CORNEAL EDEMA AFTER CATARACT SURGERY - CHANGES IN CORNEAL ENDOTHELIUM CELL CHARACTERISTICS

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Abstract: Corneal edema is a common complication of cataract surgery, although tremendous improvements have been made in the surgical techniques, which decreased surgical eye trauma and complication rates. Normal endothelial cell density is 2000-3000 cells/mm2 in older individuals, which maintains the corneal clarity. Even 'perfect' cataract surgery does some damage to the endothelium. A significant postoperative endothelium density decrease can impair its ability to maintain corneal clarity, resulting in corneal edema, blurring of vision and ocular pain. Aggressive topical treatment in the first month after surgery may lead to recovery of the endothelial cells. Our aim was to establish the effects of cataract surgery on the characteristics of the corneal endothelium. We performed a prospective interventional clinical study of 30 patients, mean age 65±12 years, with senile cataract. Over 80% were hard cataracts: 18% hypermature, 66% grade 4 (brunescent) and 16% grade 3 cataracts. Uneventful phacoemulsification with IOL implantation was performed by one experienced phaco-surgeon in an outpatient setting. Preoperative parameters included: best-corrected visual acuity (BCVA) in Snellen decimal units, IOP, cataract density (slit lamp examination), corneal endothelium cell density (ECD) and hexagonality measured with a specular microscope. Intraoperative parameters included: phacoemulsification time and energy, irrigation-aspiration suction time. Standard phacoemulsification cataract surgery was performed with in-the-bag IOL implantation. Mean baseline parameters were: BCVA=0.1±0.13, IOP=15.7±2.7 mmHg, ECD=2,497±290 cells/mm2, cell hexagonality was 54.3±9.4%. Mean surgical parameters were: surgical time=9,3±2.9 minutes, phacoemulsification time=35.6±26.1 seconds, phacoemulsification energy=13.3±10.9J, irrigation-aspiration suction time=81.3±45.9 seconds. Acute postoperative corneal edema occurred in 4 eyes (13.3%). After one-week BCVA was 0.5±0.2.9 eyes (30%) had visual acuity <0.5. They were treated aggressively with antibiotics (moxifloxacine), corticosteroid (dexamethasone) and hypertonic eye drops (sodium chloride (5%) and mannitol (20%)), every hour during the first week and gradually tapered in the 1 month. Antiglaucomatose evedrops (timolol, brinzolamide) were used to control the IOP below 20 mmHg. After 1 month mean BCVA increased to 0.85±0.15 and all eyes reached BCVA higher than 0.6. IOP was stable at 15.4±2.0 mmHg. The mean endothelial cell loss was 19,1%. None of the eyes progressed to chronic edema. Corneal edema is a common complication after surgery of difficult cataracts. Even though the cataract density directly influences the postoperative condition of the corneal endothelium, surgical trauma is still considered the most common cause of corneal endothelial decompensation. Preoperative specular microscopy is very important to predict possible postoperative complications of the corneal endothelium and apply appropriate surgical techniques and materials. Modern phaco-techniques (low phaco-energy, small incision site, new irrigation solutions and OVDs) can significantly reduce endothelial cell loss after cataract surgery. It is recommended to treat postoperative corneal edema and inflammation with topical corticosteroids, topical hypertonic agents and to maintain intraocular pressure below 20 mmHg.

Keywords: corneal edema, phacoemulsification, corneal endothelium Field: Medical sciences and Health, Ophthalmology

INTRODUCTION

The corneal endothelium is a monolayer of hexagonal cells, which functions as a barrier between the corneal stroma and aqueous humour and is crucial in maintaining the transparency of the cornea. Previous studies have shown that the endothelial cell count is approximately 5000 cells/mm2 at birth, with a rate of physiological cell decline with aging of about 0,6% per year, which leaves enough endothelial cells for normal corneal function during lifetime. The endothelium sustains corneal clarity by maintaining the hydration of the corneal stroma at 78%, through a mechanism of active ion pump. Corneal edema due to inadequate endothelial pump function is one of the most common complications of cataract surgery.

Every cataract surgery (even the 'perfect' one) damages the endothelial cells (Rosado-Adames, Afshari, 2012), although if this endothelial cell loss is small, it doesn't cause any permanent damage.

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Sometimes cataract surgery causes disruption of the endothelial cells pump function, which leads to corneal blurring, poor vision and discomfort. At this stage the endothelial cells may recover with aggressive topical treatment in the first months after surgery. Irreversible corneal edema (bullous keratopathy) occurs only if a significant number of endothelial cells were damaged during surgery, resulting in permanent decrease of endothelial cell density, which can no longer maintain corneal transparency. The patient will experience permanent blurring of vision and ocular pain (Choi, Han, 2019).

The main predisposing factors for corneal edema in cataract surgery are: intraoperative mechanical endothelial trauma, corneal dystrophy (ex. Fuchs' endothelial dystrophy), prolonged postoperative inflammation, and high intraocular pressure (IOP). Surgical trauma is the most common cause of corneal decompensation during phacoemulsification. Several factors can be identified in this process. The phacoemulsification probe increases the local temperature, leading to thermal damage of the cornea. The endothelium can be damaged during the irrigation/aspiration phase due to turbulent flow of air bubbles and lens particles in the anterior chamber. Prolonged phaco-time and high ultrasound energy used is associated with the production of free radicals, that can cause damage to the corneal endothelium through a process known as oxidative stress (Cameron et al, 2001).

In the last decade, the advances in phacoemulsification techniques and materials, such as new and improved IOLs and ophthalmic viscosurgical devices, significantly reduced the number of cases of chronic edema (bullous keratopathy) after cataract surgery.

MATERIAL AND METHODS

We conducted a prospective interventional clinical study of 30 patients, operated in February/March 2022. All patients had senile cataract, of which over 80% were advanced cataracts: 18% hypermature, 36% brunescent, 30% grade 4 and 16% grade 3 cataracts. Uneventful phacoemulsification with IOL implantation was performed by one experienced phaco-surgeon in an outpatient setting. Exclusion criteria were: pathologies of the cornea (cornea guttata, scars), pseudoexfoliation, intraocular inflammatory diseases, macular degeneration, proliferative vitreoretinopathy, insufficiently treated glaucoma and other pathologies of the eye that could negatively impact the surgical outcome.

Preoperative parameters included: best-corrected visual acuity (BCVA) in Snellen decimal units, intraocular pressure (IOP), cataract density (slit lamp examination), corneal endothelium cell density (ECD) and hexagonality measured with a specular microscope. The preoperative examination also included optical coherence tomography of the retina (where available), echography and immersion biometry for IOL calculation. Intraoperative parameters included: phacoemulsification time and energy, irrigation–aspiration suction time.

The cataract surgery was performed as follows: topical anesthesia (0,5% Alkaine), superior clear-cornea incision (2.4 mm), two side-ports, anterior chamber injection of ophthalmic viscosurgical device (OVD) (Alcon DisCoVisc®), anterior capsulorhexis, hydrodissection, pha¬coemulsification (Alcon INFINITI® Vision System), IOL implantation (Alcon AcrySof®) in-the-bag with single-use injector, intracameral antibiotic (vancomycin).

Postoperative follow-up was done on 1-day (IOP, slit-lamp exam), 1-week (BCVA, IOP, slit-lamp exam) and 1-month after surgery (BCVA, IOP, slit-lamp exam, specular microscopy).

RESULTS

Mean age of the patients was 65 ± 12 years, 61% male, 39% female. Mean baseline parameters were: BCVA=0.1\pm0.13, IOP=15.7\pm2.7 mmHg, ECD=2,497\pm290 cells/mm2, cell hexagonality was $54.3\pm9.4\%$.

Mean surgical parameters were: surgical time= $9,3\pm2.9$ minutes, phacoemulsification time= 35.6 ± 26.1 seconds, phacoemulsification energy= 13.3 ± 10.9 J, irrigation–aspiration suction time= 81.3 ± 45.9 seconds.

Acute postoperative corneal edema occurred in 4 eyes (13.3%). After 1-week BCVA improved to 0.5±0.2. 9 eyes (30%) had visual acuity ≤0.5. They were treated aggressively with antibiotics (moxifloxacine), corticosteroid (dexamethasone) and hypertonic eye drops (sodium chloride (5%) and mannitol (20%)), every hour during the first week and gradually tapered in the 1 month. Antiglaucomatose eyedrops (timolol, brinzolamide) were used to control the IOP below 20 mmHg. Artificial tears were prescribed to reduce the discomfort.

After 1 month mean BCVA increased to 0.85±0.15 and all eyes reached BCVA higher than 0.6. IOP was stable at 15.4±2.0 mmHg. ECD decreased to 1996±612 cells/mm2, whereas cell hexagonality

dropped to 43.6±12.5%. The mean endothelial cell loss was 19,1%. In 5 (16.7%) eyes we recorded improvement in ECD/hexagonality after the surgery. None of the eyes progressed to chronic edema.

DISCUSSION

Improvement in phacoemulsification techniques and materials can reduce permanent endothelial damage after cataract surgery. A comprehensive preoperative preparation, including patient characteristics such as age, stage of the cataract, presence of corneal disease is crucial in this process and influences the intraoperative parameters (incision size, phacoemulsification technique, total ultrasonic energy, materials etc.) that are optimal for corneal safety.

In previous studies endothelial cell loss after cataract surgery ranges from 4% up to 25%. In a prospective randomised study (Bourne, 2004), endothelial cell loss showed no difference between patients treated with phacoemulsification or with extracapsular cataract extraction (ECCE), although higher amounts of cell loss was found in eyes with hard cataracts, capsule break, and vitreous loss.

Regarding phacoemulsification procedures, to reduce the amount of used ultrasonic energy most major platforms use power modulation and torsional phacoemulsification to emulsify the lens, instead of longitudinal motion of the phaco tip, thus reducing the necessary phaco-power for all grades of cataract. Regarding the materials, the composition of the irrigating solution is more important to endothelial safety than is the duration of irrigation. Fortified BSS (BSS Plus,Alcon), in contrast to standard BSS and Ringer's solution, has pH, osmolarity, bicarbonate, glucose and glutathione content similar to those of the aqueous and can therefore improve corneal safety in cases of prolonged surgery or corneas with endothelial disease.

The ophthalmic viscosurgical devices used in cataract procedures can also influence the risk of acute endothelial cell loss (Storr-Paulsen et all, 2007). While low viscosity dispersive OVDs (Viscoat), provide protection against air-bubble damage because they adhere to corneal endothelium, they are not as good at maintaining anterior chamber depth and stability as cohesive OVDs (Healon, Provisc). They are also harder to remove from the eye at the end of surgery (Moschos et al, 2011). Therefore, the soft-shell technique which combines dispersive with cohesive OVDs was developed in 1999, to facilitate the surgery and enhance the safety for the corneal endothelium. In comparison, the new viscous dispersive OVDs (DisCoVisc) which have been developed in the last decade provide better endothelial cell protection during phaco and are easier to remove from the eye at the end of surgery.

Regarding the size, site of incision or different nuclear fractioning technique, studies conducted so far have not found significant differences in the rates of endothelial cell loss, but generally, smaler incision sizes are considered safer for the corneal endothelium incisions (Walkow et al, 2000).

Certain corneal diseases (cornea guttata, Fuchs' dystrophy etc.), may be a predisposing factor to endothelial cell loss after cataract surgery (Chang et al, 2020). Therefore, preoperative specular microscopy is very important to predict possible postoperative complications, adequately plan the operation course and materials and methods used and determine if combined procedure (phaco, IOL, keratoplasty) should be performed.

Postoperatively corneal edema and inflammation should be aggressively treated with topical corticosteroids. Intraocular pressure should be controlled below 20 mmHg, because increased IOP can compromise endothelial cell function, lead to epithelial edema, and cause further endothelial damage. Several treatment options are available to reduce discomfort and increase visual acuity. Epithelial oedema can be treated with topical hypertonic agents (sodium chloride 5%, mannitol 20% solution) in the form of eyedrops. Hypertonic eye ointment at night is particularly useful because the edema tends to be more severe on waking in the morning due to lack of evaporation during the night when the eyes are closed. Bandage contact lenses may be useful as an adjunct to medical treatment for the temporary relief of corneal pain and discomfort. They act to shield the cornea and epithelium from the eyelid.

Pseudophakic bullous keratopathy is a condition of irreversible edema and endothelial cell damage that occurs after cataract surgery. The definitive treatment of this chronic corneal edema is a corneal transplant. Corneal transplantation is indicated when vision is decreased significantly by corneal edema or when pain becomes intractable. Keratoplasty can be either a partial thickness transplant [e.g., Descemet's stripping endothelial keratoplasty (DSEK), Descemet membrane endothelial keratoplasty (DMEK)] or a full thickness corneal transplant.

CONCLUSION

Corneal edema is a common complication after surgery of advanced cataracts. Even though the cataract density directly influences the postoperative condition of the corneal endothelium, surgical trauma is still considered the most common cause of corneal endothelial decompensation.

Preoperative specular microscopy is very important to predict possible postoperative complications of the corneal endothelium and apply appropriate surgical techniques and materials. Modern phacotechniques (low phaco-energy, small incision site, new irrigation solutions and OVDs) can significantly reduce endothelial cell loss after cataract surgery.

It is recommended to treat postoperative corneal edema and inflammation with topical corticosteroids and topical hypertonic agents, as well as to maintain intraocular pressure below 20 mmHg.

REFERENCES

Bourne RR, Minassian DC, Dart JK, Rosen P, Kaushal S, Wingate N. Effect of cataract surgery on the corneal endothelium: modern phacoemulsification compared with extracapsular cataract surgery. Ophthalmology. 2004;111(4):679-685.

Cameron MD, Poyer JF, Aust SD. Identification of free radicals produced during phacoemulsification. J Cataract Refract Surg. 2001;27(3): 463–470.

- Chang VS, Gibbons A, Osigian C. Phacoemulsification in the Setting of Corneal Endotheliopathies: A Review. Int Ophthalmol Clin. 2020 Summer;60(3):71-89.
- Choi JY, Han YK. Long-term (≥10 years) results of corneal endothelial cell loss after cataract surgery. Can J Opthalmol. 2019;54:438-444.
- Díez-Ajenjo MA, Luque-Cobija MJ, Peris-Martínez C, Ortí-Navarro S, García-Domene MC. Refractive changes and visual quality in patients with corneal edema after cataract surgery. BMC Ophthalmol. 2022 Jun 2;22(1):242.

Farrell RA, McCally RL. Corneal transparency. In Principles and Practice of Ophthalmology, Albert DM, Jakobiec FA (Eds.), Saunders WB, Philadelphia, PA.2000;pp 629–643.

Hayashi K, Hayashi H, Nakao F, Hayashi F. Risk factors for corneal endothelial injury during phacoemulsification. J Cataract Refract Surg. 1996;22(8):1079–1084.

Kim DH, Wee WR, Lee JH, Kim MK. The comparison between torsional and conventional mode phacoemulsification in moderate and hard cataracts. Korean J Ophthalmol. 2010;24(6):336-340.

Lee HY, Choy YJ, Park JS. Comparison of OVD and BSS for maintaining the anterior chamber during IOL implantation. Korean J Ophthalmol. 2011;25(1):15–21.

Moschos MM, Chatziralli IP, Sergentanis TN. Viscoat versus Visthesia during phacoemulsification cataract surgery: corneal and foveal changes. BMC Ophthalmol. 2011;11:9.

Pirazzoli G, D'Eliseo D, Žiosi M, Acciarri R. Effects of phacoemulsification time on the corneal endothelium using phacofracture and phacochop techniques. J Cataract Refract Surg. 1996;22(7):967-969.

Rosado-Adames N, Afshari NA. The changing fate of the corneal endothelium in cataract surgery. Curr Opin Ophthalmol. 2012; 23(1):3-6.

Sandali O, El Sanharawi M, Tahiri Joutei Hassani R, Roux H, Bouheraoua N, Borderie V. Early corneal pachymetry maps after cataract surgery and influence of 3D digital visualization system in minimizing corneal oedema. Acta Ophthalmol. 2022 Aug;100(5):e1088-e1094.

Storr-Paulsen A, Nørregaard JC, Farik G, Tårnhøj J. The influence of viscoelastic substances on the corneal endothelial cell population during cataract surgery: a prospective study of cohesive and dispersive viscoelastics. Acta Ophthalmol Scand. 2007;85(2):183–187. Walkow T, Anders N, Klebe S. Endothelial cell loss after phacoemulsification: relation to preoperative and intraoperative

parameters. J Cataract Refract Surg. 2000;26(5):727-732.

Weinstock RJ, Diakonis VF, Schwartz AJ & Weinstock AJ. Heads-up cataract surgery: complication rates, surgical duration, and comparison with traditional microscopes. J Refract Surg. 2019;35: 318-322.