INTRODUCTION

Myopia is the commonest refractive error. The extent to which members of a population are myopic varies between races. In the United States and other Western countries, about a quarter of the population are myopic by their late teens with this figure rising to approximately one third by 40 years of age.

Pathological myopia means that there is greater than six dioptres of myopia or an axial length greater than 26-27mm ***(Pan et al., 2012)***.

It is a progressive, probably autosomal recessive disorder where serious ocular complications can develop such as chorioretinal degeneration, posterior staphyloma, retinal detachment, primary open angle glaucoma and cataract ***(Lee et al., 1999 and Miller & Singerman, 2001).***

According to the Beaver Dam Eye Study and the Blue Mountains Eye Study published in 2014, there is an association between myopia and nuclear cataract. The Blue Mountains Eye Study found that moderate and high myopia, especially with onset prior to age 20, are associated with posterior subcapsular cataract formation ***(Panchapakesan et al., 2003 and Meuer et al., 2014).***

For a number of years, phacoemulsification has been the method of choice for cataract extraction in developed countries.

With increasingly advanced microprocessors, phacoemulsification can offer safe and elegant disassembly and aspiration of the lens and a rapid recovery for patients. However, pseudophakia changes the physiology of the eye and immediate changes include release of inflammatory cytokines. Deformation of the globe and intraoperative fluctuations of intraocular pressure may induce microlesions and alteration of ocular perfusion. The barrier function between the anterior and the posterior segment is compromised after surgery, especially the vitreous body which is subject to increased destruction during the years after surgery ***(Herrmann et al., 2010).***

These changes in the posterior segment are best recognized by Optical Coherence Tomography (OCT). OCT was first reported in 1991 as a non-invasive, cross-sectional ocular imaging technology and today is the most promising non-contact, high resolution tomographic and biomicroscopic imaging device in ophthalmology ***(Huang et al., 1991)***.

It is a computerized instrument structured on the principle of low-coherence interferometry generating a pseudo-color representation of the tissue structures, based on the intensity of light returning from the scanned tissue. As the resolution of OCT has been improving with time, the localization and quantification of the tissues has accordingly, become more refined, faster and predictable ***(Hrynchak & Simpson, 2000 and Keane & Sadda, 2014).***

AIM OF THE WORK

To assess the impact of cataract surgery by phacoemulsification on the posterior segment in myopic patients by using an OCT device.

HIGH MYOPIA

A) Introduction:

Myopia is the state of refraction in which parallel rays of light are brought to a focus in front of the retina of a resting eye (*Curtin, 1985*). Myopia is measured by the spherical power in Diopters (D) of the diverging lens needed to focus light onto the retina as shown in figure 1 ***(Curtin, 1988, Angle and Wissmann, 1980).***

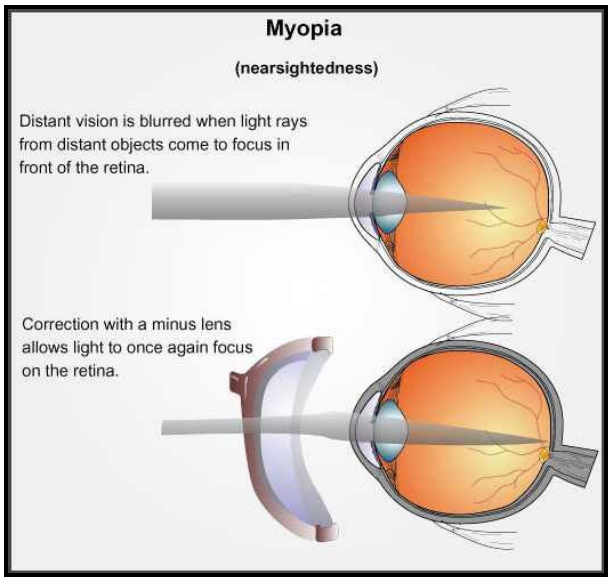

Figure (1): Myopia (Nearsightedness)

Myopia is present when either the refractive power of the eye, dependent mainly on the corneal curvature and lens, is too high or when the axial length (AXL) of the eyeball is too long.

One classification of myopia in relation to the power of refraction distinguishes low (up to-3 D), moderate (-3 to -6 D),

and high (above-6 D) myopia. According to another classification, the respective ranges for myopia are to -4 D (low), -4 to -8 D (moderate), and above -8 D (high) ***(Edwards and Lam, 1999, Zając-Pytrus, 2000).***

Myopia can also be divided according to the age at onset, into congenital myopia (often accompanied by prematurity) and acquired myopia. Acquired myopia may appear before the age of 20 (youth-onset myopia), between the ages of 20 and 40 (early adult-onset myopia), and over 40 (late adult-onset myopia) ***(Zając-Pytrus, 2000 and Niżankowska, 1999).***

High axial myopia is characterized by an elongation of the globe, predominantly at the posterior pole. One may define the cutoff value for high axial myopia as the refractive error or AXL at which the size of the optic disc and the parapapillary atrophy markedly enlarges. Hospital-based and population-based investigations have revealed that the optic disc and the parapapillary atrophy start to enlarge at about a value of -6.00 Ds of refractive error or an AXL of ~26.5 mm ***(Wang et al., 2006; Xu et al., 2010 and Logan et al., 2011).*** Beyond these values, the prevalence of myopic retinopathy and glaucomatous optic neuropathy steeply increases ***(Liu et al., 2010 and Jonas, 2005)***. One may therefore consider high axial myopia as a myopia refractive error of >-6.00 Ds or an AXL >26.5 mm.

Whether pathologic myopia and high myopia are two separate types of myopia or whether pathologic myopia is a subset of high myopia is still subject to debate. Pathologic myopia is best defined as high myopia with degenerative changes in the eye. Degenerative changes include: posterior staphyloma, retinal detachment, retinal holes, chorioretinal atrophy, Fuchs spot, subretinal neovascularisation, vitreous degeneration, LC, and RPE changes as shown in figure 2 ***(Spaide et al., 2014)*.**

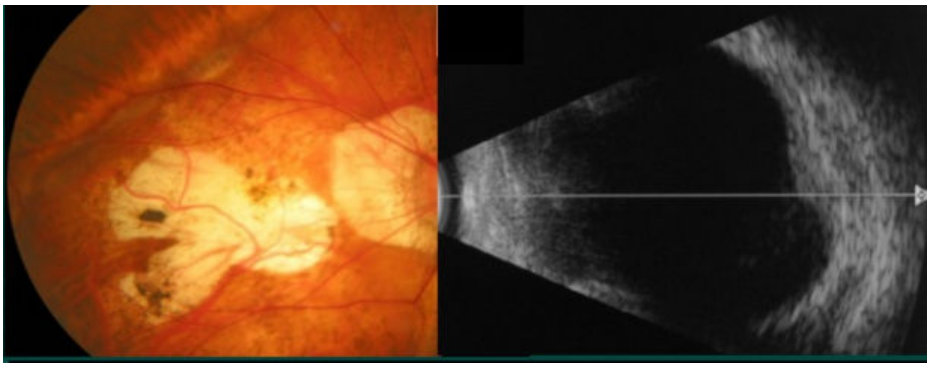

Figure (2): High axial elongation associated with degenerative changes in degenerative myopia

B) Burden and epidemiology:

Myopia is a frequent cause of visual impairment and morbidity worldwide ***(Pan et al., 2012; Pascolini and Mariotti, 2010)***. High axial myopia confers increased risk of sight-threatening ocular pathology such as RRD, glaucomatous optic neuropathy, and myopic retinopathy ***(Liu et al., 2010 and Jonas, 2005).***

Its significant burden also includes cost of correction and the association of uncorrected vision with increased risk of falls (particularly in the elderly). ***(Vitale et al., 2006; Evans & Rowlands, 2004; Harwood, 2001; Cox et al., 2005 and Rahi et al., 2008).***

The global prevalence of myopia is increasing at a disturbing rate. It has been estimated that over a quarter of the world's population in 2010 had myopia with it expected to rise to over half the population of the world by 2050 ***(Holden et al., 2015).***

Highly myopic individuals account for 2-4% of the population across Europe, Australia and United States ***(Kempen et al., 2004; Pan et al., 2013 and Williams et al., 2015).*** Its prevalence varies across populations of different regions and ethnicities being higher in urban areas and Chinese ethnicity ***(He, et al., 2009 and Lam et al., 2012)*** especially in the Asia-Pacific high income region, which includes the Republic of Korea, Japan and Singapore ***(Jung et al., 2012 and Wang et al., 2008).***

High myopia is also predicted to increase from around 3 per cent to 10 per cent in 2050, with the number of people worldwide potentially developing vision impairment or permanent blindness associated with higher levels of myopia expected to be at least one billion by 2050 ***(Holden et al., 2015).***

C) Risk Factors:

The exact pathogenic mechanisms of myopia remain unclear. Recent evidence suggests that myopia is likely to result from the combined and interacting effects of hereditary and environmental factors ***(Chua et al., 2015 and Saw et al., 2001).***

Many factors have been documented such as parental myopia, gender, ethnicity, education, occupation, income, near-work load, outdoor activities, lens opacity, and ocular dimensions ***(Lyu et al., 2015; Morgan et al., 2006 and Wong et al., 2001).***

D) Pathological changes in the posterior segment of the globe associated with high axial myopia:

In non-axially elongated eyes with an AXL of ≤26.5 mm, mean scleral thickness decreases from the limbus (0.82±0.15mm) to the ora serrata (0.43±0.14 mm) and the equator (0.42±0.15 mm), and then increases to the midpoint between posterior pole and equator (0.65±0.15 mm) to the peri-optic nerve region at the merging point of the dura mater with the posterior sclera (0.86±0.21 mm), and finally to the posterior pole (0.94±0.18 mm). The thickness of the peripapillary scleral flange (i.e. bridge between the lamina cribrosa of the RPE and the posterior sclera) is the lowest of all measurements (0.39±0.09 mm) ***(Vurgese et al., 2012 and Nangia et al., 2010).***

High axial myopia is associated with numerous anatomical changes in the posterior pole of the globe. These changes that can also be imaged by OCT may explain some of the associations between high myopia and vision-threatening diseases such as glaucoma and myopic retinopathy ***(Jonas and Xu L, 2014).***

- Scleral Changes:

In highly axially myopic eyes, mean scleral thickness is significantly lower than in the non-highly myopic eyes for measurements taken at and posterior to the equator, whereas scleral thickness anterior to the equator does not differ significantly ***(Vurgese et al., 2012 and Nangia et al., 2010).***

This scleral thinning at the posterior pole and thinning of the peripapillary scleral flange are significantly correlated with thinning of the lamina cribrosa but not with corneal thickness measurements. Interestingly, globes with secondary high axial myopia due to congenital glaucoma show a tendency to a thinning of the sclera in all regions including the region anterior to the equator ***(Wei et al., 2013).***

Posterior staphyloma can be defined as local bulging of the sclera at the posterior pole of the eye that has a radius of less than the surrounding curvature of the wall of the eye as shown in figure 3 ***(Ohno-Matsui et al., 2015).***

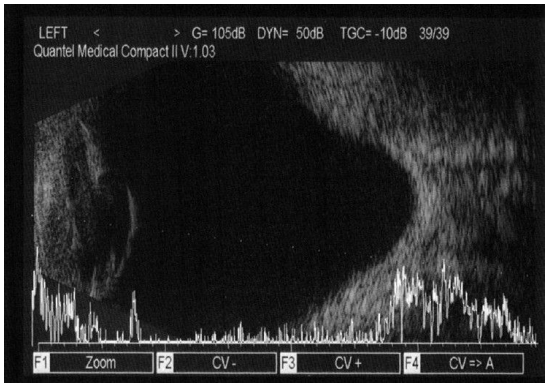

Figure (3): B scan U/S showing posterior staphyloma

- Choroidal Changes:

The choroid shows marked thinning with increasing axial elongation with the mean choroidal thickness decreasing from 250μm in emmetropic subjects to <30μm in highly axially myopic patients ***(Jonas et al., 2013 and Wang et al., 2015).***

Highly axially myopic eyes shows defects in the Bruch's membrane in the macular region called secondary defects (primary defects are defects in the Bruch's membrane in the region of the RPE). These defects are associated with a complete loss of Retinal Pigment Epithelium (RPE) cells and choriocapillaris, and an almost complete loss of the large choroidal vessel layer and photoreceptors. They can histologically be differentiated from myopic chorioretinal atrophic areas in the macular region where there is a complete loss of RPE cells and choriocapillaris, but with the Bruch's membrane present ***(Reis et al., 2012).***

- Optic Nerve Head (ONH) changes:

In axial myopia, the elongation of the globe takes place more in the posterior segment so that the position of the exit of the optic nerve moves from a location close to the posterior pole more to a location at the nasal wall of the globe. This shift in the position may not completely be followed by the Bruch's membrane, with the Bruch's membrane opening keeping a location closer to the posterior pole. This leads to an overhanging of the Bruch's membrane in the nasal region of the ONH and to its absence at the temporal disc border. The region without the Bruch's membrane (and correspondingly, without RPE and choriocapillaris) has been called parapapillary gamma zone ***(Reis et al., 2012; Jonas et al., 2012 and Dai et al., 2013).***

The optic disc in highly axially myopic eyes is enlarged and is so called 'secondary or acquired macrodiscs', whereas the lamina cribrosa shows a stretching and thinning ***(Jonas et al., 2004).***

With the increase in the aperture of the peripapillary scleral flange opening (called scleral canal of the RPE) but with the optic nerve remaining constant in size, the peripheral posterior surface of the lamina cribrosa is no longer covered and buffered by the solid tissue of the RPE. Instead, it gets exposed to the orbital cerebrospinal fluid space, pressure-

related pathological changes may then predominantly take place in the periphery of the lamina cribrosa close at the optic disc border. This is demonstrated in figure 4 ***(Jonas et al., 2003).***

Figure (4): RPE changes associated with high axial myopia

E) <u>Complications of high myopia:</u>

The pathophysiology of pathologic myopia is not fully understood, but the axial elongation of the eye followed by chorioretinal thinning is suggested as a key mechanism. Pathologic myopia may lead to many complications. Some complications affect visual acuity significantly, showing poor visual prognosis ***(Cho et al., 2016).***

1) Vitreous changes and PVD:

High myopia is associated with vitreous liquefaction, and posterior vitreous detachment (PVD) which occurs at a younger age than in non-myopic eyes. Residual cortex on the retina is occasionally encountered during the surgery performed to treat MFS despite the apparent PVD with a Weiss ring, this is due to the presence of posterior precortical vitreous pocket (PPVP); which is a boat-shaped vitreous lacuna in front of the posterior pole. This is shown in figure 5 ***(Akiba, 1993, Itakura and Kishi, 2011).***

The PPVP has a key role in the development of various vitreomacular disorders; its presence with partial PVD around the macula is a precursor of complete PVD. It was reported that a perifoveal vitreous detachment is the primary pathogenic event in idiopathic macular hole formation ***(Spaide et al., 2002).***

Recently introduced swept-source OCT (SS-OCT) clearly showed the PPVP increases along with increasing the myopic refractive error ***(Mojana et al., 2010, Itakura et al., 2013).***

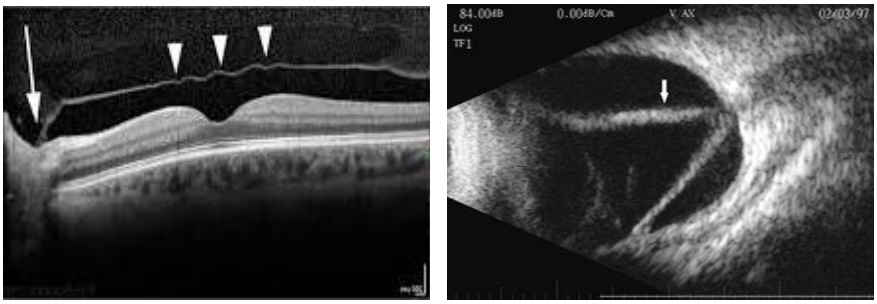

Figure (5): B scan U/S (left side) and SD-OCT (right side) showing PVD

2) Macular foveoschisis (MFS) and holes:

MFS is the splitting of the retinal layers in the macula, OCT is used to diagnose the splitting of the neural retina into a thicker inner layer and a thinner outer layer, but compound variants of the splits have been identified. It is often asymptomatic initially but progresses slowly, leading to loss of central vision from foveal detachment or macular hole formation, as shown in figure 6 & 7.

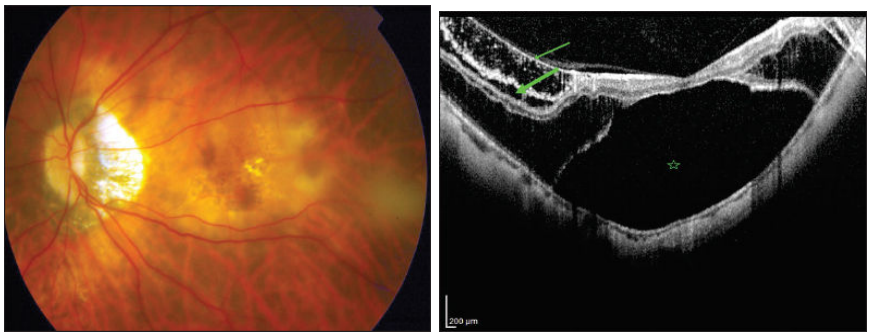

Figure (6): Coloured fundus photograph showing MFS (left side) and SD-OCT image (right side) demonstrating typical MFS and foveal detachment

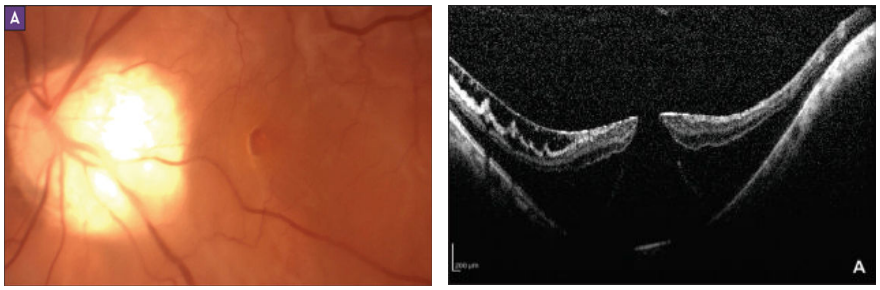

Figure (7): Coloured fundus photograph showing macular hole (left side) and SD-OCT showing full thickness macular hole

Vitrectomy with peeling of the internal limiting membrane and gas tamponade is the preferred approach for eyes with vision decline ***(Ikuno et al., 2004 and Cheung et al., 2007)***. However, reopening of the macular hole and retinal redetachment may develop and some patients will require multiple surgeries to achieve attachment due to the loss of chorioretinal tissue and RPE atrophy ***(Gohil et al., 2015; Takano and Kishi S, 1999).***

3) <u>Tessellated (Tigroid) Fundus and Chorioretinal atrophy:</u>

In eyes with high myopia, hypoplasia and thinning of the RPE following axial elongation reduces the pigment, allowing the choroidal vessels to be seen ***(Harman et al., 1999).***

Diffuse chorioretinal atrophy is observed as ill-defined yellowish lesion in the posterior fundus of highly myopic eyes. This lesion begins to appear around the optic disc and increases with age and finally covers the entire area within the staphyloma.

Patchy chorioretinal atrophy is observed as a grayish-white, well defined atrophy. Due to the absence of RPE and most of the choroid, the sclera can be observed through transparent retinal tissue, which is considered to show white color. Large choroidal vessels seem to course within the area of patchy atrophy. Pigment clumping is observed within the area of patchy atrophy especially along the margin of the atrophy or