Correlating Preoperative Biometric Data, Capsulorhexis size and different types of IOLs with Post-phacoemulsification Dysphotopsia

Thesis

Submitted for Partial Fulfillment of Master's Degree in Ophthalmology

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2018

List of Contents

List of Tables

List of Figures

List of Abbreviations

Abb.	Meaning
AC	Anterior Capsule
ACD	Anterior Chamber Depth
ACO	Anterior Capsule Opacification
AL	Axial Length
CCC	Capsulorhexis
CS	Contrast Sensitivity
FVA	Functional Visual Acuity
HOA	High Order Aberration
IOL	Intra Ocular Lens
Kf	Flat Keratometry
Kmean	Mean Keratometry
Ks	Steep Keratometry
LECs	Lens Epithelial cells
PC	Posterior Capsule
PCLI	Partial coherence laser interferometry
PCO	Posterior Capsule Opacification
SA	Spherical Aberration
VA	Visual Acuity
WTW	White To White

ABSTRACT

Purpose of this study is to outline the correlation between preoperative biometric data extracted from partial coherence laser interferometry (PCLI), capsulorhexis (CCC) size and the type of implanted IOL with dysphotopsia symptoms that might occur after phacoemulsification.

Methods: 100 patients were included in our study. All patients had uneventful phacoemulsification with implantation of square edged posterior chamber IOL (Sensar (AMO, USA), Tecnis one (ZCB00, AMO) and Acrysof single piece (SA60AT, Alcon labs, USA) IOLs).

Results: Our study demonstrated that age of patients, corneal optics, IOL haptic axis, Postoperative cylinder, temporal CCC from limbus and printed A constant of IOL affect the incidence of dysphotopsia.

Conclusion: This study showed that the age of patients, corneal steep keratometry, postoperative cylinder, temporal CCC from the limbus and printed A constant are important factors affecting the incidence of dysphotopsia.

Key words: IOL, CCC

INTRODUCTION

Optical side effects are well-documented associated symptoms of visually undesired photic phenomena following an intraocularlens (IOL) implantation surgery. The manifestations of the photic phenomena include transient glare, streaks, arcs, halos, and edge effects and have been collectively described as dysphotopsia ***(Hong et al., 2011)***.

The photic phenomena can occur either as a positive or negative dysphotopsia. Positive dysphotopsia is perceived by patients as brightness, halos, orstreaks, and rays emanating from a central point source of light, sometimes with a diffuse hazy glare. Conversely, negative dysphotopsias characterized by the perception of dark shadows of crescent shape, which can be arc-shaped, usually in the temporal field of vision ***(Hong et al., 2011)***.

The mechanism of negative dysphotopsia has remained a clinical enigma, with proposed explanations that include IOL material with a high index of refraction, optics with a sharp or truncated edge, and idiosyncratic predisposition ***(Holladay et al., 2012)***.

The explanations also include a cataract incision located temporally in clear cornea, brown irides, a prominent globe, a shallow orbit, an IOL anterior surface that is more than 0.46 mm from the plane of the posterior iris, a negative

afterimage, neural adaptation, and reflection of the anterior capsulotomy edge projected onto the nasal peripheral retina ***(Holladay et al., 2012)***.

The reflection of anterior capsulotomy edge was evidenced to be the cause of negative dysphotopsiaby the finding that reverse optic capture or placing a piggyback IOL in the ciliary sulcus eliminated negative dysphotopsia but exchanging IOL material or design did not ***(Cooke et al., 2013)***.

Negative dysphotopsia has not been reported aftersurgery complicated by malpositioned IOLs, has not been associated with astigmatic corneal incisions, radial corneal incisions, laser in situ keratomileusis flaps, or penetrating keratoplasty, it's symptoms are generally reduced with pharmacologic pupil dilation and increasedwith pupil constriction ***(Masket and Fram, 2011)***.

Several additional articles and letters with case reports showing the absence of some of these suggested mechanismshave also been published ***(Holladay et al., 2012)***.

Sharp or truncated optic edge is the most significant factor in positive dysphotopsiadue to an intense peak of reflected glare light on the retina ***(Holladay et al., 2012)***.

It was found that reflectionsfrom the front and back surfaces of both equiconvex, unequal biconvex designs and using materials of higher index ofrefraction were also factors

that increasedthe relative intensity of the reflected lightfrom 300 to 3500fold above that of the crystallinelens ***(Holladay et al., 2012)***.

Several subsequent studies confirmed thesefactors to be important in producing positivedysphotopsia ***(Holladay et al., 2012)***.

AIM OF THE WORK

The aim of the study was to outline the correlation between preoperative biometric data extracted from partial coherence laser interferometry (PCLI), capsulorhexis size and the type of implanted IOL with dysphotopsia symptoms that might occur after phacoemulsification.

REVIEW OF LITERATURE

Proposed road-map for the review:

1-Crystaline lens

Size and shape

position in relation to ciliary body, zonules and pupil

Epithelial proliferation, PCO, ACO and phimosis

2- IOLs

Material & RI

Shape: asphericity, edges, haptic angles, haptic-optic junction

Implantation techniques: bag, sulcus, button-holing, OVD removal

3- Functional vision

VA, C/S

SA

How to measure: objective and subjective

Dysphotopsia: causes/RF, theories, prevention and ttt

The crystalline lens:

It is a transparent, biconvexintraocular lens. Its nourishment is obtained from the surrounding aqueous and vitreous, and the same media must also remove waste products. The adult crystalline lens measures approximately 9.6 ±0.4 mm in diameter, with an approximate anteroposterior diameter of 4.2 ±0.5 mm. The anterior and posterior poles form the optical and geometrical axis of the lens ***(Vargas, Peng, Escobar-gomez, Schmidbauer and Apple, 1984)***.

The lens is surrounded by the aqueous humor, suspended posterior to the iris by zonular fibers which attach it to the ciliary body. It has no blood supply and no connective tissue, has a thick capsule that is the enlarged original basement membrane of the embryonic ectoderm and composed partly of type IV collagen ***(Goodenough, 1992)***.

Its capsule is a delicate membrane composed of a number of extremely fine layers which form an inner region or capsule proper, and an outer region, termed the zonular lamella which is amorphous structure. The capsular thickness is not uniform all over the lens; an annular zone of increased thickness occurs both in front and behind the lens at the insertion of the zonule ***(Street, 1972)***.

It is generally divided into the nucleus and the cortex.More detailed studies showed that the anterior and

posterior cortex show alternating light and dark zones, these cortical zones could be sub-divided into four zones (C1–C4). Zone C1, located behind the anterior and just in front of the posterior capsule, and C3 are zones of high light scatter, while C2 and C4 are dark zones of low light scatter. The thickness of zone C4 cannot be usually determined accurately. Most of the growth of the lens occurs in zone C2, while zone C3 shows little increase in thickness after being established during the second decade of life. The thickness of zone C1 tends to decrease with age ***(Dubbelman and Heijde, 2003)***.

The lens epithelium is confined to the anterior surface and the equatorial lens bow. It consists of a single row of cuboidal and cylindricalcells that can be divided biologically into two different zones withtwo different types of cells: anterior epithelial cells (A-cells) and equatorial cells (E-cells); A-cells are located in the anterior to central zone (corresponding to the zone of the anterior lens capsule) and are relatively quiescent epithelial cells with minimal mitotic activity. When disturbed, they tend to remain in place and not to migrate.The primary response of the A-cells is to proliferate and form fibrous tissue by undergoing fibrous metaplasia.E-cells form the equatorial lens bow with the germinal cells. They normally show mitotic capability, and new lens fibers are continuously produced there. Because cell production in this region is relatively active, the cells are rich in enzymes and have extensive protein metabolism ***(Vargas et al., 1984)***.

The stem cell population of the lens continues to divide throughout life generating daughter cells which undergo cytodifferentiation, massive synthesis of tissue-specific proteins, the soluble crystalline, and loss of the nucleus and other organelles and cause the lens to grow throughout life.

The crystalline is gene product unique to the lens, and due to the isolation of the lens during early embryonic development from both the blood supply and the process of self recognition by the immune system, these unique gene products are never recognized as self and can elicit an autoimmune reaction following eye injury ***(Goodenough, 1992)***.

Contrast sensitivity deteriorates with advancing age even in the absence of ocular pathology such as cataract, glaucoma, and macular degeneration. The pathogenesis of this decline in vision likely involves decreased retinal image quality caused by changes in the spherical aberration of the crystalline lens. Spherical aberration is a property of all spherical lenses, occurs when the lens bends peripheral rays more strongly (positive aberration) or less strongly (negative spherical aberration) ***(Packer, Fine, Hoffman and Piers, 2003)***.

The crystalline lens undergoes changes with age. The lens grows in size and weight throughout life.It has been estimated that the thickness of a human lens increases about 0.02 mm per year.As new fiber cells are formed, older cells are

displaced towards the center, or the lens nucleus, which becomes denser with age ***(Alió, Schimchak and Negri, 2005)***.

Important age-related changes in the lens are in the degree of high angle light scattering. It is likely that this scatter, whether considered as a function of the aging of the entire lens or of particular regions within the lens, is the primary cause of glare. There is an increase in both the amount of insoluble material and the size of the soluble material with age, and it is likely that these two classes of material are responsible for the age-dependent increase in scatter ***(Koretz and Hyun, 1994).***

The cornea has positive spherical aberration, which does not vary significantly with aging. In young people, the internal ocular surfaces, especially the crystalline lens, compensate for this positive value as they are dominated by negative spherical aberrationbecause its index of refraction is lower in the periphery than near the visual axis ***(Beiko, 2007).***

So the prolate cornea and the refractive gradient of the lens reduce spherical aberration ***(Packer et al., 2003).***

The crystalline lens changes with age, which causes a shift in spherical aberration toward positive. The negative spherical aberration in the young lens gradually approaches zero at approximately 40 years of age and continues to become increasingly positive as aging continues. This adds to the positive spherical aberration of the cornea, with possible