Oculoplastic Challenges in Patients with Glaucoma

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Abstract

Glaucoma is typically a disease that occurs in advanced age, requiring lifelong monitoring and treatment with topical medications, laser procedures, or surgery. Patients with glaucoma may also experience oculoplastic issues due to the natural aging process or as a result of glaucoma treatment or surgery. Eyelid surgery in these individuals can lead to complications and undesirable results. Therefore, it is crucial for oculoplastic surgeons to be aware of the incidence and risk factors associated with oculoplastic problems specific to glaucoma patients. Understanding these potential complications is essential for taking necessary precautions and achieving successful surgical outcomes. The purpose of this review is to raise awareness among ophthalmologists specializing in oculoplasty and glaucoma and to contribute to the quality of life of glaucoma patients.

Keywords: Ectropion, eyelid surgery, glaucoma, oculoplastic problems, periorbitopathy, prostaglandin analog, punctum stenosis

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Introduction

Glaucoma is a chronic, progressive optic neuropathy that usually occurs later in life, characterized by irreversible vision loss due to damage to retinal ganglion cells.[1,2] With global population aging, the prevalence of glaucoma is expected to increase.[2] Age-related anatomic and functional changes in the eyelids and orbital region are often accompanied by oculoplastic problems that can occur as a result of both medical and surgical glaucoma treatments.[3,4]

This review aims to comprehensively examine the oculoplastic complications that can arise iatrogenically during the treatment of glaucoma, as well as oculoplastic problems that may occur in glaucoma patients as a natural result of the aging process. Another aim was to guide physicians in their clinical practice by presenting current approaches to the diagnosis, follow-up, and treatment of these issues. Oculoplastic problems specific to glaucoma patients can be grouped under four main headings: (1) oculoplastic problems resulting from medical treatments, (2) iatrogenic oculoplastic problems resulting from surgical treatment, (3) glaucoma-related conditions developing after oculoplastic surgeries, and (4) appropriate treatment of involutional oculoplastic problems in patients with glaucoma.

Oculoplastic Problems Associated with the Medical Treatment of Glaucoma

Currently, the first-line treatment of glaucoma is primarily medical treatment with topical antiglaucoma agents, as indicated by international guidelines.[5,6] Therefore, good knowledge of the changes to the eyelid and orbit that can result from the use of antiglaucoma medications is important for the early recognition and appropriate management of potential oculoplastic problems.

Prostaglandin-Associated Periorbitopathy

Prostaglandin analogues (PGAs) are often preferred as the first choice in the medical treatment of glaucoma due to their potent intraocular pressure (IOP)-lowering effects, ease of once-daily use, and lower systemic side effect profile.[1,5,6,7,8,9]

However, PGA use can lead to various local side effects that affect the patient's facial appearance and may reduce life comfort, such as iris and periorbital hyperpigmentation, eyelash elongation and discoloration, deepening of the upper eyelid sulcus (DUES) due to orbital adipose tissue atrophy, and enophthalmos with upper eyelid ptosis—conditions collectively referred to as prostaglandin-associated periorbitopathy (PAP) (Figures 1, 2).[3,8,9,10,11,12,13,14,15,16]

Peplinski and Albiani Smith[8] first described DUES in patients using bimatoprost unilaterally. DUES was also reported to develop in fellow eyes in which bimatoprost treatment was initiated and regress after discontinuing bimatoprost, with authors emphasizing the possibly of overlooking DUES in patients using a PGA bilaterally.[8,10,11] Later reports also documented the occurrence of DUES with other PGAs.[3,11,12] Its incidence was found to be highest with bimatoprost, intermediate with travoprost, and lower with latanoprost.[11,13,14]

Lipolysis and reduced collagen fibers in the levator complex caused by PGAs and levator aponeurosis dehiscence due to fibrosis have been implicated as causes of DUES and ptosis associated with PGA use.[3,11,12,14,15] However, the regression of DUES after PGA discontinuation suggests that the pathogenesis cannot be fully explained by Müller muscle fibrosis and that aponeurotic and deep orbital adipose tissue atrophy play a more important role in this process.[14,17] Lipoatrophy of the eyelid and subsequent enophthalmos were also found to be associated with fibroblast apoptosis resulting from inflammatory changes in the orbital extracellular matrix due to PGA use.[3,14,17]

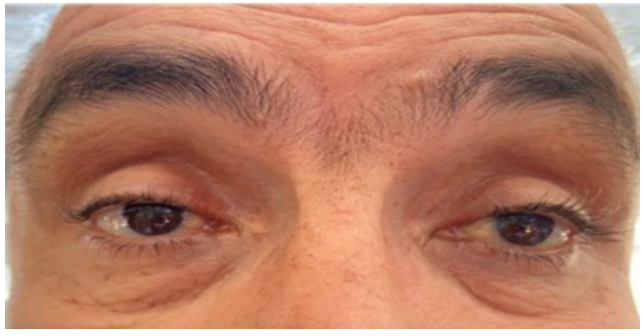

Figure 1. Male patient after bilateral prostaglandin analogue use exhibiting significant bilateral deepened upper eyelid sulci, eyelash elongation, ptosis, and associated pronounced horizontal forehead creases secondary to compensatory frontal muscle use

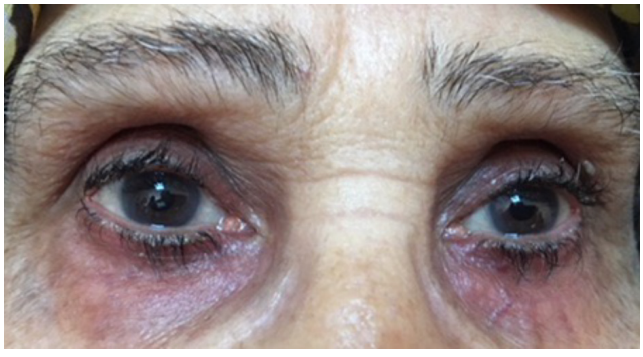

Figure 2. Female patient with significant prostaglandin-associated periorbitopathy secondary to prostaglandin analogue use. Deepening of the upper eyelid sulci, prominent periorbital fat atrophy, and hyperpigmentation in the periorbital region are observed

PGA use also leads to certain changes in the lower eyelid.[10,12,13] On examination, the first symptom of PAP in the lower lid is periorbital fat pad loss, which is especially common in older patients.[13,18] In Hertel exophthalmometer measurements of PAP patients using bimatoprost, Kucukevcilioglu et al.[13] detected enophthalmos due to loss of the periorbital fat pads, but did not find the same results with other PGAs. Fat atrophy and enophthalmos as a result of unilateral bimatoprost use have also been demonstrated by magnetic resonance imaging.[18,19] The onset of DUES and orbital fat atrophy may occur immediately after PGA initiation, or it may occur after a year of treatment.[8,20]

Prostaglandins are known to be potent stimulators of melanogenesis.[21] The increased pigmentation in the eyelid skin, lashes, and iris resulting from PGA use has been attributed to its stimulation of melanogenesis in the lid skin.[2,21] Hyperchromatic changes in iris pigmentation appear to be more permanent than pigmentary changes in the periorbital skin or eyelashes.[2] In the literature, it is reported that eyelid pigmentation occurs least with the use of latanoprost (0-5.9%), while rates of 2.9-15.4% and 1.6-25.9% have been observed with travoprost and bimatoprost, respectively.[9]

PGA-associated hypertrichosis of the eyelid and surrounding skin with thickening and elongation of the eyelashes were described in a series of 43 cases using unilateral latanoprost.[22] It was later noted that the rate of these eyelid changes observed in different studies varied widely for all PGAs (0-77%).[9] All hair follicles in the body, including the eyelid hairs and eyelashes, go through repeated cycles of regression and growth. PGAs are thought to stimulate the transition to the anagen phase, which is the active growth phase of this cycle, thus leading to eyelash hypertrophy and increased number.[2] This hypothesis is also supported by a case in which hypertrichosis associated with travoprost was unexpectedly observed in a graft obtained from the inner surface of the upper arm due to basal cell carcinoma.[23] In addition to the eyelashes and lid, increased hair growth on the upper cheek has also been reported with travoprost use (Figure 3).[24]

DUES and other PAP findings were reported to regress within 1-12 months of PGA discontinuation.[2,3,16,19,24,25] In a series of 25 patients who initially received latanoprost and did not have DUES, Sakata et al.[26] reported that DUES occurred in 15 (60%) of the patients when they switched to bimatoprost for greater IOP reduction and completely resolved in 11 of 13 patients who subsequently switched back to latanoprost. In addition, in two different studies, it was reported that PIP findings such as DUES and periocular pigmentation decreased and patient satisfaction increased after treatment with omidenepag isopropyl, a selective prostaglandin-EP2 agonist, in patients who developed PAP while using conventional prostaglandin F2α analogue drugs.[27,28]

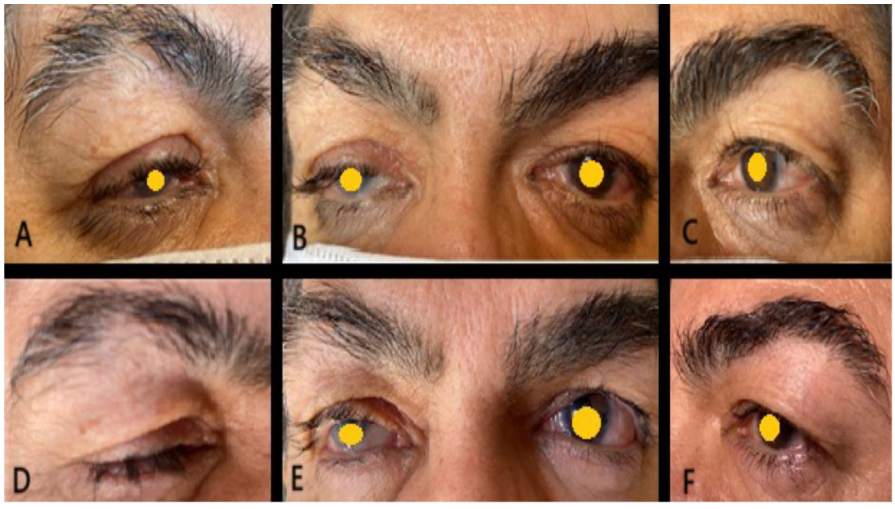

Figure 3. A patient using bilateral travoprost exhibits significant eyelash elongation and hypertrichosis in the periorbital region (A, B, C). Significant reduction in periorbital hypertrichosis was observed at 6 months after treatment discontinuation (D, E, F)

Punctum and Lacrimal System

Topical antiglaucoma drugs can cause inflammatory and fibrotic changes in the ocular surface.[29,30,31] These changes may be related to the active ingredients of the medications, as well as the preservatives used in commercial formulations or the duration of use.[29] The occurrence of similar inflammatory and fibrotic changes in the epithelial and subepithelial tissue of the lacrimal drainage system can lead to narrowing and occlusion of the lumen of the nasolacrimal system.[29,30] Obstruction associated with the use of topical antiglaucoma medications can occur in any part of the lacrimal drainage system.[30,32,33,34] Fourteen cases of punctal and canalicular stenosis were first reported in patients using long-term timolol maleate, betaxolol, and pilocarpine for the treatment of glaucoma, and were attributed to the cicatrizing effects of these agents resulting from the inflammatory changes they induce.[32] Seider et al.[33] determined that 23% of patients scheduled to undergo surgery for symptomatic nasolacrimal duct obstruction had a history of medical treatment for glaucoma. Quinn et al.[34] emphasized in a large-scale population-based study that the use of topical antiglaucoma agents increased the need for surgery to address stenosis in the punctum and other parts of the lacrimal canal. It was also noted that these patients had a higher frequency of entropion and trichiasis. Ulusoy et al.[35] also reported a relationship between the use of topical antiglaucoma agents and the prevalence of punctal stenosis. However, they provided no detailed information about the active ingredient or duration of use in their study.[35] Kashkouli et al.[30] found that punctal obstruction was more common in patients using fixed combination dorzolamide/timolol (26/130, 20%) compared to controls (24/280, 8.6%). Their study also showed that timolol/dorzolamide combination therapy had a more negative effect on the lacrimal duct system compared to monotherapy. Moreover, the authors noted a significantly higher incidence of upper canalicular system obstruction, which they suggested may be attributable to the closer proximity of the upper lacrimal system to the conjunctiva and the fornix, resulting in greater exposure to the inflammatory effects of topical drugs compared to the lower lacrimal system.[30] For this reason, the upper lacrimal system should also be examined in detail in glaucoma patients presenting with epiphora.[30]

Ectropion, Entropion, and Trichiasis

Prolonged use of topical antiglaucoma medications can cause entropion, ectropion, and trichiasis as a result of inflammation and cicatrization of the eyelid and ocular surface caused by both the active ingredients and the preservatives in these medications.[29,34,36,37] In this patient group, which is already predisposed to lid changes due to age-related involutional alterations, chronic antiglaucoma medication use and frequent scratching due to contact dermatitis caused by the drug increase lower lid laxity.[37,38] Cicatrizing changes in the eyelid also cause shortening of the anterior lamella of the lower lid, creating conditions suitable for ectropion.[29,34,36,37] Cases of entropion, ectropion, and trichiasis associated with different antiglaucoma agents have been reported in the literature. In the past, the development of drug-induced ectropion was attributed to agents that are no longer widely used, such as dipivefrin[39] and apraclonidine,[40] whereas cases of entropion, ectropion, and trichiasis are now frequently reported in association with dorzolamide,[29,38] brimonidine,[38,41] and timolol.[37] Altieri and Ferrari[42] compared lid changes between three different PGAs and a control group and indicated that lid laxity did not occur after two years of PGA use. However, there have been reports of entropion, ectropion, and trichiasis due to PGA use.[37,43,44] Among 644 eyes presenting for entropion or ectropion, Serbest Ceylanoğlu and Malkoç Şen[36] reported glaucoma in 2.2% (14 eyes total, 10 entropion/4 ectropion). Golan et al.[37] observed a higher rate (13.2%) in their study, but their inclusion of only lid malposition cases requiring surgery may explain the difference.

In addition, the high number of antiglaucoma medications (2.7 on average) used by these patients indicates the important role of these drugs in the development of lid malpositions.

Ectropion associated with topical drops is known to improve clinically after discontinuing the drug, thus reducing the need for surgery.[36,37] In contrast, entropion and trichiasis are more likely to require surgical correction.[34] Hegde et al.[38] reported regression after drug discontinuation in a series of 13 patients who developed ectropion due to antiglaucoma therapy. However, success rates are low for lid surgery performed without discontinuing or changing the topical antiglaucoma drop causing inflammation and allergy (Figure 4).[36,37,38] In multidrug regimens, inflammation and cicatrization may regress if the antiglaucoma medication causing lid malposition is identified and discontinued and treatment is continued with appropriate topical drops (preferably preservative-free antiglaucoma medications) and short-term topical steroids.[36,38] Alternative treatment options such as laser trabeculoplasty may also be considered.[34] However, patients should be informed that lid and glaucoma surgery may still be required.

Oculoplastic Problems Associated with the Surgical Treatment of Glaucoma

Upper Eyelid Ptosis

As with other anterior segment surgeries, ptosis is a possible complication of filtering or seton surgery that impacts patients' visual function and reduces their quality of life (Figure 5).[15,45,46,47,48,49] While some studies reported that the rate of ptosis development was higher in patients who underwent seton surgery with glaucoma drainage implant (GDI) compared to trabeculectomy and cataract surgery,[50,51] another study showed no significant difference in the frequency of ptosis after GDI surgery (13.7%) and trabeculectomy (10.5%).[52] In a study investigating the incidence of ptosis in patients who underwent trabeculectomy using an antimetabolite, the rate of ptosis at 6 months following surgery was reported as 19%.[45] In another study that included a 2-year follow-up period, 11 (6.7%) of 163 patients who underwent trabeculectomy developed ptosis, 9 of whom required surgical treatment.[46] Authors have emphasized that in cases where trabeculectomy and cataract surgery were combined, the incidence of ptosis was not higher than in patients who underwent cataract surgery alone.[15,47,48] Song et al.[47] stated that the development of ptosis was independent of whether trabeculectomy was performed before or after cataract surgery or combined with phacoemulsification, and was not affected by the size of the conjunctival flap or whether it was limbus- or fornix-based. Koh et al.[48] reported that in addition to bleb morphology and total bleb area, factors affecting the prevalence of postoperative ptosis included the type of anesthesia used during glaucoma surgery, the temporary suture placed in the limbus or upper rectus for eye fixation during surgery, and levator aponeurosis dehiscence resulting from the lids being held open by the speculum over a prolonged surgical time. Fukushima et al.[15] stated that the most important risk factor for ptosis after filtering surgery was the presence of DUES preoperatively, whereas the type of glaucoma, the number of glaucoma drugs used, or the need for postoperative needling were not significant in terms of ptosis development. On the other hand, some studies have emphasized that the risk of ptosis increases in patients who need postoperative needling, undergo external bleb massage, and have frequent eye itching due to ocular surface allergy caused by antiglaucoma agents and preservatives.[45,49]

Although ptosis may resolve spontaneously after glaucoma surgery, it is sometimes persistent. In a series of 339 eyes that underwent trabeculectomy, Malkoç Şen and Serbest Ceylanoğlu[49] reported transient ptosis in 30 eyes (8.8%) and persistent ptosis in 5 eyes (1.5%). Ptosis after glaucoma surgery is considered persistent if it lasts longer than 6 months, and surgical intervention can be planned accordingly.[3] Transient postoperative ptosis mostly occurs due to eyelid edema, hematoma, inflammation, or the effect of anesthetic agents on the oculomotor nerve branches and levator muscle.[3,53] Persistent ptosis usually occurs as a result of levator aponeurosis dehiscence. Age-related soft tissue and orbital fat atrophy and structural changes such as DUES occurring as a result of long-term PGA use before surgery are other contributing factors. Additionally, bleb needling, prolonged lid speculum and fixation suture use with extended surgical time, and a history of eye scratching due to antiglaucoma drug allergy may trigger levator aponeurosis dehiscence and pose a risk for the development of ptosis.[3,48,49,53,54]

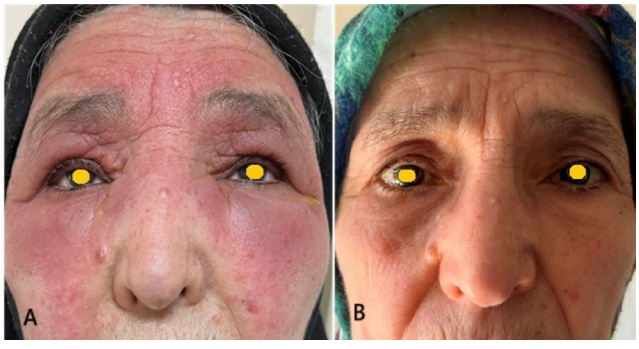

Figure 4. A) Allergic reaction spreading to the periorbital region and face following the use of brinzolamide/brimonidine tartrate fixed combination. B) Regression of the allergic findings was observed after discontinuing treatment

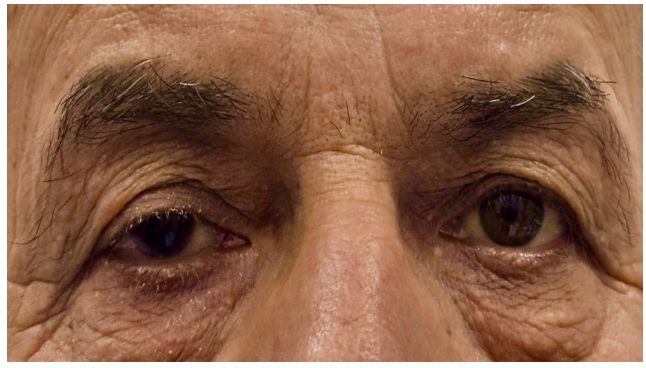

Figure 5. Ptosis following trabeculectomy surgery in the right eye. Marginal reflex distance 1 was 1 mm in the right eye and 4 mm in the left eye

When evaluating the visual field in patients with ptosis before or after surgery, it is important not to ignore the effect of the upper eyelid on the visual field.[3,4] For example, in a patient with an inferior arcuate visual field defect, concomitant ptosis or blepharochalasis may mimic a superior arcuate defect, inadvertently leading to a diagnosis of advanced glaucoma.[49] In such cases, repeating the visual field test while lifting the upper eyelid may demonstrate that ptosis surgery can significantly improve the patient's quality of life. Similarly, new ptosis developing after glaucoma surgery can create the false impression that disease progression continues postoperatively. Repeating the test after eliminating the eyelid's effect on the visual field will also provide guidance in terms of correct treatment management in these cases.

There are some important points to consider in the correction of persistent postoperative ptosis:

• The antimetabolites used during filtering surgery lead to a thinner and avascular bleb structure postoperatively.[55] Therefore, excessive correction should be avoided when planning ptosis surgery in these patients.[3] Otherwise, lagophthalmos may occur and the risk of serious complications such as blebitis and endophthalmitis will increase because the bleb is not adequately protected by the lid.

• Anterior or posterior conjunctival approaches may be preferred in the surgical treatment of ptosis after glaucoma surgery.[56,57,58] Song et al.[56] reported similar results with levator surgery via an anterior approach and Müller muscle resection via a conjunctival approach. Ben Simon et al.[57] stated that this method may be preferrable because there is less need for revision and more satisfactory cosmetic results with the conjunctival approach. However, when applying this technique, caution should be exercised during eyelid inversion to avoid potential iatrogenic traumas that may adversely affect bleb function.[3] The main purpose of surgical correction in patients who develop ptosis after filtering surgery should be to provide aesthetic and functional improvement without compromising bleb function.[3] In fact, Yunoki et al.[58] demonstrated that levator surgery performed via anterior approach in cases of new ptosis following trabeculectomy is completely safe in terms of filtration bleb function. In contrast, Putthirangsiwong et al.[59] stated that the conjunctivomullectomy method applied with the posterior approach may be an effective and safe option but reported that bleb failure occurred in 10.3% of patients with this method, emphasizing the need for caution.

Eyelid Retraction

Compared to ptosis, upper eyelid retraction following trabeculectomy is a very rare complication.[60,61] Therefore, all other neurogenic, myogenic, and mechanical causes that may cause retraction should be ruled out, especially thyroid orbitopathy.[60,61,62,63] First described by Putterman and Urist[64] in 1975, several cases have been subsequently reported by different authors.[60,61,62,63] Lid retraction may occur within 1 week after trabeculectomy[61] or after 20 years.[62] A large and cystic filtration bleb is an important risk factor for the development of lid retraction (Figure 6).[60] However, the fact that the retracted lid returns to its original position when pulled down and the development of unilateral lid retraction following bilateral trabeculectomy indicate that the pathogenesis cannot be explained by mechanical factors alone.[60,61,64] Nevertheless, Putterman and Urist[64] hypothesized that adrenergic substances in the aqueous lead to lid retraction by causing Müller muscle hyperactivation, while Awwad et al.[61] proposed that mitomycin C used during trabeculectomy has a toxic effect on the Müller muscle, leading to fibrosis in the long term. These mechanisms may help at least partially elucidate the pathogenesis of lid retraction in these patients.

The treatment approach to lid retraction following filtering surgery should be individualized according to the patient's symptoms.[60] Cases with mild retraction and bleb dysesthesia can be managed with artificial tears and bleb-related interventions. Mechanical closure at night, topical steroids, and sympatholytic agents may also alleviate retraction. However, these approaches provide symptomatic and temporary relief.[60] The "graded full-thickness anterior blepharotomy" method described by Elner et al.[65] can be implemented in these patients. Shue et al.[62] pointed out that for the surgical correction of lid retraction, methods such as Müller muscle surgery via posterior approach or "full-thickness anterior blepharotomy" may impair bleb function, as in ptosis surgery.[56,58] Aiming to reduce this risk, they modified these techniques and reported that a higher success rate with fewer complications could be achieved with the "conjunctiva-sparing anterior blepharotomy" method they described.[62] In a patient with lid retraction following trabeculectomy who already underwent transconjunctival mullerectomy, Vásquez and González-Candial[63] reported that filling with hyaluronic acid injection provided temporary anatomical and functional improvement and reduced the need for repeated surgery due to recurrent retraction. Clark et al.[66] used mathematical vector analysis to determine the forces affecting upper eyelid position in patients with blebs and showed that injection of botulinum toxin A into the upper lid inhibited retraction.

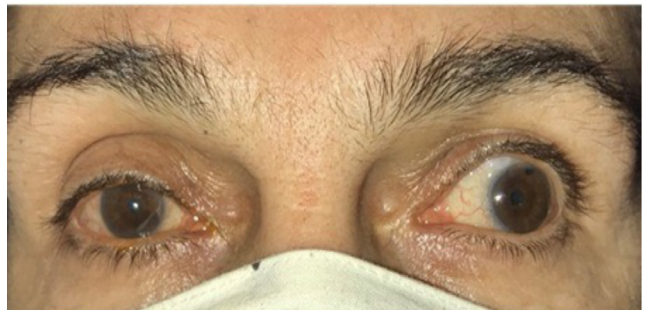

Figure 6. Female patient with mechanical upper lid retraction due to a large cystic bleb located in the superonasal region of the left eye, which also exhibits marked exotropia

Lacrimal Gland Changes Due to Glaucoma Drainage Implants

A GDI is usually implanted in the superotemporal region, near the lacrimal gland. In a study examining the effect of GDIs on lacrimal gland position with magnetic resonance imaging, lacrimal gland measurements in the unoperated orbit were similar to those of the normal population in terms of size and volume, whereas the lacrimal glands on the GDI side had significantly smaller volume, flatter morphology, and more posterior placement.[67] These findings may be directly due to the mechanical pressure caused by the GDI, as well as the gradual development of lacrimal gland atrophy. However, no significant relationship was found between lacrimal gland size and clinical symptoms of dry eye in patients with GDI.[67] Gobeka et al.[68] also reported that lacrimal gland volume was smaller in eyes with GDI compared to trabeculectomized eyes in their high-resolution computed tomography study. Additionally, they observed that lacrimal gland volume was lower in the eyes with GDI compared to unoperated side, as expected. However, lacrimal gland volume in the trabeculectomized eyes was surprisingly higher than in the unoperated eyes. The authors also emphasized that intraoperative mitomycin C application had no effect on lacrimal gland volume or size.[68]

Glaucoma Drainage Implant Exposure

One of the main complications of GDIs is exposure resulting from gradual erosion of the conjunctival tissue over the tube or implant plate, often due to inadequate or incorrect placement (Figure 7).[69,70] This undesirable situation can occur immediately after surgery or over the course of years, leading to serious infectious complications such as orbital cellulitis and endophthalmitis.[70] Tamçelik et al.[71] developed the "Tenon advancement and reproduction technique" to prevent GDI exposure. This technique can reduce the risk of implant exposure by performing it with a short scleral tunnel, as described by the authors,[71] or with a long scleral tunnel[72] or scleral flap.[73] The scleral tunnel technique has been reported to be more advantageous than the scleral flap in patients with GDIs.[74] In addition, various graft materials such as lyophilized pericardium, fascia lata, lyophilized sclera, dura mater, amniotic membrane, and cornea are also used to prevent GDI exposure.[70]

Phthisis Bulbi/Enophthalmos

In the long term, some complications that develop after glaucoma surgery may lead to irreversible ocular conditions such as phthisis bulbi and enophthalmos (Figure 8).

Evisceration and Prosthesis Requirement

Eyes that are completely blind and painful due to uncontrolled IOP despite using all available medical and surgical options for glaucoma treatment may require evisceration and a removable ocular prosthesis. The primary goal in these patients is to relieve chronic and severe ocular pain rather than aesthetic concerns.

Glaucomatous Conditions Following Oculoplasty Surgery

In addition to oculoplastic problems that may develop as a result of medical and surgical glaucoma treatment, there are also glaucomatous conditions that occur following oculoplastic surgery. This phenomenon is an important point that is often overlooked and warrants caution. For example, Osaki et al.[75] reported a statistically significant increase in IOP after upper lid blepharoplasty surgery. The results of their study indicate that the possible risks in terms of glaucoma after blepharoplasty must be carefully evaluated in glaucoma patients and glaucoma suspects. Publications in the literature about the development of acute angle closure following blepharoplasty are also noteworthy.[76,77,78] A common feature of these cases is reports of pupil dilation after surgery. In fact, a study conducted by Koçer and Sen[79] with automatic pupillometry in patients who underwent blepharoplasty surgeries demonstrated significant changes in static and dynamic pupil measurements postoperatively. Although there is still uncertainty regarding the role of other factors that contribute to pupil dilation, such as anxiety, pain, postoperative eye closure, or the pharmacological effects of anesthetic agents, the risk of angle closure after oculoplastic surgery is important.[79] A complete preoperative ophthalmological examination in which the anterior chamber is also evaluated is necessary for all patients undergoing oculoplastic surgery.

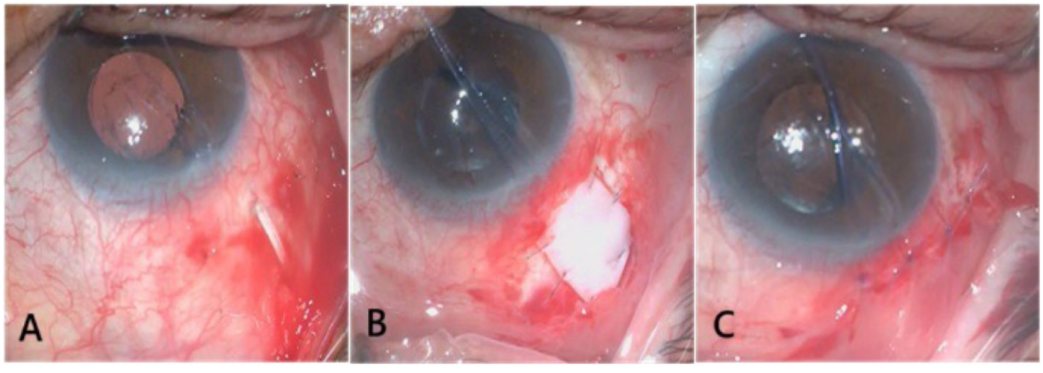

Figure 7. A) Conjunctival dehiscence and subsequent tube exposure in an eye with an Ahmed FP7 glaucoma valve. B) Lyophilized bovine pericardium was fixed over the exposed area using 10-0 nylon suture. C) The conjunctiva was closed primarily

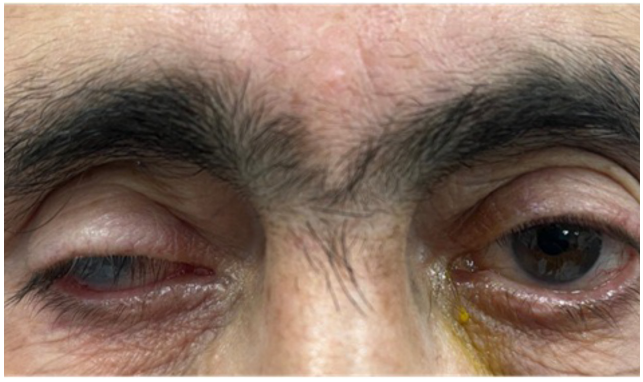

Figure 8. Enophthalmos of the right eye due to absolute glaucoma and phthisis bulbi

Appropriate Treatment of Oculoplastic Problems in Patients with Glaucoma

Beyond the oculoplastic complications associated with glaucoma treatment, it is also important to careful select treatment for glaucoma patients who already have oculoplastic problems. For example, knowing the patient's history of PGA use when planning blepharoplasty or ptosis surgery can be decisive in determining the surgical approach.[3,4] While PAP can be diagnosed more easily in patients using PGAs unilaterally, conditions such as DUES can be overlooked in patients with bilateral use. In such cases, DUES and lipoatrophy that may occur in the present or future due to PGA use should also be taken into account when planning blepharoplasty in addition to age-related periorbital volume loss. A more conservative approach is also recommended, as PGA treatment may accelerate orbital adipose tissue atrophy. In this context, it is prudent to protect the fat pads and minimize the skin excision compared to standard practices. Otherwise, there may be undesirable aesthetic consequences such as a sunken eye appearance after surgery.[3,4]

Concomitant glaucoma is among the important points for oculoplastic surgeons to consider in cases of eyelid malposition. With aging, the incidence of both glaucoma and eyelid malpositions such as ptosis, entropion, and ectropion increases.[3] Therefore, it is critical in treatment planning to distinguish whether lid changes in these patients are a result of age-related physiological mechanisms or a side effect of the topical glaucoma drugs used.[36] In addition, the mechanical effect exerted when instilling topical medication may exacerbate the existing horizontal and vertical lid laxity in these patients. Epiphora is another potential side effect that may cause skin irritation, chronic inflammation, and eventually scarring due to frequent eyelid wiping. Early recognition of side effects related to medical therapy in cases of eyelid malposition allows the timely termination of topical agents before irreversible fibrotic changes, thereby facilitating treatment without surgery and increasing success when surgery is required.

However, in glaucoma patients with ectropion, the oculoplastic surgeon may overlook this effect. If lid surgery is performed while topical drug treatment is ongoing, surgical failure and recurrence are inevitable because of persistent drug-induced inflammation.[4] Therefore, topical antiglaucoma drugs should be discontinued, inflammation should be controlled with low-potency steroid drops, and the desired IOP reduction should be managed with oral acetazolamide before surgical planning. In patients with severe allergic symptoms, oral antihistamines may be added to treatment. It should also be noted that these patients may need trabeculectomy.[4] In patients with entropion accompanied by glaucoma, the use of preserved topical drops in particular may increase corneal exposure and cause serious ocular surface diseases. Therefore, correcting the entropion with an appropriate surgical method and in a timely manner is essential to avoid interrupting glaucoma treatment.

As discussed above in the relevant section, the frequency of punctal obstruction was found to be higher in patients using dorzolamide/timolol fixed combination.[30,31] It would be a rational approach to avoid these drugs in patients who have developed punctal obstruction for any reason and undergone surgery for its correction. On the other hand, in patients with nasolacrimal duct obstruction and/or lacrimal sac abscess, the presence of a cystic, avascular bleb resulting from the use of antimetabolites during trabeculectomy may significantly increase the risk of blebitis and endophthalmitis. Therefore, the risk of infection should be carefully considered during surgical planning for such patients.

Conclusion

Oculoplastic problems and other complications that may occur due to medical and surgical treatment in glaucoma patients can affect eye health not only in terms of function, but also in terms of aesthetics and comfort. Awareness of these problems and careful management with consideration of risk factors are critical both in terms of medicolegal aspects and treatment success. During the glaucoma treatment process, regular oculoplastic evaluation and early diagnosis of possible complications contribute significantly to both vision and quality of life. It should be noted that glaucoma patients may present unique and challenging surgical conditions compared to other oculoplastic cases. When planning oculoplastic surgery in those who have undergone filtering surgery, the primary focus should be to not disrupt bleb function, and attention should be paid to the risks of blebitis and endophthalmitis in the presence of cystic bleb. It is important to increase the awareness of oculoplastics complications among clinicians planning glaucoma surgery and to determine multidisciplinary management strategies.

Ethics

Informed Consent: Patient consents have been obtained.

Declarations

Authorship Contributions

Surgical and Medical Practices: E.Ş., S.B., Concept: E.Ş., S.B., Design: S.B., K.S.C., Data Collection or Processing: E.Ş., S.B., Analysis or Interpretation: E.Ş., S.B., K.S.C., Literature Search: S.B., K.S.C., Writing: S.B., K.S.C.

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