Evaluating a Totally Femtosecond Laser Assisted Descemet Stripping Automated Endothelial Keratoplasty

Thesis

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CONTENTS

ABSTRACT

Totally femtosecond assisted DSAEK is a new, easy and effective technique in treating pseudophakic bollous keratopathy and fuch's endothelial dystrophy, it overcome the potential complications of the currently used technique for DSAEK as graft detachment, dislocation and centration and avoids the extra burden by the over swollen edematous cornea on the newly implanted endothelium. But it has its own set of potential complications that can be avoided easily to enhance its results, as thickness disparity, thickness irregularity, air trapping and the prominent stromal interface.

Key words: Totally Femtosecond Laser, Descemet Stripping Automated, Endothelial Keratoplasty

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List of Abbreviations

ABZ	:	Anterior banded zone
ACC	:	Artificial anterior chamber
CCT	:	Central corneal thickness
CHED	:	Congenital hereditary endothelial dystrophy
DLEK	:	Deep lamellar endothelial keratoplasty
DMEK	:	Descemet's membrane endothelial keratoplasty
DSAEK	:	Descemet's stripping automated endothelial keratoplasty
DSEK	:	Descemet stripping endothelial keratoplasty
EK	:	Endothelial keratoplasty
FECD	:	Fuchs endothelial corneal dystrophy
IOL	:	Intraocular lens
LASIK	:	laser in situ keratomileusis
LogMAR	:	Logarithm of the minimum angle of resolution
mm	:	Millimeter
nm	:	Nano-meter
OCT	:	Optical coherence tomography
OR	:	Operative room
PAS	:	Periodic acid-Schiff
PCL	:	Posterior collagenous layer
Pg	:	Page
PK	:	Penetrating keratoplasty
PLK	:	posterior lamellar keratoplasty
PNBZ	:	Posterior nonbanded zone
PPCD	:	Posterior polymorphous corneal dystrophy
VA	:	Visual acuity
µm	:	micrometer

List of Figures

List of Tables

INTRODUCTION

The endothelium is a single layer of cells present on the back of the cornea. The site of the metabolic pump is within the lateral cell membrane; it is temperature dependent, it is associated with the enzyme Na^+/K^+ ATPase, and it is inhibited by ouabain. Endothelial cells produce a basement membrane (the Descemet membrane), and they are of neuroectodermal origin. Cell density at birth can be as high as 7500 cells/mm^2, decreasing to an average of about 2500-2700 cells/mm^2 in older adults. Endothelial cells are not capable of significant mitotic activity. The normal rate of endothelial loss after age 20 years is approximately 0.5% per year. Surgical trauma as pseudophakic and aphakic bullous keratopathy, inflammation, and corneal dystrophies as Fuchs dystrophy can accelerate this normal aging loss. The final common pathway in the development of bullous keratopathy is damage to the corneal endothelium; when the cell density reaches a critically low level of about 300-500 cells/mm^2, fluid begins to accumulate within the cornea. As a result, the cornea loses its transparency and the individual suffers a reduction in vision. **(99)**

Fuchs endothelial dystrophy (FED) is a condition in which there is premature degeneration of corneal endothelial cells.**(100)**

Descemet stripped automated endothelial keratoplasty (DSAEK), has become the preferred method of treating endothelial dysfunction, after penetrating keratoplasty (PKP) had long been the gold standard for treatment due to its limitations including delayed visual recovery and unpredictable reftactive changes. DSAEK provides faster visual recovery

with less induced surgical astigmatism with lower rate of intraoperative and postoperative complications. **(101)**

The femtosecond laser technique allows completely new trephination procedures in penetrating and lamellar keratoplasty. Thus, it is easier to get a watertight wound closure intraoperatively and due to the larger wound surface wound healing is faster**.** In lamellar keratoplasty the femtosecond laser enables the surgeon to cut to any depth in the corneas resulting in thin corneal donor buttons, e.g. for DSAEK.**(102)**

One of the main causes of the poorer than expected vision after DSAEK microkertome assisted was usually associated with the presence of folds or wrinkles that can develop in the graft as it conforms to the host cornea.**(103)** The eye banks do not measure the curvature of the donor cornea and no attempt is made to match donor and recipient curvatures, so in some cases the curvature mismatch may be substantial leading to wrinkles in the graft.**(104)**

AIM OF THE WORK

The aim of this study is to evaluate the structural and functional effect of totally femtosecond assisted DSAEK on bullous keratopathy and Fuch's dystrophy.

CHAPTER I

Anatomy of the Cornea Related to Endothelial Keratoplasty

The cornea is a complex structure which, as well as having a protective role, is responsible for about three-quarters of the optical power of the eye.[1] The average corneal diameter is 11.5 mm vertically and 12 mm horizontally.[1] It is 540 µm thick centrally on average, and thicker towards the periphery to reach around 1000 µm, this thinner center results from greater compression of the corneal lamellae centrally rather than a difference in the number of lamellae.[2] In order to carry its optical function, the cornea is totally transparent by having a very smooth non-keratinized outer epithelial layer, the stromal layer has a deeply compact regularly arranged collagen fibers with regular spacing, no blood vessels, the cells are minimal and the nerve fibers are non-myelinated, and that the cornea has a low water content (detergescent) which is the function of the inner most layer of the cornea, the endothelium.[3] (Figure 1)[1]

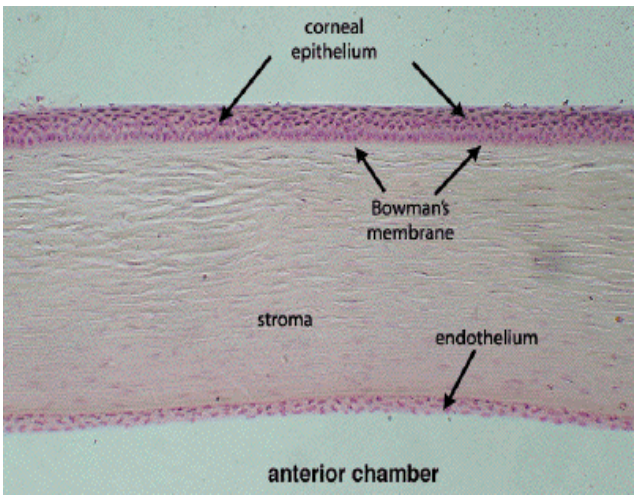

Figure 1

The endothelium, is embryologically derived from the neural crest. The postnatal total endothelial cellularity of the cornea (approximately 300,000 cells per cornea) is achieved as early as the second trimester of gestation.[4] Thereafter the endothelial cell density rapidly declines, as the fetal cornea grows in surface area.[4] achieving a final adult density of approximately 2400 - 3200 cells/mm^2.[5] The number of endothelial cells in the fully developed cornea decreases with age.[5]

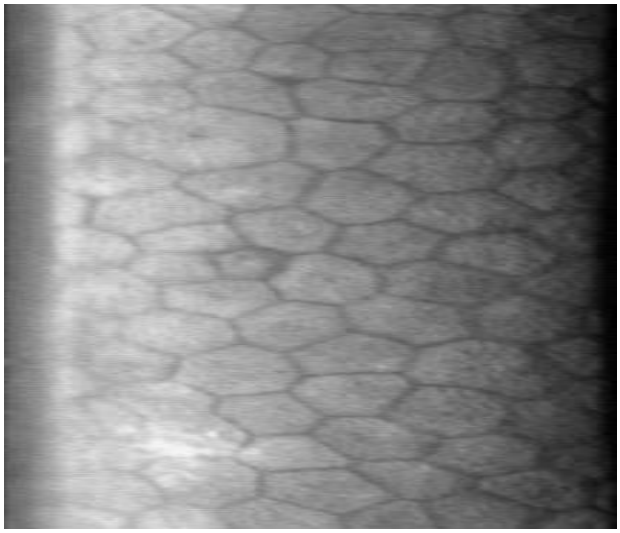

Figure 2

The normal corneal endothelium is a single layer of uniformly sized cells with a predominantly hexagonal shape (figure 2).[6] The corneal endothelium is attached to the rest of the cornea through Descemet's membrane. Descemet's membrane is the basement membrane of the corneal endothelium and is continuously deposited throughout life by the underlying endothelium, becoming gradually thicker with age. Ultra structurally, it can be divided into two zones: an anterior banded zone, and a posterior banded zone. It is composed mainly by type IV collagen . The principal physiological function of the corneal endothelium is to allow

leakage of solutes and nutrients from the aqueous humor to the more superficial layers of the cornea while at the same time actively pumping water in the opposite direction, from the stroma to the aqueous.[6] This dual function of the corneal endothelium is described by the "pump-leak hypothesis. [7] Since the cornea is avascular, which renders it optimally transparent, the nutrition of the corneal epithelium, stromal keratocytes, and corneal endothelium must occur via diffusion of glucose and other solutes from the aqueous humor, across the corneal endothelium. The corneal endothelium then actively transports water from the stromal-facing surface to the aqueous-facing surface by an interrelated series of active and passive ion exchangers.[7] Critical to this energy-driven process is the role of Na^+/K^+ATPase and carbonic anhydrase. Bicarbonate ions formed by the action of carbonic anhydrase are translocated across the cell membrane, allowing water to passively follow.[8]

The number of cells decreases at about 0.6% per year and neighboring cells enlarge to fill the space as the cells cannot regenerate.[9] Corneal endothelial cells are post-mitotic and divide rarely, if at all, in the post-natal human cornea.[9] Endothelial cell loss, if sufficiently severe, can cause endothelial cell density to fall below the threshold level needed to maintain corneal deturgescence.[9] This threshold endothelial cell density varies considerably amongst individuals, but is typically in the range of 300-500 cells/mm^2.[10] Typically, loss of endothelial cell density is accompanied by increases in cell size variability (polymegathism) and cell shape variation (polymorphism).[10] Excess hydration of the corneal stroma disrupts the normally uniform periodic spacing of Type I collagen fibrils, creating light scatter.[11] In addition, excessive corneal hydration can result in edema of the corneal epithelial layer, which creates irregularity at the