Short communication

Uveal effusion induced by escitalopram


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ARTICLE INFO

Article history:
Received 1 September 2014
Accepted 6 November 2014
Available online 25 August 2015

Keywords:
Uveal effusion
Escitalopram
Antidepressants
Acute glaucoma
Acute myopia

ABSTRACT

Case report: A 73 year-old woman with depression treated with escitalopram developed acute secondary angle closure glaucoma related to uveal effusion after duplicating the drug dose 3 days before. She evolved favorably once the antidepressant treatment was suspended and a new treatment with topical hypotensive therapy and oral prednisone was used.

Discussion: The uveal effusion syndrome associated to medicines is rare; it may be associated with acute myopic shift and acute angle closure glaucoma. The correct diagnosis and discontinuation of the drug lead to the resolution of this nosology.

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EFUSIÓN UVEAL INDUCIDO POR ESCITALOPRAM

RESUMEN

Caso clínico: Mujer de 73 años en tratamiento con escitalopram que presentó glaucoma agudo de ángulo cerrado secundario a efusión uveal tras duplicar la dosis de dicho fármaco 3 días antes. Evolucionó favorablemente tras la suspensión del antidepresivo además de tratamiento hipotensor tópico y prednisona vía oral.

Discusión: La efusión uveal secundaria a fármacos es un síndrome infrecuente. Se puede acompañar de miopización y glaucoma agudo por cierre angular. El diagnóstico correcto y la suspensión del fármaco conducen a la resolución de esta nosología.

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Introduction

Uveal effusion or ciliochoroidal detachment is an abnormal accumulation of serous material derived from choriocapillary vessels between the sclera and the uvea. Diagnostic is based on Uyama’s criterion. Uveal effusion has been associated to a number of drugs such as sulphanamides, above all topiramate. A case of uveal effusion caused by escitalopram is presented, which was resolved with the interruption of administration and is the prescription of corticoids.

Case report

Female, 73, who visited the ophthalmological emergency department due to diminished visual acuity and frontal headache since the previous day.

The patient was under treatment with escitalopram due to depression. Three days before onset the dose had been doubled at 20 mg per day. Ophthalmic history comprised hypermetropia, with anteroposterior length of 22.5 mm in both eyes (BE) and bilateral pseudophakia.

Ophthalmological reports revealed –3D bilateral myopiaization with corrected visual acuity of 0.3 in the right eye and 0.16 in the left eye. Biomicroscopically, narrowed anterior chamber was observed with advanced intraocular lens (IOL) in BE (Fig. 1). Intraocular pressure (IOP) was of 29 and 30 mmHg, respectively. Ocular fundus revealed abundant chorioretinal folds in the posterior pole, with blisters in the anterior region at the exit of the vorticose veins (Fig. 2). Anterior pole ultrasound biomicroscopy and optic coherence tomography with Visante revealed bilateral choroidal detachment (Fig. 3).

The diagnostic was acute glaucoma secondary to uveal effusion due to escitalopram.

Treatment was established with timolol and brimonidine ocular hypotensors together with topical atropine, in addition to oral prednisone 1 mg per kg of weight in descending pattern.

Three weeks later, the patient exhibited a vision of 1 in BE, bilateral IOP of 14 mmHg and absence of liquid in the suprachoroidal space (Fig. 4).

Fig. 1 – Photograph taken at admission, showing IOL anteriorization with diminished anterior chamber depth.

Fig. 2 – Photograph taken with Starenghi wide field lens in HRA 2, showing anterior choroidal blisters (Arrow) reaching up to the emergence of the vorticose veins. The posterior region shows choroidal-retinal folds.

Fig. 3 – Photograph, showing ciliary body detachment and edema (white arrow), prolonged with chorioretinal effusion; ciliary body rotation produced the closure of the iridocorneal angle (red arrow).

Fig. 4 – Photograph taken after 3 weeks of evolution, showing the disappearance of the subchoroidal liquid, application of the choroids and the ciliary body to the sclera (white arrow), with the ensuing opening of the iridocorneal angle and increased anterior chamber depth.
Discussion

The single result of a literature search was a case secondary to escitalopram.4

Selective serotonin reuptake inhibitors (SSRIs) are the most widely used antidepressants. Escitalopram is an SSRI with 300 times greater affinity for serotoninergic receptors. It is metabolized through the liver by CYP2C19 enzyme and excretion is hepatic and renal.

Three of the 7 serotonin receptor families are in the eye: 5-HT7 causes midriasis, 5-HT1A diminishes the production of aqueous humor, while 5-HT2A/2C increases its secretion.5

SSRIs increase IOP approximately 4mmHg after a single dose. This variation does not affect healthy patients but could modify ocular function and anatomy in predisposed patients.

Expelled choroidal capillaries are permeable to water and proteins, although compensating mechanisms maintain the balance. Any deterioration of these processes due to different factors (drugs, traumatism, surgery) causes an accumulation of fluid in the suprachoroidal space.5

The patient reported herein exhibited choroidal blisters in the anterior regions and only retinal folds in the posterior pole. This could be because the vorticose veins limit uveal effusion into the anterior pole due to the presence of long fibers of connective tissue between the choroids and the sclera, in contrast with the short fibers of the posterior pole (Figs. 2 and 5).

Some drugs produce lens edema, giving rise to myopization.7 The latter effect was not observed in the present patient as she was pseudophakic. Acute myopia occurs due to the anterior rotation of the ciliary body, the iris and the IOL, increasing the distance between the lens and the retina. As the result of treatment and the subsequent application of the ciliary body, anterior chamber depth was recovered together with the adequate position of the bag-lens complex (Fig. 6).

The authors assume that escitalopram caused a dose-dependent adverse reaction that increased the production of aqueous humor which could not be compensated due to patient factors (hypermetropia). The accumulation of the edema produced uveal effusion with the anterior rotation of the iris and lens, causing myopization and diminished anterior chamber depth, with the subsequent angle closure inducing acute glaucoma.

Conservative treatment includes the administration of corticoids, NSAIDs and laser photoagulation. Prostaglandin analogs are not clinically proven.8 In the present case, even though corticoids were administered, the main factor in the treatment was suspending escitalopram.

If necessary, surgical treatment includes vorticose vein decompression and total or subscleral sclerotomy.

Escitalopram is a commonly prescribed drug and accordingly ophthalmologists should be aware that the adverse effects of this drug include the induction of uveal effusion with secondary acute glaucoma. This would allow quick and effective action. On the other hand, it is necessary to inform the patient about the adverse effects of this SSRI before prescribing it.

Conflict of interests

No conflict of interests was declared by the authors.

REFERENCES