Bilateral Ocular Hypotony Secondary to Severe Dehydration and Uremia

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Dear Editor,

Ocular hypotony, defined as a sustained intraocular pressure (IOP) below approximately 6 mmHg, is a pathological condition that may compromise ocular structure and function. Prolonged hypotony can result in irreversible visual impairment through mechanisms such as corneal edema, choroidal effusion, retinochoroidal folds, and hypotonic maculopathy. Although surgical, traumatic, and inflammatory causes are most frequently encountered, systemic disturbances may also impair aqueous humor dynamics. Rare cases have been reported in association with severe dehydration, hypovolemia, and uremia, in which reduced aqueous humor production appears to play a central role.[1,2,3,4]

Herein, we report a case of bilateral ocular hypotony following severe systemic dehydration secondary to gastrointestinal bleeding. A 73-year-old man presented with bilateral ocular pain and decreased vision. Best-corrected visual acuity, assessed using a Snellen chart and subsequently converted to logarithm of the minimum angle of resolution (logMAR) units, was 1.00 logMAR (20/200) in the right eye and 0.30 logMAR (20/40) in the left eye. Goldmann applanation tonometry revealed an IOP of 5 mmHg in both eyes. Slit-lamp examination showed conjunctival hyperemia, Descemet membrane folds, and +2 anterior chamber cells bilaterally (Figure 1). Gonioscopy demonstrated open angles without evidence of cyclodialysis cleft or angle recession, with blood observed within Schlemm's canal (Figure 2). The right eye exhibited a dense corticonuclear cataract, whereas nuclear sclerosis was noted in the left. Fundus examination was limited in the right eye because of lens opacity but revealed normal optic discs and maculae, with attached retina and choroid in both eyes. No clinical signs of hypotonic maculopathy were identified. B-scan ultrasonography and optical coherence tomography showed a normal posterior segment in both eyes (Figure 3). Despite the presence of an anterior chamber reaction, no keratic precipitates, fibrin formation, posterior synechiae, or posterior segment inflammatory findings were observed.

The patient had experienced an upper gastrointestinal hemorrhage approximately 2 weeks earlier. Emergency endoscopy revealed a Forrest IIa gastric ulcer, and endoscopic hemostasis was performed. During subsequent follow-up, marked fluid loss and deterioration in hematological parameters necessitated intensive care admission. Laboratory findings demonstrated severe dehydration, anemia, and uremia secondary to acute kidney injury. Values (with normative ranges) were as follows: hemoglobin 7.8 g/dL (13.5-16.9), hematocrit 24.4% (40.0-49.4), serum creatinine 1.51 mg/dL (0.7-1.3), urea 83.46 mg/dL (19-49), and estimated glomerular filtration rate (eGFR) 45.18 mL/min/1.73 m^2 (>90). Serum sodium level was 142 mmol/L at presentation and remained stable during follow-up. Calculated serum osmolarity at presentation

Keywords: Ciliary ischemia, dehydration, hypovolemia, ocular hypotony

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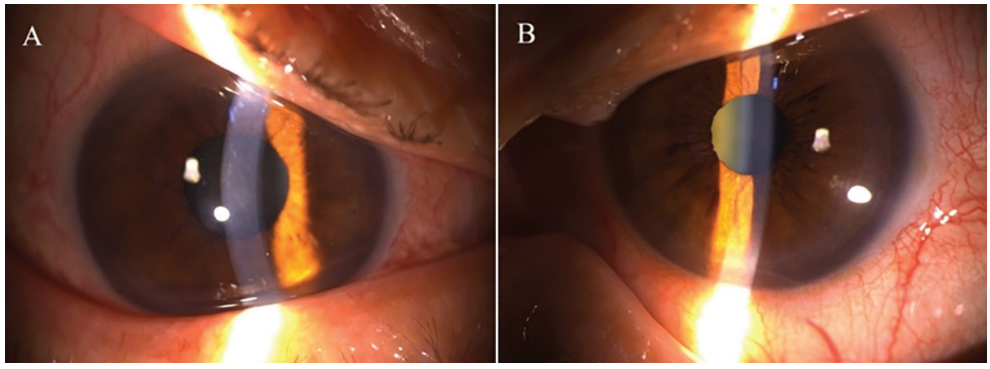

Figure 1. Anterior segment examination showed bilateral conjunctival hyperemia, corneal Descemet membrane folds, and +2 anterior chamber cells (A: right eye, B: left eye)

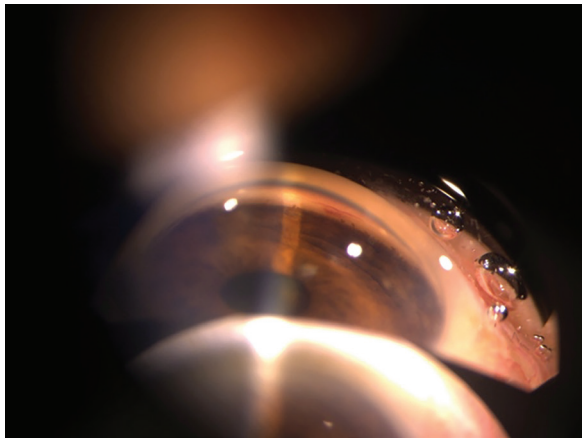

Figure 2. Gonioscopic examination revealed open angles in both eyes, with notable visualization of blood within Schlemm's canal in the left eye

was approximately 307 mOsm/kg, consistent with hyperosmolarity related to dehydration and uremia. The patient received intravenous fluid replacement, erythrocyte suspension transfusion, a proton-pump inhibitor (PPI), and intravenous iron supplementation. He was discharged after gradual systemic improvement on oral PPI and iron therapy.

No topical or systemic medications known to reduce aqueous humor production or to precipitate ocular hypotony were identified before or during the hypotony episode. Accordingly, the bilateral ocular hypotony was primarily attributed to hypovolemia-induced impairment of ciliary body perfusion. In addition, uremia-related osmotic alterations may have further exacerbated the hypotony. Topical dexamethasone 0.1% (Maxidex®, Novartis, Puurs, Belgium) was administered hourly together with atropine sulfate 1% (Atropin Sülfat®, Biofarma, İstanbul, Türkiye) three times daily. Systemic corticosteroids were withheld because of the recent gastrointestinal bleeding.

After 1 week, systemic and ocular findings improved, with IOP values of 8 mmHg and 11 mmHg, respectively. Conjunctival hyperemia, corneal folds, and anterior chamber inflammation decreased markedly. At the 1-month follow-up, IOP stabilized at 10-11 mmHg in both eyes. Endothelial pigment deposition diminished, and corneal folds and inflammatory cell infiltration resolved completely. Topical medications were gradually tapered and discontinued. Final laboratory tests confirmed systemic recovery, with hemoglobin 11.4 g/dL, hematocrit 38.2%, serum creatinine 1.10 mg/dL, urea 45.36 mg/dL, and eGFR 66.26 mL/min/1.73 m^2.

This case illustrates a fully reversible episode of bilateral ocular hypotony secondary to systemic dehydration and hypovolemia, further exacerbated by uremia associated with acute kidney injury. The concurrence of volume depletion and azotemia likely resulted in transient ciliary body hypoperfusion and suppression of aqueous humor production. The marked reduction of IOP to 5 mmHg, in the absence of prior ocular surgery, trauma, or hypotony-inducing medications, together with gradual normalization following systemic rehydration and renal recovery, supports transient aqueous hyposecretion due to ciliary ischemia as the principal mechanism.

In states of profound dehydration or hypovolemia, reduced perfusion of the ciliary circulation and altered plasma osmolality may impair aqueous production.[1,2,4] As described by Wang et al.[1], hypotony may arise from reduced aqueous production or increased aqueous outflow. In this patient, normal gonioscopic findings without cyclodialysis or angle recession favored a production-deficient mechanism. The presence of blood in Schlemm's canal was interpreted as a reflux phenomenon caused by a relative pressure gradient in the setting of marked hypotony, whereby episcleral venous pressure exceeded IOP, rather than a primary elevation of episcleral venous pressure. Similar cases reported by Schleis and Atanasoff[2] demonstrated IOP normalization

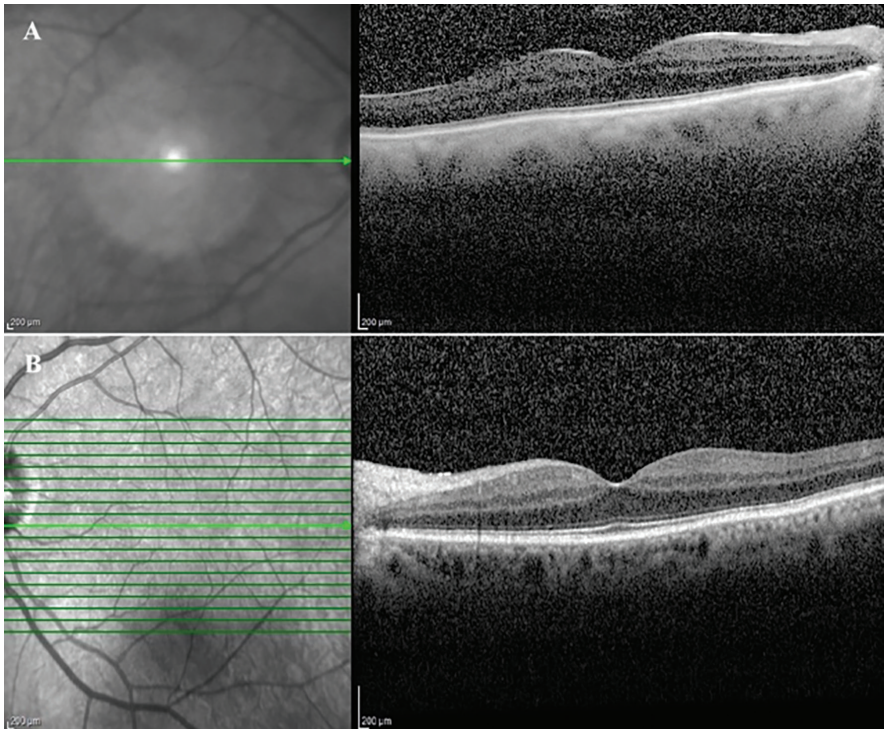

Figure 3. Macular optical coherence tomography. A) Right eye: Reduced image quality due to dense cataract, with preserved retinal architecture and no hypotony maculopathy. B) Left eye: Normal foveal contour and retinal layers, with no hypotony maculopathy

following intravenous fluid therapy, supporting this pathophysiologic explanation.

Although prolonged hypotony carries a risk of hypotonic maculopathy and visual loss,[4,5,6] early recognition and prompt systemic correction likely prevented structural macular changes in this patient. In hypovolemia-associated cases, restoration of intravascular volume and hemodynamic stability remains the cornerstone of management. In our case, systemic rehydration was considered the primary therapeutic intervention responsible for restoration of IOP. Topical therapy was used as an adjunctive treatment to mitigate ciliary body ischemia-related ocular inflammation and to support recovery of aqueous humor production.

In conclusion, dehydration-associated ocular hypotony may be fully reversible when promptly recognized and appropriately managed. Routine IOP monitoring should be considered in systemically compromised patients, particularly those with acute volume depletion or renal dysfunction. Timely diagnosis and systemic correction are essential to preserve visual function and prevent irreversible hypotonic complications.

Ethics

Informed Consent: Written informed consent was obtained from the patient.

Declarations

Authorship Contributions

Surgical and Medical Practices: A.M.K., Concept: B.E.A., A.M.K., Design: B.E.A., A.M.K., Data Collection or Processing: B.E.A., M.M.U., A.M.K., K.D., Ç.Ç., Analysis or Interpretation: B.E.A., A.M.K., Literature Search: B.E.A., A.M.K., Writing: B.E.A., A.M.K.

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