CASE REPORT

A complicated case of altered sensorium

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SUMMARY

Tuberculosis and malaria are two common diseases afflicting the population of the Indian subcontinent. Both are associated with fatal neurological complications. Clinically, it may be difficult to differentiate between the two, except for that the focal neurological complications are more common with central nervous system (CNS) tuberculosis than with cerebral malaria. We report the case of a 48-year-old woman who presented with a history of fever and altered sensorium of short duration. She was initially treated for falciparum malaria on the basis of positive peripheral smear and normal CT scan of the brain, but later on diagnosed to have CNS tuberculosis on the basis of persistence of symptoms, development of focal neurological symptoms and MRI of the brain showing multiple tuberculomas. She improved significantly with a course of antitubercular treatment (ATT) and systemic corticosteroids.

BACKGROUND

Tuberculosis of the central nervous system (CNS) accounts for approximately 5% of all extrapulmonary cases. It has a much longer course than bacterial meningitis and may present either subtly as headache or acutely with confusion, altered sensorium and neck rigidity. Focal neurological deficits especially cranial nerve palsies are much more common. Other complications include hydrocephalus as well as vasculitis. Tuberculomas are an even more uncommon manifestation of CNS tuberculosis.

Cerebral malaria is a complication of infection caused by *Plasmodium falciparum*. It manifests usually as a diffuse symmetric encephalopathy and focal neurological signs are unusual. Seizures are a more common manifestation of this condition, especially in children.

This case report highlights the similar and often confusing presentation of both the clinical conditions. In both conditions, early diagnosis and prompt institution of treatment can ensure satisfactory recovery, although in some of the cases, some of the residual neurological deficits may persist, even after completion of the full course of treatment.

CASE PRESENTATION

A 48-year-old housewife presented with a history of fever for 5 days followed by headache and altered sensorium for 3 days. She did not have any significant premorbid illnesses or addictions. There was no history of use of any prescription drugs. There was no family history of any significant illnesses. Examination showed a regular pulse rate of 86 bpm, blood pressure 130/80 mm Hg, conjunctival pallor

Figure 1 Livedo reticularis.

and livedo reticularis over both lower limbs (figure 1). Systemic examination revealed positive meningeal signs, while cardiovascular, respiratory and abdominal examinations were normal.

The patient had been evaluated in an outside hospital with a CT of the head, which proved to be a normal study (figure 2). The treatment she received in the other hospital included antipyretics and antibiotics; however, details were not available. She was investigated and found to have low haemoglobin. Her total leucocyte counts, platelets and erythrocyte sedimentation rate (ESR) were all within normal limits. Her biochemical parameters including the serum electrolytes, renal functions and liver function tests were also within normal limits. HIV serology and immunoglobulin M toxoplasma were negative. Her chest X-ray was normal. Ultrasound abdomen revealed the presence of mild hepatosplenomegaly. Blood smear showed trophozoites of *Pfalciparum* with a microcytic hypochromic picture of RBCs and mild thrombocytopenia.

The common causes of altered sensorium in an immunocompetent adult in our geographical region are stroke, sepsis, cerebral malaria, tubercular meningitis, bacterial meningitis, meningoencephalitis, hepatic encephalopathy, uraemic encephalopathy and other metabolic encephalopathies like hypoglycaemia and hyponatraemia.

Our working diagnosis was cerebral malaria, and she was started on treatment with arte-ether and mefloquine. On the second day, her sensorium improved slightly, but fever persisted and subsequently her consciousness level deteriorated further. Lumbar puncture was performed and the CSF analysis showed low glucose (19 mg/dL), high protein (206 mg/dL), elevated leucocyte count (250 cells/cumm³) with a lymphocytic predominance (78%). Cerebrospinal fluid (CSF) adenosine

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Figure 2 CT scan of the brain.

deaminase level was 26 units/L. CSF Gram stain, culture and Ziehl-Neelsen (ZN) staining did not show any positive result. Given the CSF picture and in view of the short history and the fact that she had received antibiotics previously, she was treated as a case of partially treated bacterial meningitis with a

differential diagnosis of tubercular meningitis and she was started on intravenous ceftriaxone, ampicillin as well as gentamicin (as vancomycin was very expensive). Her symptoms improved slightly, but she subsequently developed focal neurological deficits in the form of right-sided Horner's syndrome

Figure 3 Right-sided Horner's syndrome.

(figure 3), left-sided upper motor type of facial nerve palsy (figure 4) as well as left-sided extensor plantar response.

MRI of the brain was taken, which revealed multiple ring-enhancing lesions in the various parts of the brain as well as obstructive hydrocephalus (figure 5).

INVESTIGATIONS

MRI report

Multiple ring-enhancing focal lesions were noted involving the left cerebellar hemisphere, right temporal lobe, right frontoparietal lobe and left temporo-occipital region causing effacement of adjacent sulci with possible exudates within the sulci (predominantly in the right frontoparietal lobe).

No significant perilesional oedema was seen, both lateral ventricles and third ventricles were dilated. Fourth ventricle appears normal—suggestive of obstructive hydrocephalus.

Possibility of tubercular meningitis with multiple tuberculomas and obstructive hydrocephalus.

TREATMENT

In the wake of the new clinical findings as well as the MRI picture, a diagnosis of CNS tuberculosis was made and she was started on antitubercular therapy (ATT) as per directly observed

Figure 4 Left-sided upper motor neuron type facial paralysis.

treatment short course (DOTS) category I regimen, including isoniazid, rifampicin, ethambutol and pyrazinamide, along with systemic corticosteroids.

OUTCOME AND FOLLOW-UP

The patient improved significantly after she was started on ATT regimen and some of her neurological deficits improved. She was subsequently discharged on ATT and oral corticosteroids. On her latest follow-up, about 1 year after her admission, she had completed her course of ATT and was asymptomatic except for a mild residual facial paralysis.

DISCUSSION

A case of altered sensorium is one of the toughest challenges that can test a physician. Among the infectious causes of altered sensorium, bacterial meningitis is the commonest cause. In India, especially with the high prevalence of tuberculosis, CNS tuberculosis is another common cause. CNS tuberculosis (chronic meningoencephalitis) has a prolonged prodrome. Cranial nerve palsies and hydrocephalus may be seen often due to vasculitis and basal exudates. On the other hand, focal neurological deficits are much less common with cerebral malaria.

When our patient initially presented, she was in altered sensorium and had a smear positive for malarial parasites. As she hailed from an area endemic for malaria and as her other preliminary investigations failed to show any other cause for her symptoms, she was initially treated for cerebral malaria. Since she did not improve subsequently, an alternative diagnosis was suspected. CSF analysis was suggestive of either partially treated bacterial meningitis or tubercular meningitis. However, once she developed focal neurological deficits, there was a strong suspicion of CNS tuberculosis, corroborative evidence of which was provided by the MRI.

This case draws a very strong analogy to another case report¹ concerning a 32-year-old HIV-positive man who had a similar initial presentation, and was treated for cerebral malaria as per the positive smear report. As he did not show any clinical improvement with antimalarials and antibiotics, but rather developed right sixth cranial nerve paresis, he was then started on ATT and steroids, with positive clinical response. Perhaps the only clue as to the tubercular aetiology was the presence of bilateral lung infiltrates, unlike in our patient with normal chest X-ray.

It is of utmost importance to make an early diagnosis of CNS tuberculosis. Case fatality is the highest among cases of tuberculosis meningitis (TBM), as much as 68.4%, as compared with the other causes of CNS infections.² The diagnostic tests available presently are not sensitive enough for the diagnosis of CNS tuberculosis. CSF culture is 71% sensitive, ZN staining of CSF is 20% sensitive, which can be increased to 80% by repeated sampling of large volumes of CSF.² Among the imaging modalities, MRI is far more sensitive for the diagnosis of parenchymal involvement by tuberculosis.³ This is highlighted in our case, while the initial CT of the brain was normal, MRI showed multiple ring-enhancing lesions in the brain parenchyma.

Finally, a word about the role of steroids in the management of CNS tuberculosis. The exact usefulness of steroids has not been proven, but studies have shown that while institution of steroids may not prevent the development of focal neurological deficits (even if started early in the course of treatment), it still reduces the incidence of mortality due to CNS tuberculosis.

Figure 5 An MRI study showing multiple ring enhancing lesions (marked by red arrows).

Learning points

- ▶ Central nervous system (CNS) tuberculosis and cerebral malaria may have similar and often confusing clinical presentations, especially in patients hailing from endemic areas.
- ▶ Focal neurological deficits are more common in CNS tuberculosis as compared to cerebral malaria.
- ▶ An MRI is a far better imaging modality, as compared with other imaging modalities, in the detection of CNS parenchymal involvement by tuberculosis.

Contributors All authors listed have actively contributed to the writing of the case report submitted.

Competing interests None.

Patient consent Obtained.

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