A Case of Quinine Induced Blindness

Quinine toxicity is commonly encountered in various forms, the serious types being blindness, arrhythmias, acute renal failure and death [1,2]. We describe a case of quinine induced blindness during management of malaria.

A 23 year old serving paramilitary soldier, teetotaler and with no significant past history, was admitted to his paramilitary hospital with history of fever and headache of two days duration. Blood slide test was positive for *Plasmodium falciparum* malaria. He was treated with parenteral quinine (with loading dose) as per standard dose protocol. About 8-9 hours after quinine administration, he developed tinnitus and rapidly progressive bilateral, painless loss of vision. Intravenous quinine was continued for another 12 hours before he was transferred to our hospital.

On admission, systemic examination revealed a 2 cm palpable liver. Ophthalmic examination revealed right eye vision of finger counting at three feet and left eye vision of 6/60 with accurate projection. Pupils were mid dilated with very sluggish direct and consensual reflex. Fundus examination revealed bilateral pale retina with optic disc pallor. Retinal arterioles appeared diffusely constricted. Intra-ocular pressure was 17mm mercury both eyes. Perimetry and visual evoked potential (VEP) were not done due to non-availability of the investigative modalities.

Plasmodium falciparum malaria was reconfirmed at our hospital. Quinine was discontinued and he was started on injection artesunate and oral doxycycline. Oral nifedipine 10 mg tid and oral prednisolone @ 1 mg/kg body weight were started for management of quinine blindness. Vision improved over next 24 hours (Right eye 5/60 and left eye 6/24). He was continued on nifedipine and prednisolone for seven days followed by gradual tapering off. Eye evaluation at 30 and 60 days after the onset of symptoms was normal with vision of 6/9 unaided both eyes.

Quinine blindness is a rare complication seen in the management of malaria and may occur even with a single dose of the drug in susceptible persons with a mean time from ingestion to onset of blindness being 9 hours [2]. Visual loss occurs in every patient with a plasma quinine concentration above 10 mg/l [3]. The clinical findings in quinine blindness are more or less uniform. The pupils are dilated and immobile, severely contracted retinal vessels, a pale disc and edema of the retina in early stages may be seen. The natural course of the condition is for vision to improve somewhat over subsequent hours and days leaving a residual constricted field in some cases and occasional permanent blindness with optic atrophy in few. The discs may remain pale for years or become normal. Fundus photographs show mild venous distention and retinal

haziness, but normal arteries. The electroretinogram (ERG), electrooculogram (EOG) and VEP are usually abnormal [4,5].

The mechanism of quinine blindness is unclear. The postulated mechanisms being an indirect mechanism of ischemic retinopathy (by way of central retinal artery occlusive spasm) and a direct mechanism of toxicity to the neuroretina (the photoreceptors and ganglion cell layer of the retina).

Therapies like gastric lavage and use of activated charcoal, if applied within 2 hours of intake, are of proven benefit [2]. Other techniques like forced acid diuresis, charcoal column haemoperfusion, haemodialysis and exchange transfusion or techniques attempting to reverse retinal vasoconstriction like intravenous sodium nitrite, carbon dioxide inhalation, retrobulbar vasodilators, anterior chamber paracentesis and Stellate ganglion block have been unsuccessful. Vascular dilatation therapy and use of steroids have shown variable results [6]. We thus used steroids and nifedipine (as vasodilator) in our patient, though it may not have contributed to the improvement.

In summary, quinine, although invaluable in malaria management, should be used judiciously and be stopped immediately on first symptom of toxicity.

References

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