CASE REPORT

Ocular complications of malaria treatment

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Abstract

Malaria is endemic in Nigeria. With the emergence of chloroquine resistance various modes of treatment including parenteral quinine are employed with consequent untoward effects. This article reports two cases of severe ocular toxicity, including mimicry of intracranial space-occupying lesion, from treatment of malaria with various drugs including quinine. Medical practitioners are advised to exhaust other less toxic modes of therapy before using drugs with great potential for severe untoward effects such as quinine.

Key words: Eye, malaria, quinine

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Introduction

Malaria is an infectious illness caused by protozoa of the plasmodium species.[1] The infective agent (plasmodium) is innoculated into the blood stream through the bite of mosquito that is the vector of plasmodium.[1] Malaria, which is endemic in Nigeria, has protean symptoms including bitter taste, nausea, vomiting, high fever, and headache.

For a long time, chloroquine administration was the standard treatment.[1,2] However, in the past few decades chloroquine resistance has become widespread. This has led to the use of other modes of therapy including quinine, amodiaquine, artemisin, doxycycline, pyrimethamine-sulfonamide, and combination of these.[1,2]

Treatment regimen involving quinine may be associated with ophthalmic, cardiac, and oto-rhinolaryngological complications.[3] This article reports on two cases of serious ocular side effects associated with malarial treatment with parenteral quinine.

Case Reports

Case 1

A 20-year-old female undergraduate presented on 28 August 2003 at the Ebony Eye Specialist Clinic Onitsha with a week history of double vision, eye ache, and nuchal ache. Four days earlier she had developed acute febrile illness for which a blood film examination was remarkable for malaria parasite. She was admitted and treated by a general medical practitioner with intravenous infusion of 5% dextrose solution, injections of analgin, diclofenac, promethazine, quinine sulfate, and vitamin B-complex.

Examination showed a well nourished but anxious lady. The right visual acuity was 6/12 and the left was 6/60. The right eye had florid papilledema and peripapillary retinal hemorrhage. The left eye had relative afferent pupillary defect, convergent squint, complete absence of abduction, florid papilledema, and retinal hemorrhage. There was no demonstrable diplopia. The intraocular pressure in each eye was 12 mmHg. An initial impression of intracranial space-occupying lesion was made. Visual fields and computerized tomography (CT scan) were ordered.

She was advised to stop all her previous medication and we commenced her on hydrocortisone eye drops; oral dexamethasone (0.5 mg, three times daily), paracetamol, and folic acid. Visual fields showed tunnel vision in the

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right eye and the left could not fixate the test target. CT scan was normal.

Three days later, the nuchal and eye aches had subsided; degree of papilledema clinically reduced and visual acuity had improved to 6/9 in the right eye and 6/36 in the left. Six weeks after presentation the visual acuity was 6/6 in each eye; each eye had full extraocular muscle movements; the retinal; hemorrhages and papilledema had completely resolved and other systemic symptoms abated. A repeat perimetry showed normal visual fields in each eye.

Case 2

A 32-year-old female factory worker presented at the Guinness Eye Center Onitsha on 9 June 2009 with a 3 week history of visual obscuration following treatment for acute malaria. She was referred from the Medical Outpatients Department of the Nnamdi Azikiwe University Teaching Hospital Nnewi. The referral letter documented that she had been treated for laboratory proven chloroquine-resistant malaria with injections of quinine sulfate, oral ciprofloxacin, and pyrimethamine-sulfonamide (maloxine).

Examination showed a fully conscious but anxious woman. The right visual acuity was 6/12 and the left was 6/9. Each eye had florid papilledema, preretinal hemorrhage, and widespread cotton wool spots. In addition, the right eye had convergent squint, reduced abduction, and macula edema. The intraocular pressure in the right eye was 16 and 14 mmHg in the left. A clinical impression of drug-induced combined retinopathy and optic neuropathy was made. She was sent for visual fields test that showed marked constriction and paracentral scotoma in the right eye and peripheral depression in the left.

Treatment was with oral prednisolone (80 mg per day in divided doses), analgesics, and multivitamin and she was followed up weekly. Four weeks after presentation the right visual acuity had improved to 6/9 and the left to 6/6. The papilledema and macula edema had clinically reduced; the cotton wool spots had completely resolved. There was improved extraocular muscle movement though 15° esotropia was still present. At this point the steroid was tapered off for the next 4 weeks. A year after initial presentation the convergent squint had completely subsided and all the retinal and optic nerve features had fully resolved.

Discussion

Malaria is a disease every medical practitioner in our environment is bound to treat since it is endemic. However, the emergence of resistance to the hitherto standard treatment with chloroquine has led to the use of other modes of therapy.[1,2] Quinine is one of the earliest antimalarial drugs.[1] But due to its systemic toxicity and the availability of the relatively less toxic drugs it lost its pride of place as the first line drug for treating malaria.

However, with the emergence of resistance to the routine malarial drugs such as chloroquine and pyrimethamine-sulphonamide combinations, there is a surge toward the use of quinine as an initial therapeutic modality for malaria. Reports abound in the literature of the untoward effects of quinine (cinchonism) on the heart where it could cause death in overdose; the central nervous system toxicity manifests as stupor, delirium, coma and seizures; ear toxicities include tinnitus and deafness.[3-5] Ocular toxicity from quinine includes visual impairment and permanent blindness. Other ophthalmic abnormalities include reduced central visual acuity, pupillary abnormalities (vermiform motion), constricted visual fields, and dyschromatopsia.[4]

It is to be emphasized that our patients had, in addition to quinine, taken other drugs including antibiotics (ciprofloxacin) and chemotherapeutic agents (promethazine, sulphonamides, and pyrimethamine). However, none of these drugs is known to cause the type of severe visual symptoms that our patients presented with. We had suspected space-occupying lesion in our first patient, but the CT scan was unremarkable. The knowledge gained from managing our first patient made us to emphasize perimetry alone for our second patient. It is not certain that the medicines we gave to our patients had any specific effect in counteracting the effect of quinine. We recommend basic science research for an answer to this. While oral charcoal, laxatives, and plasmapheresis will lower serum quinine it is not clear that such measures in any way ameliorate the retinal toxicity which is speculated to be due to blockade of cholinergic transmission.[4]

Drug resistant malaria is a great challenge to the medical practitioner in an endemic area such as Nigeria. When confronted with life-threatening malaria, he may be forced to apply drugs with severe side effects. Quinine may not be tolerated by some patients even when given in therapeutic doses, it could cause permanent blindness and even death with overdose.[5] Therefore, medical practitioners using this drug should regularly monitor the patients' kidney function and target organ toxicity including ocular toxicity.

Finally given the poor therapeutic safety of quinine it is advised that less toxic modes of therapy be exhausted before the use of quinine. While patients also need be educated on the many side effects of quinine, the medical practitioner, including the ophthalmologist, should be aware that ocular manifestation of these untoward effects could mimic intracranial space-occupying lesion.

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