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CENTRAL NERVOUS SYSTEM TUBERCULOSIS - CASE REPORT

Tatjana Cepreganova Cangovska[1], Ilija Zdravkov[2], Angela Aleksovska[2], Daniela Ristic-Stomnaroska[1, 2], Elena Joveva[3]
[1]Medical Faculty, University "Goce Delchev" Shtip, North Macedonia,
[2]GOB "8 Septemvri" Skopje, Macedonia, [3]Clinical Hospital-Shtip, North Macedonia

Abstract

Mycobacterium tuberculosis is the leading cause of morbidity and mortality worldwide. Tuberculosis is the second most common cause of death due to an infectious agent worldwide after COVID-19 infection. Central nervous system tuberculosis, accounts for approximately 1% of all cases of tuberculosis, and the most common clinical presentation is tuberculous meningitis. Tuberculosis of the central nervous system is a devastating disease that, even under appropriate antituberculin therapy, leads to a high mortality rate.

In this paper, we present a case report of a patient in whom the disease begins slowly, progressively, and chronically, with the initial clinical manifestation of dementia syndrome. Usually, tuberculous meningitis presents with a subacute progressive febrile illness. In 2 to 3 weeks defines meningitis phase with headache, meningismus, vomiting, mild confusion. Rare is the atypical presentation of CNS TB, as in our case. During the hospitalization, brain magnetic resonance was performed with suspicious findings for neuro infection. Due to the suspected finding of magnetic resonance imaging, a lumbar puncture was performed and the cerebrospinal fluid was sent for analysis for specific causes, including Mycobacterium tuberculosis. Correct diagnosis and therapy in these patients are a challenge for neurologists.

Keywords: central nervous system tuberculosis, mycobacterium tuberculosis, neuro infection.

Introduction

Mycobacterium tuberculosis (MBT) is the leading cause of morbidity and mortality worldwide. The global burden of tuberculosis is huge. It is estimated that two billion people (33% of the world's population) are infected with tuberculosis. Tuberculosis is the second most common cause of death worldwide after COVID-19 infection, due to an infectious agent. Central nervous system tuberculosis (CNS TB) accounts for approximately 1% of all cases of tuberculosis, and the most common clinical presentation is tuberculous meningitis with a prevalence of 3 to 14% of cases.

Tuberculosis of the central nervous system is a devastating disease that, even under appropriate antituberculosis therapy, leads to high mortality rates of up to 42%.

Clinical CNS TB infection is seen to comprise three categories of illness: subacute or chronic meningitis, intracranial tuberculoma, and spinal tuberculous arachnoiditis. Of necessity, this requires some knowledge of the causes and clinical features of granulomatosis meningitis, the pathology that subserves the neurologic manifestations of disease and expected radiographic and laboratory (cerebrospinal fluid) findings [1].

Pathogenesis

Clinical neurologic illness begins with the hematogenous dissemination of Mycobacterium tuberculosis that follows primary pulmonary infection or late reactivation MBT elsewhere in the body. Sparse numbers of bacilli are scattered through the substance of the brain, meninges, and adjacent tissues, leading to the formation of multiple small granulomatous foci of various sizes and degrees of encapsulation (tubercles).

The continued proliferation and coalescence of tubercles result in larger caseous foci ("Rich focus"). This conceptual understanding of the pathogenesis of tuberculous meningitis is derived from the observations of Rich and McCordock, who performed meticulous autopsy examinations of tuberculous meningitis patients [2].

Clinical Presentation

Common symptoms and signs

The usual patient with TBM presents with subacute, progressive febrile illness that passes through three discernible phases. Illness begins with a prodrome of malaise, lassitude, low-grade fever and intermittent headache. Sometimes patients present a vague discomfort in the neck or back and subtle personality change. In 2 to 3 weeks a more well-defined meningitis phase protracted headache, meningismus, vomiting, mild confusion and various degrees of CN palsy and long-tract signs [3]. At this stage the place the illness may accelerate rapidly to the paralytic phase: delirium followed by stupor and coma, seizures, multiple CN deficits, hemiparesis and hemiplegia. In the untreated case, death commonly occurs within 5 to 8 weeks of the onset of illness. In children, headache is less common, while irritability, restlessness, anorexia and protracted vomiting are prominent symptoms especially in the very young [4]. Seizures are common in children in the early stage of illness [4, 5].

Atypical presentations

A small subset of patients presents without the characteristic prodrome and subacute progression described above. In the occasional adult, TBM takes the form of a slowly progressive dementia over many months, marked by personality change, social withdrawal and memory deficits. An “encephalitic” syndrome has been described to occur in children and occasionally adults manifesting as stupor, coma and convulsions with neither meningitis signs nor significant CSF abnormalities [6]. At times focal neurologic deficits (CN palsies, hemiparesis and seizures) or symptoms of hydrocephalus (headache, papilloma, diplopia, and visual disturbance) precede the signs of meningitis.

Clinical stages and prognosis

For purpose of prognosis and therapy, it is useful to categorize patients into clinical stages according to the degree of illness at presentation.

Stage 1 comprises patients who are conscious and rational with or without meningismus but having no focal neurologic signs or evident hydrocephalus;

Stage 2 patients exhibit lethargy and confusion and may have mild focal neurologic signs such as CN palsy and hemiparesis; and

Stage 3 patients exhibit signs of advanced disease such as stupor, coma, seizures multiple CN palsies and dense hemiplegia [2]. The prognosis for treated TBM is greatly influenced by the clinical stage at which treatment is initiated.

Case report

In this paper we report a case of a patient 52-years-old male. The disease begins slowly, progressively and chronically, with the initial clinical manifestation of dementia syndrome, 4 months before hospital admission.

At admission the patient has a somatic status in the rank of cachexia, with signs of diffuse cerebral suffering, facies hypomimia et oleosa are present, associated with severe bulbar syndrome. Increased spasticity-type tone is noted on the skeletal muscles. The patient has an infectious syndrome and confirmed pulmonary consolidation.

During the hospitalization, routine blood test was performed and mild anemia and leukocytosis were determined. Magnetic resonance imaging of the brain was made with suspicious finding is obtained for neuroinfection. Neuroradiologist could not assess what type of neuroinfection it was. Possible differential diagnosis could be- fungal meningitis, neuroborreliosis, neurobrucellosis, neurosyphilis, neurocysticercosis, CNS toxoplasmosis, neoplastic meningitis.

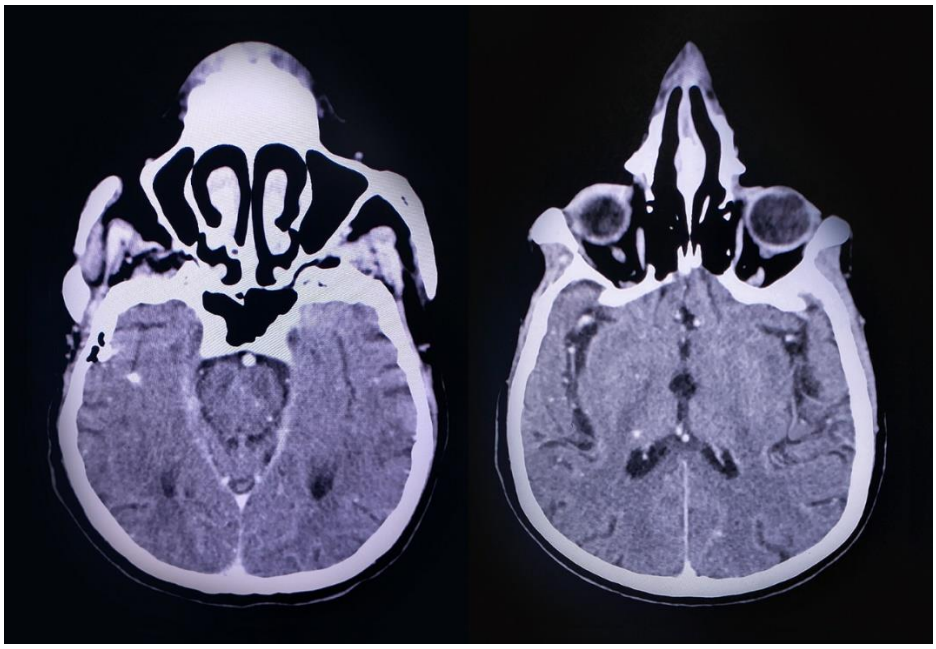

Figure 1. Computed tomography scan, multiple calcifications are registered.

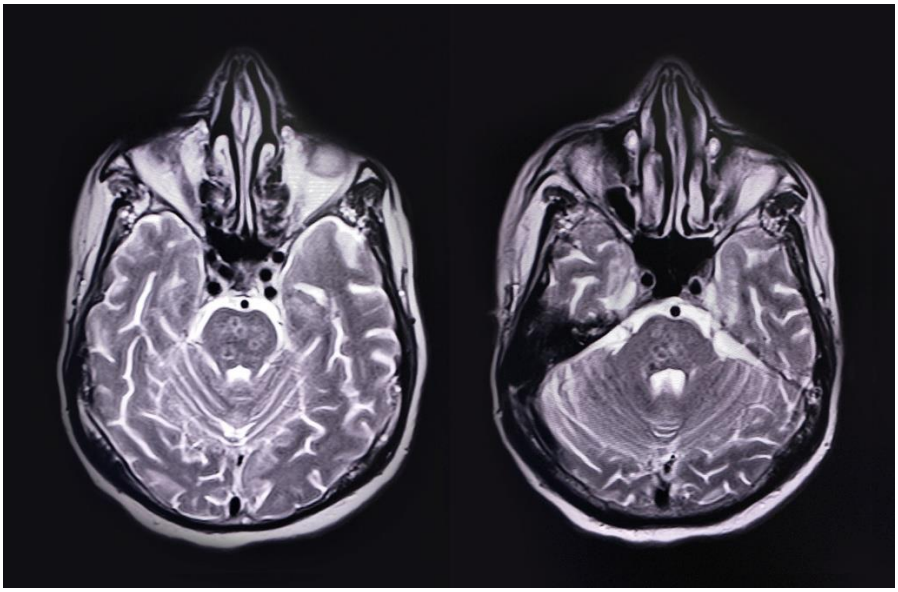

Figure 2. Magnetic resonance imaging (transverse T2-weighted MRI),with contrast revealed multifocal ring-enhancing lesions in medulla oblongata, pons and cerebellum.

Due to the suspected findings of magnetic resonance imaging, a lumbar puncture was performed. The cerebrospinal fluid was sent for analysis for specific causes, including Mycobacterium tuberculosis. After obtaining the result of the GENEEXPERT MTB / RIF assay method, the presence of the genome of Mycobacterium tuberculosis was proved. After this result, anti-tuberculosis therapy (isoniazid, rifampin, pyrazinamide, ethambutol) was immediately included in the patient's therapy.

Discussion

The clinical and radiologic manifestations of CNS tuberculosis may mimic other infectious and noninfectious neurological conditions. The application of computed tomography (CT) and magnetic resonance imaging (MRI) has greatly facilitated the assessment and management of patients with CNS TB [7].

MRI is superior to CT for defining lesions of the brain stem, midbrain and basal ganglia in patients of all ages [8, 9], as exemplified in our patient. The key to the diagnosis in most instances rests with the proper interpretation of the spinal fluid cellular characteristics and chemistries (the CSF formula) combined with the demonstration of mycobacteria in the CSF by stained smear or culture.

The demonstration of M. Tuberculosis by stained smear and culture establishes the specific diagnosis. Cultures are positive in about 75% of cases but require 3 to 6 weeks for detectible growth.

The prognosis is good when treatment is begun before the development of focal neurologic signs and altered state of consciousness.

Treatment is most effective when started in the early stages of disease, and should be initiated promptly on the basis of strong clinical suspicion without waiting for laboratory confirmation. The initial 4 drug regimen (isoniazid, rifampin, pyrazinamide, ethambutol) covers the possibility of infection with a resistant strain, maximizes antimicrobial impact, and reduces the likelihood of emerging resistance on therapy. Adjunctive corticosteroid therapy has been shown to reduce morbidity and mortality in all but late-stage disease [9, 10].

Conclusion

Central nervous system tuberculosis is a significant diagnostic challenge for neurologists. It should be emphasized that dementia as a syndrome, especially in patients with early onset, requires additional examination to detect etiology. Neuroimaging methods are not superior in diagnostic dilemma in patients with nonspecific signs of central nervous system tuberculosis. The atypical presentation of symptoms in CNS-TB, leads you in another direction of thinking. This wastes time for correct diagnosis and timely initiation of AB-therapy.

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