Myopia: a Review of Literature

*Maduka Okafor F C MBBS, FMCOph **Okoye O I, MBBS, FMCOph ***Eze B I MBBS, FMCOph

*Department of Ophthalmology, College of Medicine, University of Nigeria, Enugu Campus, Enugu State, Nigeria
**Department of Ophthalmology, College of Medicine University of Nigeria, Enugu Campus, Enugu State, Nigeria
***Department of Ophthalmology, College of Medicine, University of Nigeria, Enugu Campus, Enugu State,
Nigeria

Abstract

Introduction: Among the refractive errors, myopia is a leading cause of visual impairment throughout the world and its prevalence is on the increase. Pathological myopia is a genetically determined refractive error and a growing body of evidence shows that visual experiences early in life may affect ocular growth and eventual refractive error. In addition to the human cost of visual debility, there is a profound economic cost to the society. The current review highlights recent advances in the management of myopia.

Methods: Information was obtained by searching Medline for citations of articles in English using the Keyword "Myopia" in addition to current literature review.

Result: Myopia can be classified into two groups, low to moderate degree of myopia (refered to as simple myopia 0.5 to -6.0 dioptres) and high or pathological myopia (greater than 6.0 dioptres). Simple myopia can be corrected with spectacles or contact lenses whereas high myopia may be complicated by potentially blinding conditions such as vitreous and macular degeneration and retinal detachment.

Conclusion: Recent advances in the management of myopia have made it possible to restore vision to a reasonable degree.

Key words: Myopia, Refractive error, Visual impairment

Date Accepted for publication: 11th March 2009

Nig J Med 2009; 134-138

Copyright ©2009 Nigerian Journal of Medicine

Introduction

Aristotle (384 322BC) is credited with first distinguishing myopia¹. Galen (131-201 A.D.) coined the Greek term Myein (to close) and ops (eye) as he observed that the individual closed the eye (squinted) to see¹.

Myopia (Shortsightedness) is the type of refractive error in which parallel rays of light come to a focus infront of the retina, when the eye is at rest. A myopic patient sees near objects clearly while objects in the distance are blurred. Myopia commonly occurs when the physical length of the eye is greater than the optical length. For this, myopia often develops in the rapidly growing school aged child or teenage² and progresses during the growth years requiring frequent changes in glasses or contact lenses. It usually stops progressing as growth is completed in the early twenties. Uncorrected myopia leads to physical disability and economic disadvantage. The psychological outlook of the individual is also affected. Myopes have poor distant vision and because of this they develop in a limited world wherein they are at a disadvantage in comparison with others, a handicap which may entail a seeming limitation of intelligence and a curtailment of interests which are frequently put down to stupidity and backwardness or to naughtiness. while they are really due to visual defect. Many activities that are going on in the world especially the more subtle things escape them, avoiding outdoor sports and prone to introspection and finding free interaction with their fellows difficult. They frequently tend to grow up with distinct mental habits and peculiarities.

Myopia affects males and females equally and those with a family history of myopia are more likely to develop it³⁻⁵.

Aetiology

Myopia may be caused by increased anteroposterior diameter of the eyeball (axial myopia). Most cases are axial in type. Acquired myopia can be caused by uncontrolled diabetes mellitus or cataract. Pseudomyopia is a rare disorder that is usually due to a spasm of the ciliary muscles (spasm of accommodation). Degenerative myopia is genetic and of recessive inheritance. An increased refractivity of the lens nucleus is responsible for the myopia found in cataract. Curvature myopia may be associated with an increase in the curvature of the cornea or one or both surfaces of the lens.

Classification

A) By Origin

 Correlative (simple, Benign) is the customary type mildly progressive, if progressing at all, occurring during the years of growth, often called stationary. Simple myopia can be axial or refractive. It is axial when the eye is relatively too long for its refractive status.

Refractive in which the refractive system of the eye is too strong for the axial length, subclassified as

- (I) Index in which the indices of refraction at the various media are anomalous as
- (A) Too high-aqueous, lens nucleus, cornea
- (B) Too low -lens cortex, vitreous.
- (li) Curvature -in which the radii of curvature of the various surfaces are too short.
- (Iii) Aqueous chamber in which a decrease of the depth of the anterior chamber increases the refractive power of the eye.
- Component (variously described as pathological, progressive, malignant, degenerative) due to an abnormal development of one of the components of the ocular refracting mechanism or the malignant effect of one or more component of diseases.
 - (A) Congenital axial revealing colobomas, staphylomas, choroidal degeneration, optic and choroidal atrophy
 - (B) Developmental degenerative in which degenerative changes occur with loss of vision due to rapidly persisting progression of myopia after puberty.
 - (C) Disease acquired in which development is caused by goitres, tuberculosis, measles and other debilitating diseases.
 - B). By Amount: Classically myopia can be classified into very low (up to minus one dioptre), low (minus one dioptre to three dioptres), medium (minus three dioptres to six dioptres), high (minus six dioptres to minus ten dioptres) and very high (above minus ten dioptres).
 - C). Age of onset: Myopia could be congenital, youth onset, early adult onset or late adult onset. Congenital myopia is myopia present at birth that persists through infancy and childhood. It is often high in amount. Youth onset myopia is the most common form. The onset is about five years of age to the teenage years or physical maturity. Once youth onset myopia appears, the amount

of myopia increases, a phenomenon sometimes referred to as myopia progression⁶. The prevalence of myopia increases from about 2% at six years of age to about 20% at fifteen years of age. ^{6,7} Early adult onset myopia is myopia with onset in adulthood up to forty years of age. Myopia that appears after the age of forty years is late adult onset myopia. It seems to be less common than youth onset and early adult onset myopia.

Besides age, other factors affect myopia prevalence. One of these is ethnicity, the highest prevalence are among the Japanese and Chinese. 8,9,10 The prevalence of myopia increases with increase in family income. The prevalence is greater among persons who have more years of education and among persons who spend more time doing near work in their vocations. Greater progression of myopia has been reported to be related to greater amount of time spent on close work and to closer working distance. These associations of myopia with more near work have led many to assume that near work plays a role in the development of myopia. Some other people believe that greater involvement in near work could be a way of adaptation behaviour by the myopic patient.

Myopic Refractive Changes with Age

A wide range of refractive errors can be present at birth. Small amounts of myopia at birth usually disappear in the first year or two of life. The presence of myopia in infancy may be a risk factor for the reappearance of myopia in the school years. The largest change in refractive error generally seen by the clinician in otherwise normal healthy eye is the increase in myopia seen among children after the youth onset myopia. In most young adults, refractive error is relatively stable, although the onset of and increases in myopia are not uncommon. After the age of 45 years, there is a shift towards hyperopia. Shifts in the sixth decade or later can also be associated with the development of age related cataracts.

Progression of Myopia

Once myopia appears in childhood, it increases in amount, the myopia progression usually stopping or slowing in the middle to late teens. The earlier in childhood the onset of myopia, the faster is the rate of progression and the greater is the amount of myopia that has developed by the end of childhood. The ocular optical component associated with childhood myopia progression is an increase in the depth of the vitreous chamber. The ocular optical component associated with childhood myopia progression is an increase in the depth of the vitreous chamber.

Although childhood myopia progression typically stops or slows in the middle to late teens, further myopia progression can occur in adulthood.

Clinical Features

Symptoms: The visual defect is the most prominent symptom of myopia. Most commonly, simple or school myopia starts manifesting between 7 to 10 years and is bilateral. The greater the degree of myopia, the greater the visual defect. In small degrees of error, symptoms of eye strain are present. In progressive myopia there may be pseudoproptosis with large pupil.

Signs: Ophthalmoscopically, the major findings in a progressive myopia are

- i) Temporal crescent (myopic crescent). The bulging backwards of the posterior pole results in a separation of the retina and choroids from the temporal margin of the disc leaving an atrophied part through which the sclera is seen as a white area (the myopic crescent) while on the nasal side, the retina extends over the edge of the disc thus blurring its margin and conditioning the supertraction crescent. The myopic crescent is usually temporal, but it may run an annular ring all the way round the disc.
- ii) Tigroid Fundus in which there is loss of pigment from pigment epithelium of the retina and as a result the choroidal vessels are well seen
- iii) Choroidal atrophy is seen in the posterior pole. Myopic choroidal atrophy is present in high degree of myopia but its severity is not necessarily parallel to that of myopia. It is genetically determined and is usually recessive.
- iv) Muscae Volitantes: Vitreous opacities due to premature liquefaction and degeneration may be seen
- Forster Fuchs Fleck. Fuch's spot, the dark pigmented macular lesion results from combined effects of retinal pigment epithelial hyperplasia and pigment derived from haemorrhage. Other macular changes include atrophy, pigmentation or haemorrhage.
- vi) Posterior staphyloma. Rarely, posterior staphyloma may be present due to increased length of the anteroposterior diameter of the eyeball.
 - The following are associated with high myopia in different structures.
- (a) Retina and choroids: Atrophy, haemorrhage, break, detachment of retina and macular degeneration.
- (b) Vitreous: Liquefaction, opacities and detachment.
- (c) Lens: Cataract.
- (d) Intra-ocular pressure: High myopia is sometimes associated with open angle glaucoma.

Treatment

Treatment can be optical or surgical

- 1) Optical treatment: Optical correction consists of
 - (a) Provision of appropriate concave lenses. Glasses which give best vision with maximum comfort are prescribed. Full correction is advised in young patients with low degree of myopia up to 6.0D. In adults, under correction is advised especially for reading because the ciliary muscle becomes weak and can not tolerate normal accommodative effort offered by the correcting lenses. In high myopia, a full correction can be rarely tolerated.
 - (b) Contact lens. In very high degree of myopia where diminution in size of image and optical aberrations of the correcting glasses render it difficult for the full correction to be prescribed, contact lens is of real help. This eliminates prismatic effects and provides a greater field than the glasses.
 - (c) Telescopic glasses. This may be helpful in cases with macular degeneration.
- 2) Surgical Treatment:
 - (a) Corneal Surgeries:
 - (i) Radial keratotomy (RK). This involves making radial incisions in peripheral cornea. The procedure works well for low degrees of myopia.^{28, 29} However, since the advent of laser procedure its use has diminished.
 - (ii) Photorefractive Keratectomy (PRK). This is performed with the excimer laser which can accurately ablate corneal tissue to an exact depth with minimal disruption of surrounding tissue. The central cornea is ablated so that it becomes flatter. Approximately 10im of ablation corrects ID of myopia. The photorefractive Keratectomy is able to correct myopia up to 6D.
 - (lii) Laser in-situ keratomileusis (LASIK). Laser insitu keratomileusis is currently the most frequently performed refractive procedure. It is more versatile than PRK and can correct myopia up to 12D depending on corneal thickness. The procedure is performed by combining a lamellar incision with laser ablation of the central cornea.³¹
 - (Iv) Intrastromal plastic rings which cause central flattening can be used to correct low myopia but are at a very early stage of development. The procedure avoids the visual axis and is reversible.^{30,32}

- (v) Epikeratophakia is a form of corneal surgery in which the patient's corneal epithelium is removed and a donor minus lenticle of desired power is sutured onto the cornea. The donor lenticle becomes re-epithelialized within a few days. About 4 12 weeks are required for stabilization of the graft epithelium. Best visual acuity is usually reached by 2 3 months. The complications include glare, chronic epithelial defects and suboptimal final visual acuity.³³
- (vi) Keratomileusis is a surgical procedure in which the refractive power of the eye can be modified by using a lathe to reshape the deep surface of a lamellar disc of corneal tissue. The disc is cut from the anterior surface of the cornea with an electric keratome before freezing in liquid carbon dioxide to enable it to be ground on the lathe. If a convex section is carved away, the anterior surface of the cornea is flattened, reducing myopia. With care, the unfrozen disc is replaced, and secured with nylon sutures. The operation can be accurate to within 2 dioptres, ³⁴ but becomes impossible if the cornea is thin or irregular.
- (b) Lens Extraction: The aphakic eye is normally strongly hypermetropic. If an eye with an axial myopia is deprived of its lens, the amount of myopia becomes reduced without any correcting lens and there will be improvement in visual efficiency.³⁰ However, the highly myopic eye is often a diseased eye and is not an ideal one to withstand operative procedures. Extraction of the crystalline lens in high myopes reduces the degree of myopia. Implantation of intraocular lens in the anterior or posterior chamber improves visual acuity.³⁰
- (c) Phakic Intraocular Lens Implantation: Intraocular lens implantation in the anterior or posterior chamber improves acuity in high myopes. However, this procedure should be used with caution because it may be associated with uveitis, endothelial cell loss and cataract formation.
- (D) Scleral Reinforcement Surgeries:S u r g i c a l measure employed to prevent axial elongation is the reinforcement of the posterior sclera with fascia lata. Excision of a ring of sclera and suture of the two hemispheres has been practiced in cases of staphyloma in high myopia. Most of these surgical methods are too radical to apply to minor myopic states which can be more certainly and readily corrected by optical means.

Prognosis

About 80% of children are born hyperopic, 5% myopic and 15% emmetropic.35 After age 7 or 8, myopia gradually increases until about 25 years. Any degree occurring in a child under the age of 4 years should be regarded as potentially serious requiring observation. Above this age and certainly above the age of eight or ten, low degrees up to 6D should be looked upon with less alarm. Care should be exercised especially about the time of puberty, and if the age of twenty-one is passed without serious progression, the condition may be expected to remain stationary and the prognosis may be taken as good. In high myopia, the prognosis should always be guarded. It must be based on the appearance of the fundus and the visual acuity after correction. In all cases, the possibility of sudden haemorrhage or retinal detachment should be borne in mind. There is a higher incidence of cataract and secondary glaucoma among the high myopes.

Prevention

The hereditary nature of both myopia and the degenerative retinal condition associated with it warrants that preventive measure is necessary. There need be no restraint on marriage and procreation among simple myopes. However, parents with degenerative myopia should be warned that any offspring will be liable to the same disability according to the laws of recessive Mendelian inheritance. Two highly myopic adults with degenerated fundi should never have children because they will give birth to pathological myopes.

Conclusion

Distant visual impairment is the chief complaint of myopic patients. It has to be borne in mind that a highly myopic eye is a diseased eye. In the choice of a career, if it seems possible that progressive myopic changes will involve visual deterioration in later life, it is obviously economically unwise to choose work requiring constant good vision. It is better that interest should be on a vocation of less visually demanding nature, preferably one which can to some extent be continued if the central vision fails. Blindness from pathological myopia has profound psychological, social and economic cost to the society and should be managed adequately to restore vision. Optical or surgical treatment improves visual acuity.

Acknowledgement

We wish to thank Mrs. Ann Chiawa who painstakingly typed the manuscript.

References

- Borish I.M.: Clinical Refraction, 3rd Ed, Chicago, Professional Press, 1970: 83.
- 2. Khoo C. Y, Ng R. F.: Methodologies for interventional myopia studies Ann, Acad. Med. Singapore. 2006 Apr., 35: 282-6.
- Teikari J. O'Donnell J, Kaprio J, Koskenvuo M.: Genetic and environmental effects on Oculometric traits. Optom Vis Sci, 1980; 66:594 9
- Young T. I., Ronan S. M., Drahozal I. A., Wildenberg S. C., Alvear A. B., Octting W. S et al: Evidence that a locus for familial high myopia maps to chromosome 18p. Am J. Hum Genet. 1998; 63:10919.
- Young T. I., Ronan S. M., Alvear A. B., Wildenberg S. C., Octting W. S., Attwood I. D., et all: A second locus for familial high myopia maps to chromosome 12q. Am. J. Hum. Genet. 1998; 63: 1419 24.
- Grosvenor T.: A review and a suggested classification system for myopia on the basis of age related prevalence and age of onset. Am. J. Optom. Physiol Opt 1987; 64: 545
 554.
- Sperduto R. D., Seigel D., Roberts J., Rowland M., Prevalence of myopia in the United States. Arch Ophthalmol 1983; 101:405 407
- 8. Borish I. M.: Clinical Refraction, 3rd ed., Chicago, Professional Press, 1970:19-20.
- Baldwin W. R.: A review of statistical studies of relation between myopia and ethnic, behavioural, and physiological characteristics. Am. J. Optom Physiol Opt. 1981; 58:516-527.
- Bear J. C.: Epidemiology and genetics of refractive anomalies.
 In: Grosvenor T, Flom M. C., Refractive Anomalies: Research and Clinical Applications. Boston, Butterworth Heinemann. 1991: 57 80.
- 11. Angle J., Wissman D. A.: The epidemiology of myopia. Am.J. Epidemiol. 1980; 111:220 228.
- 12. Goldschmidt E.: On the Etiology of Myopia: An Epidemiological study. Copenhagen, Nunksgaard, 1968; 25 59.
- 13. Peckham C. S., Gardiner P.A., Goldstein H.: Acquired Myopia in 11 year old children. Br Med. J. 1977; 1: 542 544.
- 14. Angle J., Wissman D. A.: Age, reading and Myopia. Am. J. Optom Physiol Opt. 1978; 55: 302 308.
- Richler A., Bear J. C.: Refraction, nearwork and education: a population study in Newfoundland. Acta Ophthalmol 1980; 58:468 478.
- Parssinen O., Lyyra A. L.: Myopia and Myopic progression among school children: a three year follow-up study. Invest. Ophthalmol Vis. Sci. 1993; 34:2794 2802.
- 17. Ingram R. M., Barr. A.: Changes in refraction between the ages of 1 and ³¹_p years. Br. J. Ophthalmol. 1979; 63:339 342.

- 18. Mohindra I., Held R.: Refraction in humans from birth to five years Doc Ophthalmol 1981; 28:19 27.
- 19. Goss D. A.: Variables related to the rate of childhood myopia progression. Optom Vis Sci. 1990; 67:631 636.
- Hofstetter H. W.: Some inter-relationships of age, refraction and rate of refractive change. Am.J. Optom Arch Am Acad Optom. 1954; 31:161 169.
- Goss D. A., Winkler R. L.: Progression of myopia in youth: age of cessation. Am. J. Optom physical Opt. 1983; 60:651
 658.
- 22. Septon R. D.: Myopia among Optometry Students. Am.J. Optom Physio Opt. 1984; 61:745 751.
- 23. Mantyjarvi M. I.: Predicting of myopia progression in school children. J. Pediatr Ophthalmol strab. 1985; 22:71-75.
- Grosvenor T., Perrigin D. M., Perrigin J., Maslovitz B.: Houston myopia control study: a randomized clinical trial II. Final report by the patient care team. Amj Optom Physiol Opt. 1987; 64:482 498.
- 25. Tokoro T., Kabe S.: Relation between changes in ocular refraction and refractive components and development of the myopia. Acta Soc. Ophthalmol Jpn 1964; 68:1240 1253.
- 26. Fledelius H. C.: Ophthalmic changes from age 10 to 18 years: a longitudinal study of sequel to low birth weight IV, Ultrasound Oculometry of vitreous and axial length. Acta Ophthalmol. 1982; 60:403 411.
- Goss D. A.: Childhood myopia. In Grosvenor T., Flom M. C. Refractive Anomalies: Research and Clinical Applications. Boston, Buterworth Heinemann. 1991: 81 103
- 28. Forstot S. L.: Radial Keratotomy. International Ophthalmic Clinics 1988; 28:116 25.
- 29. Waring G. O., Lynn M. J., Gilbertson W.: Three year results of the prospective evaluation of radical keratotomy study. Ophthalmology 1987; 94:1339 54.
- 30. Kanski J. J.: Clinical Ophthalmology, ^{5th} ed., Boston, Butterworths, 2002:150 51.
- 31. Kanski J.J.: Clinical Ophthalmology, 5th ed., Boston, Butterworths, 2002; 152.
- 32. Schwartz A. P., Tinio B. O., Babayan A., Naikoo H. N., Roberts B, Asbell P. A.: Intrastromal Corneal Ring Implantation (360 degree Ring) for myopia: A 5 year follow up. Eye Contact lens. May 2006; 32: 121 123.
- 33. Kanski J. J.: Clinical Ophthalmology, 3rd Ed, Boston, Butterworths, 1994:144 45.
- 34. Dorrel E. D.: Surgery of the Eye, London, The Whitefriars Press, 1978:36.
- 35. Vaughan D., Asbury T.: General Ophthalmology, 9th Ed; California, Lange Medical Publications, 1980: 12 13.