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CASE REPORT/OLGU SUNUMU

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Multiple Intracranial Tuberculomas and Hydrocephalus in an Immunocompetent Patient: A Case Report and Literature Review

İmmünkompetan Bir Hastada Multipl İntrakraniyal Tüberkülom ve Hidrosefali: Bir Olgu Sunumu ve Literatürün İncelenmesi

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ABSTRACT

Intracranial tuberculomas are a rare and diagnostically challenging form of central nervous system tuberculosis (TB). In this report, we present a case of multiple intracranial tuberculomas in an immunocompetent young adult, accompanied by pulmonary TB and TB lymphadenitis. A 21-year-old male patient was admitted to the hospital with facial numbness and recurrent episodes of speech arrest lasting a few seconds. Radiological imaging revealed multiple intracranial nodular lesions, initially raising suspicion for metastatic disease. However, growth of Mycobacterium tuberculosis in the bronchoalveolar lavage specimen and the finding of necrotizing granulomatous lymphadenitis in the lymph node biopsy led to a presumptive diagnosis of tuberculosis, and a two-month course of quadruple anti-tuberculosis therapy was initiated. Despite this treatment, a ventriculoperitoneal shunt was placed due to hydrocephalus that developed as a result of the mass effect of the cerebellar lesion. The diagnosis was confirmed by the presence of caseating granulomas in the intraoperatively obtained lesion biopsy, detection of Mycobacterium tuberculosis DNA, and positive acid-fast bacilli staining. Following shunt-related complications and progression of the lesion, subtotal resection of the cerebellar mass was performed along with placement of a ventriculoatrial shunt. On follow-up, the patient's clinical findings showed marked improvement. This case highlights the diagnostic and therapeutic challenges of multiple intracranial tuberculomas, even in immunocompetent individuals, and underscores the importance of a multidisciplinary approach in their management.

Key Words: *Intracranial tuberculoma; Hydrocephalus; Tuberculosis; Ventriculoperitoneal shunt*

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ÖZ

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İntrakraniyal tüberkülomlar, merkezi sinir sistemi tüberkülozu (TBC)'nun nadir görülen ve tanısı güç bir formudur. Bu yazıda, immünkompetan genç bir erişkinde pulmoner TBC ve TBC lenfadenitin eşlik ettiği multipl intrakraniyal tüberkülom olgusu sunulmaktadır. Yirmi bir yaşındaki erkek hasta, yüzde uyuşma ve konuşma bozukluğu şikayetleriyle hastanemize başvurmuştur. Yapılan radyolojik incelemelerde intrakraniyal multipl nodüler lezyonlar saptanmış, başlangıçta bu lezyonların metastatik olabileceği düşünülmüştür. Ancak bronkoalveolar lavaj örneğinde Mycobacterium tuberculosis üremesi ve lenf nodu biyopsisinde nekrotizan granülomatöz lenfadenit izlenmesi üzerine hastaya TBC ön tanısı ile dörtlü anti-TBC tedavisi ve kortikosteroid tedavisi başlanmıştır. Ancak mevcut tedaviye rağmen serebellar lezyonun bası etkisine bağlı olarak gelişen hidrosefali nedeniyle ventriküloperitoneal şant uygulanmıştır. Şant komplikasyonları ve lezyonun progresyonu üzerine serebellar kitleye subtotal rezeksiyon yapılmış ve ardından ventriküloatriyal şant cerrahisi gerçekleştirilmiştir. İntraoperatif olarak alınan lezyon biyopsisinde kazeifiye granülomların saptanması, TBC DNA'nın gösterilmesi ve ARB pozitifliği ile tanı doğrulanmıştır Hastanın takiplerinde klinik bulgularında iyileşme saptanmıştır. Bu olgu, multipl intrakraniyal tüberkülomların immünkompetan bireylerde de görülebileceğini vurgulamasının yanında tanı ve tedavisinde karşılaşılan güçlükleri ve multidisipliner yaklaşımın önemini vurgulamaktadır.

Anahtar Kelimeler: *İntrakraniyal tüberkülom; Hidrosefali; Tüberkülozis; Ventriküloperitoneal şant*

INTRODUCTION

Tuberculosis (TB) remains a major global health concern, affecting over 10 million people annually[1]. Central nervous system (CNS) involvement, which represents about 5-10% of extrapulmonary TB and 1% of all TB cases, is associated with high morbidity and mortality[2,3]. While CNS-TB usually presents as tuberculous meningoencephalitis, intracranial tuberculomas are a much rarer manifestation, often seen in high-risk individuals such as those with immunosuppression or malnutrition[4].

In recent years, studies from endemic regions have documented an increasing number of extrapulmonary TB cases presenting with atypical neurological manifestations, highlighting the evolving spectrum of CNS involvement[5]. This trend underscores that CNS TB remains a significant diagnostic challenge, even among immunocompetent patients, due to its nonspecific clinical and radiological features[6]. Accordingly, a recent review by Rajshekhar et al. emphasizes that early radiological suspicion combined with timely empirical therapy is crucial for improving patient outcomes[7].

Despite advances in diagnostic tools and treatment, CNS TB-particularly intracranial tuberculomas-remains a challenging condition to diagnose and manage due to its rarity and variable presentation. In this context, we present a case of an immunocompetent patient with multiple intracranial tuberculomas and hydrocephalus, aiming to contribute to the existing literature by highlighting diagnostic challenges and therapeutic considerations in such rare presentations.

CASE REPORT

A 20-year-old male patient presented with facial numbness and brief, recurrent episodes of speech arrest lasting a few seconds. These neurological symptoms had persisted for approximately two months, during which he also experienced systemic manifestations, including unintentional weight loss and night sweats. He lived with both parents. There was no family history of chronic illnesses or contact with any known cases of TB. Vital signs were as follows: temperature

36 °C, pulse 92 bpm, blood pressure 110/70 mmHg, and respiratory rate 21/min. On physical examination, he was oriented and cooperative. Neurological examination revealed no focal deficits. Other system examinations were normal. Laboratory investigations showed a leukocyte count of 5.85 x 10^9/L, hemoglobin of 12.5 g/dL, platelet count of 199 x 10^9/L, creatinine of 0.92 mg/dL (normal range= 0.7–1.2 mg/dL), alanine aminotransferase of 11 U/L, aspartate aminotransferase of 14 U/L, C-reactive protein of 22 mg/dL (normal range= 0–5 mg/dL), eritrocyte sedimentation rate= 28/h. Contrast-enhanced cranial magnetic resonance imaging (MRI) demonstrated multiple nodular lesions with heterogeneous signal intensities located in the right cerebral hemisphere (frontoparietal and temporal regions), left cerebral hemisphere (frontal and temporal lobes), and right cerebellar hemisphere. The largest lesion, measuring approximately 22 x 18 mm, was identified in the right cerebellar hemisphere. Most lesions exhibited peripheral enhancement, while some showed homogeneous solid contrast uptake in post-contrast sequences. Surrounding the lesions, there was widespread perilesional vasogenic edema within the cerebral parenchyma, suggesting aggressive pathology (Figure 1).

Given these findings, metastatic disease was initially suspected, prompting thoracic computed tomography (CT) to investigate a possible primary neoplastic focus. CT imaging revealed multiple spiculated nodular consolidations with fibrotic extensions to the pleura, located primarily in the subpleural region of the left upper lobe, the largest measuring 124 x 14 mm (Figure 2). Based on these radiologic findings, the differential diagnosis included primary pulmonary malignancy or metastatic disease, and histopathological confirmation was recommended.

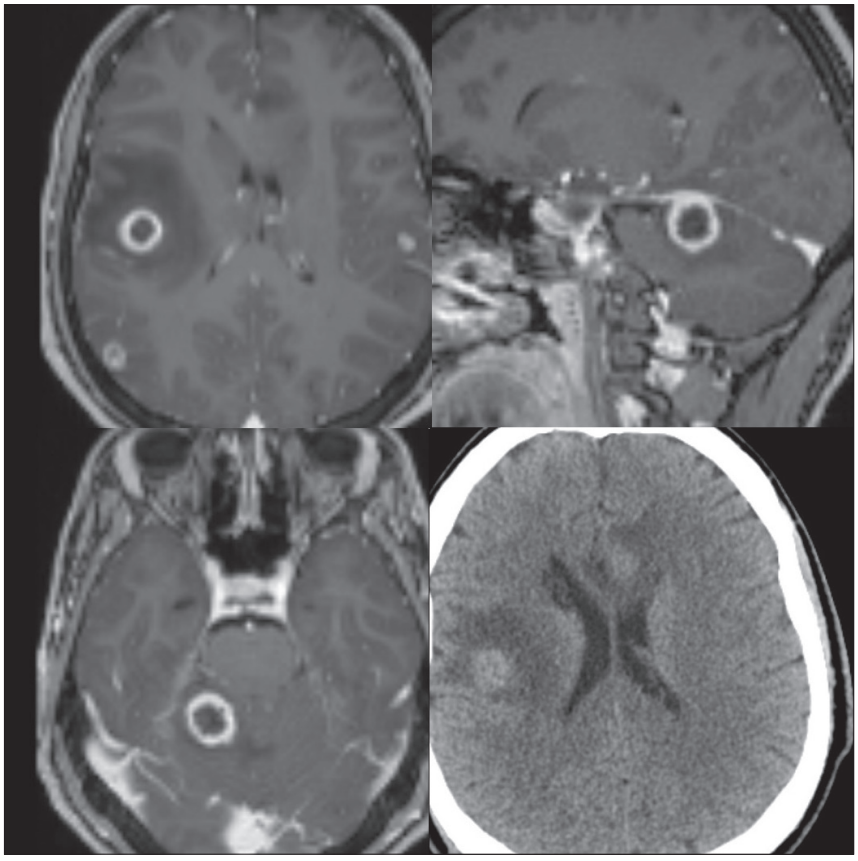

Figure 1. T1 contrast-enhanced MRI axial view shows multiple lesions in the right parietal lobe and tuberculoma lesions in the left parietal lobe, left frontal lobe, and right cerebellum tuberculoma lesion.

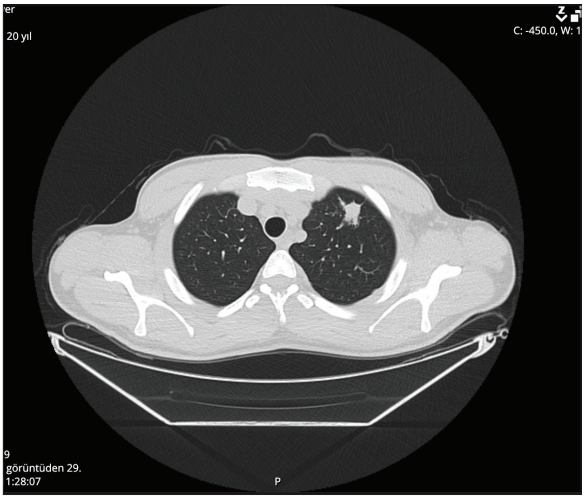

Figure 2. Thorax CT shows areas of minimal ground-glass opacity and reticular density increases about three nodular consolidated lesions with spiculated contours and fibrotic extensions to the pleura, the largest of which is 124 x 14 mm in size in the subpleural area in the anterior subpleural area in the apical-posterior segment of the left lung upper lobe.

Subsequent fluorodeoxyglucose positron emission tomography-computed tomography (FDG PET-CT) revealed increased 18F-FDG uptake in multiple lymph node stations, including prevascular, paratracheal (bilateral), paraaortic, left hilar, right infrahilar, and pericardiac lymph nodes (maximum SUV= 10.6) (Figures 3,4). Additional FDG-avid lymphadenopathy was observed in celiac, peripancreatic, perihilar, bilateral paraaortic, mesenteric, iliac, and inguinal regions, raising suspicion for a lymphoproliferative disorder such as lymphoma.

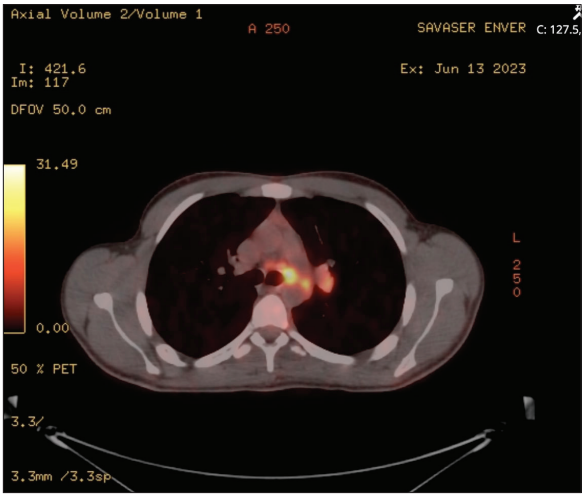

Figure 3. PET CT shows increased 18F-FDG uptake in lymph nodes in the mediastinum and left hilar region.

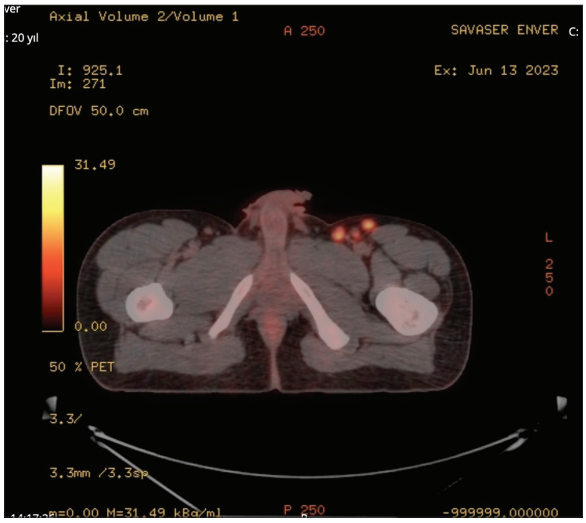

Figure 4. PET CT shows increased/intensely increased 18F-FDG uptake in multiple lymph nodes in the left inguinal lymphatic nodes.

Bronchoalveolar lavage (BAL) obtained via fiberoptic bronchoscopy was reported as benign cytology. An excisional biopsy of the inguinal lymph node performed by general surgery revealed chronic necrotizing granulomatous lymphadenitis. Moreover, BAL culture grew *Mycobacterium tuberculosis*, sensitive to isoniazid, rifampin, ethambutol, and streptomycin. Lumbar puncture and cerebrospinal fluid (CSF) sampling were not performed due to the mass effect of intracranial lesions. The diagnosis of pulmonary TB, along with necrotizing granulomatous inflammation in the lymph node, suggested that the intracranial lesions were likely tuberculomas. Additionally, the absence of malignant foci on PET-CT helped exclude metastasis from the differential diagnosis. An anti-human immunodeficiency virus (HIV) test was performed and yielded a negative result. Hematological evaluation revealed no evidence of a primary immunosuppressive condition.

The treatment of intracranial tuberculoma consists of an initial two-month intensive phase with quadruple anti-TB therapy, similar to that of TB meningitis, followed by a prolonged continuation phase, with a total treatment duration of 9 to 12 months[8]. Therefore, the patient was started on quadruple anti-TB therapy (isoniazid 300 mg, rifampin 600 mg, ethambutol 1250 mg, pyrazinamide 1500 mg) along with methylprednisolone (80 mg) once daily. Directly

observed therapy was initiated for close monitoring. He was scheduled for monthly follow-up evaluations, including clinical assessments and radiological examinations as needed.

One month after treatment initiation, he presented with headache and vomiting. Neurological examination revealed a Glasgow Coma Scale score of 13 and upward gaze palsy. CT of the brain demonstrated obstructive hydrocephalus due to compression of the fourth ventricle by the cerebellar lesion (Figure 5). Emergency ventriculoperitoneal (VP) shunting was performed, which resulted in clinical improvement. A total of 120 erythrocytes were observed, with no leukocytes detected in CSF. CSF protein was 100 mg/dL, CSF glucose was 49 mg/dL, and simultaneous blood glucose was 136 mg/dL. Culture results showed no growth, and acid-fast bacillus (AFB) staining was negative. TB polymerase chain reaction was negative.

Since clinical response was achieved during the second month of the four-drug regimen, the treatment was switched to isoniazid 300 mg and rifampin 600 mg once a day, and corticosteroids were discontinued. In the second month of dual treatment, the patient presented with abdominal redness and swelling at the shunt site. Revision of the distal shunt catheter was performed. Approximately five months later, the patient developed recurrent headaches, and brain imaging revealed worsening edema (Figure 6). A total of 10 leucocytes were observed (60% neutrophils) in the CSF sample obtained from the shunt. CSF protein was 31 mg/dL, CSF glucose was 50 mg/dL, and simultaneous blood glucose was 124 mg/dL. No bacteria were observed on Gram staining, and no growth was detected in the CSF culture. To reduce the pressure on the ventricles, subtotal surgical resection of the cerebellar lesion was planned. The procedure was successfully performed without any early postoperative complications. Intraoperative sample taken from the cerebellar lesion showed grade 2 positivity on AFB staining, *M. tuberculosis* complex DNA was positive, but TB culture showed no growth. Histopathological examination revealed necrotizing granulomatous inflammation (Figure 7-9).

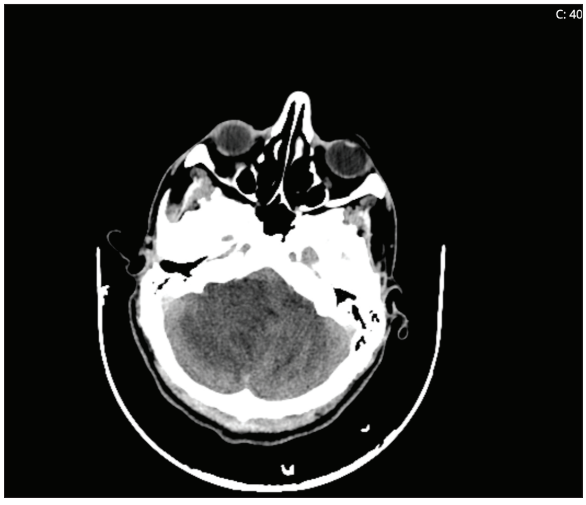

Figure 5. Brain CT shows compression of the fourth ventricle secondary to a mass lesion in the cerebellum.

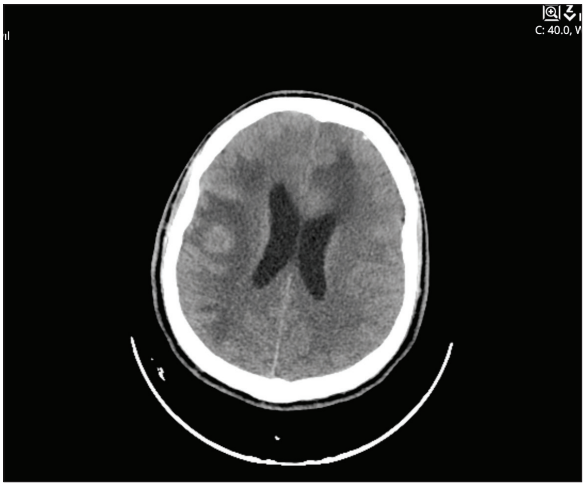

Figure 6. Brain CT shows dilatation of the posterior horn and temporal horn of the lateral ventricles due to compression of the fourth ventricle by the mass lesion in the cerebellum.

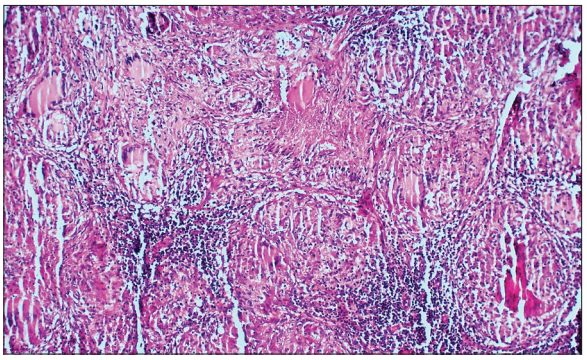

Figure 7. Microgranulomas in the cerebellum (H&E, x100).

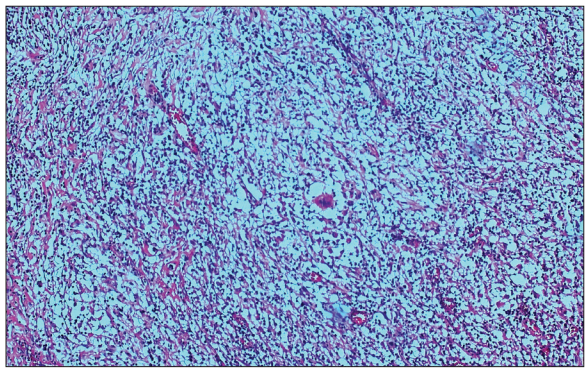

Figure 8. CD68 staining in cerebellar microgranulomas (CD68, DAB, x10).

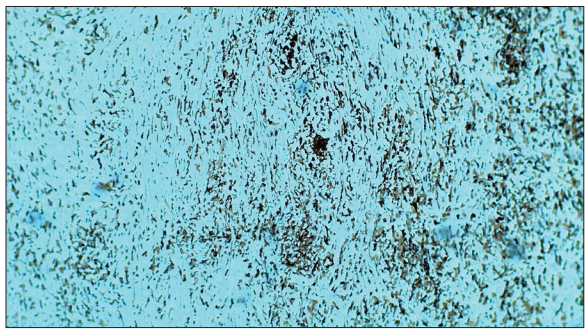

Figure 9. Caseating necrotizing granulomas in a lymph node (H&E, x100).

By the sixth month of anti-TB treatment, the patient presented again with fever, nausea, and vomiting. Suspecting a shunt infection, the VP shunt was removed. After confirming the absence of hydrocephalus on follow-up imaging, the patient was discharged. Two weeks later, he returned with signs of raised intracranial pressure. A repeat CT revealed ventricular dilatation, and a ventriculoatrial (VA) shunt was inserted. The postoperative period was uneventful, and the patient was discharged after suture removal. Follow-up imaging showed marked regression of both the ventricular dilatation and pulmonary lesions (Figs 10,11). The patient remains under anti-tubercular treatment with good clinical progress.

Literature Review

We searched the PubMed, Scopus, and Web of Science databases using the keywords

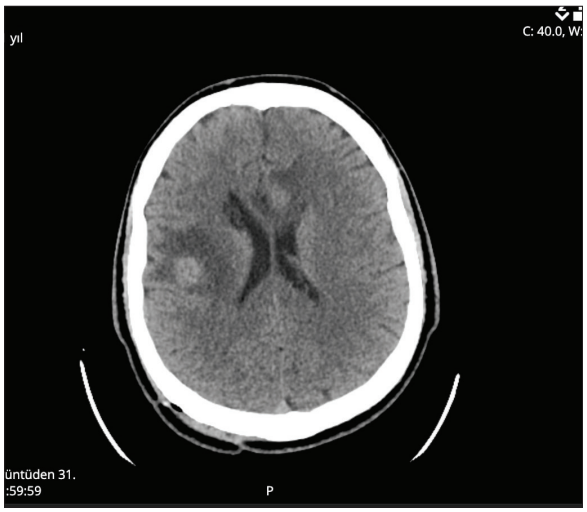

Figure 10. Brain CT shows a marked reduction in dilatation of the ventricular system.

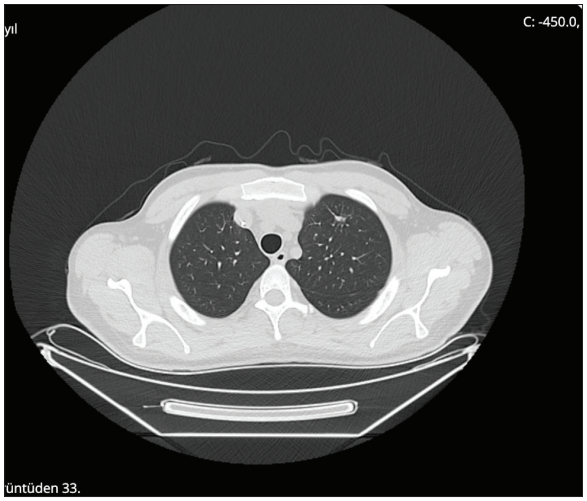

Figure 11. Thorax CT shows marked regression of the nodular lesion at the apex of the right lung.

"tuberculoma", "tuberculosis", "brain", "intracranial", "multiple", and "immunocompetent". Only English-language case reports were included, and pediatric patients were excluded. A total of 27 case reports were identified that met the inclusion criteria. Table 1 summarizes the demographic, clinical, radiological, and treatment characteristics of these cases (Table 1). Detailed information regarding the literature review is presented in Figure 12.

Table 1. Characteristics of multiple intracranial tuberculosis cases in immunocompetent patients

Reference	Age/ Gender	Symptoms	Intracranial Localization	Other TB	Treatment	Outcome
Guzel et al. (2005)[11]	32/F	Progressive left hemiparesis	Right frontoparietal and occipital regions	None	Anti-tuberculosis therapy 18 months, dexamethasone four weeks	All lesions disappeared
Oncul et al. (2005)[12]	21/M	Fever, cough, headache, weakness, loss weight	Cerebellar hemisphere, right thalamus, corpus callosum	None	Anti-tuberculosis therapy 12 months	Complete resolution of all the lesions
Başar et al. (2005)[13]	21/F	Fever, nausea, diarrea, weight loss abdominal pain, night sweats	Cerebral hemispheres	Abdominal TB	Anti-tuberculosis therapy* morphozynamide, dexamethasone	Complete resolution of the granulomas
Zavasck et al. (2006)[14]	33/F	Headache, nausea, and vomiting	Cerebral hemispheres and cerebellum	None	Anti-tuberculo-sis therapy nine months	Complete healing of the lesions
Alkhani et al. (2006)[15]	25/F	Progressive headache, malaise	Cerebral hemispheres and cerebellum	None	Anti-tuberculosis therapy 18 months, ventriculoperitoneal shunt	Complete resolution of the lesions
Ogunrin and Adeyeku, (2007)[16]	28/M	Unsteadiness of gait, dysarthria, weakness of the left extremites	Both cerebral peduncles, cerebellum	None	Anti-tuberculosis therapy 18 months, prednisolone	Clinical improvement
Gupta and Singhal. (2011)[17]	60/F	Weakness and headache	Cerebral hemisphere	Pulmonary TB	Anti-tuberculosis therapy*, dexamethason, mannitol	Clinical recovery
Matsumoto et al. (2013)[18]	70/M	Loss of memorizing power	Cerebral hemisphere	None	Anti-tuberculosis therapy*, dexamethason, mannitol	Reduction in the size and extent of the lesion
	30/M	Low bac pain, vomiting and vertigo	Cerebral hemisphere	Pulmonary TB	Anti-tuberculosis therapy*	N/A
Monteiro et al. (2013)[19]	55/M	Sudden onset of diplopia	Cerebral hemisphere	None	Anti-tuberculosis therapy 24 months, prednisolone eight weeks	Total resolution of the tuberculomas
Iqbal et al. (2014)[20]	24/M	Fever, headache, neck pain loss of appetite	Cerebral hemisphere	Miliary TB	Anti-tuberculosis therapy*, dexamethasone	Clinical recovery
Prasad et al. (2015)[21]	28/M	Progressive weakness of both lower limbs	Cerebral hemisphere, cerebellum, brainstem	Miliary TB	Anti-tuberculosis therapy for 12 months, steroids	Near complete resolution of the tuberculomas.

Table 1. Characteristics of multiple intracranial tuberculosis cases in immunocompetent patients (continue)

Reference	Age/ Gender	Symptoms	Intracranial Localization	Other TB	Treatment	Outcome
Machida et al. (2018)[22]	60/F	Headache, short-term memory impairment, and mild consciousness disturbance.	Bilateral lateral ventricle, bilateral triangle, right anterior horn, and body of the ventricle	None	Anti-tuberculosis therapy*, steroid	Clinical improvement
Venter et al. (2018)[23]	24/M	Headaches and seizures	Cerebral hemispheres	Pulmonary TB	Anti-tuberculosis therapy nine months, dexamethasone eight weeks	Some of the tuberculomas had already resolved.
Mohamm adian and Butt. (2019)[2]	69/M	Fever, weakness, recent unintentional weight loss	Cerebellar hemispheres	None	Anti-tuberculosis therapy 20 months, prednisone eight weeks	Resolution of brain lesions.
Vu et al. (2019)[24]	23/M	Chronic cough presented for a witnessed, new-onset seizure.	Right frontal and temporal lobes, cerebellum, and lower pons	Pleural effusion	Anti-tuberculosis therapy*, steroid	Clinical improvement
Velásquez-Rimachi et al. (2020)[25]	21/F	Headaches, nausea, vomiting, dizziness, and ataxia	Cerebral hemisphere, cerebellum, brainstem and spinal cord	Miliary TB	Anti-tuberculosis therapy 12 months and dexamethasone three weeks	Clinical recovery and resolution of tuberculomas
Rezaee et al. (2020)[26]	38/F	Headache, nausea, fever, chills, and back pain	Cerebral hemisphere	TB spondylitis and meningitis,	Anti-tuberculosis therapy 12 months, surgical resection	Clinical recovery
Arif et al. (2020)[27]	65/M	Anorexia, fever, headache and weight loss	Cerebral hemisphere	None	Anti-tuberculosis therapy 12 months, dexamethasone eight weeks	Complete resolution of lesions
Saleh et al. (2021)[28]	22/F	Memory loss, vision abnormalities, and intermittent tonic-clonic seizures	Left parietal lobe, right occipital lobe	None	Anti-tuberculosis therapy 24 months,	Clinical improvement
Boularab et al. (2022)[29]	57/M	Severe headaches, altered consciousness, and vomiting	Cerebellum, bilateral frontal, parietal, and temporal lobes, the left thalamus	None	Anti-tuberculosis therapy*, steroid	Clinical improvement
	22/M	Degradation of consciousness, which was preceded by headache dysarthria of progressive onset along with lower limb weakness	Frontal and parietal lobes, right thalamus.	None	Anti-tuberculosis therapy*, steroid	Clinical improvement

Table 1. Characteristics of multiple intracranial tuberculosis cases in immunocompetent patients (continue)

Reference	Age/ Gender	Symptoms	Intracranial Localization	Other TB	Treatment	Outcome
Chang et al. (2022)[30]	66/F	Intermittent headaches and neck pain	Bilateral temporoparietal	Pulmonary TB	Anti-tuberculosis therapy*,	Clinical improvement
Diker et al. (2022)[31]	20/F	Tonic-clonic seizure	Cerebral hemisphere	None	Anti-tuberculosis therapy 18 months	Near complete resolution of the tuberculomas
Huq et al. (2022)[32]	27/M	Fever and headache	Left supra and right infra-tentorial	None	Anti-tuberculosis therapy*, dexamethasone	Clinical improvement
Farrokhnia et al. (2024) [33]	67/M	Fever, malaise, myalgia, headache, and seizure episode	Cerebrum, cerebellum, and brainstem	None	Anti-tuberculosis therapy* dexamethasone	Clinical improvement
Yadav et al. (2024)[34]	28/F	Headache, nausea and vomiting	Cerebellar hemisphere and left thalamus	Pulmonary TB	Anti-tuberculosis therapy 12 months,	N/A
Present case	20/M	Facial numbness and episodes of inability to speak lasting	Cerebel lar hemisphere, right cerebellar region	Pulmonary TB, TB lymphadenitis, hydrocephalus	Anti-tuberculosis therapy, dexamethasone eight weeks	Clinical improvement

TB: Tuberculosis, N/A: Not available.
* The article does not detail how long the patient was treated.

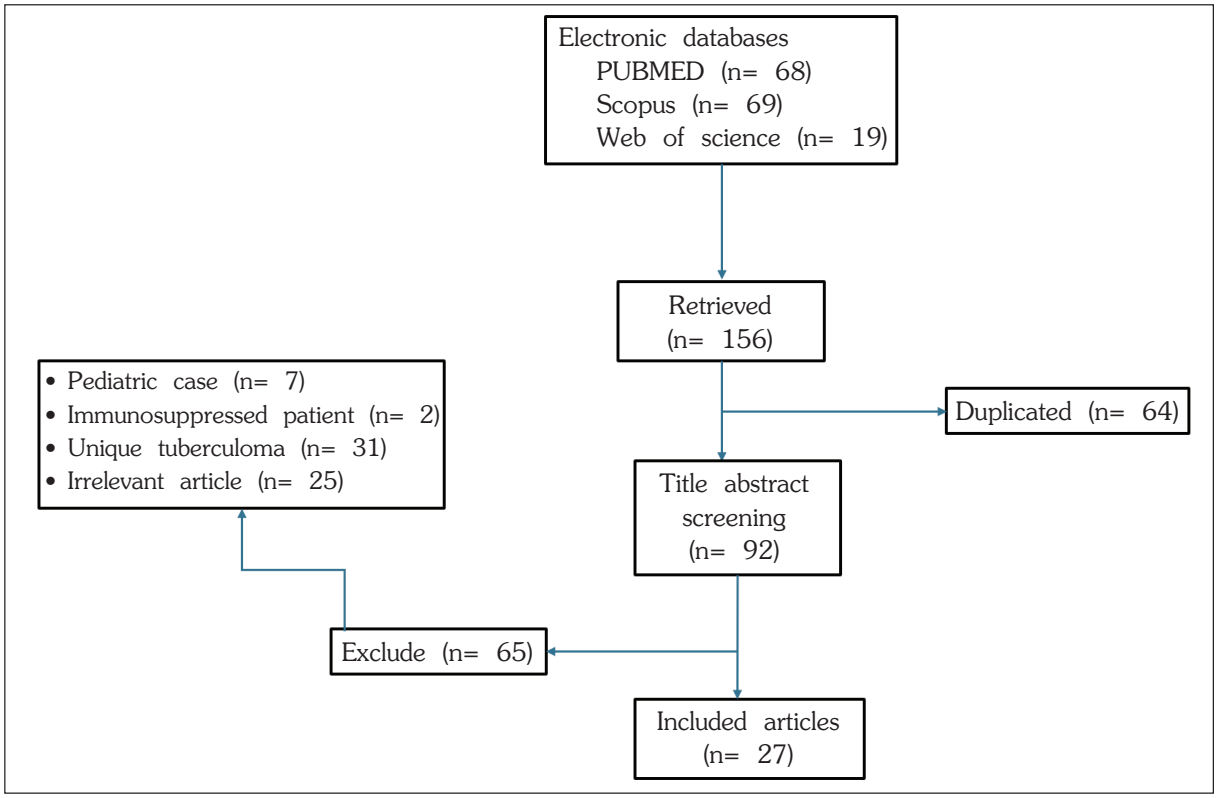

Figure 12. Flowchart of the literature review process.

DISCUSSION

Intracranial tuberculoma is defined as a granulomatous mass lesion formed by TB and other inflammatory cells in brain tissue. Tuberculomas are well-defined, solitary or multiple (ratio 4:1), firm, nodular, avascular lesions that can grow from a few millimeters to 3-4 cm, causing significant mass effects such as perifocal edema and gliosis[9]. While usually solitary, immunocompromised patients can develop multiple lesions[10]. In the present case, despite the absence of an underlying immunosuppressive condition, the patient had multiple intracranial tuberculomas. Based on this finding, we reviewed the current literature on multiple intracranial tuberculoma cases in immunocompetent adult patients. A total of 27 adult cases were identified in the literature, and our case represents the 28th (Table 1)[11-34].

In previously reported cases of intracranial tuberculoma, the mean age was 38.2 ± 18 years, with a slight male predominance (56%, n= 15). The most frequent symptoms were headache (59%, n= 16) and fever (37%, n= 10). The most frequently observed neurological findings were limb weakness (22%, n= 6) and seizures (19%, n= 5). Weight loss was reported in 15% (n= 4) of the patients. The present case was a 21-year-old male patient who presented with neurological symptoms, including facial numbness and speech impairment. The patient's medical history revealed weight loss and night sweats. The variation in clinical presentation may be explained by lesion localization and host immune response, suggesting that tuberculomas can manifest differently depending on the site of CNS involvement and the degree of inflammation they provoke.

CNS involvement in TB typically occurs through hematogenous dissemination from a pulmonary or extrapulmonary focus. In the literature, no additional TB focus is identified in 59% of cases, while the lungs are the most frequently affected concomitant site (22%). In the present case, the presence of intracranial tuberculomas in conjunction with pulmonary tuberculosis and mediastinal lymphadenitis suggests systemic TB infection with hematogenous spread to the central nervous system. Conversely, the absence of systemic involvement in many reported cases demonstrates that TB can present solely with neurological symptoms even in immunocompetent individuals. These heterogeneous presentations underscore the variable clinical course of TB and highlight the potential for disseminated forms to cause multiple intracranial lesions regardless of host immune status.

In tuberculosis-endemic regions, tuberculomas are a critical consideration in the differential diagnosis of multiple intracranial lesions[35]. These lesions may occur in both supratentorial and infratentorial regions, but their ability to mimic both infectious and neoplastic conditions often complicates diagnosis. While imaging techniques such as CT and MRI are sensitive in detecting mass lesions, they are generally insufficient to distinguish tuberculomas from other etiologies. Therefore, a multidisciplinary approach that integrates clinical, laboratory, and radiological findings is essential[36]. According to the literature, the most affected regions are the cerebral hemispheres (56%, n= 15), followed by the cerebellum (30%, n= 8) and, less frequently, the thalamus (11%, n= 3). This distribution may reflect the hematogenous spread of *M. tuberculosis*, which tends to involve highly vascularized brain regions. In the present case, the lesions were in both the cerebral hemispheres and the cerebellum, consistent with the multifocal involvement described in previous reports. Initially, due to the multiplicity of lesions and their radiological features, metastatic disease was suspected. However, the identification of *M. tuberculosis* in the BAL culture and the presence of necrotizing granulomatous inflammation in a lymph node biopsy suggested the diagnosis of tuberculoma. This was confirmed by the detection of TB DNA and the histopathological finding of caseating granulomas in the specimen obtained during neurosurgery. This case underscores the critical role of microbiological and histopathological confirmation in establishing causality and making a definitive diagnosis of intracranial tuberculoma.

The treatment of neurotuberculosis generally parallels that of pulmonary and other extrapulmonary forms of tuberculosis. It typically involves

an initial two-month intensive phase with isoniazid, rifampin, pyrazinamide, and ethambutol (HRZE), followed by a continuation phase with isoniazid and rifampin. The World Health Organization (WHO) recommends extending the HR regimen for up to 12 months following the initial HRZE phase[37]. Adjunctive therapy with dexamethasone or prednisolone is also advised for 6 to 8 weeks. In the literature on multiple intracranial tuberculomas, treatment duration was unspecified in 44% of cases (n= 12). Among the remaining 15 cases (56%) reporting treatment length, the average duration was 14 months, ranging from 9 to 24 months (Table 1). Steroid therapy was administered in 67% of cases (n= 18). In the present case, the patient received an initial two-month treatment with HRZE combined with methylprednisolone, followed by maintenance therapy with isoniazid and rifampin. Steroid therapy was continued for a total of eight weeks. The total planned treatment duration is at least 18 months and remains ongoing. These variations in treatment duration and approach likely reflect differences in disease severity and clinical progression.

Large tuberculomas located in the posterior fossa, as well as the presence of multiple lesions, can result in increased intracranial pressure. The mass effect of a tuberculoma may be disproportionately large due to significant surrounding cerebral edema[38]. In cases presenting with symptoms of raised intracranial pressure and hydrocephalus caused by obstruction secondary to the mass effect, surgical resection of the lesion or VP shunting is recommended for prompt symptom relief[39]. In the literature, one case underwent surgical resection and another received VP shunting. In the present case, despite anti-tuberculous therapy, the cerebellar lesion's mass effect increased, leading to hydrocephalus. Initially, a VP shunt was placed; however, due to insufficient clinical improvement, the cerebellar mass was largely resected, and a VA shunt was subsequently inserted. This clinical course, consistent with some reported cases, underscores that medical therapy alone may not suffice in all patients, and surgical intervention, including shunting, may be necessary in complicated cases.

In conclusion, although rare in immunocompetent individuals, multiple intracranial tuberculomas should be considered in the differential diagnosis of intracranial mass lesions, particularly in endemic regions or patients with systemic TB symptoms. Overlapping clinical and radiological features with other intracranial pathologies make diagnosis challenging, necessitating microbiological and histopathological confirmation. Treatment involves prolonged anti-TB therapy combined with corticosteroids to manage inflammation. Surgical intervention and cerebrospinal fluid shunting are essential in cases complicated by significant mass effect or hydrocephalus. Early diagnosis and individualized treatment planning are critical for achieving favorable neurological and clinical outcomes.

INFORMED CONSENT

Informed consent was obtained from the patients for this study.

CONFLICT of INTEREST

The authors declare that they have no conflicts of interest.

AUTHORSHIP CONTRIBUTIONS

Concept and Design: YÇK, EK

Analysis/Interpretation: SAB, ÜÖ

Data Collection or Processing: ZB, YÇK

Writing: EK, YÇK

Review and Correction: ÜÖ, SAB, ZB

Final Approval: ÜÖ, SAB, YÇK, EK, ZB

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