PRIMARY BILATERAL LIPID KERATOPATHY

QUERATOPATÍA LIPOIDEA PRIMARIA BILATERAL

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ABSTRACT

Case report: Observational, case report study. A 45-year-old woman complained of bilateral irritation and photophobia since childhood. Slit-lamp examination revealed yellow-whitish deposits involving the deep stroma of the peripheral half of the cornea in both eyes. The evolution was slowly progressive with a moderate impairment of her visual acuity.

Discussion: Lipid deposits adopt symmetric and bilateral configurations; the absence of corneal vascularization and ocular pathology as well as any lipoprotein disorder support the diagnosis of primary lipid keratopathy. The slow progressing disease is also confirmed (Arch Soc Esp Oftalmol 2009; 84: 263-266).

Key words: Primary lipid keratopathy, corneal vascularization, primary lipid degeneration, etiopathogenesis, lipid metabolism.

INTRODUCTION

Lipoid keratopathy (LK) is an infrequent disease which courses with yellow-whitish fatty deposits affecting all the stroma layers of the cornea. In its primary form, the lipids are deposited in an apparently normal cornea without any associated systemic disease. Said deposits are bilateral, with slow and somewhat symmetrical progression. However, in the majority of cases there is a previous history of ocular pathology or traumatism with deep neovascularization, or a return to the systemic disorder of the lipidic metabolism. In this case, it is secondary LK.
This paper presents a case of primary bilateral LK without significant reduction of visual acuity (VA) after over 10 years follow-up, evidencing the slowly progressive nature of this disease.

CASE REPORT

A 45-year-old woman who referred bilateral ocular irritation and photophobia since childhood, without previous ocular pathology or traumatism. Upon ophthalmological exploration, she exhibited a VA of 0.9 in both eyes (BE). Biomicroscopy (BMC) revealed whitish-yellowish deposits in BE in the deep stroma of the middle cornea periphery in ring-shaped arrangement respecting the pupil axis (fig. 1). The intra-ocular pressure was of 12 mm Hg. in BE and the ocular fundus exploration was normal. The lab tests, including a lipidgram, gave normal results. The familial study was negative. After a follow up period of 13 years, her VA was of 0.6 and 0.7 respectively in BE. BMC revealed a slight increase of corneal deposits (fig. 2).

DISCUSSION

A case of LK with lipidic deposits at the level of the deep stroma, bilateral and approximately symmetrical, in ring-shaped arrangement in the middle periphery of the cornea, respecting the pupil axis. Our patient referred slight episodes of ocular irritation and photophobia in her childhood, but no clear episode of keratitis or recurring ocular inflammation. In addition, there was no ocular traumatism.

Fig. 1: Whitish-yellowish lipidic deposits in ring-shaped arrangement in the deep stroma of the middle cornea periphery in A) right high and B) left eye. February 1994.

Fig. 2: Notice a slight increase of corneal lipidic deposits, which maintained the same ring-shaped symmetrical arrangement after 10+ years of follow up. A) in right eye and B) left eye. March 2007.
history. BMC did not reveal superficial or deep corneal neovascularization. These clinical data, associated to seric lipidic values within normal ranges, allow us to establish the diagnostic of primary LK.

The etiopathogenesis of primary LK is unknown. The small number of cases published with a histopathological study generally show the presence of vascularization in the corneal stroma, even though this neovascularization is not evident to BMC (1). One possible explanation for this finding is the existence of a subclinical inflammation of the corneal-scleral limbus, undetected by the patient, which causes vascularization of the cornea and the ensuing lipidic deposit (2,3). However, other authors consider that the initial course could be an intrinsic local alteration of the keratocyte with necrosis leading to inflammation, vascularization and later on deposit of lipids in the corneal stroma (2,3).

In addition, it has been considered that in the absence of vascularization the corneal lipidic deposits could proceed from the aqueous humor (4). Silva-Araújo et al (5) presented a case of primary LK with cholesterol values in the aqueous humor similar to those of control subjects but with significantly higher levels of cholesterol and sphingomyelin in the cornea. For this reason, the authors suggested a local alteration of the cholesterol and/or sphingomyelin metabolism restricted to the cornea as an etiopathogenesis hypothesis. However, more extensive biochemical and molecular studies are required to reach a more conclusive etiopathogenic hypothesis.

By way of conclusion, the clinical symptoms exhibited by our patient and discussed above, similar to those of other clinical cases in the literature, allow us to establish a diagnostic of primary LK. In our case, we were able to verify the slowly progressive evolution of this entity without significant repercussions on visual acuity.

REFERENCES