OCULAR HYPERTENSION: ETIOPATHOGENESIS, STATISTICAL ANALYSIS, AND THE RISK OF TRANSFORMATION INTO GLAUCOMA

S.Fazildinova
Superviser: Teacher of CAMU, International Medical University
sevarafazildinova6@gmail.com
+998915410377
Ahmadjon Yo'ldoshaliyev
student of CAMU, International Medical University
yoldoshaliyevahmadjon@gmail.com
+998-90-058-08-13

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Abstract: This article examines the concept of ocular hypertension (OHT), its epidemiological indicators, and its impact on the visual apparatus. The risk factors associated with increased intraocular pressure (IOP) and their clinical significance are highlighted based on global statistical data.

Keywords: Ocular hypertension, Intraocular pressure (IOP), Glaucoma progression, OHTS, Optic nerve head, Tonometry, CCC

Ocular hypertension (OHT) is defined as a clinical condition where intraocular pressure (IOP) consistently exceeds the statistical norm of 21 mmHg in one or both eyes, in the absence of detectable optic nerve damage or visual field loss [1;1711]. In modern ophthalmology, OHT is classified as a *"pre-glaucomatous"* state, requiring rigorous monitoring to prevent irreversible vision loss.

The prevalence of ocular hypertension increases significantly with age. According to global statistics, approximately 4.5% to 9.4% of the population over the age of 40 exhibit elevated IOP levels [2;262].

The Ocular Hypertension Treatment Study (OHTS), one of the most comprehensive clinical trials in the field, provides critical evidence regarding the progression of this condition. In the absence of medical intervention, there is a 9.5% cumulative probability of OHT progressing to primary open-angle glaucoma within 5 years [3;703]. The use of topical hypotensive medication to reduce IOP by approximately 20% lowers this progression risk to 4.4% [3;710].

Patients with a central corneal thickness (CCT) of less than 555 microns have a three-fold higher risk of developing glaucoma compared to those with thicker corneas (above 588 microns) [4;719]. Pathophysiological Mechanisms OHT the elevation of IOP is primarily governed by the balance between aqueous humor production and its drainage. Two main factors contribute to this imbalance. Hydrodynamic dysfunction is where excessive secretion of aqueous humor by the ciliary body [1;1715].

In outflow resistance: there will be observed increased resistance in the trabecular meshwork and Schlemm's canal. This leads to elevated hydrostatic pressure, which exerts mechanical stress on the retinal ganglion cells and the optic nerve head [5;213].

Talking about diagnostics and differential analysis we can consider accurate diagnosis requires more than simple tonometry. Modern clinical protocols must include.

Corneal pachymetry, the precise measurement of central corneal thickness (CCT), serves as a critical diagnostic parameter in modern ophthalmology, particularly in the management of glaucoma and ocular hypertension. The fundamental clinical relevance of CCT lies in its significant

influence on the accuracy of Goldmann Applanation Tonometry (GAT), which remains the global gold standard for measuring intraocular pressure (IOP).

According to the principles of the Imbert-Fick law, GAT assumes a uniform corneal thickness; however, structural variations among individuals introduce systematic measurement errors. A thin cornea possesses less structural resistance to applanation, which frequently leads to an underestimation of the true manometric IOP, potentially masking a diagnosis of glaucoma. Conversely, a thick cornea provides greater biomechanical resistance, resulting in an overestimation of the internal pressure, which may lead to an unnecessary diagnosis of ocular hypertension.

Beyond its role as a confounding variable in tonometry, CCT has been identified by the Ocular Hypertension Treatment Study (OHTS) as a potent independent risk factor for the progression from ocular hypertension to primary open-angle glaucoma. Consequently, the integration of pachymetric data is indispensable for clinicians to derive a "corrected" IOP, ensuring a more nuanced and accurate risk profile for the patient

In medicine the Optical Coherence Tomography (OCT) is used. High-resolution monitoring of the Retinal Nerve Fiber Layer (RNFL) thickness helps to detect early structural damage.

Visual field testing helps to ensure the absence of glaucomatous defects or blind spots (scotomas).

The systematic identification of specific biometric and clinical variables is essential for predicting the conversion from ocular hypertension to primary open-angle glaucoma and the subsequent rate of functional decline. Large-scale longitudinal clinical trials, most notably the Ocular Hypertension Treatment Study (OHTS) and the Early Manifest Glaucoma Trial (EMGT), have substantiated that elevated intraocular pressure (IOP) remains the primary modifiable risk factor, where every 1 mmHg increase significantly correlates with a higher probability of optic nerve damage.

Beyond manometric pressure, advanced age serves as a critical non-modifiable predictor, reflecting the cumulative biological vulnerability of the trabecular meshwork and retinal ganglion cells over time. Structural indicators, specifically a high vertical cup-to-disc ratio (VCDR) and an increased pattern standard deviation (PSD) in visual field testing, are highly sensitive markers of early neuro-retinal rim thinning and localized functional loss. Furthermore, vascular dysregulation and systemic comorbidities, such as diabetes mellitus and systemic hypertension, are increasingly recognized as contributory factors that compromise ocular perfusion pressure. The integration of these multifactorial predictors into a unified risk profile allows clinicians to implement aggressive therapeutic interventions for high-risk individuals while avoiding over-treatment in low-risk patients.

The risk profile for glaucoma progression is influenced by a combination of biological and hereditary factors. Age serves as a significant physiological marker, with the risk of the condition advancing increasing by a factor of 1.2 per decade. Furthermore, genetics play a pivotal role in determining susceptibility; individuals with first-degree relatives suffering from glaucoma face a 2-3 times higher risk of developing the condition themselves. Vascular Comorbidities: Conditions such as arterial hypertension and diabetes mellitus can impair ocular microcirculation, accelerating nerve damage.

In conclusion, while ocular hypertension does not cause immediate blindness, it serves as the primary predictor for the development of glaucoma. Data from the OHTS indicates that timely diagnosis and appropriate risk stratification can prevent irreversible optic nerve damage in 50-60%

of cases. Clinicians must adopt an individualized approach, considering corneal thickness and optic nerve morphology when deciding on a therapeutic strategy.

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