CASE REPORT

Ocular quinine toxicity in a sleepwalker

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SUMMARY

A 55-year-old woman presented to the emergency department following an episode of severe visual impairment, headache, dizziness and confusion. The patient had been taking quinine sulfate as long-term medication for leg cramps. During an episode of sleepwalking, the patient had taken an overdose of quinine sulfate. Following a thorough investigation and assessment, a diagnosis of ocular quinine toxicity was made. We present this case and highlight the risks of quinine prescription.

BACKGROUND

Ocular toxicity is a known side effect of quinine sulfate. Guidelines regarding its prescription have been written. However, cases of ocular quinine toxicity continue to present and it is evident that guidelines regarding prescription are not being followed. Often the drug is prescribed to patients such as the elderly who are at risk of inadvertent overdose. We have written this case to highlight the dangers of quinine prescription and particularly those in a vulnerable patient group. We would urge practitioners to exercise caution when prescribing this drug and to follow appropriate guidelines.

CASE PRESENTATION

A 55-year-old woman presented to the emergency department after waking up and being unable to see anything. This was accompanied by a generalised headache, dizziness and confusion. She denied having any fever or neck stiffness and had been otherwise well. There was no history of trauma.

The patient had a medical history of chronic obstructive pulmonary disease (COPD), osteoporosis and nocturnal leg cramps. She had no other neurological or ophthalmic history. Her regular medication consisted of salbutamol inhaler 100 mg PRN, tiotropium inhaler 2.5 mg once daily, prednisolone 45 mg once daily, quinine sulfate 300 mg nocte and thiamine 100 mg once daily. She had been taking this medication for several years without any recent changes. She had no known drug allergy. She lived with her daughter but was independent in her activities of daily living.

On assessment in the emergency department she was non-feverish and all other baseline observations were in the normal range. An examination of the cranial nerves 2–4 was difficult due to her inability to see. However, her pupils were fixed and dilated. There was no abnormality in cranial nerves 1 and 5–12. A full clinical examination including neurological assessment of the limbs, was unremarkable.

Differential diagnosis included subdural haematoma, cerebrovascular accident involving the occipital lobe, cavernous sinus thrombosis, optic neuropathy and ocular quinine toxicity. Blood samples were sent for a full blood count, urea and electrolytes, liver function tests and a coagulation screen. The patient underwent CT scan and MRI of the head. The scans did not show any abnormality and the blood results were in the normal range. The patient was subsequently transferred to the acute medical ward where she was reviewed by a senior physician and admitted for regular neurological observations overnight and an ophthalmology review the following morning.

The patient had an ophthalmology review the following morning. Her vision had subjectively improved but was still very dark. Her visual acuity measured 6/12 bilaterally. Pupils were equal and reactive to light, although sluggish. There was no relative afferent pupillary defect. The patient was unable to perform a test of colour vision. Visual field testing was attempted but had to be abandoned as the patient was unable to complete it due to discomfort. The rest of the ocular examination was normal including that of the fundus and optic disc. There was no optic disc pallor or swelling.

Ocular quinine toxicity seemed like a possible cause at this stage. On further questioning, the patient mentioned that she may have accidentally taken nine quinine tablets rather than the nine prednisolone tablets (for COPD) she was supposed to. She had experienced episodes of sleepwalking in the past and felt she had taken some tablets while sleepwalking. Indeed the patient's daughter had found an empty box of quinine tablets on the kitchen counter.

The patient had a further ophthalmology review 48 h after initial presentation. Her visual acuity had improved to 6/9 bilaterally. Pupillary reactions had improved and her colour vision was normal. However, she had noticed a restricted visual field.

INVESTIGATIONS

Formal visual field testing was attempted but had to be abandoned due to pain and discomfort. She was also referred for electrodiagnostic testing. This included optical coherence tomography (OCT) which revealed an almost total loss of retinal ganglion cells and thinning of the inner plexiform layer in both eyes. Such findings are typical of quinine toxicity.¹ A normal OCT scan is demonstrated in figure 1 while an OCT demonstrating thinning of the inner plexiform layer and loss of ganglion cells is demonstrated in figure 2.

In addition to OCT, visually evoked potentials (VEPs) to flash stimulation were normal from both

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Figure 1 Normal optical coherence tomography scan demonstrating human retina in cross section.

Figure 2 Optical coherence tomography scan demonstrating thinning of the inner plexiform layer and loss of ganglion cells.

eyes. Pattern responses were reduced from both eyes but were not delayed with P100 around 100–110 ms from either eye.

Pattern electroretinograms (ERGs) were poorly formed but appeared to show slight reduction of P50 and absence of N95 components in both eyes. ERGs to scotopic stimulation reveal normal a waves, but a bilateral reduction in b wave amplitude, and loss of oscillatory potentials in both eyes.

Photopic and 30 Hz (cone) responses were grossly reduced from both eyes revealing almost absent b waves in either eye.

OUTCOME AND FOLLOW-UP

At 1-month follow-up, her visual acuity was 6/9 in the right eye and 6/7 in the left eye. Only foveal vision was present. Her vision remained like this at 3, 6, 9 and 12 months of follow-up appointments.

DISCUSSION

Ocular quinine toxicity has been known about for some time with a case being reported as far back as 1980.² The drug is commonly used for the treatment of nocturnal leg cramps. Overall efficacy is modest despite variation in patient response. The Medicines and Health products Regulatory Agency (MHRA) recommend that quinine should not be used routinely but only when cramps affect sleep. It also mentions that non-pharmacological methods of management should be used first, that the prescription should be stopped if benefits are not felt in 4 weeks and that the prescription should be reviewed every 3 months otherwise. Despite this advice, quinine is still routinely prescribed in the UK without appropriate review. In other countries, more stringent rules are in place. In the USA, the food and drug administration have restricted the indications only for uncomplicated malaria while in Spain, the drug is not commercialised due to its dangerous side effects.

A paper published 16 years ago reported that six cases of severe ocular toxicity were observed over a 4-year period.³ Furthermore, a case of an overdose of quinine with alcohol was reported 10 years ago in which the authors highlighted the toxic effects and dangers of routine prescribing.⁴ The publication of cases of quinine toxicity over a wide time period,

suggests that lessons are not being learned from adverse outcomes of routine prescription.

Our case presents the challenge of prescribing to a potentially vulnerable patient group. Patients such as sleepwalkers, the elderly and those influenced by recreational drugs and alcohol, are more likely to inadvertently take an overdose. Particular caution must be made when prescribing to such patients. Prescription of quinine in such patient groups should be more restricted and more stringently reviewed.

Learning points

- ▶ The prescription of quinine should be avoided in vulnerable patient groups such as the elderly.
- ▶ Patients should not be given repeat prescriptions of quinine without very regular medication review.
- ▶ A thorough drug history can prove to be vital in the differential diagnosis and subsequent management of patients presenting with a vague history or clinical signs.

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