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SIMILARITIES AND DIFFERENCES BETWEEN PRIMARY OPEN-ANGLE AND ANGLE-CLOSURE GLAUCOMA

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Summary

Primary open-angle glaucoma (POAG) and primary angle-closure glaucoma (PACG) are the most prevalent forms of glaucoma and major causes of irreversible vision loss worldwide. Although both conditions lead to retinal ganglion cell death, optic nerve damage, and progressive visual field loss, they differ significantly in etiology, anatomical mechanisms, clinical presentation, and management strategies. POAG is characterized by an open anterior chamber angle with increased trabecular outflow resistance and typically progresses slowly and asymptomatically. In contrast, PACG results from anatomical angle narrowing or closure and may present acutely or chronically with rapid intraocular pressure (IOP) elevation. Accurate diagnosis requires combined structural, functional, and gonioscopic assessment. While IOP reduction remains the primary therapeutic goal in both conditions, treatment selection depends on the underlying mechanism of aqueous outflow impairment.

Introduction

Glaucoma is a progressive optic neuropathy characterized by structural damage to the optic nerve head, resulting in impairment of the patient's visual field [1,2]. Although the primary risk factor for developing glaucoma is high intraocular pressure (IOP), the disease itself is multifactorial and can be influenced by systemic, neurodegenerative, vascular dysfunctions and many more. Therapeutic interventions for reducing the IOP have been effective, but the lack of successful treatments for preventing the progression of glaucoma continue to make this a significant public health problem as the population ages and the number of people developing glaucoma increases [1,3,4].

The most prevalent types of glaucoma are primary open-angle glaucoma (POAG) and primary angle-closure glaucoma (PACG). POAG is among the most common types of glaucoma in the world [1]. It is characterized by impaired aqueous humor outflow through an anatomically unobstructed anterior chamber angle, leading to a progressive elevation of IOP and subsequent optic nerve damage [2]. The situation usually gets worse over time, and if it isn't treated, it might cause permanent loss of vision. POAG often manifests asymptomatically in its early stages, leading to delayed diagnosis [1,2]. PACG occurs when the anterior chamber angle becomes anatomically narrowed or occluded, obstructing aqueous outflow and causing a rapid rise in IOP. This sudden or gradual rise in pressure may severely harm the optic nerve and retina, leading to sudden and major vision loss [1,5].

Even though POAG and PACG are distinct types of glaucoma, they produce similar end-stage effects including the death of retinal ganglion cells, damage to the optic nerve head, and permanent loss of visual fields [1,3]. Understanding the difference between POAG and PACG are critical for making the proper diagnosis and selecting the correct treatment plan to prevent vision loss [2,5]. This article will provide information about the development and pathophysiology of POAG and PACG, and how each can be diagnosed and treated. It also examines the distinguishing features of their clinical presentation, forming the basis for a comparative discussion of therapeutic strategies.

Aim: The aim of this review is to compare the similarities and differences between POAG and PACG.

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Materials and Methods

A comprehensive literature review was performed using the PubMed database to identify relevant studies comparing POAG and PACG. Additional articles were retrieved from the reference lists of relevant publications to identify additional studies. Only original studies, written in English, were used. A total of 26 studies were included in the final analysis. All retrieved references were managed using the Mendeley reference management software for citation organization, duplicate removal, and consistent referencing.

Results

Etiology and pathophysiology. The main cause of optic nerve injuries in both POAG and PACG is a multifactorial combination of mechanical stress and pressure independent factors. Both conditions arise from a complicated interaction between IOP, vascular dysfunction, oxidative stress and retinal ganglion cell susceptibility to injury [1,3]. While IOP is the major modifiable risk factor for the onset of both diseases, the pathways to the impaired aqueous humor outflow and increased IOP associated with each type of glaucoma are distinctly different.

In POAG, aqueous humor drains through the trabecular meshwork, but increased outflow resistance impairs this process. The persistent obstruction of the drainage mechanism results from aging and/or pathological alterations in the trabecular meshwork, including extracellular matrix remodeling, dysfunctional cellular communication, and diminished endothelial cell activity [6]. These factors collectively increase the outflow resistance thus leading to an elevated IOP, which exerts chronic mechanical stress on retinal structures, leading to the degeneration of retinal ganglion cells [2,6]. Additional chronic damages to these cells include oxidative stress, mitochondrial dysfunction, neuroinflammation, and impaired autoregulation of ocular blood flow thus explaining why certain patients with clinically normal intraocular pressure may still experience disease progression [7–9].

Unlike POAG, the principal pathogenic mechanism in PACG is a relative pupillary block, characterized by heightened resistance to aqueous humor passage from the posterior to the anterior chamber at the pupil, creating a pressure gradient between the two chambers. This difference in pressure makes the peripheral iris bow forward (iris bombe), which closes the trabecular meshwork by apposition or synechia and quickly raises the pressure inside the eye [1,5]. Other anatomical features, such as the shape of the plateau iris or crowding around the lens, may also cause angle closure [10].

Clinical symptoms and course. Glaucoma remains a clinically challenging disease because noticeable symptoms often appear only after substantial, irreversible optic nerve damage has already occurred [11]. In POAG, progression is typically slow, with gradual peripheral visual field loss that affects one or, more commonly, both eyes. Because this vision loss develops in a characteristic pattern, many patients do not recognize meaningful impairment until central vision is involved, at which point the damage is permanent [1]. Even so, patient-reported outcomes suggest that functional visual difficulties can emerge in earlier stages of the disease. In a study of 99 patients, most of which were diagnosed with POAG, the most frequently reported complaints were a need for brighter illumination and blurred vision. As visual field loss became more advanced, patients were significantly more likely to describe problems such as reduced side vision, vision described as seeing through dirty glasses, and difficulties distinguishing object edges and colors. This supports a clear relationship between symptom severity and the extent of visual field damage [12].

PACG, by contrast, is defined largely by ocular anatomy and can present either as an acute emergency or as a more chronic condition [13]. Acute angle-closure glaucoma typically has a sudden onset, with symptoms such as headaches, nausea, severe eye pain and halos around lights. This is an ophthalmic emergency, since a rapid rise in IOP can lead to irreversible central and/or peripheral vision loss within a short time if treatment is delayed [14]. Chronic angle-closure glaucoma may be either symptomatic or asymptomatic. It involves a more gradual elevation of IOP and can still cause progressive optic nerve damage. Because it may develop without obvious warning signs, it is sometimes described as “silent” angle-closure glaucoma [15].

Diagnostic methods. The clinical diagnosis of glaucoma is primarily established through an evaluation of a patient’s eyes. It includes the measurement of IOP, the examination of the optic nerve anatomy, the assessment of ocular function, and the analysis of the anterior chamber angle configuration [2,5]. Given that optic nerve damage from glaucoma is irreversible and often asymptomatic until advanced stages, early diagnosis is essential to initiate treatment and avert further progression and permanent vision loss [1–3].

A prevalent technique for evaluating IOP is doing an applanation tonometry. IOP is the most critical and modifiable risk factor for the onset of POAG and PACG. However, IOP alone is inadequate for diagnosing glaucoma due to physiological variations, diurnal fluctuations and the occurrence of glaucoma in individuals with statistically normal IOP levels [1,3]. Consequently, the interpretation of IOP measurements must occur simultaneously with structural and functional evaluations.

The assessment of the optic nerve head is important for diagnosing glaucoma. Clinical fundoscopy is used to assess the optic disc for signs of cupping, neuroretinal rim thinning,

hemorrhages, and interocular asymmetry [2,16]. Optical coherence tomography (OCT) is a crucial instrument for evaluating the retinal nerve fiber layer and macular ganglion cell complex, both of which are commonly impacted in the initial phases of glaucoma [2,17,18]. OCT offers objective and reproducible assessments of these structures, enabling earlier identification of glaucomatous damage and the capacity for longitudinal monitoring of disease progression in both POAG and PACG [2,17–20].

The use of standard automated perimetry (SAP) remains the gold standard for detecting and monitoring glaucomatous visual field loss [2]. This includes factors such as nasal steps, arcuate scotomas and paracentral defects, which assist in determining the degree of disease. Because functional and structural changes in glaucoma do not always progress in parallel, repeated SAP is required to confirm progression and evaluate treatment effectiveness [1,2].

Gonioscopy, is another diagnostic tool that allows clinicians to assess the anterior chamber angle through direct visualization. It is an essential test for distinguishing between POAG and PACG. Through gonioscopy, the doctor can evaluate the width of the angle, the amount of iridotrabecular contact, the number of peripheral anterior synechiae and degree/mechanism of angle closure. The physician needs to be aware of this distinction because the angle configuration will determine the treatment option and long-term management plan for the patient [2,5].

Ultrasound biomicroscopy (UBM), anterior segment optical coherence tomography (AS-OCT), can provide the ophthalmologist with additional information regarding the angle anatomy and the potential mechanisms of closure (e.g., pupil block or plateau iris configuration) [21,22]. UBM and AS-OCT are useful when the gonioscopic findings are ambiguous and/or when the clinician desires more anatomical information regarding the angle. Although UBM and AS-OCT are highly sensitive for detecting angle closure, they do not replace gonioscopy but instead serve as adjunctive diagnostic tools [21,22].

Treatment principles. Glaucoma management is highly individualized and depends on the specific type of glaucoma and how far it has progressed [1]. Today's treatments can't restore vision that's already been lost, the main goal is to protect the vision that remains. This is done by lowering IOP and keeping it consistently controlled using medication, laser treatment, and, when necessary, surgery [15].

In POAG, treatment often starts with eye drops, which are selected and adjusted to reach a target IOP that is individual to the patient [2]. In everyday practice, five main medication classes are used to lower intraocular pressure: prostaglandin analogues, beta-blockers, alfa-agonists, carbonic anhydrase inhibitors and cholinergic agents. While the goal is to preserve the optic nerve, relatively few studies have shown neuroprotective benefits that go beyond pressure reduction itself [23]. It's also important to note that the target IOP isn't one universal number, it's a personalized pressure level below which progression is less likely, and it must be reassessed over time through follow-up examination [24].

If topical therapy does not lower the IOP enough, or the target isn't reached, laser treatment can be additionally introduced [25]. Laser trabeculoplasty is a well-established option for appropriate patients. This approach generally reduces IOP to a similar degree as medication-based therapy [26]. A systematic review conducted by Samantha So and co-authors reported better long-term IOP reduction with laser trabeculoplasty and a meaningful decrease in the number of medications required, whereas medication-only groups did not show a significant drop in medication use over time [27].

If a patient has a more advanced form of glaucoma, or when the IOP remains too high despite previous topical or laser treatment, surgery may be needed to achieve a stronger and more dependable pressure reduction. Such cases are often treated using trabeculectomy and glaucoma drainage devices [28]. However, in modern clinical practice, new procedures, such as stents, canaloplasty, and ciliary body ablation are used. These methods are prioritized because they are minimally invasive, allowing for minor tissue damage and quick recovery [29]. These intervention methods provide an alternative when the risk of progression or inadequate IOP control demands a more invasive solution [14].

Treatment of PACG slightly differs from the management of POAG. As with POAG, treatment usually starts with topical IOP-lowering medications. In acute or symptomatic cases, a fixed combination such as dorzolamide and timolol can be used to achieve rapid initial pressure reduction. After the IOP has decreased, laser peripheral iridotomy (LPI) is typically performed, often in both eyes, to lower the risk of recurrence. However, this requires the cornea to be clear enough and for the patient to be cooperative [30]. This is the key difference from the treatment of POAG, where laser trabeculoplasty is commonly used as an adjunct or even as an early alternative to eye drops [2]. LPI targets the pupillary block, while laser iridoplasty is used for closure caused by non-pupillary block mechanisms, such as plateau iris configuration or plateau iris syndrome [31]. According to a trial done by the EAGLE study group, compared to laser peripheral iridotomy, clear-lens extraction was both more efficient and cost-effective. For these reasons, it should be considered as the initial treatment method for PACG [32].

Conclusion

1. POAG and PACG are the most prevalent types of glaucoma, which account for the majority of irreversible

vision loss. Although they both result in retinal ganglion death, optic nerve damage and visual field impairment they differ in etiology, clinical course and management course.

2. Anatomically, POAG is characterized by an open anterior chamber angle with chronically reduced aqueous outflow and a gradual, often asymptomatic progression. In contrast, PACG results from a narrowed or closed angle and may present with an acute rise in IOP or develop more gradually, carrying a high risk of vision loss.

3. Accurate differential diagnosis of both conditions is important for a complete examination which includes IOP measurement, structural and functional evaluation of the glaucomatous optic neuropathy and gonioscopic evaluation. Although lowering IOP is the primary treatment goal in both types of glaucoma, the choice of medical, laser, or surgical therapy depends on the underlying mechanism of outflow obstruction.

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PIRMINĖS ATVIROJO IR UŽDAROJO KAMPO GLAUKOMOS PANAŠUMAI BEI SKIRTUMAI

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Raktažodžiai: pirminė atvirojo kampo glaukoma, pirminė uždarojo kampo glaukoma, glaukomos klinikinė eiga, glaukomos klinikiniai požymiai, perimetrija, glaukomos gydymas, glaukomos etiologija.

Santrauka

Pirminė atvirojo kampo glaukoma (PAKG) ir pirminė uždarojo kampo glaukoma (PUKG) yra dažniausios glaukomos formos ir vienos pagrindinių negrįžtamo regėjimo praradimo priežasčių pasaulyje. Nors abi ligos sukelia tinklainės ganglinių ląstelių žūtį, regos nervo pažeidimą ir progresuojantį regos lauko defektą, jos skiriasi etiologija, anatominiu mechanizmu, klinikine eiga ir gydymo principais. PAKG būdingas atviras priekinės kameros kampas ir padidėjęs akies skysčio nutekėjimo pasipriešinimas, o liga dažniausiai progresuoja lėtai ir be simptomų. Tuo tarpu PUKG išsivysto dėl anatomiškai susiaurėjusio ar užsidariusio kampo ir gali pasireikšti ūmine arba lėtine forma su staigiu akispūdžio padidėjimu. Tiksli diagnostika grindžiama struktūriniu, funkciniu ir gonioskopiniu ištyrimu. Nors pagrindinis gydymo tikslas abiem atvejais yra akispūdžio mažinimas, gydymo pasirinkimas priklauso nuo akies skysčio nutekėjimo sutrikimo mechanizmo.

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