Case Report

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A Case Report of Cerebellitis with Cranial Nerve Involvement in a Patient with Scrub Typhus Encephalitis

Internal Medicine Section

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ABSTRACT

Meningoencephalitis refers to inflammation involving both the meninges and brain parenchyma and represents a critical neurological emergency. The clinical picture can be diverse, often including altered sensorium, fever, seizures, and focal neurological deficits. The presence of both cerebellar and cranial nerve involvement leads to a complex presentation, causing the diagnosis to be more difficult due to the overlap with other neurological disorders. This case study aims to describe an uncommon presentation of scrub typhus-related meningoencephalitis with bilateral sixth nerve palsy and pan-cerebellar clinical presentation in a young adult. A 20-year-old female presented with high-grade fever, severe headache, vomiting, diplopia, and unsteady gait. Systemic examination revealed signs of cerebellar dysfunction, including ataxia and dysdiadochokinesia, along with involvement of the 6th cranial nerve with bilateral lateral rectus palsy and papilloedema. Laboratory workup included haematological and biochemical investigations, Cerebrospinal Fluid (CSF) analysis. Specific serological testing for tropical infections was also performed, which showed scrub typhus IgM positivity. Magnetic Resonance Imaging (MRI) showed meningeal enhancement involving the bilateral tentorium cerebelli. The patient received intravenous antibiotics, cephalosporins and tetracyclines, corticosteroids and anti-oedema measures with appropriate therapy, and neurological deficits gradually resolved over the next 10 days. In endemic areas, scrub typhus should be considered in the differential diagnosis of meningoencephalitis, particularly when accompanied by atypical features like cerebellar signs and cranial nerve palsies. Early detection and appropriate management often lead to the resolution of neurological symptoms and prevent long-term complications.

Keywords: Ataxia, Lateral rectus palsy, Leptomeningeal enhancement, Papilloedema, Rickettsial cerebellitis

CASE REPORT

A 20-year-old female presented to the emergency room with a 2-day history of high-grade fever, headache with projectile vomiting, followed by an acute onset of difficulty in walking due to unsteadiness and diplopia. She was unable to do daily activities due to a lack of coordination in performing tasks. There was no prior history of comorbidities or similar episodes.

On examination, the patient was drowsy but arousable with a Glasgow Coma Scale (GCS) of 13/15 (E3V4M6). Vitals were stable with a pulse rate of 100/min, Blood Pressure (BP) of 130/80 mm Hg, and a respiratory rate of 18/min. Neurological evaluation showed preserved higher mental functions with hypotonia in all four limbs and scanning and staccato speech. There was involvement of the 6th cranial nerve as evidenced by bilateral lateral rectus palsy. The other cranial nerve examination was normal. Meningeal signs were positive, with evident neck stiffness and a positive Brudzinski's sign. Cerebellar testing revealed bilateral impairment of the finger-nose and finger-nose-finger tests, dysdiadochokinesia, and swaying on both sides with an ataxic gait. Motor examination revealed no weakness and there was no sensory impairment on examination, with intact bowel and bladder function. Fundus examination showed bilateral papilloedema, which indicated the presence of raised intracranial pressure.

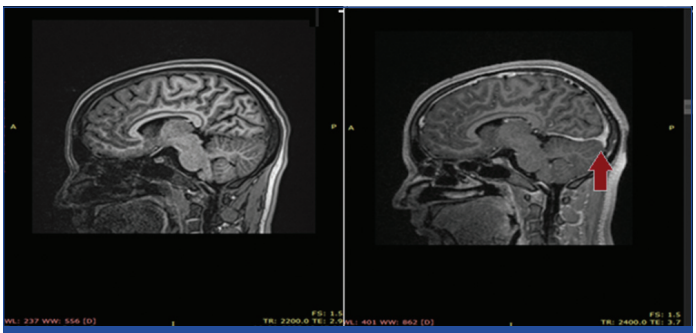
Differential diagnoses considered at this point were infectious encephalitis due to viral/bacterial/fungal or protozoal organisms, autoimmune encephalitis such as Acute Disseminated Encephalomyelitis (ADEM), Cerebrovascular Accident (CVA)-involving posterior circulation, metabolic encephalopathy, space-occupying lesions as a tumour, toxin-induced encephalopathy- as a possible differential diagnosis, even though history did not reveal any toxin exposure.

Initial laboratory investigations showed leukocytosis, elevated liver enzymes and mild anaemia. CSF analysis revealed elevated protein, normal glucose. CSF Adenosine deaminase was negative. Anti-Nuclear Antigen (ANA) was negative. Serum procalcitonin was positive. Scrub typhus IgM was significantly positive (66.97). Leptospira and dengue serologies were negative [Table/Fig-1] [1,2]. MRI brain with contrast best showed smooth leptomeningeal and pachymeningeal enhancement involving the bilateral tentorium cerebelli, consistent with cerebellitis [Table/Fig-2].

Test	Parameter	Result	Normal Values [1,2]
Complete haemogram	Haemoglobin (Hb) (g/dL)	9.7	Female: 12.1-15.1
	White blood cell (WBC) (cells/mcL)	14320	4,500-11,000
	Platelet count (cells/mcL)	220000	150,000-450,000
	Haematocrit (Hct) (%)	32	Female: 36.1-44.3
	Mean Corpuscular Volume (MCV) (fL)	80	80-100
Liver function analysis	Alanine Aminotransferase (ALT) (U/L)	375	7-56
	Aspartate Aminotransferase (AST) (U/L)	284	10-40
	Alkaline Phosphatase (ALP) (U/L)	212	44-147
	Total bilirubin (mg/dL)	1.08	0.1-1.2
	Albumin (g/dL)	3.24	3.5-5.0
	Total protein (g/dL)	6.99	6.3-7.9
Renal function analysis	Blood Urea Nitrogen (BUN) (mg/dL)	13	7-20

	Serum creatinine (mg/dL)	0.6	Female: 0.59-1.04
	Glomerular Filtration Rate (GFR) (mL/min/1.73 m^2)	132	≥90
Random blood glucose	Glucose (mg/dL)	88	70-140
Iron studies	Serum iron	55	60-180
	Total Iron Binding Capacity (TIBC)	368	250-425
	Transferrin saturation	15	15-50
	Ferritin	9	11-306
Serology	Dengue NS1 and IgM	Negative	
	Leptospira IgM/ IgG	Negative	
	Scrub typhus IgM	Positive	
	HbsAg/HCV/HIV	Negative	
	Serum procalcitonin (ng/mL)	0.22	
CSF analysis	Albumin	25.36	10 -30
	Glucose	77	40-70
	Lactate Dehydrogenase (LDH)	20	<200
	Protein	54.3	15-40 mg/dL
	Malignant cells	Negative	
	Red Blood Cell (RBC)	No cells	
	White Blood Cell (WBC)	30cells/ mm3	
	CBNAAT	Not detected	
Autoimmune profile	Anti-Nuclear Antigen (ANA)	Negative	

[Table/Fig-1]: Initial laboratory parameters [1,2].



[Table/Fig-2]: Sagittal T1-weighted: a) Plain; b) Contrast-enhanced MRI brain demonstrating leptomeningeal and pachymeningeal enhancement (marked with red arrow) over bilateral tentorium cerebelli.

A diagnosis of acute meningoencephalitis with pancerebellar features with bilateral sixth cranial nerve palsy was made, secondary to scrub typhus. The patient also had iron deficiency anaemia.

Treatment included intravenous ceftriaxone 2 g BD for 10 days, doxycycline 100 mg i.v. BD for seven days, dexamethasone 8 mg i.v. BD, and mannitol 100 mL i.v. BD. Supportive medications included proton pump inhibitors as pantoprazole and hepatoprotectives as ursodeoxycholic acid.

The patient showed gradual neurological recovery with resolution of cerebellar signs as ataxia, dysmetria and diplopia, over 10 days and at the time of discharge patient was able to walk without support, carry out activities as eating, handling objects by self and was advised outpatient follow-up.

On follow-up after a month, the patient had complete neurological recovery with no residual symptoms.

DISCUSSION

Scrub typhus is a tropical illness caused by Orientia tsutsugamushi, which is an obligate intracellular bacterium present in mites. The bite of larval trombiculid mites is the mode of transmission to humans, referred to as chiggers. This disease is prevalent in the "tsutsugamushi triangle," which spans across South and Southeast Asia, Northern Australia, and up to the Russian Far East. In India, scrub typhus has become increasingly prevalent, especially in rural and suburban regions, where seasonal outbreaks are frequently observed during and after the monsoon [3,4].

At the time of onset, scrub typhus infection presents with non-specific findings, usually as fever, malaise, myalgia, headache, and gastrointestinal disturbances. A characteristic eschar may be present at the site of the chigger bite, but it is not necessary for diagnosis. As the disease progresses, it can lead to multiple organ system involvement, including the liver, lungs, kidneys, and Central Nervous System (CNS) [5].

Neurological complications are less commonly reported than other systemic effects but are clinically significant and include variable manifestations as meningitis, meningo-encephalitis, seizures, altered sensorium, cerebellar dysfunction, and cranial nerve involvement. These complications arise from direct microbial invasion, immune-mediated responses, or vasculitis affecting cerebral blood flow, causing localised ischaemia or oedema. Among these, cerebellitis- a condition characterised by inflammation of the cerebellum- is an unusual and relatively rare manifestation in scrub typhus infections [6]. Patients with cerebellar involvement typically present with symptoms such as imbalance, difficulty in speech, intentional tremors, and difficulty with coordinated movements. In some rare instances, involvement of the cranial nerves, particularly the sixth (abducens) and fourth (trochlear) nerves, may occur, often presenting as diplopia or other ocular motor dysfunctions [7,8]. These signs suggest either direct involvement of the brainstem or the effects of increased intracranial pressure due to inflammation and oedema [9].

It is crucial to recognise neurological involvement early in the course of disease, which requires a high index of suspicion at the time of presentation, particularly in scrub typhus endemic areas. MRI of the brain is instrumental in detecting cerebellar inflammation and meningeal enhancement. Additionally, CSF analysis often reveals findings consistent with aseptic meningitis and serological testing- especially IgM ELISA for scrub typhus- serves as a key diagnostic modality [10]. While meningeal irritation and encephalitic features are the most commonly observed CNS presentations, the presence of both cerebellitis and cranial nerve palsy remains a less frequent occurrence and represents a relatively rare neurological phenotype of this infection.

This report describes an unusual case involving a young adult female with acute cerebellitis and bilateral involvement of the sixth cranial nerve in the setting of scrub typhus encephalitis. The clinical course, as indicated by neuroimaging and serological findings, emphasises the need for suspicion of scrub typhus infection in the differential diagnosis of patients with cerebellar signs, unexplained encephalopathy and other neurological manifestations in endemic regions as the suburban and rural areas of Chengalpattu district, Tamil Nadu. Early diagnosis and appropriate treatment with antibiotics such as doxycycline are necessary to alleviate complications and for favourable outcomes [11].

The underlying mechanisms of neurological impairment in scrub typhus are multifactorial. Orientia tsutsugamushi may invade the CNS directly or cause inflammation through perivascular infiltration, endothelial damage, and cytokine-mediated alterations in vascular permeability. These processes contribute to cerebral oedema and focal neurological dysfunction. The cerebellum, due to its dense vascular network and anatomical sensitivity to microvascular injury, is particularly susceptible to inflammatory damage in certain cases [12]. Cerebellar involvement exhibits classical signs such as ataxia, intention tremor, dysmetria, and scanning speech. In the case discussed, prominent signs like bilateral dysdiadochokinesia and truncal instability indicated diffuse cerebellar dysfunction.

Additionally, the presence of bilateral sixth cranial nerve palsy pointed toward either direct involvement of the brainstem or a secondary effect of raised intracranial pressure. This was further supported by the detection of bilateral papilledema on fundoscopic examination.

Imaging modalities, especially contrast-enhanced MRI, serve as a vital tool in evaluating such cases. MRI often reveals leptomeningeal or pachymeningeal enhancement and signal changes in the cerebellum or brainstem. The smooth enhancement pattern involving the tentorium cerebelli in this patient was consistent with cerebellitis secondary to infection [13]. Serological confirmation is critical for diagnosis in endemic regions. IgM ELISA for scrub typhus is widely used and highly sensitive. Due to the overlapping of clinical features, Exclusion of other potential infectious causes such as leptospirosis, dengue, or tuberculosis is necessary [14].

Early initiation of empirical antibiotic therapy is indispensable for management. Doxycycline remains the drug of choice for treating scrub typhus. In severe cases with CNS involvement, adjunctive corticosteroids are employed to modulate inflammatory responses and mitigate cerebral oedema. Osmotherapy with agents like mannitol is helpful in controlling intracranial hypertension [15]. Majumdar S et al., 2025 contributed to the expanding clinical spectrum of scrub typhus by documenting a rare presentation with cerebellar signs and bilateral cranial nerve palsies. It emphasises the importance of including scrub typhus in the differential diagnoses of acute cerebellar syndromes [16].

This case shows the importance of identifying rare neurological complications of scrub typhus, particularly cerebellitis with cranial nerve involvement, especially in endemic regions. Early diagnosis is facilitated by a high index of suspicion, especially during post-monsoon periods. MRI acts as a tool in finding cerebellar inflammation and meningeal involvement. Timely treatment significantly shifts outcomes, often leading to complete recovery.

CONCLUSION(S)

Scrub typhus, though usually present as a febrile illness, can manifest with severe neurological complications, including cerebellitis and cranial nerve palsies. This case report highlights the rare occurrence of cerebellar involvement with bilateral sixth nerve palsy, diagnosis being established by radio-imaging and serological evidence. Early recognition, prompt antibiotic therapy, and supportive care led to favourable outcomes. Physicians in endemic areas must maintain high suspicion for scrub typhus in patients presenting with unexplained cerebellar signs and encephalopathy. MRI and CSF analysis, along with scrub serology, are crucial for accurate diagnosis and effective treatment initiation.

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