EDITORIAL

Trabecular Wound Healing—the Nemesis of Trabecular MIGS

Tanuj Dada

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Minimally invasive glaucoma surgery on the trabecular meshwork (Trabecular MIGS) is one of the most popular forms of MIGS procedures worldwide. Most of the current trabecular MIGS procedures work by stenting (iStent, Hydrus), incising (GATT, microhook), or excising (KDB, BANG) the trabecular meshwork (TM), which is the site of maximum resistance for aqueous outflow. These procedures are usually done in the nasal quadrant with the surgeon sitting temporally. The surgery is considered a success when the pigmented TM is cut and the canal of Schlemm (SC) is deroofed with visibility of white scleral tissue, or when an implant is inserted into the TM. However, the trabecular response to this injury actually determines the final outcome (which is not impressive!).

Injury to the TM incites a wound healing response: the cut edges of the TM may again adhere together (especially with microhook/GATT), the strip of TM may readhere to its original position,[1] the iris may attach to the outer wall of the canal (PAS),[2] excessive pigment dispersion may deposit in the outer wall of the SC, or a fibrotic membrane may form and eventually close the outflow channels in the outer wall of the canal. Over time, this results in complete functional closure of the outflow pathways and cessation of any aqueous outflow in a region which may previously have had some outflow[3] (as trabecular MIGS is usually done in early glaucoma where trabecular outflow is decreased compared to healthy individuals but not completely absent). This can result in a rebound increase in intraocular pressure (IOP) on long-term follow-up after 1–2 years, especially when the significant hypotensive effect of cataract surgery wanes off.[4]

In addition, it is important to understand that the pathological process which caused primary open-angle glaucoma (POAG) in the first place, with accumulation of extracellular debris/plaques on the TM, continues unabated (actually worsens with age) and now moves further downstream, which may directly impact the collector channels, increasing the intrascleral outflow resistance.

Another issue that has recently come into focus is the effect of prior SLT (selective laser trabeculoplasty) on reducing the efficacy of MIGS. Mitchell et al. reported on the effectiveness of angle-based minimally invasive glaucoma surgery after laser trabeculoplasty and concluded that prior laser trabeculoplasty may be associated with a higher chance of subsequent glaucoma surgery following angle-based MIGS, either with or without concurrent phacoemulsification. This study indicates that prior SLT may reduce the effectiveness of subsequent trabecular MIGS, and one of the mechanisms may be accentuated wound healing.[5]

Certain modifications may help in attenuating/delaying the fibrotic response. First, dilating the canal with viscoelastic prior to cutting it may help to prevent injury to the outer wall of the SC and reduce bleeding, which in turn can reduce the fibrotic response.[6] The use of systemic and topical steroids in the initial postoperative period can help to reduce the inflammatory response and deposition of aqueous protein content into the outflow pathways. MIGS procedures that involve cutting the TM should only remove a small segment of the TM (45–60°) in the superonasal region so that the inferonasal quadrant, which usually harbors the maximum aqueous outflow channels, is not disturbed.[7] The smaller the incision, lesser the bleeding, inflammation, and subsequent healing response.

Performing angle-based MIGS earlier in the course of the disease, prior to use of SLT or multidrug eye drop therapy (especially as BAK also induces trabecular inflammation and may promote fibrosis), may be helpful.

Another option that needs to be further explored is the impact of rho kinase inhibitors on trabecular MIGS outcomes. In addition to decreasing episcleral venous pressure, these drugs reduce cell contraction and stiffness and increase the effective filtration area of the TM. This may have an additive effect on MIGS procedures, which dilate or stent the canal.

The TM is a vital tissue involved in regulation of IOP, and the IOP-lowering efficacy of any surgery targeting the TM-SC complex is governed by its proper placement within the canal with access to both the aqueous in the anterior chamber and the outflow pathways and is limited by the outflow resistance distal to the SC. Thus, achieving drug-free IOP in low teens is not possible with MIGS, and in the absence of any available antifibrotic agents targeting the TM, the IOP-lowering efficacy of MIGS surgery is bound to decrease over time. There is always a risk of surgical failure and a rebound increase in IOP, which must be discussed with patients prior to any MIGS surgery. Surgery should be restricted to not more than one quadrant (cut extending 45–60°) to get the best fit in terms of IOP-lowering efficacy vs tissue destruction-repair and retain functional TM tissue, which can even be acted upon in the future by lasers or by novel medical therapies.[7] This is especially important as MIGS is often performed in early glaucoma where the TM is still operational,

Department of Ophthalmology, Dr Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, India

Corresponding Author: Tanuj Dada, Department of Ophthalmology, Dr Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, India, Phone: 011-26593101, e-mail: tanujdada@gmail.com

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and effort should be made to retain as much functional tissue as possible. Further research is required to evaluate the best location for IOP vs excisional MIGS, the MIGS which leads to the least fibrotic response (usually maximum with GATT),[8] use of antifibrotic therapies targeting the TM, and impact of SLT on trabecular MIGS.[9]

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