with glaucomatous optic neuropathy. Because of the insidious nature of this condition, vision loss may be the presenting symptom. Accordingly, this disease tends to be diagnosed in its later stages and is a major cause of blindness in Asia. The clinical course of PACG usually resembles that of open-angle glaucoma in its lack of initial symptoms, modest elevation of IOP, progressive glaucomatous optic nerve damage, and characteristic patterns of visual field loss. Over time, however, IOP can rise precipitously and become more difficult to control. The diagnosis of PACG is frequently overlooked, and this condition is commonly confused with POAG. As previously noted, gonioscopic examination of all glaucoma patients is important to establish an accurate diagnosis.

Management

As with PAC, LPI is considered the standard of care. It has similar precautions related to eyes with extensive PAS in which a paradoxical rise in IOP can occur (see Treatment Controversies).

Medical treatment for PACG can include both aqueous suppressants and outflow drugs. Prostaglandin analogues are very effective for lowering IOP in angle-closure glaucoma, with efficacy similar to or exceeding that of β-blockers. The degree of IOP reduction does not seem to correlate with the amount of permanent angle closure. The efficacy of other outflow drugs, such as Rho kinase inhibitors, has yet to be established.

Cataract surgery alone is beneficial in reducing IOP and use of medications, and it compares favorably to cataract extraction combined with trabeculectomy. The recent EAGLE study showed that lens extraction can be an effective option in treating PACG. (See also Treatment Controversies.)

Chen R, Yang K, Zheng Z, Ong ML, Wang NL, Zhan SY. Meta-analysis of the efficacy and safety of latanoprost monotherapy in patients with angle-closure glaucoma. *J Glaucoma.* 2016;25(3):e134–144.

Tham CC, Kwong YY, Leung DY, et al. Phacoemulsification versus combined phacotrabeculectomy in medically controlled chronic angle closure glaucoma with cataract. *Ophthalmology.* 2008;115(12):2167–2173.e2.

Tham CC, Kwong YY, Leung DY, et al. Phacoemulsification versus combined phacotrabeculectomy in medically uncontrolled chronic angle closure glaucoma with cataracts. *Ophthalmology.* 2009;116(4):725–731, 731.e1–3.

Symptomatic Primary Angle Closure

IOP elevation with acute or subacute blockage of most of the angle can cause symptomatic angle closure.

Subacute primary angle closure

Subacute, or *intermittent, angle closure* is a condition characterized by episodes of blurred vision, halos, and mild pain caused by elevated IOP. Vague symptoms of pain or headache not associated with visual symptoms have a low specificity for angle closure. The visual symptoms resolve spontaneously, especially during sleep-induced miosis, and the IOP is usually normal between episodes, which occur periodically over days, months, or years. These episodes are often confused with headaches or migraines, so obtaining a careful history is required. The correct diagnosis can be made only with a high index of suspicion and

gonioscopy. The typical history and the gonioscopic appearance of a narrow angle with or without PAS help establish the diagnosis. The management of subacute primary angle closure is similar to that of PAC.

Acute primary angle closure

In *acute primary angle closure (APAC),* IOP rises rapidly as a result of relatively sudden blockage of the trabecular meshwork by the iris. APAC, which is sometimes called *acute angle-closure crisis,* is typically manifested by ocular pain, headache, blurred vision, and rainbow-colored halos around lights. Acute systemic distress may result in nausea and vomiting. The rise in IOP to relatively high levels causes corneal epithelial edema, which is responsible for the visual symptoms. Signs of acute angle closure include

- high IOP
- mid-dilated, sluggish, and irregularly shaped pupil
- corneal epithelial edema
- congested episcleral and conjunctival blood vessels
- shallow peripheral anterior chamber
- mild amount of aqueous flare and cells

Diagnosis Definitive diagnosis depends on gonioscopic verification of angle closure. Gonioscopy should be possible in almost all cases of APAC, although clearing of corneal edema with topical IOP-lowering therapy, topical glycerin, or paracentesis may be necessary to allow visualization of the angle. Dynamic gonioscopy, with indentation of the central cornea, may help the clinician determine whether the iris–trabecular meshwork blockage is reversible *(appositional closure)* or irreversible *(synechial closure),* and it may also be therapeutic in breaking the attack of acute angle closure. Gonioscopy of the fellow eye in a patient with APAC usually reveals a narrow, occludable angle. The presence of a deep angle in the fellow eye should prompt the clinician to search for secondary causes of elevated IOP, such as a posterior segment mass, zonular insufficiency, anterior segment neovascularization, or the iridocorneal endothelial syndrome, among others. When performing gonioscopy, the clinician should note the effect of the examination light on the angle recess; the slit-lamp beam can cause pupillary constriction, thus artificially opening the inherently narrow angle recess (see Fig 9-2).

During an acute attack, the IOP may be high enough to cause glaucomatous optic nerve damage, ischemic optic neuropathy, and/or retinal vascular occlusion. PAS can form rapidly, and IOP-induced ischemia may produce sector atrophy of the iris, releasing pigment. This causes pigmentary dusting of the iris surface and corneal endothelium. Iris ischemia, specifically of the iris sphincter muscle, may cause the pupil to become permanently fixed and dilated. *Glaukomflecken,* characteristic small anterior subcapsular lens opacities, may also develop as a result of necrosis. These findings are helpful in the detection of previous episodes of APAC.

Management The definitive treatment for APAC associated with pupillary block is usually LPI (discussed in Chapter 13). Once an iridotomy has been performed, the pupillary block is relieved and the pressure gradient between the posterior and anterior chambers is