Clinical Approach to Angle-recession Glaucoma

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

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Collins provided the first pathologic description of angle recession resulting from blunt trauma. 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. 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%. Approximately 2.4 million ocular injuries occur in the United States each year. One study reported a 19% risk of developing glaucoma after closed-globe contusion, a rate approximately six times higher than after a penetrating injury. 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. Greater recession of the angle (180º or more) may be predictive of a higher incidence of developing glaucoma. Interestingly, up to 50% of patients whose angle recession progresses to glaucomatous optic neuropathy will develop glaucoma in the fellow uninjured eye. 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.

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, 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.

Pathologically, the recession of the anterior chamber angle appears as a separation between the longitudinal and circular fibers of the ciliary body muscle. 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 2.  A gonioscopic examination of an eye with angle recession shows deepening and widening of the ciliary body.