Short communication

Fingerprint microcystic dystrophy: Episodes of irregular astigmatism and their topographical representation

G. Pérez-Carro a,*, R. Fau a, L. González-González b

a Oftalmología, Hospital de la Cruz Roja, Gijón, Asturias, Spain
b Enfermería, Hospital de la Cruz Roja, Gijón, Asturias, Spain

ARTICLE INFO

Article history:
Received 23 September 2010
Accepted 3 June 2011
Available online 22 April 2012

Keywords:
Microcystic corneal dystrophy
Cogan keratopathy
Fingerprint lines

ABSTRACT

Case study: A 44-year-old male patient suffering from painless bouts of blurred vision and with no visible ophthalmological repercussions initially. After one of these clinical episodes we managed to visualise fingerprint images in the anterior pole, as well as corneal, pachymetric and topographical changes, which in turn produce the symptomatic refractive changes.

Discussion: Fingerprint keratopathy is a condition diagnosed through recurring corneal erosion. The pathogenic origin of the condition –an altered epithelial basal membrane –may encourage the separation of the corneal epithelium from its underlying layers. Depending on whether this separation is partial or total, this will lead to spontaneous corneal erosion or, less frequently, episodes of blurred vision caused by oedema and corneal swelling.

© 2010 Sociedad Española de Oftalmología. Published by Elsevier España, S.L. All rights reserved.

Distrofia en huella dactilar: brotes de astigmatismo irregular y su demostración topográfica

R E S U M E N

Caso clínico: Varón de 44 años con brotes indoloros de visión borrosa sin repercusión oftalmológica visible inicialmente. Tras uno de sus episodios, se visualiza en polo anterior imágenes en huella digital así como cambios corneales, paquimétricos y topográficos que originan cambios refractivos sintomáticos.

Discusión: La distrofia en huella dactilar es una entidad diagnosticada por erosiones corneales recurrentes. Su base patogénica, una membrana basal epitelial alterada, favorece la separación del epitelio corneal de las capas subyacentes. En la medida que ésta sea total o parcial ocasionará erosiones corneales recurrentes o, con menor frecuencia, episodios de visión borrosa por edema y engrosamiento corneal.

© 2010 Sociedad Española de Oftalmología. Publicado por Elsevier España, S.L. Todos los derechos reservados.

* Corresponding author.
E-mail address: gemurri27@yahoo.es (G. Pérez-Carro).

2173-5794/$ – see front matter © 2010 Sociedad Española de Oftalmología. Published by Elsevier España, S.L. All rights reserved.
Introduction

Dystrophy or fingerprint keratopathy (FK) is an entity that is frequently diagnosed due to painful and recurring corneal erosions (RCE) caused by alterations in the epithelial basal membrane. Painless episodes predominate occasionally, coursing with blurred vision without obvious ophthalmoscopic signs.

This paper presents a case of unilateral visual loss, which, after months of controls, was labeled as fingerprint dystrophy on the basis of biomicroscopic signs which would be confirmed with topographic and pachymetric data.

Clinic case

A, male, aged 44 years, visited the practice due to painless blurred vision episodes in the left eye (LE) starting a few days back. Initially, all the explorations gave normal results without direct or indirect signs of ophthalmological pathologies. However, due to the progressive increase of said episodes, a sub-epithelial central cyst was observed, initially of 1 mm, posteriorly annular (Fig. 1a–c) and through transillumination, tenuous fingerprint lines beyond the visual axis (Fig. 2a and b).

With the diagnosis of presumed unilateral keratopathy in map-dot-fingerprint, corneal topographs and pachymetries were carried out to confirm the assumption. Due to the histopathology underlying FK, where the epithelial basal membrane is aberrant, the blurred vision crises were related

Fig. 1 – (a) Sub-epithelial cyst in the central area of the left eye (LE). (b) In just a few days, subepithelial annular cyst. Clinic episode. (c) Anterior image seen through transillumination.

Fig. 2 – (a and b) “Fingerprint” visible with dilatation and transillumination in LE. Located beyond the pupilar area. Asymptomatic.
with corneal epithelial detachments and changes in topographic and refractive corneal thickness. Accordingly, the pachymetric data at asymptomatic times were: 574 μm in RE and 584 μm in LE, and during the episodes, said data were: 600 μm in LE (US Alcon Pachymeter). The topographic images corresponding to Fig. 1 show corneal flattening and irregular astigmatism only in the LE (corneal topograph CA-100 Corneal Analyser Topcon) (Fig. 3a and b). We also found a hypermetropic change of +1.00 dioptres of refractive spherical change. After prescribing 0.5% NaCl eyedrops and lubricant 3 times a day, a reversal of the symptoms and topographic changes were observed (Fig. 3c). After one year, greater topographic stability was observed (Fig. 4a) although occasionally the patient visits due to blurred vision outbreaks (Fig. 4b).

Discussion
Initially described by Cogan in 1964, fingerprint keratopathy shares similarities with familial recurring corneal erosion described by Franceschetti in 1928, epithelial basal membrane dystrophy and Cogan microcystic dystrophy in map-dot-fingerprint. It is the most common dystrophy with a prevalence in the population of 2–42%, the majority of which...
are non-hereditary sporadic cases to which the term “keratoconal” is applied.1

The clinical signs range from grayish configuration in geographic form (map), discrete spots (dots) up to fingerprint patterns made up by concentric lines. The latter are better visualized with transillumination although they could go unnoticed. At the clinical level, these lesions course in a fluctuating and evanescent manner, hindering diagnostic when painless blurred vision episodes arise, as in the instant case.2

The most common debut of this keratoconal is painful corneal erosion,1–4 although some patients do not refer symptoms or only a foreign body feeling or photophobia. On other occasions, blurred vision, diploia, ghost images, irregular astigmatism2 or structural lachrymal film alterations1 predominate. It has been described with greater prevalence in females, ranging between 40 and 70 years of age.

The abnormal synthesis of the basal membrane (BM)1 or the abnormal migration of the basal epithelial cells2 and the loss of desmosomes and intercellular adhesion are the causes that will produce corneal epithelial detachment with a simple “scan”.5 In these episodes subepithelial and intraepithelial detritus accumulate to form pseudo-cysts or dots. The fingerprints are linear projections of fibrinogranular material between the BM and Bowman’s membrane.1–3

During sleep, due to lack of evaporation and oxygen, the physiological subepithelial corneal edema will worsen adherence and originate corneal erosion with the first morning blinking.

The most widely applied treatments are osmotic and lubricant hypertonic solutions that diminish edema and palpebral friction which, in our case, enabled prolonged asymptomatic periods.1 Additional treatment options include contact lenses,1,2,5 the typical epithelium debridement,4 mechanical micropunctures or with diathermia with Nd-Yag laser and therapeutic photokeratectomy with Excimer laser, although with the latter symptoms tend to reappear.1

Accordingly, the case is a sub-clinic Cogan keratoconal that courses with painless and self-limited outbreaks and fluctuating refractive changes due to corneal edema. Axial topographs and pachymetries will enable an understanding of the histopathogenetic events underlying this disease as well as for monitoring corneal changes in this patient at different times of its evolution, with and without treatments.

Conflict of interests

The authors have no conflict of interests to declare.

REFERENCES