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Case Report

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CNS Tuberculoma in a Patient of Acute Lymphoblastic Leukemia: A Rare Case Report

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ABSTRACT

Patients with acute leukemia are immunocompromised and highly susceptible to infections. Central nervous system (CNS) tuberculoma is a rare but serious complication in this population, particularly among those undergoing treatment for hematological malignancies. Early diagnosis is often challenging due to non-specific symptoms.

We report the case of a 28-year-old female recently diagnosed with CALLA-positive B-cell acute lymphoblastic leukemia, who presented with a two-month history of low-grade fever. Induction chemotherapy was initiated; however, during the third week, she developed new-onset seizures. A computed tomography scan of the brain revealed a heterogeneous ring-enhancing lesion in the right parietal lobe. Magnetic resonance imaging with spectroscopy demonstrated characteristic lipid peaks, supporting the diagnosis of a tuberculoma.

Antitubercular therapy comprising rifampicin, isoniazid, pyrazinamide, and ethambutol, along with pyridoxine and dexamethasone, was commenced. The patient showed a favorable response, with resolution of fever and no recurrence of seizures.

This case underscores the importance of considering CNS tuberculosis in the differential diagnosis of unexplained neurological symptoms and fever in patients with acute leukemia, particularly in tuberculosis-endemic regions. Prompt recognition and treatment can lead to favorable outcomes.

Keywords: Acute leukemia; Seizures, Magnetic resonance spectroscopy; Tuberculoma

INTRODUCTION

In 2022, approximately 10.6 million individuals worldwide developed tuberculosis (TB)—equivalent to 133 cases per 100,000 population—reflecting a 2.9% increase from 10.3 million cases (131 per 100,000) in 2021. TB-related deaths in 2022 totaled 1.3 million, a 6.4% decrease from 1.39 million in 2021[1]. Patients with acute leukemia have significant impairment of both humoral and cellular immunity, predisposing them to new infections and reactivation of latent infections, including TB. Although mycobacterial infections are more common in lung, head, and neck cancers, they can also occur in leukemia patients, albeit less frequently. Despite the global burden of TB, diagnosis in individuals with leukemia is often delayed due to atypical and subacute clinical presentations. The coexistence of CNS tuberculosis and leukemia is particularly rare, and a high index of suspicion is essential, as diagnostic or therapeutic delays can be life threatening, especially in acute leukemia[2].

Case presentation

A 28-year-old woman presented with a two-month history of low-grade fever and progressive pallor over the past six weeks, requiring multiple blood transfusions. Physical examination revealed bilateral cervical lymphadenopathy involving levels I to V, as well as mild splenomegaly. She had no prior history of tuberculosis, no known exposure to an infected individual, and no previous antitubercular treatment.

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Initial hematological investigations revealed significant abnormalities. Hemoglobin was critically low at 6 g/dL. The total leukocyte count was elevated to 18,500 cells/µL, with an absolute neutrophil count of 5,000 cells/µL. Peripheral smear revealed approximately 30% medium-sized blasts with condensed chromatin and scant cytoplasm. Platelet count was also reduced to 50,000/µL. Serum biochemistry showed normal renal and liver function, though lactate dehydrogenase was markedly elevated at 1,120 U/L, suggestive of high cell turnover.

Immunophenotyping of peripheral blood confirmed precursor B-cell acute lymphoblastic leukemia (CALLA+ B-ALL), with blast cells positive for CD34, CD45, CD10, CD19, CD20, CD22, CD24, and CD79. Following diagnosis, she began induction phase A of the BFM 2002 protocol, comprising prednisolone, vincristine, daunorubicin, L-asparaginase, and intrathecal methotrexate. Despite completing four weeks of induction therapy, the patient continued to have persistent fever, unresponsive to broad-spectrum antibiotics tailored to blood culture sensitivities. On day 28 of treatment, however, she experienced two episodes of generalized tonic-clonic seizures, each lasting approximately 20 minutes.

A thorough metabolic workup, including serum calcium, magnesium, phosphorus, and potassium, revealed no electrolyte abnormalities to explain the seizures. To investigate further, computed tomography (CT) of the brain was performed and revealed an inhomogeneous ring-enhancing lesion in the right parietal lobe (Figure 1A). Subsequent brain magnetic resonance imaging (MRI) with spectroscopy showed multiple ring-enhancing lesions with perilesional edema in the right parietal and occipito-temporal regions. A characteristic lipid peak on spectroscopy was suggestive of CNS tuberculoma (Figures 1B, 2).

In light of these findings, the patient was started on standard anti-tubercular therapy (rifampicin, isoniazid, pyrazinamide, and ethambutol) on day 29. Pyridoxine was added to prevent isoniazid-induced neurotoxicity, and she received dexamethasone 0.6 mg/kg/day to manage cerebral edema. Five days after initiating anti-tubercular treatment, the patient showed marked clinical improvement. Her fever resolved by day 34, and no further seizures occurred. After her condition stabilized, she successfully completed the remainder of her chemotherapy regimen.

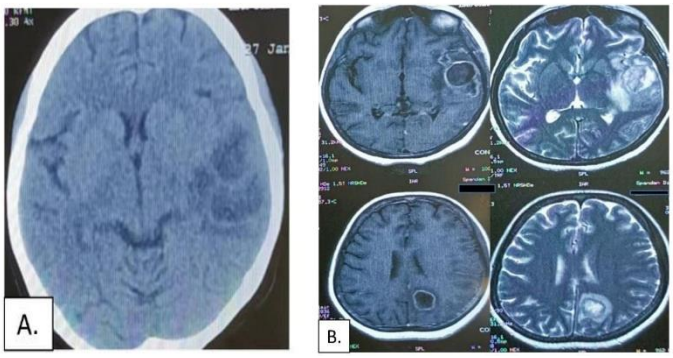

Figure 1. A 28-year-old woman with CALLA+ B-ALL (A) CT Head showing inhomogeneous lesion in right partial lobe (B) MRI brain showing multiple ring enhancing lesions (hypointense T1) (hypointense T2) with perilesional edema

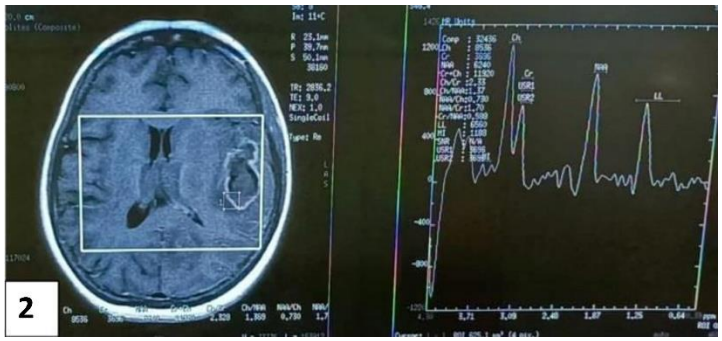

Figure 2. MR spectroscopy of a 28-year-old woman with CALLA+ B-ALL showing characteristic lipid peak suggestive of tuberculoma

DISCUSSION

Tuberculomas represent approximately 20–30% of all intracranial space-occupying lesions globally and predominantly affect young individuals. They arise from coalesced granulomatous foci formed during early hematogenous dissemination of tuberculosis, often without accompanying meningitis. Clinically, patients may present with headache, vomiting, new-onset seizures, focal neurological deficits, or papilledema. Neuroimaging is indispensable for diagnosis. On CT, these lesions typically appear as ring-enhancing masses with perilesional edema. MRI

usually demonstrates ring-shaped lesions that are hypointense on T1-weighted and hyperintense on T2-weighted images. In patients with acute leukemia, persistent fever and new-onset seizures pose a diagnostic challenge, as the differential includes leukemic CNS involvement, fungal abscesses, toxoplasmosis, drug-related neurotoxicity, and posterior reversible encephalopathy syndrome. In tuberculosis-endemic regions, CNS tuberculoma should be strongly considered. MR spectroscopy plays a crucial role in this setting by demonstrating a lipid peak characteristic of tuberculoma. This aids differentiation from neoplastic and pyogenic lesions and reduces the need for invasive diagnostic procedures[3].

Histopathological confirmation was not obtained due to the deep parenchymal location of the lesions and the high procedural risk in this immunocompromised, thrombocytopenic patient. The diagnosis was therefore based on characteristic MRI and MR spectroscopy findings—notably the lipid peak—and a rapid, sustained clinical response to antitubercular therapy. This approach is widely accepted in clinical practice when tissue diagnosis is not feasible. The management of CNS tuberculosis requires prolonged antitubercular therapy (ATT). The standard regimen comprises four first-line drugs—rifampicin, isoniazid, pyrazinamide, and ethambutol—administered over 9 to 12 months. Adjunctive corticosteroids (e.g., dexamethasone) are recommended to reduce inflammatory edema and improve clinical outcomes. Dexamethasone dosing typically ranges from 0.6 mg/kg/day in children to 0.4 mg/kg/day in adults[4]. Tuberculosis is an uncommon but important cause of febrile neutropenia in patients with acute leukemia. Previous studies, including those by Chen et al. 5 and Mishra et al. [6], have demonstrated a higher incidence of tuberculous infections, especially in patients with acute myeloid leukemia (AML), compared to other hematological malignancies. However, CNS tuberculosis in patients with acute lymphoblastic leukemia (ALL), particularly the CALLA-positive B-cell subtype described in this case, remains rare. This heightened vulnerability is largely due to profound immunosuppression from both the underlying malignancy and intensive chemotherapy. Such immunosuppression predisposes to new infections and can reactivate latent TB. Latent TB poses a significant clinical challenge, as symptoms are often subtle or atypical, which can delay diagnosis and treatment.

Timely initiation of antitubercular therapy is critical in these immunocompromised patients. With early and appropriate treatment, favourable outcomes are achieved in up to 90% of cases, underscoring the importance of prompt diagnosis. Prompt therapy not only reduces the risk of mortality from CNS tuberculosis but also allows continuation of chemotherapy without significant interruption, which is essential for controlling the primary leukemia[7].

This case is unique among previously reported cases (Table 1) for its occurrence during induction therapy, MR spectroscopy-based diagnosis, and successful chemotherapy continuation after prompt ATT. Prior reports (e.g., Zivanovic et al.[8]) describe TB in ALL as predominantly pulmonary/pleural, occurring 20 months post-immunosuppression. Conversely, our patient developed early isolated CNS tuberculoma without systemic disease—demonstrating a distinct timing, organ involvement, and diagnostic strategy.

Table 1: Comparison of the results/outcome of the present study with different other studies published in the literature

Study	Patient age/ sex	Presentation	Diagnosis	Treatment
Thomas et al.[7]	58 years male	Fever, cough, anorexia, weight loss, and cervicallymphadenopathy	Acute myeloid leukemia with pulmonary tuberculosis	Four drug regimen ATT (Isoniazid, rifampicin, pyrazi-namide, and ethambutol with pyridoxine) and 3+7 induction chemotherapy
Zivanovic et al.[8]	14 years male	Fever, sweating, cough, and pallor	T-Acute lymphoblastic leukemia with tuberculous pleuropneumonia	Four drug regimen ATT (Isoniazid, rifampicin, pyrazi-namide, and ethambutol with pyridoxine) and prednisolone
Present study	28 years female	Fever, seizures, pallor, cervical lymphadenopathy, splenomegaly	CALLA+ B- Acute lymphoblastic leukemia with CNS tuberculoms	Four drug regimen ATT (Isoniazid, rifampicin, pyrazi-namide, and ethambutol with pyridoxine), dexamethasone and chemotherapy (vincristine, daunorubicin, L-asparaginase, and intrathecal methotrexate)

CONCLUSION

CNS tuberculosis is a rare but frequently overlooked cause of febrile neutropenia in acute leukemia patients. Diagnosis is often delayed due to its subtle, subacute progression and atypical features, which are easily mistaken for other infections or leukemia-related complications. Prompt and accurate diagnosis is crucial, as early ATT leads to rapid clinical improvement and prevents serious complications. In immunocompromised leukemia patients, failure to diagnose and treat CNS TB promptly can increase morbidity and mortality. Furthermore, once ATT is initiated, chemotherapy should be started or resumed as soon as the patient's condition allows, as early continuation is essential for controlling the underlying malignancy and reducing cancer-related mortality. This approach underscores the need for a high index of suspicion for CNS TB in leukemic patients with persistent fever and neurological symptoms, particularly in TB-endemic regions. Ultimately, prompt diagnosis and coordinated management of both tuberculosis and leukemia significantly improve patient outcomes.

Data availability statement

The datasets generated and/or analysed during the current study are available from the corresponding author upon reasonable request.

Declaration of patient consent

The authors certify that they have obtained all appropriate written informed consent from the patient for publication of this case report.

Consent to publish

The patient provided written informed consent for the publication of this case report and any accompanying images.

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Nil.

CONFLICTS OF INTEREST

There are no conflicts of interest.

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