

Clinical Approach to Angle-recession Glaucoma

Early diagnosis and the aggressive management of elevated IOP after blunt trauma are essential.

BY QUANG H. NGUYEN, MD

Collins provided the first pathologic description of angle recession resulting from blunt trauma.¹ In 1962, Wolff and Zimmerman astutely connected the pathologic entity of the angle's recession with the clinical phenomenon of unilateral chronic glaucoma following trauma.²

Physicians must consider the possible development of glaucoma after any ocular trauma, which may damage the trabecular meshwork and/or other ocular structures relating to aqueous outflow. The consequent transient or prolonged elevation of IOP may lead to glaucomatous optic neuropathy. Ophthalmologists must also be cognizant that the treatment for the ocular injury, such as the use of steroid therapy, can further complicate the management of elevated IOP.

Ocular trauma may be classified as either blunt or penetrating. This article focuses on the former—namely, angle-recession glaucoma (ARG). It can be further subdivided into two stages of injury: early and delayed onset. In cases of early-onset ARG, a clinical examination may reveal iritis with or without hyphema. Clues such as hyphema with or without an iridodialysis and/or cyclodialysis cleft should alert physicians that the trabecular meshwork has sustained damage. The delayed onset of angle recession will cause a permanent elevation in IOP months to years after the initial blunt injury.

EPIDEMIOLOGY

The lifetime prevalence of ocular trauma is estimated to be 19.8%, with a 5-year incidence of 1.6%.³ Approximately 2.4 million ocular injuries occur in the United States each year.⁴ One study reported a 19% risk of developing glaucoma after closed-globe contusion, a rate approximately six times higher than after a penetrating injury.⁵ Whereas a patient who sustains a penetrating injury will immediately seek medical care, those who suffer a blunt injury will delay or may not seek medical attention. The latter group

therefore may not be appropriately educated about their injury and the prognosis.

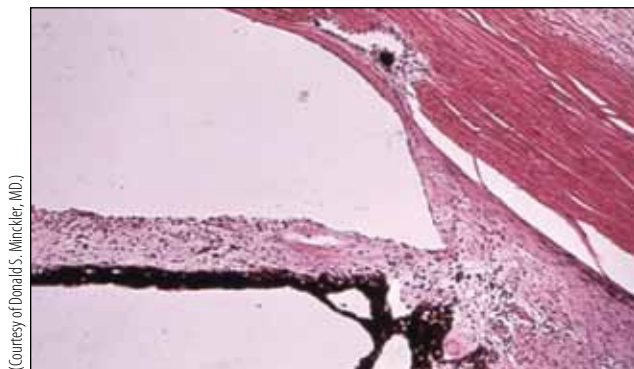
Among patients who experience traumatic angle recession, 5% to 20% will develop glaucomatous optic neuropathy.⁶ Greater recession of the angle (180° or more) may be predictive of a higher incidence of developing glaucoma.⁷ Interestingly, up to 50% of patients whose angle recession progresses to glaucomatous optic neuropathy will develop glaucoma in the fellow uninjured eye.⁸ This observation suggests that some patients are predisposed to developing the disease and that the trauma initiates a cascade of events leading to glaucomatous optic neuropathy.

MECHANISM AND PATHOPHYSIOLOGY OF INJURY

Blunt force indents the anterior aspect of the globe and rapidly transmits massive energy throughout the internal structures of the eye. Those structures that cannot withstand this energy will be damaged, producing various patterns of injury.⁹

Secondary open-angle glaucoma associated with angle recession represents the subtlest yet most devastating form of traumatic glaucoma. The angle's recession itself is not necessarily responsible for the damage to outflow structures, but it is a precursor to microscopic trabecular damage. The mechanism of IOP elevation in ARG appears to be a decrease in aqueous filtration. As suggested by Herschler,¹⁰ the tear in the ciliary body muscle is a marker of significant injury, and the glaucoma is related to scarring of the trabecular meshwork. Outflow facility, measured by tonography, is reduced and correlates with the degree of angle recession and glaucoma.¹¹

Pathologically, the recession of the anterior chamber angle appears as a separation between the longitudinal and circular fibers of the ciliary body muscle.² Histologically, the iris root is retrodisplaced, and there is a tear between the longitudinal and circular fibers. The longitudinal muscle remains attached to the scleral spur (Figure 1).



(Courtesy of Donald S. Minckler, MD.)

Figure 1. A histological section of angle recession illustrates the tear between longitudinal and circular fibers, with longitudinal fibers still attached to the scleral spur. There is retrodisplacement of the iris root.

CLINICAL APPROACH AND TREATMENT

Patients who suffer blunt trauma to the eye will require a thorough slit-lamp examination and a careful, detailed characterization of the angle structures via gonioscopic examination. Initially, gonioscopy may not be possible, owing to iritis or pain. This examination may be delayed until the patient can cooperate. Clinical clues such as hyphema, iridodialysis of the iris root, and/or cyclodialysis will guide the clinician to pursue gonioscopy sooner than later to confirm angle recession.

A gonioscopic examination reveals a deepening of the angle in which the exposed face of the ciliary body appears to be wider than usual and the iris root seems to be posteriorly displaced. In the example presented in Figure 2, exaggerated widening of the ciliary body face is visible after a blunt injury.

The initial treatment of ARG is medical. Medications typically used to treat open-angle glaucoma sometimes suffice; aqueous suppressants such as α -agonists, topical carbonic anhydrase inhibitors, or β -blockers are options. Of course, the judicious use of steroid therapy with fast



Figure 2. A gonioscopic examination of an eye with angle recession shows deepening and widening of the ciliary body.

tapering is required to address the concomitant iritis from the blunt injury. Laser trabeculoplasty is relatively ineffective and should be avoided. Cyclodestructive procedures should also be avoided, except in cases where there is limited visual potential.

When maximally tolerated medical therapy fails to control the IOP adequately, filtering surgery may be indicated. Mermoud et al compared standard trabeculectomy, trabeculectomy with antimetabolites, and the implantation of a Molteno device (IOP Ophthalmics) in the eyes of patients with uncontrolled ARG.¹² Trabeculectomy with antimetabolites was the most effective at controlling the IOP with the fewest postoperative antiglaucoma medications, but the rate of bleb-related infection was also highest in this study group.

CONCLUSION

Glaucomatous optic neuropathy can be a devastating consequence of angle-recession blunt injury. Early diagnosis and aggressive intervention to lower the IOP are of the utmost importance. Once the sequela of the injury (eg, hyphema) resolves, appropriate counseling is the next crucial step. Physicians must educate patients on their injury so that they understand their lifetime risk of developing glaucoma. Careful lifelong monitoring of their IOP and examinations of their optic nerves is recommended for patients who experience angle recession, because glaucoma is usually an asymptomatic disease. ■

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