Editorial

Descemetorhexis and corneal clearing: A new perspective on the treatment of endothelial diseases

Descemetorrexis y aclaramiento corneal: una nueva perspectiva en el tratamiento de las enfermedades del endotelio

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The corneal endothelium, made up of a uniform monolayer of polygonal cells having a width of approximately 20 μm, is a fundamental structure for maintaining corneal transparency by means of active transport of water in the reverse direction to avoid corneal edematization.1 Primary endothelial disorders such as Fuchs’ endothelial dystrophy or acquired lesions such as those secondary to surgical trauma produce alterations in this layer with ensuing (and most frequently irreversible) visual loss. Loss or severe damage of these cells has been considered irreparable up to now as the endothelium does not regenerate in case of injury. In addition there is no possibility for mitosis and damaged cells are substituted by hypertrophy of adjacent cells and collagen, without the capacity for actively carrying water.1

However, in the last decade the above theory has been questioned because doubts are being cast on what in the past was considered to be irreparable. Endothelial dystrophy is fatal for maintaining said endothelial transparency and, until very recently, it was deemed that incision and complete loss of the Descemet-endothelium complex in a healthy cornea produce irreversible corneal edema. However, in 2003, Braunstein et al.2 described a spontaneous recovery of corneal edema after an accidental traumatic detachment with Descemet membrane excision after cataract surgery. It was verified that the exposed stromal area had been covered in the course of a few months by polymorphic and hypertrophic endothelial cells which maintained corneal transparency. A similar case was published in 2004 by Patel.3 In our own practice we have evidenced the evolution of this spontaneous endothelial recovery through anterior segment optic coherence tomography (OCT). The patient, an 89-year-old male, experienced complete traumatic tearing out of the Descemet-endothelium complex during left eye cataract surgery, with the ensuing corneal edema in the post-surgery period. Surprisingly, corneal recovery occurred spontaneously and was virtually complete, with total disappearance of the edema. The descemetorhexis edges could be seen in biomicroscopy. As with the other described cases, the endothelial count has evidenced the repopulation of this area by large-sized polymorphic cells which, at the


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functional level, fulfill the role of maintaining corneal transparency (Fig. 1).

Up to very recently, these cases were considered exceptional and unimportant, but they have already given rise to doubts about the nature of the endothelium and its allegedly limited regeneration capacity. Only with the recent publication of similar cases in patients with Fuchs’ endothelial dystrophy this spontaneous anatomic and functional recovery process has been brought to our attention.

At present, endothelial keratoplasty and its different variants (DMEK, DSEK and DSAEK) are the procedures of choice for treating corneal edema secondary to primary endothelial disorders, in which the apposition of healthy donor endothelium to the receptor stroma is considered essential. However, since 2009 some cases have been described in which the functional recovery of the endothelium took place despite the failure or absence of donor graft. This observation gave rise to many questions and doubts about the physiology and behavior of this cellular line, along the lines of the questions posed by the isolated cases of accidental descemethorhexis.

In 2009, Balachandran et al. published two cases of Fuchs’ endothelial dystrophy in which, after performing DMEK and losing graft adherence almost immediately, endothelial functional recovery was observed together with endothelial cell regeneration and improved patient visual acuity, which had never been observed in these transplants. Subsequently, in 2012, Dirisamer et al. published a study with 12 cases (which included patients with Fuchs’ endothelial dystrophy and others with bullous keratopathy due to post-surgery damage) in which they carried out the technique named Descemet membrane endothelial transfer. This new technique, a variant of DMEK, consisted of the injection of a free Descemet membrane graft (free roll) in the patient anterior chamber. Only the Fuchs’ endothelial dystrophy patients exhibited corneal clearing, with repopulation of endothelial cells demonstrated by mirror microscopy.

There have been many speculations about possible endothelial regeneration mechanisms in these cases. However, none of the proposed theories have been demonstrated to date. As early as 2006 the possibility was proposed that endothelial cells could undergo a certain degree of mitosis and repair as observed in vitro. Due to the traumatic loss of Descemet membrane, which acted as a physical factor inhibiting mitosis, adjacent endothelial cells were able to proliferate and repair the damaged area. This dynamic nature of the endothelium was subsequently confirmed by Lagali et al. who demonstrated in a controlled environment that receptor endothelial cells could completely substitute donor cells after penetrating keratoplasty, giving rise to graft failure. Up to now these speculations did not seem to arouse a great deal of interest, and all data and clinical experience suggested that in endothelial keratoplasty and all its variants, donor endothelium needed to adhere to the receptor for recovering corneal transparency. However, some authors believe that the corneal clearing and regeneration demonstrated after the implant of a free endothelial graft mean that we are in the presence of a stimulus and free migration within the anterior chamber of graft cells, or perhaps cells coming from the receptor as a possible source of this re-endothelialization in severe baseline endothelial disease cases.

At this date, research on this topic has gone further: in 2012, Shah et al. described the endothelial functional regeneration and recovery of a patient with Fuchs’ endothelial dystrophy who after descemethorhexis (as part of DSEK) did not receive donor graft implant. The above-described polygonal hypertrophic cells were observed a few months later in the Descemet-free area. This case goes as far as questioning the need of a donor graft and suggests that, as with our patients and the described traumatic descemethorhexis cases, the cornea of endothelial dystrophy patients could spontaneously regenerate and recover its function.

The above findings open the door to new treatments and prognostics in patients with endothelial damage and a disease which until now has been considered irreparable, in which the complicated endothelial transplant techniques might no longer be a necessary treatment.

REFERENCES


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