

Special Edition  
"Recent Advances in the Diagnosis  
and Management of Glaucoma"

## Editorial

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# An Introduction to Glaucoma

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Glaucoma is a disease known to mankind since antiquity. The word glaucoma is derived from the Greek word "glaukos". When we look into the pupil of an individual who has no ocular disease, we see a black pupillary reflex. However, patients with "glaukos" are said to have a greenish or bluish hue. Hippocrates, in his aphorisms in 400 BC, also used the term to describe "a kind of blindness which came with aging and was associated with a glazed look of the pupil". However, when we critically analyze the definition of "glaukos", we can conclude that this glazed look could have come from glaucoma but also seen in patients with cataract.

It was only in the 10<sup>th</sup> century AD that the Arabic scholar Abul Hasan Al-Tabari, in his "Book of Hippocratic Treatments", suggested the association of glaucoma with high intra-ocular pressure (IOP). Subsequently, in 1622, Richard Bannister, an English oculist described glaucoma as a triad. According to him, glaucoma is characterized by: raised IOP, increased cup: disc ratio (C:DR) and visual field defects. Thus, the main etiological factor in the pathogenesis of glaucoma was assumed to be only high IOP.

Later on, we came to know that glaucoma can occur even when the IOP is in a statistically normal range. This condition is called normal tension glaucoma (NTG) or low tension glaucoma (LTG). It was also seen that despite lowering of IOP some patients continued to progress. Conversely, some individuals with IOP above 21 mmHg do not show glaucomatous changes. This condition is called ocular hypertension. This shows that there are other factors apart from IOP which can contribute to the development of glaucomatous optic atrophy (GOA). Therefore, now glaucoma is defined as a multi-factorial neurodegenerative disorder. IOP however remains the only risk factor which can be controlled.

Glaucoma is widely present across the world. According to the World Health Organization (WHO), glaucoma is the second leading cause of blindness in the world. Estimates suggest the total number of suspected cases of glaucoma at over 60 million worldwide. The global prevalence rate for glaucoma in the age group 40-80 years is 3.54%. The prevalence of primary open angle glaucoma (POAG) is highest in Africa at 4.20% and the prevalence of primary angle closure glaucoma is highest in Asia (1.09%). Globally, the number of people with glaucoma in 2013 was estimated to be 64.3 million; this is expected to increase to 76.0 million in 2020 and 111.8 million by 2040.<sup>1-4</sup>

The pathogenesis of glaucoma is attributed to a number of factors and various theories have been presented to explain the causation of GOA. These theories include the following:

1. Mechanical theory
2. Vascular theory
3. Biochemical theory
4. Genetic theory

The mechanical theory is based on the assumption that high IOP causes mechanical compression of the optic nerve head (ONH). It suggests that distortion and displacement of the lamina cribrosa in the ONH causes damage to the axons, resulting in blockade of axonal transport and later axonal death.

The vascular theory attempts to explain glaucoma causation on the basis of factors such as: reduced perfusion pressure to the optic nerve, faulty vascular auto regulation or loss of neurovascular coupling.<sup>5</sup>

Certain biochemical molecules are also presumed to play a vital role in the causation of GOA. The biochemical mechanisms postulated to play a role include: excitatory amino acids, caspases, protein kinases, oxygen free radicals, nitric oxide, tumor necrosis factor- $\alpha$ , neurotrophins and matrix metalloproteins.<sup>6</sup>

Genetic linkage studies on familial and congenital glaucoma have revealed a handful of genes with a very strong effect on the disease. These include the *MYOC* and *OPTN* genes for familial POAG and *CYP1B1* for congenital glaucoma. However, sporadic glaucoma occurring in the population has not shown any consistent genetic association.<sup>7</sup>

It is assumed that a number of different mechanisms are active in different sets of patients leading to GOA.

Glaucoma is a heterogeneous group of disorders. It can be categorized into various types by different classification methods. Based on etiology, glaucoma can be of primary or secondary forms. Primary glaucomas are those where the initial events leading to outflow obstruction and IOP elevation are confined to the anterior chamber angle (ACA) or conventional outflow pathways with no apparent contribution from other ocular or systemic disorders.

Glaucoma can be open angle, developmental or closed-angle type. Open angle glaucoma can be further divided into primary, secondary and NTG. Similarly, developmental and closed angle glaucomas can also be primary or secondary. Other classification methods are based on mechanisms, events or staging.

The diagnosis of glaucoma is facilitated by various investigations. These include:

1. Tonometry: Measurement of IOP
2. Gonioscopy: Assessment of anterior chamber angle (ACA)
3. Ophthalmoscopy: To measure C:DR
4. Pachymetry: Required to measure central corneal thickness
5. Perimetry: For visual field assessment
6. Imaging studies: Techniques like GDx, heidelberg retina tomograph (HRT) and optical coherence tomography (OCT) are used to study the optic nerve head and retinal layers.

Diagnosis of glaucoma is based on clinical judgment with prudent use of available investigation techniques to detect structural and functional damages. However, at times it may be difficult to reach a diagnosis of glaucoma and patients are kept in the category of “glaucoma suspect”.<sup>8,9</sup>

The management of glaucoma is still limited to control of IOP. This is regarded as the surrogate treatment goal in treatment of glaucoma. There are a number of methods available to reduce IOP.<sup>10,11</sup> These include the following:

1. Medical or pharmacologic
2. Lasers
3. Minimally invasive glaucoma surgeries (implants)
4. Glaucoma drainage devices (valved and non-valved)
5. Surgical methods
6. Cyclodestructive methods.

Glaucoma is a multifactorial disorder with the common denominator of retinal ganglion cell loss. There are a number of theories which attempt to explain the causation of this multi-spectrum disorder called “glaucoma”. However, the only risk factor which can be modulated presently is intra-ocular pressure (IOP). Thus, IOP is often used as a surrogate treatment goal to assess the efficacy of any glaucoma management strategy. It is necessary to fully understand the pathogenesis of glaucoma in order to manage this disease effectively.

## REFERENCES

1. Thylefors B, Négrel AD. The global impact of glaucoma. *Bull World Health Organ.* 1994; 72(3): 323-326. Web site. <http://www.who.int/blindness/publications/glaucoma/en/>. Accessed March 25, 2017.

2. Glaucoma Research Foundation (GRF). Glaucoma Facts and Stats. 2016. Web site. <http://www.glaucoma.org/glaucoma/glaucoma-facts-and-stats.php>. Accessed March 25, 2017.
3. Vajaranant TS, Wu S, Torres M, Varma R. A 40-year forecast of the demographic shift in primary open-angle glaucoma in the United States. *Invest Ophthalmol Vis Sci*. 2012; 53: 2464-2466. doi: [10.1167/iovs.12-9483d](https://doi.org/10.1167/iovs.12-9483d)
4. Tham YC, Li X, Wong TY, Quigley HA, Aung T, Cheng CY. Global prevalence of glaucoma and projections of glaucoma burden through 2040: A systematic review and meta-analysis. *Ophthalmol*. 2014; 121(11): 2081-2090. doi: [10.1016/j.ophtha.2014.05.013](https://doi.org/10.1016/j.ophtha.2014.05.013)
5. Ahmad SS. Controversies in the vascular theory of glaucomatous optic nerve degeneration. *J Ophthalmol*. 2016; 6(4): 182-186. doi: [10.1016/j.tjo.2016.05.009](https://doi.org/10.1016/j.tjo.2016.05.009)
6. Ahmad SS, Ghani SA, Rajagopal TH. Current concepts in the biochemical mechanisms of glaucomatous neurodegeneration. *J Curr Glaucoma Pract*. 2013; 7(2): 49-53. doi: [10.5005/jp-journals-10008-1137](https://doi.org/10.5005/jp-journals-10008-1137)
7. Aung T, Khor CC. Glaucoma genetics: Recent advances and future directions. *Asia Pac J Ophthalmol (Phila)*. 2016; 5(4): 256-259. doi: [10.1097/APO.0000000000000229](https://doi.org/10.1097/APO.0000000000000229)
8. Allingham RR, Damji KF, Freeman S, et al. *Shields Textbook of glaucoma*. 6<sup>th</sup> ed. Philadelphia, USA: Wolters Kluwer/Lippincott Williams & Wilkins; 2012.
9. Sharma P, Sample PA, Zangwill LM, Schuman JS. Diagnostic tools for glaucoma detection and management. *Surv Ophthalmol*. 2008; 53(Supp 1): S17-S32. doi: [10.1016/j.survophthal.2008.08.003](https://doi.org/10.1016/j.survophthal.2008.08.003)
10. Schwartz K, Budenz D. Current management of glaucoma. *Curr Opin Ophthalmol*. 2004; 15(2): 119-126. Web site. [http://journals.lww.com/co-ophthalmology/Abstract/2004/04000/Current\\_management\\_of\\_glaucoma.11.aspx](http://journals.lww.com/co-ophthalmology/Abstract/2004/04000/Current_management_of_glaucoma.11.aspx). Accessed March 25, 2017.
11. Shahid SM, Naqib SM, Crawley L. Management of glaucoma. *Brit Undergrad Jou Ophthal*. 2014; 1: 1-7. Web site. <http://bujo.buos.co.uk/documents/2/bujo.2014.004.pdf>. Accessed March 25, 2017.