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THE SIGNIFICANCE OF CENTRAL VISUAL FIELD DEFECTS IN PRIMARY OPEN-ANGLE GLAUCOMA

Introduction: In primary open-angle glaucoma (POAG), the focus has traditionally been on peripheral visual field defects. However, over the past 15 years, it has become apparent that the central visual field is affected at an early stage in POAG. The fundamentals and the most important risk factors will be presented.
Methods: This study is a literature review. Databases including PubMed, Scopus, Web of Science, and Springer were searched using the terms "central visual field" and "glaucoma" for the period from 2000 to 2025. Duplicates were removed. The selected literature focused on the role of lamina cribrosa, ocular microcirculation, and neurodegeneration in glaucoma.
Results: Approximately 50% of retinal ganglion cells (RGCs) are responsible for the central 16° of the visual field alone, and around 40% of these cells are affected in POAG. This leads to abnormalities in the central visual field even in the early stages of POAG. The problem is that the 24-2 or 30-2 visual field program does not adequately detect these central disturbances. The degeneration of RGCs and their nerve fibers is a complex process involving mechanical (intraocular pressure), vascular, and, above all, inflammatory processes.
Conclusion: Central RGC loss is not only associated with early central visual field defects, but also leads to color vision defects, contrast sensitivity defects, crowding, and reading difficulties, which significantly impair patients' everyday lives. Therefore, in addition to adjusting intraocular pressure to the individual target pressure range, it is particularly important to have general systemic diseases such as arterial hypertension and diabetes mellitus optimally controlled by an internist and to avoid a nocturnal drop in blood pressure.
Keywords: central visual field, lamina cribrosa, glaucoma, neurodegeneration, microcirculation.

Introduction

Central visual field defects in primary open-angle glaucoma (POAG) are of great clinical significance, but they are generally given too little attention. The aim of this article is therefore to describe this perimetric feature in more detail.

Primary open-angle glaucoma (POAG) is not an isolated optic neuropathy, but rather a systemic neurodegeneration [1,2]. In addition to the glaucoma-typical changes in the optic nerve, including involvement of the entire visual system (lateral geniculate nucleus, optic radiation, visual cortex), there are also generalized cerebral impairments with synaptic connection disorders [3,4] involving both the central autonomic network and the peripheral autonomic nervous system [3,4,5].

The importance of the visual system for humans is reflected in its large involvement in the cerebral cortex. In addition to the primary visual cortex (V1), which accounts for about 15% of the entire cerebral cortex, more than 30 different visual areas have been described to date. In total, about 60% of the cerebral cortex is involved in the perception, interpretation, and response to visual stimuli [6].

Visual impairment in glaucoma is primarily determined by visual field defects. There are various types of classifications, such as the visual classification according to Aulhorn and Karmeyer [7], the classification using the mean defect and the number of defective test locations (Hodapp–Parrish classification) [8], and a classification using the mean defect and the pattern standard deviation (Brusini classification) [9]. These classifications are used to classify perimetric findings according to the severity of functional optic nerve damage, but they do not correspond to individual loss patterns and do not allow the findings to be localized, i.e., whether they originated locally in the optic nerve or in the central nervous system in glaucomatous neurodegeneration.

Material and methods

This study is an evaluation review. The literature was searched in the databases PubMed, Scopus, Web of Science, and Springer. Duplicate publications have been checked and deleted. The literature search was carried out using the terms "central visual field" and "glaucoma" in the period 2000 to 2025. The selected literature focused on the role of lamina cribrosa, ocular microcirculation, and neurodegeneration in glaucoma.

Results and discussion

The human optic nerve contains an average of 1.023 million nerve fibers with a wide range of ± 310,000 nerve fibers [10]. There is a retinotopic organization in the prelaminar optic nerve, where neighboring fibers from neighboring retinal areas enter it [10]. Basically, the peripheral retinal areas are located at the outer edge of the optic nerve, while the central parts are located in the center. The axons from the retinal ganglion cells in the macular run via the papillomacular bundle directly to the optic nerve head and are thus located centrally-temporally [10]. The special feature of the macular region is that no axons run over the macula [11]. In the prelaminar section, the retinal nerve fibers (RNF) are unmyelinated, while after the lamina cribrosa they are myelinated by oligodendrocytes [10]. The cellular composition of the lamina cribrosa consists mainly of microglia cells, astrocytes, and alpha-actin-positive lamina cribrosa cells, which are bounded toward the sclera by the Elschnig ring [12]. Its main function is to allow the RNF to pass through the sclera and to equalize the pressure between the prelaminar intraocular pressure (IOP) and the postlaminar cerebrospinal fluid pressure (translaminar pressure difference).

Blood supply to the optic nerve

The optic nerve is supplied with blood via the ophthalmic artery, which branches off from the internal carotid artery. The outer parts of the optic disc and the lamina cribrosa are supplied by the posterior short ciliary arteries via the Zinn–Haller vascular ring, while the central nerve fibers are supplied by branches of the central retinal artery [13]. The special feature of papillary blood supply is that it is autoregulated, i.e., it can maintain a constant flow within certain limits. A distinction is made between metabolic autoregulation, which is based on the current metabolic activity of the retinal ganglion cells (RGC), and pressure-dependent autoregulation [14]. The latter maintains a constant blood flow at the optic nerve head under increased intraocular pressure [15] and intraluminally under both increased and decreased blood pressure [14,16]. The endothelium of the retinal vessels [17] and its interaction with the pericytes [18] play an important role here, as they are regulated by the peripheral autonomic nervous system [19].

Central visual field

In perimetric testing, the central 10° are defined as the central visual field. This primarily examines the macula and fovea. Due to the high importance of this retinal region for visual acuity, contrast, and color vision, 50% of all ganglion cells are located within the central 16° of the visual field, an area that corresponds to only 7.3% of the total surface area of the retina [20]. These findings were confirmed by Zhang [21] using optical coherence tomography (OCT): the average estimated number of RGCs in the macula is 520,678 ± 106,843 cells in healthy eyes, with an average estimated total number of RGCs of 1,042,019 ± 185,243 cells; the estimated number of RGCs in the macula corresponds to approximately 50% of the estimated total number of RGCs. These figures underscore the great importance of the central visual field for the overall evaluation of a perimetric finding. In addition, the central 10 degrees occupy at least 50–60%, and the central 30 degrees of the visual field occupy 83% of the visual cortex [22].

POAG and central visual field defects

Typically, visual field defects in glaucoma are primarily assumed to be peripheral visual field defects as the first signs of damage in the visual field, as it is generally accepted that increased IOP first mechanically impairs the peripheral nerve fibers in the optic nerve head. Compression at the Elschnig ring causes disturbances in the axoplasmic flow of these nerve fibers [23], which ultimately leads to the onset of peripheral visual field defects. This view, which focuses on damage caused by intraocular pressure, has meant that the central visual field is still not routinely assessed in POAG patients. In addition, the standard perimetric examination for glaucoma using the 24-2 or 30-2 visual field with the Humphrey Field Analyzer (Zeiss Meditec AG, Jena) does not take sufficient account of the central area, as there are only 12 test points in the 10° range with the 6° grid in the 24-2/30-2 program. In comparison, the G-1 program in the Octopus (Haag-Streit AG, Koeniz, Switzerland) covers the central visual field better with 17 test points in the 10° range and is considered a good compromise to the special macula program [24].

In addition, in achromatic perimetry, the first scotomas only appear when there is a loss of approximately 30% of retinal ganglion cells [25]. As a result, early visual changes are not detected in time. Motion perimetry methods such as frequency doubling perimetry (Matrix, Zeiss Meditec) or flicker perimetry (Pulsar, Haag-Streit) have proven to be good complementary perimetric methods that can detect visual changes earlier [26,27].

In general, a distinction can be made between POAG-independent and POAG-associated central visual field defects. The first group includes additional pathologies of the macula that are not directly related to POAG, such as age-related macular degeneration, macular epiretinal gliosis, or myopic maculopathy.

On the other hand, POAG itself causes a significant decrease in RGCs, including in the macula. While the average estimated number of RGCs in the macula in healthy eyes was 520,678 ± 106,843 cells, it was already lower in eyes with suspected glaucoma at 410,003 ± 83,887 cells, but in patients with glaucoma it was significantly reduced to 306,010 ± 109,447 cells. Thus, compared to the estimated number of RGCs in the macula, eyes with glaucoma had an average of 41% fewer RGCs in the macula of healthy eyes [21].

These figures clearly demonstrate the significant impact of glaucomatous neurodegeneration on the macular region, which is not solely affected by individually elevated intraocular pressure. This is because the oxidative stress triggered by primary mitochondrialopathy [28,29], which has been significantly detected in both aqueous humor and serum [30], causes retinal and systemic neuroinflammation [31]. This also leads to oxidative stress and neuroinflammation in the optic nerve and lamina cribrosa with activation of microglia, astrocytes, and connective tissue lamina cribrosa cells [12]. In addition to these direct impairments of the RGC and their corresponding nerve fibers, the inflammatory changes in the cellular environment lead to disturbances in the metabolic autoregulation of papillary perfusion and, due to extensive disruption of the central nervous system in POAG [32], to a negative influence on the peripheral autonomic nervous system [33], so that the pressure-dependent autoregulation of papillary perfusion is also disturbed in POAG [34]. In addition, mitochondrial dysfunction itself causes vascular endothelial dysfunction [35], which can further worsen papillary perfusion. In this respect, the degeneration of RGCs and their nerve fibers is a complex process involving mechanical, vascular, and, above all, inflammatory processes [36] that have a direct influence on the development of perimetric disturbances [37,38].

Since 75% of POAG patients have arterial hypertension, 50% have dyslipidemia, and 30% have diabetes mellitus [39], all three of which trigger secondary mitochondriopathy [40,41,42], this leads to a further increase in oxidative stress, neuroinflammation, and small vessel disease. Clinically, these considerations are confirmed by the fact that central visual field defects in POAG occur more frequently in the presence of arterial hypertension or diabetes mellitus [43]. In normal-tension glaucoma, a connection between central visual field defects and migraine, Raynaud's disease, sleep disorders, low blood pressure [44], and nocturnal blood pressure drops 45] has been demonstrated. In advanced glaucoma, the use of antihypertensive therapy is an additional risk factor for central visual field defects [46], which underscores the importance of arterial hypertension. Other risk factors for central visual field defects include optic disc hemorrhages [47,48], reduced choroidal vascular density in the parapapillary β-atrophy zone [49], nasalization of the central retinal vessel trunk [50,51], and reduced vascular density in the foveal avascular zone as well as its enlargement [52]. In the context of biomechanical instability of the lamina cribrosa in POAG [53], a connection with reduced corneal hysteresis and central visual field defects has also been demonstrated [54].

Usually, the nasal step is the earliest visual field defect in POAG patients, followed by paracentral and arcuate-like scotomas, respectively, and the superior hemifield is more often affected than the inferior hemifield [55]. However, central visual field defects occur even in the early stages of glaucoma and are about as common as peripheral visual field defects [56,57,58]. It has also been shown that optic discs with localized notches, known as the focal ischemic type, are significantly more likely to have central visual field defects than optic discs of the senile sclerotic type [59]. Since central visual field defects can progress more rapidly than peripheral scotomas [60], their detection is of great importance, as central visual functions are directly threatened. Temporal papillomacular bundle visual subfield defects were significantly associated with future deterioration in visual acuity in advanced glaucoma [61].

Conclusion

Central visual field defects in glaucoma are given far too little attention in everyday life, even though approximately 50% of retinal ganglion cells are re-

sponsible for the central 16° of the visual field alone, and 40% of these cells are affected in glaucoma. The early clinical effects of these central RGC losses are color vision disorders, contrast sensitivity disorders, crowding (difficulty recognizing objects when they are close together), and reading difficulties [62,63], which, like color perception disorders [64], can be detected before the onset of scotomas in achromatic threshold perimetry

However, early perimetric changes in the central visual field can be detected with special glaucoma programs, such as the G-1 program, or with macula programs before structural changes are visible in OCT [65], which is also to be expected from the biological sequence of neurodegenerative events [66]. It is clinically important to consider accompanying vascular system diseases in POAG, which have a significant influence on the development of central visual field defects in glaucoma. It is therefore essential to evaluate patients with POAG with a blood count (LDL cholesterol, HbA1c) and a 24-hour blood pressure profile and to adjust their treatment accordingly [67]in order to keep central visual field defects to a minimum. This is because the primary goal of glaucoma therapy is not purely to reduce intraocular pressure numerically, but to preserve visual function [68], which requires a holistic therapeutic approach combining local and systemic therapy.

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