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CASE REPORT

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Brimonidine Induced Hypertensive Acute Granulomatous Anterior Uveitis

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ABSTRACT

Brimonidine is a medication used to treat open-angle glaucoma, ocular hypertension, and rosacea. It is used as eye drops or applied to the skin. Common side effects when used in the eyes include itchiness, redness, and a dry mouth. We present a case of Brimonidine induced Hypertensive acute granulomatous anterior uveitis.

KEYWORDS: Brimonidine, Uveitis, Adverse Drug Reaction

INTRODUCTION

Some of the most common side effects of brimonidine are photophobia, conjunctival hyperemia, follicular conjunctivitis, itchy eyes, allergic conjunctivitis, and allergic blepharitis.[1,2] These can cause to patients discontinue treatment. Allergic reactions are more common with brimonidine than with Beta-blockers. They resolve with the withdrawal of treatment. Another less common but potentially more dangerous side effect than those mentioned previously is the occurrence of acute anterior granulomatous uveitis.[1-5] This uveitis often tends to be hypertensive and associated with bilateral follicular conjunctivitis.

CASE REPORT

A 78-year-old female who presented to us with a clinical picture of eye discomfort and bilateral eye redness for several days. The patient's systemic medical history included in particular the presence of non-insulin-dependent diabetes mellitus. Where ophthalmology is concerned, the patient had been receiving topical treatment with Brimonidine/timolol every 12 h by primary open-angle glaucoma from 2 years prior. She had undergone phacoemulsification in both eyes 6 years ago without complications. At the time of consultation, intraocular pressure (IOP) using applanation tonometry was 40 mmHg in the right eye (RE) and 42 mmHg in the left eye (LE). Best-corrected visual acuity was 1/10 in both eyes (BE). The slit lamp examination showed conjunctival hyperemia accompanied by signs of blepharitis and chronic conjunctivitis with a follicular reaction in the tarsi and inferior symblepharon in BE. The cornea presented with fine and diffuse epithelial keratitis; and thick, endothelial keratitic precipitates, with an appearance of mutton fat bilaterally. The anterior chamber was wide and there was an inflammatory reaction of 2+. The iris was normal, with no transillumination defects, with a round pupil without synechiae. The intraocular lens showed no alterations. There was no vitritis or inflammatory chorioretinal foci. Faced with bilateral anterior granulomatous hypertensive uveitis, we decided to start topical treatment with 1% prednisolone acetate 6 times daily in a descending regimen, cyclopentolate 3 times daily, artificial tears and a 1 g valacyclovir tablet three times daily. While there was no infectious ophthalmological history, given that the uveitis was hypertensive, antiviral treatment was started for possible herpes. Two days after the consultation in the emergency room IOP was 46 mmHg in the RE, and 44 mmHg in the LE. The anterior pole was of the same appearance as in the previous visit and the inflammation of the anterior chamber continued. The systemic diagnostic screening for uveitis and HLA-B27 typing were negative. The irritative reaction presented by the conjunctiva in both eyes and the fact that uveitis was bilateral with negative results in the systemic diagnostic screening for uveitis made us consider the possibility of brimonidine-associated hypertensive uveitis on finding literature that supported us in our clinical suspicion. For this reason, the suspect drug was discontinued and a fixed combination of dorzolamide and timolol was added. The corticosteroid eye drops regimen cyclopentolate, was continued and artificial tears and antiviral treatment via the oral route was discontinued. Two weeks after withdrawing brimonidine, the best

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corrected visual acuity was 2/10 in RE, and 5/10 in LE. The IOP 25 and 24 mmHg in RE and LE respectively. The conjunctival hyperemia had disappeared, the corneal epitheliopathy had lessened, as well as the mutton-fat precipitates in the endothelium. There was no inflammation in the anterior chamber. Due to the absence of inflammation in the anterior chamber and the clear improvement of the endothelial precipitates, we decided to add a single daily of latanoprost to the treatment. One week after incorporating prostaglandin to the topical treatment and three weeks after withdrawing brimonidine, IOP was 20 mmHg in BE. The endothelial precipitates were sparse and pigmented and the corneal epithelium was of better appearance. There was no inflammation in the anterior chamber. Three months after withdrawing brimonidine, IOP was 17 mmHg in the RE and 14 mmHg in the LE. The corneal epithelium showed no positive lesions in the fluorescein stain and with regard to the endothelium, residual remnants of the keratic precipitates persisted. The descending corticosteroid treatment regimen had ended 2 months prior, and there was no evidence of new inflammatory activity.

DISCUSSION

The pathophysiology of brimonidine-induced anterior uveitis is unknown. A correlation has been established in the literature between allergic conjunctival reactions and predisposition to anterior uveitis.[2] The first manifestations of allergic conjunctivitis from brimonidine usually appear between 6 and 12 months after the commencement of treatment. It is thought that there is a decrease in the density of the conjunctival cells associated with an enlargement of the intercellular spaces induced by alpha-adrenergic agonists, thus favoring the flow of the mediators of inflammation responsible for the allergic reaction.[2]

Some authors argue that uveitis appears systematically after a phase of chronic allergic conjunctivitis and would be related to prolonged application of treatment despite ocular surface irritation symptoms. This could justify the lower frequency of anterior uveitis cases with respect to the cases of brimonidine induced conjunctivitis diagnosed in daily clinical practice. The increase of proinflammatory cytokines in the aqueous humor after a long period of brimonidine treatment may contribute to the development of brimonidine-induced uveitis, and therefore be an essential part of its pathophysiology.[3] The literature suggests that there is usually a time frame of between 6 to 18 months from starting treatment with brimonidine until the anterior granulomatous uveitis appears, with a run-in phase of allergic conjunctivitis. There is a predominance of females in the patients affected and in a third of cases the uveitis is hypertensive.

Once the suspected diagnosis is reached, the effective treatment is withdrawal of the brimonidine eye drops, with the addition or not topical corticosteroids to control the inflammatory process, depending on the severity of clinical picture.

CONCLUSION

Acute anterior granulomatous uveitis secondary to treatment with eye drops containing brimonidine is a rare adverse effect, but one that must be considered. In general, there is little knowledge amongst ophthalmologists of this potential effect of brimonidine. It would be worthwhile for medical professionals who prescribe this drug to indicate to their patients the need to seek medical attention rapidly if symptoms compatible with the disease appear. It is a process with an excellent prognosis. Therefore, in cases with a strong clinical suspicion that the uveitis is induced by brimonidine, withdrawal of the eye drops is the step required for the definitive diagnosis and corresponds to the treatment itself.

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