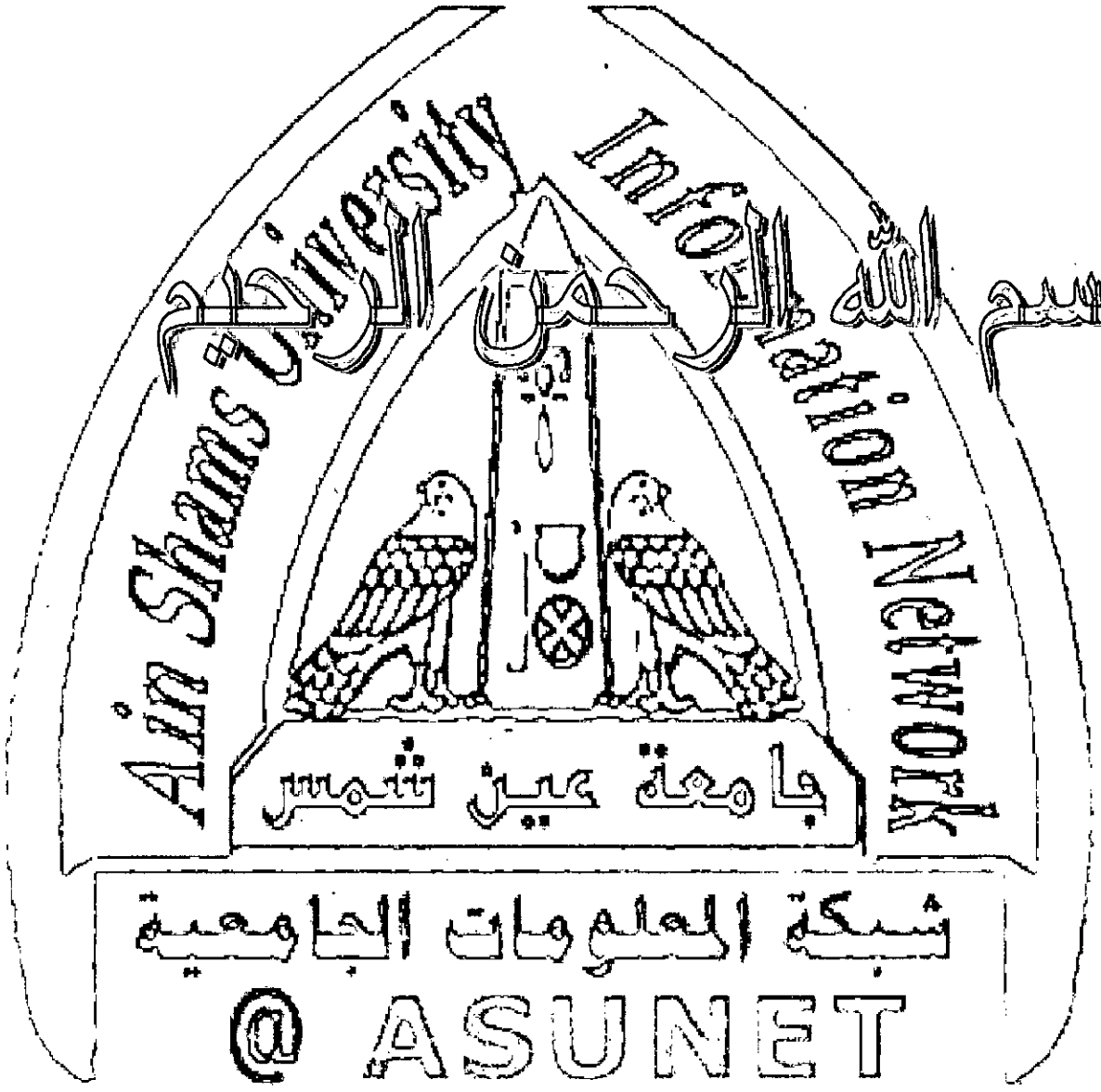
شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم



شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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INDOCYANINE GREEN ANGIOGRAPHIC CHORIORETINAL CHANGES IN PATHOLOGIC MYOPIA WITH AND WITHOUT LASIK PROCEDURE

Thesis

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INTRODUCTION

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Myopia:

The world "myopia" is derived from the term "muopia" which, in Greek, means to "close the eyes". It manifests itself as blurred distance vision; hence the popular terms "shortsightness" and "nearsightness". Myopia is a condition whereby images come into focus in front of the retina, resulting in a blurred image. The more severe the nearsightness, the farther the image is from the retina, which results in more blurry vision in the distance. [1]

In the myopic eye, the focusing power of the cornea (the major refracting structure of the eye) and the lens is too great with respect to the length of the eyeball. Light rays are bent too much, and they converge in front of the retina. Common types of myopia include: physiologic, pathologic and acquired. Physiologic myopia is a less common abnormality. This condition begins as physiologic myopia and then the eye continues to enlarge to an abnormal rate (progressive myopia). This more advanced type of myopia may lead to degenerative changes in the fundus. Acquired myopia occurs after infancy and it may be associated with uncontrolled diabetes and certain types cataract. [2]

A variety of genetic patterns for inheriting myopia have been suggested, ranging from a recessive pattern with complete penetrance in people who are homozygotic for myopia to an autosomal dominant pattern, an autosomal recessive pattern and various mixtures of these patterns.[3]

Pathological Myopia

The genetic profile of high myopia (defined as a refractive error greater than -6 diopeters) may differ from that of low myopia. Some researchers think that high myopia is determined by genetic factors to a greater extent than low myopia. Since even small deviations from normal structure cause significant refractive errors, it may be difficult to single out any specific genetic or environmental factor as their cause.

More than one gene is proved to be involved in the transmission of familial high myopia.[4]

Pathologic myopia (Degenerative myopia) is more severe than other forms of myopia and is associated with retinal changes and severe vision loss. It progresses rapidly and visual outcome depends on the extent of fundus changes.

Pathological myopia has been associated with other ocular and systemic diseases. These conditions include Down's syndrome, ocular albinism, infantile glaucoma, Marfan's syndrome Retinopathy of prematurity, Ehler's Danlos syndrome, low birth weight and maternal alcholism.

These conditions should be considered:"at risk" for pathological myopia and carefully monitored.

Pathological myopes may present with decreased visual acuity, an unusually large exophoria, strabismus (typically exotropia), open angle glaucoma, premature lenticular opacification and increased axial length (26.533 mm).[5]

Pathological myopia causes the eye to elongate, which in turn stretches and thins the retina and the sclera of the eye. The earliest fundoscopic sign of pathologic myopia is diffuse tessellation and pallor of the fundus as a result of thinning of the retinal pigment epithelium.[3]

Degenerative myopia is one of the leading causes of blindness in the world Macular haemorrhage is a major complication of degenerative myopia. Hallmarks of this degeneration are visible breaks in Bruch's membrane, which usually herald a decrease in

central vision especially when associated with haemorrhages. The occurrence of haemorrhage can also be a symptom of choroidal neovascular membrane: central visual function may be affected by serous or hemorrhagic detachments of the retina. [6]

Myopia is the seventh greatest cause of registered blindness in adults in Europe, [7] and in the United States. The prevalence of myopia in various published reports has varied from 11% to 36%. [8] It shows a marked change with age, the peak being at 20 years. [7] The frequency of pathologic myopia has been reported to range from 27% to 33.2% of the myopic population. [8]

Pathogenesis

The pathogenesis of the degenerative changes in and about the macula of the myopic eye is not clearly understood, although the lesions are thought to be either biomechanical abnormalities or here-dodegenerative. In the biomechanical concept, the chorioretinal lesions are viewed as a consequence of excessive axial elongation. [9]

It is believed that progressive distension of the posterior pole stretches the ocular coats, as evidence by straightening of the temporal retinal vessels, the appearance of a supertractional crescent and thinning of the retina and choroid. [10]

Crescent formation and chorioretinal atrophy have been directly related to increased axial length. [11]

Although the pathophysiology of staphydoma formation is unknown, it has been suggested that sclera is abnormal, has a low mechanical resistance, and gradually stretches or creeps in response to forces such as Intraocular pressure. [10]

Histopathology

The extensive histologic changes found in the posterior pole of the eye with pathologic myopia are characteristic:

Sclera:

The sclera is thinned with localized ectazia of the posterior pole. The architectural changes in the longitudinal fibers consist of thinning of the collagen bundles, reduced diameter of collagen fibrils, loss of striations and diminished scleral lamellae. The modification of elastic tissue remains controversial. Ultra-microscopic alterations seen in myopic sclera indicate a derangement of the diameter, the number, and organisation of the fibrils [12]

Choroid and retinal pigment epithelium

Changes affecting the choroid are essentially degenerative and atrophic. Ultramicroscopic alterations include a pronounced thinning of the choriocapillaris. The RPE cells appear flatter and larger than