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이 동 국

A Case of Tuberculous Meningitis Combined with Acute Cervical Epidural Abscess and Cervical Spondylitis

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Neurologic sequelae of tuberculous meningitis include hemiparesis, paraparesis, quadriparesis, aphasia, developmental delay, dementia, blindness, visual field defect, deafness, cranial nerve palsies, epilepsy, and hypothalamic and pituitary dysfunction. But cervical epidural abscess and cervical spondylitis are rare.

A 64-year-old woman who was diagnosed as tuberculous meningitis presented a severe neck pain and stiffness after 3 weeks of anti-tuberculous medication. Electromyography and cervical X-ray showed a cervical spondylosis with polyradiculopathy. But cervical MRI showed an acute cervical epidural abscess and mild cervical spondylitis. After continuous anti-tuberculous medication with supportive care, she showed a slow clinical improvement. But about 1 month of anti-tuberculous therapy, she presented a more aggravation of neck pain, neck stiffness, radicular pain, and neck motion limitation. Follow-up cervical MRI showed an more advanced cervical spondylitis. Afterthen she has recovered slowly by cervical laminectomy with posterior stabilization and continuous anti-tuberculous medication.

Key Words : Tuberculous meningitis, Epidural abscess, Spondylitis

Tuberculous meningitis(TM) is a common disorder in neurological field and generally have a good prognosis by appropriate anti-tuberculous therapy. But sometimes TM showed various neurological sequelae include hemiparesis, paraparesis, quadriparesis, aphasia, developmental delay, dementia, blindness, visual field defect, deafness, cranial nerve palsies, epilepsy, and hypothalamic and pituitary dysfunction, And TM also complicated with spinal tuberculosis as myelitis, arachnoiditis, vasculitis, spinal cord atrophy, and occasionally the development of syringomyelia.[1-3] But TM combined with acute cervical epidural abscess and cervical spondylitis is rare. Only a case of tuberculous epidural abscess developed during the treatment of disseminated intracranial tuberculosis was reported in Korea.[4]

So I reported a case of TM combined with acute cervical epidural abscess and cervical spondylitis who was improved by anti-tuberculous therapy with supportive care, but finally has recovered by cervical laminectomy with stabilization and continuous anti-tuberculosis medication.

Case Report

A 64-year-old woman was diagnosed as TM by clinical symptoms of headache, nausea, vomitting, fever, and neck stiffness, and CSF findings of elevated cell counts and protein level. Past history revealed that she had hypertension and osteoarthritis at both knee joints. After diagnosis of TM, she was treated by anti-tuberculous therapy including isoniazid, rifampin, pyrazinamide, and kanamycin injection. Her symptoms and vital

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signs were improved gradually after anti-tuberculous therapy. After 3 weeks of anti-tuberculous therapy, she presented severe neck pain, radicular pain with neck motion limitation. Neurological examination showed neck stiffness and positive Lhermitte sign. But deep tendon reflexes were bilaterally symmetrical and urinary and fecal sphincter dysfunction were absent. Her body temperature was 38-39。C. Electromyography showed a cervical polyradiculopathy. Cervical X-ray revealed a cervical spondylosis. Median and ulnar somatosensory evoked potential were normal. But her symptoms were not improved by supportive care. Finally cervical MRI which was checked after 4 days of neck symptoms onset showed an acute cervical epidural abscess and mild cervical spondylitis(Fig. 1). Afterthen she was improved slowly by continuous anti-tuberculous therapy with supportive care. But after 1 month, she presented a more aggravation of neck pain, neck stiffness, radicular pain, and neck motion limitation. She was diagnosed as more advanced cervical spondylitis by follow-up MRI. Afterthen she has recovered slowly by laminectomy of C1 with posterior stabilization of occiput to C4 and continuous anti-tuberculous medication.

Discussion

TM is a quite common subacute meningitis and delay in treatment is associated with poor prognosis with various neurological sequelae.[5,6] Spinal TM can originate in three ways as a primary tuberculous lesion from hematogenous spread from a source outside the central nervous system, via secondary extension caudally of cranial tuberculous meningeoencephalitis, and by secondary intraspinal extension from osseous or discal tuberculosis.[7-10]

A variety of infectious disease processes may occur in the paraspinal and spinal regions, among the most important of which is nontuberculous spinal epidural abscess. Although uncommon, this problem demands urgency in diagnosis and therapy. But Mycobacterium tuberculosis is an also important cause of subacute or chronic epidural infection, at times occurring without other overt evidence of tuberculosis. At present, tuberculous involvement of the spine has become less common, but still accounts for a significant number of cases of epidural spinal infections.[11-13]

Spinal epidural abscess(SEA) is a rare but potentially devastating condition in which the prognosis for survival and prevention of permanent neurologic deficit is related to the rapidity of the decision-making process. SEA typically results from direct hematogenous seeding of the epidural space from a cutaneous, pulmonary, or urinary tract source.[14,15] Staphylococcus aureus is the most common organism responsible for spine

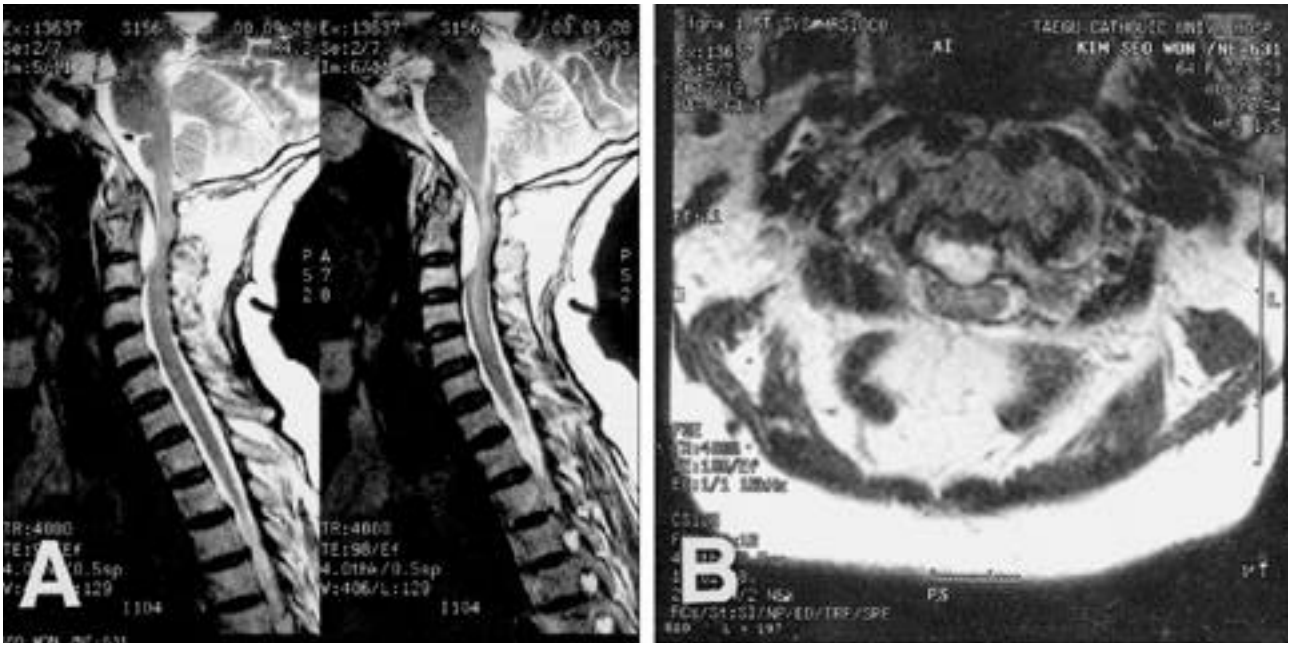

Figure 1. Sagittal(A) and axial(B) cervical MRI reveals a large anterior epidural abscess and cervical spondylitis at upper cervical level.

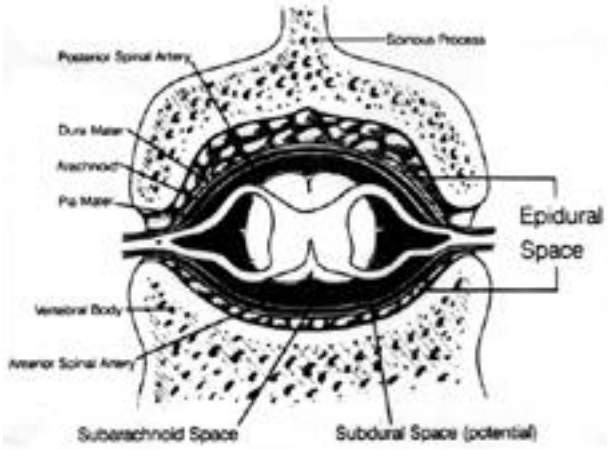

Figure 2. Anatomic localization of the epidural space.

infections of all types.[13,16]

In order to understand the pathogenesis of SEA, it is important to know the anatomy of this region.(Fig. 2)[12] Anteriorly, the dura is tightly adherent to the surface of the vertebral bodies and to the ligaments that bound the spinal canal. Thus in the anterior region the epidural space is actually only a potential space. This fact likely accounts for the relatively rarity of anterior SEA. And anterior epidural inflammatory masses are commonly associated with discitis. But in the posterolateral area the size of the epidural space is determined by the diameter of the spinal cord.[12,15,17,18] These anatomic features may partly explain the relative frequency of the location in which SEA occur. SEA is usually posterior and lateral and occur most often in the larger epidural space in the thoracic region. The lumbar region is the next most frequently involved, and abscesses in the cervical area are even less frequent.[12] But my patient showed a SEA without discitis in anterior upper cervical region.

SEA can be seperated into acute and chronic forms of infection on the basis of the rapidity of progression of symptoms and signs. And Rankin and Flothow[19] and Huesner[20] summerized the classic clinical syndrome and divided the progression of disease into 4 phases(Table 1).[12] My patient presented a fever, radicular pain, and nuchal rigidity. Therefore I thought she was at phase II state.

The most important step in the diagnosis of SEA is a heightened awareness of this disease. This diagnosis should be considered in any patient with back pain, especially if it is accompanied by other signs of infection, motor weakness, sphincter disturbance, or radicular pain. Because of the mild or nondescript initial symptoms, variety of presentations, and the rarity of this disease process, the diagnosis can be easily overlooked. In fact, most patients who eventually are diagnosed as having a SEA are initially misdiagnosed as musculoskeletal pain, arthritis, neuritis, spinal tumors or metastatic cancer, bacterial infection on other sites, vertebral osteomyelitis or infected disc, cholecystitis, pyelonephritis, intra-abdominal abscess, meningitis, infectious polyneuritis or transverse myelitis.[12,13,15,21,22] My patient also initially diagnosed as a cervical spondylosis with polyradiculopathy.

Table 1. Clinical progression of acute spinal epidural abscess

Phase **I**	Spinal ache	Back pain at affected spinal level
		Localized tenderness
		Fever may be present
Phase **II**	Root pain	Radicular pain, fever and leukocytosis prominent
		Headache and/or nuchal rigidity common
		Abnormal deep tendon reflexes
Phase **III**	Weakness	Motor impairment
		Sensory abnormalities
		Bowel and bladder dysfunction
Phase **IV**	Paralysis	Complete paralysis and sensory level

Therefore, if SEA with or without spondylitis is suspicious, simple X-ray, myelography, CT or MRI studies are considered. Plain spine film may disclose osteomyelitis and disk space narrowing. Myelography or CT-myelography demonstrates an extradural soft tissue mass with blockage of normal CSF flow.[23,24] MRI show an extradural, soft tissue mass that is iso- to hypointense compared to spinal cord on T1WI and hyperintense on proton density and T2-weighted sequences.[25] The differential diagnosis also includes epidural hematoma and epidural metastasis. Rare epidural angiolipomas and hemangiomas could also cause a similar appearance.[15]

The standard treatment for SEA includes urgent surgical decompression as laminectomy with decompression and drainage, however, in recent years a nonoperative approach has been advocated for patients without any signs of neuronal compression.[13,26] Leys and Petit[27] suggest that antibiotics are sufficient in patients not suffering from severe loss of spinal cord or cauda equina

functions, in patients whose abscess involves a considerable length of the vertebral canal, from the cervical to lumbar level, in patients who have a complete paraplegia for several days, and in patients in poor state of health because of severe concomittant medical problems. Moreover the causative agent is often isolated from blood or epidural puncture, and surgery cannot be performed only to isolate it.

And Latronico et al.[26] also reported a case of tuberculous SEA and signs of cord compression, but was successful treated without surgery. And rapid clinical improvement after the onset of the anti-tuberculous treatment decisively indicated spinal tuberculosis. So they suggested that tuberculous SEA can be fully managed with medical therapy even if a neuroligical deterioration is present, provided that the patient is closely monitored. But the same is not true for bacterial SEA, in which the signs of neural compression unquestionably demanding urgent surgical decompression. Therefore, the distinction between these two entities is more than academic, so cerrelation of clinical and MRI findings may provide sufficient discriminatory elements. And MRI, in addition to its role as a diagnostic tool, was helpful in monitoring the resolution of inflammation. And MRI is also more reliable and sensitive than clinical judgement. Therefore, urgent surgical treatment is only indicated if the spinal cord or cauda appear to be compressed with progressive loss of function. My patient presented only an fever, radicular pain, nuchal rigidity, and neck motion limitation by SEA. So she has improved after continuous anti-tuberculous therapy with supportive care.

Most tuberculous disease involving the paraspinal and spinal cord is associated with spondylitis. Spread of the tubercle bacillus to bone is generally thought to be hematogenous. The development of the vertebrae and their blood supply is important to understanding the pathogenesis of this infection. Each vertebra develops from two different sclerotomes, and each sclerotome contributes to the growth of the two adjacent vetebral bodies and the intervening disc during the embryogenesis. The blood supply established during the embryonic period remains fixed during life, with the intercostal or lumbar arteries supplying a part of each of two adjacent vertebral bodies, the lower half of the one and the upper half of the other. This may explain why most cases of tuberculous spondylitis involve adjacent vertebrae[28]. The subchondral area of the vertebral body adjacent to the disc is the most common initial site of involvement of tuberculosis in the spine[12].

Tuberculous spondylitis is a disease of decreasing incidence, and when the incidence is compared to that of developing nations, affects an older popullation. Although pulmonary tuberculosis has decreased, the incidence of bone and joint tuberculosis remains unchanged. Tuberculous spondylitis now account for 6% of new extrapulmonary tuberculosis cases. Debilitation, immunosuppression, alcoholism, and drug addiction are predisposing conditions.[29-31] Granulomatous spondylitis is most commonly caused by Mycobacterium tuberculosis. The lower thoracic and lumbar spine is most frequently involved, but cervical spine is an uncommon site. And in advanced disease several vertebrae may be destroyed with resulting significant morbidity and mortality. Bone changes generally occur slowly over 2 to 5 months and cross the disc space, spreading to adjacent vertebrae and eventually causing progressive vertebral collapse.[13,23] My patient only presented a neck pain, neck stiffness, and motion limitation. So early operation could prevented a further progressive vertebral damage.

The usual clinical presentation of spondylitis consists of fever, back pain, and nonspecific systematic symptoms of varying duration. Tuberculous spondylitis is typically more indolent than pyogenic osteomyelitis. Onset is often insidious, and symptom duration frequently ranges from months to years. More advanced disease presents with neurological deficits, kyphotic deformities of the spinal column, and paravertebral abscess. Therefore early diagnosis is crucial in the management of spine infections because delayed treatment can lead to increased morbidity and mortality.[13,21]

Plain film findings in tuberculous spondylitis include bone destruction in nearly all cases and associated soft tissue masses in most. Loss of disk height is present in more than three quarters of patients, and vertebral body fusion eventually occurs in most cases. CT scans show extensive

bony destruction and large paraspinous abscesses that are disproportionate to the amount of bone destruction. And MRI show loss of cortical definition of the affected vertebrae. But affected vertebrae are often at least partially maintained in pyogenic spondylitis.[23,30,31]

The major differential diagnosis of tuberculous spondylitis is pyogenic vertebral osteomyelitis or other spondylitides such as brucellosis, actinomycosis, and hydatid disease. Tumor is also a diagnostic consideration when a paraspinal mass is associated with bone destruction.[23,30]

Treatment has changed due to the introduction of highly effective anti-tuberculous chemotherapeutic agents and the development of new surgical methods. But these new data, controversy remains regarding the indications for surgical treatment, since anti-tuberculous therapy alone is successful in a large proportion of patients. However, when rapid loss of neurologic function is evident or progressive deterioration in spinal cord function continues in spite of adequate drug therapy, it appears that surgical decompression of the spinal cord is indicated to prevent irreversible neurologic deficits.[12,13,23,32] My patient showed a more aggravation of neck symptoms again after 1 month of anti-tuberculous therapy. And the symptoms were not improved after continuous anti-tuberculous therapy. So upper cervical laminectomy and posterior stabilization to occiput were done.

Prevention of the major paraspinal infectious processes is largely dependent upon control of the specific etiologic agents and associated epidemiologic and host factors. Tuberculous epidural infection can be decreased only by rapid detection of cases and early optimal treatment of primary infection and the use of antimicrobial agents in the treatment. Improvement in socioeconomic conditions and nutrition, along with attention to health care, are important in diminishing the spread of tuberculosis. A recent complicating factor in control of tuberculosis is its increased incidence in areas where significant HIV infection is present. So, the early detection of infection of the paraspinal area through combining a high index of clinical suspicion with the appropriate use of techniques such as myelography, CT, and MRI can be expected to diminish the morbidity and mortality of these extremely serious disease processes[12].

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