

Tuberculoma and disseminated tuberculosis infection presenting as status epilepticus: A Case Report

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Introduction

In this article we describe an unusual presentation of extrapulmonary tuberculosis infection in a 42 gentleman who presented with seizures. We also discuss various presentations of disseminated tuberculosis and guidelines on management

Case

A 42 year old Indian male with residence in Ireland for 5 years presented to hospital by ambulance in status epilepticus on a background of recent alcohol abuse. The patient had no previous medical records and no collateral history was available at time of presentation.

Initial laboratory investigations showed mild pancytopenia with profound lymphopenia of $0.1 \times 10^9/L$ on admission. CT Brain showed vasogenic oedema within the inferolateral right frontal lobe and some surrounding ill-defined enhancement.

Lumbar puncture was performed, and CSF showed a raised protein (125mg/dL) with normal white cell count and no bacterial or fungal growth. Viral PCR panel was negative for HSV1, HSV2, EBV and Enterovirus. He was commenced on IV ceftriaxone, vancomycin and metronidazole to treat possible intracranial infection, and IV dexamethasone for cerebral oedema. He was also given chlordiazepoxide and IV thiamine for alcohol withdrawal

MRI subsequently revealed a 2cm intra-axial ring enhancing lesion in the subcortical white matter of the right frontal lobe and a small lesion in the left thalamus (Figure 1 and 2). He was extubated successfully and moved to a medical ward following 2 days in ICU. His HIV test was negative, immunoglobulin counts were in normal range and lymphocyte subsets revealed deficiency in all subgroups.

Case Continued..

CT axial imaging revealed mediastinal and right supraclavicular lymphadenopathy with some omental thickening and free fluid in the pelvis suggestive of peritoneal disease. The right supraclavicular node was amenable to biopsy, and this was carried out with radiological guidance. TB PCR was subsequently positive from this tissue sample, and rifampicin resistance gene was not detected. EBUS was performed to sample a right paratracheal node and later proved to be non-diagnostic. He was commenced on rifampicin, isoniazid, pyrazinamide, and ethambutol.

Following haematology advice regarding the persistent and marked lymphopenia, a bone marrow biopsy was carried out to investigate possible marrow involvement or underlying malignant process. The bone marrow aspirate did not show any blasts or other malignant cells, and there were no granulomas or evidence malignancy on analysis of the trephine. Mycobacterial cultures from the aspirate are negative to date.

The patient's cognitive status initially fluctuated, however this gradually improved over the course of his admission. He suffered no further seizures on a combination of levetiracetam and lacosamide. At present he continues on a 4 drug regimen and mycobacterial sensitivities are

MRI Brain

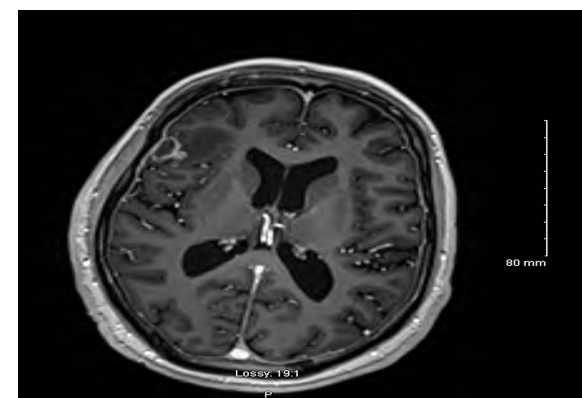


Figure 1: T₁ weighted image

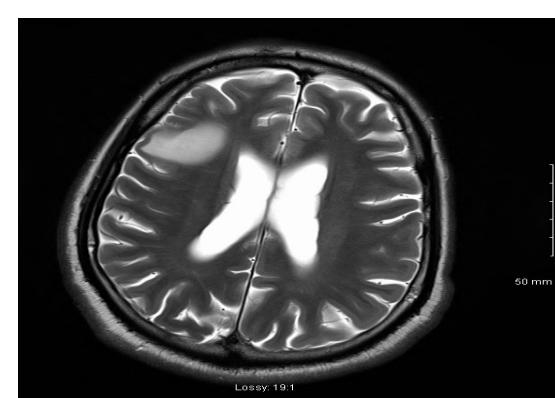


Figure 2: T₂ weighted image

Ring enhancing lesion R Frontal Lobe

Cerebrospinal Fluid Results

Test	Result	Normal Range
Protein	125	15-45mg/dL
Glucose	3.6	2.8-4.4 mg/dL
TB-PCR	Not Detected	Not detected
Mycobacterial Culture	Pending	Sterile

Discussion and Conclusion

CNS TB infection occurs in 10% of all cases of disseminated TB and arises in both immunocompromised and immunocompetent hosts. It is further subdivided into three categories, tuberculoma, mycobacterium tuberculosis meningitis and spinal tuberculosis arachnoiditis. [1] The clinical presentation of tuberculoma is similar to that of any space occupying lesion which may or may not include involvement of other organ systems. With our patient, CSF contained mildly elevated protein but no inflammatory cells indicating the infection was confined to brain parenchyma and absence of meningitis. Poor prognostic indicators include advanced age, concomitant haematogenous disseminated tuberculosis, low GCS score at the time of admission and hydrocephalus[2]

Prompt tissue sampling is required to confirm diagnosis and commence appropriate therapy as soon as possible. Fortunately in this case, we were able to obtain a sample from a superficial node which provided a positive TB PCR result and culture with low risk to the patient. In some cases brain biopsy must be carried out to confirm diagnosis and carries greater risk to the patient. [2] It is of vital importance to rule out underlying immunosuppression from other causes, especially HIV which is frequently implicated in disseminated tuberculosis infection. TB can also be associated with a number of haematological manifestations. [3] In the aforementioned case, the pancytopenia was concerning for bone marrow infiltration or haematological malignancy however this was absent on bone marrow biopsy. Treatment for all forms of CNS tuberculosis should consist of 4 drugs for 2 months followed by 2 drugs for at least 10 months. [4] Culturing of mycobacterium tuberculosis is necessary to guide anti-TB therapy, especially in patients from endemic areas of multidrug-resistant TB. Corticosteroids are an important component of therapy and have been shown to reduce mortality. [5]

In summary intracranial TB infection carries a high risk of morbidity and mortality. Confirming a diagnosis can be challenging and requires prompt treatment with anti-TB agents and corticosteroids.

References

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