EMERGENCIES IN GLAUCOMA: A REVIEW

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ABSTRACT

Most severe eye diseases and injuries ultimately require intervention by an ophthalmologist. The urgency of referral depends on various factors, including level of vision loss, duration of symptoms, and presence of comorbid diseases. Of special importance are five acute eye problems in which emergency management by primary care physicians can be critical to visual outcome: high-velocity injuries, chemical injuries, acute angle-closure glaucoma, arteritic ischemic optic neuropathy, and central retinal artery occlusion. This paper will focus on emergencies in glaucoma, especially primary and secondary closed-angle glaucoma and secondary open-angle glaucoma. Delay in presentation, and unresponsiveness to medical treatment of the acute ocular hypertension attack carry a significant risk of chronic glaucoma, whatever the mechanism of disease is. Whenever the acute glaucoma crisis does not cease with an appropriate medical treatment, a laser and/or surgical intervention needs to be urged.

RÉSUMÉ

La plupart des maladies ou traumatismes de l'œil nécessitent l'intervention d'un opthalmologue. L'urgence de la consultation dépendra de plusieurs facteurs tels que le niveau de la perte visuelle, la durée des symptômes avant la présentation aux urgences du patient, la coexistence de maladies morbides. Cinq maladies oculaires nécessitent une prise en charge urgente: les traumatismes oculaires, les brulures chimiques, la crise de glaucome aigu, la neuropathie optique ischémique artéritique et l'occlusion de l'artère centrale de la rétine.

Différentes présentations de glaucome nécessitent une prise en charge immédiate, que ce soit les glaucomes primitifs et secondaires par fermeture de l'angle irido-cornéen et les glaucomes secondaires avec ouverture de l'angle irido-cornéen. Le délai de présentation et l'échec du traitement médical de l'hypertension intra-oculaire représentent un risque significatif au développement d'un glaucome chronique, quel que soit le mécanisme de l'augmentation de la pression intra-oculaire. Devant une crise de glaucome réfractaire au traitement médical classique, une intervention au laser et/ou chirurgicale devra être réalisée sans délai.

KEY WORDS:

Emergency, glaucomas, treatment, classification.

MOTS-CLÉS

Urgence, glaucomes, traitement, classification.

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INTRODUCTION

Emergencies in glaucoma gather a variety of eye diseases but acute angle-closure glaucoma remains of special importance. In fact visual outcome following an acute attack depends on the emergent referral by primary care physicians. The patients will generally complain about eye pain and redness, accompanied by nausea and vomiting making difficult the differential diagnosis with gastrointestinal pathologies. Sometimes the patients report blurred vision as multicoloured halos around lights. Besides primary closed-angle glaucoma, this paper will focus on secondary closed or open-angle glaucoma. Generally speaking secondary closed angle glaucomas are usually caused by peripheral anterior synechiae and secondary open angle glaucomas are developed by cells or debris blockage of the trabecular meshwork or by damaged outflow channels or secondary to corneoscleral and extraocular diseases or by combined mechanisms. Whatever the cause of the acute glaucoma, if the crisis does not cease with an appropriate medical treatment, laser and/or surgical interventions are recommended to avoid a chronic outcome and though alive treatment.

Part I. Clinical presentation

A. ADULT-ONSET CLOSED-ANGLE GLAUCOMAS

I. Primary acute closed-angle glaucoma (with pupillary block)

BACKGROUND

Primary angle closed-angle glaucoma is initiated by excessive iris-lens apposition, which impedes the flow of aqueous humor from the posterior chamber to the anterior chamber with the development of relative pupillary block. The resultant pressure gradient between the posterior and anterior chamber causes a forward bowing of the peripheral iris so that the iris occludes the filtering portion of the trabecular meshwork (appositional angle closure), which can lead to elevation of intraocular pressure (IOP). Prolonged contact of the peripheral iris with the trabecular meshwork may lead to formation of peripheral anterior synechiae and

functional damage to the trabecular meshwork (6).

CLINICAL CHARACTERISTICS OF THE DISEASE

I. Acute onset of elevated intraocular pressure. Aqueous humor dynamics are usually normal prior to the onset of the acute attack.
II. Closed iridocorneal angle on gonioscopic examination.
III. Shallow anterior chamber on slit-lamp examination. The fellow eye usually will manifest a narrow and potentially occludable iridocorneal angle.
IV. The conjunctiva is hyperemic, the pupil is mid-dilated and nonreactive to light, and the cornea may be cloudy.
V. Vision is reduced and the patient may complain of seeing colored halos around lights due to the corneal edema.
VI. Ocular pain is usually present.
VII. The patient may also have systemic complaints secondary to autonomic stimulation, including nausea, vomiting, and bradycardia.
VII. Many patients with primary acute angle-closure glaucoma have a past neglected history of intermittent episodes of angle-closure symptoms.
IX. The basic ocular abnormality is present in both eyes, which is manifested by the following:
    a. A small anterior segment with hyperopia, a small cornea, and a well developed ciliary body. The iris may insert anteriorly on the ciliary body.
    b. A reduced anterior chamber depth with an increased diameter to the lens which is located anteriorly.

MANAGEMENT

Management of suspects

I. Patients with asymptomatic narrow angles do not usually require medical therapy. We rarely use prophylactic pilocarpine therapy in these patients and *we prefer to perform a prophylactic laser iridotomy* if we have the following concerns regarding the narrow-angle suspect:
    a. the presence of signs or symptoms compatible with intermittent angle- closure. This includes the finding of peripheral anterior synechiae on indentation gonioscopy.
    b. appositional closure of the angle for greater than 180°. This may lead to trabecular dysfunction and progressive decrease in outflow facility.
    c. the systemic use of medications which are associated with the precipitation of an acute attack.
    d. a strong family history of acute angle-closure glaucoma or if the patient lives in a remote area, distant from ophthalmologic care.
II. If laser iridotomy is to be performed, a miotic should be used before treatment to facilitate the surgery.

Management of an acute angle-closure attack

I. The acute attack is treated medically in conjunction with the performance of a laser iridotomy. Sometimes topical agents (a beta-blocker and pilocarpine) are effective in terminating an acute attack. However, the iris sphincter muscle may not respond to miotics in the presence of a high intraocular pressure.
    For this reason the primary treatment usually is a hyperosmotic agent such as intravenous mannitol or oral glycerol (5).
    Carbonic anhydrase inhibitors and beta-blockers may also be effective during an acute attack in lowering intraocular pressure and permitting the miotic to function to open the iridocorneal angle.
II. Termination of the attack is attended by
    a. an opening of the closed iridocorneal angle and a deepening of the anterior chamber. This must be determined by gonioscopy.
    b. the pupil becoming miotic.
III. If the attack cannot be broken with medical therapy, a laser iridotomy or a surgical iridectomy should be performed (5).
IV. In case of refractive attack, the removal of the lens by an experienced surgeon should be done (5).

Management of the fellow eye

I. The fellow eye is at increased risk to have a spontaneous attack of acute angle-closure glaucoma, approximatively an 80 % risk within 5 years.
II. Prophylactic pilocarpine eye drops in the fellow eye associated with a laser iridotomy reduce the risk of a spontaneous attack.

Chronic closed-angle glaucoma (creeping angle-closure) and management

I. This is an insidious form of closed-angle glaucoma in which the iridocorneal angle slowly closes from the periphery to Schwalbe's line without signs or symptoms of intermittent or acute attacks of angle closure.
II. This condition may be more common in Asian and Black patients and presents as an open-angle glaucoma. However, gonioscopy demonstrates synechial closure of the angle. Closure usually begins superiorly and progresses inferiorly.
III. This condition can be cured by laser iridotomy if detected early but medical therapy is usually required after the iridotomy. Unfortunately, many patients with this type of glaucoma are diagnosed after total synechial angle closure, and filtration surgery is the only effective method of therapy (5).

II. Primary closed-angle glaucoma (without pupillary block)

Primary plateau iris

I. Shows gonioscopic finding of a closed anterior chamber angle with a flat iris plane as opposed to the forward bowing observed in closed-angle glaucoma with pupillary block (6).
II. The central chamber depth is normal in contrast to the shallow depth observed in primary closed-angle glaucoma.
III. The mechanism for angle-closure involves angle crowding. Relative pupillary block may be only a minor factor in the cause of the acute attack. This factor is eliminated by performing a laser iridotomy.
IV. Long-term treatment with pilocarpine is required after the iridotomy to prevent angle crowding and angle closure. A peripheral laser iridoplasty may be performed in order to reduce the angle crowding and to prevent the angle closure. If significant peripheral anterior synechiae are present, as a result of previous episodes of angle closure, treatment with other agents, similar to the therapy of chronic angle-closure glaucoma, may be needed.
V. If trabecular damage or peripheral anterior synechiae with reduced outflow angle glaucoma are present, filtration surgery is the only effective method of therapy.

B. ADULT-ONSET SECONDARY GLAUCOMAS

Various conditions can cause secondary open-angle or closed-angle glaucoma. In some conditions management is in a similar manner to primary open-angle glaucoma, e.g., pigmentary and pseudoexfoliative glaucoma. Other conditions require therapy of the underlying etiology, e.g., uveitic or steroid-induced glaucoma. However, many times, the underlying cause for the glaucoma cannot be removed.
In addition, the disease process may cause alterations in outflow facility and the glaucoma persists following treatment of the underlying disease process. In these cases, continued medical therapy is required. While there are a large number of conditions which can be associated with secondary glaucoma in the adult patient, only the more frequently encountered conditions are covered in this section.

SECONDARY CLOSED-ANGLE GLAUCOMA

Many conditions relating to pupillary block formation, a secluded pupil or on anteriorly dislocated lens, result in secondary closed-angle glaucoma. In addition, peripheral anterior synechiae can result in chronic closure of the iridocorneal angle. It is important to determine whether or not pupillary block is present. If not, iridotomy will be of no benefit and is not indicated (6).
Ocular disorders which can lead to secondary closed-angle glaucoma include the following:
1. Rubeosis iridis

2. Ciliary body swelling, cysts, or inflammation
3. Posterior segment tumors
4. Scleral buckling procedures and panretinal photocoagulation
5. Lens induced conditions: either a swollen and mature lens or a lens which is dislocated or which is subluxated into the anterior chamber
6. Posterior synechiae to the lens (iris bombé), e.g., uveitis, or to the vitreous in aphakia, or to an intraocular lens in pseudophakia

7. Malignant glaucoma (ciliary block of aqueous misdirection)

A. Malignant glaucoma is observed most frequently following a filtering surgery and less often a cataract extraction This is a rare complication consisting of a flat anterior chamber with an elevated intraocular pressure in the presence of a patent iridectomy. Aqueous humor appears to flow posteriorly into the area of detached vitreous and forces forward the iris-lens diaphragm in the phakic eye, or the iris-intraocular lens implant in the pseudophakic eye.
B. The condition is more common in eyes with chronic angle-closure glaucoma. If the condition develops in one eye it may be more common in the fellow eye.
C. Medical treatment is successful in about 50 % of the cases and includes the following:
    1. Administration of a systemic hyperosmotic agent
    2. Maximal mydriasis achieved with cycloplegics. If this therapy is effective in treating the attack, atropine must be continued permanently.
    3. Topical beta-blocker and systemic carbonic anhydrase inhibitor administrated to lower the intraocular pressure.
D. Argon laser treatment of visible ciliary processes through the iridectomy may be successful in the management of phakic malignant glaucoma.
E. Nd-Yag laser treatment to the anterior hyaloid face may be successful in pseudophakic malignant glaucoma.
F. If conservative treatments, including the use of laser treatment, are not effective, a pars plana vitrectomy in the phakic eye is required. The vitrectomy should remove central vitreous as well as the pocket of posterior fluid. The anterior chamber is reformed following the procedure. Occasionally lens removal is needed in combination with the vitrectomy in the phakic eye.

8. Iridocorneal endothelial (ICE) syndrome

I. The syndrome is a spectrum of disorders which are typically unilateral and occur predominantly in white women. The condition is most often recognized in early to middle adulthood (3).
II. The syndrome is divided into three clinical variations as characterized by alterations in the iris.

A. Progressive (essential) iris atrophy.
Corectopia and atrophy of the iris lead to hole formation in quadrants away from the direction of the pupillary displacement. In addition, hole formation can occur independent of corectopia, secondary to ischemia of the iris.

B. Chandler's syndrome.
Corectopia is mild or absent and iris atrophy is mild and limited to the superficial stroma. Corneal abnormalities are the major component of this syndrome.

C. Cogan-Reese (iris nevus) syndrome.
Areas of nodular or diffuse pigmented lesions appear on the surface of the iris. Iris atrophy can be mild of severe.

III. ICE syndrome also has the following additional characteristecs.
    A. Corneal alterations demonstrated by diffuse endothelial cell defects on specular microscopy. This is the major defect in Chandler's syndrome and can be associated with corneal decompensation with minimally elevated intraocular pressure.
    B. Peripheral anterior synechiae which extend to or beyond Schwalbe's line.
    C. Histologically a Descemet-like basement membrane covers portions of the chamber angle and may be associated with synechial closure.
IV. Secondary glaucoma is related to impaired outflow facility due to involvement of the angle with the Descemet-like membrane or by synechiae formation.

This may develop into secondary angle-closure glaucoma.

V. Management of patients with the ICE syndrome consists of therapy of the corneal edema with topical hyperosmotic agents and bandage soft contact lens.
Management of the glaucoma can usually be achieved initially with medical therapy; agents which reduce the rate of aqueous humor formation (e.g.topical beta-blockers and systemic carbonic anhydrase inhibitors) being more effective than agents which increase aqueous humor outflow. Surgical intervention is required to control the glaucoma in many eyes with this condition but has a lower success rate than in primary glaucoma.

SECONDARY OPEN-ANGLE GLAUCOMA

Pigmentary glaucoma

I. Pigmentary glaucoma is a bilateral disorder usually affecting young myopic men. The disease is very rare in black patients.

II. It is characterized by pigment loss (pigment dispersion syndrome) from the iris pigment epithelium. This occurs in a radial pattern, particularly in the midperiphery. The gonioscopy reveals a concave-shaped iris creating a reverse pupillary block (1,2).

III. Pigment is deposited in characteristic locations in the anterior segment: on the endothelial surface of the cornea, in the pattern of a base-down triangle (Krukenberg's spindle), on the iris surface, in the trabecular meshwork and on the lens equator and zonules.

IV. Pigmentary dispersion syndrome can occur with or without an elevated intra-ocular pressure. The relative risk for the development of glaucoma in patients with pigment dispersion is unknown.

V. Pigment release into the anterior chamber can occur after pupil dilation or physical exercise. Pigment release decreases with increasing age. Pigment accumulates in the trabecular meshwork and decreases outflow facility, which results in the increase in intraocular pressure.

VI. Management of the condition includes early Yag-laser iridotomy to release the inverse pupillary block with, if necessary, laser Argon trabeculoplasty. Finally trabeculectomy may be performed in case of uncontrolled intraocular hypertension.

Pseudoexfoliation (exfoliation) glaucoma

I. An unilateral or bilateral (37 %) disorder which increases in frequency with age, particularly after the age of 65 years. Incidence is higher among people of Danish, Scandinavian and Greek extractions. The condition is uncommon in American blacks.

II. Pseudoexfoliation is found incidentally without evidence of glaucoma in 2 % to 20 % of elderly patients. The incidence of pseudoexfoliation in patients with open-angle glaucoma may be as high as 50 %, depending on the population under study.

III. It is characterized by flakes and sheets of exfoliative material on structures of the anterior segment. The material has a close association with amyloid. The origin of the material is an abnormal epithelial basement membrane with a multifocal origin: lens, ciliary body, iris pigment and trabecular epithelium.

IV. Exfoliative material is deposited on these structures. Deposition within the trabecular meshwork results in reduced outflow facility and an associated elevation in intraocular pressure.

V. Pseudoexfoliation is also associated with increased pigment release within the anterior segment. A pigment line (Sampaolesi's line) can be detected anterior to Schwalbe's line on gonioscopy. Iris transillumination may reveal defects near the pupillary sphincter (1,2).

VI. Management of pseudoexfoliation glaucoma is similar to that described for ocular hypertension and primary open-angle glaucoma.

Steroid-induced glaucoma

I. Topical, periocular, intravitreal and all forms of systemic administration of corticosteroids (i.e. per os, cutaneous ointments, intraarticular injection) can result in a sec-

ondary open-angle glaucoma in susceptible individuals (4).

II. Diagnosis of steroid-induced glaucoma requires a high index of suspicion and the questioning of patients specifically about their use of steroid eyedrops, ointments, skin preparations, and pills.

III. The key to management is stopping the corticosteroid. If the drug is used for treatment of intraocular inflammation, e.g., uveitis, or postoperatively after ocular surgery, the steroid preparation should be tapered or switched to a less potent preparation such as fluorometholone.

IV. Intraocular pressure returns to baseline over a few weeks to several months after the steroid is stopped. During this period, treatment with standard antiglaucoma medications may be required to control the intraocular pressure. Steroid-induced glaucoma responds poorly to argon laser trabeculoplasty. Steroid-induced-glaucoma resistant to standard medical management requires filtration surgery.

Lens-induced glaucoma *(4)*

I. Secondary open-angle glaucoma can result from
   A. Phacolytic (lens protein) glaucoma.
   B. Retained lens protein following extracapsular cataract surgery.
   C. Phacoanaphylaxis.

II. Secondary closed-angle glaucoma can result from
   A. An intumescent lens
   B. A dislocated lens either into the anterior chamber or into the vitreous.
      This can be secondary to trauma, Marfan's syndrome, homocystinuria, and numerous other rare conditions.
   C. A microspherophakic lens (Weill-Marchesani syndrome).

Neovascular glaucoma

I. Rubeosis iridis and growth of a fibrovascular membrane over the anterior chamber angle causes neovascular glaucoma. The glaucoma is initially open-angle with abnormal blood vessels and fibrous tissue overlying the trabecular meshwork. This progresses to peripheral anterior synechiae with continuing closure ("zippering") of the angle (1,2).

II. The earliest detectable finding of rubeosis iridis is the breakdown of the blood-aqueous barrier with fluorescein leakage from the iris vessels. The new vessels usually first make their appearance at the pupillary margin and progress onto the surface of the iris resulting in ectropion uveae. Finally the vessels will grow into the iridocorneal angle and onto the trabecular meshwork.

III. Various systemic and ocular diseases can cause rubeosis iridis. The more common include retinal occlusive vasculopathies associated with extensive retinal ischemia as diabetic retinopathy, and central retinal vein occlusion.

IV. Therapy
   Treatment of neovascular glaucoma is directed to therapy and prevention of rubeosis iridis. Glaucoma treatment is dictated by the visual potential of the eye.
   A. Panretinal photocoagulation.
      Neovascular glaucoma is very difficult to manage. Retinal photocoagulation is aimed to eliminate the ischemic retinal area and cause regression of the rubeosis iridis. If performed at a stage prior to synechial closure of the angle, panretinal photocoagulation can prevent the severe secondary angle-closure glaucoma. The importance of treatment in central retinal vein occlusion relates to the ischemic type with areas of capillary nonperfusion that is highly associated with the development of rubeosis iridis.
   B. Medical management.
      Topical corticosteroids and atropine are administered in response to intraocular inflammation due to breakdown of the blood-aqueous barrier. Atropine also increases uveoscleral outflow, the only mechanism by which aqueous humor can exit the eye if the iridocorneal angle is totally sealed, and may therefore lower intraocular pressure. Many patients without visual potential are comfortable with this combined therapy in spite of an elevated intraocular pressure. Topi-

cal beta-blockers and systemic carbonic anhydrase inhibitors are used to lower the intraocular pressure. Miotics should be avoided since they are ineffective if the angle is totally closed, may cause additional intraocular inflammation and decrease uveoscleral outflow.

C. Other procedures: Diode laser, vitrectomy or filtrating surgery, tubes or valves. More recently, intravitreal triamcinolone injection has been proposed for its anti-inflammatory, analgesic and anti-angiogenic actions which are usually helpful to manage this painful condition.

Uveitis glaucoma

I. Uveitis can cause a secondary open- or closed-angle glaucoma due to the following mechanisms.
   A. Open-angle due to blockage of the trabecular meshwork by cellular debris or protein as well as a direct inflammatory effect (trabeculitis) on the outflow channels.
   B. Closed-angle glaucoma due to posterior synechiae and pupillary block or peripheral anterior synechiae.

II. Various uveitis conditions associated with glaucoma include the following (1,2):
   A. Glaucomatocyclitic crises (Posner-Schlossman syndrome) corresponds to an *unilateral* elevated pressure associated with reccurent attacks of mild cyclitis. The iridocorneal angle is open and the pupil is larger, not miotic, in the involved eye. The attacks last hours to weeks. While the intraocular pressure is usually normal between attacks, this condition can be associated with later development of open-angle glaucoma.
   B. Fuchs' syndrome of heterochromic cyclitis consisting of the triad of heterochromia, a mild and chronic form of cyclitis which characteristically does not respond to corticosteroids, and the late development of cataract.
   C. Syphiliis can cause glaucoma during the active inflammatory stage. Also, in eyes with interstitial keratitis, secondary open- or closed-angle glaucoma may later develop.
   D. Sarcoid uveitis frequently causes a secondary open-or closed-angle glaucoma.
   E. Ankylosing spondylitis, adult rheumatoid arthritis and juvenile rheumatoid arthritis are frequently associated with uveitis and secondary glaucoma.
   F. Herpes simplex, herpes zoster, rubella, and mumps can have an associated uveitis and secondary glaucoma.

III. Management
   A. Open-angle
      Therapy is a balance between treatment of the inflammation and the elevated intraocular pressure.
      The uveitis is treated with a combination of topical, periocular or systemic corticosteroids and topical cycloplegics. The ophthalmologist must be aware of a possible steroid-induced rise in intraocular pressure.
      Occasionally, systemic immunosuppressive therapy is required. Topical beta-blockers, epinephrine drugs and systemic carbonic anhydrase inhibitors are used to treat the glaucoma. Miotics (and prostaglandines) should be avoided since they increase intraocular inflammation. Argon laser trabeculoplasty is not effective, and filtration surgery has a lower rate of success in uveitic glaucoma.
   B. Closed-angle
      Following treatment of the inflammation, pupillary block requires a *laser iridotomy or a surgical iridectomy* with the subsequent medical management of glaucoma as described above. This sequential treatment is crucial in order to avoid a catastrophic inflammatory intraocular reaction. Filtration surgery may be required, especially if there are extensive peripheral anterior synechiae.

Ocular trauma *(4)*

I. Blunt trauma to the globe can result in an elevated intraocular pressure due to
   A. Mechanical obstruction of the outflow pathways by red blood cells (hyphema), blood products (hemolytic glaucoma), or degenerative red cells (ghost cell glau-

coma or Kakhi cells). In addition, inflammatory products can obstruct the trabecular pathways.
   B. Lens subluxation (lens-induced).
   C. Direct trauma to the trabecular meshwork resulting in a tear in the ciliary body and an angle recession.

II. Penetrating injuries can cause secondary glaucoma due to
   A. A flat anterior chamber resulting in permanent peripheral anterior synechiae.
   B. Hyphema
   C. Penetration or dislocation of the lens.
   D. Sympathetic ophthalmia
   E. A retained metallic intraocular foreign body, e.g., iron (siderosis) or copper (chalcosis).

III. Chemical injuries due to acid or especially alkaki compounds can cause extensive ocular damage resulting in secondary glaucoma.

Part II. Therapeutical approach

N-d YAG LASER IRIDOTOMY

Creation of a full-thickness hole in the iris with the laser has become an acceptable and preferred alternative to surgical iridectomy. Iridotomy, whether by a laser or a surgical technique, functions by eliminating pupillary block. The term laser "iridotomy" rather than laser "iridectomy" is used in this review (4).

Indications

Laser iridotomy is indicated, as well as surgical iridectomy, in all cases of angle-closure glaucoma in which pupillary block is the causative mechanism. Specific indications include the following.

I. Acute angle-closure glaucoma.
   A. Following medical termination of the acute attack
   B. To terminate a medically unresponsive acute attack.

II. Fellow eye of a patient who had an attack of acute angle-closure.

III. Chronic angle-closure glaucoma

IV. Subacute or intermittent angle-closure glaucoma

V. Prophylactic treatment of an anatomically narrow iridocorneal angle

VI. Combined mechanism glaucoma

VII. Secondary angle-closure glaucoma including ciliary block (malignant glaucoma) and aphakic pupillary block glaucoma.

VIII. To help in the diagnosis of the plateau iris syndrome.

Contraindications (surgical iridectomy is preferred in these cases)

I. An uncooperative patient.
   Inability of the patient to sit at the slitlamp.

II. Corneal edema
   A. This limits the precise focus of the laser beam on the iris surface.
   B. It reduces the amount of laser energy delivered to the iris surface.
   C. It increases the susceptibility to laser-induced corneal burns.
   D. The administration of topical glycerin may result in sufficient corneal dehydration and clearing of the edema to allow the safe performance of laser surgery.

III. A flat anterior chamber
   Contact of the iris with the cornea eliminates the normal dissipation of laser-generated heat through the aqueous. Corneal endothelial burns will therefore occur immediately, preventing further attempts at the laser procedure.

Technique

I. Topical pilocarpine 1 % or 2 % administered prior to the procedure places the iris under stretch and reduces the iris thickness, facilitating penetration.

II. Topical anesthesia is usually sufficient for performing the procedure.

III. Antireflective-coated iridotomy contact lens (e.g. the Abraham lens)

IV. Location of the iridotomy
   A. While the procedure can be performed at any clock hour, the superior iris between 10.30 and 1.30 is selected so as to have the iridotomy covered by the upper lid. We prefer the area in the superior nasal quadrant with the laser aimed away from the posterior pole.

B. The 12.00 location is avoided because of possible bubble formation and migration, which can prevent visualization of the treatment site.
C. With the pupil constricted with miotics, a site between one-half and two-thirds of the distance between the pupil margin and the visible periphery is chosen. This is approximately equivalent to one half the distance between the edge of the collarette and the limbus.
D. Treatment through a corneal arcus senilis is avoided since the density of the arcus reduces the laser power across the cornea and interferes with a clear focusing of the laser beam.
E. For dark irides, select the base of an iris crypt or a depigmented area with thinner stroma for easier penetration. In light blue or grey irides, the base of a crypt is also selected for treatment. In light-colored irides, a surface freckle that may provide improved laser absorption can be selected for treatment.

V. Laser settings
A. With certain Nd-Yag lasers the only variables are the amounts of energy used and the number of bursts per shot. Duration and spot size are constant.
B. Setting of the Nd-Yag laser is not dependent upon the color of iris, as it is with the argon laser.
C. Power settings between 2 and 8 mj are used.
D. A burst of one or two pulses per shot is sufficient to create an iridotomy. Higher numbers of pulses per shot may increase the chance of lens injury.
E. In case of acute congestive crisis, *three argon laser spots on the peripheral iris prior the iridotomy are recommended in order to avoid iris hemorrhage*

Postoperative treatment

I. Glaucoma medications are continued
II. Topical corticosteroids are administered four times a day for 3-5 days.
III. Intraocular pressure is measured 1-2 hr after treatment to detect and treat possible acute increases.

Operative complications

I. Corneal epithelial and endothelial burns
II. Iris hemorrhage
III. Laser injury to the lens
IV. Retinal burn
V. Failure to penetrate the iris
VI. Pupillary distortion

Postoperative complications

I. Visual alterations
II. Acute intraocular pressure crisis
III. Iritis
IV. Late closure of the iridotomy

SURGICAL IRIDECTOMY

I. The indications for a surgical iridectomy are similar to those for laser iridotomy and include elimination of pupillary block in acute, subacute, chronic, and secondary angle-closure glaucoma as well as in combined mechanism glaucoma (4).
II. Laser iridotomy has replaced surgical iridectomy in most patients with closed-angle glaucoma. However, surgical iridectomy may still be indicated in the following situations:
A. Inability to produce a patent laser iridotomy
B. When a laser iridotomy closes repeatedly, as in chronic uveitis.
C. Unavailability or nonfunctioning of a laser.
D. Opacification of the media preventing laser treatment, e.g. edema or scarring of the cornea, striate keratopathy or hyphema.
E. During an acute attack of angle-closure glaucoma that cannot be stopped medically in which the anterior chamber is very shallow and the iris is inflamed and thickened.
F. When a patient is unable to sit at the laser slit-lamp because of physical or mental disability.

CONCLUSIONS

Emergent glaucoma as just discussed is defined as any condition leading to a rise in in-

traocular pressure with or without associated damage to the optic nerve head or visual field. The distinction between ocular hypertension and glaucoma when one is dealing with primary open-angle glaucoma is not always applicable to the secondary glaucomas. We have no doubt that some patients with a secondary elevation of intraocular pressure are more resistant to glaucomatous optic atrophy than other individuals. However, it is difficult to apply general concepts to all of the scondary glaucomas because of the wide variation in etiologic factors, treatments and prognoses.

Therefore, the gonioscopy is probably the most important exam to perform in order to define the causes of the ocular hypertension.

This complete and summarized review of emergent glaucoma may help to clarify the etiology and guide the management.

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