



Long-term Effects of Uncomplicated Traumatic Hyphema on Corneal and Lenticular Clarity

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Purpose: To evaluate the long-term effects of uncomplicated traumatic hyphema on endothelial morphology, anterior segment structure, and corneal and lenticular densitometry

Methods: In this retrospective comparative study, eyes with a history of uncomplicated traumatic hyphema were compared with the healthy contralateral unaffected eyes. The corneal endothelial cell properties were captured using specular microscopy. Anterior segment analysis, corneal densitometry (12-mm corneal diameter), and lens densitometry measurements were performed using the Pentacam imaging system.

Results: Measurements were obtained at a mean follow-up of 49.5 ± 15.8 months after injury. The average endothelial cell density was significantly lower in the study group than in the control group ($2,506.6 \pm 294.0$ cells/mm² vs. $2,665.7 \pm 195.0$ cells/mm², $p = 0.020$). There was no difference between the groups in respect of polymegathism and pleomorphism ($p = 0.061$ and $p = 0.558$, respectively). All the investigated corneal tomographic and angle parameters were similar in both groups (all $p > 0.05$). The corneal densitometry values in all concentric zones and layers showed no statistically significant difference between the groups ($p > 0.05$ for all). The lens zone 1 densitometry value was significantly higher in the study group than in the control group ($9.6\% \pm 1.1\%$ vs. $8.9\% \pm 1.2\%$, $p = 0.031$). No difference was observed in zone 2 and 3 ($p = 0.170$ and $p = 0.322$, respectively). The degree of hyphema was not correlated with endothelial cell and lenticular clarity loss ($p = 0.087$ and $p = 0.294$, respectively).

Conclusions: Even if traumatic hyphema is not complicated, long-term outcomes indicate endothelial cell loss and increased lenticular density.

Key Words: Corneal diseases, Corneal endothelium, Densitometry, Hyphema, Lens diseases

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Traumatic hyphema is one of the most common complications after blunt trauma to the eye. As a result of blunt trauma, an equatorial globe extension occurs simultaneously with exposure of the globe to increased anteroposterior pressure. This equatorial expansion causes stretching on anterior chamber angle structures, and consequently, rupture develops in the stromal vascular structures of the iris or ciliary body [1,2]. Complications of hyphema include increased intraocular pressure (IOP), peripheral anterior synechia, corneal blood staining, optic atrophy, and secondary hemorrhage [2]. Apart from the complications caused by hyphema, other simultaneous findings resulting from blunt trauma are also important in terms of prognosis. Conditions such as sphincter rupture, iridodialysis, angle recession, trabecular meshwork tear, zonular rupture, cataract, vitreous hemorrhage, retinal dialysis, and choroid rupture may develop following blunt trauma [3,4]. However, there is controversy in the literature between publications indicating a decrease in corneal endothelial cell density (ECD) related to traumatic hyphema [5,6] and publications reporting no significant change [7,8]. In one study, high corneal densitometry values were reported in the posterior corneal layer of eyes with hyphema in the early period [9], whereas previous studies did not distinguish whether eyes with hyphema were complicated or not [5-9].

The aim of this study was to investigate the long-term effects of uncomplicated traumatic hyphema on endothelial cell morphology, anterior chamber parameters, corneal clarity, and lens transparency by comparing these results to those of the fellow unaffected eyes of the same patients, and to determine whether there is a clinical correlation between the amount of change and hyphema grade.

Materials and Methods

Ethics statement

The study adhered to the tenets of Declaration of Helsinki and was approved by the Institutional Review Board/Ethics Committee of Numune Training and Research Hospital, Ankara, Turkey (No. E-18-1787). Written informed consent was obtained from all patients prior to enrollment. All participants were of Caucasian origin.

Study population

The study included a total of 30 consecutive patients with a history of nonpenetrating traumatic hyphema who presented at the emergency eye clinic and were followed up for approximately 4 years. The patients were followed up until hyphema resolved and during this time, a record was kept of medical treatment, increased IOP control, and the need for surgical treatment. The diagnosis of traumatic hyphema was made on the basis of the typical clinical visualization of layered or dispersed blood in the anterior chamber following blunt trauma. The hyphema was graded by measuring the vertical height of the layered blood as previously described [10]: grade 1, hyphema filling <one-third of the anterior chamber; grade 2, hyphema filling one-third to one-half of the anterior chamber; grade 3, hyphema filling more than one-half but less than the total volume of the anterior chamber; and grade 4, total hyphema with either red or black blood clots. No patient with microhyphema was included. Eyes with corneal scarring/diseases, contact lens wear, long-term topical eye drop usage, active intraocular infection, history of ocular surgery, glaucoma, uveitis, secondary hemorrhage, and trauma history other than nonpenetrating traumatic hyphema were excluded from the study. Uncomplicated hyphema was defined as a case without complications such as corneal blood staining, glaucoma, iridodialysis, synechia, and cataract and optic atrophy in the traumatic eye after anterior chamber hemorrhage due to blunt trauma. The control eyes were the fellow healthy eyes of the same patients, without any ocular disorder or trauma.

The data of traumatic hyphema patients were reviewed according to the inclusion and exclusion criteria and patients who met the criteria were called for a final examination, at which age, sex, cause of injury, and eye laterality were recorded in all cases. None of the patients had any ocular symptoms, visual impairment, or medical treatment at the time of measurements. All cases underwent a comprehensive ophthalmological examination, including best-corrected visual acuity tests using the Snellen chart, IOP measurement with Goldmann applanation tonometry, slit-lamp biomicroscopy, and dilated fundus examination. After the ophthalmological examination, specular microscopy and Scheimpflug corneal tomography measurements were performed.

Endothelial cell properties

ECD, coefficient of variation (CV; polymegathism indicator), and the ratio of hexagonal cells to other different geometric shaped cells (HEX; pleomorphism indicator) were evaluated as morphometric analysis of the corneal endothelium by the same blinded experienced clinician once with specular microscopy (Tomey EM-4000; Tomey Corp., Nagoya, Japan). Patients were asked to look at the central fixation target and the automatic alignment function which provides reproducible and reliable measurements was performed. All corneal endothelial cells clearly visible on the picture were marked manually. The center method, a common technique incorporated into specular microscopy, was used. At least 110 cells/measurement were included in each analysis [11]. Endothelial cell loss (ECL) was assessed as follows: $ECL = [(cell\ count\ of\ fellow\ eye - cell\ count\ of\ traumatic\ eye) / cell\ count\ of\ fellow\ eye] \times 100$, stated as a percentage.

Anterior segment analysis

Two consecutive measurements were obtained from all participants with a rotating Scheimpflug camera system (Pentacam HR; Oculus Optikgeräte GmbH, Wetzlar, Germany) by a single specialist. The first was performed without pupil dilation to evaluate the tomographic and corneal densitometric indices, and the second was performed after pupil dilation to assess the lens densitometry values. The following tomographic parameters were recorded: the power of the corneal astigmatism, flat keratometry and steep keratometry for the central 3.0 mm of the cornea, maximum keratometry, thinnest corneal thickness, corneal volume, anterior chamber volume, anterior chamber angle, and anterior chamber depth.

Corneal and lenticular densitometry analysis

Corneal and lens densitometry parameters were obtained using the densitometry software of the Pentacam HR. For corneal densitometry, the whole 12-mm corneal area was further divided into four concentric zones. The first zone comprises a circular area 2 mm in diameter at the center of the cornea. The second zone consists of a 2-mm to 6-mm annular area around the first zone. The third zone is the 6-mm to 10-mm annular area around the second zone, and the fourth zone is the 10-mm to 12-mm annular area around the third zone. This analysis also yields densitometric values of the cornea at three different depths. The anterior layer contains the superficial 120 μ m, and the posterior layer consists of 60 μ m of the innermost cornea. The central corneal layer is located between these two layers. The corneal densitometry values are expressed as the pixel luminance per unit volume in the Scheimpflug image and are given in grayscale units, ranging from a minimum light scatter of 0 (maximum transparency) to a maximum light scatter of 100 (completely opaque cornea) [12]. The lens densitometry was measured in predefined three-dimensional zones (zone 1 at 2.0 mm, zone 2 at 4.0 mm, and zone 3 at 6.0 mm), which were located around the center of the pupil. The change in lens zone 1 densitometry was calculated as follows: $change\ in\ lens\ zone\ 1\ densitometry = [(lens\ zone\ 1\ densitometry\ value\ of\ traumatic\ eye - lens\ zone\ 1\ densitometry\ value\ of\ fellow\ eye) / lens\ zone\ 1\ densitometry\ value\ of\ fellow\ eye] \times 100$, stated as a percentage.

Statistical analysis

Data obtained in the study were analyzed statistically using IBM SPSS ver. 22.0 (IBM Corp., Armonk, NY, USA). Descriptive data were presented as mean \pm standard deviation values, frequency, and percentage. Visual (histogram and probability graphs) and analytical methods

Table 1. Corneal endothelial cell properties of the traumatic versus fellow healthy eyes

Variable	Traumatic eye (n = 30)	Control eye (n = 30)	p-value
Endothelial cell density (cells/mm ²)	2,506.6 \pm 294.0	2,665.7 \pm 195.0	0.020 ^{*†}
Coefficient of variation (%)	42.3 \pm 5.6	39.9 \pm 3.9	0.061 [‡]
Hexagonal cell percentage (%)	43.8 \pm 4.9	44.5 \pm 4.1	0.558 [‡]

Values are presented as mean \pm standard deviation.

^{*}Mann-Whitney *U*-test; [†]Statistically significant result; [‡]Independent samples *t*-test.

Table 2. Anatomic characteristics of anterior segment parameters in traumatic versus fellow healthy eyes

Variable	Traumatic eye (n = 30)	Control eye (n = 30)	p-value
Astigmatism (D)	0.8 ± 0.6	0.7 ± 0.5	0.827*
Keratometry (D)			
K ₁	42.5 ± 1.8	42.5 ± 1.8	0.965†
K ₂	43.2 ± 1.8	43.2 ± 1.8	0.937†
K _{max}	44.2 ± 2.0	43.9 ± 1.9	0.619†
Thinnest corneal thickness (μm)	537.2 ± 23.8	538.8 ± 18.6	0.788†
Cornea volume (mm ³)	58.9 ± 2.9	59.3 ± 2.1	0.538†
Anterior chamber volume (mm ³)	172.0 ± 34.4	167.4 ± 33.9	0.610†
Anterior chamber angle (°)	33.7 ± 5.1	34.1 ± 5.0	0.763†
Anterior chamber depth (mm)	2.8 ± 0.4	2.7 ± 0.3	0.503*

Values are presented as mean ± standard deviation.

D = diopters; K₁ = flat keratometry; K₂ = steep keratometry; K_{max} = maximum keratometry.

*Mann-Whitney U-test; †Independent samples t-test.

Table 3. Corneal densitometry measurements (grayscale units) in traumatic versus fellow healthy eyes

Variable	Traumatic eye (n = 30)	Control eye (n = 30)	p-value
Anterior 120 μm (mm)			
0–2	21.1 ± 1.1	21.1 ± 1.6	0.859*
2–6	18.4 ± 1.3	18.4 ± 1.7	0.946*
6–10	21.6 ± 3.4	22.0 ± 3.7	0.719*
10–12	33.4 ± 7.2	34.6 ± 6.9	0.690†
Total diameter	21.2 ± 2.4	21.6 ± 2.9	0.525*
Center (mm)			
0–2	14.4 ± 2.0	14.3 ± 2.0	0.734†
2–6	13.6 ± 2.1	13.6 ± 2.1	0.918†
6–10	16.0 ± 2.9	16.2 ± 3.0	0.706†
10–12	19.6 ± 3.9	20.0 ± 3.6	0.667*
Total diameter	15.6 ± 2.3	15.7 ± 2.2	0.892*
Posterior 60 μm (mm)			
0–2	9.7 ± 1.5	9.7 ± 1.5	0.813†
2–6	9.4 ± 1.4	9.5 ± 1.3	0.818*
6–10	11.5 ± 2.3	11.7 ± 2.3	0.680*
10–12	15.5 ± 3.2	15.9 ± 3.2	0.645*
Total diameter	10.6 ± 2.0	10.8 ± 2.1	0.774*
Total thickness (mm)			
0–2	15.2 ± 2.1	15.2 ± 2.3	0.836†
2–6	12.7 ± 1.3	12.8 ± 1.4	0.916*
6–10	16.8 ± 2.9	17.1 ± 3.0	0.657†
10–12	22.2 ± 3.9	23.0 ± 3.9	0.451*
Total diameter	16.3 ± 2.0	16.5 ± 2.0	0.742*

Values are presented as mean ± standard deviation.

*Independent samples t-test; †Mann-Whitney U-test.

(Kolmogorov-Smirnov test and Shapiro-Wilk test) were used for all data samples to check normality of distribution. To compare quantitative variables, the independent samples *t*-test was used for data showing parametric distribution, and the Mann-Whitney *U*-test for data with non-parametric distribution. Correlations were tested with Spearman correlation analysis. A value of $p < 0.05$ was considered statistically significant.

Results

Evaluation was made of 26 male and four female patients with traumatic hyphema, with a mean age of 34.7 ± 11.0 years (range, 20–56 years). The right eye was affected in 63.3% of cases, and the left eye in 36.7%. All patients presented with unilateral hyphema. The level of hyphema was evaluated as grade 1 (63.3%), grade 2 (20%), grade 3 (13.3%), and grade 4 (3.3%). The mean visual acuity on presentation due to traumatic hyphema was 1.6 ± 1.2 logarithm of the minimum angle of resolution, and the mean IOP was 19.3 ± 8.0 mmHg. Elevated IOP (>21 mmHg) requiring topical antiglaucomatous treatment was determined in 36.7% of the eyes with hyphema. The mechanisms of the injuries were falling (3.3%), sports injury (26.7%), fighting (10.0%), occupational accident (23.3%), home accident (26.7%), and others (10.0%). No patient underwent surgery for hyphema. Measurements were taken on average 49.5 ± 15.8 months (range, 30–78 months) after the injury. No clinically significant ocular complication such as glaucoma, iridodialysis, synechia, corneal blood staining, and optic atrophy developed in any patient. At the time of measurement, the best-corrected visual acuity of

eyes with history of trauma and the fellow eyes was 20 / 20 and the IOP values of both groups were similar (13.97 ± 3.54 mmHg vs. 13.40 ± 3.44 mmHg, $p = 0.532$, respectively).

The endothelial cell properties of eyes captured via specular microscopy are illustrated in Table 1. The average ECD was significantly lower in eyes with a history of traumatic hyphema than in the control eyes ($2,506.6 \pm 294.0$ cells/mm² vs. $2,665.7 \pm 195.0$ cells/mm², $p = 0.020$). The endothelial cell count of the traumatic eyes showed a drop of 5.8% compared to the control eyes. Polymegathism and pleomorphism were more impaired in the study group, but this difference was not statistically significant. ($p = 0.061$ and $p = 0.558$, respectively).

The comparisons of the anatomic characteristics of the anterior chamber between the traumatic hyphema and fellow control eyes are summarized in Table 2. Evaluation of the anterior segment parameters revealed no significant difference in astigmatism, keratometry, thinnest corneal thickness, corneal volume, anterior chamber volume, anterior chamber angle, and anterior chamber depth values between the traumatic and fellow control eyes ($p > 0.05$ for all values).

The corneal densitometry values in the study and control groups are presented in Table 3. There was no statistically significant difference in any zone and layer of corneal densitometry measurements in eyes with a history of traumatic hyphema and the control eyes ($p > 0.05$ for all values). The lens densitometry values in the hyphematic and control eyes are shown in Table 4. In comparison with the control group, the study group had significantly higher lens zone 1 densitometry values ($8.9\% \pm 1.2\%$ vs. $9.6\% \pm 1.1\%$, $p = 0.031$) and higher lens average densitometry values ($9.3\% \pm 1.3\%$ vs. $10.2\% \pm 1.5\%$, $p = 0.017$). There was no

Table 4. Lens densitometry measurements in traumatic versus fellow healthy eyes

Variable	Traumatic eye (n = 30)	Control eye (n = 30)	<i>p</i> -value
Pentacam Nucleus Staging (score)	0.3 ± 0.5	0.2 ± 0.4	0.543*
Zone 1	9.6 ± 1.1	8.9 ± 1.2	0.031**
Zone 2	8.1 ± 0.3	7.9 ± 0.3	0.170†
Zone 3	7.9 ± 0.3	7.8 ± 0.2	0.322†
Average	10.2 ± 1.5	9.3 ± 1.3	0.017**
Standard deviation	3.3 ± 2.1	3.1 ± 2.3	0.798*
Maximum	53.3 ± 33.1	52.1 ± 33.3	0.826*

Values are presented as mean \pm standard deviation and percentage, unless otherwise indicated.

*Mann-Whitney *U*-test; †Independent samples *t*-test; **Statistically significant result.

significant difference between the groups in respect of nucleus staging, zone 2 densitometry value, and zone 3 densitometry value ($p > 0.05$).

No correlation was observed between the grade of hyphema and ECL ($\rho = 0.330$, $p = 0.087$). No significant correlation was determined between the grade of hyphema and the change in lens zone 1 in the eyes with hyphema ($\rho = 0.202$, $p = 0.294$).

Discussion

Erythrocytes contain hemosiderin, which may have a detrimental effect on corneal endothelial cells and keratocytes [13,14]. It has been demonstrated that there was a decrease in the number of corneal endothelial cells and keratocytes in the pathological samples of corneas undergoing penetrating keratoplasty as a result of prolonged hyphema and increased IOP [13,14]. Therefore, in the present study, an evaluation was made of the anterior segment findings including anatomic characteristics of the anterior chamber, corneal and lens densitometry values, and endothelial cell morphology in eyes with a history of hyphema compared to fellow healthy control eyes.

Slingsby and Forstot [5] reported that blunt trauma significantly reduces corneal ECD by 6.4% depending on the degree of the first contusion. However, increased IOP, the amount of hemorrhage in the anterior chamber, and intraocular inflammation are also considered to be other factors causing increased endothelial loss in some patients [5]. Lee et al. [6] observed a significant decline of 5.6% in ECD in injured eyes compared to the normal fellow eyes. This ECD reduction was not reported to be correlated with the amount of hyphema, and degree of angle recession [6]. Conversely, Brooks et al. [7] found similar corneal endothelial cell count and morphology between fellow and hyphema eyes when hyphema was resolved. Pong et al. [8] observed no statistically significant association between corneal endothelial cell count reduction and the duration/severity of trauma. In the current study, a significant decrease of 5.8% in ECD values was found in the eyes with a history of uncomplicated traumatic hyphema compared to the control eyes. In our study, high CV and low HEX values, which are indicators of unhealthy corneal endothelium, were observed in traumatic eyes, but these values were not significant. This may be related to the small num-

ber of patients and/or the variability of specular microscopy measurements [15].

There was no significant change in the anatomic and tomographic characteristics of the anterior chamber between hyphematic and fellow healthy eyes in the long-term follow-up. This absence of difference in anterior segment parameters, such as anterior chamber volume, angle, and depth, could be attributed to the fact that complicated cases such as those with iridodialysis or phacodonesis were not included in this study.

Densitometry provides an estimation of corneal transparency with quantitative data at different depths and in different regions [16]. Collagen fibrils should be arranged in a narrow, uniform diameter and exactly organized, and the keratocyte distribution and extracellular matrix organization should be in a regular structure for corneal transparency [17-19]. Various disorders, such as corneal scars, corneal infiltrates, corneal deposits, and corneal ectatic diseases can cause increased light backscatter. These opacity increments are reflected in the form of increased corneal densitometry values [11,16,20]. In addition, the endothelium plays a substantial role in maintaining corneal transparency by pumping water from the stroma into the aqueous. Disruption of the endothelial function leads to stromal swelling and corneal edema [21,22]. Thus, increased corneal densitometry can be seen in endothelial alterations due to decreased transparency. Kiziltoprak et al. [9] performed corneal densitometry analysis in the 1st week and 1st month after complete resolution of the anterior chamber blood and drug discontinuation in 28 patients with traumatic hyphema. Higher corneal densitometry values were determined in all zones of the posterior layer in the 1st week compared to the control group. However, the posterior layer corneal densitometry values decreased by the end of the 1st month, and the values at 10-mm to 12-mm, and in the total zones were not significantly different from those of the control group. They proposed that corneal densitometry analysis may be used in clinically normal cases to detect possible early corneal blood staining [9]. Patients with traumatic hyphema are prone to complications such as corneal blood staining and ECL because of the concussion effect of trauma and the toxicity of erythrocytes to keratocytes and endothelium. However, it is not clear whether these densitometric changes persist or return to normal values in the long term. Although densitometry was not measured throughout follow-up in the current

study, according to the densitometry measurement taken at 49.5 months after hyphema, there was no significant difference in terms of corneal densitometry values despite the decrease in ECD. Thus, it can be thought that the ECD change may not have been sufficient to cause a permanent change in corneal densitometry values. Traumatic hyphema may cause clinically significant changes in a case with previously impaired endothelium or insufficient ECD by increasing endothelial loss.

The lens densitometry values only in zone 1 were significantly higher in eyes with a history of traumatic hyphema compared to the control eyes. The role of trauma in increasing the risk of cataractogenesis has been highlighted in many studies [23-26]. The increase in the lens densitometry can be considered as early-stage cataract without clinical manifestation. Concussion of the lens without tearing the capsule can cause a cataract that is initially subcapsular [26]. To the best of our knowledge, there is no pathological sample study showing that toxicity of erythrocytes in hyphema affects the anterior lens capsule. In the slit-lamp examination of the current study, no blood staining was observed on the lens. The increase in densitometry values in lens zone 1 and not in other lens layers can be considered to be possibly due to the uncomplicated nature of the cases and the direct mechanical effect of trauma initially in zone 1.

The development of corneal staining as a result of hyphema is a finding related to the amount of blood in the anterior chamber and is a precursor for endothelial cell decompensation. However, no correlation was found between both ECL and change in lens zone 1 densitometry and the degree of hyphema in this study. Although factors such as the amount of hemorrhage in anterior chamber, increased IOP, and intraocular inflammation are accepted in ECL, the nature of initial trauma may be dominant.

Former studies have reported an association between high IOP and ECL, indicating the mechanism of direct compression damage and glaucoma medication toxicity [27,28]. Due to the absence of cases with a permanent IOP increase, it was not possible in this study to reveal a potential association between high IOP values, antiglaucomatous medications, and ECL. Addressing this issue in future studies with large sample sizes may clarify the situation.

The primary limitation of this study was the relatively small sample size due to the low number of patients with both uncomplicated hyphema and long-term regular follow-up. That complicated traumatic hyphema patients

were not included as a separate group could also be seen as a limitation. However, this is one of the few studies in the literature with long-term follow-up of endothelial cell morphology and corneal/lenticular clarity after uncomplicated traumatic hyphema.

In conclusion, a significant decrease was detected in the endothelial cell count of the eyes with uncomplicated traumatic hyphema. In preoperative cataract surgery evaluation, endothelial cell count and health should be considered in patients with a history of trauma even if corneal clarity remains. A significant increase in zone 1 lens densitometry value was determined in eyes with a history of uncomplicated traumatic hyphema, and this increase suggests that trauma affects zone 1 even if no clinically significant cataract has developed. However, further prospective studies with larger sample sizes are needed to elucidate the association between hyphema and optical clarity by evaluating the effects of diverse grades of hyphema in different age groups.

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References

1. Wilson FM. Traumatic hyphema: pathogenesis and management. *Ophthalmology* 1980;87:910-9.
2. Walton W, Von Hagen S, Grigorian R, Zarbin M. Management of traumatic hyphema. *Surv Ophthalmol* 2002;47:297-334.
3. Pass AF. Traumatic hyphema. In: Onofery BE, Skorin L Jr, Holdeman NR, editors. *Ocular therapeutics handbook: a clinical manual*. 1st ed. Philadelphia: Lippincott Williams & Wilkins; 1998. p. 329-34.
4. Lenihan P, Hitchmoth D. Traumatic hyphema: a teaching case report. *Optom Educ* 2014;39:110-8.
5. Slingsby JG, Forstot SL. Effect of blunt trauma on the corneal endothelium. *Arch Ophthalmol* 1981;99:1041-3.
6. Lee HD, Kim JW, Kim BK. Corneal endothelial changes in patients with traumatic hyphema. *J Korean Ophthalmol Soc* 2002;43:1730-7.
7. Brooks AM, Grant G, Gillies WE. The identification of different vascular cells on the corneal endothelium by specular microscopy. I. Red blood cells and corneal endo-

- thelial changes in hyphaema secondary to contusion injury. *Aust N Z J Ophthalmol* 1988;16:3-6.
8. Pong J, Lai J. Effect on corneal endothelial cell count of traumatic microhyphaema and hyphaema. *Acta Ophthalmol* 2009;87:559-61.
 9. Kiziltoprak H, Atesoglu HI, Tekin K, et al. Evaluation of densitometric analysis for early detection of corneal blood staining in hyphema. *Cornea* 2021;40:467-71.
 10. Edwards WC, Layden WE. Traumatic hyphema: a report of 184 consecutive cases. *Am J Ophthalmol* 1973;75:110-6.
 11. McCarey BE, Edelhauser HF, Lynn MJ. Review of corneal endothelial specular microscopy for FDA clinical trials of refractive procedures, surgical devices, and new intraocular drugs and solutions. *Cornea* 2008;27:1-16.
 12. Lopes B, Ramos I, Ambrosio R Jr. Corneal densitometry in keratoconus. *Cornea* 2014;33:1282-6.
 13. Messmer EP, Gottsch J, Font RL. Blood staining of the cornea: a histopathologic analysis of 16 cases. *Cornea* 1984-1985;3:205-12.
 14. McDonnell PJ, Green WR, Stevens RE, et al. Blood staining of the cornea: light microscopic and ultrastructural features. *Ophthalmology* 1985;92:1668-74.
 15. Chaurasia S, Vanathi M. Specular microscopy in clinical practice. *Indian J Ophthalmol* 2021;69:517-24.
 16. Otri AM, Fares U, Al-Aqaba MA, Dua HS. Corneal densitometry as an indicator of corneal health. *Ophthalmology* 2012;119:501-8.
 17. Jester JV, Moller-Pedersen T, Huang J, et al. The cellular basis of corneal transparency: evidence for 'corneal crystallins'. *J Cell Sci* 1999;112(Pt 5):613-22.
 18. Meek KM. Corneal collagen-its role in maintaining corneal shape and transparency. *Biophys Rev* 2009;1:83-93.
 19. Ma J, Wang Y, Wei P, Jhanji V. Biomechanics and structure of the cornea: implications and association with corneal disorders. *Surv Ophthalmol* 2018;63:851-61.
 20. Enders P, Holtick U, Schaub F, et al. Corneal densitometry for quantification of corneal deposits in monoclonal gammopathies. *Cornea* 2017;36:470-5.
 21. Edelhauser HF. The balance between corneal transparency and edema: the Proctor Lecture. *Invest Ophthalmol Vis Sci* 2006;47:1754-67.
 22. Bourne WM. Corneal endothelium: past, present, and future. *Eye Contact Lens* 2010;36:310-4.
 23. Churchill AJ, Noble BA, Etechells DE, George NJ. Factors affecting visual outcome in children following unioocular traumatic cataract. *Eye (Lond)* 1995;9(Pt 3):285-91.
 24. Khattry SK, Lewis AE, Schein OD, et al. The epidemiology of ocular trauma in rural Nepal. *Br J Ophthalmol* 2004;88:456-60.
 25. Sinha R, Kumar C, Titiyal JS. Etiopathogenesis of cataract: journal review. *Indian J Ophthalmol* 2009;57:245-9.
 26. Gupta VB, Rajagopala M, Ravishankar B. Etiopathogenesis of cataract: an appraisal. *Indian J Ophthalmol* 2014;62:103-10.
 27. Gagnon MM, Boisjoly HM, Brunette I, et al. Corneal endothelial cell density in glaucoma. *Cornea* 1997;16:314-8.
 28. Yu ZY, Wu L, Qu B. Changes in corneal endothelial cell density in patients with primary open-angle glaucoma. *World J Clin Cases* 2019;7:1978-85.