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Quinine Induced Temporary Visual Loss - A Case Report

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Background
Cinchona bark, which contains quinine, is known to have been used in Europe since 1633 for prophylaxis and treatment of malaria. The commonest symptoms of overdose are tinnitus, nausea, vomiting, hearing impairment, vasodilation and sweating [1,2] .While all of the cinchona alkaloids can cause visual disturbances, only quinine is known to cause blindness. [3] The commonest permanent disability is visual field restriction, which is frequently severe and may require the patient to be placed on the blind register [1, 2, 4]. The vast majority of patients show some improvement with transient blindness lasting for 1 h to 50 days. Between 8 and 14 h is the typical of the length of time between developing blindness and the first perception of light. In general, visual recovery is only partial, with the peripheral vision being the most affected.
Key words: Quinine, Visual loss.

DISCUSSION

Disturbance of vision have been reported in 17% of patients with quinine overdose[5], 75% of these patients were completely blind. However, once daily dose of Quinine may cause alteration in colour vision, visual field restriction or blurring of vision. Typically, ocular symptoms develop 4-15h after overdose [1, 5, 6]The exact mechanism of visual loss has been debated since the 1880's [1,7] and is still uncertain. It was initially felt that blindness occurred due to retinal ischemia secondary to retinal arteriolar constriction. [8, 9, 10] However, blindness has been observed in patients whose retinal arteriolar calibre remained normal [6, 11] and others with normal retinal arteriolar calibre when blindness occurred, several days or weeks later after sight had returned, developed arteriolar constriction.[1,2,5,9,6,12,13,4,15] This suggests that blindness is not due to retinal arteriolar constriction. Support for the hypothesis that blindness results from a direct toxic effect of quinine on the retina comes from electroretinographic (ERG) studies following quinine overdose. Measures to dilate retinal arterioles have been used in the treatment of blindness due to quinine overdose for 45 years, [16, 17] the commonest method used has been SGB (Stellate ganglion block).Other methods have been used to achieve retinal arteriolar vasodilation including intravenous[8], inhaled [1,4,9, 18,19] and retrobulbar[2,19,20] vasodilators, carbon-dioxide inhalation [4] and reducing intraocular pressure by anterior chamber paracentesis [4].

Indian Journal of Pharmacy Practice
Received on 08/09/2008 Modified on 22/09/2008
Accepted on 24/09/2008 *© APTI All rights reserved*

These methods have never been studied in a controlled trial but case reports do not show any clear evidence of benefit. This is further support for the idea that blindness following quinine overdose is not due to vasoconstriction. Ocular massage, recumbent posture [1] and hyperbaric oxygen [22] have also been reported as beneficial in single case reports. Peritoneal dialysis, haemodialysis, exchange transfusion and charcoal and resin haemoperfusion (if oral ingestion of quinine tablets) only remove a very small quantity of quinine and have not been proven to be of any therapeutic benefit [23, 24,25,26,27]. This is presumed to be due to quinine being strongly bound to plasma proteins and having a large volume of distribution[26,27].

The case

A 24y year old male, suffering from moderate-high grade fever intermittently since a week having tested Malaria falciparum positive was treated with oral chloroquine 15mg & Paracetamol tablets. The symptoms still persisted and hence reported to the emergency at around 6.30p.m. Upon investigation, the patient was diagnosed to have cerebral Malaria. He was then shifted to the MICU, at 8.30 p.m and treated with a loading dose of 1200mg quinine IV in 5% dextrose infusion, artisunate 120mg IV in 0.9% normal saline, along with IV omeprazole 20mg & IV ondansetron 8mg.The patient had acute attacks of psychoses for which the patient was given IV haloperidol. In the morning at around 6.45 a.m, the patient was given a second dose of 600 mg of quinine IV. Within two hours after administration of second dose

of quinine intravenously, the patient complained his inability to see objects and people attending him. Also, the patient was not responding to finger counting. The resident doctor upon thoroughly examining the patient called on the ophthalmologist. Fundoscopic examination revealed that there was no evidence of retinal ischemia with vasoconstriction or retinal pallor and the ophthalmological examination was essentially normal. Later, the physician suspected that it could be due to other causes & hence spontaneously reported as an ADR. Upon systematically analyzing the ADR report, the vision loss was suspected to have been caused by quinine. Careful literature survey was carried out to assess the causality of the reported ADR. Literature survey revealed that quinine can cause temporary to permanent visual loss at plasma concentration levels of 10-15mcg/ml. Upon confirmation of the causality, quinine was withdrawn from the treatment of the patient. Literature survey revealed that this type of ADR induced by quinine can be managed by administering hyperbaric oxygen to the patient. Although there is no known definite treatment for quinine induced blindness, early withdrawal of quinine from the treatment regimen and by supplemental oxygen administration is suggested in the literature. Supportive management was done by maintaining good fluid and electrolyte balance. This treatment was adopted by the physician attending the patient. By evening of the same day, the patient gradually could see faintly and finally, regained his vision. The patient was treated for Malaria only with Artisunate 60mg OD and oflaoxacin and was discharged two days later after he was found to be stable.

CONCLUSION

Though commonly not encountered, quinine overdose may lead to temporary or permanent blindness at a plasma concentration of 10-15mcg/ml. Hence, it is essential to monitor the plasma concentration of quinine in patients treated for Malaria especially with IV quinine. Though the plasma concentration of quinine was not monitored in the present case, timely reporting of this ADR helped the patient to regain his sight. The present case emphasizes the importance of monitoring of the plasma concentration of potentially harmful drugs and more importantly it highlights the importance of adverse drug reaction monitoring.

ACKNOWLEDGEMENT

Authors wish to thank the Medical superintendent, consultants and nursing staff of St.Martha's Hospital, Bangalore for their support and encouragement. The authors also thankful to the principal & Management of Al-Ameen College of Pharmacy, Bangalore for their support.

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