INTRODUCTION

Phacoemulsification (phaco) is one of the most widely used cataract surgery techniques nowadays. Various factors involved in phaco can influence the tissue structures of the eyeball. Ultrasonic energy and fluidics produce mechanical effects that cause an inflammatory reaction, compression, and hypoxia on the tissue. Every step of this maneuver can cause direct effects on tissue and instantaneous pressure fluctuation. Fluidics also has radiating pressure effects which resemble a miniature shock wave and jet stream that point directly onto the anterior chamber tissue and are forwarded in all directions. Ultrasonic energy should also be taken into account as a risk factor that may affect the structure of the tissue in the eyeball. In this case, phaco surgeons can measure the ultrasonic energy that was emitted through the phaco time ***(Fine et al., 2001).***

Phaco time consists of two components: effective phaco time (EPT) and absolute phaco time (APT). Both EPT and APT are representatives of any countable ultrasonic energy used during phaco. EPT is a parameter that is calculated by multiplying the total APT by the average percentage of power used, and represents how long phaco time would have been at 100% phaco power ***(Park et al., 2010).***

The retina is a sensory array that requires more oxygen than the brain; the structure is highly sensitive to changes in oxygen levels and changes in the condition of the eyeball.

Microchanges in the retina appear as normal on visual inspection using visual ophthalmoscopy. Microchanges are often not felt by the patient, even after severe damage; only later does the patient begin to notice a decline in visual function ***(Hee et al., 1995).***

The macula is an important structure in charge of the 30-degree field of view, which greatly determines the quality of sharp vision, communication, interpersonal relationships, and color vision, including contrast sensitivity. The central macula has been widely studied, and is closely related to the function of the fovea in visual acuity and color vision ***(Cabrera et al., 2009).***

Optical Coherence Tomography (OCT) is a diagnostic tool which is noncontact, noninvasive, and capable of displaying slices of living tissue with high resolution (high definition). It operates on the principle of coherence interferometry using infrared light with high reliability and high validity, and does not require immersion. OCT has evolved through several generations from the prototype of time domain OCT to the latest generation of Fourier domain OCT or Spectral DomainOptical Coherence Tomography (SD-OCT).

SD-OCT is a diagnostic tool for the tissue layer of the macula and retina with >87% sensitivity and >98% specificity. It uses the principle of spectral detectors for infrared light with a wavelength of about 840 μm. Its display performance can exceed 28,000 A-scans per second with an axial resolution of 1–5 μm and

a transverse resolution of 5–10 μm. The maximum A-scans per B-scan is 8,000 to a depth of 2 mm scan, thus presenting a higher resolution, examination 50–100 times faster, and providing more information than its predecessor ***(Tatrai et al., 2011).***

Corneal endothelial damage following phacoemulsification is still one of the major concerns of modern day cataract surgery. Introduction of different techniques and certain innovations in the phaco instrumentation have reduced the incidence of postoperative corneal endothelial loss. At every stage of cataract surgery, the surgeon has options to optimize corneal endothelial safety. This includes relevant parameters in various phaco platforms, surgical techniques, materials used and patient characteristics such as the grade of the cataract, age of the patient and the presence of corneal disease. Several mechanisms have been proposed for endothelial cell damage during phacoemulsification; these include mechanical contact with nuclear fragments, irrigation flow, turbulence and movement of fluid, direct trauma caused by instruments or lens fragments, and formation of cavitation bubbles ***(Linebarger et al., 1999).***

In phacoemulsification procedures the most important predictor of corneal clarity in the early postoperative period is inversely proportional to the amount of ultrasound energy used. For this reason, all of the major platforms now use power modulation to reduce the amount of phaco power necessary, either by varying the duty cycle and pulse energy or by varying the pulse intervals with microburst techniques ***(Steinert, 2010).***

Several phacoemulsification techniques have been developed and modified. Although many techniques have been proposed, the risks of posterior capsular rupture and corneal endothelium damage persist. In theory, damage to the corneal endothelium is minimized by delivering the lowest phaco energy only in the direction necessary to emulsify the lens nucleus. Furthermore, phacoe-emulsification should occur in the posterior chamber rather than in the iris plane or theanterior chamber. There is a difference of opinion among ophthalmologists with respect to the phaco tip position during phaco-emulsification. Hence, it has traditionally been believed that the bevel of the needle should be turned towards the nucleus or the nuclear fragment (ie, bevel down). However, some studieshypothesize that in the bevel-up technique is better as therefore the source of heat, are farther from the endothelial cells than in the bevel-down technique and this decreases the chance of endothelial cell damage. Further clinical trials concluded in their study that bevel-up tip position has a negative effect on corneal endothelial cells compared with the bevel-down position ***(Faramarzi et al., 2011).***

Corneal topography provides highly detailed information about corneal curvature. Topography is evaluated using keratoscopic images, which are captured from Placido disk patterns that are reflected from the tear film overlying the corneal surface and then converted to computerized color scales ***(Roberts, 1996).***

AIM OF THE WORK

To evaluate the effect of phacoemulsification on cornea, retina and choroid and to compare these effects in diabetic and non-diabetic patient to reach a conclusion whether diabetic patient are more vulnerable to these effects or not.

We can also evaluate the effect of diabetes on cornea retina and choroid through measuring central corneal thickness, central foveal thickness & sub foveal choroidal thickness preoperatively.

To achieve this study purpose we had to evaluate and compare central foveal thickness (CFT) changes, sub foveal choroidal thickness changes, using Spectral Domain optical coherence tomography (SD-OCT), and central corneal thickness (CCT) changes using pentacam, before and after cataract surgery (after one week and after one month) and compare the results between normal and diabetic patients without retinopathy.

REVIEW OF LITERATURE

Cornea

In the average adult, the horizontal diameter of the cornea is 11.5 to 12.0 mm and about 1.0 mm larger than the vertical diameter. It is approximately 0.5 mm thick at the center and gradually increases in thickness toward the periphery. The shape of the cornea is prolate flatter in the periphery and steeper centrally which creates an aspheric optical system. Corneal shape and curvature are governed by the intrinsic biomechanical structure and extrinsic environment. Anterior corneal stromal rigidity appears to be particularly important in maintaining the corneal curvature ***(Muller et al., 2001).***

The human cornea consists of 5 recognized layers, 3 cellular (epithelium, stroma, endothelium) and 2 interface (Bowman membrane, Descemet membrane), recently a sixth layer is believed by some authors to exist between the Descement membrane and the stroma called Dua'slayer as shown in figure (1) ***(Dua et al., 2015).***

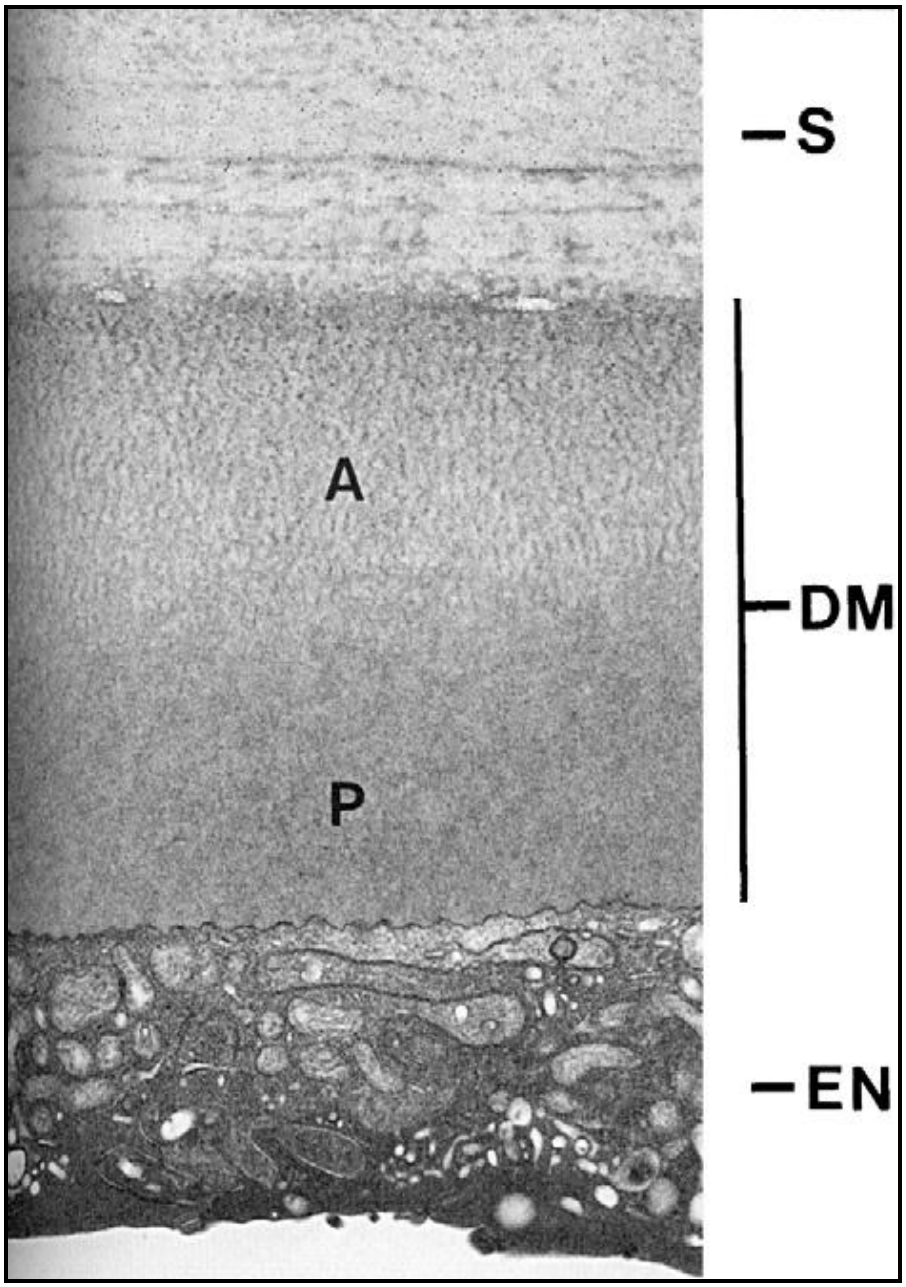

Figure (1): Micrograph illustrating Descemet membrane (DM) located between the posterior aspect of the corneal stroma (S) and the underlying endothelium (EN). The anterior "banded" region (A) is secreted by the endothelial cells during fetal development and is more highly organized than the posterior "amorphous region" (P), which is secreted after birth ***(Farjo et al., 2008).***

Epithelium

The epithelial surface of the cornea creates the first barrier to the outside environment and is an integral part of the tear film–cornea interface that is critical to the refractive power of the eye. It is a stratified, nonkeratinizing squamous layer characterized by extreme uniformity. Embryologically, the corneal epithelium is derived from surface ectoderm between 5 and 6 weeks of gestation. It is composed of 4 to 6 cell layers thick (40 mm to 50 mm).The epithelium is covered with a tear film, which is optically important in smoothing out micro irregularities of the anterior epithelial surface. The air–tear film interface, together with the underlying cornea, provides two thirds of the total refractive power of the eye as shown in figure (2) ***(Farjo et al., 2008).***

The corneal epithelium and overlying tear film have a symbiotic relationship both anatomically and physiologically. The mucinous layer of the tear film, which is in direct contact with the corneal epithelium, is produced by the conjunctival goblet cells and interacts closely with the corneal epithelial cell glycocalyx to allow hydrophilic spreading of the tear film with each eyelid blink. It has been suggested that the epithelium itself may contribute to this mucinous layer, but this is unproven ***(Gipson et al., 1992).***

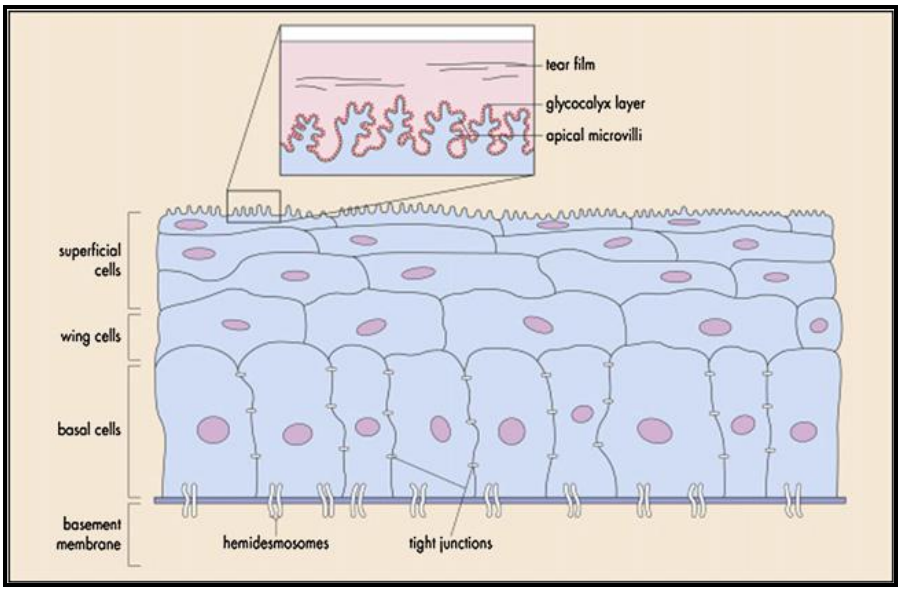

Figure (2): Cross-sectional view of the corneal epithelial cell layer ***(Farjo et al., 2008).***

Corneal epithelial cells have an average lifespan of 7 to 10 days7 and routinely undergo orderly involution, apoptosis (programmed cell death), and desquamation. This process results in complete turnover of the corneal epithelial layer every week as deeper cells replace the desquamating superficial cells in an orderly, apically directed fashion. The most superficial corneal epithelial cells form a mean of 2 to 3 layers of flat polygonal cells. These cells have extensive apical microvilli and microplicae, which in turn are covered by a fine, closely apposed, charged glycocalyceal layer. This layer's apical membrane projections increase the surface area of contact and adherence between the tear film's mucinous layer and the cell membrane ***(Farjo et al., 2008).***

Bowman Layer

Bowman layer (or Bowman membrane) lies just anterior to the stroma and is not a true membrane but rather the acellular condensate of the most anterior portion of the stroma. This smooth layer is approximately 15 mm thick and helps the cornea maintain its shape. When disrupted, it will not regenerate and can form a scar

Stroma

The corneal stroma provides the bulk of the structural framework of the cornea and comprises roughly 80% to 85% of its thickness. Embryologically, it is the result of a second wave of neural crest migration that occurs in the seventh week of gestation, after establishment of the primitive endothelium. The stroma differs from other collagenous structures in its transparency, which is the result of precise organization of the stromal fibers and extracellular matrix (ECM) ***(Boote et al., 2003).***

The collagen fibers are arranged in parallel bundles called fibrils, and these fibrils are packed in parallel arranged layers or lamellae. The stroma of the human eye contains 200 to 250 distinct lamella, each layer arranged at right angles relative to fibers in adjacent lamellae. The peripheral stroma is thicker than the central stroma, and the collagen fibrils may change direction to run circumferentially as they approach the limbus. This highly organized network reduces forward light

scatter and contributes to the transparency and mechanical strength of the cornea ***(Meek et al., 2004).***

An additional feature of the stroma is that the ultrastructure within the organization of the lamella appears to vary based on the depth within the stroma. Deeper layers are more strictly organized than superficial layers, and this difference accounts for the greater ease of surgical dissection in a particular plane as one approaches the posterior depths of the corneal stroma. This variation in stromal organization also accounts for the differences in response to corneal edema, as mentioned previously. Descemet folds are the result of asymmetric swelling of the posterior stroma imposed by the structurally more rigid anterior cornea and structural restriction imposed by the limbus ***(Gipson et al., 2008).***

Stromal swelling is therefore directed posteriorly and results in relative flattening of the posterior surface, which can push Descemet membrane into multiple folds that become visible as striae. Stromal collagen fibrils are composed of type I collagen in a heterodimeric complex with type V collagen to obtain their unique and narrow diameter ***(Fini and Stramer, 2005).***

These complexes are surrounded by specialized proteoglycans, consisting of keratan sulfate or chondroitin sulfate/dermatan sulfate side chains, which help regulate hydration and structural properties. Keratocytes are the major

cell type of the stroma and are involved in maintaining the extra cellular matrix (ECM) environment. They are able to synthesize collagen molecules and glycosaminoglycans while also creating matrix metalloproteases (MMPs) helping in maintaining stromal homeostasis ***(Jester et al., 1999).***

Descemet Membrane

Beginning in utero at the 8-week stage, endothelial cells continuously secrete Descemet membrane. The anterior 3 µm secreted prior to birth has a distinctive banded appearance when viewed by electron microscopy, but Descemet membrane produced after birth is unbanded and has an amorphous ultrastructural texture. Descemet can accumulate up to 10 mm in thickness with age.

Dua's layer

The initial publication on Dua's layer created some Controversy. Many corneal surgeons recently consider it as a plane of cleavage in deep anterior lamellar keratoplasty (DALK) and others contested its identity as a distinct layer ***(Goweida, 2015).***

Endothelium

The endothelial layer of the cornea maintains corneal clarity by ensuring it remains in a relatively deturgesced state. The intact human endothelium is a monolayer, which appears as a honeycomb-like mosaic when viewed from the posterior

side. In early embryogenesis, the posterior cornea is lined with a neural crest-derived monolayer of orderly arranged cuboidal cells as shown in figure (3) ***(Beebe and Coats, 2000).***

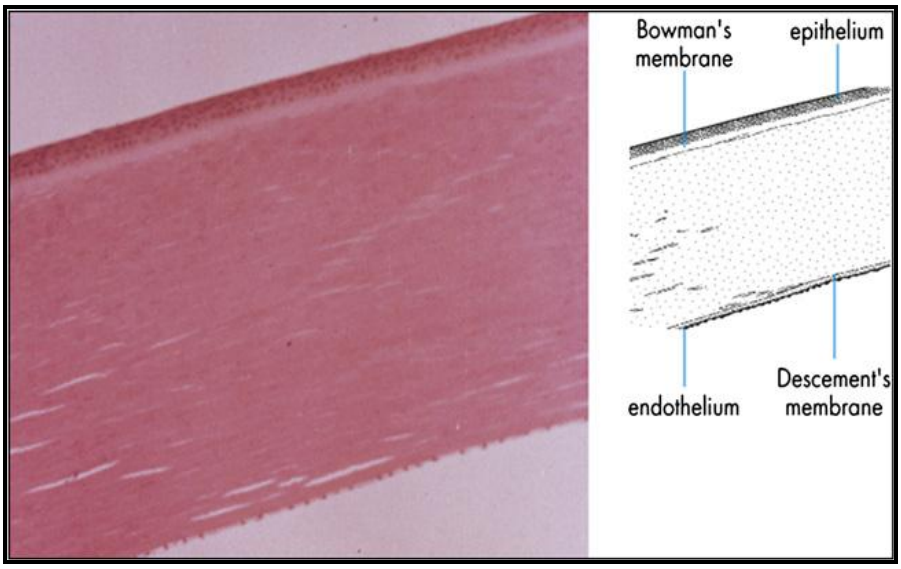

Figure (3): Light micrograph of normal endothelium (original magnification_100). Note the single-cell endothelial layer with a Descemet membrane of uniform thickness (epithelial surface at top of figure) ***(Beebe and Coats, 2000).***

At birth, the endothelial monolayer is approximately 10 mm thick and consists of a uniform thickness layer of cells that spans the entire posterior corneal surface and fuses with the cells of the trabecular meshwork. Similarly, Descemet membrane becomes continuous and uniform, fusing peripherally with the trabecular beams. The fusion site, known as Schwalbe line, is a gonioscopic landmark that defines the end of Descemet membrane and the start of the trabecular meshwork ***(Watsky et al., 1989).***

Adjacent endothelial cells share extensive lateral interdigitations and possess gap and tight junctions along their lateral borders. The lateral membranes contain a high density of Na-K ATPase pump sites. The basal surface of the endothelium contains numerous hemidesmosomes that promote adhesion to Descemet membrane ***(Stiemke et al., 1991).***

Endothelial cell density and topography continue to change throughout life. From the second to eighth decades of life, the cell density declines from 3000 to 4000 cells/mm2 to around 2600 cells/mm2, and the percentage of hexagonal cells declines from approximately 75% to approximately 60%. The central endothelial cell density decreases at an average rate of 0.6% per year in normal corneas ***(Bourne et al., 1997).***

Endothelial cells have no mitotic activity in vivo; however, humans are born with a significant reserve. Cell density is approximately 3500 cells/mm2 at birth, but this number decreases gradually throughout life at approximately 0.6% per year. It has been observed that eyes with endothelial cell counts below 500 cells/mm2 may be at risk for the development of corneal edema. Endothelial cell morphology (size and shape) also appears to correlate with pump function. An increase in cell size (polymegathism) and an increase in variation of cell shape (pleomorphism) correlate to reduced ability of the endothelial cells to deturgesce the cornea. The number of endothelial cells decreases with age, trauma, inflammation, and other disease processes (ie, Fuchs endothelial dystrophy), but the remaining

cells have the capacity to "stretch" and take over the space of the degenerated endothelial cells. As this process occurs, the remaining cells grow in size (polymegathism) and lose their hexagonal shape (pleomorphism) ***(Polse et al., 1990).***

Corneal Responses to Injury

Epithelial Injury

When any portion of an epithelial cell is disrupted, the entire cell is usually lost, leaving a defect in the epithelial layer. The most common form of injury to the epithelium is mechanical, but thermal and chemical injuries are also possible. When a mechanical force creates a break in the epithelial barrier, cells at the edge of the abrasion begin to cover the defect within minutes by a combination of cell migration and cell spreading. This process is preceded by almost immediate preparatory cellular changes of an anatomical, physiological, and biochemical nature, including the creation of cell membrane extensions, disappearance of hemidesmosome adhesions from the basal cells, and increase in mitochondrial energy production ***(Matsuda et al., 1985).***

This early nonmitotic wound coverage phase can proceed at the remarkable rate of 60 to 80 mm per hour. Studies have shown that the migrating sheet of epithelial cells is attached most firmly to the underlying substrate at the leading margin, possibly suggesting that the leading cells are "pulling" the epithelial sheet as it migrates. The ECM protein fibronectin is