcium, creatine kinase, and lactate dehydrogenase levels were normal, as were her urinalysis and liver function test results. An electrocardiogram was significant only for evidence of sinus tachycardia.

Radiographic Studies

A chest film showed no abnormalities. A pelvic film showed a small lytic lesion on the left superior pubic ramus. Thoracic and lumbar spine films showed grade 1 spondylolisthesis involving lumbar vertebra 5 and sacral vertebra 1. Femur films showed no abnormalities.

To evaluate the lytic lesion and other bony structures and to further investigate the abdomen for potential sources of infections, computed tomography (CT) scans of the abdomen, pelvis, and lumbosacral spine region were performed. The scans were scheduled to be performed both without and with an intravenous contrast medium. The noncontrast phase of the abdominal CT scan was only significant for evidence of diverticulosis; the aorta and viscera appeared normal. Also, the images of the lumbosacral spine and pelvis showed no abnormalities, including in the region of the left superior pubic ramus. Unfortunately, the patient refused the scheduled CT scans with contrast medium. No further imaging studies were ordered. A magnetic resonance imaging (MRI) study and CT myelogram were considered, but the patient would not consent to either of them.

Empiric Therapy

A pan-culture was performed, and the patient was empirically treated with ceftriaxone to cover the central nervous system. She was subsequently admitted to the hospital for intravenous antibiotic treatment and diagnostic evaluation. The differential diagnosis at the time of admission was broad and included multiple myeloma, occult intra-abdominal infection, osteomyelitis, and spinal epidural abscess.

Continued Clinical Course

On the second hospital day, the patient had several febrile episodes. Four out of 4 blood cultures grew gram-positive cocci, which were later identified as *Streptococcus pneumoniae*. Urine cultures were negative for bacteria. An echocardiogram was obtained to rule out endocarditis and revealed no valvular abnormalities. Owing to the unexplained bacteremia, bone pain, and pelvic lytic lesion, the leading diagnosis was multiple myeloma. Serum immunoprotein electrophoresis (IPEP) was subsequently performed and showed a monoclonal gammopathy suggestive of multiple myeloma. The patient's antibiotic coverage was changed to vancomycin and ampicillin/sulbactam.

On the fourth hospital day, the patient was still experiencing fevers. The *Streptococcus pneumoniae* was found to be sensitive to penicillin G, and antibiotic coverage was changed accordingly. Because multiple myeloma was the leading diagnosis, a bone marrow analysis was performed but did not show evidence of abnormal plasma cells. A urine protein analysis was also performed but showed no evidence of Bence-Jones proteins. In light of these new findings, the diagnosis of multiple myeloma was reconsidered. The possibility of osteomyelitis was entertained, and a bone scan was performed, which yielded normal results. A gallium scan was also performed and yielded normal results, as well.

By the eighth hospital day, the patient continued to experience fevers and back pain. An MRI scan of her spine was subsequently performed and showed an L5 facet joint infection, an L5-S1 disc space infection, and an epidural abscess at L5, which extended to encircle the left L5 nerve root. (The MRI scan revealed an abscess, whereas the previous noncontrast lumbosacral CT scan failed to do so.) There was no evidence of cord compression, and it was decided that there was no need for emergent neurosurgical intervention.

Intravenous antibiotic therapy was continued, and on the 12th hospital day, the patient's fever remitted. Her pain was also greatly diminished, and she was walking better. The patient remained in the hospital for 9 more days of intravenous antibiotic therapy and was subsequently discharged to a rehabilitation setting with a peripherally inserted central catheter (PICC) line in place for protracted intravenous antibiotic treatment.

Outcome

A follow-up MRI study, 25 days after the initial MRI study, showed complete resolution of the spinal epidural abscess, with lingering evidence of the original

L5-S1 discitis and osteopenia. The patient continued to receive intravenous antibiotics for a total of 6 weeks, followed by 4 weeks of oral antibiotics. She is doing well today with no lasting sequelae. The lytic lesion on the pelvic film is now thought to be of no clinical significance and has not been further evaluated. The gammopathy has persisted but has not progressed and is thought to be a monoclonal gammopathy of undetermined significance (MGUS).

DISCUSSION

Spinal epidural abscesses are rare but can cause severe neurologic impairment if not promptly diagnosed or treated. They occur in approximately 2.5 patients per 10,000 hospital admissions annually.¹ A high degree of suspicion assists with early identification, but frequently, as with the case patient, the diagnosis is delayed.

Etiology

As previously stated, spinal epidural abscesses can be caused by the hematogenous spread of bacteria from a remote site of infection to the spinal epidural space.⁴ Intravenous drug abuse and recent intravenous catheter insertion have also been noted to be common sources of hematogenous spread.⁵ Abscess may also result from direct extension of local infection from sources such as vertebral osteomyelitis, stage IV decubitus ulcers, and posttraumatic or procedural infected spinal fluid collections.⁵

Infectious endocarditis, pneumonia, pharyngitis, sinusitis, and dental, cutaneous, and urinary tract infections are all commonly recognized predisposing conditions. Other predisposing factors include degenerative joint disease, intravenous drug and alcohol abuse, diabetes mellitus, HIV infection, corticosteroid therapy, renal failure, and cellulitis.² However, the incidence of predisposing factors varies from series to series. The presence of any of these factors could suggest a spinal epidural abscess in a patient with fever, back pain, and reproducible spinal tenderness.

The case patient had no clear predisposing condition. Her only potential source of seeding infection was an episode of uncomplicated primary varicella-zoster virus infection, which had resolved 1 month prior to presentation. The patient had not had any lesions during the outbreak that appeared to be secondarily infected, but normal skin flora on any of the varicella-zoster lesions may have penetrated the dermal layer of the skin. An extensive work-up for another source of infection did not reveal a definitive source. A review of the literature on spinal epidural abscesses reveals that skin and

soft-tissue diseases are reported as the most frequent sources of infection. It is therefore likely that the case patient's previous varicella-zoster virus infection resulted in the development of the spinal epidural abscess. It is important to note, however, that a source of infection remains unidentified in up to 46% of patients.²

Which organism causes the abscess is largely dependent upon the location in which the primary bacterial infection develops. *Staphylococcus aureus* is the organism reported most often, especially when there is a primary cutaneous or subcutaneous infection or mild blunt abdominal trauma.² Streptococcal species, including *Streptococcus pneumoniae*, may also cause the abscess. These organisms may be associated with dermal infections as well. Numerous other organisms have been reported and include enterococcal species, Enterobacteriaceae, coagulase-negative staphylococci, *Mycobacterium tuberculosis*, anaerobic gram-negative bacilli, *Pseudomonas aeruginosa*, and *Eikenella corrodens*.²

Clinical Features

Heusner recognized 4 stages of increasing morbidity associated with spinal epidural abscesses: (1) spinal ache (fever, back pain, and tenderness), (2) root pain (headache, nuchal rigidity, and reflex changes), (3) impaired cord function (paresis), and (4) complete paralysis.⁶ However, the specific signs and symptoms that are manifested and the rate of symptom progression can differ for each patient. Symptoms have been observed to progress within weeks in some patients and within hours in other patients. The case patient's symptoms progressed over 2 days. Many authors believe that a rapid progression of symptoms reflects acute infection and a very poor prognosis.⁷

Most patients with spinal epidural abscesses present with back or spinal pain, weakness, and fever. The pain may exist in the absence of palpation, but marked reproducible local tenderness of the spine at the level of the abscess is frequently present.⁸ Other prominent symptoms include paresthesia, radiculopathy, and paralysis.

Laboratory study results indicative of a spinal epidural abscess include an elevated leukocyte count with bandemia, an elevated ESR, and an abnormal cerebrospinal fluid level.² The patient may or may not present with systemic illness. As the abscess enlarges however, compression of the spinal cord occurs, and myelopathic symptoms develop rapidly.⁹ The case patient had many of the hallmark features of the disease such as fever, subjective back pain, reproducible spinal tenderness with palpation, weakness, an elevated leukocyte count with bandemia, and an increased ESR.

Differential Diagnosis

Despite the case patient's signs and symptoms, the ultimate diagnosis of spinal epidural abscess was delayed for 8 days. The lack of a clear predisposing condition certainly contributed to diminished suspicion regarding the existence of such an abscess. Perhaps more important in causing a delay was the initial evidence that pointed to the possibility of multiple myeloma.

Multiple myeloma is a relatively rare disorder in which malignant plasma cells produce monoclonal immunoglobulin (Ig) proteins, usually IgG or IgA. Osteolytic lesions, anemia, renal insufficiency, and recurrent bacterial infections are the most common clinical features associated with multiple myeloma.¹⁰ Several of these features were indeed found in the case patient. Myeloma should be suspected in anyone 40 years of age or older with clinical features of unexplained bone pain or fracture, osteoporosis, osteolytic lesions, lethargy, anemia, red cell rouleaux, increased ESR or plasma viscosity, hypercalcemia, renal dysfunction, proteinuria, or recurrent infection. The classic triad of clinical signs consists of infiltration of the bone marrow by plasma cells, lytic bone lesions as observed via skeletal radiology, and the presence of M protein in the serum, urine, or both.¹¹

Multiple myeloma was initially included in the differential diagnosis for the case patient because of unexplained bony pain in the presence of an apparent lytic lesion on the inferior portion of the left pubic ramus and a presumptive underlying bacterial infection. The presence of weakness did not rule out multiple myeloma as the etiology, because myeloma may cause neurologic abnormalities. As the abnormal plasma cells of multiple myeloma divide uncontrollably, plasmacytomas form within the bone marrow. Some of these may develop into frank tumors that stimulate osteoclastic activity.¹² It is this excess osteoclastic activity that results in osteoporosis, osteolytic lesions, and susceptibility to fractures.¹² Neurologic deficits may be noted if compressive osteolytic lesions or pathologic fractures are present within the spinal column.

The diagnosis of myeloma seemed to be supported by the positive result on serum IPEP. If myeloma is suspected, however, a urine sample should be analyzed for Bence-Jones proteinuria because solitary free light chains are commonly undetected by routine serum electrophoresis.¹¹ In addition, a bone marrow biopsy is essential because infiltration of the bone marrow by plasma cells is pathognomonic of the disease. The case patient's urine was negative for Bence-Jones protein, and the bone marrow biopsy specimen showed no abnormalities, thereby essentially ruling out a diagnosis

of multiple myeloma. Although no treatment is recommended for the monoclonal gammopathy, it must be monitored closely as a proportion of patients with MGUS will develop pure multiple myeloma.¹¹

Diagnosis

The preliminary choice of radiographic modalities used in the analysis of the case patient was appropriate for the initial broad diagnostic differential but was not sensitive enough to properly assess for the presence of a spinal epidural abscess. It is unclear why the noncontrast CT scan failed to show the lytic lesion observable on the plain film of the pelvis. The use of a noncontrast CT scan followed by a contrast phase may be helpful in delineating contrast-enhancing lesions and may have aided in the evaluation of the case patient. A CT scan alone is inferior to other existing technologies when evaluating suspected spinal cord lesions.

The use of CT myelography or an MRI scan with and without gadolinium contrast is favored. MRI is preferable because it is less invasive and displays more defining images of the abscess. MRI has a distinct advantage over CT and myelography for viewing soft tissues.¹³ Prior data have shown that the extent and location of an abscess are best visualized by using MRI.^{14,15} In addition, MRI allows better identification of other potential spinal cord lesions.^{14,15} If a spinal epidural abscess is clearly thought to be the cause of a patient's condition, an emergent MRI study is warranted. It is important to remember that multiple spinal epidural abscesses may coexist in discrete locations; thus, an MRI scan of the entire spine is recommended.¹⁵ Other studies that are frequently ordered include plain radiographs, which usually have negative results (unless concomitant osteomyelitis exists), and bone scans, which may show uptake of radioisotopes due to inflammation.

Treatment

Once the diagnosis is made, treatment must begin immediately. Because of the wide spectrum of flora that potentially may be found in spinal epidural abscesses, initial empiric antibiotic therapy should be broad enough to cover gram-positive bacteria, especially *Staphylococcus aureus*, as well as gram-negative bacteria.⁵ In general, a third-generation cephalosporin and an aminoglycoside are preferred, although other regimens may be used as well. Once wound or blood cultures identify the specific organism and sensitivities are defined, the antibiotic coverage should be tailored. The duration of recommended intravenous antibiotic therapy varies but generally ranges from 6 to 12 weeks

followed by 4 weeks of oral antibiotics. This course may be further protracted in the presence of concomitant osteomyelitis.

Traditional management of spinal epidural abscesses has been decompressive laminectomy with operative débridement followed by long courses of antibiotics given intravenously and orally.^{16,17} Recent evidence, however, has both challenged and supported the necessity of emergent surgical intervention. Khanna et al¹⁸ studied prognostic factors affecting outcome in patients with spinal epidural abscess and found that there was no statistically significant difference in the outcomes of their patients treated either surgically or medically. They concluded that patients can possibly be treated medically, if they are neurologically stable, have minimal sensory deficits, and are known to be infected with a specific organism with known antibiotic susceptibilities. Heightened awareness on the part of the medical team and patient for detecting evidence of neurologic decline was included as a prerequisite to medical treatment, because rapid neurologic decline may require surgical treatment.¹⁸

Darouiche¹ showed that a delay in surgical treatment might have devastating consequences. In his analysis, patients who underwent surgery within 24 hours of the onset of paralysis did much better than those who had surgery after 3 days.¹⁸ Because of the lack of consensus, we do not recommend one approach over the other. However, patients considered to be poor surgical candidates because of underlying medical conditions, those with abscesses so extensive that laminectomy could potentially destabilize the spinal column, and those for whom the risks of surgery would outweigh the benefits should receive medical treatment alone.¹⁹ In general, the clinical situation and neurosurgeon's predilection will be the major factors that determine the treatment in the majority of cases.

Patient Outcomes

Several factors may affect patient outcomes, including the patient's presenting signs and symptoms, the duration of the signs and symptoms prior to presentation, the time delay to treatment, the degree of spinal canal compression, the patient's age, and the presence of any underlying medical conditions.^{18,20} In Khanna's review, patients presenting with only back pain or radiculopathy had a better outcome compared with those presenting with paresis or plegia.¹⁸ In Maslen's analysis, it was shown that those who were plegic preoperatively for greater than 12 hours tended not to recover neurologic function, and those plegic for greater than 36 hours had an increased risk for death.¹²

The case patient ultimately did well despite many factors that supported a poor prognosis. Her case is unusual, owing to the predisposing condition (that of the primary varicella-zoster virus infection) and the early suspicion of multiple myeloma as the cause of her signs and symptoms. Medical management was chosen for her because there was minimal neurologic impairment and no clear benefit to surgical intervention.

CONCLUSION

Spinal epidural abscesses are rare but can have serious sequelae if not appropriately diagnosed and treated. The time taken to make the diagnosis and begin treatment may affect the patient's prognosis. Misdiagnosis occurs relatively frequently because the abscesses occur only rarely and patient presentations are varied. The initial diagnoses given to patients who are later found to have spinal epidural abscesses are also varied and include musculoskeletal pathology, disc disease, tumor, osteomyelitis, infectious endocarditis, upper urinary tract infection, prostate pathology, soft-tissue infection, and disease completely unrelated to the spine.² Although complications associated with spinal epidural abscesses are frequent, patients can do well with appropriate management. Surgical with medical management, or medical management alone, is warranted depending on the clinical scenario. **HP**

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