Glaucoma: The Perspective of Oculoplastic Surgery

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ABSTRACT

Glaucoma is a leading cause of irreversible blindness in the world and follow-up is life-long in order to prevent and detect visual loss. Patients with glaucoma may encounter some oculoplastic conditions that might associate with decrement of functional ability or cosmetic problems. Detailed understanding and addressing the risk factors is essential to minimize complications after glaucoma surgery. An oculoplastic surgeon should be aware of the problems of a glaucoma patient, to get good cosmetic and functional results given its interference with the patient's visual field and quality of life.

Keywords: Glaucoma, Eyelid, Ptosis, Oculoplastic surgery.

INTRODUCTION

Glaucoma has become the most frequent cause of irreversible blindness worldwide.[1-3] The global prevalence of glaucoma was roughly 3-5% for people aged 40-80 years.[3] This chronic disease requires life-long monitoring as well as treatment with topical medications, laser, or surgery. Each of the treatment models have its own effects on intraocular pressure, and ocular and adnexal complications as well. A patient with glaucoma may encounter many problems including periorbital changes of lipodystrophy, eyelid malpositions, periocular dermatitis, and lacrimal drainage system pathologies.

In this text, the effect of the disease and treatment strategies on the adnexal structures will be discussed from perspective of oculoplastic surgery.

1. TOPICAL GLAUCOMA MEDICATION AND ITS ADNEXAL EFFECTS

1.1. Prostaglandin-associated Periorbitopathy

Patients with glaucoma are usually prescribed topical medications. Several categories of topical drugs for management of glaucoma are present in the market. In general, prostaglandin analogues which lower intraocular pressure by improving uveoscleral outflow, are the first-line medical treatment. The main advantage of this group of drugs is their dosing schedule as using once daily can provide a significant decrease in intraocular pressure. However, they come with local side-effects including elongation and darkening of eyelashes, loss of orbital fat (prostaglandin-associated periorbitopathy) with resulting enophthalmos, and periocular skin pigmentation.

Peplinski and Smith described these findings first in 2004 as deepening of the upper eyelid sulcus (DUES) in patients on bimatoprost.[4] Today, periocular changes are also encountered with other PG analogues.[5] Prostaglandin-associated periorbitopathy (PAP), has been proposed to describe the periocular changes associated with topical PGA therapy and includes DUES, upper eyelid ptosis/retraction, loss of inferior orbital fat pads, and enophthalmos, eyelid pigmentation, and eyelash changes.[6-9] The incidence of DUES who were treated with bimatoprost and travoprost eyedrops was reported over 50% of the patients experiencing periocular changes.[10-11] Bimatoprost has been found to have the highest associations in several studies [8,12] There are different mechanisms that have been hypothesized to explain the presence of DUES (**Fig. 1**). First, the chronic use of eye drops with a mechanical insult to the eyelid causing levator dehiscence or reduction in collagens leading to Muller's muscle degeneration may explain deepening of the eyelid sulcus and upper lid ptosis.[13] Second, the induction of apoptosis of orbital fibroblasts

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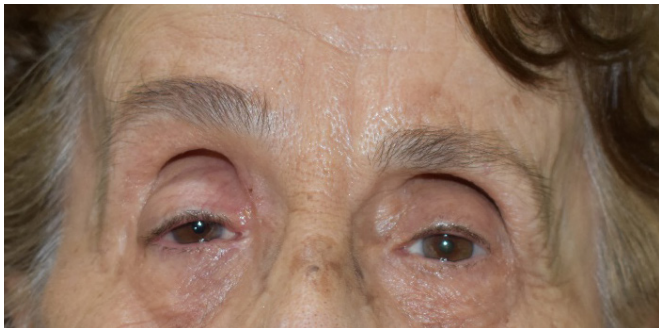

Figure 1: *A 82-year-old woman treated with prostaglandin analogue. Note the bilateral deepening of the superior eyelid sulcus and upper eyelid ptosis.*

with a remodeling of the extracellular matrix was proposed to decrease the volume of eyelid fat pads.[14] This hypothesis is further supported by in-vitro studies that reported the suppression of adipogenesis with PGA in knockout mice.[15] The mechanism of increased pigmentation in both the eyelids and iris appears to be due to the stimulation of melanogenesis in melanocytes.[16] Finally, hypertrichosis has been reported with PGA use.[17-18] Even though this was first evaluated as a side effect dermatological use of these molecules has also been studied.[19] The mechanism of hypertrichosis has been linked to the ability of PGA to prolong anagen in resting hair follicles while inducing hypertrophic changes in the involved follicles. Previous case reports have noted that these PAP changes may be reversible, ranging from 1 month to 6 months after discontinuation of PGA.[20]

Other anti-glaucomatous drugs are β adrenergic blockers (eg, timolol, betaxolol), topical carbonic anhydrase inhibitors (eg, dorzolamide, brinzolamide), α adrenergic agonists (eg, brimonidine), and miotics (eg, pilocarpine). They have different mechanisms for the decrease of intraocular pressure and may show systemic side effects systemic side-effects including bradycardia, arrhythmias, a drop in blood pressure, reduced libido, and increased obstructive bronchial problems that can lead to an asthmatic attack more significantly seen with beta-blockers. However, adnexal side effects are usually encountered due to topical exposure of the main agent or preservatives used in the drug.

A preservative is an agent that extends the shelf-life of a drug with its bacteriostatic or sterilizing properties. Most preservatives also have surfactant activity which destabilizes cell membranes. This causes destruction of the cell membrane, inhibition of cell growth, and reduction of cell adhesiveness, mainly used for bacterial membranes. However, preservatives also show these effects on normal corneal and conjunctive cells, resulting in ocular surface and adnexal disorders. These include superficial punctate keratitis, corneal erosion, conjunctival allergy, conjunctival inflammation, punctal stenosis, eyelid skin inflammation. Patients using antiglaucoma eye drops usually have lower Schirmer's test scores and reduced tear break-up times that may be the cause of ocular surface disorders, however punctal stenosis, eyelid malpositions may also end with occasional or constant epiphora. In this text, we will evaluate punctal stenosis and eyelid malpositions. [21-23]

1.2. Punctal Stenosis and Obliteration

Acquired punctal stenosis related to topical medication can be isolated or associated with canalicular stenosis, nasolacrimal duct obstruction, eyelid laxity, or malposition (**Fig. 2**). The incidence and prevalence are still unknown because of the lack of large population-based studies; however lower punctal stenosis appears to be more frequent than upper punctal stenosis The level of punctal stenosis can be visually graded with slit -lamp examination.

Grading of Punctal Stenosis:[27]

Congenital absence of the punctum (atresia) has been defined as grade 0,

severe punctal stenosis as grade 1,

less severe punctal stenosis as grade 2,

a normal punctal opening as grade 3,

slit punctal opening of 2 mm and more as grade 5

Ocular surface infections by chlamydia, herpes simplex, actinomyces, and human papillomavirus may cause punctal stenosis.[28-29] Systemic medications like 5-fluorouracil, docetaxel, and paclitaxel, and topical agents like prednisolone acetate, dexamethasone, phenylephrine hydrochloride, adrenaline, antibiotics (chloramphenicol

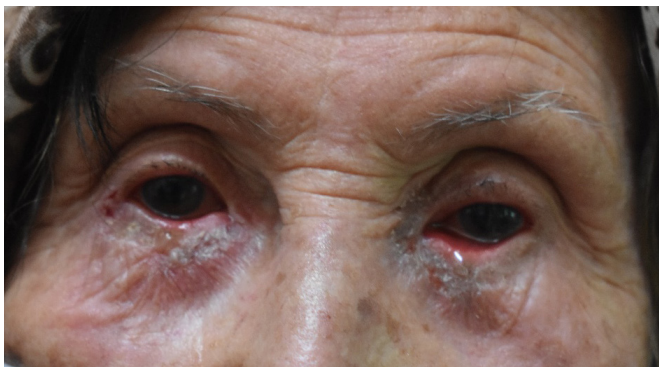

Figure 2: *A 85-year-old woman treated with prostaglandin analogue. Note the bilateral periocular dermatitis associated with punctal stenosis and left lower lid ectropion.*

and tobramycin), indomethacin, tropicamide have also been associated with punctal stenosis.[27, 30,31] Chronic usage of antiglaucoma agents like pilocarpine, beta-blockers, dorzolamide, prostaglandin analogues can result in histological findings, including conjunctival metaplasia, decrease of goblet cells, and increase of sub-conjunctival fibroblasts, macrophages, and other inflammatory components.[32] Eyelid malpositions like ectropion might also trigger the progression of punctal stenosis due to local inflammation and external punctum exposure and keratinization.

1.2.1. Management of Punctal Stenosis and Punctal Obliteration

The treatment of punctal stenosis aims to achieve an anatomically normal and functioning lacrimal punctum. This is usually provided with adequate size and a correct position with the globe and the tear lake. The most commonly used treatment options are perforated punctal plugs, bicanalicular stents, 1 snip to 4 snip punctoplasty with or without Mitomycin C.[33-34] Konuk et al.[35] retrospectively evaluated 44 eyes of 26 patients treated with dilation and placement of a perforated punctal plug for acquired punctal stenosis left in place for 2 months, and epiphora resolved in 84.1% of cases. Another study proposed the use of mini-Monoka stents for punctal and canalicular stenosis. The functional success rate was favorable but the reported mean follow-up of 6 weeks was very short.[36]

Currently, two-snip procedure with removing a V-shape wedge from the vertical portion of the canaliculus on the conjunctival surface, and 3-snip punctoplasty, consisting of two vertical cuts at either side of the vertical canaliculus and one horizontal cut at the base, are common procedures performed in punctal stenosis. A retrospective study on 169 reported an anatomical success rate of 91% and 94% using 2-snip and 3-snip procedures; interestingly, the 2-snip procedure showed significantly higher functional success.[37] In another punctal surgery, Singh et al. [38] showed good anatomical and functional outcomes with punctal dilatation and Mini-Monoka stent. This technique can be used as an effective alternative to punctoplasty as it is minimally invasive and preserves the punctal anatomy Maluf et al.[39] used intraoperative topical Mitomycin C combined with posterior punctectomy and achieved an anatomical and functional success rate of 100% and 96%, respectively in 1 year follow up.

2. EYELID MALPOSITIONS

2.1. Ptosis

A patient with glaucoma may be presented with blepharoptosis which can decrease the visual quality of the patient by inducing refractive errors and blocking the visual field. It may also complicate intraocular pressure monitoring and performing accurate visual field tests which have an important role in a follow-up of a glaucoma patient.

Other factors beyond age-related changes contribute to the development of involutional ptosis in these patients.

Upper eyelid ptosis can result in obstruction of the peripheral vision and cause with-the-rule corneal astigmatism.[40] Clinicians investigating for poor vision in glaucoma patients should therefore keep in mind astigmatic and corneal topographic changes as a cause.

Patients with ptosis often require additional manual eyelid elevation during tonometry potentially putting pressure on the globe and falsely elevating IOP as well.

Studies on the effects of particular ophthalmic surgeries in the development of involutional ptosis are well documented and it is well acknowledged that anterior segment surgery is a significant risk factor for the development of ptosis. Postoperative ptosis has been defined as a 2 mm decrease in palpebral fissure or margin reflex distance-1 (MRD-1) after surgery in the presence of good levator function and high lid crease (**Fig. 3**).[41] It can be classified as transient in case of lasting shorter than 6 months, on the other hand when it lasts for more than 6 months it is classified as persistent ptosis. The incidence of postoperative ptosis has been reported to be between 10-44% with different study designs, criteria, and surgical techniques.[42] A recent meta-analysis had demonstrated that the incidence of ptosis following ocular surgery was 11.4% and occurs at a higher frequency following glaucoma surgery than that following other ocular surgery.[43] According to the previously reported publications on the ptosis after glaucoma surgery which has at least 6 months a follow-up duration time, the incidence of persistent ptosis after trabeculectomy ranged from 12.5 to 19%. Transient ptosis was observed in 18-32% of the

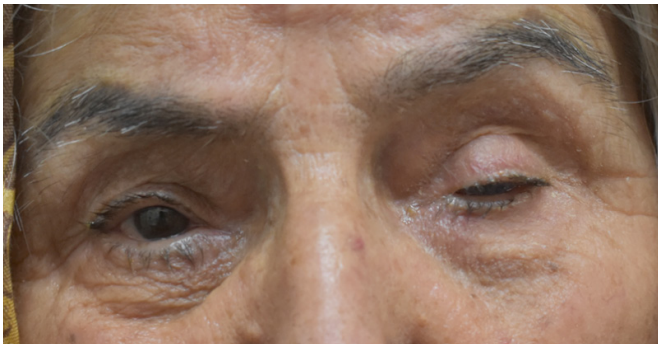

Figure 3: *Left upper eyelid ptosis of a patient with previous glaucoma surgery.*

patients after glaucoma drainage device implantation and persistent ptosis was reported to be 10.8%.[44-46]

There are many possible theories focused on explaining the development of postoperative ptosis. Myogenic, neurogenic, mechanical, and aponeurotic factors were attributed.[47] Trauma to the levator aponeurosis and scarring of the levator complex are considered to be the main reasons for ptosis following ocular surgeries. Bridle suture traction on the superior rectus muscle, eyelid speculum, and globe inferior traction might be the potential maneuvers leading to the dehiscence.[48] Some of the proposed mechanisms behind the cause of ptosis after surgery include lid edema from the locally administered anesthetic, initial myotoxic effects, and the compression of the upper eyelid against the orbital bones from the eyelid speculum reducing blood flow to the levator muscle contributing to the edema. Park et al. investigated whether shunting procedures, duration of surgery, or use of steroids contributed to ptosis after surgery. [44] The use of mitomycin C in glaucoma surgery also increases this risk. Naruo-tscuchiaska et al. have shown that the rates of ptosis after trabeculectomy were 10.7%-12% without mitomycin C (MMC) and as high as 19% with MMC 6 months after surgery. Contrary to the findings of Naruo-tscuchiaska et al, Park et al. did not show statistically significant rates for ptosis with the use of MMC.[44,49]

In a study which contributing factors to ptosis after glaucoma surhery was evaluated, ptosis was seen in 10.3% of those with filtering surgery and 22.5% in those with shunting procedures. [44] There was a statistically significant association between shunting surgery being more common than filtering surgery in patients with ptosis without a significant difference in the percentage of shunting or filtering surgery across all our age categories. There was also a statistically significant association between ptosis and shunting surgery, regardless of whether the patient had prior surgery.

The potential cause of increased ptosis in patients with shunting surgery could be due to the need for increased exposure required during shunting surgery to place a glaucoma drainage device 8-10 mm posterior to the limbus. The need for increased exposure possibly resulted in more pressure on the levator palpebrae aponeurosis by the lid speculum compared with the fornix-based filtering surgery.

Ptosis and the type of lid speculum have been evaluated and believed to play a greater role in patients with smaller palpebral fissures. Crosby et al. noted that a more rigid speculum can cause greater compression and the greatest risk of eyelid malposition involving those with the smallest palpebral apertures.[50]

Even if the patient's intraocular pressure remains controlled after surgery, the quality of life may be limited by postoperative ptosis. Blepharoptosis may interfere with accurate visual field assessment, especially of superior total deviation. Visual field assessments of glaucoma patients need to be as accurate as possible because they are involved in the assessment of glaucoma progression and IOP treatment strategies.

The diagnosis and treatment of blepharoptosis in glaucoma patients may be important for accurate assessment of visual field progression and improvement of quality of life. Visual field assessments of glaucoma patients need to be as accurate as possible because they are involved in the assessment of glaucoma progression and IOP treatment strategies.

Acquiring a reliable visual field is particularly important in glaucoma patients with ptosis, as ptosis may affect upper hemifield test results and contribute to a spurious interpretation that visual function has deteriorated.

In their study, Taniguchi et al examined parameters of the visual field test in glaucoma patients with blepharoptosis before and after blepharoptosis.[51] They have shown that margin reflex distance 1 (MRD-1) and the superior TD values were significantly improved after blepharoptosis surgery whereas there were no significant differences in best-corrected visual acuity, intraocular pressure, mean deviation, and pattern standard deviation values. If there is worsening of the visual field in glaucoma despite well-controlled IOP, blepharoptosis may be the reason. Therefore, if progressive visual field defects are observed, the influence of blepharoptosis should be considered. In the follow-up of glaucoma patients, it is necessary to actively perform blepharoptosis surgery if there is a possibility that blepharoptosis is affecting visual field testing results, not to mention the patient's quality of life. When performing the visual field tests in eyes with ptosis or blepharochalasis, it has been common practice to tape the ptotic eyelid to the skin at the level of the eyebrows. This traditional way of elevating the ptotic eyelid may be adequate for most patients.

1.1.1. Ptosis surgery in patients with glaucoma

The choice of ptosis surgery technique is arguably an important question to be addressed. Different surgical techniques are available for correction and depending on treatment goals the appropriate technique is chosen.

Both anterior (external) and posterior approaches are effective and safe techniques for ptosis treatment. The external levator approach is performed through an anterior approach which provides effective results without any

manipulation of the conjunctiva. A conjunctival incision, eversion of the eyelid, and sutures for closure of the conjunctiva may have a potential risk of mechanical damage to bleb which makes external approach preferable and more convenient in patients with previous glaucoma surgery. However, this surgery is more prone to over-elevation and poor eyelid closure.[52]

A more systematic analysis is required for comparing 2 approaches specific to patients with previous glaucoma surgery. The results of the previous studies focused on comparing external levator advancement and Muller muscle conjunctiva resection for correction of upper eyelid involutional ptosis may not be adopted to the patients with filtering blebs because the aim of the ptosis surgery in these patients may differ from traditional involutional ptosis surgeries. The surgeon should plan a more conservative surgery considering the presence of filtering bleb or possible glaucoma filtration surgery. It is important to remember that, in some patients with filtering blebs, undercorrection of their ptosis may be the ideal outcome. Overcorrection can lead to bleb exposure and subsequent complications of bleb failure, blebitis, and possible endophthalmitis. If the bleb is particularly thin or rests on the superior aspect of the cornea, the desired outcome may be a subtotal ptosis correction with the eyelid resting slightly lower than its anatomically correct position, approximately 2 mm below the limbus.

Some oculofacial plastic surgeons feel MMCR provides a more predictable eyelid height and better eyelid contour compared with an external levator advancement. [53] On the other hand it has been widely accepted that the presence of a filtering bleb or a prior history of corneal disease or corneal surgery makes Muller muscle conjunctiva resection (MMCR) is relatively contraindicated. The main problem is that the sutures in the palpebral conjunctiva can cause erosion of the filtering bleb. Also in the case of glaucoma filtering blebs, one could consider overcorrection and exposure problems to be a significant risk to the maintenance of a glaucoma filtering bleb and cause a poor aesthetic result. This theoretical benefit to MMCR surgery, in conjunction with the safety and efficacy data from this and other studies, argues for the utility of MMCR as a primary surgical approach for appropriately selected cases of ptosis after glaucoma surgery. [54-55]

There has been less previous evidence for postoperative bleb-related complications or bleb failure. Seminal contributions have been made by Putthirangsiwong et al. and they reported the overall bleb failure rate of 10.3% in the patients who underwent MMCR surgery after glaucoma filtering surgery which was similar to that noted in the Tube Versus Trabeculectomy (TVT) study. [56]

Michels et al. aimed to determine whether any bleb- or cornea-related complications in high-risk patients who underwent MMCR. In their study, no patient with a prior history of a filtering procedure had neither overcorrection of their ptosis nor bleb-related complications.[57]

Care should be taken to avoid damaging the accessory lacrimal glands in the MMCR surgery in patients with glaucoma. These patients may already have ocular surface problems because of the topical medications and any damage to accessory lacrimal glands may aggravate dry eye.

Scar formation following eyelid surgery, in general, can be attributed to several predisposing factors such as previous radiation or chemical burns, race, an autoimmune patient profile, and systemic or topical medications.[58]

Among topical medications, the most commonly linked with the development of progressive

cicatrizing conjunctivitis or pseudopemphigoid are long-term topical intraocular pressure-lowering medications.[59] Lee et al. reported 3 cases who were on multiple anti-glaucoma medications and developed conjunctival scarring leading to symblepharon and diplopia following Mueller's muscle conjunctival resection (MMCR).[60] In patients treated with multiple antiglaucoma medications who require ptosis repair surgery, consideration should be given to an external approach to avoid the potential for

postoperative symblepharon and diplopia. However, Bae et al. drifted from one point of view to another by reporting a case presented with glaucoma drainage implant erosion after bilateral ptosis repair. The authors concluded that caution and conservative lid elevation may be warranted when performing ptosis repair in patients with a glaucoma drainage implant.[61]

2.2. Eyelid Retraction

In addition to ptosis, glaucoma filtration surgery is a noticed cause of upper eyelid retraction. Its incidence is lower than that of blepharoptosis, with approximately 20 cases reported in the literature thus far. Lid retraction following glaucoma filtering surgery was first described by Putterman and Urist in 1975. [63] Putterman and Urist theorized the presence of an unknown sympathomimetic agent in the aqueous that stimulates the Müller's muscle. They described 2 cases that improved after a müllerectomy in one case and cautery-induced fibrosis to the upper bulbar conjunctiva in the other so it would block the supposed agent migration. However, there is no evidence of the existence of this adrenergic substance in aqueous humor. A recent study has shown that eyelid retraction was present in

8% of patients who underwent unilateral glaucoma surgery whereas ptosis was seen in 11% of patients.[64]

It should only be considered a diagnosis of exclusion over more common causes of retraction such as thyroid eye disease, trauma and enophthalmos with globus, midbrain disease, contralateral ptosis, cutaneous scarring, and topical sympathomimetic drugs. Three pathophysiologic theories based on chemical, myogenic and mechanical factors have been postulated. The chemical hypothesis theorizes that there might be a sympathomimetic agent in the aqueous that would stimulate Müller's muscle and cause an overaction. The myogenic theory defends that Müller's muscle is fibrous and retracted due to inflammation. And finally, the mechanical theory suggests that a prominent bleb would mechanically lift the eyelid. This argument was further supported by the case series reported by Shue et al. demonstrated retracted eyelids assuming the contours of the bleb. However, Putterman, Urist, and Awwad argued against the mechanical etiology by demonstrating immediate retraction of the eyelid on release after pulling the eyelid down over the bleb. Moreover, Awwad suggested a myogenic cause which Müller's muscle fibrosis was a possible cause.[52, 63-70]

A medical cause of bilateral upper eyelid retraction in a glaucoma patient was reported by Kazunami et al. in 2012. Topical bimatoprost 0.03% was administered to a 69-year old woman with bilateral normal-tension glaucoma and right upper eyelid retraction occurred 1 week after the initiation of the therapy and left upper eyelid retraction occurred 63 days after starting treatment on the left side. The upper eyelid retraction was reversed 1 week after switching therapy to once-daily latanoprost 0.005%. It seems that eyelid retraction could present any time after a trabeculectomy, as soon as 1 week per Awwad et al. and up to decades later as in the case reported by Shue et al. [65,67]

Management of the upper eyelid retraction needs to be tailored to the patient's problems and can be divided into conservative, medical or surgical.[72] Conservative measures such as artificial tears and lid taping are suitable for mild cases. Medical options have included steroids and sympatholytics. The anti-inflammatory effects of steroids have been used to reduce orbital inflammation and reduce lid retraction but are not effective in longstanding disease and are associated with complications. Conjunctiva-sparing levator/muller recession, full-thickness anterior blepharotomy, full-thickness transconjunctival mullerectomy, posterior levator disinsertion, are surgical options for trabeculectomy bleb-induced upper eyelid retraction. A large number of techniques reflects the fact that none is entirely satisfactory in terms of consistency, reproducibility, and obtaining desired post-operative eyelid height.

Conjunctival-sparing surgical techniques minimize the risk of damaging the blebs whereas full-thickness methods carry increased risks associated with damage to conjunctiva glaucoma filtering bleb. Full-thickness anterior blepharotomy is considered an effective procedure whereas postoperative eyelid crease recession, asymmetry greater than 10 mm, and contour abnormalities were reported.[73] Some authors further modified full-thickness methods with an anterior approach by the preservation of a central pedicle of conjunctiva and Muller's muscle to achieve a natural eyelid contour.[74]

To sum up, upper eyelid retraction is a rare complication that can occur after glaucoma filtering surgery. The most plausible causes are Müller muscle fibrosis and Müller muscle overaction associated with a diffuse hyperfiltering bleb. Mechanical hindrance and early Graves disease, however, should be considered. MMC appears to be a facilitator rather than the sole perpetrator of the overall process. We hope that we have shed more light on this under-reported complication. It is essential to look for changes in eyelid level after glaucoma filtering surgery and to better understand the possible etiologic factors.

3. PSEUDOEXFOLIATION SYNDROME

Pseudoexfoliation syndrome (PEX) is characterized by the deposition of proteinaceous material in the anterior ocular segment and also by several systemic manifestations. This disease is now recognized as a systemic disease with ocular manifestations. The involvement of peri-ocular tissues in PEX, including the eyelid skin, lacrimal gland, conjunctiva, orbital fat, and vessels, as well as the optic nerve, has been reported by several previous studies. The peri-ocular effects of PEX include the development of eyelid laxity, conjunctival chalasis, tear film abnormalities, pronounced orbital fat atrophy in response to the administration of prostaglandin analogues in pseudoexfoliative glaucoma, deficient orbital vascular supply, and biomechanical changes in both the eyeball and the optic nerve.

Pseudoexfoliative material or associated abnormalities have been detected in several extra-ocular sites, such as visceral organs, vascular tissue, and the brain.

Previous studies have reported the detection of PEX material in biopsies from eyelid skin and the accumulation of pseudoexfoliative material in the peri-ocular connective tissues including the medial and lateral canthal tendons, tarsal plates, and orbicularis oculi muscle. [76,77] A study has reported that PEX may be associated with atonic changes

of the orbicularis oculi and compromise the stability of medial and lateral canthal tendons, resulting in horizontal and vertical lid laxity and predisposing to the development of eyelid margin malpositions, such as entropion or ectropion.

The presence of PEX material in the orbits has been reported by several previous studies. Apart from the detection of pseudoexfoliative material in the orbital soft tissues, the effect of PEX is particularly pronounced in the orbital vasculature. Eyelid laxity, often resulting in changes of the eyelid margin position such as entropion or ectropion, as well as in lacrimal deficiencies, such as DED or OSD, may also be a source of both intra-operative and post-operative complications in cataract or other forms of intraocular surgery in the presence of PEX. Such changes may be more detrimental to the ocular surface, taking into account the reported mechanical corneal sensitivity defects in PEX.[78]

Salvá-Palomeque et al. has shown an association between PXG and ptosis. 54.5% of ptosis were present in PXG and 35% of patients with PXG showed ptosis after filtering surgery. Being pseudoexfoliation syndrome is a systemic condition, condition, we hypothesize that pseudoexfoliation material could accumulate among the fibers of either the levator aponeurosis, weakening them and rendering the eyelid more prone to drooping. This is in line with several reports which have shown accumulation of pseudoexfoliative material in extraocular organs (including liver, lungs, and heart), especially in areas rich in elastic fibers.[79-80] The presence of pseudoexfoliation has shown a significant risk factor for this complication. Further studies with a larger number of patients, a prospective design as well as possible correlations with other factors would be necessary to reach more definitive conclusions.

4. CONCLUSION

The worldwide incidence of glaucoma is increasing. These patients may also have oculoplastic issues, which may be age-related or secondary to medical or surgical glaucoma treatment. Patients who underwent glaucoma surgery are more likely to have postoperative ptosis than patients. High ptosis rates in patients undergoing glaucoma surgery may warrant preoperative counseling and post-operative referral to oculoplastic surgeons.

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