International Journal of Contemporary Pediatrics
Sinha R et al. Int J Contemp Pediatr. 2026 Aug;13(8):1545-1547
https://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20262435

Case Report

Novel electroencephalographic delta brush pattern in typhoid encephalitis

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Received: 07 June 2026
Revised: 09 July 2026
Accepted: 14 July 2026

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ABSTRACT

Extreme delta brush is a rare interictal electroencephalography (EEG) pattern first described in anti-N-methyl-D-aspartate receptor (NMDA) receptor encephalitis and initially considered pathognomonic for this autoimmune subtype. It has since been reported, rarely, in other forms of encephalitis. Typhoid encephalitis commonly presents with acute confusion and behavioral changes and may also cause severe neurologic complications, including seizures, coma, and cerebral edema. Typhoid toxins may increase blood–brain barrier permeability and contribute to neurologic dysfunction. We report this rare EEG finding in a 13-year-old girl with typhoid encephalitis. Our case highlights the diagnostic value of EEG in unexplained encephalopathy or suspected encephalitis. Although extreme delta brush is specific to anti-NMDAR encephalitis, its presence should prompt evaluation for other autoimmune or infectious encephalitis when the anti-NMDAR antibodies are negative.

Keywords: Encephalitis, Delta brush, NMDA, Typhoid, EEG, Autoimmune

INTRODUCTION

Extreme delta brush is a rare interictal electroencephalography (EEG) pattern first described in anti-N-methyl-D-aspartate receptor (NMDA) receptor encephalitis and initially considered pathognomonic for this autoimmune subtype. It has since been reported, rarely, in other forms of encephalitis. Typhoid encephalitis commonly presents with acute confusion and behavioral changes and may also cause severe neurologic complications, including seizures, coma, and cerebral edema. Typhoid toxins may increase blood–brain barrier permeability and contribute to neurologic dysfunction. Typhoid fever is a bacterial infection caused by *Salmonella enterica* serovar typhi (*Salmonella typhi*). It spreads by the fecal–oral route, usually through contaminated food or water. Typical features include acute fever, headache, abdominal pain, diarrhea or constipation, and malaise. Severe complications include intestinal perforation, hepatitis, pneumonia, and tissue abscesses.[1] Neurologic involvement, most often acute encephalopathy or meningitis, has also been reported. Other manifestations include acute neuropsychiatric symptoms, spasticity, clonus, ataxia, aphasia, and cerebritis.[2] Although confusion is common, severe neurologic complications such as seizures, coma, and cerebral edema may occur. Typhoid toxins may increase blood–brain barrier permeability and contribute to neurologic dysfunction. Acute encephalitis syndrome is increasingly recognized. Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis is the best-known form, and its characteristic electroencephalography (EEG) pattern is extreme delta brush (EDB). This rare EEG pattern has recently also been described in other forms of encephalitis. We report this finding in a 13-year-old girl with typhoid encephalitis.

CASE REPORT

A developmentally normal 13-year-old girl presented with high-grade fever for 5 days, followed by reduced speech

output and behavioral changes, including inappropriate laughter and labile mood, for 3 days. She also had excessive sweating and generalized pallor for 3 days. There was no history of seizures, vomiting, headache, focal weakness, altered sleep–wake cycle, rash, drug intake, animal bite, neck pain, photophobia, recent travel or loose stools. She was age-appropriately immunized and had good scholastic performance. On examination, her height was 156 cm (0 to +1 SD), weight 50 kg (0 to -1 SD), and body mass index, BMI 20 (0 to +1 SD). Her pulse was 90/min, blood pressure 110/70 mm Hg in the right arm while supine with no significant postural drop, respiratory rate 18/min, SpO_2 96% on room air, and temperature 100°F. She had pallor but no icterus, cyanosis, clubbing, or lymphadenopathy. Excessive sweating was the only feature suggestive of dysautonomia. Neurologic examination showed a pediatric mini-mental state examination score of 28/35. She was conscious but disoriented to time and place, with bouts of inappropriate laughter and dysarthria. There was no cranial nerve deficit, and fundus examination was normal. Motor and sensory examination was normal, with no cerebellar signs. There was no neck stiffness or involuntary movement. No bladder or bowel involvement was noted. She had mild hepatomegaly; respiratory and cardiovascular examinations were otherwise normal. She was admitted with acute encephalitis syndrome, with infectious, demyelinating, autoimmune, and neurometabolic causes considered. Laboratory evaluation showed hemoglobin 9.0 g/dl, total leukocyte counts 4600/cmm (polymorphs 74%, lymphocytes 20%), platelet count 1.0 lakh/cmm, and elevated liver enzymes (total serum bilirubin 1.0 mg/dl; serum glutamic oxaloacetate transaminase (SGOT)/serum glutamic pyruvic transaminase (SGPT)/alkaline phosphatase (ALP) 238 IU/l/136 IU/l/165 IU/l. Electrolytes and renal function tests were normal. Serologic tests for dengue, malaria, and scrub typhus were negative. Thyroid profile, anti-thyroid perioxidase (TPO) antibodies, and anti nulear antibody (ANA) were normal. Chest X-ray, echocardiography, and electrocardiography were normal. Abdominal ultrasonography showed mild hepatomegaly with a liver span of 15 cm. Contrast-enhanced magnetic resonance imaging (MRI) of the brain and spine was normal. The interictal sleep EEG with longitudinal bipolar montage (sweep 30 mm/s; sensitivity 75 µV/cm; bandpass 0.5–70 Hz) showed high-frequency beta activity superimposed on periodic blunt delta waves, especially in the frontocentral and frontopolar leads (Figure 1). Her arterial lactate, ammonia, glucose and ketones were in the normal range. The human immunodeficiency virus (HIV), hepatitis B (HBsAg) and hepatitis C serology were negative. The workup for tuberculosis was negative. Cerebrospinal fluid (CSF) showed no cells, 31 mg/dl protein and 64 mg/dl sugar (random blood sugar 96 mg/dl), sterile CSF culture, and negative CSF viral panel. CSF titres of anti N-methyl-D-aspartate (NMDA) receptor antibody and serum antibodies against alpha-amino-3-hydroxy-5-methyl-4-isoxazole (AMPA), gamma-aminobutyric acid type A and B (GABA), contactin-associated protein-like 2 (CASPR2), glutamic acid decarboxylase (GAD), and dipeptidyl-peptidase-like protein (DPPX) were negative. Blood culture grew *Salmonella typhi*, which was sensitive to ceftriaxone and meropenem. She was initially started on intravenous ceftriaxone, acyclovir, and doxycycline. Immunotherapy was being considered pending the initial results. She improved with antibiotics, and acyclovir was stopped after the viral panel was negative. Her sensorium and behavioral symptoms improved after 7 days of antibiotics, and EEG normalized by day 10 of admission. Antibiotics were continued for 14 days. She recovered fully before discharge and is currently doing well academically.

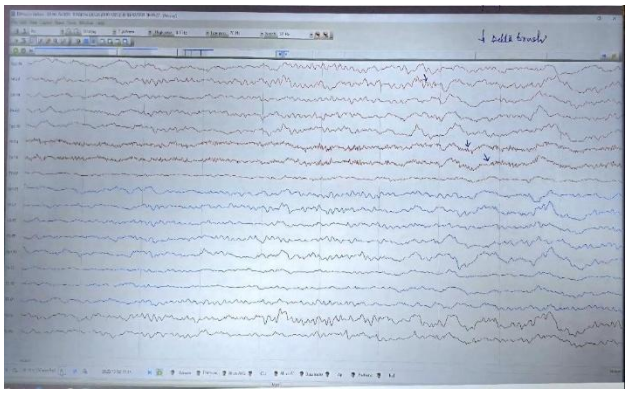

Figure 1: Interictal sleep EEG longitudinal bipolar montage; sweep 30 mm/s; sensitivity 75 microvolt/cm; bandpass 0.5-70 Hz shows the typical pattern of high frequency beta activity superimposed on periodic blunt delta waves (marked with arrow) especially in frontocentral and frontopolar leads.

DISCUSSION

Extreme delta brush is reported in 30%–60% of anti-NMDAR encephalitis.[3,4] Although initially considered pathognomonic for anti-NMDAR encephalitis, it has since been rarely described in other encephalitic syndromes.[5,6] To our knowledge, this is the first reported case of typhoid encephalitis associated with extreme delta brush on EEG. Although headache and confusion are common in typhoid and were reported in more than 50% of patients in some historical cohorts, severe neurologic manifestations are uncommon.[7,8] When present, they may include encephalitis, myelitis, psychosis, cerebellitis, and parkinsonism. The mechanism underlying neurologic involvement in typhoid fever remains unclear. Host susceptibility or environmental factors may contribute to the development of neurologic complications during severe systemic infection. EEG is a valuable adjunct to the clinical neurologic examination because it can detect subtle or subclinical cerebral dysfunction and enables serial monitoring of brain activity. Continuous EEG, combined with quantitative analysis and machine learning, may help identify evolving changes in real time before clinical signs appear and may aid assessment of response to treatment. EEG is rarely pathognomonic in

encephalopathy or encephalitis, but certain patterns may suggest specific pathophysiology when interpreted in context (e.g., lateralised periodic discharges in herpes simplex virus (HSV-1), generalised periodic discharges in sporadic Creutzfeldt-Jakob disease, and extreme delta brush in anti-N-methyl-D-aspartate receptor encephalitis). In practice, patients with subacute encephalopathy, neurologic symptoms, and psychiatric features require exclusion of reversible causes such as neoplastic, infective, toxic, prion, and metabolic disorders. EEG is often part of this evaluation but may be normal early in autoimmune encephalitis. Most EEG abnormalities, such as slowing or sharp waves, are nonspecific, except for the occasional extreme delta brush pattern in anti-NMDA receptor encephalitis.[9] Delta brush variance consists of generalized delta activity with superimposed fast spikes. It may represent a variant of extreme delta brush during the florid phase of disease. Cortex–subcortical interactions may contribute to the transition between delta brush variance and extreme delta brush.[10] In routine practice, typhoid encephalitis may present with mild CSF pleocytosis, nonspecific MRI changes, and diffuse EEG slowing. Although extreme delta brush is mostly specific to anti-NMDAR encephalitis, however when the serum or CSF anti-NMDAR antibody is negative then it should prompt evaluation for other autoimmune or infectious encephalitis. EEG remains useful in evaluating encephalopathy and encephalitis. Although rarely pathognomonic, it can help estimate cerebral dysfunction and support neuro prognostication.

CONCLUSION

Our case highlights the diagnostic value of EEG in unexplained encephalopathy or suspected encephalitis. Although extreme delta brush is specific to anti-NMDAR encephalitis, its presence should prompt evaluation for other autoimmune or infectious encephalitis when the anti-NMDAR antibodies are negative.

ACKNOWLEDGEMENTS

The authors acknowledge the contribution of all nursing and paramedical staff of Dept of Pediatrics, Base Hospital, Delhi Cantt.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Sinha R, Kumar P, Selvakumar N, Sharma D, Gupta S, Popli H. Novel electroencephalographic delta brush pattern in typhoid encephalitis. Int J Contemp Pediatr 2026;13:1545-7.