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Review

The impact of intraocular pressure fluctuations on the progression of glaucoma and associated factors

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ABSTRACT

Background: In recent years, remarkable advancements in the comprehension of glaucoma pathophysiology have highlighted the necessity of looking beyond the conventional focus on mean intraocular pressure (IOP) levels. IOP fluctuations have been identified as a potential factor that could exert a substantial impact on the progression of glaucoma.

Main text: IOP exhibits dynamic variations throughout a 24-h cycle. Glaucoma patients exhibit substantially more pronounced IOP fluctuations compared to healthy individuals, even when the mean IOP remains within the normal range. This implies that IOP fluctuations may play a role in glaucoma progression through mechanisms independent of elevated mean IOP. In this review, an exhaustive examination of studies spanning the past decade was conducted to analyze the relationship between IOP fluctuations and disease progression in primary open-angle glaucoma (POAG), ocular hypertension (OHT), normal tension glaucoma (NTG), and primary angle-closure glaucoma (PACG). While a few studies present conflicting results, the majority of research supports the notion that elevated IOP fluctuations significantly contribute to disease progression in POAG patients. This association has also been confirmed in PACG patients. However, in NTG or OHT patients, other risk factors may outweigh IOP fluctuations in disease progression or glaucoma conversion. Additionally, we summarized common factors affecting IOP fluctuations to provide a basis for the identification of patients prone to significant daily IOP variations. Finally, the efficacy of various IOP-lowering interventions in modulating IOP fluctuations is concisely summarized, offering insights for the formulation of comprehensive treatment strategies that incorporate IOP fluctuation management.

Conclusions: IOP fluctuations play a significant role in disease progression in POAG and PACG. Individuals with certain systemic or ocular characteristics are more predisposed to pronounced and recurrent IOP fluctuations. Consequently, a comprehensive assessment of IOP fluctuation that transcends mean IOP values, as well as the integration of IOP fluctuations management into glaucoma treatment strategies are of paramount importance.

1. Introduction

Glaucoma, a progressive optic neuropathy, is characterized by the degeneration of retinal ganglion cells and is typically associated with elevated intraocular pressure (IOP).[1] IOP demonstrates dynamic variations throughout the circadian cycle,[2] which can be classified into transient, short-term, and long-term fluctuations. Transient IOP fluctuations, persisting from seconds to minutes, are elicited by physiological activities such as respiration, ocular movements, and postural changes.[3] Short-term IOP fluctuations encompass 24-h IOP patterns (diurnal variations) as well as changes occurring over a period ranging from days to weeks.[4] These are of critical clinical importance as a single IOP

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measurement generally fails to capture the 24-h peak IOP. Long-term IOP fluctuations are derived from follow-up records spanning from months to years.[4] Studies indicate that these long-term fluctuations are strongly correlated with short-term fluctuations.[5]

Conventionally, the calculation of IOP fluctuations has been based on the difference between maximum and minimum IOP values.[6] However, this approach neglects the dynamic process of IOP change and furnishes only fragmentary information. Moreover, IOP parameters are interrelated. The range of IOP demonstrates a positive association with both mean IOP[7] and peak IOP,[4] a relationship that can confound statistical analysis and interpretations. Consequently, alternative metrics that are less sensitive to mean IOP and outliers, such as standard deviation (SD)[4,8] and the coefficient of variation,[9] are more favorable. Relative IOP fluctuation, defined as the ratio of IOP fluctuation to mean IOP,[10] also serves to mitigate the impact of absolute IOP values.

In recent years, the analysis of IOP fluctuation has accentuated the transition from simplistic range-based measurements to more clinically relevant metrics. For example, Zhai et al.[11] proposed the concept of "overall IOP", incorporating dimensions that encompass the duration, variability, and rate of IOP elevation, thereby underscoring the greater damage inflicted by sustained IOP elevation. Another investigation[12] defined "relevant IOP fluctuations" as IOP differences exceeding the 24-h IOP SD. This definition aligns with the theoretical postulation that only fluctuations surpassing a certain threshold bear clinical significance.

2. Evaluation of IOP fluctuation

2.1. 24-Hour IOP fluctuations

In clinical practice, reliance on isolated IOP measurements during office visits may lead to an underestimation of actual IOP exposure given the inherent dynamic nature of IOP. This highlights the significance of a comprehensive IOP evaluation, which should incorporate mean IOP, peak IOP, and fluctuation patterns. Twenty-four-hour IOP monitoring, providing the aforementioned information, is of vital importance in validating glaucoma diagnosis and evaluating treatment efficiency.

2.1.1. Intermittent measurement of 24-hour IOP

Current 24-h IOP monitoring generally entails 12 intermittent measurements at 2-h intervals during hospitalization. However, technical constraints present formidable challenges. The acquisition of reliable measurements in the prone position is restricted to a limited number of specialized tonometers.[13] Consequently, in certain instances, measurements are confined to the sitting position even during the night. This divergence from physiological conditions substantially compromises the accuracy and reliability of nocturnal IOP measurements. Another significant concern for nocturnal IOP measurements pertains to the sleep disruption induced by frequent awakenings.[14] While one study has indicated that sleep disturbance exerts minimal influence on the 24-h IOP rhythm,[15] this conclusion necessitates further experimental validation. To mitigate measurement variability and enhance data reliability, it is also recommended to employ the same tonometer throughout the entire monitoring period.

2.1.2. Continuous 24-hour IOP monitoring technology

In light of the limitations of conventional 24-h IOP measurement, continuous IOP monitoring has emerged as a research hotspot, offering a more accurate evaluation of physiological IOP.[16] Continuous IOP monitoring devices can be classified into invasive and non-invasive types.[16] Invasive devices necessitate the surgical implantation of a sensor within the eye,[17] whereas non-invasive devices measure IOP directly by detecting corneal deformation.[18] A significant advantage of these devices lies in their capability for high-frequency sampling, which enables the capture of subtle and transient IOP fluctuations that are overlooked by conventional methods.[19] Nevertheless, the widespread implementation of continuous IOP monitoring devices confronts several technical and practical impediments. These encompass challenges in data interpretation, concerns regarding biocompatibility, issues related to measurement sensitivity, signal drift artifacts, and cost-related considerations.[14,20]

2.2. Posture-induced IOP fluctuation

IOP is substantially affected by changes in head and body postures. A marked increase in IOP can be detected when an individual transitions from a sitting or upright position to a supine one.[21] Further elevation in IOP occurs during head flexion or rotation, potentially attributed to jugular vein compression.[22,23] The postural IOP changes that occur rapidly within minutes are considered to be independent of aqueous humor production.[21] These changes are predominantly ascribed to the sympathetic response,[23] along with alterations in episcleral venous pressureand choroidal blood volume.[24] Moreover, in the supine position, the vertical distance between the eyes and the heart is diminished. This reduction leads to a decrease in the gravitational force that facilitates aqueous humor drainage through the venous system into the circulatory system, thereby contributing to an elevation in IOP.[23]

2.3. Provocative Tests

Glaucoma provocation tests represent a specialized category of diagnostic procedures designed to evaluate the ocular response to induced IOP fluctuations. For open-angle glaucoma, methods like increasing aqueous humor production (water-drinking, drug tests) are used for provocation. For angle-closure glaucoma, methods causing anterior chamber angle closure (dark-room, prone, mydriatic tests) are applied.

2.3.1. Water-drinking test (WDT)

WDT entails the rapid consumption of 800–1000 mL of water within a short time frame, followed by serial IOP measurements.[25] This test serves to assess the facility of aqueous humor outflow.[26] In the course of WDT, glaucoma patients exhibited more pronounced IOP fluctuations compared to healthy controls,[27] with a strong correlation to the 24-h mean and peak IOP values.[25,28]

2.3.2. Darkroom prone test (DRPT)

During the DRPT, patients are instructed to lie prone in a darkroom for 1 h with no additional pressure applied to the eyeballs.[29] The typical IOP elevation during DRPT ranges from 4 to 6 mmHg in primary angle-closure glaucoma (PACG) patients, with more significant increases observed in eyes with compromised angle function.[29,30] Nevertheless, studies have demonstrated that IOP also increases in primary open-angle glaucoma (POAG) patients.[31]

2.3.3. Mydriasis test

Certain medications can increase IOP by inducing mydriasis.[32–34] These alterations exhibit a significant correlation with the diurnal peak IOP.[34] Ibopamine, for instance, has been observed to increase IOP by 3–11 mmHg in glaucomatous eyes through mydriasis and the stimulation of aqueous humor production.[32]

3. The impact of IOP fluctuation on glaucoma progression

Elevated mean IOP has long been recognized as a well-established risk factor for glaucoma progression.[4] Despite this, studies have demonstrated that some patients with severe glaucomatous damage present with normal IOP levels.[8] IOP exhibits distinct circadian patterns. In healthy individuals, the physiological fluctuations of IOP typically remain within 5 mmHg over a 24-h period.[35] Conversely, glaucoma patients, even when their average IOP levels fall within the normal range, generally exhibit more significant IOP fluctuations.[36]

The human body sustains a state of homeostasis, and deviations from this equilibrium can lead to pathological consequences. Researchers have

put forward the hypothesis that excessive IOP fluctuations, indicative of regulatory dysfunction,[37] could disrupt the homeostasis of retinal ganglion cells and potentially exacerbate glaucomatous neurodegeneration.[19,38] This effect might be independent of elevated mean IOP levels. Therefore, extensive research has been dedicated to elucidating the role of IOP fluctuations in glaucoma development. The following sections provide a comprehensive review of studies conducted over the past decade that address this issue, categorized separately according to glaucoma subtypes (Supplementary Table 1).

3.1. POAG

Compared to healthy individuals, POAG patients demonstrate higher mean IOP levels, a delay in the peak IOP timing, and more significant fluctuations.[35,39,40] The relationship between IOP fluctuations and glaucoma progression in POAG patients has yielded heterogeneous results. While some studies have reported inconclusive or conflicting results,[8,41–44] the preponderance of evidence supports a positive correlation.[4,37,45–48] This association is further corroborated by IOP-related variability data obtained through contact lens sensor, a continuous IOP monitoring device.[19,49–51]

In general, statistically significant findings are more likely to be associated with larger amplitudes of IOP fluctuations, as evaluated by both SD and range. This implies the potential existence of intrinsic regulatory mechanisms that respond to IOP fluctuations. When IOP fluctuations surpass certain physiological thresholds, they may exert a more pronounced pathological impact on glaucomatous optic neuropathy. The elevated IOP fluctuations observed in glaucoma patients, as opposed to the physiological fluctuations in healthy individuals, may further corroborate this hypothesis.

3.2. Ocular hypertension (OHT)

OHT patients are characterized by consistently elevated IOP levels without glaucomatous optic nerve damage.[52] The transition from OHT to glaucoma represents a distinct progression pathway. Existing evidence suggests that IOP fluctuations play a limited role in the risk of this conversion.[53,54] For instance, through secondary analysis of two early OHT clinical trials, Gordon et al.[53] demonstrated that long-term IOP variability provides minimal predictive value for identifying untreated OHT patients who will progress to POAG. It's suggested that the mean IOP may supersede the pathological effects of IOP fluctuations at higher IOP levels, exerting the predominant influence on disease progression.

3.3. Normal-Tension Glaucoma (NTG)

The IOP level of NTG patients consistently remains below 21 mmHg. The 24-h IOP patterns in these patients are comparable to those of healthy individuals, with a slightly elevated peak IOP and increased fluctuation amplitudes.[36] When compared to glaucoma patients with elevated IOP, they exhibit significantly lower mean, maximum, and fluctuation values.[55] Although a few studies have demonstrated a positive correlation between IOP fluctuation and disease progression in NTG patients,[56,57] the majority of investigations have revealed no significant associations, regardless of whether short-term or long-term fluctuations were examined.[7,58–60] One possible explanation is that the minor IOP fluctuations in NTG patients fall within the physiological safety thresholds.

Interestingly, one study has demonstrated that a greater seasonal IOP fluctuation between summer and winter suppressed the thinning rate of the retinal nerve fiber layer (RNFL).[61] Given the characteristic seasonal pattern of higher IOP levels in winter and lower levels in summer, this finding likely reflects the beneficial impact of the reduction in summer IOP.[61] This observation highlights the importance of taking the baseline IOP into account when evaluating the pathological impact of IOP fluctuation. The challenge persists in determining whether sustained IOP elevation or marked fluctuations between significantly elevated and reduced levels impose more detrimental effects on the optic nerve.

3.4. PACG

In comparison with other glaucoma subtypes, PACG patients typically present with more pronounced elevation in both mean IOP and diurnal IOP fluctuations.[38] Given the dominant role of elevated IOP, investigations into additional risk factors contributing to disease progression have received relatively less attention. Nevertheless, the detrimental impact of IOP fluctuations on disease progression in PACG patients has been confirmed. Current evidence demonstrates significant positive correlations between greater IOP fluctuations and both visual field deterioration and RNFL loss.[9,62] These associations remain statistically significant even after adjusting for mean IOP levels.[9] Notably, even ultra-short-term IOP fluctuation of 1-h intervals differ significantly between progressive and stable cases, which can potentially be attributed to its capacity to reflect the status of ocular microcirculation.[63]

In the process of comparing results among diverse investigations, several critical factors demand particular consideration. The definition of IOP fluctuation exerts a substantial influence on the interpretation of statistical analyses and ultimate outcomes. For example, when contrasted with short-term IOP fluctuations, long-term measurements obtained from routine office visits are inherently less sensitive in detecting IOP peaks. Moreover, the SD may represent a more reliable metric than the IOP range.[9] Population characteristics constitute another crucial factor influencing study outcomes. This underscores the significance of ensuring inter-group comparability, formulating appropriate inclusion and exclusion criteria, and minimizing selection bias. Notably, patients with advanced glaucoma generally display a limited capacity for further detectable progression owing to severe pre-existing optic nerve damage.[8] This can potentially lead to their misclassification as "stable" cases.

4. Related factors of IOP fluctuations

Given the potential significance of IOP fluctuations in glaucoma progression within certain patient subgroups, identifying individuals who are more susceptible to significant IOP fluctuations in daily life is of great importance. IOP exhibits dynamic variations throughout the day under the influence of physiological factors such as respiration,[64] blinking,[3] and physical activities,[65] as well as environmental factors like seasonal changes.[10] Existing studies indicate that the amplitude of IOP fluctuation is modulated by multiple factors, which can be categorized into ocular and systemic factors (Supplementary Table 2). Certain patients may encounter frequent and pronounced IOP fluctuations in their daily lives, increasing their risk of repeated ischemia-reperfusion injury and disruption of retinal ganglion cell homeostasis.[19,38] Comprehending the factors that influence IOP fluctuations is crucial for identifying these high-risk patients.

4.1. Ocular factors

4.1.1. Refraction and Axial Length (AL)

The relationship between refractive status and IOP fluctuations remains conflicting. Some studies have suggested that myopic patients with longer AL exhibit reduced IOP fluctuations compared to hyperopic individuals.[55] Conversely, other research has reported no significant correlation.[21] Theoretically, myopic eyes with longer AL possess structural features that may mitigate IOP fluctuations. These include enlarged anterior chamber dimensions, which enhance aqueous humor outflow; choroidal thinning, which leads to a reduction in vascular volume changes[66]; as well as scleral thinning and stiffening.[55] Collectively, they contribute to diminished postural IOP variations in myopic eyes.[55] It is particularly noteworthy that the structural alterations associated with myopia may increase their susceptibility to glaucomatous damage, regardless of the magnitude of IOP fluctuations.[66]

4.1.2. Anterior chamber structures

The anterior chamber constitutes parts of the aqueous humor outflow pathway and can impact IOP fluctuations. Although an earlier study reported no statistically significant differences in postural IOP fluctuations between POAG and PACG patients,[67] recent research has demonstrated an inverse correlation between narrower angle and postural IOP changes in eyes experiencing acute angle-closure attacks.[68] Additionally, a study conducted on patients undergoing hemodialysis revealed a significant IOP elevation 2 h subsequent to the initiation of dialysis in eyes with narrow anterior chamber angles.[69] The magnitude of IOP fluctuations was positively correlated with changes in lens thickness and negatively correlated with changes in angle opening distance and trabecular-iris angle during hemodialysis.[69]

4.2. Systemic factors

4.2.1. Age

The relationship between aging and IOP dynamics presents a complex pattern. While mean IOP typically decreases with age,[70] diurnal IOP fluctuations exhibit a tendency to increase in elderly populations.[71] This can be attributed to several age-related physiological changes. These encompass alterations in hormonal secretion patterns (e.g., cortisol and melatonin),[72] a reduction in aqueous humor production combined with an increase in trabecular meshwork outflow resistance,[71] and progressive rigidification of ocular structures.[73] Notwithstanding, the advancing age may not exert a discernible impact on the amplitude of postural IOP fluctuations.[72] Nevertheless, the increased vulnerability of elderly patients to IOP fluctuation remains a critical concern.[65]

4.2.2. Obesity

A 5-years longitudinal investigation has revealed an inverse correlation between baseline Body Mass Index and long-term IOP fluctuations.[71] This may be attributed to obesity-induced oxidative stress and elevated epidural venous pressure, which contribute to trabecular meshwork degeneration.[71] Consequently, these factors may attenuate ocular sensitivity to further IOP fluctuations in obese individuals. In contrast to this long-term pattern, obese patients exhibit more pronounced acute IOP elevation upon changing from a sitting to a supine position.[74] This immediate response could be a consequence of increased thoracic and abdominal cavity pressure caused by adipose tissue accumulation, which raises the central venous pressure and increases resistance to aqueous humor outflow through the aqueous veins.[74]

4.2.3. Diabetes

Systemic diseases such as diabetes mellitus have been identified as risk factors for glaucoma development.[75] However, Cheung et al.[76] reported that, despite having elevated mean IOP levels, diabetic patients exhibited reduced long-term IOP fluctuations over a two-year period. This phenomenon may be ascribed to the impairment of IOP regulatory mechanisms induced by diabetes, which leads to a diminished capacity for IOP modulation and renders individuals sustained elevated IOP levels.[76]

4.2.4. Blood pressure (BP)

BP represents another critical systemic factor in glaucoma pathophysiology. Aberrations in BP, whether elevated or reduced, have been associated with the progression of glaucoma.[77] Clinical studies have demonstrated significant correlations between elevated mean arterial pressure and increased 24-h or long-term IOP fluctuations.[71,78] Huang et al.[79] also proposed that more pronounced daytime IOP fluctuations, in contrast to those during the night, might be ascribed to heightened physical activity patterns and corresponding BP variations. These findings underscore the importance of comprehensive management of BP and IOP in glaucoma treatment.

4.2.5. Endocrine hormone

Endocrine hormones have been recognized as potential regulators of IOP fluctuations, yet research in this domain remains scarce. Gubin et al.[80] undertook a comprehensive investigation into the effects of melatonin supplementation over a 90-day period in POAG patients. The results of their findings demonstrated significant reductions in both mean IOP and IOP fluctuations, with more pronounced effects observed in patients with higher baseline IOP and more advanced disease stages.[80]

5. Clinical management strategies for IOP fluctuations

5.1. Medication application

Despite the presence of conflicting results in some studies,[81,82] the preponderance of research findings supports the beneficial effect of IOP-lowering eye drops in reducing 24-h IOP fluctuation.[83–86] In general, combination therapies demonstrate superior efficacy compared to monotherapy.[82,86] However, a consensus regarding the optimal medication selection remains elusive.[87,88] The timing of administration also exerts an influence on therapeutic outcomes. Early investigations indicated that evening administration of travoprost and timolol provided a superior effect in regulating IOP fluctuations,[89,90] whereas the combination of latanoprost and timolol demonstrated greater effectiveness when administered in the morning.[91]

5.2. Surgical interventions

Current evidence indicates a differential efficacy of diverse IOP-lowering interventions in controlling IOP fluctuation. Trabeculectomy, for example, has been demonstrated to be effective in reducing both diurnal and postural IOP fluctuations.[92] However, such an effect is attenuated in pseudophakia eyes.[93] Other surgical approaches, including canaloplasty,[94] selective laser trabeculoplasty (SLT),[95] and Ex-PRESS filtration device implant,[96] also lead to a reduction in short-term IOP fluctuations. Among these, SLT exhibits inferior efficacy compared to medication therapy,[97] with 360-degree SLT outperforming 180-degree procedures.[98] Conversely, phacoemulsification[21] and laser peripheral iridotomy,[99] despite reducing mean IOP, failed to reduce IOP fluctuation. Notably, certain procedures may inadvertently increase IOP fluctuations. For example, a rise in 24h IOP fluctuation has been observed following vitrectomy,[100] and postural IOP fluctuations may increase during the early postoperative period of microcatheter-assisted trabeculotomy and modified canaloplasty.[24]

5.3. Comprehensive treatment strategies

Theoretically, the concurrent use of multiple eye drops with different onset and duration timing may be efficacious in reducing 24-h IOP fluctuations while minimizing mean IOP. However, the implementation of supplementary therapeutic interventions requires careful consideration of potential pharmacological side effects and patient compliance. To address this, ancillary strategies for mitigating IOP fluctuations should be explored. For example, optimizing the temporal sequence of medication administration may represent a viable approach, though further investigations are needed to establish evidence-based guidelines. As previously elucidated (see Section 2.3, Provocative Tests), the avoidance of acute water intake and prolonged prone positioning can prevent drastic transient IOP fluctuations. Additionally, maintenance of a healthy weight and glycemic control may reduce postural IOP fluctuations, thereby promoting more stable IOP status during daily activities. Similarly, the management of BP can contribute to long-term IOP stabilization.

6. Conclusions and prospects

IOP displays constant dynamic fluctuations. Current evidence suggests that elevated IOP fluctuations significantly contribute to disease progression in POAG and PACG patients. This accentuates the necessity of evaluating both IOP fluctuations and mean IOP for these patients. Nevertheless, in NTG or OHT patients, other risk factors may exert a more conspicuous influence.

IOP fluctuations are influenced by both ocular and systemic factors, leading to a more frequent and substantial alterations in certain patients. The identification of such high-risk individuals is critical, as IOP that appears to be well-controlled may still exhibit significant variations in daily life. Effective glaucoma management strategies necessitate consideration of not only the amplitude, duration, and frequency of IOP fluctuations but also individual susceptibility profiles. Modifications in lifestyle can also contribute to the reduction of IOP fluctuations, and treatment regimens should be adjusted accordingly.

The underlying mechanisms of IOP fluctuations remain elusive, and current hypotheses lack robust experimental validation. Future research endeavors should concentrate on elucidating these mechanisms, determining optimal therapeutic thresholds, and developing personalized management strategies to enhance the clinical outcomes for glaucoma patients.

Study approval

Not applicable.

Author contributions

The authors confirm contribution to the paper as follows: TL and YLC conceived and designed the review; MC, MYH and XL searched and selected references of the review; TL, YLC, ZYW and KJW drafted and revised the manuscript. All authors reviewed and approved the final version of the manuscript.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Abbreviations

AL	Axial length
BP	Blood pressure
DRPT	Darkroom prone test
IOP	Intraocular pressure
NTG	Normal tension glaucoma
OHT	Ocular hypertension
POAG	Primary open-angle glaucoma
PACG	Primary angle-closure glaucoma
RNFL	Retinal nerve fibre layer
SD	Standard deviation
SLT	Selective laser trabeculoplasty
WDT	Water-drinking test

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.aopr.2025.03.002.

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