

## Corneal Damage after Glaucoma Drainage Device Use

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### Commentary

Since the introduction of the glaucoma drainage device (GDD), the success rate of surgical treatment for refractory glaucoma, such as failed trabeculectomy, neovascular glaucoma, uveitic glaucoma, and glaucoma with wide conjunctival scarring, has improved significantly, compared with conventional trabeculectomy. However, GDD surgery results in unique complications as well as common ones shared with trabeculectomy. Exposure or movement of the implant, limitation of ocular movements and resultant diplopia, and corneal damage are well-known complications resulting from use of a large GDD [1-3].

Corneal damage after surgery, such as cataract surgery, trabeculectomy, vitrectomy, laser iridotomy, and cyclophotocoagulation, are thought to occur only during treatment [4-8]. However, corneal endothelial cell loss after GDD surgery is particularly significant due to its continuity [9-11].

The mechanism of corneal damage after ocular surgery is not fully understood, although mechanical damage from direct contact by the iris or air bubbles at the time of and shortly after surgery may be an important factor. In contrast to trabeculectomy, GDD surgery involves retention of a foreign body, the tube, within the anterior chamber, resulting in the possibility of it touching the cornea during forceful blinking or squeezing of the eyelid. Jet flow of the aqueous humor around the tube end that occurs with heartbeat may also stress the cornea at any time. Moreover, the cornea in refractory glaucoma patients has sometimes been damaged previously by inflammation, surgery, or trauma.

Differences in the drainage mechanism (open tube or valved tube) or plate material (mostly silicone or polypropylene) of the GDD may possibly cause a different tissue response and different effect on the corneal endothelium. There are a few studies comparing corneal complications of different types of GDDs. Most studies found no difference in the frequencies of corneal complications among implants of different material and type, while corneal edema was more frequent in silicone plate implants than polypropylene plate implants in some reports [12-14]. However, there are many confounding factors making this comparison difficult, such as differences in plate size, intraocular pressure course, and other complications after surgery, which may affect the cornea in addition to the materials and types of GDDs.

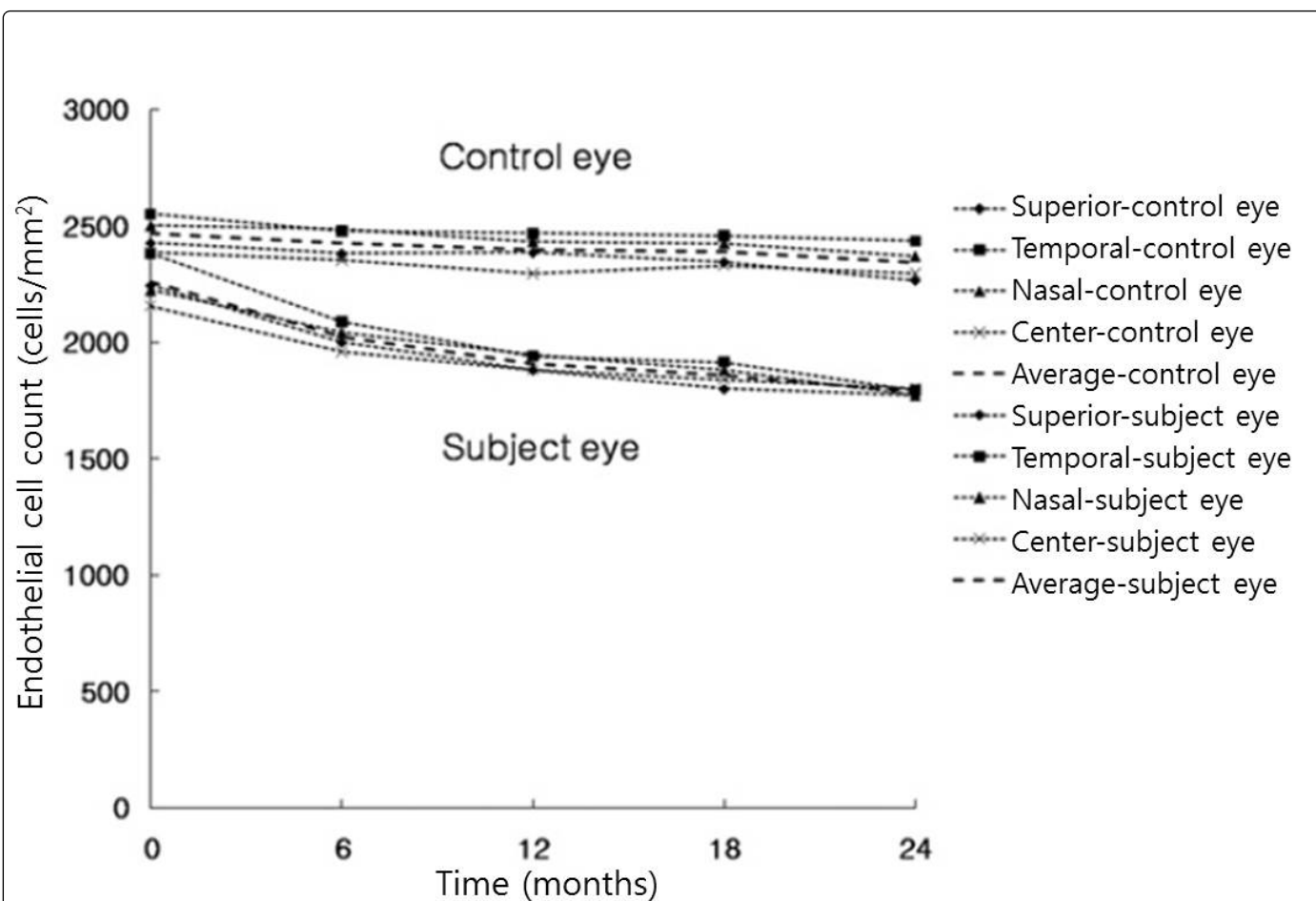
Previous studies have reported corneal damage after use of a GDD. McDermott et al. reported endothelial cell loss averaging two cells per

mm<sup>2</sup> per postoperative month after Molteno implant drainage procedures, but no clinically significant progressive trend in endothelial cell loss was seen in patients undergoing uncomplicated procedures [15]. However, Topouzis et al. reported that the most frequent complication was corneal decompensation or corneal graft failure, and most failures after 12 months of Ahmed glaucoma valve (AGV) implantation follow-up resulted from corneal complications [16]. The frequency of corneal decompensation after glaucoma implant surgery with long-term ( $\geq 2$  years) follow-up ranged from 5-27% [15-17].

A longitudinal study using a baseline preoperative specular microscopic examination reported a cell loss of 10.6% a year after AGV implantation [9]. That study enrolled patients who could complete a 1-year follow-up, and excluded patients who required a second intervention within 1 year after surgery, but this could have led to an underestimation of the degree of damage by excluding patients with a poor short-term postoperative course. A second study by the same group reported a 15.3% cell loss at 1 year and an 18.6% cell loss at 2 years after AGV implantation. The cell losses at 1 and 2 years after surgery were significantly greater than those of controls [10] (Figure 1).

In this study, the degree of cell loss was averaged over four areas, involving the superior, superotemporal, superonasal, and central regions of the cornea. Because the damage was greatest at the superotemporal side and smallest in the central area, averaging the loss from these areas can overemphasize the damage as compared to conventional specular microscopic examination of the central cornea.

A third 5-year report stated that the endothelial cell loss was significant only up to 2 years after surgery, compared with control eyes without AGV. Although there was a more rapid loss of endothelial cells in the AGV group compared with the control group for 5 years (-7.0%/year and -0.1%/year, respectively;  $p < 0.001$ ), the average cell loss, measured at the central cornea, decreased with time from -10.7% during the first year to -2.7%/year from 3 years to the final follow up (45.3  $\pm$  20.6 months), and the statistically significant difference compared with the control group was maintained only during the first 2 years after surgery (Figure 2). However, although there was no statistical significance compared to the control eyes, the endothelial cell density decreased 2 years after surgery: -4.2% 2-3 years after surgery and -2.7% 3 years after surgery. Nevertheless, there is a possibility that a prospective study with a larger number of cases would yield different results.



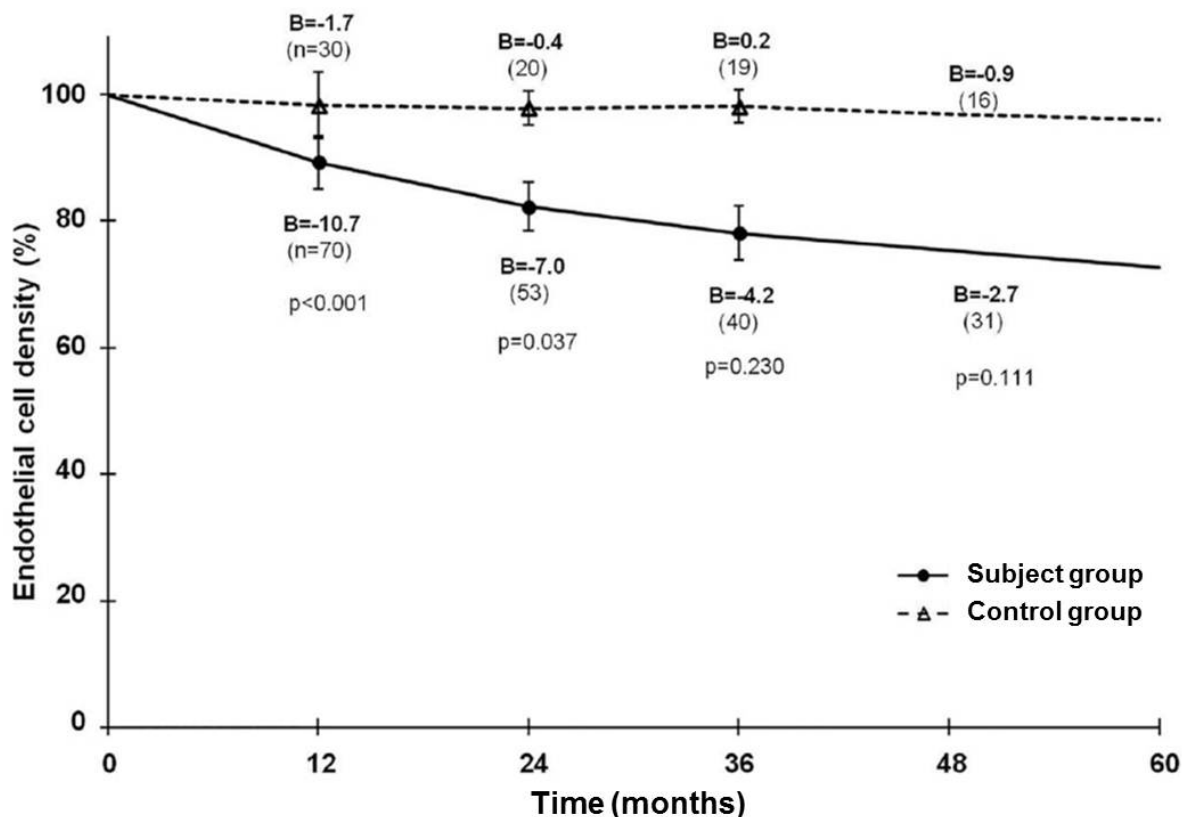
**Figure 1:** Change in endothelial cell count after Ahmed glaucoma valve (AGV) implantation over a two-year period. There was a statistically significant mean percentage decrease in the corneal endothelial cell counts in operated eyes in all measurement areas compared with control eyes during the follow-up ( $p < 0.05$ , Mann-Whitney U-test).

After excluding previous penetrating keratoplasty cases, the cumulative risk of corneal decompensation was 3.3% at 5 years after surgery in uncomplicated cases that did not involve apparent contact between the tube and the cornea. Despite efforts to find clinical variables associated with the rapid loss of endothelial cells, there was no variable other than the presence of the AGV itself.

Because the rate of endothelial cell loss remained the same, regardless of the baseline cell count, patients with low endothelial cell densities before surgery were at a higher risk of corneal decompensation. Although patients with previous penetrating

keratoplasty were excluded from this study to determine the course of uncomplicated cases, it is well known that penetrating keratoplasty involves a greater chance of corneal decompensation.

In conclusion, surgeons must be very careful not to damage the cornea during GDD surgery for the treatment of refractory glaucoma, and all patients treated with the GDD procedure should be monitored for possible corneal problems for more than 2 years. During surgery, extensive efforts should be made to minimize corneal damage. This involves avoiding corneal traction suture, if possible, which may induce wrinkling of the cornea and damage endothelial cells.



**Figure 2:** Corneal endothelial cell densities at different time intervals following Ahmed glaucoma valve (AGV) implantation. The solid line and dashed lines represent the endothelial cell densities in 72 patients with an AGV implant and 31 controls (fellow glaucomatous eyes without AGV implantation that received glaucoma medications), respectively. The annual rate of the endothelial cell density changes [the regression coefficient (B)], for the patient group decreased gradually: -10.7% from preoperatively to 1 year ( $p < 0.001$ , Mann-Whitney U-test), -7.0% from year 1 to year 2 ( $p = 0.037$ ), -4.2% from year 2 to year 3 ( $p = 0.230$ ), and -2.7% from year 3 to the follow-up ( $p = 0.111$ ; mean follow-up,  $45.3 \pm 20.6$  months). Standard error bars are shown at 1, 2, and 3 years.

General anesthesia without a traction suture may be more helpful for compromised patients. Avoiding the flat anterior chamber and avoiding gas injection into the anterior chamber during paracentesis and corneoscleral block excision are important. Use of viscoelastic material in these procedures may be helpful to protect the cornea. Positioning the tube away from the cornea may be the most important procedure in protecting the corneal endothelium. After surgery, all patients should be warned not to squeeze their eyelids, to avoid forceful blinking, and to avoid lid compression during face-down sleeping. For patients with low corneal endothelial cell counts or previous penetrating keratoplasty, surgical options other than GDD should first be considered, and the patient warned of possible complications from corneal decompensation.

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