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Intraocular pressure elevation associated with blood in Schlemm's canal after strabismus surgery

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ABSTRACT

Purpose: To report a case of increased intraocular pressure (IOP) associated with blood in Schlemm's canal following strabismus surgery.

Observations: A 43-year-old female presented with acquired comitant esotropia. The patient had undergone an uneventful bilateral medial rectus recession and right lateral rectus resection operation under general anesthesia. Routine post-operative follow-up at day 3 detected a marked chemosis at the temporal side of the conjunctiva, an elevated IOP of 30 mmHg, and the presence of blood in Schlemm's canal in the temporal angle of the right eye. Episcleral venous outflow impairment was hypothesized to be the cause of secondary ocular hypertension in this patient. IOP was controlled with anti-glaucoma drops. Conjunctival chemosis, IOP, and blood in Schlemm's canal gradually decreased, and all topical medications were ceased at 11 weeks after the surgery.

Conclusion and importance: An IOP elevation can be an early complication after strabismus surgery. The presence of blood in Schlemm's canal suggests that the cause is impairment of episcleral venous flow. Although the episode can be transient, this report underlines the importance of IOP examination during the early post-operative period.

1. Introduction

Intraocular pressure (IOP) can change following strabismus surgery, and the literature has reported that IOP can change in both directions, an increase or decrease.[1–11] The postulated causes of IOP reduction includes a decrease of aqueous humor production due to anterior ciliary artery injury after rectus muscle detachment, and a release of globe pressure from rectus muscle repositioning.[1] Increased IOP in strabismus surgery may be attributed to globe rotation,[2] steroid-induced ocular hypertension,[4–6] and, occasionally, malignant glaucoma.[7] However, a sudden IOP elevation is rarely seen following strabismus surgery. In this report, we present a case with an increase of IOP associated with the presence of blood reflux into Schlemm's canal.

2. Case report

A 43-year-old female presented with gradual onset of binocular horizontal diplopia for 1 year. Her general condition was healthy with no underlying disease. She denied any past trauma but had a history of pterygium excision surgery on both of her eyes 10 years ago. Her best corrected-distance visual acuity was 20/20 in both eyes. An orthoptic evaluation revealed esotropia 55 prism diopters at both distant and near targets. Forced duction testing was negative. Slit lamp examination showed diffuse conjunctival scarring at the nasal bulbar conjunctiva of both eyes. Other examinations were unremarkable. Her baseline IOP was 15 mmHg in both eyes. Her cycloplegic refraction was plano in the right eye and −0.25 diopters in the left eye. Investigations for acquired strabismus had been conducted, which included MRI of the brain with orbits and blood tests for acetylcholine receptor antibody and thyroid function. All the workups were negative. The diagnosis was acquired comitant esotropia.

She underwent left medial rectus recession 6 mm, right medial rectus recession 6 mm on adjustable suture, and right lateral rectus resection 6 mm under general anesthesia. During the surgery, an extensive scar was found in both eyes, presumably from her past pterygium surgery, requiring a slightly greater amount of cauterization than

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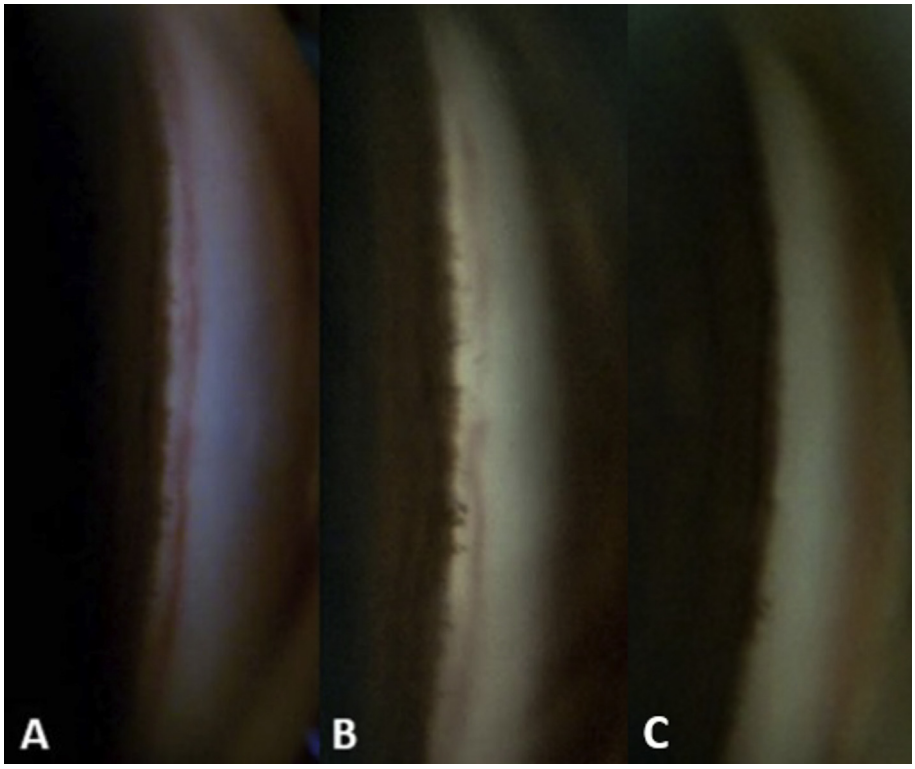

Fig. 1. Gonioscopic view of temporal angle (nasal mirror) in right eye (A) Blood in Schlemm's canal at day 3 post-operation (B) Decreased blood in Schlemm's canal at day 10 post-operation (C) Complete resolution of blood in Schlemm's canal at week 4 post-operation.

usual. On day 1 postoperatively, suture adjustment was done with no complication and the patient was orthotropic. The conjunctiva was mildly injected in both eyes. There was mild chemosis on the right eye. An IOP measurement was not performed on that day. No cells or flares in the anterior chamber were noted. The postoperative medications were a combination of tobramycin and dexamethasone ophthalmic suspension four times daily and preservative-free artificial tears every hour.

The patient came back for a scheduled routine follow-up at day 3 post-operation. She did not complain of any abnormal symptoms except for mild discomfort in her right eye. The visual acuity was 20/32 right eye and 20/20 left eye. The IOP was 30 mmHg in the right eye and 12 mmHg in the left eye. There was a marked temporal conjunctival chemosis with neither hematoma nor abnormally dilated vessels in the right eye. No proptosis was found by Hertel exophthalmometer. The gonioscopic examination revealed open angle with blood in Schlemm's canal at the temporal angle in the right eye (Fig. 1A). The pupil and anterior chamber were normal. The left eye was unremarkable. The cup-to-disc ratio was 0.4 in both eyes.

Episcleral venous obstruction was hypothesized to be the cause of secondary ocular hypertension in this patient. The patient was prescribed 0.05% latanoprost (Pfizer, New York, New York) at bedtime and a combination of 0.5% timolol and 2% dorzolamide (Santen Pharmaceutical Co. Ltd., Osaka, Japan) twice daily to the right eye. One week later, the IOP had decreased to 22 mmHg in the right eye. Conjunctival chemosis, and blood in Schlemm's canal were also decreased (Fig. 1B). All the medications were continued. By 1 month postoperatively, the IOP was 11 mmHg, with a complete resolution of conjunctival chemosis and blood in Schlemm's canal (Fig. 1C). The steroid eyedrops were discontinued at 6 weeks post-operation. The antiglaucoma medications were gradually tapered off and completely stopped at 11 weeks post-operation. At 3 months post-surgery, IOP remained controlled in the mid-teens in both eyes. No change of the optic disc was observed. The alternate prism cover test revealed orthophoria at both near and distance fixations. There were slight limits of adduction - to 40 prism diopters pass midline in the right eye and to 50 prism diopters pass midline in the right eye.

3. Discussion

The IOP is maintained by an equilibrium between production and drainage of aqueous humor. Regarding the conventional outflow pathway, the aqueous humor exits Schlemm's canals through collector channels and subsequently drains into the episcleral venous plexus through aqueous veins.[12] The pressure in the episcleral venous plexus determines the resistance of the post-trabecular meshwork outflow structure and, thus, is also another factor that regulates the IOP.

Steroid-induced ocular hypertension was one of the causes of elevated IOP following strabismus surgery. The mechanism is thought to be related to deposition of glycosaminoglycans in the trabecular meshwork. Topical steroids have been shown to produce a steroid response over a period of weeks. However, IOP elevation almost never occurs sooner than 5 days and rarely in less than 2 weeks of steroid treatment.[13] This case developed high IOP at day 3 post-operation which may be too early to be the steroid side effect. Rather, the presence of blood in Schlemm's canal suggested the pathology that is related to the pressure in the episcleral venous system.

Blood can enter Schlemm's canal when the IOP becomes lower relative to the episcleral venous pressure.[14] This can be caused by two main etiologies – (1) pathologies that lead to an increased resistance of episcleral venous flow and (2) pathologies that cause intraocular hypotony. Determining the manifested IOP is a straightforward approach, as the former usually shows high IOP, while the latter's IOP is often less than 6–8 mmHg. Our case presented with high IOP, so the initial differential diagnosis included conditions with increased episcleral venous pressure, such as carotid-cavernous sinus fistula, orbital arteriovenous fistula, and orbital congestion. The patient had previously received brain and ocular imaging as a part of her strabismus investigation. No preexisting abnormalities of the brain or orbit were detected. The clinical examination revealed no other clinical signs of increased

episcleral venous pressure, such as dilated or tortuous episcleral vessels. Moreover, the Schlemm's canal blood reflux was noted at only a single quadrant of the angle. These suggested that the pathology of episcleral venous obstruction in this case was likely to be confined to the local episcleral area rather than more distal venous drainage system which should affect a more extensive area with greater signs of venous back pressure.

Disruption of the episcleral venous flow, either by occlusion or compression, can increase IOP by increasing resistance in the post-trabecular meshwork outflow network. Several surgical procedures have been validated as successful methods for inducing ocular hypertension in animal models.[15–17] These techniques involved cauterizing or ligating 2–4 episcleral veins. The results showed significant increases in the IOP, with the onset ranging from immediately after the procedures to 5 weeks post-operation[16](Zhong L. ARVO 2019. Abstract 638-B0116). The degree and duration of IOP elevation correlated with the extent and number of veins being occluded.[16] It was estimated that cauterization of three vessel trunks reduced venous outflow up to 50%.[16] In eyes with three veins cauterized, the IOP elevation was stably maintained at least until 12 weeks (Zhong L. ARVO 2019. Abstract 638-B0116). It is interesting to note that, according to a study from Shareef et al., cauterizing a single episcleral vein in adult Wistar rats did not show any appreciable IOP change.[16]

In humans, the understanding of the patterns of aqueous veins and exact pathway of aqueous outflow distal to Schlemm's canal is still limited. A recent *in vivo* study in the human eye, which observed episcleral venous fluid waves in eyes that underwent the Trabectome procedure or iStent implantation, suggested that the distal outflow is largely segmented and limited circumferentially.[18] Their findings corroborated previous laboratory studies.[19,20] Given that the outflow is arranged in a segmental pattern, localized damage to episcleral vessels, hence, can give rise to backward pressure over the corresponding segment of Schlemm's canal. Thus, this supports the finding of confined blood in Schlemm's canal in our case.

Post-operative tissue swelling can cause transient compression of episcleral vessels. In addition, electrocauterization, which is routinely used to control bleeding during muscle surgery, can also impair the episcleral venous outflow. In our case, the degree of episcleral damage, either form the surgery itself or the cauterization, might have been significant enough to impair the episcleral venous outflow and subsequently induce the IOP increase. In this case, the episode of IOP elevation subsided within 11 weeks with the complete resolution of blood in Schlemm's canal. In animal models, a decrease in the IOP after an initial spike was also observed before the IOP reached a plateau.[15–17] The onset of an IOP decrease ranged from the first few days to 4 weeks after the procedure. The reduction was thought to be due to the development of venous collateral vessels.[17] Moreover, the cauterization during the surgery was limited to the anterior scleral area. Unlike the occlusion of the posteriorly located main venous trunks that were studied in the animal models, interference with the venous outflow over the anterior smaller plexus may cause a subtler IOP effect and allow easier venous re-anastomosis over time.

4. Conclusion

IOP elevation is an uncommon complication following strabismus surgery. Due to the presence of blood in Schlemm's canal in this case, the cause was postulated to be impairment of episcleral venous drainage from the surgery. Fortunately, in this patient, the episode of IOP elevation was transient and the IOP could be controlled with medication. However, this report underlines the importance of IOP examination during the early post-operative period, especially in those with preexisting glaucoma.

Patient consent

The patient consented to publication of the case in writing.

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Authorship

All authors attest that they meet the current ICMJE criteria for authorship.

Declaration of competing interest

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