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AYURVEDIC PERSPECTIVE OF PRIMARY OPEN ANGLE GLAUCOMA W.S.R TO VATAJA TRUTEEYA PATALAGATA TIMIRA

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ABSTRACT

Glaucoma is a group of disorders characterized by a progressive optic neuropathy which ultimately results in irreversible blindness. At present, there is no way to functionally restore vision lost from glaucoma but regenerative therapy may be possible in the future. The goal of medical treatment for glaucoma is to lower an individual's eye pressure to a level that preserves visual function to reduce morbidity such as diminished psychosocial functioning and falls, while maintaining a good quality of life. Modern treatment aims at controlling IOP but yet the disease progresses even when IOP is under control. And hence modern treatment is looking at neuroprotective strategies. POAG can be considered as *margavarana janya vata vyadhi* in Ayurveda and correlated to *truteeya patalagata timira*. Ayurveda treatment aims at both *srotoshodhana* and *chakshushya rasayana* which helps prevent further glaucomatous neuropathy changes. Here is an attempt made for understanding the pathogenesis and treatment of POAG in Ayurveda.

KEYWORDS: POAG, *vataja timira*, *chakshushya rasayana*, *truteeya patalagata timira.*

INTRODUCTION

Glaucoma is a leading cause of blindness in the world, affecting almost 5% of persons >70 years. It is a progressive neurological disease of the retina characterized by optic nerve head remodeling, loss of optic nerve axons, and loss of retinal ganglion cells. These effects cause a slow progressive decline of vision, starting in the periphery, moving over time toward central vision, and resulting in complete vision loss.[1]

Primary open angle glaucoma is the most common type of glaucoma accounting for three-quarters (74%) of all glaucoma cases. A recent review estimated the global number of POAG cases in 2013 at 4million, rising to 53 million by 2020 due to population ageing.[2]

Glaucoma is a neurodegenerative disorder in which degenerating retinal ganglion cells (RGC) produce significant visual disability. Clinically, glaucoma refers to an array of conditions associated with variably elevated intraocular pressure (IOP) that contributes to RGC loss via mechanical stress, vascular abnormalities, and other mechanisms, such as immune phenomena.[3]

At present, there is no way to functionally restore vision lost from glaucoma but regenerative therapy may be possible in the future. The goal of medical treatment for glaucoma is to lower an individual's eye pressure to a level that preserves visual function to reduce morbidity such as diminished psychosocial functioning and falls, while maintaining a good quality of life. An alternate way of looking at target IOP is to follow a more dynamic treatment paradigm in which, instead of setting a periodically modified target IOP goal, a clinician makes an assessment on each patient visit regarding whether lowering a patient's IOP with additional therapy is justified on the basis of the risk-benefit tradeoff at a given point in time based on a variety of patient-specific factors. With multiple OCT measurements of the retinal nerve fiber layer and visual field index information for individually calculated rates of progression, therapy can be personalized to each patient.[4]

Here is an attempt made in understanding the Ayurvedic approach in the management of POAG W.S.R to *vataja truteeya patalagata timira* in which field defects are explained.

AIMS AND OBJECTIVES

1. To study pathophysiological aspects of POAG in Ayurveda and modern medicine.
2. To determine the treatment aspects for POAG in Ayurveda.

MATERIALS AND METHODS

Literature reviewed from ancient Ayurveda classical texts, contemporary science and online sources.

Etiopathogenesis of glaucoma[5]

Etiological factors

Factors involved in the etiology of retinal ganglion cell death and thus in the etiology of glaucomatous optic neuropathy can be grouped as below:

A. Primary insults

1. Raised intraocular pressure (Mechanical theory). Raised intraocular pressure causes mechanical stretch on the lamina cribrosa leading to axonal deformation and ischaemia by altering capillary blood flow. As a result of this, neurotrophins (growth factors) are not able to reach the retinal ganglion cell bodies in sufficient amount needed for their survival.
2. Pressure independent factors (Vascular insufficiency theory). Factors affecting vascular perfusion of optic nerve head in the absence of raised IOP have been implicated in the glaucomatous optic neuropathy in patients with normal tension glaucoma (NTG). However, these may be the additional factors in cases of raised IOP as well. These factors include:

i. Failure of autoregulatory mechanism of blood flow. The retina and optic nerve share a peculiar mechanism of autoregulation of blood flow with rest of the central nervous system. Once the autoregulatory mechanisms are compromised, blood flow may not be adequate beyond some critical range of IOP (which may be raised or in normal range).

ii. Vasospasm is another mechanism affecting vascular perfusion of optic nerve head. This hypothesis gets credence from the convincing association between NTG and vasospastic disorders (migranous headache and Raynaud's phenomenon).

iii. Systemic hypotension particularly nocturnal dips in patients with night time administration of antihypertensive drugs has been implicated for low vascular perfusion of optic nerve head resulting in NTG.

iv. Other factors such as acute blood loss and abnormal coagulability profile have also been associated with NTG.

B. Secondary insults (Excitotoxicity theory)

Neuronal degeneration is believed to be driven by toxic factors such as glutamate (excitatory toxin), oxygen free radicals or nitric oxides which are released when RGCs undergo death due to primary insults. In this way the secondary insult leads to continued damage mediated apoptosis, even after the primary insult has been controlled.

Etiopathogenesis

Etiopathogenesis of POAG is not known exactly. Some of the known facts are as follows:

(A) Predisposing and risk factors: These include the following:

1. Intraocular pressure is the most important risk factor for development of POAG.
2. Family history (heredity): the approximate risk of getting disease is 10% in the siblings and 4% in the offspring of patients with POAG. POAG has a polygenic inheritance, approximately two dozen loci have been identified for POAG out of which only following three genes have been coloned:

- Myocilin C (MYOC)
- Optineurin (OPTN)
- WD repeat domin 36 (WDR 36)

3. Age: the risk increases with increasing age. The POAG is more commonly seen in elders between 5th and 7th decades.
4. Race. POAG is significantly more common, develops earlier and is more severe in black people than in white.
5. Myopes are more predisposed than the normals.
6. Central corneal thickness (CCT): A thinner CCT, apart from causing underestimation of IOP by applanation tonometer, is being considered as an independent risk factor for POAG.
7. Diabetics have a higher prevalence of POAG than non-diabetics.
8. Cigarette smoking is also thought to increase its risk.
9. High blood pressure is not the cause of rise in IOP however the prevalence of POAG is more in hypertensives than the normotensives. Most meaningful blood pressure variable related to glaucoma diastolic perfusion pressure (diastolic blood pressure-IOP). A diastolic perfusion pressure of <55mmHg is an important risk factor for glaucoma.
10. Thyrotoxicosis is also not the cause of rise in IOP, but the prevalence of POAG is more in patients suffering from Graves' ophthalmic disease than the normals.
11. Corticosteroid responsiveness. Patients with POAG and their offspring and sibilings are more likely to respond to six weeks topical steroid therapy with a significant rise of IOP.

(B) Pathogenesis of rise in IOP: It is certain that rise in IOP occurs due to decrease in the aqueous outflow facility. Recently it has been proposed that reduced aqueous outflow facility occurs due to failure of aqueous outflow pump mechanism owing to trabecular meshwork stiffening and apposition of Schlemm's canal wall. Such changes are caused by:

- Thickening and sclerosis of trabecular meshwork with faulty collagen tissue.
- Narrowing of intertrabecular spaces.
- Deposition of amorphous material in the juxtacanalicular space.
- Collapse of Schlemm's canal and absence of gaint vacuoles in the cells lining it.

The exact cause of these changes is uncertain. Detection of increased gammaglobulin and plasma cells in trabecular meshwork on immunohistochemistry and positive antinuclear, antibody reaction in some cases support an immunogenic mechanism in POAG.

(C) Pathogenesis of optic neuropathy: It has now been recognized that progressive optic neuropathy results from the death of retinal ganglion cells (RGCs) in a typical pattern which results in characteristic optic disc appearance and specific visual field defects.

Pathogenesis of retinal ganglion cell death: Retinal ganglion cell (RGC) death is initiated when some pathologic event blocks the transport of growth factors (neurotrophins) from the brain to the RGCs. The blockage of these neurotrophins initiates a damaging cascade, and the cell is unable to maintain its normal function. The RGCs losing their ability to maintain normal function undergo apoptosis and also trigger apoptosis of adjacent cells. Apoptosis is a genetically controlled cell suicide programme whereby irreversibly damaged cells die, and are subsequently engulfed by neighbouring cells, without eliciting any inflammatory response.

Retinal ganglion cell death is, of course, associated with loss of retinal nerve fibres. As the loss of nerve fibres extend beyond the normal physiological overlap of functional zones the characteristic optic disc changes and specific visual field defects become apparent over the time.

Clinical Features[6]

Symptoms

1. Asymptomatic: The disease is insidious and usually asymptomatic, until it has caused a significant loss of visual field. Therefore, periodic eye examination is required after middle age.
2. Headache and eyeache of mild intensity may be experienced in the course of the disease.
3. Scotoma may be noticed occasionally, by some observant patients.
4. Difficulty in reading and close work often persistently increasing, is experienced by most patients. This occurs due to increasing accommodative failure as a result of constant pressure on the ciliary muscle and its nerve supply. Therefore, patients usually complain of frequent changes in presbyopic glasses.
5. Delayed dark adaptation may develop, a disability which becomes increasingly disturbing in the late stages.
6. Significant loss of vision and blindness is the end result of untreated cases of POAG.

Signs

I. **Anterior segment signs:** Ocular examination including slit-lamp biomicroscopy may reveal normal anterior segment. In late stages pupil reflex becomes sluggish and cornea may show slight haze. A low (<555µm) central corneal thickness (CCT) is a significant risk factor for POAG.

II. **Intraocular pressure changes:** In the initial stages the IOP may not be raised permanently, but there is an exaggeration of the normal diurnal variation. Therefore, repeated observations of IOP (every 3-4 hour), for 24 hours is required during this stage (Diurnal variation test). In most patients IOP falls during the evening, contrary to what happens in closed angle glaucoma. A variation in IOP of over 5 mm Hg (Schiotz) is suspicious and over 8 mm of Hg is diagnostic of glaucoma. In later stages, IOP is permanently raised above 21 mm of Hg and ranges between 30 and 45 mm of Hg.

III. **Optic disc changes:** Optic disc changes, usually observed on routine fundus examination, provide an important clue for suspecting POAG. These are typically progressive, asymmetric and present a variety of characteristic clinical patterns. It is essential, therefore, to record the appearance of the nerve head in such a way that will accurately reveal subtle glaucomatous changes over the course of follow-up evaluation.

Optic disc changes

Glaucomatous changes in the optic disc can be described as early changes, advanced changes and glaucomatous optic atrophy.

(a) Early glaucomatous changes should be suspected to exist if fundus examination reveals one or more of the following signs:

1. Vertically oval cup due to selective loss of neural rim tissue in the inferior and superior poles.
2. Asymmetry of the cups. A difference of more than 0.2 between two eyes is significant.
3. Large cup i.e., 0.6 or more (normal cup size is 0.3 to 0.4) may occur due to concentric expansion.
4. Splinter hemorrhages present on or near the optic disc margin.
5. Pallor areas on the disc.
6. Atrophy of retinal nerve fibre layer which may be seen with red free light.

(b) Advanced glaucomatous changes in the optic disc:

1. Marked cupping (cup size 0.7 to 0.9), excavation may even reach the disc margin, the sides are steep and not shelving (c.f. deep physiological cup).
2. Thinning of neuroretinal rim which occurs in advanced cases is seen as a crescentric shadow adjacent to the disc margin.
3. Nasal shifting of retinal vessels which have the appearance of being broken off at the margin is an important sign (Bayonetting sign). When the edges overhang, the course of the vessels as they climb the sides of the cup is hidden.
4. Pulsations of the retinal arterioles may be seen at the disc margin (a pathognomic sign of glaucoma), when IOP is very high.

5. Lamellar dot sign the pores in the lamina cribrosa are slit-shaped and are visible up to the margin of the disc.

(c) Glaucomatous optic atrophy. As the damage progresses, all the neural tissue of the disc is destroyed and the optic nerve head appears white and deeply excavated.

IV. Visual field defects[7]

Visual field defects usually run parallel to the changes at the optic nerve head and continue to progress if IOP is not controlled. These can be described as early and late field defects.

Progression of field defects. Visual field defects in glaucoma are initially observed in Bjerrum's area (10-25 degree from fixation) and correlate with optic disc changes. The natural history of the progressive glaucomatous field loss, more or less, takes the following sequence:

1. Isopter contraction. It refers to mild generalized constriction of central as well as peripheral field. It is the earliest visual field defect occurring in glaucoma. However, it is of limited diagnostic value, as it may also occur in many other conditions.
2. Baring of blind spot. It is also considered to be an early glaucomatous change, but is very non-specific and thus of limited diagnostic value. Baring of the blind spot means exclusion of the blind spot from the central field due to inward curve of the outer boundary of 30° central field.
3. Small wing-shaped paracentral scotoma. It is the earliest clinically significant field defect. It may appear either below or above the blind spot in Bjerrum's area (an arcuate area extending above and below the blind spot to between 10o and 20o of fixation point).
4. Seidel's scotoma.With the passage of time paracental scotoma joins the blind spot to form a sickle shaped scotoma known as Seidel's scotoma.
5. Arcuate or Bjerrum's scotoma. It is formed at a later stage by the extension of Seidel's scotoma in an area either above or below the fixation point to reach the horizontal line (Fig. 9.13D). Damage to the adjacent fibres causes a peripheral breakthrough.
6. Ring or double arcuate scotoma. It develops when the two arcuate scotomas join together.
7. Roenne's central nasal step. It is created when the two arcuate scotomas run in different arcs and meet to form a sharp right-angled defect at the horizontal meridian.
8. Peripheral field defects. These appear sometimes at an early stage and sometimes only late in the disease. The peripheral nasal step of Roenne's results from unequal contraction of the peripheral isopter.
9. Advanced glaucomatous field defects. The visual field loss gradually spreads centrally as well as peripherally, and eventually only a small island of central vision (tubular vision) and an accompanying temporal island are left. With the continued damage, these islands of vision also progressively diminish in size until the tiny central island is totally extinguished. The temporal island of the vision is more resistant and is lost in the end leaving the patient with no light perception.

Treatment

Therapeutic choices include:

- Medical therapy
- Argon or diode laser trabeculoplasty and
- Filteration surgery.

But all these have certain complications like haemorrhage, uveitis, peripheral anterior synechiae, reduced accommodation etc.

Truteeya patalgata timira

According to *Sushruta acharya*, when the *doshas* invade the third layer, the person will see objects present above but not those present below, sees even big objects as though covered with cloth, sees the face of others as though devoid of ears, nose and eyes; as the *doshas* become more aggravated, disorders of vision also increase; when the *doshas* are localized at the bottom the person will not see objects; when localized in the sides he will not see objects as though combined; when localized in the centre, he finds one object as two, two objects as three; when the *doshas* are not localized(but are moving from place to place) he sees one object as many.[8]

According to *Vagbhatta acharya*, person sees objects present above but not those present below, objects are seen as covered by thin cloth. And few of the *sushruta* symptoms of *third patalagata timira* are included in second *patalagata timira*. And while explaining *vataja timira*, he has mentioned that the person sees the objects as though covered (with thin cloth), unsteady, dirty, slightly red, sometimes and some other times as clear and clean. And when *kacha* has developed, the area of vision is slightly red, the person sees the face as noseless, sees the moon, lamp etc. as many, understands curved things as straight; *kacha* when grows older, makes the sight of objects as though covered with dust and smoke, of well defined red colour, wide spread or small in size and loss of vision.[9]

Samprapti

Achakshushya nidana sevana

↓

Leads to *vata pradhana tridosha prakopa*

↓

Jatharagni mandya

↓

Jatharagni mandya janya aama

↓

Urdhwagamana of *doshas* through *siras*

↓

Sthanasamshraya in *netra patalas*

↓

Sthanika dhatwagni mandya

↓

Dhatwagni mandya janya aama

↓

Sanga and *vimargagamana* of *ama* causing *srotoavarodha*

↓

Margavarana

↓

Vataja timira manifesting in *truteeya patala*

- Understanding in terms of *rasa dushti* w.r.t *aharaja nidana*:

Rasa dhatu is formed from *annarasa* from which *avalambaka kapha* is initially formed. It nourishes the other *kaphas*. If this is not formed properly, then *tarpaka kapha* also gets affected i.e. more of *kitta bhaga*(waste byproduct) gets merged with *saara bhaga*(essence) producing defective *rasa*. This defective *rasa* would not meet the normal functions of *rasa* and inturn leads to *meda, majja dhatu* vitiation which gives rise to *timira darshana*.

- *Viharaja nidana*: According to *Ashtanga Sangraha*, in *nidanasthana* 16th chapter he has mentioned that the *nidanas* like *rukshata*, *vyayama*, *langhana*, *ati ahara*, *abhighata*, *adhwa*, *vega udeerana dharana* leads to *prana vata dushti* leading to *chakshu upaghata*.

- Also due to *dhatu kshaya*, the *uttarottara dhatus* are not formed properly where again *meda majja dhatu kshaya* is seen in which *tarpaka kapha* gets affected and hence affecting the nutrition of all the structures of the eye because the nutritive property of *kapha* is *anugraha* to the sense organs through circulation influenced by *vyana vata*.[10]

Samprapti ghataka

Dosha:	*Prana vata, vyana vata, alochaka pitta, tarpaka kapha*
Dooshya	*Rasa, rakta, mamsa, meda, asthi majja*
Agni	*Jatharagni, dhatwagni*
Ama	*Jatharagni mandya janya, dhatwagni mandya janya*
Srotas	*Rasavaha, raktavaha*
Srotovaha dushti	*Sanga*
Rogamarga	*Madhyama*
Udbhava sthana	*Amashaya*
Adhishtana	*Netra*
Sanchara sthana	*Rupavaha siras*
Vyakta sthana	*Drushti mandala*
Sadhyaasadhyata	*Yapya*
Vyadhi swabhava	*Chirakaaleena*

Chikitsa

General measures like *snehana* (oleation), *rakta visravana* (blood letting), *nasya*(nasal medication), *anjana* (collyrium), *murdha basti* (making medicated oil stand on the head for sometime), *basti* (rectal enema), *tarpana* (nourishing of the eyes therapy), *lepa* (topical application) and *seka* (pouring liquids on the body parts) – should be administered as suitable to the *doshas*.[11] And different formulations have been explained for the same.

For *vataja timira*, medicated ghee prepared with the *dashamoola kwatha*, 4 parts of milk, paste of *sreshtha* should be consumed. Next, *triphala* and *panchamula* added with milk and *eranda taila* should be administered to produce purgations.[12]

DISCUSSION

Owing to the etiopathogenesis of POAG, it can be correlated to the *vataja truteeypatalagata timira* where visual defects are explained. The aqueous humour is considered as the *rasa dhatu* for it nourishes the other avascular structures and fills anterior and posterior chamber (It fulfills the functions of *rasa* like *aharahar gacchati, aharahar yaapayati* and *aharahar dharayati*) and with the help of *vyana vayu* it moves to other structures of the eye and nourishes them.

According to *Sushruta acharya*, after 40 years *ishat parihani* of all the *avayas* begin.[13] Due to the *vata vruddhi* (mainly as the age progresses and due to the lifestyle adopted), changes are seen in the ocular structures of the filtering regions like Trabecular meshwork, Schlemm's canal, Episcleral veins (along with non- filtering regions too). This will lead to the reduced aqueous outflow.

The main cause of elevated IOP in primary open angle glaucoma (POAG) is thought to be an increased outflow resistance via the pressure-dependent trabecular outflow system by an increased accumulation of extracellular matrix (ECM) material in the trabecular meshwork, due to a disturbed balance between ECM deposition and degradation.[14]

Some studies suggest that glycosaminoglycans, which constitute the fundamental substance of the ECM of the TM, are partly responsible for increased resistance to outflow. The osmotic forces exerted by glycosaminoglycans may induce hydration (edema) of the TM, which can cause obstruction of the trabecular structure. Catabolic enzymes released from lysosomes depolymerize glycosaminoglycans and prevents this obstruction. This effect is also inhibited by corticosteroids, which prevent the release of the enzymes by stabilizing the lysosomal membranes and has been associated with a role in outflow obstruction and glaucoma pathogenesis. In glaucomatous eyes, an increase in the ECM thickness beneath the inner wall of Schlemm's canal and in the juxtacanalicular meshwork compared with age-matched healthy controls has been observed. Other studies suggest that the interaction of ECM components with different proteins may induce formation of deposits that obstruct aqueous humor outflow through the TM.[15]

The accumulation of these glycosaminoglycans and ECM in the form of *mala* leads to obstruction of the *srotas* i.e., the normal flow of aqueous is disturbed which indicates *vyana vayu dushti*; hampering its *rasa vikshepana karma*. This is understood as *rasapradoshaja vikara* in which *srotorodha* is one of the *lakshanas.* And further kapha gets dushti. And the increased ECM deposition in the outflow pathway can be considered as *aama*. The *srotodushti prakara* is *sanga* as there is resistance to the aqueous outflow.

This hampers the aqueous homeostasis, which subsequently cascades into glaucoma and its sequelae i.e, the changes of ECM at optic nerve head can be understood as *vimargagamana*.

The theories put forth, factors like elevated intraocular pressure (IOP) which causes mechanical damage to RGC axons in the region of optic nerve head which progresses to retrograde damage of the RGC body and damage to RGC bodies' leads to enhanced secretion of MMPs, which in turn causes ECM changes and apoptosis.Vascular dysregulation primarily contribute to the initial insult during glaucomatous atrophy in the form of obstruction to axoplasmic flow within the RGC axons at the lamina cribrosa, altered optic nerve microcirculation at the level of lamina and changes in the laminar glial and connective tissue. The factors leading to secondary insult include excitotoxic damage caused by glutamate or other toxins released.

This can be understood in 2 ways: the IOP related pathology can be understood as *margavarana janya vyadhi* and pathology due to vascular dysregulation can be considered with respect to *margavarana* elsewhere in the body or due to *dhatu kshaya* which is effecting the circulation at the level of optic nerve leading to deprivation of neurotrophic signals i.e., *abhava* of these growth factors is seen. Hence all these factors prevent *indriyartha sannikarsha* that is *prana vata* does the *indriya* and *buddhi dharana*, the compression of the nerve fibres can be attributed to *prana vata dushti* where different optic disc signs can be seen. And it does *dhamani dharana* where *dhamani* can be considered as nerves and the conductivity of their signals to and from the brain is disturbed leading to visual disturbances.

This *truteeya patalagata timira* can be considered under the *margavaranjanya vata vyadhi*. i.e., in C*haraka samhita chikitsa sthana*, the samprapti of *vatavyadhi* is that when *vata* gets aggravated, it occupies the susceptible srotas and cause *vatavyadhi*. And purvaroopas remain avyakta.

The *prana vata* is the one which moves upwards (*prano atra murdhaga*) i.e., the conductivity of neural signals to the brain can be attributed to *prana vata*. That is due to the mechanical damage of RGC axons by the compression of the optic nerve, *prana vata dushti* occurs. i.e, the function of *vata- kriyanam apratighatam amoha buddhikarmanam*(*su.sha.*7/8) is not seen. The blood circulation is mainly by *vyana vata.* Due to the *prana vata dushti* there will be abnormal dilatation and constriction of vessels which leads to *vyana vayu dushti*

where *rasa vikshepana* is hampered i.e., axonal deformation and ischemia by altering capillary blood flow. . This inturn leads to *rasa* and *rakta kshaya*. Also there will be failure of autoregulatory mechanism of blood flow (*khale kapota nyaya*). Because of this there is no *poshana* to the *dhatus* by the *tarpaka kapha* (which is referred to the neurotropins). This inturn leads to *sanchaya* of *malarupi kapha*. (Toxic nitric oxide, glutamate, free radicles). Henceforth, they lose the ability to maintain normal function and undergo apoptosis which induces further apoptosis of adjacent cells. This leads to the visual field defects. These are explained in the *truteeya patalagata timira* which says the defects in the the superior part leads to lower vision loss and vice versa. And if the defect is present in nasal side, temporal side is not seen and vice versa.

Adopting treatment

Ayurveda aims at maintaining the health of healthy individual and curing the disease of the afflicted one. Hence treatment can be of preventive and curative type, where preventive aspects if adopted in daily life helps prevent any of the *netra rogas*. Also when included during treatment phase, they promote the treatments and further progression of the disease can be prevented.

1. Preventive aspect

According to a study conducted, it is reported that "A continuous decrease in trabecular meshwork cellularity normally occurs throughout life. This decline represents a loss of approximately 43% of the trabecular cell density between the ages of 20 and 80 or a loss of 58% of the trabecular cell density from birth to 81 years of age."[16] In a healthy eye, a constant flow of blood is required in the retina and optic nerve head so as to meet the high metabolic needs in these vital parts of the eye. To maintain a constant rate of blood flow an efficient autoregulatory mechanism operates in arteries, arterioles and capillaries over a wide range of day-to-day fluctuations in ocular perfusion pressure that is dependent on both the systemic blood pressure and IOP. These autoregulatory mechanisms are not as robust in aging individuals as in youth. Thus, deficient autoregulatory mechanisms leading to ischemia contribute to the development of glaucomatous neuronal damage with increasing age.[17]

According to *Sushruta acharya*, nourishment of eye and prevention of *timira* is possible with the following *pathyas* mentioned under *timira pratisheda adhyaya*.[18]

- *Purana ghrita*, *triphala*, *shatavari*, *patolapatra*, *mudga*, *amalaki*, *yava* – the one who consumes these will not have fear of *timira*.
- Consuming daily - milk processed with *shatavari*/*amalaki kalka* or *swarasa*, mixing of excess quantity of *ghrita in triphala kashaya*, *odana* prepared by mixing *yava* in water and adding excess quantity of ghee.
- *Jeevanti shaaka*, *sunishannaka*, *tanduliyaka*, *vaastuka*, *chilli*, *moolakapotika*, *mamsa* of *jaangala pakshi* – are all *hita* for *drishti*.
- *Patola*, *karkotaka*, *karavella*, *vaartaku*, *tarkari*, *kareera phala*, *shigru aartagala* – frying of all these in ghee are *drishti hita*.

Similarly According to *Ashtanga hrudaya*, use of *triphala*, *rakta visravana* (blood letting), *shodhana* (purificatory procedures), withdrawing the mind from sensual actions, use of *anjana* (collyrium), *nasya* (nasal medication), consuming meat of birds, worshipping the feet like anointing feet, using footwear etc. and regular use of ghee protects the eyes always. Abstaining always from ingestion of unsuitable foods, from observing things which are very shining, quick moving and minute are advised in order to protect the eyes.[19]

Following of the *dinacharya* and *rutucharya* helps to maintain the healthy structures and removal of the *avarodha* of the *srotas* timely. Also following of the *kriyakalpas* like *anjana*, *tarpana* maintains the eye health and prevents diseases.

Curative aspect

It is considered as *margavaranajanya vatavyadhi*. The treatment should be selected based on the *doshas* involved in the *avarana* w.r.t mechanical cause or vascular factor. Under *Charaka Samhita Chikitsa Sthana* 28th chapter, while explaining the *pranavrita vyana vata*, he has mentioned the "*sarvendriya shoonyatwa*" and *bala kshaya*. And he further adds to adopt *urdhwajatrugata karmas* for the same.

For *dhatukshaya janya*, *deepana pachana* can be done and then *snehana* line of treatment can be adopted.

And in *margavarana janya timira*, main aim is to relieve *avarana* first and once relieved *snehana* line of treatment can be adopted. The initial step is to achieve *sthanika* and *deha shodhana*. For the same *virechana* and *shirovirechana* procedures can be adopted by selecting the drugs in a way which wouldn't provoke *vata* and clear the *avarodha* too. Generally for *koshta shodhana*, *eranda paya* can be given as advised for *vataja timira*. And *navana nasya* can be adopted with *anutaila*. Different procedures and medications can be used according to *rogi roga bala*. Even the *rukshana karmas* like *udvartana* can be adopted to relieve the *srotoavarodha* and then depending on the *lakshanas* further procedures can be administered. Further treatment can be concentrated on *kriyakalpa* procedures like *tarpana* where selective drugs are used to act as both *srotoshodhaka* and *vatahara* e.g. *purana ghrita*. Once the *srotoshodhana* is achieved one can go for the *snehana* lines of treatment like *murdhni tailas* based on *yukti*. But it shouldn't be given at the initial stage when the *avarodha* persists. And the use of *chakshushya rasayanas* which are in the neurodegenerative conditions

act best. Further the general lines of *vatavyadhi* which are suitable for *timira pratishedha* should be used.

CONCLUSION

POAG can be related to *vataja truteeya patalagata timira* in Ayurveda. Though there is no direct reference, it can be understood by the visual field effects and the *patala* involved. Hence to understand the pathogenesis in terms of *margavarana janya* or *dhatu kshayajanya* becomes inevitable with a view to adopt the appropriate treatment. In *truteeya patalagata timira*, for *margavarana janya timira*, treatment is aimed at clearing the *avarana* and then treating the *vata* whereas in *dhatukshayajanya*, *snehana* line of treatments should be adopted.

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