ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 9, Issue 2, 2016

Online - 2455-3891
Print - 0974-2441

Case Report

THORACIC EPIDURAL ABSCESS DUE TO COMMUNITY-ACQUIRED METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS*

RAGHAVENDRA RAO S[1]*, SHUBHA SESHADRI[1], KARTHIK RAO N[1], NAVIN PATIL[2], A AVINASH[2], SUSHIL KIRAN KUNDER[2], TALHA AHMED[3]

[1]Department of Medicine, Kasturba Medical College, Manipal - 576 104, Karnataka India. [2]Department of Pharmacology, Kasturba Medical College, Manipal - 576 104, Karnataka, India, [3]Kasturba Medical College, Manipal - 576 104, India. Email: ragsmanipal1983@gmail.com

Received: 16 October 2015, Revised and Accepted: 21 December 2015

ABSTRACT

A spinal epidural abscess threatens the spinal cord or cauda equina by compression and also by vascular compromise. If left untreated, an expanding suppurative infection in the spinal epidural space can impinge on the spinal cord, producing sensory symptoms and signs, motor dysfunction, and ultimately paralysis, followed by death. Hence, early intervention undoubtedly improves the outcome. The authors report a case of a 27-year-old female patient, who presented with paraplegia, and was found to have thoracic epidural abscess due to community-acquired methicillin-resistant *Staphylococcus aureus*.

Keywords: Spinal cord, Cauda equina, Infection, Laminectomy, Drainage.

INTRODUCTION

Epidural abscess is a rare infectious disorder, affecting 0.2-2 patients a 10,000 hospital admissions [1]. *Staphylococcus aureus* is the most common pathogen implicated in the condition, being cultured in about 75% of the cases seen in any hospital setup. Methicillin-resistant *S. aureus* (MRSA) is responsible for 26% of these cases, making it a very significant microorganism in this area [2]. Most patients with spinal epidural abscess have one or more predisposing conditions, like an underlying disease or risk factor (which may be diabetes mellitus, alcoholism, or HIV infection), a spinal abnormality or intervention, or a potential local or systemic source of infection [3,4]. Bacteria gain access to the epidural space through contiguous spread (about one-third of cases) or hematogenous dissemination (about half of cases); in the remaining cases, the source of infection is not identified. Less common causative pathogens include coagulase-negative staphylococci, such as *Staphylococcus epidermidis* and Gram-negative bacteria, particularly, *Escherichia coli* and *Pseudomonas aeruginosa* [5].

Epidural infection can injure the spinal cord either directly by mechanical compression or indirectly as a result of vascular occlusion caused by septic thrombophlebitis [6]. Because abscesses are more likely to develop in larger epidural spaces that contain infection-prone fat, they are a more common in posterior than in anterior areas and in thoracolumbar than in cervical areas [7]. Spinal epidural abscesses, generally, extend over three to four vertebrae [8], but in rare cases, they involve the whole spine, resulting in the so-called panspinal infection [9].

CASE REPORT

A 27-year-old female had presented with a backache involving the lower thoracic region and fever for a duration of 12 days. On the 9th day after the onset of backache, she developed weakness of the lower limbs, mainly involving the distal muscles. A day later, she developed band-like sensation above the umbilicus, with diminished sensory perception below the level of the umbilicus. On the same day, she also developed urinary retention and was taken to a local hospital where she was catheterized. She was then referred to our hospital for further management. There was no history of a cough, breathlessness, anorexia, weight loss, burning micturition, intravenous drug abuse, or administration of vaccine/spinal anesthesia prior to the development of these symptoms.

On examination, the patient was febrile. There was no evident pallor or lymphadenopathy. Power in her lower limbs was 0/5, while it was 5/5 in her upper limbs. Deep tendon reflexes in lower limbs were absent, with plantar reflex also being unresponsive. Only the upper abdominal reflexes were present, with Beevor's sign positive (seen with the selective weakness of lower abdominal muscles). All modalities of sensation were diminished below the level of umbilicus. There was also localized swelling and tenderness at the level of D_9 vertebra.

Investigations done showed hemoglobin of 10.6 g/dL, total leukocyte count of 15,600 cells/cu.mm. Differential leukocyte counts showed 81% of neutrophils and 19% of lymphocytes. ESR was raised at 92 mm/hr. Liver function tests, renal function tests, serum electrolyte levels were within normal limits. Her chest X-ray was also normal. Mantoux test was negative. Magnetic resonance imaging (MRI) of the spine showed an epidural abscess, extending from D_{9-12}, measuring 9.4 cm × 0.8 cm × 0.9 cm, causing compression and anterolateral displacement of the dorsal spinal cord (Fig. 1). There was also paraspinal soft tissue collection.

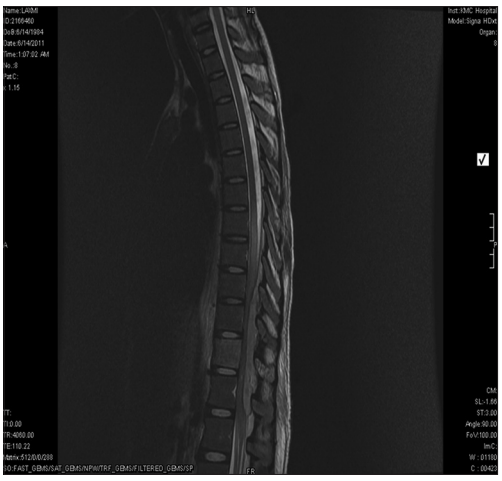

Fig. 1: Magnetic resonance imaging of thoracic spine showing D_{9-12} epidural abscess, with no evidence of vertebral osteomyelitis

A neurosurgical opinion was sought, and the patient was taken up for surgery. D_{9-12} laminectomy was done with the aspiration of the pus collection in the epidural space. The pus culture from the abscess site showed growth of MRSA. Blood culture also showed significant growth of MRSA. The patient was treated with linezolid. On the 30th post-operative day, her lower limb power had improved to 3/5 as depicted in Table 1. She was then discharged with advice to continue linezolid for 2 more weeks.

DISCUSSION

Clinical presentation of an epidural abscess may be quite variable. The clinical triad of fever, back pain, and neurologic deficit may not be present in most patients [10]. A diagnosis of a spinal epidural abscess is suspected on the basis of clinical findings and supported by laboratory data and imaging studies, but can be confirmed only by drainage. Although leukocytosis is detected in about two-thirds of patients and inflammatory markers are almost uniformly elevated, they are not specific for spinal epidural abscess. Bacteremia causing or arising from the spinal epidural abscess is detected in about 60% of the patients, more so in those infected with *S. aureus* than with other organisms [11]. Both MRI with intravenous administration of gadolinium and myelography followed by computed tomography of the spine are highly sensitive in diagnosing spinal epidural abscess [12].

Management of such patients consists of both medical and surgical therapy. Empirical antibiotic coverage should include antistaphylococcal antibiotics. With the increasing incidence of MRSA infections, coverage that includes antibiotics effective against MRSA is highly recommended.

Table 1: Progression of neurological status in the patient

Neurological status	1 day before operation	1 month after operation
C8: Hand intrinsic muscles	5/5 (R), 5/5 (L)	5/5 (R), 5/5 (L)
L2: Hip flexion	0/5 (R), 0/5 (L)	3/5 (R), 3/5 (L)
L3: Knee extension	0/5 (R), 0/5 (L)	3/5 (R), 3/5 (L)
L4: Ankle dorsiflexion	0/5 (R), 0/5 (L)	3/5(R), 3/5(L)
L5: Great toe extension	0/5 (R), 0/5 (L)	3/5 (R), 3/5 (L)
S1: Ankle plantar flexion	0/5 (R), 0/5 (L)	3/5 (R), 3/5 (L)
Sensations	Anesthesia below T_{10}	Anesthesia below L_4
Ankle reflex	Absent	+
Knee reflex	Absent	+

R: Right, L: Left, +: Present

The majority of retrospective studies provide support for the overwhelming consensus that surgical drainage together with systemic antibiotics is the treatment of choice [13]. Because the preoperative neurologic stage is the most important predictor of the final neurologic outcome, and because the rate of progression of neurologic impairment is difficult to predict, decompressive laminectomy, and debridement of infected tissues should be done as soon as possible, as evidenced by several other studies [10]. The single most important predictor of the final neurologic outcome remains the patient's neurologic status immediately before and after surgery [14].

REFERENCES

1. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: A meta-analysis of 915 patients. Neurosurg Rev 2000;23(4):175-204.
2. Louis A, Fernandes CM. Spinal epidural abscess. CJEM 2005;7:351-4.
3. Pereira CE, Lynch JC. Spinal epidural abscess: An analysis of 24 cases. Surg Neurol 2005;63 Suppl 1:S26-9.
4. Nussbaum ES, Rigamonti D, Standiford H, Numaguchi Y, Wolf AL, Robinson WL. Spinal epidural abscess: A report of 40 cases and review. Surg Neurol 1992;38(3):225-31.
5. Soehle M, Wallenfang T. Spinal epidural abscesses: Clinical manifestations, prognostic factors, and outcomes. Neurosurgery 2002;51(1):79-85.
6. Browder J, Meyers R. Pyogenic infections of the spinal epidural space: A consideration of the anatomic and physiologic pathology. Surgery 1941;10:296-308.
7. Darouiche RO, Hamill RJ, Greenberg SB, Weathers SW, Musher DM. Bacterial spinal epidural abscess. Review of 43 cases and literature survey. Medicine (Baltimore) 1992;71(6):369-85.
8. Lu CH, Chang WN, Lui CC, Lee PY, Chang HW. Adult spinal epidural abscess: Clinical features and prognostic factors. Clin Neurol Neurosurg 2002;104(4):306-10.
9. Solomou E, Maragkos M, Kotsarini C, Konstantinou D, Maraziotis T. Multiple spinal epidural abscesses extending to the whole spinal canal. Magn Reson Imaging 2004;22(5):747-50.
10. Davis DP, Wold RM, Patel RJ, Tran AJ, Tokhi RN, Chan TC, *et al.* The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. J Emerg Med 2004;26(3):285-91.
11. Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: A ten-year perspective. Neurosurgery 1990;27(2):177-84.
12. Rigamonti D, Liem L, Sampath P, Knoller N, Namaguchi Y, Schreibman DL, *et al.* Spinal epidural abscess: Contemporary trends in etiology, evaluation, and management. Surg Neurol 1999;52(2):189-96.
13. Baker AS, Ojemann RG, Swartz MN, Richardson EP Jr. Spinal epidural abscess. N Engl J Med 1975;293(10):463-8.
14. Heusner AP. Nontuberculous spinal epidural infections. N Engl J Med 1948;239(23):845-54.