

Atypical Presentation of Spinal Tuberculosis Misdiagnosed as Metastatic Spine Tumor

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We present a case of a 68-year-old female with an atypical form of spinal tuberculosis, which involved posterior elements with multiple foci in two noncontiguous vertebral levels. The lesions caused spastic paraparesis and urinary hesitation. There was no evidence of pulmonary or other extrapulmonary tuberculous disease. Based on clinical and radiographic findings, this atypical spinal tuberculosis was preoperatively misdiagnosed as metastatic spine tumor. The histopathologic finding confirmed tuberculosis and the patient was treated successfully with surgery and antituberculous therapy. In case of a spinal lesion of unknown origin, it is important to be aware that atypical spinal tuberculosis can mimic metastatic spine tumor and tuberculosis should always be considered.

KEY WORDS : Spinal tuberculosis · Atypical · Metastatic spine tumor.

Introduction

Typical spinal tuberculosis affects the anterior part of two or more continuous vertebral levels and causes narrowing of the adjacent disc space and bone destruction, which in turn leads to anterior compression of the spinal cord. But atypical presentations are uncommon including involvement of the central part of the vertebral body^{14,21)}, involvement of the posterior elements^{13,14,16)}, neurological complication without any radiographic evidence of tuberculosis^{14,19)}, and multiple foci of infection^{5,6)}. These unusual presentations are likely to cause a delay in the diagnosis and management. And these findings lead to a misdiagnosis to metastatic spine tumor.

We report a rare case of spinal tuberculosis which involved posterior elements with multiple foci, therefore it was misdiagnosed as metastatic spine tumor.

Case Report

A 68-year-old female presented with a upper thorax pain for one month, gait disturbance for one week, and urinary hesitation for two days. Thoracic and lumbar spine magnetic resonance images(MRI) were checked in local clinic and the MRI of thoracic spine showed a bulky mass, which involved

the bodies, left transverse processes, pedicles and posterior elements of T3 and T4 vertebrae. The mass was bulging to spinal canal and compressed the spinal cord and extended to costovertebral junction and posterior part of ribs and another signal change of L3 vertebral body and pedicle was detected on lumbar spine MRI(Fig. 1). The multiple spinal metastasis were suspected in local clinic and she was transferred to hemato-oncologic department of our hospital.

On the initial neurologic examination, she had spastic paraparesis(right : power grade III and left : power grade II), Chest radiograph was normal and laboratory studies showed the C-reactive protein(CRP) 0.25mg/dL(reference range, 0-5) and several tumor markers were normal, but the erythrocyte sedimentation rate(ESR) was only slightly elevated at 38mm/hour (reference range, 0-30).

Whole body computed tomography(CT) scans were performed for the detection of primary lesion. Among them, only the chest CT showed that there were extensive osteolytic lesions with circumferential involvement of T3 and T4 vertebrae and these lesions were extended into spinal cord and ribs. CT-guided needle biopsy was done but failed to establish a diagnosis. Based on the CT scan and MRI findings, a provisional diagnosis was multiple spinal metastasis with unknown origin. Radiotherapy was planned to prevent further deterioration of

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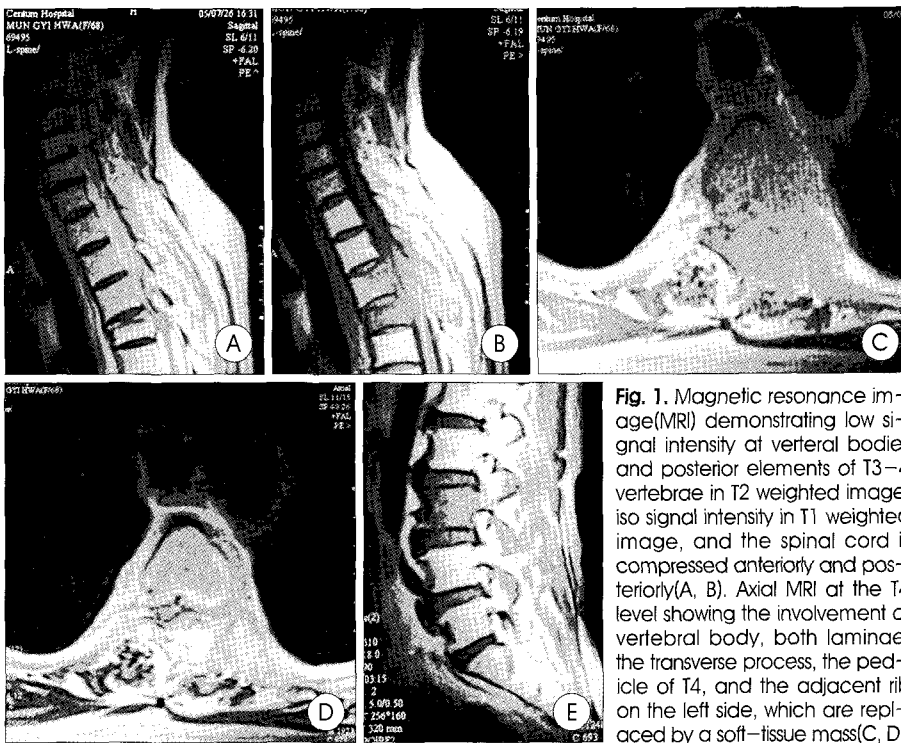


Fig. 1. Magnetic resonance image(MRI) demonstrating low signal intensity at vertebral bodies and posterior elements of T3-4 vertebrae in T2 weighted image, iso signal intensity in T1 weighted image, and the spinal cord is compressed anteriorly and posteriorly(A, B). Axial MRI at the T4 level showing the involvement of vertebral body, both laminae, the transverse process, the pedicle of T4, and the adjacent rib on the left side, which are replaced by a soft-tissue mass(C, D).

And also, T1 weighted MRI of lumbar spine revealing low signal intensity of L3 vertebral body and pedicle(E).

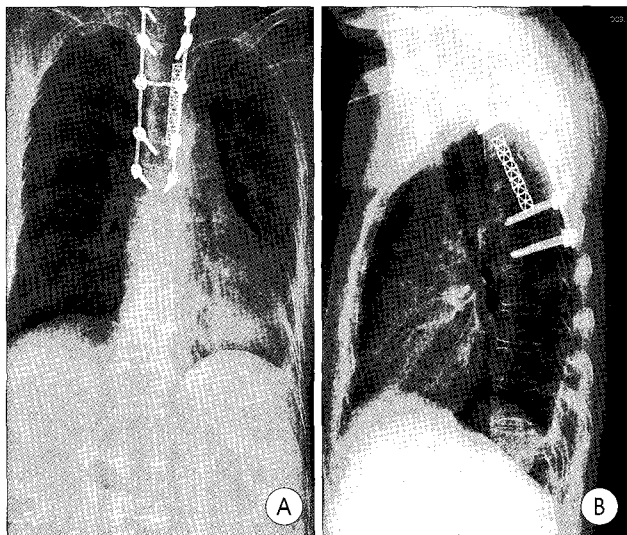


Fig. 2. Postoperative plain anteroposterior(A) and lateral(B) x-ray showing the three-level spinal reconstruction by using mesh cage and posterior stabilization by using tranpedicular screws from T1 to T6.

neurologic deficits. At two days after radiation therapy (total 600 rad), neurological symptoms had worsened. The motor weakness aggravated (right lower extremity : power grade 0, left lower extremity : power grade I) and the voiding sensation was also lost.

To release her neurological symptoms, emergency operation for mass removal was done through lateral extracavitary app-

roach. The operation began in the prone position supported by standard positioning systems and padding. A posterior midline skin incision centered on the affected levels was made long enough to expose at least two levels above and below the tumor. We used sharp dissection around posterior tumor mass, taking particular care to keep the dissection outside of that. The mass was attached to paraspinial muscle, covered posterior neural arches and attached to left-sided 3rd and 4th ribs. Then we performed a total laminectomy of T3 and T4 segments. The tissues of the mass looked pinkish gray, brown color but bleeding was less than other metastatic tumors. And not only the mass was solid with no sign of abscess formation but, looked like a solid mass. After removal of pos-

terior neural arches and facet joints, we removed the left costovertebral joints of T3 and T4 vertebrae, using a combination of rongeurs and a high-speed air drill. Once the posterolateral thecal sac had been decompressed, the spinal nerve roots of left-sided T3 and T4 were sacrificed. Posterior partial corpectomy of T3 and T4 vertebrae were done with a combination of curettes and a high-speed air drill. Disc materials adjoining the affected segments were also removed to allow proper mating of graft material to the bone surface. And then interbody fusion was done between T2 and T5 by insertion mesh cage and posterolateral fusion was done between T1, T2, T5 and T6 with using transpedicular screws(Fig. 2). The histopathologic finding showed chronic granulomatous inflammation that is suspected for tuberculosis, and the result of polymerase chain reaction for Mycobacterium tuberculosis is positive (Fig. 3).

After the surgical treatment, neurological symptoms were improved. Right leg motor recovered to power grade IV, left leg to power grade III, and self voiding was also possible. An antitubercular treatment consisting of isoniazid, rifampin, etambutol, pyrazinamide, and B6 50mg per day was started immediately and 2 weeks later, rehabilitation was also started.

Histopathologically, L3 vertebra was not confirmed. But the patient was able to walk without any support 3 months after surgery, and normal alignment of the lumbar spine was maintained. Based on radiographic finding and clinical symptom, we suspected that the lesion was another pathologic lesion of tuberculosis.

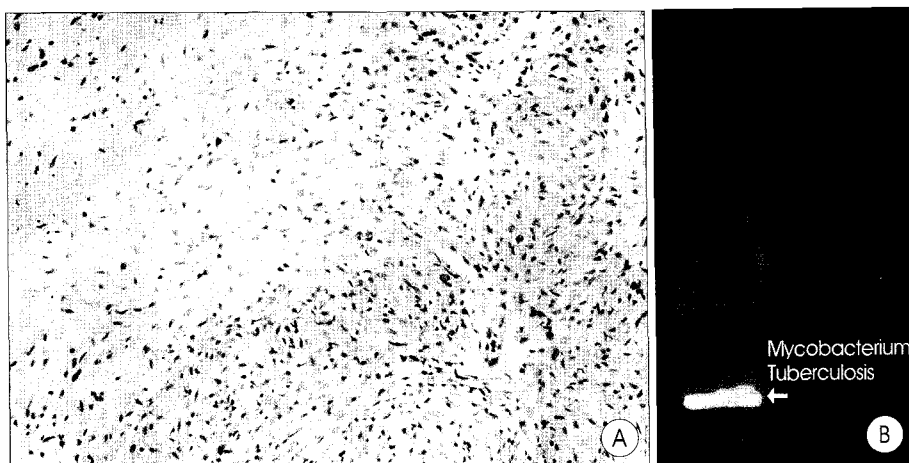


Fig. 3. A : Histological showing chronic granulomatous inflammation composed of epithelioid histiocytic aggregation (H&E, $\times 200$). B : The result of polymerase chain reaction for *Mycobacterium tuberculosis* is positive.

Discussion

The tuberculosis patients are increasing today than ever before^{3,10} due to the increasing of the aging population and other immunocompromised conditions. And in these cases the spine is a common site for extrapulmonary tuberculosis^{3,5,10}.

Spinal tuberculosis is usually initiated as a tuberculous spondylitis by hematogenous spread of organisms. Classically, there are destructions of the anterior parts of the adjacent vertebral bodies and intervertebral discs leading to the kyphotic deformity^{5,6,16,21}. Diagnosis of classic spinal tuberculosis is readily made due to the typical radiographic findings^{5,6,21}, although its differentiation from pyogenic spondylitis may still pose problems^{5,11,18}.

Atypical forms of spinal tuberculosis, including single body disease^{13,21}, skip lesions^{4,5}, neural arch tuberculosis^{12,13,15} and extradural spinal cord compression without radiographic evidence of bony involvement (spinal tumor syndrome)^{13,19}, are uncommonly reported and because of insufficient description in the medical literature, it still continues to be the source of diagnostic delays and error^{2,12,13,15}. The delay in the diagnosis may account for continuing morbidity in some case, of an otherwise curable disease.

Tuberculosis spreads to the spine from a primary focus by way of the vertebral venous system as described by Batson¹ and by arterial spread¹⁹. In the majority of the patients, there have been typical spinal lesions characterized by destruction of the adjacent bone and end plates of the bodies and the intervertebral disc²⁰. Subsequent involvement of the soft tissues results in the formation of paraspinal cold abscesses that may be the most prominent radiographic feature of the disease²¹. Other vertebrae are involved by infection tracking beneath the anterior longitudinal ligament. However, the atypical spinal

tuberculosis involving of the posterior elements is better explained by venous spread⁸. The posterior external venous plexus of vertebral veins is located on the posterior surface of the laminae and around the spinous, transverse, and articular processes¹. They anastomose freely with the other vertebral venous plexuses and constitute the final pathway for the infection to reach the neural arch¹². The most common site of atypical spinal tuberculosis is the pedicle¹⁷. Hematogenous spread of infection to the pedicle is thought to be the initial event of atypical spinal tuberculo-

sis; this assumption is based on Kumar's report that approximately 60% of the patients in a series had pedicle involvement alone⁹. We think that at the T3 or T4 level, the pedicle was the first to be involved, followed by extension of the infection from the pedicle, to the posterior element then vertebral body circumferentially. It seems that in our patient the extent of the damage was more serious in the posterior elements and less invasive to the vertebral bodies. On the other hand, most of the large series of spinal tuberculosis include few patients with skip lesions^{6,16,18}. Skip lesions, that is two or more regions of spinal tuberculosis separated by uninvolved vertebrae, were rare and most of them were separated by only a few uninvolved vertebrae^{4,7,16}. But as seen in our case, long skip lesions which were separated by more than 10 uninvolved vertebrae¹⁴, were extremely rare.

For these reason, the differential diagnosis of atypical spinal tuberculosis from metastatic spinal tumors is still difficult^{13,15,21}. In our case, we made a misdiagnosis to metastatic spine tumor, considering radiographic and clinical findings. even at operation, the lesion was solid with no sign of abscess formation and looked like a tumor. The true diagnosis was established only by histopathology. The patient was treated successfully with posterolateral decompression and antituberculosis therapy, the functional results following treatment were improved dramatically.

Conclusion

Radiographic features of typical spinal tuberculosis are well known and so the diagnosis is not so difficult in majority of cases. However, atypical spinal tuberculosis are often misdiagnosed as primary or metastatic tumors of the spine. In case of a spinal lesion of unknown origin, it is important to be aware

that atypical spinal tuberculosis can mimic metastatic spine tumor and tuberculosis should always be considered, as in our case. It is desirable that increased awareness of the abnormal features of the atypical spinal tuberculosis in the differential diagnosis of metastatic spine tumors will improve diagnostic accuracy at an early stage of disease before irreversible neurologic deficits and spinal deformities have occurred.

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