



Acute Disseminated Encephalomyelitis Following an Attack of Falciparum Malaria

Dr. Syed Khaja Waheed Hussain ^{1*}, Dr. K. Vasudev ², Dr. K. Sravan ³

1. Postgraduate, Department of Paediatrics, Kakatiya Medical College, Hanumakonda.
2. Professor, Department of Paediatrics, Kakatiya Medical College, Hanumakonda.
3. Assistant Professor, Department of Paediatrics, Kakatiya Medical College, Hanumakonda.

Corresponding Author: Dr. Syed Khaja Waheed Hussain, Postgraduate, Department of Paediatrics, Kakatiya Medical College, Hanumakonda.

Copy Right: © 2022, Dr. Syed Khaja Waheed Hussain, This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Received Date: September 14, 2022

Published Date: October 01, 2022

Introduction

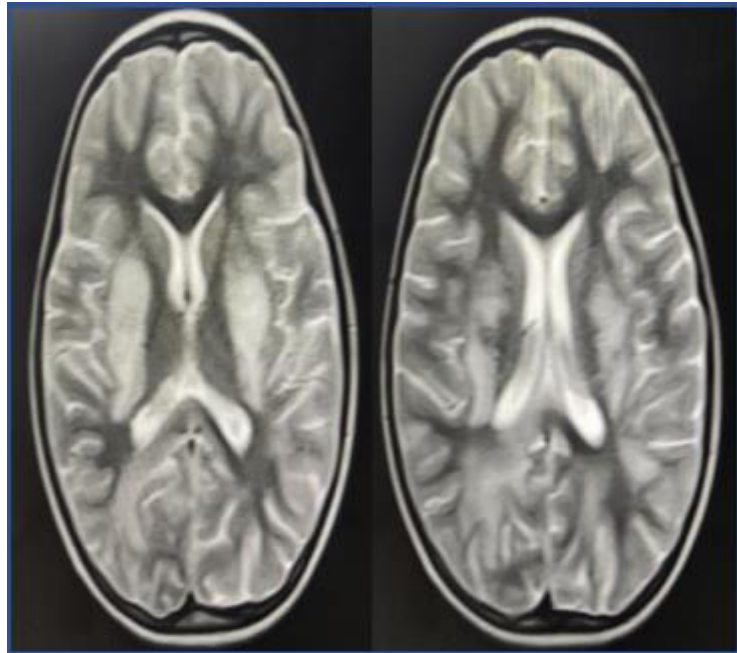
Acute disseminated encephalomyelitis (ADEM) is an immune mediated disease of the central nervous system (CNS) that produces inflammatory damage at multiple locations in the brain and spinal cord, particularly in the white matter. ADEM as a neurological manifestation after complete recovery from severe malaria, particularly caused by *Plasmodium falciparum*, is rare yet reported.

Case Details

A six-year-old male child presented with ten days of history of difficulty in walking, decreased vision in both eyes (painful and progressive). At admission, the child was in altered sensorium. The next day after gaining sensorium, examination revealed that he had ataxic gait (swaying to right), dysdiadokokinesia, past pointing and mild global hypotonia. Eye exam revealed decreased acuity in both eyes, left > right and bilateral Relative Afferent Pupillary Defect (RAPD), fundus exam showed bilateral papillitis. Child was admitted one month prior for fever, pain abdomen and persistent vomiting and diagnosed with *Plasmodium falciparum* malaria on blood smear. He was given intravenous artesunate and treatment completed with oral Artemisinin Combined Therapy (ACT) and single dose primaquine. Child had completely recovered and discharged with no apparent complications of malaria. For ADEM the child was given intravenous methylprednisolone for five days. Child improved with normal gait and vision returning to normal in both eyes. The child was put on tapering dose of oral steroids and discharged with complete recovery.

Investigations

Smear for MP negative, CSF (oligoclonal bands) – normal, Serum NMO antibodies negative. MRI brain - symmetrical bilateral hyperintensities in capsuloganglionic regions, parieto-occipital region, basal ganglia and mesial temporal lobe with asymmetrical involvement of splenium of corpus callosum and adjacent deep white matter. Cortex and subcortical U fibres were not spared. Right thalamus, right cerebellar hemisphere in anterior portion were involved. Spinal cord was normal to extent visualised. MRS - elevated Cho, reduced NAA, no lactate peak, reduced NAA/Cr ratio and elevated Cho/Cr ratio.



Discussion

ADEM is a rare complication of severe malaria. Most cases are self-limited and benefit from steroid therapy. In patients with recent history of malaria that present with neuropsychiatric symptoms, ADEM should be one of the differential diagnoses. Repeat MRI brain on follow up showed lesions had partially resolved and hyperintensities were restricted to peritrigonal areas, sple.

References

1. Yun Jin Lee. Acute disseminated encephalomyelitis in children Korean J Pediatr. 2011 Jun; 54(6): 234– 240.
2. Agrawal A, Goyal S. Acute demyelinating encephalomyelitis in a child following malaria. Indian Pediatr 2012; 49:922-3. 4.
3. van der Wal G, Verhagen WI, Dofferhoff AS. Neurological complications following Plasmodium falciparum infection. Neth J Med 2005; 63:180-3.
4. Schnorf H, Diserens K, Schnyder H, et al. Corticosteroid-responsive post malaria encephalopathy Arch Neurol 1998; 55:417-20.