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Case Report

An atypical presentation of scrub typhus

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ABSTRACT

Scrub typhus is caused by *Orientia tsutsugamushi*, characterised by focal or disseminated vasculitis and perivasculitis which may involve the lungs, heart, liver, spleen and central nervous system. The clinical picture and severity of the symptoms varies widely. The neurological manifestations of scrub typhus are diverse. Meningoencephalitis is classical manifestation of scrub typhus but cerebellitis, cranial nerve palsies, plexopathy, transverse myelitis, neuroleptic malignant syndrome and Guillain-Barre syndrome are other manifestations reported in literature. The availability of literature on the neurological manifestations of scrub typhus is limited to case reports mainly. This article shows a case report of neurological manifestations of scrub typhus.

Keywords: Scrub typhus, Encephalitis, Chigger

INTRODUCTION

Scrub typhus is caused by *Orientia tsutsugamushi*, which is an obligate, intracellular gram-negative coccobacilli and is distributed throughout the Asia Pacific.

Scrub typhus is an acute febrile disease characterised by an eschar, lymphadenopathy, rash, fever, headache, myalgia and cough. Severe complications include encephalitis, interstitial pneumonia, acute respiratory distress syndrome (ARDS), myocarditis and pericarditis, cardiac arrhythmia, acute renal failure, acute hepatic failure and acute hearing loss.[1]

The disease is characterized by focal or disseminated vasculitis and perivasculitis. CNS involvement is rare but it should be considered as an important differential in the setting of known areas of scrub typhus outbreak.

Because of non-specific presentation of the disease, absence of eschar and paucity of confirmatory diagnostic tests, scrub typhus is the grossly under-diagnosed in the India.[2]

CASE REPORT

A 51-year-old male patient presented to our hospital with history of vomiting, loose stools, fever and giddiness of 1 day duration. Personal history was significant with patient having continuous consumption of alcohol approximately 180 ml per day since the past 10 years. At the time of presentation patient was conscious, restless and agitated. General physical examination revealed mild icterus and conjunctival congestion. Vitals were, temperature of 99.6 F, pulse of 110 bpm, regular and blood pressure of 170/110 mm of hg. Systemic physical examination was unremarkable.

Routine lab parameters were sent and significant findings were thrombocytopenia of 63000/cu mm, elevated ESR of 105 mm/hr, elevated liver enzymes of AST 179 U/l, ALT 39 U/l, ALP 669 U/l, TB 2.1 mg/dl, IB 1.2 mg/dl, Amylase 280 U/L. In view of the above, we initiated treatment with a provisional diagnosis of Alcoholic liver disease in alcohol withdrawal, gastroenteritis and probable bacterial meningitis. A differential diagnosis of viral haemorrhagic fevers was considered. Further investigations done for evaluation including MP (malarial parasite), IgM

leptospira, IgM dengue, HIV, HBsAg, HCV were reported as negative. USG Abdomen done showed mild hepatomegaly. Peripheral smear showed mild normocytic normochromic anaemia with severe thrombocytopenia.

Meanwhile patient continued to have remittent fever and his consciousness level worsened. Psychiatry opinion was sought and it was suggested that the patient may be suffering from alcohol withdrawal and he was started on benzodiazepines (lorazepam injection). Since the patient continued to have fever, patient was initiated on parenteral piperacillin tazobactam and azithromycin.

After a week of initial presentation patient continued to be febrile, disoriented and restless. Further work up was done with blood samples being sent for ANA, repeat Malarial parasite, widal test, scrub typhus and Weil Felix test. 2D ECHO was done to rule out infective endocarditis and it was reported as normal. In view of persisting fever and consistently elevated inflammatory markers (ESR was always above 100 mm/hr), ANA done was reported as moderately positive (26.2 units). since patient continued to be in altered sensorium, possibility of CNS tuberculosis and CNS vasculitis was considered and hence he was started on trial of steroids following which fever subsided. MRI brain done was normal and CSF analysis done was unremarkable, Mantoux test was also negative. But patient's sensorium did not improve. Repeat MP, Widal test and Weil Felix was reported as normal.

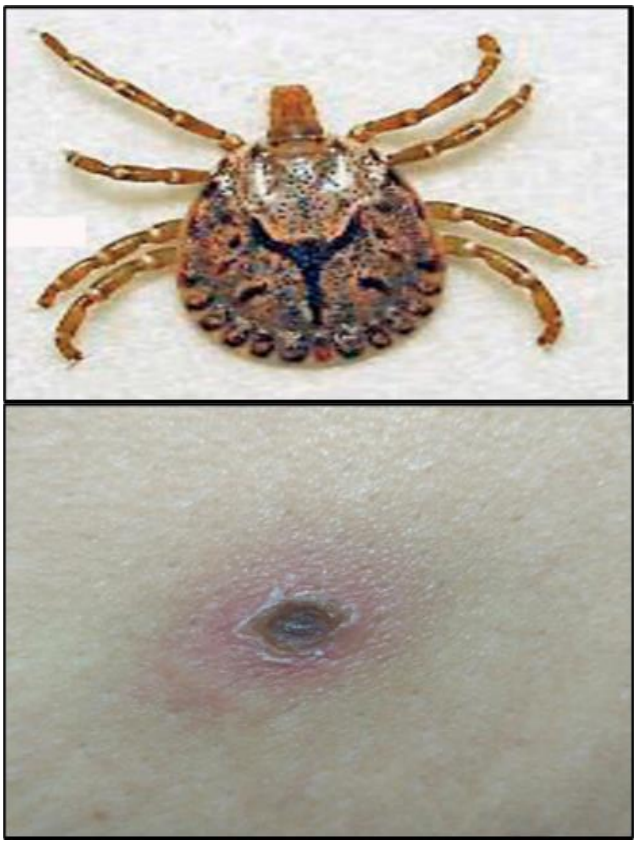

Figure 1: Scrub typhus and clinical presentation.

On day 18 of his admission the IgM scrub typhus reported as positive-(2.62 ratio; non-reactive <1). He immediately started on oral doxycycline therapy. Patient responded very well to treatment and fever spikes settled down.

He regained consciousness and was fully oriented after 3 days of doxycycline therapy. The treatment was continued for a duration of 2 weeks and patient was discharged from the hospital. At the time of discharge patient was afebrile, conscious, fully oriented with no neurological deficits. On follow up inflammatory markers had returned to normal.

DISCUSSION

Cerebellar involvement in scrub typhus has been noticed in adult and paediatric patients.[3,4] One report discovered isolated cerebellar involvement in a 21-year-old man presenting with ataxia and slurred speech.[5]

Case with both cerebral and cerebellar manifestations involved a 53-year-old female who was referred to a neurology clinic for progressive neurological deficits exhibited signs of cerebellar dysfunction and presented with lateral gaze palsy, drowsiness and slurred speech.[6]

Another report highlighted a 6-year-old girl presenting with 5-day history of fever and difficulty walking, slurring of speech as well as single episode of tonic-clonic seizure.[7]

The neurological manifestations of scrub typhus are not uncommon but are diverse.

Meningoencephalitis is classical manifestation of scrub typhus but cerebellitis, cranial nerve palsies, plexopathy, transverse myelitis, neuroleptic malignant syndrome and Guillain-Barre syndrome are other manifestations reported in literature.

Recommended treatment of scrub typhus is doxycycline (100 mg twice a day for 7-15 days), azithromycin (500 mg orally for 3 days) or chloramphenicol.

CONCLUSION

In patients with pyrexia of unknown origin presenting with neurological manifestations diagnosis of scrub typhus can be missed since it is an uncommon presentation. In countries with high prevalence of scrub typhus it is advisable for clinicians to work up for the disease routinely since delay in treatment can be disastrous.

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