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DIAGNOSTIC AND SURGICAL TECHNIQUES

Intraocular Lens Complications Requiring Removal or Exchange

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Abstract. Intraocular lens (IOL)-related complications are caused primarily by mechanical trauma, inflammatory or infectious complications, or optical problems. Complications may occur at the time of surgery or be the result of an ongoing postoperative process. Mechanical and inflammatory injury may produce corneal decompensation, cystoid macular edema, hyphema, uveitis, and glaucoma, causing reduced vision and in some cases chronic pain. Optical problems may be due to a wrong power of the IOL or to postoperative decentration or dislocation of the lens. Ophthalmologists should be aware of the indications for IOL removal or exchange in those patients who have ongoing IOL-induced injury or impairment. Removal or exchange of an IOL frequently involves a complex decision-making process and is often associated with immense technical challenge. Various medical and surgical treatments may be tried to correct IOL problems before the decision is made to remove or exchange the lens. (**Surv Ophthalmol 42**:417–440, 1998. © 1998 by Elsevier Science Inc. All rights reserved.)

Key words. bullous keratopathy • cataract surgery • complications of intraocular lenses • corneal edema • cystoid macular edema • glaucoma • hyphema • malposition of intraocular lenses • removal or exchange of intraocular lenses • uveitis

Although complications from intraocular lenses (IOLs) were common in the early days of cataract surgery,[8,215] improvements in surgical technique and IOL design and quality have reduced the problems. Mechanisms for IOL-related complications fall into four groups: mechanical trauma, chronic inflammation, infectious disease, and optical problems (Table 1).

Mechanical trauma from an IOL may occur at the time of original surgery or it may be the result of ongoing intraocular injury. Surgical contact with the corneal endothelium causes endothelial cell loss, which may cause transient or persistent dysfunction with corneal decompensation. Anterior chamber (AC)-IOLs also may produce ongoing mechanical trauma to the cornea, iris, ciliary body, or the anterior chamber angle. Mechanical injury to the iris may produce atrophy and chronic uveitis, potentially compromising aqueous outflow and leading to glaucoma. Erosion of the IOL into the ciliary body also may cause ocular discomfort.

A more subtle form of injury may result from chronic inflammation produced by ongoing mechanical trauma. The IOL may erode into uveal tissue, liberating inflammatory mediators that damage other ocular structures; this is especially common with oversized or older AC-IOLs with a closed-loop design. These inflammatory products may contribute to progressive corneal endothelial cell loss, cystoid macular edema (CME), and glaucoma.

A more serious form of inflammatory injury occasionally results from infectious endophthalmitis. Acute infectious endophthalmitis usually is not di-

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0039-6257/98/$19.00
PII S0039-6257(97)00140-9

TABLE 1

Mechanisms for IOL-Related Complications

Mechanical
Intraoperative
Postoperative
Inflammatory
Noninfectious
Infectious
Optical
IOL Power
IOL Malposition

IOL 5 intraocular lens.

rectly attributable to IOL implantation, although epidemiologic data suggest that polypropylene loops are a risk factor for endophthalmitis.[166] Menikoff et al reported that one-piece polymethylmethacrylate lenses appear less likely to be associated with infectious endophthalmitis than multi-piece design lenses with polypropylene loops.[166] Successful treatment of endophthalmitis seldom requires IOL removal, although some patients with localized endophthalmitis are resistant to treatment until the IOL and the capsular bag are removed.[165]

Optical problems may be caused by improper power selection or abnormal IOL position. Improper IOL power may produce an unacceptable refractive error, which may require IOL exchange. Malposition may produce symptoms associated with the optic edge, IOL tilt, or other IOL structures, such as positioning holes, lens loops, or laser ridges.

Failure of an IOL represents a great disappointment to the patient, who is usually aware of many successful outcomes of cataract/IOL surgery among friends. The patient often blames the surgeon and seeks a new ophthalmologist for follow-up care of the complications; thus, the original surgeon may not even be aware of the eventual need for IOL explantation. The consulting physician can help the patient to understand that the original surgeon shared a high level of optimism and that, although an IOL may currently be recognized as a poor design, it may have been acceptable or even the state-of-the-art IOL at the time of the original surgery.

Corneal Complications

Corneal complications were the leading indication for IOL explantation before the development of newer IOLs and surgical techniques.[8,143] The corneal endothelium consists of a monolayer of nonregenerative cells responsible for maintaining corneal clarity by its pump function. The cells that make up this layer are among the most delicate type in the human body.[252]

Corneal endothelial dysfunction after cataract surgery may be mild-to-moderate and transient (resulting in temporary microcystic edema and visual impairment), or it may be severe and permanent. Persistent endothelial dysfunction with corneal decompensation is currently the most common indication for penetrating keratoplasty.[22,24,43,51,90,99,117,132,151,157,174,176,177,208,217,236,238,241]

Surgical trauma can cause corneal decompensation,[15,142,189,218,256] but the condition may be hastened by pre-existing corneal disease (e.g., Fuchs' dystrophy). Endothelial cell loss related to the IOL may be caused by direct mechanical trauma, including excessive vaulting of an AC-IOL, lens mobility (pseudophakodonesis) of an undersized or poorly fixated IOL (tilting, rocking, or rotating [propellering]), or IOL malposition (e.g., migration through a peripheral iridectomy). Even peripheral corneal-IOL contact has the potential to cause central endothelial dysfunction. Peripheral cell loss may stimulate endothelial migration and reduce endothelial cell density centrally, causing corneal decompensation.[106,107,110–112]

Erosion of an IOL into uveal tissue may incite inflammation and cause the liberation of inflammatory mediators, such as prostaglandins, which may damage the corneal endothelium.[8] Inflammation is often the mechanism for late corneal decompensation associated with closed-loop AC-IOL designs that erode into peripheral iris, anterior chamber angle, and ciliary body.[44,135,162,163,211,236] This mechanism can explain the late corneal decompensation that may occur in patients who appear to have stable IOLs that are not directly traumatizing the corneal endothelium. Because the inflammation often is very mild, careful examination is required to reveal subtle cell and flare in the anterior chamber and fine inflammatory precipitates on the IOL surface. Inflammation associated with the semiflexible, closed-loop style AC-IOLs contributed to their removal from the USA market.[9] Corneal failure may also result from continuing postoperative endothelial cell loss associated with aging, ocular disease, laser treatment, or iris or vitreous contact with the endothelium.

PSEUDOPHAKIC CORNEAL EDEMA

Intraocular Lens Design

We prefer *pseudophakic corneal edema* over the term *pseudophakic bullous keratopathy* to describe patients with corneal decompensation, because not all patients develop bullae. Pseudophakic corneal edema is the leading indication for penetrating keratoplasty.[22,58,217,236,241,271] Kraff et al reported that 36% of IOL removals are performed because of pseudophakic corneal edema.[143] In a series of 101 eyes of 98 consecutive patients, Lyle and Jin reported bullous keratopathy as the causative indication in 86.7% of patients who underwent combined penetrating keratoplasty and IOL exchange.[153]

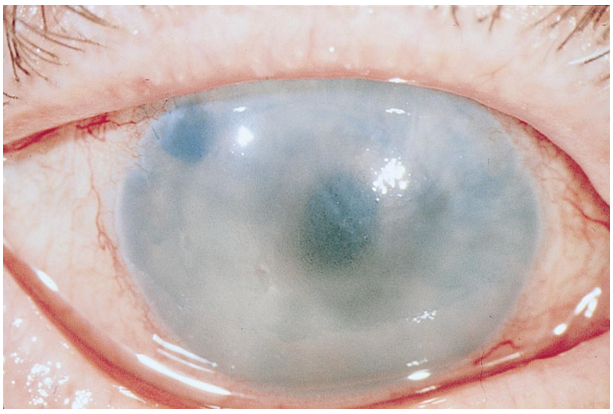

Fig. 1. Pseudophakic corneal edema associated with a Stableflex-style, closed-loop design AC-IOL. Successful treatment requires IOL exchange at the time of penetrating keratoplasty.

When corneal decompensation is associated with the IOL design (e.g., the semiflexible, closed-loop design [Fig. 1]),[26,44,47,118,135,162,163,211,226,236,243,255,272,274] the IOL should always be exchanged or removed when penetrating keratoplasty is performed. Such designs include the Leiske Style 10 (Surgical Corp., Goleta, CA), Azar 91Z (IOLAB Corp., Covina, CA), Optical Radiation Corp. model 11 Stableflex (Azusa, CA), the Intermedics Hessburg model 024 (Intraocular Inc., Pasadena, CA), and the Pannu AC-IOL (Allergan Medical Optics, Irvine, CA).[8] Also to be avoided are AC-IOL designs featuring positioning holes or eyelets, which may erode into the angle and act as a smaller version of the closed-loop designs.[8,155] Flexible, open, thin-loop lenses, such as the Dubroff-style (Intermedics Pharmacia, Monrovia, CA) AC-IOL, also may erode into the peripheral angle and create low-grade, chronic inflammation with concomitant corneal endothelial damage and CME.[147]

Surgical results of penetrating keratoplasty after removal or exchange of closed-loop AC-IOLs have been good. Kornmehl et al reported an average recovery of corrected visual acuity to 20/44 in 40 consecutive patients who underwent exchange of a closed-loop AC-IOL (30 Leiske, 10 Stableflex) for a Kelman Omnifit AC-21 AC-IOL (Allergan Medical Optics) at the time of penetrating keratoplasty.[135] Koenig et al reported good results in 20 consecutive patients with corneal decompensation associated with a Stableflex-style AC-IOL who underwent penetrating keratoplasty, anterior vitrectomy, and IOL exchange for a Kelman Multiflex-style, open-loop AC-IOL (Cooper Vision Cilco Inc., Pomona, CA).[133] Thirty-five percent (7 of 20) attained a spectacle correction of 20/40 or better, and all grafts remained clear during a follow-up period of 4–45 months. Zaidman and Goldman reported a 90% chance of allograft clarity at the end of 12–18 months in patients undergoing penetrating keratoplasty and IOL exchange with a Kelman-style, open-loop AC-IOL or posterior chamber (PC)-IOL.[285]

Price et al noted that 20.6% (7 of 34) patients developed allograft failure with retained AC-IOLs compared with 5.1% (4 of 78) in patients with retained PC-IOLs at the time of keratoplasty.[205] In contrast, Speaker et al reported a large number of graft failures in patients who retained closed-loop AC-IOLs at the time of keratoplasty.[243] Allograft failure was noted in 60% of these patients at 1 year, 75% at 2 years, and 100% at 5 years. Smith et al,[236] Insler et al,[118] and Cohen et al[47] reported series in which allograft outcomes were compromised by retention of closed-loop AC-IOLs.

Removal of iris-supported lenses also should be considered at the time of penetrating keratoplasty, particularly if the IOL appears poorly fixated or excessively mobile. Despite initial reports suggesting a favorable surgical outcome in patients who retained these lenses, subsequent longer-term studies invariably demonstrated a higher rate of complications.[12,40,74,145,167,226,258,270] Speaker et al reported that 75% of the allograft failures in patients with retained iris-supported IOLs occurred after the second postoperative year.[243] Exchange did not appear to offer significant advantage over retention in their study. Rao et al found endothelial cell loss to occur at a greater rate with iris-supported IOLs than with Choyce-style AC-IOLs.[207] Several additional authors noted that patients with retained iris-supported IOLs had the worst prognosis for maintenance of a clear graft.[137,191,226]

Reports by Meyer and Sugar,[167] Kozarsky et al,[137] and Waring et al[274] support leaving a properly positioned, secure PC-IOL or modern flexible open-loop AC-IOL at the time of keratoplasty. Notably, Price et al reported that grafts for pseudophakic bullous keratopathy with retained posterior chamber lenses had a significantly higher failure rate from rejection and endothelial decompensation, 5.1%, compared with less than 1.0% in keratoconus, Fuchs' dystrophy, or bullous keratopathy with secondary implants.[205]

Several authors have addressed the clinical and technical strategies associated with IOL exchange during penetrating keratoplasty.[27,54,59,62,92,99,114,133,135,146,199,203,205,227,241,272,281,285]

Instability of the Intraocular Lens

Excessive IOL mobility (pseudophakodonesis) jeopardizes allograft success. An unstable IOL should be removed at the time of keratoplasty unless it can be safely stabilized in a secure position and is considered a safe IOL design. Even modern, acceptable IOLs should be removed if they cannot be properly

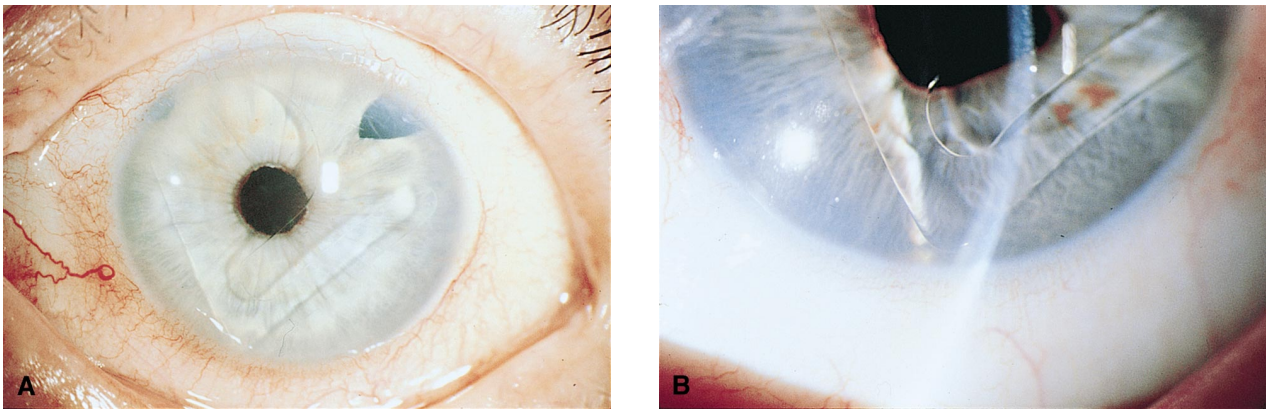

Fig. 2. Left: Migration of a Kelman-style II AC-IOL through a peripheral iridectomy. *Right:* This migration results in IOL tilt and corneal endothelial contact inferiorly. Intraocular lens exchange was performed to decrease the risk of corneal endothelial decompensation.

fixated, e.g., because of inadequate anatomic support, improper sizing, etc.

Clinically, the best indication of unacceptable pseudophakodonesis is "shimmering" from mobile Purkinje-Sanson images III and IV from the anterior and posterior IOL surfaces, which can be observed during examination of subtle eye movements at the slit lamp (Holladay J: personal communication, 1996). Miller and Doane[169] and Jacobs et al[121] used high-speed cinematography to demonstrate pseudophakodonesis with corneal touch associated with iris-supported IOLs. Rao et al identified progressive corneal endothelial cell loss over time.[207]

PERIPHERAL CORNEAL EDEMA

Corneal decompensation due to ongoing mechanical injury may be caused by central and/or peripheral endothelial contact with the IOL. Intermittent or constant central corneal touch may cause accelerated loss of endothelial cells.[106,107,112–212] Corneal touch is more commonly associated with AC-IOLs and may be caused by improper IOL positioning, IOL migration due to improper IOL size, ocular trauma (including eye rubbing), excessive IOL vaulting, or prolonged head-down position.[60] Migration of an AC-IOL through a wound dehiscence or a peripheral iridectomy (Fig. 2A) may also cause IOL-corneal touch (Fig. 2B).

Patients with peripheral IOL-corneal touch may initially have no signs or symptoms of corneal decompensation. However, although the central cornea may appear clear, the peripheral contact may cause local cell loss, which can stimulate cell migration from the central cornea to the periphery. Eventually, continued endothelial cell loss will result in central corneal decompensation unless the source of damage is removed.

To undergo IOL repositioning, removal, or exchange, the cornea must have adequate endothelial reserve. Clinical signs of inadequate endothelial reserve may include a low cell density, (e.g., less than 500 cells/mm^2), abnormal cell morphology (e.g., polymegathism or pleomorphism), or increased corneal thickness by pachymetry. Clinical symptoms may include decreased vision in the morning secondary to microcystic corneal epithelial edema. Microcystic corneal epithelial edema is frequently worse in the morning because of reduced evaporation and slight endothelial hypoxia from eyelid closure during sleep (Fig. 3). Patients with reduced endothelial cell density who develop microcystic corneal epithelial edema with normal intraocular pressure (IOP) are at high risk for corneal decompensation after intraocular surgery and may require penetrating keratoplasty. Patients who have IOL-corneal touch should be considered for IOL repositioning, removal, or exchange despite a clear central cornea provided that adequate endothelial cell reserve exists, as this may prevent the need for penetrating keratoplasty.[106,107,160]

Fig. 3. Microcystic corneal epithelial edema is best seen under high magnification using indirect retroillumination.

CLEAR CORNEA WITH INTRAOCULAR LENS-INDUCED ACCELERATED ENDOTHELIAL CELL LOSS

The decision to exchange an IOL is controversial when a patient has normal vision and no IOL-corneal touch but shows evidence of IOL pseudophakodonesis and/or chronic low-grade inflammation.[28,186] Such patients may be at risk for corneal decompensation because of their IOL design, e.g., a closed-loop AC-IOL.

When accelerated endothelial cell loss (i.e., cell loss at a much more rapid rate than could be attributed to aging) can be documented by serial specular cell photomicrography in a patient with a closed-loop–style AC-IOL, removal or exchange of the IOL should be considered. Coli et al reported a series of 102 eyes with anterior chamber or iris-plane IOLs and signs of progressive endothelial damage and concluded that corneal decompensation may be prevented if the IOLs are removed before a critical degree of endothelial cell loss or dysfunction occurs.[48] This requires serial evaluation with documentation of a reliable trend that considers the accuracy and reproducibility of each test. Evaluation should include assessment of changes in cell density, morphology, or pachymetry, as well as examination for factors that would suggest IOL-induced corneal compromise (IOL design, position, stability, proper size, evidence of erosion, or inflammatory precipitates on the IOL surface or corneal endothelium). If a patient has adequate endothelial reserve but demonstrates a clinical course that indicates likelihood of corneal decompensation, an early exchange procedure may forestall corneal transplantation. This spares the patient from potential problems, such as allograft rejection, corneal astigmatism, and prolonged corneal wound healing.

Considering IOL exchange in these patients requires an assessment of the risks and benefits of observation compared with intervention. A patient with a closed-loop–style AC-IOL who continues to demonstrate good vision and a stable cell count is certainly at less risk with continued observation than a patient with documented progression of endothelial cell loss with marginal cell reserve (e.g., 500 cell/mm^2 centrally [Fig. 4]). Intervention carries the risk of making the patient acutely worse, through surgically induced corneal decompensation, hemorrhage, iridodialysis, and failed or incomplete IOL removal. This is because the closed-loop AC-IOL designs may erode into the angle and ciliary body. Removal techniques must respect this erosion to preserve the integrity of the anterior segment and to avoid complications.[5]

The goal of IOL exchange in the presence of a clear cornea is to avoid the need for a more involved procedure later, which will be more expensive, require a longer recovery, and be associated with more potential complications. However, patients with low endothelial cell counts (less than 500 cells/mm^2) often demonstrate increased stromal thickness by pachymetry and are likely to develop acute corneal decompensation after surgical intervention. These patients are better managed with IOL exchange combined with penetrating keratoplasty after visually significant corneal decompensation occurs.

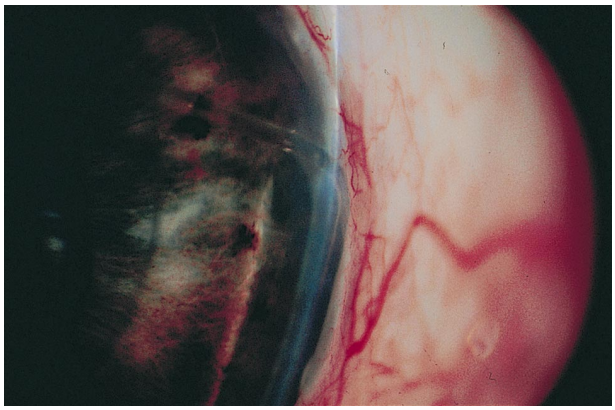

Fig. 4. Erosion of a Leiske-style, closed-loop AC-IOL into the peripheral cornea produced "corneal fixation." This was the patient's best eye and it maintained a stable endothelial cell count and excellent vision for over 10 years with observation. Deterioration in vision or accelerated endothelial cell loss would provide motivation for IOL exchange.

Patients with pre-existing progressive corneal disorders, such as Fuchs' corneal endothelial dystrophy, would obviously continue to demonstrate progression associated with their underlying corneal disorder despite IOL removal.

It appears that all AC-IOLs accelerate endothelial cell loss to some degree. This may be an acceptable situation in older patients receiving modern versions of the Kelman-style implant (e.g., Multiflex style) that are properly sized and do not have positioning holes at the end of the haptic loop that can cause synechiae.[14,155]

Retinal Indications

RETINAL DETACHMENT

Retinal detachment rarely requires explantation of the IOL.[14] Lens removal should be considered to repair retinal detachment in patients 1) who have concomitant pathology creating other indications for IOL removal, 2) whose IOL obscures visualization of the detachment (which is more common in eyes with AC-IOLs and severe anterior proliferative vitreoretinopathy) or mechanically obstructs the surgery, 3) whose IOL may create specific intraoperative or postoperative management problems (e.g., condensation of a gas bubble on a silicone optic or

the creation of silicone oil "droplets" on the surface of a silicone optic),[17,65,76,91,144,235,253] or 4) who have significant capsular pathology (localized endophthalmitis, severe capsular contraction, or limited IOL support) that might contribute to IOL-related intraoperative or postoperative complications.

CYSTOID MACULAR EDEMA

Cystoid macular edema is a well-recognized complication of intraocular surgery with or without IOL implantation.[3,53,64,68,74,81,83,96–98,100,101,104,245] Potential mechanisms of pathogenesis include increased permeability of the perifoveolar capillaries, an ischemic tissue injury, a secondary response to intraocular inflammation, and direct traction on the macula after vitreous shifts.[8]

Incidence and Mechanisms

Clinically, CME can be divided into three groups: 1) angiographic CME, in which vision is usually unaffected; 2) clinically significant CME, in which vision is decreased; and 3) chronic CME, defined as chronic or recurrent CME of more than 6 month's duration. The presence of angiographic CME alone is of uncertain importance. In a study by Stark et al, 78% of patients with fluorescein-positive angiographic evidence of CME had 20/20 vision, and the rest had 20/25 vision.[245] Clinically significant CME is associated with transient decrease in vision in up to 8% of patients after uncomplicated cataract extraction and is associated with chronic decrease in 1–2% of patients.[49] A clinical study at the Wilmer Eye Institute found that clinically significant CME developed in a smaller percentage of cases (2% total incidence and 0.3% chronic) if the IOL implantation was uncomplicated.[245] Visual potential probably correlates best with thickness of edema rather than size or severity of angiographic leakage.[184]

The peak incidence of CME after cataract surgery is approximately 4–16 weeks postoperatively, but the onset may be delayed months or even years after surgery.[49,173] Spontaneous resolution occurs in 75% of cases within 6 months.[49,79,82,83,104,105,173,221,284] Cystoid macular edema that causes permanent structural damage to the macula is an important cause of poor vision that persists despite IOL removal or exchange.[26,153]

Chronic CME may be associated with photoreceptor atrophy, lamellar hole formation, and reactive retinal pigment epithelium (RPE) changes in the fovea.[80] Histopathologic examination supports the role of inflammation in the development of CME by documenting the presence of inflammation in the iris, ciliary body, vitreous, and retinal blood vessels. Breakdown of the blood-retinal barrier can be seen with iris angiography and vitreous fluorophotometry.[136,171] The degree and duration of breakdown of the blood-retinal barrier may correlate with the amount of intraoperative damage to the iris and the extent of the posterior synechiae.[73] For this reason some authors have suggested that IOLs placed in the capsular bag may be better tolerated than those placed in the ciliary sulcus.[73,172]

Epidemiologic studies suggest that surgical techniques and IOL design play an important role in the development of CME. Jaffe et al found that only 3 patients in a series of 103 who underwent extracapsular cataract extraction (ECCE) with posterior chamber IOL implantation developed angiographic CME.[125] The lower incidence of CME after ECCE versus intracapsular cataract extraction (ICCE) is supported by studies by Binkhorst,[23] Kraff et al,[139] Taylor et al,[259] Stark et al,[245] and Jaffe et al.[124,125] Iris-supported IOLs are associated with a higher incidence of CME. In a retrospective study by Taylor et al, clinical CME developed in only 2% of 630 eyes that underwent ICCE alone, versus 9.9% of 850 eyes that underwent ICCE combined with iris-supported IOL implantation.[259] Rigid closed-loop AC-IOLs have also been associated with an increased incidence of CME. In a study by Maynor, CME developed in 1.9% of 207 patients who underwent ICCE and implantation of a Leiske-style IOL and in 5% of 302 patients undergoing ICCE and implantation of an Azar 91Z lens.[162] Choyce found a 3% incidence of CME in a series involving 1,000 Mark IX rigid AC-IOL (Coburn, Clearwater, FL).[41]

Maintaining the integrity of the posterior capsule and vitreous appears to reduce the risk of CME.[37] Nikica et al reported CME in 35.7% of cases of extracapsular cataract extraction complicated by posterior capsular rupture requiring anterior vitrectomy and insertion of an AC-IOL.[183] This compares with a 1.5% incidence of CME in cases of uncomplicated extracapsular cataract extraction with insertion of a PC-IOL. Kraff et al randomly performed primary capsulotomies in 152 patients and left the capsule intact in 136 patients.[138] Cystoid macular edema was evident, by angiography, in 5.6% of those with an intact capsule, as opposed to 21.5% of those with capsulotomy. However, overall visual acuity results were virtually identical in the two groups. Angiographic findings in patients with intact capsules did not correlate with visual acuity.

Winslow and colleagues found angiographic evidence of CME in 48% of patients with vitreous prolapse after capsulotomy, whereas edema was demonstrable in only 10% of patients without vitreous loss and with intact capsules.[280] Jaffe found that if capsulotomy is delayed for a year or longer after cataract extraction, CME is less likely to occur.[126]

Management

Cystoid macular edema per se is only a relative indication for IOL removal or exchange, and it often occurs in the presence of other ocular inflammatory conditions, such as pseudophakic corneal edema, IOL-induced uveitis, and localized or generalized endophthalmitis. Therefore, chronic CME is infrequently the sole indication for IOL removal or exchange.[158,234,239] The true incidence of CME as an associated finding in patients undergoing IOL exchange is unknown because of coexisting conditions, such as pseudophakic corneal decompensation. Isolated studies report the overall incidence of CME before IOL removal or exchange to be 3.3–5.5%.[48,133,153,191,227,234]

Although many authors report improvement in clinically observed CME and visual acuity after IOL removal or exchange, it is difficult to predict the response in an individual patient. Visual outcome may be limited by permanent structural changes of the fovea or from perpetuation of CME by mechanisms that are not directly attributed to the IOL. Smith reported significant improvement in visual acuity after IOL removal or exchange in some patients with chronic CME attributed to an IOL.[239] In their study, 50% of the patients with CME achieved a significant improvement in vision after removal of the IOL.

Lyle and Jin reported resolution of CME in 7 of 14 patients (50%) after IOL exchange; however, in their series of 101 eyes in 98 patients, the overall incidence of postoperative CME was higher than the preoperative incidence.[153] In that study, the incidence of postoperative CME was significantly associated with the preoperative CME. Lyle and Jin also found that new CME was more likely to develop after penetrating keratoplasty combined with IOL exchange. They identified CME and glaucoma as the cause of a poor visual outcome in patients undergoing IOL exchange without penetrating keratoplasty. This is consistent with the findings of Busin et al, who identified glaucoma and CME as the most common cause of a final visual acuity worse than 20/60 after IOL exchange.[26] In a retrospective analysis by Mamalis et al of 102 patients who underwent IOL removal or exchange, the postoperative vision improved in 39%, remained unchanged in 46%, and worsened in 15%.[158] The most common cause for worsening of vision postoperatively was corneal decompensation, then glaucoma and CME, respectively.

Coli et al studied 102 patients with AC-IOL–induced corneal endothelial damage who underwent IOL exchange.[48] This group contained 47 patients (46%) with concomitant CME before IOL exchange. Patients with low endothelial cell counts (less than 900 cell/mm^2) and either a rigid-loop– or closed-loop–style AC-IOL had a higher incidence (71%) of preoperative CME. Posterior chamber-IOLs were substituted for AC-IOLs in all cases, with 87 sutured to the iris, 3 sutured to the sclera, and 12 inserted into the ciliary sulcus. Among all patients with CME, visual acuity improved in 30 eyes (64%), remained unchanged in 6 eyes (13%), and worsened in 11 eyes (23%) after exchange for an iris-sutured PC-IOL and vitrectomy.

In another study, penetrating keratoplasty, AC-IOL removal, anterior vitrectomy, and insertion of an iris-sutured PC-IOL resulted in angiographic resolution of CME in 18 of 25 patients diagnosed with CME and pseudophakic corneal edema.[203] Improvement of visual acuity to 20/40 or better was achieved in 16 patients, with visual recovery usually occurring within a year of surgery.

Stark et al reported three patients who underwent IOL exchange because of pain with CME.[246] Exchange of an AC-IOL for a sutured PC-IOL in the absence of capsular support relieved the pain in all the patients, and the CME improved in two of them.[246] Two additional patients who underwent IOL exchange because of CME alone also demonstrated improved vision and reduced CME.

Insertion of a three-piece–C-loop PC-IOL in the anterior chamber has been associated with inflammatory complications, including CME and epimacular membrane formation. Liu et al reported improvement in visual acuity and reduced CME in two patients who underwent exchange of a three-piece–C-loop PC-IOL that had been inserted into the anterior chamber.[152]

Many of the data addressing CME and visual outcome after IOL exchange are retrospective, and there are little data from prospective controlled interventions. Schein et al compared IOL exchange techniques in a randomized prospective study of 176 consecutive patients undergoing penetrating keratoplasty with IOL exchange.[227] They found that the cumulative risk of CME with iris-fixated PC-IOLs was significantly less than that with AC-IOLs or transsclerally sutured PC-IOLs. However, the number of resolved cases and the number of newly developed cases of CME were not reported. Also, the cumulative risk of CME after penetrating keratoplasty and IOL removal alone was not determined.

Three retrospective uncontrolled studies have been reported, two comparing open-loop–Kelman-style AC-IOLs with iris-fixated lenses and one comparing open-loop–Kelman-style AC-IOLs, iris-fixated PC-IOLs, and transscleral fixated PC-IOLs.[54,99,146] None of these studies reported any differences in visual outcome or complication rates after IOL exchange among the different techniques.

Harbour et al treated 24 consecutive patients with chronic pseudophakic CME with pars plana vitrectomy without IOL removal.[94] Preoperatively, the pa-

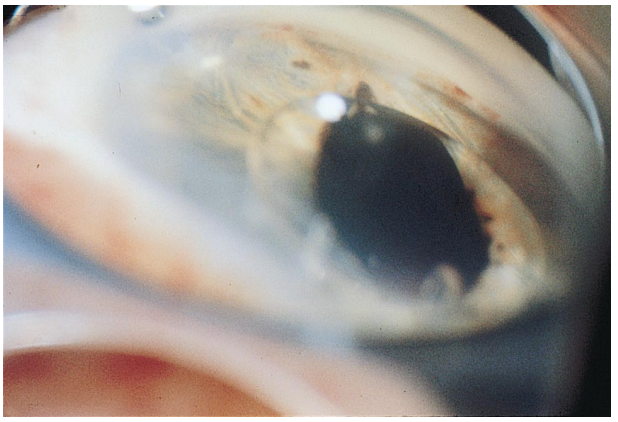

Fig. 5. Erosion into the peripheral iris is common with closed-loop AC-IOL designs, such as the Azar, producing low-grade inflammation with the potential for CME and corneal decompensation.

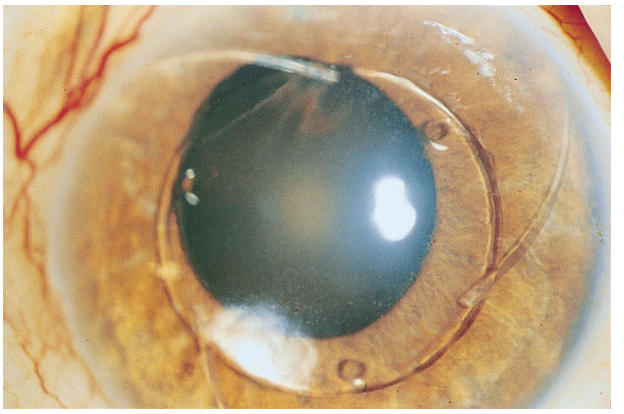

Fig. 6. Vitreous entanglement with the IOL (traction at superior optic near the loop—optic junction) may produce traction with low-grade inflammation, producing CME.

tients had evidence of either vitreous adhesions to anterior segment structures (23 patients) or iris capture of the IOL (1 patient), and all had responded poorly to medical therapy. In all patients, pars plana vitrectomy with removal of vitreous adhesions improved the visual acuity, with a mean improvement of 4.6 Snellen lines.

The surgeon performing IOL removal or exchange for the treatment of visually significant CME should direct meticulous attention toward alleviating potential causes of chronic inflammation, such as vitreocorneal or iridocorneal adhesions, iris incarceration, IOL capture, IOL-induced iris chafing, or any vitreous traction (Figs. 5–8). This may reduce the sources of inflammation responsible for CME. Cystoid macular edema is a potential complication of any intraocular procedure, and not all pseudophakic patients with CME benefit from IOL removal or exchange. Patients with concomitant inflammation and clinical signs that suggest that the IOL is the cause of ongoing CME are the patients most likely to benefit from IOL removal or exchange. Surgical intervention should be reserved for cases of CME refractory to medical treatment. Cystoid macular edema may also be complicated by the development of an epimacular membrane or foveal hole formation.

Inflammatory (Noninfectious) Indications

MICROHYPHEMA

Microhyphema has been noted in approximately 2–5% of anterior chamber and iris-fixated lenses after cataract extraction.[66,150,283] Microhyphema associated with increased IOP and uveitis in the presence of an anterior segment lens (uveitis-glaucoma-hyphema syndrome) is discussed in the next section.

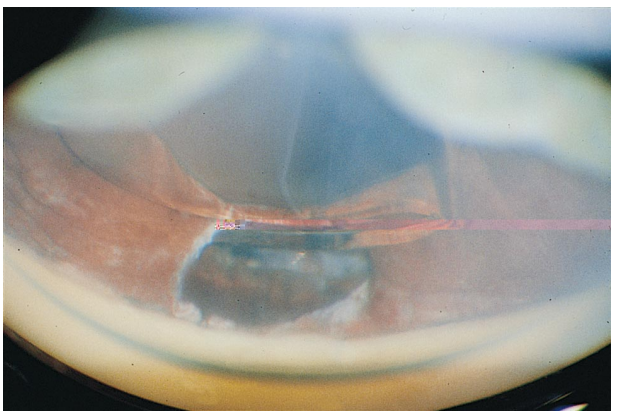

Fig. 7. Inflammation results from vitreous traction in addition to IOL migration through a peripheral iridectomy, producing erosion into the ciliary body.

Fig. 8. Synechiae encases the loop and peripheral eyelet of a Pannu-style AC-IOL, producing CME from low-grade inflammation.