Reviews

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Compressive optic neuropathy in the setting of tumors of the chiasmal and sellar region: a review

K. S. Iegorova

SI " Romodanov Neurosurgery Institute, National Academy of Medical Sciences of Ukraine"

Kyiv (Ukraine)

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This is a review on compressive optic neuropathy (CON) in the setting of tumors of the chiasmal and sellar region. CON is a difficult to manage neuro-ophthalmological condition that occurs when the optic nerve is damaged due to compression of the anterior visual pathway, commonly by scull-base tumors of the middle and anterior fossae. Compression of the optic nerve/ chiasm complex is accompanied by reduced vision, visual field defects and descending optic atrophy, which, if not treated properly, can lead to permanent vision loss and blindness. The literature data on the prevalence and features of visual disturbances in chiasmal-sellar tumors which differ in histology have been reviewed.

Introduction

Compressive optic neuropathy (CON) is a condition that occurs when the optic nerve is damaged due to gradual or abrupt compression of the anterior visual pathway. Skull-base tumors of the middle and anterior fossae typically cause mass effect on the optic nerve/chiasm complex (ONCC), which is accompanied by reduced vision, visual field defects and descending optic atrophy (OA). CON is relatively rare, with an estimated incidence of about 4 cases per 100,000 individuals per year [1].

Clinical manifestations of sellar and parasellar tumors vary and depend on tumor involvement, nature of growth pattern, and rate of growth. Due to the proximity of the visual pathways, visual disturbances can develop in early disease, and are the basic clinical findings in most tumors of the chiasmal and sellar region (CSR) [2, 3, 4, 5, 6]. A multidisciplinary team approach is recommended for effective diagnosis and management of a patient with CON, since it is an essential prerequisite for a low rate of severe complications and increased duration of active life in patients with tumors of the CSR. The opthalmological aspect of CON is associated with frequent ocular manifestations of these tumors, which, when combined with late diagnosis of the tumors, can lead to complete loss of visual functions and legal visual disability. The literature data on the prevalence and features of visual disturbances in chiasmal-sellar tumors which differ in histology have been reviewed.

Anatomy of the chiasmal and sellar region

The CSR is located in the center of the cranial base, posteriorly to the posterior wall of the sphenoid sinus, and between cavernous sinuses. The region is basically composed by the body of the sphenpoid bone, in the central portion of which is located the sella turcica. The pituitary gland formed by the adenohypophysis (anterior pituitary) and neurohypophysis (posterior pituitary), lies in the sella turcica. The sella turcica is separated from the cavernous sinus by a thin and weak wall of the connective tissue so that sellar tumors frequently extend to the cavernous sinus. The anterior borders of the sella are formed by the anterior clinoid processes of the lesser sphenoid wing and the tuberculum sellae. Above the tuberculum sellae is the chiasmatic sulcus; the latter is bounded anteriorly by the chiasmatic ridge, which forms the posterior border of the planum sphenoidale (Fig. 1) [7, 8, 9].

Anatomy of the chiasm

The optic chiasm is an important neuroanatomy structure which lies over the sella turcica. The anterior cerebral and anterior communicating arteries, the lamina terminalis, and the third ventricle are located above the chiasm, and the diaphragma sellae (a portion of the dura that covers the sella but that contains an opening for the pituitary stalk), below the chiasm. Laterally the optic chiasm is abutted by the supraclinoid internal carotid arteries (Fig. 2) [10, 11]. The anatomical location of the chiasm renders it susceptible to compression damage from lesions arising from the surrounding structures. However, anatomic variations are common and influence the clinical presentation and type of visual field defects. The relation between the chiasm and the sella is determined by the length of the intracranial optic nerve and is important in the

development of visual disturbances. The length of the optic nerve varies between 35 and 55 mm from the globe to the chiasm, and the length of the intracranial optic nerve is the most variable (4-17 mm) [12]. A study by Zander (1896), however, has shown that the length of the intracranial optic nerve varied from 6 to 21 mm, with a mean value of 13 mm [13]. The optic nerves rise from the optic canal to the chiasm at an angle of 15–45° [14] (Fig. 3). The average distance between the tubercle of the saddle and the anterior edge of the optic chiasm is 6.28 mm with variations of 1 to 10 mm depending on the length of the intracranial optic nerve [15]. The optic chiasm is 5-12 mm (average, 8.73 mm) long, 12.33-15.0 mm wide anteroposteriorly, 1.93-3.5 mm hight, and 2-8 mm (average, 4.13 mm) in thickness, with its area ranging from 27.07 to 43.7 mm2), as assessed by magnetic resonance imaging (MRI) [15, 16, 17, 18, 19]. Measurements of chiasmal width have been cited as the most clinically useful marker for optic atrophy and is defined as a width of <13.5 mm [20].

The normal chiasm overlies the diaphragm sellae and the pituitary gland, the prefixed chiasm overlies the tuberculum, and the postfixed chiasm overlies the dorsum (Fig. 4). When the chiasm is prefixed, the optic nerves are short, the chiasm sits forward over the sella, and the optic

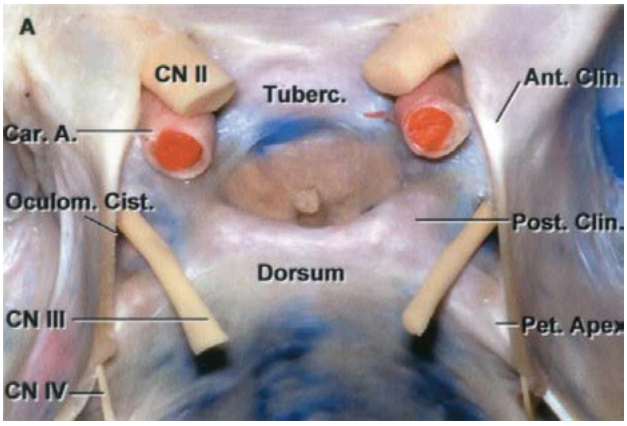

Fig. 1. Superior view of the anatomy of the chiasmal and sellar region (Rhoton AL Jr, 2002)

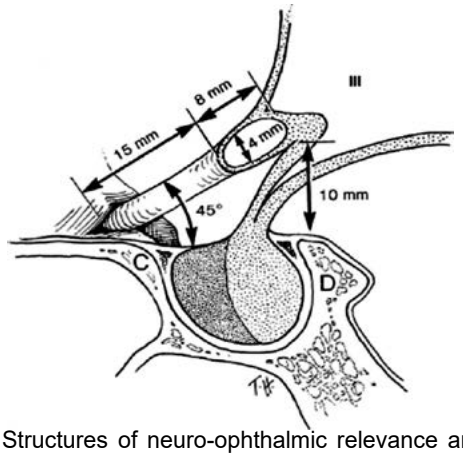

Fig. 3. Structures of neuro-ophthalmic relevance and their relative positions in the chiasmal and sellar region (Schiefer U, Wilhelm H. Clinical Neuro-Ophthalmology: A Practical Guide. Berlin, Germany: Springer; 2007)

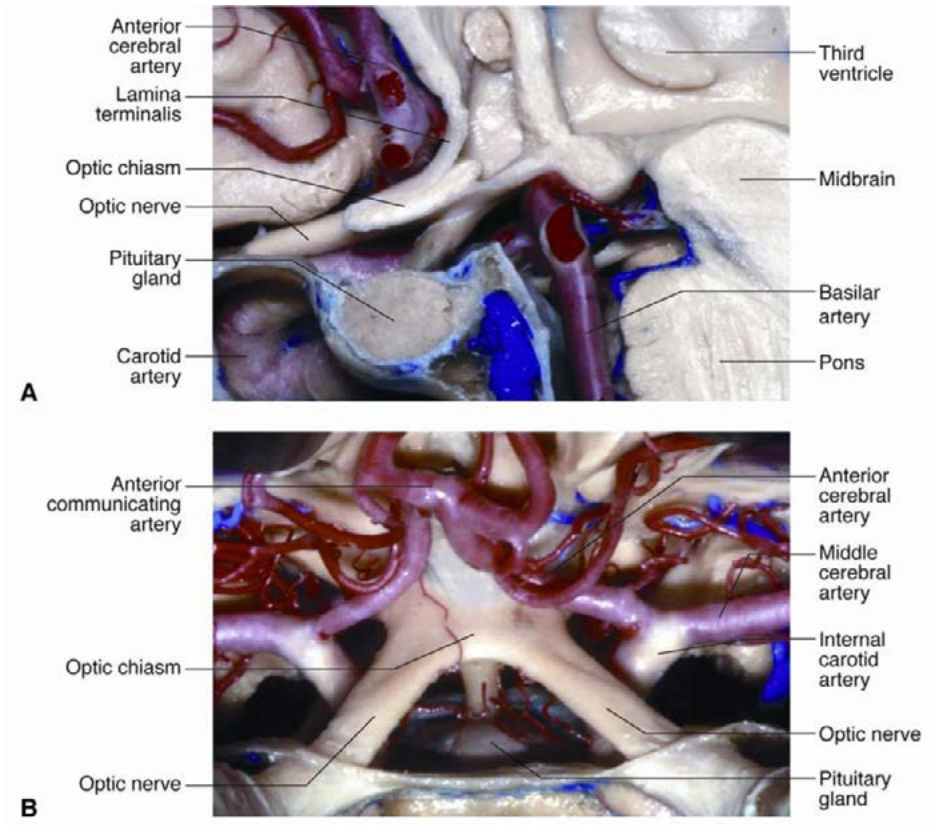

Fig. 2. Sagittal view (A) and superior view (B) of the anatomy of the optic nerve/chiasm complex (Rhoton AL Jr, 2002)

tracts are long. When the chiasm is postfixed, the optic nerves are long, the chiasm sits posteriorly over the sella, and the optic tracts are short. The chiasm is in the normal position in approximately 70-80% of cases, prefixed in 9–15% of cases, and postfixed in 11–15% of cases [9, 14, 21, 22, 23, 24].

The blood supply to the chiasm and the entire optic nerve/chiasm complex varies depending on the individual's anatomy. The chiasmal blood supply appears to be separated into a superior and an inferior group of arteries. The superior group of vessels is derived from the two anterior cerebral arteries and, occasionally, from the anterior communicating artery above the optic pathways. The inferior group is derived from the basilar, the posterior communicating, the posterior cerebral, and the internal carotid arteries [25].

Blood is supplied to the intracranial portion of the optic nerve and chiasm by the arteries arising from the ophthalmic, posterior communicating, and choroidal branches of the C4 segment of supraclinoid internal carotid artery [26]. The hypophyseal and infundibular arteries play the most important roles. The superior hypophyseal arteries (SHA) are a group of one to five small branches that arise from the C4's ophthalmic segment and terminate on the pituitary stalk and gland, but also send branches to the optic nerve and chiasm. The largest of the branches is often referred to as the SHA.

The infundibular arteries are a group of arteries that originate from the posterior communicating artery to the pituitary stalk. The SHA and infundibular arteries intermingle and form an anastomotic plexus called the circuminfundibular anastosmosis, and it is the small ascending arteries arising from the plexus that supply the inferior surface of the optic chiasm.

There is a map of the visual field on the retina, and a map of the retina on the various central visual nuclei. A general principle of retinotopicity is respected throughout the visual pathway. Peripheral neuronal fibers are definitely arranged in different compartments; this affects clinical manifestations if the pathological process develops [22].

The anterior visual pathway is composed of axons from retinal ganglion cells (RGC), which form the optic nerve, optic chiasm, and optic tract. The retinal nerve fiber layer (RNFL) axons are derived from the ganglion cells of the macular retina, which become organized at the neural-retinal rim (the tissue between the papilla of the optic disc and the edge of the optic cup), beyond which point they are consolidated as the optic nerve (composed of about 1.2-1.5 million axons). Within the retina, the RGC axons follow a particular pathway as they enter the optic disc. The axons from the nasal macula project directly to the disc, forming the papillomacular bundle. These nasal macula retinal fibers decussate at the chiasm. The axons from the temporal macula arch around the nasal macular axons, forming the superior and inferior parts of the papillomacular bundle [27].

The axons in the optic nerve are arranged in a distinct pattern. Immediately behind the globe, papillomacular fibers are arranged peripherally in the inferior portion of the outer optic nerve. The fibers are triangular in shape, and the triangle base is attached to the periphery of the cross-section. Uncrossed optic nerve fibers behind the globe appear like two isolated bundles separated by papillomacular fibers. In that part of the optic nerve where the papillomacular bundle occupies a central position, both bundles of uncrossed fibers merge with each other, forming one crescent-shaped bundle, which occupies a ventrolateral position. The crossed fibers along the entire length of the optic nerve are presented in the form of a single bundle located dorsomedally (Fig. 6) [28, 30].

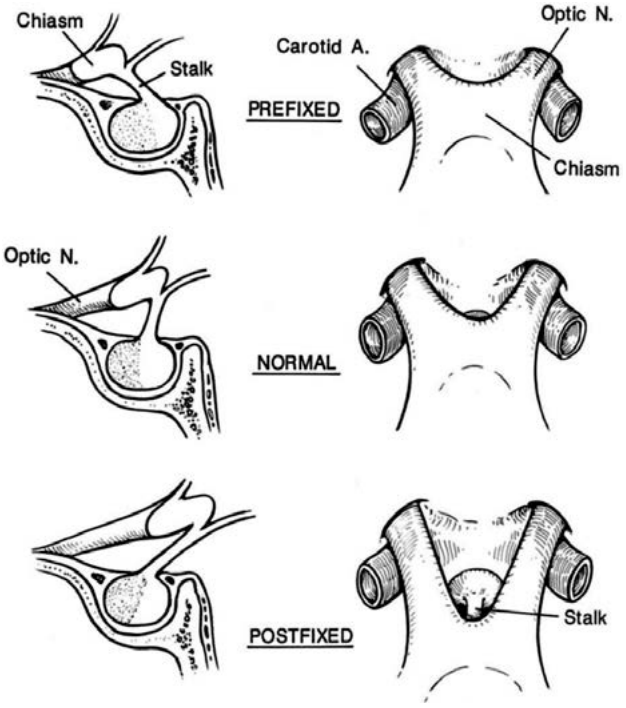

Fig. 4. Locations of the optic chiasm (Rhoton AL Jr, 2002)

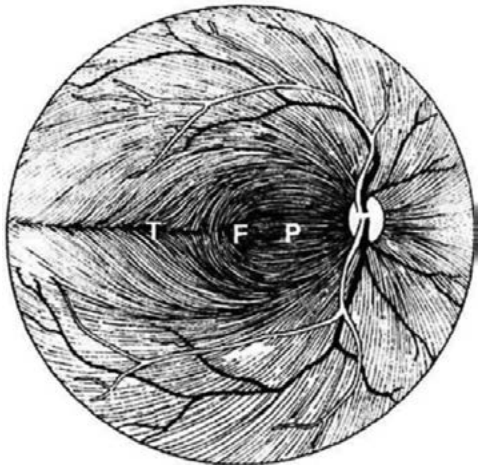

Fig. 5. Retinal nerve axons in the retina. Note: F, fovea; P, papillomacular bundle; T, temporal axons (Miller et al., 2005)

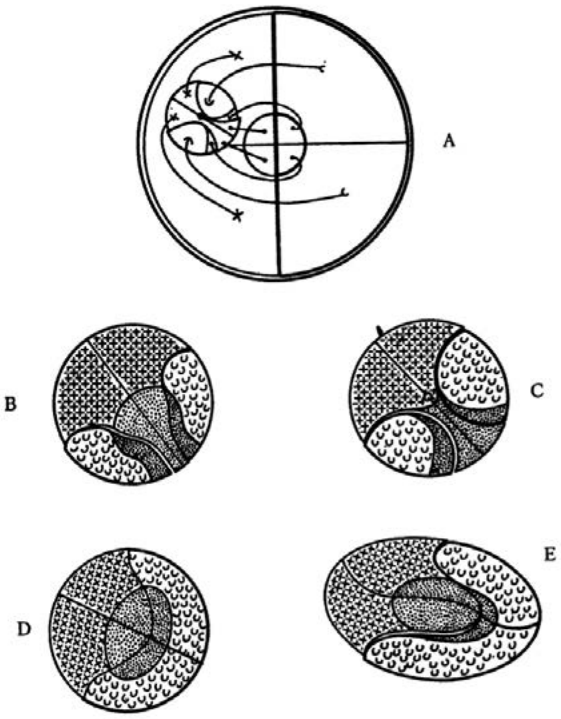

Fig. 6. Schematic representation of the pathway of fibers in the optic nerve (Henschen S, 1990; Vit VV, 2003). Note: A, retina and optic disc; B, optic disc behind the globe; optic disc behind the entrance of the central vessels; C, posterior orbital optic nerve; E, intracranial portion

The optic chiasm contains approximately 2.4 million nerve fibers as the two optic nerves join. Within the chiasm, the crossing fibers from the superior retina cross in the caudal chiasm and enter the medial side of the optic tracts, whereas those from the inferior retina cross more rostrally entering the optic tract laterally. Macular fibers make up a large proportion of the chiasm and contain both crossed and uncrossed fibers. Macular fibers are located superiorly and centrally, and are not present in the inferior regions of the chiasm [27, 29, 30, 31, 32, 33, 34].

The peripheral axons from the temporal retina arch around the papillomacular bundle and enter the disc at the superior and inferior poles. These fibers from the retina temporal to the fovea remain uncrossed in the lateral part of the chiasm and project directly to the ipsilateral optic tract. The empirical evidence informing the proportion of crossed versus uncrossed fibers is limited. One autopsy study of a single human chiasm found a ratio of 53:47 [35], while a further study of a single human chiasm found a ratio of 56:43 using an indirect assessment by looking at the ratio of crossed to uncrossed laminae in the lateral geniculate nucleus [36].

Wilbrand's knee was initially described as a loop of crossing fibers from the inferonasal retina, deviating into the pre-chiasmal part of the contralateral optic nerve before entering the optic chiasm. Wilbrand first identified these fibers in silver-stained sections from a human chiasm where one eye had been enucleated a number of years before death. Wilbrand's knee was accepted as an explanation for the anterior junctional syndrome (ipsilateral central visual field defect and a contralateral temporal hemianopia respecting the vertical meridian) [37]. Hoyt examined the fiber organization of the optic nerve and chiasm in 1963. He found no evidence for Wilbrand's knee and rejected its importance for the topical diagnosis of chiasmal lesions. His conclusion is supported by new data suggesting that Wilbrand's knee is absent normally, but appears after atrophy of one optic nerve [38, 39, 40, 41].

Chiasmal-sellar tumors

Chiasmal-sellar tumors comprise approximately 10–15% of all intracranial tumors [42, 43]. The clinical picture depends on whether or not the tumor is secreting one or more of a variety of hormones, as well as on tumor size, and direction of tumor extension.

Benign chiasmal-sellar tumors include pituitary adenomas (PAs), tuberculum sellae meningiomas, craniopharyngiomas, germinomas, dermoid and epidermoid cysts, lipomas, teratomas and germinomas; malignant chiasmal-sellar tumors include chordomas, chordosarcomas, gliomas, astrocytomas, and cancer metastases [44, 45]. The chiasm can be compressed by several types of lesions given its unique location [21].

Pituitary adenoma

PAs are the most common primary benign intracranial chiasmal-sellar tumors that can affect the chiasm and cause CON; they comprise approximately 10–15% of all extracerebral intracranial tumors [44, 46, 47, 48]. Ezzat and colleagues [49] conducted a meta-analysis of all existing English-language articles in MEDLINE to determine the prevalence of pituitary adenomas. They found an overall estimated prevalence of pituitary adenomas of 16.7% (14.4% in autopsy studies and 22.5% in radiologic studies). Epidemiologic data prior to 1969 indicated an annual incidence of 1.85 per 100,000 population. More recent epidemiologic studies show that PAs are increasing in incidence (between 3.9 and 7.4 cases per 100,000 per year) [50, 51]. The estimated annual incidence of PA in Ukraine was about 2.4-3.0 per 1,000,000 population [52].

In a functioning (i.e., secreting) pituitary adenoma (SPA), a severe clinical disorder or syndrome (acromegaly, Cushing's disease, or hyperprolactinemia) develops, and, therefore, the diagnosis can be made early, when the tumor is small. In a non-secreting pituitary adenoma (NSPA), the early stage is often asymptomatic. In elderly patients, NSPA and prolactinomas may be large and even giant-sized, which causes difficulties in their management [53, 54].

The clinical picture depends on the PA growth direction. The Yasargil classification of 1996 [55] reflects PA growth directions in details and is important in extracellular pituitary adenomas. Suprasellar growth is the main axis of extension of sellar tumors, with the tumor expanding upward through the sellar diaphragm and displacing the ONCC [43]. PAs can reach the anterior edge of the chiasm, where they can compress the crossing fibers going from the

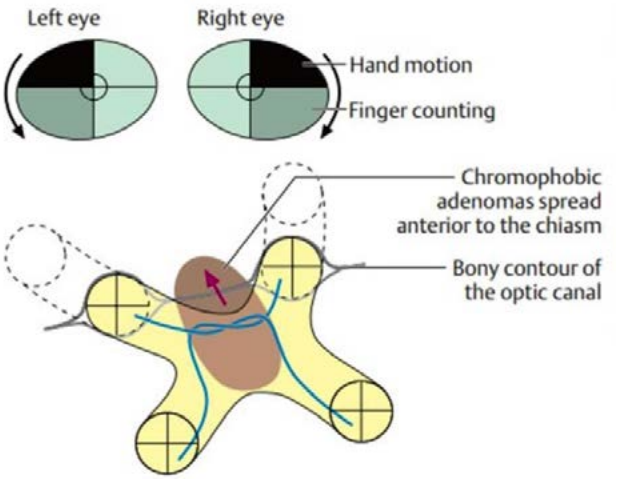

Fig. 7. Visual field defect caused by inferior compression of the optic chiasm by a pituitary adenoma (Lang G, 2000).

inferior nasal retina. Thereby, the visual field defect begins as a bilateral superior temporal defect and may progress to complete bilateral temporal hemianopsia (Fig. 7).

In pituitary adenomas with a supraparasellar extension, a tumor grows upward, causing ONCC and lateral cavernous sinus (CS) compression. The Knosp classification is an MRI tool used to define CS invasion in the 2017 World Health Organization classification [56].

PAs can be divided by size into microadenoma (< 10 mm) and macroadenoma (> 10 mm). Microadenomas can be subdivided by size into four types: small (16–25 mm), moderate (26–35 mm), large (36–59 mm) and giant (> 60 mm). Giant pituitary adenomas are tumors 4 cm or greater in maximal diameters, although there is no consensus for the criteria, and they account for 5-27% of all adenomas in surgical series [57, 58, 59, 60, 61].

Due to their size, these tumors have a special clinical course, and managing them is a challenge. Most giant tumors are diagnosed late and cannot be excised radically.

Anterior visual pathway compression typical for macroadenoma develops at the ophthalmological phase of the disease and is an absolute indication for surgical intervention.

Pituitary apoplexy

Pituitary apoplexy is a clinical condition characterized by sudden headache, nausea, vomiting, impaired consciousness, visual and oculomotor disturbances, and meningeal symptoms resulting from the hemorrhage or infarction of a pituitary adenoma. Changes in tumor tissue such as necrosis, hemorrhagic leakage, hemorrhage, formation of hematomas and intratumoral hemorrhagic cysts result in a rapid increase in tumor volume and changes in tumor relationships with surrounding brain structures [62, 63, 64]. Pituitary apoplexy is a rare emergency that occurs in a small but significant percentage (0.6-16.6%) of patients with pituitary adenomas. Impaired circulation in pituitary adenoma (pituitary apoplexy) causes an atypical clinical course of the disease which includes general brain symptoms, sudden substantial loss of visual function, and oculomotor impairment. This determines a high (above 45%) rate of false primary diagnosis of stroke or meningoencephalitis, which substantially worsens outcomes. Severe visual function impairment and persistent oculomotor symptoms are more common in delayed surgical interventions [69].

The most common symptom of pituitary apoplexy is headache, with the incidence of 90-100%. Neuro-ophthalmic manifestations are seen in 78-82.6% of cases of pituitary apoplexy, and include loss of vision, visual field defects and oculomotor disturbances [62, 69,71].

Craniopharyngioma

Craniopharyngiomas (CP) are benign epithelial tumors of the chiasmal and sellar region and/or the third ventricle region which arise from embryonic remnants of Rathke's pouch [72, 73].

The clinical features of the disease depend on tumor location with respect to the pituitary stalk, chiasm, sella turcica diaphragm and third ventricle. Yassargil (1996) [55] divided craniopharyngiomas into a) purely intrasellar, infradiaphragmatic; b) intra- and suprasellar, infra- and supradiaphragmatic; c) supradiaphragmatic, parachiasmatic, extraventricular; d) intra- and extraventricular; e) paraventricular w/ respect to 3rd ventricle and f) purely intraventricular. Because endosuprasellular craniopharyngiomas arise in the pituitary stalk, they can extend in various directions and cause the compression of the ONCC [55, 72, 73, 74].

On a population scale, however, craniopharyngiomas are relatively rare lesions, with an incidence of only 0.17-0.2 per 100,000 person-years. They comprise 0.8% of all intracranial neoplasms and constitute 13% of all suprasellular tumors. They are most common among children (0 to 14 years of age) and among older adults (aged 65-74 years), comprising 5-10% of all intracranial neoplasms and 56% of the sellar and suprasellar tumors in these age groups, although can occur in any age [75, 76].

Craniopharyngiomas are in close proximity to the surrounding nervous, endocrine and vascular structures of the brain such as the chiasm, optic nerves and tracts, hypothalamus, pituitary stalk, major vessels and their branches, including perforating branches of the anterior cerebral artery, posterior communicating artery, and posterior cerebral artery. The pathogenesis of visual disturbances is associated with the chiasmal compression by the cystic tumor component, devascularization of the optic chiasm, and direct tumor growth into the chiasm. Craniopharyngiomas compress the chiasm from above and behind with initial involvement of the upper nasal fibres, giving rise to bilateral inferotemporal defects. The defects then spread to involve upper temporal fields (Fig. 8).

Tumor relation to the optic tracts is an important aspect of surgery for craniopharyngioma. In large tumors, the sites of the ONCC are compressed, expanded and dislocated. The blood supply to the ONCC is usually not affected, which allows maintaining it perioperatively; it is, however,

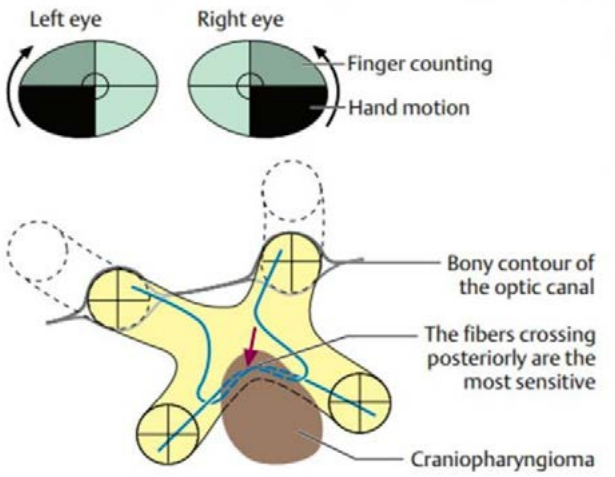

Fig. 8. Visual field defect caused by superior compression of the optic chiasm by a craniopharyngioma (Lang G, 2000).

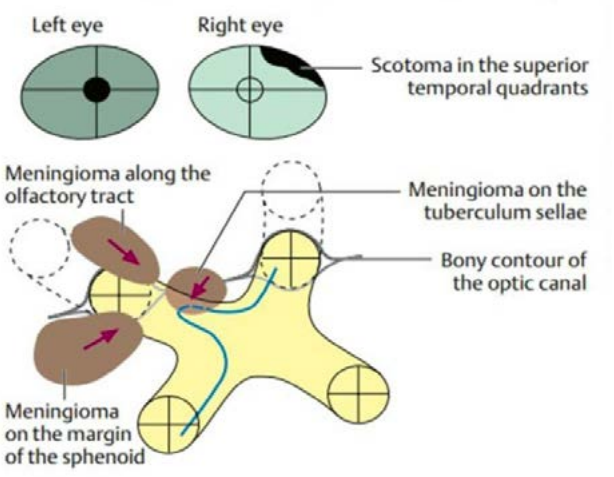

Fig. 9. Visual field defect caused by possible compression of the optic nerve by a meningioma (Lang G, 2000).

sometimes difficult to distinguish the branches supplying the tumor from those supplying the visual pathways.

Parasellar meningiomas

Meningiomas comprise approximately 20–25% of all intracranial tumors. The morphological features of the disease vary, although meningiomas commonly grow slowly [77]. Parasellar (or sellar and suprasellar) meningiomas are less common than pituitary adenomas or craniopharyngiomas [78, 79, 80]. The incidence of parasellar meningioma is 2.0 per 100,000 person-years [45]. Parasellar meningiomas can affect the ONCC; frequent origins of parasellar meningiomas include the tuberculum sellae, diaphragma sellae, and dorsum sellae. Tuberculum sellae meningiomas usually occur in the midline suprasellar region; as they enlarge, they displace the optic nerves laterally and the chiasm posteriorly and somewhat superiorly [81]. They cause not only mass effect on the ONCC, but also optic nerve compression in the optic canal due to the features of extension of parasellar meningiomas [82], with the compression affecting much more severely one optic nerve that the chiasm, which results in a mild visual field defect in the superior temporal quadrant in the ipsilateral eye and a severe visual field defect in the contralateral eye (Fig. 9).

Surgical removal of parasellar meningiomas is very difficult due to their proximity to the optic nerve, chiasm, anterior cerebral and anterior carotid arteries and their branches which are usually involved in the pathological process. Decompression of the ONCC usually does not result in improved visual functions if the disease progresses to blindness, which is a major cause of legal visual disability [82, 83, 84, 85].

Chiasmal syndrome

Compression of the ONCC results in visual impairments through the mechanisms involving metabolic, ischemic, mechanic and other lesions [86]. An experimental study by Kanamori et al. (2012) [27, 87] demonstrated retrograde and anterograde axonal degeneration occurring in response to injury to nerve fiber axons.

Impaired visual functions are observed in 67.8-83% of patients. Specifically, reduced visual acuity and visual field impairment have been found in 38-68.5% and 68-70%, respectively, of patients [11, 53, 88, 89]. 30-88.9% of cases manifest loss of visual acuity or visual fields [11, 89]. Visual disturbance was a major symptom in 38-72% of patients with a tumor of the chiasmal and sellar region who received surgical decompression of the chiasm [90, 91, 92].

Mean duration of visual disturbance symptoms until establishing a correct diagnosis ranged from 6 to 24 months [93, 94, 95, 96].

Late-diagnosed but mostly benign processes causing compression of the ONCC may result in temporary or permanent visual function loss leading to visual disability. Prolonged chiasmal compression results in the development of OA in 16 to 72% of patients, leading to blindness in 3.5% to 25% of cases [2, 90, 95, 96, 97, 98, 99].

Ocular motility disorders (OMD) are common in PA with CS invasion, occurring in 1.4% to 17% of patients with a conventional disease course and in 45 to 57% of patients with pituitary apoplexy [100, 101, 102, 103]. OMD are more typical for parasellar extension of pituitary adenoma and sphenoid meningioma, and develop as a result of compression or infiltration of the medial wall of the CS by the tumor, with damage to cranial nerve (CN) III, IV, and/or VI. Paralytical strabismus is accompanied by diplopia, dizziness, headache, and nausea instability of gait. Oculomotor (CN III) injury is more common than those of CN IV and/or VI, and is evident by ptosis, mydriasis, and limited upward, downward and inward movements of the globe. OMD develop gradually with an increase in tumor size or abruptly in pituitary apoplexy.

The gold standard of surgical treatment for a tumor in the chiasmal and sellar region is endoscopic transnasal

excision of the lesion which aims to decompress the anterior visual pathway and improve or restore the visual function [44]. Studies of the last century indicated that the visual function may recover within a month, with a relatively high recovery rate immediately after decompression of the chiasm, and a lower recovery rate subsequently until month 1 [12]. Recent studies, however, have demonstrated that the visual function may improve within three years, and this improvement can be divided into three phases: fast-recovery phase (several minutes to several days), delayed recovery phase (several weeks to several months), and late recovery phase (6 months to 3 years). Many researchers argue that the fast recovery phase occurs due to the recovery of the physiological conduction and improvement of axoplasmic flow in peripheral neuron fibers. The delayed and late recovery phases occur due to remyelination of nerve fibers [104, 105].

Longitudinal changes in visual function after decompression of the ONCC depend on patient's age, baseline visual acuity and visual fields, duration of the compression of the visual pathway, presence of the optic atrophy, and size of the tumor [47, 106]. It is the endoscopic skull-base surgery that has been reported to be the best option for visual recovery [44]. Predicting visual acuity and visual field outcomes after removal of chiasmal-sellar tumors is still an important task [107,108,109,110].

Until late last century, patients with chiasmal-sellar tumors were believed to be inoperable. Microsurgical techniques, neuronavigation, endoscopic techniques, neuroanesthesiology and neurovisualization are helpful in successful treatment of chiasmal-sellar tumors [111,112, 113].

Despite the development of advanced techniques for ophthalmological diagnosis, neurovisualization, endoscopy and microsurgery, there is an increase in the annual incidence of first diagnosed compressive optic neuropathy due to chiasmal-sellar tumors with partial or total loss of visual function [11, 97].

Conclusion

Therefore, management of ONCC lesions in the presence of CON is still a challenge and there is a need for developing effective techniques for their early diagnosis and selection of the most appropriate treatment plan. Further research is required for better understanding the clinical course of CON in the presence of chiasmal-sellar lesions depending on tumor size and extension, type of tumor growth, and whether or not the tumor is secreting one or more of a variety of hormones. There are a number of CON-related issues to be clarified and solved. The issue of early diagnosis of CON has not been completely resolved. Diagnostic criteria of CON have not been defined, and diagnostic sensitivity of several CON examination techniques has not been determined. Defining the diagnostic criteria and determining the diagnostic sensitivity of these techniques will be helpful for the early diagnosis of the disease. Impoving early diagnosis is essential for adequate and timely treatment, preventing irreversible changes, arresting the pathological process, and preventing optic atrophy and its sequelae. Live studies of imaging features of the ONCC using advanced neuroimaging techniques and morphological and structural parameters of the optic nerve and retina, with the comparison of the results obtained with visual acuity and perimetry findings, will enable better understanding of CON stages, and offering new diagnostic methods for early diagnosis and timely treatment of the condition. Since CON issues have not been addressed sufficiently in the literature, research should be undertaken to develop an algorithm for the examination, treatment and follow-up of patients with the condition.

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Disclosures

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Corresponding Author. *Kateryna Iegorova, Email: iegorova_katya@ukr.net*

Author Contribution. *KSIe: Conceptualization, Writing-original draft, Writing-review & editing*

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Abbreviations. *CON, compressive optic neuropathy; CP, craniopharyngioma; CSR, chiasmal and sellar region; NFPA, non-functioning pituitary adenoma; OA, optic atrophy; OMD, ocular motility disorders; ONCC, optic nerve/chiasm complex; OD, optic disc; PA, pituitary adenoma; PAP, pituitary apoplexy; PM, parasellar meningioma; PMB, pupillomacular bundle*