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Acute Disseminated Encephalomyelitis Following Falciparum Malaria: A Rare Case Report

Authors
Dr Preetam Ghosal[1*], Dr Shantisena Mishra[2]
[1]Junior Resident, [2]Associate Professor
Department of Pediatrics, MKCG Medical College, Bramhapur, Odisha
*Corresponding Author
Dr Preetam Ghosal
Room no- 14, PG Hostel no 2 MKCG Medical College, Bramhapur, Ganjam, Odisha, 760004, India
Email: *preetam.mkcg.ghoshal@gmail.com*, Phone no- 8895121819

Abstract
Acute disseminated encephalomyelitis is an autoimmune, demyelinating disease of CNS caused by viral infections, vaccinations, some bacterial infections and rarely by malarial infection too. Early diagnosis and prompt treatment can reduce the neurological complications and hence morbidity and mortality.
Keywords: *ADEM, Falciparum malaria, Post malarial neurological syndrome.*

Introduction

ADEM is an immune mediated, inflammatory and demyelinating disease of central nervous system with diffuse neurological signs and multifocal white matter lesions on neuroimaging[1,2]. An autoimmune reaction of T-cells against myelin basic protein, trigerred by viral infection or vaccination has been suggested as a possible pathogenetic mechanism[3]. ADEM is quite rare after malarial infection[4]. Few case reports of ADEM following P. Falciparum infection in children are present till date. We report a case of complicated malaria developing ADEM during its recovery phase.

Case Report

A 3 year old female child was admitted to our hospital with chief complaints of high grade fever for 5 days, lethargy and generalised weakness for 5 days, black coloured urination for 3 days and 3 episodes of generalised tonic clonic seizure on day 4 of fever. There was no H/O similar illness before. No H/O previous blood transfusion or contact with Tuberculosis.

On examination the child was obtunded and drowsy with GCS being 10/15, severely pale and prostrated. There was mild icterus but no cyanosis, clubbing, lymphadenopathy and edema. Cardiovascular and Respiratory system was normal except for tachycardia and tachypnea. Abdominal examination revealed hepatosplenomegaly. Neurological examination showed bilateral plantar extensor response, deep tendon reflexes were brisk. Cranial nerves were intact, no meningeal or cerebellar signs and bilateral pupils were normal in size, equal and reacting to light.

Investigations revealed Hb- 5.3 g%, WBC - 12,300, TPC-1.9 lakhs. Plasma glucose, serum electrolytes, RFT were normal.LFT was mildly derranged. Sickling test was negative. Rapid malaria antigen test was positive for P.falciparum and slide microscopy was also positive (2+) for P. falcipaum. CSF study was normal and fundoscopy revealed B/L macular edema. The child was negative for IgM Dengue and IgM Scrub typhus.

Child was shifted to PICU and treated with iv fluids, artesunate, cefotaxime, phenytoin injections. Packed RBC was transfused @ 10ml/kg. 3% NS infusion was given for 48 hours. Gradually the fever subsided and the clinical and biochemical parameters improved but there was no improvement in sensorium for which the child was subjected to neuroimaging which showed hyperintense signals at B/L parieto- occipital region on FLAIR T2 weighted MRI. The child was given i.v methyprednisolone @ 30mg/kg for 5 days following which her GCS improved drastically. The patient was shifted to general ward and discharged after 3 days with oral prednisolone @ 1mg/kg and physiotheraphy advice.

Fig 1 – showing cerebral malaria Child with ADEM Admitted in PICU

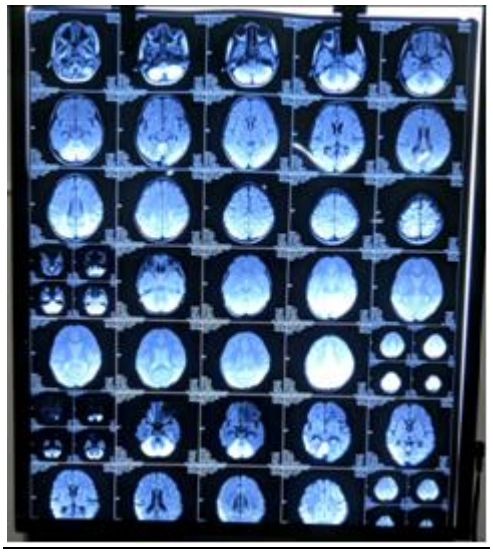

Fig 2 – MRI Picture of same Child showing hyper intense signals with T2 FLAIR

Discussion

ADEM is characterised by abrupt onset of multifocal neurological dysfunction with signs of encephalopathy[5]. It is common in childhood and young adults following infections and vaccinations[5]. ADEM following cerebral malaria has been reported in literature as a part of Post Malaria Neurological Syndrome (PMNS)[6]. PMNS was first described by Nguyen et.al as symptomatic malaria infection whose parasites have cleared from peripheral blood fully but developing neurological or psychiatric symptoms within 2 months of acute illness[7]. Schnorf et.al classified PMNS based on severity as i) Mild and localised encephalopathy ,ii) Diffuse and mild encephalopathy, iii) Severe corticosteroid responsive encephalopathy characterised by motor aphasia, cranial nerve palsy, seizures, cerebellar ataxia, optic neuritis closely resembling ADEM[8]. Reccurence or new appearance of neurological complications in a known case of severe malaria should arouse the suspicion of ADEM warranting specific investigations and definite treatment[9].

Conclusion

ADEM can be regarded as a neurological complication of severe falciparum malaria[10]. The latency to neurological involvement and response to steroid treatment in our patient supports an

immunological mechanism. This case report emphasizes the importance of recognizing P. falciparum as an etiological cause of ADEM especially in tropical countries.

Conflict of interest none.

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