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Optic Disc Edema and Posterior Globe Flattening Secondary to Ocular Hypotony: Case Report and Discussion Regarding Pathophysiology and Clinical Findings

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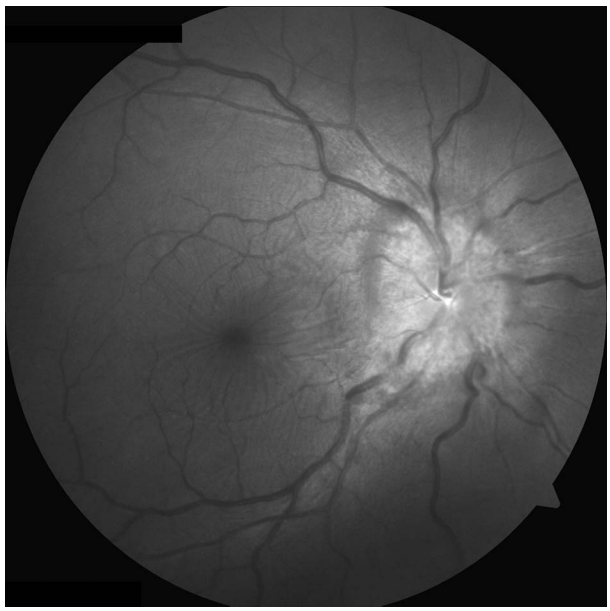

FIG. 1. Fundus scan of the right eye, highlighting optic disc edema and hypotonic maculopathy.

Abstract: We describe a case of a young female patient presenting with ocular hypotension (4 mm Hg) secondary to cyclodialysis, and optic disc edema (ODE) after a blunt trauma in the right eye (right eye). MRI showed posterior globe flattening of the right eye, drawing our attention to the pathophysiology behind these findings. The combination of ODE and posterior globe flattening, as observed in the present case of ocular hypotony, is known from other conditions such as intracranial hypertension and spaceflight neuro-ocular syndrome, pointing to a common pathophysiological mechanism, possibly resulting from axoplasmic stasis at the level of the lamina cribrosa due to a high translaminar pressure difference.

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A 28-year-old female patient was referred 2 months after a blunt eye trauma in the right eye caused by a bottle cap. Visual acuity (VA) was finger count in the right eye and

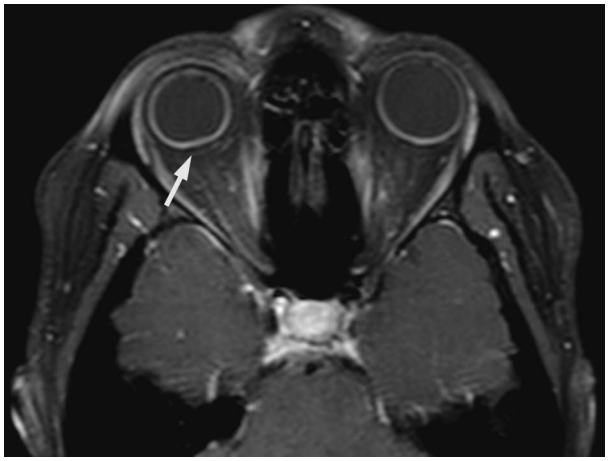

FIG. 2. Orbital magnetic resonance scan. Axial imaging at the level of the pituitary. Note the posterior sclera flattening in the right eye (*arrow*).

1.0 in the left eye. Intraocular pressure (IOP) was 3 mm Hg in the right eye and 16 mm Hg in the left eye on Goldmann applanation tonometry. Gonioscopy showed 90° of cyclodialysis in the right eye. Fundoscopy evidenced optic disc edema (ODE) and macular chorioretinal folds in the right eye (Fig. 1). No abnormalities were observed in the left eye. Oral prednisone, topical corticosteroid, and cycloplegic eyedrops were initiated as treatment for hypotony. An orbital MRI was requested to rule out other causes of ODE, such as optic neuritis and compressive lesions. The right posterior sclera was found to be flattened (Fig. 2), a finding commonly reported in intracranial hypertension. Empty sella was not observed. Trabecular laser photocoagulation was performed in areas of cyclodialysis and, 2 months after the procedure, IOP was 10 mm Hg and VA was 1.0 in the right eye.

INTERPRETATION OF THE FINDINGS

The most widely acknowledged theory on the pathogenesis of ODE in intracranial hypertension attributes the condition to axoplasmic flow stasis in the optic nerve at the level of the lamina cribrosa (1–3). The edema is believed to be induced by a high translaminar pressure difference (TLPD) between the IOP and the cerebrospinal fluid pressure (CSFp) in the optic nerve subarachnoid space (ONSAS) (1,3,4). The optic nerve sheath is continuous with the dura mater, and the compartment formed around the optic nerve is an extension of the intracranial subarachnoid space filled with cerebrospinal fluid (4). TLPD is thought to be an important factor in the pathogenesis of ODE in certain conditions, including intracranial hypertension, spaceflight neuro-ocular syndrome (SANS), and ocular hypotony (3–6). In patients with ocular hypotony, ODE may involve a set of factors, including choroidal effusion (5), but in the present discussion, we will focus on the role of TLPD in the pathophysiology of specific optic disc conditions, such as papilledema, ocular hypotony, and SANS.

A high CSFp or reduced IOP will produce a high TLPD with a force vector toward the intraocular compartment, resulting in axoplasmic stasis, nerve fiber edema, and ODE. Edema generates compression of the vessels of the optic nerve head, leading to venous stasis and fluid leakage into the extracellular space (1–4). Axoplasmic stasis and CSF accumulation, in turn, generate ischemia and accumulation of toxic substances in the optic disc head, contributing to edema (5,7). Lawlor et al recently published 2 case reports that exemplify the role of TLPD in the pathogenesis of ODE. In one, a patient with intracranial hypertension developed ODE after the introduction of hypotensive eye drops, with improvement after discontinuation and further worsening on reintroduction. In the other, a patient with intracranial hypertension and asymmetric papilledema presented with ODE, especially in the eye with reduced IOP due to a previous trabeculectomy (8). It should pointed out that a high IOP or reduced CSFp will also produce a high TLPD, but with the force vector in the opposite direction, potentially contributing to glaucoma-related optic nerve damage (3,4).

As for SANS, astronauts exposed to microgravity over extended periods (usually over 6 months) have a high incidence of ODE. It seems that, under microgravity conditions, fluid normally diverted to the caudal spine is distributed throughout the subarachnoid space and exhibits reduced drainage due to the higher pressure in the intracranial venous sinuses, contributing to a slight elevation of CSFp at the optic disc level (3,4,9–11). The ONSAS consists of a cul-de-sac compartment with a complex ultrastructure composed of numerous septa, velums, and trabeculae that limit the flow of CSF, which is believed to be drained by the lymphatic system of the optic nerve termination (1–4,12,13). Structural changes in the ONSAS secondary to toxic substances accumulated during axoplasmic flow stasis might explain the presence of ODE in the absence of increased CSFp and the persistence of ODE in astronauts after their return (1–3,12). Choroidal effusion, as in patients with ocular hypotony, is probably also involved in the pathogenesis of SANS (3,5,9,10,14).

The main MRI findings in idiopathic intracranial hypertension (IIH) and SANS are optic nerve sheath enlargement, optic nerve tortuosity, and posterior flattening of the eyeball (3–5,9,15). The latter finding in patients with hypotony is described by Westfall et al (6) after trabeculectomy and prolonged low IOP. The description fits the imaging findings of the case presented in this article.

Our case shows that the pathophysiological mechanism of ODE may be the same in patients with ocular hypotony, IIH, and SANS. Further explorations of the anatomy and

physiology of the optic nerve, lamina cribrosa, and ONSAS, especially regarding TLPDs, may help clarify the pathophysiology of optic disc conditions such as papilledema, ocular hypotony, and SANS.

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