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Evaluatıon and Management of Hypotonıa and Endothelıal Damage after Glaucoma Draınage Implants

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ABSTRACT
Glaucoma remains a leading cause of irreversible blindness globally, necessitating effective management strategies to reduce intraocular pressure (IOP) and mitigate associated complications. This study investigates the efficacy and potential complications of Ahmed Glaucoma Valve (AGV) implantation, with a particular focus on two critical postoperative challenges: corneal endothelial damage and hypotony. The research assesses both immediate and long-term outcomes of AGV surgeries, specifically examining the effects of tube placement on corneal health and the success of different surgical modifications designed to manage hypotony and loss of endothelial cells. The use of ophthalmic viscosurgical devices (OVDs) is evaluated for their role in managing postoperative hypotony by stabilizing the anterior chamber and maintaining appropriate IOP levels. Additionally, the paper discusses new, less invasive suture-assisted tube revision techniques that address the prevalent issue of tube-endothelial contact, which frequently leads to corneal endothelial decompensation. The adoption of these methods marks a significant advancement in glaucoma surgical practices, particularly beneficial for patients with systemic conditions or inflamed glaucoma, where more complex surgeries pose greater risks. The manuscript delves into various AGV placement strategies, emphasizing the benefits of ciliary sulcus placement over anterior chamber placement to reduce corneal complications. Overall, the findings underscore the necessity for meticulous surgical planning and proactive management to effectively mitigate severe complications associated with AGV implantation.
Key words: *glaucoma, intraocular pressure, Ahmed Glaucoma Valve, cornea, endothelial cells*

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Original article

Оценка и лечение гипотонии с повреждением эндотелиального слоя после хирургии глаукомы с применением дренажных имплантатов

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РЕФЕРАТ
Глаукома остается ведущей причиной необратимой слепоты во всем мире, что требует эффективных стратегий лечения для снижения внутриглазного давления (ВГД) и нивелирования выраженности связанных с этим осложнений. В данном исследовании представлены результаты изучения эффективности и потенциальных осложнений имплантации клапана Ахмеда для глаукомы (КАГ). Уделяется особое внимание двум критическим послеоперационным проблемам: повреждению эндотелия роговицы и гипотонии. В исследовании проведена оценка как непосредственных, так и отдаленных результатов КАГ, в частности, при изучении влияния имплантации трубки на состояние роговицы и успех различных хирургических модификаций, предназначенных для лечения гипотонии и потери эндотелиальных клеток. Использование офтальмологических вискоэластических растворов (ВЭР) позволяет оценить их роль в лечении послеоперационной гипотонии путем стабилизации передней камеры и поддержания соответствующих уровней ВГД. Кроме того, в статье обсуждаются новые, менее инвазивные методы ревизии трубки с использованием швов, которые решают распространенную проблему контакта трубки с эндотелием, часто приводящему к декомпенсации эндотелия роговицы. Внедрение этих методов знаменует собой значительный прогресс в хирургии глаукомы, что особенно полезно для пациентов с системными заболеваниями или глаукомой на фоне воспаления, где более сложные хирургические вмешательства сопряжены с большим риском. В статье подробно рассматриваются различные стратегии размещения клапана Ахмеда, подчеркиваются преимущества размещения в область цилиарной борозды, минуя переднюю камеру, для снижения рисков развития осложнений со стороны роговицы. В целом полученные результаты подчеркивают необходимость тщательного хирургического планирования и превентивного лечения для эффективного снижения частоты тяжелых осложнений, связанных с имплантацией клапана Ахмеда в хирургии глаукомы.
Ключевые слова: *глаукома, внутриглазное давление, клапан Ахмеда, роговица, эндотелиальные клетки*

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INTRODUCTION

Glaucoma is a leading cause of irreversible blindness worldwide, with the primary treatment strategy being the reduction of intraocular pressure (IOP). In cases where IOP cannot be medically controlled, glaucoma filtration surgeries are performed [1]. Today, trabeculectomy and glaucoma drainage devices (GDD) are the main surgical treatment methods [2]. GDDs are typically preferred as a second-line treatment after a failed trabeculectomy or as a primary procedure in clinical situations considered high-risk for trabeculectomy failure, such as previous vitreoretinal surgery, penetrating keratoplasty, uveitic glaucoma, or neovascular glaucoma (NVG) [3]. In our cases, the selection of the Ahmed Glaucoma Valve (AGV) as the primary treatment option was guided by the specific types of glaucoma, the ages of the patients, and their extensive histories of medical treatment.

Although the Ahmed Glaucoma Valve (AGV) is a valved device, ocular hypotony (temporary or permanent) can occur in the early postoperative period. Hypotony may be due to over-priming of the tube and valve insufficiency, leakage of aqueous humor around the silicone tube, or decreased aqueous production due to ciliary body dysfunction. In hypotonic eyes, hypotonic maculopathy, choroidal detachment, and a shallow or flat anterior chamber (AC) can develop. Therefore, to prevent early ocular hypotony, partial ligation of the silicone tube with a vicryl suture may be applied intraoperatively [4]. Despite these precautions, hypotony remains a frequent complication and often necessitates further interventions, particularly in cases of inflamed glaucomas.

Corneal endothelial cell loss and subsequent corneal edema and decompensation are well-known complications of tube shunt surgery. The rate of corneal complications following tube shunt surgeries has been reported at high rates of 16 to 27% in previous reports [5–7]. Factors such as postoperative hypotony, a shallow anterior chamber (AC), tube-cornea contact, and chronic inflammation contribute significantly to the loss of corneal endothelial cells after these procedures [6, 8, 9]. The risk of endothelial loss increases notably when the tube is implanted into the AC [7, 10, 11]. In these cases, removal of the tube and repositioning it through a new route is usually the preferred treatment method, but the management of hypotony and endothelial loss, especially in complicated cases, can be challenging [12, 13].

Our analysis delves into these complications associated with the Ahmed Glaucoma Valve (AGV), including hypotony and endothelial decompensation. We provide a comprehensive review of the existing literature and discuss two complex clinical scenarios, aiming to improve the understanding and management strategies for these serious complications in glaucoma treatment.

CASE PRESENTATIONS

Case 1:

A 61-year-old woman with a long-standing history of diabetes mellitus presented with significant pain and visual impairment in her right eye. An extensive evaluation revealed her right eye's visual acuity was notably reduced to 0.2, with an alarmingly high IOP of 60 mm Hg. The presence of neovascularization on both the iris and the angle indicated advanced NVG. Despite the severe presentation, her retinal nerve fiber layer (RNFL) maintained a thickness of 104 μm, showing no signs of deterioration. She was diagnosed with NVG and commenced on an intensive treatment regimen that included topical brinzolamide/timolol three times daily, topical brimonidine three times daily, and oral acetazolamide four times daily, complemented by panretinal photocoagulation to treat retinal ischemia. Subsequently, an AGV was implanted to enhance control over her condition, taking into account her high visual potential and stable RNFL. As the patient was phakic, the tube was inserted into the AC without ligation.

However, her postoperative trajectory was fraught with complications. She returned on the tenth day with a significant decline in vision. Examination revealed a shallow anterior chamber, direct contact between the tube and the iris, and an edematous lens complicated by posterior synechiae. A Seidel test confirmed a leaking surgical wound, her IOP had dropped to 4 mmHg, and an ultrasound showed choroidal detachment *(Figure 1)*.

These developments required immediate attention to prevent further deterioration of her condition. Ophthalmic viscosurgical device (OVD) was introduced into the AC, and the conjunctiva was sutured to repair the wound site. Subsequent follow-ups indicated that the eye remained normotensive with signs of regressing choroidal detachment. However, swelling of the lens was observed, which pushed the tube forward, resulting in contact between the tube and the corneal endothelium. Pentacam corneal topography (Oculus, Germany) revealed a shallow anterior chamber and the development of diffuse corneal edema, underscoring the complexity of managing this patient's postoperative recovery *(Figure 2)*.

The severity of these findings prompted additional surgical measures to address the complications. Phacoemulsification with intraocular lens implantation was performed, along with synechiotomy, to relieve mechanical pressure on the tube and restore the eye's anatomical configuration. Follow-up examinations indicated that the direct contact between the tube and the endothelium in the primary position had been successfully resolved. However, localized non-healing edema persisted in the superior portion of the cornea. Suspecting that contact between the tube and the endothelium during blinking or other manipulations might be contributing to the problem, a decision was made to reposition the tube

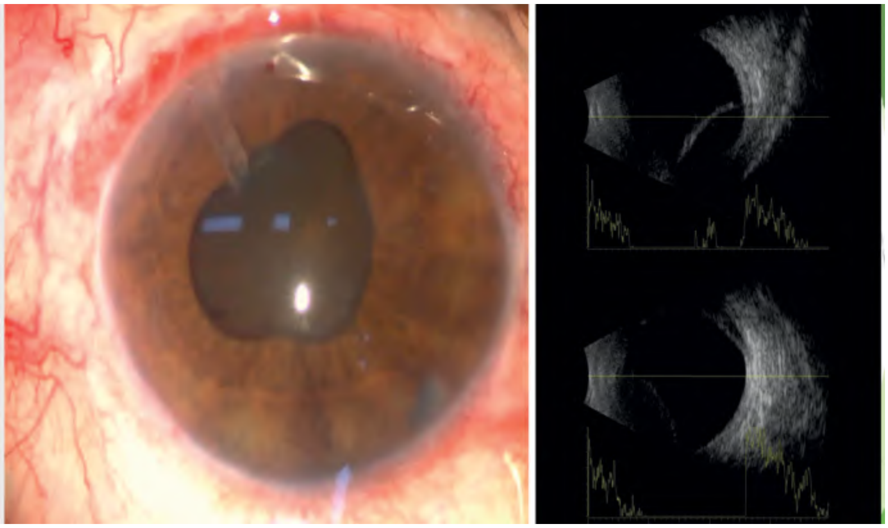

Fig. 1. Ocular examination revealed shallow AC, direct contact between the tube end and the iris, and an edematous, swollen lens with posterior synechiae. An accompanying ultrasound image shows a choroidal detachment

Рис. 1. Биомикроскопическое исследование глаза выявило обмеление передней камеры, прямой контакт между концом трубки и радужной оболочкой, а также набухание хрусталика с задними синехиями

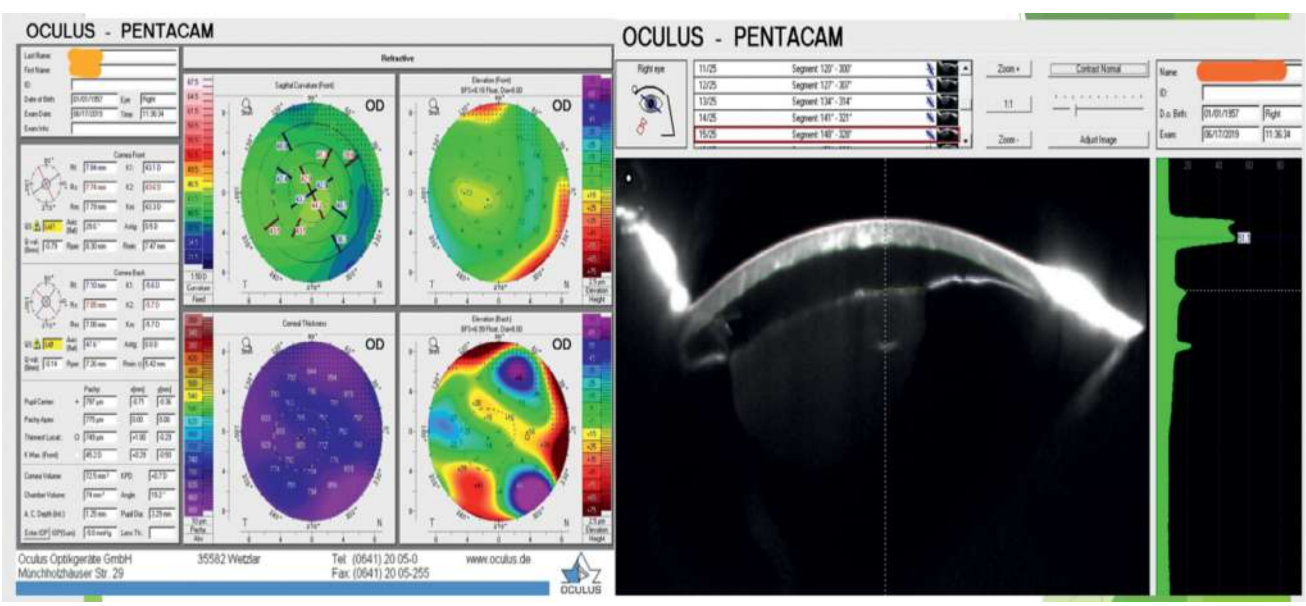

Fig. 2. Corneal topography map highlights the diffuse corneal edema resulting from contact between the tube and the corneal endothelium, as well as a shallow anterior chamber caused by a swollen lens

Рис. 2. Кератотопографическая карта роговицы демонстрирует диффузный отек роговицы, возникший в результате контакта трубки с эндотелием роговицы, а также мелкую переднюю камеру, вызванную набуханием хрусталика

behind the iris into the ciliary sulcus (CS), as illustrated in *Figure 3*. This strategic adjustment aimed to enhance patient outcomes and stabilize the postoperative situation.

Case 2:

A 59-year-old male, currently undergoing oncology treatment for colon cancer, presented with a long-standing history of pseudoexfoliative glaucoma (PXG) and pigment dispersion syndrome (PDS). He had been adhering to a comprehensive anti-glaucoma regimen for 18 years, which included topical dorzolamide/timolol, brimonidine, and oral acetazolamide. During the ophthalmologic examination, the visual acuity in his left eye was recorded at 0.1, with an IOP of 36 mm Hg and a RNFL thickness of 35 μm. In contrast, his right eye showed a visual acuity of 1.0 with IOP well-controlled by the current treatment. Given his relatively young age, extensive history of medication use, and severe conjunctival inflammation, it was decided to implant an AGV in his left eye to achieve better IOP management. Being phakic, the AGV tube was positioned in the AC without ligation.

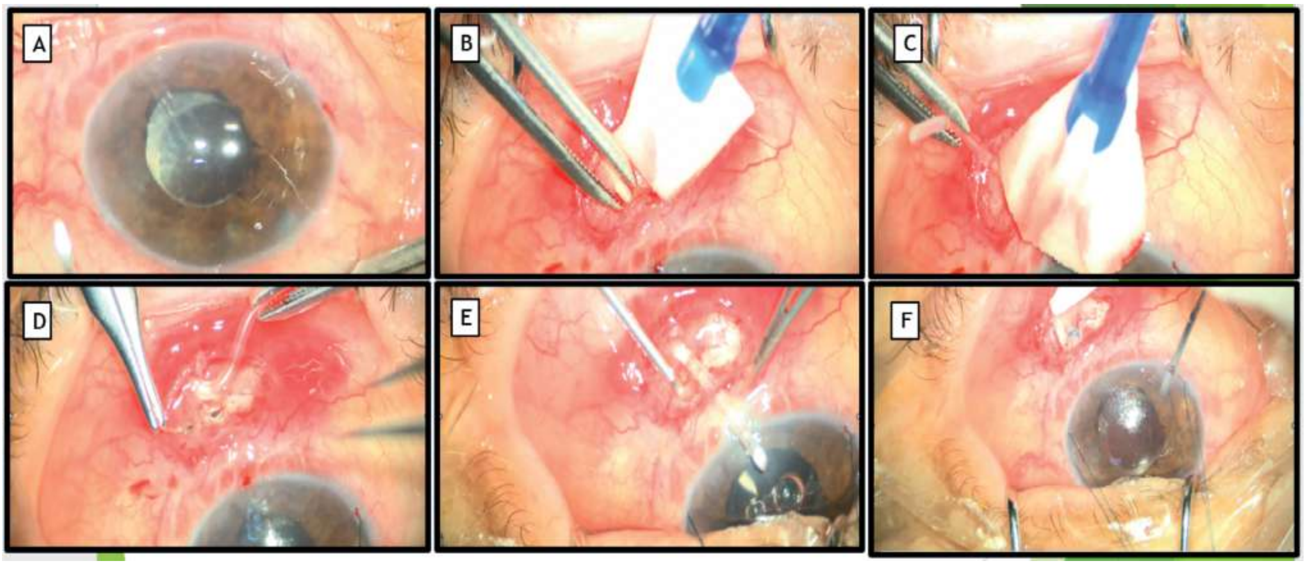

Fig. 3. Repositioning the Tube into the Ciliary Sulcus

A. Preoperative View: Displays the original position of the tube prior to repositioning.

B, C. Exposure and Repositioning: The conjunctiva is opened and the tube is gently retracted from the anterior chamber towards the posterior.

D. Suturing the Former Scleral Entry: The original scleral entry site is sutured closed.

E. Creating a New Entry Site for Sulcus Implantation: A new entry is made for positioning the tube in the sulcus.

F. Final Placement and Suturing: After placing the tube in the sulcus, the site is checked for leakage and then sutured to ensure stability and proper sealing.

Рис. 3. Повторное позиционирование трубки в цилиарную борозду

A. Предоперационный вид: отображает исходное положение трубки в передней камере до ее репозиции.

B, C. Выведение и изменение положения трубки: конъюнктиву открывают и трубку осторожно выводят из передней камеры и реимплантируют в заднюю камеру глаза.

D. Исходное место входа в склеру зашивают.

E. Создается новый склеротомический доступ для позиционирования трубки в цилиарной борозде.

F. Окончательное размещение и наложение швов: после введения трубки в цилиарную борозду место входа в склере проверяют на предмет утечек, а затем зашивают, чтобы обеспечить стабильность и правильное прилегание.

Two days after the implantation of the AGV, the patient developed significant hypotony and choroidal detachment, which continued beyond the initial postoperative period. In an effort to address these complications, an OVD was administered into the AC on the fifth day post-surgery. This procedure successfully stabilized the choroidal detachment and restored the IOP to normal levels. During this intervention, a thorough inspection of the surgical site was also conducted to check for any potential leaks. The Seidel test was negative, confirming the integrity of the wound and indicating that there were no leaks.

At the two-week follow-up, the patient was found to have developed extensive corneal edema due to contact between the tube and the endothelium. Fortunately, the earlier issue of hypotony had resolved by this time, which led to considerations for a surgical revision of the tube to prevent further complications. However, the patient's complex systemic health issues contraindicated the use of general anesthesia, and his limited capacity to attend frequent follow-up visits necessitated a less invasive approach. Consequently, a simpler procedure was chosen involving the repositioning of the tube away from the endothelium using a polypropylene suture [14]. After obtaining informed consent, the procedure was performed under topical anesthesia for minimal discomfort. A 10/0 polypropylene suture equipped with double-armed 3-inch long straight needles was used to transcamerally secure the tube from limbus to limbus. This technique effectively depressed the tube into an optimal position, ensuring it did not contact any intraocular structures. The ends of the suture were then securely tied, with the knot buried under the conjunctiva in the limbal region to minimize any potential irritation or infection. This strategic adjustment aimed to stabilize the tube's position while accommodating the patient's health constraints and reducing the need for more invasive interventions *(Figure 4)*.

Three months following the suture-assisted revision of the tube, the sutures unfortunately loosened, resulting in the tube once again contacting the endothelium. Three months after the suture-assisted tube revision, the sutures loosened, and the tube contacted the endothelium, necessitating a procedure similar to the previous case to relocate the tube into the CS *(Figure 5)*. Despite managing the hypotony and

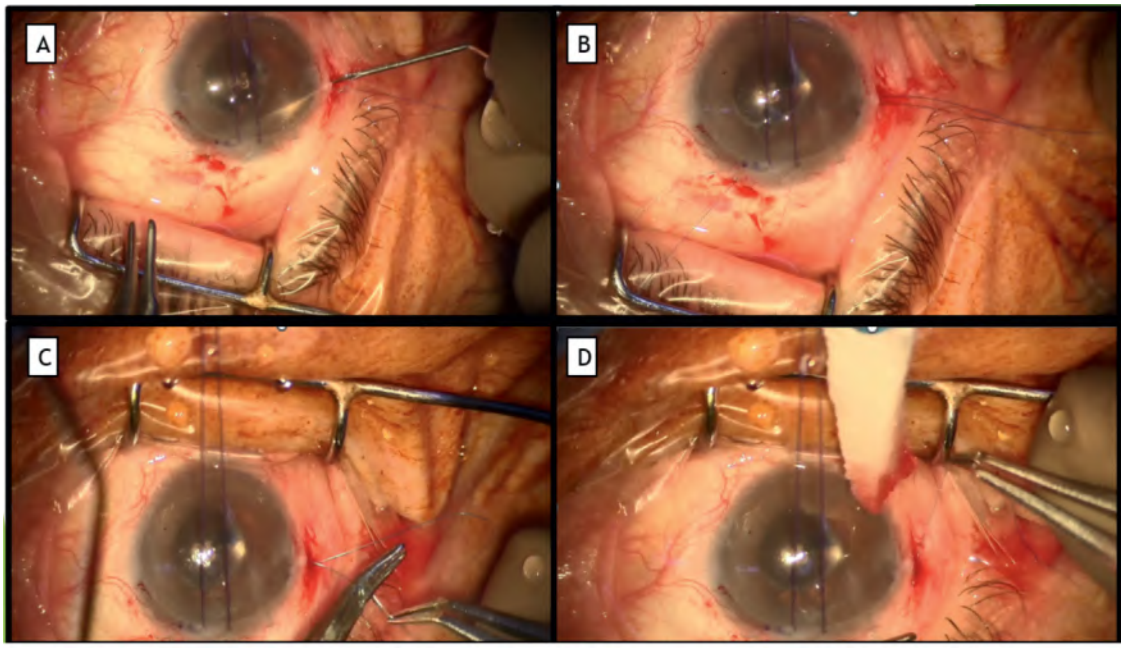

Fig. 4. Suture-Assisted Tube Revision Technique

A. Marking Limbus: Marks are made on both sides of the Ahmed Glaucoma Valve (AGV) tube near the limbus.

B. Suture assisted tube flattening: A 10-0 polypropylene suture is placed trans-camerally, running from limbus to limbus over the tube to depress it into an optimal position.

C. Securing the Suture: Once sufficient tension is achieved, the suture ends are cut and tied.

D. Final Positioning: The procedure concludes after confirming that the tube has flattened and moved away from the endothelium, ensuring reduced risk of corneal damage.

Рис. 4. Техника ревизии трубки с помощью шовного материала

A. Маркировка лимба: маркировка делается с обеих сторон трубки клапана Ахмеда (КАГ) рядом с лимбом.

B. Уплощение трубки с помощью наложения швов: полипропиленовый шов 10-0 накладывается транскамерально от лимба к лимбу над трубкой, чтобы привести ее в оптимальное положение.

C. Закрепление нити: как только будет достигнуто достаточное натяжение, концы нити обрезаются и завязываются.

D. Окончательное позиционирование: процедура завершается после подтверждения уплощения трубки и ее отдаления от эндотелия, что обеспечивает снижение риска повреждения роговицы.

tube-endothelial contact, the patient developed established endothelial decompensation and was referred to the cornea unit.

DISCUSSION

Glaucoma affects approximately 3.5% of the global population aged between 40 and 80, necessitating a variety of medical and surgical interventions aimed at slowing the disease's progression. However, these treatments can inadvertently impact the corneal endothelium, a critical layer whose full response to such interventions remains inadequately explored [15]. The corneal endothelium itself is composed of a single layer of hexagonal cells essential for maintaining clear vision by regulating corneal hydration through active ion transport. Disruptions in this delicate balance can result in corneal swelling and a subsequent loss of visual clarity. Despite the prevalence of glaucoma and its treatments, the precise mechanisms by which it induces changes in corneal endothelial cells remain elusive, highlighting a significant gap in our current understanding [16]. Gagnon et al. have described three potential mechanisms for this: direct compression from higher IOP, congenital alterations in the endothelium and trabecular meshwork, and toxicity from glaucoma medications [17].

Glaucoma drainage devices (GDDs) are increasingly favored for managing complex and refractory glaucoma, often being the first choice for surgical intervention among many clinicians [18]. According to the findings of the Tube Versus Trabeculectomy Study, GDDs generally show higher success rates than trabeculectomy with mitomycin C over a five-year follow-up, achieving similar reductions in IOP while reducing the reliance on additional glaucoma medications [7]. The use of GDDs is particularly growing among high-risk patients where alternative treatments like trabeculectomy are likely to fail [19]. Despite their benefits, the placement of GDDs into the AC angle can lead to significant complications, including corneal endothelial decompensation, with incidence rates reported between 7 and 27% [7, 18]. To mitigate these risks, inserting the tube through the CS has emer-

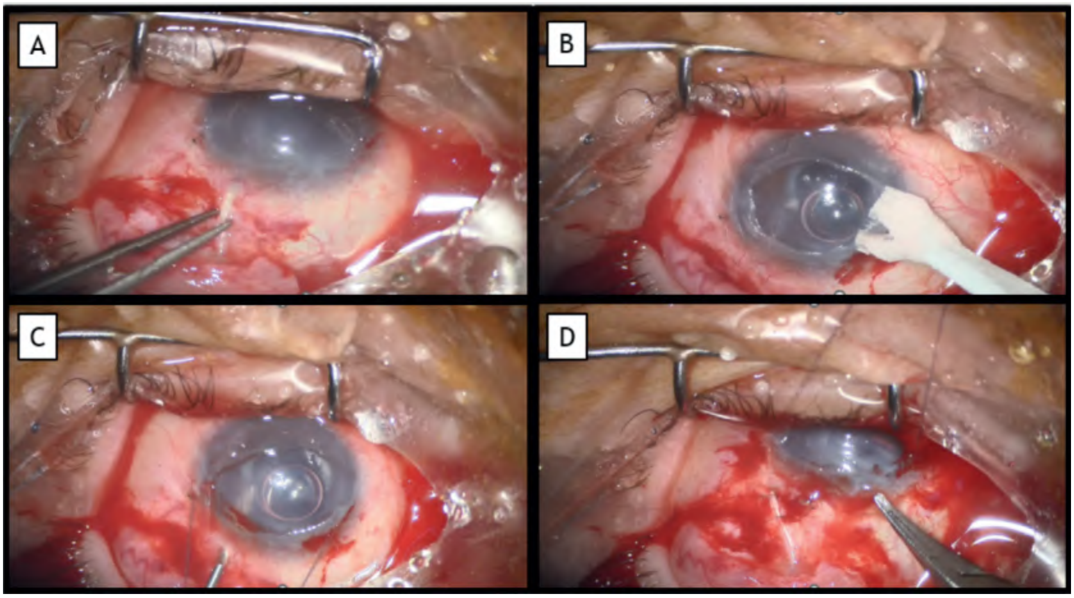

Fig. 5. Repositioning the Tube into the Ciliary Sulcus in a Case of Intensely Inflamed Glaucoma

*A. Exposure***:** The surgical field is prepared by opening the conjunctiva, followed by gentle retraction of the tube from the anterior chamber.

B. Corneal deepitheliazation: Due to severe edema obscuring anterior chamber details, the cornea is deepithelialized to improve visibility and handling during the procedure.

C. Creating a New Entry Site for Sulcus Implantation: A new entry point is surgically created to reposition the tube into the ciliary sulcus, aimed at achieving better positional stability and reducing endothelial contact.

D. Final Placement and Suturing: The tube is meticulously placed in the newly created sulcus position, followed by careful inspection for any leaks. The surgical site is then sutured to ensure both stability and proper sealing of the tube. Additionally, the silicone tube is ligated to prevent potential hypotony.

Рис. 5. Изменение положения трубки в цилиарной борозде на фоне воспаления после хирургии глаукомы.

A. Выведение трубки: операционное поле подготавливается путем вскрытия конъюнктивы с последующим осторожным выведением трубки из передней камеры.

B. Деэпителизация роговицы: из-за сильного отека, скрывающего визуальную детализацию в передней камере, эпителий роговицы удаляют для улучшения видимости и удобства во время процедуры.

C. Создание нового места входа для имплантации в цилиарную борозду. Новая точка входа создается хирургическим путем для размещения трубки в цилиарной борозде с целью достижения лучшей позиционной стабильности и уменьшения эндотелиального контакта.

D. Окончательное размещение и наложение швов. Трубку основательно размещают в новом положении, созданном в цилиарной борозде с последующей тщательной оценкой на предмет фильтрации. Затем место операции зашивают, чтобы обеспечить стабильность и правильную фиксацию трубки. Кроме того, на силиконовую трубку накладывают шов, чтобы предотвратить потенциальную гипотонию.

ged as a viable alternative, especially in pseudophakic/aphakic patients or those with peripheral anterior synechiae (PAS). This approach aims to preserve corneal health while effectively managing the glaucoma [20].

Corneal decompensation is a well-documented complication of common glaucoma procedures, yet the specific causes and risk factors are not fully understood. It is increasingly evident that the pathophysiology extends beyond mere mechanical injury and involves ongoing processes. Research into corneal edema post-glaucoma interventions has identified multiple mechanisms for endothelial cell damage. These include mechanical trauma from the surgery itself, alterations in the composition of aqueous humor, and shifts in the dynamics of aqueous humor flow [21]. A notable two-year study demonstrated a significant and progressive decline in endothelial cell density (ECD) following AGV implantation, with an average loss of 18.6%. The most substantial decrease occurred in the superotemporal quadrant, directly impacted by the tube placement, showing a loss of 22.6%. Such declines are clinically significant, with corneal decompensation often being the most common complication arising from this procedure [6]. Comparatively, while other intraocular surgeries like cataract and vitreoretinal surgeries also cause endothelial cell loss, such losses are generally one-time events. In contrast, ECD loss from GDD implantations tends to be progressive [15, 22]. Additionally, studies indicate that the position of the tube relative to the cornea, which may shift over time, correlates with the degree of endothelial cell loss. For instance, a study tracking 70 eyes with Baerveldt tubes freely placed in the anterior chamber showed a significant decrease in the distance between the tube and the cornea over 24 months, suggesting that tube migration is a critical factor affecting endothelial health [23].

Given the inflammatory nature of the glaucoma in our cases and the long-term medical treatment these patients had undergone, the likelihood of successful trabeculectomy was anticipated to be low; thus, AGV implantation was the preferred initial strategy. A significant limitation in our preoperative preparation was the absence of endothelial cell count and morphological analysis. Had specular microscopy been feasible before surgery, we might have considered alternative surgical approaches given the elevated risk of endothelial loss. Unfortunately, the high preoperative IOP in these patients caused corneal edema, which hindered such diagnostic assessments. Furthermore, research suggests that the incidence of corneal edema is comparably high following any IOP-lowering procedure, whether it involves device placement or not. This indicates that corneal edema is a potential risk inherent to all such interventions. To better understand the nuances of corneal decompensation across different treatments, it is crucial to conduct long-term follow-up studies comparing outcomes between GDD implantations and trabeculectomy [21].

The relationship between glaucoma and endothelial damage extends beyond the effects of surgical intervention; our examination reveals that certain types of glaucoma inherently predispose patients to endothelial dysfunction. Particularly in complex cases like NVG and pseudoexfoliation (PEX), the endothelial cell count and functionality are notably inferior compared to those in healthy individuals [24]. Research shows that patients on chronic therapy with multiple IOP-lowering medications experience molecular alterations in the corneal endothelium [15]. From a molecular perspective, these drugs are thought to affect endothelial cells particularly by altering intracellular calcium balance [25]. Therefore, the risk of endothelial decompensation following GDD implant surgery should be kept in mind for these patient groups [26].

Anterior chamber (AC) tube placement, while effective in reducing IOP, poses corneal endothelial risks [12]. Conversely, CS and vitreous cavity placements reduces the risk of mechanical damage to the cornea, which can result from blinking, eye rubbing, or other external force on the eye [20, 27, 28]. Positioning the tube in the CS increases the distance between the tube and the corneal endothelium and the effects from the turbulent flow at the tip of the implant or intermittent tube-corneal touch are mitigated. The iris may also act as a barrier to mechanical factors that cause ECD loss [29]. Studies, including those by Zhang et al., have demonstrated that the iris effectively shields the endothelium from tube-related damage in CS placements, maintaining ECD across different corneal areas [28]. Retrospective and interventional studies have documented a lesser decline in ECD in CS placements over time, emphasizing its benefits over AC placements [30, 31]. For instance, a 2021 study noted a more pronounced decrease in ECD in the AC group compared to the CS group over 24 months, with the AC group experiencing a 20% reduction versus 10% in the CS group [29]. While CS tube placement offers considerable advantages in reducing endothelial cell loss, it does present challenges such as difficulty in accurately placing the tube during surgery, and the potential for iris pigmentation due to friction [32]. Because of these constraints, most GDDs are inserted into the AC [33]. In cases where the tube is placed in the AC, surgeons aim to minimize the tube's length and align it as close and parallel to the iris as possible to reduce the risk of endothelial damage. However, progressive endothelial damage remains a concern with AC placements. While pars plana placement requires a vitrectomy, introducing potential complications, CS placement has been increasingly favored for its ability to preserve endothelial integrity by increasing the separation from the cornea and minimizing the effects of turbulent flow at the tube tip [29].

Several hypotheses have been suggested to explain the loss of ECD following AGV surgery. The proposed theories include turbulent flow at the implant's tip, postoperative inflammation, depletion of nutrients and oxygen, intermittent contact between the tube and the cornea or uveal tissue, and the foreign body effects of the silicone tube [29, 34]. Rososinski and colleagues have posited that the mere presence of the tube in the AC can trigger inflammation, either from physical contact or simply from its presence, leading to the formation of peripheral anterior synechiae (PAS). PAS, in turn, has been negatively correlated with endothelial cell count, and can lead to endothelial decompensation either through direct iris-endothelial contact or indirectly by increasing IOP due to impaired outflow [30]. Inflammation alters the composition of the aqueous humor, increasing oxidative stress and endothelial loss. Research indicates that GDDs can themselves exacerbate inflammation and increase inflammatory cytokines within the aqueous humor, potentially intensifying inflammation [35]. Additionally, the jet stream of aqueous humor flowing through the silicone tube during the heartbeat could contribute to endothelial loss, even without direct mechanical contact [34, 36].

Freedman and colleagues have reported that cytokines from a foreign-body reaction in the bleb of aqueous drainage implants can diffuse back into the AC. This interaction, combined with the altered flow dynamics near the tube's internal tip—where back-and-forth motion due to pulse-induced IOP changes can occur—may mechanically damage the nearby corneal endothelium. Normally, aqueous fluid enters the AC through the pupil, flows forward toward the endothelium, and then peripherally to the trabecular meshwork, facilitating efficient nutrient delivery and waste removal. However, when flow is redirected through a GDD tube, these metabolic exchanges are likely compromised [37].

Risk factors for endothelial decompensation when a shunt tube is present include high IOP, an initially low endothelial cell count (observed in conditions like Fuchs endothelial dystrophy or post-cataract surgery or trabeculectomy with mitomycin C), a shallow AC, synechial angle closure, direct tube-endothelial contact, and the presence of a corneal graft [38]. Even when direct endothelial contact is eliminated, endothelial loss may continue, suggesting the influence of the tube's dynamic movements and intermittent endothelial contact. Dynamic tube migration, particularly prevalent in inflammatory glaucomas such as uveitis, can occur in various gaze positions, as exemplified by our cases [39]. Regarding the compensation mechanisms of corneal endothelial cells, it is expected that the central ECD reflects the superotemporal ECD to some extent. Since corneal endothelial cells cannot reproduce, they compensate for damage through sliding, rearrangement, and enlargement. Therefore, central ECD loss might not only result from direct central damage but also as a compensation for damage in the superotemporal area [11]. This might explain the diffuse corneal edema in such patients. Koo et al. have also focused on the mechanism of endothelial loss involving the aqueous jet stream, noting that the positioning of the tube end ('bevel up') exposes endothelial cells more directly to these currents, thereby increasing the risk of endothelial loss [5].

Our initial case involved NVG, a form of secondary glaucoma characterized by inflammatory processes, where hypotony ensued following the placement of an AGV into the AC. Hypotony following drainage implantation commonly arises from leaks at the tube entry sites [40]. In this instance, a progressive sequence of events unfolded, involving tube-lens contact, the onset of cataract formation, and the tube exerting pressure against the endothelium due to lens swelling. Throughout the management process, cataract surgery was performed, and the tube's position was initially assessed as appropriate and unchanged. However, given the inflammatory nature of NVG, compounded by the presence of synechiae, the likelihood of endothelial contact increased. Managing NVG poses significant challenges, and as time progresses, angle closure can potentially direct the tube end towards the endothelium, exacerbating the risk of endothelial damage [41].

In cases involving AC AGV, immediate intervention is necessary upon noticing endothelial decompensation due to tube-endothelial contact. Prior to opting for tube placement in the CS, less invasive techniques might have been introduced. For instance, as demonstrated in our second case, suture-assisted tube revision serves as a feasible alternative for patients who cannot undergo anesthesia due to systemic conditions. On the other hand, if the conjunctiva is fibrosed, reopening it for tube reinsertion can be challenging. This technique, employed by Bochmann et al. in a patient with significant subconjunctival scarring and no scleral support , may induce complications such as astigmatism. In their case, while visual acuity remained stable, surgically induced astigmatism was noted. Other potential complications include suture erosion at the limbus, long-term degradation of prolene, or ocular infection [14]. Despite Bochmann et al. reporting no complications aside from induced astigmatism over a 20-month follow-up period, our case exhibited loosening of sutures shortly after the procedure.

Ma et al. proposed a technique for repositioning the tube with scleral fixation, which does not necessitate dissection or retrieval from the original scleral pathway. This method involves creating a scleral flap at the fixation site and maintaining the AC with a chamber maintainer. A precise incision is made just above the point where the tube enters the AC, allowing the tube end to be carefully flipped out using a Sinskey hook. Subsequently, a double-armed 10/0 Prolene straight needle is employed to penetrate through the tube end, with one needle entering the AC through the incision and being drawn through the scleral flap, followed by the other needle. This meticulous technique aligns the tube end parallel to the corneal surface, thereby minimizing potential complications associated with conventional methods [13].

Another suture assisted revision technique also involves using a scleral flap. Initially, the tube's position is identified, typically situated in the superotemporal quadrant. Subsequently, a meticulous placement of a 9-0 Prolene (Ethicon; Johnson and Johnson, USA) straight transchamber needle is executed across the cornea to overlay the body of the AGV tube. Following this, fornix-based conjunctival peritomies are meticulously performed, and partial-thickness scleral flaps are created. The Prolene suture is then inserted at the 10 o'clock limbus under the scleral flap and exits at the 2 o'clock limbus. This precise needle placement ensures that it lies above the AGV tube. Subsequently, the suture is securely fastened to displace the tube away from the cornea. Both scleral flaps and conjunctival peritomies are carefully closed to ensure that the suture knot remains adequately covered [42]. In this technique, different from the one we applied, the sutures passing further back from the limbus and being held under the scleral flaps might increase the tension and flattening effect of the suture.

In managing endothelial decompensation and tube-endothelial contact, revising the tube's placement into the CS has been the definitive method in both cases presented. Initially, it might be questioned why placement in the sulcus was not chosen. One reason was the phakic nature of the patients. Additionally, it's important to consider that sulcus implantation doesn't entirely prevent endothelial loss and may contribute to inflammation. A recent study comparing endothelial loss after trabeculectomy and drainage implant surgery showed significantly higher endothelial loss post-implantation, even years later, with continued higher levels of inflammation indicated by persistent elevated flare levels [43]. In the study group with sulcus AGV, monthly ECD loss was notably higher in the superotemporal corneal region compared to the central and inferonasal areas. This suggests that, while CS placement reduces trauma to central endothelial cells, it still induces endothelial loss and corneal changes near the tube [28]. Hypotheses for this ongoing loss include turbulence from altered aqueous humor flow, immune reaction from foreign body presence, and intermittent tube contact with the endothelium [43, 44]. Moreover, CS placement could exacerbate intraocular inflammation in uveitic glaucoma cases due to microscopic and continual contact between the tube and the iris, thereby promoting further inflammation [18]. Therefore, AC implantation might be considered beneficial, especially in inflamed or complex glaucoma cases, and was deemed appropriate in our cases for this reason.

Murakami et al. demonstrated that CS placement with a GDD led to decreased central endothelial cell density. Factors like previous ocular surgeries and high preoperative IOP were linked to this reduction. The long-standing glaucoma in our cases also posed a risk for post-surgical endothelial loss. They emphasized that the number of past intraocular surgeries was directly associated with endothelial decompensation. Hence, the suture revision surgery applied to our second case could have negatively impacted the process as an additional intraocular surgical intervention [10].

Mechanical contact between the tube and the endothelium is a primary cause of endothelial damage following the implantation of a GDD. If the tube is left excessively long, resulting in evident contact with the corneal endothelium, tube trimming should be considered. Traditionally, this procedure is viewed as a major surgical intervention involving multiple steps: dissection through scarred conjunctiva, navigating beneath the scleral patch graft, extracting the tube from the AC, cutting it to the appropriate length, and then reinserting and securing it with sutures. This comprehensive approach can be extensive and prone to complications.

However, Asrani and colleagues [45] have proposed a less invasive method requiring just two paracenteses, while Soebijantoro and associates [46] have introduced an even more minimally invasive technique involving only a single paracentesis. This streamlined procedure minimizes the surgical footprint and potential for complications.

Hypotony, characterized by IOP below 5 mmHg, poses a significant concern following glaucoma surgeries, including AGV implantation Factors such as insufficient capsule creation, excessively wide sclerostomy, or weak aqueous production due to postoperative iritis can contribute to hypotony [40]. In such cases, it is crucial first to investigate and rule out the possibility of leakage [40, 47]. The AGV is specifically designed to minimize postoperative hypotony through its valve mechanism, demonstrating fewer severe complications compared to the Baerveldt glaucoma implant [8]. Despite the precautionary designs, studies indicate that while AGV effectively reduces hypotony risks, it does not eliminate them. Surgical precautions to prevent persistent hypotony include avoiding over-priming the tube and excessive manipulation of the valve housing during surgery [48]. In the realm of clinical outcomes and predictive factors for postoperative hypotony, Rachmiel et al. observed lower IOP levels in patients with uveitic glaucoma compared to those with primary open-angle glaucoma in the initial months post-surgery [41]. However, Kaderli et al. found no significant differences in risk factors such as age, sex, lens status, history of previous ocular surgeries, preoperative glaucoma medication usage, or type of glaucoma affecting the prevalence of postoperative hypotony [47]. Chronic hypotony, which persists beyond four weeks, can lead to serious complications such as accelerated cataract formation, choroidal detachments, hypotony maculopathy, and even suprachoroidal hemorrhages [49, 50]. In these situations, surgical interventions may be necessary, such as ligating sutures for tube shunts or revising the surgery to reduce outflow, ensuring vision preservation [51, 52]. Non-physiological IOP levels, whether high or low, can disrupt endothelial function. Elevated or decreased IOP can impair endothelial cell function, leading to decompensation through the disruption of desmosomes and junctional complexes [53]. Furthermore, long-term complications like mechanical tube corneal rubbing might go unrecognized. A long AC tube, more prone to movement in eyes with low IOP, could lead to intermittent corneal contact during eye movements such as blinking or rubbing, potentially exacerbating endothelial damage over time [21].

Ophthalmic viscosurgical devices (OVDs) play a crucial role in managing complications associated with ocular surgeries by blocking the trabecular meshwork, closing ciliary body detachments, and interrupting the cycle of hypotony and choroidal effusion. These devices also increase the viscosity of aqueous humor, effectively slowing the rate of filtration through tube shunts or sclerotomies [51, 54]. In our cases, postoperative hypotony was managed by injecting OVD into the AC. For the first patient, a Seidel positive wound with leakage was identified and subsequently repaired. It is important to note that if wound leakage is not addressed, the effectiveness of the OVD injection will be limited, thus checking and repairing the wound site is crucial initially.

In their research comparing AGVs placed in the AC and the CS, Bayer and colleagues observed that shallow AC and the need for AC revisions with OVD were more frequent in implants placed in the AC. They provided three possible explanations for this observation, suggesting that the AC tends to be deeper in pseudophakic or aphakic eyes compared to phakic eyes, influencing the rate of AC reformation. Secondly, the threshold for reforming the AC may have been lower in the AC group to prevent mechanical contact between the tube and the corneal endothelium or the crystalline lens. Thirdly, they noted that peritubular filtration of aqueous is less likely in CS implantations due to the longer pathway through sclera and ciliary body tissues, compared to the shorter pathway through just scleral tissue in AC implantations [18].

Given the challenges associated with sulcus implantation, Chey and colleagues recently published a study on a technique to reduce endothelial damage by utilizing a guided-assisted AGV implantation in the AC. In this technique, a 4-0 nylon suture is used as an intraluminal guide to facilitate accurate placement. Their findings revealed that none of the cases with guided implantation required repositioning of the tube into the CS, in contrast to standard implantation where such repositioning was necessary in 10 out of 79 cases. The postoperative complications did not differ significantly between the two groups, except for instances of flat AC, potentially due to leakage at the sclerotomy site following multiple punctures made to achieve the desired tube positioning in the non guided AGV group. Furthermore, the study indicated that guided implantation resulted in less endothelial loss over a two-year follow-up period [55]. Given that this is a recent and innovative technique, there is a need for long-term comparative studies with traditional sulcus implantation to fully evaluate its efficacy and safety.

Research indicates that both CS and AC tube shunt placements are effective and safe for reducing IOP. However, studies also show that CS implantation leads to significantly lower rates of corneal endothelial cell loss compared to AC placements. This makes CS implantation particularly advantageous for patients at high risk of corneal decompensation [3, 18, 20, 29]. When complications such as tube-endothelial contact occur, the recommended interventions include trimming or repositioning the tube tip. Nonetheless, it is important to note that endothelial decompensation resulting from such contact may become irreversible, potentially necessitating corneal transplantation to restore vision [38].

CONCLUSION

The findings from this study underscore the critical nature of strategic AGV placement and postoperative management to optimize patient outcomes in glaucoma surgery. While AGV is effective in reducing IOP, its association with significant risks such as hypotony and corneal endothelial decompensation necessitates careful surgical planning and follow-up. The comparison between AC and CS placements reveals a clear preference for the latter, given its reduced impact on corneal endothelial health. Furthermore, the introduction of guided-assisted AGV implantation presents a promising technique to reduce endothelial damage, although long-term studies are required to establish its efficacy fully. Despite being a valved system, AGV implantation, especially in high-risk cases, may benefit from intraoperative ligation to prevent postoperative hypotony and subsequent tube-endothelial contact.

These findings highlight the complexity of managing high-risk glaucoma cases and emphasize the importance of tailored surgical approaches that consider individual patient anatomy and disease severity. Future research should continue to explore innovative surgical techniques and postoperative management strategies to enhance the safety and effectiveness of glaucoma filtration surgeries, thereby improving quality of life for affected patients.

REFERENCES/ЛИТЕРАТУРА

1. Tham YC, Li X, Wong TY, Quigley HA, Aung T, Cheng CY. Global prevalence of glaucoma and projections of glaucoma burden through 2040: a systematic review and meta-analysis. Ophthalmology. Nov 2014;121(11): 2081–2090. doi: 10.1016/j.ophtha.2014.05.013
2. Patel S, Pasquale LR. Glaucoma drainage devices: a review of the past, present, and future. Semin Ophthalmol. 2010;25(5–6): 265–270. doi: 10.3109/08820538.2010.518840
3. Arikan G, Gunenc U. Ahmed Glaucoma Valve Implantation to Reduce Intraocular Pressure: Updated Perspectives. Clin Ophthalmol. 2023;17: 1833–1845. doi: 10.2147/OPTH.S342721
4. Kee C. Prevention of early postoperative hypotony by partial ligation of silicone tube in Ahmed glaucoma valve implantation. J Glaucoma. 2001;10(6): 466–469.
5. Koo EB HJ, Han Y, Keenan JD, Stamper, BH RJ. Effect of glaucoma tube shunt parameters on cornea endothelial cells in patients with Ahmed valve implants. Cornea. 2015;34: 37–41.
6. Lee EK, Yun YJ, Lee JE, Yim JH, Kim CS. Changes in corneal endothelial cells after Ahmed glaucoma valve implantation: 2-year follow-up. Am J Ophthalmol. Sep 2009;148(3): 361–367. doi: 10.1016/j.ajo.2009.04.016
7. Gedde SJ, Schiffman JC, Feuer WJ, et al. Treatment outcomes in the Tube Versus Trabeculectomy (TVT) study after five years of follow-up. Am J Ophthalmol. 2012;153(5): 789–803e2. doi: 10.1016/j.ajo.2011.10.026
8. Christakis PG, Kalenak JW, Zurakowski D, et al. The Ahmed Versus Baerveldt study: one-year treatment outcomes. Ophthalmology. 2011;118(11): 2180–2089. doi: 10.1016/j.ophtha.2011.05.004
9. Chen J, Gedde SJ. New developments in tube shunt surgery. Curr Opin Ophthalmol. 2019;30(2): 125–131. doi: 10.1097/ICU.0000000000000549
10. Murakami Y, Hirooka K, Yuasa Y, et al. Determinants of corneal endothelial cell loss after sulcus placement of Ahmed and Baerveldt drainage device surgery. Br J Ophthalmol. 2021;105(7): 925–928. doi: 10.1136/bjophthalmol-2020-316379
11. Kim KN, Lee SB, Lee YH, Lee JJ, Lim HB, Kim CS. Changes in corneal endothelial cell density and the cumulative risk of corneal decompensation after Ahmed glaucoma valve implantation. Br J Ophthalmol. 2016;100(7): 933–938. doi: 10.1136/bjophthalmol-2015-306894
12. Weiner A, Cohn AD, Balasubramaniam M, Weiner AJ. Glaucoma tube shunt implantation through the ciliary sulcus in pseudophakic eyes with high risk of corneal decompensation. J Glaucoma. 2010;19(6): 405–411. doi: 10.1097/IJG.0b013e3181bdb52d
13. Ma KT, Kim JH, Seong GJ, Jang DS, Kim CY. Scleral fixation of Ahmed glaucoma valve tube tip for adjustment of cornea-touching malposition. Eye (Lond). 2014;28(1): 23–25. doi: 10.1038/eye.2013.214
14. Bochmann F, Azuara-Blanco A. Transcameral suture to prevent tube-corneal touch after glaucoma drainage device implantation: a new surgical technique. J Glaucoma. 2009;18(8): 576–577. doi: 10.1097/IJG.0b013e3181911284
15. Janson BJ, Alward WL, Kwon YH, et al. Glaucoma-associated corneal endothelial cell damage: A review. Surv Ophthalmol. 2018;63(4): 500–506. doi: 10.1016/j.survophthal.2017.11.002
16. Edelhauser HF. The balance between corneal transparency and edema: the Proctor Lecture. Invest Ophthalmol Vis Sci. 2006;47(5): 1754–1767. doi: 10.1167/iovs.05-1139
17. Gagnon MM BH, Brunette I, et al. Corneal endothelial cell density in glaucoma. Cornea. 1997;16: 314–318.
18. Bayer A, Onol M. Clinical outcomes of Ahmed glaucoma valve in anterior chamber versus ciliary sulcus. Eye (Lond). 2017;31(4): 608–614. doi: 10.1038/eye.2016.273
19. Vinod K, Gedde SJ, Feuer WJ, et al. Practice Preferences for Glaucoma Surgery: A Survey of the American Glaucoma Society. J Glaucoma. 2017;26(8): 687–693. doi: 10.1097/IJG.0000000000000720
20. Eslami Y, Mohammadi M, Fakhraie G, Zarei R, Moghimi S. Ahmed glaucoma valve implantation with tube insertion through the ciliary sulcus in pseudophakic/aphakic eyes. J Glaucoma. 2014;23(2): 115–118. doi: 10.1097/IJG.0b013e318265bc0b
21. Mehta AA, Tu EY, Vajaranant TS. Beyond Mechanical Trauma-Why the Cornea May Decompensate After Glaucoma Surgery. JAMA Ophthalmol. 2019;137(5): 479–480. doi: 10.1001/jamaophthalmol.2019.0173
22. Hau SSA, Bunce C, Barton K. Corneal endothelial morphology in eyes implanted with anterior chamber aqueous shunts. Cornea. 2011;30: 50–55.
23. Tan AN, De Witte PM, Webers CA, et al. Baerveldt drainage tube motility in the anterior chamber. Eur J Ophthalmol. 2014;24(3): 364–370. doi: 10.5301/ejo.5000379
24. Kandarakis SA, Togka KA, Doumazos L, et al. The Multifarious Effects of Various Glaucoma Pharmacotherapy on Corneal Endothelium: A Narrative Review. Ophthalmol Ther. 2023;12(3): 1457–1478. doi: 10.1007/s40123-023-00699-9
25. Wu KY, Hong SJ, Wang HZ. Effects of antiglaucoma drugs on calcium mobility in cultured corneal endothelial cells. Kaohsiung J Med Sci. 2006;22(2): 60–67. doi: 10.1016/S1607-551X(09)70222-0
26. Yuksel N, Emre E, Pirhan D. Evaluation of Corneal Microstructure in Pseudoexfoliation Syndrome and Glaucoma: In Vivo Scanning Laser Confocal Microscopic Study. Curr Eye Res. 2016;41(1): 34–40. doi: 10.3109/02713683.2014.1002046
27. Qin VL, Kaleem M, Conti FF, et al. Long-term Clinical Outcomes of Pars Plana Versus Anterior Chamber Placement of Glaucoma Implant Tubes. J Glaucoma. 2018;27(5): 440–444. doi: 10.1097/IJG.0000000000000931
28. Zhang Q, Liu Y, Thanapaisal S, et al. The Effect of Tube Location on Corneal Endothelial Cells in Patients with Ahmed Glaucoma Valve. Ophthalmology. 2021;128(2): 218–226. doi: 10.1016/j.ophtha.2020.06.050
29. Kim JY, Lee JS, Lee T, et al. Corneal endothelial cell changes and surgical results after Ahmed glaucoma valve implantation: ciliary sulcus versus anterior chamber tube placement. Sci Rep. 2021;11(1): 12986. doi: 10.1038/s41598-021-92420-8
30. Rososinski A, Wechsler D, Grigg J. Retrospective review of pars plana versus anterior chamber placement of Baerveldt glaucoma drainage device. J Glaucoma. 2015;24(2): 95–99. doi: 10.1097/IJG.0b013e31829d9be2
31. Godinho G B-BJ, Oliveira-Ferreira C, Madeira C MA, Falca o-Reis F, Estrela-Silva S. Anterior chamber versus ciliary sulcus Ahmed glaucoma valve tube placement: longitudinal evaluation of corneal endothelial cell profiles. J Glaucoma. 2021;30(2): 170–174.
32. Moon KK, Kim KS. Ciliary Sulcus Ahmed Valve Implantation. KJO. 2007;21: 127–130.
33. Kim Y, Cho WJ, Kim JD, et al. Tube-Iris Distance and Corneal Endothelial Cell Damage Following Ahmed Glaucoma Valve Implantation. J Clin Med. 2022;11(17). doi: 10.3390/jcm11175057

34. McDermott ML, Swendris RP, Shin DH, Juzych MS, Cowden JW. Corneal endothelial cell counts after Molteno implantation. Am J Ophthalmol. 1993;115: 93–96.
35. Rosenfeld C, Lai X, et al. Distinctive and pervasive alterations in aqueous humor protein composition following different types of glaucoma surgery. Mol Vis. 2015;21: 911–918.
36. Law SK, Coleman AL, Caprioli J. Dynamic tube movement of Ahmed glaucoma valve. J Glaucoma. 2009;18(8): 628–631. doi: 10.1097/IJG.0b013e3181996f33
37. Freedman J, Iserovich P. Pro-inflammatory cytokines in glaucomatous aqueous and encysted Molteno implant blebs and their relationship to pressure. Invest Ophthalmol Vis Sci. 2013;54(7): 4851–4855. doi: 10.1167/iovs.13-12274
38. Bersudsky V, Rumelt S. Management of endothelial decompensation because of glaucoma shunt tube touch by Descemet membrane endothelial keratoplasty and tube revision. Cornea. 2011;30(6): 709–711.
39. Lopilly Park HY, Jung KI, Park CK. Serial intracameral visualization of the Ahmed glaucoma valve tube by anterior segment optical coherence tomography. Eye (Lond). 2012;26(9): 1256–1262. doi: 10.1038/eye.2012.131
40. Stein JD, McCoy AN, Asrani S, et al. Surgical management of hypotony owing to overfiltration in eyes receiving glaucoma drainage devices. J Glaucoma. 2009;18(8): 638–641. doi: 10.1097/IJG.0b013e31819aa4e0
41. Rachmiel R, Trope GE, Buys YM, Flanagan JG, Chipman ML. Ahmed glaucoma valve implantation in uveitic glaucoma versus open-angle glaucoma patients. Can J Ophthalmol. Aug 2008;43(4): 462–427. doi: 10.3129/i08-082
42. Kumar H, Gupta A, Gupta V. A microinvasive technique for management of corneal edema secondary to glaucoma drainage device tube-corneal touch. Indian J Ophthalmol. 2018;66(6): 861–862. doi: 10.4103/ijo.IJO_987_17
43. van Kleij JM, Islamaj E, Vermeer KA, Lemij HG, de Waard PWT. The long-term postoperative effect of the Baerveldt glaucoma drainage device and of a trabeculectomy on the corneal endothelium. Acta Ophthalmol. 2022;100(2): 212–217. doi: 10.1111/aos.14815
44. Islamaj E, Wubbels RJ, de Waard PWT. Primary baerveldt versus trabeculectomy study after 5 years of follow-up. Acta Ophthalmol. 2020;98(4): 400–407. doi: 10.1111/aos.14265
45. Asrani S, Herndon L, Allingham RR. A newer technique for glaucoma tube trimming. Archives of Ophthalmology. 2003;121(9): 1324–1326.
46. Soebijantoro I, Noor NA. Tube Length Adjustment and Tube Trimming Technique in Refractory Glaucoma. Case Rep Ophthalmol Med. 2020;2020: 8889448. doi: 10.1155/2020/8889448
47. Kaderli A, Demirok G, Uney G, Yakin M, Gunal B, Eksioglu U. Assessing risk factors for postoperative hypotony in Ahmed glaucoma valve implantation surgery. Int Ophthalmol. Oct 2021;41(10): 3381–3386. doi: 10.1007/s10792-021-01900-3
48. Gupta S, Jeria S. A Review on Glaucoma Drainage Devices and its Complications. Cureus. 2022;14(9): e29072. doi: 10.7759/cureus.29072
49. Schubert HD. Postsurgical hypotony: relationship to fistulization, inflammation, chorioretinal lesions, and the vitreous. Surv Ophthalmol. 1996;41(2): 97–125. doi: 10.1016/s0039-6257(96)80001-4
50. Tuli SS WD, Ciulla TA, Cantor LB. Delayed suprachoroidal hemorrhage after glaucoma filtration procedures. Ophthalmology. 2001;108: 1808–1811.
51. Xia T, Khouri AS. Intracameral viscoelastic treatment for hypotony after glaucoma incisional surgery. Taiwan J Ophthalmol. 2019;9(4): 292–294. doi: 10.4103/tjo.tjo_31_18
52. Sarkisian SR Jr. Tube shunt complications and their prevention. Curr Opin Ophthalmol. 2009;20(2): 126–130. doi: 10.1097/ICU.0b013e328323d519
53. Hatton MP, Perez VL, Dohlman CH. Corneal oedema in ocular hypotony. Exp Eye Res. Mar 2004;78(3): 549–552. doi: 10.1016/j.exer.2003.06.003
54. Tosi GM, Schiff W, Barile G, Yoshida N, Chang S. Management of severe hypotony with intravitreal injection of viscoelastic. Am J Ophthalmol. 2005;140(5): 952–954. doi: 10.1016/j.ajo.2005.06.019
55. Chey JH, Lee CK. Effect of guided Ahmed glaucoma valve implantation on corneal endothelial cells: A 2-year comparative study. PLoS One. 2023;18(2): e0278340. doi: 10.1371/journal.pone.0278340

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