INTRODUCTION

Striate melanokeratosis (SM) is a corneal pigmentation which may associate to a healing process of a corneal epithelial erosion in dark skinned races. This paper presents a Caucasian patient that developed SM in relation to recurring corneal erosion in his left eye (LE).

RESUMEN

Caso clínico: Se presenta el caso de una mujer de raza blanca y piel clara, con erosión corneal recurrente y con melanosis conjuntival en OI, que desarrolló una opacidad corneal por melanoqueratosis estriada. La melanosis conjuntival se trató con mitomicina C tópica, manteniendo la pigmentación corneal.

Discusión: La melanoqueratosis estriada se presenta en personas de piel oscura, con un único caso descrito en personas de raza caucásica. Ésta se origina por daño corneal o por migración de pigmento desde melanosis conjuntivales, debiendo tratar ambas condiciones para detener la pigmentación corneal.

Palabras clave: Melanoqueratosis estriada, erosión corneal recurrente, pigmentación corneal, mitomicina C tópica, melanosis conjuntival, raza caucásica.

ABSTRACT

Clinical case: A fair-skinned woman presented marked striate melanokeratosis in her left eye related to recurrent corneal erosion. The source of pigmentation was a conjunctival melanosis. The conjunctival melanosis responded to treatment with topical mitomycin, while the corneal pigmentation persisted.

Discussion: Striate melanokeratosis is a condition described in dark-skinned patients who show a well-defined pigmentation of the limbal area, with only one case of striate melanokeratosis reported previously in a Caucasian person. The stimuli for this proliferation are corneal lesions or melanosis close to the limbus. Avoiding both stimuli are the main steps in its management (Arch Soc Esp Oftalmol 2009; 84: 213-216).

Key words: Striate melanokeratosis, recurrent corneal erosion, corneal pigmentation, topical mitomycin C, conjunctival melanosis, caucasian race.
CASE REPORT

A 50-year-old woman with a history of corneal erosions in her LE dating six years back and of unknown origin. In November 2004 she visited the practice due to pain in the LE and a temporal-superior erosion close to the limbus, with a visual acuity (VA) of 1. The LE bulbar conjunctiva exhibited two conjunctival melanosis patches in the temporal and inferior quadrants (fig. 1). The cornea exhibited diffuse subepithelial pigmentation accentuated in the periphery and the superior and temporal limbus, without corneal vascularization (fig. 2). The erosion was treated with de-epithelization, therapeutic contact lens (TCL) and hyaluronic acid eyedrops, autologous serum 20%, antiedema and fluorometalone, the latter for two weeks. Subsequently, micropunctures were performed due to recurrence of the erosion in this area. In May 2005, doxicicline 100 mg/24 h and vitamin C 1 gr/24 h were added to the treatment due to peripheral nasal-superior erosion. In July 2005 the patient exhibited a temporal erosion. In December of the same year she exhibited a superior nasal corneal erosion with extreme pain and broad de-epithelization. Pigment was observed at the base of the ulcer. The corneal epithelium was referred to pathological anatomy due to exhibiting positive HMB-45 melanic cells characteristics of melanocytes. The re-epithelization of the area was accompanied with intense local pigmentation. In February 2006 an inferior temporal limbar conjunctival biopsy was carried out, revealing a conjunctival mucous with squamous metaplasia and base melanocytic hyperpigmentation without abnormal melanocytes and corneal biopsy of the same quadrant which showed base melanocytic hyperpigmentation due to melanocyte deposits (fig. 3).

In her evolution, the patient remained with continuous irritation in her LE and progression of corneal pigmentation with slight reduction of VA to 1 (-3). In March 2006, treatment with mitomycin C eyedrops at 0.02%/8h was established together with fluorometalone/8h eyedrops for two weeks, repeating the cycle an additional week with a three-week interval. Biomicroscopically, the conjunctival pigmentation disappeared but the corneal melanosis persisted, accompanied in 2008 with recurrence of superior-temporal and superior-nasal corneal erosion (fig. 4).

DISCUSSION

Around the corneal limbus there is a strip of pigment with a varying density according to the

Fig. 1: Temporal conjunctival Melanosis in LE.

Fig. 2: Diffuse limbal and corneal pigmentation.

Fig. 3: corneal biopsy with deposits of melanocytes in the basal layers of the corneal epithelium.
Most of the pigment is contained in melanoblasts located in the basal layer of the conjunctival epithelium. A normal cornea does not contain pigmented cells. However, in some circumstances the melanoblasts proliferate and migrate to the base and layers of the corneal epithelium and reproduce their pigment. The main stimulus for this unusual proliferation is corneal aggression, particularly if located at a critical distance of the limbus under 4 mm (1). In dark skinned people with markedly pigmented limbus, this pigmentation progresses under the intact epithelium and is distributed in the cornea forming strips based on the limbus and an apex directed towards the corneal lesion, producing a permanent local opacity or a diffuse corneal pigmentation (2) known as striated melanokeratosis (SM). Frequently, SM is accompanied by corneal vascularization and depigmentation of the limbar area where the melanocytes originate (3).

In the Caucasian race, there is one described case of SM associated to corneal erosion after receiving eight subconjunctival injections of 5-fluorouracil post-trabeculectomy (4). Said drug as well as topical epinephrine and phenothiazines have been associated to cutaneous and ocular pigmentation. Corneal invasion by pigment can also originate from conjunctival cancerous and pre-cancerous melanosis close to the limbus.

Our patient is a white skin woman with two pigmented conjunctival lesions which, when biopsied, did not show atypia or abnormal melanocytes. SM, described in colored people, has been related to a broad range of corneal aggressions such as anterior chamber paracentesis, keratoplasty, cataract surgery, traumatisms and keratitis of varied origins. The most severe cases were those associated to penetrating keratoplasty or cataract surgery which developed with prolonged inflammation (3). In our case, the SM which was only in the eye with recurring corneal erosion, suggests that this was the stimulus responsible for the migration of limbar or conjunctival melanocytes towards the cornea. We do not know if this sub epithelial pigmented deposit could weaken re-epithelization and therefore predispose the patient to corneal erosions. We do not have either information about its management apart from avoiding the stimulus promoted by SM. Topical application of mitomycin C has demonstrated its efficiency in the treatment of conjunctival primary acquired melanosis (5). Although its effect on the corneal extension of this disease is not detailed, these cases have exhibited alterations of the corneal epithelium which were resolved by suspending the treatment (5). In our case, this treatment reduced the source of conjunctival pigmentation even though the biopsy (which was possibly insufficient) did not exhibit conjunctival atypia. However, it did not affect the course of the corneal melanosis.

REFERENCES