Visual Field Changes in Chronic Simple Glaucoma

Essay
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Mohamed Sarwat Sayem
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Supervisors

Prof. Dr. Amin Gadel-Rab Atta
Professor of Ophthalmology
Ain Shams University

Dr. Mervat Salah Murad
Ass. Professor of Ophthalmology
Ain Shams University

Faculty of Medicine
Ain Shams University
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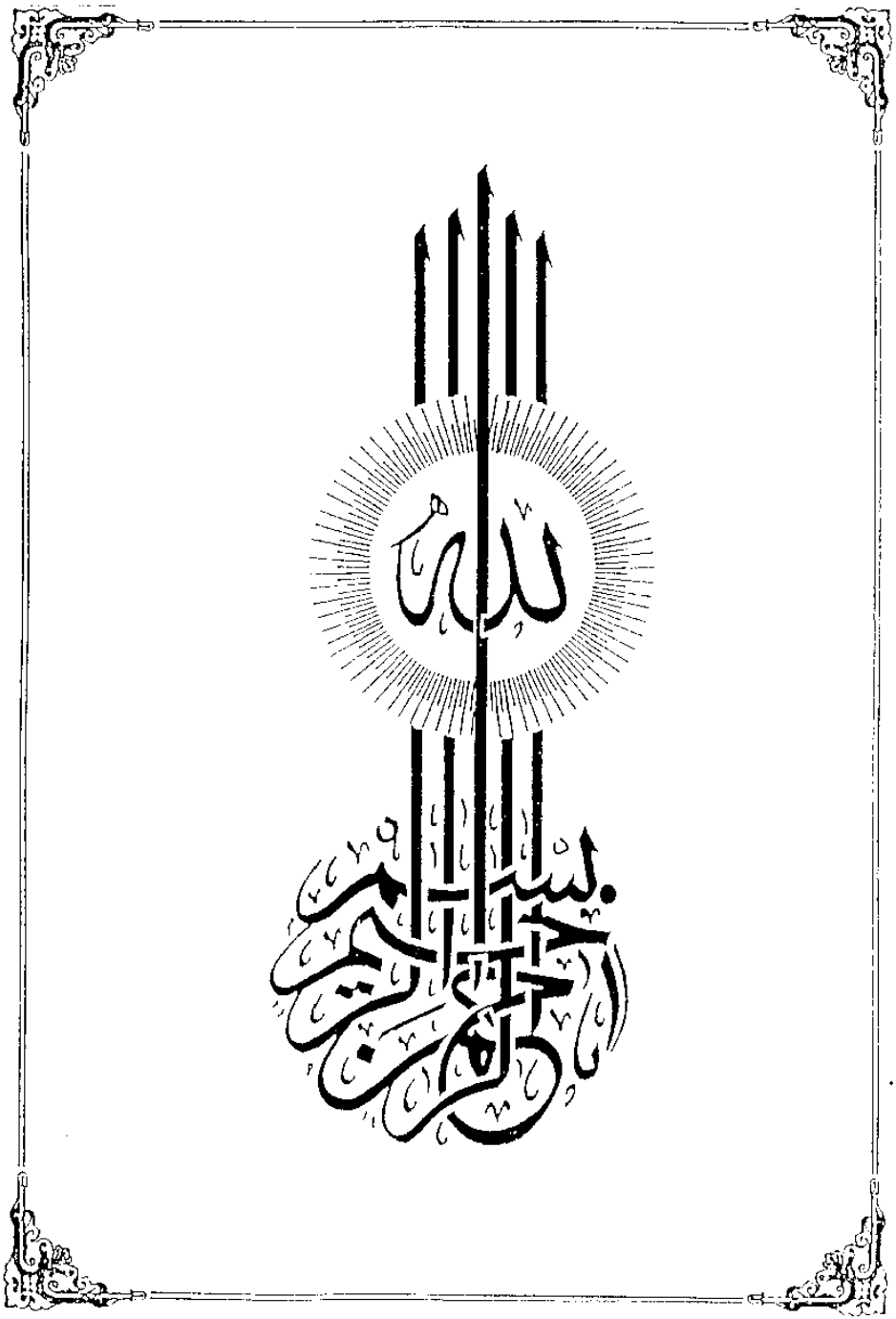

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ANATOMY OF THE NERVE FIBER LAYER OF THE RETINA

This layer is almost entirely made up of nerve fibers about 1.000.000. ***(Polyak 1941)***

It consists essentially of the axons of the ganglion cells which pass centripetal through the lamina cribrosa to form the optic nerve but there are also;

a) Centrifugal fibers.

b) Fibers of müller cells.

c) Neuroglial cells.

d) Retinal vessels.

The nerve fiber layer is thickest around the margin of the optic disc 20-30 um, but here it differs in the different quadrants, while thinnest directly lateral, it becomes thicker at the upper and lower quadrants, then the medial edge of the disc. The thickest parts are the upper and lower medial quadrants.

From the disc, the nerve fiber layers becomes thinner as we it passes towards the periphery and finally near the ora it is invaded by sparse ganglion cell, the two layers becoming one. The macular fibers constitute a well defined papillo-macular bundle which assume an oval shape extending from the macular region to the temporal region of the optic disc, the fibers from the nasal edge of the macula travel directly while those from the temporal hemi-macula with those from the

upper and lower part take an arcuate course to reach the disk. ***(Duke Elder 1961)***

The peripheral fibers from the nasal hemi-retina converge regularly and directly upon the nasal edge of the disc with little or no overlap between the upper and lower nasal quadrants. The fibers belonging to the nasal hemi-retina that arise between the temporal margin of the disc margin and the macula, arch sharply above and below the disc to reach its supero- and infero-nasal segments as the juxta-pupillay bundles. ***(Posner and Schlosman, 1948).***

The peripheral fibers from the temporal hemi-retina do not pass through the macula but have to go around it, the fibers above the horizontal meridian pass above the macula and those below under it. Those 1-2 mm temporal to the macula encircle it and the fibers cross the horizontal meridian. ***(Dimmer 1921)***

As we pass temporaly from the edge of the disc towards the macula it becomes thinner and at the bottom of the fovea it seems to disappear entirely. The axons of the ganglion cells throughout their course in the retina do not normally possess a myelin sheath - they vary from 0.5 to 2.0 um. in diameter. The cytoplasm of the axons contains microtubules, fine fibrils, mitochondria and occasionally vesicles. Individual fibers are separated by neuroglial processes, the nerve fibers are arranged in bundles which run parallel to the surface of the retina, they inter-weave with each other forming a network in those meshes are the processes of Muller and other glial cells. ***(Wolf 1976).***

Arrangement of the nerve fibers :-

All the visual fibers of the retina which are the axons of the ganglion cells converge towards the optic disc. However, a more logical and consistent scheme is obtained if the retina is considered to be divided vertically through the macula into a nasal hemi-retina including a nasal hemi-macula and a temporal hemi-retina including a temporal hemi-macula. ***(Polyak 1957)***

More temporal they pass above and below the macula in ever increasing arcs, the arcuate fibers in a pennate manner. ***(Greef 1900)***

There is no or little overlap between the upper and lower temporal fibers for a sharp horizontal raphe is formed from which the fibers diverge, this shape looks like a fold in the macula with its edge falling into apposition. ***(Roenne 1927)***

It follows that at the margin of the disc, since the greater part of its temporal edge is occupied by the papillo-macular bundle, the superior and inferior arcuate bundles which subserve the greater part of the temporal periphery must necessarily become aggregated heaped up to a greater degree then the corresponding fibers on the nasal side, each bundle in fact being concentrated into an arc occupying only about one sixth of the temporal half of the margin. ***(Duke Elder 1961)***

Since the center of the optic nerve is 1.0 mm above the horizontal raphe, it follows that the inferior arcuate bundle will

PATHOGENESIS OF GLAUCOMATOUS OPTIC ATROPHY

The pathogenesis of glaucomatous optic atrophy has remained a controversy since the mid 19th century when two concepts were introduced in the same year. In 1858, Muller proposed that elevated IOP leads to direct compression and death of neurons ***(The mechanical theory)***, while Von Jaeger suggested that a vascular abnormality was the underlying cause of optic atrophy ***(The vascular theory)***. The mechanical theory received the greatest support and held away through the first quarter of this century. Beauvieux popularizes the vascular theory in 1925. In 1968 the role of axoplasmic flow in glaucomatous optic atrophy was introduced. ***(Shields 1987)***

The mechanical theory

This theory was found by Muller in 1858, who postulated that optic nerve atrophy in glaucoma is a consequence of the nerve fiber compression by elevated intraocular pressure. He stressed that relief of the elevated intraocular pressure is the only aim of glaucoma therapy. ***(Carins 1986)***

The mechanical theory of nerve damage implies that glaucomatous excavation and the functional deficit are a direct result of the mechanical stretching and compression of tissue caused by a pressure imbalance across the lamina cribrosa. This pressure imbalance is thought to control and compress

the layers of the lamina cribrosa, thus compressing the axon bundles that pass through the pores of the lamina. Later, after one century when the role of axoplasmic flow was introduced in 1968, the mechanical theory could be more explained. The mechanical compression of neural tissue across the lamina causes blockage of the axoplasmic flow with subsequent functional deterioration and structural atrophy of the nerve fibers and their supporting elements. A corresponding reduction in the blood supply follows the tissue atrophy because the atrophied tissue no longer requires nutrition. ***(Quigley et al., 1981)***

Against this mechanical theory is the observation that elevated IOP in monkeys neither caused obstruction of rapid axoplasmic flow, nor prevented it in response to elevated IOP despite reduction in the pressure gradient across the lamina cribrosa. This suggest that more than a simple mechanical or hydrostatic mechanism may be involved. Also, against the simple mechanical theory, the observation that axon damage is diffuse within bundles, rather than focal as might be expected with a kinking effect and the location of transport interruption does not correlate with the cross-section area of fiber bundle, the shape of laminar pores or the density of inter-bundle septa. ***(Shields 1987).***

The vasogenic theory :-

This theory was introduced by Von Jaeger in 1858. He considered defects in the blood circulation to be the main cause of optic nerve damage in chronic glaucoma. A

hypothesis that has been recently supported by several authors.

Said et al., 1967, conclude that the main protective part in systemic hypertension is the diastolic blood pressure, as lowering of the systolic blood pressure in cases with only systolic hypertension does not lead to field damage.

Ernest and Potts 1968, found that the blood supply to the prelaminar part of optic nerve comes from the short posterior ciliary arteries, which are not autoregulated.

Hayreh et al., 1970, revealed that in animal experiments, acute elevation of IOP produces decreased ocular blood flow. He also, found that ischaemia of optic disc from disease such as temporal arteritis can produce cupping and visual loss indistinguishable from primary open angle glaucoma.

Klewin et al., 1978, said that a haemodynamic crises (example of sever bleeding in an accident or operation) may produce optic nerve cupping and visual field loss that closely resembles primary open angle glaucoma.

Adam and Schwartz, 1980, noticed fluorescein filling defect of the optic disc in primary open angle glaucoma and correlated in location with the visual field loss.

Hoskin and Kass 1989, found that marked elevation of intraocular pressure produces optic nerve ischaemia with pallor, hemorrhage and arteriolar attenuation. Also they noticed that systemic hypertension protects the eye against

damage produced by raised intraocular pressure. Visual field loss can progress rapidly when blood pressure is reduced too rapidly from extensive treatment in hypertensive patients. Also they found that diabetes and other vascular diseases, through their damaging effect of the vascular beds, are risk factors for the development of primary open angle glaucoma.

There is significant discrepancy between the findings of different investigators and still more controversy in their explanation. However, it is generally accepted that these two theories are the main factors responsible for optic nerve damage in glaucoma, which of them is the primary and leading factor may be different in various patients and different stages of glaucoma. ***(Carins, 1986)***

Axoplasmic flow

It was demonstrated that elevation of intraocular pressure can stop rapid axoplasmic transport in monkey optic nerve at the level of lamina cribrosa. ***(Anderson and Henrickson 1974)***

Later it was confirmed that both orthograde and retrograde axonal flows are blocked at the posterior border of lamina cribrosa after acute elevation of intraocular pressure. ***(Minkler et al., 1978)***

The degree of block increases with the increase of intraocular pressure and blockade is reversible. The mechanism of this phenomenon is not yet known but several hypothesis have been suggested;