·Commentary·

Keratorefractive surgery and glaucoma

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Abstract

• Keratorefractive surgery changes the central corneal thickness (CCT) and corneal curvature, which could influence the Goldmann applanation tonometer (GAT) and non-contact tonometer (NCT) measurements of intraocular pressure (IOP), but not dynamic contour tonometer (DCT). During the procedure of LASIK, there is a transient rise of IOP, which increases the risks of optic nerve damage. Meanwhile, the presence of functioning filtering blebs may affect the choice and outcome of refractive surgery, or even becomes a contraindication of surgery. Steroids are typically used after keratorefractive surgery, which could lead to IOP elevation. Hence it is important to monitor IOP after LASIK and to be aware of inaccurate IOP readings due to corneal flap interface fluid. Treating patients with postoperative elevated IOP after keratorefractive surgery is similar to that for patients with glaucoma. This review will address the issues surrounding the safety, relevant complications and implications of keratorefractive surgeries on glaucoma and relevant diagnostic tests.

• KEYWORDS: keratorefractive surgery; glaucoma; intraocular pressure; steroid-induced glaucoma

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INTRODUCTION

The morbidity of glaucoma among myopes is two to three times more prevalent than that of the general population[1]. Patients with glaucoma who seek keratorefractive surgery present a unique set of considerations, because high intraocular pressure (IOP) and frail optic nerve create less ideal situation for such procedures. One investigation on 2 784 patients who requested refractive surgery showed that 705 patients were rejected for various reasons, 0.7% of them for glaucoma [2]. The presence of functioning filtering blebs may affect the choice and outcome of refractive surgery or even become a contraindication of the operation [3]. Because of special features of glaucoma, the risks and benefits must be weighed before recommending such surgeries to a patient with glaucoma, and the treatment should be modified to avoid postoperative complications.

EFFECT ON INTRAOCULAR PRESSURE MEASUREMENT

Goldmann applanation tonometer (GAT) is the "gold standard" of IOP measurement. GAT is based on the Imbert-Fick principle, which assumes the external force is equal to the internal pressure. The principle assumes that the cornea is a dry, infinitely thin, and perfectly spherical surface with diameter of 3.06mm, central corneal thickness (CCT) of 520μm, and surfaces with stable tension and resistance; hence the results are in a linear relationship between external force and IOP. But the cornea is not an ideal subject; therefore modification of the Imbert-Fick principle is applied. Besides, many studies indicated that CCT had an important influence on IOP measurement [4-12]. When CCT is thicker than 520μm, the real IOP is overestimated because the high pressure is required to flatten the cornea, and the thinner CCT leads to a decrease of IOP reading. There is a fluctuation in CCT among the general population, especially in patients after refractive surgery, which may affect the IOP measurement significantly. What's more, many reports showed that corneal flap interface fluid after laser-assisted *in situ* keratomileusis (LASIK) caused a lower IOP reading and could mask a dangerous elevated IOP[13].

Relationship Between CCT and IOP Measurement In refractive surgery, the central cornea is cut and rebuilt to correct myopia. In the early postoperative period, there is certain fluctuation in CCT, which seems to remain stable after the first 24 hours. Several studies have confirmed a decrease in measured IOP with GAT or non-contact tonometer (NCT) [8-12]. Montés-Micó *et al* [8] found IOP decreased obviously with GAT or NCT after photorefractive keratectomy (PRK) and LASIK (P<0.01), while the control

group remained the same, and there was no difference between PRK and LASIK. Park *et al* [11] found an average 3.9mmHg decrease in IOP on the central cornea (P <0.01, 25.2%), and an average 2.0mmHg decrease in IOP on the nasal side of cornea. The change in IOP measurement may be caused by the removal of Bowman's membrane and thinning of corneal stroma, which results in decreased resistance of cornea.

The reduction in IOP after LASIK is related to the changes of corneal thickness, corneal curvature, and correction degree. Montés-Micó *et al* [8] found a 0.5mmHg reduction in IOP with each diopter correction of myopia. Gimeno *et al* [14] found a remarkable decrease in measured IOP after surgery if the preoperative refraction was greater than -5.00D. Although not stable, there was a linear relationship between IOP and CCT, which was about 45μm change in CCT with 2 to 3mmHg reduction of IOP. Emara *et al* [15] found a 0.25mmHg reduction in IOP with each 10μm change in CCT, while Duch *et al* [9] found a change of 0.41mmHg with each 10μm change in CCT. Nevertheless, Chatterjee *et al* [16] and Rosa *et al* [17] found a decrease in IOP of 0.46mmHg and 0.71mmHg respectively with each 10μm change in CCT after PRK. Several authors have proposed that when the ablated cornea reaches a certain proportion, it will cause an apparent IOP decrease [18]. It is obvious that we can't apply a precise correction coefficient to patients who have undergone LASIK.

Influence of Flap Interface Fluid after LASIK on IOP Measurement In the first week after LASIK, a few patients have clinic symptoms similar to diffuse lamellar keratitis (DLK), which is due to the accumulated fluid in the flap interface. Flap interface fluid masks an elevated pressure, so clinicians should give much attention to this phenomenon. Hamilton *et al*[19] reported that in 4 steroid-induced glaucoma patients (six eyes), the interface fluid masked the elevated IOP, which resulted in a difficult diagnosis. Fogla *et al* [20] reported a case of presumed DLK after LASIK, which was treated with prednisolone 10g/L once every two hours for one week, and then increased to once an hour with the addition of ketorolac. One week later, the patient had bilateral microcystic edema and a clear fluid-filled space in the interface between the flap and stromal bed. The IOP measured by GAT was 3.0mmHg, but 54.7mmHg by Schiotz. Later, with a reduction of steroids, the interface fluid of the patient diminished and the IOP became normal. Although many physicians do not measure IOP on the first day after surgery in order to prevent the displacement of the flap, IOP should be measured in subsequent visits.

Reliability of Different Tonometers in IOP Measure–ment

Dynamic contour tonometer For dynamic contour tonometer (DCT) we design a special concave tip that matches the corneal curvature. A microchip-enabled solid-state sensor integrated in the DCT's contoured surface may conduct direct transcorneal measurement of IOP. DCT is used to measure IOP and ocular pulse amplitude (OPA) simultaneously, dynamically showing measured data through the screen. Unlike the GAT plane probe, the concave tip achieves a contour which matches the minimal corneal morphologic alteration, allowing all of forces to be directed to the pressure sensor surface. DCT measures IOP automatically, and displays data on the screen, when the pressure is equal on both sides of curved surface. It is suitable for the patients whose corneal thickness varies from 300μm to 700μm and the radius of curvature from 5.5mm to 9.2mm. DCT is less affected by corneal properties than GAT, such as corneal thickness and corneal rigidity [21]. Kniestedt *et al* [22] found that IOP readings by DCT had deviation less than 0.5mmHg. However, IOP results by GAT had deviation of nearly 4mmHg, which appeared that DCT was more accurate than GAT. Kaufmann *et al* [23] found the IOP variation degree inside patients was 0.65mmHg by DCT and 1.1mmHg by GAT(P=0.008), whereas the variation degree outside patients was 0.44mmHg by DCT and 1.28mmHg by GAT (P =0.017), which illuminated smaller variety and higher repeatability of DCT. Pepose *et al* [10] found DCT eliminated most of the systematic errors arising from physical variables of the eye. What's more, the standard deviation and variance of IOP by DCT are smaller than that by GAT. The main advantage of DCT is that the IOP measurements are not significantly affected by changes in the pressure on cornea or corneal properties, resulting in stabilization and accuracy.

IOP is measured accurately with DCT, which is little affected by LASIK. Siganos *et al* [24] monitored IOP in 118 eyes at 1 week and 4 weeks after LASIK using GAT, NCT and DCT, and then found no obvious change in the observed value of IOP by DCT, but the contrary outcomes by NCT and GAT. Thickness changes in the cornea have little effect on IOP measurement by DCT, especially to the patients with thinner CCT. Moreover, DCT reading, on average, is close to the real IOP. In summary, DCT may be more suitable for monitoring IOP in eyes that have been done LASIK.

Non-contact tonometer Non-contact tonometer (NCT) uses an air pulse to flatten the central area of cornea (the diameter is 3.6mm) and records the spent time, then transferring to IOP reading by computer. Without using anesthetics and contacting cornea, NCT avoids infections caused by anesthetics and instruments after the cornea epidermis is damaged. It also has other advantages, such as easy operation, stable IOP measurements even after performing repeatedly. In contrast to GAT, NCT is more accurate after keratorefractive surgery [9]. Zadok *et al*[25] measured IOP before and after LASIK, and found a greater decrease in measured IOP with GAT than with NCT, whereas there was no correlation between change in IOP and change in corneal curvature or CCT when measured with NCT and GAT.

However, the NCT is relatively similar to GAT, which is affected by both corneal properties and the stability of lacrimal film, resulting in imprecise IOP measurement. Hsu *et al* [26] used NCT to measure IOP of 60 eyes (30 patients), found the average IOP is 14.9mmHg before surgery and 8.3mmHg at three months after the surgery, an average of 6.6mmHg reduction. Morgan *et al*[27] also found IOP reduction was influenced by CCT when measuring with NCT, which demonstrates the change in CCT also influences IOP.

EFFECT ON THE IMAGE ANALYSIS OF RNFL AND VISUAL FIELD EXAMINATION

Effect on the Image of RNFL Image analysis of the optic nerve head and peripapillary retinal nerve fiber layer(RNFL) is a common examination in glaucoma patients. The first generation of never fiber analyzer (NFA) is GDx, which estimates the thickness of the peripapillary RNFL by measuring changes of polarized light reflected from the retina. Keratorefractive surgery may change the corneal birefringence, which in turn will affect the accuracy of the GDx measurement. But corneal compensation can prevent the deviation of measurement. However, researches to date [28-30] have shown no significant change in the RNFL measurement after LASIK using either GDx(without corneal compensation), Heidelberg retinal tomography (HRT), or optical coherence tomography (OCT). Nevertheless, the potential influence of refractive surgery on image analysis must be taken into account, especially with the earlier GDx that lacks corneal compensation.

Effect on Visual Field Weiss *et al* [31] and Bushley *et al*[32] found some patients presented with visual field loss after LASIK, which might be caused by the transient rise of IOP. It was reported [33] that the central visual field remained after PRK, but the peripheral visual field was damaged. This is mainly because of a blur zone in the cornea caused by the tissue ablation. However, Vetrugno *et al*[34] found no change before and after PRK when testing the visual fields of 16 patients with myopia using frequency doubling technology. Chan *et al*[35] also found that there was no obvious change in visual function or visual field in a short time after LASIK. Lleó-Pérez and Sanchis Gimeno [36] found patients with mild myopia presented a transient diffuse visual field defect within six months after LASIK, but self-recovered after 12 months. However, there is no consensus about whether visual field will be affected after LASIK, but we should still be careful about the possibility of diffuse visual field defect. In a word, it is prudent to be alert to possible changes in visual fields after keratorefractive surgery.

The Possible Mechanism of Visual Field Change -a Transient Elevation of IOP In LASIK, suction ring is mounted on eyeball by vacuum, around which the microkeratome makes a rotatory movement. Suction on the cornea can raise the IOP to more than 90mmHg. It was documented that this high IOP affected retinal circulation, but one study[37] found no changes in neither the structure nor the function of the optic nerve after LASIK. Hamada *et al* [38] reported that the transient elevation of IOP didn't change the morphology of optic disc or the thickness of RNFL one year after LASIK. However, Cameron *et al*[39] reported a case of bilateral optic neuropathy after LASIK, which could be attributed to the sharp, transient IOP rise during flap construction. Damage of nerve fiber and visual field defects were associated with a sudden rise in IOP which resulted in physical press on retinal ganglion cells (RGCs), nerve fiber layer and lamina cribrosa sclerae. Besides, transient IOP rise can cause optic nerve ischemic damage because of transient block of the blood flow of retinal central artery and posterior ciliary artery. The cause of such complications can be identified only by subsequent examination or angiography of fundus after surgery. Although the risk of transient IOP rise in glaucoma patients is not clear yet, LASIK should be avoided in patients with serious glaucomatous optic neuropathy, or physicians may consider other possible operations to avoid the transient elevation of IOP, such as PRK.

STEROID-INDUCED GLAUCOMA AFTER REFRACTIVE SURGERY

Steroids are commonly used after LASIK to reduce the synthesis of collagen, decrease corneal haze and avoid myopia relapse. Although many researchers reported that steroids did not improve corneal haze after PRK, topical and

oral steroids are being used with large amount and high frequency in the management of postoperative DLK, which has an incidence ranging from 0.75% -32% . It is more dangerous for patients with glaucoma to use steroids which can induce IOP elevation. Galal *et al* [40] retrospectively studied 11 cases of DLK after LASIK who had steroid-induced IOP elevation and consequently developed flap interface fluid and corneal edema. After steroids was tapered off and replaced by aqueous suppressants, the interface fluid was cleared and IOP became normal. Severe steroid-induced glaucoma can apparently damage the visual function. Davidson *et al*[41] reported that when a patient took steroids for DLK after LASIK, his optic nerve was damaged by steroid-induced glaucoma. Shaikh *et al* [42] reported two steroid-sensitive patients who developed end-stage glaucoma after LASIK requiring trabeculectomy to control IOP.

The manifestation of steroid-induced glaucoma is similar to primary open angle glaucoma (POAG), but postoperative optic papilla change of high myopia is different from that of POAG, without deepening and enlargement of optic cup, which is not easily identified. Probably it is because high myopia is vulnerable to elevated IOP. Therefore it is not wise to judge the optic papilla damage of steroid-induced glaucoma in myopia patients according to the changes of optic papilla.

The clinical manifestation of pressure-induced interlamellar stromal keratitis (PISK) is similar to DLK, which is characterized by corneal opacity without clear reasons. Topical steroids are not only ineffective, but also may aggravate the corneal haze and increase IOP. Elevated IOP for a few weeks may cause optic nerve damage and visual field defect, which can be solved by decreasing the dose of steroids and administering aqueous suppressants. Miyai *et al*[43] reported two cases with PISK after LASIK and found that using aqueous suppressants instead of steroids was effective to clear interface fluid and decrease IOP. Cheng *et al* [44] studied two cases of steroid-induced glaucoma after LASIK by confocal microscopy and found that it was no related to inflammatory cells in the flap interface.

For patients after refractive surgery, it is recommended to use steroids that are of low density and with less side-effect of increasing IOP, and the time can't last too long. Once IOP elevation occurs, steroids should be stopped. After refractive surgery, it is a routine to measure IOP regularly. It should be more prudent to perform LASIK for patients with one eye high myopia (the other eye is blind).

CAN KERATOREFRACTIVE SURGERY BE DONE IN PATIENTS WITH GLAUCOMA?

Glaucoma is not an absolute contraindication to LASIK, but a relative contraindication. The disadvantages of patients with glaucoma undergoing LASIK are as follows: ① Eyes with glaucomatous nerve damage are more sensitive to the transient IOP elevation in the surgery, which could cause the damage of optic nerve and visual field [35]. ② The use of steroids after surgery could aggravate the elevated IOP. Furthermore, patients with glaucoma are more sensitive to steroids, and hence the risk of steroid-induced glaucoma and damage of visual function are much greater than general population. ③ IOP elevation could cause the relapse and development of myopia, which is a main reason for the regression after LASIK. ④ The risk to the filtering bleb with the microkeratome makes LASIK a contraindication in most cases, and PRK would be preferable, if any keratorefractive surgery is to be used. Further caution is required when prophylactic mitomycin C is employed with PRK [3]. In summary, high IOP not only affects the postoperative application of steroids, but also could cause regression. Therefore, intensive preoperative counseling and informed consent is mandatory.

For the sake of safety, preoperative IOP should be controlled in normal range, preferably lower than 24mmHg, because some reports show that the incidence of POAG is much higher among people whose IOP is higher than 26mmHg. Secondly, preoperative ratio of C/D should be less than 0.5, with normal visual field. Thirdly, surgeons should shorten the time of vacuum suction. Corneal thickness could be used as a predicting factor for the prognosis, and therefore preoperative CCT measurement is important to patients with high IOP, which could predict the risk of POAG. The difference between preoperative and postoperative IOP is useful to guide the postoperative steroids use. Once dangerously high IOP appears, steroids should be stopped immediately, in case further damages occur to optic nerve.

TREATMENT FOR THE ELEVATED IOP AFTER REFRACTIVE SURGERY

There is no evidence that patients undergoing refractive surgery with postoperatively elevated IOP should have special medical management protocols different from chronic glaucoma. Nagy *et al*[45] compared timolol (twice per day), dorzolamide (three times per day) and combined timolol and dorzolamide (twice per day) in patients after PRK and found that the combination was the most effective. Vertugno *et al*[46] reported that latanoprost 0.05g/L and timolol maleate 5g/L were effective in lowering IOP after PRK. In recent years,

the treatment for managing the rise of IOP has greatly improved and physicians should be aware of the contraindications and side effects of those drugs. In brief, treatment for the elevated IOP after keratorefractive surgery is similar to those for patients with glaucoma.

In conclusion, patients with glaucoma require considerable preoperative counseling and postoperative monitoring. Furthermore, postoperative steroids and underestimation of tonometric IOP measurements are extremely important considerations. Keratorefractive surgery influences the accuracy of GDx measurements of the RNFL, but there is no significant change in the RNFL measurements after LASIK using GDxVCC apparatus. Glaucoma medications in the management of patients who have had keratorefractive surgery are similar to the medications used in other glaucoma patients. The present data regarding the safety of keratorefractive surgery in patients with glaucoma show that glaucoma is not an absolute contraindication to LASIK, but a relative contraindication.

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