PARTICULAR SPECULAR MICROSCOPY ASPECTS OF THE CORNEAL ENDOTHELIUM REGARDING CATARACT SURGERY

Adina Berghian

INTRODUCTION

A vast amount of information about the eye can be gathered through a slit-lamp exam. Using a focused slit beam of light we can single out the epithelium, stroma or endothelium for close examination. In doing so, we can discover deposits on the endothelial layer that can even be identified as guttae that may indicate Fuchs corneal dystrophy and keratic precipitates that point towards uveitis. However, in order to have a full and clear image of the endothelium we have to turn to specular microscopy. Before 1970, the corneal endothelium was studied at high magnification only in vitro. The development of the clinical specular microscope is making possible qualitative and quantitative in vivo evaluation of corneal endothelium.

Cornea guttata consists of focal accumulations of collagen on the posterior surface of Descemet membrane. When the lesions involve the corneal periphery they are of no particular significance except as an indication of ageing. The term “cornea guttata” is reserved for the same lesions, involving the central cornea, which are also usually innocuous although rarely they are indicative of the early stages of Fuchs endothelial dystrophy. Fuchs dystrophy is more common in women and usually presents in older patients.
MATERIAL & METHODS

A total of 23 patients were selected from the Clinic of Ophthalmology, Timișoara. Eighteen subjects (nine with "cornea guttata" and nine with no corneal involvement) were operated using the method of phacoemulsification with posterior intraocular lens (IOL) implantation and five using mini-Nuc technique with anterior chamber implant in one eye and posterior chamber implant in the other.

In cases where phacoemulsification was used, we performed specular microscopy before and after surgery. The period of survey was of 3 month after surgery.

The specular microscopy was performed using a non contact specular microscope TOPCON SP 2000P. This specular microscope has also a pachimeter, helping us to determine the corneal thickness, too. The specular parameters noted were minimum (MIN), maximum (MAX) and average (AVG) endothelial cell area (they give the range of variation in cell size); the standard deviation (SD) of the endothelial cell size from the mean (is a measure of polymegathism); the coefficient of variation (CV) is calculated by dividing the standard variation of the endothelial cell area by the mean endothelial cell area. CV is a dimensionless index which gives a measure of polymegathism independent of cell size.

Two significant cases from the nine patients with cornea guttata will be presented.

The first case is a 74 years old woman who preoperatively presented guttae in the central corneal area. (Fig. 1) Postoperatively corneal thickness increased, but not significant (from 526µ to 545µ), but the cell density loss is noticed (from 2486 cells/mm² to 1678 cells/mm²). (Fig. 2) Coefficient of variation indicates the degree of polymorphism, more pronounced postoperatively (35 preop compared to 45 postop).

The second case is a 72 years old woman presenting few guttae in the central corneal area; with a better endothelium aspect than the one presented before (less guttae, higher cell density, lower CV). (Fig. 3) Postoperatively she presented corneal oedema which contraindicated specular microscopy to be performed the first week after surgery.

Still, after three months the corneal thickness returned to normal, but cell density remained low (1039 cells/mm²) with very pronounced polymorphism (CV 20 preop compared to 47 postop). (Fig. 4) This shows that the surgical trauma was larger than in the previous case.
Other nine cases were with no clinically visible corneal pathology.

The following case presents a 50 years old male patient with very good cell density preoperatively = 3195 cells/mm², but a high coefficient of variation for his age (39 years). (Fig. 5) Postoperatively he presented a growth in corneal central thickness (from 661µ to 752 µ) and folds in Descemet membrane, suggesting a decompensation in the endothelial pumping function. (Fig. 6)

![Figure 5. Patient N.P., 50 years, preop.](image)

![Figure 6. Same case, folds in Descemet membrane.](image)

![Figure 7. Anterior chamber implant.](image)

We have also studied five cases with anterior chamber implant in one eye (implanted five, four or three years ago) and posterior chamber implant in the other eye. We have examined both eyes using specular microscopy.

One of these patients had an anterior chamber implant for over five years. (Fig. 7) Using the specular microscope, a difference was noticed between the eye with posterior chamber implant, and the one with the anterior chamber implant, which presented greater endothelial cell loss. (Fig. 8, 9)

![Figure 8. Patient L.P., 56 years, posterior chamber implant.](image)

![Figure 9. Same case, anterior chamber implant for 5 years.](image)

**RESULTS**

The group with cornea guttata (nine cases) was compared with the group with no corneal involvement (nine cases).

Preoperatively, significant statistical differences were noticed (p = 0.012) in endothelial cell density – the cell density was lower in the “cornea guttata” group (1899.5 cells/ mm² compared to 2687.88 cells/ mm²). Also in mean endothelial cell area (p = 0.012) – the area was bigger in “cornea guttata” group (700 µm² compared to 398 µm²).

The decrease of endothelial cell density was statistically significant (p = 0.039) postoperatively – 1600.4 cells/ mm² in cornea guttata group compared to 2269.88 cells/ mm² in the other group.

Postoperatively, we have noticed a statistically
significant difference (p = 0.039) in mean endothelial cell area. The mean cell area was bigger in cornea guttata (711.71 µm²) than in the other group (461 µm²).

The corneal thickness had no statistically significant difference (p = 0.916) three months postoperatively compared to preoperative values, as reported in other studies as well.48

In all cases with anterior chamber implants we observed a significant (p < 0.001) increase in endothelial cell loss (30%) in the eye with anterior chamber implant, compared with the one with posterior chamber implant (17.5%).

DISCUSSION

Following cataract surgery, endothelial cell loss occurs due to surgical trauma. This indicates the need to protect the endothelium during surgery and also advocates for a careful examination of the endothelial cell population before surgery, including specular microscopy.

In fact, otherwise healthy elderly individuals may have guttae that resemble those seen in Fuchs’ dystrophy, indicating a relatively decompensate endothelium due to age. All this factors suggest the need to completely evaluate the cornea before exposing it to the trauma of a cataract surgery.1

Endothelial cell loss caused by cataract surgical trauma has been correlated with cataract incision size and location, density of nucleus, total ultrasound energy used, and volume of fluid irrigated into the eye at the time of surgery. It also depends on the surgeon’s technique and skills. Directly touching the endothelium during cataract surgery with instruments, nuclear fragments, or the intraocular lens should be avoided.2 Routine use of viscoelastic agents has resulted in a dramatic decrease in endothelial cell loss. This offers a practical and effective means of protecting the cornea from inadvertent trauma during cataract surgery.9 Dispersive viscoelastics may offer more protection to the endothelium than cohesive viscoelastics (especially if the surgeon’s technique is such that nuclear fragments are removed with phacoemulsification more anterior, above the iris plane).8,10

Certain diseases that damage the corneal endothelium, such as Fuchs corneal dystrophy, lead to endothelial changes such as guttae and eventually lead to corneal oedema. Additional injuries that can damage the corneal endothelium usually come from trauma; often, a case with a long evolution of cataract, especially when extracting a large, mature cataract, may lead to endothelial damage and cell loss.1

Corneal dystrophies (eg. Fuchs endothelial dystrophy) sometimes are overlooked on the preoperative exam, where the finding of cornea guttata may be subtle. If cornea guttata are noted on slit lamp examination, specular microscopy and ultrasound pachymetry should be performed to help quantify endothelial reserve and to aid in risk assessment.

Pre-operative polymegathism may indicate the likelihood of developing corneal oedema than patients without cells size abnormalities.11 In some cases this corneal oedema may be irreversible and may even develop into bullous keratopathy, one of the most unwanted postoperative complications.8,10,12,13

Older style intraocular lenses have been associated with accelerated endothelial cell loss following cataract surgery. In particular, closed-loop anterior chamber intraocular lenses (Leiske, Hessburg style) have been implicated with this problem. The haptics with these lenses tended to be stiff and erode through uveal tissue, causing chronic low-grade inflammation and continued endothelial cell loss. Modern flexible open-loop anterior and posterior chamber intraocular lenses have proven to be much safer alternatives.12 (Fig. 7)

Phacoemulsification and aspiration (PEA) has become the most popular cataract surgery, due to the establishment of safe surgical techniques and development of associated instruments. However, corneal endothelial damage still represents a serious complication, as excessive damage can lead to irreversible bullous keratopathy.10

CONCLUSIONS

Specular microscopy is extremely useful in evaluating the cornea before surgery.

Patients with endothelial pathology (such as guttae) or relatively few endothelial cell counts undergoing cataract surgery may require extensive care for protecting the endothelial layer in order to avoid unwanted complications.

Anterior chamber implants seem to be harmful for the cornea, damaging the corneal endothelium and causing endothelial cell loss.

REFERENCES


