



Tuberculous meningomyelitis in magnetic resonance imaging: A Chinese case report

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ABSTRACT

Tuberculous meningomyelitis is a relatively rare but serious type of nervous system tuberculosis. This disease is caused by invasion of the spinal cord or the spinal meninges tuberculosis. The early symptoms are not typical and lack specificity. It can cause early changes in the MRI. Analysis of the MRI manifestations combined with the clinical manifestations and cerebrospinal fluid examination can facilitate accurate diagnosis of the disease. Early treatment has a clear effect, we want to increase knowledge of the disease by sharing this case in order to reduce clinical misdiagnosis and allow more patients to be treated in time.

1. Introduction

Tuberculous meningomyelitis is commonly seen in adolescents. This disease presents with a low incidence, accounting for approximately 3% [1] of systemic tuberculosis. Tuberculous meningomyelitis often presents with a history of tuberculosis or a history of contact. Patients with tuberculous meningomyelitis are primarily characterized by paralysis of the lower limbs, sensory disturbances and urination disorder. A significant increase of cerebrospinal fluid protein is the primary characteristic of the disease. The pathogenesis of this disease are tuberculous meningitis followed by spread of spinal tuberculosis to the spinal canal. The thoracic segment is the most common site followed by the lumbar and neck segments [2]. The diagnosis of the disease depends primarily on CSF and MRI examination. MRI can show spinal cord swelling, thickening of the spinal membrane. The swelling spinal cord usually without obvious enhancement, The spinal membrane is clearly irregular, patchy and circular enhancement [3,4]. In the CSF examination, it was found that acid-resistant bacillus was the gold standard for the diagnosis of tuberculous meningomyelitis, however, the positive rate of acid-fast staining was very low. CSF examination usually indicates that the vertebral canal is free or obstructed, the protein is elevated, sugar and chloride are reduced, and the total number of cells is changed.

2. Case report (clinical findings)

A 20-year-old female presented to the emergency department with

weakness of the lower limbs and activity disorder. After 10 days, the condition was aggravated with paralysis of the lower limbs, urinary and fecal incontinence, low-grade fever and night sweats, weight loss, and no cough and expectoration.

Physical examination: significantly decreased bilateral nipple medial shallow feel, abated bilateral abdominal reflex, double lower limbs muscle level 0 and 1, increased muscle tone, bilateral knee and ankle reflex disappeared, positive bilateral pap, positive ankle clonus, and negative meningeal stimulation.

Auxiliary examination: chest CT with dual lung infiltrative pulmonary tuberculosis with tuberculosis pleuritis. MRI showed that the thoracic spinal cord was markedly swollen. The T2WI shows patchy long T2 signal without enhancement. The thoracic segment and lumbar spinal membrane had clear thickening and enhancement with multiple small abscess formation.

CSF biochemical and routine examination was normal without acid fast bacilli. The erythrocyte sedimentation rate (ESR) was 34 mm/h. Fiber bronchoscope sputum smear demonstrated acid bacillus, while bronchial mucosa biopsy showed tuberculosis granuloma formation. At the same time, three CSF examinations were performed repeatedly, and the acid-proof bacillus was found in the last CSF examination of cerebrospinal fluid images. The patient was diagnosed with miliary tuberculosis and tuberculous meningomyelitis of the thoracolumbar spinal cord. The patient was given the following anti-tuberculosis treatment: Rifampicin 0.45 g/d + ethambutol 0.75 g/d + pyrazinamide 1.5 g/d + isoniazid 0.6 g/d. After re-examination after two months, the lesion of the spinal cord was significantly reduced, and the

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Fig. 1. T2WI of the spine: the lesions in the spinal cord (red arrow), spinal meninges (yellow arrow) and the subdural abscess of lumbar spine (white arrow) all appeared as long T2 signals.

small abscess of the spinal membrane was significantly reduced. The strength of lower limbs was restored to 3 +, and the patient was able to walk approximately 1 km with her family's help.

3. Imaging finding

The thoracolumbar spinal cord was swollen, and there were long T2 and T1 signals. The spinal meninges were markedly thickened, and multiple microabscesses were seen on the spinal membrane. Moreover, we also observed a cystic long T2 signal under the subdural space of the lumbar segment. Enhanced scanning showed no enhancement of intramedullary lesions, clear enhancement of the spinal meninges, circular enhancement of small spinal abscesses and subdural lesions in the lumbar segment (Figs. 1–3).

4. Discussion

Tuberculosis meningomyelitis is often seen in adolescents, and its pathogenesis primarily includes the following aspects [5]. First, tuberculosis bacillus (TB) in the lung, kidney and bone can disseminate through the circulatory system. Second, tuberculosis of the spine can directly involve the spinal cord and spinal membrane. Thirdly, the pathogenic bacteria and inflammatory exudate of tuberculosis meningitis can spread to the spinal cord or meninges via the cerebrospinal fluid.

As a result, the spinal cord and spinal nerve roots are damaged due to the spinal cord vascular inflammatory response, thereby leading to spinal cord compression ischemia [6].

The disease is rare in clinical practice and has low morbidity [7]; however, it develops rapidly. Once confirmed, anti-tuberculosis treatment should be given immediately, and delayed diagnosis and treatment will cause irreversible spinal cord injury. Therefore, early

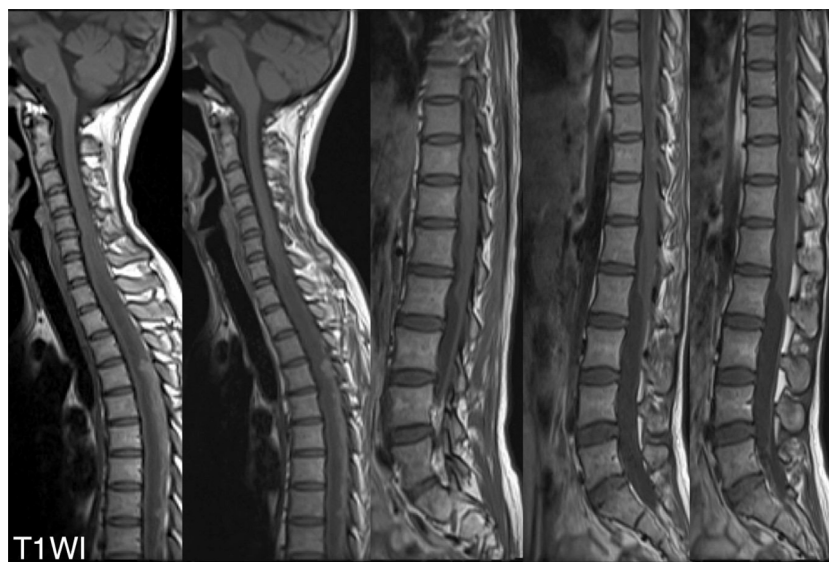


Fig. 2. T1WI of the spine: the lesions in the medullary, spinal meninges and lumbar subdural space all appeared as long T1 signals.

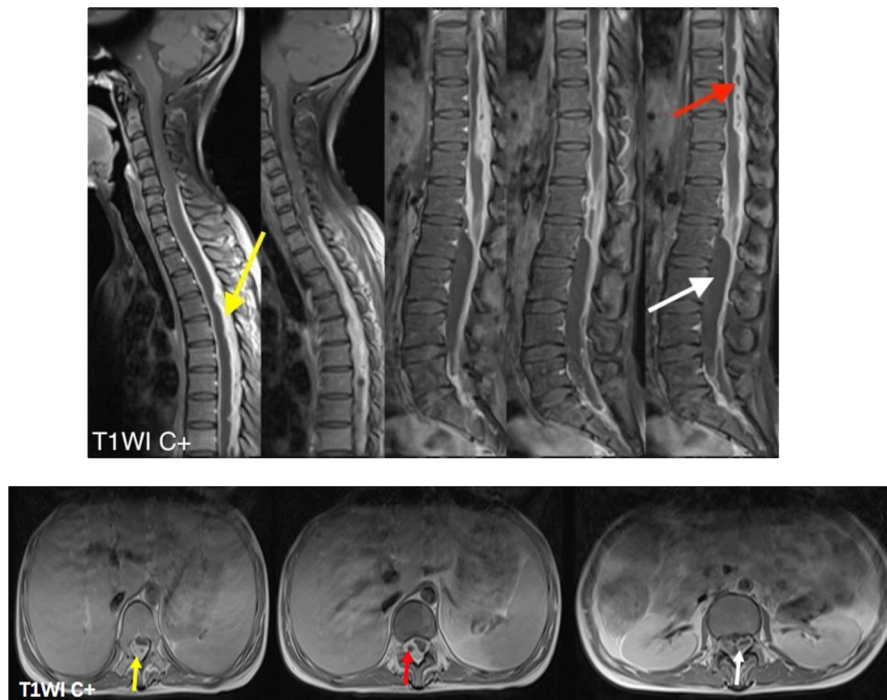


Fig. 3. Enhanced T1WI of the spine: intramedullary lesions were not enhanced. The thickened spinal membrane was markedly strengthened (yellow arrow). The abscess on the spinal membrane (red arrow) and the lumbar subdural space (white arrow) showed clear ring enhancement.

diagnosis and treatment are of utmost significance to the prognosis.

According to the statistics of the literature, treatment that is started in the first week of onset of the disease results in 70% alleviation, while initiation in the second week can alleviate 50% of cases. However, if the treatment is started more than 3 weeks after onset, the curative effect is significantly reduced.

The diagnosis of the disease primarily relies on CSF examination and MRI, where CSF is the gold standard; however, the positive rate is low, and enhanced MRI scan can clearly show the size and scope of the lesion site, MRI can identify the disease early and improve the diagnostic rate of this disease [8,9]. MRI performance is primarily divided into the following kinds: 1. Changes in the spinal membrane: the spinal membrane is mostly thickened with irregular nodules, plaques or strips, and enhanced scanning is clearly enhanced. 2. Changes in subarachnoid space: subarachnoid space narrows or disappears, cerebrospinal fluid presents a separated change or cyst formation, and enhanced scanning is not clear [10,11]. 3. Changes in nerve roots: the affected nerve roots are thickened, showing slightly longer T1 and long T2 signals, irregular scabbard sac, and mildly to moderately enhanced scanning.

4. Changes in the spinal cord: spinal cord limitations or diffuse swelling, showing a slightly long T1 and long T2 signals with slight enhancement or without enhancement.

Several patients can present with merged syringomyelia. Spinal intramedullary tuberculoma represent isolated nodular lesions, and the signal intensity depends on the maturity of the tuberculoma. The mature nodules showed long T1 and short T2 signals, and iso-T1 and long T2 signal rings were seen around them. The enhanced scanning lesions clearly exhibited ring enhancement. The immature nodule showed long T1 and long T2 signals, and the enhanced scanning lesion was uniformly enhanced.

We hope that through the introduction of this case report, clinicians can improve their understanding of this disease, reduce misdiagnosis and missed diagnosis, and enable more patients to be diagnosed in time in order to receive timely and effective treatment.

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