

A CLINICAL EVALUATION OF PATHOLOGICAL MYOPIA

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Certificate

This is to certify that the dissertation entitled “A CLINICAL STUDY OF PATHOLOGICAL MYOPIA” is the bonafide original work of Dr. KUMARAVEL.T. in partial fulfilment of the requirements for M.S. Branch – III (Ophthalmology) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in April 2013

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Declaration

I, Dr. KUMARVEL.T., solemnly declare that dissertation titled, “A CLINICAL STUDY OF PATHOLOGICAL MYOPIA” is a bonafide work done by me at Govt. Thanjavur Medical College & Hospital during 2011-2013 under the expert guidance and supervision of Prof. P.S.GIRIDHAR, M.S.,D.O. Head of the Department, Department of Ophthalmology.

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HISTORY

Myopia is well known even since ancient period. The word Myopia is a Greek word, which means “to shut” or “to close the eye”. The original meaning of the word Myopia was meant to describe the condition where the patient attempts to see clearly by partially shutting or closing their eyes.

Aristotle was the first person who was thought to have distinguished between myopia and hypermetropia. Gallen(138-201 AD) who belongs to Rome , used the word “myopia”. It is known by other terminologies like “near – sightedness” or “short sightedness”

Initially the term hypometropia was used to differentiate from hypermetropia, an opposite condition(2). But the term “myopia” still holds good. Environmental factors were first blamed to cause myopia.

Cohn was the first to describe the “environmental theory of Myopia.

Later the “genetic theory” was also proposed. The two theories, genetic and environmental were together referred to as “Nature vs Nurture theory”

DEFINITION

Myopia or short sight is a type of refractive error in which parallel rays of light coming from infinity are focussed in front of the retina when the accommodation is at rest.(2)

CLASSIFICATION (17)

Various classification systems have been described for myopia

1. Clinical classification:

Simple myopia, nocturnal myopia, pseudo myopia, degenerative myopia, and induced (acquired) myopia.

2 Other systems classify myopia by degree

(i.e., low, medium, or high) or

3. by age of onset

(i.e., congenital, youth onset, early adult-onset, late adult-onset)

Classification Systems for Myopia

Type of Classification	Classes of Myopia
Clinical entity	Simple myopia Nocturnal myopia Pseudo myopia Degenerative myopia Induced myopia
Degree	Low myopia (<3.00 D) Medium myopia (3.00 D-6.00D) High myopia(<6.00D)
Age of onset	Congenital myopia (present at birth and persisting through infancy) Youth-onset myopia (<20 years of age) Early adult-onset myopia (2-40 years of age) Late adult-onset myopia (>40years of age)

CLINICAL VARIETIES OF MYOPIA

1. Congenital myopia
2. simple or developmental myopia
3. pathological or degenerative myopia
4. acquired myopia

CONGENITAL MYOPIA

Congenital myopia is present since birth, however usually diagnosed by the age of 2-3 years. It is seen more frequently in children born prematurely or with various birth defects such as marfans syndrome or homocystinuria. Usually the error is about 8 – 10 D dioptries.

In most of the them the error is unilateral and may be associated with convergent squint. The condition is not progressive and full cycloplegic refractive error including astigmatic correction should be prescribed.

SIMPLE MYOPIA

Simple or developmental myopia is the commonest variety. It is considered as a physiological error not associated with any disease of the eye. It results from normal biological variations in the development of eye. Inheritance is considered to be autosomal dominant. However there are number of reports which claim recessive mode of inheritance is common.(5)

Simple myopia begins between 7 and 10 years of age and may increase during the years of growth until stabilising round mid teens. Refractive error is usually less than 6 D.

DEGENERATIVE MYOPIA

Pathological/degenerative/progressive myopia as the name indicates, is a rapidly progressive condition resulting in high myopia during early adult life which is usually associated with degenerative changes in the eye, particularly in the posterior segment of globe.

It is usually but not invariably results from a rapid axial growth of the eyeball which is outside the normal biological variations of development.

Duke elder(1) says 'myopes should be classified not by retinoscopy but by ophthalmoscopy'.

Low myopes and indeed eyes with normal axial length may show degenerative changes characteristic of myopia while over -17 dioptré may show no abnormal changes.

From medical point of view degenerative myopia is the most important of all refractive errors for it is relatively common, leading frequently to much visual disability and not infrequently to eventual blindness. Its economic and social implications are therefore considerable.

TYPES OF ACQUIRED MYOPIA:

INDEX MYOPIA:

This occurs in condition such as nuclear sclerosis, incipient stage of cortical cataract and in diabetes.

CURVATURE MYOPIA:

Conditions where there is increase in corneal curvature as in keratoconus may produce curvature myopia.

POSITIONAL MYOPIA:

This may occur in conditions producing anterior subluxation of lens.

CONSECUTIVE MYOPIA:

The condition follows overcorrection of hypermetropia or wrong implantation of intraocular lens.

PSEUDOMYOPIA:

So called artificial myopia may be produced in excessive accommodation or spasm of accommodation.

SPACE MYOPIA:

The condition occurs when the individual has no stimulation for distant fixation.(5)

DRUG INDUCED MYOPIA:

Seen in patients using cholinergic drugs such as pilocarpine.

PREVALENCE

The prevalence of myopia is difficult to access from literature since the available data deals with all types of short sight. Myopes of over-6 dioptre represents 27-32% of myopic population and of over -8 dioptre 6-18%. Although there are no studies regarding prevalence of pathological myopia in India, according to national program for control of blindness and world health organisation (NPCB-WHO) study(8) refractive errors are the second leading cause of low vision and blindness counting for 18.87% of low vision and 7.35% of both low vision and blindness. This is second most common cause of blindness only secondary to cataract.

SEX

Sex appears to have an influence on the incidence. Although males and females are equally affected in lower degrees, in higher degrees females are more prone to degenerative changes

RACE

High degree with degenerative changes is more common in Chinese Japanese Arabs and Jews. Uncommon among Negroes.

ETIOLOGY

As already said, degenerative myopia is usually but not invariably associated with increased axial length of the eyeball and degenerative changes in the posterior segment of eye, various theories has been put forward to explain the facts.

ROLE OF HEREDITY

Genetic factors play a major role in progressive myopia. in one study hereditary element was present in 32.89%.father alone affected in 12.43% mother alone in 17.36% and both parent in 3.1% neither present in 67.09%.(1)

It is presumed the heredity like growth of retina is the determinant in the development of myopia. The sclera due to its distensibility follows retinal growth but the choroid undergoes degeneration due to stretching, which in turn causes degeneration of retina. The classical view is that due to mechanical stretching of the posterior part of the globe causing to the weakness of sclera. Inherited weakness of the sclera must also be postulated.

Yet not known whether mesoderm or ectoderm is the primary fault(5)

.Mesoderm theory-

This theory says that there is disparity between sclera and extraocular muscles. When the muscles cease to grow they exercise a strain on the sclera. Arrest of the development of sclera at the fifth month might result in thinness and weakening of this structure.

Neuroectodermal theory-

Each coat of the retina, choroids and the sclera has its own growth potential. In myopia overgrowth of retina is usually genetically determined. As the retina enlarges it pushes towards the posterior pole, the sclera, adapting to this growth becomes thinned.

The choroid rendered susceptible to stretching becomes atrophied and the retina which depends on the choroid for its nutrition degenerates secondarily.

Another theory considers ciliary muscle as a pivotal mechanism in controlling ocular growth by acting as a counter force to excessive ocular expansion. According to this theory, underdevelopment of ciliary muscle leads to excessive axial growth due to a decrease in the inhibitory activity of the ciliary body on ocular growth.

Inheritance in lower degrees of myopia is autosomal dominant. In higher degrees various forms of inheritance exists of which recessive form appears to be common. Autosomal dominant type associated with nystagmus and sex inheritance has also been reported.

In a study by institute of genetics, Fudan University, china it was found that HLA-DQB1 gene was altered in pathological myopia

ROLE OF GENERAL GROWTH PROCESS

The role of general growth process though minor cannot be denied in the progress of myopia. Lengthening of the posterior segment of globe commences during the period of active growth and probably ends with the termination of active growth.(4) Therefore, factors such as nutritional deficiency, debilitating illness and endocrine disturbances which affect

general growth process may also have some influence on the progress of myopia.

In myopia which develops after the period of active growth, environmental factors probably exert some influence.

Scleral weakness attributed to dietary deficiency of calcium, vitamin D and proteins. Endocrine influence on myopia is mainly attributed to pituitary and partly to thyroid. This is because these gland functions reaches maximum around puberty, so is progressive myopia.(1)

General diseases like tuberculosis and syphilis probably increases the weakness of sclera. Thus logical treatment of myopia by improving the general health is a very good thing

NEAR WORK AND MYOPIA

There is a strong belief that excessive near work aggravates myopia. The fact that progressive myopia starts around 7 – 10 years , which is also the schooling period also necessitates more near work ,adds more to the confusion whether excessive near work aggravates myopia or not.

Prevalence studies show while myopia is more common among literate students of Japan, it is as common in illiterate Jews. Also the prevalence is equal among both educated and illiterate Negroes.

The influence of close work is secondary and incidental in the aetiology of the condition which is essentially predetermined.(4) As regards the development of myopia to processes of growth, it is said that the lengthening of the posterior segment of the eye commences only during the period of active growth. The eye and the brain show precocious growth at the age of 4 years; the brain is 84% and the eye 78% and the rest of the body is 21%. After this, both the eye and the brain increase slowly while the body grows more rapidly. However, when axial myopia continues to progress, it is interpreted as a precocious growth which has failed to get arrested.

PATHOLOGICAL CHANGES IN DEGENERATIVE MYOPIA:

The gross appearance of highly myopic eye is characteristic both in size and shape. Instead of being globular it is egg shaped.

It is enlarged but the elongation of eye is almost entirely confined to the posterior pole.

Initially thought to be inflammatory condition, it is now established all changes characteristic of myopia are those of degenerative process.

SCLERA:

Electron microscopic studies of the sclera in high myopes show thinning of meridional bundles with separation and splaying of cross bundles. In

classical view, the cause of thinning sclera was due to mechanical stretching but atrophic element does co exist.

CHOROID:

The choroidal changes are essentially atrophic in nature (myopic choroidal degeneration). The chief change is a generalized thinning of the choroidal coat which becomes very attenuated and may even disappear completely in larger area.

First change is usually a disappearance of lumina of small vessels, which eventually appear as solid sclerotic white threads.

Vessels contain leucocytes which are not normally seen. As the degeneration continues the choriocapillaries fails completely and the larger vessels are alone left. Finally they too become obliterated(1)

Along with vascular changes the chromatophores lose their pigment, disintegrate, and last of all the elastic elements disappear.

Slits occur in the elastic lamina, usually have clefts which may form branching or reticular figures resembling cracks in lacunae.

RETINA:

Atrophy involving retinal pigment epithelium occurs before atrophy of rods and cones and chorioretinal fusion occurs. The regular hexagonal pattern of normal cells is replaced by irregular pattern of misshapen cells with much of the pigment lying extracellularly.

In association with dehiscence in Bruch's membrane the pigment proliferates to form branched pigment figures or large conglomerate masses. It is a localized proliferation of this type which forms the Forster Fuchs black spots in the macula (possibly associated with choroidal hemorrhage). (1)

Peripheral cystoid degeneration (Blessing cyst) more characteristic of senile changes is also seen.

OPTIC DISC:

The optic disc is large. Myopic crescent(Conus Myopicus) is seen.

The nerve fibres usually transverse the disc in an obliquely nasal direction. The choroid terminates from the margin of the disc and is completely or partially absent in the area of the crescent. the outer retinal layers and the pigimentary epithelium may be absent in the area of crescent and only the inner layer continue.

Regarding crescent, in the classical view it is caused simply by stretching, the choroids being dragged back into the ectasia into the posterior pole of the eye. Conversely, it is a result of an excessive response of the sclera to the stimulation of retinal growth. Alternatively, it may be due to localized atrophy.

Super traction of the retina on the nasal side of the disc may reach up to the middle of the disc before the nerve fibres bend sharply backwards into the nerve.

Sometimes inverse crescent (inverse myopia) does occur where the myopic crescent is situated on the nasal side and super traction on the temporal side.

Although many ocular and systemic conditions are associated with pathological; myopia, certain entities such as retinal venous thrombosis, hypertensive and diabetic retinopathy are rare. Arterial occlusions, papilloedema are also rare, while closed angle glaucoma is an exception. In case of unilateral myopia the pathological changes may be confined to the normal eye alone.

SYMPTOMS OF DEGENERATIVE MYOPIA(3)

- Decreased visual acuity. this may greatly vary from 6/9 to loss of perception of light
- muscae volitantes .this may be really annoying to the patients.
- Defective dark adaptation
- Colour vision defects –particularly for blue colour
- Night blindness
- Visual field defects

Visual field defects

Visual defects in degenerative myopia is divided in to typical and atypical

Typical visual field defects

Enlargement of blind spot.

Loss in the superior temporal quadrant of peripheral field.

Atypical visual field

Centrocaecal scotoma

Hemianopia

Nasal scotoma

Annular scotoma

Tubular vision

FUNDUS CHANGES:

Evaluation of fundus is the most reliable method to differentiate simple or physiological myopia from pathological or degenerative myopia, which determines the prognosis for vision.

Examination by direct ophthalmoscopy is of limited value and both stereoscopic examination with +78D or +90D lens and indirect ophthalmoscopy after full dilatation is necessary in these patients.

Ophthalmoscopically four signs are indicative of excess axial elongation of eyeball (18)

1. crescent formation
2. supertraction
3. tessellation with pallor
4. posterior staphyloma

posterior staphyloma is path gnomonic of pathological myopia and was first described by Scarpa.

MYOPIC CRESCENT:

Myopic crescent formation occurs as a result of disparity in area between sclera and retinal pigment epithelium- choriocapillaries complex. The most frequent location of crescent is temporally but may be annular, inferior or superior.

Crescent may be of three types,

1. scleral crescent which is white in color
2. choroidal crescent which has a mottled appearance
3. mixed crescent

In rare cases, crescent may be seen even on the nasal side which is called as inverse crescent (inverse myopia). Supertraction result from dragging of retina nasally on the surface of the optic nerve. This gives rise to a light reflex which is best seen by direct ophthalmoscope. They appear as thin streaks with concavity towards disc called Weiss Streaks. With advancing myopia these streaks disappear.

STAPHYLOMA:

Posterior staphyloma is pathognomonic of pathological myopia.(1) They occur at the site of maximal scleral thinning and weakening (posterior pole). At the staphyloma margin the retinal vessels make a sharp bend and within the staphyloma they appear straight. Staphyloma is best detected by indirect ophthalmoscopy and their prevalence increase with the increase in the axial diameter of globe.

CHORIORETINAL CHANGES:

Fundus appears tessellated or tigroid because of the degenerations of the retinal pigment epithelium. In the early stages, Lacquer Cracks, retinal hemorrhages and small focal areas of chorioretinal atrophy characterize chorioretinal changes. Lacquer cracks are seen as yellow white irregular lines in the posterior pole. They are usually multiple and horizontally oriented. They also form a reticular pattern:. Lacquer cracks are fissure in the retinal pigment epithelium – choriocapillary complex and are due to mechanical tear.

Focal areas of chorioretinal atrophy appear as white to yellow round lesions with or without pigment clumping. Initially, discrete, in the later stages they become confluent. The atrophic changes are probably due to vascular occlusion and abiotropic degeneration.⁹¹⁰ At the macula, earliest change is unusual degree of hyperpigmentation. Macular holes are more frequent in degenerative myopes. Associated hemorrhage in the macular region as such or a hyperplasia of retinal pigment epithelial cells or both may give rise to a dark spot called Forster Fuch's spot. Later they appear gray in color when retinal pigment epithelial detachment occurs.

PERIPHERAL FUNDUS CHANGES:

Four types of peripheral fundus abnormalities are found to be associated with axial elongation of the eye. They are

1. white without pressure
2. lattice degeneration
3. pigmentary degeneration
4. Paving stone degeneration.

In summary, the fundus changes in degenerative myopia(16) is as follows,

1. Myopic crescent-temporal crescent consisting of area of bare sclera surrounded by a crescent in which the choroidal structure is visible
2. Super traction on the nasal side of disc
3. Tessellated (trigoid) appearance – due to diffuse attenuation of RPE with the visibility of large choroidal vessels.
4. Focal chorioretinal atrophy
5. Lacquer cracks – rupture in RPE-choriocapillaries complex characterized by fine,irregular,yellowlines often branching and criss-crossing
6. Lattice degeneration
7. Sub retinal coin shaped haemorrhages
8. Fuchs spot – raised circular pigmented lesion at macula developing after a Sub retinal haemorrhage has absorbed.
9. Posterior staphyloma

10. Macular hole
11. Rhegmatogenous retinal detachment
12. Choroidal neovascularisation
13. Foveal retinoschisis

RETINAL DETACHMENT IN PATHOLOGICAL MYOPIA:

A dangerous complication of degenerative myopia is the development of rhegmatogenous retinal detachment, where the incidence in the general population is 0.005 to 0.01%, the risk of retinal detachment increases four fold in subjects with spherical equivalent refractive error of -1D to -3D and ten fold in patients with -10D. Overall incidence of retinal detachment in myopia varies from 8 to 32%.

The risk factors for retinal detachment in degenerative myopia are

1. vitreous liquefaction
2. posterior vitreous detachment
3. peripheral retinal degeneration, especially lattice degeneration.
4. retinal break
5. Macular hole.

Macular holes are more frequent in myopic eyes with retinal detachment and before considering them as a cause of retinal detachment, peripheral retinal detachment should be ruled out.

CHOROIDAL NEOVASCULARISATION IN DEGENERATIVE MYOPIA:

Choroidal neovascularisation is a major cause of vision loss in pathological myopia. CNV may take origin from lacquer cracks. The prevalence of choroidal neovascularisation is about 5-10% in degenerative myopia. The subfoveal location is quite frequent, accounting for 58 -74% of cases.

Myopic CNV is generally small, <1 disc area, flat, greyish, with hyper pigmented margins. Most myopic CNVs are type II, located in the space between sensory retina and the retinal pigment epithelium

In CNVs due to age related macular degeneration, it is mainly of type I, which is situated in the subretinal pigment epithelial space.

Angiography in CNV:

Fundus fluorescein angiography may demonstrate abnormally slow choroidal and retinal blood flow in myopic patients. It is helpful in identifying and locating the site of choroidal neovascularisation. Characteristic pattern of myopic CNV is an early hyperfluorescence with a little to moderate leakage in late phase. (3)

Lacquer cracks can be detected early by FFA and ICG which appear as hyperfluorescence in FFA and hypofluorescence in ICG.

GLAUCOMA AND MYOPIA

There is definitely an increased incidence of open angle glaucoma in myopic individuals and myopia is considered as an important risk factor for open angle glaucoma.

The prevalence of glaucoma is 3% in eyes with axial length less than 26.5mm 11% in eyes with axial length between 26.5 and 33.5 mm, and 28% in eyes with axial length >33.5 mm.(6)

The clinical importance of the association lies in the fact that the glaucoma is usually of an insidious type without high tension and therefore readily missed by indentation tonometry.

The importance of using applanation tonometry is important owing to the low ocular rigidity.

Another fact in the concept of normal or low tension glaucoma is that individuals with normal or low tension actually have a higher value of intraocular pressure and is falsely measured due to low ocular rigidity as in myopia.

Previously it was thought that increased axial length in degenerative myopia is due to increased intraocular pressure, which determines or accentuates myopia. Also pathological myopia and glaucoma are of the same degenerative process. It was thought that glaucoma is sequelae to a generalised atrophy of choroids.

Not only the measurement of intraocular pressure makes difficult in the diagnosis of glaucoma, the appearance of disc and fundus adds to the problem. Myopic eyes have abnormal disc and their diagnosis represents special problem in the management of glaucoma.

The distance between the level of lamina cribrosa and the level of retina is much less when compared to myopic eyes. The average value is 0.7mm whereas in myopics it is 0.2-0.5mm. therefore a completely cupped disc in myopia will have only half the depth of the usual

glaucomatous cup. Such shallow excavation is difficult to appreciate clinically.

Also incidence of pigmentary glaucoma is more common among young myopic males.

OCULAR AND SYSTEMIC ASSOCIATIONS OF PATHOLOGICAL MYOPIA(7)

OCULAR ASSOCIATIONS

Retinopathy of prematurity

Congenital glaucoma

Albinism

Congenital stationary night blindness

Ectopia lentis

Retinitis pigmentosa

Wagner's syndrome

SYSTEMIC ASSOCIATIONS

Marfans syndrome

Ehlers danlos syndrome

Downs syndrome

Alport's syndrome

Albinism

Congenital rubella

De Lange's syndrome

Foetal alcohol syndrome

Gyrate atrophy-hyper ornithinemia

Laurence Moon beidel Bardet syndrome

Stickler syndrome

Pierre robin syndrome.

DIFFERENTIAL DIAGNOSIS:

RETINITIS PIGMENTOSA

A very careful history and a meticulous clinical examination will have no difficulty in diagnosing myopia.,

Although patients with retinitis pigmentosa are frequently myopic, show secondary cataract and vitreous liquefaction, can develop macular degeneration and have peripheral visual field defects. These are easily differentiated by other fundus changes.

OCULAR HISTOPLASMOSIS

Peripapillary atrophic changes, retinal pigment epithelial punched out defects and macular neovascularisations are also seen in Ocular Histoplasmosis Syndrome.

ARMD:

May develop CNV and a similar macular appearance to high myopia, but typically drusen are present and typically myopic disc features are absent.

TILTED DISC:

Anomalous disc with scleral crescent inferonasally and an irregular vascular pattern as the vessels emerge from the disc (Situs Inverses) Is associated with areas fundus ectasia in the direction of tilt. Most patients have myopia and astigmatism but no chorioretinal degeneration or lacquer cracks.

TOXOPLASMOSIS:

A well circumscribed chorioretinal scar that does not typically develop CNV. Active disease show retinitis and vitritis which are absent in high myopia.

GYRATE ATROPHY:

The condition is Rare. Well demarcated multiple areas of chorioretinal atrophy beginning in the mid periphery in childhood. Later they coalesce to involve a larger portion. Blood levels of ornithine are increased. Patients are often high myopic.

INVESTIGATIVE WORK –UP(15)

1. Visual acuity measurement
2. IOP measurement by applanation tonometry.(indentation tonometry like Schiötz may underestimate IOP in high myopes.
3. Refractive error estimation
4. Slit lamp examination and fundus stereoscopic examination with 78D or 90D lens of the macula to identify CNV which may appear as grey or green lesion beneath the retina.
5. Detailed evaluation of fundus periphery with indirect ophthalmoscopy to search for retinal breaks or detachment. Care should be taken in doing a scleral depression over a staphyloma.
6. FFA and ICG in suspected CNV patients and in those where lacquer cracks transverse the macula
7. Ocular coherence tomography when a macular detachment is suspected

MANAGEMENT:

Degenerative Myopia as such cannot be prevented by any known treatment. It is advisable to forewarn a couple with high myopia about a strong possibility to be affected. At present, treatment is focused on controlling the possible complications related to the progression of degenerative myopia, with special attention to choroidal neovascularisation and retinal detachment.

A periodic examination is always indicated in all these patients

.

Management of degenerative myopia should run along the following general guidelines.(3)

1. correction of the refractive error
2. early identification and treatment of the complications so as to prevent the patient from becoming virtually blind
3. building up of adequate visual hygiene and general health and visual rehabilitation in case of low vision patients.

REFRACTIVE CORRECTION:**SPECTACLE CORRECTION:**

Degenerative myopic patients usually present with greater than -6 D. Spectacles in such patients are cumbersome to wear because of the appearance and weight of the spectacles. Also the visual fields are grossly restricted.

CONTACT LENS CORRECTION:

Contact lens of choice in high myopia is rigid gas permeable lenses. Other lenses like poly methyl methacrylate and daily wear soft contact lenses may be used. Optical demands of high myopic correction requires thick lens edge. Side effect related to thick lens edge is neovascularisation. Rigid gas permeable lenses are the lenses of first choice because the complication rate due to reduced oxygen transmission can be minimized.

With the invent of newer softer contact lenses like daily wear, monthly wear disposable lenses, contact lenses make definitely a good alternative to spectacles.

Orthokeratology:

Otherwise called corneal refractive therapy refers to overnight use of gas permeable contact lens to temporarily reduce low degrees of myopia. Corneal flattening results from redistribution of corneal epithelium(14).

REFRACTIVE SURGERY

Keratorefractive surgeries may be helpful only to correct the refractive component of the degenerative myopia but does not arrest the degenerative changes.

Refractive surgery includes any procedure done to alter the refractive condition of the eye to improve uncorrected refractive error. The history dates back to nineteenth century when both contact lenses and laser were not available. With the discovery of contact lenses in 1950's refractive surgery became secondary interest due to simplicity and relative safety of contact lenses. Interest in refractive surgeries renewed in 1970 because occupations which demanded good vision without glasses and contact lenses like pilots and athletes sought refractive surgeries.

PRINCIPLES OF REFRACTIVE SURGERIES:

The anterior surface of cornea is responsible for 60 – 70% of optical power of eye. This significant contribution makes it the most operated part upon the eye in most refractive surgeries. Myopia can be corrected by making the central cornea more flat. Phakic intraocular lenses change the refractive state by changing the refractive media of the eye. Surgical techniques can be classified into incisional, thermal, lamellar and intraocular.

Incisional methods include procedures such as radial keratotomy.

Thermal based refractive surgeries use heat to shrink the collagen in the corneal stroma. The latest technique uses Holmium: Yag to produce the shrinkage.

Lamellar procedures alter directly the shape of cornea by ablation or placement of pre designed corneal lenticules in the cornea. These include keratomileusis, keratophakia, epikeratoplasty, photorefractive keratectomy and intrastromal photoablation. Among these procedures laser assisted in situ keratomileusis (LASIK) is the most popular.

INCISIONAL CORNEAL SURGERIES:

Since 1980s incisional corneal surgeries have had periods of adoption, refinement, and abandonment. Incisional surgery for myopia has been replaced by laser procedures. The role of incisional surgeries reserved for correcting astigmatism after cataract surgery (limbal relaxing incision) and after penetrating keratoplasty(arcuate keratotomy). Contribution to incisional corneal surgeries were made by Sato of japan and Fyodorov of Russia. Sato observed central corneal flattening and improvement of vision after healing of spontaneous rupture of descemets membrane in keratoconus patients. Fyodorov established that the central corneal clear zone was inversely related to the amount of refractive correction.

In radial keratotomy, radial peripheral corneal incision are made either posterior (Japanese technique) or anteriorly (Russian technique). Upon healing of the incision, flattening of the incision occurs.(14)

Radial keratotomy was used to treat patients with refractive error of -1 to -4 D of myopia and its role in pathological myopia is insignificant.

ONLAY AND INLAY TECHNIQUES:

In these techniques, the refractive error is corrected by preformed tissue or synthetic material onto or into the cornea. Corneal ring segments are under investigation to treat ectactic condition such as keratoconus or ectasia after refractive surgeries. As for as now laser procedures replaced all of the older procedures.

PHOTOABLATIVE TECHNIQUES:

The Argon fluoride Excimer Laser reduces the refractive error by ablating the anterior corneal stroma to a new radius of curvature.

There are three major refractive surgical techniques that employ Excimer Laser ablation(14)

1. photorefractive keratectomy(PRK), where the epithelium is derided
2. LASEK , where the epithelium is preserved as a flap

3. LASIK. here the Excimer laser ablation is performed under a lamellar flap made with a microkeratome or using Nd:Yag femtosecond laser (Intralase)

PHOTOREFRACTIVE KERATECTOMY:

Here the epithelium is removed by a sharp blade or blunt spatula after application of diluted absolute alcohol (20%) to the corneal surface to loosen the epithelium. Epithelium can also be removed by transepithelial ablation by the Excimer Laser itself. Photo ablation is then applied and postoperatively the patient is fitted with bandage soft contact lens for 48 to 72 hrs.

The popularity of PRK decreased when LASIK came because of faster visual recovery and decreased post operative discomfort with LASIK. PRK is an alternative to LASIK in conditions where very low refractive correction is needed, epithelial basement disease, thin cornea and for treatment of LASIK flap related complications like button holed flap.

With the advent of wave front guided laser ablation the popularity of PRK may increase.

LASIK in high myopia:

Although LASIK is done for myopia up to -12 to -16D, in this high range the predictability of the procedure is markedly reduced. In addition, while treating such a high myopia, there was a high incidence of loss of

best corrected visual acuity than in the correction of low levels of myopia. However, patients with high myopia often gain best corrected visual acuity after LASIK, probably due to decreased image magnification preoperatively due to spectacles. The required ablation depths for high corrections may leave an inadequate stromal bed for long term instability of cornea. The stromal bed thickness after refractive surgery should be at least 300 microns in the minimum. In addition, higher order corrections have an unacceptable of high side effects, including halos, glare and loss of contrast sensitivity. These side effects are due to increased high order aberrations like spherical aberrations.

CONTRAINDICATIONS OF LASIK(5)

1. thin cornea
2. ectactic corneal disorder
3. dry eyes
4. blepharophimosis
5. glaucoma
6. large pupil size
7. monocular patients
8. retinal vascular disease
9. autoimmune disease
- 10.Pregnancy.

COMPLICATIONS OF LASIK:

Intraoperative complications

Flap related complication:

Flap of variable and suboptimal thickness and diameter

Incomplete flap:

Tear or hole in the flap

Free flap

Damage, destruction or dislocation of flap

Complications during laser application:

Decentration of ablation

Incorrect ablation

Ablation of hinge

Complication during flap reposition

Incorrect placement of flap

Wrinkling on reposition of flap.

Damage of flap due to increased handling

Postoperative complication:

poor adhesion and anchoring of flap with the stromal bed

Infective keratitis

Non-specific intrastromal / intralamellar keratitis (Sands of Sahara)

Epithelial ingrowth under the flap

Undercorrection or overcorrection

Regression

Haze at the interface

Corneal ectasia

INTRAOCULAR SURGERIES:

1. PHAKIC IOL
2. CLEAR LENS EXTRACTION(REFRACTIVE LENS EXCHANGE)

PHAKIC IOL represents a new category of IOL that expand the range of keratorefractive surgery. While LASIK surgery can only be done for refractive error up to -14D , PIOL can be done for up to -20D. PIOL can be an alternative if PRK and LASIK are contraindicated.(14)

Types of phakic IOL

1. angle supported PIOL
2. iris supported PIOL
3. sulcus supported PIOL

The combination of corneal and intraocular refractive surgeries is called BIOPTICS. This may ultimately allow patients at the extremes of refractive error to achieve predictable outcome.

PIOL are contraindicated in pre-existing intraocular diseases such as corneal endothelial decompensation, iritis, rubeosis iridis, and cataract with glaucoma.

CLEAR LENS EXCHANGE may be preferable to PIOL in the presence of lens opacity.

EARLY IDENTIFICATION AND TREATMENT OF COMPLICATIONS:

The most important causes of vision loss in degenerative myopia are choroidal neovascularisation, macular degeneration and retinal detachment. Every effort should be made to examine thoroughly all highly myopic patients by indirect ophthalmoscopy to identify the peripheral retinal degenerations. Prophylactic laser treatment of the identified patients may avoid retinal detachment.

Choroidal neovascularisation in early stages may not be detected by clinical examination alone. Special investigations like fundus fluorescein angiography, indocyanine green angiography and optical coherence tomography are necessary to identify early CNV.

Laser photocoagulation has been applied successfully in juxtrafoveal and extrafoveal choroidal neovascularisation. Laser is not indicated in subfoveal choroidal neovascularisation. Treatment options other than laser include surgery, photodynamic therapy and anti-Vascular Endothelial growth Factors.

Successful surgery for choroidal neovascularisation is surgical removal of CNV and macular translocation with 360 degree retinotomy. Vertiporfin in photodynamic therapy trials have shown that it is effective in retaining vision by stabilizing or improving visual acuity and contrast sensitivity particularly when used in combination with anti Vascular Endothelial Growth factors. This is also useful in recurrent cases.

OTHER MODES OF APPROACH

The management of stretching of the sclera and the arrest of the degenerative process to date is not rewarding. Attempts are made to strengthen the thinned out sclera by patch graft but was not very successful. Bearing in mind, the scleral thinning is due to intra ocular pressure, use of anti glaucoma medications have been tried with variable results. Treatment in animal models based on biochemical and genetic approach are promising. One such is the modification of the scleral proteoglycan synthesis which might be able to inhibit the development of globe enlargement

VISUAL REHABILITATION:

One of the major causes of visual disability is pathological myopia which is largely unaddressed.

Rehabilitation is as important as the prevention and control of blindness.

The visually challenged persons may need the following types of rehabilitation

1. Medical rehabilitation

By low vision aids many visually challenged persons may have some useful vision. A low vision patient is a person who because of an irreversible disorder of the visual system cannot perform customary visual activities without special vision enhancing devices.

A low vision aid refers to an optical device that improves the residual vision by magnifying the image of the object at the retinal level. Also there are some non optical aids which may be helpful in improving the residual vision.

Principle of low vision aids is based on the fact that with sufficient magnification the normal retina surrounding the damaged retina can be used for central vision. Though the surrounding retina is less sensitive than the fovea or parafoveal region some useful vision may be obtained.

Types of optical low vision aids(5)

The currently available optical low vision aids are as follows

1. Magnifying spectacles
2. Hand held magnifiers
3. Stand magnifiers
4. Telescopes
5. close-circuit television

Magnifying spectacles are the most commonly prescribed low vision aids and many patients achieve a high degree of success with their use. They may be unioocular or binocular spectacles. the vantages of magnifying spectacles re more comfortable to use ,both hands are free field of vision is large, simultaneous near and distant vision possible and are less expensive.

Closed circuit television (CCTV)

In CCTV the camera picks up the reading material, magnifies and displays it on the television screen. They provide excellent contrast and magnification. These can be modified for a variety of purposes like reading, writing, computer works, crafts etc.

A CCTV provides a number of advantages over the optical systems. it provides a distortion free , brighter, magnified image with enhanced

contrast on a magnified screen. The letters on the back screen also helps in image improvement in some cases. The major limiting factor is that it is expensive, heavy, and difficult to mobilise.

Non optical devices

The various types of non optical methods are

Approach magnification

Proper lightning

Contrast enhancement

Auditory aids such as talking clocks, computers with speech synthesizers are available for use by visually challenged persons.

Fibre tipped pens- with black ink provides best contrast while writing. The writing ids are quit useful for signature and cheque writing.

2. TRAINING AND PSYCHOLOGICL REHABILITATION

It is the most important aspect. First of all they should be assured and made to feel that they are equally useful and not inferior to sighted persons. Their training should include mobility training with help of a stick, training in daily activities like washing, putting clothes, shaving, cooking and other household works.

3. EDUCATIONAL REHABILITTION

It includes education in blind schools with the facility of Braille system of education.

4. VOCATIONAL REHABILITATION

It will help them to earn their livelihood. Blind persons can be trained in making handicrafts, book binding, candle and chalk making, etc.

To conclude, the strategies must ensure that

No citizens go blind needlessly due to preventable causes

All avenues are exhausted to restore the best of possible vision to curable blind persons.

Blinds not amenable to curable measure receive comprehensive rehabilitation.

PART – II

AIM OF THE STUDY

1. To analyse the ocular parameters associated with degenerative myopia
2. To study the corneal thickness, axial length and Refractive parameters with reference pathological myopia
3. To assess the ocular associations of pathological myopia
4. To assess the visual status associated with pathological myopia
5. To determine the visual disability and the major causes of vision loss in pathological myopia
6. To categorise and assess the percentage of visual disability

PURPOSE OF STUDY

Degenerative myopia is common in India and is a major cause for visual disability. According to NPCB-WHO SURVEY 1980 uncorrected refractive errors, particularly pathological myopia, accounts for 7.355 of all causes of bilateral blindness which is only second to cataract among all leading causes of blindness. In Tamilnadu the percentage goes to 21.5 % .also in the survey for causes of low vision in India, refractive errors accounts for 18.875 and is the second leading cause for low vision.

While prevention is not possible at least blindness from pathological myopia may be prevented, at least to some extent. this is possible by early detection of the most common cause of vision loss the choroidal neovascularisation by appropriate investigation and treating them. also early detection of peripheral retinal degeneration And prophylactic laser treatment would be helpful.

Vision loss in myopes is not only because of their high refractive error but also due to the associated conditions with it.

One among them which gains importance is open angle glaucoma which is easily missed because of the falsely recorded visual acuity and the misled optic disc apperaence.prompt recognition of these associated conditions and addressing them will be greatly helpful to them in retaining their vision.

INCLUSION AND EXCLUSION CRITERIA

INCLUSION CRITERIA

1. All patients with refractive error greater than -6 dioptre spherical equivalents in one or both eyes were included in the study.
2. Patients with age 10 – 40 who filled the above criteria were included in the study.
3. Both sexes, males and females ,were included in the study
4. Patients were included irrespective of the visual acuity

EXCLUSION CRITERIA

1. Patients with previous intraocular surgery were excluded from the study.
2. Patients aged less than 10 years and greater than 40 years were excluded from the study
3. Patients wearing contact lenses were excluded from the Study.

MATERIALS AND METHODS

This is a retrospective study done in eye department, Thanjavur medical college, Thanjavur from June 2011 to October 2012.

A total of fifty patients attending outpatient department were selected.

Informed consent was obtained from all the patients.

All patients underwent a complete ocular examination including

1. Visual acuity measurement- by snellens visual acuity chart
2. Anterior segment examination by torch light followed by slit lamp examination.
3. Corneal curvature measurement and retinoscopy by automated refractometer.
4. Intraocular measurement by goldmann applanation tonometer after instillation of 4 % lignocaine and staining with sterile fluorescein strips.
5. Central corneal thickness measurement by pachymeter

6. Axial length measurement by ultrasound A scan
7. Gonioscopy by Goldman single mirror
8. Dilated fundus examination using direct, bio microscopic examination with + 78 dioptre lens, indirect method.
9. In selective cases, ultrasound –B scan and was also done.
- 10 .In selective cases visual field examination was done on another day with Octopus automated perimeter.

All the measurements and examination were done by single trained person to avoid inter observer variations.

Visual disability assessed as per the chart given by government of India

categories	Vision in better eye (best corrected)	Vision in worse eye (best corrected)	Percentage impairment
Category 0	6/9-6/24	6/24-6/36	20%
Category I	6/18-6/36	6/60-nil	40%
Category II	6/60-4/60 Or Field of vision 10-20 degree	3/60-nil	75%
Category III	3/60-1/60 Or Field of vision <10 degree	CFCF to nil	100%
Category IV	CFCF to nil Or Field of vision <10 degree	CFCF to nil Or Field of vision <10 degree	100%
One eyed persons	6/6	CFCF -nil	30%

KEY TO MASTER CHART:

SL. NO	SERIAL NUMBER
V/A	VISUAL ACUITY
M	MALE
F	FEMALE
RE	RIGHT EYE
LE	LEFT EYE
BCVA	BEST CORRECTED VISUAL ACUITY
Sph	SPHERE
Cyl	CYLINDER
K VALUE	KERATOMETRY VALUE
DEG	DEGREE
AXL	AXIAL; LENGTH
IOP	INTRAOCULAR PRESSURE
CCT	CENTRAL CORNEAL THICKNESS
OA	OCULAR ASSOCIATIONS
DIS	VISUAL DISABILITY
CFCF	COUNTING FINGERS CLOSE TO FACE
HM	HAND MOVEMENTS
PL	PERCEPTION OF LIGHT
RP	RETINITIS PIGMENTOSA

MASTER CHART

TABLE 1.1

SL. NO	NAME	AGE/SEX	V/A			REFRACTION			K VALU E	I O P	CCT	AXL	OA	DIS %
				UC	BCV A	Sph	Cyl	AXI S						
1	MANIKANDAN	17/M	RE	2/60	6/60	-6.00	-1.00	70	45.75 45.75	14	0.527	24.07	-	30
			LE	6/6	6/6	-	-	-	45.25 45.25	18	0.528	23.45		
2	JOSHI	20/M	RE	CFCF	4/60	-19.75	-1.0	25	43.25 43.00	16	0.512	33.25	-	75
			LE	HM	HM	-22.00	-	-	43.00 43.00	16	0.487	35.06		
3	PERIYAVOTT AIYI	46/F	RE	6/60	6/12	-6.50	-	-	42.25 44.25	16	0.512	22.18	-	30
			LE	CFCF	3/60	-9.00	-	-	45.25 45.50	14	0.496	27.17		
4	VIGNESH	17/M	RE	5/60	6/18	-11.00	-2.00	180	42.00 44.50	14	0.476	26.47	-	-
			LE	5/60	6/18	-11.00	-1.50	120	42.75 44.75	12	0.475	28.99		
5	MANIKANDAN	15/M	RE	2/60	6/60	-10.00	-	-	41.25 43.00	16	0.528	24.12	-	75
			LE	1/60	1/60	-12.00	-	-	41.30 42.25	14	0.562	24.56		
6	SIVARANJINI	9/F	RE	CFCF	3/60	-18.00	-2.0	172	47.00 48.75	12	0.419	26.55	RP, KER AT OC ON US	100
			LE	CFCF	2/60	-24.00	-3.50	29	47.25 48.75	12	0.465	26.42		
7	VAISHNAVI	22/F	RE	5/60	6/18	-3.75	-4.75	32	40.50 42.25	18	0.465	23.75	-	-
			LE	4/60	6/18	-3.75	-3.25	179	40.00 42.75	18	0.479	24.01		
8	MURALI	32/M	RE	4/60	6/36	-5.00	-2.00	120	43.25 43.25	16	0.512	23.82	-	-
			LE	6/60	6/18	-4.50	-2.00	30	44.12 44.25	18	0.514	23.89		
9	BOSE	18/M	RE	3/60	6/60	-8.00	-	-	42.25 43.00	14	0.504	24.18	-	-
			LE	3/60	6/60	-9.00	-	-	41.25 43.75	14	0.502	24.25		
10	SUJATHA	30/F	RE	6/60	6/12	-8.50	-2.50	70	42.00 43.75	14	0.482	27.79	-	30
			LE	CFCF	CFCF	-6.00	-3.50	180	42.00 44.75	12	0.481	28.13		

TABLE 1.2

S.NO	NAME	AGE/ SEX	V/A			REFRACTION			K VALUE	IOP	CCT 0.521	AXL	OA	DIS %
				UC	BCVA	Sph	Cyl	AXIS						
11	KASTHURI	24/M	RE	1/60	1/60	-7.00	- 2.00	70	41.30 42.25	26	0.521	24.81	GLAUCOMA	75
			LE	2/60	6/60	-6.00	- 1.50	150	41.75 43.00	20	0.493	25.01		
12	SHANKAR	22/M	RE	6/60	6/12	-6.00	-	-	44.15 44.50	18	0.518	23.18	-	-
			LE	5/60	6/12	-7.00	-	-	44.25 44.25	18	0.522	23.45		
13	KRISHNAN	42/M	RE	CFCF	2/60	-8.00	-	-	43.25 42.75	22	0.488	24.26	GLAUCOMA	100
			LE	CFCF	2/60	-8.00	-	-	42.50 43.00	22	0.482	24.18		
14	MANI	18/M	RE	6/60	6/18	-7.00	-	-	43.25 43.50	18	0.522	24.03	-	30
			LE	1/60	1/60	- 18.00	-	-	44.75 45.25	16	0.514	25.72		
15	JAMEELA	28/F	RE	6/60	6/24	-6.00	- 1.00	180	42.25 42.75	16	0.522	23.72	-	-
			LE	4/60	6/36	-6.00	- 1.50	180	43.25 43.25	16	0.514	23.68		
16	SANDIYAGU	26/M	RE	HM	HM	-5.25	- 2.50	16	44.25 43.75	14	0.469	22.21	RP, KERATO CONUS	100
			LE	HM	HM	-6.75	- 2.00	150	43.25 43.25	14	0.467	24.38		
17	JOSEPH	40/M	RE	6/60	6/24	-7.00	-	-	44.25 43.75	18	0.524	24.01	-	-
			LE	6/60	6/18	-6.00	-	-	44.75 44.50	18	0.518	23.82		
18	PREMA	29/F	RE	1/60	1/60	-9.00	- 1.00	90	45.25 44.75	14	0.498	25.12	-	30
			LE	6/60	6/12	-6.00	-	-	43.25 43.75	16	0.504	24.01		
19	SIVAKUMAR	22/M	RE	5/60	6/12	-7.00	-	-	43.50 43.75	18	0.510	24.13	-	-
			LE	5/60	6/12	-7.00	-	-	44.25 44.25	16	0.515	24.18		
20	RANI	28/F	RE	NO PL	NO PL	- 18.00	- 2.00	73	46.75 47.25	12	0.482	25.88	OPTIC ATROPHY	30
			LE	6/60	6/12	-6.50	-	-	44.25 44.50	18	0.522	24.14		

TABLE 1.3

S.NO	NAME	AGE/ SEX	V/A			REFRACTION			K VALUE	IOP	CCT	AXL	OA	DIS
				UC	BCVA	Sph	Cyl	AXIS						
21	JEYA JANSIRANI	38/F	RE	1/60	4/60	-7.00	- 3.00	180	46.00 46.25	14	0.512	24.18	ALBI NISM	75
			LE	1/60	3/60	-9.00	- 2.50	180		14	0.523	24.32		
22	SELVAM	44/M	RE	1/60	2/60	- 11.00	- -	-	41.25 42.00	14	0.498	25.31	GLAU COMA	100
			LE	1/60	1/60	- 13.00	- -	-	42.50 42.00	14	0.498	25.45		
23	ELIZABETH	18/F	RE	4/60	6/18	-6.00	- 0.50	180	43.50 43.50	16	0.522	23.82	-	-
			LE	4/60	6/24	-7.00	- 0.50	180	42.25 42.25	16	0.521	23.71		
24	KUMAR	23/M	RE	1/60	1/60	-5.75	- 0.50	169	41.50 41.50	16	0.483	24.18	-	30
			LE	6/6	6/6	-0.25	- 0.50	23	43.50 43.50	18	0.516	23.43		
25	ROSELIN	21/F	RE	5/60	6/18	-6.50	- -	-	42.75 42.75	16	0.511	23.82	-	-
			LE	5/60	6/18	-7.00	- -	-	43.25 43.25	16	0.523	23.94		
26	MOHAN	27/M	RE	3/60	6/36	-7.00	- -	-	41.75 43.25	18	0.522	23.56	-	40
			LE	2/60	6/60	-7.00	- 1.00	180	42.50 44.00	18	0.523	24.03		
27	VANATHI	31/F	RE	1/60	3/60	-9.00	1.00	23	44.25 45.00	18	0.474	25.15	-	75
			LE	2/60	5/60	-7.00	- 1.50	100	43.75 43.25	16	0.492	24.34		
28	JEYARAMAN	40/M	RE	CFCF	3/60	-8.75	- 1.25	44	41.75 42.25	14	0.535	27.13	-	100
			LE	CFCF	2/60	- 10.75	- 2.00	90	41.25 43.25	10	0.481	26.78		
29	KASTHURI	32.F	RE	6/60	6/24	-5.00	- 2.50	180	44.25 44.00	18	0.533	24.05	-	-
			LE	6/60	6/18	-6.00	- 1.00	180	43.75 43.50	16	0.534	24.12		
30	BALA KRISHNAN	39/M	RE	HM	HM	- 18.00	- -	-	41.25 41.25	22	0.488	25.83	GLAU COMA	100
			LE	HM	HM	- 19.00	- -	-	42.00 41.50	24	0.492	26.12		

TABLE1.4

S.NO	NAME	AGE/ SEX	V/A	REFRACTION					K VALUE	IOP	CCT	AXL	OA	DIS
				UC	BCVA	Sph	Cyl	AXIS						
31	ARUL ANAN DHAM	35/M	RE	PL	PL	-14.00	-	-	41.00 43.25	14	0.489	26.12	RP	100
			LE	PL	PL	-16.00	-	-	41.00 41.75	14	0.492	25.63		
32	MALLIKA	40/F	RE	6/60	6/12	-5.00	-	68	43.25 43.25	18	0.531	23.82	-	-
			LE	6/60	6/18	-6.00	-	121	43.25 43.50	18	0.533	23.75		
33	ANTONY	41/M	RE	6/60	6/24	-6.00	-	21	43.50 42.75	18	0.522	23.51	-	-
			LE	5/60	6/24	-6.00	-	104	42.50 42.75	16	0.523	23.13		
34	JEEVA	18/M	RE	3/60	6/36	-4.00	-	90	43.50 43.75	18	0.521	23.82	-	-
			LE	6/6	6/6	-	-	-	44.25 44.25	18	0.521	23.82		
35	DEVAKI	38/F	RE	2/60	6/60	-5.50	-	42	43.50 42.75	18	0.528	23.18	-	40
			LE	4/60	6/36	-5.00	-	117	43.50 43.50	16	0.518	23.22		
36	ANANDRAJ	17/M	RE	4/60	6/36	-6.00	-	37	41.50 42.00	16	0.513	24.08	-	40
			LE	6/60	6/18	-6.00	-	134	41.00 42.25	18	0.522	24.83		
37	JOSEPH	28/M	RE	6/60	6/18	-5.50	-	90	42.25 41.75	18	0.512	23.72	-	-
			LE	5/60	6/18	-6.00	-	-	42.00 42.25	18	0.513	23.66		
38	ARUL	18/M	RE	6/60	6/18	-6.00	-	-	42.25 43.00	16	0.522	23.12	-	20
			LE	4/60	6/36	-7.00	-	-	41.75 42.25	16	0.522	23.18		
39	BALASUBRAMA NIYAM	22/M	RE	3/60	6/60	-7.00	-	90	41.25 43.75	16	0.511	23.89	-	40
			LE	4/60	6/36	-7.00	-	90	41.50 42.50	18	0.507	24.03		
40	SELVAM	40/M	RE	6/60	6/12	-5.50	-	180	43.50 44.50	18	0.522	23.54	-	-
			LE	5/60	6/18	-6.00	-	-	43.50 43.25	18	0.523	23.81		

TABLE 1.5

S.NO	NAME	AGE/ SEX	V/A			REFRACTION			K VALUE	IOP	CCT	AXL	OA	DIS
				UC	BCVA	Sph	Cyl	AXIS						
41	KOWSALYA	31/F	RE	CFCF	2/60	-8.00	-1.50	4	41.75 43.25	16	0.489	25.72	-	100
			LE	CFCF	2/60	-9.00	-	-	42.00 41.75	16	0.491	25.18		
42	MARY	38/F	RE	5/60	6/24	-6.00	-0.50	90	43.50 43.25	18	0.503	24.08	-	-
			LE	4/60	6/18	-6.00	-	-	44.00 44.25	18	0.506	24.13		
43	MOHAN	35/M	RE	CFCF	1/60	-13.00	-	-	41.00 41.25	12	0.472	27.11	-	30
			LE	5/60	6/12	-7.00	-	-	43.25 45.25	18	0.514	24.28		
44	VIMALA	23/F	RE	HM	HM	-16.00	-3.00	82	41.25 41.75	12	0.466	26.83		100
			LE	HM	HM	-19.00	-3.00	90	41.00 40.75	12	0.473	26.56		
45	ISMAIL	41/M	RE	4/60	6/18	-6.00	-1.00	180	42.75 42.50	16	0.512	24.12	-	-
			LE	5/60	6/24	-6.00	-1.00	180	43.25 42.75	16	0.518	24.66		
46	DEEPA	16/F	RE	5/60	6/18	-5.50	-1.50	180	41.50 43.25	16	0.502	23.92	-	-
			LE	4/60	6/18	-6.50	-	-	42.75 44.25	16	0.496	24.16		
47	RANI	32/F	RE	1/60	5/60	-8.00	-	-	43.25 43.00	18	0.492	24.82	-	40
			LE	1/60	6/60	-7.50	-	-	43.25 43.25	18	0.488	24.67		
48	BALAJI	14/M	RE	3/60	6/36	-6.00	-1.50	180	44.25 44.00	18	0.511	24.12	-	20
			LE	4/60	6/18	-6.00	-	-	43.75 43.75	18	0.513	24.08		
49	PAREETHA	18/F	RE	5/60	6/18	-5.50	-1.00	180	41.75 43.25	18	0.512	23.98	-	-
			LE	5/60	6/18	-5.50	-1.50	180	41.50 43.25	18	0.522	23.82		
50	RAJKUMAR	21/M	RE	6/60	6/12	-6.00	-	-	43.50 43.50	18	0.523	24.07	-	-
			LE	6/60	6/18	-6.00	-	-	43.25 43.50	18	0.534	24.13		

INTERPRETATION OF RESULTS

SEX DISTRIBUTION

TABLE 2

SEX	TOTAL
MALE	29
FEMALE	21

Sex appears to have an influence in the incidence. Although males and females are equally affected in the lower degree of myopia, in higher degrees females are more prone for degenerative changes .In the study among 50 patients 29 are males and 21 were females. As the patients were randomly selected from the outpatient in the study conclusion regarding prevalence could not be obtained.

FAMILY HISTORY OF MYOPIA

TABLE 3

POSITIVE FAMILY HISTORY	NO OF PATIENTS	PERCENTAGE
FATHER ALONE	4	8
MOTHER ALONE	12	24
BOTH PARENTS	1	2
NEITHER PARENT	35	70

The role of heredity is well linked to pathological myopia.

In the study out of 50 patients 15 patients had positive family history.

Father alone in 4, Mothers alone in 12 and both of them in 1 patient. This

amounts to a total of 30 % heredity element which correlated well with

duke elder's heredity element of 32.89%.

Although heredity element is found in 30% there was no family history in 70% which again adds more to the conclusion that factors other than heredity like environmental factors ,visual hygiene,general health, increased near work, increased intraocular pressure etc., may play role in pathogenesis of Pathological myopia.

LATERALITY

According to duke elder unilateral high myopia is relatively rare.

In the study two cases of unilateral myopia was found among 50 patients.

Analysis from literature tends to show predominance for higher degrees of myopia and unilateral myopia in right eye.

In the study no laterality for higher degrees of myopia in right eye was found and out of 48 cases of bilateral myopia 24 cases had higher degrees in right eye and 24 patients had higher degrees in left eye.

However in the study the affected eye was the right side in both two cases of unilateral myopia.

R)	mm	D	AX
R1)	7.28	46.50	173
R2)	7.20	47.00	83
AVE	7.24	46.75	
CYL		+0.50	83
S:-	0.25	C:-	-0.50
L)	mm	D	AX
R1)	7.37	45.75	169
R2)	7.17	47.00	79
AVE	7.27	46.50	
CYL		+1.25	79
S:-	5.75	C:-	-0.50
PD =	62		

PRESS START S

PICTURE 1 (AR value showing unilateral high myopic refractive error in left eye. Case no. 24)

AXIAL LENGTH

TABLE 4

AXIAL LENGTH	NO OF EYES
<22.50 mm	7
22.51-24.00	25
24.01-26.00	41
>26.00	25

The axial length well correlated with both the retinoscopy values and the amount of visual impairment. The more the axial length the more was the retinoscopy value and poorer is the visual acuity.

Among the 98 eyes (with the exception and two eyes in patients with unilateral myopia) 25 eyes and axial length >26 mm, 41 eyes between 24 and 26 mm, 25 eyes Between 22.51 to 24.00 mm and only 7 eyes < 22.50 mm. Also patients with higher axial length had decreased central corneal thickness and the intraocular pressure relatively low.



PICTURE 2 (Ultrasound A Scan value in LE
Showing 35.06mm Case no.2)

In my study the maximum recorded axial length was 35.06mm.

Even patients whose axial length were normal showed fundus changes of Pathological myopia confirming the fact that the degeneration of retina is not only due to increase in axial length and stretching of sclera but in fact may be genetically determined.

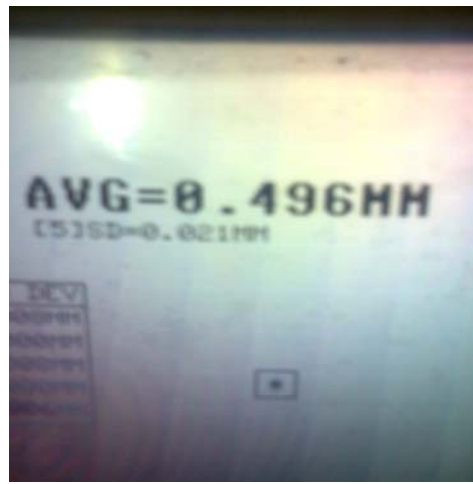
CENTRAL CORNEAL THICKNESS

TABLE 5

CCT	NO.OF EYES
<500	34
501-525	56
526-550	8
>550	NIL

The normal central corneal thickness varies from 540 – 565 microns.

The central corneal thickness in pathological myopes in my study is found to decreased. None out of the 98 eyes in 50 patients had central corneal thickness greater than 550 microns.56 eyes had central corneal thickness between 501 and 525 microns and 34 eyes had values less than 500 microns.8 eyes had values between 526 and 550 microns and none had values greater than 550 microns.



PICTURE 3(pachymeter showing CCT

Value of 0.496 mm.Case no 46)

This is an important fact because the decreased CCT in pathological myopes results in falsely low recording of a really high intraocular pressure and thus a coexisting glaucoma may be easily missed. Recording of intraocular pressure by applanation tonometry becomes essential in all pathological myopes. This may give amore accurate value than the falsely recorded low value of indentation tonometry because of decreased CCT and low ocular rigidity.

Previously it was said that corneal thickness maybe thicker than normal in pathological myopes. But studies have shown varying thickness may occur in degenerative myopia and in fact thinner corneas are more common in myopes .Too thin corneas in these patients makes contraindication for the laser corrections in these patients

The decrease in CCT makes to think that the stretching may not be limited to the posterior segment alone and may be a generalised process involving whole eyeball. Other anterior segment changes such as rupture in descemets membrane, zonular dehiscence adds more to the fact that degeneration may be a generalised process but much attention is given to the posterior segment changes because of the effect on vision caused by it.

REFRACTIVE POWER OF CORNEA IN DEGENERATIVE MYOPIA:

TABLE6

AVERAGE REFRACTIVE POWER IN DIOPTRE	NO. OF EYES
<42.00D	17
42.01 – 43.00D	16
43.01- 44.00D	34
44.01 – 45.00D	20
>45.00D	11

In the study 17 out of the 98 eyes had refractive power less than 42 D, 16 eyes had had refractive power between 42 and 43 D , 34 out of 98 eyes had values between 43 and 44D and 20 eyes between 44 and 45 D . Only 11 eyes had values greater than 45 D.

The refractive power of cornea in degenerative myopia in this study is more clumped in the lower normal values with about 75%o having refractive power <44D . This is correlating with the previous facts that the cornea in the degenerative myopia is mire flatter than normal to compensate the axial elongation. However. two patients with keratoconus had refractive power of about 48D

However values greater than 45 D should always arise suspicion of keratoconus and along with gross change in refractive error within short period without degenerative changes in patients aged more than 18 years corneal topographic studies should always be done.

FUNDUS ASSOCIATIONS

TABLE 7

FUNDUS FINDING	NO OF EYES
TESSELATION	92
PERIPHERAL RETINAL DEGENERATION	28
TEMPORAL CRESENT	71
RETINAL DETACHMENT	7
CHOROIDAL NEOVASCULARISATION	12
PETINITIS PIGMENTOSA	6
POSTERIOR STAPHYLOMA	2

Tessellation of fundus was the most common finding seen in about 92% of eyes myopic crescent comes second with temporal crescent in all except 2 eyes where a nasal or inverse crescent is seen.

Peripheral retinal degeneration was documented in about 28% of eyes with lattice degeneration being more common.

The two most important cause of vision loss in degenerative myopia choroidal neovascularisation and retinal detachment were seen in about 12% and 7% of patients respectively.

The incidence of choroidal neovascularisation varies between 5 -10 % and in the study it is 12% which also correlated with the early documentation

The incidence of retinal detachment in literature is that 5% of myopes develop retinal detachment and in my study also the percentage of retinal detachment is 7%.



PICTURE 4 (Ultrasound B scan showing retinal detachment In RE . Case no 43)

The incidence of choroidal neovascularisation is 8% which is diagnosed by slit lamp bio microscopic examination of fundus with 78 dioptre lens under high magnification. The actual prevalence of CNV may be much higher as in the early stages may be missed by ophthalmoscopy,

This necessitates the need for special investigations like fundus Fluroscein angiography, indocyanin green angiography and ocular coherence tomography to diagnose early CNV and their prompt treatment with Photodynamic therapy and anti VGEF may prevent the patient from Vision loss and becoming virtually blind. Posterior staphyloma is seen in 2 eyes which is confirmed by Ultrasound – B Scan.

OCULAR ASSOCIATIONS

TABLE 8

OCULAR ASSOCIATIONS	NO OF EYES
OPEN ANGLE GLAUCOMA	8
RETINITIS PIGMENTOSA	6
KERATOCONUS	4
ALBINISM	2

The most common association was open angle glaucoma which was found in four patients out of fifty of which three of them were diagnosed newly and one was already on antiglaucoma medications.

According to Becker and Shaffer the incidence of glaucoma is directly related to the axial length .the incidence is 3% in eyes with axial length less than 26.5 mm, 11% in eyes with axial length between 26.5 and 33.5 mm, 28% in eyes with axial length > 33.5mm.

The percentage of open angle glaucoma in the study was found to be 8% in pathological myopes. The other ocular associations were retinitis pigmentosa in 6%, keratoconus in 2% and albinism in 1%



PICTURE 5 (iris transillumination in
Myopic patient with albinism case no.21)



PICTURE 6 (keratoconus in myopic patient

Case no. 16)

Although the corneal curvature in pathological myopia is said to be flatter than normal, its thickness is very much less when compared to normal which may result in bulging of the cornea. The phenomenon of keratoconus in myopia may be well explained. Similar to the posterior staphyloma where the sclera gets thinned and protrudes out. The prevalence of both keratoconus and posterior staphyloma is 4% although both do not occur in the same individuals.

VISUAL DISABILITY

TABLE 9

DISABILITY PERCENTAGE	NO OF PERSONS	PERCENTAGE
NO DISABILITY	25	50%
20% DISABILITY	2	4%
30% DISABILITY	7	14%
40% DISABILITY	3	6%
75% DISABILITY	5	10%
100% DISABILITY	8	16%

Out of 50 patients 25 patients had some percentage of visual disability which means 50% of all pathological myopes are visually disabled. 8 patients had 100% disability, 5 had 75% disability, and 3 had 40% disability. 7 had 30% disability and 2 had 20 % disability. In East Asian cities, myopia is very common and appears to be rising in some parts of the world. The most common causes for blindness in pathological myopia in my study were choroidal neovascularisation , retinal detachment and optic Atrophy due to open angle glaucoma. While the occurrence of pathological myopia cannot be prevented early recognition of the above said cause and treatment of the same may much reduce the visual disability.

SUMMARY AND CONCLUSION

The literature on degenerative myopia was reviewed.

Materials and methods employed are stated.

In the study there was 30% association of family history in pathological myopia. The ocular parameters were greatly altered in pathological myopia. The axial length is increased and their increase is proportional to the refractive error and vision loss in majority of cases.

The central corneal thickness is reduced to less than 525 microns in most of the patients which is very important finding because it may results in the false recording of intraocular pressure. The common ocular associations found in pathological myopia are 1) open angle glaucoma, 2) retinitis pigmentosa , 3) keratoconus and 4)albinism. The common cause of vision loss associated with pathological myopia were choroidal neovascularistion, and retinal detachment.

The associated conditions which were responsible for vision loss were Optic atrophy due to glaucoma and retinitis pigmentosa.

Regarding visual disability in pathological myopia 50% of all myopic patients suffered from some form of visual disability.

8 patients had 100% disability (16%), 5 patients had 75% disability (10%), 3 patients had 40% disability (6%), 7 patients had 30% disability (14%) and 2 patients had 20 % disability (4%)

Degenerative myopia deserves recognition as one of the truly neglected areas of ophthalmology. Degenerative myopia is an important world-health issue with an unfortunate history of ineffectual treatments that have led most eye specialists to believe that it is something of a "lost cause." As a result, this condition, which is responsible for the loss of vision in so many people during the middle years of life and in old age, seems destined to run its natural course, save for attention to correction of the refractive error . Much of the sight loss in high axial myopia is due to the complications such as choroidal neovascularisation and retinal detachment. If early diagnosis of the CNV is done in pathological myopes by doing fundus Fluorescein angiography and indocyanine green angiography, prompt treatment with laser photocoagulation, photodynamic therapy and anti- VGEF factors, vision loss due to these major complications may be prevented. Early diagnosis of peripheral retinal degeneration by indirect ophthalmoscopy and prophylactic treatment with lasers may prevent a retinal detachment.

Diagnosis of coexisting factors such as open angle glaucoma in myopes should not be missed as it is one of the leading causes of blindness associated with degenerative myopia. In doubtful cases prophylactic antiglaucoma medications can be prescribed.

There is an increased vulnerability for trauma in these patients due to prominent globe. So necessary preventive measures should be taken. Myopia related visual impairment may affect the productivity, mobility, quality of life and activities of daily living of the individuals. Potentially blinding-myopia related pathologies are often irreversible in nature, especially if diagnosed late. So early recognition of the coexisting conditions and complications, and their management may prevent the degenerative myopic patients from becoming essentially blind.

PART- III

ABBREVIATIONS:

D	DIOPTRE
NPCB	NATIONAL PROGRAMME FOR CONTROL OF BLINDNESS
WHO	WORLD HEALTH ORGANISATION
HLA	HUMAN LEUCOCYTE ANTIGEN
RPE	RETINAL PIGMENT EPITHELIUM
CNV	CHOROIDAL NEOVASCULARISATION
VEGF	VASCULAR ENDOTHELIAL GROWTH FACTOR
FFA	FUNDUS FLOURESCEIN ANGIOGRAPHY
ICG	INDOCYANINE GREEN ANGIOGRAPHY
ARMD	AGE RELATED MACULAR DEGENERATION
PRK	PHOTOREFRACTIVE KERATECTOMY
LASER	LIGHT AMPLIFICATION BY STIMULATED EMISSION OF RADATION
LASIK	LASER ASSISTED INSITU KERATOMILEUSIS
LASEK	LASER SUBEPITHELIAL KERATOMILEUSIS
Nd;YAG	NEODMYIUM YITTIRUM ALUMINIUM GARNET
PIOL	PHAKIC INTRAOCULAR LENS
UC	UNCORRECTED
SL. NO	SERIAL NUMBER
V/A	VISUAL ACUITY
M	MALE
F	FEMALE
RE	RIGHT EYE
LE	LEFT EYE
BCVA	BEST CORRECTED VISUAL ACUITY
Sph	SPHERE
Cyl	CYLINDER
K VALUE	KERATOMETRY VALUE
DEG	DEGREE
AXL	AXIAL; LENGTH
IOP	INTRAOCULAR PRESSURE
CCT	CENTRAL CORNEAL THICKNESSS
OA	OCULAR ASSOCIATIONS
DIS	VISUAL DISABILITY
CFCF	COUNTING FINGERS CLOSE TO FACE
HM	HAND MOVEMENTS
PL	PERCEPTION OF LIGHT
RP	RETINITIS PIGMENTOSA

PROFOMA
DISSERTATION ON
A CLINICAL EVALUATION OF PATHOLOGICAL MYOPIA

DATE-

NAME -
ADDRESS -

AGE/ SEX -
OP NO -
PRESENTING COMPLAINTS -

H/O PREVIOUS OCULAR SURGERY-
H/O CONTACT LENS USE -

FAMILY HISTORY-

OCULAR EXAMINATION-

HEAD POSTRE
EYEBALL POSITION
EOM

RIGHT EYE

LEFT EYE

VISUAL ACUITY

UNCORRECTED

BCVA

FOREHEAD
EYEBROWS
EYELIDS
COJUNCTIVA
CORNEA
ANTERIOR CHAMBER
IRIS
PUPIL
LENS

RIGHT EYE

LEFT EYE

KERATOMETRY

CCT

IOP

USG-A SCAN

RETINOSCOPY

FUNDUS

USG-B SCAN

VISUAL FIELDS

OCULAR ASSOCIATIONS

VISUAL DISABILITY%

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