"A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF THE ASSOCIATION BETWEEN THE PATTERN OF MYOPIC FUNDAL CHANGES AND THE AXIS OF ASTIGMATISM OF THE EYE"

By

REG. NO. BK0112002

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Endorsement by the Head Of Department, Principal/ Head of the Institution

This is to certify that the dissertation entitled **"A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF THE ASSOCIATION BETWEEN THE PATTERN OF MYOPIC FUNDAL CHANGES AND THE AXIS OF ASTIGMATISM OF THE EYE"** is a bonafide research work done by **REG. NO. BK0112002.**

Seal & Signature of the HOD

Dr. S. B. Patil M.S, DOMS
Professor & Head,
Department of Ophthalmology,
J. N. Medical College,
Belgaum – 590010.
Karnataka, India.

Date :

Place: Belgaum

Seal & Signature of the Principal

Dr. (Mrs.) N. S. Mahantshetti MD (Paed)
Principal,
J. N. Medical College,
Nehru Nagar,
Belgaum - 590010.
Karnataka, India.

Date :

Place: Belgaum

LIST OF ABBREVIATIONS

M	-	Male
F	-	Female
D	-	Dioptres
DS	-	Dioptric Sphere
DC	-	Dioptric Cylinder
CF	-	Counting fingers
BCVA	-	Best Corrected Visual Acuity
UCVA	-	Uncorrected Visual Acuity
AL	-	Axial Length
Mm	-	Millimetre
WTR	-	With-the-rule
ATR	-	Against-the-rule
CNVM	-	Choroidal Neovascular Membrane
AC	-	Anterior chamber
PDT	-	Photodynamic therapy

ABSTRACT

Purpose of the study:

The study was conducted as a cross sectional study to assess the correlation, if any, between the pattern of myopic fundal changes and the axis of astigmatism and to find an etiopathogenic association in the occurrence of myopic fundal changes.

Methods:

A total of 97 eyes of 64 patients having myopic disc changes, i.e., peripapillary atrophy or myopic crescents were studied. Each eye had undergone subjective refraction testing, measurement of corneal curvature, axial length and anterior chamber depth using A – Scan Biometer (Echorule2, Biomedix).

Fundoscopic examination was carried out thoroughly by indirect ophthalmoscopy with +20 D lens. The fundus photograph was recorded with a Canon CF–1 Digital Retina Camera. The deep/longest axis of the crescent/peripapillary atrophy was determined and the axis of myopic crescent or peripapillary atrophy was measured using the annotations incorporated within the software attached to the fundus camera and was ascribed a value in degrees, in a manner similar to the axis of astigmatism in refraction.

The long axis of peripapillary changes and of the disc crescent were correlated with the axis of astigmatism in patients with compound myopic astigmatism and the coefficient of correlation determined.

Results:

A statistically significant correlation was found between the axis of myopic astigmatism and the long axis of the myopic retinal degeneration (crescent or peripapillary atrophy). According to Spearman's rank correlation co-efficient, $p < 0.001$ which was considered significant.

Conclusion:

A positive correlation is present between the axis of astigmatism of the eye and of the angle of the myopic fundal changes.

Astigmatism may have an aetiopathogenic association in the occurrence of myopic fundal changes.

Key words: Myopia; Astigmatism; Peripapillary Atrophy; Crescent; Axial Length.

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INTRODUCTION

Refractive Error is the most common clinical condition in ophthalmology. Myopia is a common optical aberration and the most common visual disorder in the world. Physiological myopia, is by far the most prevalent and is less than -6D in magnitude. It is considered a normal biological variation.

Eyes with errors more than -6D are said to have high myopia. Not all the eyes that have myopia more than -6D progress, nor does every eye with progressive myopia develop degenerative complications. High Myopia associated with secondary degenerative changes is known as Pathological Myopia.

Pathological Myopia is the most important of all refractive errors. It is associated with increased risk of the development of sight threatening eye diseases, such as glaucoma, macular hemorrhage, retinal detachment, visual impairment, and blindness.

It leads to visual disability and may eventually lead to blindness.

As per the 2001-02 national survey on blindness in India, refractive errors account for 19.7% of total blindness.[1] The prevalence of pathological myopia is estimated to be 1-3% in population based studies.[2]

Prevalence of myopia is different among different ethnic groups and is least in blacks and greatest in Asians. Out of the mixed population of USA, 25% were myopic, among a subset of African-Americans. When 1,20,000 Chinese individuals were studied, 70% were found to be myopic.[3]Also, myopia was seen more often in women (48%) than men (41%). Pathological myopia is also influenced by ethnicity but is observed less frequently.

High myopia, also refers to myopic eyes with very long axial lengths (26 mm) or a high degree of myopic refractive error (-6D).

Recently, the incidence of high myopia has been increasing all over the world, especially in the younger East Asian population. Different countries show considerable variation of prevalence rates.[4]

The clinical diagnosis of pathological myopia is simple. When there is myopia of more than -6D, the fundus may reveal characteristic changes, some of which can be accredited to increased axial elongation along with subsequent mechanical stretching as well as thinning of the choroid and retinal pigment epithelium with vascular and degenerative changes.[6] In pathological myopia, the early changes include localized tessellations and myopic crescent formation. Peripapillary atrophy (PPA) may also follow around the disc.[7]

Chorioretinal degenerative changes also appear along the outer margin of the crescent. The area of these changes and staphyloma formation conform to the area of retinal pigment epithelium thinning. Eventually, the mechanical aspects associated with ectasia of the fundus become more evident. There is increased loss of choroidal circulation and increased chorioretinal degeneration.

The treatment for simple myopia is easy, but for pathological myopia it may pose a challenge. Although corrective glasses may improve vision to a certain extent, the patient is often not satisfied with the quality of vision.

Also, high myopic eyes may harbor chronic simple glaucoma, which is difficult to diagnose and can lead to gradual loss of visual fields.

Myopic eyes are also predisposed to complications such as Retinal Detachment, which may result in complete loss of vision.

Therefore, a highly myopic eye is always a susceptible eye and is always prone to risks. The course of a high myopic eye and its prognosis is always unpredictable.

Astigmatism is defined as the cylinder power of more than 0.5DC. The astigmatism axis is classified as "with-the-rule" if the axis is between 75° and 105° and "against-the-rule" if the axis is between 0° and 15° or between 165° and 180° and oblique if itis at any other meridian.

Myopic astigmatism is a common and significant cause of visual impairment.[8,9]

It has been reported that an uncorrected astigmatic error can induce myopia and even promote its progression.[10-13]

Astigmatism is the most common refractive error in certain countries such as Indonesia[14], Taiwan[15] and Japan[16] and approximately half of the people in these areas suffer from astigmatism. Age, gender, genetics and even environmental factors have been shown to affect astigmatism in different studies.[17,18,19,16-28]

Astigmatism has major differences compared to myopia and hyperopia. The prevalence of myopia and hyperopia is presented in amount and percentage, however, there are different types of astigmatism mainly related to cornea.

Furthermore, the astigmatic axis is an important indicator of this refractive error which in some cases, is more important than the magnitude of astigmatic power.[22,29]

To our knowledge, the correlation between pathological changes in high myopia and the axis of astigmatism has not been studied in India before. This study was conducted to assess the correlation, if any, between the pattern of myopic fundal changes and the axis of astigmatism.

AIMS AND OBJECTIVES

1. To assess the correlation, if any, between the pattern of myopic fundal changes and the axis of astigmatism.
2. To find an etiopathogenic association in the occurrence of myopic fundal changes.

REVIEW OF LITERATURE

Historical Review

The subject of the cause and prevention of myopia is possibly the most highly discussed topic in ophthalmology. High degrees of hyperopia and astigmatism have always been explained rather satisfactorily by attributing them to a congenital deformation of the eyeball. The rare cases of high myopia at birth fall into the same category. But acquired myopia, has always been an intriguing discussion. Because the affliction is so widespread and has affected millions, many statistics have been assembled and a lot of time and labour has been devoted to studying it.

The first person to distinguish between the conditions called myopia and hyperopia is thought to be Aristotle (384-321 BC). However, the credit of using the word *myopia*, goes to Galen (138-201 AD) who lived for many years in Rome. The first written record of the term is in Libris Pandectorum, which is a collection of Roman Law that was completed in 65 AD.

In Greek, *myein* means to close and *opos* means the eye. Hence, the original meaning of the Greek word *myopos* and the Latin word *myops* was to describe a condition in which a person would attempt to see clearly by partially closing the eyes. This squinting is often observed in myopes, since it restricts the vision to a narrow band across the center of the pupil, cutting off the light rays passing above and below the lens (rays which would only increase the amount of blur at the retina) and thus resulting in temporary improvement of vision. In addition, if the narrowing of the eyelids can be used to put sufficient pressure on the front of the eye, the eye can temporarily be made shorter and less myopic. [30]

As early as 1813, James Ware noted that the men under his command were not nearsighted, whereas the officers of the Queen's Guard were. He realized that they became nearsighted because they were all educated and could read, whereas the men in his company could not. It led him to believe that reading was the cause of the myopia. [30]

Cohn (1864), presented evidence that myopia developed in many youngsters after they began attending school, leading him to conclude that it was attending school and the accompanying overuse of eyes for close work which produced myopia. Cohn is therefore considered to be the originator of the environmental theory of myopia development. He tried to point out the need for improved reading habits and illumination in the schools.[30]

In Germany, rules were set regarding the size of type in schoolbooks, the distance at which the book was to be held and the amount of lighting. These efforts failed however since they did not go far enough, although they were a step in the right direction. Also, since these efforts took place before electric lights came into widespread use, it is unlikely that the lighting in the schools could have been very good. [30]

Hirschberg (1982) accredited the first discussion of myopia and presbyopia to Aristotle. The discussions were entitled in Aristotle's book 'Problemata'. Similarly, Plato's book constituted the oldest citations explaining the concept of myopia. [31]

Johannes Kepler (1571-1630) a physicist and astronomer explained that that convergence of light in front of the retina is cause of myopia. [10]

Plempius (1632) proved that increase in axial length of eye was a cause for myopia. Other causes that had been postulated were increased thickness of lens, an increase in its refractive index and change in its position.

Tron (1934-1935) in a study of 275 eyes confirmed the wide range of axial lengths in emmetropia (22.4mm-27.3mm) and concluded that the axial length was the determinant factor for cause of myopia. [31]

Epidemiology

Myopic progression in children is a cause of concern in Asian countries because theprevalence of myopia in Asia is high and may be increasing.[32,33] The prevalence of myopia for Singapore children aged 3 to 7 was 8.6%.[34]

It was 30% in Hong Kong children aged 6 to 7.[35] The annual myopic shift was reported to be 0.63D (age 5 to 16years) in 2004 in Hong Kong.[32]

High myopes have also been reported to show a faster myopic progression.[32,34]

Degenerative myopia has an incidence of 2% in the United States, and is the seventh leading cause of blindness. It seems to affect the Chinese, Jewish, Japanese and Arab populations more and affects women twice as often as men. Blacks are virtually free of pathological myopia.

The prevalence of myopia has been reported in several Caucasian populations: 28.1% in the Baltimore Eye Survey (aged 40–80+ years), 26.2% in the Beaver Dam Eye Study (aged 43–84 years), 15% in the Blue Mountains Eye Study (aged 49–97 years) and 17% in the Visual Impairment Project (aged 40–98 years).[36-39]

In East Asia, the prevalence of myopia is higher. It is 38.7% in Singapore Chinese, aged 40–79 years and is 41.5% in Japanese aged over 40 years.[40,41]

In Indian population-based studies, the Andhra Pradesh Eye Disease Studyand the Chennai Glaucoma Studyreported age-adjusted prevalence rates for myopia of 34.6% and 31.0%, among adults aged 40 years or older, respectively.[42,43]

The prevalence of myopia of adults over 30 years of age was 17% in Central India.[44]

It was also noted that the prevalence of myopia was significantly higher among rural than urban residents (38.0% vs. 31.9%)[42] contrary to the perception that myopia is less common in rural communities.[45,46]

However, the higher prevalence of myopia in rural India could be explained by the more severe nuclear cataract.[42] Nuclear cataract has been found to cause myopic shift in many studies, which reflects the increased power of the lens rather than increased axial length.[39,47-49]

Definition

Helmholtz (1871) described the myopic eye as "one for which the far point is a short distance away, sometimes only a few inches from the eye". Donders (1864) defined it as "one in which the focus of the dioptric system lies in front of the retina".[50]

More recently, Curtin (1985) defined myopia as "the state of refraction in which parallel rays of light entering the eye at rest are brought to a focus in front of the retina".[7]

Myopia occurs as a result of a mismatch between the power and axial length of the eye, due to which, the images of distant objects are brought to focus in front of the retina, leading to blurred vision. In many cases, the structural cause of myopia is excessive axial length of the eye or the vitreous chamber depth.

In 2% of the general population, the myopia degree is over 6 dioptres (D) and is termed high myopia. The chances of sight threatening ocular pathology significantly increases in eyes with high myopia. The excessive elongation of the eye involves the outer coat (sclera).[51]

Also, high myopia is reported as a leading cause of registered blindness and partial sight. The present theories of refractive development accept that there is a pivotal role of the sclera in the control of eye size and the development of myopia.[51]

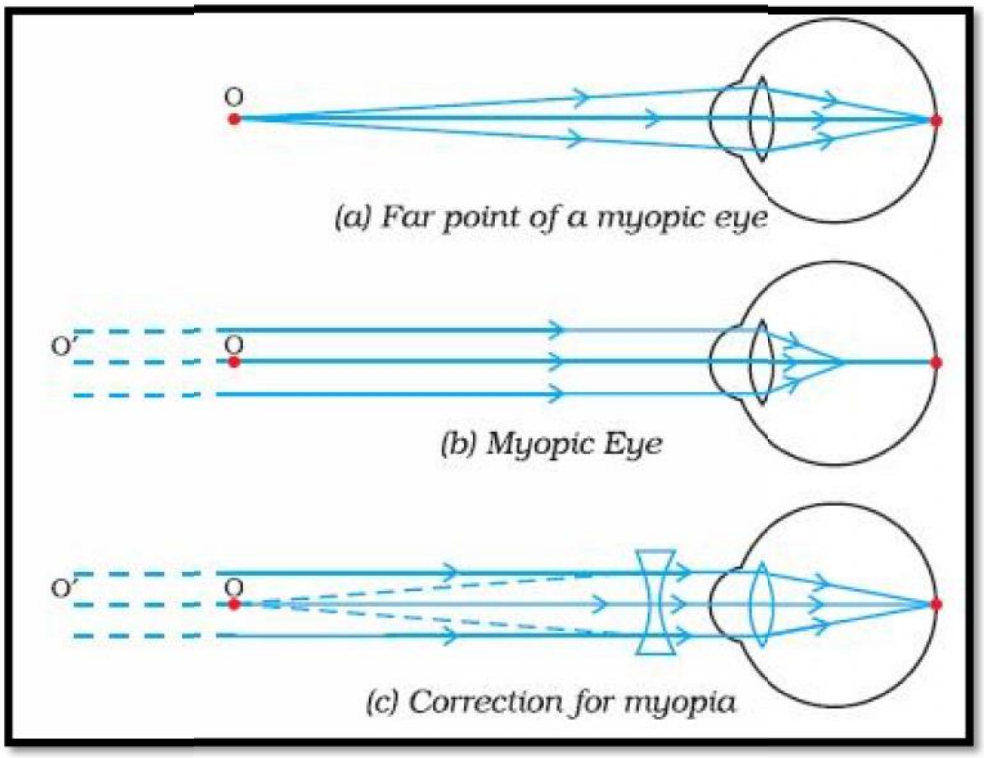


FIGURE 1: MYOPIA

Anatomical Correlates of Myopia

There are four main components of the eye, which together produce the overall refractive power. These are:

1. Corneal Power
2. Anterior Chamber Depth
3. Lens Power
4. Axial Length.

The increase in axial length (due to elongation of the posterior vitreous chamber) is thought to be the most common refractive element to cause myopia.[7]

It is also observed that Vitreous chamber depth and axial length are greater for myopes.

Theories about Myopia:

I. Mechanical Theory:

This theory affirms that mechanical tension caused by the crystalline lens or ciliary body limits equatorial ocular expansion and causes accelerated axial elongation. It suggests that there are factors that produce a large eye size in children, making them susceptible for myopia. Ciliary-choroidal tension, anterior to the globe, reaches a precarious point where proportional expansion of the globe during eye growth is no longer possible. After the equatorial growth is restricted, there is accelerated axial growth.

II. Environmental Theory:

This theory is also known asthe “use abuse” theory. It is stated that the incidence of myopia is high during school age when the eyesight is used mainly for near work.[52]

It states that near work causes myopia. This is proved by higher prevalence of myopia among highly educated people and people in white-collar occupations.

Age-related Axial length difference was also discovered in some investigations.[53]

The pathophysiological basis for this could be that accommodation is strained in prolonged use of eyes for near vision. The oblique muscles contract, producing a constant traction on the sclera. This can lead to lengthening of the anteroposterior axis of eyes, hence causing myopia.[50]

III. Biological Theory:

It states that all the coats of the eye have their own growth potential. Hence,there is enough coordination among the different tissues to achieve emmetropia. While enlarging, the retina pushes towards the posterior pole. The sclera tries to adapt itself to this growth and becomes thinned out in the process and characteristics of myopia are produced. After this, the choroid stretches and gets atrophied and the retina degenerates.

IV. Endocrine Theory:

The Endocrine Theory of Aging claims that aging is due to declining levels of hormones, especially HGH (Human Growth Hormone) and Sex Hormones. However,

some say that light through the eyes controls hormonal production in the brain via the Retinohypothalamic Tract (RHT). So, by controlling ocular light, it is possible to control hormonal production in the biological organism. With age, the lens shifts away from the ability to focus light on the fovea, causing development of nearsightedness.

Pathophysiology

The pathogenesis of pathological myopia is still unclear. Prior reports have identified a locus for autosomal dominant pathologic myopia to gene 18p11.31. Recent findings support the genetic heterogeneity of myopia by establishing linkage to a second locus at the 12q21- 23 regions.[54]

Pathological myopia has two stages, developmental and degenerative.

Damage in the developmental stage is due to axial lengthening, accompanied by damage due to vascular changes. Elongation of the globe, known as posterior staphyloma, occurs as a result of scleral thinning. This progressive scleral ectasia can form in the posterior pole (disc and macula), inferiorly, nasally or in multiple, complex patterns. Breaks in Bruch's membrane with accompanying choroidal atrophy create lesions known as lacquer cracks. These dehiscences are associated with increased risk for choroidal neovascularization.[54]

The term degenerative myopia is used mainly due to the posterior staphyloma (ectasia). The progressively myopic eye expands in all its posterior dimensionsand the formation of an equatorial staphyloma with scleral dehiscence is quite common, especially in the superotemporal quadrant. As the scleral shell expands, the pigment

epithelium, the neural retina and the choroid stretch and thin out to contain the area they cover.

Due to attenuation of the tissues, the fundus gets a pale, tessellated appearance. The pigment epithelial cells flatten and a reduction occurs in the thickness of the choriocapillaries and in the larger vessel layers and choroid pigment.

As time progresses, the phenomenon of traction and tension come to act. The first is a pale, temporal crescent at the disc as the pigment epithelium and choriocapillaries are retracted from the disc margin toward the deepest area of the staphyloma.

Bruch's membrane is non-cellular and elastic but can only stretch upto a certain limit. If this elastic limit is exceeded, the internal tension is relieved by formation of microdehiscences or focal, linear breaks called lacquer cracks.

If a choroidal neovascular membrane invades a crack, an abrupt macular haemorrhage could be produced. The hyperpigmented fibrovascular scar which evolves, can cause a central or paracentral scotoma or Foster Fuch's spot, although, it is generally self-limited.

Around this scar, an area of choroidal and pigment epithelial atrophy develops. This extends and coalesces with areas of atrophy which advance from other lacquer cracks,to eventually produce large geographical areas in which sclera can be seen through the transparent neural retina. The process is usually bilateral and insidious.

Progressive myopia is associated with systemic diseases such as Marfan's syndrome, retinopathy of prematurity, Ehler's-Danlos syndrome, Stickler's syndrome and albinism.[25]

Myopia and Race

High degrees of myopia with degenerative changes are common amongst Chinese, Japanese, Arabs and Jews and rare among Negroes and Sudanese. The variation is probably due to heredity rather than habit or culture.[7]

Myopia and Inheritance

Inheritance has a big influence on myopia. The lower degrees of myopia are transmitted by a dominant trait, whereas in higher degrees, the transmission is by recessive trait. Thus, consanguinity increases the incidence.

Refractive properties of the eye are determined more often by genetic factors, rather than environmental ones. The inheritance of refraction and its components in a general population follows a pattern set by a number of genes with additive effects.[7]

There is a higher prevalence of myopia in children of myopic parents than in children of non-myopic parents.[55]

Genetic studies of myopia are mainly twin studies, pedigree studies and studies of familial correlation.

It is difficult to separate hereditary factors from environmental factors such as similar work patterns in parents and their children.[56]

However, early environmental factors may also have led to longer eyes.[57]

The role of heredity is postulated to be more significant in persons with higher degrees of myopia.

TYPES OF MYOPIA

1. ***Axial myopia***

   It is seen in majority of the cases and is due to an increase in the antero-posterior diameter of the eye. The etiology of axial myopia is possibly due to physiological variation from normal, which is stationary and hereditary or postnatal factors concerned with higher degrees tend to be progressive.

2. ***Curvature myopia***

   It is either due to increase in the curvature of the cornea or one or both the surfaces of the lens. A 1mm change in the curvature of the cornea leads to a 6D refractive change. Increase in lenticular curvature as a whole or of its axial portion causes myopia, in anterior lenticonus and lentiglobus. The anterior surface of the lens assumes a conical or spherical form. As a result, the central area is highly myopic. A decrease in lens power is correlated with theelongation of axial length.[58]

   Axial length was also reported to have a negative correlation with corneal power, a positive correlation with anterior chamber depth and a negative correlation with lens thickness.[59]

3. ***Index myopia***

   It occurs due to a change of refractive index of lens because of senile sclerosis. This is a result of an increase in the optical density of lens. But a change of refractive index of aqueous or vitreous is negligible.

4. ***Positional myopia***

   This is due to forward displacement of lens from its normal anatomical position.

Myopia is the refractive anomaly of the eye in which the conjugate focus of the retina is at some finite point in front of the eye, when the eye is not accommodating.

It is the refractive condition in which parallel light rays from an object at optical infinity are focused by the eye in front of the retina, with accommodation at rest.

The neonatal eye is about 18mm in length as investigated earlier. By the age of 3 years the antero-posterior diameter is about 25 mm. From the ages of 3 and 14 years, elongation is only about 0.5 mm per year. This represents a decrease in ocular refraction of about 3D for the entire period between 3 and 14 years when the growth appears to be complete. During growth, as the axial length increases, the cornea and lens become flatter.[60]

CLASSSIFICATION

Myopia can be classified using different criteria.[61]

Myopia has been classified aseither:

1. Physiological

2. Pathological

Physiological myopia is due to an increase in the axial diameter of the eye, over which it is attained by normal growth.

Physiological myopia and pathological myopia are differentiated by the presence of degenerative changes and the degree of refractive error.

Intermediate myopia includes all types of myopia that present with an abnormal axial length.

The development of early signs of globe enlargement such as temporal crescent and supertraction of disc aretowards a more severe category of intermediate myopia.

Pathological myopia is caused by an abnormal lengthening of the eyeball and is often associated with thinning of the scleral wall. [7]

Based on the age of onset:

1. Youth-onset myopia (less than 20 years old)

2. Early adult-onset myopia (aged between 20 and 40)

3. Late adult-onset myopia (over 40 years old)

Other types of myopia:

1. **Congenital or Infantile Myopia**:

   It occurs at birth, with a reported prevalence in the full-term newborn varying from 0.0 to 24.2 percent. This variability is because of technical difficulties in measuring refraction in newborns.[62]

   Childhood myopia (school myopia) occurs at approximately 7-17 years of age and stabilizes by the late teens or early twenties. Both school and adult-onset myopia are mainly the result of idiopathic causes, whereas congenital myopia is generally associated with other abnormalities.

2. **Myopia of prematurity:**

   Premature and low birth-weight infants have a higher risk of developing myopia later in life.[63]The degree of myopia decreases as they reach maturity. It is due to the change in the corneal curvature, refractive index of media and axial length of eye.

3. **Pseudomyopia:**

   It occurs due to spasm of the ciliary muscles which cause parallel rays of light to converge to a point focus in front of the retina. This may occur with excessive accommodative effort in uncorrected hypermetropia. The diagnosis is confirmed by relaxation of the ciliary muscle by using a cycloplegic and correcting the myopia.[64]

4. **Night myopia**:

   The patients cannot see well in the distance at night or dusk and have good vision in the day time. This is also called twilight myopia.[65]

Myopia can also be classified as:

- Axial myopia and non-axial myopia
- Syndromic myopia and non-syndromic myopia.

Based on degree:

1. Low myopia (0 to -3 D)
2. Moderate myopia (-3 to -6 D)
3. High myopia (≤ -6 D)

SYMPTOMS AND SIGNS

The most common symptom related to uncorrected myopia is blurred distance vision. In simple myopia and degenerative myopia, the distance blur is constant. In nocturnal myopia, distance vision is blurred only in dim illumination. In pseudomyopia, the blurred distance vision canbe constant or intermittent, but greater distance blur occurs after near work.

Blurred distance vision in induced myopia can vary from transient (lasting a few hours) to constant and depends on the particular agent or condition causing it.

With the exception of pseudomyopia and some forms of induced myopia, asthenopic symptoms are not characteristic of myopia. If asthenopia is present in a patient with myopia, it is usually due to some other cause, such as astigmatism, anisometropia, an accommodative dysfunction or due to a vergence disorder.

Children with simple myopia often do not know that they have reduced distance vision until they discover that other children can see better than they can.

The definitive sign of pseudomyopia is significantly more minus power on the manifest refraction than on the cycloplegic refraction. This additional minus power cannot be eliminated with the standard refraction procedures used to relax accommodation at distance.

In myopes, the eyes are quite often prominent. They appear elongated and might even appear like exophthalmos, especially in unilateral cases. This elongation of the eyeball mainly affects the posterior pole and surrounding area. The part of the eye anterior to the equator may be normal.

In myopia, the anterior chamber is deeper than the normal.

Pupils are generally large and a may react sluggishly

Degenerative or pathological myopia is generally congenital or of early onset. Corrected visual acuity may be reduced as a result of pathological changes in the posterior segment. Abnormal or adverse ocular changes in degenerative myopia can include:

1. Peripapillary atrophy: appear as rings around the optic disc.
2. Tilting or malinsertion of the optic disc.
3. Vitreous liquefaction and posterior vitreous detachment.
4. Lattice degeneration in the peripheral retina.
5. Thinning of the retinal pigment epithelium with resulting atrophic appearance of the fundus.
6. Ectasia of the sclera posteriorly (posterior staphyloma).
7. Breaks in Bruch's membrane and choriocapillaris, resulting in lines across the fundus called "lacquer cracks".
8. Fuchs' spot in the macular area.[66]

The observation of some of these signs alone does not necessarily indicate pathological myopia. For instance, very often, small choroidal crescents are often noticed on one side of the optic disc.

These crescents are commonly seen on the temporal side. This is because greater postnatal expansion of the sclera results in a shearing type of displacement on the lamina vitrea temporally.

As a counter part of the temporal crescent, choroidal and retinal tissues pile up on the nasal side resulting in asupertraction crescent.

Patients with degenerative myopia may complain of floaters or flashes of light associated with retinal changes.

Media and fundus findings:

1. **Vitreous:**

The vitreous liquefies due to degenerative changes. Muscae volitantes are almost invariable with large floaters. Detachment of the vitreous can occur at the posterior pole presenting as a lacy opacity with a circular opening about the size of the disc. The lacy film represents the posterior condensation layer of the vitreous that has become detached. The hole in the membrane represents the line, where under normal conditions the vitreous is attached to the margins of the disc.[64]

2. **Fundus:**

Both the central and peripheral fundus should be evaluated by binocular indirect ophthalmoscopy.

Central posterior fundus:

The pigmented layer of retina loses a lot of its pigment so that the fundus is tigroid and the choroidal vessels are well seen. This thinning is often inferior to disc and localized in the lower fundus. Ectasia, called staphylomas occurs eventually and becomes increasingly noticeable towards the end of the first decade and thereafter.[67]

Posterior staphyloma:

Five types are known:

1. Type-I: It affects the posterior pole and is the most frequent posterior staphyloma.
2. Type-II: Macular Staphyloma. It involves a smaller area, temporal to the macula and is the second most common.
3. Type-III: The Peripapillary staphyloma. It is the least common and involves a sharply circumscribed area about the optic nerve.
4. Type-IV: Staphylomas of the nasal and the inferonasal aspect originate at the optic nerve.
5. Type-V: It involves an elliptical area below the optic nerve.

Macular changes

The irregularity of the pigmentation is represented histologically by proliferation and depigmentation of the hexagonal cells. In highly myopic cases, the atrophic changes are limited to the macular area and are liable to be accompanied by subretinal or deep retinal haemorrhages. The blood absorbs slowly and may ultimately be replaced by a patch of organized exudates or a pigment. The latter is a rational explanation of the black spot described by Fuchs.[64]

The peripheral fundus

The retinal changes in the periphery are produced by excessive ora-equatorial elongation.[68]

It is initially marked by the presence of extensive areas of white without pressure that affect the temporal quadrant of fundus. Within the second decade, an increase in incidence of lattice degeneration is found. Later, during the fifth and sixth decade, pigmentary and paving stone degenerative changes occur with former lesions predominantly affecting the temporal quadrant and the latter affecting the inferior quadrant.[67]

Lattice degeneration:

Lattice degeneration is present in about 8% of the population. It probably develops early in life, with a peak incidence during the second and third decades. It is found more commonly in moderate myopes and is the most important degeneration directly related to retinal detachment. It is usually bilateral and most frequently located in the temporal rather than the nasal fundus. It is an important cause of retinal detachment in young myopes.

A characteristic feature is an arborizing network of white lines within the islands.

Some lattice may be associated with snowflakes. Small holes within the lattice are common and are usually innocuous.

Lattice may be complicated by a tear developing along the posterior edge of an island of lattice.

Tears typically occur in myopes over the age of 50 years. Atrophic holes may rarely lead to retinal detachment in young myopes.[72]

Snailtrack degeneration:

It is characterized by sharply demarcated bands of tightly packed ‘snowflakes’ which give the peripheral retina a white frost-like appearance. They are usually longer than the islands of lattice and may be associated with overlying vitreous liquefaction.

White with pressure :

It is a translucent grey appearance of the retina, induced by indenting the sclera. Each area has a fixed configuration, which does not change when the scleral indenter is moved to an adjacent area. It is observed along the posterior border of islands of lattice degeneration and snailtrack degeneration.

White without pressure:

Pale, discrete areas of the retinal periphery without the application of any external pressure are thought to be the result of vitreous traction, which could result in the formation of retinal breaks.

COMPLICATIONS:

High myopes are more prone to having a retinal detachment than patients with hyperopia. The risk for retinal detachment increases as myopia increases.[69]

According to Sorsby's reports, the probability of retinal detachment is 4 times greater for the patients with refractive error more than -10 diopters, compared to those whose refractive error is of +5.00 diopters.[70]

Patients with myopia are also more likely to have most forms of glaucoma.

Loss of vision can occur at lower intraocular pressures when the patient is myopic.[69]

Glaucoma is significantly more prevalent in myopic eyes, particularly in pathological myopia and the incidence of cataract in myopia is twice of that in the normal population.[69]

Because of these associations with retinal detachment and glaucoma, degenerative myopia is one of the leading causes of blindness in the United States, United Kingdom and Canada.[71]

MANAGEMENT

Both spectacles and contact lenses can be effective in treating the refractive error. High-index glass, plastic and polycarbonate lenses are suitable for these prescriptions. Special edge polishing and buffing can also improve lens cosmetics.

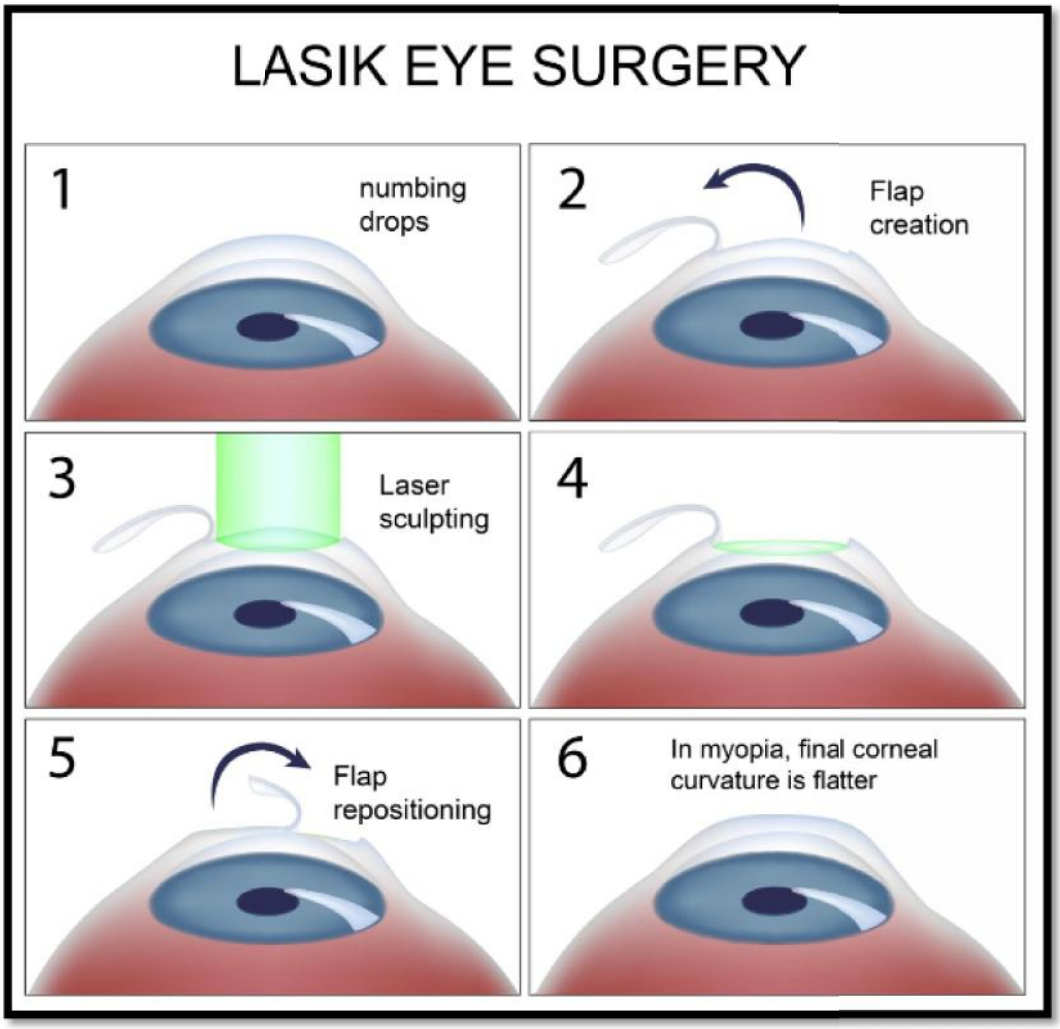


FIGURE 2: LASIK SURGERY

Both soft and gas-permeable contact lens designs are acceptable. Although, soft lenses provide increased comfort and convenience, patients need to be monitored closely for hypoxia. Gas-permeable lenses offer solid optics and excellent physiology. Though LASIK is used often for treatment of myopia, it is not a reasonable solution for patients with pathological myopia. Intraocular lens implantation may be a viable refractive surgery alternative.

No treatment can regress or arrest the progression of the staphyloma in pathological myopia. Experimentally, atropine has been used in children to ease the stress and strain from accommodation. Others have attempted to alleviate this mechanism with bifocals. Neither modality has been successful.

Anytime a posterior staphyloma, lacquer crack or Fuch's spot is seen on the fundus, fluorescein angiography is appropriate. A-scan and B-scan ultrasonography can confirm the presence of increased axial length and posterior staphyloma.

Patients with pathological myopia should be routinely monitored for choroidal neovascularization membrane (CNVM) formation. Angiographically definable extrafoveal or juxtafoveal subretinalneovascular membranes may demand treatment with argon laser photocoagulation. But due to the globe elongation and tissue stretching, the laser scars can stretch and enlarge and may adversely impact vision.

Some clinicians use photodynamic therapy (PDT) off-label in degenerative myopia for patients with juxtafoveal and extrafoveal membranes. PDT and macular translocation may be options for subfoveal CNVM in pathological myopia.[54]

Patients must also be monitored for retinal detachment and should receive at least annual dilated fundus examinations.

Because pathological myopia results from stretching of the globe, it compromises ocular stability and strength. Patients should be counseled to avoid dangerous circumstances and activities. Contact sports or activities that jolt the body increase the risk for retinal detachment.

Crosslinking of scleral components has the potential to halt progression of degenerative myopia since it tackles both of the underlying causes that are currently hypothesized. It increases tissue strength and hinders tissue remodeling.[72-74]

Interventions for myopia

The exact cause for the onset or progression of myopia has not yet been identified and the mechanism underlying myopic development is not well understood. Research is still ongoing with the aim of finding some effective methods to slow down or control the increasing trend of progression of myopia. There is no single treatment or management that can be of significant effectiveness. Many studies are being conducted using randomized trial method to explore the efficacy and side effects of interventions, but the results are inconsistent. Progressive addition lenses and rigid gas-permeable contact lenses, have yielded both negative and positive results.[75,76]

While refractive errors induced by progressive myopia are quickly corrected with spectacles, contact lenses, corneal refractive surgery or intraocular lenses, these modalities do not prevent visual loss induced by stretching of chorioretinal tissues. Current means to treat choroidal neovascularization in degenerative myopia, such as

photodynamic therapy, are minimally effective.[77]

Recent studies have begun to test injections of anti-angiogenic drugs like Bevacizumab (Avastin®) or Lucentis®.[78-82]

Attempts are being made to treat expansion of the eye due to myopia, including the use of scleroplasty, scleral reinforcement and even an attempt to polymerize foam around the eye. [82-90]

Since these modalities remain unproven in well-controlled clinical trials, none have been widely adopted to manage patients with degenerative myopia. Current therapies are essentially palliative, attempting to mitigate visual loss in this condition.

ASTIGMATISM

Astigmatism is defined as the cylinder power of more than 0.5DC. The astigmatism axis is classified as "with-the-rule" if the axis is between 75° and 105° and "against-the-rule" if the axis is between 0° and 15° or between 165° and 180° and oblique if itis at any other meridian.

Age-related evolution of ocular astigmatism in terms of power and axis has been observed in epidemiologic studies.[11,91-93]

The degree of astigmatism is higher in preterm newborns and has an inverse association with postconception age and birth weight.[94]

In near retinoscopy, without cycloplegia, astigmatism of at least 1D was found in about 55% of infants younger than 5 months, 10% of who displayed a cylinder power of 3D or more.[95]

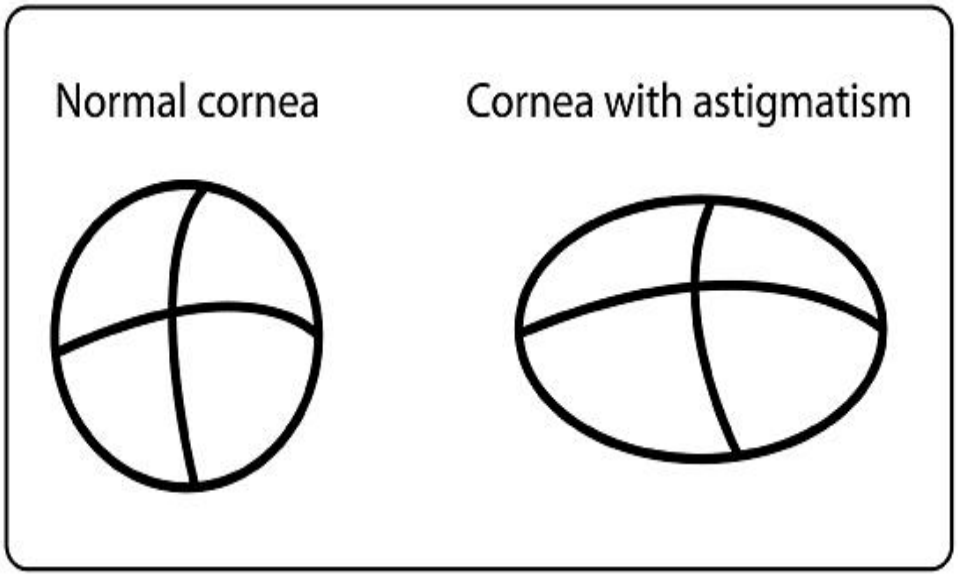


FIGURE 3: CORNEA IN ASTIGMATISM

In another study, photorefractive techniques showed that almost all infants at the age of 3 months had at least 1D of astigmatism, which had decreased to adult levels by the age of 18 months.[91] Likewise, a longitudinal study found astigmatism of at least 1D in about 40% of infants at 3 months of age, with a significant decrease to 4% by the age of 36 months.

This reduction seems to be due to the decrease in toricity of the cornea and the anterior lens.[96] The corneal shape changes throughout life. The linear reduction of the astigmatism to lower values with age is apparently a part of normal eye maturation and emmetropisation.[94] It has been suggested that the high astigmatism in early life induces and activates accommodation.[97]

Reports on the axis of astigmatism in infants are contradictory. According to Gullstrand, the natural form of the cornea is against the rule.[98]

Several studies have found a plus cylinder axis at 180±20 (i.e., against the rule) in the majority of infants.[92]

Some studies have shown that with the rule astigmatism is more frequent among infants.[93,96]

As the child grows older, a lot of the early astigmatism gradually disappears and transforms into with the rule owing to eyelid pressure.[95]

Most changes occur at ages 1-3 years. This is when the vertical and horizontal diameters of the cornea and its elasticity attain adult size and amount.[99]

The with the rule astigmatism in preschool children gets stabilized while nearing adolescence.[100]

In early adulthood, astigmatism of more than one diopter is infrequent and is still with the rule.

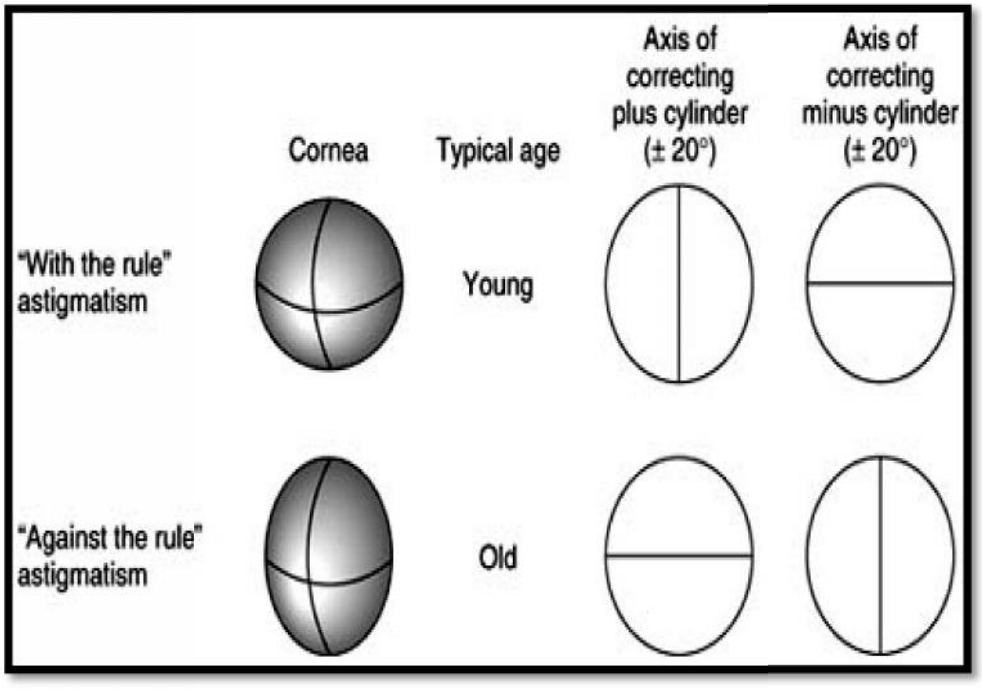


FIGURE 4: ASTIGMATISM: WTR, ATR

Nuclear sclerosis cataract and the change in refractive index of the crystalline lens at older ages may contribute to myopic astigmatism.[101]

Anterior corneal (and total) astigmatism shows flattening in the vertical meridian with age, in contrast to a trend towards with-the-rule astigmatism on the posterior corneal surface.[102]Against-the-rule astigmatism is the most common type of astigmatism in adults over 40 years of age. Interestingly, men are significantly more likely to develop against-the-rule astigmatism.[103]

Corneal toricity accounts for the major component of total astigmatism.[102]

With age, the upper eyelid pressure on the cornea and the tone of orbicularis muscle decreases.

Also, with-the-rule astigmatism decreases when eyelids are retracted from the cornea. When relative steepening in the vertical meridian subsides, the intrinsic lenticular, against the rule astigmatism manifests. There is a decrease in the action of extraocular muscles, especially the medial rectus. Vitreous syneresis and liquefaction may also contribute.[104]

The contribution of the lens to the ocular astigmatism is relatively constant throughout life.[92] Lenticular astigmatism develops due to emmetropisation phenomenon, i.e., it effectively decreases manifest astigmatism in the early decades of life. But in older ages, lenticular astigmatism is manifested as an against-the-rule astigmatism when the corneal astigmatism is decreased.[93]

Factors affecting Astigmatism:

Diurnal changes of astigmatism in the normal eye:

The magnitude and axis of astigmatism vary during the day, due to multiple factors, such as:

- Changes in eyelid pressure
- Extraocular muscle tension
- Pupil size
- Accommodation

It is postulated that generally the cornea has its flattest shape on waking up and steepens slightly until the evening. Recently, diurnal variations in corneal topography have been studied.[105]

Corneal wavefront error analysis revealed significant changes in astigmatism during the day.[105]

Effects of lid pressure and near work:

Changes in corneal contour exerted through eyelid pressure have been widely discussed since the mid-1960s.

There occurs a transient bilateral monocular diplopia after near work due to temporarily induced toricity in the cornea has been reported by a number of investigators

It is known that visual tasks that require a significant downward gaze, such as reading, can alter corneal curvature owing to eyelid pressure.

This leads to horizontal bands on red reflex during retinoscopy with concomitant topographic changes and corresponding distortions in Zernike wavefront analysis.

Effects of eyelid slant and tension

The overalleffect of the eyelids contribute to naturally occurring astigmatism in healthy adults.

Palpebral fissure slanting is an important factor that can affect thetoricity of the cornea. The magnitude of astigmatism increases as the palpebral fissure diverges from the horizontal plane.[105]

Thicker or tighter eyelids can be correlated to higher degrees of astigmatism as well, which is probably why Asians and Native Americans show higher degrees of corneal astigmatism than other races.[106]

Corneal rigidity can also add to the amount of astigmatism caused by eyelid pressure.

Nutritional deficiencies may also decrease corneal rigidity and result in flattening of the horizontal meridian and steepening of the vertical meridian.[107]

Pupil dynamics

Three important axes have been described as part of the optical system of the eye:

1. Optical Axis (corneal optical center of cornea to optical center of lens)
2. Visual Axis (object of regard to fovea, i.e., line of sight)
3. Pupillary Axis

There can be a mild physiological pupil decentration in the nasal direction.

The pupil is the aperture from which light enters the eye.

The size of the pupil and its (centroid) lateral position around the optical axis of the eye changes according to ambient light, accommodative effort and emotional status.[108]

The pupil size correlates with both the magnitude as well as orientation of astigmatism.

Larger mesopic pupil sizes are generally associated with with-the-rule astigmatism.

Accommodation and convergence

The term 'accommodative astigmatism' was introduced years ago. Lenticular astigmatism changes can neutralize corneal astigmatism and reduce the eye's overall toricity. Accommodative astigmatism is related to lens distortion.

There is homogeneous lens elasticity, variable constriction in ciliary muscles and nonhomogeneous tension of the extraocular muscles during convergence (leading to corneal distortion).

These can explain 'lag of accommodation', the phenomenon of less accommodative response than the accommodative stimulus in the horizontal meridian and the resultant with-the-rule astigmatism.

In a more recent study, it was found that all emmetropic subjects became astigmatic during accommodation, 93% with the rule (mean -1.96 D). The eyes became emmetropic, just after relaxation.[109]

Wavefront aberrations in a large adult population have been studied and changes in astigmatism have been found towards with-the-rule with an average of -0.1

D during maximum accommodation.[15]

The mentioned pupillary and accommodative effects interact with the factors considered above during near tasks

Accommodation and convergence always go hand in hand during near-vision tasks. Slight changes in cylinder power and axis (towards with-the-rule) occur during convergence alone.[109]

Cyclotorsion and binocularity

Rotation of the eye around the Z-axis (rolling or cyclotorsion) modifies the axis of ocular astigmatism. The features of accommodation, baseline astigmatism and torsional alignment contribute to binocular fusional potential, stereopsis and field of vision.

Although several studies have shown significant in-cyclotorsion or ex-cyclotorsion of about 2-4 degrees (maximum 9 to 14 degrees) as a result of changing the body position from seated to supine.[110]

It has been suggested that axis misalignment of about 4 degrees will lead to 14% cylinder undercorrection during laser ablation.[110]

Aside from body position and monocularity, which account for static eye rotational alignment, dynamic cyclotorsion also occurs during laser ablation and may result in astigmatic undercorrection and/or induced astigmatism. Blurring of the fixation target happens during ablation (after removal of epithelium in surface ablation and following flap lifting in LASIK).[110]

It is an important factor for dynamic cyclotorsion. The magnitude seems to be significantly higher in supine position. Modern eye trackers now are designed to dynamically follow the eye during laser ablation.

Retinal astigmatism

From a historical point of view, directional variability in photoreceptor arrangement was considered a source of astigmatism. Functional retinal elements may be more abundant or thicker in one axis than the other.

Also a 'tilted' retina was simulated and it was observed to manifest as some degree of cylindrical error.[111]

This could be the result of unequal lengthening of the sclera in different meridians during axial growth.

Myopic Astigmatism is a common and significant cause of visual impairment. The myopic retinal changes have been studied in great details, but there is very little literature on the correlation between the myopic fundal changes and the axis of astigmatism of the eye.

METHODOLOGY

After signing the informed consent, data was collected in a prescribed data collection form. General data (age, gender) was obtained. History of using glasses, the duration of using them and any other significant history was noted down.

Visual acuity, both uncorrected and corrected were recorded on Snellen's chart. Near vision was recorded on standard Jagger's near type chart.

Anterior segment examination was carried out on each patient.

Refractive error was estimated by Priestly Smith retinoscope, either by dry refraction or under full mydriasis and cycloplegia with cyclopentolate hydrochloride 1%. Spherical equivalent and the cylindrical equivalent along with the axis of astigmatism were taken for analysis.

Fundoscopic examination was done on patients who had undergone cycloplegic refraction on the same visit.

Thorough indirect ophthalmoscopic examination was performed with a +20D lens and the disc changes were observed and noted.

Any peripheral degenerative changes in the retina were also noted down.

Corneal curvature was measured using a Bausch and Laumb Keratometer. Average keratometric reading was noted.

Axial length and central anterior chamber depth were measured using an A scan Biometer(Echorule2, Biomedix).

Fundus photograph was taken using a Canon CF-1 Digital Retinal Camera, during which the patient was asked to look straight ahead and avoid torsional movement of the globe.

The deep/longest axis of the crescent/peripapillary atrophy was determined. The centre of the disc was first determined as the point of intersection of two lines across the long and short axis in case of oval discs or vertical and horizontal axis in case of round disc.

A vertical and horizontal perpendicular was drawn across the point to give the 90° and 180° axis respectively. The deep/long axis of crescent was drawn to the axis of the disc.

The axis of myopic crescent or peripapillary atrophy was measured using the annotations incorporated within the software attached to the fundus camera and was ascribed a value in degrees, in a manner similar to the axis of astigmatism in refraction.

The long axis of peripapillary changes and of the disc crescent were correlated with the axis of astigmatism in patients with compound myopic astigmatism and the coefficient of correlation was determined using Spearman's rank correlation coefficient. P value < 0.001 was considered statistically significant.

Source of data:

Participants attending the Ophthalmology Out Patient Department in KLE's DR.PRABHAKAR KORE HOSPITAL AND MRC, BELGAUM

Study design:

A cross – sectional study

Sample size: 64

Since there are no studies done previously showing the prevalence of myopic fundal changes with astigmatism, we took into account 80 % of all patients coming to the OPD with myopic fundal changes and astigmatism, which going by thehospital refraction records of the previous three years, is ~80 patients.

Therefore, sample size = 80/100 * 80

= 64 patients

Inclusion criteria for analysis:

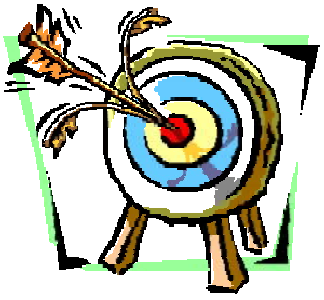
Participants who on initial examination in the Out Patient Department were found to be having myopia, along with one or more characteristic myopic fundal changes, i.e., myopic crescent or peripapillary atrophy, as well as myopic astigmatism.

Participants already wearing an optical correction for myopia and myopic astigmatism, as well as having one or more myopic fundal change and attending the Out Patient Department for change of glasses.

Exclusion criteria for analysis:

- Participants not willing to give consent.
- Participants who had undergone any intraocular surgeries
- Patients with congenital anomalies
- Amblyopic patients
- Patients with strabismus

Introduction



Objectives

Review of Literature

Methodology

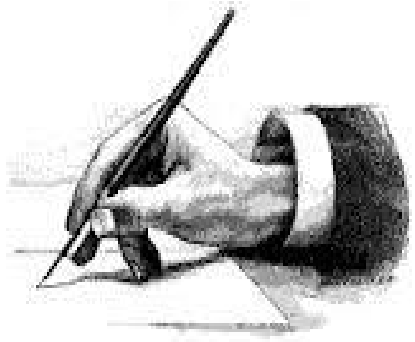
Results

Discussion

Conclusion

Summary

Bibliography



Annexure-I

Annexure-II

Annexure-III

Annexure-IV

Annexure-V

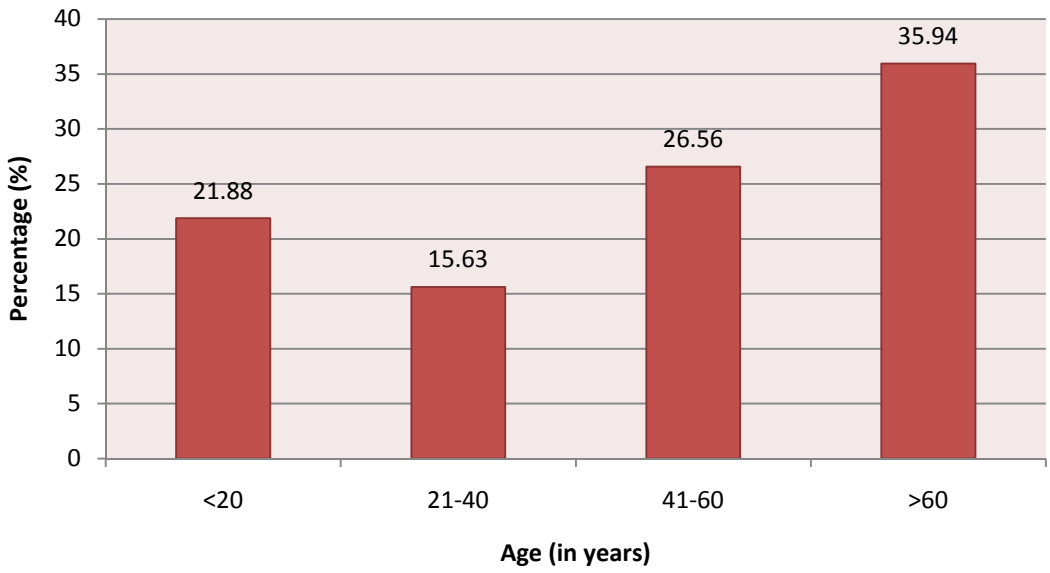
OBSERVATIONS AND RESULTS

The present study was conducted in the Department of Ophthalmology, KLE's Dr. Prabhakar Kore Hospital And Medical Research Centre, Belgaum, on patients with myopic fundal changes and astigmatism, during the period of 1st January 2013 to 31st December 2013. In this study, 97 eyes of 64 patients with myopic astigmatism and myopic fundal changes were studied and observed and the findings were recorded and tabulated as below.

TABLE 1: AGE DISTRIBUTION

AGE (YEARS)	NO. OF SUBJECTS	PERCENTAGE(%)
≤ 20	14	21.88
21 - 40	10	15.63
41 - 60	17	26.56
> 60	23	35.94
TOTAL	**64**	**100**

GRAPH 1: AGE DISTRIBUTION



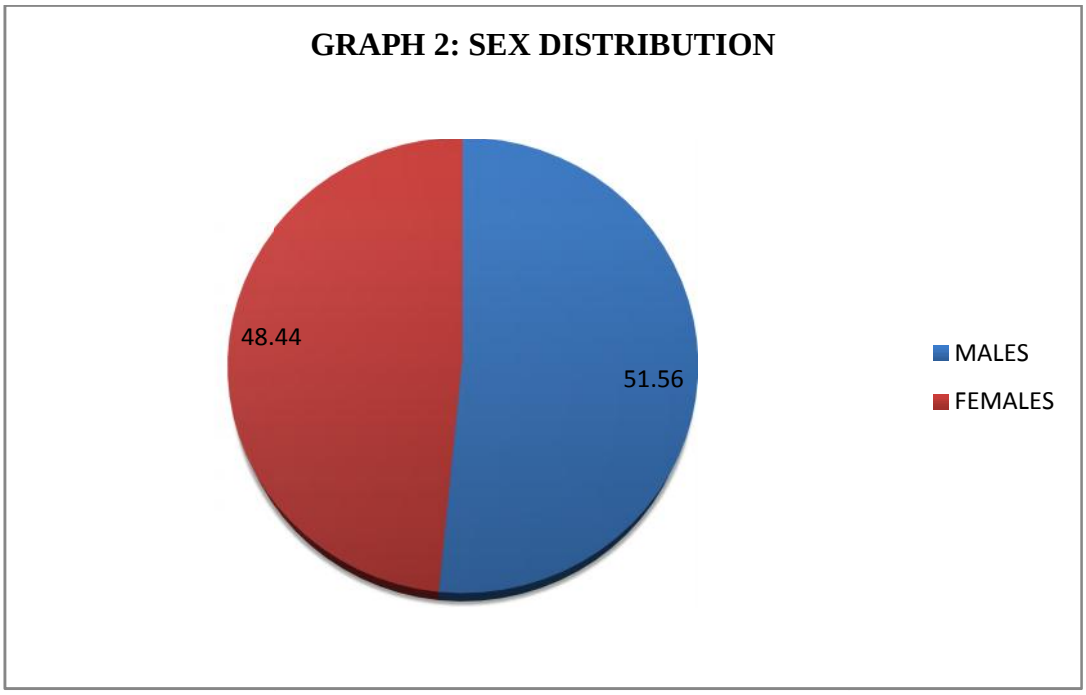
Out of the 97 patients, 14 patients belonged to the age group of less than 20 years (21.88%), 10 patients belonged to the age group between 21-40 years (15.63%), 17 patients belonged to the age group of 41-60 years (26.561%), 23 patients were above 60 years of age (35.94%).

Mean age was 46.91 years

TABLE 2: SEX DISTRIBUTION

GENDER	NO.OF SUBJECTS	PERCENTAGE (%)
MALES	33	51.56
FEMALES	31	48.44
TOTAL	64	100

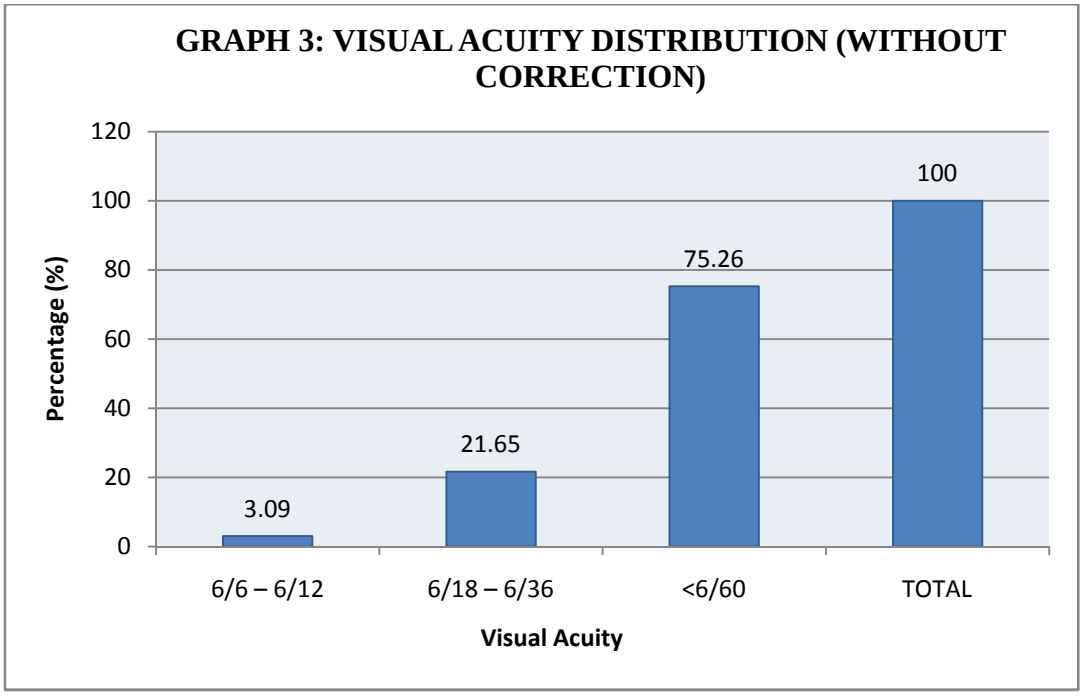
GRAPH 2: SEX DISTRIBUTION



Out of the 64 patients, 33 (51.56%) were males and 31 patients (48.44%) were females.

TABLE 3: VISUAL ACUITY DISTRIBUTION WITHOUT CORRECTION

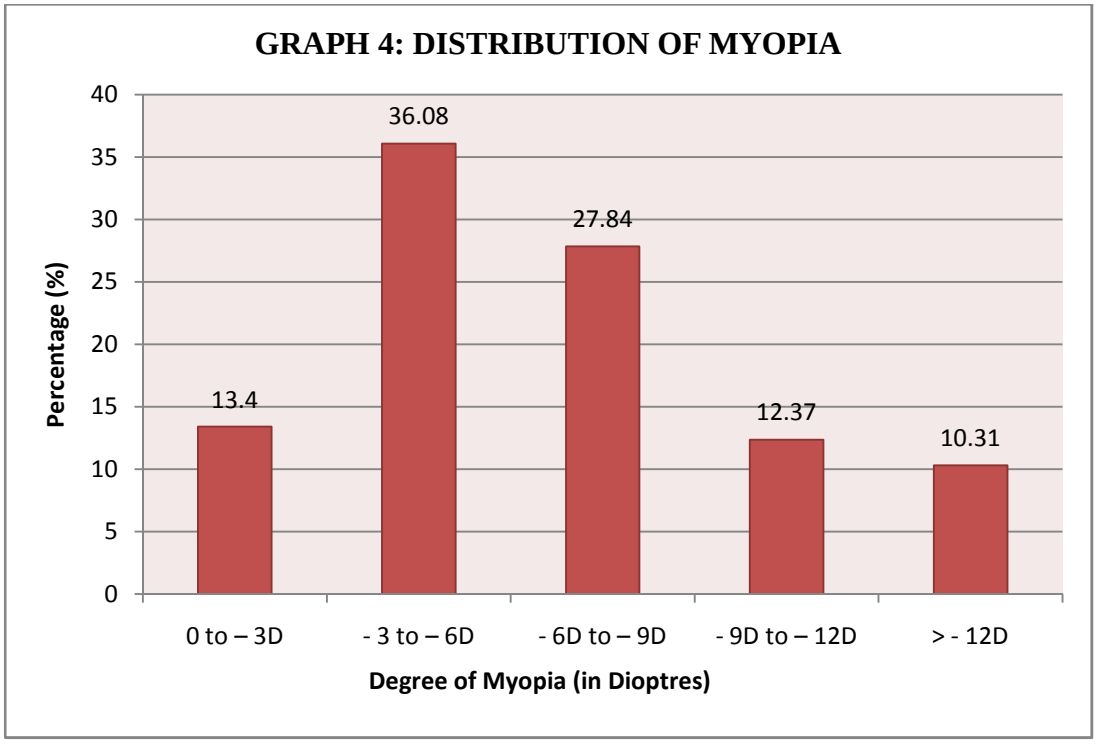
VISUAL ACUITY	NO. OF EYES	PERCENTAGE (%)
6/6 – 6/12	03	3.09
6/18 – 6/36	21	21.65
≤6/60	73	75.26
TOTAL	97	100



Out of the 97 eyes, majority, 73(75.26%) eyes had an unaided visual acuity of less than or equal to 6/60, 21 eyes (21.65%) had a visual acuity between 6/18-6/36 and only 3 eyes (3.09%) were having visual acuity between 6/6-6/12.

TABLE 4: MYOPIA DISTRIBUTION

DEGREE OF MYOPIA	NO. OF EYES	PERCENTAGE(%)
0 to -3D	13	13.40
-3D to -6D	35	36.08
-6D to -9D	27	27.84
-9D to -12D	12	12.37
> -12D	10	10.31
TOTAL	97	100



Out of the 97 eyes, 48 eyes (49.48%) belonged to the group of myopia of less than or equal to -6D and 49 eyes (50.52%) belonged to the group with myopia of more than -6D.

13 eyes (13.40%) were having myopia, less than or equal to -3D, 35 eyes (36.08 %) belonged to the group having myopia between -3D to -6D.

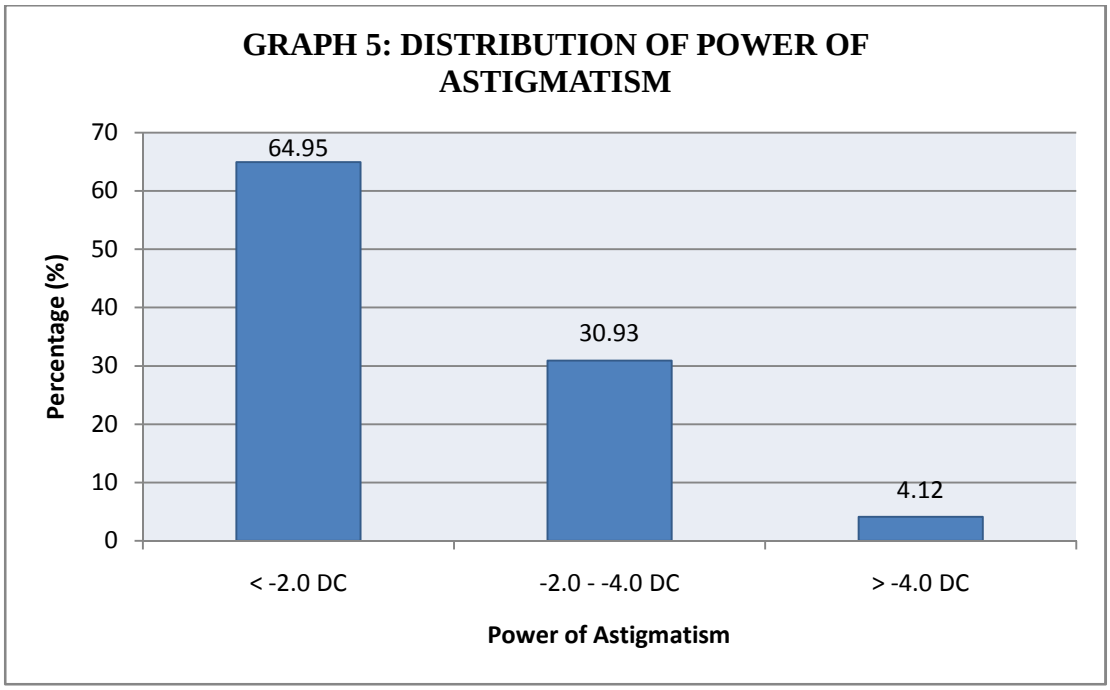
27 eyes (27.84%) were having myopia between -6D to -9D, 12 eyes belonged to the group having myopia between -9D to -12D and 10 eyes (10.31%) were having myopia of more than -12D.

The least myopic value was -0.25D and the greatest was -20D.

TABLE 5: DISTRIBUTION OF ASTIGMATISM

TABLE 5a: DISTRIBUTION OF POWER OF ASTIGMATISM

POWER OF ASTIGMATISM	NO. OF EYES	PERCENTAGE(%)
≤ -2.0DC	63	64.95
-2.0 to -4.0DC	30	30.93
> -4.0DC	04	4.12
TOTAL	97	100

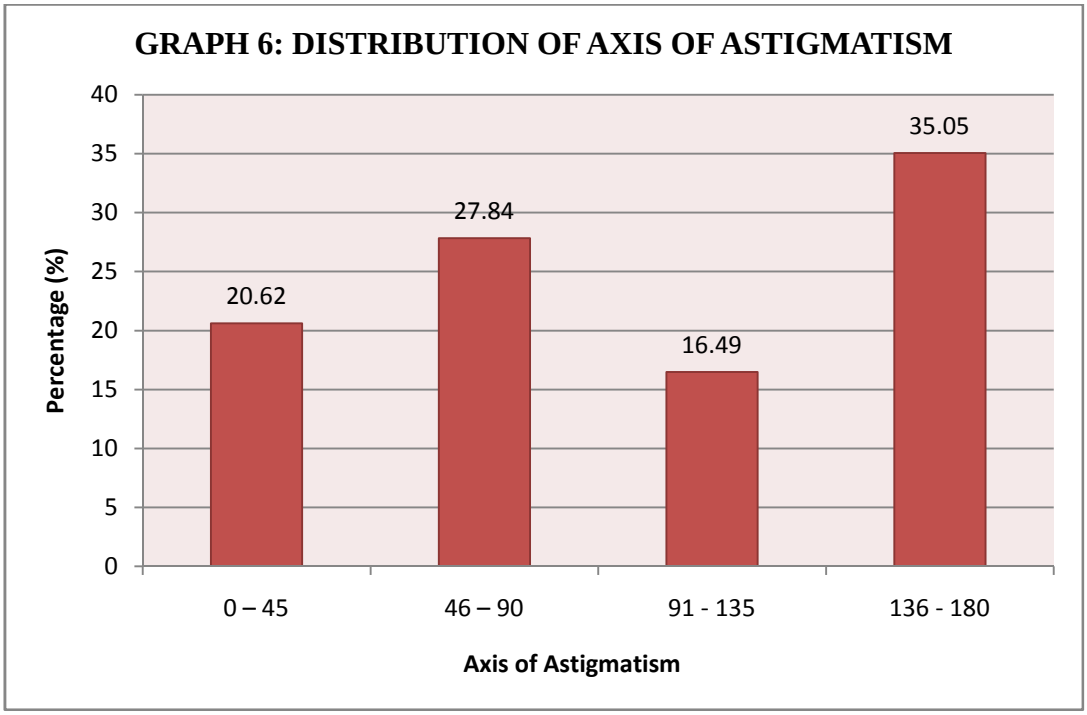


Out of the 97 eyes, 63 eyes (64.95%) belonged to the group having cylindrical power of less than or equal to -2DC, 30 eyes (30.93%) belonged to the group having cylindrical power between -2 to -4DC and only 4 patients (4.12%) had a cylindrical power of more than -4DC.

TABLE 5b: DISTRIBUTION OF AXIS OF ASTIGMATISM

AXIS OF ASTIGMATISM	NO. OF EYES	PERCENTAGE(%)
0° – 45°	20	20.62
46° – 90°	27	27.84
91° - 135°	16	16.49
136° - 180°	34	35.05
TOTAL	97	100

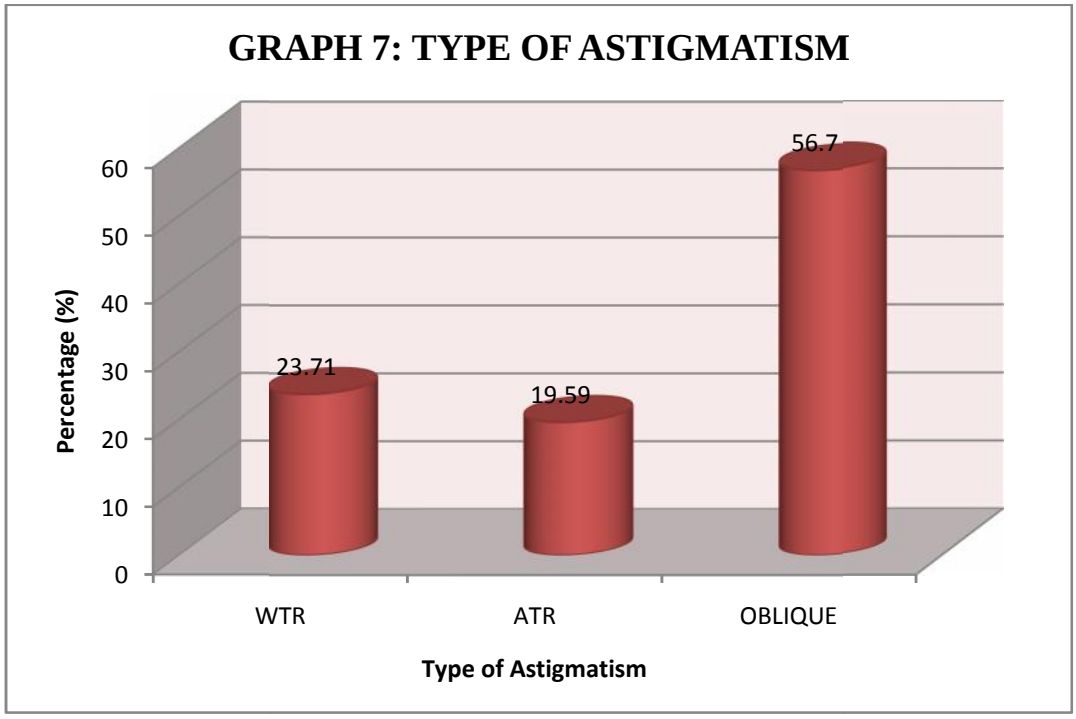
GRAPH 6: DISTRIBUTION OF AXIS OF ASTIGMATISM



Out of the 97 eyes , 20 eyes (20.62%) had an axis of astigmatism of less than 45°, 27 eyes (27.84%) belonged to the group with astigmatic axis between 46° – 90°. 16 eyes (16.49%) had an astigmatic axis between 91° – 135° and 34 eyes (35.05%) belonged to the group having an axis of astigmatism between 136° – 180°.

TABLE 5c: DISTRIBUTION OF TYPE OF ASTIGMATISM

AXIS OF ASTIGMATISM	NO. OF EYES	PERCENTAGE(%)
WTR	23	23.71
ATR	19	19.59
OBLIQUE	55	56.7
TOTAL	97	100

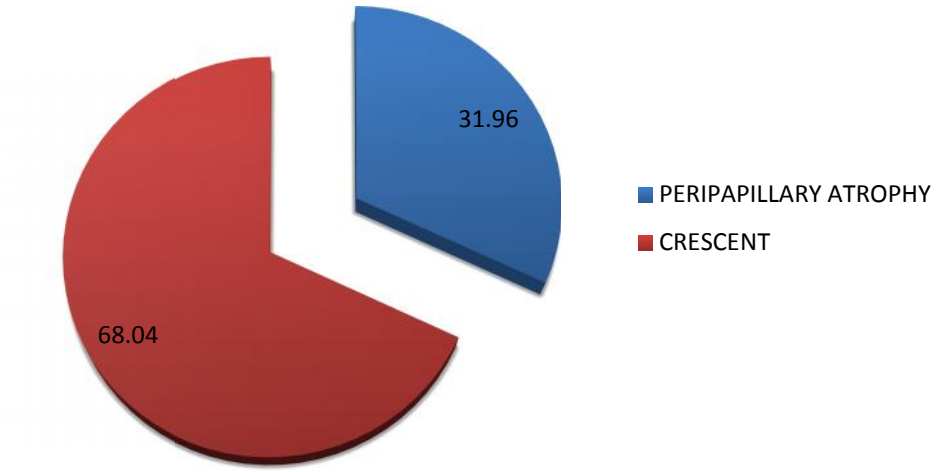


Out of 97 eyes, 23 (23.71%) had with-the-rule astigmatism. 19 eyes (19.59%) showed against the rule and most of the eyes, i.e., 55 (56.7%) had oblique astigmatism.

TABLE 6a: DISTRIBUTION OF DISC CHANGES

DISC CHANGES	NO. OF EYES	PERCENTAGE(%)
PERIPAPILLARY ATROPHY	31	31.96
CRESCENT	66	68.04
TOTAL	97	100

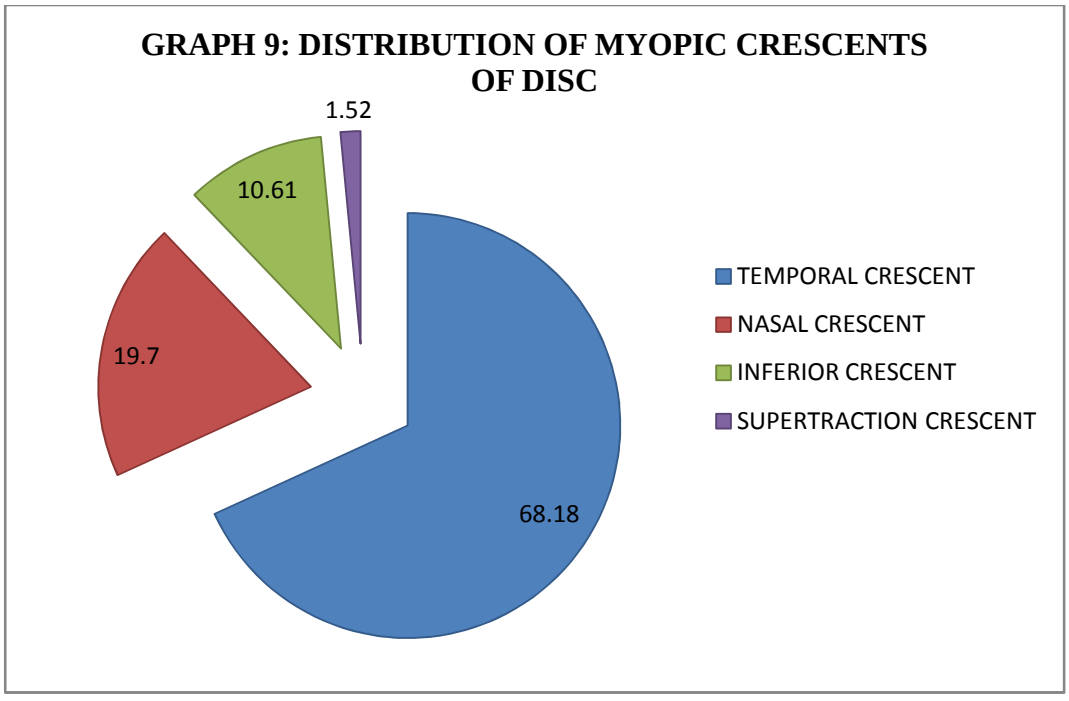
GRAPH 8: DISTRIBUTION OF DISC CHANGES



Out of the 97 eyes, 31 eyes (31.96%) had peripapillary atrophy around the disc, whereas 66 eyes (68.04%) had a myopic crescent around the disc.

TABLE 6b: DISTRIBUTION OF MYOPIC CRESCENTS OF DISC

DISC CHANGES	NO. OF EYES	PERCENTAGE(%)
TEMPORAL CRESCENT	45	68.18
NASAL CRESCENT	13	19.70
INFERIOR CRESCENT	07	10.61
SUPERTRACTION CRESCENT	01	1.52
TOTAL	66	100



Out of the 66 eyes having myopic crescents, majority of them had a temporal crescent 45 eyes (68.18%), 13 eyes (19.70%) had nasal crescents, 7 eyes (10.61%) had inferior crescents and only 1 eye had a supertraction crescent (1.52%).

TABLE 7: FOR 97 EYES CORRELATION BETWEEN AXIS OF ASTIGMATISM AND ANGLE OF CRESCENT/PERIPAPILLARY ATROPHY (Spearman's Rank Correlation Coefficient)

SPEARMAN'S RANK CORRELATION COEFFICIENT (δ)	p VALUE	n
0.495	<0.001 (S)	97

The above table shows a positive correlation between the axis of astigmatism of the eye and of the angle of the myopic fundal changes.

TABLE 8: CORRELATION BETWEEN AGE, DEGREE OF MYOPIA, AXIS OF ASTIGMATISM AND MYOPIC FUNDAL CHANGES

AGE (in years)	DEGREE OF MYOPIA	NO. OF EYES	NO. OF EYES WITH POSITIVE CORRELATION BETWEEN AXIS OF ASTIGMATISM AND MYOPIC FUNDAL CHANGES	PERCENTAGE (%)
≤ 20	≤ -6D	5	3	60
	> -6D	18	5	27.78
21 - 40	≤ -6D	8	5	62.50
	> -6D	6	3	50
41 - 60	≤ -6D	14	10	71.43
	> -6D	13	8	61.54
> 60	≤ -6D	21	10	47.62
	> -6D	12	9	75

In the age group of less than or equal to 20 years, 5 eyes had a spherical equivalent of less than -6D, of which 3 eyes (60%) had a direct correlation between axis of astigmatism and fundal changes, whereas 18 eyes were above -6D, out of which 5 eyes (27.78%) had a positive correlation.

Between the age group of 21-40 years, 8 eyes had a spherical equivalent of less than -6D, of which 5 eyes (62.50%) had a direct correlation between axis of astigmatism and fundal changes, whereas 6 eyes were above -6D, out of which 3 eyes (50%) had a positive correlation.

In the age group of 41-60 years, 14 eyes had a spherical equivalent of less than -6D, of which 10 eyes (71.43%) had a direct correlation between axis of astigmatism

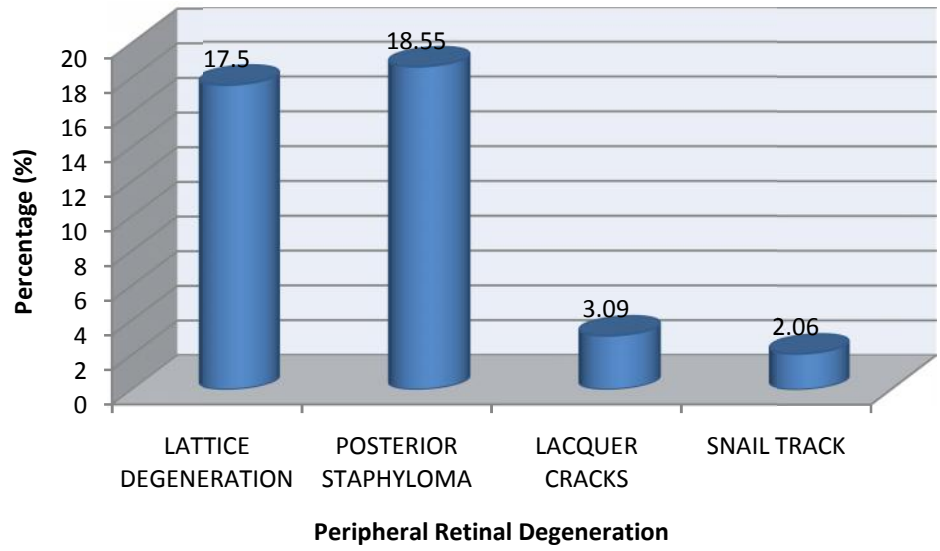
and fundal changes, whereas 13 eyes were above -6D, out of which 8 eyes (61.54%) had a positive correlation.

In age group above 60 years, 21 eyes had a spherical equivalent of less than -6D, of which 10 eyes (47.62%) had a direct correlation between axis of astigmatism and fundal changes, whereas 12 eyes were above -6D, out of which 9 eyes (75%) had a positive correlation.

TABLE 9: PERIPHERAL RETINAL DEGENERATION DISTRIBUTION

TYPE OF PERIPHERAL DEGENERATION	NO. OF EYES	PERCENTAGE(%)
LATTICE DEGENERATION	09	17.5
POSTERIOR STAPHYLOMA	12	18.55
LACQUER CRACKS	03	3.09
SNAIL TRACK	02	2.06

GRAPH 10: PERIPHERAL RETINAL DEGENERATION DISTRIBUTION

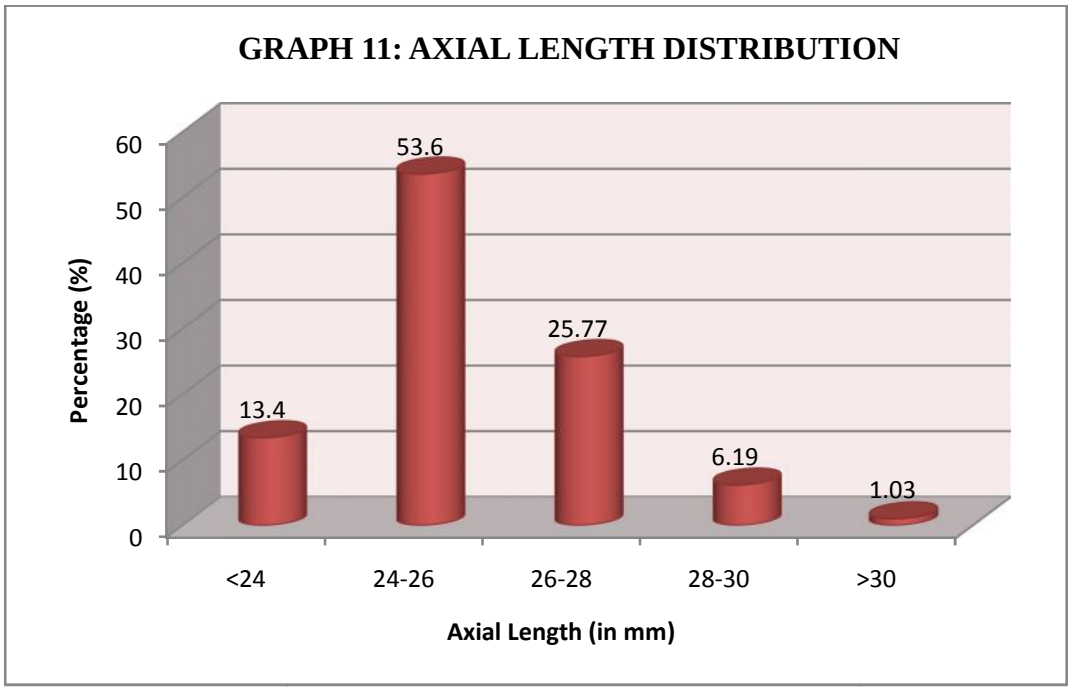


Out of the 97 eyes, all eyes had a tessellated background. 9 eyes (17.5%) out of the 97, had Lattice degeneration, 12 eyes (18.55%) had Posterior staphyloma, 3 eyes had Lacquer cracks (3.09%) and only 2 eyes (2.09%) had snail track degeneration changes present.

TABLE 10a: AXIAL LENGTH DISTRIBUTION

AXIAL LENGTH (mm)	NO. OF EYES	PERCENTAGE(%)
<24	13	13.4
24-26	52	53.6
26-28	25	25.77
28-30	06	6.19
>30	01	1.03
TOTAL	97	100

GRAPH 11: AXIAL LENGTH DISTRIBUTION

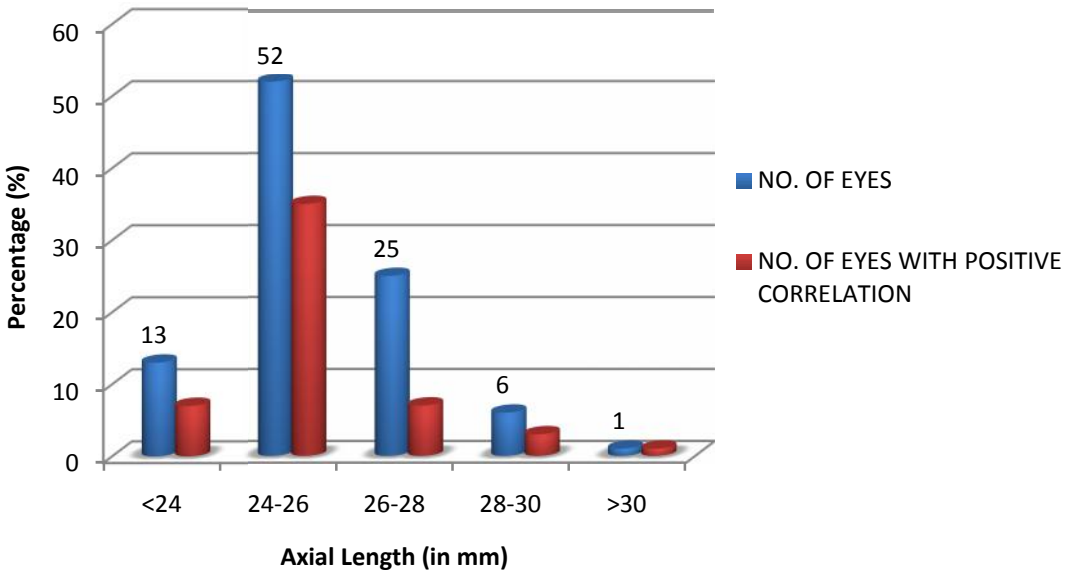


Out of the 97 eyes, most of the eyes i.e., 52 (53.6%) had an axial length between 24 – 26 mm, 25 eyes were in the group of axial length between 24 - 26 mm, 13 eyes (13.4 %) had an axial length of less than 24 mm, 6 eyes (6.19%) had an axial length between 28 – 30 mm and only 1 eye had an axial length more than 30 mm.

TABLE 10b: CORRELATION OF AXIAL LENGTH DISTRIBUTION

AXIAL LENGTH (mm)	NO. OF EYES	PERCENTAGE (%)	NO. OF EYES WITH POSITIVE CORRELATION	PERCENTAGE (%)
≤ 24	13	13.4	07	53.85
24-26	52	53.6	35	67.31
26-28	25	25.77	07	28
28-30	06	6.19	03	50
>30	01	1.03	01	100
TOTAL	97	100	53	-

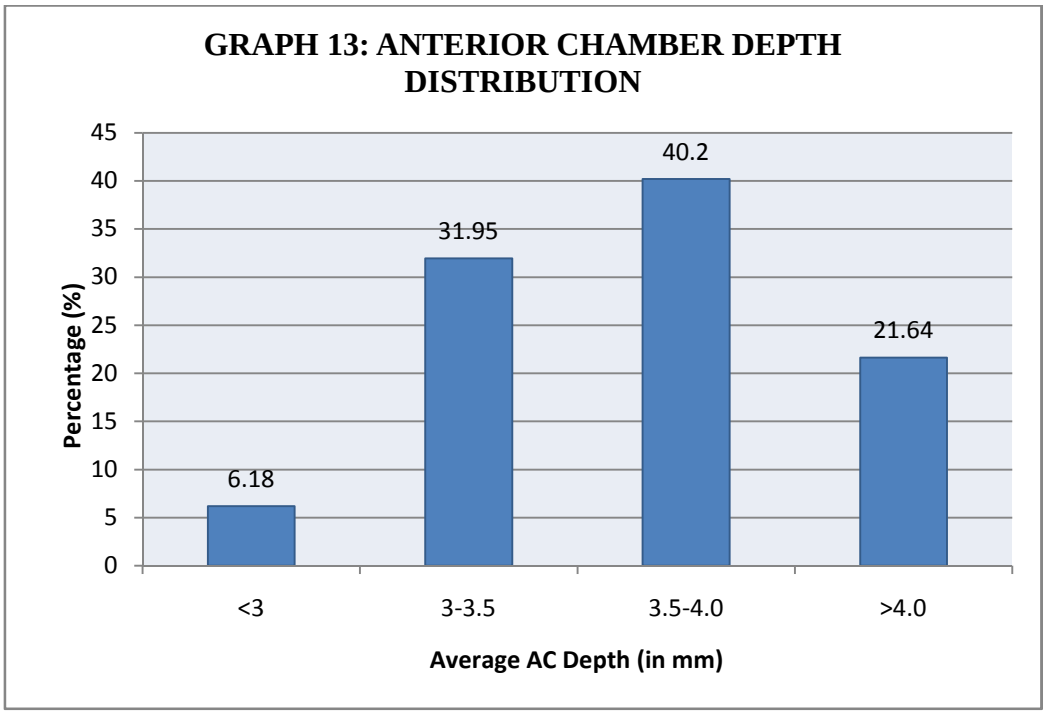
GRAPH 12: CORRELATION OF AXIAL LENGTH DISTRIBUTION



In the group having axial length ≤ 24 mm, 7 eyes (53.85%) had a positive correlation between axis of astigmatism and myopic fundal changes, between axial length 24-26 mm, 35 eyes out of 52 (67.31%) had a positive correlation, 7 eyes (28%) out of 25 having axial length between 26-28 mm had a positive correlation, 3 eyes out of 6 (50%) with axial length between 28-30 mm had a positive correlation and the one eye that had axial length more than 30 mm had a positive correlation.

TABLE 11a: ANTERIOR CHAMBER DEPTH DISTRIBUTION

AC DEPTH	NO. OF EYES	PERCENTAGE(%)
<3	06	6.18
3-3.5	31	31.95
3.5-4.0	39	40.20
>4.0	21	21.64
TOTAL	97	100

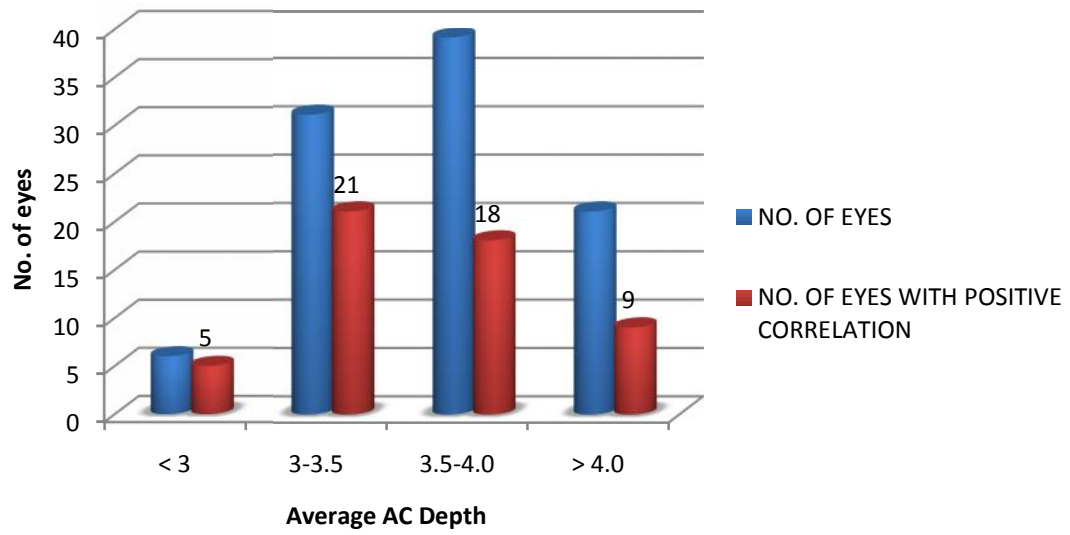


Out of 97 eyes, maximum number of eyes, i.e., 39 (40.20%), had anterior chamber depth between 3.5 – 4 mm, 31 eyes (31.95%) belonged to the group having anterior chamber depth between 3 – 3.5 mm, 21 eyes (21.64%) had anterior chamber depth above 4 mm and only 6 eyes (6.18%) had anterior chamber depth less than 3 mm.

TABLE 11b: CORRELATION OF ANTERIOR CHAMBER DEPTH DISTRIBUTION

AC DEPTH	NO. OF EYES	PERCENTAGE (%)	NO. OF EYES WITH POSITIVE CORRELATION	PERCENTAGE (%)
≤ 3	06	6.18	5	83.33
3-3.5	31	31.95	21	67.74
3.5-4.0	39	40.20	18	46.15
>4.0	21	21.64	9	42.86
TOTAL	97	100	53	-

GRAPH 14: CORRELATION OF ANTERIOR CHAMBER DEPTH DISTRIBUTION



5 out of 6 eyes (83.33%) with anterior chamber depth less than or equal to 3mm had a positive correlation between axis of astigmatism and myopic fundal changes. 21 out of 31 eyes (67.74%) with anterior chamber depth between 3-3.5 mm had a positive correlation, 18 out of 39 eyes (46.15%) having anterior chamber depth between 3.5-4 mm had a positive correlation and 9 out of the 21 eyes (42.86%) with anterior chamber depth over 4 mm had a positive correlation.

DISCUSSION

The present study was conducted as a cross sectional study to assess the correlation, if any, between the pattern of myopic fundal changes and the axis of astigmatism and to find an etiopathogenic association in the occurrence of myopic fundal changes.

The study included patients who attended the Out Patient Department of Ophthalmology at K.L.E.S Dr. Prabhakar Kore Hospital and MRC, Belgaum. The study consisted of 97 eyes of 64 patients having the refractive error of myopia with pathologic fundus changes like myopic crescent or peripapillary atrophy, along with myopic astigmatism.

Myopia is a common refractive error affecting all age groups of either sex.

We studied patients in the age group of 5-80 years. No age limit was set.

Mean age was 46.91 years and a majority of the patients (35.94%) belonged to the age group above 60 years.

Out of the 64 patients, 14 patients belonged to the age group of less than 20 years (21.88%), 10 patients belonged to the age group between 21-40 years (15.63%), 17 patients belonged to the age group of 41-60 years (26.561%), 23 patients were above 60 years of age (35.94%).

According to a study conducted in rural South India, myopia (SE < −0.50 DS) had a prevalence rate of 26.99% and high myopia (SE < −5.00 DS) was 3.71%. [43]

The prevalence of emmetropia decreased significantly with age (P < 0.0001),

and the prevalence of myopia and high myopia increased significantly with age ($P < 0.001$) and were significantly associated with nuclear sclerosis ($P < 0.001$). [43]

According to the Andhra Pradesh Eye Disease Study, in the participants who were less than or equal to 15 years of age, the prevalence of myopia was 3.19%. Myopia increased with increasing age and was more prevalent in the urban study area. In participants > 15 years of age, the prevalence of myopia was 19.45%.[112]

There were 33 male patients and 31 female patients included in our study. We did not emphasise on correlating sex with pathological myopic changes.

According to the Andhra Pradesh Eye Disease Study, myopia was more common in males.[112]

The findings of our study are in the line with published literature. According to literature, myopia prevalence varies with age, race and sex, increasing through age.

In a given patient there can be a difference of refractive error between the two eyes of the same individual. Hence we analyzed each eye separately in an individual.

Out of the 97 eyes, majority of the eyes, 73 (75.26%) had an unaided visual acuity of less than or equal to 6/60, 21 eyes (21.65%) had a visual acuity between 6/18 - 6/36 and only 3 eyes (3.09%) were having visual acuity between 6/6-6/12.

In another study, low myopia (0 to –3 D) patients had uncorrected visual acuity of 6/9 to CF 2m whereas intermediate myopia (-3 to -6 D) patients had uncorrected visual acuity of 6/24 to CF 2m and high myopia (< -6D) patients had uncorrected visual acuity of 6/60 to CF 2m.[113]

In our study, out of the 97 eyes, 48 eyes (49.48%) belonged to the group having myopia of less than or equal to -6D and 49 eyes (50.52%) belonged to the group having myopia of more than -6D.

Out of these, 13 eyes (13.40%) were having myopia, less than or equal to -3D, 35 eyes (36.08 %) belonged to the group having myopia between -3D to -6D.

27 eyes (27.84%) were having myopia between – 6D to – 9D, 12 eyes belonged to the group having myopia between – 9D to – 12D and 10 eyes (10.31%) were having myopia of more than – 12D.

We observed that maximum number of eyes, i.e., 62 eyes (62.92%) with myopic fundal changes were in the range of -3D to -9D.

Out of the 97 eyes, 63 eyes (64.95%) belonged to the group having cylindrical power of less than or equal to – 2 DC, 30 eyes (30.93%) belonged to the group having cylindrical power between – 2 to – 4 DC and only 4 patients (4.12%) had a cylindrical power of more than – 4 DC.

Out of the 97 eyes, 20 eyes (20.62%) had an axis of astigmatism of less than 45°, 27 eyes (27.84%) belonged to the group with astigmatic axis between 46° – 90°. 16 eyes (16.49%) had an astigmatic axis between 91° – 135° and 34 eyes (35.05%) belonged to the group having an axis of astigmatism between 136° – 180°.

Out of 97 eyes, 23 (23.71%) had with-the-rule astigmatism and 19 eyes (19.59%) showed against-the-rule astigmatism.

In a study conducted in rural South India, out of the participants with astigmatism (cylindrical error greater than 0.50 DC), 9.80% had with-the-rule (WTR) and 77.44% against-the-rule (ATR) astigmatism. [43]

Out of the 97 eyes, 31 eyes (31.96%) had peripapillary atrophy around the disc, whereas 66 eyes (68.04%) had a myopic crescent around the disc.

Out of the 66 eyes having myopic crescents, majority of them had a temporal crescent 45 eyes (68.18%), 13 eyes (19.70%) had nasal crescents, 7 eyes (10.61%) had inferior crescents and only 1 eye had a supertraction crescent (1.52%).

After getting the value of the axis of astigmatism by subjective refraction, fundus photographs were taken, on which the axis of long axis of the disc crescent or peripapillary atrophy was measured.

The two readings were co related using Spearman's Rank correlation co-efficient.

According to Spearman's Rank correlation co efficient, $\delta=0.495$.

Hence $p<0.001$

A positive correlation has been noted between the axis of astigmatism of the eye and of the angle of the myopic fundal changes.

This is in accordance with a study conducted in Queen's Medical Centre, Nottingham, showing that there is a significant correlation between the axis of astigmatism and the early myopic chorioretinal degenerative changes.[114]

This suggests that astigmatism may have an aetiopathogenic association in the occurrence of myopic fundal changes.

In the age group of less than or equal to 20 years, 5 eyes had a spherical equivalent of less than -6D, of which 3 eyes (60%) had a direct correlation between axis of astigmatism and fundal changes, whereas 18 eyes were above -6D, out of which 5 eyes (27.78%) had a positive correlation.

Between the age group of 21-40 years, 8 eyes had a spherical equivalent of less than -6D, of which 5 eyes (62.50%) had a direct correlation between axis of astigmatism and fundal changes, whereas 6 eyes were above -6D, out of which 3 eyes (50%) had a positive correlation.

In the age group of 41-60 years, 14 eyes had a spherical equivalent of less than -6D, of which 9 eyes (64.29%) had a direct correlation between axis of astigmatism and fundal changes, whereas 13 eyes were above -6D, out of which 8 eyes (61.54%) had a positive correlation.

In age group above 60 years, 21 eyes had a spherical equivalent of less than -6D, of which 10 eyes (47.62%) had a direct correlation between axis of astigmatism and fundal changes, whereas 12 eyes were above -6D, out of which 9 eyes (75%) had a positive correlation.

It was observed that a majority of the participants (66.46%) having a positive correlation between the axis of astigmatism and myopic fundal changes belonged to the age group of 41-60 years.

It was also observed that the correlation in eyes with a spherical equivalent of ≤-6D was more than in eyes with >-6D.

When we screened for peripheral retinal degenerative changes with an indirect ophthalmoscope and recorded the findings with a fundus camera, we observed that all of the 97 eyes had a tessellated background, 26 eyes (26.8%) showed other peripheral degenerative changes. Of these, 9 (17.50%) eyes had Lattice Degeneration in the periphery, 12 eyes (18.55%) had Posterior Staphyloma present, 3 eyes (3.09%) had Lacquer Cracks and 2 of the eyes (2.06%) had Snail Track degeneration.

Pathological myopia usually refers to a condition where there is greater than six dioptres of myopia or an axial length greater than 26-27 mm.

In a study conducted by Celorio and Pruett, in patients with myopia of six dioptres or more in both eyes, it was found that one third had lattice degeneration, with the greatest prevalence being in eyes having six to nine dioptres of myopia.[66]

Lattice degeneration represents vulnerable areas of retinal thinning.

Epidemiological studies have demonstrated increased prevalence of peripheral retinal degenerations in association with high myopia and increased axial length.[115]

Among the different types of peripheral retinal degenerations in high myopia, lattice degeneration is the most important peripheral retinal degeneration which can predispose to rhegmatogenous retinal detachment.

This is because retinal tears can develop at the posterior and lateral margins of the lattice degeneration caused by strong vitreoretinal adhesions following posterior vitreous detachment.

Excessive axial elongation of the globe in high myopia can cause mechanical stretching and thinning of the choroid and retinal pigment epithelium layers, resulting in various retinal degenerative changes.

High myopes have increased risks of retinal complications such as peripheral retinal degenerations, retinal tears, retinal detachment, posterior staphyloma, chorioretinal atrophy, retinal pigment epithelial atrophy, lacquer cracks, choroidal neovascularisation (CNVM) and macular hemorrhage.

Out of the 97 eyes, most of the eyes, i.e., 52 (53.6%) had an axial length between 24 – 26 mm, 25 eyes were in the group of axial length between 24 - 26 mm, 13 eyes (13.4 %) had an axial length of less than 24 mm, 6 eyes (6.19%) had an axial length between 28 – 30 mm and only 1 eye had an axial length more than 30 mm.

In the group having axial length ≤ 24 mm, 7 eyes (53.85%) had a positive correlation between axis of astigmatism and myopic fundal changes, between axial length 24-26 mm, 35 eyes out of 52 (67.31%) had a positive correlation, 7 eyes (28%) out of 25 having axial length between 26-28 mm had a positive correlation, 3 eyes out of 6 (50%) with axial length between 28-30 mm had a positive correlation and the one eye that had axial length more than 30 mm had a positive correlation.

It was observed in our study that maximum number of eyes had axial length between 24-26mm.

It was also observed that the maximum number of eyes having a positive correlation between axis of astigmatism and myopic crescent, i.e., 35 (67.31%) also belonged to the group with axial length 24 – 26mm, although the one eye that had axial length >30mm also showed a positive correlation.

Axial length is one of the largest determinant of refractive error.[56]

The distribution of axial length is reported to be positively skewed in the general population and it is under a normal distribution in some selected cohorts.[60]

Out of 97 eyes, maximum number of eyes, i.e., 39 (40.20%), had anterior chamber depth between 3.5 – 4 mm, 31 eyes (31.95%) belonged to the group having anterior chamber depth between 3 – 3.5 mm, 21 eyes (21.64%) had anterior chamber

depth above 4 mm and only 6 eyes (6.18%) had anterior chamber depth less than 3 mm.

5 out of 6 eyes (83.33%) with anterior chamber depth less than or equal to 3mm had a positive correlation between axis of astigmatism and myopic fundal changes. 21 out of 31 eyes (67.74%) with anterior chamber depth between 3-3.5 mm had a positive correlation, 18 out of 39 eyes (46.15%) having anterior chamber depth between 3.5-4 mm had a positive correlation and 9 out of the 21 eyes (42.86%) with anterior chamber depth over 4 mm had a positive correlation.

It has been suggested in the early 19th century that astigmatism has a causal role in myopia.[10]

Corneal nebulae, which are a common cause of astigmatism, were also suggested to influence the development and progression of myopia.[10]

This association of myopic axial elongation of the globe with corneal nebulae has been demonstrated by ultrasonic measurements.

The obliquity of the retina is also considered one of the causes of astigmatism. The posterior pole of the eye may be placed obliquely, due to a staphyloma.

However, in this study even early peripapillary atrophy was associated with and had a significant correlation with the axis of astigmatism.

Astigmatism produces a distortion of the retinal image. In compound myopic astigmatism, the meridians of maximum and minimum power are both too strong and fall short of the retina.

If the elongation of the posterior segment of the globe is symmetrical round the visual axis, the chorioretinal changes will be peripherally along the ora and infero-temporal to the disc, the fovea being infero-nasal to the disc.

In our observation, when the location of the disc crescent or the shape of the peripapillary atrophy did not correspond to the symmetrical elongation of the globe, it had a correlation with the axis of astigmatism.

These suggest that astigmatism may have an etiopathogenic association in the occurrence of myopic fundal changes.

CONCLUSION

Myopia is a common refractive error affecting all age groups of either sex.

It can be unilateral or bilateral.

Myopia increases with age. Most cases were noted above 60 years in our study.

Majority of the patients with myopic fundal changes have a poor visual acuity, i.e., most of them had an uncorrected visual acuity ≤6/60.

Most of the patients with myopic fundal changes had a spherical equivalent of -3D to -9D and ≤ -2 DC

Among the myopic disc changes, more cases had crescents than peripapillary atrophy, and of these crescents, temporal crescents were the most predominant.

A positive correlation is present between the axis of astigmatism of the eye and the angle of the myopic fundal changes.

This correlation is highest in the age group of 40-60 years and is more predominant in eyes with spherical equivalent ≤ -6D than those with a spherical equivalent > -6D.

26.8% eyes had peripheral degenerative changes, but these changes have no correlation with the disc changes and astigmatism.

Also, the maximum number of eyes having a positive correlation between axis of astigmatism and myopic crescent, have an axial length between 24 – 26 mm and average anterior chamber depth between 3 – 3.5 mm.

Hence, it was concluded from our study that there is a positive correlation between the axis of astigmatism and myopic fundal changes and astigmatism may have an aetiopathogenic association in the occurrence of myopic fundal changes.

SUMMARY

The present study was conducted as a cross sectional study to assess the correlation, if any, between the pattern of myopic fundal changes and the axis of astigmatism and to find an etiopathogenic association in the occurrence of myopic fundal changes.

The study included patients who attended the Out Patient Department of Ophthalmology at KLES Dr. Prabhakar Kore Hospital and MRC, Belgaum.

A total of 97 eyes of 64 patients having myopic disc changes, i.e.,peripapillary atrophy or myopic crescents were studied. Each eye had undergone subjective refraction testing, measurement of corneal curvature, axial length and anterior chamber depth using A – Scan Biometer (Echorule2, Biomedix).

Fundoscopic examination was carried out thoroughly by indirect ophthalmoscopy with +20 D lens. The fundus photograph was recorded with a Canon CF–1 Digital Retinal Camera.

The deep/longest axis of the crescent/peripapillary atrophy was determined and the axis of myopic crescent or peripapillary atrophy was measured using the annotations incorporated within the software attached to the fundus camera and was ascribed a value in degrees, in a manner similar to the axis of astigmatism in refraction.

The long axis of peripapillary changes and of the disc crescent were correlated with the axis of astigmatism in patients with compound myopic astigmatism and the coefficient of correlation determined.

The summary of the results obtained is as follows,

Myopia is a common refractive error affecting all age groups of either sex.

We studied patients in the age group of 5-80 years. No age limit was set.

Mean age was 46.91 years with majority of the patients (35.94%) being in the age group above 60 years.

14 patients belonged to the age group of less than 20 years (21.88%), 10 patients belonged to the age group between 21-40 years (15.63%), 17 patients belonged to the age group of 41-60 years (26.561%).

There were 33 (51.56%) male patients and 31 (48.44%) female patients included in our study. We did not emphasize on correlating sex with pathological myopic changes.

Myopia prevalence varies with age, race and sex and increases throughout age.

In a given patient there can be a difference of refractive error between the two eyes of the same individual. Hence we analyzed each eye separately in an individual.

Out of the 97 eyes, majority of the eyes, 73 (75.26%) had an unaided visual acuity of less than or equal to 6/60, 21 eyes (21.65%) had a visual acuity between 6/18 - 6/36 and only 3 eyes (3.09%) were having visual acuity between 6/6-6/12.

In our study, out of the 97 eyes, 48 eyes (49.48%) belonged to the group having myopia of less than or equal to -6D and 49 eyes (50.52%) belonged to the group having myopia of more than -6D.

13 eyes (13.40%) were having myopia, less than or equal to -3D, 35 eyes (36.08 %) belonged to the group having myopia between -3D to -6D.

27 eyes (27.84%) were having myopia between -6D to -9D, 12 eyes belonged to the group having myopia between -9D to -12D and 10 eyes (10.31%) were having myopia of more than -12D.

We observed that maximum number of eyes, i.e., 62 eyes (62.92%) with myopic fundal changes were in the range of -3D to -9D.

Out of the 97 eyes, 63 eyes (64.95%) belonged to the group having cylindrical power of less than or equal to -2 DC, 30 eyes (30.93%) belonged to the group having cylindrical power between -2 to -4 DC and only 4 patients (4.12%) had a cylindrical power of more than -4 DC.

20 eyes (20.62%) had an axis of astigmatism of less than 45°, 27 eyes (27.84%) belonged to the group with astigmatic axis between 46° – 90°. 16 eyes (16.49%) had an astigmatic axis between 91° – 135° and 34 eyes (35.05%) belonged to the group having an axis of astigmatism between 136° – 180°.

23 eyes (23.71%) had with-the-rule astigmatism and 19 eyes (19.59%) showed against-the-rule astigmatism.

Out of the 97 eyes, 31 eyes (31.96%) had peripapillary atrophy around the disc, whereas 66 eyes (68.04%) had a myopic crescent around the disc.

Out of the 66 eyes having myopic crescents, majority of them had a temporal crescent, i.e., 45 eyes (68.18%), 13 eyes (19.70%) had nasal crescents, 7 eyes (10.61%) had inferior crescents and only 1 eye had a supertraction crescent (1.52%).

After getting the value of the axis of astigmatism by subjective refraction, fundus photographs were taken, on which the axis of long axis of the disc crescent or peripapillary atrophy was measured.

The two readings were co related using Spearman's Rank correlation co-efficent.

According to Spearman's Rank correlation co-efficient, $\delta=0.495$, $p<0.001$ was calculated, which was considered statistically significant.

A positive correlation was noted between the axis of astigmatism of the eye and of the angle of the myopic fundal changes.

In the age group of less than or equal to 20 years, 5 eyes had a spherical equivalent of less than -6D, of which 3 eyes (60%) had a direct correlation between axis of astigmatism and fundal changes, whereas 18 eyes were above -6D, out of which 5 eyes (27.78%) had a positive correlation.

Between the age group of 21-40 years, 8 eyes had a spherical equivalent of less than -6D, of which 5 eyes (62.50%) had a direct correlation between axis of astigmatism and fundal changes, whereas 6 eyes were above -6D, out of which 3 eyes (50%) had a positive correlation.

In the age group of 41-60 years, 14 eyes had a spherical equivalent of less than -6D, of which 9 eyes (64.29%) had a direct correlation between axis of astigmatism and fundal changes, whereas 13 eyes were above -6D, out of which 8 eyes (61.54%) had a positive correlation.

In age group above 60 years, 21 eyes had a spherical equivalent of less than -6D, of which 10 eyes (47.62%) had a direct correlation between axis of astigmatism and fundal changes, whereas 12 eyes were above -6D, out of which 9 eyes (75%) had a positive correlation.

It was observed that a majority of the participants (66.46%) having a positive correlation between the axis of astigmatism and myopic fundal changes belonged to the age group of 41-60 years.

It was also observed that the correlation in eyes with a spherical equivalent of ≤-6D was more than in eyes with >-6D.

When screened for peripheral retinal degenerative changes with an indirect ophthalmoscope it was observed that all the 97 eyes had a tessellated background, 26 eyes (26.08%) had other peripheral degenerative changes. Of these, 9 (17.50%) eyes had Lattice Degeneration in the periphery, 12 eyes (18.55%) had Posterior Staphyloma present, 3 eyes (3.09%) had Lacquer Cracks and 2 of the eyes (2.06%) had Snail Track degeneration.

The peripheral changes did not have any correlation with the axis of astigmatism and disc changes.

Out of the 97 eyes, most of the eyes i.e., 52 (53.6%) had an axial length between 24 – 26 mm, 25 eyes were in the group of axial length between 24 - 26 mm, 13 eyes (13.4 %) had an axial length of less than 24 mm, 6 eyes (6.19%) had an axial length between 28 – 30 mm and only 1 eye had an axial length more than 30 mm.

In the group having axial length ≤ 24 mm, 7 eyes (53.85%) had a positive correlation between axis of astigmatism and myopic fundal changes, between axial length 24-26 mm, 35 eyes out of 52 (67.31%) had a positive correlation, 7 eyes (28%) out of 25 having axial length between 26-28 mm had a positive correlation, 3 eyes out of 6 (50%) with axial length between 28-30 mm had a positive correlation and the one eye that had axial length more than 30 mm had a positive correlation.

It was observed in our study that maximum number of eyes had axial length between 24-26mm.

It was also observed that the maximum number of eyes having a positive correlation between axis of astigmatism and myopic crescent, i.e., 35 (67.31%) also belonged to the group with axial length 24 – 26mm, although the one eye that had axial length >30mm also showed a positive correlation.

Out of 97 eyes, maximum number of eyes, i.e., 39 (40.20%), had anterior chamber depth between 3.5 – 4 mm, 31 eyes (31.95%) belonged to the group having anterior chamber depth between 3 – 3.5 mm, 21 eyes (21.64%) had anterior chamber depth above 4 mm and only 6 eyes (6.18%) had anterior chamber depth less than 3 mm.

5 out of 6 eyes (83.33%) with anterior chamber depth less than or equal to 3mm had a positive correlation between axis of astigmatism and myopic fundal changes. 21 out of 31 eyes (67.74%) with anterior chamber depth between 3-3.5 mm had a positive correlation, 18 out of 39 eyes (46.15%) having anterior chamber depth between 3.5-4 mm had a positive correlation and 9 out of the 21 eyes (42.86%) with anterior chamber depth over 4 mm had a positive correlation.

Astigmatism produces a distortion of the retinal image. In compound myopic astigmatism, the meridians of maximum and minimum power are both too strong and fall short of the retina.

If the elongation of the posterior segment of the globe is symmetrical round the visual axis, the chorioretinal changes will be peripherally along the ora and infero-temporal to the disc, the fovea being infero-nasal to the disc.

In our observation, when the location of the disc crescent or the shape of the peripapillary atrophy did not correspond to the symmetrical elongation of the globe, it had a correlation with the axis of astigmatism.

These results and findings suggest that there is a positive correlation between the axis of astigmatism of the eye and of the angle of the myopic fundal changes and suggest that astigmatism may have an etiopathogenic association in the occurrence of myopic fundal changes.

BIBLIOGRAPHY

1. Park.K, Park's Textbook of Preventive and Social Medicine, 19th edition:337

2. Fredrick DR. Myopia. British J Ophthalmol. 2002; 324: 1195-1199.

3. Pruett R C,Pathological Myopia, Albert Jackobiec, Principles and Practice of Ophthalmology 2nd Edition vol. 3; 2062-2064.

4. Rens GHMBV, Arkell SM. Refractive errors and axial length among Alaskan Eskimos. ActaOphthalmologica. 1991;69:27-325

5. The Prevalence of Astigmatism and its Determinants in a Rural Population of Iran: The "NooravaranSalamat" Mobile Eye Clinic Experience.Hassan Hashemi, FarhadRezvan, Abbas Ali Yekta1, Maryam Hashemi2, Reza Norouzirad3, Mehdi Khabazkhoob4

6. Pierro L, Camesasca FI, Mischi M, et al. Peripheral retinal changes and axial myopia. Retina 1992;12:12e7.

7. Curtin BJ. The myopias: basic science and clinical management. Philadelphia: Harper & Row, 1985.

8. Huynh SC, Wang XY, Ip J, et al. Prevalence and associations of anisometropia and aniso-astigmatism in a population based sample of 6 year old children. Br J Ophthalmol 2006; 90:597e601.

9. Williams C, Miller LL, Gazzard G, et al. A comparison of measures of reading and intelligence as risk factors for the development of myopia in a UK cohort of children. Br J Ophthalmol 2008;92:1117e21.

10. Duke Elder. System of Ophthalmology, Chapter-V, Anomalies of the optical System, London, Henry Kimpton; 1967; Vol. 5: 241-253.

11. Gwiazda J, Grice K, Held R, et al. Astigmatism and the development of myopia in children. Vision Res 2000;40:1019e26.

12. Fan DS, Rao SK, Cheung EY, et al. Astigmatism in Chinese preschool children: prevalence, change, and effect on refractive development. Br J Ophthalmol 2004;88:938e41.

13. Heidary G, Ying GS, Maguire MG, et al. The association of astigmatism and spherical refractive error in a high myopia cohort. Optom Vis Sci 2005;82:244e7.

14. Saw SM, Gazzard G, Koh D, Farook M, Widjaja D, Lee J, et al. Prevalence rates of refractive errors in Sumatra, Indonesia. Invest Ophthalmol Vis Sci 2002;43:3174-80.

15. Cheng CY, Hsu WM, Liu JH, Tsai SY, Chou P. Refractive errors in an elderly Chinese population in Taiwan: The Shihpai Eye Study. Invest Ophthalmol Vis Sci 2003;44:4630-8.

16. Sawada A, Tomidokoro A, Araie M, Iwase A, Yamamoto T. Refractive errors in an elderly Japanese population: The Tajimi study. Ophthalmology 2008;115:363-70.

17. Yekta A, Fotouhi A, Hashemi H, Dehghani C, Ostadimoghaddam H, Heravian J, et al. Prevalence of refractive errors among schoolchildren in Shiraz, Iran. Clin Experiment Ophthalmol 2010;38:242-8.

18. Rezvan F, Khabazkhoob M, Fotouhi A, Hashemi H, Ostadimoghaddam H, Heravian J, et al. Prevalence of refractive errors among school children in Northeastern Iran. Ophthalmic Physiol Opt 2012;32:25-30.

19. Liang YB, Wong TY, Sun LP, Tao QS, Wang JJ, Yang XH, et al. Refractive errors in a rural Chinese adult population the Handan eye study. Ophthalmology 2009;116:2119-27.

20. Ostadimoghaddam H, Fotouhi A, Hashemi H, Yekta A, Heravian J, Rezvan F, et al. Prevalence of the refractive errors by age and gender in Mashhad, Iran: The Mashhad eye study. Clin Experiment Ophthalmol 2011;39:743-51.

21. Hashemi H, Khabazkhoob M, Yekta A, Jafarzadehpur E, Emamian MH, Shariati M, et al. High prevalence of astigmatism in the forty to sixty-four year old population of Shahroud, Iran. Clin Experiment Ophthalmol 2012;40:247-54.

22. Fotouhi A, Hashemi H, Yekta AA, Mohammad K, Khoob MK. Characteristics of astigmatism in a population of schoolchildren, Dezful, Iran. Optom Vis Sci 2011;88:1054-9.

23. Schellini SA, Durkin SR, Hoyama E, Hirai F, Cordeiro R, Casson RJ, et al. Prevalence of refractive errors in a Brazilian population: The Botucatu eye study. Ophthalmic Epidemiol 2009;16:90-7.

24. Li Z, Sun D, Cuj H, Zhang L, Lju P, Yang H, et al. Refractive error among the elderly in rural Southern Harbin, China. Ophthalmic Epidemiol 2009;16:388-94.

25. Gupta A, Casson RJ, Newland HS, Muecke J, Landers J, Selva D, et al. Prevalence of refractive error in rural Myanmar: The Meiktila Eye Study. Ophthalmology 2008;115:26-32.

26. Hashemi H, Hatef E, Fotouhi A, Mohammad K. Astigmatism and its determinants in the Tehran population: The Tehran eye study. Ophthalmic Epidemiol 2005;12:373-81.

27. Fan DS, Rao SK, Cheung EY, Islam M, Chew S, Lam DS. Astigmatism in Chinese preschool children: Prevalence, change, and effect on refractive development. Br J Ophthalmol 2004;88:938-41.

28. Read SA, Collins MJ, Carney LG. A review of astigmatism and its possible genesis. ClinExpOptom 2007;90:5-19.

29. Mandel Y, Stone RA, Zadok D. Parameters associated with the different astigmatism axis orientations. Invest Ophthalmol Vis Sci 2010;51:723-30.

30. The Myopia Myth: The Truth about Nearsightedness And How to Prevent It: Donald S. Rehm; International Myopia Prevention Assn.

31. Daniel M Albert, Diane D Edwards. History of Ophthalmology, Blackwell Science, 1996.

32. Fan DS, Lam DS, Lam RF, Lau JT, Chong KS, Cheung EY, Lai RY and Chew SJ (2004). Prevalence, incidence, and progression of myopia of school children in Hong Kong. Invest. Ophthalmol. Vis. Sci. 45 (4): 1071-5.

33. Cheng D, Schmid KL and Woo GC (2007). Myopia prevalence in Chinese-Canadian children in an optometric practice. Optom Vis Sci. 84 (1): 21-32.

34. Saw SM, Nieto FJ, Katz J, Schein OD, Levy B and Chew SJ (2000). Factors related to the progression of myopia in Singaporean children. Optom Vis Sci. 77 (10): 549-54.

35. Lam CS and Goh WS (1991). The incidence of refractive errors among school children in Hong Kong and its relationship with the optical components. Clin. Exp. Optom. 74: 97-103.

36. Katz J, Tielsch JM, Sommer A. Prevalence and risk factors for refractive errors in an adult inner city population. Invest Ophthalmol Vis Sci. Feb 1997;38(2):334-340.

37. Wang Q, Klein BE, Klein R, Moss SE. Refractive status in the Beaver Dam Eye Study. Invest Ophthalmol Vis Sci. Dec 1994;35(13):4344-4347.

38. Attebo K, Ivers RQ, Mitchell P. Refractive errors in an older population: the Blue Mountains Eye Study. Ophthalmology. Jun 1999;106(6):1066-1072.

39. Wensor M, McCarty CA, Taylor HR. Prevalence and risk factors of myopia in Victoria, Australia. Arch Ophthalmol. May 1999;117(5):658-663.

40. Wong TY, Foster PJ, Hee J, et al. Prevalence and risk factors for refractive errors in adult Chinese in Singapore. Invest Ophthalmol Vis Sci. Aug 2000;41(9):2486-2494.

41. Sawada A, Tomidokoro A, Araie M, Iwase A, Yamamoto T. Refractive errors in an elderly Japanese population: the Tajimi study. Ophthalmology. Feb 2008;115(2):363-370 e363.

42. Krishnaiah S, Srinivas M, Khanna RC, Rao GN. Prevalence and risk factors for refractive errors in the South Indian adult population: The Andhra Pradesh Eye disease study. ClinOphthalmol. 2009;3:17-27.

43. Raju P, Ramesh SV, Arvind H, et al. Prevalence of refractive errors in a rural South Indian population. Invest Ophthalmol Vis Sci. Dec 2004;45(12):4268-4272.

44. Nangia V, Jonas JB, Sinha A, Matin A, Kulkarni M. Refractive error in central India: the Central India Eye and Medical Study. Ophthalmology. Apr 2010;117(4):693-699.

45. Garner LF, Owens H, Kinnear RF, Frith MJ. Prevalence of myopia in Sherpa and Tibetan children in Nepal. Optom Vis Sci. May 1999;76(5):282-285.

46. Zhan MZ, Saw SM, Hong RZ, et al. Refractive errors in Singapore and Xiamen, China--a comparative study in school children aged 6 to 7 years. Optom Vis Sci. Jun 2000;77(6):302-308.

47. Wu SY, Nemesure B, Leske MC. Refractive errors in a black adult population: the Barbados Eye Study. Invest Ophthalmol Vis Sci. Sep 1999;40(10):2179-2184.

48. Samarawickrama C, Wang JJ, Burlutsky G, Tan AG, Mitchell P. Nuclear cataract and myopic shift in refraction. Am J Ophthalmol.Sep 2007;144(3):457-459.

49. Cheng CY, Hsu WM, Liu JH, Tsai SY, Chou P. Refractive errors in an elderly Chinese population in Taiwan: the Shihpai Eye Study. Invest Ophthalmol Vis Sci. Nov 2003;44(11):4630-4638.

50. Donder FC. Accommodation and refraction of eye, 1864; Reprint 1979.

51. McBrien N, Gentle A. Role of the sclera in the development and pathological complications of myopia. Progress in retinal and eye research. 2003;22(3):307--338.

52. Goldschmidt F. On the etiology of myopia. Copenhagen, Munksgaard 1968.

53. Wong TY, Foster PJ, Ng TP, Tielsch JM, Johnson GJ, Seah SK: Variations in ocular biometry in an adult Chinese population in Singapore: The TanjongPagar Survey. Invest Ophthalmol Vis Sci 2001; 42: 73–80.

54. W. Sowka J. handbook of ocular disease management. 1st ed. 2011.

55. Hui J, Peck L, Howland HC. Correlations between familial refractive error and children's non-cycloplegic refractions. Vision Res 1995, 35: 1353-8.

56. Keller JT. A comparison of the refractive status of myopic children and their

parents. Am J Optom Arch Am AcadOptom 1973; 50: 206-ll.

57. Zadnik K, Satariano WA, Mutti DO, et al. The effect of parental history of myopia on children's eye size. JAMA 1994; 271: 1323-7.

58. Mutti DO, Mitchell GL, Jones LA, Friedman NE, Frane SL, Lin WK, Moeschberger ML, Zadnik K. Axial growth and changes in lenticular and corneal power during emmetropization in infants. Invest Ophthalmol Vis Sci2005; 46: 3074–3080.

59. Su DH, Wong TY, Foster PJ, Tay WT, Saw SM, Aung T. Central corneal thickness and its associations with ocular and systemic factors: the Singapore Malay Eye Study. Am J Ophthalmol 2009; 147: 709–716.

60. Sorsby A, Leary GA, Fraser GR. Family studies on ocular refraction and its components. J Med Genet 1966; 3: 269-73.

61. Zejmo M, Forminska-Kapuscik M, Pieczara E, Filipek E, Mrukwa-Kominek E, Samochowiec-Donocik E et al. Etiopathogenesis and management of high-degree myopia. Part I. Med SciMonit 2009; 15: 199–202.

62. Ruben M, Khoo CY. Contact lenses: medical aspects. Singapore: PG Publishers, 1989.

63. Graham MV, Gray OP. Refraction of premature babies' eyes. Br Med J 1963; 1: 1452-4.

64. Text of the fundus of the eye, 2nd Edition, Arthur J Ballantyne, Issac C Michaelson, Edinburgh & London, E&S Livingstone, 1970: 480.

65. Patrick D Trevor, Roper peter V Curran. The eye and its disorders, 2nd Edition, chapter-15; PG Asian Economy Edition, 1984; PG Publishing Pte Ltd. 1984: 194-198.

66. Celorio JM, Pruett RC. Prevalence of lattice degeneration and its relation to axial length in severe myopia. Am J Ophthalmol 1991; 111: 20-3.

67. Brain J Curtin. Physiologic Vs Pathologic Myopia: Genetics vs Environment. Survey Ophthalmology 1979; 86: 681-691.

68. Karlin DB, Curtin BJ. Peripheral chorioretinal lesions and axial lengths of the myopic eye. Am. J.Ophthalmol 1976; 81(5): 625-35.

69. Perkins ES. Morbidity from myopia. Sight-Saving Rev 1979; 49: 11-9.

70. Thylefors, B. Negrel, A.D. Available data on blindness. Ophthal. Epidemiol. 1995;2, 5-39.

71. Perkins ES, Phelps CD. Open angle glaucoma, ocular hypertension, low tension glaucoma and refraction. Arch Ophthalmol 1982; 100:1464-7.

72. Bailey, A.J. Molecular mechanisms of ageing in connective tissues. Mechanisms of Ageing and Development, 2001;735-755.

73. Singh, R., Barden, A., Mori, T., Beilin, L. Advanced glycationend-products: a review. Diabetologia 2001;129-146.

74. Spoerl, E., Wollensak, G., Seiler, T. Increased resistance of crosslinked cornea against enzymatic digestion. Current Eye Research 2004; 35-40.

75. Gwiazda J, Hyman L, et al. A randomized clinical trial of progressive addition

lenses versus single vision lenses on the progression of myopia in children. Invest Ophthalmol Vis Sci 2003; 44:1492–500.

76. Edwards MH, Li RW, Lam CS, et al. The Hong Kong Progressive Lens Myopia Control Study: study design and main findings. Invest Ophthalmol Vis Sci 2002; 43:2852–8.

77. Blinder, K.J., Blumenkranz, M.S., Bressler, N.M., Bressler, S.B., Donati, G., Lewis, H., Lim, J.I., Menchini, U., Miller, J.W., Mones, J.M., Potter, M.J., Pournaras, C., Reaves, A., Rosenfeld, P., Schachat, A.P., Schmidt-Erfurth, U., Sickenberg, M., Singerman, L.J., Slakter, J., Strong, H.A., Virgili, G., Williams, G.A. Verteporfin therapy of subfovealchoroidal neovascularization in pathologic myopia - 2-year results of a randomized clinical Trial - VIP report no. 3. Ophthalmolog.2003;667-673.

78. Chan, W.M., Ohji, M., Lai, T.Y.Y., Liu, D.T.L., Tano, Y., Lam, D.S.C. Choroidalneovascularisation in pathological myopia: an update in management. British Journal of Ophthalmology,2005;1522-1525.

79. Buys, Y.M. Bevacizumab: the need for controlled studies to move forward. Canadian Journal of Ophthalmology-Journal Canadien D Ophtalmologie2007;42:789-789.

80. Rosenfeld, P.J. IntravitrealAvastin for choroidalneovascularisation in pathological myopia: the controversy continues. British Journal of Ophthalmology 2007;91:128-130.

81. Yamamoto, I., Rogers, A.H., Reichel, E., Yates, P.A., Duker, J.S. Intravitreal

bevacizumab (Avastin) as treatment for subfoveal choroidal neovascularisation secondary to pathological myopia. Br J Ophthalmol 2007;91,157-160.

82. Sakaguchi, H., Ikuno, Y., Gomi, F., Kamei, M., Sawa, M., Tsujikawa, M., Oshima, Y., Kusaka, S., Tano, Y. Intravitreal injection of bevacizumab for choroidalneovascularisation associated with pathological myopia. British Journal of Ophthalmology 2007;91:161-165.

83. Tarutta, Y.P., Iomdina, Y.N., Shamkhalova, E.S., Andreyeva, L.D., Maximova, M.V. Sclera Fortification In Children At A High-Risk Of Progressive Myopia. VestnikOftalmologii.1992;108:14-17.

84. Politzer, M. Experiences In Medical-Treatment Of Progressive Myopia. Klinische Monatsblatter Fur Augenheilkunde1977;171:616-619.

85. Belyaev, V.S., Ilyina, T.S. Late Results Of Scleroplasty In Surgical Treatment Of Progressive Myopia. Eye Ear Nose And Throat Monthly 1975;54: 109-113.

86. Chauvaud, D., Assouline, M., Perrenoud, F. Scleral reinforcement. Journal Francais D Ophtalmologie 1997;20, 374-382.

87. Jacob, J.T., Lin, J.J., Mikal, S.P. Synthetic scleral reinforcement materials .3. Changes in surface and bulk physical properties. Journal Of Biomedical Materials Research 1997;37: 525-533.

88. Korobelnik, J.F., D'Hermies, F., Chauvaud, D., Legeais, J.M., Hoang-Xuan, T., Renard, G. Expanded polytetrafluoroethyleneepiscleral implants used as encircling scleral buckling - An experimental and histopathological study. Ophthalmic Research 2000;32: 110-117.

89. Mortemousque, B., Leger, F., Velou, S., Graffan, R., Colin, J., Korobelnik, J.F. S/e-PTFE episcleral buckling implants: An experimental and histopathologic study. Journal Of Biomedical Materials Research 2002;63: 686-691.

90. Jacoblabarre, J.T., Assouline, M., Conway, M.D., Thompson, H.W., McDonald, M.B. Effects Of Scleral Reinforcement On The Elongation Of Growing Cat Eyes. Archives Of Ophthalmology 1993;111: 979-986.

91. Atkinson, J., Braddick, O. & French, J. (1980), 'Infant astigmatism: its disappearance with age', Vision Res, 20 (11), 891-3.

92. Baldwin, W.R. & Mills, D., 'A longitudinal study of corneal astigmatism and total astigmatism', Am J Optom Physiol Opt 1981;58 (3), 206-11.

93. Ehrlich, D. L., et al., 'Infant emmetropization: longitudinal changes in refraction components from nine to twenty months of age', Optom Vis Sci, 1997;74 (10), 822-43.

94. Friling, R., et al., 'Keratometry measurements in preterm and full term newborn infants', Br J Ophthalmol 2004;88 (1), 8-10.

95. Gwiazda, J., et al., 'Astigmatism in children: changes in axis and amount from birth to six years', Invest Ophthalmol Vis Sci, 1984;25 (1), 88-92.

96. Mutti, D. O., et al., 'Refractive astigmatism and the toricity of ocular components in human infants', Optom Vis Sci, 2004;81 (10), 753-61.

97. Campbell, F.W., Westheimer G., ‘Factors influencing accommodation responses of thehuman eye’, J Opt Soc Am, 1959;49 (6), 568–71.

98. Gullstrand, A, The cornea, In: Helmoltz”s Treatise on Physiological Optics, Helmoltz, H.V., Southal, J.P.C., Wade, N., Vol, 1, Dover, New York.1962.

99. Karesh, J.W. (1994), “Topographic Anatomy of the eye: an overview”, In:

Duane”s Foundations of Clinical Ophthalmology, Jeager, E.A., Tasman, W., pp.(1-30), JB Lippincott, Philadelphia.

100. Huynh, S. C., et al., 'Astigmatism in 12-year-old Australian children: comparisonswith a 6-year-old population', Invest Ophthalmol Vis Sci 2007;48 (1), 73-82.
101. Fotedar, R., Mitchell, P., Burlutsky, G. & Wang, J.J., ‘Relationship of 10-year change in refraction to nuclear cataract and axial length findings from an older population’, Ophthalmology 2008;115 (8), 1273-8.
102. Ho, J. D., et al., 'Effects of aging on anterior and posterior corneal astigmatism', Cornea, 2010;29 (6), 632-7.
103. Goto, T., et al., 'Gender- and age-related differences in corneal topography', Cornea, 2001;20 (3), 270-6.
104. Mehdizadeh, M., 'Age and refraction', Ophthalmology, 2008;115 (11), 2097; author reply 2097-.
105. Read, S. A., Collins, M. J. & Carney, L. G., 'The diurnal variation of corneal topography and aberrations', Cornea, 2005;24 (6), 678-87.
106. Osuobeni, E.P. & Al Mijalli, M.H., 'Association between eyelid thickness and corneal astigmatism', Clin Ex Optom 1997;80 (1), 35-9.
107. Lyle, WM, Grosvenor, T. & Dean, KC, 'Corneal astigmatism in Amerind children', Am J Optom Arch Am Acad Optom 1972;49 (6), 517-24.
108. Walsh, G. ‘The effect of mydriasis on the pupillary centration of the human eye’, Ophthalmic Physiol Opt, 2008 (2), 178-82.
109. Tsukamoto, M., et al., 'Accommodation causes with-the-rule astigmatism in emmetropes', Optom Vis Sci, 2000;77 (3), 150-5.

110. Neuhann, I. M. et al., 'Static and dynamic rotational eye tracking during LASIK treatment of myopic astigmatism with the Zyoptix laser platform and Advanced Control Eye Tracker', J Refract Surg, 2010;26(1), 17-27.
111. Flüeler, U. R. & D. L. Guyton. 'Does a tilted retina cause astigmatism? The ocular imagery and the retinoscopic reflex resulting from a tilted retina', Surv Ophthalmol, 1995;40 (1), 45-50.
112. Dandona R, Dandona L. Review of findings of the Andhra Pradesh Eye Disease Study: policy implications for eye-care services. Indian J Ophthalmol 2001;49:215.
113. Ci A. A Clinical Study Of Axial Myopia Using Ophthalmic A-Scan Ultrasonography. 2011;14:139-159.
114. Ahmad R., Al-Aqaba M.A., Dua H.S. et al. Correlation between the pattern of myopic fundal changes and the axis of astigmatism of the eye. Br J Ophthalmol 2010;94:307-310.

ANNEXURE – I: CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Patient I.D. No.:

Mr/Mrs/Ms:_____

You are invited to participate in our research study titled **ASSOCIATION BETWEEN MYOPIC FUNDAL CHANGES AND ASTIGMATISM OF THE EYE** conducted by **Dr. _____**, Post Graduate in M.S. OPHTHALMOLOGY under the guidance of **Dr. _____,** Professor, Department of Ophthalmology, J.N. Medical College, Belgaum.

Respected sir/ madam. We request you to enroll yourself to participate in our study as you are eligible for doing so.

Your participation in research is voluntary. Your decision whether or not to participate in the study will not affect your relationship with the hospital. If you decide to participate, you are free to withdraw at anytime.

Purpose of the study:

The purpose of this research is to assess the association between the pattern of myopic fundal changes and the axis of astigmatism of the eye.

Procedure involved:

If you agree to enroll yourself in this study I will ask your present, past and family history. Then you will be clinically examined in detail by slit lamp examination. Then you will undergo refraction testing, after which your axial length and anterior chamber depth will be measured with an A-Scan Biometer. Finally, after dilating your pupils, your fundus photograph will be taken with a fundus camera.

Risks and benefits:

There are no risks involved. Your participation may benefit you and others suffering from same ailment in future by helping us learn more and by establishing certain facts from this study.

Cost for participation in this research:

There will not be any extra cost incurred by the participant. The participant will however have to pay for the investigations, which are part of the existing management protocol for this ailment. There is no commitment for reimbursement or any other compensation for the participant.

Privacy and confidentiality:

Your privacy is guaranteed. The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission. However, your medical records can be directly accessed and reviewed by authorized individuals or by the ethics committee.

Authorization to publish results:

When the results of the research are published or discussed in a conference no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

CONSENT FOR PARTICIPATION IN RESEARCH TRIAL

I,Mr/Ms/Mrs voluntarily agree for the participation as a subject of study. By signing this consent form I am not giving up any of my legal rights. I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name :_____

Signature or the left thumb print of subject:_____

Witness Name :_____

Signature of Witness :_____

Investigators Name: Dr. _____

Signature of investigator:_____

Name of Guide :_____

Signature of Guide :_____

Date: Place:

ANNEXURE – II : PROFORMA

ASSOCIATION BETWEEN MYOPIC FUNDAL CHANGES AND AXIS OF ASTIGMATISM OF THE EYE

Patient I.D. No.

Name (in capital letters)

Age (in yrs)

Sex : (1=Male; 2=Female)

O.P. No.

Address:

Telephone No.

Inclusion Criteria: (1=Met; 2=Not met)

Informed consent: (1=Taken; 2=Not taken)

Chief Complaints (1=Yes; 2=No)

Complaint	RE	LE	BE
Diminution of vision			
Headache			
Watering			
Pain			
Redness			
Discharge			
Itching			
Ocular irritation			
Photophobia			
Floaters			
Diplopia			
Coloured halos			
Other			

Other complaints: (if present):

Past History (1=Yes; 2=No)

	RE	LE	BE
Intra-ocular Surgery			
Trauma			
Other			

Other past history (if present):

Medical History (1=Yes; 2=No)

Diabetes	
Hypertension	
Others	

Other medical history (if present):

Family History ☐ (1=Significant; 2=Insignificant)

If 1, specify:

Personal History(1=Sig…cant; 2=Insignificant)

If 1, specify:

General Physical Examination

Vitals

- Pulse (per min) ☐☐☐
- Blood Pressure ☐☐☐☐☐☐
  (systolic/diastolic)
- Temperature (1=Febrile; 2=Afebrile) ☐
- Respiratory Rate (per min) ☐☐☐

(1=Yes; 2=No)

Pallor	
Icterus	
Cyanosis	

Clubbing	
Lymphadenopathy	
Oedema	

Systemic Examination (1=Normal; 2=Abnormal)

C V SIf 2, specify	
R SIf 2, specify	
C N SIf 2, specify	
P / AIf 2, specify	

Ocular Examination

- Head posture ☐ (1=Erect; 2=Tilted)
- Facial symmetry ☐ (1=Symmetrical; 2=Asymmetrical)
- Visual axes ☐ (1=Parallel; 2=Deviated)
- Extra-ocular movements (1=Normal; 2=Restricted)
    - Uniocular RE ☐ LE ☐
    - Binocular ☐
- Vision

	RE	LE
Unaided		
Pin-hole		
Spectacles		

- Retinoscopy

RE	LE

- Refraction

	RE				LE			
	Sphere	Cylinder	Axis	Vision	Sphere	Cylinder	Axis	Vision
Distance								
Near								

- Anterior segment examination

	RE	LE
Adnexa (1=Normal; 2=Abnormal) If 2, specify		
Conjunctiva (1=Normal; 2=Abnormal) If 2, specify		
Cornea		

(1=Clear; 2=edematous; 3=other) If 3, specify		
Sclera (1=Normal; 2=Abnormal) If 2, specify		
Anterior chamber (1=Normal depth; 2=shallow; 3=deep)		
Iris (1=Normal; 2=Atrophic patches; 3=other) If 3, specify		

	RE	LE
Pupil • Size (1=normal; 2=constricted; 3=dilated) • Reactions: o Direct		

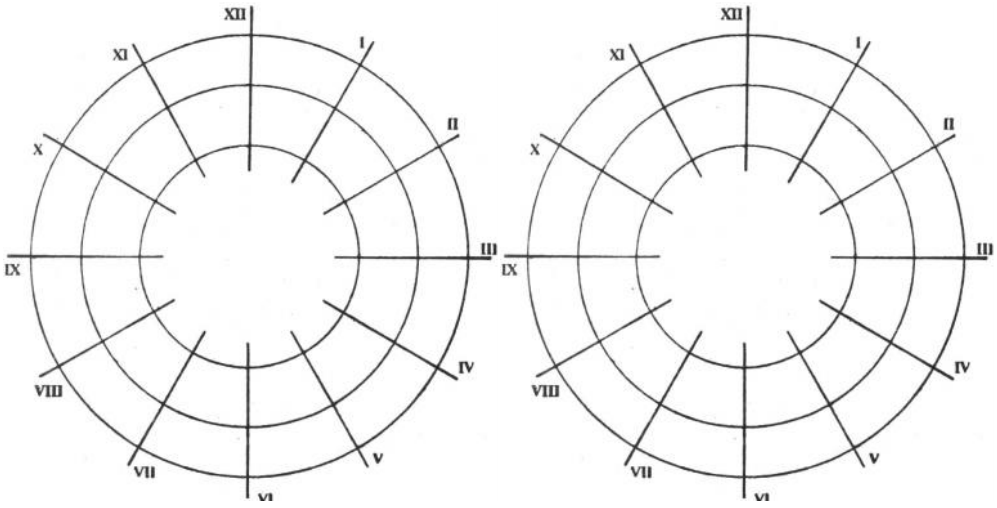
o Indirect (1=present; 2=absent; 3=sluggish)		
Lens (1=Clear; 2=Cataractous) (If 2: 1=immature; 2=mature; 3=hypermature)		

- Fundus

	RE	LE
Glow (1=Good; 2=Faint; 3=Absent)		
Media (1=Clear; 2=Hazy)		
Disc • Size (1=Normal; 2=small; 3=large) • Shape (1=Normal; 2=Abnormal) • Margins (1=Normal; 2=Abnormal) • Cup (1=Normal; 2=Abnormal)		

• CDR (1=0.2; 2=0.3; 3=0.4; 4=0.5; 5=0.6; 6=0.7; 7=0.8; 8=0.9; 9=1.0) • NRR (1=Normal; 2=Thin)		
Blood vessels (1=Normal; 2=Abnormal)		
Background (1=Normal; 2=Tessellated; 3=Other)		
Macula (1=Normal; 2=Abnormal)		

FUNDUS DIAGRAM



RIGHT EYE **LEFT EYE**

Angle of Axis of Crescent/Peripapillary Atrophy:

Investigations

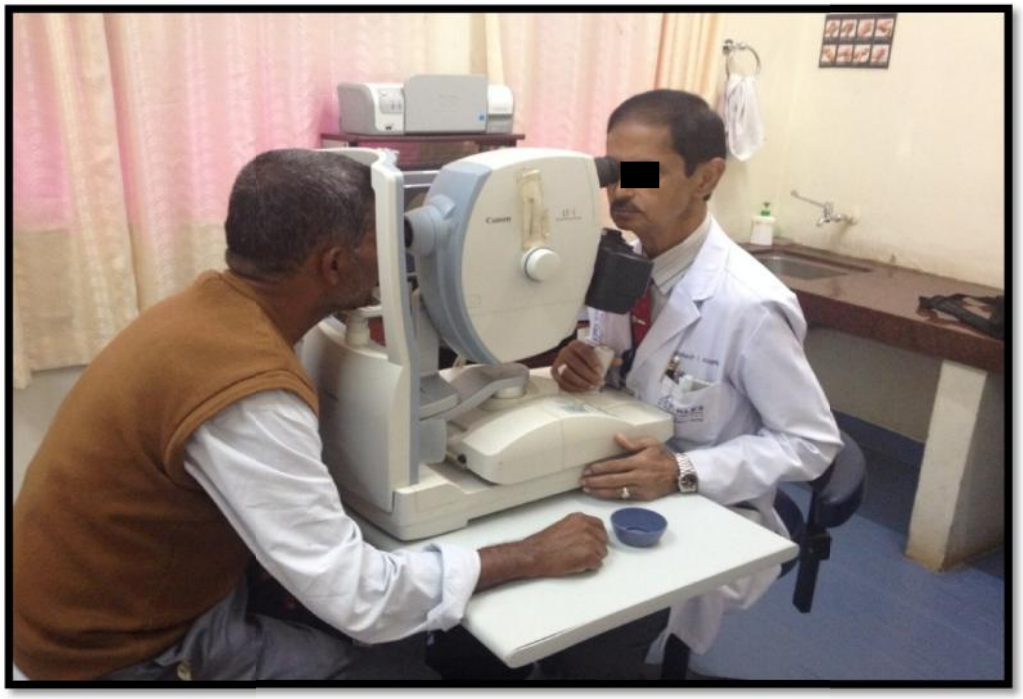
- A-scan Ultrasonography

Readings (mm)	RE	LE
K 1		
K 2		
Axial Length		
Average AC Depth		

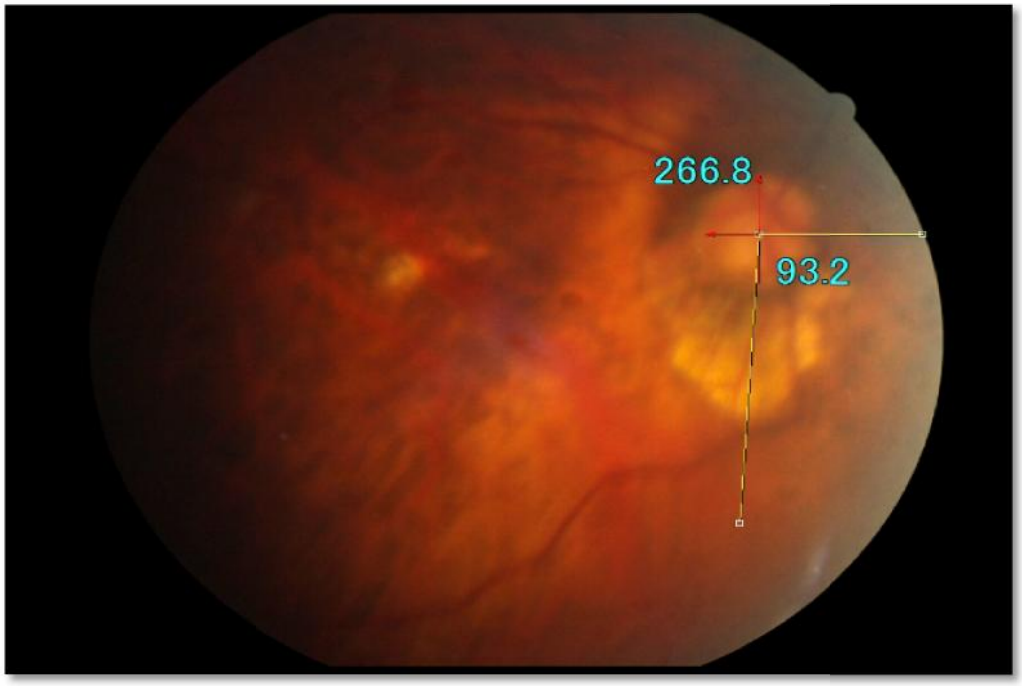
DIAGNOSIS:

ANNEXURE - III :PHOTOGRAPHS

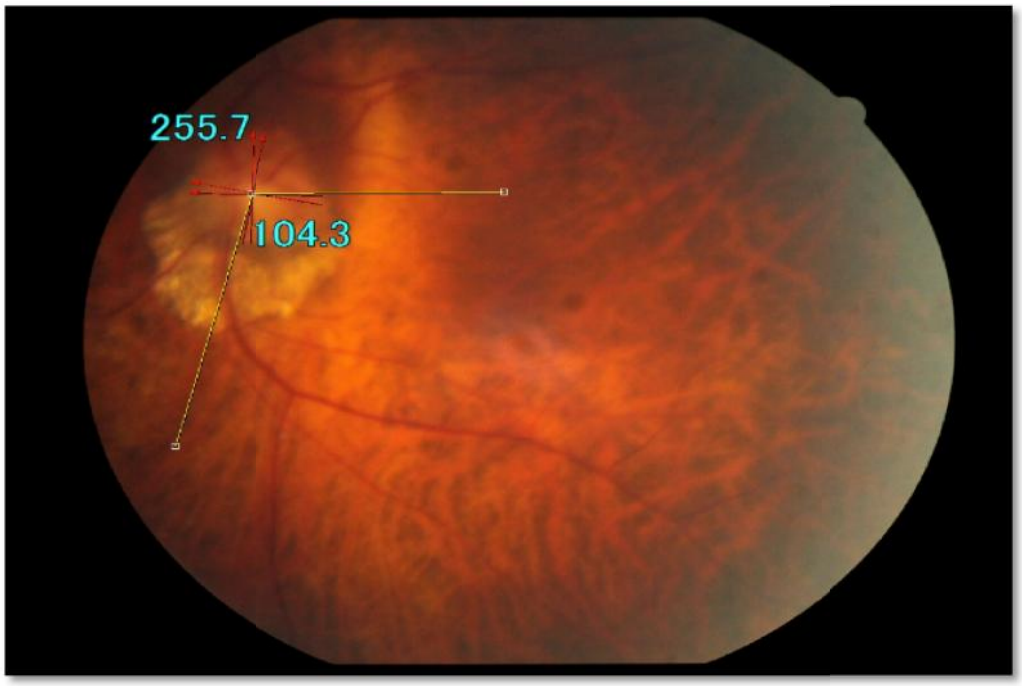
Photograph No.1: Patient undergoing Refraction



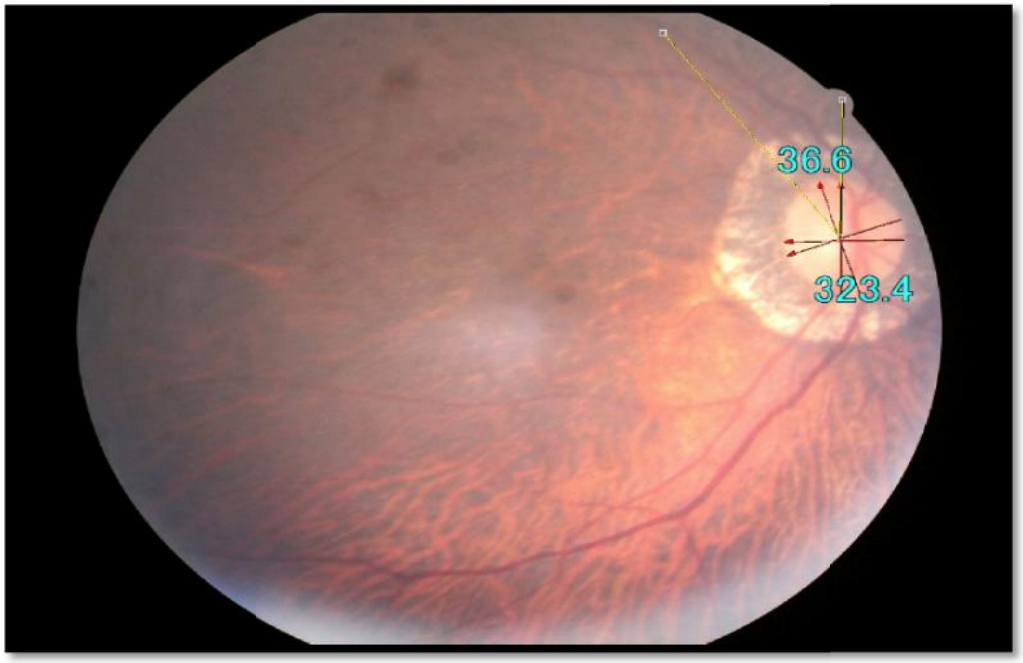
Photograph No.2:Patient undergoing Fundus Photography



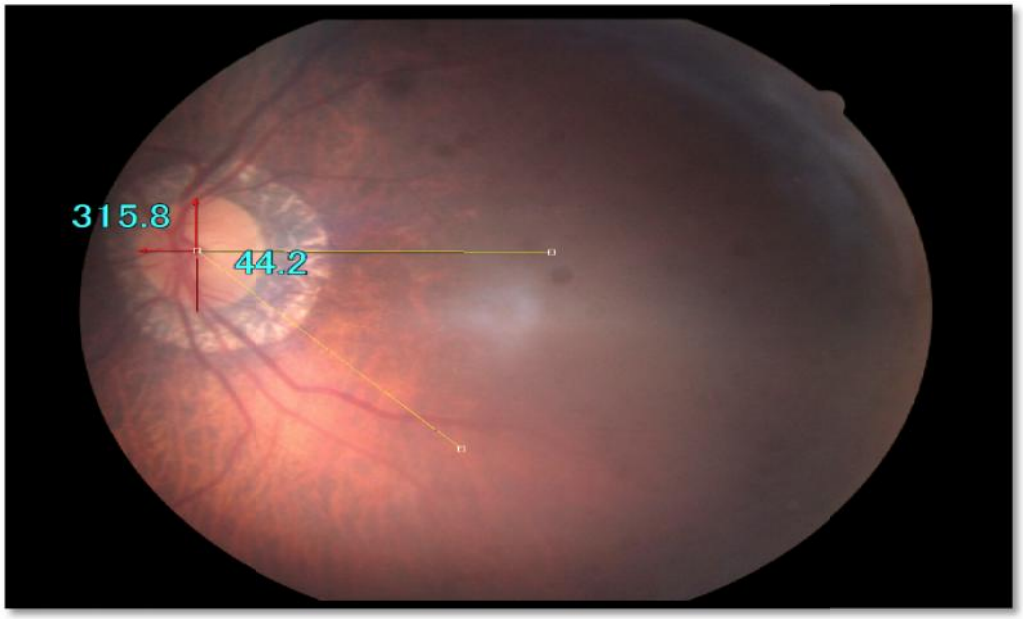
Photograph No.3: Fundus photograph showing measurement of angle of Inferior Crescent (Right Eye)



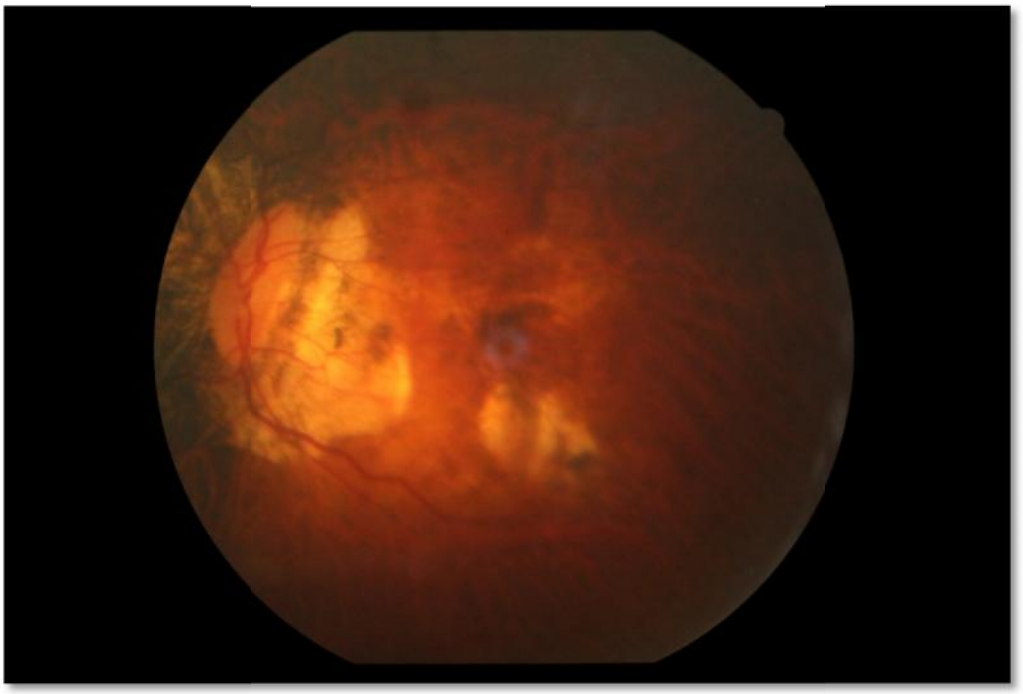
Photograph No.4: Fundus photograph showing measurement of angle of Inferior Crescent (Left Eye)



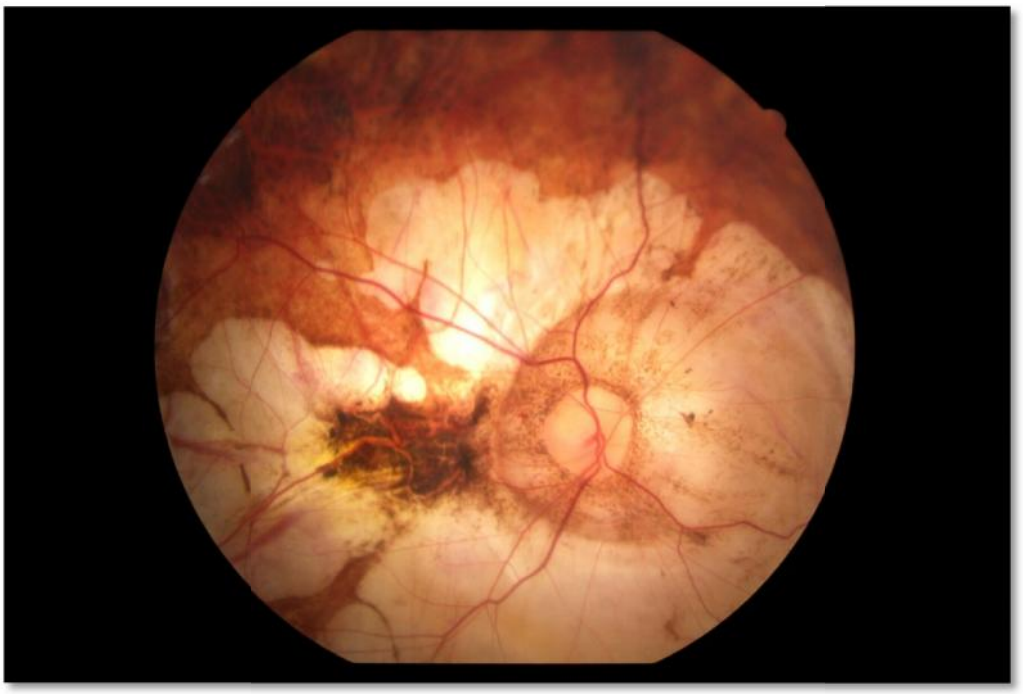
Photograph No.5: Fundus photograph showing measurement of angle of Peripapillary Atrophy (Right Eye)



Photograph No.6:Fundus photograph showing measurement of angle of Peripapillary Atrophy (Left Eye)



Photograph No.7:Fundus photograph showing Posterior Staphyloma



Photograph No.8: Fundus photograph showing Gyrate Atrophy

KEY TO MASTER CHART

O.P NO.	-	Out Patient Number
SPH. EQ. RE	-	Spherical equivalent of right eye
SPH. EQ. LE	-	Spherical equivalent of left eye
CYL. RE	-	Cylindrical power of right eye
CYL. LE	-	Cylindrical power of left eye
AST. AXIS RE	-	Astigmatic axis of right eye
AST. AXIS LE	-	Astigmatic axis of left eye
RE AXL	-	Right eye axial length
LE AXL	-	Left eye axial length
ACD RE	-	Anterior chamber depth right eye
ACD LE	-	Anterior chamber depth left eye
M	-	Male
F	-	Female
CF	-	Counting fingers
HMCF	-	Hand movements close to face
N	-	Normal
PPA	-	Peripapillary atrophy
TC	-	Temporal crescent
NC	-	Nasal crescent
IN	-	Inferior crescent
SC	-	Supertraction crescent
LD	-	Lattice degeneration
PS	-	Posterior staphyloma
LC	-	Lacquer cracks
ST	-	Snail track degeneration
NA	-	Not applicable

SR.NO	O.P NO.	AGE	SEX	VISUAL ACUITY RE	VISUAL ACUITY LE	SPH EQ RE	CYL. RE	AXIS RE	SPH EQ LE	CYL LE	AXIS LE	DISC RE	DISC LE	PERIPHERY RE	PERIPHERY LE	RE AXL	LE AXL	AC DEPTH RE	AC DEPTH LE	ANGLE RE	ANGLE LE
1	2385764	18	M	CF 3m	CF 3m	-7	-0.5	90	-7	-0.5	90	TC	NC	LD	N	25.04	25.23	3.44	3.25	120	80
2	2409528	20	M	6/60	6/36	-4	-3	60	-4.5	-2.5	150	N	PPA	N	N	24.32	24.27	3.93	4.13	NA	151.1
3	2415833	52	F	CF 3m	6/24	-6	-2	140	-7	-1.25	140	TC	N	PS	N	25	24.99	3.44	3.44	145	NA
4	2188630	51	M	CF 1/2m	6/24	-8	-2.5	140	-7	-2.5	80	PPA	PPA	ST	N	24.34	24.32	3.44	3.44	144.2	80.6
5	2464724	59	F	6/60	6/12	-5	-2.5	150	-4.5	-1.25	20	TC	PPA	PS	PS	25.04	25.23	3.44	3.25	150.8	19.4
6	1652501	68	M	6/24	6/24	-5	-1.5	100	-5.5	-1.25	70	TC	N	N	N	25	25	3.44	3.25	143.6	NA
7	2510413	22	M	6/18	6/18	-4.5	-0.75	90	-3.5	-2.5	90	IC	N	LD	N	24	24	3.25	3.25	90	NA
8	817058	5	M	CF 4m	6/36	-3.5	-0.25	90	-1	-0.25	30	PPA	N	N	N	25.04	25.23	3.93	4.13	40.8	NA
9	2526122	30	F	HMCF	CF 3m	-10	-3.5	30	-12	-2	30	N	NC	RD	LD	24.32	24.27	3.25	3.25	NA	30
10	2543314	60	F	CF 2m	CF 2m	-9	-2.75	74	-9	-2.5	108	PPA	PPA	PS	PS	23.9	24	3.44	3.25	170	8.7
11	2064937	67	M	CF 1/2m	6/60	-6	-2.25	90	-2	-2.5	90	PPA	PPA	LC	LC	22.93	23.08	3.36	3.36	65	136
12	2547827	34	F	CF 3m	CF 2m	-6	-1	110	-6	-0.75	70	TC	N	N	N	23.91	23.78	3.74	2.96	130	NA
13	531815	62	M	CF 2m	CF 2m	-5	-3	40	-5	-2.5	10	NC	N	N	N	26.08	26.07	3.88	3.52	21.1	NA
14	2562028	20	M	CF 1 m	6/60	-12	-2	175	-5.5	-0.75	60	NC	N	N	N	27.1	25.62	3.29	3.13	170	NA
15	2562544	65	M	CF 1 1/2m	CF 3m	-2	-0.5	28	-3	-0.5	30	TC	N	N	N	24.86	24.82	3.83	3.78	27.8	NA
16	2571745	60	F	CF 2m	CF 2m	-6	-0.5	50	-7	-2.5	90	PPA	PPA	N	N	24.32	24.27	3.44	3.25	112	96.3
17	2536050	70	M	CF 1/2m	6/36	-14	-2	20	-3	-1	90	PPA	TC	PS	N	25.73	23.97	4.2	4.06	18.9	21.2
18	2599298	60	F	CF 1/2m	6/12	-7	-3.5	145	-0.75	-0.5	180	TC	NC	PS	N	23.69	23.76	3.52	3.57	147.3	160
19	2611451	33	F	6/36	6/36	-7	-1.75	80	-7	-1	40	TC	TC	LD	N	24.24	23.86	3.51	3.17	125.3	40
20	2567310	23	F	CF 2m	CF 2m	-16	-0.5	180	-13	-0.5	130	TC	N	N	N	24.84	23.98	3.43	3.4	163.8	NA
21	2613241	14	F	CF 2m	CF 2m	-10	-2	170	-11	-2	160	TC	N	N	N	24.1	24.05	3	3.05	170	NA
22	2613240	25	F	CF 4m	6/36	-4	-1	120	-15	-1.25	10	TC	TC	N	N	25.05	24.98	3.53	3.53	118.9	7.4
23	2613139	80	M	6/60	6/60	-2	-1.5	170	-2.25	-1	30	NC	N	N	N	25.2	24.98	3.46	3.53	172	NA
24	2614742	60	M	CF 1 m	6/18	-6	-1.75	170	-0.75	-1.25	70	PPA	N	N	N	24.98	25.02	3.43	3.46	170	NA
25	2134657	45	F	CF 2m	CF 1m	-12	-3.5	145	-12	-2.5	30	TC	TC	N	PS	26.2	26.81	3.53	4.01	144.3	5

SR.NO	O.P NO.	AGE	SEX	VISUAL ACUITY RE	VISUAL ACUITY LE	SPH EQ RE	CYL. RE	AXIS RE	SPH EQ LE	CYL LE	AXIS LE	DISC RE	DISC LE	PERIPHERY RE	PERIPHERY LE	RE AXL	LE AXL	AC DEPTH RE	AC DEPTH LE	ANGLE RE	ANGLE LE
26	2484604	80	F	6/24	CF 3m	-4	-1	120	-5	-1	50	ST	TC	N	N	24.84	23.98	3.46	3.53	120	50
27	2013079	25	M	6/36	6/24	-6	-1.25	130	-5	-1.25	30	PPA	TC	N	LD	28.55	28.36	3.31	3.45	132.8	28.1
28	2621001	20	F	6/6	CF 1m	-0.25	-1.25	40	-9	-1.75	160	NC	NC	N	N	24	24.98	3.53	3.46	13.3	165.7
29	2638458	65	F	CFCF	CF 1/2	-14.75	-2.25	90	-17.25	-1.25	100	IC	IC	PS	N	24.93	24.12	1.87	3.5	93.2	104.3
30	2013802	33	M	6/18	6/24	-5	-1.25	145	-5	-2.5	90	TC	N	N	N	25.04	25.23	3.44	3.25	161.8	NA
31	2664288	60	M	CF 4m	6/60	-6	-2.25	170	-5.5	-2.5	30	TC	N	N	N	24.32	24.27	3.93	4.13	156	NA
32	2629232	56	F	CF 1m	CF 2m	-12	-1	140	-9	-1	40	TC	TC	N	N	23.78	24.08	2.76	3.02	140	65.9
33	2659518	60	F	6/36	CF 3m	-4	-1.5	90	-8	-4.5	118	N	TC	N	N	24.73	25.1	3.43	3.72	NA	15.2
34	2676530	12	F	CF 2m	CF 2m	-6	-1	130	-11	-1	180	PPA	PPA	N	LD	23.3	23.29	3.57	3.93	130	152.4
35	2684180	68	M	CF 3m	CF 2m	-4	-1.25	170	-7.5	-1.5	170	TC	TC	N	N	27.1	26.16	3.66	3.88	170.4	102.6
36	2688423	61	M	CF 2m	CF 2m	-5	-2	120	-5	-1	90	PPA	N	N	N	24.98	24.5	3.1	3.53	120	NA
37	2692489	19	M	6/36	6/60	-1.5	-1.25	90	-2.5	-1.5	90	TC	N	N	N	27.31	27.3	2.99	2.99	90	NA
38	269931	35	F	CF 1m	CF 2m	-18	-1.25	34	-4.5	-3.5	152	PPA	TC	PS	N	25.51	25.17	2.77	2.88	175.4	16.6
39	2706746	66	F	CF 1/2m	CF 1/2	-8	-2.25	130	-6	-1.25	130	TC	N	N	N	25.51	25.17	2.77	2.88	134	NA
40	2712192	55	F	CF 2m	CF 1 1/2	-6	-2.25	130	-5	-1.25	130	PPA	N	N	N	24.3	25	3.52	3.33	130	NA
41	2713537	11	M	CF 3m	CF 3m	-7	-1	90	-7	-1	90	TC	TC	N	N	25.98	26	4.12	4.14	156.2	16.8
42	2713541	6	M	CF 2m	CF 4	-10	-1.25	19	-7	-0.5	180	TC	TC	N	N	28.65	29.91	3.48	3.61	198	40
43	2713544	8	F	CF 2m	CF 2m	-8	-0.5	27	-8	-1.25	180	NC	TC	N	N	28.95	29.33	3.22	3.26	163.3	25.4
44	2742267	45	F	6/36	CF 1/2	-5	-2.25	145	-5	-1.25	145	PPA	TC	N	N	24.56	24.32	3.59	3.63	143.9	30
45	2742110	70	F	6/18	6/24	-4	-2.5	155	-7.5	-2.5	155	NC	TC	N	N	24.71	24.6	3.51	3.62	155.2	33.8
46	2742595	19	F	6/60	CF 2m	-10	-2	3	-7.5	-3.25	5	TC	TC	LD	LD	26.78	27.01	4.01	3.53	158.8	24.6
47	2751805	68	M	6/60	6/36	-1.5	-1.25	120	-12	-9	70	IC	N	N	N	24	23.65	3.53	3.46	85.6	NA
48	2762029	65	M	6/18	6/36	-2	-0.5	90	-2	-2	90	PPA	N	N	N	27.32	27.08	3.46	3.53	123.3	NA
49	2763107	65	F	CF 3m	CF 3m	-8	-2.5	90	-6	-2.5	80	TC	N	N	N	25.8	25.4	4.02	4.02	127.7	NA
50	2753419	80	M	CF 1m	CF 1m	-2.5	-5.0	90	-3	-1	40	PPA	PPA	N	N	25	25	4.02	4	122	44.2

SR.NO	O.P NO.	AGE	SEX	VISUAL ACUITY RE	VISUAL ACUITY LE	SPH EQ RE	CYL. RE	AXIS RE	SPH EQ LE	CYL LE	AXIS LE	DISC RE	DISC LE	PERIPHERY RE	PERIPHERY LE	RE AXL	LE AXL	AC DEPTH RE	AC DEPTH LE	ANGLE RE	ANGLE LE
51	2696115	70	F	CF 1m	CF 1m	-5	-5.0	40	-6	-2.5	50	N	PPA	N	PS	24.28	24.3	4.02	4.02	NA	52.9
52	2772405	72	M	CF 1m	CF 2m	-6	-2	60	-8.5	-1.25	108	NC	N	N	N	24.48	24.4	3.38	3.74	137.2	NA
53	2779593	20	M	CF 1 m	CF 1m	-12	-1	10	-14	-1.5	170	PPA	PPA	N	LD	24.98	24.84	4.2	3.95	19.6	36.5
54	2778794	69	F	CF 1 m	CF 2m	-20	-6	130	-20	-4	40	PPA	TC	ST	PS	26	26	4.02	4	130	41
55	2789573	76	M	CF 2m	CF 2m	-6	-0.75	90	-7	-0.5	130	N	NC	N	N	27.6	26.54	3.35	3.26	NA	130
56	2797866	73	M	CF 4m	CF 1 1/2	-5	-2.25	155	-6	-2.5		TC	N	N	N	25	25	3.35	3.26	155	NA
57	2463154	64	M	CF 2m	CF 2m	-8	-3	170	-7.5	-1.25	160	TC	TC	N	N	27.29	26.57	3.78	3.69	173	160
58	2805923	53	M	CF 3m	CF 2m	-10	-2.25	65	-8	-0.5	80	IC	IC	N	LD	28.97	30.93	3.79	3.75	65	80.6
59	2814573	14	M	CF 2m	CF 1 1/2	-14	-0.25	170	-11	-2.5	35	TC	TC	LC	LC	25.98	26	4.12	4.14	170.6	34.8
60	2818731	58	F	CF 2m	CF 1 1/2	-6	-3	90	-6	-1.25	108	PPA	IC	N	N	25.98	26	4.12	4.14	90	107
61	2820636	28	F	6/6	6/36	plano	plano	plano	-4	-1	160	N	NC	N	N	24.52	23.56	3.54	3.47	NA	167.7
62	2823580	70	F	6/24	6/24	-3	-0.25	90	-2.5	-0.5	90	PPA	PPA	N	N	25.5	25.5	4.2	4.2	112.7	54.3
63	2143536	60	M	CF 2m	6/60	-4.5	-2.25	160	-6	-1.5	70	PPA	N	LC	N	24.52	23.56	3.54	3.47	162	NA
64	2515757	49	M	6/24	6/24	-4	-1	140	-6	-2.5	90	TC	N	N	N	26	25.5	4.2	4.2	140	NA