Falciparum malaria induced retrobulbar neuritis

Sir,

Commonly seen ophthalmic complications in falciparum malaria include retinal haemorrhages and oedema, papilloedema, disc pallor and vitreous haemorrhage. We describe here a rare complication of falciparum malaria leading to sudden blindness, thus warranting its clinical awareness for early diagnosis and intervention.

A 25-year-old lady presented with high-grade fever and malaise of 3 days duration. On examination, she was febrile, normotensive, had mild pallor with other systemic examination being normal.

The haemogram showed haemoglobin of 9.6 gm%, total leucocyte count of 8.5x10^9/L and a platelet count of 23x10^9/L. Total bilirubin was 1.2 mg/dl and direct bilirubin was 0.5 mg/dl. The aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase were 48, 36 and 43 IU/L respectively. The serum creatinine was 0.8 mg/dl. Thin blood smear showed falciparum malarial parasites (gametocytes and schizonts) with parasitic index of 8%. She received intravenous quinine 10 mg/kg dose, 3 doses on first day. The next morning (day 2), patient complained of sudden painless loss of vision in both eyes. Ophthalmic examination showed dilated pupils with absence of reaction to light. Visual acuity was reduced to light perception in all 4 quadrants bilaterally. Fundus examination was completely normal and a diagnosis of retrobulbar neuritis was made.

Although rare, Quinine can cause toxic amblyopia and blindness. Hence it was stopped and she was given injection methylprednisolone 1gm/day for 5 days. She also received IV methylprednisolone 1gm/day for 3 days. Her falciparum parasitic index was 2% on day-2. Rest of the haemogram was normal. On day-3, her vision improved to perception of hand movements with a sluggish reaction of the pupils to light. The peripheral smear was negative for malarial parasites with no clinical deterioration otherwise. On day-4, vision improved to finger counting at 2 feet with persistence of abnormal pupil reaction. Repeat smears were negative for malarial parasites. On day-7, the patient was discharged having completed her treatment for malaria, with further improvement in vision to finger counting at 6 feet. Acuity on Snellen’s chart was 6/60 bilaterally. She was advised tapering dose of oral steroids for 6 weeks.

Follow-up ophthalmic assessment at 8 weeks showed considerable improvement in vision to 6/18 on Snellen’s chart in both eyes. Colour vision was normal bilaterally. Fundus showed mild disc pallor with venous sheathing bilaterally. Perimetry showed loss of vision in peripheral fields with good central vision. These findings reconfirmed the diagnosis of retro-bulbar neuritis.

Sudden visual loss in falciparum malaria is commonly caused by vitreous haemorrhages or cortical blindness. Retrobulbar neuritis is a rare complication of falciparum malaria causing blindness and has been described only once in literature. It shows a normal fundus on early examination, which helps to differentiate from loss of vision due to vitreous haemorrhages. An afferent pupillary defect rules out cortical blindness where pupillary reaction to light is expected to be normal. A normal fundus in this case also ruled out quinine amblyopia as a possible cause of blindness (observation of retinal oedema, macular cherry red spots, arteriolar narrowing followed later by disc pallor). It is important to be aware of retrobulbar neuritis complicating falciparum infection as early diagnosis and steroid treatment may help reverse the vision loss considerably as seen in our case and reported cases.

Although the precise mechanism is not known, retrobulbar neuritis possibly results from tissue hypoxia due to RBCs adhering to the vascular endothelium via membrane proteobactenches called “knobs”. These “knobs” contain a high-molecular weight, adhesive protein (PfEMP1) that mediates attachment to receptors on endothelium causing obstruction of vessels including ophthalmic vessels - an event termed as cytoadherence. Infected RBCs also adhere to non-infected red cells to form “rosettes”. These two events cause obstruction of microcirculatory flow and tissue hypoxia. Tissue hypoxia in turn causes loss of capillary endothelial integrity and leakage of fluid and protein into the interstitial spaces causing further compression of blood vessels, setting up a vicious cycle of worsening hypoxia. This damages optic nerve fibres causing loss of vision. Whether the treatment of falciparum malaria alone, thereby relieving the obstruction of vessels to optic nerves or the addition of steroids as for other causes of retrobulbar neuritis helped improvement in vision in our patient remains debatable in view of rarity of such a complication and experience in its management.

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Received : 10-02-04
Review completed : 16-03-04
Accepted : 20-04-04