SEVERE OCULAR SIDE EFFECTS WITH TOPAMAX

TOXICIDAD OFTALMOLÓGICA SEVERA POR TOPAMAX

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ABSTRACT

Case reports: We describe 2 patients who developed ocular side effects during treatment with topiramate. One was a 16-year-old woman with generalized seizures who developed a homonymous hemianopia after using topiramate 125 mg per day for 12 weeks, and the other was a 24-year-old epileptic female who developed a bilateral maculopathy after using topiramate 150 mg per day for 8 weeks.

Discussion: We identified two severe ocular adverse reactions from topiramate. Both reactions necessitated discontinuing the treatment, but the topiramate-associated maculopathy was irreversible (Arch Soc Esp Oftalmol 2006; 81: 345-348).

Key words: Topiramate, homonymous hemianopia, maculopathy.

INTRODUCTION

Topamax® (Topiramate; Janssen-Cilag, Madrid, Spain) is a relatively new anti-epileptic classified as a monosaccaride sulphamate utilized in untreatable epileptic syndromes. The most recent studies show numerous adverse effects, although at the ophthalmological level said effects are mainly inducing myopia, acute glaucoma due to angular closure and choroid detachment (1,2). This communication presents two patients, one with hemi-apnosic alterations of the visual field and the other with maculopathy, both related to Topamax.

CASE REPORTS

Case report 1

A 16-year old girl referred to the ophthalmology section due to a visual difficulty condition, in her own words: «there is a part of the blackboard I can’t
see, particularly for about 3 weeks». Her mother refers the girl «has gone from being active to slow, with difficulty in thinking and speaking, and now her eyesight is bad». Since age 7, the girl had difficult to control epileptic episodes. Her treatment was carbamazepine and hydantoine, and since 3 months ago 125 mg per day of Topamax. The previous ophthalmological assessment performed one year ago was normal. In the assessment, her spontaneous visual acuity (VA) was of 1 in both eyes, with normal anterior biomicroscopy. The pupils were isochoric and with normal reactions. The funduscopy did not exhibit alterations. The computerized cam-pimetry showed a non-congruent left hemianopsia (fig. 1). The colors test did not reveal alterations in any of the eyes. A nuclear magnetic resonance (NMRI) of the brain with gadolinium gave normal results as well as a MRI with diffusion. The rest of the neurological assessment was normal. Due to a suspected intoxication of Topamax the medication was gradually withdrawn, with subjective improvements referred by the patient and partial recovery of the visual field (fig. 2).

Case report 2

A 24-year old woman referred by primary care due to bilateral loss of vision which started 6 weeks ago. The patient had no family history of interest. Her personal history includes treatment with 150 mg per day of Topamax started two months ago due to epilepsy which did not respond to conventional treatment. The maximum VA in the right eye was of 0.6 and 0.7 in the left eye (LE).

DISCUSSION

Topiramate (Topamax®) is an anti-epileptic drug introduced in Europe in 1995 for treating difficult to control epileptic conditions.
At present, the indications of Topamax have increased, being prescribed for treating obesity, as prophylaxis in migraines and in certain psychiatric diseases (2,3). The latest research on this drug describes increases in undesirable effects, particularly at the ophthalmological level although generally referring to ciliar-choroidal detachment with increase of myopia and glaucoma attacks due to angular closure (1,2). The Medline database comprises only one article referring to Homonime Hemianopsia (HH) due to Topamax (3) and a single reference to maculopathy due to Topamax (4). Foroozan et al (3) describe a patient (32 years old) who, with a 100 mg/day dosage developed in 6 weeks a HH condition and a certain degree of cognitive deterioration. The patient of the first case is very similar, starting with difficulty in speaking and slow movements, subsequently referring visual problems. In this patient no lesions were found. The visual field loss associated to slow motor and cognitive responses led us to suspect Topamax-induced toxicity.

Thompson et al (2) demonstrated that topiramate has a negative impact in the patient’s cognitive functions, particularly in the verbal capacity and in movements, this impact being dependent on the dosage and cumulative. Vaphiades et al (4) described a patient with Topamax-induced maculopathy, although they admit their lack of knowledge about the its mechanism of induction. The WHO database includes four cases of maculopathy associated to topiramate (data not published). The patient described in the second case previously was normal and developed maculopathy concurrently with the two-month treatment with Topamax.

The action mechanism of Topamax could be dual; on the one hand, it inhibits carbon anhydrase and on the other it enhances the inhibiting action of GABA, which exhibits an analogy with another anti-epileptic such as vigabatrine, which has a well-proven retinal toxicity (5), particularly in cones. Topamax is an efficient anti-epileptic but, considering the severity of the ophthalmological lesions (which could be irreversible), it is recommended to utilize this drug only with a strict and well justified reason, coupling it with neurological and ophthalmological control of the patient.

REFERENCES