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CASE REPORT

Tuberculous meningitis in a former inmate, a case report

Meningitis tuberculosa en un exrecluso, reporte de un caso

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ABSTRACT

Introduction: tuberculous meningitis is the most severe form of extrapulmonary tuberculosis, with mortality rates approaching 50 %. Neurological damage is primarily driven by dysregulated host neuroinflammatory responses following the invasion of the central nervous system by Mycobacterium tuberculosis.
Case report: a case is presented of a 45-year-old male, a former inmate, with a one-month clinical course characterized by constitutional symptoms, headache, and progressive neurological impairment. Physical examination revealed meningeal syndrome and sixth cranial nerve palsy. Cerebrospinal fluid analysis showed lymphocytic pleocytosis (410 cells/mm³, 88 % lymphocytes), hyperproteinorrachia (260 mg/dL), and hypoglycorrachia (18 mg/dL). The diagnosis was confirmed via GeneXpert and neuroimaging, which evidenced basal leptomeningeal enhancement and hydrocephalus. Treatment was initiated with the HRZE regimen and adjuvant dexamethasone, achieving significant clinical improvement after 35 days of hospitalization. Discharge was coordinated to complete the total planned duration of antituberculous treatment through the Directly Observed Treatment, Short-course (DOTS/TAES) program. At the time of discharge, the patient was oriented and able to walk with minimal assistance, with the sixth cranial nerve palsy showing signs of progressive improvement.
Conclusions: early diagnosis based on epidemiological and clinical suspicion is vital. Timely management with antitubercular drugs and corticosteroids significantly reduces mortality and permanent sequelae.

Keywords: Tuberculosis Meningeal; Mycobacterium tuberculosis; Cerebrospinal Fluid.

RESUMEN

Introducción: la meningitis tuberculosa es la forma más grave de tuberculosis extrapulmonar, con una mortalidad cercana al 50 %. El daño neurológico es impulsado principalmente por respuestas neuroinflamatorias desreguladas del huésped ante la invasión del sistema nervioso central por Mycobacterium tuberculosis.
Reporte de caso: se presenta el caso de un paciente masculino de 45 años, exrecluso, con un cuadro clínico de un mes de evolución caracterizado por síntomas constitucionales, cefalea y deterioro neurológico progresivo. El examen físico reveló síndrome meníngeo y paresia del sexto par craneal. El estudio del líquido cefalorraquídeo mostró pleocitosis linfocítica (410 células/mm³, 88 % de linfocitos), hiperproteinorraquia (260 mg/dL) e hipoglucorraquia (18 mg/dL). El diagnóstico se confirmó mediante GeneXpert y neuroimagen, que evidenciaron realce leptomeníngeo basal e hidrocefalia. Se inició tratamiento con el esquema HRZE y dexametasona adyuvante, logrando una mejoría clínica significativa tras 35 días de hospitalización. Se coordinó el alta para completar la duración total planificada del tratamiento antituberculoso a través del programa de Tratamiento Acortado Estrictamente Supervisado. Al momento del alta, el paciente se encontraba orientado y era capaz de deambular con mínima asistencia, con la paresia del sexto par craneal mostrando signos de mejoría progresiva.
Conclusiones: el diagnóstico precoz basado en la sospecha epidemiológica y clínica es vital. El manejo oportuno con antifímicos y corticosteroides reduce significativamente la mortalidad y las secuelas permanentes.

Palabras clave: Meningitis Tuberculosa; Mycobacterium Tuberculosis; Líquido Cefalorraquídeo.

INTRODUCTION

Tuberculous meningitis represents the most devastating form of extrapulmonary tuberculosis, with a mortality rate reaching 50 % of affected patients, even with the best available treatment. This disease has been recognized as a distinct clinical entity for nearly two centuries, although its pathophysiological understanding and therapeutic management continue to pose significant challenges for contemporary medicine.[1,2]

The history of tuberculous meningitis is marked by controversies and progressive discoveries spanning from the 18th century to the present day. In 1734, the Scottish physician John Paisley first described "dropsy of the cerebral ventricles," followed by Robert Whytt in 1738, who documented clinical manifestations of what would later be recognized as tuberculous meningitis. However, it was not until 1836 that The Lancet published a description of six children with fatal "acute hydrocephalus," in whose autopsies "an inflammation of the meninges, with a deposit of tubercular matter in the form of granulations or caseous material" was found.[3]

This publication generated considerable controversy by proposing "tuberculous meningitis" as a new diagnosis, combining the growing number of diseases characterized by the presence of "tubercles." The unitary theory of tuberculosis was not widely accepted until 1882, when Robert Koch first stained and cultured Mycobacterium tuberculosis, demonstrating that it was the bacterium transmitted in tuberculosis. This fundamental discovery transformed the understanding of the disease, although debates persisted as to whether tuberculous meningitis resulted from direct hematogenous invasion of the meninges by the bacilli or from inoculation from contiguous lesions resulting from prior bacillemia.[4]

The global prevalence of tuberculous meningitis is estimated to be 2 per 100,000 inhabitants, with the mortality rate reaching 42 % among hospitalized patients.[5] The geographic distribution of the disease is markedly heterogeneous, with the highest burden in low- and middle-income countries. More than three-quarters of cases are reported in Southeast Asia, the Western Pacific, and Africa.[6] Tuberculous meningitis has become the leading cause of bacterial brain infections in regions with a high burden of tuberculosis, especially following the success of vaccines against pyogenic bacterial meningitis.[2]

The populations most vulnerable to tuberculous meningitis include young children and immunocompromised individuals, particularly those with HIV infection.[1,2,4] The incidence is directly related to the prevalence of pulmonary tuberculosis in the population.[5] In HIV-positive patients, mortality reaches 53 %, whereas it is 22 % in HIV-negative patients. Children aged 2 to 4 years have the highest incidence of the disease.[7]

Although the prison setting is a critical reservoir for tuberculosis due to overcrowding and poor ventilation, there is a gap in the clinical literature addressing the management of tuberculous meningitis during the transition of care for recently released individuals. The relevance of reporting this case lies in illustrating the diagnostic challenge posed by this vulnerable population, where early epidemiological suspicion and social health coordination are essential to prevent fatal outcomes. Therefore, this article aims to describe a case of tuberculous meningitis in a formerly incarcerated patient, analyzing the clinical and therapeutic aspects and the importance of supervised follow-up to ensure adherence in this specific social context.

CASE REPORT

A 45-year-old male patient who was unemployed and a temporary resident of a social welfare shelter. A key epidemiological factor in his history is that he is a former inmate who was released exactly four months ago after serving a five-year sentence in a prison known to be severely overcrowded. During his time in prison, he lived in a shared cell with individuals who had active chronic coughs, and no contact tracing was conducted at the time.

The patient reported having had a chronic cough with occasional sputum production during his final year of incarceration; however, he was never evaluated or screened for tuberculosis (TB) by the prison medical service. His BCG vaccination status was confirmed by the presence of a characteristic scar on his right arm and previous medical records.

The current illness began insidiously three months ago (one month after his release), with profound asthenia, profuse night sweats, anorexia, and weight loss of eight kilograms. The condition progressed to include a disabling holocranial headache and, in the week prior to admission, marked neurological deterioration with disorientation, lethargy, and diplopia.

Physical examination

On admission, the patient presented with pallor, evident muscle wasting, and poor general condition. He was somnolent but aroused by loud verbal stimuli, disoriented in time and place, with a Glasgow Coma Scale score of 13/15. His vital signs included a temperature of 38,5 °C, a heart rate of 102 beats per minute, a respiratory rate of 20 breaths per minute, and a blood pressure of 105/65 mmHg.

A detailed neurological examination revealed clear meningeal syndrome, manifested by severe nuchal rigidity and positive Kernig's and Brudzinski's signs. Additionally, sixth cranial nerve (abducens nerve) palsy was documented in the left eye, accounting for the reported diplopia. Funduscopic examination revealed no papilledema or choroidal tubercles. The remainder of the physical examination, including cardiopulmonary auscultation, showed no acute findings of consolidation, although a slight decrease in vesicular breath sounds was noted at the pulmonary apices.

Diagnostic evaluation

Initial laboratory studies revealed a complete blood count with a leukocytosis of 12 200 cells/µL (78 % neutrophil predominance), mild normocytic normochromic anemia (hemoglobin concentration of 11,2 g/dL), and markedly elevated inflammatory markers: an erythrocyte sedimentation rate (ESR) of 78 mm/h and a C-reactive protein (CRP) concentration of 55 mg/L. A screening panel for HIV, VDRL, and hepatitis B and C viruses was negative.

A posterior-anterior chest X-ray revealed the presence of fibrous tracts and apical retraction in the right hemithorax, suggesting residual or chronic pulmonary granulomatous disease, which was consistent with his history of coughing in prison. Given the urgency of ruling out focal intracranial hypertension, an uncontrasted computed tomography (CT) scan of the skull was performed, which revealed mild dilation of the lateral ventricles and third ventricle, without a midline shift.

A lumbar puncture was performed. The cerebrospinal fluid (CSF) had a slightly xanthochromic appearance. Cytochemical analysis confirmed the classic pattern of tuberculous meningitis: pleocytosis of 410 cells/mm³ (with 88 % lymphocytes), severe hyperproteinorrachia of 260 mg/dL, and profound hypoglucorrachia of 18 mg/dL (simultaneous serum glucose concentration of 92 mg/dL). Ziehl–Neelsen staining of CSF was negative; however, the adenosine deaminase (ADA) concentration was reported to be 22 U/L (elevated). A definitive diagnosis was obtained through molecular biology testing (GeneXpert MTB/RIF) of the CSF, performed at the Pedro Kouri Institute of Tropical Medicine in Havana, which detected *Mycobacterium tuberculosis* DNA without rifampicin resistance.

Subsequent contrast-enhanced brain magnetic resonance imaging (MRI) confirmed the presence of dense leptomeningeal enhancement at the level of the basal cisterns, the optic chiasm, and the brainstem, accompanied by moderate communicating hydrocephalus.

Treatment and Clinical Course

On the basis of the high index of epidemiological and clinical suspicion, empirical antituberculosis treatment was initiated immediately after the lumbar puncture, which was confirmed and continued upon receipt of the molecular report. The intensive phase of first-line treatment with isoniazid, rifampicin, pyrazinamide, and ethambutol (HRZE regimen) was initiated. As a crucial measure to mitigate the inflammatory reaction in the subarachnoid space and prevent neurological complications such as arachnoiditis and cerebral infarction, intravenous dexamethasone was administered at a dosage of 0,4 mg/kg/day, with a tapering schedule planned for eight weeks.

The course of the disease during the first fourteen days was sluggish. The patient experienced occasional fever spikes, and his level of consciousness fluctuated; however, he did not have seizures or require a ventricular shunt, as his level of hydrocephalus remained stable on follow-up neuroimaging. Starting in the third week, a positive turnaround was observed: the fever subsided completely, the patient achieved a sustained state of alertness, the patient cooperated with questioning, and the sixth cranial nerve palsy showed signs of progressive improvement.

Liver function tests were strictly monitored twice weekly, with no elevation in transaminases indicating hepatotoxicity from the HRZE regimen. After 35 days of hospitalization and with marked neurological improvement (ability to ambulate with minimal assistance and oriented in all three domains), discharge was scheduled. The gradual tapering of dexamethasone was strictly initiated during the fourth week of treatment after neurological stability was observed, with a gradual reduction regimen completed over a total of eight weeks.

Given his socially vulnerable situation and status as a former inmate, discharge was closely coordinated with the Social Work Department and the local health secretariat to ensure the patient's inclusion in the Strictly Supervised Shortened Treatment (TAES) program at his assigned health center, thereby ensuring treatment adherence and outpatient neurological monitoring.

At the end of the full one-year treatment, the patient was discharged from epidemiological monitoring. As a residual neurological sequela, minimal paresis of the sixth cranial nerve in the left eye persists, which does not limit his daily activities, with intact cognitive functions and the ability to walk independently.

This case report was conducted in accordance with the ethical principles established in the Declaration of Helsinki for medical research involving human subjects. The patient provided written informed consent, authorizing the publication of his clinical information and laboratory results for academic and scientific purposes, with the goal of protecting his identity and ensuring the confidentiality of his personal data. Furthermore, the protocol for this study was reviewed and approved by the Ethics Committee of the Faculty of Medicine at the University of Medical Sciences of Villa Clara.

DISCUSSION

This case exemplifies the classic presentation of tuberculous meningitis in a patient with multiple epidemiological and clinical risk factors. A history of prolonged incarceration in an overcrowded prison constitutes a critical risk factor for acquiring tuberculosis, as prisons function as reservoirs and amplifiers of Mycobacterium tuberculosis transmission because of overcrowding, poor ventilation and air, and suboptimal screening practices.(Studies have demonstrated that length of incarceration is an independent risk factor for tuberculosis acquisition, with incidence rates of latent infection reaching 2,402 cases per 100 000 person-months in newly incarcerated individuals.[9]

The patient's clinical presentation is characteristic of tuberculous meningitis. The insidious course over one month with constitutional symptoms (asthenia, night sweats, weight loss) followed by progressive neurological manifestations (headache, deterioration of mental status, sixth cranial nerve palsy) corresponds to the typical pattern described in the literature. Abducens nerve palsy is a well-documented neurological complication of tuberculous meningitis resulting from inflammatory involvement of the basal cisterns. The history of chronic, uninvestigated cough during his incarceration suggests prior or concomitant pulmonary tuberculosis, which is present in approximately 50 % of cases of tuberculous meningitis.[4,5]

The cytochemical profile of the cerebrospinal fluid confirmed the classic pattern of tuberculous meningitis: lymphocytic pleocytosis (410 cells/mm³ with 88 % lymphocytes), severe hyperproteinorrachia (260 mg/dL), and profound hypoglucorrachia (18 mg/dL). Although the Ziehl–Neelsen staining was negative, this does not rule out a diagnosis, as the sensitivity of smear microscopy

in CSF is less than 30 %. The elevated adenosine deaminase level (22 U/L) provided additional evidence, although it is not specific for tuberculosis.[4]

Diagnostic confirmation using GeneXpert MTB/RIF represents the current standard for the rapid diagnosis of tuberculous meningitis. Recent meta-analyses report a sensitivity of 85 % and specificity of 98 % for definitive tuberculosis meningitis.[10] However, recognizing that a negative GeneXpert result does not rule out tuberculosis meningitis is crucial, and many patients (30–50 %) should initiate empirical treatment on the basis solely of clinical suspicion. The GeneXpert Ultra version, with a lower detection limit (16 CFU/mL versus 131 CFU/mL), shows slightly higher sensitivity, especially when large volumes of centrifuged CSF are processed.[2,11]

Discrepancy between Ziehl–Neelsen staining (negative) and GeneXpert MTB/RIF (positive) is a common phenomenon because of the paucibacillary nature of MTB (). It is estimated that between 5000 and 10 000 bacilli/mL are required for a positive smear, whereas GeneXpert has a significantly lower detection limit (approx. 131 CFU/mL). In this patient, the low bacterial load in the CSF, typical of meningeal dissemination, prevented direct visualization of the bacillus but not the detection of its DNA.[12]

Neuroimaging findings are highly suggestive of tuberculous meningitis. Dense leptomeningeal enhancement in the basal cisterns, optic chiasm, and brainstem, along with communicating hydrocephalus, constitute pathognomonic radiological features. The presence of hydrocephalus in combination with meningeal enhancement is strongly suggestive of tuberculous meningitis and occurs in 50–90 % of patients. Apical fibrous tracts on chest radiography support the diagnosis of chronic or residual pulmonary granulomatous disease.[5]

The therapeutic management implemented was appropriate and evidence-based. HRZE regimens (isoniazid, rifampicin, pyrazinamide, and ethambutol) constitute the first-line treatment recommended by international guidelines. The administration of the adjuvant dexamethasone is strongly recommended in patients with tuberculous meningitis, as it reduces mortality in patients without HIV coinfection. Randomized clinical trials have demonstrated a survival benefit with corticosteroids, especially in children and adults without HIV. The dosage used (0,4 mg/kg/day) with gradual tapering over 8 weeks is in line with current recommendations.[13]

Although some guidelines suggest extending treatment to 12 weeks in cases of severe arachnoiditis, the 8-week regimen aligns with the evidence from the seminal clinical trial by Thwaites et al., which demonstrated a significant reduction in mortality in HIV-negative patients with this treatment duration. The use of the adjuvant aspirin, which has been proposed in some studies to reduce the incidence of cerebral infarcts due to vasculitis, was not considered; however, its use is not yet a universal standard of care because of inconsistent results.[14]

Neurological complications of tuberculous meningitis are common and affect prognosis. Approximately 88,9 % of patients develop complications, including hydrocephalus (40 %), cerebral infarcts (33 %), hyponatremia (49 %), and epileptic seizures (18 %).(Cerebral infarcts, resulting from vasculitis of the basal perforating arteries, occur in more than 65 % of cases and are predictors of poor prognosis. The overall mortality rate for tuberculous meningitis remains high (20–50 %), even with the best available treatment, and early diagnosis and treatment before the onset of coma substantially reduces mortality and disability.[4]

The patient's initial sluggish course during the first two weeks is common and does not necessarily indicate treatment failure. Paradoxical reactions occur in approximately 20 % of patients (>30 % in people with HIV), typically between days 20–60 of treatment, and may require the intensification of corticosteroid therapy. The clinical improvement observed starting in the third week, with resolution of fever and progressive neurological recovery, is encouraging.[4]

To assess the prognosis, the Thwaites Prognosis Index was calculated retrospectively. The patient scored 0 (age 45 years: +2; blood leukocytes 12,200: 0; duration of illness: -2 (>6 days); CSF cells 410: -1; % lymphocytes 88 %: -1). A score of ≤ 4 is highly suggestive of tuberculous meningitis, and within this range, the patient is classified as having an intermediate stage of severity (British Medical Research Council Stage II), which is consistent with his recovery but with the presence of minor sequelae.[16]

Coordinated discharge with enrollment in the Strictly Supervised Short-Course Treatment (SSCT) program is essential to ensure treatment adherence and prevent the development of resistance. The population of former prisoners faces multiple barriers to completing treatment, including housing instability, unemployment, and social stigma. Outpatient neurological follow-up is essential, as long-term neurological sequelae are common and include motor deficits, intellectual disability, epilepsy, behavioral disorders, and sensory deficits.[17]

Barriers to care among former prisoners are critical. Prisons act as "time bombs" for tuberculosis due to overcrowding. Upon release, a lack of stable housing, undiagnosed mental health disorders, and potential substance use create an environment of "social disaffiliation" that compromises treatment adherence. As noted by Kamarulzaman et al., therapeutic success in this population depends not only on the drug but also on a care transition that includes comprehensive social support to prevent the patient from falling through the cracks in outpatient follow-up.[18]

This case highlights the importance of a high index of clinical suspicion in at-risk populations, the early initiation of empirical treatment when suspicion is high (without waiting for microbiological confirmation), the appropriate use of adjuvant corticosteroids, and the need for structured follow-up strategies to ensure adherence and detect late complications.

CONCLUSIONS

The timely diagnosis of tuberculous meningitis requires a high index of clinical suspicion, especially in socially vulnerable populations with a history of incarceration. Early initiation of empirical antituberculosis therapy and the adjunctive use of corticosteroids are critical for halting neurological damage and reducing mortality.

On the basis of the lessons learned from this case, it is recommended that prison health systems implement systematic screening protocols for active and latent tuberculosis prior to the release of inmates, ensuring a "bridge of care" or coordinated transition with

public health services to prevent loss to follow-up during the postincarceration period.

Similarly, it is imperative to recognize that clinical recovery at discharge does not mark the end of the disease. Survivors of tuberculous meningitis require long-term neurological and neuropsychological follow-up to monitor and rehabilitate persistent sequelae, such as cranial nerve palsies, cognitive deficits, or behavioral disorders, thereby ensuring optimal social and functional reintegration. Therapeutic success among former inmates depends on a multidisciplinary approach that combines pharmacological rigor with robust social and occupational support.

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CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest.

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