

Myopic shift and acute glaucoma associated with topiramate

T Chang¹, M M Dissanayake²

Journal of the Ceylon College of Physicians, 2011, 42, 39-40

Introduction

Topiramate, a sulphamate-substituted monosaccharide, is primarily an anti-epileptic agent, but is also used in the management of migraine, neuropathic pain and bipolar disorders. In Sri Lanka, topiramate is increasingly being prescribed as first-line prophylaxis for migraine. Nephrolithiasis, severe acidosis, hyperthermia and glaucoma are rare but serious side-effects of topiramate. Secondary angle-closure glaucoma associated with topiramate was first reported in 2001¹ and several such cases have been reported since². We report a case of bilateral acute glaucoma and induced myopia occurring within 7 days of initiating topiramate for headache.

Case report

A 32-year old man presented to a neurologist with almost-daily headaches for 2 months. Although he had suffered from migraine with aura for about 12 years, the frequency of migraine headaches had reduced over the years with the last episode being in 2006. His current headaches were different to the migraines in that they were less severe, protracted and not associated with aura or photo / phonophobia. However, given the history of migraine, he had been prescribed topiramate 25 mg daily in addition to a short course of NSAIDs. One week after commencing treatment, he had experienced blurred vision with halos around lights and figures, and complete obscuration of distal vision associated with frontal headache. However, his near vision was intact. On examination by the ophthalmologist, he was found to have bilateral corneal oedema and the intraocular pressures (IOP) were 44 mmHg bilaterally. His visual acuity was 3/60 in the right and 6/60 in the left eye. Refraction revealed - 3.00 spheres in either eye with corrected vision of 6 /12. He was treated with intravenous mannitol, oral acetazolamide and topical timolol whilst anti-migraine medication was discontinued. His IOP normalised and his vision returned to normal. Anti-glaucoma therapy was discontinued. One week later, his IOPs were 12

mmHg bilaterally and the visual acuities were 6/9 in both eyes. He saw a second neurologist (author) for want of alternative medication for his headaches, which were tension-type and not migrainous, and did not warrant topiramate.

Discussion

The exact mechanism of topiramate in seizure control, migraine prophylaxis or mood stabilisation is unknown. Weight loss (up to 10% of patients), cognitive slowing (up to 20% of patients), paraesthesia (up to 50% of patients), nephrolithiasis, severe acidosis, hyperthermia and glaucoma are some of its notable side-effects.

Our patient with no previous history of ocular disease developed bilaterally raised IOP and myopic shift following a week of topiramate therapy, which resolved completely after medical treatment of the raised IOP and discontinuation of topiramate suggesting that the ocular effects were drug-related. Since our patient was on only 25 mg of topiramate per day, it appears that the effect on IOP is not dose-dependent.

Acute elevation of IOP after topiramate use was only discovered post-marketing^{1,3}. In a review of 115 case reports of ocular side-effects associated with topiramate, 86 cases of angle-closure glaucoma (83 bilateral) were reported². The onset was acute with 85% of cases occurring within 2 weeks of treatment with a mean of 7 days from the onset of therapy. Forty seven percent of patients were on 50 mg or less of topiramate. Five cases were precipitated within hours when the dose was doubled.

The pathogenic mechanisms underlying angle-closure glaucoma and myopia after topiramate are not fully understood. Visual outcome is usually good and the episode resolves within a few days to weeks after discontinuation of the drug. However, if unrecognized as a drug-related event, permanent visual loss could occur². The latter is likely to occur when treating migraine since the visual symptoms of raised IOP and the associated headache could be misdiagnosed as migrainous.

Thus, it is important for clinicians to be aware of this serious, albeit rare side-effect of topiramate and

¹ *University Medical Unit, National Hospital of Sri Lanka and Department of Clinical Medicine, University of Colombo*

² *Department of Anatomy, Faculty of Medicine, University of Colombo, Sri Lanka.*

instruct patients to seek attention should they develop blurred vision following initiation or escalation of dosage. Furthermore, topiramate should not be over-used in migraine prophylaxis and must be reserved as a second-line agent for patients in whom other prophylactic agents with less serious side-effect profiles such as beta blockers, calcium channel blockers and tri-cyclic antidepressants have failed⁴.

References

1. Banta JT, Hoffman K, Budenz DL, Ceballos E, Greenfield DS. Presumed topiramate-induced bilateral acute angle-closure glaucoma. *American Journal of Ophthalmology* 2001; **132**: 112-4.
2. Fraunfelder FW, Fraunfelder FT, Keates EU. Topiramate-associated acute, bilateral, secondary angle-closure glaucoma. *Ophthalmology* 2004; **111**: 109-111.
3. Thambi L, Kapcala LP, Chambers W, Nourjah P, Beitz J, et al. Topiramate-associated secondary angle-closure glaucoma: a case series. *Archives of Ophthalmology* 2002; **120**: 1108.
4. British Association for the Study of Headache. Guidelines for all doctors in the diagnosis and management of migraine and tension-type headache. 2nd edition (revised). August 2004. <http://www.bash.org.uk>.