INTRACEREBRAL HEMORRHAGE IN TUBERCULOUS MENINGITIS: A CASE REPORT

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BACKGROUND

Involvement of the central nervous system (CNS) by tuberculosis (TB) is a rare but devastating manifestation of TB because of its high mortality rate and possible serious neurological sequelae. It can manifest as meningitis, cerebritis, cerebral abscess, tubercolomas, miliary tubercolosis and spinal involvement. **Tuberculous meningitis** (TBM) is the most common presentation of neurotuberculosis and it can be usually complicated by hydrocephalus, vasculitis, infarction and cranial neuropathies. We describe an atypical case of TBM complicated by intracerebral hemorrhage.

CASE REPORT

- 40-year old indian man
- One-month history of left hemi-parhesthesias
- No systemic diseases or family history of neurological diseases
- Neurological examination: slight neck stiffness.

Cerebrospinal fluid	
Color	transparent
White blood cells	150/mm^3 (95% lymphocytes)
Proteins	73 mg/dl
Glucose level	71 mg/d
Glucose ratio CSF/serum	0,5
Lactate	17mg/dl

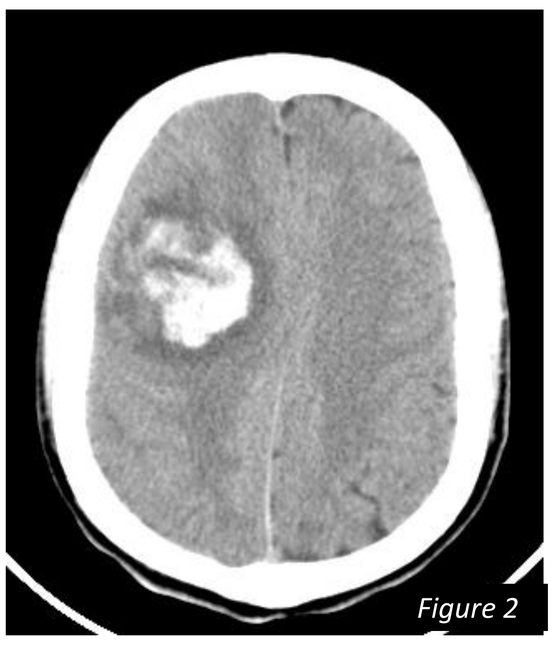

Figure 2

RADIOLOGICAL EXAMS

- Brain computer tomography (CT) scan: normal
- While brain magnetic resonance imaging (MRI): right frontotemporal meningeal contrast enhancement *(Figure 1)*
- Intracranial MRI angiography: normal
- Total-body CT scan showed chest multiple nodular opacified areas with tree-in-bud appearance.

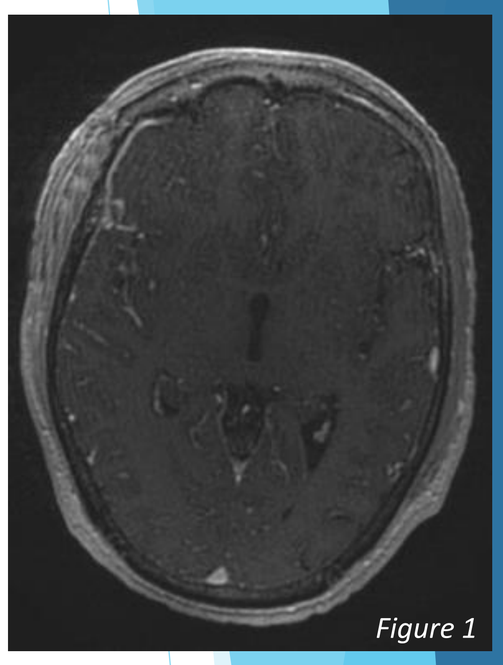

Figure 1

LABORATORY TESTS

- Standard blood tests: normal
- HIV, CSF and serum viral tests and common bacterial and fungal culture were negative.
- QuantiFERON-TB-gold test: positive

Four days after the admission he suddenly developed **left hemiplegia** and **fever.** The brain CT-scan detected right frontotemporal intracranial hemorrhage *(Figure 2)* that was treated surgically. Culture for **mycobacterium tuberculosis** from **meningeal biopsy** was positive. Histopathologic examination confirmed the diagnosis of TBM.
Anti-tubercular therapy and rehabilitation were started with clinical improvement.

One week after surgery brain MRI was performed again, revealing an **acute asymptomatic ischemic lesion in the right basal ganglia** *(Figure 3 and 4).*

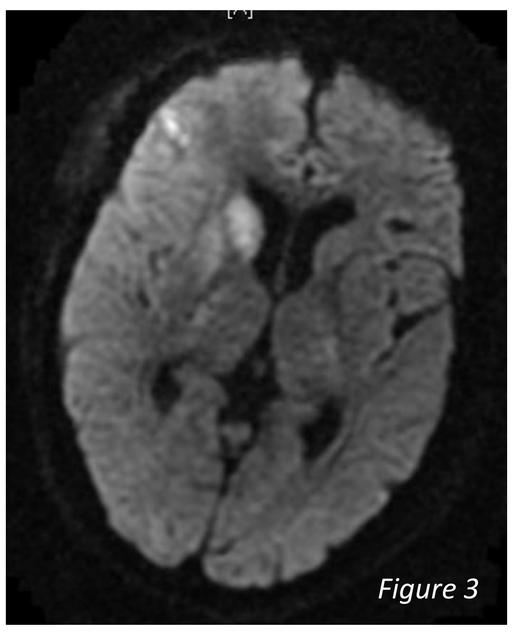

Figure 3

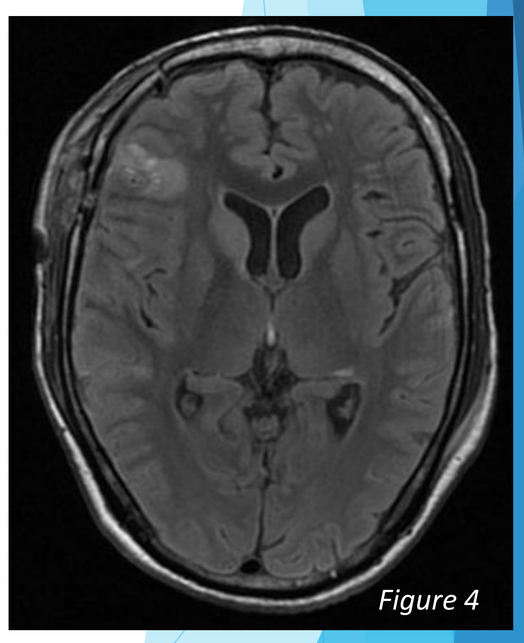

Figure 4

DISCUSSION AND CONCLUSION

TBM usually presents as enhancing exudate in the basal cisterns on CT and MR images. Occasionally meningeal enhancement is seen over the cerebral convexities, the sylvian fissures and the tentorium.

Cerebral infarction in TBM is due to vasculitis and its commonest location is the "tubercolar zone" which includes basal ganglia and internal capsule, due to involvement of perforating vessels. **Hemorrhagic lesion** in TBM is rare and has been attributed to aneurysmal rupture following the formation of mycotic aneurysms, or to non-aneurysmal rupture as a consequence of intimal fibrinoid degeneration due to vasculitis.

Our case is characterized by **nonspecific presenting symptom**s, **atypical neuroimaging** without basal cisterns enhancement, and sudden clinical worsening due to **cerebral haemorrhage**, which is a rare complication of TBM. As this disease has no unique characteristics at presentation, a diagnosis of TBM at an early stage may be very difficult, but it is crucial to allow a prompt treatment and to reduce morbidity and mortality.

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