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Modern neuro-ophthalmological evaluation of patients with pituitary disorders

Anne-Laure Vié, MD, Ophthalmologist [a, *],
Gérald Raverot, MD, PhD, Endocrinologist [b, 1]

[a] Neuro-ophthalmologist Service, Pierre Wertheimer Hospital, 59 Boulevard Pinel, 69677, Bron, France
[b] Endocrinology Federation Louis Pradel Hospital, 59 Boulevard Pinel, 69677, Bron, France

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Pituitary adenomas can manifest as ophthalmological symptoms, such as decreased vision, impaired visual field or diplopia. It is important to recognize these neuro-ophthalmological syndromes to achieve early diagnosis and treatment and to improve prognosis. Currently, ophthalmological examination includes precise measuring instruments, such as optical coherence tomography (OCT), which allows the evaluation of optic atrophy related to compression of the anterior optic tract. These measurements are reproducible and are useful for diagnostic and prognostic evaluation.

In this review, we describe the ophthalmological syndromes associated with pituitary tumours: anterior optic pathway compression, followed by oculomotor disorders and pituitary apoplexy.

Introduction

In 1978, Klaubet et al. [1] reported that the main complaint of patients with pituitary adenoma was a decrease in visual function. Nowadays, with accurate tests for hormone detection and advances in neuroimaging, the diagnosis of pituitary adenomas is made earlier. Only 30% of patients presenting with pituitary tumours complain of visual problems, and neuro-ophthalmological manifestations are

* Corresponding author. Fax: +33 472118014.
E-mail addresses: annelaurevie@yahoo.fr (A.-L. Vié), gerald.raverot@chu-lyon.fr (G. Raverot).
[1] Fax: +33 472681307.

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the initial symptoms in less than 10% of cases. Their incidence is dependent on the type of pituitary tumour. Functioning adenomas are usually diagnosed secondarily to systemic symptoms that are linked to hormone excess, before mass effect [2,3]. Non-functioning pituitary adenomas are often diagnosed later than functioning adenomas, have a larger tumour volume and represent 58% of pituitary adenomas with visual impairment [4]; however, they constitute only 28–33.2% of the pituitary adenoma population [5,6].

The pituitary gland is located in a dural sac in the sella and is surrounded laterally by the cavernous sinuses [7]. The optic chiasm, corresponding to the point at which optic nerves partially cross after passing through the optic foramen, is directly above the pituitary gland. When the tumour grows superiorly beyond the sella, compression of the central optic chiasm may be responsible for bitemporal visual field defects, and can cause a decrease in visual acuity [3,7,8]. Pituitary adenomas may also expand laterally and compress the cavernous sinus, causing oculomotor impairment. Occasionally, this can present as pituitary apoplexy, one of a few true ophthalmic emergencies. Apoplexy presents as severe headache with a sudden onset or rapid progression, associated with visual and ocular motor disorders.

In this review, we describe the ophthalmological symptoms associated with pituitary adenomas: anterior optic pathway compression, oculomotor disorders and pituitary apoplexy. Knowledge of pituitary adenomas and their recognition allows earlier diagnosis and can influence the management and prognosis of the tumour.

Anterior optic pathway compression

Anatomy

The optic nerves enter the intracranial space through the optic foramen in the sphenoid bones. After continuing for 8–15 mm upward and backward they join to form the optic chiasm. The chiasm measures 8 mm from the anterior to the posterior commissure, and is about 12–18 mm wide and 4 mm thick (Fig. 1).

Fibres arising from the nasal hemiretina (temporal visual field) of both eyes (nasal to the fovea-bisecting vertical meridian), corresponding to 53% of fibres, decussate in the chiasm to the

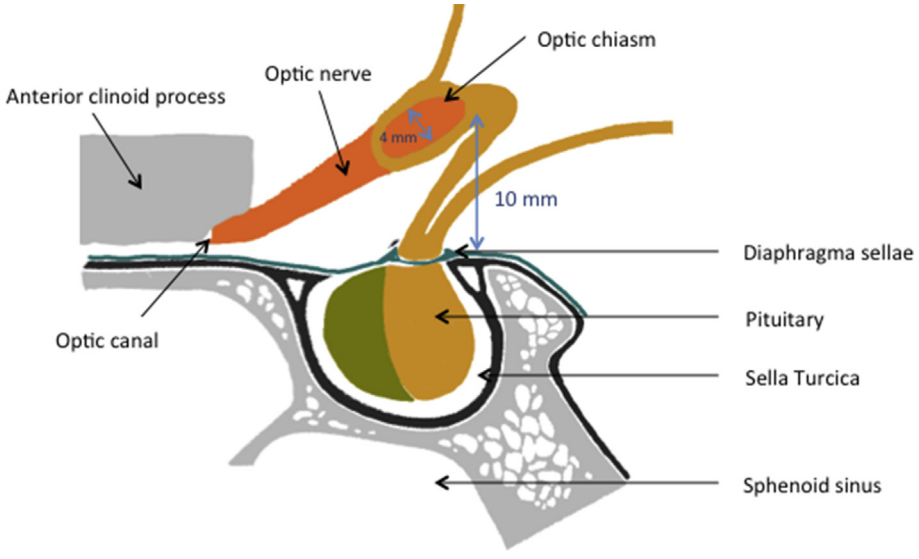

Fig. 1. Anatomy and relative positions of the peri-chiasmal region.

contralateral side. Fibres arising from the temporal hemiretina (nasal visual field) remain ipsilateral as they enter the optic tracts. Visual information corresponding to the right hemifield is encoded in the left optic tract, and *vice-versa*. Central chiasmal compression affects crossed nerve fibres associated with the nasal hemiretina, leaving uncrossed nerve fibres that originate in the temporal hemiretina relatively preserved [9,10]. Also, its compression is responsible for bitemporal visual field defects that respect the meridian line.

As they enter the chiasm, however, some ventral crossed fibres, primarily from the inferonasal retinal of the contralateral eye and serving the superotemporal portion of the contralateral visual field, were historically believed to loop anteriorly 1–2 mm into the terminal portion of the opposite optic nerve before turning posteriorly to continue through the chiasm and into the optic tract. This loop is called Wilbrand's knee [11,12]. Damage to these fibres is the cause of junctional Traquair syndrome (junctional scotoma).

An important factor influencing the nature of early visual field defect in chiasmal compression is the positional relationship between the chiasm and the diaphragm sellae. This varies significantly from one person to the other: in 75–80% of people the, optic chiasm is positioned just above the diaphragma sellae; in about 10% of people, the optic chiasm is positioned anteriorly, or 'pre-fixed', and it sits above the tuberculum sellae. This occurs when the intracranial optic nerve is shorter than about 12 mm. Compression of the posterior part of the chiasm is more likely to cause homonymous patterns of loss by damaging the optic tract; in 10–15% of people, the chiasm is positioned posteriorly to the dorsum sellae or 'post-fixed' [11,13], when the intracranial optic nerve is long, over 18 mm. Compression of the chiasm by masses approaching from the anterior aspect are more likely to cause monocular or highly asymmetric visual field loss.

Pathophysiology

Compression of the anterior visual pathways by a pituitary adenoma is responsible for visual impairment. The pituitary gland must rise a full 10 mm above the sellar diaphragm before abuting the chiasm. This can be suspected on imaging when the tumour induces a displacement of the visual pathways. Initially, compression induces axoplasmic flow disorder, blockage of conduction and demyelination, all of which are reversible. Longer or more intense compression induces axonal fibre degeneration and optic atrophy, which are irreversible.

A small degree of atrophy may have no functional effect. Advanced optic atrophy, however, induces decreased vision and visual impairment that can persist even after surgical decompression.

Clinical examination

The usual ophthalmological assessments that need to be carried out include refraction, visual acuity, intra-ocular pressure, ocular motor assessment, examination for ptosis, pupil, anterior segment slit-lamp examination and eye fundus examination. Visual field and OCT provide additional information essential to the clinical examination.

Signs and symptoms

Visual field defects and decreased vision occur in 9–32% and 4–16% of patients with pituitary adenoma, respectively [8,14,15]. The actual incidence of these symptoms, however, is likely to be higher because visual field defect and subtle vision loss are not always identified [1]. The extent of visual field and visual acuity loss at the time of diagnosis varies from minimal deficits in temporal hemifields to catastrophic, binocular loss of all light perception. Patients may also complain of difficulty in reading, headaches and abnormal colour vision (red-green defect).

The presence of bitemporal hemianopsia can associate different manifestations, such as the 'hemifield slide' phenomenon that produces fluctuating diplopia with no oculomotor impairment owing to anomalous retinal correspondence [11,13]. It is responsible for a disturbance in depth perception. There is no longer a shared area of vision between the two eyes, the right eye perceiving only the left hemifield and *vice-versa*. This neutralizes the normal fusional vergence reflex that

maintains ocular alignment, producing nasal visual hemifields that variously overlap (in those with esodeviations), separate (in those with exodeviation) or shift vertically (in those with hyperdeviations).

Initial assessment

Visual acuity may be normal even if visual field defects are present, but is more likely to be lower if the visual field loss is significant. Therefore, patients cannot be monitored only by assessment of visual acuity.

When the visual field defect in the two eyes is similar, the pupillary reactions will be equal, whereas relative afferent pupillary defect may be found in asymmetric involvement.

Fundus examination can show normal appearance or atrophy of the optic discs (Fig. 2). Atrophy can be diffuse or, more typically, in a horizontal band across the disc with relative sparing of the superior and inferior portions. Pallor of the optic disc is usually a sign of an old and chronic compression of the anterior visual pathway. Papilledema is rare and is generally associated with suprachiasmal tumours that compress the third ventricle and obstruct the flow of cerebrospinal fluid.

Fundus examination allows identification of ophthalmic conditions that may mimic bitemporal visual field defect, notably optic nerve coloboma, severe myopia with tilted disc syndrome or nasal retinitis pigmentosa. These pathologies, however, rarely respect the vertical midline.

Visual field assessment

As the opposite sides of the visual field are affected in each eye, the visual field remains full binocularly for a long period of time. Patients may not notice impairment of their visual field and tumours may progress for months or years before being detected.

The automatic visual field test is classically used. The test assesses only the central 24–30° but it can detect most visual field defects (VFD) induced by pituitary adenomas. The Goldmann visual field, assessing the periphery up to 90° laterally, is mainly used in cases of severely impaired acuity or with non-compliant patients.

Various patterns of VFD have been described in patients with pituitary adenomas, with the precise type of defect depending on the anatomy of the optic chiasm and its relation to the tumour (Fig. 3):

Central chiasmal syndrome, causing bitemporal hemianopia is the most frequent (Fig. 4). The defect may be complete, involving the whole hemi-field or parts of it. In pituitary adenoma, as the compression is inferior, it usually begins superiorly and progresses inferiorly, depending on the degree of compression. The hemianopsia can be congruous, if the damage is symmetrical, or incongruous.

Anterior chiasmal syndrome is more common in post-fixed chiasms. The compression in the anterior angle of the optic chiasm affects the Wilbrand's knee fibres and produces temporal and superior visual field defects. In cases of non-centred tumours, the anterior junction syndrome of Traquair (junctional scotoma) can be observed, involving the optic nerve immediately next to the chiasma. It

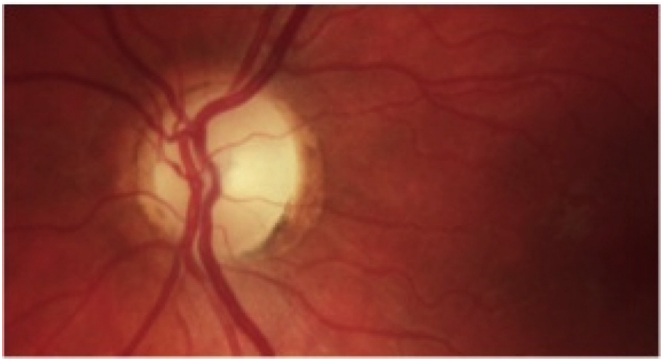

Fig. 2. Optic disc of a patient with a pituitary adenoma, showing a nasal and temporal pallor, and a relative sparing of the superior and the inferior areas of the disc.

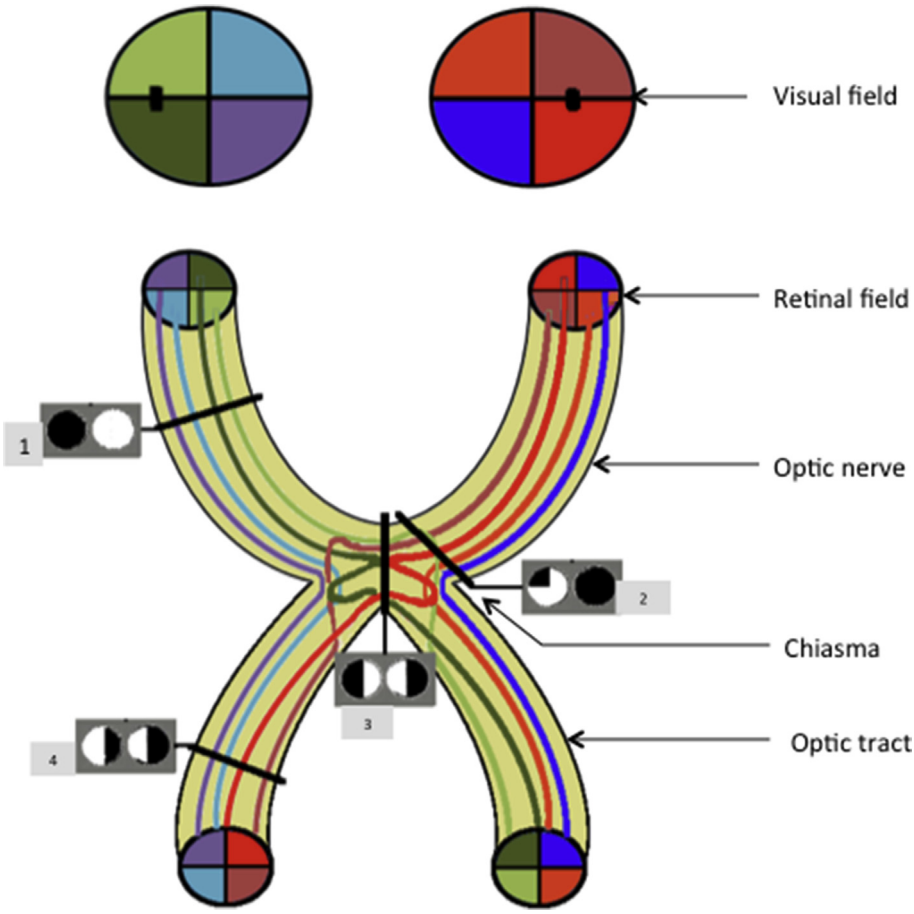

Fig. 3. Schema of the course of the ganglion cell axons in the anterior optic pathway and the corresponding visual field defects: -1: Compressive optic neuropathy. -2: Anterior junctional syndrome of Traquair with damage to anterior Wilbrand's knee. -3: Central chiasma syndrome, causing bitemporal hemianopsia. -4: Lesion of optic tracts, inducing a homonymous hemianopsia.

presents as a combination of severe central visual loss in one eye and subtle defects in the superior temporal visual field respecting the vertical midline in the contralateral eye [12].

Posterior lesions may involve the optic tracts producing a homonymous hemianopsia.

Optical coherence tomography (OCT)

In ophthalmology clinics, OCT is a new device used daily to study the retina and optic disc. It is a non-invasive ocular imaging technique, without contact, based on infrared light, enabling in-vivo assessment of eye tissue structure. Nowadays, OCT is used to establish and quantify the axonal loss in several neurological disorders.

In the case of anterior visual pathway compression, two structures are of interest to analyse (Fig. 5): papillary OCT can measure peripapillary retinal nerve fiber layer (RNFL) thickness and estimate the number of ganglion cell axons constituting the optic nerve. Its analysis provides a mean RNFL value and values per quadrant (temporal, nasal, superior and inferior); and macular OCT allows the measurement of macular ganglion cell complex (GCC). The GCC includes the retinal ganglion cell layers (composed of the nuclei of the ganglion cells), and the inner plexiform layer (formed by the dendrites of ganglion cells).

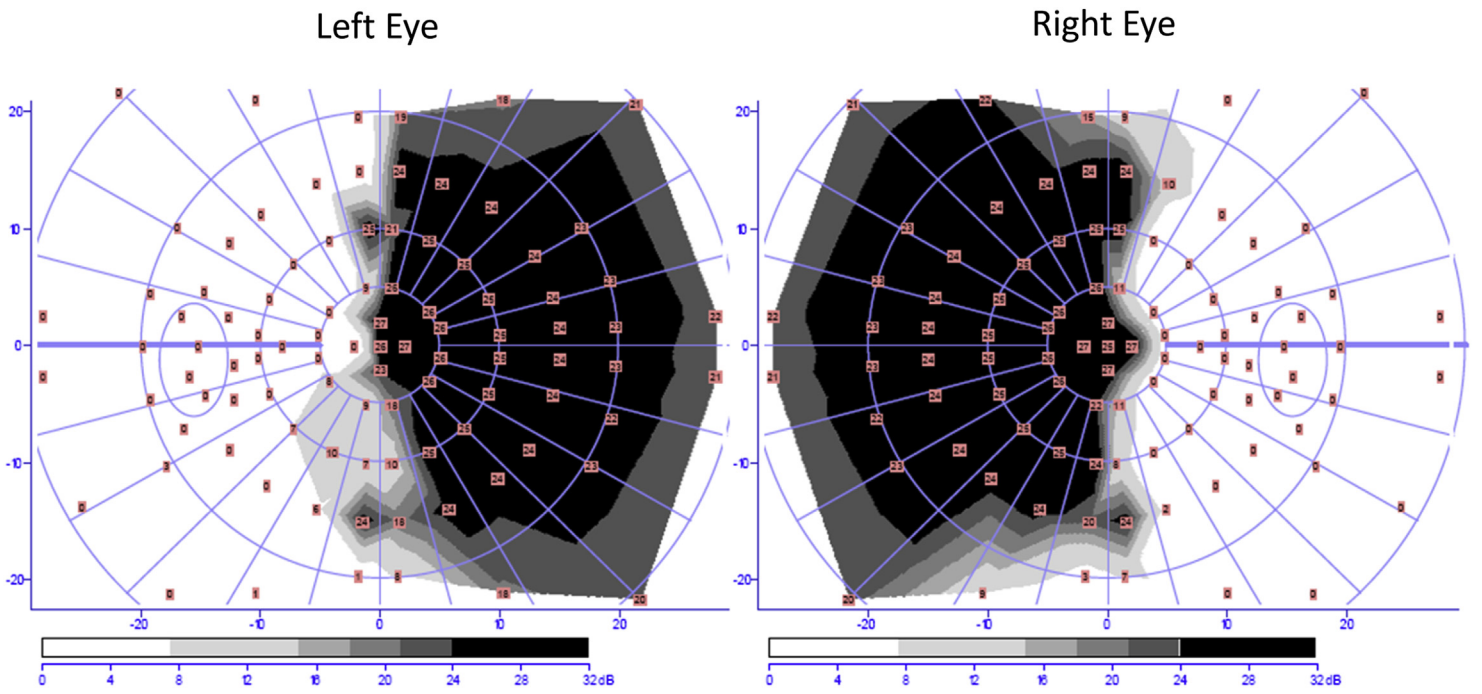

Fig. 4. Typical bitemporal hemianopsia revealing pituitary macroadenoma. The vertical meridian is well respected.

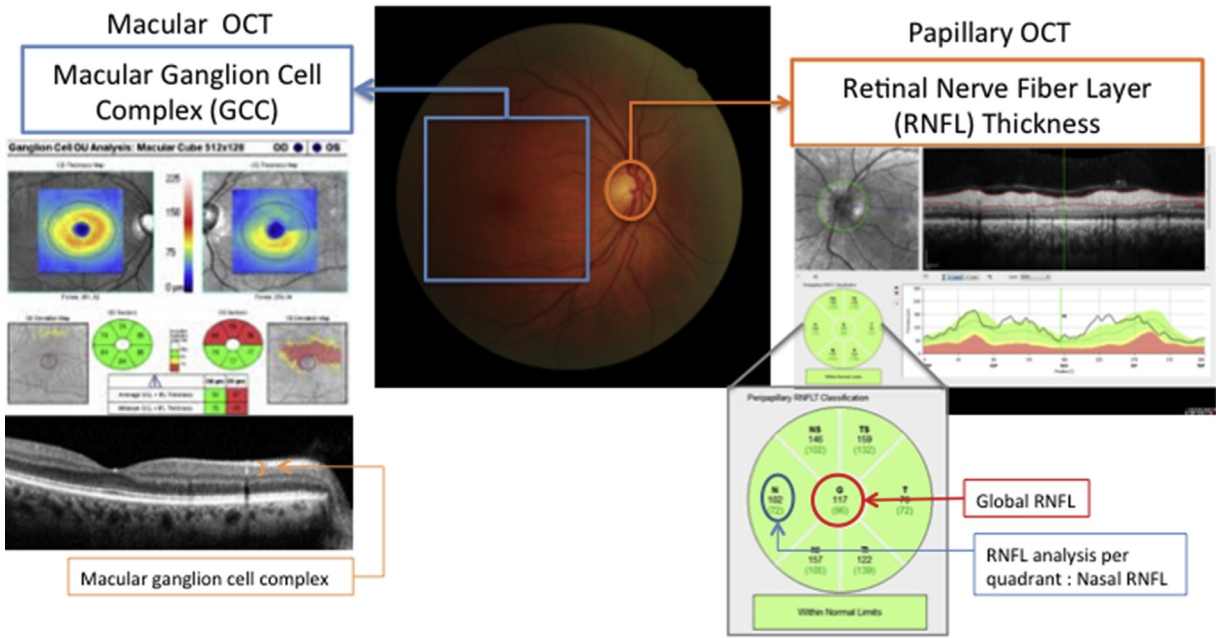

Fig. 5. Schema of the structures analysed by the OCT in neuro-ophthalmological pathologies: the RNFL (Retinal Nerve Fiber Layer) and the GCC (Ganglion Cell Complex).

- RNFL thickness

Pituitary adenoma can induce RNFL thinning, which reflects axonal degeneration caused by compression of the anterior visual pathways. RNFL loss is a reproducible and useful marker for diagnosis and follow-up of optic nerve axonal damage in several neurological diseases [16–19], including pituitary adenomas [20].

Compression of the optic chiasm is associated with thinner nasal and temporal RNFL sectors whereas, in glaucoma, superior and inferior sectors are the most affected [21]. Its measurement is objective and quantifiable, whereas determination of optic nerve pallor on funduscopic examination is subjective and non-quantifiable. In early compressive chiasmopathy, however, RNFL analysis is less sensitive than visual field assessment as there is, as yet, no axonal degeneration. These patients may have visual field defects but show a normal RNFL measurement.

RNFL thickness is also a useful tool to evaluate tumour severity and prognosis. The degree of reduction in RNFL thickness has been shown to correlate with visual field defects [22]. It can predict the recovery of visual function after decompressive surgery in patients with chiasmal visual field defects caused by compression [23–26]. Patients with normal RNFL thickness show an increased propensity for visual recovery. This effect has been described to continue after long-term follow-up [27].

- GCC-thickness

Newer OCT technology, with macular segmentation, measures macular ganglion cell layer complex (GCC) thickness, which correlates with visual field defects. Compression of the optic chiasm is associated with preferential ganglion cell loss in the nasal hemiretina, respecting the vertical meridian [19] (Fig. 6). This pattern of nasal defects in macular OCT is more specific than RNFL measurements [28]. It helps to distinguish chiasmal compression from other disorders [29]. It is also particularly useful in patients with unreliable automated perimetry [30]. GCC thinning seems to appear early, even when visual fields and RNFL thickness are normal, suggesting that the GCC analysis may be more sensitive for detecting compressive damage. When chiasmal compression is extended and severe, temporal hemiretinal thinning will also be found. GCC-thickness is also a predictive factor after surgical decompression [30].

Differential diagnoses

It can be difficult to diagnose patients who have suffered a pre-existing ophthalmological impairment that was responsible for the loss of global vision (e.g. cataracts, corneal and retinal pathologies) or who have had optic nerve pathologies. Glaucoma is a common optical neuropathy that can mimic pituitary adenoma, but the ophthalmologist can find atypical elements that lead to suspicion of a neurological etiology for example: in glaucoma, damage to the disc is responsible for a vertical excavation, optic disc haemorrhage can be found and there is no pallor of the neuro-retinal ring, the visual field defects do not respect the vertical meridian and OCT shows a thinner superior and inferior RNFL sector. Therefore, the presence of atypical elements must encourage the physician to perform a brain MRI.

Other tumoural conditions in the region such as craniopharyngioma, meningioma, suprasellar glioma, metastasis, Rathke's cleft cyst, arachnoid cyst, granulomatous diseases and chordoma may be responsible for ophthalmologic symptoms similar to those caused by pituitary adenomas.

Prognostic factors

Numerous prognostic factors for visual recovery after surgery have been explored and are shown in Table 1, although the results are variable depending on the study. The most reliable prognostic factors seem to be RNFL and ganglion cell complex thickness on OCT, as this can measure the degree of axonal loss and thus the possibility of visual function recovery after treatment.

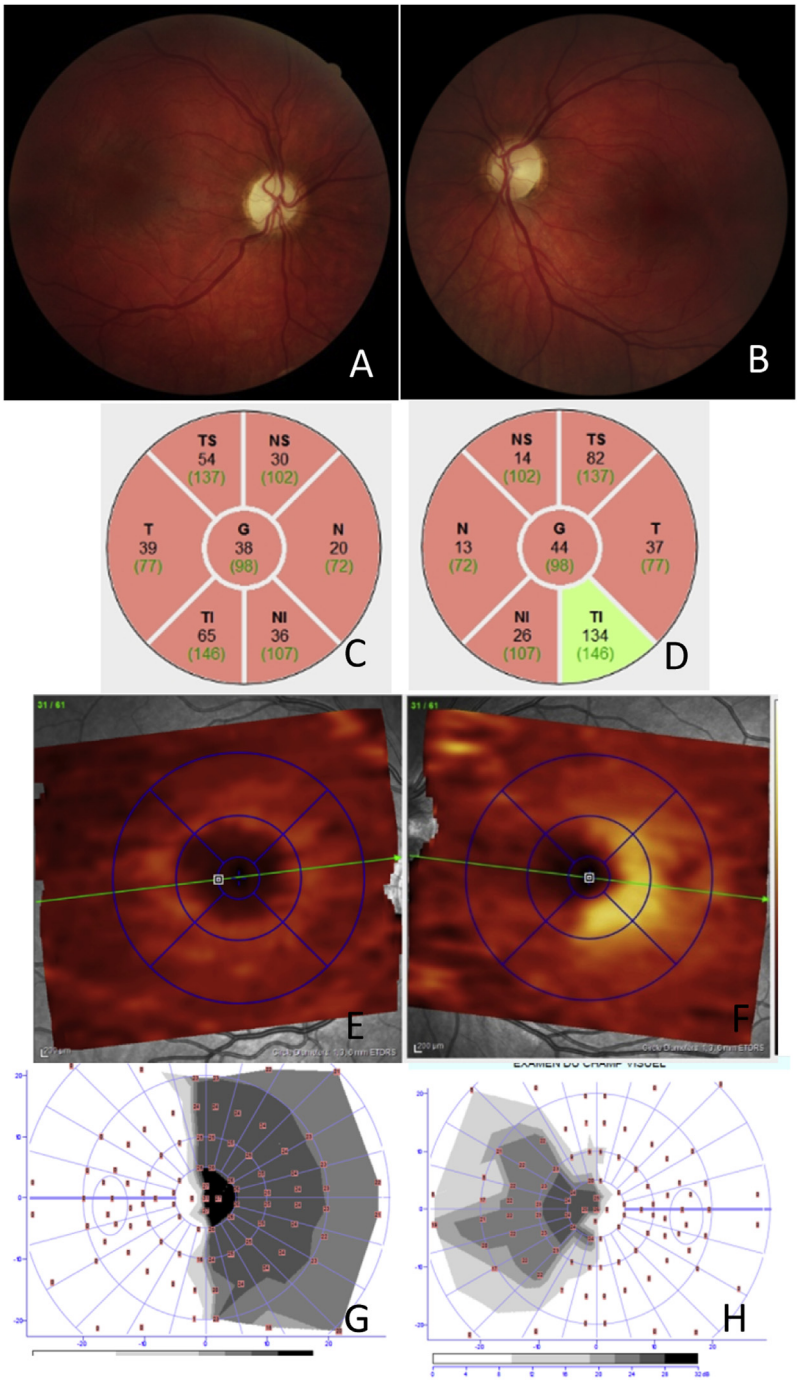

Fig. 6. Example of patient presenting chiasmal compression secondary to macroadenoma. Funduscopy of both eyes demonstrates the optic disc atrophy of the right eye (OD) (A) and the left eye (OS) (B). Papillary-OCT shows a thinning of the RNFL affecting both OD (C) and OS (D). Macular OCT shows a ganglion cell loss, severe and global in the OD (E), and in the nasal hemiretina, respecting the vertical meridian in the OS (F). Visual field shows a bitemporal hemianopsia; OS(G) – OD(H).

Table 1
Clinical signs and symptoms to predict post-operative visual function after transsphenoidal pituitary surgery.

Good predictive factor	References
Younger age	[4,31]
Short duration of symptoms	[4,31]
Less pre-op visual field loss	[4,31,32]
Less pre-op visual acuity loss	[4,33]
No pallor of optic disc	[13,32]
Tumour size < 3 cm	[34,35]
Normal ERG	[36,37]
Less RNFL thinning	[4,24,27,34]
Normal ganglion cell complex parameters	[29,30,37,38]

Follow-up

- Before treatment

If the tumour abuts the anterior visual pathways without giving any visual complication (visual acuity, visual field, ocular motor examination), ophthalmologic evaluation can be scheduled twice a year. The risk for visual function is low, except in the case of pituitary apoplexy.

Treatment is often considered if visual function is impaired.

- After surgery

Visual acuity recovers rapidly after surgical decompression, particularly during the day. Gradual improvement continues over the next few months. The visual field improves in a triphasic manner (Table 2). It can take months to years to recover [27]. No international recommendation has been made for the follow-up but a visit 3 months after surgery and then every 6 months until visual function stabilizes seems to be necessary [39]. It is important to maintain annual monitoring thereafter.

Ocular motor impairment

A large pituitary adenoma may be associated with three types of ocular motor impairment: ocular palsy, which is rare compared with visual field defect (<5% of cases) except in the context of pituitary apoplexy [see below]), abnormal eye movement (notably see-saw nystagmus), and dorsal midbrain syndrome (in very large tumours compressing the upper brainstem). Ocular motor impairment strongly suggests pituitary apoplexy, being exceptionally found at diagnosis of macroadenoma [41].

Anatomical bases of ocular motor impairment

The cavernous sinus limits laterally the sella turcica. A growing adenoma may expand laterally and eventually compress it. The cavernous sinus is a large confluence of veins, and receives blood via the ophthalmic vein through the superior orbital fissure and from superficial cortical veins. It is connected to the basilar plexus of veins posteriorly. It drains by the superior and inferior petrosal sinuses,

Table 2
Triphasic recovery of the visual field following transphenoidal pituitary tumour surgery according to Kerrison et al. [40].

Phase	Period	Physiopathology
Initial rapid recovery	Early phase, up to a week	Release of the conduction block caused by the compression
Delay - slower phase	Up to 4 months	Phase of remyelination
Late mild recovery	4 months to 3 years	Remyelination and possible neuronal plasticity

ultimately into the internal jugular vein. The internal carotid artery, and cranial nerves III (common ocular motor nerve), IV (trochlear nerve), V1 (the ophthalmic nerve), V2 (the inferolateral portion of the maxillary nerve) and VI (abducens nerve) and the sympathetic contingent innervating the eye, all pass through this blood-filled space (Fig. 7). These nerves, with the exception of V2, pass through the cavernous sinus to enter the orbital apex through the superior orbital fissure [42,43].

Nerve III innervates the elevator palpebrae superioris, the superior, inferior and medial rectus muscles, the inferior oblique and the iris sphincter muscle. Complete external nerve III palsy induces complete ptosis of the upper lid with paralysed elevation, lowering and adduction of the eye. With passive elevation of the upper lid, the examiner will find the eye resting in an abducted position of 20–30°. It is associated with internal ophthalmoplegia that induce paretic mydriasis, pupillary sphincter paralysis and paralysis of accommodation. Partial nerve III palsy is frequent, inducing partial or complete palsy in only some of the muscles innervated by the nerve. Nerve IV innervates the superior oblique that intorts, depresses and weakly abducts the eye. This palsy causes a loss of depression in adduction with vertical separation of doubled images that increases on downgaze and on gaze to the contralateral side. Nerve VI innervates the lateral rectus and palsy induces abduction deficit.

Dorsal midbrain syndrome is due to compression of the superior and posterior midbrain, which may also be associated with hydrocephalus caused by compression of the 3rd ventricle.

See-saw nystagmus is caused by compression of the diencephalic–mesencephalic region at the interstitial nucleus of Cajal or rostral interstitial nucleus of the medial longitudinal fasciculus [44].

Pathophysiology of ocular motor impairment

Ocular motor nerve involvement can result from a direct compression of the nerve by the tumour, or indirect compression through the wall of the cavernous sinus [45]. This latter is observed in the case of rapid tumour growth but mainly in apoplexy, following intra-tumoural haemorrhage/necrosis or ischaemic infarction.

The location is usually in the cavernous sinus; however, some investigators have hypothesized nerve compression in the oculomotor trigone, which is a dural weak-spot, limited laterally by the anterior petroclinoid ligament, medially by a line above the interclinoid ligament and posteriorly by the posterior petroclinoid ligament [43]. This is assumed to be a dural weak-spot. Ocular motor nerve compression may also be localized in the brainstem or subarachnoid spaces [46]. Other hypotheses include ischaemia and vascular occlusion of the perforating arteries and infiltration of the nerve by the tumour [46]. The common ocular motor nerve (III) is the most medial ocular motor nerve and is the most frequently involved [47]. Fourth and VIth nerves are less frequently involved and affected mostly in cases of massive compression of the cavernous sinus, involving all the ocular motor nerves [48].

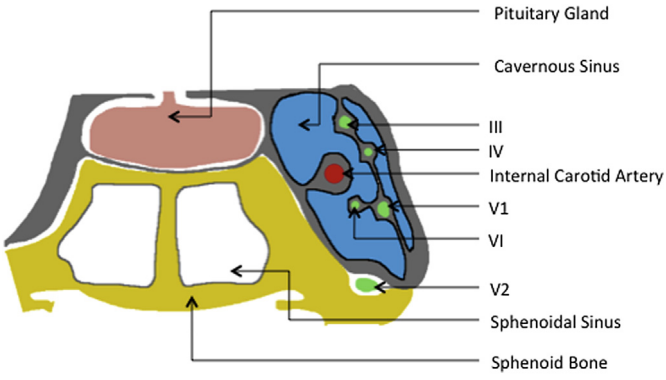

Fig. 7. Cavernous sinus in the coronal plane.

Initial ocular motor assessment

Ocular motor restriction

The primary symptom of paretic strabismus is binocular diplopia. Diplopia can be horizontal or vertical. It is perceived when the gaze is focused in the field of action of the paralysed muscle.

The oculomotor examination begins after examining visual acuity and visual fields. Physical signs may note a head tilt or turn. The important parameters for assessment here are version (conjoint movement of both eyes), duction (movement eye by eye), cover test measuring the deviation with prisms, eyelid and pupil examination. The Hess–Weiss coordimetric graph measures ocular motor defects reproducibly and allow the follow-up of the paresis over time.

Nystagmus

Nystagmus is responsible of oscillopsia. The rare phenomenon of 'see-saw' nystagmus may occur in cases of diencephalic tumour. See-saw nystagmus comprises a cycle of elevation and intorsion of one eye with synchronous depression and extorsion of the other, followed by an inverse cycle [49].

Other manifestations

Large tumours with posterior expansion toward the third ventricle, which block cerebrospinal fluid circulation, may induce hydrocephalus. Papilledema is secondary to hydrocephalus more than to tumour volume.

In exceptional circumstances, dorsal midbrain syndrome may manifest. This is associated with one or more of the following: Parinaud's syndrome (gaze elevation defect); nystagmus retractorius; abnormal eyelid position (ptosis or retraction of the upper eyelids); abnormal pupil size, loss of the pupillary light reaction and pupillary light/near dissociation; convergence disorder; and skew deviation.

Treatment

In the case of binocular diplopia, the objective is to relieve diplopia in primary position and in lowered gaze, but it is usually not possible to restore single vision in all directions of the gaze. In the case of a wide-angle strabismus, occluding one eye is the only means of relieving the patient's diplopia. If the discrepancy is moderate, prisms can be adapted. If it persists long after treatment, ocular motor muscle treatment can be proposed.

Pituitary apoplexy

Pituitary apoplexy is a potentially life-threatening disease associated with pituitary tumours, in which neuro-ophthalmologic signs are in the forefront of the clinical presentation. The incidence of this phenomenon has been described as being as high as 10% in some series [50]. Pituitary apoplexy can reveal the existence of the pituitary tumour or may occur during its follow-up [51]. The pituitary tumour generally presents as a sudden onset expanding mass with an intralesional haemorrhage, followed rapidly by ischaemic necrosis. This increases intrasellar pressure and is responsible for a stretching of the diaphragm, causing headache, and for compression of neighboring structure; specifically the optic chiasm and cavernous sinuses. The most common initial symptom is a sudden headache, associated with a rapid impairment of visual acuity and visual field defect, double vision (caused by compression of the cranial nerves surrounding the gland) and altered mental status. This is followed in many cases by acute metabolic symptoms caused by lack of secretion of essential hormones.

Clinical examination

Headaches are usually retro-orbital or frontal in location, are intense and may be accompanied by nausea and vomiting. This is attributed to stretching of the sellar diaphragm [52]. Visual impairment is

found in 75% of cases, secondary to a compression of the optic chiasm or optic nerve [53] and responsible for decreased visual acuity and bitemporal visual field defects. It is, however, noteworthy that optic atrophy is often already present in the eye fundus at diagnosis owing to prior compression of the visual pathway. Visual impairment may be already advanced even if the patient does not realise it at onset of apoplexy [54].

Ocular motor palsy occurs in almost 70% of cases [53], with most frequently involvement of the third nerve. The next most frequent nerve involved is VIth nerve palsy, followed by IVth nerve palsy, and then Vth nerve palsy [55]. Other less common symptoms are related to possible brainstem damage, such as light-near dissociation or convergence retraction nystagmus.

Treatment

The optimal management of acute pituitary apoplexy remains controversial. Given the rarity of pituitary apoplexy, no prospective studies have been feasible, and the retrospective studies had several biases (small series, lack of data on the initial degree of neuro-ophthalmologic deficit or differences in severity according to the group, and time to surgery variable between teams) [56].

Surgery is indicated in cases of severe neuro-ophthalmologic impairment affecting visual acuity, visual field, or both, disorders of consciousness, or where rapid degradation occurs. Early surgery obtains a better result than late surgery. In moderate neuro-ophthalmologic impairment, it is unclear whether conservative or surgical management provides the best outcome. Retrospective studies have confirmed that mild neuro-ophthalmic signs tend to improve spontaneously in most patients with pituitary apoplexy who are managed conservatively [57–59]. Patients should be closely monitored by a daily clinical and neurological examination, including cranial nerves and visual fields, as well as assessment of visual acuity. Surgical intervention must be considered if neuro-ophthalmic signs fail to improve or deteriorate.

Evolution

The outcome of acute apoplexy is variable and difficult to predict. Clinical status may deteriorate dramatically, secondary to a subarachnoid haemorrhage from the apoplectic adenoma, or cerebral ischaemia secondary to cerebral vasospasm, or the patient may recover spontaneously, with or without sequelae. The patient may subsequently suffer from visual defects, neurological disorders and pituitary insufficiency.

Visual acuity, visual field defects and ophthalmoplegia have been reported to improve in most patients after surgical decompression. Such improvement is observed in the immediate postoperative period and often continues for several weeks after surgery [40]. Visual recovery is less frequent in patients presenting with complete loss of vision, with a negative light perception. Significant improvement, however, has been observed in patients rendered blind by pituitary apoplexy treated with surgery [60]. Thus, positive prognostic factors for oculomotor recovery are early treatment, absence of afferent pupillary defect and low-intensity oculomotor signs [61].

Recurrent apoplexy and tumour regrowth have been documented both in surgically and conservatively managed groups of patients. Therefore, all patients require long-term follow-up imaging to detect recurrent growth.

Summary

Pituitary adenomas have a wide spectrum of clinical features. Therefore, management of pituitary adenomas is necessarily multidisciplinary. Neuro-ophthalmological manifestations of pituitary adenomas are frequent and variable. In some cases, they are the first symptoms. Physicians must be aware of these visual problems in order to refer such patients to an ophthalmologist. Ophthalmologic examination should include visual acuity, visual field test and ocular misalignment measures. OCT is a new technology that can allow an earlier diagnosis and allowow prognosis to be assessed. Treatment is

basically aetiological, but symptomatic treatment, notably in cases of diplopia, may be undertaken by the ophthalmologist.

Practice points

- Each ophthalmologic evaluation should include visual acuity, eye fundus, visual field test, OCT and study of ocular motility.
- OCT measurement (RNFL and GCC) is a useful tool to diagnose chiasmal compression and to evaluate tumour severity and prognosis.
- Ocular motor impairment is rarely found at diagnosis of macroadenoma and strongly suggests pituitary apoplexy.
- Pituitary apoplexy can be life-threatening for the patient and thus requires urgent treatment. It associates visual disorder, ocular motor disorder and headache with a sudden onset.

Research agenda

- Comparative trials of the efficacy of medical or surgical treatment on pituitary apoplexy are needed.

Conflict of interest statement

None declared.

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