Short communication

Angle-closure glaucoma secondary to topiramate use

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ABSTRACT

Case report: We describe a 42-year-old patient who developed acute myopia and closed-angle glaucoma one week after beginning treatment with topiramate. Ultrasound biomicroscopy (UBM) revealed a bilateral angle closure and choroidal effusion. The clinical findings resolved with withdrawal of the topiramate.

Discussion: Topiramate may cause acute myopia and closure angle glaucoma in some patients due to a choroidal effusion. UBM seems to be a useful tool for monitoring the progression of the clinical lesions and their resolution when the drug is withdrawn.

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Glaucoma de ángulo cerrado secundario a topiramato

RESUMEN

Caso clínico: Se presenta el caso de una mujer de 42 años que desarrolló un cuadro de miopía aguda y glaucoma de ángulo cerrado bilateral una semana después del inicio del tratamiento con topiramato. La biomicroscopía ultrasonora (BMU) reveló que la paciente presentaba un ángulo cerrado y una efusión coroidea bilateral. Con la retirada del fármaco se resolvió el cuadro.

Discusión: El topiramato puede desencadenar miopía aguda y glaucoma de ángulo cerrado en algunos pacientes debido a una efusión coroidea. La realización de la BMU parece ser una herramienta útil para observar la evolución del cuadro y su resolución tras la suspensión del fármaco.

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Introduction

Topiramate (Topamax®, Janssen-Cilag, Madrid, Spain) is a sulphonamide monosaccharid utilized for treating some types of epilepsy, although it recently began to be used for treating bipolar disorder, migraine and even for losing weight. Ocular side effects have been described with this drug, including temporary myopia (TM) or closed-angle glaucoma (CAG)1-3 as well as campimetric alterations or maculopathies.4


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This paper presents a case of a patient with topiramate-induced TM and CAG describing the probable pathogenic mechanism by means of UBM as well as the evolution of the condition, correlating clinical findings with echography images.

**Clinic case**

A patient, aged 42, visited the Emergency section due to diminished visual acuity (VA) with a 24-h evolution. Personal history included treatments for depression. No relevant ophthalmological history was found and the patient denied using spectacles. Said treatment consisted in olanzapin 10 mg, lorazepam 2 mg and venlafaxin 150 mg retard for several months. One week before he had started taking topiramate to lose weight.

VA was finger counting at 1 m in both eyes (BE), which improved to 20/40 with stenopic. The biomicroscopic assessment revealed corneal edema and conjunctival chemosis, narrow anterior chamber and angular closure, evidenced by means of gonioscopy. Intraocular pressure (IOP) was of 55 mmHg in BE. Accordingly, bilateral CAG diagnostic is established, administering topical treatment with pilocarpin 2% (Colicursi Pilocarpina 2%, Alcon-Cusí, Barcelona, Spain), timolole 0.5% (Cusimolol 0.5%, Alcon-Cusí, Barcelona, Spain), brimonidine 0.2% (Alphagan®, Allergan, Madrid, Spain) as well as 1 acetazolamide capsule 250 (Edemox®, Chiesi, Barcelona, Spain). Two hours later, IOP was of 30 and 32 mmHg, respectively.

Twenty-four hours after the first visit, the patient eyesight remained the same. At the biomicroscopic level, the corneal edema had disappeared (Fig. 1a and b), and IOP was of 28 and 30 mmHg. UBM was performed on BE by means of the UBM system (AVISO, Quantel, Clermont-Ferrand, France) with a 50 MHz probe (Fig. 1c) which evidenced angle closure, narrowed anterior chamber (2.18 mm measured from the corneal epithelium to the lens anterior capsule) as well as an uveal effusion condition which pushed forward the ciliary body and the iris-lens diaphragm in BE. The patient was diagnosed with secondary CAG associated to ciliary-choroidal detachment. Pilocarpin was suspended and treatment with ocular hypotensors was continued. Topiramate treatment was suspended in agreement with the prescribing physician.

Five days after terminating topiramate treatment, the patient exhibited a VA of 20/40 in BE, which improved to 20/20 with a grade of –2 spherical in BE. IOP was of 10 and 12 mmHg. At the biomicroscopic level, the width of the anterior chamber returned to normal (Fig. 2a). An additional UBM was performed (Fig. 2b) which revealed a total disappearance of the uveal effusion and conjunctival chemosis as well as normalization of the anterior chamber width (3.54 mm) together with an increased angle. The papillae were normal in BE. Three months after the onset of the condition, the patient remained stable.

**Discussion**

Bilateral CAG and myopic changes have been described in the literature as side effects of topiramate. The use of UBM has allowed us to determine the etiopathogeny of both processes. Recently, Scheimpflug images have also been applied to obtain graphical documentation of this phenomenon. It is known that the uveal effusion syndrome, as well as the ciliary body edema cause anterior displacement of the iridocrystalline diaphragm, leading to myopization. As a result of said displacement the anterior chamber narrows and an acute glaucoma crisis ensues. In our case, due to the use of UBM we were able to confirm that the cause of both processes was in the uveal effusion syndrome which was resolved 5 days after suspending topiramate treatment.

As there was no pupil obstruction, peripheral iridectomy or pilocarpine treatment were not useful. Myotic eyedrops are not advised because they could produce relative pupil obstruction due to anterior iridocrystalline diaphragm displacement. The recommended treatment is mainly suspending topiramate. Initially, we began treatment with pilocarpine due to lack of certainty about the existence of pupil obstruction, but UBM discarded the possibility and evidenced an uveal effusion condition. Accordingly, treatment with miotics was withdrawn. It is not clear if the CAG was a consequence of initiating treatment with topiramate on its own or if venlafaxin
contributed to the mechanism which produced or worsened the process.1

By way of conclusion, it is necessary to determine the relationship between bilateral GAC and the establishment of recent medication with topiramate. Assessment with UBM will assist in confirming the diagnosis and following up the evolution of the condition.

**Conflict of interests**

No conflict of interest has been declared by the authors.

**REFERENCES**


Fig. 2 – (a) Right eye photograph evidencing the increased width of the anterior chamber (AC). (b) Right eye ultrasound biomicroscopy; a composition comprising 3 images evidencing normal AC depth, angular aperture with reabsorption of the supraciliary choroidal liquid, ciliary body (CB) repositioning and lens plane. The subconjunctival bags have disappeared.