ARCHIVOS DE LA SOCIEDAD ESPAÑOLA DE OFTALMOLOGÍA

www.elsevier.es/oftalmologia

Short communication

Anterior stromal puncture for the treatment of Brown–McLean syndrome☆,☆☆

P. Rodríguez-Ausín, K. Pachkoria*

Clínica Oftalmológica Suárez Leoz, Madrid, Spain

ARTICLE INFO

Article history:
Received 18 December 2011
Accepted 8 April 2012
Available online 28 June 2013

Keywords:
Peripheral corneal edema
Anterior stromal puncture
Bullous keratopathy
Brown–McLean syndrome
Keratoplasty

ABSTRACT

Case report: The Brown–McLean syndrome is defined by peripheral corneal edema sparing the central cornea. We report a patient with bullous annular keratopathy following phacoemulsification and implantation of posterior chamber intraocular lens. The uncorrected and best corrected visual acuity was 20/30. The patient required the use of a bandage contact lens for almost 5 years to relieve the ocular discomfort.
Discussion: Anterior stromal puncture was performed using a cystotome. Two weeks after the procedure the ocular discomfort disappeared and the use of bandage contact lens was no longer required. Anterior stromal puncture is a simple procedure that is performed under the slit lamp, and can alleviate the symptoms in patients with Brown–McLean syndrome.

Micropunción estromal anterior como tratamiento del síndrome de Brown–McLean

Palabras clave:
Edema corneal periférico
Punción estromal anterior
Queratopatía bullosa
Síndrome de Brown–McLean
Queratoplastia

RESUMEN

El síndrome Brown–McLean consiste en la presencia de edema corneal periférico respetando la córnea central. Presentamos un paciente con queratopatia bullosa anular años después de facoemulsificación e implante de lente en cámara posterior. Su agudeza visual espontánea y corregida era 20/30 y precisaba lente de contacto blanda para aliviar molestias desde hacía 5 años. Practicamos micropunción estromal anterior con cistitomo, y en 2 semanas las molestias desaparecieron y no precisa porte de lente blanda. La micropunción estromal es un procedimiento simple que se realiza en lámpara de hendidura y que puede eliminar la sintomatología del síndrome de Brown–McLean.

☆ Please cite this article as: Rodríguez-Asuín P, Pachkoria K. Micropunción estromal anterior como tratamiento del síndrome de Brown–McLean. Arch Soc Esp Oftalmol. 2013;88:193–6.
☆☆ Presented as a verbal communication at the 87.° Congreso de la Sociedad Española de Oftalmología, Oviedo, 2011.

* *Corresponding author.*
E-mail address: pazrausin@telefonica.net (P. Rodríguez-Ausín).

Introduction

The Brown–McLean syndrome (BMS) consists in the appearance of peripheral corneal edema sparing the central area. It was first described in 1969[1] and is generally associated to previous intracapsular lens surgery.

BMS patients are usually asymptomatic although some documented cases exhibited chronic irritation symptoms, pain and septic corneal ulcers. Various treatment options with varying degrees of success have been published for symptomatic patients including hypertonic saline solutions or lubricants,[2] extended use soft contact lenses, penetrating keratoplasty, removal of the phakic lens in the anterior chamber and anterior stromal micropuncture.[3]

This paper presents a symptomatic BMS case successfully treated with anterior stromal micropuncture (ASM).

Case report

Male patient, 72, intervened for phacoemulsification with intraocular lens implant in both eyes 12 years before, also requiring vitrectomy for removing nuclear remains in the right eye. The patient had to use soft contact lenses starting 5 years before with substitution every 2 months due to persistent discomfort in the right eye (RE). Non-corrected and best corrected visual acuity was of 20/30 in both eyes (BE). Biomicroscopy revealed pseudophakia in the anterior chamber of BE and a 3 mm peripheral annular corneal edema in the RE sparing the central cornea with peripheral endothelial pigment dispersion (Fig. 1A and B). No gutattas were observed in BE. The LE cornea did not exhibit pathological findings. Intraocular pressure (IOP) was normal in BE and central endothelial cell density measured with SP-3000P TOPCON® mirror microscope was of 1300 and 1500 cells/mm^2 in RE and LE respectively. The peripheral density was not displayed due to edema in RE.

In April 2011, ASM was performed in slit lamp under topical anesthesia with 0.5% tetracaine (Colircusí Anestésico®, Alcon-Cusí, Barcelona, Spain), consisting in 40–50 superficial micropunctures (one fourth thickness, approximately 150 μm) in each quadrant utilizing a 25 G cystotome (BD Visitec™, Waltham, USA) sparing the central cornea (Fig. 2A and B). Antibiotic prophylaxis was applied with moxifloxacin (Vigamox®, Alcon-Cusí, El Masnou, Barcelona, Spain) before and after the procedure.

2 weeks later, the patient was able to stop using the contact lens undisturbed and in 3 weeks subepithelial fibrosis was observed in the treated area. The patient remains asymptomatic after 12 months of follow-up (Fig. 3A and B).

Discussion

BMS consists in peripheral corneal edema with circumferential progression which typically spares the central cornea. In addition, dotted brownish-orange pigmentation may appear in the peripheral endothelium. Central endothelial density is somewhat below normal values and cornea gutatta may coexist. It has been described mainly after intracapsular lens extraction and with less frequency after extracapsular extraction, phacoemulsification, keratoplasty, IOL implant in CA and vitrectomy. In addition, it has been reported in the absence of surgery associated to ocular traumatism, closed angle glaucoma, myotonic dystrophy and lens subluxation.[3,4]

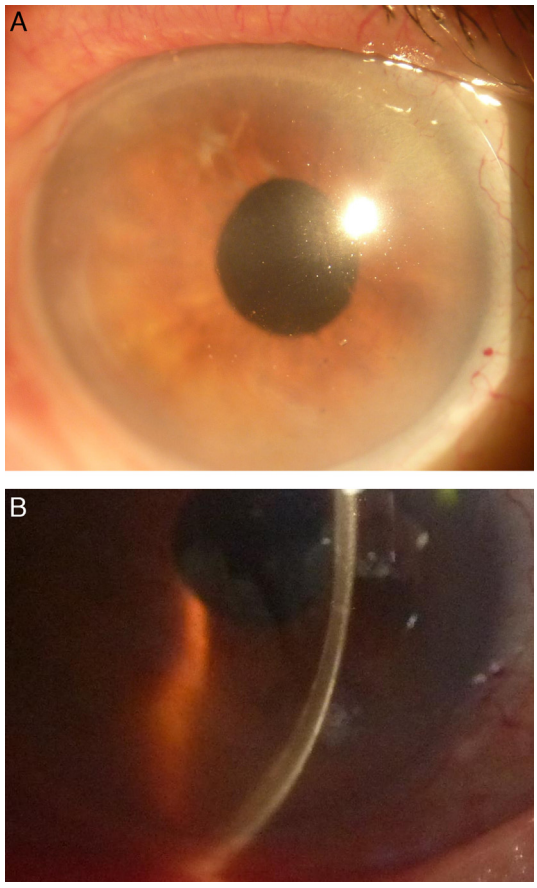

Fig. 1 – (A) Cornea with blurred ring and presence of soft contact lens and (B) peripheral edema with pigmented deposits.

Suggested pathogenics include iris peripheral endothelial traumatism, IOL haptics, dislocated vitreous or lens which causes endothelial abrasion and cell loss in the periphery, without discarding associated genetic predisposition.[4]

The period of time in which BMS appears after surgery has been reported between 1.5 and 34 years. In the instant case it appeared after 10 years and unilaterally. This extended period of time has made us consider progressive RE endothelial damage without knowing the intra- and post surgery details of previous surgeries which could have played a role in the unilateral expression of BMS.

In many cases the patient remains asymptomatic although persistent pain may appear due to bullous keratopathy. This pain is related to the rupture of the bullae and the ensuing exposure of corneal nervous endings. If treatment with hypertonic saline solution or lubricants is not enough, extended use soft contact lenses may be required with the ensuing risk of infection. In the case of progressive decompensation which could affect the center of the cornea, as can occur particularly with anterior chamber phakic lenses, penetrating keratoplasty could be indicated. However, this would not be adequate in