

Differences between the characteristics of normal tension glaucoma and high tension glaucoma

Abstract

Normal tension glaucoma is a type of primary open angle glaucoma in which the intraocular pressure is in the normal range. Although both normal tension glaucoma and high-tension glaucoma resemble clinically each other, there are some differences between both of them. In this editorial, I aim to summarize the differences between the characteristics of these glaucoma types. If these are well-known, the diagnosis and management of normal tension glaucoma will be easier.

Keywords: normal tension glaucoma, normal pressure glaucoma, normotensive glaucoma, high-pressure glaucoma, high tension glaucoma, differences, characteristics

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Abbreviations: NTG, normal tension glaucoma; NPG, normal pressure glaucoma; POAG, primary open angle glaucoma; HPG, high pressure glaucoma; HTG, high tension glaucoma; RNFL, retinal nerve fiber layer; OD, optic disc; RGCs, retinal ganglion Cells; IOP, intra-ocular pressure; GON, glaucomatous optic neuropathy; CNTGS, collaborative normal tension glaucoma study; ODH, optic disc hemorrhage; CCT, central cornea thickness; CSFP, cerebro-spinal fluid pressure; ONPP, optic nerve perfusion pressure; PVD, peripheral vascular dysregulation; OBF, ocular blood flow; OSAS, obstructive sleep apnea syndrome

Editorial

Glaucoma is defined as a multifactorial and progressive optic neuropathy which usually caused by the effects of elevated

intraocular pressure (IOP). Glaucomatous optic neuropathy (GON) is characterized by the excavation of the optic nerve head (ONH), thinning of the retinal nerve fiber layer (RNFL) and the axons of by retinal ganglion cells (RGCs) and eventually the specific loss of visual field (VF).¹ Primary open-angle glaucoma (POAG) is the most common type of chronic progressive GON with the absence of the association of any ocular disease. POAG includes two types as ‘‘high pressure glaucoma (HPG)/high-tension glaucoma (HTG)’’ and normal-pressure glaucoma (NPG)/normal tension glaucoma (NTG).¹⁻⁵ There are significant overlapping characteristics between both glaucoma types. However, they may be separated by some marked differences in the aspects of risk factors, pathogenesis, OD findings, RNFL thickness and VF defects.⁶⁻⁴⁷ Comparison of the characteristics of both glaucoma types has been given in Table 1.

Table 1 The comparison of the characteristics of NTG and HTG^{1,2,4,8,9,11,12,15-20,22-47}

Characteristics	NTG	HTG
IOP	Normal with wide diurnal fluctuations, nocturnal spikes	High with normal range diurnal fluctuations
Sexual Predisposition	Female	N/A
Possible Specific Risk Factor	ODH, ischemic/occlusive vascular disease (Migraine, Raynaud, diabetes, cardiac arrhythmia, stroke) addition to elevated IOP	High IOP (primary continuous causative risk factor), Genetic mutation, advanced age, black race, older age, systemical vascular diseases
Age	Older average 10 years than HPG	Usually over 50 years old
OD Sign	Narrow NRR, a larger OD surface area, thinner inferior/inferotemporal NRR, deep, focal NRR thinning/notching, OD Diffuse NRR thinning, classical GON signs pit	
GON Pathogenesis	Neuro-degeneration, IOP-independent mechanisms (LC weakness or abnormalities, abnormal easily triggered apoptosis, low ONBP, low CSFP, local PVD, enhanced sensitivity to physiologic IOP, hypotension, hematologic abnormalities)	Neurodegenerative and IOP-dependent mechanisms, vascular, genetic, and biochemical mechanisms
PPA	More frequently beta-zone PPA	Beta-zone PPA
ONPP	Frequently lower	Maybe lower

Table Continued...

Characteristics	NTG	HTG
RNFLT Loss	Earlier, inferotemporal	N/A
VF Defects	More focal, central, deeper and closer to fixation	Deeper, superior nasal step, inferior and superior paracentral
Suggested VF testing protocol I0-2		30-2
CCT	Maybe thinner than average	N/A
Genetic Mutation in which	Optineurin	TIGR, Myocilin
Systemical BP	Increased diastolic BP and larger dips in BP overnight	N/A
ODH	More common	Less common
OBF	Strongly impaired	Maybe impaired
OSAS	Strongly associated	May be associated
Progression	Slower to HPG in the absence of beta-blocker treatment	N/A

NTG, normal tension glaucoma; HTG, high tension glaucoma; RNFLT; retinal nerve fiber layer thickness; VF, visual field; OD, optic disc; IOP, intra-ocular pressure; GON, glaucomatous optic neuropathy; NRR, neuro-retinal rim; ODH, optic disc hemorrhage; CCT, central cornea thickness; PPA, peripapillary atrophy; OBF, ocular blood flow; cerebro-spinal fluid pressure; ONPP, optic nerve perfusion pressure; OSAS, obstructive sleep apnea syndrome; HRT, heidelberg retinal tomography; BP, blood pressure; LC, lamina cribrosa; BP, blood pressure

It has been considered that NTG might occur due to IOP-independent pathogenic factors such as peripheral vascular dysregulation (PVD), hypotension, mechanical factors such as lamina cribrosa (LC) abnormalities and weakness, and enhanced sensitivity of the optic nerve to physiologic IOP. However, CNTGS showed that a 30% or more reduction of the IOP value in normal range belonging the patient achieved a significant slowdown in the progression of the NTG.⁶ On the other hand, HTG is often an IOP-dependent glaucoma type. IOP plays a great role in the pathogenesis of this glaucoma typ.¹ In conclusion, as seen in Table 1, compared to those with HTG, the common distinctive findings in the patients with NTG are an IOP value in normal range; female predisposition; optineurin gene mutation; thinner CCT; larger OD size; larger/deeper OD cupping; thinner LC; frequent NRR notching; earlier and focal RNFLT loss; frequent beta zone peripapillary atrophy; frequent OD hemorrhage; frequent pit; deeper, closer to fixation, steeper slopes, more localized and central VF defects; peripheral vascular dysregulation (low nocturnal optic nerve perfusion pressure, cold extremities, impairments in the nail fold capillary, retinal and choroidal blood flow), ocular perfusion abnormalities and vasospastic/ischemic disorders in pathogenesis; associated hematologic abnormalities, obstructive sleep apnea syndrome and blood loss.^{2,3,7-47}

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Conflicts of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

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