Short communication

Herpetic endotheliitis and trabeculitis with delayed corneal involvement

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ABSTRACT

Case report: A 62-year-old man with previous renal transplant and immunosuppressive treatment presented with decreased visual acuity (20/100) in his left eye, corneal edema and intraocular pressure of 46 mmHg. One month later an inferior marginal dendritic keratitis appeared. Corneal scraping and real-time polymerase chain reaction showed herpetic simplex virus (HSV).

Discussion: The autoimmune corneal endotheliopathy or acute idiopathic corneal endotheliitis is characterized by corneal stromal edema and keratic precipitates. HSV might be sequestered from the trabeculum, innervated by the trigeminal nerve. This hypothesis is supported by the clinical observation that the corneal stromal edema usually starts from the periphery.

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Endoteliitis y trabeculitis herpética con afectación corneal tardía

RESUMEN

Caso clínico: Varón de 62 años de edad con trasplante renal y tratamiento inmunosupresor que acude por disminución de la visión (20/100) en ojo izquierdo, edema corneal y presión intraocular de 46 mmHg. Un mes después aparece queratitis dendrítica marginal inferior. El raspado corneal y la proteína C reactiva demuestran la presencia de un virus del herpes simple (HVS).

Discusión: La episoteliopatía corneal autoinmunitaria o endoteliitis idiopática corneal se caracteriza por edema corneal y precipitados queráticos. Se cree que el HVS podría ser secretado desde el trabeculum, innervado por el nervio trigémino, apoyado clínicamente por la progresión del edema estromal desde la periferia.

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Introduction

In 1982 Khodadoust and Attarzadeh defined the autoimmune corneal endotheliopathy, characterized by a stromal edema that progress inwardly from the periphery.\(^1\) It was subsequently proven that this endotheliopathy was caused by the herpes simplex virus (HSV). Herpetic keratitis is a complex ocular disease with highly variable forms of expression. Trabeculitis is defined by some authors as a preferred variant of endothelitis while others consider it a form of herpetic uveitis.\(^2\) It appears that the physiopathological mechanism consists of an obstruction of the aqueous humor drainage by inflammatory cells and occasionally trabecular scarring with persistent glaucoma. Increased intraocular pressure (IOP) generally responds to topical corticoids treatment, making filtering surgery frequently unnecessary.\(^3\)

Clinic case

A male, aged 62, visited the practice due to painless diminished visual acuity (VA) in the left eye (LE) with 2 days of evolution. The patient had visited 2 months earlier due to red eye, diagnosed as conjunctivitis and treated with a fixed combination of dexamethasone and tobramycin. At that time, the VA was of 1 and IOP of 14 mmHg in both eyes. Personal history of the patients included diabetes mellitus, arterial hypertension, obesity, hepatic steatosis, hyperthyroidism, ischemic cardiopathy and kidney transplant due to chronic renal insufficiency secondary to chronic pyelonephritis. The systemic and medical treatment included prednisone 5 mg/24 h (Prednisona Alonga\(^\text{®}\), Sanofi Aventis), tacroliimus (Prograf\(^\text{®}\), MSD) and mycophenolate motefitil (Cellcept\(^\text{®}\), Roche).

The initial exploration gave a VA of 1 in the right eye (RE) and 0.1 in LE, normal biomicroscopy in RE with intense conjunctival injection in LE and lackcluster corner with central predominance with slight edema and negative fluorescein staining, without Tyndall, synechiae, keratic precipitates or secretion. IOP was of 18 mmHg in RE and 46 mmHg in LE. The ocular fundus could be seen with difficulty due to the corneal edema, but the papilla and the macular were normal with the retina applied without peripheral lesions. The gonioscopy showed open angle without synechiae or neovessels. During the exploration, we observed that the patient was drowsy and had difficulty breathing, in addition to exhibiting low height, moderate obesity and a short neck.

The initial diagnosis was ocular hypertension in LE, and treatment was established with a fixed combination of 0.2% brimonidine and 0.5% timolole maleate (Combigan\(^\text{®}\), Allergan). The patient was referred to the glaucoma and neurology services where he was subsequently diagnosed with severe apnea-hypopnea syndrome.

One week later, the VA remained at 0.1 with persisting conjunctival hyperemia, slight corneal edema, slight superficial keratitis punctata, with IOP at 40 mmHg (Fig. 1). Pachymetry was 512 \(\mu\)m in RE and 574 \(\mu\)m in LE (this increase was explained by the corneal edema). A differential diagnosis was proposed with a carotid-cavernous fistula or an orbital apex syndrome and a computerized axial tomography...
(CAT) was requested. The treatment was changed to brinzolamide (Azopt®, Alcon Cusi), tafluprost (Saflutan®, MSD) and sodium diclofenac (Diclofenaco-lepori®, Angelini Farmacéutica). Two days later, the IOP remained at 30 mmHg and therefore acetazolamide was added (Edemox®, Chiesi-Spain) and oral prednisone 60 mg (Prednisona Alongs®, Sanofi Aventis).

The hyperemia diminished progressively (Fig. 2), and the corticoids dosage was gradually reduced although the IOP remained above 26 mmHg. The CAT discarded any arbitrary and/or cranial disease. Five weeks later a peripheral ulcer emerged with pseudodendritic appearance which gave positive fluorescein staining with infiltration of edges. A corneal sample was taken for culture and detection of proteins C reactive to virus herpes (Figs. 3 and 4), initiating treatment with topical aciclovir (Zovirax®, Glaxo Smith Kline) and oral aciclovir (Virex®, Biogen). The microbiological analysis was positive for HSV. With said treatment, the corneal lesion closed leaving a small peripheral leukemia, the IOP returned to normal values and the visual acuity reached 0.8.

Discussion

The definitive diagnosis of our case was endotheliitis and trabeculitis with secondary ocular hypertension and epithelial involvement in the form of late corneal ulcer. The absence of clear signs caused a delay in the diagnosis which became obvious when the corneal alteration emerged, with microbiological confirmation and the favorable response to the treatment.

Ohashi et al.4 proposed the hypotheses that, in each reactivation, the HSV latent in the trigeminal ganglion could traverse said nerve and reach the trabecular mesh from where copies of the virus would be secreted to the other ocular structures. These hypotheses are supported by the onset of the corneal edema in the periphery, the presence of HSV DNA in the aqueous humor of patients with endotheliitis and immunoreactivity for HSV described in the trabeculum of a patient intervened with trabeculectomy due to a secondary glaucoma of this type.5 It has also been postulated that the cicatrization of the trabecular mesh would be associated to a certain degree of necrosis produced directly by the virus similar to the iris atrophy or the necrotizing stromal keratitis. In our case, the absence of keratic precipitates, ocular pain and typical epithelial or endothelial injuries made the diagnosis difficult, although the multiple pathologies should have suggested an evident possibility, i.e., a recurring and opportunistic infection in an immunodepressed patient. Errorneously we considered that the corneal edema was explained by the increased IOP although the sequence was the contrary: an endotheliitis that caused corneal edema accompanied by trabeculitis which increased ocular pressure. With the diagnostic suspicion of herpetic endotheliitis and trabeculitis, treatment with antivirals and corticoids must be established without delay because the response is generally quick and prevents possible cicatricial lesions.

Conflict of interest

The authors declare no conflict of interest.

REFERENCES