Bilateral Choroidal Detachment in the Absence of Previous Intraocular Surgery

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Keywords
Choroidal detachment · Choroidal effusion · Acute angle-closure glaucoma · Dorzolamide · Timolol

Abstract
The aim of this report was to present a case of bilateral choroidal detachment following treatment with topical therapy dorzolamide/timolol without history of previous surgery. An 86-year-old woman, with intraocular pressures of 40.00/36.00 mm Hg, was treated with preservative-free double therapy with dorzolamide/timolol. One week later, she presented with bilateral vision loss and irritative symptoms in the face, scalp, and ears, with well controlled pressures. The anterior exam showed LOCS III N4C3 cataracts, and the fundus and ultrasound exams revealed a bilateral infero-temporal choroidal detachment in the absence of neoplasia or other systemic cause. One week in absence of hypotensive treatment and receiving topical prednisolone, she showed reattachment of the choroidal detachment. Six months after cataract surgery, the patient remains stable, without choroidal effusion regression. Hypotensive treatment following chronic angle closure can lead to choroidal effusion similar to cases of acute angle closure treated with oral carbonic-anhydrase inhibitors. The combined strategy of removing hypotensive treatment and topical corticosteroids could be useful for the initial management of choroidal effusion. Also, performing cataract surgery after choroidal reattachment can help with stabilization.
Introduction

The goal of medical management of glaucoma is to lower intraocular pressure through topical or oral medication, laser therapy, or surgery. Glaucoma surgery involves different techniques to decrease the intraocular pressure in patients whose disease is not well controlled with topical treatment. Filtration surgery has been related to numerous postoperative complications derived from the sudden drop in intraocular pressure. This effect can be increased by some topical medications that decrease the formation of aqueous humor. Double topical therapy with beta-blockers and carbonic-anhydrase inhibitors has been widely spread following glaucoma surgery. Among the complications derived from the sudden drop in intraocular pressure is choroidal detachment [1–3].

Several cases of choroidal detachment associated with hypotensive topical treatment after glaucoma drainage device implantation or trabeculectomy have been described. This effect tends to be produced by the marked hypotensive effect of glaucoma surgery, enhanced by the decrease in the formation of aqueous humor provided by this drug.

Yang et al. [1] described a case series of uveal effusion after acute primary angle-closure glaucoma. These were associated with treatment with oral acetazolamide and pilocarpine and are attributed to the anatomical changes of the iris-crystalline diaphragm after a marked hypotensive effect. However, few cases of choroidal detachment after the application of topical dorzolamide/timolol, without a history of previous intraocular surgery or systemic treatment with carbonic-anhydrase inhibitors have been described in the literature [4].

Case Report/Case Presentation

An 86-year-old woman was admitted in the Glaucoma Department with progressive symptoms of visual loss and redness in the right eye, associated with erythematous face, ears, and scalp after 1 week of topical treatment with dorzolamide/timolol 20 mg/mL + 5 mg/mL (Duokopt, Laboratoires Thea, Clermont-Ferrand Cedex, France) in both eyes. Treatment was initiated in a different hospital due to the incidental finding of intraocular pressure 40.00 mm Hg in the right eye and 36.00 mm Hg in the left eye. Her personal medical history included arterial hypertension, hypercholesterolemia, and Parkinsonism, which were already under treatment with acetylsalicylic acid 100 mg, ezetimibe/rosuvastatin 10 mg/10 mg, carbidopa monohydrate/levodopa 25 mg/100 mg, and amlodipine besilate/olmesartan medoxomil 40 mg/5 mg.

Best-corrected visual acuity was 0.1 in the right eye (RE) and 0.3 in the left eye (LE) (Table 1). She presented intraocular pressure (Goldmann) of 17.00 mm Hg RE and 16.00 mm Hg LE and normoreactive pupils after 3 days without topical hypotensive treatment. The anterior segment exam revealed: anterior chamber depth grade 2 (Van Herick), cortical and nuclear cataract (LOCS III N4C3), and pseudoexfoliation in both eyes (Fig. 1). She presented punctata keratitis in the exposure area, more pronounced in her left eye. The fundus exam showed slightly pallid optic discs, with cup/disc ratio 0.4 and prominent lobulated inferotemporal choroidal detachments in both eyes, no alterations were observed in the retinal vessels or macula. Abnormalities were also seen in the B-scan ultrasonography and macular optical coherence tomography (Fig. 2, 3). The 24-2 visual field test (Humphrey, Carl Zeiss Meditec, Dublin, CA) revealed severe constriction of the visual field in RE and a central isolated preserved area in LE. (RE: VFI 0%, MD −30.83 dB, PSD 1.75 dB, LE: VFI 20%, MD −26.21 dB, PSD 6.97 dB). Iridocorneal angle opening in both eyes was grade 2 of Shaffer’s classification. Based on these findings, an expectant attitude without topical treatment was adopted and treatment with topical prednisolone was initiated.
One week later, visual acuity remained stable, intraocular pressure rose to 38.00 mm Hg RE and 36.00 mm Hg LE, and choroidal detachments remitted. Thus, topical treatment with Bimatoprost 0.3 mg/mL was initiated in both eyes. Since pressure decreased to 24.00 mm Hg RE and 22.00 mm Hg LE and fundus exam remained stable, performing phacoemulsification and monofocal intraocular lens implantation was decided.

Table 1. Clinical data of the case report

| Day       | BCAV       | IOP       | BMC                      | Fundus                      | Treatment                      |
|-----------|------------|-----------|--------------------------|-----------------------------|--------------------------------|
| Day 0     | 0.10/0.20  | 17/16     | Cataract (LOCS III N4C3), VH2, Shaffer II, PEX | Bilateral choroidal detachment infero-temporal, E/P 0.4 pallor | Prednisolone                     |
| Day 7     | 0.05/0.20  | 38/36 =X  | No choroidal detachment, E/P 0.4 pallor | Bimatoprost 0.3               |
| Day 14 Surgery | 0.05/0.20  | 24/22  | = | r | Cataract surgery |
| Day 1 post-op | 0.17/0.24  | 24/22  | Pseudophakia, VH4, Shaffer III-IV, PEX | No choroidal detachment, E/P 0.4 pallor | Ciprofloxacine, dexamethasone, latanoprost |
| Day 30 post-op | 0.17/0.24  | 22/21  | = | r | Timolol 0.5 mg/mL + bimatoprost 0.2 mg |
| Day 40 post-op | 0.17/0.24  | 16/15  | X | r | Timolol 0.5 mg/mL + bimatoprost 0.2 mg |

BCAV, best-corrected visual acuity; IOP, intraocular pressure; BMC, biomicroscopy; =, remains stable.

Fig. 1. Anterior segment exam. Shallow anterior chamber, cortical and nuclear cataract (LOCS III N4C3), pseudoexfoliation and superficial punctata keratitis in the exposure area, more pronounced in her left eye.
Cataract surgery was performed with no significant findings. Visual acuity after surgery improved to 0.17 RE and 0.24 LE. Intraocular pressure was 24.00 mm Hg RE and 22.00 mm Hg LE 24 h after surgery, so treatment with topically latanoprost 50 μg/mL was initiated with good response. However, 1 month after surgery, the pressure rose again to 22.00 and 21.00 mm Hg. Then, treatment was increased to bimatoprost/timolol 0.3 mg/mL + 5 mg/mL. Patient remained stable 6 months after surgery with no progression in the visual field and no changes in the fundus exam.

**Discussion/Conclusion**

The topic agent employed to reduce the intraocular pressure should be selected according to the current type of glaucoma. Thus, some drugs increase the elimination of aqueous humor and other reduce its formation. The selection of the hypotensive agent becomes more relevant after glaucoma surgery. There is evidence in the literature of some cases of choroidal effusion with topical or systemic carbonic-anhydrase inhibitor drugs [2–5] and prostaglandin analogs [6–9] after trabeculectomy surgery and glaucoma drainage device implantation. However, there are few cases of choroidal detachment related to topical hypotensive medication in the absence of an intraocular surgical history.
Goldberg et al. [4] reported one case of choroidal detachment in a 76-year-old woman with open angle glaucoma, 12 h after initiation of dorzolamide eyedrops. Withdrawal of the drug and initiation of corticosteroid drops resulted in prompt resolution of the choroidal detachment. In this case report, the presence of a narrow angle with probable intermittent angle closures related to environmental circumstances and the fact of not having previously used any hypotensive treatment could have caused an enhanced effect of both drugs, inhibitors of aqueous humor production [2].

Between 2015 and 2018, some case series of choroidal effusion after initiation of systemic hypotensive therapy for primary acute angular closure were reported [1, 10, 11]. The choroidal vessels’ congestion during the startup hypertensive episode, as well as the choroidal expansion and reduction of hydrostatic pressure gradient produced after the beginning of systemically hypotensive treatment, lead to choroidal effusion. Other implicated mechanisms are edema and anterior rotation of the ciliary body, and anterior displacement of the lens-iris diaphragm [1]. Topical or systemic corticosteroids along with the interruption of hypotensive treatment have been described as two efficacy strategies to achieve the choroidal reattachment [1, 10, 11].

Recently, cases of choroidal effusion produced by zonisamide and other sulfa-derived drugs with or without acute angle closure has been reported [12]. Additionally, three types of choroidal detachment after surgery have been reported. Berke et al. [13], described that all subjects showed a complete resolution of choroidal detachment after performing cataract surgery.

In most cases, entities such as the plateau iris should be valued. Besides, the presence of neoplastic or space-occupying lesions that could justify the presence of folds or choroidal detachment should be dismissed through ultrasonography and magnetic resonance imaging. The reported case has some limitations such as an associated ischemic neuritis, which is suspected due to the cardiovascular history of the patient and the presence of slightly pale optical discs, as well as the absence of study through ultrasonic biomicroscopy (UBM) to detect the ciliary body and lens-iris diaphragm changes.

**Statement of Ethics**

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. The research was conducted in accordance with the tenets of the Declaration of Helsinki. Ethical approval is not required for this study in accordance with local or national guidelines.

**Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

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**Author Contributions**

Maria J. Chaves-Samaniego, Pedro Amat Peral, and José M. Ruiz Moreno observed and followed the subject of the case report and contributed to the conception and design of the study. Zaira Cervera Sanchez and Aleyda Molina Lespron provided and interpreted the patient data.
and were involved in drafting the work. Maria J. Chaves-Samaniego and Aleyda Molina Lespron revised critically the manuscript. All the authors read and approved the final manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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