

Corneal Endothelial Changes Following Anterior Chamber Intraocular Lens Implantation in Extracapsular Cataract Surgery

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Abstract

Original Research Article

Aim: To determine & quantify corneal endothelial changes after primary or secondary implantation of anterior chamber intraocular lenses (AC IOLs) in extracapsular cataract surgery complicated by loss of capsule support. **Setting:** Ibn Al-Heythem teaching eye hospital, Baghdad. **Patients & Methods:** This prospective study comprised 125 patients underwent extracapsular cataract surgery complicated by loss of capsular support. Of them 76 patients had primary implantation of open-loop AC IOLs. The other 49 patients were kept aphakic after similar complicated cataract surgery & had secondary AC IOL implantation (open-loop), this was performed 10 weeks to 2 years from initial cataract surgery. At least 2 weeks post AC IOL implantation, non-contact specular microscopy was performed at the center of the cornea for eyes with primary & secondary AC IOLs implantation, their fellow eyes (phakic or pseudophakic with posterior chamber PC IOL) taken as control. **Results:** The mean endothelial cell density (ECD) was significantly lower in eyes with primary or secondary AC IOL implantation than in unoperated phakic eyes. Patients with primary AC IOL implantation in 1 eye & PC IOL implantation in fellow eye had a difference in ECD that was not statistically significant. The ECD difference was significantly greater in eyes with secondary AC IOL implantation compared to fellow pseudophakic eyes (PC IOL). The coefficient of variation (CV) in cell size & percentages of hexagonality showed no significant changes in the various groups. **Conclusion:** AC IOL implantation didn't appear to alter corneal endothelial function. Results indicate that endothelial cell loss was related to surgical trauma rather than the presence of AC IOL.

Keywords: Extracapsular cataract surgery, endothelial cell density (ECD), coefficient of variation (CV).

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INTRODUCTION

The corneal endothelium is essential for maintenance of normal corneal hydration, thickness, and transparency [1]. This cellular monolayer is highly vulnerable and has only limited regenerative capacity.

The corneal endothelium can be damaged by many factors during cataract surgery, and its status is an important parameter in evaluating the quality of anterior segment surgery [2]. One of the main causes of cataract is diabetes mellitus [3, 4].

Specular microscopy has become a standard technique to determine endothelial cell density and morphology in vivo.

Trauma to the endothelium reduces cell density, increases the mean cell size, and disrupts the normal morphological pattern. Analysis of cell shape and pattern is a more sensitive indicator of endothelial damage than cell density alone [5].

This study used specular microscopy to clarify the extent of corneal endothelial injury in the early period after primary or secondary implantation of anterior chamber intraocular lenses (AC IOLs) after extracapsular cataract surgery complicated by loss of capsule support.

The corneal endothelial cell layer cannot regenerate after injury. Repair processes involve enlargement of residual cells, amitotic nucleus division, migration, and the rosette phenomenon, which leads to a reduction in cell density, a proportional increase in mean cell size, and disruption of the normal hexagonal cell pattern.

The normal corneal endothelial cell density is approximately 2500 cells per mm², with corneal edema and decompensation occurring when cell density falls to 500 cells per mm² or below [6]. The corneal endothelial cell count diminishes after cataract surgery.

The amount of loss depends on the procedure, the type of intraocular lens (IOL) implanted, and intraoperative and postoperative complications [7, 8].

Cheng *et al.*, calculated a mean endothelial cell loss of 2% per annum for cataract operated (ECCE) patients [2], measurements were made 2 years apart), whereas a loss of between 0.9% and 1% was found in the unoperated eye of the same group of patients [9].

PATIENTS & METHODS

This prospective study comprised 125 patients were chosen randomly, 76 of them underwent extracapsular cataract extraction complicated by loss of capsular support, anterior vitrectomy with automated director was performed, anterior chamber open loop PMMA IOL was implanted (primary implantation)

The other 49 patients had cataract surgery complicated by loss of capsular support & they were kept aphakic until later on they had secondary AC IOL implanted of the same type as in the primary procedure, where corneal incision was performed with anterior vitrectomy using automated vitrector if necessary. Secondary implantation was performed from 10 weeks to 2 years after the initial cataract surgery.

These surgeries were performed by different seniors & residents at Ibn Al-Heythem teaching eye hospital, Baghdad.

Non-contact specular microscopy with TOPCON SP_3000P shown in figure (7) was performed at the center of the cornea for the eyes underwent primary or secondary AC IOL implantation, & their contralateral eye phakic (49 patients) or pseudophakic with PC IOL implanted (76 patients), taken as control, this examination was performed at least 2 weeks after AC IOL implantation.

Endothelial structure was evaluated by densitometric & morphometric analysis. Mean Endothelial cell density (mean ECD), mean ECD difference was calculated as percentage of the mean ECD of the eyes with AC IOL versus that of the fellow control eye. Coefficient of variation (CV) of cell size with the standard deviation SD, Percentage of hexagonality. These parameters were evaluated in both eyes of each patient at time of examination.

Criteria including: corneal pathology, uveitis, glaucoma, inflammatory eye disease, diabetic retinopathy, previous ocular surgery, intraoperative or postoperative complications (except capsular support loss) were not taken in consideration in this study.

The use of air or viscoelastic substance during surgery was also not taken in consideration.

Data referring to right & left eyes were considered together. The student t-test was used to compare between the eyes with AC IOL (primary or secondary) data & that of their fellow control eyes (phakic or pseudophakic with PC IOL). A p value less than 0.05% was considered significant.

Table 1: Patients demographic parameters parameter Primary AC IOL implantation

parameter	Primary AC IOL implantation	Secondary AC IOL implantation
Number of patients	76	49
Mean age	62±10	58±13
Sex (M/F)	36/40	24/25
Right / Left eye	30/46	21/28
Control (Phakic)	23	26
Control (Pseudophakic PC IOL)	53	23

RESULTS

For eyes with primary AC IOL implantation, mean endothelial cell density (mean ECD) was 1840 cells/mm², SD was 343. Their control phakic eyes had mean ECD 2123 cells/mm², SD was 345. Mean ECD difference (%) was 13.3 ± 6.7. P value less than 0.01 which is statistically significant, indicating significant reduction in the mean ECD after such surgery.

Eyes with secondary AC IOL implantation had mean ECD 1669 cells/mm², SD was 379. Their control phakic eyes had mean ECD 2187, SD was 363. Mean ECD difference (%) was 23.7 ± 8.3. P value less than 0.01 which is statistically significant, also indicating reduction in the mean ECD after similar surgery. As

shown in table [2].

For eyes with primary AC IOL implantation their mean ECD was 1763 with SD of 501. Control fellow eyes (pseudophakic with PC IOL) had mean ECD 1873, SD 516. Mean ECD difference (%) 5.9 ± 2.8. P value was statistically not significant.

Regarding eyes with secondary AC IOL implantation their mean ECD was 1679, SD was 463. Control pseudophakic eyes (PC IOL) had mean ECD 1965, SD was 412. Mean ECD difference (%) 14.6 ± 6.2. P value less than 0.05 is statistically significant, so the endothelial cell density significantly reduced following secondary AC IOL implantation. This is shown in table

[3].

Regarding the Coefficient of variation (CV) in cell size in eyes with primary AC IOL was 0.29, SD 0.03. Control phakic eyes had CV in cell size 0.30, SD 0.02. P value was not significant.

Eyes with secondary AC IOL had CV in cell size 0.30, SD 0.03. Control phakic eyes their CV in cell size was 0.30, SD 0.03. P value was also not significant. As shown in table [4].

For eyes with primary AC IOL their CV in cell size was 0.29, SD 0.02. Control pseudophakic eyes (PC

IOL) had CV in cell size 0.29, SD 0.03. P value showed to be not significant.

Eyes with secondary AC IOL had CV in cell size 0.30, SD 0.02. Fellow control pseudophakic eyes (PC IOL) their CV in cell size was 0.30, SD 0.03. Also P value was not significant. This is shown in table [5].

No significant difference in the percentage of hexagonality was found in eyes with primary or secondary AC IOL implantation compared to their control fellow eyes whether phakic or pseudophakic (PC IOL).

Table 2: Mean corneal ECD in eyes with primary or secondary AC IOL implantation & in fellow phakic eyes with the difference in ECD

Group	Number of patients	Mean ECD AC IOL	Mean ECD Control (Phakic)	Mean ECD difference AC IOLs VS. Control (%)	P value
Primary AC IOL	23	1840± 343	2123± 345	13.3± 6.7	<0.01
Secondary AC IOL	26	1669± 379	2187± 363	23.7± 8.3	< 0.01

Mean ECD (cells/mm²)
Means ± SD
ECD = endothelial cell density

Table 3: Mean corneal ECD in eyes with primary or secondary AC IOL implantation & in fellow pseudophakic eyes (PC IOL) and the difference in ECD Group Number Of patients

Group	Number Of patients	Mean ECD AC IOL	Mean ECD Control (PC IOL)	Mean ECD Difference AC IOLs Vs Control (%)	P value
Primary AC IOL	53	1763± 501	1873± 516	5.9 ± 2.8	NS
Secondary AC IOL	23	1679± 463	1965± 412	14.6 ± 6.2	< 0.05

Mean ECD (cells /mm²)
Means ± SD
ECD = endothelial cell density
NS = not significant

Table 4: Coefficient of variation in cell size in eyes with primary or secondary AC IOL implantation & in fellow Phakic eyes

Group	Number of patients	AC IOL	Control (Phakic)	P value
Primary AC IOL	23	0.29± 0.03	0.30± 0.02	NS
Secondary AC IOL	26	0.30± 0.03	0.30± 0.03	NS

Mean CV in cell size (SD/μm²)
Means ± SD
CV = Coefficient of variation in cell size
NS = not significant

Table 5: Coefficient of variation in cell size in eyes with primary or secondary AC IOL implantation & in fellow pseudophakic eyes (PC IOL)

Group	Number of patients	AC IOL	Control (PC IOL)	P value
Primary AC IOL	53	0.29± 0.02	0.29± 0.03	NS
Secondary AC IOL	23	0.30± 0.02	0.30± 0.03	NS

Mean CV in cell size (SD/μm²)
Means ± SD
CV = coefficient of variation
NS = not significant

DISCUSSION

The normal thickness & transparency of the cornea are maintained by the barrier function & active

fluid pump of the corneal endothelium [1]. It is a fragile cell layer whose integrity must be guarded to ensure the success of any intraocular procedure.

Corneal endothelial cells can be damaged by many factors during & after cataract surgery [10, 11]. Intraoperative factors associated with corneal endothelial injury include turbulence of the irrigation solution [11, 12], mechanical trauma by instruments [13], anemia [14, 15], infection and the presence of lens fragments and IOLs [16].

In contrast, hyaluronic acid has binding sites on the endothelium and may provide mechanical and chemical protection during surgery [17].

Endothelial alterations are considered important parameters of surgical trauma and are essential in estimating the safety of surgical techniques [18]. Mishima showed that endothelial damage diminishes cell density, increases corneal thickness, and alters the normal morphometric endothelial pattern [19].

Morphological changes are more sensitive indicators of endothelial damage & function than change in cell density [20]. The results in many studies suggest that endothelial cell morphometry (pleomorphism & polymegathism) is the most sensible index of corneal endothelial functional reserve [5-21]. A high CV of cell area may be an early sign of continuing endothelial cell loss [20]. After cataract surgery corneal ECD decreases [22, 23]. Many studies document increased complications associated with AC IOL implantation, especially endothelial corneal damage [24-26]. Alterations in the corneal endothelium depend on surgical technique & style. Kaufman & Katz [27, 28], showed that contact between the IOL surface & the endothelium can result in adhesion & stripping of endothelial cells from Descemet's membrane.

Intermittent touch of a malpositioned or inappropriately vaulted IOL is more frequent with AC IOLs. Drews [29], describes intermittent touch syndrome including ciliary flush, corneal changes & cystoid macular edema. Persons in occupations involving rapid head jerking or a head-down positions are at risk for intermittent contact between the lens & cornea.

Intrinsic factors contribute to endothelial touch are poor IOL position, IOL dislocation, incorrect IOL size, excessive anterior vaulting of the IOL & vitreous adhesion to the wound. Duffin & Olson [30], compared vaulting characteristics of various AC IOLs & found that lenses with closed loops vaulted more per unit of loop compression than open loop lenses.

Late corneal decompensation often occurs in the absence of direct intraoperative endothelial trauma. This can be explained by assuming inflammation with toxic effect of inflammatory mediators on corneal endothelium [31, 32], or exposure to components of vitreous humor [33]. Ravalico *et al.*, reported 63 patients following primary & secondary AC IOL implantation at first examination mean ECD 1749 ± 341 (cells/mm²), mean CV in cell size 0.31 ± 0.03 (SD/ μ m²). Second examination 2 years later revealed mean ECD 1738 ± 333 (cells/mm²), mean CV in cell size 0.31 ± 0.02 (SD/ μ m²), with the absence of statistically significant differences in the ECD & CV of cell area in both examinations.

In our study, eyes with primary AC IOL implantation during complicated cataract surgery had reduced ECD, with statistically significant difference in the mean ECD from their fellow phakic control eyes.

The mean ECD difference was even greater in eyes with secondary AC IOL implantation, indicating the role of double surgical trauma in reducing cell density. The CV of cell size was less than 0.30 in both groups of eyes with AC IOLs with non-significant difference compared to their control groups.

Our data appear more or less similar to those reported by other authors regarding primary & secondary AC IOL implantation in complicated cataract surgery. Long-term follow up is required to confirm these results.

Selection of well-designed AC IOLs of the correct size with proper IOL positioning have markedly decreased the incidence of intraoperative or postoperative endothelial damage, confirming the safety of a well-positioned AC IOL & pointing that surgical trauma as the main cause of endothelial cell loss.

Table 6: Mean ECD & CV in cell size in eyes with primary or secondary AC IOL implantation, first examination versus examination 2 years later.

Group	Mean ECD (cells/mm ²)	Mean CV In cell size (SD/ μ m ²)	P value
Primary & secondary AC IOL implantation First examination	1749 \pm 341	0.31 \pm 0.03	NS
Primary & secondary AC IOL implantation Second examination*	1738 \pm 333	0.31 \pm 0.02	NS

CONCLUSION

Corneal endothelial cell density decreased significantly in eyes with primary & secondary AC IOL implantation compared to fellow phakic eyes, ECD

decreased with greater significance in eyes with secondary AC IOL implantation compared to their fellow phakic or pseudophakic eyes (PC IOL) indicating the role of double surgical trauma in reducing cell density.

Non-significant differences in CV of cell size of eyes with primary & secondary AC IOLs compared to fellow control eyes whether phakic or pseudophakic (PC IOL). Thus AC IOL implantation didn't appear to alter corneal endothelial function, suggesting the safety of a well-positioned AC IOL & pointing to surgical trauma as the main cause of corneal endothelial cell loss.

The use of AC IOLs in complicated cataract extraction is justified if capsular support is lost. It can be safely used as a primary or secondary procedure.

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