CASE REPORT

Bimatoprost (0.03%)-induced accommodative spasm and pseudomyopia

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SUMMARY

Bimatoprost is a prostaglandin analogue used topically in the treatment of glaucoma. Commonly known side effects include eyelash growth, iris pigmentation and conjunctival hyperemia. While pseudomyopia is reported to be caused by parasympathomimetics, such an effect precipitated by bimatoprost has not yet been reported. We report a case demonstrating pseudomyopia and accommodative spasm caused after starting bimatoprost 0.03% in a young patient with glaucoma.

BACKGROUND

Bimatoprost is an ocular hypotensive that is considered to be a prostamide rather than an ester such as latanoprost or travoprost.¹ It reduces the intraocular pressure (IOP) in humans by increasing outflow of aqueous humour through the trabecular meshwork and uveoscleral routes. It has been associated with many ocular and systemic side effects,^{1 2} however, accommodative spasm due to this drug is unknown.

CASE PRESENTATION

A 47-year-old man with a history of primary open angle glaucoma (POAG) presented to us with inability to focus on distant objects while wearing his regular glasses, for 5–7 days, with relatively fewer problems with his near vision. His glasses (near add +1.5DS) had been prescribed 1 year prior. The problems began 2 weeks earlier, after he was started on bimatoprost 0.03% in both eyes once at night. On examination, his unaided visual acuity was 20/30 in the right eye and 20/50 in the left eye with near vision N6 in both eyes. Refraction showed a myopic shift of $-1.00\text{DS}/-0.5\text{DC}\times 90^\circ$ in the right eye and -1.25DS in the left eye (with near add +1.75DS, N6 both eyes). His previous prescription for glasses included only near addition of +1.75DS in both eyes with no distance correction. Slit lamp evaluation showed clear lens and open angles on gonioscopy in both eyes, with no anterior chamber activity. His IOP by Goldmann applanation tonometry was 18 and 20 mm Hg in right and left eye, respectively. Fundus evaluation showed glaucomatous cupping with cup disc ratio of 0.8:1 with inferior notch in the right eye and 0.9:1 with bipolar notch with corresponding visual field defects.

INVESTIGATIONS

Cycloplegic refraction (1% cyclopentolate) was repeated, which showed complete nullification of the myopia seen on dry refraction. This suggested

accommodative spasm as the cause for the refractive error seen in the undilated state. In view of sudden onset myopia starting after initiation of the bimatoprost drops, a possibility of drug-induced accommodative spasm was considered and the patient was reviewed the next day in an undilated state for accommodative amplitude to confirm the diagnosis. Ultrasound biomicroscopy was advised, which was, however, unsuccessful, because the patient was unable to cooperate.

Table 1 shows the biometry parameters, which were suggestive of accommodative spasm.

TREATMENT

Since the patient was highly symptomatic in his daily office work (he was a clerk by profession), we discussed the possibility of bimatoprost-induced accommodative spasm and switched his treatment to brominidine 0.15%. Since he was an active professional with routine near sight work, he opted against use of intermittent cycloplegics and chose to stop the drug and observe the symptoms.

OUTCOME AND FOLLOW-UP

The patient was reviewed 2 weeks later and had an IOP of 10 mm Hg on the aforementioned treatment in both eyes. His accommodative amplitude and other parameters had improved significantly with complete resolution of symptoms for distance (table 2). He had no recurrence of symptoms with the aforementioned treatment and final IOP was 10 mm Hg at follow-up of 3 months.

DISCUSSION

Prostaglandin (PG) analogues comprise the firstline of drugs in glaucoma medical treatment owing to their greater IOP reduction. These molecules are physiologically involved in the inflammatory process.^{3–6} Since they are inflammatory mediators causing disruption of the aqueous barrier by release of arachidonic acid, their ocular side effects suggestive of inflammatory process after topical administration include flare, hyperaemia, cystoids macular oedema, anterior uveitis and reactivation of herpetic infections.^{5 7–11} To the best of our knowledge, accommodation spasm by bimatoprost, however, has not been reported earlier. The proinflammatory effect of PG analogues makes them unsuitable for treatment of patients with underlying inflammation.^{2 4} Our patient did not have any underlying inflammation at the time of presentation or later. Subtle early inflammation causing ciliary irritation may presumably be responsible for the accommodative spasm in our patient, which

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Table 1 Biometry parameters with the use of topical bimatoprost 0.03%

Parameters	OD	OS
Axial length (mm)	24.6	24.7
Anterior chamber depth (mm)	3.05	2.98
Lens thickness (mm)	4.09	4.25
Amplitude of accommodation (dioptres)	6.00	6.00

therefore, in our case, resolved after stopping the offending drug, without the need for cycloplegics. It is, however, unclear if this is an idiosyncratic reaction or dose dependent, which may be confirmed by pharmacokinetic studies.

Anterior chamber flare has been reported in patients treated with latanoprost.⁶ Though all three, including bimatoprost, travoprost and latanoprost, induce hyperaemia, apart from many other ocular side effects,^{4 6} Cellini *et al*⁷ postulated that eyes treated with bimatoprost and travoprost have less inflammation and fewer disruptions of the blood aqueous barrier than other PG analogues. Stjernschantz⁹ suggested that travoprost and bimatoprost may have higher incidence of hyperaemia owing to difference in chemical structure. We feel that our case did not demonstrate significant inflammation because the changes may be more pronounced at the ciliary level rather than iris level, therefore making it difficult to observe anterior segment inflammation.

Guenoun *et al*¹⁰ showed that the direct toxic effect of PG analogues was mild and theorised that most of the side effects may be mediated using preservatives such as benzalkonium chloride (BAC). We are unsure if the accommodative spasm induced in our patient was a result of preservatives in the drug (BAC in bimatoprost as compared to purite in brimonidine drops). Nevertheless, the pro-inflammatory effect in clinical practise is not uncommon. Uveal effusion has also been reported after travoprost and latanoprost.¹¹ It is possible that a very mild inflammation may have presumably caused swelling of the ciliary body causing forward displacement of iris and lens, further causing accommodative spasm. We are unsure if a lower concentration of bimatoprost drops (0.01%) would have caused similar effects with less inflammation. Ciliary irritation may be another possibility for induced spasm of the muscle. Absence of obvious

Table 2 Change in biometry parameters after stopping bimatoprost 0.03%

Parameters	OD	OS
Axial length (mm)	24.6	24.7
Anterior chamber depth (mm)	3.15	3.07
Lens thickness (mm)	3.9	4.1
Amplitude of accommodation (dioptres)	5.00	5.00

inflammation in our case could also imply a direct toxic effect of the drug on the ciliary body rather than iris. These explain the therapeutic resolution of the spasm as seen on improvement in biometric properties and refraction complemented with complete relief from symptoms.

Learning points

- Patients started on prostaglandin analogues should be advised to report any signs of blurred vision especially in the first few days of treatment. Accommodative spasms by direct effect of the drug on the ciliary muscle must be kept in mind in patients who are active professionals.
- Cycloplegic and dry refraction with measurement of accommodation amplitude would help confirm accommodation spasm.
- Bimatoprost 0.03% may have a direct toxic effect without evidence of anterior segment inflammation.
- Stopping the drug may help resolve the symptoms, if detected early.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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