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Neuro-Ophthalmology Findings in Pituitary Disease (Review of Literature)

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Abstract

Visual symptoms often accompany pituitary diseases. Pituitary tumors may compress surrounding structures such as optic chiasm leading to visual field defects including bitemporal hemianopia and visual disturbance. They also may compress cranial nerves III, IV, and VI, leading to ocular motility abnormalities. Pituitary adenomas are the most common cause of chiasmal compression. Patients with nonsecreting tumors present initially with vision loss, and these tumors can reach large size without causing other symptoms; however, hormonally active tumors are detected before vision loss because of systemic symptoms. Acute hemorrhage or infarction of the pituitary tumor known as pituitary apoplexy causes diplopia, loss of vision, and visual field. Thus, the ophthalmologist's role is crucial in diagnosis and treatment of pituitary tumors. As visual loss may be the first sign of recurrence after treatment, it is essential to repeat visual field and visual acuity testing every 6–12 months.

Keywords: pituitary tumors, pituitary adenoma, pituitary apoplexy, chiasmal compression, bitemporal hemianopia, ocular motility abnormalities, craniopharyngioma, suprasellar meningioma, parasellar meningioma

1. Introduction

Pituitary diseases are the most common causes of extrinsic compressive chiasmal syndromes leading to visual disturbances. Chiasmal syndromes most commonly occur secondary to pituitary adenomas, craniopharyngiomas, meningiomas, and pituitary apoplexy.

Visual field defects may be one of the first signs of nonfunctional pituitary tumors growing in the vertical direction [1]. Visual disturbances are much less frequent in functional adenomas,

where systemic hormonal aberrations such as Cushing's syndrome, galactorrhea, and acromegaly usually predate the compressive signs [2].

However, pituitary tumors may grow in the horizontal direction to invade the cavernous sinus. This will cause dysfunction of cranial nerves III, IV, V, and VI, with less visual damage [1].

2. Anatomy

2.1. Optic nerve

The optic nerve extends from the retina to the optic chiasm; it is approximately 5 cm long and is divided into four portions: intraocular or optic disk (1 mm), intraorbital (25 mm), intracanalicular within the optic canal (9 mm), and intracranial which terminates in the optic chiasm (12–16 mm) [3].

2.2. Optic chiasm

The optic chiasm measures approximately 12 mm wide, 8 mm long in the anteroposterior direction, and 4 mm thick. It is inclined at almost 45°, located just anterior to the hypothalamus and anterior to the third ventricle, and situated about 10 mm above the pituitary gland, which rests in the sella turcica of the sphenoid bone. The chiasm and sella are separated by a space called the suprasellar or inferior chiasmatic cistern [4].

As a result of variations in the length of the optic nerves, the exact location of the chiasm with respect to the sella is variable [4]. Most of the time, (80%) it is central and lies directly over the sella (expanding pituitary tumors involves chiasma first). In approximately 10% of individuals, it is prefixed as it lies more anteriorly over tuberculum sellae (pituitary tumors involves optic tract first), and in approximately 10%, it is posterior (postfixed) lying more posterior over dorsum sellae (pituitary tumors damage the optic nerve) [5–8].

The pituitary infundibulum, which arises from the hypothalamus (ventral diencephalon) behind the chiasm, extends downward to the posterior lobe of the pituitary (neurohypophysis). The anterior lobe of the pituitary (adenohypophysis) forms embryologically from Rathke's pouch, an embryological structure connected to the pharynx. The chiasm is flanked laterally by the supraclinoid segments of the carotid arteries and inferolaterally by the cavernous sinuses [8, 9].

At the optic chiasm, retinal ganglion cell axons from the temporal retina remain ipsilateral, and those from the nasal retina cross the chiasm and course toward the contralateral brain [10]. However, there are intricacies in the chiasmal crossing [11]. In the process of decussating, fibers from the inferior nasal quadrant loop forward into the opposite optic nerve for a short distance before turning back again, forming Wilbrand's knee. In addition, some of the upper nasal fibers loop back briefly into the ipsilateral optic tract before decussation. In the chiasm, the fibers from the upper retinal quadrants lie superior, and those from the lower quadrants inferior. Inferior nasal fibers decussate anteriorly and inferiorly in the chiasm, while superior

nasal fibers cross posteriorly and superiorly. This accounts for the difference in the pattern of evolution of the field defect in infrachiasmatic versus suprachiasmatic lesions. Macular fibers more or less decussate as a group, forming a miniature chiasm within the chiasm, primarily in the posterior superior portion [11].

2.3. Visual field testing

The primary role of the clinician in the diagnosis of chiasmal disorders is to assess visual function accurately, interpret the results correctly, and, thus, localize the anatomical region that is affected. Visual field tests may provide a strong indication of direct chiasmal involvement, and failure to perform and properly interpret visual field tests is a common cause for delay in the diagnosis of chiasmal disorders [12].

Chiasmal injury is classically associated with bitemporal visual field defects due to damage to crossing fibers that carry temporal visual field information from the nasal retinas. Retinal ganglion cell axons from either side (right or left) of the macula travel together through the optic nerve but separate at the optic chiasm. This divergence creates the hemianopic midline and is the reason that all chiasmal and retrochiasmal visual field defects respect the vertical meridian [13].

Bitemporal defects that respect the vertical meridian indicate damage to the visual pathways at the optic chiasm [14]. It is important to establish that the vertical midline forms the border of the field depression and accordingly to rule out nonchiasmal temporal field loss that does not respect the vertical midline [12].

Bitemporal hemianopias may be complete (with no vision temporal to fixation in either eye), but when compression is early or mild, the hemianopia may be denser superiorly (when the chiasm is compressed from below) or inferiorly (when the chiasm is compressed from above) [13].

Because crossing macular fibers travel posteriorly, a lesion of the posterior chiasm may result in a central bitemporal hemianopia, sparing the more peripheral temporal fields [13].

Rarely, an anterior lesion at the junction of the optic nerve and the optic chiasm can injure crossing fibers from the opposite inferior retina (which loop forward into the optic nerve, forming Wilbrand's knee, before projecting posteriorly to the optic tract). As a result, the optic nerve visual field defected in one eye may be accompanied by a superior temporal scotoma in the other eye: the junctional scotoma [13].

Lesions causing compression of the optic tract produce dissimilar incongruous homonymous defects in the hemifields contralateral to the affected optic tract [15].

3. Ophthalmologist's role in pituitary tumors

Pituitary tumors are common benign tumors that represent about 12% of intracranial tumors [12].

Studies of several old series of pituitary tumors led to the old classical ophthalmology teaching that visual field abnormalities were an important early diagnostic sign of pituitary tumor [16].

Henderson [17] reviewed Cushing's series of 338 pituitary tumors in 1939. He found that chromophobe adenoma was almost never diagnosed before it had grown large enough to compress the anterior visual pathways causing field defects. Furthermore, he felt that no operation should be undertaken unless vision was at risk. In 1955, Chamlin et al. [18] reviewed 156 cases of pituitary tumors and found that 86% of these cases had field defects. Hollenhorst et al. [19] analyzed 1000 cases of pituitary tumors, which were diagnosed between 1940 and 1962, and found visual field defects in 70% of patients. Klauber et al. [20] reviewed 51 patients with pituitary tumors seen between 1967 and 1974 and reported a 69% incidence of field defects. Wray [21] examined 100 pituitary tumor patients between 1974 and 1976 and demonstrated field defects in 31%. Anderson et al. [16] reported, in their study of 200 patients with pituitary tumors seen between 1976 and 1981, 9% incidence of field defects.

In comparing the above six studies done before 1981 and including all pituitary tumors regardless of their origin, size, and presenting symptoms, it was obvious that the incidence of ophthalmologic findings was decreasing in patients with pituitary tumors. This can be explained by the following several developments which enabled earlier diagnosis and treatment before visual disturbance had occurred [16]:

1. Availability of prolactin assays: Prior to prolactin era, though the clinical syndromes of amenorrhea/galactorrhea in women had been described in pituitary tumors, their cause was not understood, as these tumors could be identified on plain skull x-rays, only in minority of patients when they were large enough to be seen. It is not surprising then that many of these patients received no specific therapy until they developed visual field defects.

2. Progress in radiologic imaging techniques: Prior to 1980, access to high-resolution CT scanning was limited, so this examination was carried out only in patients with large visual field defects. Since 1980 development of CT scanning techniques made it possible to visualize lesions smaller than 3 to 4 mm in diameter.

3. Development of surgical techniques: Before the development of selective transsphenoidal adenectomy techniques in which only the adenoma is removed and the rest of the pituitary is left intact, surgical removal of an adenoma was accomplished transfrontally by total hypophysectomy, with the resultant higher morbidity and mortality. Since the iatrogenic hypopituitarism was a major medical problem, surgical removal of pituitary adenomas was often not recommended until visual manifestations developed.

As a result of these developments, the ophthalmologist's role in the diagnosis of pituitary tumors appeared to be changing from the classical teaching [16]. Patients became less likely to present primarily because of visual complaints but more likely to be referred to the ophthalmologist by an endocrinologist, gynecologist, or neurosurgeon during the course of a workup of a suspected pituitary tumor. However, for the small group of nonsecreting tumors, the ophthalmologist still has a primary role in that these patients will often present first with field defects.

Later on Poon et al. [22] performed a prospective study on 29 patients with visual deficits who presented to the neurosurgical unit for removal of pituitary macroadenomas between

1992 and 1994. All patients had visual field defects. It is noteworthy that out of the 29 patients, only 11 (37.9%) were referred by an ophthalmologist, which means that visual disturbance and visual field defects were the first presenting symptoms in only 37.9% of patients with macroadenomas. Other patients were referred by general practitioners, neurologists, endocrinologists, gynecologists, general physicians, and optometrists in 34.5, 10.3, 6.8, 3.4, 3.4, and 3.4%, respectively.

Ren-Wen Ho et al. [1] reviewed retrospectively the records of 78 patients with pituitary adenomas who were referred for transsphenoidal adenectomy between 2009 and 2011. Among these patients, 24 had small macroadenomas (>1 cm to ≤2 cm), 37 had large macroadenoma (>2 cm to ≤4 cm), and 17 had giant adenomas (>4 cm). Abnormal visual fields were found in 65.4% of all patients. Patients with large macroadenomas and giant adenomas had a higher rate of abnormal visual field in comparison with patients with small macroadenomas (81.1, 94.1, and 20.8%, respectively).

The increase in incidence of visual field defects in patients with pituitary tumors in reports published after the 1980s (varies from 65.4–100%) [1, 22] may be explained by the fact that recent studies were more specific and included in general only patients with macroadenomas, who were referred to neurologists for transsphenoidal adenectomy (these patients are more susceptible to have visual field defects), while reports before 1980s were more general and included all pituitary tumors regardless of their size, presenting symptoms, and indication for surgery.

Another way in which patients with pituitary tumors may present to the ophthalmologist is investigation of ocular causes of headache. Besides searching for primary ocular causes of headache, the ophthalmologist should be aware of performing careful visual fields in all headache patients. In addition to performing careful visual field examinations on routine headache patients, the ophthalmologist must be willing to question these patients about reproductive and sexual dysfunction and to refer these patients for appropriate neuroendocrine and neuroradiologic investigation [16].

4. Pituitary adenomas

Pituitary adenomas make up 12–15% of symptomatic intracranial neoplasms [12]. They are the most common causes of chiasmal compression and may occur at any adult age; they are rare in childhood [15].

Typically, nonfunctioning adenomas present as macroadenomas that cause neurological symptoms due to intracranial mass effects, since hormonal inactivity leads to a delay in diagnosis compared with functioning pituitary adenomas [23]. If pituitary adenomas are not treated, vision will continue to deteriorate and blindness might result [24].

A relationship exists between severity of visual impairment and tumor size [25]. Pituitary adenomas are usually classified into microadenomas, macroadenomas, and giant adenomas according to their size [26–29]. The larger the pituitary adenoma is, the higher is the risk of optic chiasm or optic nerve compression [16, 19, 30]. In general, the size of macroadenomas

range from 1 to 4 cm. Smaller macroadenomas typically will not result in any visual field defect or visual impairment, whereas the larger ones will usually cause severe visual disability. Several studies clearly illustrated that patients with larger tumors tended to have visual field abnormality and that the severity of visual field defects was closely related to tumor size [31, 32]. Investigation of the relation between the severity of the visual impairment and the different sizes of macroadenomas by Ren-Wen Ho et al. [1] indicated that pituitary adenomas less than 2 cm usually have no or only a minimal effect on the visual pathway.

The typical visual field defect, bitemporal hemianopia, is due to the anatomical compression of the optic chiasm, which contains the crossing nasal fibers of each optic nerve. Nevertheless, the visual field defect actually depends on the relation between the optic chiasm and the tumor itself. If the tumor is anterior to the optic chiasm, or if the patient has an anatomical postfixed chiasm, conditions such as central scotoma, arcuate scotoma, and monocular visual constriction can be noted. If the tumor compresses the optic tracts, or the patient has a prefixed chiasm, a homonymous hemianopia might be seen [31, 33, 34].

Ren-Wen Ho et al. [1] found that large macroadenomas and giant adenomas leading to visual impairment are mostly nonfunctioning adenomas (97.8%). This result is consistent with other reports [16, 35] and can be explained by the absence of endocrine symptoms, which often result in a delay of the diagnosis since there are no early visual symptoms [16]. This explanation is consistent with what Monteiro et al. [32] have mentioned previously that nonfunctioning and prolactin-secreting adenomas are the most likely pituitary tumors associated with visual impairment.

In general, larger volume tumors will usually result in a higher risk of compression at the optic chiasm; however, this relationship is not found when tumor extension mainly occurs at the infrasellar or parasellar region instead of the suprasellar region. It was found that if the adenoma grows in the vertical direction, it will usually result in more severe visual impairment. If the adenoma grows in the horizontal direction, it will usually cause less vision damage. Horizontal growth of the adenoma may invade the cavernous sinus [1].

Furthermore, some studies have already discussed the relationship between the optic chiasm position and visual loss. Ikeda and Yoshimoto [36] found that visual impairment occurred when the displacement of the optic chiasm was more than 8 mm above the reference line on the sagittal image and more than 13 mm above on the coronal image on brain MRI. Monteiro et al. [32] have also shown that tumor exceeding 10 mm above the sagittal standard line and 12 mm above the coronal standard line had a significant effect on visual loss. Ren-Wen Ho et al. [1] found that significant visual impairment occurred when the optic chiasm was moved by the tumor more than 11.2 mm above the reference line on the sagittal view and more than 15.3 mm on the coronal image.

With regard to visual improvement after surgery for pituitary adenoma, Ren-Wen Ho et al. [1] reported visual improvement in 88.7% with complete recovery of vision in 27.67% of patients. They found that patients with large macroadenomas or giant adenomas experienced greater visual improvement after surgical resection compared with patients who had micro- or small macroadenomas, but patients with smaller pituitary adenomas still had a better visual outcome. Other series have reported that visual improvement depends on the surgical approach,

ranging from 74.7 to 93.4% [24, 37, 38]. Gnanalingham et al. [35] believed that better preoperative visual acuity and a smaller degree of impairment in preoperative visual field would have a better effect on the visual outcome.

There have been contradictory results regarding predictive factors for recovery of vision. Müslüman et al. [24] found that tumor size was not significantly associated with the postoperative visual impairment score, but preoperative visual deficit and the time interval between the initial visual symptom and surgery were the factors significantly associated with the postoperative visual impairment score. A shorter duration of symptoms, younger age, and a better preoperative best-corrected visual acuity (BCVA) have been reported to be associated with better postoperative recovery of visual field by some investigators [35].

Undoubtedly, vision can rapidly improve within minutes or days after tumor resection [39]. Among all surgical resection procedures, transsphenoidal adenectomy is likely most effective for providing rapid relief of visual symptoms in patients with a pituitary adenoma [40, 41]. Thus, early surgical resection of the tumor should be considered for patients with a large or giant macroadenoma causing visual loss, in order to preserve their vision.

5. Pituitary apoplexy

Pituitary apoplexy is the sudden enlargement of a pituitary gland, as a result from hemorrhage or infarction (most commonly hemorrhagic infarction) of a pituitary adenoma [42]. Pituitary apoplexy is typically associated with acute headache, visual field or vision loss, ophthalmoplegia, facial pain, or facial numbness.

Sudden expansion of the tumor into the adjacent cavernous sinuses can cause dysfunction of cranial nerves III, IV, V, and VI, with cranial nerve III as the most commonly affected one. Superior extension causes visual field loss; it also may cause central visual loss up to no light perception vision. Extravasation of blood into the subarachnoid space causes numerous symptoms, including a decreased level of consciousness and vasospasm with secondary stroke. The acute endocrine abnormalities may lead to numerous complications, including adrenal crisis. Therefore, recognition of pituitary apoplexy is crucial, as treatment is initiated emergently. Treatment includes immediate administration of corticosteroids, surgical decompression of the sella, and appropriate supportive measures. Some authorities recommend conservative management when neuro-ophthalmic signs are absent or mild [15].

The normal pituitary gland also may undergo hemorrhagic or nonhemorrhagic infarction, but such episodes generally do not cause visual loss and may go unrecognized until hypopituitarism develops. Predisposing factors include pregnancy, estrogen therapy, obstetrical hemorrhage (Sheehan's syndrome), diabetes mellitus, bleeding disorders, long-term anticoagulation, blood dyscrasias, radiation therapy, trauma, angiography, atheromatous emboli, cardiac surgery, coughing, positive pressure ventilation, and vasoactive agents [12].

As presentation of acute pituitary apoplexy is variable and its course is unpredictable, it should be considered in any patient with abrupt neuro-ophthalmological deterioration associated with headache. Although early investigators suggested that pituitary apoplexy occurs primarily

in patients with large macroadenomas, it is now evident that tumors of almost any size may undergo hemorrhagic necrosis [43, 44].

6. Lymphocytic adenohypophysitis

Lymphocytic adenohypophysitis is an immune-mediated diffuse lymphocytic infiltration of the pituitary gland; it has been reported to cause chiasmal compression from suprasellar extension [45]. This uncommon condition was reported in women only, and over one-half of the cases was found to occur during the perinatal period.

7. Craniopharyngioma

Craniopharyngiomas comprise 3% of all intracranial tumors, 13% of intracranial neoplasms in childhood, and 30% of all new growths in the hypophyseal area [46]. Since ocular signs are frequently the presenting features of these tumors, it is clear that their recognition by ophthalmologist is of great importance for establishing early diagnosis and treatment and more favorable prognosis.

Craniopharyngioma is a benign tumor, of ectodermal origin, arising from squamous cell remnants of Rathke's pouch. These cell remnants occur mainly on the infundibulum, between the undersurface of the brain and the upper surface of the pituitary gland [46]. The tumor may be entirely suprasellar or partially intrasellar, resulting in avascular solid tissue mass in which calcification may occur, as well as cysts filled with cellular debris and cholesterol crystals [46]. About 90% of tumors contain cysts, some of which may be very large [47]. These tumors may produce variable symptoms and signs depending on their size, direction of growth, and degree of compression of the optic pathways, pituitary area, hypothalamus, and third ventricle.

One of the striking features in craniopharyngioma is the variation in visual acuity and visual field loss, observed with disease progression [46]. Thus, information should be gathered from repeated field testing rather than from any single measurement [46]. These fluctuations can occur in both solid tumors (perhaps due to local edema) and in cystic variety (from intermittent emptying of the cyst into the ventricles) [48].

Kennedy and Smith [46] found in their study of 45 patients with craniopharyngioma that the majority of adults presented with visual failure and optic atrophy. Bitemporal hemianopia was fairly frequent (found in 27% of patients at the time of diagnosis) but was asymmetrical and unpredictable in its evolution. Homonymous hemianopia was relatively common (found as presenting feature in 11% of patients). Full fields were found initially in 20% of patients, a high incidence compared with pituitary adenomas or suprasellar meningiomas. Furthermore, Kennedy and Smith [46] detected pleomorphism (a distinct change from one type of field defect to another, with progress of the disease), which is a characteristic feature of the tumor (as indeed are fluctuations in the clinical state and visual acuity), in 22% of their patients.

In children, there are two main presenting syndromes [48]: (1) symptoms and signs of raised intracranial pressure, resulting from obstruction of the third ventricle by the tumor, and (2) visual failure from compression of the visual pathways. Kennedy and Smith [46] pointed out that, in those patients who have raised intracranial pressure, optic atrophy may be found rather than papilledema.

In addition, strabismus is a common finding in craniopharyngioma patients, and it can be classified into three categories [46, 49]:

1. Cases of concomitant esotropia or exotropia following marked loss of central vision in one or both eyes:

Kennedy and Smith [46] reported this type of squint as an early feature of craniopharyngioma in 30% of children included in their study. As the presence of optic atrophy or sluggish pupil may be missed in a young child, the poor vision in the squinting eye may be wrongly attributed to strabismic amblyopia until it is realized that the visual acuity is deteriorating despite treatment.

2. Cases of paralytic strabismus:

The incidence of paralytic strabismus was variable between different case series of cranio-pharyngioma. Love and Marshall [49] found in year 1950 third nerve palsy in 10% of their cases, while Hoff and Patterson reported in 1972 the incidence of cranial nerve palsies in 25% of children and 33% of adults [46]. Wybar found in 1971 two cases of third nerve palsy and one case of sixth nerve palsy, in 72 cases [46]. Kennedy and Smith [46] had only one patient in their series with transient sixth nerve paresis and were surprised not to have more cases of ophthalmoplegia, in spite of the massive size of the cystic tumor present in many of their craniopharyngioma patients.

3. Cases of non-paretic diplopia:

It is characterized by episodes of diplopia in the absence of demonstrable ocular palsy or defect of ocular movement [46], which was attributed to disturbance of binocular vision, secondary to bitemporal hemianopia and reduction of the area of visual field overlap common to the two eyes.

Endocrine disorders are also common in craniopharyngioma especially in adults, and mental deterioration is a frequent presenting feature in patients over 30 years of age [46]. Straight x-rays of the skull are practically diagnostic of craniopharyngioma in children, but in adults a normal x-ray does not exclude the diagnosis.

8. Sellar and suprasellar meningiomas

Meningiomas in the region of the sella turcica are rare, representing about 1% of all sellar masses [50]. They typically originate and involve the suprasellar region but in unusual cases arise from within the sella. Sellar/suprasellar meningiomas can mimic both clinically and radiologically, any of the other nonhormone-secreting sellar region masses, in particular the nonfunctioning pituitary adenomas. They can cause loss of visual acuity, visual field distur-bances, hypopituitarism, hyperprolactinemia, or a combination of the above [51].

Kwancharoen et al. [52] analyzed a retrospective series of subset of 44 patients with sellar/suprasellar meningiomas and adequate MRI imaging study, who underwent surgery at the Johns Hopkins Hospital during a 12-year period (between 2000 and 2012). They were mostly diagnosed in the sixth decade (age ranges between 30 and 78 years). The female/male ratio was 6:1, quite higher than the reported one in the previous series and higher than the ratio found in the entire meningioma cohort (2.7:1) [53–55]. The most common presenting symptoms were visual disturbance (85.96%), although some series report them in an even higher proportion (82.4–100%) [53, 54], and this was followed by headaches (49.12%), diplopia (12.28%), and ptosis (7.02%). A small proportion of patients had symptoms of endocrine dysfunction. Among these, hyperprolactinemia was the most common one, highlighting the fact that prolactin can be above the threshold in nonprolactin-secreting sellar masses. An additional meningioma was found in two cases (3.5%), one in the region of the falx cerebri and one in the middle cranial fossa [52].

MRI is the investigation of choice in these cases because it can often show the dural origin of the tumor [52]. In agreement with the previous series [56, 57], most of the tumors (81.25%) reported by Kwancharoen et al. [52] were iso-intense in T1. On T2-weighted imaging, the tumors were hyperintense in 35%, iso-intense in 30%, and hypointense in 25% of the cases [52].

Due to the high vascularity, gadolinium enhancement of meningiomas is usually marked, homogeneous, and rapid [58, 59]. This enhancement pattern may be helpful in distinguishing them from adenomas. However, in Kwancharoen et al.'s sellar/suprasellar series [52] in almost 11% of cases, the mass was thought to be a pituitary adenoma. This fact points out the significant challenges of establishing the diagnosis through radiology alone and necessitates further work to establish more accurate clinical and radiologic means of distinguishing between meningiomas and macroadenomas in the region of the sella turcica.

Because of the fibrous content in histology and expression of angiogenesis factors, meningiomas are usually adhesive, firm, and high vascular and may have major intraoperative bleeding and worse surgical outcome when compared to pituitary adenomas [60].

Previous series report worsening of vision in approximately 20–30% after surgery for sellar and parasellar meningiomas [53, 61–63]. Kwancharoen et al. [52] found deterioration of vision in 10.5% of cases after surgery (79.6% had improvement of one or more symptoms, improvement of vision occurred in 79.6%, and improvement of headache in 7.14%). It was proposed that surgical techniques might affect this outcome, as optic nerve manipulation and devascularization of perforating arteries supplying the optic apparatus are important factors for visual outcome [63, 64]. As Kwancharoen et al. [52] reported outcomes of more recent surgeries, the lower prevalence of vision deterioration when compared with previous reports might reflect the historical improvement in surgical techniques.

9. Parasellar meningiomas

Parasellar meningiomas occur most often in middle-aged women; arise most frequently from the tuberculum sella, planum sphenoidale, or anterior clinoid; and often produce asymmetric

bitemporal vision loss. Parasellar meningiomas may also enlarge and produce chiasmal compression during pregnancy [15].

Parasellar involvement manifests with abnormalities of ocular motility, pupillary function, or facial sensation from injury to cranial nerves III, IV, V1, V2, or VI or the ocular sympathetic nerves in the parasellar region [12]. Injury to these structures within the cavernous sinus may be associated with complaints of diplopia, ptosis, unequal pupil size, accommodative difficulty, facial pain or numbness, or eye pain. Signs include ocular motor nerve palsies, decreased sensation in the areas innervated by the first and second divisions of the trigeminal nerve, or Horner's syndrome [12]. Multiple cranial nerve involvement is more suggestive of invasive malignant tumors [12].

Therapy of parasellar tumors is complex and depends on the age of the patient; the nature, location, and extent of the tumor; its hormonal activity; and the severity of symptoms [15].

The ophthalmologist's role in the management of parasellar tumors is crucial, in that the first sign of recurrence may be vision loss [15]. Baseline visual field and visual acuity testing should be performed 2–3 months after treatment and at intervals of 6–12 months thereafter, depending on the course. Visual acuity and visual fields should be rechecked more often (immediately if necessary) if the patient notes any ongoing change. Periodic neuroimaging is essential [15].

Delayed vision loss after therapy for parasellar lesions should prompt the following considerations [15]:

• Tumor recurrence.

• Delayed radio necrosis of the chiasm or optic nerves.

• Chiasmal distortion due to adhesions or secondary empty sella syndrome, with traction on the chiasm.

• Chiasmal compression from expansion of intraoperative over packing of the sella with fat.

Neuroimaging effectively helps differentiate among these entities and guides further management decisions [15].

10. Conclusion

Due to developments in laboratory assays, radiological imaging, and surgical techniques, patients with pituitary tumors became less likely to present primarily to the ophthalmologist because of visual complaints. These patients are more likely referred to ophthalmologists by the endocrinologist, gynecologist, or neurosurgeon at earlier stages of pituitary disease during the course of workup. However, the ophthalmologist still has a primary role in patients with nonsecreting tumors, where patients will present first with visual and field defects. Patients with pituitary apoplexy usually present with acute headache, visual field or vision loss, ophthalmoplegia, facial pain, or facial numbness. In craniopharyngioma, strabismus is a common finding. It can be concomitant or paralytic. In addition, the patient may complain

of non-paretic diplopia. The most common presenting sign in sellar/suprasellar meningiomas is visual disturbance, followed by headaches, diplopia, and ptosis. Parasellar meningiomas manifest with ocular motility abnormalities, abnormal pupillary reaction, as well as abnormal facial sensation; these complaints result from injury to cranial nerves III, IV, V1, V2, or VI and ocular sympathetic nerves in the parasellar region.

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