J Microbiol Immunol Infect.
2008;41:215-221

Original Article

Spinal epidural abscess due to *Staphylococcus aureus*: clinical manifestations and outcomes

Wan-Chin Chen, Jiun-Ling Wang, Jann-Tay Wang, Yee-Chun Chen, Shan-Chwen Chang

Division of Infectious Diseases, Department of Internal Medicine, National Taiwan University Hospital College of Medicine, Taipei, Taiwan

Received: May 12, 2007 Revised: June 14, 2007 Accepted: August 31, 2007

Background and Purpose: Despite advances in diagnosis and treatment, spinal epidural abscess due to *Staphylococcus aureus* remains a challenge to clinicians. In this study, we describe the clinical features and outcomes of patients with spinal epidural abscess due to *S. aureus*.

Methods: Thirty one cases of spinal epidural abscess due to *S. aureus* treated at the National Taiwan University Hospital from January 2001 to December 2006 were retrospectively reviewed, using a standardized case collection form. Spinal epidural abscess was diagnosed by computed tomography or magnetic resonance imaging of the spine.

Results: The median age of subjects was 55 years (range, 20 to 90 years) and the male-to-female ratio was 4.2. All patients had spine pain and 18 (58.1%) had fever. Lumbar or lumbosacral region was the most frequently involved site of spinal epidural abscess (61.3%), and 83.9% of the patients also had vertebral osteomyelitis. Sixteen patients (51.6%) were treated successfully with antibiotics alone for a median duration of 70 days (range, 23 to 274 days), whereas the median duration of antibiotic therapy in patients undergoing surgical intervention was 102 days (range, 40 to 227 days). Renal failure, malignancy or underlying comorbid illness estimated by Charlson score was predictive of a poor prognosis with treatment failure or mortality.

Conclusion: Although medical treatment alone might benefit selected patients with spinal epidural abscess due to *S. aureus* and minimal neurologic sequelae, close monitoring of the evolution of neurologic deficits with radiographic imaging follow-up is necessary, since the rate of progression of neurologic impairment is difficult to predict.

Key words: Comorbidity; Epidural abscess; *Staphylococcus aureus*; Treatment outcome

Introduction

Spinal epidural abscess, first described in 1761 [1], remains a challenge in early diagnosis and treatment despite advances in imaging and surgical techniques. The incidence of this disease was estimated at 1 of 20,000 hospital admissions two decades ago; however, it has doubled in the past two decades owing to an aging population, increasing use of spinal instrumentation and vascular access, and drug injection [2]. Predisposing conditions to spinal epidural abscess may include the presence of certain underlying diseases, such as diabetes mellitus, liver cirrhosis, malignancy, chronic renal failure, and autoimmune disease; receipt of chemotherapy, corticosteroids and immunosuppressant agents; spinal intervention; and a potential local or systemic source of infection [2-5].

While a multitude of pathogens can cause spinal epidural abscess, *Staphylococcus aureus* remains the leading etiology. Other etiologies are coagulase-negative staphylococci, Gram-negative bacteria, particularly *Escherichia coli* and *Pseudomonas aeruginosa*, streptococci, fungi and mycobacteria [3]. Rarely, anaerobic bacteria [6] or parasitic infections [7] have been reported to be causative. Among all etiological pathogens, methicillin-resistant *S. aureus* (MRSA) accounts for about 15% [8].

Corresponding author: Dr. Shan-Chwen Chang, Division of Infectious Diseases, Department of Internal Medicine, National Taiwan University Hospital College of Medicine, 7 Chung-Shan South Road, Taipei 100, Taiwan.
E-mail: changsc@ntu.edu.tw

In recent years, the mortality rate of spinal epidural abscess has been estimated at 3-20% [8-13]; delayed diagnosis, degree of cord compression, preoperative neurologic deficits and suboptimal treatment may contribute to a poor outcome [8,11,13-21]. Optimal treatment of spinal epidural abscess may include a combination of medical and surgical interventions [2]. In most instances, once the diagnosis is made, laminectomy for drainage of epidural abscess becomes a surgical emergency [3]. However, in recent years, some studies have reported a similar outcome in patients who were treated with systemic antibiotics alone [9,10,12,20,22]. Because of the rare occurrence of spinal epidural infection, it may be difficult to conduct randomized clinical trials to determine the optimal treatment.

In this study, we aimed to describe clinical manifestations, diagnosis, treatment and outcome of adult patients with epidural abscess due to *S. aureus*, and the consequences of non-surgical therapy.

Methods

Patient population

From January 1, 2001 to December 31, 2006, patients who were aged ≥18 years and diagnosed as having infective spondylitis, infective spondylodiskitis, vertebral osteomyelitis, or epidural abscess were reviewed. Definite diagnosis of spinal epidural abscess was made by computed tomography or magnetic resonance imaging of the spine. Those with spinal epidural abscess and positive cultures of *S. aureus* from blood samples or aspirate from computed tomography-guided aspiration/biopsy were enrolled. Cases of polymicrobial infection were excluded.

A standardized case collection form was used to record demographics and clinical data, which included: presenting signs and symptoms, predisposing conditions, intervals from the first visit in our hospital with associated clinical symptoms to definite diagnosis, methods of diagnosis, laboratory and radiographic features, type of management, length of hospital stay, neurologic sequelae, duration of treatment and follow-up, and clinical outcome.

The presence of the following comorbid conditions was calculated using the Charlson score: cardiac vascular disease, congestive heart failure, peripheral vascular disease, cerebrovascular disease, dementia, chronic pulmonary disease, connective tissue disease, ulcer disease, liver disease, diabetes mellitus with or without end-organ damage, hemiplegia, renal disease, solid organ or hematologic malignancy, and acquired immunodeficiency syndrome [23].

Staging and definitions

Onset of clinical symptoms was defined as acute (≤3 days), subacute (>3 days but ≤14 days), and chronic (>14 days). A staging system was used to outline clinical symptoms, as follows [2]: stage 1, back pain at the level of the affected spine; stage 2, nerve-root pain radiation from the involved spinal area; stage 3, motor weakness, sensory deficit, and bladder and bowel dysfunction; stage 4, paralysis.

In outcome assessment, complete recovery or improvement of symptoms and signs of stage 1, 2, or 3 condition and no marked residual weakness (i.e., motor strength grades 1-2/5) was defined as a good outcome. Marked residual weakness, stage 4 conditions and recurrence or worsening clinical symptoms after discharge, or mortality due to infection, was defined as a poor outcome.

Statistical analysis

Comparisons were made between patients with good outcomes and those with poor outcomes. Continuous data were compared using Mann-Whitney *U* test. Categorical data were compared using Fisher's exact test or chi-squared test. The two-tailed significance level was set at $p<0.05$.

Results

Thirty one patients with spinal epidural abscess due to *S. aureus* were identified during the 6-year study period. The median age was 55 years (range, 20-90 years), and the male-to-female ratio was 4.2. The demographics and clinical characteristics are shown in Table 1.

Causative microorganisms were methicillin-susceptible *S. aureus* (MSSA) in 21 patients (67.7%) and MRSA in 10 patients (32.3%). In patients with MRSA infection, one was due to contiguous surgical wound infection, one intravenous drug use, and two intravascular devices. Concurrent *S. aureus* bacteremia was found in 29 patients (94%). Spinal specimens were obtained for bacterial cultures in 20 patients (64.5%), and growth of *S. aureus* was found in 12 patients (60.0%). The median serum white blood cell count was 10.86×10^9/L (range, 3.24-35.71 $\times 10^9$/L) and the median C-reactive protein level was 120 mg/dL (range, 20.1- 246.4 mg/dL).

Table 1. Baseline demographic and clinical data of 31 patients with spinal epidural abscess due to *Staphylococcus aureus*

Variable	Number of patients (%)
Median age (years) [range]	55 (20-90)
Male/female	25/6
Underlying diseases	
Diabetes mellitus	6 (19.4)
Liver cirrhosis	3 (9.7)
Chronic renal failure	5 (16.1)
Malignancy	4 (12.9)
Human immunodeficiency virus infection	2 (6.5)
Presentation	
Acute	4 (12.9)
Subacute	13 (41.9)
Chronic	14 (45.2)
Initial neurologic deficits	
Stage 1	11 (35.5)
Stage 2	9 (29.0)
Stage 3	11 (35.5)
Stage 4	0 (0.0)
Laboratory findings	
White blood cell count ($\times 10^9$/L; median) [range]	10.855 (3.240-35.710)
Alkaline phosphatase (U/L; median) [range]	272 (120-458)
C-reactive protein (mg/L; median) [range]	120 (20.1-246.4)
Median diagnosis interval (days) [range]	4 (1-71)
Location of epidural abscess	
Cervical	7 (22.6)
Thoracic	7 (22.6)
Lumbar/lumbosacral	19 (61.3)
Median abscess size (vertebral levels; range)	2 (1-6)
Associated with vertebral osteomyelitis	26 (83.9)
Etiology	
Methicillin-sensitive *Staphylococcus aureus*	21 (67.7)
Methicillin-resistant *Staphylococcus aureus*	10 (32.3)

Six patients (19.4%) had diabetes mellitus, three (9.7%) had liver cirrhosis, five (16.1%) had chronic renal failure, four (12.9%) had malignancy and two (6.5%) had human immunodeficiency virus infection. Their median Charlson score was 0 (range, 0-6). Nine patients (29%) had no underlying diseases or any associated risk factors. Risk factors for *S. aureus* infection included intravenous drug use (n = 3), preceding skin or soft tissue infection (n = 3), preceding surgical site infection (n = 2), use of central vascular device (n = 2), and recurrent *S. aureus* bacteremia (n = 2). Five patients received corticosteroids or immunosuppressive agents, and three underwent chemotherapy or radiotherapy before the onset of spinal epidural abscess.

The most commonly involved locations of spinal epidural abscess were lumbar or lumbosacral spine (n = 19, 61.3%). Two patients had abscess extending from one anatomic region to another: one had spinal epidural abscess with involvement from the level of C2 through T1; and the other from the level of T12 through L1. Twenty six patients (83.9%) had concurrent vertebral osteomyelitis. Twenty three patients (74%) underwent echocardiography, and two (6%) were diagnosed as having infective endocarditis.

All 31 patients had spine pain, and 18 (58%) had fever. Four patients (12.9%) had acute onset of symptoms, 13 patients (41.9%) had subacute onset and 14 patients (45.2%) had chronic onset. Twenty patients (65%) presented with stage 1 or stage 2 neurologic deficits. However, three of these 20 patients had progressive neurologic symptoms before the definite diagnosis of spinal epidural abscess was made, including two patients who had paraplegia or quadriplegia.

The median interval from the first hospital visit for the spinal symptoms to the diagnosis of spinal epidural abscess was 4 days (range, 1-71 days). All patients were diagnosed by magnetic resonance imaging. The characteristics of spinal epidural abscess were quite

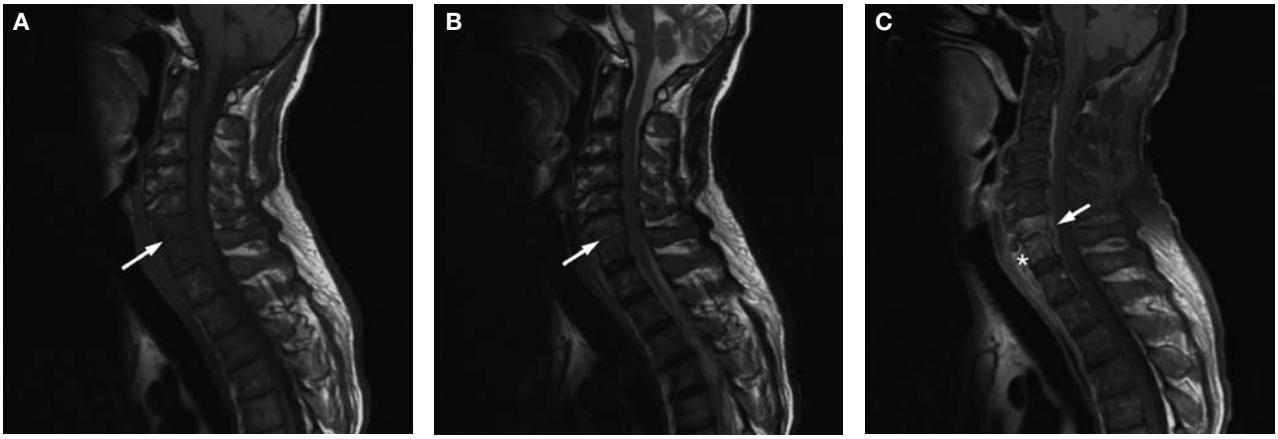

Fig. 1. Sagittal T1-weighted image (A) demonstrates low signal intensity over C6 and C7 vertebrate body (arrow), whereas in T2-weighted image (B), heterogenous high signal intensity is noted (arrow). Anterior epidural abscess is also noted from C2 through T1, with hypointensity on T1-weighted imaging, and obviously enhanced on post-gadolinium (C) T1-weighted image with rim enhancement over C6-C7 (arrow). In addition, there is soft tissue infiltration of abnormal signal and enhancement at the prespinal area of C6-T1 levels (*).

distinctive with iso- or hypointensity on T1-weighted imaging, and iso- or hyperintensity in T2-weighted imaging. Gadolinium enhancement can aid in the definition of abscess (Fig. 1). Before definite diagnosis of spinal epidural abscess was made, the initial impression gained from the presenting symptoms was: myofascial pain (n = 6), spondylosis (n = 4), radiculopathy (n = 2), compression fracture (n = 2), spondylolisthesis (n = 1), spinal tuberculosis (n = 1), malignancy (n = 1), and fever of unknown origin (n = 1).

Twenty patients (64.5%) received medical treatment alone, and 11 patients (35.5%) had combined medical treatment and surgical intervention (Table 2). More severe neurologic deficits, smaller abscess size and longer hospital stay were observed in the combined medical and surgical treatment group. In the medical treatment group, median length of hospital stay and treatment duration was 45.5 and 70 days, respectively. Two patients had deteriorating neurologic deficits while they received follow-up as outpatients, and one of them subsequently developed paralysis. One had relapse of spinal epidural abscess after discontinuation of antibiotics after a 42-day treatment course. One patient died due to infectious complications related to paralysis.

In the combined medical and surgical treatment group, the median time from diagnosis to operation was 14 days (range, 1-35 days). One patient (9.1%) died of postoperative complications of hematoma and nosocomial infection and one had relapse of disease after 119 days of treatment during outpatient follow-up. Operation was performed for intractable bone pain (n = 3), uncontrolled infection (n = 3), cord compression (n = 3), compression fracture (n = 1), or no improvement in follow-up imaging (n = 1).

In our patients, a total of 17 magnetic resonance imaging studies were performed for follow-up, and imaging showing improvement was correlated with the clinical status and final outcome (Table 3). Two patients died, the overall mortality rate being 6.5%. There was no significant difference between patients with medical treatment and those with medical and surgical treatment. Renal failure, malignancy or underlying comorbid illness defined as Charlson score >1 was strongly predictive of a poor outcome (12% vs 100%, p=0.0001).

Discussion

Despite improvement in diagnosis and treatment in recent decades, the mortality rate of spinal epidural abscess remains around 15%, and 15% of the survivors had neurologic sequalae of paresis or complete paralysis [2,3]. *Staphylococcus* spp. cause 59.2-93.0% of spinal epidural abscess cases [3]. MRSA has been reported to account for 15% of staphylococcal spinal epidural infection [8]. The risk of MRSA infection is particularly high in patients with implantable spinal or vascular devices [2]. In this era of increasing incidence of community-acquired MRSA infection, spinal epidural infection due to community-acquired MRSA has been reported recently [24,25]. More studies are needed to evaluate ongoing trends in the prevalence of community-acquired MRSA infections.

Table 2. Treatment of spinal epidural abscess: medical treatment alone versus combined medical and surgical treatment

Variable	Medical treatment (n = 20) No. (%)	Combined medical and surgical treatment (n = 11) No. (%)	p^a
Age (years; median)	55.5	52	0.89
Male/female	17/3	8/3	0.64
Charlson score			0.70
≤1	13 (65.0)	9 (81.8)	
>1	7 (35.0)	2 (18.2)	
Presentation			1.00
Acute	3 (15.0)	1 (9.1)	
Subacute or chronic	17 (85.0)	10 (90.9)	
Neurologic deficits			0.02
≤Stage 2	16 (80.0)	4 (36.4)	
>Stage 2	4 (20.0)	7 (63.6)	
Etiology			0.42
Methicillin-sensitive *Staphylococcus aureus*	15 (75.0)	6 (54.5)	
Methicillin-resistant *Staphylococcus aureus*	5 (25.0)	5 (45.5)	
Abscess location			0.68
Cervical spine	4 (20.0)	3 (27.3)	
Non-cervical spine	16 (80.0)	8 (72.7)	
Abscess extent			0.03
≤2 Vertebral levels	13 (65.0)	11 (100.0)	
>2 Vertebral levels	7 (35.0)	0 (0.0)	
Vertebral osteomyelitis	16 (80.0)	10 (90.9)	0.63
Length of stay (days; median)	45.5	58	0.02
Total duration of treatment (days)	70	102	0.19
Outcome			1.00
Good outcome	16 (80.0)	9 (81.8)	
Poor outcome	4 (20.0)	2 (18.2)	

[a]Fisher's exact test or Mann-Whitney *U* test.

The classic triad of fever, spine pain, and neurologic deficits is the hallmark of spinal epidural infection. However, not all patients have the above symptoms. Over one-half of our patients had both fever and spine pain, not including 26% who were afebrile after taking antipyretics. Misdiagnosis was common, particularly in patients without neurologic deficits or symptoms localized to the spine. Delay in diagnosis may occur, especially when fever is masked by antipyretic agents. Neither leukocytosis nor elevation of inflammatory markers is sensitive or specific enough for definitive diagnosis of the disease. Besides, 29% of our patients had no comorbid illness or any other associated risk factors for spinal epidural abscess, which is different from the findings of a previous study [14].

To most clinicians, once the diagnosis of spinal epidural abscess is made, laminectomy for drainage of the epidural abscess becomes a surgical emergency [3-5, 8,13,15,16,18,19,26,27]. Several studies have reported similar outcomes in patients who were treated with systemic antibiotics alone and patients who received both medical and surgical treatment [9,10,12,22], as we found in the present study. Several factors have been proposed as contributing to a poor prognosis, including delayed diagnosis, degree of cord compression, preoperative neurologic deficits or suboptimal treatment [8,11,13-21]. In our study, renal failure, malignancy or underlying comorbid illness calculated by Charlson score was a feature predictive of a poor outcome.

In our case series, nearly one-half of patients could be treated successfully with antibiotics alone. However, over one-half of our patients had neurologic deficits less than stage 2 at initial presentation to our hospital. In addition, most of our patients did not undergo surgical intervention within 36 h after neurologic deficits developed, and their preoperative muscle strength was 4/5. The decision regarding medical treatment or surgical intervention may depend mainly on the neurologic deficits at presentation and their evolution over time, the extent to which spinal epidural abscess involves the presence of spinal instrumentation, and the comorbidity of the patients. Some experts have suggested that

Table 3. Outcomes analysis in 31 patients with *Staphylococcus aureus* spinal epidural abscess

Variable	Good outcome (n = 25) No. (%)	Poor outcome (n = 6)[a] No. (%)	p^b
Age (years; median)	51	64.5	0.11
Male/female	20/5	5/1	1.00
Underlying disease[c]			
Diabetes mellitus	6 (24.0)	0 (0.0)	0.31
Liver cirrhosis	3 (12.0)	0 (0.0)	1.00
Chronic renal failure	2 (8.0)	3 (50.0)	0.04
Malignancy	1 (4.0)	3 (50.0)	0.02
Charlson score			<0.001
≤1	22 (88.0)	0 (0.0)	
>1	3 (12.0)	6 (100.0)	
Presentation			1.00
Acute	3 (12.0)	1 (17.0)	
Subacute or chronic	22 (88.0)	5 (83.3)	
Neurologic deficits			0.15
≤Stage 2	18 (72.0)	2 (33.3)	
>Stage 2	7 (28.0)	4 (66.7)	
Laboratory findings			
White blood cell ($\times 10^9$/L; median)	11.82	10.07	0.23
Alkaline phosphatase (U/L; median)	255	288	0.57
C-reactive protein (mg/L; median)	120	164	0.10
Diagnosis interval (days; median)	4	6.5	0.57
Etiology			
Methicillin-sensitive Staphylococcus aureus	17 (68.0)	4 (66.7)	1.00
Methicillin-resistant Staphylococcus aureus	8 (32.0)	2 (33.3)	
Abscess location			1.00
Cervical spine	6 (24.0)	1 (16.7)	
Non-cervical spine	19 (76.0)	5 (83.3)	
Abscess size			
≤2 Vertebral levels	19 (76.0)	5 (83.3)	1.00
>2 Vertebral levels	6 (24.0)	1 (16.7)	
Treatment			1.00
Medical alone	16 (64.0)	4 (66.7)	
Medical and surgical	9 (36.0)	2 (33.3)	

[a]Defined as marked residual weakness, stage 4 conditions and recurrence or worsening clinical symptoms after discharge or mortality due to infection.

[b]Fisher's exact test or Mann-Whitney *U* test.

[c]Not all patients had underlying disease.

spinal epidural abscess without neurologic deficits can be safely managed with medical treatment as long as the patient can be closely monitored [28]. However, two of our patients with medical treatment developed treatment failure with worsening complications. Therefore, urgent surgical intervention should always be considered in patients with marked motor deficits or rapid deterioration of general condition.

In conclusion, spinal epidural abscess often requires a prolonged course of antibiotic therapy, regardless of surgical intervention. Medical treatment alone can be beneficial for patients with minimal neurologic deficits, and renal failure, malignancy or underlying comorbid illness calculated by Charlson score is predictive of poor outcome. Close monitoring of the evolution of neurologic deficits with radiographic imaging follow-up is necessary.

References

1. Morgagni GB. De Sedibus et Causis Morborum per Anatomen Indagatis. In: Alexander B, ed. The seats and causes of diseases investigated by anatomy. New York: 1960:220-2.
2. Darouiche RO. Spinal epidural abscess. N Engl J Med. 2006;355:2012-20.
3. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev.

2000;23:175-204; discussion 205.
4. Baker AS, Ojemann RG, Swartz MN, Richardson EP Jr. Spinal epidural abscess. N Engl J Med. 1975;293:463-8.
5. Heusner AP. Nontuberculous spinal epidural infections. N Engl J Med. 1948;239:845-54.
6. Lechiche C, Le Moing V, Marchandin H, Chanques G, Atoui N, Reynes J. Spondylodiscitis due to *Bacteroides fragilis*: two cases and review. Scand J Infect Dis. 2006;38: 229-31.
7. Kaufman DM, Kaplan JG, Litman N. Infectious agents in spinal epidural abscesses. Neurology. 1980;30:844-50.
8. 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;52:189-96; discussion 197.
9. Savage K, Holtom PD, Zalavras CG. Spinal epidural abscess: early clinical outcome in patients treated medically. Clin Orthop Relat Res. 2005;439:56-60.
10. Sørensen P. Spinal epidural abscesses: conservative treatment for selected subgroups of patients. Br J Neurosurg. 2003;17:513-8.
11. Soehle M, Wallenfang T. Spinal epidural abscesses: clinical manifestations, prognostic factors, and outcomes. Neurosurgery. 2002;51:79-85; discussion 86-7.
12. Siddiq F, Chowfin A, Tight R, Sahmoun AE, Smego RA Jr. Medical vs surgical management of spinal epidural abscess. Arch Intern Med. 2004;164:2409-12.
13. Curry WT Jr, Hoh BL, Amin-Hanjani S, Eskandar EN. Spinal epidural abscess: clinical presentation, management, and outcome. Surg Neurol. 2005;63:364-71; discussion 371.
14. 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;26:285-91.
15. Danner RL, Hartman BJ. Update on spinal epidural abscess: 35 cases and review of the literature. Rev Infect Dis. 1987;9:265-74.
16. Lu CH, Chang WN, Lui CC, Lee PY, Chang HW. Adult spinal epidural abscess: clinical features and prognostic factors. Clin Neurol Neurosurg. 2002;104:306-10.
17. Akalan N, Ozgen T. Infection as a cause of spinal cord compression: a review of 36 spinal epidural abscess cases. Acta Neurochir (Wien). 2000;142:17-23.
18. Darouiche RO, Hamill RJ, Greenberg SB, Weathers SW, Musher DM. Bacterial spinal epidural abscess. Review of 43 cases and literature survey. Medicine (Baltimore). 1992;71:369-85.
19. Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: a ten-year perspective. Neurosurgery. 1990;27: 177-84.
20. Tung GA, Yim JW, Mermel LA, Philip L, Rogg JM. Spinal epidural abscess: correlation between MRI findings and outcome. Neuroradiology. 1999;41:904-9.
21. Khanna RK, Malik GM, Rock JP, Rosenblum ML. Spinal epidural abscess: evaluation of factors influencing outcome. Neurosurgery. 1996;39:958-64.
22. Wheeler D, Keiser P, Rigamonti D, Keay S. Medical management of spinal epidural abscesses: case report and review. Clin Infect Dis. 1992;15:22-7.
23. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. J Chronic Dis. 1987;40:373-83.
24. van Hal SJ, Post JJ. Community-acquired MRSA epiduritis in an Australian prison inmate. Med J Aust. 2004;180: 650-1.
25. Kaplan SL, Hulten KG, Gonzalez BE, Hammerman WA, Lamberth L, Versalovic J, et al. Three-year surveillance of community-acquired *Staphylococcus aureus* infections in children. Clin Infect Dis. 2005;40:1785-91.
26. Pereira CE, Lynch JC. Spinal epidural abscess: an analysis of 24 cases. Surg Neurol. 2005;63(Suppl 1):S26-9.
27. 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;38:225-31.
28. Rigamonti D, Metellus P. Spinal epidural abscess. N Engl J Med. 2007;356:638; author reply 638-9.

