# Tuberculosis with Meningitis, Myeloradiculitis, Arachnoiditis and Hydrocephalus: A Case Report

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**Abstract-**

***Purpose:*** Involvement of the central nervous system (CNS) by tuberculosis is rare; it can affect either immunocompromised or immunocompetent people.

***Case report:*** Here, we report a case of tuberculosis with CNS involvement. We present the case of an immunocompetent young man who developed fever, subacute headache, disturbance of consciousness, paraparesis, sphincter dysfunction, and hypoesthesia. The final diagnosis was tuberculous meningitis, myeloradiculitis and arachnoiditis based on clinical signs, imaging studies, and cerebrospinal fluid culture. The patient received antituberculosis medication with adjunct intravenous steroid therapy. Although his clinical condition improved significantly, some neurological sequelae persisted.

***Conclusion:*** Methods for detection of CNS TB and treatment protocols should be constantly re-evaluated to improve treatment outcome and reduce likelihood and severity of neurological sequelae.

**Key Words:** arachnoiditis, meningitis, radiculomyelitis, tuberculosis

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## INTRODUCTION

Tuberculosis (TB) is a common infectious disease that most frequently affects the lungs. Approximately 1% of TB cases are complicated by infection within the nervous system[(1)]. Central nervous system (CNS) TB includes three clinical categories: meningitis, tuberculoma, and myeloradiculitis. CNS TB has the highest mortality rate (20% to 50%) among all forms of TB, and it is associated with more serious complications and sequelae[(2)]. Consequences of TB meningitis are hydrocephalus and development of vasculitis of the circle of Willis, the vertebrobasilar system, and the perforating branches of the middle cerebral artery, resulting in infarctions[(2)]. Presentation and clinical course of CNS TB vary from case to case. We present a case involving an immunocompetent young man with profound meningitis, myeloradiculitis, and arachnoiditis related to CNS TB.

## CASE REPORT

A 30-year-old Taiwanese man who had studied in Berlin, Germany presented to our infectious disease

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clinic with fever and generalized weakness of five days' duration. Laboratory data (complete blood count with differential and chemical analysis) and urinalysis were within normal limits, and chest X-ray showed no particular abnormalities. He was admitted to the infectious disease ward the following day. Ceftriaxone was started as broad-spectrum coverage for suspected salmonellosis. Fever waxed and waned (peak, roughly 38.5˚C) during the initial days of admission and was accompanied by by dizziness, vomiting, and neck stiffness. Cerebrospinal fluid (CSF) study showed an opening pressure of 400 mm$H_2O$, white blood cell count 110/$mm^3$ (78% lymphocytes), protein 289 mg/dL, and glucose 29 mg/dL. CSF India ink stain, acid-fast stain, bacterial culture, cryptococcal antigen and bacterial antigen study (Escherichia coli, Group B streptococci, Hemophilus influenzae) were all negative. Serum testing for HIV and syphilis was negative; thyroid function and autoimmune profile were within normal limits. TB meningitis was suspected. On the tenth day after admission, the patient developed numbness and weakness of his left leg. Myoclonus, bilateral hyperreflexia, and urinary retention were noted. The patient became agitated and irritable as symptoms progressed. Brain magnetic resonance imaging (MRI) revealed leptomeningeal enhancement (Fig. 1) and mild hydrocephalus. Repeat CSF study showed an opening pressure of 235 mm$H_2O$, white blood cell count 90/$mm^3$ (75% lymphocytes), protein 2.3g/dL, glucose 49 mg/dL, and adenosine deaminase (ADA) 37 IU/L (0~20 IU/L). Treatment for highly suspected TB meningitis switched to Rifater (120 mg rifampicin + 80 mg isoniazid + 250 mg pyrazinamide) five tablets per day, ethambutol (400 mg) 2.5 tablets per day and intravenous dexamethasone. Consciousness worsened to 11 points on the Glasgow coma scale (E3M4V4), and the patient had a generalized seizure on day 11 after admission. His breathing pattern became shallow and he developed clinical signs and symptoms consistent with the syndrome of inappropriate antidiuretic hormone secretion (serum sodium 118 mmol/L, urine sodium 94 mmol/L, serum osmolarity 249 osmol/kg, urine osmolarity 549 osmol/kg). Emergent brain computed tomography (CT) (Fig. 2) showed worsened hydrocephalus. External ventricular drainage (EVD) was performed immediately to relieve CSF pressure. Level of consciousness improved dramatically soon after the emergent EVD procedure.

Within three weeks of admission, muscle strength of the legs decreased to 1/5 (Medical Research Council [MRC] grade). The patient had reduced vibration, joint-position and proprioception over both lower limbs and

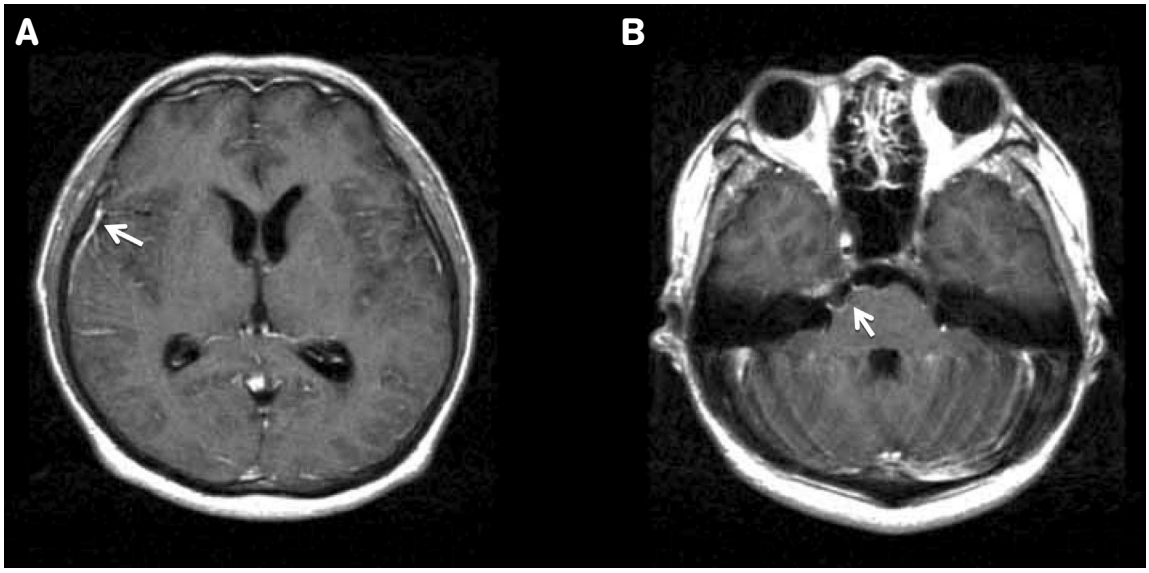


**Figure 1.** (A, B) T1-weighted magnetic resonance images of the brain after administration of gadolinium contrast show mild leptomeningeal enhancement (arrow).

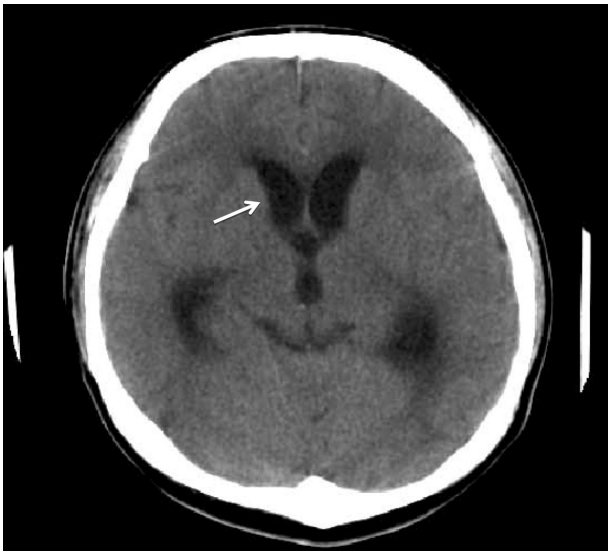

**Figure 2.** Computed tomography scan of the brain on the eleventh hospital day shows dilatation of both lateral ventricles (arrow).

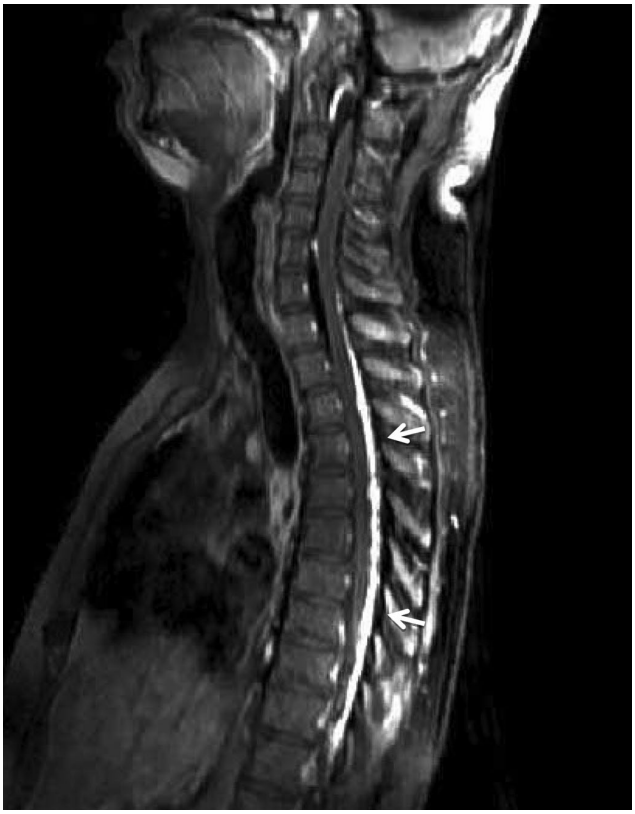

**Figure 3.** T1-weighted magnetic resonance image (sagittal view) of the cervical-thoracic spine after gadolinium enhancement shows contiguous leptomeningeal enhancement from C7-T10 levels (arrow).

on the trunk below his mid-thoracic level. A band zone dermatome of hypoesthesia on the left side at T5-T6 level was noted. Sphincter dysfunction, mainly severe urinary retention, persisted. CSF culture for Mycobacterium (M.) tuberculosis was reported as positive one month after initial examination. Diagnosis of TB meningitis with myeloradiculitis and hydrocephalus was confirmed based on culture results. Chest CT showed no abnormalities. Serial serum HIV testing was performed for three consecutive months and all results were negative. Sputum culture was negative for tuberculosis. The patient's fever subsided and his general condition gradually improved after anti-TB medications were started. Two months after treatment, muscle strength in his legs had improved to MRC grade 5/5. However, sphincter dysfunction and sensory impairment remained. Cervical-thoracic spine MRI revealed abnormally increased leptomeningeal enhancement with slight dural enhancement from the C7 to T10 levels (Fig. 3). The patient was treated with a anti-TB medication for a total of 15 months under careful physician supervision.

## DISCUSSION

TB meningitis is the most common manifestation of CNS TB, with clinical presentation often a subacute febrile illness with generalized neurological syndrome. The British Medical Research Council devised a three-stage system to assess severity of CNS TB[3]. Hydrocephalus may occur in the early or latent stage of the disease even after commencement of anti-TB drugs[4,5], and its management may influence prognosis[6-8]. Our patient developed hydrocephalus with rapid change in consciousness by the eleventh hospital day (grade III). He underwent EVD without shunt surgery. The EVD procedure alone dramatically improved consciousness and respiratory sufficiency. Subsequent brain CT showed resolution of the hydrocephalus.

In CNS TB, the spinal cord can be affected by the inflammatory process and immune reaction[9]. An immune response may proceed even after initiation of anti-TB medication. As a result of these processes, the spinal cord is virtually strangulated due to progressive

constrictive pial fibrosis (so-called spinal arachnoiditis)[10]. This is characterized on MRI by CSF loculations, obliteration of the spinal subarachnoid space, and thickened, clumped nerve roots in the lumbar region[11]. Contrast-enhanced MRI is helpful in differentiating active TB granulomatous disease from chronic fibrotic adhesion. Chronic fibrotic tissue shows poor enhancement under normal MRI[11]. Our patient started to have symptoms of radiculomyelitis early in the course of his CNS TB. Level of consciousness, fever and weakness of the legs all improved after treatment with anti-TB medication and steroid. However, he still could not walk well due to bilateral impaired proprioception, hyperreflexia and clonus. Sphincter dysfunction remained. Follow-up cervical-thoracic spine MRI three months later revealed persistent leptomeningeal enhancement at the cervical and thoracic levels. The abnormal enhancement suggests ongoing inflammatory changes. Sequelae may be induced by adhesive arachnoiditis, which causes irreversible damage of the posterior column or secondary axonal damage of peripheral nerves. Recent medical literature in the field of CNS TB research identified a phenomenon known as the "paradoxical reaction" (PR)[12]. This phenomenon refers to observation of clinical or radiological worsening of previous TB lesions or development of new lesions after at least one month of TB treatment in a patient who initially responded to anti-TB therapy[12]. This PR may explain the abnormal enhancement signals seen in our patient's follow-up cervical-spine MRI. Adjunctive corticosteroid therapy is sometimes used in the management of PR[13]. Some authors believe that steroid therapy is probably beneficial to cerebral and spinal edema and spinal block due to its anti-inflammatory effects[14,15]. However, the benefit for PR very among different studies[13]. Our patient developed PR even though he received adjunctive steroid therapy from the outset of anti-TB medication treatment.

Although the incidence of TB infection is low in developed countries, maintaining a high degree of suspicion for TB infection is vital in order to initiate therapy as soon as possible. From our case, we realized that M. tuberculosis can cause diffuse CNS infection in immunocompetent individuals. In order to reduce likelihood of complications and sequelae of CNS TB, we should make the diagnosis as quickly as possible and initiate anti-TB medication accordingly. Early EVD and shunt surgery may prevent hydrocephalus and associated irreversible neurological damage. Further advancement in early detection and diagnosis of CNS TB is valuable for physicians in clinical practice. In conclusion, both methods for detection of CNS TB and treatment protocols should be constantly re-evaluated to improve treatment outcome and reduce likelihood and severity of neurological sequelae.

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