ANGLE RECESSION GLAUCOMA

by

Julie A. Slough
Optometry Intern

Senior Research Project
Literary Search
April 9, 1987

Contents

ANGLE RECESSION GLAUCOMA

Abstract

Angle recession glaucoma is due to blunt trauma and presents with an elevated intraocular pressure up to years after onset due to anterior chamber angle damage and a resultant decrease in aqueous outflow. A thorough case history and careful gonioscopic exam serve most valuable in the detection and diagnosis. Unilateral angle recession, widening of the ciliary band, and unusual chamber angle are common signs. Evidence of angle recession indicates a need for medical or surgical intervention depending on the degree of damage, and warrants careful yearly gonioscopic and fundus exams for the rest of the patient's life to enable early detection of late onset glaucoma.

Key Words: angle recession glaucoma, blunt injury, trabecular meshwork, iridodialysis, cyclodialysis, hyphema, aqueous outflow, secondary glaucoma, gonioscopy.

Introduction

Angle recession glaucoma (also called contusion or cleavage glaucoma) is characterized by unilateral damage to the anterior chamber angle, reduction in aqueous outflow, and secondary glaucoma.[1,2,3] Elevated intraocular pressure (IOP) in the injured eye may be present immediately following trauma, or it may be delayed for many years.[4] Clinically, patients with angle recession glaucoma are usually detected during a routine eye examination later in life.[3]

A blunt, nonpenetrating injury to the eye may lead to acute or chronic secondary glaucoma due to a variety of mechanisms.[2] These may include an acute IOP elevation due to a transient anterior uveitis, an intraocular hemorrhage, dislocation of the lens, or trabecular damage. An injury causing one or more of these manifestations should alert the clinician to the possibility of a second, delayed IOP elevation after the initial rise has subsided. Trabecular damage, in particular, may frequently lead to chronic IOP elevation.[5] This rise in pressure may occur many months, years, or even decades after the ocular injury. Once it has developed, glaucoma due to visible disruption of angle structures is usually a permanent condition.[3]

Ocular Signs

There are numerous ocular or periocular manifestations which may indicate blunt trauma and the possibility of angle-recession glaucoma. Besides the obvious signs of an abnormal or deepened angle, there may be evidence of blood staining of the cornea, flare in the anterior chamber, pigmentation of the ciliary body band and trabecular meshwork (T.M.), lens displacement, and cataracts.[4] Cataracts may occur in 24% of patients

with contusion injuries, varying from hypermature to cortical types.[6] Additional manifestations include optic nerve atrophy and glaucomatous cupping, retinal detachments and holes, hypotony, increased IOP, and vitreal hemorrhages. Any of these injuries should prompt further investigation into the possibility of an angle recession.[4]

A recessed angle is a sign of trauma to the eye and is a rough measure of the severity of the injury.[4] From 4 to 9% of patients with angle recession of up to and exceeding 180° will eventually develop a chronic glaucoma.[5] From 1 to 20% of such patients develop late onset glaucoma.[3,4]

While recessions of less than 180° don't carry much of a risk, recessions of equal to or greater than 240° are especially worrisome. Therefore, it is important to look specifically for recessed angles in all cases of unilateral glaucoma and particularly in those with a history of blunt injury.[4]

Related Injuries

A blunt blow to the eye results in a sudden increase in IOP and a variety of injuries to the intraocular contents. Angle recession is thought to occur as a result of aqueous being pushed posterior and laterally, and by the stretching of the posterior sclera causing the iris to be suddenly forced against the lens.[2,3,4] The valvelike action of the iris prevents aqueous humor from running back through the pupil, and the resulting pressure may cause a blowout or tear of the anterior face of the ciliary body. This is called an iridodialysis.[2,4,7] This usually occurs between the circular and radial (longitudinal) muscles of the ciliary body.[3,4,5]

Another associated injury is the separation of the ciliary body from the scleral spur, called a cyclodialysis.[5,7,8] This may lead to severe hypotony and late cataract formation.[2]

Trauma that is sufficient to cause an angle recession is often also associated with a hyphema due to the rupture of ciliary vessels. This may, in fact, be the initial finding when a patient presents for evaluation of a blunt eye injury.[3,5,7] The majority of patients with angle recession glaucoma have had a prior hyphema, with a prevalence ranging from 60-94%.[4,5] It is interesting to note that even after 10 years, 6% of hyphema patients have been reported to develop late onset glaucoma.[4]

Although angle-recession is very common after traumatic hyphema, the vast majority of eyes with recessed angles fail to develop glaucoma or any evidence of outflow impairment.[2,4]

Mechanism of Outflow Impairment

It has been suggested that although an angle recession provides evidence of past injury, it is possibly not the actual cause of the glaucoma. Initial trauma to the T.M. may stimulate proliferative and/or degenerative changes in the trabecular tissue which could lead to obstruction of aqueous outflow.[5]

Suggested mechanisms causing these changes may involve cyclodialysis, meshwork sclerosis or fibrosis, hyaline membrane formation, or, in some patients, an underlying open-angle glaucoma.[6]

It has been demonstrated through human observations and animal studies that tears in the T.M. during the early post-trauma period produce a flap of trabecular tissue which is hinged at the scleral spur.

Over time, scarring occurs causing the initial trabecular injury to be less apparent while leading to chronic obstruction in portions of the aqueous outflow system.[5]

This theory is supported by the fact that the majority of eyes which eventually develop glaucoma after blunt injury appear to have an underlying predisposition to reduced aqueous outflow, as evidenced by frequent alterations of IOP in the fellow eye. Along with these changes within the T.M., an endothelial layer with a Descement's-like membrane may extend from the cornea over the anterior chamber angle. These alterations can make a diagnosis based on gonioscopic appearances of the T.M. difficult.[5]

Often, there is a delay in onset of elevated IOP. This period of low pressure before onset of glaucoma may be attributable to a tear through the ciliary muscle to the suprachoroidal space. This allows the aqueous to have a direct access to Schlemm's canal resulting in a pressure-lowering effect until the wound in the T.M. heals. Acute onset of severe glaucoma following the hypotony may be related to spontaneous closing of the traumatic cyclodialysis cleft.[8]

Eyes in which traumatic recession or disruption of the angle is limited to a small portion of the circumference seldom develop late secondary changes or intractable glaucoma.[8]

Types of Angle Recession Glaucoma

There are three types of glaucoma associated with damage to angle structures--the early type, the late type, and the very late type.[2]

The most common is the early type which can develop up to two years after the injury. The severity relates to the visible extent of

the injury. When mild, this glaucoma may persist indefinitely without getting worse. This early type is thought to be directly caused by damage to the T.M. or from cyclodialysis, either of which can result in a decrease in aqueous permeability and outflow.[2]

The late type of glaucoma develops in a small percentage of injured eyes several years after the original trauma, and is usually seen in eyes with disruption of at least 3/4 of the angle circumference. It is also characterized by a subnormal tonographic facility of outflow from the time of injury.[8] This decrease in outflow may be a type of mechanism used to compensate for obstruction of outflow channels.[4]

The very late type of glaucoma may develop decades after injury and is due to secondary changes affecting the corneoscleral meshwork. These changes may involve sclerosis or scarring within the meshwork or the development of an endothelial and hyaline membrane covering due to unknown mechanisms.[8]

It is not known how to distinguish between the early and late types of glaucoma by clinical examination, but only by history and by the course of development. However, gonioscopy reveals signs in the angle that are important in establishing the diagnosis of traumatic glaucoma in both types.

Gonioscopic Findings

An accurate history and a comparison of the gonioscopic findings between the two eyes is essential in making the correct diagnosis of angle recession glaucoma.[3,4,7] Fortunately, this type of glaucoma is readily differentiated from primary glaucoma by the appearance of the angle and its unilaterality.[2]

Gonioscopy commonly reveals a recessed angle which may involve only one small part or up to 360° of the iris root.[4,7,8] Fortunately, in most instances, the recession occupies less than 50% of the angle.[3]

The most important characteristic sign is widening of the distance from the scleral spur to the iris root (the root is posteriorly displaced[4]) resulting in a broadened ciliary band in the area of the recession. A localized deepening of the anterior chamber is frequent, and remnants of torn iris tissue and scattered pigment may be seen along the margins of the tear.[2] This can sometimes lead to the incorrect diagnosis of pigmentary glaucoma, especially if a Krukenberg Spindle has been produced.[3]

The iris may have small radial splits or notches at the pupillary margin, or rarely, may be torn at the root.[8] The iris sphinctor may be ruptured resulting in an irregularly dilated pupil.[3,7]

In eyes having the least visible damage, the ciliary band is minimally, if even noticeably, wider than in the fellow normal eye. However, disrupted strands of uveal meshwork on the ciliary band can be seen, leaving the ciliary muscle bare and of a different appearance than the other eye. These strands rupture near their middle and the ends tend to contract into small balls of tissue which can be seen in the region of the scleral spur, both on the posterior edge of the corneoscleral T.M. and on the anterior portion of the ciliary band. Also, the scleral spur may stand out abnormally white and distinct in the injured eye as compared to the normal eye.[4]

In more severe disruptions of the angle, the affected areas of the ciliary band may appear very broad, with the space between the iris

root and angle wall greatly widened due to the tearing of the ciliary muscle itself.[4] The torn ciliary muscle is light gray and delicate, resembling wool or cotton batting (vs. pink and vascular skeletal muscle). The tear does not usually extend all the way through the muscle to the sclera, but if it does, the white scleral wall will be exposed posterior to the scleral spur and will be much whiter and solid appearing than the ciliary muscle.[4,8] Anterior ciliary arteries are often seen on the inner surface of the sclera in this case.[8]

Immediately after blunt trauma, a disruption in the filtration portion overlying Schlemm's canal can be seen as a sharp-edged, slightly gaping, linear opening resembling an incision. This rip may extend into the canal itself, but the sharp edges gradually become rounded and the whole wound in the meshwork is converted to an inconspicuous shallow depression or trough just anterior to the scleral spur.[8] In some cases this region of the cleft is filled with fibrous tissue which can obscure the recession.[3] It is at this point that there exists no direct communication from the anterior chamber to Schlemm's canal as proven by an elevation in IOP.[8]

Peripheral anterior synechias adjacent to disrupted areas of the angle can be caused by hemorrhages and an apposition of torn surfaces.[4] This is commonly accompanied by a rise in IOP.[7] However, synechias are rarely a significant factor in the development of glaucoma after blunt trauma.[8]

Occasionally the signs of angle recession glaucoma are quite subtle, and only back-and-forth comparison between the two eyes reveals the abnormality.[2] It is often helpful to use binocular Koeppe gonioscopic lenses while evaluating the anterior chamber so that comparisons

of the angles can be made more quickly and easily.[8]

Management

Response to treatment of recession-angle glaucoma varies widely and is related chiefly to the nature and extent of the changes in the angle. The more extensively the angle is damaged, the less responsive the glaucoma tends to be to treatment.[8]

This type of open-angle glaucoma is treated medically and/or surgically. Minimally injured eyes usually respond well to treatment with miotics. Pilocarpine (1 to 4%), timolol (0.25 or 0.5%), and epinephrine (0.5 to 2.0%) are the topical drugs of choice, keeping in mind that rarely a paradoxical elevation of IOP occurs in response to pilocarpine. In unresponsive cases, atropine may forestall the need for systemic or surgical therapy.[1,3,4,8] If these drugs are not effective due to scarring and a decrease in aqueous outflow, anticholinesterases or systemic carbonic anhydrase inhibitors may be of use by decreasing aqueous production.[4,5,8]

When a major part of the angle has been damaged, miotics are generally less effective since there is less pulling action on the scleral spur and T.M. due to the ruptured angle.[6,8] In cases of late onset where excessive tears have caused secondary scarring or membrane formation, the obstructive changes in the outflow channels are probably not reversible by any form of medical treatment. Surgical therapy may be required in these cases.[3] Unfortunately, there is no operation that is consistently successful in the treatment of recessed-angle glaucoma secondary to trauma.[4]

If the glaucoma is severe and maximum medical treatment fails to

control the IOP, or if there is progressive visual field loss associated with optic nerve damage, a filtering operation needs to be done.[4,5,8] Trephination of the sclera with a peripheral iridectomy is the procedure of choice. Although trabeculectomy and other filtering procedures have a lower complication rate, they are no more effective than other procedures.[4] Similarly, laser trabeculoplasty brings unreliable results, and IOP may even be worsened by this particular therapy.[8]

Patients with known damage to the angle structures from blunt trauma should be kept under observation and be followed yearly for the rest of their lives because of the possibility of late development of glaucoma.[4]

References

1. Bartlett JD, Jaanus SD. Clinical Ocular Pharmacology, Stoneham, Butterworth; 1984; pp. 907,908.
2. Kolker AE, Hetherington JJr. Diagnosis and Therapy of the Glaucomas, 5th ed. St. Louis, C.V. Mosby Co.; 1983; pp. 288, 290, 311, 312.
3. Henkind P. Atlas of Glaucoma, Medical Dialogues, Inc.; 1984; pp. 55-56.
4. Fraunfelder FT, Roy FH. Current Ocular Therapy, Philadelphia, Saunders; 1980; pp. 468, 469.
5. Shields MB. A Study Guide for Glaucoma, Baltimore, Williams and Wilkins; 1982; pp. 324, 325, 327.
6. Duane TD. Clinical Ophthalmology, vol. 3, chapter 55. Philadelphia: Harper and Row, 1982; p. 17.
7. Bellows JG. Contemporary International Concepts, Masson Publishing USA, Inc.; 1979; pp. 171-173.
8. Epstein DL. Glaucoma, 3rd ed. Philadelphia, Lea and Febiger; 1986; pp. 301-307.