Commentary: Glaucoma following penetrating keratoplasty: A double whammy

Glaucoma associated with penetrating keratoplasty (PK) is a major cause of graft failure and irreversible visual loss.[1,2] Post-penetrating keratoplasty glaucoma (PPKG) is a significant clinical challenge due to difficulty in diagnosis, accurately measuring and monitoring IOP as well as complexity in its management. It leads to significant endothelial cell loss as the endothelial reserve is already low in a recipient corneal graft. Needless to say, appropriate diagnosis and treatment are essential to preserve the clarity of the corneal graft as well as optic nerve function. The incidence of glaucoma following PK has been reported to be between 9% and 35%.[3] Few studies in India have reported on glaucoma following PK despite corneal diseases, including infectious keratitis and penetrating trauma, being a major cause of visual loss and PK being one of the most common surgical procedures to address corneal blindness.^[4,5] Post-PK glaucoma is likely to be less common in the West and economically advanced nations owing to the preponderance of non-inflammatory corneal diseases such as keratoconus, corneal dystrophies, and ectasia as predominant indications of PK. In less-developed economies such as Asia and Africa, infectious keratitis, penetrating trauma with significant peripheral anterior synechiae (PAS) and bullous keratopathy associated with complicated cataract surgeries are likely to be more common indications of PK with an estimated higher incidence of post-PK glaucoma. Outcomes of cataract surgery have significantly improved in India in the past three decades, with lower incidence of corneal complications, and the quality of graft clarity has significantly improved with lamellar keratoplasty techniques. Due to the paucity of published studies, it is not known if these improvements in outcomes of ocular surgeries and early diagnosis and management of infective keratitis have reduced the occurrence of post-PK glaucoma. Preexisting glaucoma, regrafts, peripheral anterior synechiae, and prolonged steroid therapy are major risk factors for post-PK IOP elevation or glaucoma. [6]

The incidence of glaucoma in the study reported in the current issue of the Indian Journal of Ophthalmology by Shree et al.[7] is around 40.5%, which is higher than that reported in the past. The pooled estimate for overall incidence of PPKG has been reported to be 21.5%.[8] The most common indication for PK in the study by Shree et al.^[7] was corneal ulcers, including perforated corneal ulcers (38%), which account for a high likelihood of post-PK glaucoma reported by the authors. Given the fact that infective keratitis with secondary complications is one of the most common indications of KP in India, the probability of secondary glaucoma is expected to be high, compromising the success of grafts. Early recognition and instituting appropriate management, including therapeutic KP in refractory keratitis prior to perforation is likely to minimize glaucoma and optimize success of the corneal grafts. Lamellar keratoplasties (LK) appear to lower the possibility of elevated IOP and glaucoma as these procedures generally involve less intraocular manipulation and disturbance of the anterior chamber anatomy, thus requiring short-term steroid therapy. Moreover, in clinical situations where LK is indicated, there appears to be least disorganization of the anterior chamber anatomy, inflammation, and PAS, possibly contributing to lesser IOP rise. LK appears to have replaced PK in many instances, such as early bullous keratopathy and corneal dystrophies, with lower likelihood of post keratoplasty glaucoma.

Diagnosis of glaucoma in PK offers major challenges. The optic nerve head is often difficult to assess due to a lack of adequate graft clarity and variable astigmatism. Surface irregularities, scarring, and alternations in corneal thickness render measurement of IOP with Goldman applanation tonometry, the gold standard, extremely challenging. [9] Newer techniques in lamellar corneal surgery have helped overcome some of these difficulties in diagnosing glaucoma post keratoplasty. Rebound iCare tonometers are less influenced by corneal edema compared to GAT, and agreement between these instruments was acceptable in lamellar keratoplasties and DSAEK but was poor in PK and in eyes with graft edema.[10] The evolution of transpalpebral rebound tonometers (Diaton) is likely to provide alternate modalities of accurately measuring IOP in corneal grafts, once clinically validated.[11] Monitoring IOP will play a key role in assessing glaucoma progression post PK as assessment of discs, visual fields, and OCT is not feasible in most grafted eyes.

Glaucoma following corneal transplantation continues to pose a significant challenge in diagnosis as well as in monitoring and managing progressive disease. Visual loss results from both optic disc excavation and graft rejection. However, the approach to corneal transplantation surgeries has been currently evolving with advances in selective replacement of diseased endothelium as well as anterior lamellar techniques and consideration of earlier intervention in refractory infective keratitis, all of which are likely to reduce the incidence of glaucoma following corneal surgeries. Earlier diagnosis and improved monitoring of glaucoma in lamellar corneal surgeries are also likely to significantly contain progressive visual loss due to glaucoma.

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