UVEITIC GLAUCOMA

Uveitic Glaucoma is a dreaded complication of uveitis which can cause irreversible blindness even when the uveitis has been successfully treated. While uveitis accounts for 10-35% of blindness in individuals under 65 years of age, intraocular pressure (IOP) elevation occurs in approximately 8.6-41.8% of cases. [1, 2]

Aetiology and Mechanism

In general, glaucoma may arise as a complication in all types of uveitis. However, certain uveitic syndromes are more commonly associated with elevated IOP even at the onset of the uveitis. These include Possner Schlossman Syndrome and Fuch's Heterochromic cyclitis which are usually unilateral; and herpetic uveitis and Juvenile idiopathic arthritis which commonly affect both eyes.[3]

Many factors may cause elevated IOP in uveitic glaucoma. The most notable of them would be reduced aqueous outflow, pupillary block due to synechial closure and side effect of corticosteroid treatment.

Among the reasons for reduced aqueous outflow are clogged trabecular meshwork from inflammatory debri, changes in the cytoskelton of the trabecular meshwork, trabeculitis, angle closure from synechiae or angle neovascularization; or less commonly angle closure associated with anterior rotation of the ciliary body caused by ciliary body effusion from excessive inflammation.

Pupillary block is commonly associated with secclusio pupillae, seen when enough posterior synechiae impedes aqueous outflow through the pupillary aperture. The ensuing events may mimick that of an acute angle closure attack, with iris bombe, severely elevated IOP, a congested eye and corneal edema. In these instances, a peripheral iridotomy either performed surgically or with laser may temporarily reduce the IOP before more definitive surgical intervention such as synechiolysis is performed. Often this is done together with lens extraction.

Corticosteroid-induced IOP elevation

Corticosteroids remain the mainstay of treatment for uveitis. It is a potent anti-inflammatory drug that is often used as a first line treatment. However, it also causes significant IOP elevation especially when a corticosteroid of higher potency or higher dose and frequency is used or when used in those at risk like young individuals. Unfortunately in uveitis, the necessity of corticosteroid use is paramount, although second line agents have now contributed substantially to the control of uveitic exacerbations.

Diagnosis and Monitoring

The recurrent, relapsing and remitting nature of uveitis may influence detection of glaucomatous damage from prolonged IOP elevation. Papillitis, seen in posterior uveitis may mask glaucomatous assessment of the optic disc head. Cystoid macular edema may affect perimetric assessment for monitoring of glaucoma. Other retinal changes such as that seen in multifocal choroiditis or retinitis may also result in visual field defects not attributed to glaucomatous changes. Likewise, the use of optical coherence tomography may be affected by similar changes from uveitis.

The diagnosis of uveitic glaucoma therefore has to be made based on multiple signs. When the IOP elevation only occurs after starting corticosteroids, steroid – induced IOP elevation has to be suspected. A gonioscopy may help in differentiating IOP elevation from uveitic changes from that which occurs due to corticosteroid use. Synechial closure, angle neovascularization and pigmentation of the angle suggests uveitic damage as the cause of IOP elevation, whereas the angle may appear normal when IOP elevation is purely from corticosteroid use. More often however, these two entities co-exist.

Treatment

The first line of treatment is still medical therapy. The choice of medical therapy in uveitic glaucoma is similar to any other type of glaucoma, although some experts avoid the use of prostaglandin analogues (PGAs) with the assumption that PGAs may exacerbate macula edema. However, data to support this is conflicting.[4]

Marked IOP elevation in uveitis associated with glaucomatous damage, often requires more than medical treatment. Approximately 60% of children and 40% of adults with uveitis require surgical intervention when maximum tolerable medical therapy is insufficient to control the IOP.[5] Often, surgeons who resort to surgical intervention, prefer tube implantation to trabeculectomy, as the success rate of trabeculectomies is limited by bleb scarring and closure. Laser treatment in general, is avoided as it may cause exacerbation of the inflammation.

The importance of controlling inflammation prior to any surgical intervention cannot be stressed enough. Preparing a uveitic patient for surgery, or "priming" is as important as the surgery and the post-op care itself, because insufficient preoperative control may result in excessive postoperative inflammation, which in turn can result in ciliary shutdown and hypotony. Excessive post-operative inflammation may also result in the formation of a cyclitic membrane which may close any tubes present in the anterior chamber or the internal osteum in the case of trabeculectomy.

"Priming" of a patient pre-operatively is often done with an anti-inflammatory dose of corticosteroid like Prednisolone (0.5 mg/kg body weight), given approximately 2 weeks prior to surgery, and tapered according to how well the postoperative inflammation control is. Often, the timing of surgery is planned when the eye has been as quiet as possible, for as long as possible. All these steps are taken to ensure the success of the surgery in maintaining adequate IOP control.

In conclusion, striking a balance between control of inflammation to limit lens, macula and angle damage; and avoiding IOP elevation from steroid use, is crucial in reducing the risk of permanent visual loss from glaucomatous damage.

References

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