Towards understanding ocular motility: III, IV and VI

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Abstract The study of the ocular-motor nerves must be exhaustive from their source (nuclei in the brainstem) down to the effector muscles (orbit). Visual disturbances have to be analysed by differentiating between a decrease in visual acuity and ocular-motor disorders. Imaging tests are dominated by MRI, including fine slices and gadolinium injection. A study of the Circle of Willis vessels is often useful, and essential in the case of type III impairment. A further CT scan is essential for analysis of the foramina, base of the skull and orbital walls. Impairment of CN VI requires a CT scan of the apex of petrous. The study of the cavernous sinuses must be in-depth (T2 and T1 after gadolinium and elimination of fats) and always comparative. Impairment of CN III is often complex, difficult to identify precisely (complete or partial, with or without a pupil impairment, associated with other neurological signs) and requires a reasoned study based on anatomical, semiological and pathological knowledge. Other than tumour diseases, it is necessary to consider less well known malformative, ischemic and inflammatory aetiology.

Introduction

Cranial nerve pathology requires precise anatomical knowledge as well as recognition of symptoms with a great deal of localisation value. The specific nature of examinations carried out by the imaging practitioner, acquainted with neuro-ophthalmological diseases, resides in his or her knowledge of the correct use of available imaging techniques.

Ocular motility constantly adapts the point of regard and in this, although very distinct from visual acuity, is essential to visual function.

Abbreviations: CBH, Claude Bernard-Horner; ICH, Intracranial hypertension; MLB, Median longitudinal bundle; INO, Internuclear ophthalmoplegia.

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Ocular motility, a complex combination, is based on voluntary movements and automatic or uncontrolled movements.

We will deal with each of the three ocular-motor nerves through the prism of anatomy, after which we will explain the details of investigational imaging protocols, after which we will tackle pathology.

**Clinical anatomy: general principles [1,2]**

Systematic ocular-motor control is under the command of several systems:

- an execution system, the final common route for all ocular movement: nerve and ocular-motor muscles;
- a pre-motor system that generates the movements located in the brain stem, different for each category of ocular movement;
- an ocular-motor command system sent to generators: this can be located in the stem or in the cortex according to the movement class;
- a control system ensuring good execution and precision of ocular movement or inhibiting the pre-motor system when fixing the gaze.

With regard to the imaging study, the most commonly investigated and encountered abnormalities are located in the brain stem and along the pathways of the ocular-motor nerves, possibly even within the orbital content.

Ocular movements are under the control of the efferent system. The efferent system consists of supranuclear and infranuclear pathways. This distinction is clinically important as supranuclear lesions almost always affect both eyes simultaneously while an infranuclear lesion affects the two eyes differently.

With a supranuclear lesion, bilateral impairment of the two eyes means that diplopia is most often not present. On the other hand, infranuclear impairment usually causes diplopia.

**The supranuclear pathways**

The supranuclear pathways include all pre-motor and motor regions of the frontal and parietal cortex, cerebellum, basal ganglia, superior colliculus, thalamus and brain stem centres (paramedian reticulated pontine formations, neuronal integrators) and vestibular nuclei.

**The infranuclear pathways**

The infranuclear pathways include the ocular-motor nuclei, the intramedullary segments of the emerging ocular-motor nerves, their peripheral segments (their pathway in the subarachnoidal space, cavernous sinus, superior orbital fissure and the orbit), the neuromuscular junction and extraocular muscles.

**The ocular-motor nerve: III**

The nucleus of III is located in the cerebral peduncle before the aqueduct of Sylvius in the paramedian position.

Within this nucleus there are closely linked motor fibres (for the ocular-motor muscles) and parasympathetic visceral motor fibres (for the ciliary muscle and the sphincter muscle of the iris) arising from the Edinger-Westphal nucleus. There are several sub-nuclei distributed from top to bottom from the posterior commissure down to the nucleus of IV.

The nerve emerges from the inner side of the cerebral peduncle and crosses the interpeduncular cistern (Fig. 1) on contact with numerous arteries, in particular the posterior communicating artery (major anatomical relationship in clinical application).

It passes through the upper part of the cavernous lodge (Fig. 2) and penetrates into the orbit through the superior orbital fissure before being divided into an upper branch which innervates the superior right muscles, right medial, superior eyelid elevator and into a lower branch innervating the right inferior and oblique inferior muscles.

The irido-constrictive fibres located at the upper part of the ocular-motor nerve are the most vulnerable to temporal engagement, which explains why mydriasis is the first sign [3].

If one or both of the branches are impaired, this is called extrinsic III.

Impairment of the parasympathetic component (intrin- sic III) is the source of pupil problems as it innervates the sphincter muscle of the iris.

**The trochlear nerve IV**

This is an exclusively somatic motor nerve.

The nucleus of CN IV is found within the grey matter in the caudal and dorsal part of the mesencephalon just below the aqueduct, to the side of the nucleus of III which is situated slightly higher in the mesencephalon.

The intra-axial portion of CN IV is very short with a path- way dorsally which then crosses the median line of the inferior colliculus, itself located below the pineal gland.

**Figure 1.** 3D CISS: III axial view.
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The abducens nerves VI

This is an exclusively somatic motor nerve, it is also destined for the right lateral muscle.

The nerve emerges from the caudal pontine area, exactly on its dorsal side just below the IVth ventricle (Fig. 4).

The fibres of the facial nerve go around its nucleus in a circumferential manner, directly close to the parapontine reticular formation and the medial longitudinal fasciculus.

The nucleus contains primary motorneurones for innervation of the ipsilateral lateral right muscle and interneurones intended for innervation of the ventral nucleus of contralateral CN III, crossing the median line to rejoin the ventral nucleus of CN III intended for the right medial muscle in the medial longitudinal fasciculus.

CN VI then takes a ventral direction that is somewhat lateral; in the course of its fascicular course, it passes close to the spinal pathway of the trigeminal nerve and crosses the corticonuclear pathway.

The motoneurone axons are directed frontwards and laterally, leave the brain stem around the ponto-bulbar junction (Fig. 5). On leaving the brainstem, CN VI is directed

Because of decussation, each nucleus innervates the contralateral superior oblique muscle [3].

CN IV is the only cranial nerve to emerge dorsally from the brain stem and it is the cranial nerve which has the longest pathway, unprotected, subarachnoidal (Fig. 3). It is also the thinnest which makes it more vulnerable to trauma.

CN IV circumnavigates the brainstem then passes just below the free edge of the tentorium cerebelli after which it penetrates into the lateral part of the cavernous sinus, below CN III, then reaches the lateral part of the cavernous sinus, below CN III, and enters the orbit through the superior orbital fissure, above and beyond the Annulus of Zinn.

It passes close to the optic nerve and reaches the superior oblique muscle around the area of the superior medial orbit.

Figure 2. T2 coronal: cavernous III.

Figure 3. 3D CISS: IV axial view.

Figure 4. 3D CISS: axial VI.

Figure 5. 3D CISS: VI sagittal view.
frontwards outside and above and crosses the upper edge of the tip of petrous, close to its tip.

Its close links to the tip explain frequency of CN VI paralysis in apex fractures [3] and in pathological processes affecting the base of the skull. It then penetrates the cavernous sinus where it follows a path along the lateral side of the internal carotid artery before joining the sphenoidal fissure.

**Ocular-motor muscles**

The final common pathway which determines the position of the eye in the orbit consists of the soft tissues which surround the ocular globe.

There are six ocular-motor muscles (Fig. 6): four right muscles (lateral right, medial right, superior right and inferior right) and two oblique muscles (superior oblique and inferior oblique). The right muscles and the upper eyelid elevator muscle are inserted into the ring of Zinn tissue condensation around the optic nerve at the orbital apex [4]. They then follow a course towards the front in the aponeuroses, which are linked together by means of intermuscular septae, to then pass through the capsule of Tenon and insert from the front into the sclera at various distances from the limbus.

The oblique muscles insert into the posterior and lateral section of the ocular globe. The anatomical source of the inferior oblique is located around the anteromedial peri-orbit, close to the posterior fossa.

The functional source of the superior oblique is the trochlea acting as a pulley located in a trochlear notch. The superior oblique follows a course towards the front within the superior-medial orbit in the direction of the trochlea, then the tendon changes direction at this level.

The ocular-motor muscles have very different dimensions: the inferior oblique is the thinnest and the medial right the thickest.

Consequently, as long as there is normal tonic innervation, the right medial muscle which is the strongest tends to reduce the spontaneous diversions of the ocular globes.

**Particular case of pupil interplay**

The size of the pupil is determined by the balance between sympathetic muscle tone of the iris dilator muscles and parasympathetic muscle tone for the iris sphincter muscle.

The sympathetic pathway originates in the posterolateral region of the hypothalamus while the sympathetic pathway with an ocular destination is divided into three segments (Fig. 7).

Fibres destined for the pupil continue to follow the internal carotid and penetrate the intercranial through the carotid canal.

The parasympathetic pathway originates at different points in the brain stem. The fibres which control the motility of the pupil sphincter originate in the Edinger-Westphal

![Figure 6](image1.png)  

![Figure 7](image2.png)  
nucleus beneath the nucleus of CN III in the mesencephalon (Fig. 8).

Paralysis of CN III leads to dysfunction of the extra-ocular somatic muscles (extrinsic impairment).

In a patient with complete paralysis of the oculomotor nerve, examination reveals total ptosis whereas the eye is in hypotropia position and in abduction leading to inability of adduction displacement upwards and downwards.

In addition the pupil is dilated and responds weakly to light.

This picture of complete CN III is rare, but requires emergency investigation of compression due to aneurysm.

Partial CN III paralysis is more common; it consists of varying degrees of limitation in ocular movement ± ptosis of varying severity, partial pupil impairment. These are clinical signs which correspond to investigation of various aetiologies by MRI in current practice.

Pure and isolated pupil impairment is only exceptionally a question of CN III impairment: it is more commonly a case of Addie’s tonic pupil disorder which does not require imaging, or pharmacological or mechanical problems (iris synchiae).

Thus when pupil impairment that occurs in isolation, the clinician must exclude an ocular motility problem by all means. This therefore modifies the procedure to be followed and requires neuro-imaging to be carried out [3].

**Practical and optimal imaging [3, 5]**

**Imaging protocols**

MRI is the standard examination allowing, through excellent contrast resolution and fine spatial resolution, identification of the oculomotor nerves throughout their course. There are at least three arguments in favour of carrying out an initial MRI:

- better examination for lesional topographic diagnosis;
- excellent resolution for cranial nerves, cavernous lodges;
- it is the only examination that allows an ischemic focus to be detected, even if smaller size.

MRI is far better than CT scans for detecting pathologies of the posterior fossa, in particular minor ischemic, inflammatory or neoplastic disorders (Appendix 1).

Nevertheless, ultra-high resolution CT scans are of benefit in studying the bone of the base of the cranium through the course of the oculomotor pathways in the case of discovery of an expanding lesion to MRI. Moreover, the choice of imaging method is motivated by the following clinical situations: cranial and/or orbital trauma, child who keeps moving (rapidity of acquisition), contraindication to MRI. An inconclusive MRI CT scan can be completed by an angiogram for investigation of minor ruptured aneurysm or to confirm the degree of stenosis of a dissection.

The case of a restrictive disorder needs to be distinguished, indicating a problem located in the orbit (most frequently muscular), from paralytic problem of a cranial nerve.

In fact this distinction (operated by the clinician with the aid of the forced duction test and orthoptic check-up) directs the study protocol.

In the case of orbital symptoms, many fine slices of orbital sequences need to be taken and especially a T2 sequence with fat elimination needs to be included, very sensitive to inflammatory phenomena.

**The ocular-motor pathology: clinical profiles and aetiologies**

When a patient reports binocular diplopia, anatomical localisation of the lesion is essential and the degree of urgency needs to be assessed.

Faced with ptosis and diplopia which vary in time, myasthenia needs to be excluded which never leads to sensory deficiency, pain, vegetative or pupil deficiency.

**Clinical profiles**

**Painful vertical or oblique diplopia**

This immediately suggests compression of III in the interpeduncular area as a result of aneurysm by rupture. The pain is due to subarachnoidal haemorrhage: severe headaches, often sudden and one-sided, nausea and vomiting.

Ocular-motor paralysis is complete, extrinsic and intrinsic reflected by mydriasis.

This major clinical symptom proves the compression of the irido-constrictor parasympathetic bundle by aneurysm or peri-aneurysmal haematoma (Figs. 9, 10).
The aneurysm develops at the expense of the posterior communicating artery or the posterior side of the carotid siphon, or even the posterior cerebral.

In the elderly subject, but also more rarely in the young subject [6], several cases have been observed of chronic diplopia relating to giant aneurysm of the cavernous location (Figs. 11–13).

Claude Bernard-Horner

The association of ptosis, miosis, enophthalmia (± pain) should point to carotid dissection. Haematoma of the carotid wall compresses the sympathetic innervation which is destined for the pupil. The imaging check-up includes a cervical, cerebral, parenchymatous and vascular study. It typically shows a hypersignal image of the carotid wall on sequence T1 FAT SAT and the appearance of irregular stenosis on the MRA TSA angiography sequence and/or angioscan.

In the absence of dissection over the whole of the carotid pathway (in contact with the sympathetic), a lesion of the pulmonary apex is investigated.

Specific case of bilateral CN VI, associated with other neurological symptoms

In an obese, cephalalgic patient whose eye fundus shows swollen disc, the possibility of idiopathic ICH has to be raised.
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This concerns uni- or bilateral paresis of CN VI, often associated with tinnitus.

Ocular-motor impairment is linked to stretching of CN VI in the CSF cisterns where pressure is elevated.

Imaging shows at least three of the following five signs: intrasellar arachnoidocele (Fig. 14), widening of the sub-arachnoidal spaces surrounding the optic nerves (Fig. 15), flattening of the sclera in front of the optic disc, narrowing of the cerebellar amygdala, stenosis of the transverse venous sinuses (Fig. 16).

Multiple paralysis of the ocular-motor nerves
Multiple impairment presupposes impairment of the cavernous sinus or of the superior orbital fissure.

The aetiologies suspected are neoplastic infiltration [7] (carcinomatous meningitis, lymphoma), inflammatory or infectious infiltration.

In the case of bilateral impairment, neuro-imaging is carried out to investigate a midline tumour which extends bilaterally, for example a cordoma, chondrosarcoma, nasopharyngeal carcinoma (Figs. 17–20), a meningeal lesion or viral polyradiculoneuritis or part of a sarcoidosis condition.

Investigation of impairment of CN V is systematic in clinical practice but also radiological. Aggressive or large lesions of the cavernous lodge leading to venous congestion extend along the orbital and ocular with elevation of intra-ocular pressure.

Tolosa-Hunt syndrome is a sterile idiopathic inflammatory impairment of the cavernous sinus. Considerable pain is

**Figure 13.** ARM MIP: mirror carotid aneurysm.

**Figure 14.** T2 coronal sequence: intrasellar arachnoidocele.

**Figure 15.** T2 coronal sequence: broadening of the subarachnoidal spaces surrounding the optic nerves.

**Figure 16.** Venous angiography with injection of MIP: tight stenosis of the transverse venous sinuses.
Figure 17. T1 coronal FAT SAT gd+: cavum mass, lateralised to the left, invading the left cavernous sinus by an ascending course.

Figure 18. T2 coronal mass of the cavum, lateralised to the left, invading the left cavernous sinus by ascending course.

Figure 19. T1 sagittal FAT SAT with injection: mass originating from the cavum invading the clivus explaining impairment of VI; note the retroclival meningeal enhancement.

Figure 20. Axial T1 FAT SAT gd+: syndrome of the left parapharyngeal mass, distinctly enhanced.
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often associated. The MRI shows a mass of variable volume, enhanced by gadolinium, inside the cavernous sinus, the disease is sensitive to corticosteroids. Nevertheless faced with meningeal infiltration, the diagnosis of Tolosa-Hunt is an exclusion diagnosis since one has to search hard for a specific inflammatory aetiology, lymphoma, tumour; even to the point of having a PET scan for investigation of primary foci and/or sites accessible to biopsy.

Ocular-motor impairment suggesting an ischemic process [8]
Discovery of extrinsic paralysis of CN III sparing the pupil makes it highly unlikely that an aneurysm is responsible.

The most probable and frequently encountered cause is ischemic impairment. The site of ischemic impairment of CN III is most commonly peripheral, especially in the cavernous sinus but also in the subarachnoid space, more rarely the brain stem. Pure extrinsic ocular-motor paralysis results from an ischemic phenomenon within the nutritive vessels (vasa nervosum) intended for the central path of the trunk of CN III which contains the extrinsic motor fibres.

Oclusion of the nutritive vessels causes ocular-motor paralysis of the extrinsic musculature but usually does not affect the pupil fibres located at the superficial part of the nerve trunk.

This ischemic microvascular impairment is usually not represented on the diffusion image. It does not constitute an imaging emergency but later neuro-imaging makes it possible to obtain a parenchymatous and vascular profile of this patient.

Nevertheless, the patient should be monitored as this type of ocular-motor paralysis of vascular origin should improve spontaneously or stabilise in under 3 months. If worsening occurs, the diagnosis must be reconsidered and other causes of ocular-motor impairment need to be investigated by neuro-imaging (inflammatory or compressive impairment in particular) and by paraclinical examinations.

Internuclear ophthalmoplegia
An internuclear lesion by definition affects the MLB.

Unilateral INO disorders include varying degrees of slowing down of the saccades of eye adduction associated with nystagmus of the other eye in adduction; this abnormality can be associated with a skew deviation.

Conventionally, the INO side is defined in relation to the deficient adduction side. Bilateral INO causes a bilateral delay in adduction and bilateral nystagmus.

The main lesions are represented by a demyelinising process (Figs. 21–24) [9] and an ischemic incident.

Skew deviation
This is a combination of strabismus and diplopia. It is an acquired defect of the vertical alignment of the ocular globes which results in supranuclear imbalance in input originating from the otolithic organs (the utricle and saccule). It can be caused by both central and peripheral lesions [10]. The most common lesions are central along the course of pathways originating in the otolithic organs in the posterior fossa (stem and brain). Skew deviation causes diplopia and...
Congenital pathology
A group of congenital pathologies initially attributed to restrictive limitations of “fibrosed” ocular muscles corresponds in reality to agenesis of the motorneurones of the brain stem.

Type 1 congenital fibrosis of the ocular-motor muscles is a dominant autosomal pathology (ptosis and bilateral external ophthalmoplegia) related to agenesis of the superior division of CN III. The diagnosis is clinical with small patients with characteristic head tilt but, as a result of the advances made in T2CISS sequencing methods, these abnormalities are being recognised more and more.

Other forms of agenesis are visible, in particular defects of the formation of the CN VI nucleus and the nerve itself, known under the name of Stilling-Duane syndrome [12].

The right lateral muscle is then innervated by a branch of abnormal origin because it originates from CN III. This aberrant innervation causes abnormal contractions of the right horizontal muscles. In addition, in some cases hypoplasia of CN IV is observed whose origin may be post constitutional, traumatic or compressive.

Restrictive impairment: orbital
Impairment of one or more ocular-motor muscles as a result of a limitation in the excursion of the ocular globe along with binocular diplopia. In practice, faced with an ocular-motor limitation, evidence supporting a solely muscular localisation and not a central or infranuclear one needs to be provided.

Impairment of a muscular origin is seen in a variety of clinical situations.

Additional clinical signs can be used to assist diagnosis: exophthalmia, enophthalmia, ptosis, conjunctival inflammation, medusa head dilation of the episcleral vessels, known tumour context, trauma, hereditary or congenital disorders.

In imaging, following the MRI protocol (Appendix 1) makes it possible to unmask each of these pathologies by showing the muscle or muscles affected and gives indications about volume and signal.

Dysthyroidorbitopathy
Dysthyroidorbitopathy is the most common cause (Figs. 25 and 26).

Diagnosis is often straightforward if there are accompanying orbital signs such as ptosis, chemosis, palpebral retraction, the thyroid context can be absent or unknown. MRI typically shows enlargement of the body of the ocular-motor muscles without this affecting the tendons. In the case of inflammatory impairment, a clear T2 SAT hypersignal is found sometimes with associated impairment.

Traumatic impairment
Secondly, traumatic impairment [13,14] with fracture of the orbital walls is the cause of muscle incarceration of varying degrees. Thus in the case of a trauma, the support tissue network for the ocular-motor muscles can be entrapped in the fracture site without there being real muscle entrapment.
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A particular case is that of the child which constitutes an absolute imaging emergency: considerable ocular-motor paralysis. The CT scan shows a "droplet" fracture of the orbital floor complicated by entrapment of the right inferior muscle drawn towards the maxillary sinus. Emergency surgical correction is indicated in order to limit the risk of tissue necrosis and ocular-motor sequelae.

The inflammatory or infectious impairments, tumour impairment

Next come the inflammatory or infectious impairments, tumour impairment of the facial bones or metastatic type Figs. 27–29, most commonly related to breast cancer.

The MRI CT scan for inflammatory myositis, whether idiopathic or non-idiopathic, differs in appearance from that of dysthyroidism as the impairment affects the body of the muscle and the tendon. Myositis can be idiopathic or specific, and part of a systemic disease (sarcoidosis, lupus, Wegener).

Figure 25. T2 coronal muscular impairment, predominant in the left muscle groups, within the context of Basedow’s disease.

Figure 26. T2 coronal FAT SAT: presence of inflammatory symptoms with a persistent hypersignal.

Figure 27. Axial T1 FAT SAT gd+: woman known to have breast cancer treated 20 years ago, vertical diplopia; mass developed at the expense of the right superior, left highly elevated by the contrast product.
Clinical profiles requiring emergency imaging must be recognised; MRI is the standard imaging technique, in contexts other than trauma.

When a lesion site is suspected, a pathological process must be investigated by imaging fine slices, often of a very small size.

**TAKE-HOME MESSAGES**

- A sudden and painful ocular-motor problem is an emergency (vertical or oblique diplopia): aneurysm undergoing fissuration.
- A chronic ocular-motor problem that becomes worse points towards investigation of a cavernous aetiology: meningioma, inflammation, tumour, etc.
- Presented with painful ptosis: CBH suggesting compression of the sympathetic cervical by haematoma of the carotid dissection.
- Presented with painful, post-traumatic paralysis of the elevation: absolute emergency in a child, muscle entrapment in a floor fracture.
- Painful horizontal diplopia: investigate impairment of the stem, 1 or 2 VI, right lateral muscles.
- Impairment of the 2 VI, associated with tinnitus and headaches, should lead to investigation of an abnormality of the encephalic veins: thrombophlebitis or idiopathic ICH.
- A sudden impairment suggests an ischemic incident, either of the stem or the small vessels of the nerve wall in the diabetic patient.
- A conjugated ocular-motor problem should lead to an investigation of a stem lesion (FLM) or of the posterior commissure.

**Clinical case**

M. G., aged 74, no particular medical history, presents with diplopia of the lateral gaze. Orthoptic analysis concludes impairment of the right VI. An MRI is requested.

**Questions**

1) What can we see on the course of the right VI (Fig. 30a–f)?
2) What complementary imaging examination should be requested?

**Answers**

1) Knowledge of an ocular-motor nervous impairment requires study of the core of VI, its emergence and its entire course to the orbit, including analysis of the right lateral muscle (with comparative analysis of its volume). Therefore for the brainstem, a diffusion and FLAIR study is necessary.

For the cisternal course, the CISS 3D sequence, highly weighted at T2, and the T1 FAT SAT sequence after injection of gadolinium in the axial plane in fine slices studying...
Dorello’s canal, the cavernous sinus and the orbital fissure.

For the orbits: T2 sequence as a coronal cut, allowing analysis of orbital content and analysis of the right lateral muscle (form, volume, signal). We discover here the absence of a lesion of the stem by projection of the right VI core but find a small extra-axial lesion appended to the posterior face of the clivus to the right, T1 isosignal, hyposignal for sequence CISS T2, and highly elevated after injection. The clival meningeal elevation is observed from sagittal views (Fig. 30f) (dural tail).

2) The structure of the clival bone must be analysed: a high resolution CT scan is requested. It shows thickening of the clival bone with small irregularities (linear calcifications) (Fig. 30g, h). It is also necessary to carry out a thoraco- abdomino-pelvic examination to investigate the presence of a deep lesion as meningeal thickening can correspond to a secondary carcinomatous localisation, and not always to simple meningioma. Moreover, this must be compared with an internal medicine examination (study of markers, mammography, etc).

In the present case, the aetiological check-up remained negative; meningioma is the diagnosis retained, especially as there is meningeal calcification and most importantly in the absence of bone lysis or an aggressive lesion.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.
Appendix 1. Imaging protocols as a function of clinical orientation

Restrictive orbital impairment (trauma, Basedow’s disease, myasthenia, suspected inflammatory or secondary myositie)
  Fine joint cuts centred on the orbitals and including the cavernous sinus
  T2 axial
  Coronal T1
  T2 coronal
  T2 coronal with fat elimination
III with pupil impairment (mydriasis of varying degrees)
  The anatomical proximity to the vessels (posterior communicating, posterior side of the carotid siphon) requires an exhaustive study of the Circle of Willis in addition to anatomical sequences
  Axial T1
  MRA TOF
  3D FLAIR
  T2 CISS
  Axial diffusion/T2*
  T2 coronal orbits to the cavernous sinus
  After injection of gadolinium: coronal T1 FAT SAT
  3D T1 with fat elimination
III with sympathetic impairment (myosis of variable degrees), more or less painful ptosis
  This impairment requires a double exploration:
  Investigation of an abnormality throughout the entire sympathetic course
  Encephalus
  3D FLAIR
  Axial diffusion
  T2 axial*
  T2 coronal on the visual and ocular-motor pathways
  3D T1 FAT SAT after injection
Cervical
  Axial T1 FAT SAT (for investigation of dissection)
  MRA TSA and circle after injection
  If investigating Claude Bernard Horner aetiologie: chest scan (apical mass)
IV
  Impairment of the trochlear nerve presupposes a study of the course of IV, the trunk and the effector muscle
  3D FLAIR
  Axial diffusion/T2 axial*
  T2 CISS volume
  T2 coronal centred on the orbits
  After injection: 3D T1 FAT SAT
  Coronal T1 FAT SAT fine orbital cuts
VI
  3D FLAIR
  Axial diffusion/ T2 axial*
  T2 centred on the lower topography
  Coronal and T2 axial centred on the orbits

After injection: 3D T1 FAT SAT
  Coronal T1 FAT SAT fine orbital cuts
INO: internuclear ophthalmoplegia
  Assumes impairment of the median longitudinal bundle: investigation of signs of ischemia, haemorrhage or multiple sclerosis
  3D FLAIR
  T2 axial fine cuts on the brain stem, T2 axial Dual (proton density)
  T2 axial*, axial diffusion
  after injection 3DT1 T1 spin echo
Nystagmus, cascades abnormalities:
  Investigation of a cranio-cervical abnormality: in the sagittal plane, multiple sequences T2, T1, FLAIR, fine cuts, diffusion, gadolinium; suspected myokemia MRA TOF and CISS (investigation of neurovascular compression), notably in the course of IV
Skew deviation:
  This is related to a supranuclear lesion producing problems with ocular vertical alignment
  This is not impairment of III or IV, but should be investigated by MRI, an abnormality of the posterior fossa, T2, FLAIR, diffusion, fine cuts

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