Editorial

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

Descemotorrexis 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 µ, is a fundamental structure for maintaining corneal transparency by means of active transport of water in the reverse direction to avoid corneal edematization.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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 descemotorhexis 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 trans-
parency (Fig. 1).

Up to very recently, these cases were considered excep-
tional 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 publi-
cation 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 vari-
ants (DMEK, DSEK and DSAEK) are the procedures of choice
for treating corneal edema secondary to primary endothe-
lial disorders,4 in which the apposition of healthy donor
endothelium to the receptor stroma is considered essential.5,6
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.7–10 This obser-
vation 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.7 published two cases of Fuchs’
dehiscence dystrophy in which, after performing DMEK and
losing graft adherence almost immediately, endothelial func-
tional 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.11 published a study with 12 cases (which
included patients with Fuchs’ endothelial dystrophy and oth-
ers with bullous keratopathy due to post-surgery damage) in
which they carried out the technique named Descemet mem-
brane endothelial transfer. This new technique, a variant of
DMEK, consisted of the injection of a free Descemet mem-
brane graft (free roll) in the patient anterior chamber. Only the
Fuchs’ endothelial dystrophy patients exhibited corneal clear-
ing, with repopulation of endothelial cells demonstrated by
mirror microscopy.

There have been many speculations about possible
endothelial regeneration mechanisms in these cases. How-
ever, 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,11 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.12 who demonstrated in a controlled environment that
receptor endothelial cells could completely substitute donor
cells after penetrating keratoplasty, giving rise to graft fail-
ure. Up to now these speculations did not seem to arouse
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 recov-
ering corneal transparency.5,6 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,13,14 or perhaps cells coming from the
receptor15 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.15 described the endothelial functional regen-
eration 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 poly-
ynal hypertrophic cells were observed a few months later in
the Descemet-free area. This case goes as far as question-
ing 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 sponta-
neously 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.

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Fig. 1 – Transparent cornea with central descemethorhexis
area.


