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HIGH MYOPIA: INCIDENCE, PATHOGENESIS AND MANAGEMENT

Ophthalmology

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ABSTRACT

Myopia is one of the most prevalent disorders of the eye. High myopia is associated with comorbidities that increase risks of severe and irreversible loss of vision, such as retinal detachment, subretinal neovascularization, dense cataract, and glaucoma. Myopia, commonly referred to as shortsightedness, is a common cause of visual disability throughout the world. The World Health Organization has grouped myopia and uncorrected refractive error with cataract, macular degeneration, infectious disease, and vitamin A deficiency among the leading causes of blindness and vision impairment in the world.1

KEYWORDS

High myopia, complications, prevention, management.

INTRODUCTION:

High myopia is characterized by axial length elongation, and consequent stretching of the posterior eye wall causes various specific complications including cataract, chorioretinal atrophy, and macular hole with or without retinal detachment, myopic foveoschisis, or optic nerve head changes. Most of these complications are vision threatening and often lead to irreversible retinal photoreceptor damage, and thus, central visual loss. People with myopia can be classified in two groups, those with low to modest degrees of myopia (referred to as "simple" or "school"myopia, 0 to - 6 dioptres) and those with high or pathological myopia (greater than - 6 dioptres). Simple myopia can be corrected with spectacles or contact lenses, whereas "high" (pathological) myopia is often associated with potentially blinding conditions such as retinal detachment, macular degeneration, and glaucoma (fig 1).

Incidence: The incidence of high myopia varies among ethnicity, race, and country, but with a higher incidence in Asian countries. It ranges from 2% in White Americans2 to 2.6% in China3 and 5.5% in Japan.4 The incidence of myopia is seemingly increasing,5 and it is one of the major causes of blindness, especially in Europe and East Asian countries.6,7

Definition : To obtain clear vision, the eye must accurately focus an image in space on the retina. The main ocular determinants of refraction are the focusing power of the cornea and crystalline lens and the length of the eye. In myopia, the image is focused in front of the retina because the cornea or lens curvature is too strong or the eye is too long (axial myopia). When the optical components focus the image perfectly on the retina, this is described as emmetropia, and when the eye focuses the image behind the retina, this is described as hyperopia. Refractive error is measured in dioptres (D), and myopia is designated with a minus sign. Mild myopia is 0 D to -1.5 D, moderate - 1.5 D to - 6.0 D, and high myopia - 6.0 D or more. Pathological myopia occurs with more than - 8.0 D, although retinal disease, cataract, and glaucoma—the associated threats to vision—can also occur in patients with moderate and high myopia. At birth, most infants are hyperopic, but when the eyes grow they usually become less hyperopic and by age 58 years emmetropic. This process, wherein the refractive state of children's eyes shifts in magnitude and reduces in variance to reach near emmetropia, is called emmetro pisation.

PATHOGENESIS OF MYOPIA

The incidence of myopia depends on genetic and environmental factors. Myopia is inherited in a mono- or polygenic fashion. Monogenic inheritance occurs rarely as autosomal dominant, autosomal recessive and X-linked transmitted. Polygenic inheritance occurs much more frequently. Current studies have identified genes responsible for myopia higher than 6 D on chromosomes 1–5, 7, 8, 10–12, 14, 17–22. Genes responsible for myopia lower than 6 D have been found on chromosome 7. Myopia is present in many syndromes such as: Cohen, Cornelia de Lange, Down, Ehlers-Danlos, Kniest, Knobloch, Marfan, McCune-Albright, Noonan, Prader-Willi, Rubinstein-Taybi, Stickler, Weill-Marchesani, fetal alcohol and also in homocystinuria, congenital night blindness, deficiency of ornithine aminotransferase and prolidase, lack of a sufficient amount of calcium, fluoride and selenium in food and in premature infants [8-12].

In 1997 Goss et al. [13] described the following risk factors for the development of myopia: positive family history of myopia; presence of myopia on noncycloplegic retinoscopy in infancy, decreasing to emmetropia before entry into school; refractive error of emmetropia to 0.5 D of hyperopia;against-the-rule astigmatism; decreased accommodative function or near point esophoria; substantial amount of near work on a regular basis; steep corneal curvature or high axial length to corneal radius ratio; conditions temporarily obscuring the retina from clear imagery during infancy. It is currently believed that the risk factors for myopia are: sleeping in illuminated rooms until the age of 2 years, high level of education, having a high IQ, infrequent outdoor activity, passive exposure to cigarette smoke [14,15].

Role of accommodation: The most important environmental factor for myopia is intensive near work such as reading, writing or working on a computer.It is widely believed that the more frequent incidence of myopia in people subjected to intensive near work is caused by accommodative spasm. During intensive near work the image viewed is focused slightly posterior to the retina, which may induce accommodative spasm, deformation of the image on the retina and an increase of axial length of the eyeball.It has been observed that watching television is tiring for the eyesight but does not lead to a more frequent incidence of myopia. This is most probably due to the fact that most people watch television from a distance of a few meters with little risk of developing accommodative spasm [16,17]. The correlation between level of academic achievement and the prevalence and progress of myopic refractive errors is strong; people whose professions entail much reading during either training or performance of the occupation (lawyers, physicians, microscopists, and editors) have higher degrees of myopia, and the myopia may progress not just in people's teenage years but throughout their 20s and 30s.[18-20]

MYOPIA MANAGEMENT

There are a lot of non-surgical methods of management in myopia[21,22].Myopia can be corrected with spectacles, contact lenses or by refractive surgery.

Spectacles have the advantages of:

1) being more economical in most cases;
2) providing a level of safety against injury to the better seeing eye;
3) serving as a modality for other optical modifications (bifocal or prism) in the management of residual binocular anomalies [23].

A clear method of spectacle correction has not been established (full correction or undercorrection). Therefore, this problem requires further clinical studies. The majority of doctors, however, prefer to prescribe undercorrection in patients up to the age of 18 years and after reaching 18 years of age give the full correction.

Contact lenses appear to have certain advantages, including:

1) reduction of aniseikonia in cases of refractive and axial anisometropia;
2) improved cosmetics, which encourages better compliance with wearing the optical correction;
3) elimination or reduction of prismatic imbalance, weight problems, tilt, peripherical distortions, and visual field restrictions experienced by users of spectacle lenses [23].

There are the following refractive surgeries:

incisional corneal surgery (RK), ablative corneal surgery (PRK, LASIK, LASEK, Intra-LASIK), keratoplasty (FTK, LTK, CK), other corneal surgeries and devices, crystalline lens modifications. Each method has its advantages and disadvantages [24].

J. Messmer [25] has lately demonstrated the complications after correction of refractive errors with LASIK. Many serious and less threatening complications were observed. These complications have been compiled in Table 1. According to Messmer as a result of postoperative progression of myopia, many patients need to undergo a second LASIK procedure.

Surgical methods of refractive error correction do not solve the fundamental problem of myopia, which are increased axial length and the associated complications. After surgical procedures, degenerative changes of the retina do not regress, the risk of retinal detachment is not reduced and myopia may still progress. Surgical methods only permit the individual freedom from wearing spectacles or contact lenses. However, they do not inhibit the progress of the disease.

New possibilities of myopia treatment. In 1975 David H. Hubel and Torsten N. Wiesel, both laureates of the Nobel Prize in Physiology or Medicine initiated studies on drugs inhibiting the progress of myopia [26]. Currently, it has been observed that the following pharmacological agents: atropine, oxyphenonium, pirenzepine, chlorpyrifos, 7-methylxanthine, apomorphine, reserpine, 6-hydroxy dopamine, dextromethorphan, MK-801, APV, bicuculline, SR95531, CACA, TPMPA, dextrorphanol, levorphanol, D- and L-naloxane, L-NAME, formoguanamine,-xyloside, central and peripheral antagonist of VIP, basic fibroblast growth factor and scleral crosslinking agents inhibit the progress of myopia [27,28].

CONCLUSION

Most myopic children will develop only low to moderate levels of myopia, but some will progress rapidly to high myopia. Risk factors for the development of high myopia include ethnicity, parental refraction, and rate of progession of myopia. In those children at risk, interventions should be considered.

Efforts to prevent the progression of myopia date back centuries, and eye exercises, medications, and hygiene have been proposed to prevent weak eyes. Most modern efforts have been focused on decreasing the accommodative requirements of the eyes. Anticholinergics such as atropine have been used in combination with bifocals in an attempt to slow the progression of myopia. Although progression is slowed during treatment, the long term effects seem to be a difference of no more than 12 dioptres, and no cases of pathological myopia have been prevented with this treatment.Rigid or gas permeable contact lenses may offer a mode of treatment that may be effective in slowing the progression of myopia.[29] The rate of progression of myopia is slower in patients using these contact lenses than in patients using lenses that are placed in spectacles.[30] The exact mechanism by which rigid contact lenses prevent axial myopia from developing is unclear. Laser refractive surgery can eliminate the refractive condition of myopia, but it does not decrease the rate of the blinding conditions of retinal detachment, macular degeneration, and glaucoma associated with high myopia.[31] Other interventions have included the use of vitamins, scleral surgery to provide shortening of the eye, biofeedback, ocular hypotensives, ocular relaxation techniques.

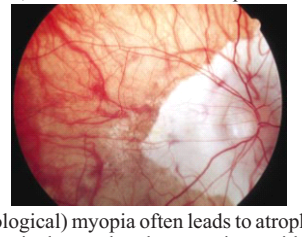

Fig 1 High (pathological) myopia often leads to atrophy of the choroid and subsequent retinal macular degeneration, with loss of central visual acuity and high incidence of retinal detachment, glaucoma, and strabismus.

Table 1. Selected complications after LASIK procedure

Complications Frequency	(%)
Dry eyes	20–40
Diffuse lamellar keratitis	2–4
Epithelial ingrowth	1–3
Flap wrinkles	0.2–4
Free cap	0.1–1
Infection (bacterial, fungal)	0.1
Over- and under-correction, poor centration, irregular astigmatism, contamination	
Total	23.4–52.1

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