Hydrochlorothiazide-Induced Hyponatremia: A Bilateral Acute Angle Closure Glaucoma Case Report

Hiponatrémia Induzida por Hidroclorotiazida: Um Caso Clínico de Glaucoma Agudo de Ângulo Fechado Bilateral

Sofia Teixeira [1], Dália Meira [1], Renata Rothwell [1]

[1] Department of Opthalmology, Centro Hospitalar Vila Nova de Gaia/Espinho, Portugal

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ABSTRACT

Our objective is to report a case of bilateral acute glaucoma associated with hydrochlorothiazide-induced hyponatremia.

An 83-year-old female patient, with arterial hypertension history, presented with severe ocular pain and decreased vision in both eyes, associated with headache, nausea and vomiting.

On examination, visual acuity was counting fingers in both eyes. Biomicroscopy showed ciliary hyperemia, marked corneal edema and shallow anterior chamber. Intraocular pressure (IOP) was 59/68 mmHg. Gonioscopy revealed bilateral angle closure and UBM ciliary body effusion. Antihypertensive eye drops and intravenous mannitol were started. Blood tests revealed profound hyponatremia and hypochloremia. After electrolyte stability, laser iridoplasty was performed in both eyes, and previous hydrochlorothiazide prescription was suspended. Thereafter, progressive clinic improvement was verified, with IOP normalization.

Bilateral acute angle closure has been associated with sulfonamide derivative medications. The development of bilateral ciliary effusions and angle closure potentially due to hydrochlorothiazide-induced-hyponatremia, had been reported only once in literature.

KEYWORDS: Antihypertensive Agents/adverse effects; Glaucoma, Angle-Closure/chemically induced; Hydrochlorothiazide/adverse effects; Hyponatremia/chemically induced.

RESUMO

O nosso objetivo é relatar um caso de glaucoma agudo bilateral associado à hiponatremia induzida por hidroclorotiazida.

Paciente do sexo feminino, 83 anos, com antecedentes de hipertensão arterial, apresenta dor ocular intensa e diminuição da visão em ambos os olhos, associada a cefaleia, náuseas e vômitos.

Ao exame físico, apresentava acuidade visual de conta dedos em ambos os olhos. A biomicroscopia revelou hiperemia ciliar, edema corneano acentuado e câmara anterior plana. A pressão intraocular (PIO) era de 59/68 mmHg. A gonioscopia mostrou ângulo fechado bilateralmente e a UBM efusão do corpo ciliar. Foram iniciados colírios anti-hipertensivos e manitol endovenoso. O

estudo analítico revelou hiponatremia profunda e hipocloremia. Após estabilidade eletrolítica, foi realizada iridoplastia laser bilateral, sendo suspensa a prescrição prévia de hidroclorotiazida. A partir daí, verificou-se melhora clínica progressiva, com normalização da PIO.

O encerramento de ângulo agudo bilateral tem sido associado a medicamentos derivados de sulfonamidas. O desenvolvimento de efusão ciliar bilateral e encerramento do ângulo, potencialmente devido à hiponatremia-induzida-pela-hidroclorotiazida, foi relatado uma única vez na literatura.

PALAVRAS-CHAVE: Anti-Hipertensivos/efeitos adversos; Glaucoma de Ângulo Fechado/induzido quimicamente; Hidroclorotiazida/efeitos adversos; Hiponatrémia/induzida quimicamente.

INTRODUCTION

Hydrochlorothiazide is today the most widely used thiazide-type diuretic, and has been used for several decades to treat hypertension. It inhibits the sodium chloride co-transporter system on distal convoluted tubules, leading to potassium loss in urine. Thus, it lowers peripheral vascular resistance and inhibits the retention of water by the kidneys.[1] It is considered a safe medication, despite several adverse effects reported as orthostatic hypotension, pancreatitis, nausea and vomiting, diarrhea, aplastic anemia, leukopenia, thrombocytopenia, fever, paraesthesias, pulmonary edema, electrolyte imbalance (including hypokalemia, hyponatremia and hypercalcemia), among others.[1]

Furthermore, sulfonamide derivative medications, like topiramate, acetazolamide and hydrochlorothiazide, have already been associated with the development of bilateral acute angle closure, through an idiosyncratic reaction, which causes transient myopia and acute angle-closure glaucoma.[2] In a still somewhat unknown and controversial mechanism, it is thought that the angle closure may be secondary to anterior uveal effusions and anterior rotation of the ciliary body that subsequently creates a forward displacement of the iris-lens diaphragm.[3]

Bilateral angle closure glaucoma due to ciliary effusions in the context of sudden induced hyponatremia by hydrochlorothiazide has anedectolly been reported in the literature. To our knowledge, the case occurred in an inuit woman,[3] and its mechanism is still mostly unknown.

This study goal is to report a case of bilateral acute glaucoma potentially associated with hydrochlorothiazide-induced hyponatremia.

CASE REPORT

An 83-year-old female patient presented at our emergency department with severe ocular pain and decreased visual acuity (VA) in both eyes, associated with headache, nausea and vomiting. She had history of arterial hypertension, dyslipidemia, multinodular goiter and bronchiectasis. Her chronic medication was amlodipine, candesartan, hydrochlorothiazide, atorvastatin, euthyrox and cetirizine, and she denied recent changes in her medical prescription.

On ophthalmological observation, visual acuity was counting fingers at 30 cm in both eyes. Biomicroscopy showed bilateral diffuse ciliary hyperemia, chemosis, marked corneal edema, shallow anterior chamber and nuclear cataracts (Figs. 1A and B). Gonioscopy revealed bilateral angle closure. Intraocular pressure (IOP) was 59/68 mmHg (rebound tonometer). UBM showed bilateral ciliary body effusion with anterior rotation of the ciliar body (Figs. 2A and B). She was started immediately on topical hypotensive drugs, atropine and dexamethasone, intravenous mannitol and paracetamol. Meanwhile, blood tests, requested due to some somnolence of the patient, revealed severe electrolyte imbalance with profound hyponatremia (Na^+ 126 mmol/L, with K^+ 4.28 mmol/L) and hypochloremia (Cl^- 93.2 mmol/L). After electrolyte stability, laser iridoplasty was performed in both eyes. The patient was discharged with topical dexamethasone, atropine, timolol and dorzolamide. Oral hydrochlorothiazide was suspended.

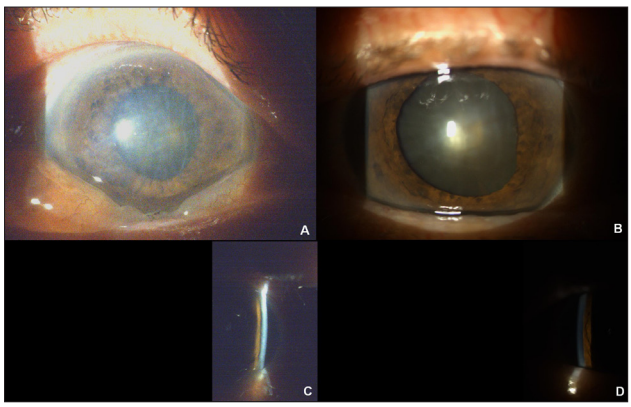

Figure 1. Photograph of the anterior surface of the eye, revealing marked corneal edema in both eyes (A, B); with shallow anterior chamber (C, D).

On the next day, electrolyte balance and intraocular pressure had returned to normal values. In subsequent reassessments, progressive clinical improvement and pain resolution, with normalization of IOP, allowed gradual tapering of the topical medication. VA returned to its baseline values, as well as biomicroscopy.

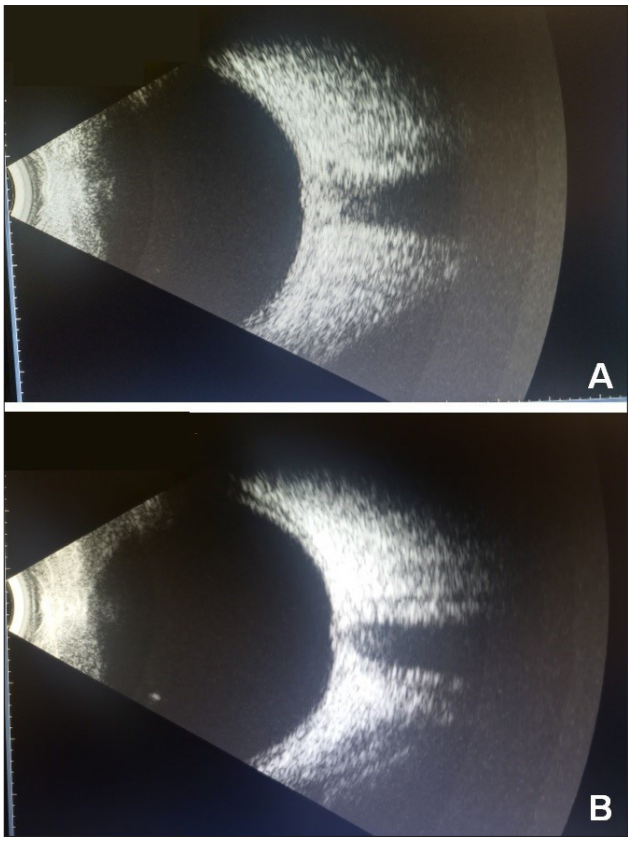

Figure 2. Ultrasound biomicroscopy reveals anterior uveal effusion, both in RE (A) and LE (B).

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

DISCUSSION

Hydrochlorothiazide has been associated with bilateral angle closure secondary to a 'posterior pushing' mechanism with the development of uveal and choroidal effusion.[3] The anterior migration of the iris-lens diaphragm leads to a myopic shift. Acute glaucoma is precipitated by appositional angle closure. However, the development of profound hyponatremia within this clinical picture was reported just once before. Our case reinforces the findings by Chen SH.[3]

Clinical suspicion is essential for this diagnosis. The typical presentation includes the easy recognizable signs and symptoms of acute glaucoma, such as blurring of vision, nausea and vomiting, red eye, ocular pain and headache, associated with conjunctival injection, corneal edema, anterior chamber inflammation and shallow anterior chamber. This clinical presentation is associated with an ultrasound biomicroscopy revealing swelling of the ciliary body. If this presentation is bilateral, side effect of any drug or systemic condition must be suspected and carefully investigated. After diagnosis, the treatment stands on three pillars, namely: (1) acute management of the electrolytic imbalance; (2) topical antihypertension medication and (3) discontinuation of hydrochlorothiazide. Peripheral iridotomy is not helpful, once the underlying mechanism is not pupillary block.[2,4-6] Instead, iridoplasty may be considered if the initial treatment is not enough.

In this particular case, the classical presentation raises clinical suspicion of iatrogenic bilateral angle closure. Hydrochlorothiazide was the only drug taken by our patient potentially associated with bilateral glaucoma and all the clinical features reverted with its suspension, which strengthens the diagnosis.

Our clinical approach was similar to that of cases involving topiramate[4,5] due to few hydrochlothiazide established cases. In fact, to our knowledge, only two acute glaucoma cases have been described so far involving hydrochlorothiazide.[7] Furthermore, the association of bilateral acute glaucoma and hyponatremia is rare and it is still unclear whether there is a causal or synergistic effect.[8]

Hydrochlorothiazide is one of the most commonly prescribed anti-hypertensive medication. Even if the described adverse effect is rare, clinical awareness for these events is essential. Early recognition will determine treatment and prognosis in cases of this potentially devastating cause of acute angle closure.

CONTRIBUTORSHIP STATEMENT / DECLARAÇÃO DE CONTRIBUIÇÃO:

All authors contributed to the study conception and design. All authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

RESPONSABILIDADES ÉTICAS

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ETHICAL DISCLOSURES

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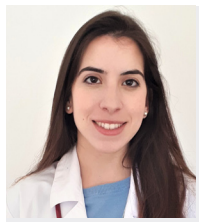

Corresponding Author/
Autor Correspondente:

Sofia Teixeira
R. Conceição Fernandes S/N,
4434-502 Vila Nova de Gaia, Portugal
E-mail: ana.cunha.teixeira@chvng.min-saude.pt

ORCID: 0000-0003-1116-7474