Clinical Features of Traumatic Corneal Endothelial Rings

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We report the clinical features and the course of traumatic corneal endothelial rings by trauma. Fourteen eyes (of fourteen patients) with traumatic endothelial rings (twelve cases of BB shot injury), were enrolled in this study. With median follow-up interval of 50 weeks, initial and final best corrected visual acuity, presence of combined injuries such as gross hyphema, and time of disappearance of traumatic endothelial rings were recorded. And specular microscopic examination was performed. The duration of existence of traumatic endothelial rings was mean 4.6 days. On the specular microscopic examination, the count of corneal endothelial cells in the injured eye decreased by mean 16.8% (ranged from 1 to 56%) than that in the opposite uninjured eye. The duration of existence of traumatic endothelial rings was 3.5 days in the group without combined angle recession and was 6.1 days in the group with combined angle recession. We suggest that the possibility of traumatic corneal endothelial rings and resultant endothelial cell loss and their clinical potential should be always kept in mind in ocular trauma, particularly BB shot injury.

Key words: BB shot, corneal endothelial cells, specular microscopy, traumatic corneal endothelial rings

INTRODUCTION

Traumatic corneal endothelial rings are annular gray opacities of the corneal endothelium caused by the impact of small projectiles on the surface of the cornea, associated with stromal edema.

Traumatic corneal endothelial rings were probably first described by Pichler in 1916, and the level of the damage of these rings were reported to be associated with corneal endothelium by Payr and Raynaud.¹

The traumatic corneal endothelial rings was reported to consist of an annular endothelial cell loss and disruption with adherent macrophages by electron microscopy.²

The purpose of this report is to illustrate the clinical manifestations of the traumatic corneal endothelial rings and the relationships between the traumatic corneal endothelial rings and the resultant endothelial cell loss.

MATERIALS AND METHODS

We retrospectively reviewed the charts of fourteen patients who were diagnosed as traumatic corneal endothelial ring at Hanyang university hospital from July, 1999 to February, 2000. Fourteen patients with traumatic corneal endothelial rings participate in this study. Of these, a dozen were
Table 1. Clinical features of the patients with traumatic corneal endothelial rings

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Sex</th>
<th>Age</th>
<th>Initial BCVA</th>
<th>Final BCVA</th>
<th>Initial recession</th>
<th>Final recession</th>
<th>Angle hyphema</th>
<th>Gross (days)</th>
<th>%*</th>
<th>duration#</th>
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BCVA: best corrected visual acuity, HM: hand motion, *: percent of decreased corneal endothelial cells in the injured eye in comparison with the opposite uninjured eye, #: duration of the presence of traumatic endothelial rings

injured by BB shot, one was injured by blade, and last one was injured by stone.

We examined the initial and final visual acuity, presence of the combined injuries, particularly with respect to gross hyphema and angle recession. We also checked out the time period of disappearance of traumatic endothelial rings. When traumatic corneal endothelial rings were not observed on the slit lamp biomicroscopy, specular photographs were taken at the area of the corneal center.

We divided patients according to the presence of the combined injuries, and compared initial and final visual acuity and the degree of decreased corneal endothelial cells.

Mann-Whitney U test was used to analyze the statistical significance of differences.

RESULTS

The mean initial best corrected visual acuity was 4/20 (ranged from HM to 20/30) and the mean final best corrected visual acuity was 20/20 (ranged from 20/30 to 20/20) (Table 1). Two cases of low final best corrected visual acuity were caused by sphincter rupture in one case and macular edema in another case.

Traumatic corneal endothelial rings were clinically very clearly visible immediately after injury. Slit-lamp examination showed central epithelial defect with stromal edema and gray endothelial ring in all cases (Fig 1).

The duration of presence was mean 4.6 days (ranged from 3 to 7 days).

There were no specific abnormal findings on the slit-lamp examination in the remaining area of cornea surrounding the rings.
On the specular microscopic examinaitain, the count of corneal endothelial cells in the injured eye decreased by mean 16.8% (ranged from 1 to 56%) than that in the opposite uninjured eye.

The percent of decreased corneal endothelial cell in the injured eye in comparison with that of the opposite uninjured eye, was 6.3% in the group without combined angle recession and was 26.7% in the group with combined angle recession (P = 0.002). And also, the percent of decreased corneal endothelial cell was 6% in the group with traumatic hyphema without angle recession, and was 6.5% in the group without both of traumatic hyphema and angle recession (P = 0.481).

The duration of existence of traumatic endothelial rings was 3.5 days in the group without combined angle recession and was 6.1 days in the group with combined angle recession (P = 0.002).

**DISCUSSION**

Traumatic corneal endothelial rings are annular gray opacities of the corneal endothelium caused by the impact of small projectiles on the surface of the cornea, associated with stromal edema. The impact of small foreign bodies onto the cornea with sufficient force, but not penetrating into the cornea stroma, results in clinically apparent gray rings on the corneal endothelium. Caspar described large, 3- to 5-mm, subepithelial, parenchymal, disciform or ring-shaped opacities. But, these lesions were not purely endothelial. Those are presumably due to more massive corneal damage. Payrau and Raynaud localized traumatic corneal endothelial ring to the level of the corneal endothelium. Payrau and Raynaud described multiple, small, ring-shaped, corneal opacities after explosion injuries. They correctly localized the opacities to the endothelium and thought they represented penetration of the cornea by tiny foreign bodies. More recently, Forstot and Gasset presented similar findings in a 14-year-old boy after he had had a fireworks injury, and they postulated that the rings resulted from transient endothelial damage secondary to concussive forces.

Initially, it was thought that the traumatic endothelial rings originated by the penetration of the cornea by microscopic foreign bodies. Later, it was revealed that these rings are a true concussion injury of the corneal epithelium and that they are due to a transference of a force from the epithelial, foreign body impact site through the corneal stroma to the endothelium. And also why the ring-shaped distribution of these lesions occurs is because maximal cell disruption occurs in an annular area where bending and distortion of endothelium and Descemet’s membrane are greatest.

Older concepts of the pathologic mechanism of these concussion injury was a temporary dysfunction of and alteration in the permeability of the endothelium. But, several studies revealed that their pathologic mechanism is an endothelial cell disruption or loss. Maloney et al described by means of specular microsopic examination in traumatic endothelial ring patients that endothelial damage from this kind of trauma results in endothelial cell loss but that a measurable endothelial cell loss occurs only in instances of severe damage. And these disrupted cells in the ring eventually recover or are displaced by normal cells. Cibis et al have been produced traumatic endothelial rings experimentally in rabbits and monkeys. They revealed that these rings consist of swollen or disrupted endothelial cells and accumulation of fibrin and leukocytes, with more normal cells at their center. Wittppen and Stulting and their co-workers have described histopathologic study of human corneal endothelial rings. Scanning electron microscopy of the endothelium showed an annular area of endothelial cell disruption with loss of cell-to-cell contact, swelling, irregular cell membranes, and absence of cells in some areas. And in the center of the lesion and outside the ring of injury, the endothelial mosaic was preserved and endothelial cells appeared to be normal by transmission electron microscopy.

According to the degree of traumatic impact in traumatic endothelial rings, variable endothelial cell disruption in the ring may occur. And these disrupted cells actually recover or under go a process of necrosis or apoptosis. And resultant gap is replaced by enlargement and sliding of normal corneal endothelial cells at the margin of the lesion. As a result, endothelial cell density decrease.

In our study, traumatic corneal endothelial rings were clinically visible immediately after injury, and
resolved within days of the injury. Biomicroscopically, there appeared to be resolution of the endothelial changes without permanent sequelae.

A significant decrease in the endothelial cell density was found in the group with gonioscopically proven angle recession. But, there was no significant difference in the endothelial cell density between the group with traumatic hyphema without angle recession and the group without both of the traumatic hyphema and angle recession. These result may suggest that angle recession may be a good marker for the degree of initial traumatic impact on the eye. The amount of endothelial cell loss also depends on the degree of initial traumatic impact on the eye rather than the presence of anterior chamber hemorrhage or intraocular inflammation. And these results were consistent with other studies.\cite{8,11} Otherwise, the factors representing the severity of injury, are the size of corneal epithelial defects, the size of corneal endothelial rings, and the amount of corneal stromal edema.

Specular microscopy counts corneal endothelial cell as one when cell boundary is intact circumferentially. The case that had decreased corneal endothelial cell by 56\% is thought to have been incompletely healed or have had blurred margin.

At the time of injury, any clinical sequelae does not occur due to endothelial cell loss in traumatic endothelial ring. The amount of remaining endothelial cell reserve in large enough to compensate for the decreased endothelial cell density because the amount of endothelial cell loss is relatively small. But corneal decompensation may occur later when the cornea is subjected to further accidental or surgical trauma or is predisposed to endothelial dystrophy.

And also traumatic endothelial rings may be used for a index of the degree of traumatic impact on the eye together with angle recession.

In conclusion, the possibilities of traumatic corneal endothelial rings and resultant endothelial cell loss and their clinical potential should be always kept in mind.

REFERENCES