Lesson of the Week

Loss of sight after self poisoning with quinine

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Quinine is often prescribed for leg cramps; 1.355 million prescriptions were issued for quinine preparations in Great Britain in 1981 (Department of Health and Social Security, personal communication). Loss of sight after overdose has been reported¹ and may arise as an idiosyncratic sensitivity to the drug.² In susceptible subjects severe reduction in visual function may follow ingestion of amounts not grossly above the therapeutic level.³ We report two cases in which visual damage occurred when an overdose was taken by a close relative of the person for whom the drug was prescribed. In both cases the self poisoning could have been a predictable risk.

Case reports

Case 1—A 38 year old man with a history of chronic alcoholism and previous overdoses took 10 tablets of quinine sulphate 200 mg, prescribed to his mother for night cramps, after a severe marital disagreement. He awoke during the night with tinnitus, nausea, and vomiting and was unable to see at all. When seen at casualty he was distressed, with striking facial flushing and profuse sweating. He was obviously deaf, able to hear only when shouted at. He was unable to see even bright lights, and his pupils were fixed and dilated with no response to light or accommodation. Fundal examination showed retinal oedema with a cherry red spot at the macula. Shortly after admission he had a stellate ganglion block and retrobulbar injection of acetylcholine with no apparent improvement, but over the subsequent days his visual acuity improved to counting fingers in either eye, and he had visual field constriction. His pupillary reactions recovered slowly. The fundal appearance of the blood vessels changed during his stay as the arterioles became considerably constricted and the discs pale. His hearing returned after one to two days. The central visual acuities improved slowly, and 16 months after the ingestion of quinine they were recorded as right 6/12 and left 6/9. Visual fields also expanded, although there was still some vertical limitation. He complained of a grey haze across his vision, especially in the dark, and this was considered to be consistent with subtle pigmentary retinal disturbance which had developed. The discs remained pale.

Case 2—A 60 year old woman with a history of depression requiring inpatient management took 20 tablets of quinine sulphate 200 mg, prescribed to her husband for night cramps. Over the next three to four hours she noticed profound deterioration of vision, nausea, and considerable loss of hearing. When seen at casualty she had a visual acuity of perception of light in either eye. The pupils were fixed and dilated, and bilateral macular oedema could be seen. The blood vessels at this stage appeared of normal calibre. She was given a stellate ganglion block. Over the next few days central vision improved to 6/12 in each eye, and hearing returned. The peripheral

Caution should be shown in prescribing quinine for patients whose close relatives may be prone to self poisoning, as this drug can cause severe and prolonged visual handicap

visual field was found to be grossly constricted, and fundal examination showed considerably attenuated retinal arteries. Her depressive condition worsened considerably, with a considerable overlay of guilt about her overdose. Central vision slowly deteriorated and six months after the event was recorded as 6/36 right and 6/60 left. The peripheral field remained grossly constricted. The retinal vessels still appeared attenuated and disc pallor had developed. She continued to need close psychiatric supervision, with a long period of inpatient treatment.

Comment

Quinine is commonly used for leg cramps as well as for more serious conditions such as resistant *Plasmodium falciparum* infections. Quinine amblyopia was first described in 1841 and has been reported since, usually in association with temporary but severe deafness, nausea, vomiting, and facial flushing.³ Severe cases develop deep coma with circulatory collapse. Some recovery of visual function is the rule, but appreciable impairment of vision may persist with secondary problems such as occurred in case 2, where severe depression was precipitated in an already susceptible subject.

In some cases the reaction to the drug is believed to be idiosyncratic,² with low doses leading to symptoms in a few patients. Toxic visual symptoms have been reported from doses as low as 0.13 g; an average individual, however, would have symptoms after 2.5–4 g,³ and this would be expected in our cases, where 2 g was taken in case 1 and 4 g in case 2.

In both our cases the drug was prescribed for patients whose close relatives could be recognised as at risk of self poisoning. Care is taken in prescribing drugs more commonly associated with self poisoning in such families, and we feel that the implications of prescribing quinine should always be carefully considered and this drug should be avoided if at all possible.

Quinine toxicity should be suspected in patients with sudden visual loss and widely dilated non-reactive pupils. There are several suggested treatments: stellate ganglion block, vasodilators, and adrenocorticotrophic hormone, but all these require rapid referral, as treatment is unlikely to be successful more than 24 hours after ingestion.

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