VNUHCM Journal of Science and Technology Development 2026, 29(3):4263-4270

Open Access Full Text Article

Review Article

Assessment of Corneal Endothelial Cell Damage in Phacoemulsification Surgery: A Clinical Perspective

Pham Thi Thuy Tien

Use your smartphone to scan this QR code and download this article

ABSTRACT

Phacoemulsification is the current standard surgical technique for cataract extraction because of its safety, minimal incision requirement, and rapid visual recovery. Despite continuous technological advances, corneal endothelial cell damage remains an important concern after surgery, particularly in patients with pre-existing endothelial dysfunction. Since corneal endothelial cells have limited regenerative capacity, excessive cell loss can result in postoperative corneal edema, endothelial decompensation, and impaired visual outcomes. This review summarizes the current understanding of corneal endothelial alterations associated with phacoemulsification, including endothelial physiology, mechanisms of intraoperative injury, postoperative morphological changes, and major risk factors affecting endothelial survival. Special attention is given to high-risk conditions such as glaucoma, pseudoexfoliation syndrome, and diabetes mellitus, which are associated with increased endothelial vulnerability and delayed corneal recovery. Current evidence suggests that endothelial cell loss after phacoemulsification is influenced by multiple factors, including cataract density, ultrasound energy, cumulative dissipated energy, surgical duration, fluidics, ocular comorbidities, and surgical technique. Modern approaches such as torsional phacoemulsification, optimized fluidics, reduced ultrasound energy, femtosecond laser-assisted cataract surgery, and the appropriate use of dispersive ophthalmic viscosurgical devices may help minimize endothelial injury. Careful preoperative endothelial assessment, individualized surgical planning, and endothelial-protective strategies are essential to preserve corneal clarity and improve postoperative outcomes, especially in eyes at high risk of endothelial decompensation.

Key words: corneal endothelial cells, phacoemulsification, endothelial cell loss, glaucoma, diabetes mellitus, pseudoexfoliation syndrome

Ho Chi Minh City Eye Hospital, Ho Chi Minh, Vietnam

Correspondence

Pham Thi Thuy Tien, Ho Chi Minh City Eye Hospital, Ho Chi Minh, Vietnam

History

- Received: 25-06-2025
- Revised: 22-05-2026
- Accepted: 10-06-2026
- Published Online: 27-09-2026

DOI : 10.32508/vnuhcmj-std.v29i3.4531

Check for updates

INTRODUCTION

Phacoemulsification is currently the standard surgical method for treating cataracts, favored for its effectiveness and quick recovery time. However, the phacoemulsification tip can generate heat, posing a risk of thermal damage to surrounding tissues, particularly the corneal endothelium. This innermost layer of the cornea comes into direct contact with aqueous humor and plays a crucial role in maintaining corneal structure and function through its fluid-pumping system.

Endothelial cells can be damaged by factors such as nuclear hardness, phacoemulsification time, ultrasound energy, and intraocular lens (IOL) implantation manipulation. Morphological indicators, such as endothelial cell density (ECD), coefficient of variation (CV), and percentage of hexagonal cells (HEX), can be used to evaluate endothelial function. Abnormalities in these parameters—combined with increased central corneal thickness (CCT)—suggest loss of endothelial function, fluid imbalance, stromal edema, and corneal decompensation. Thus, assessing corneal endothelial cells is critical for prognosis and visual outcomes following phacoemulsification surgery.

CORNEAL ENDOTHELIAL CELLS

The cornea is located at the front of the eye and accounts for about one-fifth of the outer surface area of the eyeball. Structurally, the cornea consists of five layers from anterior to posterior: the epithelium, Bowman's membrane, stroma, Descemet's membrane, and endothelium. Of these, the corneal endothelium is the innermost layer, directly contacting the aqueous humor.

At birth, the human corneal endothelium consists of a single layer of hexagonal cells, totaling approximately 500,000 cells at a density of about 7,500 cells/mm^2. Over time, this cell density declines, with cells not exhibiting regenerative capacity. The most significant reduction in ECD occurs during the first year of life, followed by a slight decrease through the twenties, and then a gradual decline with aging. From age 20 to 80, the average annual decline in ECD is estimated

Cite this article : Tien P T T. **Assessment of Corneal Endothelial Cell Damage in Phacoemulsification Surgery: A Clinical Perspective**. *VNUHCM J. Sci. Technol. Dev.* 2026; 29(3):4263-4270.

at approximately 0.52%.

At birth, density ranges from 3,500–4,000 cells/mm^2, decreasing to 2,500–3,000 cells/mm^2 in adulthood. A minimum of 500–1,000 cells/mm^2 is required to maintain corneal transparency [1].

PHYSIOLOGY OF CORNEAL ENDOTHELIAL CELLS

The endothelium plays an important role in maintaining both the shape and the function of the cornea, particularly in preserving its transparency via the endothelial pump mechanism (Figure 1). This layer has two primary functions, supporting corneal structure and clarity by regulating fluid balance within the stroma. Unlike other tissues, the corneal stroma lacks direct vascular supply; therefore, the endothelium must maintain a hydration equilibrium. It does so by serving as both a barrier to aqueous influx and a metabolic pump, actively transporting ions and drawing water out of the stroma into the aqueous humor through osmotic gradients. This stabilizes CCT at approximately 0.5 mm (with 78% stromal hydration) [1].

Tight junctions between endothelial cells act as a barrier. When the fluid inside the cornea is hypotonic relative to the aqueous humor, water moves passively through the endothelial barrier into the stroma to supply oxygen and nutrients. In addition, corneal endothelial cells actively remove excess water from the stroma via Na^+/K^+-ATPase ion pumps (see Diagram 1), maintaining the osmotic gradient. This fluid balance can be disrupted by corneal disorders, such as Fuchs' dystrophy, posterior polymorphous corneal dystrophy (PPCD), iridocorneal endothelial (ICE) syndrome, elevated intraocular pressure (IOP), or surgical/mechanical trauma.

Because endothelial cells cannot regenerate by mitosis, and their numbers naturally decline with age, any cell loss is compensated for by two main mechanisms:

Morphological Changes of Corneal Endothelial Cells

Changes in cell function

Postoperative endothelial cells are deformed into irregular polygonal cells, causing changes in the density of cell pumps and cell permeability. Each cell has 3 million endothelial pump sites on the side of the cell membrane. After surgery, corneal functional compensation is shown by an increase in the number of endothelial pumps. Cell physiology is restored after a few weeks. If cell loss exceeds the functional threshold—that is, less than 500 cells/mm^2—the ability to compensate for the number of pumps is overcome; the cells are stretched thin, and the contact area between them is not enough for the endothelial pumps, thus causing endothelial decompensation and edema.

Changes in cell morphology (pleomorphism)

In the process of filling gap at the site of endothelial cell loss, adjacent cells tend to extend the cytoplasm to slide into the exposed Descemet's membrane. This distorts the normal hexagonal shape of the endothelial cells, as shown by a decrease in HEX [1].

Cell size variation (polymegethism). When endothelial cells die, adjacent cells tend to enlarge to cover the damaged area. Therefore, after injury recovery, in addition to the decrease in ECD, the cells in the damaged area tend to be larger than other cells. The size of these cells is also uneven. This difference in cell size is called polymegethism (Figure 2). If repeated trauma or inflammation reduces the ECD below the annual standard, the healing mechanism is insufficient to maintain physiological function, leading to damage of the neighboring endothelial cells, thereby decompensating and causing corneal edema [1].

Endothelial Healing

When injury occurs, neighboring cells rapidly develop pseudopodia and migrate from all directions to cover the wound (Figure 3). Once cells meet at the center of the defect, migration stops, and mitotic processes are initiated. However, mitosis halts at the G1 phase, meaning that endothelial cells do not increase in number. Mitotic activity at the wound margin begins at approximately 16 hours and peaks at 24–36 hours post-injury. Studies show that if healing is uncomplicated, the endothelial layer stabilizes within 3 months after phacoemulsification [4].

PHACOEMULSIFICATION

Phacoemulsification is now the most commonly performed surgery for cataract treatment: it is the safest intervention, as it requires only a small incision and has a lower risk of perioperative complications and a faster recovery. Despite its many advantages, phacoemulsification surgery significantly reduces the number of corneal endothelial cells. The use of high-intensity ultrasound energy during phacoemulsification to dissociate and emulsify the nucleus is considered an important cause of corneal endothelial cell loss. The vibration of ultrasound waves in water causes the collapse of gas or vapor bubbles in the fluid. The growth of bubbles is due to a decrease in local pressure in the aqueous humor, while subsequent collapse is due to an increase in pressure. The rupture of

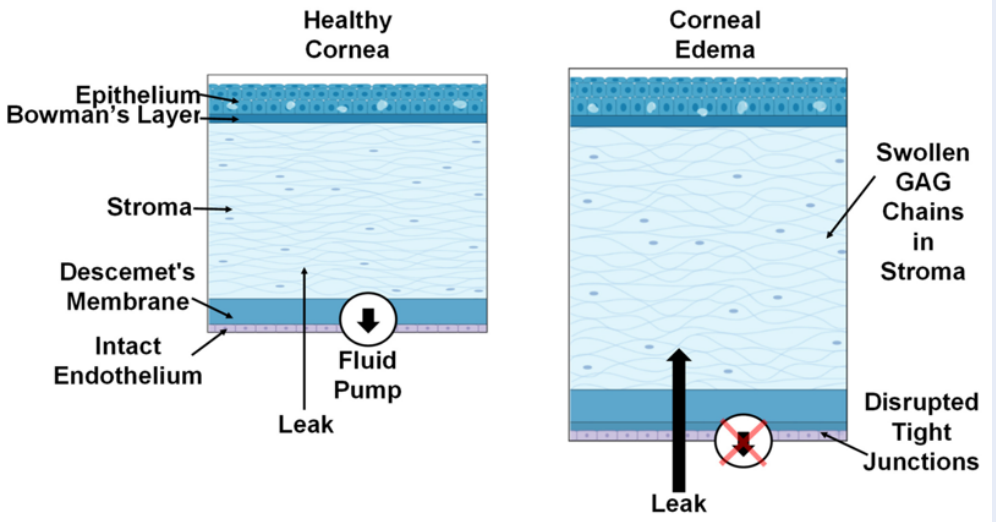

Figure 1: Fluid pump system of cornea (Source: Sensors, 2022)[2]

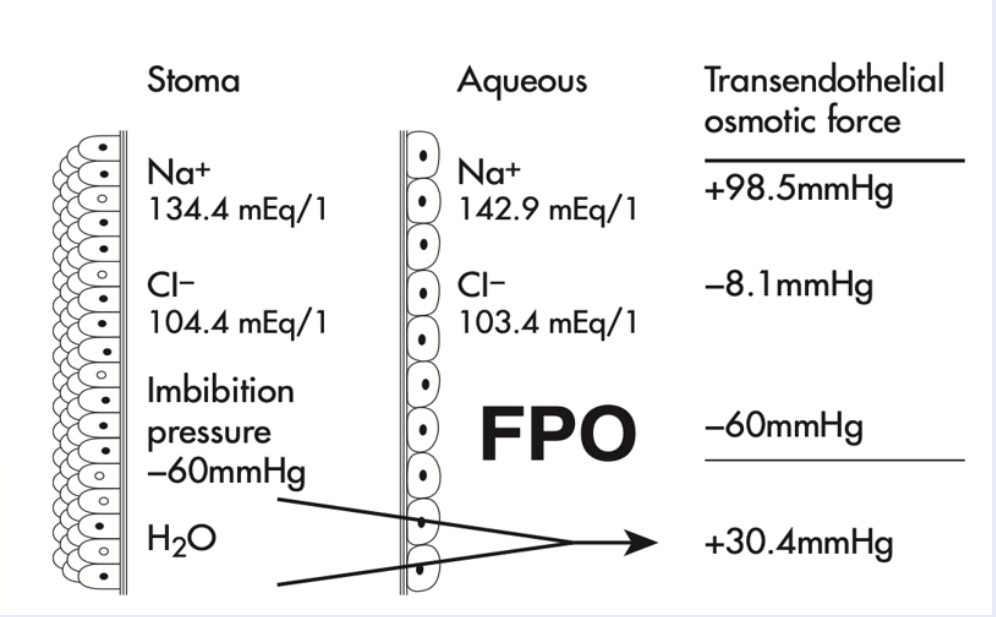

Diagram 1: Illustration of the total osmotic pressure across the endothelial cell due to Na^+ and Cl^- activity, along with osmotic pressure. Although the Na^+concentration in the aqueous humor is greater than that in the parenchyma (142.9 vs. 134.4 mEq/L, $p < 0.05$), the osmotic pressure due to Na^+ is 98.5mmHg. Similarly, the pressure for Cl^- and the osmotic pressure in the parenchyma are −8.1 and −60 mmHg, respectively. The sum of these pressures results in a total osmotic pressure of +30.4 mmHg, which ultimately results in the movement of water from the parenchyma to the outside (Source: Corneal Surgery, 2009)[1].

Table 1: Comparison of endothelial cell density (ECD) by ethnicity: Indian, American, Chinese, Filipino, Japanese [1].

	India		America		Chinese		Filipino		Japanese	
Age groups (years)	No.of eyes	ECD (cells/mm^2)	No.of eyes	ECD (cells/mm^2)	No.of eyes	ECD (cells/mm^2)	No.of eyes	ECD (cells/mm^2)	No.of eyes	ECD (cells/mm^2)
20–30	104	2,782 ± 250	11	2,977 ± 324	100	2,988 ± 243	114	2,949 ± 270	18	3,893 ± 259
31–40	96	2,634 ± 288	6	2,739 ± 208	100	2,920 ± 325	112	2,946 ± 296	10	3,688 ± 245
41–50	97	2,408 ± 274	11	2,619 ± 321	97	2,935 ± 285	112	2,761 ± 333	10	3,749 ± 407
51–60	98	2,438 ± 309	13	2,625 ± 172	97	2,810 ± 321	102	2,555 ± 178	10	3,386 ± 455
61–70	88	2,431 ± 357	8	2,684 ± 384	90	2,739 ± 316	114	2,731 ± 299	6	3,307 ± 330
> 70	54	2,360 ± 357	15	2,431 ± 339	83	2,778 ± 365	86	2,846 ± 467	15	3,289 ± 313

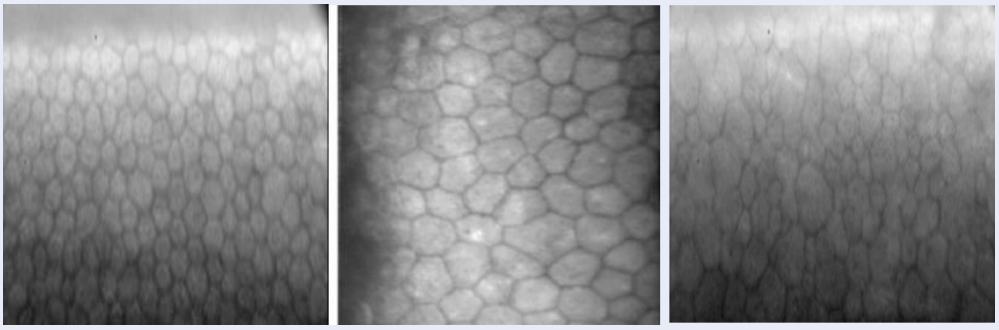

Figure 2: Corneal endothelial cell shapes: Normal endothelial cells (left), Polymegethism (middle), Pleomorphism (right). (Source: Annu Int Conf IEEE Eng Med Biol Soc, 2016) [3].

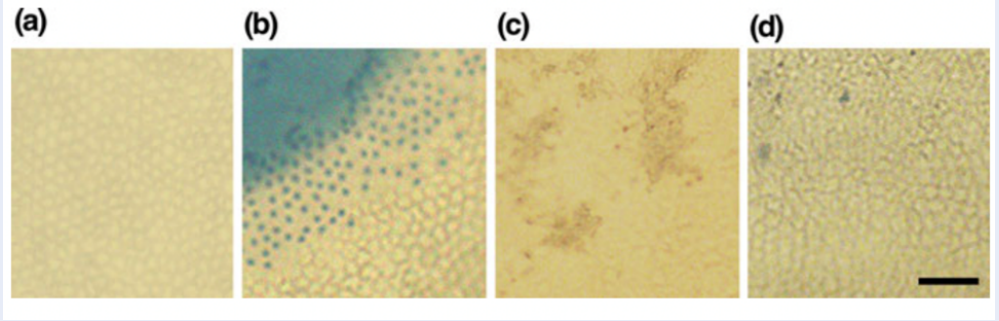

Figure 3: The repair of the corneal endothelium in organ culture. Phase contrast micrographs of the endothelium before and after the induction of a lesion in the part of the cornea: (a) before lesioning; (b) dead cells present immediately after lesion induction; (c) 7 days post lesion with no dead cells but cell debris is present; (d) the endothelium is completely repaired 14 days after induction of the lesion. Dead cells are stained by trypan blue. (Source: The Cornea, Anatomy and Function, 2017) [4].

the gas bubbles generates microscopic shock waves, resulting in a local increase in pressure and temperature, then possibly leading to the direct dissociation of water molecules. The water molecules then dissociate into hydrogen and hydroxyl radicals, which are among the most reactive free radicals that can induce oxidative stress in endothelial cells[5].

Endothelial damage after phacoemulsification is one of the most devastating corneal complications. The integrity of the endothelium is essential for complete corneal transparency. Endothelial function is threatened in all intraocular surgeries, especially for cataracts. Cataract surgery often results in immediate endothelial dysfunction, and more rarely in permanent postoperative dysfunction (bullous keratopathy) or late-onset dystrophy. The severity of endothelial damage after surgery depends on many factors, including the hardness of the lens, the time of using energy to emulsify the lens, the depth of the anterior chamber, and complications during and after surgery. The degree of cell damage also depends on the technique of cataract removal as well as the placement of an IOL.

In the 1970s, when intracapsular cataract extraction (ICCE) was common, the rate of endothelial cell loss in each uncomplicated case was 10%–15%, and in each ICCE with iris-fixated IOL placement, the average rate was about 25%–30% within a follow-up period of 3 months. The extracapsular cataract extraction (ECCE) method was popularized in the early 1980s, reducing the rate of endothelial cell loss to about 12%. The use of ophthalmic viscosurgical devices (OVDs) later reduced the rate of endothelial cell loss to 10%.

Phacoemulsification surgery has been implemented since the 1970s and has been gradually improved. Over a long period of follow-up, phacoemulsification surgery is reported to cause endothelial cell loss of about 30%. However, with advances in technology, the rate of endothelial cell loss after phacoemulsification surgery is currently about 7%. The rate of cell loss depends on the type of IOL and the placement technique; with iris claw IOL fixation, the rate of cell loss is 40%, due to direct contact between the IOL and endothelial cells. In addition, uveitis, hyphema, and secondary glaucoma are common complications, contributing to increased endothelial cell damage, and sometimes causing bullous keratopathy.

According to a study by Werblin in 1993, the rate of corneal endothelial cell loss after phacoemulsification surgery was about 9%. The technique of placing an IOL in the posterior chamber after ECCE or phacoemulsification significantly reduced postoperative endothelial damage. The rate of endothelial cell loss was reported as 20% after anterior chamber IOL fixation, compared with 12% for posterior chamber IOL fixation[6].

Various rates of endothelial cell loss after surgery have been reported in different locations. Schultz and colleagues[5] examined changes in corneal thickness, alongside the number and morphology of endothelial cells in each corneal region. The change in cell number and morphology (decreased HEX, increased cell shape, increased CV) occurred most strongly at the area near the surgical site, within 1 week postoperatively. This change occurred in the central corneal region 1 month after surgery. Studies by Schultz[5] (1986), Rao[7] (1981), and Galin[8] (1979) reported that the ECD stabilized and morphological changes stopped within 3 months after surgery in all corneal areas.

Many studies have shown that diabetic patients undergoing conventional phacoemulsification lose more endothelial cells than those undergoing femtosecond laser-assisted cataract surgery (FLACS). FLACS uses less energy, which helps reduce endothelial damage[9]. Therefore, FLACS may be a safer choice in this group of patients.

The rate of corneal endothelial cell loss after phacoemulsification surgery varies depending on many factors, including surgical technique, ultrasound energy, nucleus hardness, history of eye diseases (glaucoma, diabetes, pseudoexfoliation syndrome [PXS]), and surgeon experience. The harder the cataract nucleus (Lens Opacities Classification System III [LOCS III]: NO_4–NO_6), the higher the phacoemulsification energy used, the greater the loss of corneal endothelial cells. Cumulative dissipated energy (CDE) is a factor because the higher the accumulated energy, the greater the endothelial damage. The longer the phacoemulsification time, the more corneal edema and cell death[5–8]. A retrospective study at Chang Gung Hospital (Taiwan)[9] showed that patients with preoperative ECD of 1,000–2,000 cells/mm^2 had more cell loss than those with density < 1,000 or > 2,000 cells/mm^2. This suggests that the "intermediate" density group is at higher risk of postoperative injury. Another study that followed up for 7 years after phacoemulsification surgery showed that endothelial cell loss not only occurred immediately after surgery, but also continued to progress over a long period, especially in patients with increased CCT after surgery[10]. A review comparing the "direct-chop" and "stop-and-chop" techniques showed that the "direct-chop" technique reduced surgery time and ultrasound energy

Table 2: Rate of endothelial cell loss after phacoemulsification surgery.

Time after surgery	Average rate of endothelial cell loss
1 month	11.6%–17.9%
2 months	9.97%–16.7%
6 months	10%–15%
1 year	7.3%–12%
10 years	20.6%

used, thereby reducing endothelial cell injury and improving postoperative visual acuity [11]. Another study showed that the use of low-pressure and low-negative-pressure irrigation patterns in phacoemulsification surgery was safe and effective for patients with low ECD, reducing endothelial cell injury [12].

Corneal endothelial cell damage during phacoemulsification in glaucoma patients

Increased IOP and corneal endothelial cell damage

Glaucoma, especially acute primary angle closure (APAC), can cause endothelial cell damage resulting from increased IOP. A recent study showed that changes in IOP during phacoemulsification can cause mechanical stress on the corneal endothelium, potentially causing endothelial damage. Preoperative IOP control is therefore an important factor in protecting endothelial cells. The CV, HEX, and CCT indices also decreased compared with healthy eyes [13].

In glaucoma patients, high IOP damages endothelial cells before surgery, and glaucoma medications (such as prostaglandins and timolol) also microscopically affect endothelial cells. Now, combined glaucoma surgery with phacoemulsification increases the risk of further endothelial cell loss, causing corneal edema and prolonged endothelial loss [13].

Phacoemulsification combined with glaucoma surgery

Phacoemulsification surgery combined with glaucoma treatment procedures—such as trabeculectomy or minimally invasive glaucoma surgery (MIGS)—can affect endothelial cells. Obuchowska et al. (2022) assessed the postoperative endothelial cell loss rate of combined surgery to be 13.2% in the first 6–29 months [14].

The rate of endothelial cell loss after combined phacoemulsification and trabeculectomy in the open-angle glaucoma group after 3 months, 1 year, and 6–29 months was approximately 9%–15%, 12.6%, and 13.2%, respectively [13].

Phacoemulsification in patients with PXS

PXS is associated with the accumulation of fibrillar material on structures in the eye, including endothelial cells. This leads to a decrease in ECD before surgery compared with healthy people of the same age, increasing the risk of endothelial cell damage during and after surgery. This increased risk is due to factors such as weak zonular ligaments that can easily cause vibration or dislocation of the lens; vibration of the nucleus during phacoemulsification, increasing collision with the endothelium; difficulty in manipulating the shallow anterior chamber during surgery; and pseudoexfoliative material causing trabecular meshwork obstruction, increasing IOP [15–19].

In India, Khanday et al. (2024) reported that a PXS patient group had a decrease in ECD from 2,367.9 to 2,083.2 cells/mm^2 1 year after surgery (equivalent to a loss of about 12%), while the non-PXS group had a decrease from 2,665.4 to 2,545.8 cells/mm^2 (about 4.5%) [17]. Meanwhile, Hasegawa et al. (2016) showed that the rate of endothelial cell loss after surgery was 18.1% in their PXS group and 11.6% in their non-PXS group [18]. Similarly, Hayashi et al. (2013) showed mean endothelial cell loss after 3 months of 13.1% in the PXS group and 7.5% in the non-PXS group [19].

Cataract surgery can cause similar changes in endothelial cells without increasing the rate of endothelial cell loss after surgery. This suggests that although ECD is reduced preoperatively, phacoemulsification surgery did not significantly increase the rate of endothelial cell loss after surgery in patients with PXS [15].

The rate of endothelial cell loss after phacoemulsification surgery in eyes with PXS was significantly higher (average 25%) compared with control eyes (average 11%).

Doan Kim Thanh et al. (2024) examined 70 eyes for changes in the morphology and number of corneal endothelial cells before and after phacoemulsification surgery in patients with PXS. ECD was lower, and visual acuity improved less well at 6 months postoperatively in the PXS group compared with the control

group [20]. Phacoemulsification time and phacoemulsification energy (CDE and FP3) were found to correlate with the degree of corneal endothelial cell damage postoperatively [21].

Phacoemulsification in diabetic patients

Diabetes mellitus affects the cornea, corneal nerves, corneal epithelium, and endothelium. People with diabetes mellitus often have dry eye syndrome (50%–60%) due to damage to cranial nerves V and VII, which disrupts tear secretion. The risk of keratitis and corneal ulcers also increases due to reduced corneal sensation, dry eyes, and immunodeficiency. Re-epithelization of the corneal epithelium is delayed due to the damaged basement membrane and weak connections, leading to infection and slow healing [22]. Prolonged hyperglycemia increases sorbitol formation through the polyol pathway: the Na^+/K^+-ATPase pump is reduced in activity due to increased advanced glycation end products (AGEs) and oxidative stress. The consequence of this is the reduced ability to retain corneal tears, often leading to postoperative corneal edema. Diabetic patients have lower ECD than non-diabetics; the risk increases with disease duration and poorer glycemic control. CV increases, and HEX decreases; ECD decreases and morphological disorders appear, including altered cell size (polymegethism) and reduced HEX (pleomorphism). In addition, corneal nerve damage reduces corneal sensation, causing delayed wound healing [23].

Research has shown that the rate of endothelial cell loss in diabetics 1 month and 3 months after phacoemulsification was 14.3% and 18.2%, respectively [21]. The risk of corneal edema is higher due to the weak endothelium and the slow healing of the corneal epithelium, causing prolonged vision loss and increasing the risk of postoperative keratitis.

Prevention of complications after phacoemulsification surgery in diabetics therefore requires specific treatment strategies. To protect the epithelium and improve wound healing, artificial tears, lubricating ointments, and protective glasses should be used. Controlling blood sugar (HbA1c < 7%) helps improve corneal wound healing, and prophylactic antibiotics can prevent infection. In one study, diabetic patients lost an average of 472.7 cells/mm^2 after phacoemulsification, significantly higher than 165.7 cells/mm^2 in the non-diabetic group [24]. HEX decreased more in the diabetic group in weeks 1 and 4 after surgery. Phacoemulsification caused more endothelial damage in diabetic patients, with a slower recovery time. Indices such as ECD, CCT, CV, and HEX were more affected in the diabetic group [22]. A study of 200 Indian patients showed that the diabetic group lost more endothelial cells and recovered more slowly than the non-diabetic group. The longer the duration of diabetes, the greater the endothelial cell loss [24].

In summary, among diseases with a risk of endothelial cell damage, PXS has the highest rate of endothelial cell loss (22%–28%), with concomitant glaucoma (9%–15%), but also exhibited stability after 3 months. In contrast, diabetic patients (14%–18%) have a risk of slow-recovery corneal edema.

CONCLUSIONS

It is necessary to assess ECD before surgery, especially in high-risk patients, such as those with glaucoma, PXS, and diabetes. Follow-up after surgery is crucial, especially in the first 3 months, when the rate of endothelial cell loss is highest. For eyes at risk of endothelial cell damage, periodic endothelial cell evaluation is required to detect complications early. Choosing a phacoemulsification technique with low ultrasound energy and short surgery time can help reduce endothelial cell damage. The surgical technique must be carefully applied, maintaining a stable anterior chamber, and using an appropriate endothelial-protective OVD. For patients with high IOP or long-term glaucoma medication, IOP must be controlled before phacoemulsification stabilization, to reduce the risk of endothelial cell damage.

ABBREVIATIONS

CCT: Central corneal thickness
CDE: Cumulative dissipated energy
ECD: Endothelial cell density
FLACS: Femtosecond laser-assisted cataract surgery
HEX: Percentage of hexagonal cells
IOP: Intraocular pressure
IOL: Intraocular lens
PXS: Pseudoexfoliation syndrome

COMPETING INTERESTS

The author declares no competing interests.

AUTHORS' CONTRIBUTIONS

Conceptualization, literature review, manuscript preparation, critical revision, and final approval.

Table 3: Comparison of corneal endothelial cells between diabetic and non-diabetic patients [24].

Characteristics	Non-diabetics	Diabetics
ECD (cells/mm^2)	2,500–3,000	Decreased (~10%–15% depending on glycemic control)
HEX	60%–70%	Decrease (HEX < 50%), pleomorphism
CV	< 30%	Increased (> 35%), polymegethism
Regeneration and recovery	No regeneration, compensated by hypertrophy	Lesser, prone to postoperative corneal edema
Pump activity (Na^+/K^+-ATPase)	Normal	Decreased due to oxidative stress and AGEs
CCT	Normal (520–540 μm)	Slight increase (~10–20 μm) due to decreased endothelial cell function
Postoperative corneal edema	Rare, rapid recovery	More high risk, slower recovery

REFERENCES

1. Dawson D, Geros'ki D, Edelhauser H. Corneal endothelium: structure and function in health and disease in: Corneal Surgery. 2009;.
2. Chen A, Harris ZB, Virk A, Abazari A, Varadaraj K, Honkanen R. Assessing Corneal Endothelial Damage Using Terahertz Time-Domain Spectroscopy and Support Vector Machines. Sensors (Basel). 2022;22(23):9071.
3. Scarpa F, Ruggeri A. Automated morphometric description of human corneal endothelium from in-vivo specular and confocal microscopy. Annu Int Conf IEEE Eng Med Biol Soc. 2016;2016:1296–1299.
4. Jirsova K. The Cornea, Anatomy and Function. Light and Specular Microscopy of the Cornea. Springer; 2017.
5. Schultz RO, Glasser DB, Matsuda M, Yee RW, Edelhauser HF. Response of the corneal endothelium to cataract surgery. Arch Ophthalmol. 1986;104(8):1164–1169.
6. Werblin TP. Long-term endothelial cell loss following phacoemulsification: model for evaluating endothelial damage after intraocular surgery. Refract Corneal Surg. 1993;9(1):29–35.
7. Rao GN, Stevens RE, Harris JK, Aquavella JV. Long-term changes in corneal endothelium following intraocular lens implantation. Ophthalmology. 1981;88(5):386–397.
8. Galin MA, Lin LL, Fetherolf E, Obstbaum SA, Sugar A. Time analysis of corneal endothelial cell density after cataract extraction. Am J Ophthalmol. 1979;88(1):93–96.
9. Chen H, et al. Accelerated Corneal Endothelial Cell Loss after Phacoemulsification in Patients with Mildly Low Endothelial Cell Density. J Clin Med. 2021;10(11):2270.
10. Lundberg B. Corneal endothelial changes seven years after phacoemulsification cataract surgery. Int Ophthalmol. 2024;44(1):169.
11. Upasani D, Daigavane S. Phacoemulsification Techniques and Their Effects on Corneal Endothelial Cells and Visual Acuity: A Review of "Direct-Chop" and "Stop-and-Chop" Approaches Under Topical Anesthesia. Cureus. 2024;16(8).
12. Shen J. Impact of preoperative intraocular pressure on corneal endothelial cell loss after phacoemulsification in acute primary angle-closure glaucoma with cataracts. Sec Ophthalmology; 2025.
13. Soro-Martínez MI, de Imperial-Ollero JAM, Pastor-Montoro M, Arcos-Villegas G, Sobrado-Calvo P, Ruiz-Gómez JM. Corneal endothelial cell loss after trabeculectomy and phacoemulsification in one or two steps: a prospective study. Eye (Lond). 2021;35(11):2999–3006.
14. Obuchowska I, Konopińska J. Corneal Endothelial Cell Loss in Patients After Minimally Invasive Glaucoma Surgery: current Perspectives. Clin Ophthalmol. 2022;16:1589–1600.
15. Wirbelauer C, Anders N, Pham DT, Wollensak J. Corneal endothelial cell changes in pseudoexfoliation syndrome after cataract surgery. Arch Ophthalmol. 1998;116(2):145–149.
16. Ramage FA. Corneal endothelial evaluation after phacoemulsification in eyes with pseudoexfoliation syndrome. Delta Journal of Ophthalmology. 2022;23(4):241–247.
17. Khanday SB, et al. Corneal endothelial cell density after phacoemulsification in patients with and without pseudoexfoliation syndrome. J Popul Ther Clin Pharmacol. 2024;31(9):2149–2154.
18. Hasegawa Y, Nejima R, Mori Y, Sakisaka T, Minami K, Miyata K, et al. Risk factors for corneal endothelial cell loss by cataract surgery in eyes with pseudoexfoliation syndrome. Clin Ophthalmol. 2016;10:1685–1689.
19. Hayashi K, Manabe S, Yoshimura K, Kondo H. Corneal endothelial damage after cataract surgery in eyes with pseudoexfoliation syndrome. J Cataract Refract Surg. 2013;39(6):881–887.
20. Đoàn Kim Thành, Tuấn LM, Anh LNV, Đặng Hoàng Long. Khảo sát sự thay đổi đặc điểm tế bào nội mô giác mạc trước và sau phẫu thuật tán nhuyễn thủy tinh thể 6 tháng ở bệnh nhân hội chứng giả tróc bao. Tạp chí Y học Việt Nam. 2024;537(1):215–219.
21. Storr-Paulsen A, Singh A, Jeppesen H, Norregaard JC, Thulesen J. Corneal endothelial morphology and central thickness in patients with type II diabetes mellitus. Acta Ophthalmol. 2014;92(2):158–160.
22. Lee JS, Lee JE, Choi HY, Oum BS, Cho BM. Corneal endothelial cell change after phacoemulsification relative to the severity of diabetic retinopathy. J Cataract Refract Surg. 2005;31(4):742–749.
23. Yang Y, Chai H, Ding Z, Tang C, Liang Y, Li Y. Meta-analysis of corneal endothelial changes after phacoemulsification in diabetic and non-diabetic patients. BMC Ophthalmol. 2023;23(1):174.
24. Upadhyaya P, et al. Comparison of endothelial cell loss using specular microscopy in diabetic versus non-diabetic patients undergoing phacoemulsification surgery at an eye hospital in central India. Indian Journal of Clinical and Experimental Ophthalmology. 2024;10(4):746–753.