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Spinal Epidural Abscess Presenting as Lower Back Pain — A Case Report and Literature Review

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Spinal epidural abscess (SEA) is a rare infectious disorder often subject to a delayed diagnosis. In two United States hospitals, the incidence was between 0.2 and 1.2 per 10000 admissions. There are still no reports about the incidence of SEA in Taiwan. In this report, we present a 57-year-old diabetic woman who suffered from severe lower back pain for one week prior to admission. Neither physical infectious signs nor obvious neutrophilia was apparent. The responses to electrotherapy and nonsteroidal anti-inflammatory drugs were poor. The result of magnetic resonance imaging (MRI) study demondtrated an epidural soft tissue mass extemding from the L3-4 to S1 vertebral level. Decompressive hemiaminectomy from L3 to L5 with pus drainage from the epidural space was performed and followed by administration of antibiotics. The pathogen was proven to be *Staphylococcus aureus*. The symptoms were relieved after surgery and there was no residual neruological deficit.

Lower back pain is a common clinical complaint. This infectious disorder, though rare, should be considered as a possibility if patients have an atypical clinical course and poor reponse to treatment. MRI can offer reliable information for diagnosis. More care should be taken with diabetic patients who have decreased immune function for fear of delayed diagnosis and management.

Key words: spinal epidrual abscess

INTRODUCTION

Spinal epidural abscess (SEA) is a relatively rare infectious spinal disorder. In two United States hospitals, the incidence was between 0.2 and 1.2 per 10000 admissions from 1947 to 1974[1]. There are still no reports about the incidence of SEA in Taiwan. The rarity of this disorder may lead to delayed diagnosis and worsening of neurological symptoms before effective treatment can be initiated. The early presentation may mimic lumbar disc disease, causing back pain, leg pain or both. While conservative treatment for lumbar degenerative disc disease is the usual initial treatment, failure to identify SEA may inevitably worsen the patient's prognosis. The purpose of this report is to acquaint physicians with this condition.

CASE REPORT

A 57-year-old woman was hospitalized for management of severe lower back pain and right

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leg pain of one week's duration. The patient denied bowel or bladder dysfunction, as well as increased back pain from coughing, sneezing, or Valsalva maneuver. There were no fever, chills, or night sweats.

Past medical history revealed nothing of significance except diabetes mellitud which went untreated for several years. Physical examination showed overall normal findings except for a positive Lasegue's test on the right leg. The lumbar spine was minimally tender to percussion and no paravertebral spasm was noted. Passive range of motion of the right hip was associated with exacerbation of the lower back symptoms. The radiating radicular pain of the right leg extended from the L3-4 to S1 dermatomal level.

The admitting laboratory studies showed a white blood cell count of 11500/cm3 with normal differentiation. Radiographs of the lumbosacral spine revealed only minimal degenerative changes. Electrotherapy (TENS) and nosteroidal anti-inflammatory drugs were prescribed to the patient to relieve symptoms, but were in vain. MRI study demonstrated a well defined epidural soft tissue mass, extending from the L3-4 to S1 vertebrae and comressing the dural sac (Fig. 1 to 4). The diagno-

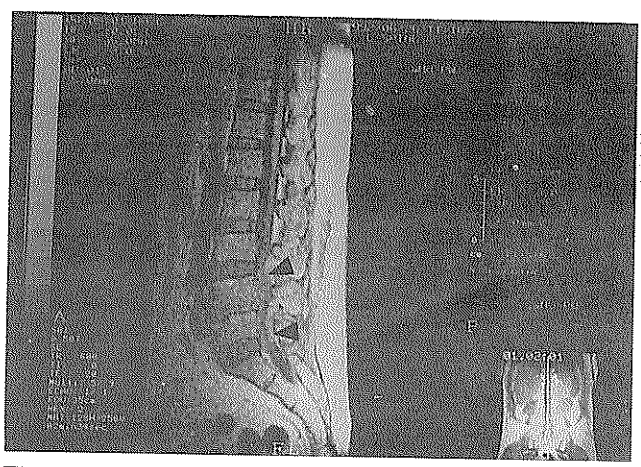

Fig 1. Magnetic resonance imaging study. Midsagittal scan of the lumbosacral spine taken with a spin-echo technique, T1-weighted image. A soft tissue mass extending from L3-4 to S1 vertebrae (arrows) and blocking the continuigy of subarachnoid space.

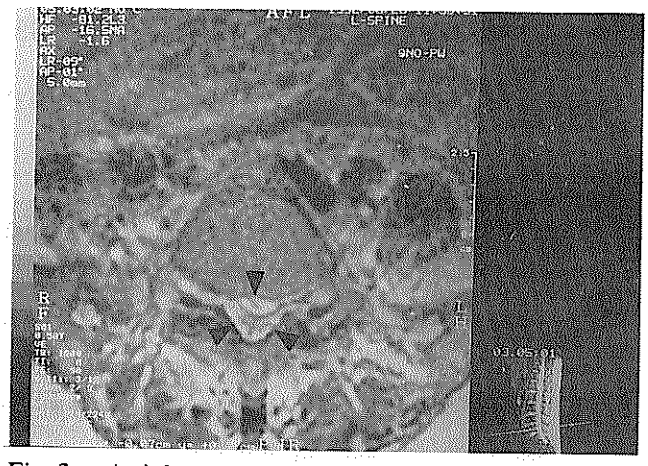

Fig 2. Axial scan, T2-weighted image. The mass has high intensity signal and encircles the dural sac (arrows).

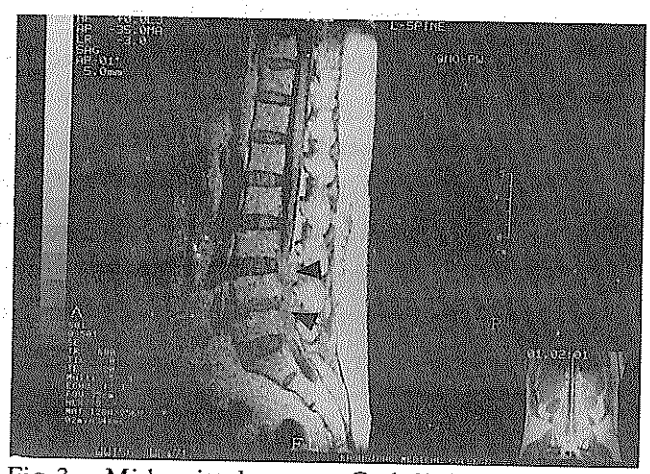

Fig 3. Midsagittal scan, Gadolinium enhanced image. The epidural mass is partially enhanced (arrows).

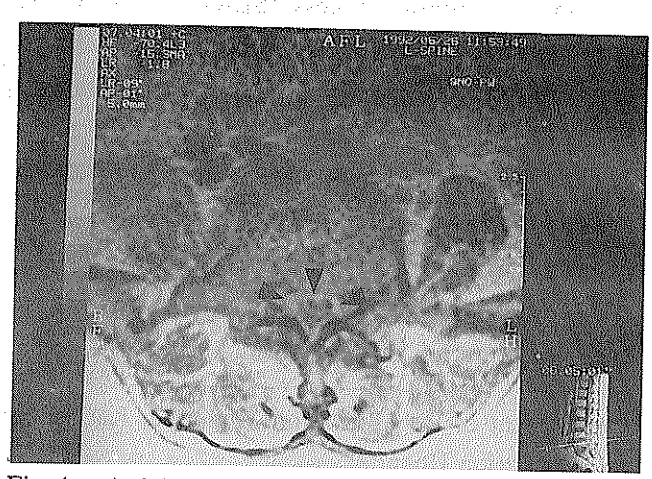

Fig 4. Axial scan, Gadolinium enhanced image. The epidural mass is enhanced (arrows).

sis of a probable epidural abscess was made and the patient underwent a decompressive hemilaminectomy from L3 to L5. Operative findings revealed a soft tissue mass above the dura from L3 to L5. Upon opening the moderately thickened membrane, purulent discharge spilled out. The decompression was completed and the wound was closed with drains. The pathogen was proven to be *Staphylococcus aureus*. During the postoperative course, she was put on a total of 2 weeks of parenteral Cefazoline® therapy, followed by oral Amoxillin® for 4 weeks. During the early postoperative period, the patient's back and right lower extremity pain diminished dramatically and little neurological deficit remained.

DISCUSSION

SEA was first described by Bergamaschi [2] in 1820. In early reports, the disease was nearly always fatal, and in most cases it was not identified until autopsy [3]. Proper surgical treatment and the advent of antibiotic therapy have improved its prognosis [2]. However, the mortality remains high, ranging from 9 to 31%, in modern series [1,2].

The routes of inoculation to the epidural space have been described previously [4]. The first, and most common, is hematogenous dissemination from distant foci of infection. Infection of the skin, particularly carbuncles and furuncles, accounts for the largest proportion [5]. The second route of infection is through local extension of primary foci. Abscesses adjacent to the spine, sush as retroperitoneal and mediastinal abscesses, have been reported to extend to the epidural space [4]. Osteomyelitis of the vertebral bodies can progress to form an epidural abscess [6]. Finally, the infection may be iatrogenically introduced during spinal procedures. These procedures include lumbar disc surgery, spinal anesthesia, epidural anesthesia, and chemonucleolysis [4,7,8]. In roughly 60% of the patients, the cultured organism from SEA is *Staphylococcus aureus* [2,4,5]. Recently, there has been an increase in the percentage of negative and anaerobic organisms [9,10]. Fungi and parasites have been reported, especially in a few immunocompromised cases [4]. This trend toward a broader source of organisms has presumably occurred secondary to the introduction of antibiotics since 1939, the increasing use of immunosuppressive agents, and the rising incidence of immunosuppressive diseases.

Trauma to the spine, occurring just before the onset of symptoms, has been reported in 10 to 40 percent of patients. However, there may be a link between the colonization of bacteria in the traumatic epidural hematomas and SEA. Most SEAs involve the dorsal dura in the thoracic region because the epidural fat, which is susceptible to bacterial invasion, lies dorsally [5]. The dura itself is resistant to bacterial penetration, so the spread of infection to the subdural or subarachnoid spaces is rare. There are two mechanisms accounting for the neurological deterioration of SEA: (1) the space occupying epidural pus compromises the spinal cord by its pressure; (2) the epidural arterial or venous thrombosis results in spinal cord infarction [3].

Individuals suffering from acute epidural abscesses are febrile and septic, whereas those with chronic processes may be afebrile and appear relatively well. Heusnen [11] has grouped the progression of symptoms into four stages according to the degree of neural involvement: (1) focal spinal pain and tenderness, (2) root pain, (3) paressis, and (4) paralysis. In acute cases, this sequence of events evolves over an average of 7 days. The duration for chronic cases may be weeks or months. Once paresis ensues, however, total paralysis generally occurs within 24 hours. The most consistent signs in acute patients are fever and spinal tenderness. These two signs are frequently found in all phases of the illness but are nonspecific. Usually, the spinal tenderness is accompanied by decreased range of motion of the entire spine [4]. Additionally, patients with SEA often experience pain during hip

flexion and exhibit a markedly positive straight leg raising sign. The presence of pain with hip flexion helps to distinguish this type of radiculopathy from that of a primary lumbar herniated disc [4].

Laboratory data suggestive of SEA are nonspecific. Patients with acute abscesses frequently have a leukocytosis. The erythrocyte sedimentation rate is nearly always markedly elevated [10]. Blood cultures are positive in approximately one out of four patients [10]. Cerebral fluid analysis generally shows an increase in total protein, pleocytosis, and a normal glucose [4,10]. Due to the great improvement of modern diagnostic imaging techiques, diagnosis no longer specifically requires lumbar puncture, which carries the risk of inducing meningitis if the needle traverses the area of infection. Whenver possible, imaging of the spine should precede lumbar puncture to avoid iatrogenic SEA.

The primary diagnosis of spinal epidural abscess lies in radiographic imaging. Plain films and bone scanning, although possibly helpful as a baseline reference in long-term follow-up, cannot be considered a useful screening procedure, given the frequent absence of abnormalities [2]. As brief reports [12,13,14] have suggested, MRI offers advantages over CT-myelograghy in the diagnosis of spinal epidural abscess. It is equally as sensitive as CT-myelography and has the advantage of distinguishing other entities in the differential diagnosis, such as herniation, syrinx, spinal tumor, spinal hematoma, cord infarct, or transverse myelitis. MRI often provides greater detail in demarcating the extent of a lesion. On plain MRI, epidural abscesses often appear as clearly defined masses that are usually isointense with the spinal cord on T1 weighted images (WI) and have a high-intensity signal on T2WI [12,13,14]. They may have a variable appearance, however, and may be seen as nonhomogenous collections of mixed signal intensities on both T1 and T2WI [12]. Most epidural abscesses can be enhanced densely and homogenously with contrast (Gd-DTPA)[15]. The total or partial enhancement of the epidural abscesses with Gd-DTPA enables a precise determination of the size and extent of the lesion, because the enhanced epidural abscess is silhouetted against the relatively lower signal of the subarachnoid sac and spinal cord [15].

Although some reports showed good results with antibiotics alone, the treatment of choice for spinal epidural abscess remains immediate surgical drainage [2]. Most reports recommend laminectomy over the entire extent of the abscess. The abscess should be drained and specimens should be sent for gram stain and aerobic and anaerobic bacterial and fungal cultures. Patients should be placed on parenteral antibiotics to cover *S. aureus* or other possible organisms. The duration of the antibiotics is empiric, with most reports recommending a minumum of four weeks of therapy after surgical debridement [1].

Some authors favor using only antibiotics for those patients who are in the early stages of the disease without evidence of neural dysfunction [16]. Leys et al. have shown good results by using CT scan for early diagnosis and a minumum period of eight weeks of parenteral antibiotics followed by oral antibiotics [17]. From those published reports, it is reasonable to suggest that the following clinical conditions should be present prior to nonperative treatment: (1) The pathogenic organism must be identified; (2) The patient should be neurologically stable; (3) Access to MRI or CT must be available for potentially rapid radiological follow up; (4) Appropriate neurosurgical consultation and nursing care must be present for sequential neurological examinations during nonoperative treatment.

In general, the neurological outcome depends on the residual cord function before drainage and decompression. Patients without paresis or with weakness of less than 36 hours' duration may have excellent neruological recoveries. Those who have been paralyzed for longer than 48 hours will make no neruological recovery.

Early intervention, before the onset of neuro-

logical deficit, has been linked to improved outcome. The key to this remains early diagnosis followed by prompt treatment. Physicians' awareness of the disease, along with an understanding of its unpredictable behavior, are vital to achieving this goal. The use of new imaging methods may play an important role in improving a patient's prognosis.

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以下背痛表現的脊椎硬膜外膿瘍 — 病例報告

汪作良　陳信穎　沈永訓*

脊椎硬膜外膿瘍(Spinal Epidural Abscess)為一相當罕見的神經系統感染疾病。其發生率國內尚無統計報告。根據美國數個醫學中心發表之統計，每10000住院人次中僅有0.2至1.2人次。因其罕見，往往造成診療之延誤。

本文報告一例57歲具有糖尿病史的中年婦女，主訴嚴重下背痛約一週，具神經根症狀，但無發燒、畏寒、倦怠、嗜中性球過多等感染表現，且對復健治療反應不佳。核磁共振掃描(MRI)發現脊椎硬膜外有T2強度訊號增加之塊狀病灶（自第三、四腰椎間至第一薦椎）。病患接受第三、四腰椎椎板切除術與膿瘍引流，並投予強效抗生素。病原菌為金黃色葡萄球菌。術後病患症狀緩解且無任何神經學後遺症。

下背痛是復健科門診常見的主訴，若患者病程不典型、對治療反應不佳，感染疾患雖罕見但務必列入鑑別診斷。核磁共振掃描常可提供優良訊息。尤其糖尿病患者，免疫能力降低，感染徵象不明顯，更須詳加檢查，以免延誤病情。

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