

## Blindness From Quinine Toxicity

S Lewallen, L Bandawe, B Wills

Treatment of febrile children with antimalarials with little or no investigation is standard practice in many areas. Since January 1993 sulfadoxine-pyrimethamine has been the recommended first line treatment for children in Malawi in whom malaria is suspected. The Ministry of Health (MOH) recommends that parenteral quinine be reserved for children with severe malaria; that is, those with altered consciousness, repeated convulsions, persistent vomiting precluding oral treatment, or very severe anaemia. Whenever possible, the diagnosis should be confirmed by blood film examination. Oral quinine is recommended for children who are allergic to sulfa drugs and for those with documented parasitaemia who show no improvement after appropriate treatment with sulfadoxine-pyrimethamine. The malaria treatment guidelines also emphasize the importance of considering alternative diagnoses in febrile children. The indiscriminate use of quinine should be discouraged and health workers should be aware of the potential hazards of quinine toxicity, particularly in small children. The following case illustrates this point.

The patient, a 15 month old male, presented with spasticity of all limbs for 2-3 weeks. The patient was the product of a normal delivery and was well for the first month of life, but started having convulsions 3-4 times a week after that. His mother thought he was afebrile during this time. He was taken to a rural hospital where he was treated with intravenous quinine and diazepam, followed by oral quinine on discharge. Over the next 8 months the patient experienced recurrent fevers, developed diarrhoea, and started losing weight. During these 8 months he was treated with 2-3 day courses of oral quinine every few weeks, according to the mother, for recurrent fevers. When the child was one year old a radiologic diagnosis of pulmonary TB was made and he began treatment with Streptomycin and HT1. Two months later the patient was given a blood transfusion "for pallor" and about a week later the mother noted that the child was not seeing properly, had stopped walking and talking, and had developed stiff limbs. At this point she brought the child to QECH.

Physical examination revealed a severely wasted child with axillary and cervical lymphadenopathy. Hyperreflexia and hypertonia were present in all limbs. The child was poorly responsive to stimulation and appeared to be blind. The right pupil did not react to light, and the left reacted only slightly. Fundus exam showed marked atrophy of both optic nerves, the right being chalk white. Retinal blood vessels were completely constricted and

sheathed, appearing as thin white threads against the dark background of an atrophied retina. There were a few vessels still patent on the left optic nerve, consistent with the minimal pupil reaction and colour of the nerve.

Investigations showed haemoglobin 8.7 and white blood count 3.5 (normal differential). Lumbar puncture yielded clear CSF which was negative on culture; Gram, india ink, and acid fast bacilli stains were all negative.

The mother requested discharge from hospital before any further investigations could be carried out.

Paediatricians consulted considered the diagnosis in this case to be most consistent with AIDS related complex (ARC). The ophthalmologic findings are those of bilateral central retinal artery obstruction, consistent with the late picture of quinine toxicity.

### DISCUSSION

The pathogenesis of blindness in quinine toxicity remains a controversial subject, particularly in the acute stages. Controversy centres on the relative contributions of retinal hypoxia secondary to vasospasm versus toxic damage directly to the retina.<sup>1</sup> Some patients with blindness and acute quinine overdose present with normal fundi and recover vision after a few days. Others present with arteriolar spasm, venous dilatation, and retinal edema. The classic late picture of quinine toxicity, however, is that of an old central retinal artery obstruction, i.e. retinal vessel constriction and sheathing with diffuse retinal atrophy and optic nerve atrophy. In an adult such a finding is generally associated with systemic vascular disease (such as hypertension or diabetes) and is bilateral in less than 5 % of cases. There are no common causes of this phenomenon in children. Specifically, such fundus changes have not been associated with AIDS or ARC.

It is possible for exogenous substances (e.g. talc, cornstarch, or other impurities in intravenous injections) to cause embolic obstruction of the central retinal artery. Neurologic examination was quite abnormal in this child and the possibility of multiple emboli to the brain and retina cannot be ruled out, particularly in light of the history of a blood transfusion shortly before the mother noted blindness. The retinal findings in this child, however, would have required several weeks or months to develop. In view of the history of frequent doses of quinine, we believe that quinine toxicity was the cause of blindness. We have seen several other cases at QECH with an identical fundus picture and history of quinine use.

Quinine is a useful drug, but can have serious toxicity. It should be given according to MOH guidelines and should not be used to treat recurrent fevers in children such as the one described here.

Queen Elizabeth Central Hospital, Blantyre, Malawi

S Lewallen, L Bandawe, B Wills

Correspondence to:  
Susan Lewallen, M.D.  
PO Box 2273  
Blantyre  
Malawi

### Reference

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