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Case Report

Lumbar Periradicular Abscess Mimicking a Fragmented Lumbar Disc Herniation : An Unusual Case

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We herein describe the case of a focal spontaneous spinal epidural abscess who was initially diagnosed to have a free fragment of a lumbar disc. A 71-year-old woman presented with history of low back and right leg pain. Magnetic resonance imaging suggested a peripherally enhancing free fragment extending down from S1 nerve root axilla. Preoperative laboratory investigation showed elevation of c-reactive protein (CRP), erythrocyte sedimentation rate (ESR) levels. She was taken for surgery and a fluctuating mass at the axilla of S1 nerve was found. When the mass was probed with a dissector, a dark yellow, thick pus drained out. Pus cultures were negative. Patients who present with extreme low back plus leg pain and increased leucocyte count, ESR and CRP levels should raise the suspicion of an infection of a vertebral body or spinal epidural space.

KEY WORDS : Abscess · Disc · Periradicular · Spinal.

INTRODUCTION

Abscess formation along the spinal epidural space is a rare condition with incidence rates of 0.2-2.8 cases per 10,000 population per year[1,3,6,7]. Pyogenic spondylodiscitis associated with epidural abscess is still a serious problem and despite wide usage of highly effective new generation antibiotics, suppuration along the epidural space is still a neurosurgical emergency[1,3,6,7,10].

In fact, the pyogenic infections of the back can be divided into three categories : those involving the paraspinal space, vertebral bodies, or the intervertebral disk space. Most cases of intervertebral disk space infection (diskitis) occur postoperatively and are managed with conservative therapy (medication, braces etc.) and surgery is rarely needed for patients with neurological complications, spinal instability, or progressive spinal deformity, or for patients who failed to respond to medication[11]. Spontaneous septic diskitis is a rare cause of back pain (accounting for less than 0.01% of cases) in a nonoperative patient[8].

We herein describe the case of a spontaneous diskitis in a non-operative patient which was associated with focal spinal epidural suppuration.

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CASE REPORT

A 71-year-old woman presented with two-month history of low back and right leg pain. Past medical history included use of coumadine for a reversible ischemic neurological deficit which occurred 8 months prior to emergence of her present symptoms. At that time she was hospitalized for 2 weeks and since her discharge has had her prothrombin time checked every 2-3 weeks over the last 10 months.

On admission, she was in the wheel chair bending to the left arm rest. Effort to stand still or any movement caused extreme pain. Straight leg-raising test was positive on the right side. There was no real power or sensation deficit yet the right ankle jerk was absent. Plain spine X-rays did not reveal a vertebral body infection or erosion. Preoperative laboratory investigation showed leucocyturia at urine analysis and elevation of C-reactive protein (CRP), erytrocyte sedimentation rate (ESR) levels without leucocytosis. Magnetic resonance (MR) imaging showed degenerated L5-S1 intervertebral disc with peripheral enhancement starting immediately from the posterior longitudinal ligament and a peripherally enhancing mass extending down to S1 nerve root axilla suggestive of a free disc frag-

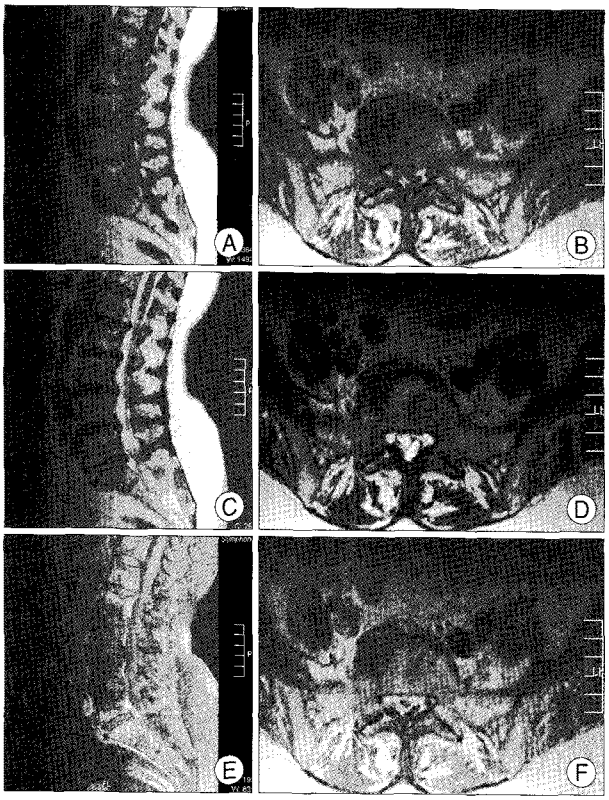

Fig. 1. Preoperative magnetic resonance (MR) images. A and B : T1-WI MR scan demontrates degenerated L5-S1 disc with a hypointense free fragment extending down from S1 nerve root axilla. C and D : This free fragment is seen hyperintense on T2-WI MR scan. E and F : It is seen hypointense with peripheral enhancement on T1-WI MR scan after gadolinium.

ment (Fig. 1). Symptoms of an insignificant bladder infection seemed to correlate well with preoperative laboratory results. Preoperative diagnosis was an extruded L5-S1 intervertebral disc herniation. She was then taken for surgery and through a right L5 hemilaminectomy and spacious S1 foraminatomy, S1 root was found to be immobile and edematous. With microscopic exploration, a fluctuating mass at the axilla of S1 nerve was found. When the mass was probed with a dissector, a dark yellow, thick pus of approximately 2-3 mL drained out. After routine sample collection for microbiology, the area was irrigated with saline and rifamycin-polyvinyl-iodine solution. Considering severly painful preoperative condition of the patient a preexisting diskitis could not be ruled out and therefore protruded L5-S1 intervertebral disc was emptied and sent for histopathology. Gram as well as Ziehl-Nielsen stains of fresh pus from the OR revealed numerous polymorpholeukocytes but was otherwise negative for microorganisms. After routine cultures, postoperative antibiotic treatment was started as intravenous vancomycin and meropenem which could effect nearly all gram negative, gram positive and anaerobic microorganisms. A chest computed tomo-

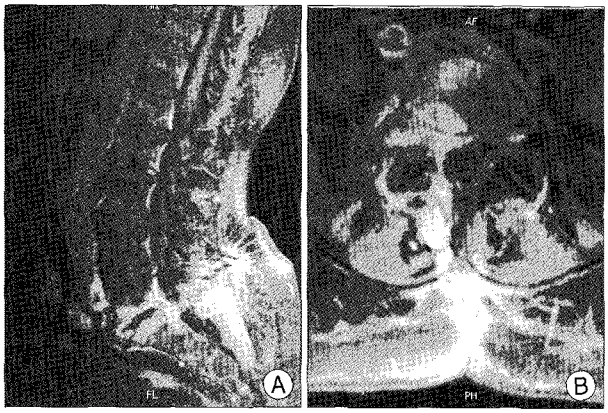

Fig. 2. Postoperative magnetic resonance (MR) images. A and B : T1-WI MR scan with gadolinium shows degenerated L5-S1 disc with no residual mass or infection at the lumbar spine.

graphy (CT) scan and abdominal ultrasound (US) to detect any other abscess formation revealed no infectious focus and blood cultures returned negative for pathogenic microorganisms. The histopathological examination of the disc specimen revealed degenerated disc material with active chronic inflammation which contained mixture of leucocytes, lymphocytes and vascularization.

The postoperative course was uneventful and two weeks later the patient was discharged from the hospital with recommendation of oral antibiotic regimen which contains amoxicillin trihidrate and potassium clavulanate combination in dosage of 2 grams per day for using two weeks. CRP level decreased to normal range and there was no fever, pain or a neurological deficit. Two months after the operation, lumbar MR imaging showed that there was still a dense inflammatory reaction at the L5-S1 intervertebral disc and the leptomeninges but the cavity previously described disappeared altogether (Fig. 2).

At one year postoperatively, she remains free of pain. CRP and ESR levels are in normal levels.

DISCUSSION

Spinal epidural suppuration is mostly seen at fifth and sixth decades of life. Risk factors mentioned in literature are infections of other body parts (skin, lung, liver), diabetes mellitus (DM), invasive procedures, IV drug abuse, alcohol abuse, chronic IV injections, spinal surgery or trauma, immunodeficiencies, lumbar puncture and other invasive procedures to the spinal column. Two-three weekly venopunctures (minimum 14-20 punctures over 10 months) for coumadine dose regulation appear to be the only risk factor in our patient[1,3,5,6,9].

Disease usually starts with fever and chills progressing to spinal and/or radicular pain. The disease usually becomes

manifest by confusion, bowel or bladder disfunction, local spinal tenderness, neck stiffness, paresthesia and paralysis. The laboratory investigations often reveal leucocytosis and elevation of erythrocyte sedimentation rate (ESR) but white blood cell count is not a reliable marker for spinal epidural suppuration and ESR is highly sensitive but less specific parameter for inflammation. Although most patients throughout the literature had moderate leukocytosis, normal serology does not preclude the diagnosis[3,5,6,9]. Most spontaneous epidural abscesses are located posterior to the dural sac of the thoracic spine (80%) followed by lumbar (30-34%) and cervical (15-19%) spine. Posterior spinal epidural abscesses often originate from an infected focus elsewhere in the body[1,3,6]. The causative microorganism reaches the spinal epidural spaces hematogenously from a focus elsewhere in the body, especially the skin. Although the most causative agent is *Staphylococcus aureus* (80% of cases), other microorganisms such as Streptococcus sp., *Escherichia coli*, Pseudomonas sp. may also be the cause of suppuration. Abscesses located anterior to the dural sac often originate from inflammation of the intervertebral disc material or from local spread of vertebral osteomyelitis. In 4-14% of patients of SEA, a causative agent may not be identified from pus cultures[1,6,9].

Radiology is often helpful for diagnosis of spinal epidural abscess (SEA). In the past CT scan has been widely used to distinguish vertebral infection and/or erosion and CT-myelography to demonstrate the compression of thecal sac. MR imaging is now the gold standard of spinal imaging and characteristics of SEA are quite distinctive. Epidural abscess may be seen iso- or hypointense on T1-weighted images (WI) and is hyperintense on T2-WI. Gadolinium enhanchement demonstrates linear enhancement surrounding non-enhancing purulent or necrotic matter[1,5,7]. MR scan characteristics of sequestrated or fragmented lumbar intervertebral discs may be quite similar to that of a focal spinal epidural abscess. A sequestrated disc will appear as low signal intensity on T1-WI and hyperintense on T2-WI because the fragment may still have a higher water content or reparative processes may lead to transient water gain. On contrast enhanced MR scans, a sequestrated or a fragmented lumbar intervertebral disc also exhibit peripheral enhancement surrounding the non-enhancing disc material. This peripheral contrast enhancement is best explained by the inflammatory response and new vascularization around the sequestrated tissue[2,4].

Despite elevated ESR and CRP counts, our patient did not have leucocytosis, and did not experience fever, chills, fatigue, spinal tenderness, neck stiffness, sphincter dysfunction or a power deficit. Preoperative radiological findings, an insignificant bladder infection and all clinical data lead us think that low back pain and the right leg pain was probably associated with lumbar disc herniation with sequestrated disc fragment rather than a rare spinal epidural suppuration.

Meticulously obtained pus cultures in our case were negative for any microorganism. This may well be due to preoperative use of single dose of prophylactic antibiotic in the OR. Gram stain of fresh pus was also negative for any microorganism. Histopathological examination of the pus revealed active chronic imflamation, but there was no granulomatous reaction or caseification. Previous series suggest that in 4-14% of patients of SEA, the causative agent may not be identified leading to sterile cultures[5,9].

The factors influencing the outcome are age, the degree of spinal cord compression, abscess location, surgical findings, septic presentation and duration of clinical symptoms[9]. In spite of two months' duration of disease our patient's outcome is excellent probably because the suppuration was too focal without lack of a reticuloendothelial system involvement (septic presentation). Focal and limited suppuration at the periradicular area, relatively early diagnosis, appropriate surgical evacuation and irrigation and postoperative use of wide-spectrum antibiotic combination (intravenous vancomycin and meropenem) as long as two weeks appear to be the key factors in the outcome of the reported patient.

CONCLUSION

Patients who present with extreme low back plus leg pain and increased leucocyte count, ESR and CRP levels should raise the suspicion of an infection of a vertebral body or spinal epidural space despite more common disc problems we see clinically. MR scan characteristics of sequestrated or fragmented lumbar intervertebral discs may be quite similar to that of a focal spinal epidural abscess and intraoperative diagnosis a focal infection can come as a surprise.

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