Clinical history: Malaria is one of the most common diseases in the tropical countries. Cerebral malaria is usually a diffuse symmetric encephalopathy with focal signs being unusual.

Methods: We present a three-year old girl lapsing into unconsciousness following a seizure while undergoing treatment for malaria. Imaging revealed a large heterogenous density, left hemispheric acute subdural haematoma with brain herniation. Investigations revealed anaemia, thrombocytopenia and positive peripheral blood smear for falciparum malaria.

Results: Treatment involved surgical evacuation of the clot and the associated subdural empyema, intravenous quinine and antibiotics.

Conclusion: This is the second case report of spontaneous subdural empyema in complicated falciparum malaria and highlights a rare but surgically manageable complication.

Key words Cerebral malaria – falciparum – subdural empyema – subdural haematoma

Malaria is one of the most common diseases in tropical countries. It has a myriad of presentations and complications with cerebral malaria being one its most devastating and lethal sequelae. Cerebral malaria can lead to a variety of complications including hemiplegia, convulsions, delirium and death\(^1,2\). Spontaneous subdural empyema in malaria is very rare with only one case being reported in available literature. We are presenting a rare case of spontaneous subdural empyema and haematoma in a case of falciparum malaria.

A 3-year old girl presented to the emergency department of our hospital in altered sensorium, following a trivial fall during a seizure. She was undergoing treatment for malaria from the past 10 days. Examination revealed afebrile, toxic and unconscious child with a Glasgow Coma Scale of 6, pupils were unequal, not reacting to light with right hemiparesis. A noncontrast computed tomographic (CT) scan of head revealed a heterogenous left hemispheric subdural haematoma with mass effect and midline shift.

There was subfalcine and transtentorial herniation and basal cisterns were effaced (Fig. 1). Laboratory investigations revealed a haemoglobin of 3.6 g/dl prothrombin time of 14/16 sec and thrombocytopenia.
Electrolytes were normal. Peripheral smear was positive for falciparum malarial parasite. The patient was taken up for emergency surgery after correction of haematological and coagulation parameters. A frontotemporoparietal osteoplastic flap craniotomy was performed. On opening the dura, there was a thick clot encased in a membrane which also contained purulent fluid. The subarachnoid cerebrospinal fluid (CSF) appeared purulent. The purulent fluid and the clot were removed and the membrane was biopsied. A left frontal lobectomy was performed since the brain was bulging. Duraplasty was performed using a pericranial graft and the bone flap was not replaced. Analysis of the purulent fluid revealed pus cells with no organism on gram stain and Ziehl Nielson stain. Pyogenic, fungal and tubercular cultures were sterile. The histopathology of the brain and membrane did not reveal any evidence of malarial parasite but revealed congested blood vessels. Postoperatively, the patient was electively ventilated and administered intravenous quinine and antibiotics (ceftriaxone, amikacin and metronidazole). Postoperation scan revealed postoperative changes, multiple infarcts and decreased mass effect. The child became afebrile and started localising to painful stimuli. She however had right sided hemiplegia. Unfortunately, she developed chest infection, bacterial meningitis and septicemia two weeks after surgery to which she succumbed.

Discussion

Malaria is caused by any of the four species (Plasmodium vivax, P. ovale, P. malariae and P. falciparum) of the genus Plasmodium, of which the falciparum being the most dangerous. It generally occurs 5–15 days after infection and is characterised by febrile paroxysms, anaemia and splenic enlargement. Severe falciparum malaria can be associated with cerebral complications, hypoglycemia, lactic acidosis, non cardiogenic pulmonary edema, renal impairment and hematological abnormalities such as blackwater fever and coagulation anomalies. Cerebral malaria is defined as any CNS disturbances in a malarial infection.

The diagnosis is supported by finding cerebral capillaries and venules-packed with parasitised red blood cells (RBC). These findings may be absent if patient dies several days after treatment as was the case with our patient. Cerebral malaria is usually a diffuse symmetric encephalopathy with focal signs being unusual. Convulsions are common particularly in children. Ophthalmoscopy may reveal retinal haemorrhages. Signs of meningeal irritations are lacking and corneal reflexes are usually preserved. Subarachnoid hemorrhage can occur and mimics meningitis. CSF pressures are raised, with evidence of brain edema on radiological imaging and post-mortem, the most likely cause of raised ICP being rise in cerebral blood flow. The mortality rate of cerebral malaria varies. Among the patients who survive, neurologic outcomes vary, the most frequent sequelae being paresis and ataxia. Other deficits include behavioural problems, hearing defects, visual field defects, aphasia and dysarthria. Brewster et al noted a 16% mortality rate, with 11% incidence of residual neurologic sequelae with hemiplegia being the commonest.
Molyneux et al\textsuperscript{10} noted 9% of children had residual sequelae and a mortality of 15%, while Lemercier et al\textsuperscript{11} in a study in 235 children observed a mortality of 9% and residual sequelae in 5%.

Focal lesions in malaria are rare with subdural empyema being extremely uncommon. The usual causes of subdural empyema (SDE) being meningitis secondary to paranasal sinusitis/otitis media, postoperative or secondary to trauma. There has been only one report of SDE in malaria recorded in available literature by Smythe & Cairns\textsuperscript{12} who described a case of subdural abscess complicated falciparum malaria which was treated with repeated aspirations, antimalarials and antibiotics. This is the second case report of spontaneous subdural empyema complicating falciparum malaria in which the primary pathology was a spontaneous SDE followed later by an associated bleed which was probably due to the associated coagulation abnormalities.

Conclusion

Cerebral malaria in childhood can have lethal and devastating implications and needs intensive management. This is the second case report of a spontaneous subdural empyema complicating cerebral malaria that highlights a devastating but surgically manageable complication and stresses the necessity of a high index of suspicion along with adequate imaging for diagnosis especially in atypical presentations.

References


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