Steroid-Induced Glaucoma

A familiarity with this entity is essential to the care of a uveitic patient.

BY SANDRA M. JOHNSON, MD

In a patient with uveitis and elevated IOP, the latter may be due either to the inflammation or to the steroids used to treat the inflammation. Becker reported the entity steroid-induced glaucoma in 1965.[1] In his landmark study, after 6 weeks of treatment with betamethasone drops, the IOP in 30% of normal eyes rose to over 20 mm Hg, and it increased to above 30 mm Hg in 4%. It took 2 weeks for the IOP to return to baseline, as reported by Tripathi et al.[2] Further studies defined the condition and demonstrated that, in patients at increased risk (eg, diabetics, patients with open-angle glaucoma [OAG], relatives of patients with OAG, and high myopes), the response occurred as early as 1 week or as late as years after the continued use of steroids.[3-5] Interestingly, individuals with connective tissue disease such as rheumatoid arthritis are more likely to be steroid responders.[6]

Steroids alter the trabecular meshwork and decrease aqueous outflow. The activation of glucocorticoid receptors in the trabecular meshwork may lead to an accumulation of materials, including glycosaminoglycans, that inhibit aqueous outflow.[7-10]

Gonioscopy is fundamental to the diagnosis: it allows the clinician to assess the status of the anterior segment angle. In the case of secondary angle closure, a steroid response is often suspected when the IOP rises. In these cases, the elevation in IOP is likely from treating the uveitis with steroids and the restoration of aqueous inflow in an eye that has poor aqueous outflow. Steroid-induced glaucoma is a secondary OAG, as gonioscopy helps to define.

STEROID TREATMENT

Administered via multiple routes for uveitis, steroids are often initiated prior to immunosuppressants but may also be prescribed as supplements to immunosuppressant therapy. Physicians may administer oral steroids perioperatively or may prescribe the agents for manifestations of associated systemic disease. Topical steroids are commonly used to treat flare-ups of uveitis, and patients may require long-term maintenance doses.

> "Steroids alter the trabecular meshwork and decrease aqueous outflow."

Triamcinolone may be administered as a sub-Tenon injection for posterior disease or cystoid macular edema or as an intravitreal injection. This treatment can raise IOP for as long as 6 months.[11] This drug is also available as a sustained-release implant. All of these ocular routes have been associated with elevated IOP but the implants most of all.[12] When uveitic inflammation has damaged the outflow pathway, the eye may be more susceptible to a steroid-induced elevation of the IOP than a healthy eye would be.[13] In addition to the risk groups mentioned, children may also be more likely steroid responders.[14]

DIAGNOSIS

Bodh and colleagues provided guidance on the differential diagnosis for steroid-induced glaucoma.[15] They noted that an acute, short-term increase in IOP may be due to trabecular obstruction by inflammatory cells and proteins from the aqueous or to swelling of the trabecular meshwork's cellular components with narrowing and cellular dysfunction. Chronically elevated IOP, however,

Figure 1. An eye of a teenager on systemic immunosuppressants who requires episodic intravenous triamcinolone acetonide to control his pars planitis. The patient's IOP rose to the 50s with central retinal artery pulsations despite oral acetazolamide and topical therapy with a β-blocker, carbonic anhydrase inhibitor, α-agonist, and prostaglandin analogue. His IOP has remained normal since the implantation of an Ahmed Glaucoma Valve (New World Medical, Inc.) and has permitted him to use steroid therapy when needed.

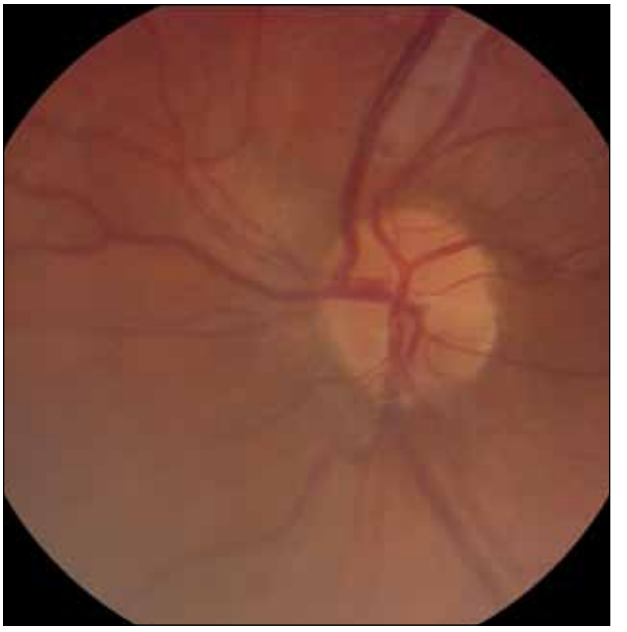

Figure 2. Preserved optic disc in the patient whose IOP rose above 50 mm Hg.

may be caused by underlying primary OAG or scarring and obstruction of outflow from multiple insults by inflammation.

In a review of 891 patients over 3 months by Sallam and colleagues, elevated IOP in uveitic eyes was more easily controlled when the increase was related to the use of steroids rather than to inflammation.[16] Their finding suggests a better prognosis for this type of secondary OAG. They also reported that the use of topical prostaglandin analogues to control IOP did not result in increased inflammation.

Besides using medical therapy to control IOP, physicians can adjust steroid therapy, especially if the patient is using topical agents. Topical difluprednate is more potent and requires less frequent dosing than prednisolone acetate 1%, and the former has been shown to produce large increases in IOP among uveitic patients. Difluprednate can be replaced with prednisolone acetate, or the latter may be exchanged for either rimexolone 1% or loteprednol etabonate, both of which have less propensity for elevating IOP.[17-19]

Little is known about the possible role of topical nonsteroidal antiinflammatory drugs for the treatment of uveitis, but this class of medication has no impact on IOP. Data support the use of oral nonsteroidal antiinflammatory drugs to treat uveitis associated with juvenile idiopathic arthritis,[20] an approach that might lessen the need for topical steroids in this group and potentially for patients with HLA-B27–associated uveitis or idiopathic anterior uveitis.[21]

Surgery is indicated for the minority of patients in whom maximally tolerated medication and an adjustment in steroid therapy fails to control the IOP while controlling the inflammation. In some instances, the surgeon can perform a vitrectomy to remove an intravitreal steroid depot or excise sub-Tenon triamcinolone.[22] When steroid treatment cannot be stopped, filtration surgery may permit the therapy to continue without the risk of glaucomatous damage (Figure 1).[23,24]

CONCLUSION

A familiarity with steroid-induced glaucoma or ocular hypertension is essential to the care of a uveitic patient.

"Comanagement with a rheumatologist or uveitis specialist is likely required."

Gonioscopy is required to define the open- or closed-angle mechanism for elevated IOP. Careful charting will help the clinician to determine if the increase in IOP relates to the inflammation or the steroid therapy. Steroid-sparing therapy or the avoidance of steroids in affected or at-risk patients can help avoid or treat a steroid response. Comanagement with a rheumatologist or uveitis specialist is likely required (Figure 2). ■

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