Fluorescien Angiographic Changes after Phacoemulsification and YAG Laser Micropunctures in the Posterior Capsule in Diabetic Patients.

Thesis

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List of abbreviations

("A" cells)	**Anterior epithelial cells.**
(ACO)	**Anterior capsule opacification/ fibrosis**
(ALL)	**anterior limiting lamina**
(ASC)	**Anterior subcapsular cataracts**
(BCVA)	**Best corrected visual acuity**
(BRB)	**Blood retinal barrier**
(CCC)	**Continuous curvilinear capsulorhexis**
(CME)	**Cystoid macular oedema**
("E" cells)	**Equatorial lens bow.**
(ECCE)	**Extracapsular cataract surgery.**
(FFA)	**Fundus fluorescein angiography**
(FGF)	**Fibroblast growth factor.**
(GFF)	**Growth factor family**
(l/A)	**Irrigation/aspiration**
(ILO)	**Intrlenticular opacification.**
(IOL)	**Intraocular lens.**
(IOLO)	**Intraocular lens opacification.**
(LCT)	**Laser posterior capsulotomy**
(LEC)	**Lens epithelial cell.**
(Nd:YAG)	**Neodymium: Yttrium-Aluminum-Garnet.**
(OCT)	**Ocular computed tomography**

(PCO)	**Posterior capsule opacification.**
(PMMA)	**Polymethylmetacrylate**
(PVD)	**posterior vitreous detachment**
(RD)	**Retinal detachment**
(SCI)	**Sealed capsule irrigation**
(TGFβ)	**Transforming growth factor-beta**

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Introduction

Posterior capsule opacification (PCO) (secondary cataract, after cataract) is a nagging complication of cataract-intraocular lens (IOL) surgery since the beginning of extracapsular cataract surgery (ECCE) and IOL implantation. PCO needs to be eliminated since deleterious sequelae of this complication occur and Neodymium:Yttrium-Aluminum-Garnet (Nd:YAG) laser treatment now constitutes a major and unnecessary financial burden on the health care system. A successful expansion of ECCE- IOL surgery in the developing world depends on eradication of PCO, since patient follow-up is difficult and access to the Nd:YAG laser is not widely available. Advances in the surgical techniques, IOL designs, biomaterials have been instrumental in gradual and unnoticed decrease in the incidence of the PCO. There is strong belief that the overall incidence of PCO and hence the incidence of Nd:YAG laser posterior capsulotomy is now rapidly decreasing from rates as high as 50 percent in the 1980s-early 1990s to less than 10 percent in the developed world nowadays. Active research and information derived from other experimental and clinical studies from several other centers have revealed that the tools, surgical procedures, skills and appropriate IOLs designs are now available to significantly reduce this complication. Opacification of the posterior capsule caused by postoperative proliferation of cells in the capsular bag remains the most frequent complication of cataract intraocular lens (IOL) surgery.[1,2] In addition to classic posterior capsule opacification (PCO, secondary cataract, after cataract), postoperative lens epithelial cell (LEC) proliferation is also involved in the pathogenesis of anterior capsule opacification/ fibrosis (ACO) and interlenticular opacification (ILO).[3,4]Secondary cataract (PCO) has been recognized since the origin of

extracapsular cataract surgery (ECCE) and was noted by Sir Harold Ridley in his first IOL implantations.[5] It was particularly common and severe in the early days of IOL surgery (in late 1970s and early 1980s) when the importance of cortical cleanup was less appreciated. Through the 1980s and early 1990s, the incidence of PCO ranged between 25-50 percent.[6] PCO is a major problem in pediatric cataract surgery where the incidence approached 100 percent.[7,8]

One of the crowning achievements of modern cataract surgery has been a gradual, almost unnoticed decrease in the incidence of this complication. Data at present show that with modern techniques and IOLs, the expected rate of PCO and the need for subsequent Neodymium: Yttrium-Aluminum-Garnet (Nd:YAG) laser posterior capsulotomy rate is decreasing to single digit (less than 10%).[9,10]

REASONS TO ERADICATE POSTERIOR CAPSULE OPACIFICATION

Although cataract is the most common cause of blindness in the world, after-cataract (PCO or secondary cataract) is an extremely common cause as well. Worst, MD has stated- "the most meaningful development in intraocular implant research in the next five years will be effective prevention of secondary cataract formation".(11) Eradication of PCO following ECCE has major medical and financial implications: Nd: YAG laser secondary posterior capsulotomy, can be associated with significant complications. Potential problems including IOL optic damage pitting, postoperative intraocular pressure elevation, cystoid macular edema, retinal detachment, and IOL subluxation.[12,13] Dense PCO and secondary membrane formation is particularly common following pediatric IOL implantation.[10-12] A delay in diagnosis can cause irreparable amblyopia. A

posterior capsulotomy can increase the risk or posterior segment complications in high myopes and patients with uveitis, glaucoma, and diabetic retinopathy. PCO of even a mild degree can decrease near acuity through a multifocal lOL, and may interfere with the function of refractive/ accommodating lOL designs.[10-12]

A significant incidence of PCO means that cataract surgery, alone, may not restore lasting sight to the 25 million people worldwide who are blind from cataract. Finally, a successful expansion of ECCE-IOL surgery in the developing world depends on eradication, or at least diminishing of PCO, since patient follow-up is difficult and access to the Nd:YAG laser is not widely available.[19]

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Literature Preview

Etiopathogenesis of posterior capsule opacification:

In the normal crystalline lens, the Lens epithelial cells are confined to the anterior surface at the equatorial region and the equatorial lens bow. This single row of cuboidal cells can be divided into two different biological zones (Figure 1). The anterior-central zone (corresponding to the zone of the anterior lens capsule) consists of a monolayer of flat cuboidal, epithelial cells with minimal mitotic activity. In response to a variety of stimuli, the anterior epithelial cells ("A" cells) proliferate and undergo fibrous metaplasia. This has been called "pseudofibrous metaplasia" by Font and Brownstein. [20] The second zone is important in the pathogenesis of "pearl" formation. This layer is a continuation of anterior lens cells around the equator, forming the equatorial lens bow ("E" cells). Unlike within the A-cell layer, cell mitoses, division, and multiplication are quite active in this region. New lens fibers are continuously produced in this zone throughout life. [20]

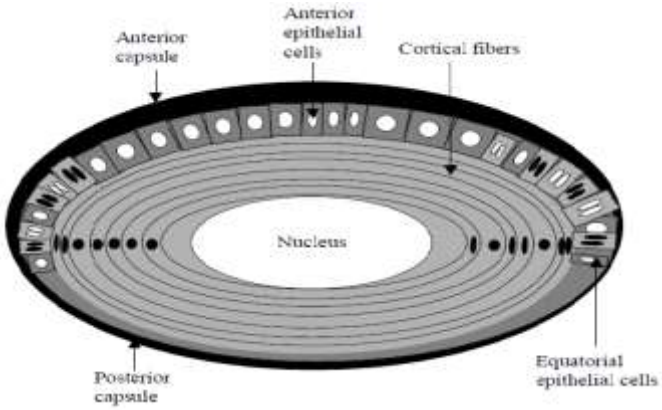

Figure 1 histology of normal lens. [20]

In addition to classic PCO, postoperative LEC proliferation is also involved in the pathogenesis of other entities, such as anterior capsule opacification/ fibrosis (ACO) [3,4] and ILO; a more recently described complication related to piggyback IOLs. [18,19] Thus, there are three distinct anatomic locations within the capsular bag where clinically significant opacification may occur postoperatively (Figure 2-3). Ophthalmic researchers are now developing surgical techniques/ devices not only to eliminate PCO, but also to eliminate capsular bag opacification, secondary to proliferation of LECs. [21]

Although both types of cells (from the anterior central zone and from the equatorial lens bow) have the potential to produce visually significant opacification, most cases of classic PCO are caused by proliferation of the equatorial cells. The term posterior capsule opacification is actually a misnomer. It is not the capsule which opacifies. Rather, an opaque membrane develops as retained cells proliferate and migrate onto the posterior capsular surface. [21]

Fig 2 capsular pearls

The opacification usually takes one of two morphologic forms. One form consists of capsular pearls (figure 2), which can consist of clusters of swollen, opacified epithelial "pearls" or clusters of posteriorly migrated equatorial epithelial (E) cells (bladder or Wedl

cells). It is probable that both LEC types can also contribute to the fibrous form of opacification. Anterior epithelial (A) cells are probably important in the pathogenesis of fibrosis PC0, since the primary type of response of these cells is to undergo fibrous metaplasia. Although the preferred type of growth of the equatorial epithelial (E) cells is in the direction of bloated, swollen, bullous-like bladder (Wedl) cells, these also may contribute to formation of the fibrous form of PCO by undergoing a fibrous metaplasia. This is a particularly common occurrence in cataracts in developing world settings where cataract surgery has been delayed for many years, and where posterior subcapsular cataracts have turned into fibrous plaques. [21] (Figure 3)

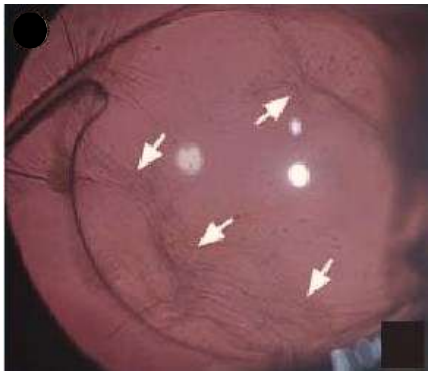

Fig 3 fibrous plaques.[21]

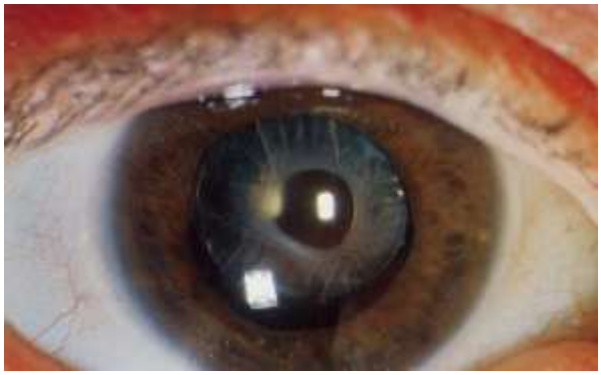

Fig 4 capsular phimosis

Capsulorhexis contraction (capsular phimosis) (Figure 4) is an important complication related to extreme fibrous proliferation of the anterior capsule. [2-4] Capsular phimosis can be avoided by not making the capsulorhexis too small. In general, a diameter less than 5.5 mm is undesirable.

In contrast to the lesions of the anterior (A cells) capsule that cause phenomena related to fibrosis, the (E cells) of the equatorial lens bow (Figure 2) tend to form cells that differentiate toward pearls (bladder cells) and cortex. Equatorial cells (E-cells) are also