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Transient Diagnostics and Therapeutic-Related Increase in Intraocular Pressure and Risk to the Glaucoma Patient

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Abstract: This review evaluates the impact of transient intraocular pressure (IOP) elevations during common ophthalmic surgical and diagnostic procedures on glaucoma patients. Elevated IOP is a key risk factor in glaucoma, and while transient IOP spikes are frequently encountered during surgeries like cataract extraction, laser in situ keratomileusis (LASIK), and femtosecond laser-assisted cataract surgery (FLACS), the clinical significance of these short-term elevations remains uncertain, particularly for eyes with compromised optic nerves. There is still a lack of data on which IOP level and duration of IOP insult the glaucoma damage occurs. Still, it is known that the combination of the degree of IOP elevation, duration of the insult and optic nerve susceptibility are important determinants of this event. While transient IOP elevations during these procedures are generally well tolerated, patients with advanced glaucoma and severely compromised optic nerves may be at greater risk for further damage. More research is needed to fully understand the long-term implications of acute IOP spikes, particularly in patients with advanced disease.

Keywords: intraocular pressure, glaucomatous damage, transient intraocular pressure rise

Introduction

Glaucoma is the leading cause of irreversible blindness worldwide, and intraocular pressure (IOP) is currently the main modifiable risk factor for this disease.[1,2] There are some theories behind the glaucoma damage mechanism and how IOP plays an important role in the pathophysiology of the disease. It is known that elevated IOP leads to an increased pressure gradient across the lamina cribrosa, causing compression and deformation of this structure, compromising axonal transport and damaging the retinal ganglion cell axons.[3,4] Previous studies have demonstrated that the impact of IOP rise in axonal transport changes occurs in a time-dependent fashion; thus, the duration of IOP insult is an important determinant of glaucoma damage.[5]

There are several situations where a transient IOP increase occurs; however, it is still unclear if this could cause direct damage to the ganglion cell axons or compromise optic nerve perfusion. Although this transient elevation in IOP appears well tolerated in healthy eyes, little is known about how eyes with glaucoma respond to this IOP insult and the time scale at which IOP-related damage occurs. Patients with a glaucomatous optic nerve are more susceptible to acute IOP elevation than patients with ocular hypertension or normal eyes and this is probably explained by the different biomechanical properties of the connective tissues with eyes having different levels of susceptibility to the same IOP insult.[6,7.] In glaucomatous eyes, the optic nerve head and lamina cribrosa exhibit greater susceptibility to IOP-related stress due to compromised connective tissue properties. The lamina cribrosa in glaucomatous eyes tends to be thinner, less resilient, and more prone to deformation in response to IOP elevation compared to healthy eyes.[6,7] These biomechanical vulnerabilities increase the risk of axonal compression and damage to retinal ganglion cell axons, even during transient IOP elevations. Additionally, glaucomatous eyes often have altered scleral rigidity and stiffness, which further influences the way the optic nerve head and surrounding structures respond to IOP stress.[6,7]

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In this review, we included studies that analyzed the IOP behaviour during surgical and diagnostic procedures. The objective of this review is to provide data on short-term IOP rise related to surgical and diagnostic procedures to understand whether this would be a real risk for glaucoma patients.

IOP Elevation During Surgical Procedures

Cataract surgery is the most common ophthalmic surgery performed in the world, and a significant percentage of these patients have glaucoma as a comorbidity.[8,9] During phacoemulsification, maintaining anterior chamber stability is crucial for surgical success, and this is done by keeping a high infusion pressure, resulting in transitory IOP elevations during the procedure.[10–12] A routine phacoemulsification procedure takes approximately 10 minutes, and up to 80% of the time IOP exceeds 60 mmHg.[11] Zhao et al showed an IOP rise of up to 96 mmHg during cataract surgery, with an IOP greater than 60 mmHg during cortical clean up and nucleus disassembly in an in vivo human study.[11] Despite these significant transient IOP elevations, there does not appear to be any conclusive evidence that this is harmful in the glaucoma patient with already compromised optic nerves. Several recent large clinical trials of phacoemulsification encompassing over 1000 patients with mild to moderate open angle glaucoma showed the procedure to be very well tolerated with no acute visual field progression postoperatively.[13–15]

A study evaluating VF changes after cataract extraction from patients from the Advanced Glaucoma Intervention Study (AGIS) showed significant mean deviation (MD) improvement after surgery in patients with mild and moderate glaucoma but no improvement in patients with advanced disease.[16] For this last group, indices of localized field loss (PSD and CSPD) mildly worsened after surgery. Still, the scotoma size and depth did not change significantly from a clinical standpoint.[16] Although glaucoma progression cannot be ruled out, the explanation for the mild decline in PSD and CPSD is that the opacity removal improved sensitivity in a few points resulting in greater unevenness when comparing improved sensitivity areas to areas with already existing defects.[16] In contrast to this previous study, Seol et al found MD improvement after cataract surgery in glaucoma patients with different levels of disease, including advanced glaucoma, with no significant changes in PSD.[17] Indeed, phacoemulsification is not contraindicated in glaucomatous eyes of all severity.

This transitory IOP elevation is also seen when creating a corneal flap for laser in situ keratomileusis (LASIK) treatment, with IOP getting as high as 90 mmHg for 10–30 seconds during the vacuum application period, as demonstrated in studies on porcine and cadaver eyes.[18–20] The first femtosecond platforms designed to create corneal flaps used a flat patient interface (PI) to induce corneal flattening to possibly create more planar flaps. Previous studies already showed a higher IOP rise during femtosecond treatment when using a flat patient interface compared to curved ones.[21,22] Bolivar et al showed, in enucleated porcine eyes, a mean IOP increase of 78mmHg and 108mmHg when using a flat patient interface (iFS 150 kHz model, Abbott Medical Optics, Inc). During the suctioning and cutting phases to create a corneal flap, respectively.[21] The IOP rise with a curved interface (Victus, Bausch & Lomb, Inc) was lower during the suction phase with a mean IOP elevation of 20 mmHg. This study also evaluated the IOP rise induced by another femtosecond platform (LenSx, Alcon Laboratories, Inc.), with a curved interface showing a less significant IOP elevation than the other two platforms. The corneal flap creation using a mechanical microkeratome induces higher IOP elevation than with femtosecond; however, it creates the corneal flap in half of the time compared to femtosecond.[22] Patients usually experience a complete blackout when microkeratome is used, which means the IOP rise is higher than the retinal arterial pressure. Chaurasia et al showed, in rabbit eyes, a mean IOP elevation of 62.25 vs 141.02mmHg ($p = 0.0009$) during the cutting phase in the femtosecond and microkeratome, respectively.[22]

Acute IOP elevation can induce transient changes in retinal nerve fiber layer (RNFL) thickness, though short-term IOP spikes, such as a 45-second rise to 100 mmHg during LASIK suction, have shown no significant impact on RNFL thickness in healthy human eyes.[23]

Studies in non-human primates have indicated that raising IOP to 45 mmHg for 60 minutes leads to a small reduction in RNFL thickness (1.4% overall, $P < 0.0001$), with more pronounced thinning (4.9%, $P = 0.001$) within 800 µm of the ONH.[24] These findings suggest that acute IOP elevation may cause minimal, temporary thinning of the RNFL but is unlikely to have long-term clinical effects when the pressure elevation is transient.[23,24]

There are a few case reports of visual field (VF) defects associated with LASIK, and the presence of ocular hypertension preoperatively or a family history of glaucoma appear to be risk factors for this condition.[25,26] A clinical trial evaluating visual field changes in non-glaucoma patients after LASIK found a decline in mean sensitivity (MS) between 15 and 30 degrees of the VF but no significant change in the MS in the central 15 degrees visual field.[27] Brown et al reported a case of VF changes following LASIK without optic nerve damage.[28] The VF defect was associated with irregular effective optic zones that caused loss of contrast in the midperipheral visual field. The visual field defects almost disappeared when pupil was constricted with brimonidine. Another study, including fifty-one patients that underwent myopic LASIK found no difference in visual field three months after the procedure.[29] A few studies associated the visual field changes following LASIK mainly with optical factors rather than RNFL damage.[27,28] Notwithstanding, there are cases in the literature where the VF defect was proved to be associated with ON damage and both causes should be considered in case of a VF defect following LASIK.[25,26] Per FDA recommendations, uncontrolled glaucoma is a contraindication for LASIK, and patients with controlled glaucoma/ocular hypertension or glaucoma suspect should consider not having the procedure. Despite the paucity of data, this seems reasonable considering the high magnitude of IOP rise associated to this procedure and the challenges to accurately follow IOP post-LASIK.

Transient IOP elevation has also been reported in femtosecond-laser-assisted cataract surgery (FLACS). Cataract surgery patients are usually older, different from LASIK patients, and more likely to have additional comorbidities that could leave them more susceptible to complications when exposed to IOP elevation. Previous studies have already shown that the IOP rise associated with FLACS is less significant than with others docking systems used for refractive surgery.[18,30] Kerr et al found, in an in vivo human study, a mean IOP of 28.9± 3.2mmHg with the Liquid Optics Interface during the vacuum phase of FLACS, with a further increase in IOP (mean IOP 36.0± 4.4mmHg) after laser capsulotomy and lens fragmentation.[30] No patients reported amaurosis during the procedure. The magnitude of IOP elevation also depends on whether a liquid-filled or rigid direct-contact docking system is being used, with the first inducing considerably less IOP elevation than the second one.[30,31,] Most studies evaluating IOP behaviour during femtosecond treatment include only healthy eyes, excluding glaucomatous eyes and ocular hypertension.

Darian-Smith et al showed a slight, but statistically significant higher IOP rise during FLACS in glaucomatous eyes compared to controls (mean IOP 34mmHg vs 29 mmHg, p=0.005).[32] The IOP 2 minutes after the procedure decreased but remained above baseline in both groups, with a higher level in the glaucomatous eyes (mean IOP 30.2 vs 27.6 mmHg, p= 0.03). There was no association between glaucoma severity and IOP increase during this study. In contrast, a study by Alvarez-Ascencio et al showed no remaining IOP rise immediately after suction docking in glaucomatous eyes, with IOP levels slightly lower than baseline.[33] A visual field (VF) analysis was also done in this study and no changes during one-year follow-up were seen in the VF parameters. Another study also compared the VF changes after transient elevation (IOP average of 64 mmHg for < 30 seconds) in eyes with and without glaucoma. No functional damage was observed in both groups.[34] Zhou et al compared FLACS with conventional phacoemulsification in patients with glaucoma; no difference in first postoperative day IOP was seen between the two groups.[35]

Glaucoma lasers can induce short-term intraoperative IOP elevation, usually caused by the pressure applied to the globe with the goniolens. A study on human cadaver eyes showed an average IOP rise of 20 mmHg when simulating selective laser trabeculoplasty (SLT) and laser peripheral iridotomy (LPI) procedures.[36] In this last procedure, IOP got as high as 130 mmHg during increased globe pressure with the iridotomy lens simulating the maneuver to tamponade the blood by the end of the procedure.[36]

IOP Elevation During Diagnostic Procedures

Diagnostic procedures such as gonioscopy can also induce short-term IOP elevation with an IOP increase of 20 mmHg during gonioscopy using a Sussman goniolens on cadaver eyes.[36] When pressure was increased against the globe and indentation was performed, IOP rose 50–90 mmHg compared to baseline. Gonioscopy is recommended to be performed in any patient who has glaucoma or is suspected of having the disease and is an essential tool for glaucoma diagnosis – yet clinically, there is little concern with the transient IOP elevation here.

Digital ocular compression (DOC) is often performed in the postoperative period after glaucoma filtering surgery to expand the filtering bleb and achieve IOP reduction; however, transient IOP elevation is observed during the period of

compression.[37,38] A study evaluating IOP behaviour during DOC found, in an in vivo human experiment, a mean IOP of 104±8 mmHg during the compression period.[37]

Transitory IOP elevation is not only seen during procedures but can also be observed after peribulbar anesthesia.[39,40] Morgan et al found a mean post-injection IOP of 32.4 mmHg in patients who underwent peribulbar block.[39] By Four minutes post-injection, IOPs no longer differed from their pre-injection levels.[39] The level of IOP spike will depend on the type of anesthetic drugs used and the volume of injected anesthetic.[39–41]

Everyday habits like squeezing the lids and rubbing the eye can cause short-term IOP elevation as high as 70 and 109 mmHg above baseline, respectively.[42,43] Many studies have reported that chronic rubbing can contribute to the pathogenesis of some diseases, such as keratoconus.[44,45] However, it is still unknown whether this transitory direct force applied to the globe is relevant to the development or progression of glaucoma.

Discussion

Elevated IOP can induce irreversible retinal ganglion cell injury with consequent permanent functional damage and is the major risk factor for the development and progression of glaucoma. This mechanism is a combination of the degree of IOP elevation, duration/consistency of the insult and optic nerve susceptibility. It is still unclear at which IOP level and duration of IOP insult that glaucoma damage occurs, and this is likely variable from eye to eye. Previous studies evaluated the time-dependent axonal transport changes in nonhuman eyes subjected to IOP elevation. Balaratnasingam et al showed that IOP elevation (IOP of 40–45 mmHg) up to 3 hours did not result in axonal transport changes.[5] However, after 12 hours of insult, there were significant changes in axonal transport. Chihara et al also found no axonal transport changes up to 3 hours of IOP elevation, while changes started to get evident after 6 hours of IOP insult.[46] Another study from Lai et al showed that no severe retina nerve fiber layer (RNFL) damage was seen in eyes that suffered a single episode of acute primary angle closure with a duration of up to 48 hours.[47] RNFL damage was detected when the duration of the attack was longer than 48 hours. Garhofer et al evaluated retinal and optic nerve head blood flow during short-term increase in IOP and showed that this insult did not alter retinal or ONH regulation during neuronal stimulation.[48] The apparent lack of long-term adverse effects after transitory IOP elevation emphasizes that the duration of IOP insult is an important determinant of optic nerve damage. Short-term moderate IOP elevations lasting a few minutes appear to unlikely to cause significant structural damage to the optic nerve.

Transitory IOP rise occurs in many ophthalmic surgeries and diagnostic procedures, including cataract surgery and laser procedures, as well as during gonioscopy. These acute IOP elevations range from high twenties to 140 mmHg and last for a few seconds or even 10 minutes.[11,12,20–22,30,31,35] The IOP usually returns to baseline shortly after the procedure.[33]

Based on basic science and clinical evidence, as well as how common they occur in glaucoma patients with diagnostic and therapeutic procedures, sporadic transient IOP elevations are unlikely to cause significant harm. While there is still a need for more evidence, it is likely of minimal risk to most glaucoma patients as transient IOP elevations appear to be well-tolerated with no obvious deleterious effects seen clinically. As with any procedure or evaluation, the risk to benefit ratio must be considered – and in most cases, the transitory IOP elevation from most ocular procedures is favorable. One exception may be LASIK, where the IOP elevation is specifically much higher than other procedures, and the risk to benefit ratio is not as favorable.

Previous studies have already reported the snuff-out phenomenon with unexplained permanent central vision loss following glaucoma surgery.[49,50] This phenomenon is more common in patients with severely compromised visual fields. Therefore, greater caution must be exerted when dealing with very advanced glaucomatous eyes with already severely compromised optic nerve structure and function, as those are more susceptible to optic nerve damage, which could result in further glaucoma damage.More studies are still necessary to have a clearer understanding of whether transitory isolated IOP elevations are clinically significant and if this represents a real risk to glaucoma patients.

This review has a few limitations, particularly the variability in reported IOP changes stemming from the diverse methodologies employed across studies, the broad range of IOP elevations, and the differing study populations (including both animal and human eyes). Furthermore, the absence of long-term studies examining the effects of transient IOP elevation represents another limitation that should be addressed in future studies.

Conclusion

This review highlights that transient intraocular pressure (IOP) rises during ophthalmic diagnostic and therapeutic procedures, although common, are generally well tolerated in both healthy and glaucomatous eyes. Most studies have not demonstrated significant structural or functional damage from these short-term IOP elevations, particularly when the duration is short, and the magnitude of increase remains moderate. However, the vulnerability of eyes with advanced glaucoma, especially those with severely compromised optic nerve structures, remains a concern. The evidence suggests that while transient IOP elevations are unlikely to cause widespread harm, patients with advanced glaucoma may require additional caution and monitoring during procedures known to induce acute IOP spikes. More research is needed to delineate the specific IOP thresholds and duration that could lead to permanent optic nerve damage, particularly in this vulnerable group. Understanding the individualized risk profile for glaucoma patients is crucial for optimizing procedural safety and minimizing the potential for irreversible damage.

Disclosure

Dr Ticiana De Francesco is a consultant and/or speaker for AbbVie, Iantrek, Elios, Nova eye Medical, Vialase, Zeiss, Alcon, and Glaukos, outside the submitted work. Dr Iqbal Ike Ahmed is a consultant for AbbVie, Ace Vision, Alcon, Aliph Medical, Aquea Health, Arcscan, Avellino Lab, Avisi, Balance Ophthalmics, Bausch and Lomb, Beaver Visitec, Belkin Vision, Bionode, Carl Zeiss, Centricity Visio, Inc, CorNeat Vision, Custom Surgical, Elios Vision, ElutiMed, eyeFlow, Inc, EyeMed, EyeO Technologies, Exhaura Limited, Glaukos, Gore, Hexiris Pharma, Iantrek, InjectSense, Iridex, iCare, iStar, Johnson & Johnson Vision, LayerBio, Liqid Medical, Long Bridge Medical, MST Surgical, Myra Vision, New World Medical, Nova Eye, Ocular Instruments, Ocular Therapeutix, Oculus Surgical, OcuSciences, Omega Ophthalmics, Peripherex, PolyActiva, PulseMedica, Radiance Therapeutics, Radius XR, Rheon Medical, Ripple Therapeutics, Sanoculis, Santen, Singapore Biodesign, Shifamed, Sight Sciences, Smartlens, Stroma, Thea Pharma, TFS Health Science, ViaLase, Visci Ltd, Visus Therapeutics, Vizzario, and Zilia Inc, outside the submitted work.

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