Quinine induced blindness

Symptoms of cinchonism are well described and consist of headache, tinnitus, nausea, abdominal pain, and hypotension. Acute intoxication has been associated with blindness, arrhythmias, acute renal failure, and death. Symptoms are likely in any single dose greater than 4 g and death has been reported with as little as 8 g. Quinine is almost completely absorbed from the gut and peak plasma concentrations are reached at about 1 to 3 hours. It is 70% protein bound and metabolism occurs in the liver.

In a study of 48 cases, the mean time from ingestion to onset of minor symptoms was 3.5 hours, blindness developed somewhat later, after a mean of 9 hours. Visual loss occurred in every patient with a plasma quinine concentration above 10 mg/l. A level above 15 mg/l is associated with increasing risk of permanent ocular damage.

The mechanism by which quinine causes ocular toxicity has yet to be defined and is reflected by a lack of effective treatment. The early observation of arteriolar narrowing and disc pallor followed later by macular oedema led to the suggestion that arterial vasodistortion may cause retinal ischaemia. Other workers have observed this as a late phenomenon occurring after the onset of blindness and favour direct toxicity to the neuroretina. Canning et al argue for a toxic mechanism involving interference with cholinergic neurotransmission causing the acute visual loss, with long term visual disturbance either the result of irreversible acute retinal toxicity or secondary to retinal vascular insufficiency.

The natural course of the condition is for vision to improve somewhat over subsequent hours and days leaving a residual constricted field. Therapies have been largely directed towards reducing quinine absorption, enhancing its elimination, and reversing retinal vasodistortion. It has been shown that continued administration of activated charcoal significantly reduces the plasma half life of quinine. Other techniques to enhance elimination including forced acid diuresis, charcoal column haemoperfusion, and exchange transfusion have been unsuccessful.

Historically, various methods have been used to attempt reversal of retinal vasodistortion including intravenous sodium nitrite, carbon dioxide inhalation, retrobulbar vasodilators, and anterior chamber paracentesis to reduce intraocular pressure. Stellate ganglion block (SGB), in an attempt to reduce intraocular pressure. SGB is not entirely discredited, it may have a place if administered early enough. Patients often present when irreversible ocular damage has already occurred. We suggest that therapies aimed at preventing ocular damage be administered to all patients with a significant history of quinine ingestion before symptoms appear.

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