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Imaging of the optic chiasm and retrochiasmal visual pathways



N. Menjot de Champfleu^{a,*},
S. Menjot de Champfleu^a, D. Galanaud^b,
N. Leboucq^a, A. Bonafé^a

^a Service de Neuroradiologie, Hôpital Gui-de-Chauliac, CHRU de Montpellier, 80, avenue Augustin-Fliche, 34295 Montpellier cedex 5, France

^b Service de Neuroradiologie, Groupe Hospitalier Pitié-Salpêtrière, Paris, France

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Abstract The exploration of the chiasmal and retrochiasmal visual pathways is based on magnetic resonance imaging. A bitemporal hemianopsia suggests a lesion of the optic chiasm while homonymous lateral hemianopsia should lead to a search for a lesion of the retrochiasmal visual pathways. The causes of chiasmal impairment are mainly tumoral. The exploration protocol is based on MRI with T1-weighted sagittal sections, then T2- and T1-weighted coronal sections with and without injection. In case of a retrochiasmal syndrome, the MRI exploration protocol is a function of the type of occurrence of the deficiency and the context.

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Exploration of a disorder in the visual field

Methods to study the visual field

The visual field is studied separately for each eye. The examination may involve a simple clinical examination where the examiner presents either one of his fingers or a white ball, starting from the periphery and moving towards the centre in different sectors of the visual field. Campimetry is a dynamic monocular exploration of the visual field on a flat screen while perimetry is a dynamic monocular exploration of the visual field.

To analyse the results, it is necessary to take into account the fact that, for each eye, the nasal hemiretina receives light rays from the temporal visual hemifield and that the temporal hemiretina receives light rays from the nasal visual hemifield. Both left

* Corresponding author.

E-mail address: nicolasdechampfleur@orange.fr (N. Menjot de Champfleu).

visual hemifields, corresponding to the right temporal and left nasal hemiretinas, project themselves on the right fissure, while the right visual hemifields corresponding to the left temporal and right nasal hemiretinas project themselves on the left calcarine fissure.

Campimetry

The campimetre screen: a test light is moved along a flat screen and its position is noted as soon as it is perceived. This simple method analyses the central visual field in a dynamic manner.

Friedmann's analyzer (static perimetry) is a fast method to explore the central visual field: static white test lights of the same diameter are presented and their light intensity is modified.

Amsler grids: the subject looks at the small dot in the centre of a 10 cm grid and specifies whether he sees wavy lines, a deformation or if lines are missing from the grid. This method detects small central scotomas.

Perimetry

Perimetry uses a bowl-shaped screen, adapted to the curve of the eye. The projected tests are thereby at equal distance from the eye.

Kinetic perimetry

Kinetic perimetry is carried out with Goldman's perimeter (Goldman bowl). The visual field registers on a diagram where the centre is the point of fixation corresponding to the macula. The Mariotte blind spot corresponds to a hole in the visual field, due to the optic papilla (zone of absolute scotoma or negative temporal paracentral physiological scotoma).

Automated perimetry

More specific, automated perimetry helps visualize the intensity of impairment. Humphrey perimetry is most often used to study the central visual field. This perimetry is static, carried out by a computer.

What imaging protocol?

The exploration of the chiasm and retrochiasm visual pathways relies on magnetic resonance imaging. If a lesion of the optic chiasm is suspected, the millimetric acquisitions in T2 and T1 weighting with and without contrast infusion are carried out in the three planes. If the level of suspected impairment is that of the optic tracts or optic radiations, the exploration will cover the entire brain, in T2 and T1 weighting, without and then after contrast infusion, and include diffusion imaging. The examination will be completed, if necessary, by perfusion imaging, spectroscopic imaging, and in case of vascular lesion, by angiographic imaging.

Anatomical-physiological review

The optic chiasm

It receives the optic nerves by its anterior angles and emits the optic tracts by its posterior angles. The nerve fibres

from the two nasal hemiretina cross over there. In fact, the nerve fibres arising in each of the temporal hemiretina reach the homolateral optic tract, while those coming from each of the two nasal hemiretina reach the contralateral optic tract, the macular bundle comprising both direct and crossed fibres. It is located in the chiasmatic cistern, behind the tubercle of sella turcica, behind the chiasmatic groove (located at the posterior part of the sphenoid planum), above the sella turcica. The normal dimensions of the chiasm are 8 mm (4–13 mm) in anterior-posterior diameter and 3–5 mm thick.

The tractus optici or optic tract

Each optic tract is formed by the temporal bundle coming from the homolateral retina, and by the nasal bundle coming from the contralateral retina as well as the macular fibres originating in both retinas.

The tractus optici or optic tract starts at the posterior-lateral angle of the chiasm. It runs laterally and behind the anterior perforated substance and the tuber cinereum, and thereby forms the anterior-lateral limit of the interpeduncular cistern. It runs around the upper part of the brain peduncle, to which it adheres (Fig. 1). In this portion of its path, it is indivisible from the uncus and parahippocampal gyrus. The optic tract is positioned directly above the posterior cerebral arteries and ends in the lateral geniculate nucleus, at the posterior-lateral side of the thalamus. Each optic tract sends two contingents of fibres, the first and most abundant to the lateral geniculate nucleus and another minority contingent of fibres to the superior colliculus. The optic tracts divide at the lateral geniculate nucleus into two pathways: a lateral pathway that enters the lateral geniculate nucleus and a medial pathway that enters the medial geniculate nucleus. The optic tracts thereby end in the lateral geniculate nucleus, where nerve fibres provide a relay, but before, the bundle of papillary fibres separates from it and reaches the pretectal region, thereby entering in the formation of the pathway of the papillary light reflex.

The lateral geniculate nucleus

The lateral geniculate nucleus is an ovoid formation associating grey matter and white matter, located at the posterior and lateral side of the pulvinar thalamus. The anterior pole mingles with the optic tracts. Optic radiations emerge from the lateral geniculate nucleus, which run towards the visual cortex.

Optic radiations or geniculocalcarine tract

Gratiolet radiations arise in the lateral geniculate nucleus. They leave the lateral geniculate nucleus by forming the optic peduncle. The optic radiations then divide into three contingents of fibres that occupy the outer part of the sagittal stratum, directly outside the atrium of the lateral ventricle. The optic radiations then run towards the striate cortex of the occipital lobe and divide into two groups of fibres:

- a ventral bundle goes around the temporal lobe of the lateral ventricle and reaches the lower lip of the calcarine fissure. This ventral bundle makes a loop within

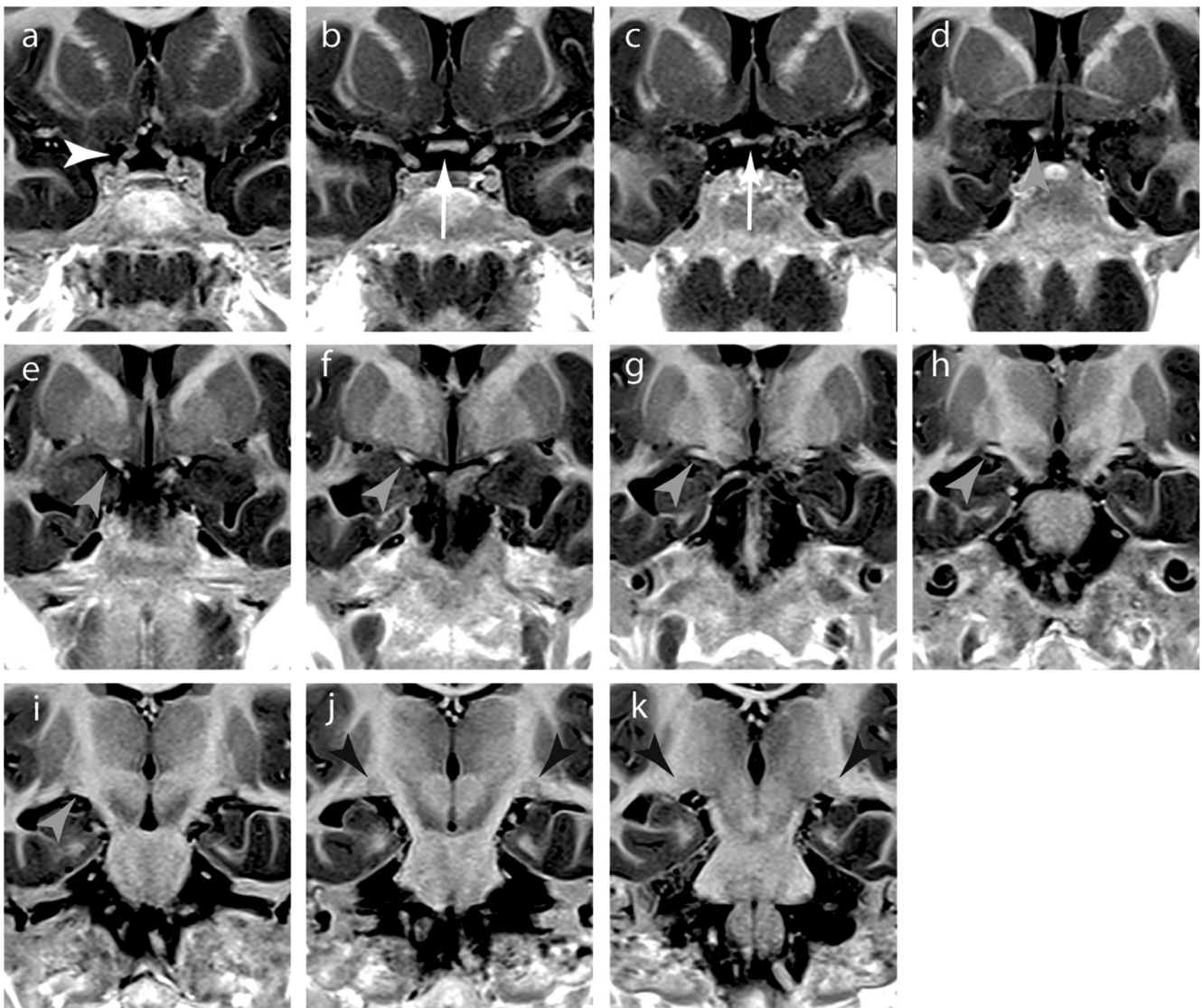


Figure 1. Anatomy of the retrochiasmal visual pathways. Series of inversion-recovery T1-weighted coronal acquisitions at 3 Tesla identifying the prechiasmal portion of the optic nerves (a, tip of white arrow), the optic chiasm (b and c, white arrows), the optic tracts (d to i, tip of grey arrows) and the lateral geniculate bodies (j and k, tip of black arrows).

the temporal lobe, moving forward and outside, around the temporal lobe of the lateral ventricle, then runs towards the rear to join the striate cortex. This anterior deviation of the lower optic radiations is more commonly called Meyer's loop. It is located at the level of the anterior end of the temporal lobe of the lateral ventricle, about 1 cm outside of it;

- a dorsal bundle goes around the occipital lobe of the lateral ventricle and ends in the upper lip of the calcarine fissure.

Cortical centre of the visual field: striate cortex

The cortical centre of the visual field (striate cortex or brodmann area 17) is located at the level of the upper

and lower lips of the calcarine fissure, at the medial side of the occipital lobe. Its anterior limit is the parieto-occipital fissure, the posterior limit is the occipital pole or, if present, the lunate sulcus. The upper lip of the calcarine fissure belongs to the cuneus, the lower lip to the lingual gyrus.

Classification of disorders of the visual field by chiasmal and retrochiasmal impairment: anatomofunctional correlation

The semiology of disorders of the visual field according to the site of the lesion is summed up in [Table 1](#).

Table 1 Summary table of visual disorders according to the site of the lesion.

Site of the impairment	Visual disorder	Associated signs
Optic chiasm	Sellar or suprasellar lesion: heteronymous bitemporal hemianopsia, central bitemporal scotoma Supra-chiasmatal lesion: heteronymous binasal hemianopsia	
Optic tract	Homonymous lateral hemianopsia	Absence or pupil contraction to light with visual stimulation of the blind hemifield
Optic radiations (temporal contingent)	Contralateral homonymous superior quadranopia without respect of macular vision	Persistence of the pupil contraction to light with stimulation of the blind hemifield
Optic radiations (parietal contingent)	Contralateral homonymous inferior quadranopia, full homonymous lateral hemianopsia	Persistence of the pupil contraction to light with stimulation of the blind hemifield Abolition of contralateral optokinetic nystagmus
Visual cortex	Deficiency in areas of the visual field: homonymous central hemianopsal scotoma Unilateral lesion: homonymous lateral hemianopsia with respect of macular vision Bilateral lesions: double hemianopsia Lesions located in the area striate (lips of the calcarine fissure): homonymous superior or inferior quadranopia Bilateral and symmetrical lesions of the upper or lower lip of the calcarine fissure: inferior or superior altitudinal hemianopsia Bilateral impairment of the visual cortex: cortical blindness	

The scotomas

Scotomas are gaps in the visual field. The ones most important to know are central scotomas related to impairment of the macular bundles. The central scotoma occupies the macular visual field around the fixation point. It induces a major loss of visual acuity resulting in discomfort in everyday life (reading). The clinical aspect varies according to the seat of the lesion:

- a single or bilateral central scotoma attests to a lesion in the optic nerve;
- a bitemporal central scotoma attests to a chiasmal lesion;
- a junctional scotoma of Traquair attests to a compression of the anterior angle of the chiasm and associates a central temporal hemiscotoma on one side and an upper temporal peripheral notch on the other side;
- a homonymous central hemianoptic scotoma attests to a retrochiasmatal lesion. Hemianoptic scotomas are gaps in the visual field affecting each side of the macular vision or peripheral vision.

The hemianopsias

This consists of a loss in visual acuity in half of the visual field. When the limit of the impairment is horizontal, the hemianopsia is said to be altitudinal, superior or inferior, depending on whether the loss in visual acuity involves the upper or lower half of the visual field.

The limit of the loss of the visual field may be vertical. The hemianopsia is said to be homonymous when the loss in visual acuity involves both right visual hemifields or both left visual hemifields. It is said to be heteronymous when it involves both nasal hemifields, or both temporal visual hemifields. Macular vision is often not affected during hemianopsia due to its bilateral projection on the visual cortex.

The altitudinal hemianopsias

These hemianopsias are rare, often due to bilateral occipital lesions affecting the optic radiations or the cortex itself, in

particular during traumas. Inferior altitudinal hemianopsia is the most common of the two.

The heteronymous hemianopsias

Bitemporal heteronymous hemianopsia, relatively frequent and characteristic of the chiasmal syndrome, provokes a loss of visual acuity in both temporal hemifields by affecting the fibres leaving both nasal hemiretinas. It attests to a lesion of the optic fibres that criss-cross in the optic chiasm and usually results from an external compression, in particular during the evolution of a tumor in the sellar region. Initially, it may only affect one quadrant, usually the upper quadrant.

Binasal heteronymous hemianopsia is rare. It provokes a loss of visual acuity in both nasal hemifields by impairment of the fibres leaving both temporal hemiretinas. It follows direct impairment of the visual fibres to the optic nerve or chiasm. This type of hemianopsia is especially found with tumors of the third ventricle.

Therefore, heteronymous hemianopsias are due to chiasmal impairment. The chiasma syndrome is based on a triad of symptoms associating campimetric deficiency, a reduction in visual acuity and optic atrophy.

The homonymous lateral hemianopsias

Definition

The homonymous lateral hemianopsias are most common, attesting to a retrochiasmal lesion of the visual pathways. It may affect all of the right or left visual hemifields and are contralateral to the lesion, that is, a left retrochiasmal lesion will induce right homonymous lateral hemianopsia affecting the nasal field of the left eye and the temporal field of the right eye.

The impairment may only involve the upper or lower half of the visual hemifields, thereby creating homonymous lateral quadrant hemianopsia (or quadranopsia). The impairment is sometimes even more localized, occurring in the form of a hemianoptic scotoma.

Location of the lesions responsible for homonymous lateral hemianopsia

The lesions are found on the retrochiasmal visual pathways and study of the papillary light reflex may specify the location of the lesion.

Lesion of the optic tract. In unilateral lesions of the optic tract, the homonymous lateral hemianopsia is characterised by the lack of pupil contraction in response to visual stimulation in the blind hemifield (Wernicke's hemianopic pupil reaction), while the pupil contraction is normal when the stimulation is on the healthy visual hemifield.

Lesion of the optic radiations. When the lesion affects the optic radiations, in their parietal or temporal pathway, the resulting hemianopsia is characterised by the persistence of the pupil contraction in light during the stimulation of the blind visual hemifield.

Lesion of the temporal lobe. In the temporal syndrome, the deficiency in the visual field is due to the impairment of the fibres that reach the lower lip of the calcarine fissure, resulting in a contralateral superior homonymous quadranopsia. It does not respect the macular vision.

The semiology of impairment to the temporal lobe is complex and heterogeneous. Other sensory and gnostic disorders

may be involved (cortical deafness by bilateral impairment of the transverse temporal gyrus, auditive agnosia, auditory illusions and hallucinations, olfactory disorders and balance disorders), aphasia, or finally seizures.

When present, these seizures may be simple partial seizures. Complex visual hallucinations and illusions of an aesthetic nature are then possible.

Lesion of the parietal lobe. The parietal lobe syndrome involves a deficiency in the visual field related to impairment of the fibres that reach the upper lip of the calcarine fissure. It consists of contralateral inferior homonymous quadranopsia, even if the occurrence of complete homonymous lateral hemianopsia is not exceptional.

The blink reflex in response to a threat may be abolished with parietal lesions even without hemianopsia. A conjugate eye deviation is possible but, above all, the abolition of contralateral optokinetic nystagmus (rapid movement towards the opposite side of the lesion) should be searched for.

The other elements of the parietal lobe syndrome may be found: objective sensory disorders and tactile agnosia, body image disorders (hemiasomatognosia, anosognosia, anosodiaphoria in case of lesion of the minor hemisphere; autotopoagnosia, digital agnosia and right-left disorientation, in case of a lesion of the dominant hemisphere), spatial agnosia, praxis disorders, language disorders and trophic disorders (amyotrophy).

Cortical lesions of the occipital lobe. Impairment of the visual cortex (Brodmann area 17) may induce homonymous lateral hemianopsia with respect of the macular vision in case of a unilateral lesion, or a double hemianopsia in case of bilateral lesions. Respect of macular vision is certainly due to its bilateral projection on the visual cortex.

Homonymous quadrant hemianopsia, whether superior or inferior, is found with localized lesions of the striate area, only damaging one of the lips of the calcarine fissure. The loss of vision affects the upper or lower half of the contralateral visual hemifield depending on whether the lesion involves the lower or upper lip of the calcarine fissure. Bilateral and symmetrical lesions of the upper or lower lip of the calcarine fissure are manifested by inferior or superior altitudinal hemianopsia.

Central homonymous hemianopic scotomas have already been mentioned and correspond to deficiencies in areas of the visual field.

Finally, the double hemianopsis is possible and is manifested by the loss of peripheral vision in the entire visual field while the macular vision may be respected.

Bilateral impairment of the visual cortex is responsible for cortical blindness. It creates a bilateral destruction of the Brodmann area 17. Here, the loss of vision is total and affects both macular vision and peripheral vision. However, the pupil reflexes are maintained. The blink reflex in response to a threat, and optokinetic nystagmus are abolished, voluntary and reflex eye movements are maintained. The duration of such cortical blindness is variable (transitory or definitive) and may be accompanied by anosognosia with recognition disorders and visual hallucinations.

The other elements of the occipital syndrome should be searched for: dyschromatopsia and achromatopsia, visual illusions or hallucinations in case of peri- and parastriated lesion, seizures with visual manifestations, visual agnosia (trouble recognizing objects, people or graphic symbols),

spatial agnosia, oculomotor and visual motor disorders and finally psychic disorders (transitory or lasting fixation amnesia).

The causes of chiasmal impairment

Chiasmal lesions mainly result from sellar tumors (pituitary adenomas) as well as suprasellar tumors (craniopharyngioma, meningioma of the tubercle of sella turcica, chiasm glioma).

Tumoral causes

Infrachiasmal tumors

Pituitary adenoma

The macroadenomas (diameter greater than 10mm) and the invasive adenomas may be responsible for chiasm compression in case of extrapituitary extension towards the suprasellar regions. Most cases are non-secretory as secreting adenomas are most often discovered at the microadenoma stage.

Solid macroadenoma. The (non-necrotic) solid macroadenomas most often appear isointense to the cerebral cortex in T1, isointense or slightly hyperintense and heterogeneous in T2. Contrast infusion determines an intense and homogenous enhancement. The dura mater may be thickened and enhanced. GH adenomas often appear hypointense in T2.

The relationship between the upper pole of the adenoma on the one hand and the chiasm and the optic nerves on the other hand is best assessed on coronal sections in T2 and T1 without injection. The chiasmal compression may be associated with a hypersignal of the optic tracts. This hyperintense signal of the optic tracts in T2 attests to an edema induced by the blocked communication of the perivascular spaces of the nervous system with the subarachnoid space, due to the tumoral compression [1]. The macroadenomas may be enclosed, thereby presenting regular limits, or invasive, where their contours are irregular and they often present an extension to the cavernous sinus space. The intracavernous extension may be assessed using the Knosp method [2].

CT imaging, carried out in bone window, reveals an enlarged sella turcica, an erosion of the dorsum sellae, thinning and depression of the sellar floor. Giant adenomas, often invasive, develop towards the suprasellar regions, the cavernous sinus spaces, the sphenoid sinus and the basiphennoid.

Necrotic macroadenoma. The necrotic macroadenomas often present a hypointense central part in T1, highly hyperintense in T2, with peripheral enhancement. These macroadenomas with necrosis are hemorrhagic in 30% of the cases. In CT imaging, they appear hypo-, iso-, or hyperdense with possibility of liquid-liquid level.

In MRI, the hemorrhagic necrosis appears iso- or hyperintense in T1. A liquid-liquid level may be identified on the sagittal and transverse sections, where the anterior part appears hyperintense in T2, and the posterior part hypointense in T2. The enhancement is usually peripheral and annular.

The sudden hemorrhagic necrosis of the macroadenoma may be associated with a clinical picture of pituitary apoplexy. In this case, the T1 hyperintensity is generally absent and the T2 gradient echo sequence alone reveals intratumoral hyperintense areas. The hemorrhagic necrosis is identified in the acute phase by CT scan in the form of intratumoral areas of hyperdensities. This pituitary apoplexy is often associated with the thickening of the sphenoid sinus.

The differential diagnosis of macroadenoma with hemorrhagic necrosis should be carried out with:

- Rathke's cleft cyst: median topography, absence of contrast enhancement, absence of liquid-liquid level;
- a cystic craniopharyngioma;
- a giant thrombotic aneurysm.

Other intrasellar lesions responsible for chiasmal compression

Fleshy lesions enhanced by the injection of contrast product: hypophysitis. The group of hypophysitis includes three entities:

- lymphocytic adenohypophysitis (touches the anterior pituitary) corresponds to a lymphocytic, plasmocytic and eosinophilic infiltration of the pituitary and the pituitary stalk, with progressive appearance of a fibrosis. It is especially found in the post-partum woman. The height of the gland seems to be higher, of iso- or hypointense signal in T1 and hyperintense in T2, with intense enhancement extending to the thickened pituitary stalk;
- lymphocytic infundibulo-neurohypophysitis reaches the posterior pituitary, pituitary stalk and the hypothalamus. It is clinically manifested by diabetes insipidus;
- giant cell granulomatous hypophysitis.

Other fleshy and enhanced pituitary lesions may finally lead to a compression of the intracranial portion of the optic chiasm or the optic nerve: pituitary metastases, germinomas, choristomas, chordomas, giant arterial aneurysms with thrombosis.

Liquid lesions. Certain liquid pituitary lesions may cause compression of the chiasmal visual pathways: intrasellar subarachnoid cysts, Rathke's cleft cyst, intrasellar craniopharyngiomas and more rarely abscesses, epidermoid cysts or colloid cysts.

Suprachiasmal and prechiasmal tumors

Fifteen to 20% of all brain tumors are located in the region of the chiasm, including 50% pituitary adenomas, 25% craniopharyngiomas, 10% meningiomas, and 5% gliomas.

Meningiomas

They account for 20% of all intracranial tumors. When they are responsible for clinical signs, the meningiomas may cause visual signs, inducing unilateral central scotoma or monocular blindness, by compression of one of the anterior angles of the chiasm. Only meningiomas with posterior development induce a chiasmal syndrome. They are sometimes responsible for a Foster-Kennedy syndrome, associating unilateral optic atrophy and contralateral papillary edema, sometimes with anosmia on the side of the optic atrophy. It is above all provoked by meningioma of the small

wing of the sphenoid, olfactory meningiomas and frontal tumors.

Craniopharyngiomas

The craniopharyngiomas are embryonal tumors usually found in the child. These tumors present a first frequency peak in the child and a second peak in the adult after the age of 50. In most cases, they are located in the suprasellar region and more rarely within the third ventricle, or even intrasellar. Adamantine craniopharyngiomas are most common in the child, associating fleshy and cystic components, and are often calcified. In MRI, the cystic portion most often appears hyperintense in T2 and FLAIR, of variable signal in T1. The walls of the cyst may be enhanced after injection. After injection, the fleshy component presents intense enhancement. Papillary craniopharyngiomas are more common in the adult. They are solid or solid and cystic and most often without calcification (Fig. 2).

Intrachiasm tumor: optic chiasm glioma

The optic chiasm glioma arises from the proliferation of neurological tissue found in nerve fibres. It is found in the child and is sometimes associated with type I neurofibromatosis. The association of a chiasmatic syndrome, bilateral optical atrophy and signs of intracranial hypertension is indicative of the diagnosis (Fig. 3).

Vascular causes

Aneurysms of the inner carotid artery and the circle of Willis (supraclinoid aneurysms, of the anterior communicating artery or anterior cerebral artery) may cause chiasmatic compression.

Post-traumatic chiasmatic syndrome

It usually follows serious cranial traumas with fracture of the lower level of the base of the skull.

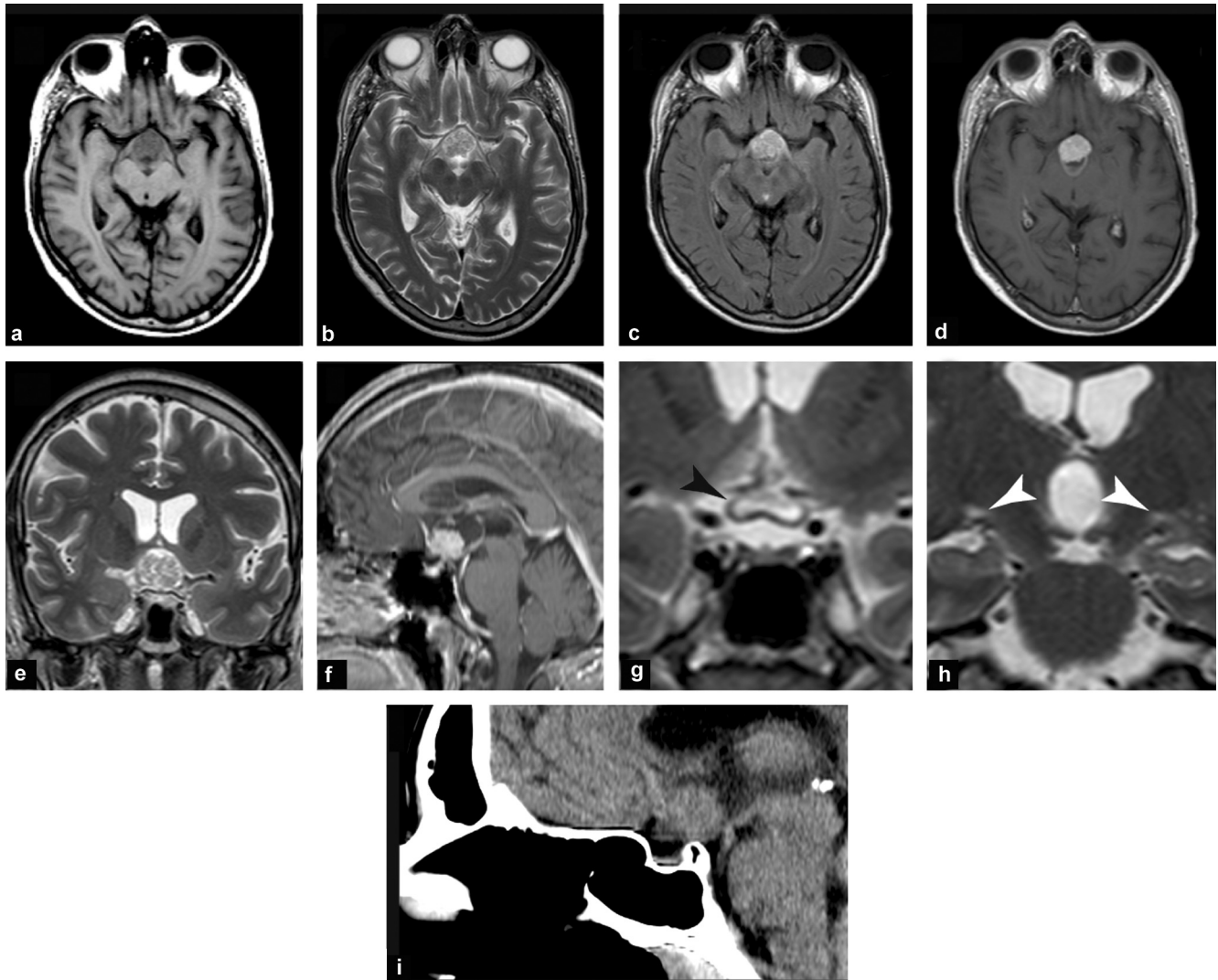


Figure 2. Craniopharyngioma in a 51-year-old patient. This nodular lesion is centred on the infundibulum of the third ventricle, in isosignal T1 (a), heterogeneous hypersignal T2 (b and e), hypersignal FLAIR (c), enhanced in a homogenous and massive way after the injection of gadolinium salts (d and f). It comes into contact with the posterior side of the optic chiasm that presents a T2 hypersignal (g, tip of black arrow). Narrowing of the retrochiasmatic visual pathways is noted in the form of a T2 hypersignal of the optic tracts (h, tip of white arrows). The CT-scan does not reveal any calcifications (i).

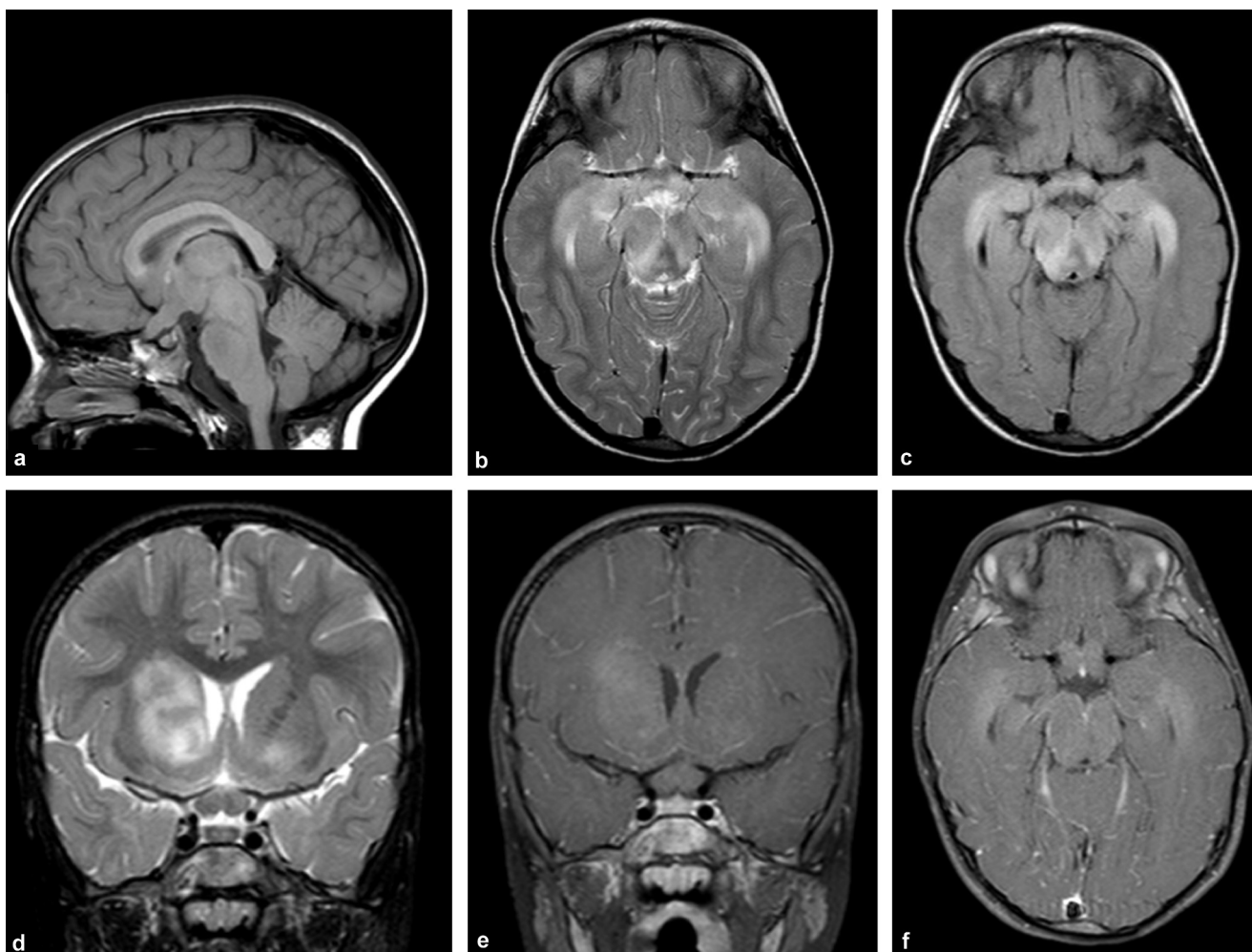


Figure 3. Mesencephalo-diencephalic glioma in a 5-year-old. Mesencephalic gliomatous infiltration in hypersignal T2, hyposignal T1, presenting several enhancements after injection, extending to the diencephalic region, the optic tracts, the optic chiasm and the prechiasmatic portion of both optic nerves.

The causes of retrochiasmatic impairment

The optic tract may be damaged by a tumor, a thrombosis of the anterior choroidal artery, or an aneurysm of the internal carotid artery.

Lesions of the optic radiations are actually included in those of the parietal and temporal lobes, strokes and tumors being most common. Transitory deficiencies may follow ischemia in the vertebrobasilar territory or a basilar migraine.

Tumoral

The symptoms most often seen with brain tumors are headache, neurological deficiency, seizures, or visual symptoms such as ocular motor disorders, colour vision disorders, papillary edema or visual field disorders. The tumors most often seen are of neuroepithelial origin (pilocytic astrocytomas, low-grade gliomas, glioblastomas), and of secondary origin (intracranial metastases).

Low-grade gliomas

Low-grade gliomas affect the subject between the age of 20 and 50. Beyond this age, high-grade gliomas are most often seen. They mainly involve the fronto-temporo-insular regions, and preferentially diffuse along the fibres of white matter [3]. This accounts for the impairment of the optic radiations. Low-grade gliomas are thereby responsible for homonymous lateral hemianopsias where the installation is slow, over several months (Fig. 4).

These lesions appear in hyperintensity on T2 and FLAIR sequences, in T1 hypointensity, not enhanced after injection, the increase in the cerebral blood volume (rCBVmax) if present, is moderate and the spectroscopy finds a moderate increase in choline, a drop in N-acetyl-aspartate (NAA), without a lipid or lactate peak. The borders of the lesions are poorly defined and the mass effect is moderate. The growth of the tumor is slow, with an average diameter of under 4mm per year [4].

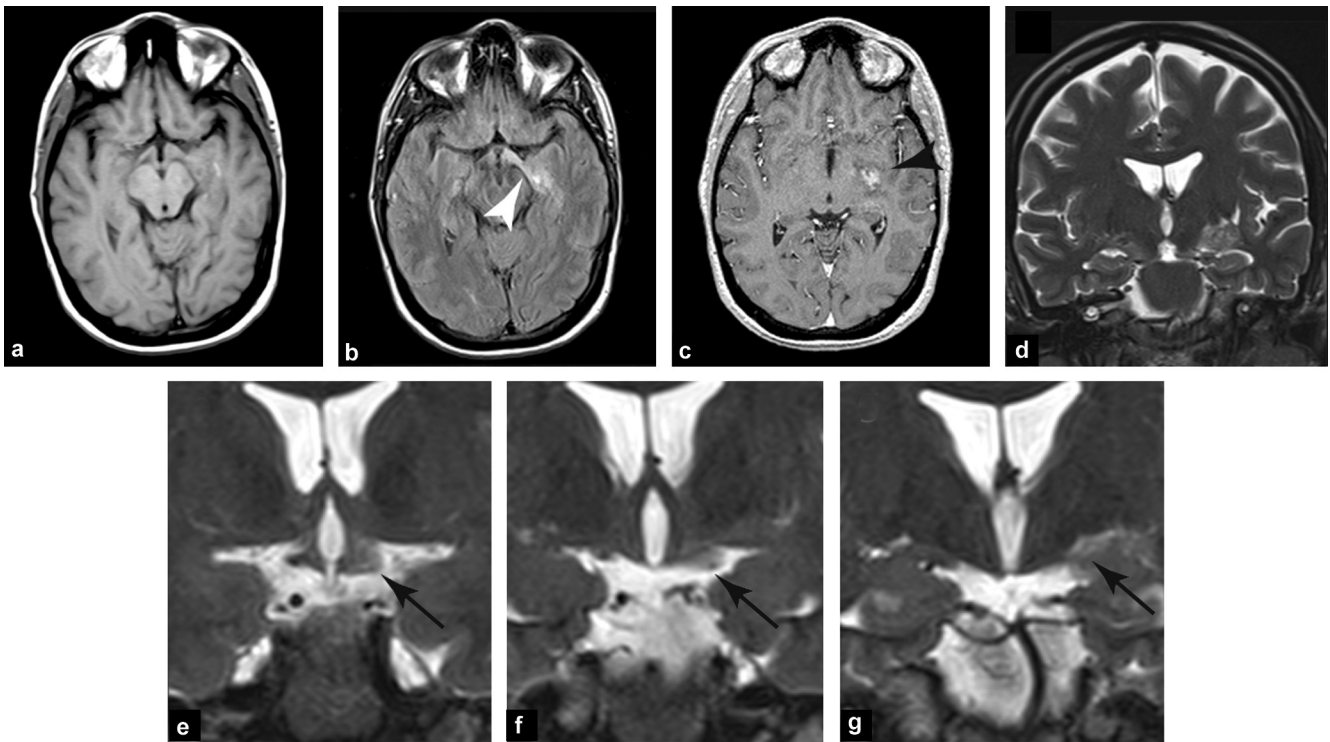


Figure 4. Homonymous lateral hemianopsia right in a 31-year-old patient, revealing a pilocytic astrocytoma of the left optic tract. The intraparenchymatous lesion in isosignal T1, hypersignal FLAIR presents an enhancement after the injection of gadolinium salts (c, tip of black arrow). It induces a hypersignal of the left optic tract on the FLAIR sequences (b, tip of white arrow) and T2 (e, f, g, black arrows).

High-grade gliomas

The anaplastic gliomas and glioblastomas are the most common high-grade gliomas. Especially found in the elderly, they all have an unfavorable prognosis.

The kinetics of tumor growth is superior than that of low-grade gliomas, provoking fast-evolving neurological deficiencies. A papillary edema of the fundus oculi may be found, attesting to intracranial hypertension. These lesions are heterogeneous, comprising hemorrhagic or necrotic rearrangements, enhanced by the intravenous injection in a nodular or annular fashion, and are surrounded by an edema. In first passage perfusion, the rCBVmax is increased. In proton spectroscopy, a choline peak of great amplitude, a drop in the NAA, the presence of lipids and/or lactate indicate malignancy.

Vascular

Any sudden deficiency in the visual field, whether regressive or not, should raise the possibility of an ischemic or hemorrhagic vascular origin. Once the vascular origin is eliminated, the main cause of transient visual disorder is migraine.

Ischemic vascular disease

An ischemic stroke in the posterior cerebral arteries may be the cause of a sudden deficiency in the visual field (Fig. 5).

Cerebral venous thrombosis

Hemianopsia is found in 4% of all cerebral venous thromboses, most often attesting to distress of the visual cortex

[5]. In the sub-acute phase, the thrombus appears in hyper-signal T1 and T2. Venous infarctions are not systematized to an arterial vascular territory, of cortical-subcortical topography and are visible in the form of sometimes hemorrhagic T2 and FLAIR hyperintensities. The presence of an endoluminal defect is corroborated by a venous angiography-MRI sequence.

Migraine with visual aura

A scintillating scotoma lasting for 5 minutes to 1 hour may sometimes precede a migraine. During the episode, the MRI is most often normal. However, the perfusion imaging [6,7] and the T1 imaging after the injection of contrast agent may be perturbed. The anomalies found are dilations of the pial vessels [8] on the one hand and, in first passage perfusion, an elongation of the mean time of transit and the time until the crest value on the other hand, while the cerebral blood volume and the blood flow remain normal. These perfusion disorders may be found in migraine with [9] or without aura [10]. Migraines are sometimes complicated by ischemic accidents [11].

Inflammatory

The other inflammatory causes group different entities. Only two of them will be mentioned here.

Multiple sclerosis

Multiple sclerosis is a common disease, affecting the young subject. The disease evolves in stages, the origin of a major

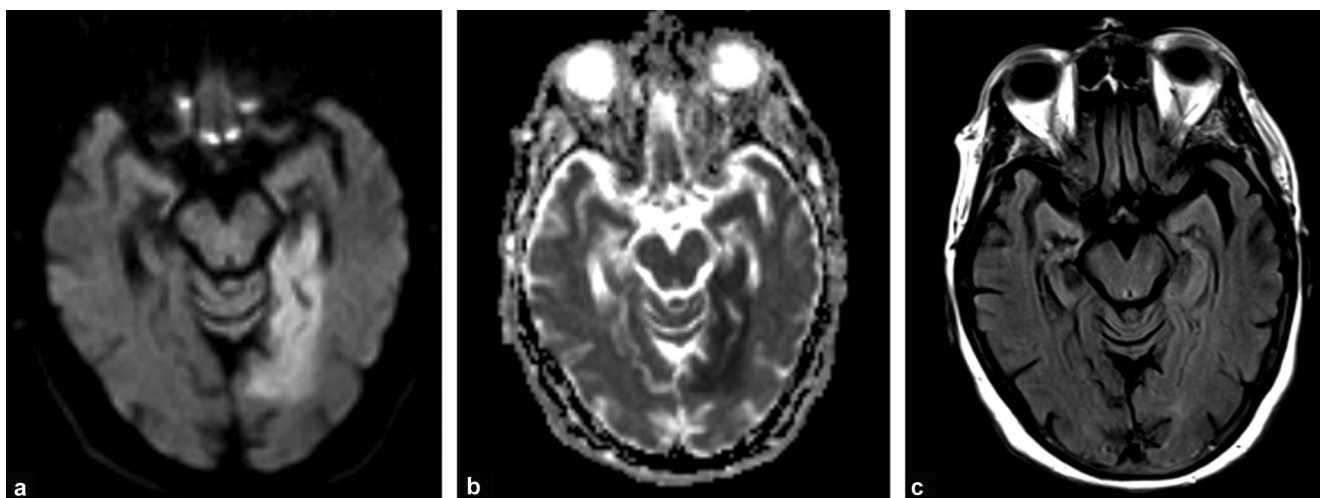


Figure 5. Ischemic stroke in the area of the posterior cerebral artery. Homonymous right lateral hemianopsia, right hypoesthesia and right hemibody weakness in an 82-year-old patient revealing an ischemic stroke in the left posterior cerebral artery. The stroke is visible in the diffusion imaging in hypersignal $b = 1000 \text{ s/mm}^2$ (a), in restriction on the ADC map (b), in slight hypersignal FLAIR (c).

disability. The lesions are typically multifocal, the seat of inflammatory phenomena, demyelination and then a gliosis. The most common seats involve the periventricular regions, the corpus callosum, the optic pathways and in particular the optic chiasm or optic nerves, the brain stem and the cerebellum and finally the spinal cord. The lesions suggesting the diagnosis are of ovoid morphology, callosofugal, with the great axis perpendicular to the ventricles. The location is preferentially periventricular predominant around the temporal horns of the lateral ventricles, or sub-tentorial (where they may involve the spinal cord). MRI provides information about the activity of the disease. The enhancement of the lesions of active multiple sclerosis is nodular, annular or in an open ring.

Sarcoidosis

Sarcoidosis is a multisystemic granulomatosis. The impairment of the central nervous system results from an infiltration of the meningeal spaces that then diffuse to the cranial and spinal nerves, to the vessels, to the hypothalamus-pituitary axis or to the brain parenchyma. The leptomeningeal impairment is manifested in the form of a contrast enhancement according to the relief of the brain. The impairment of the dura mater is revealed by a diffuse or focal thickening of the meninges. Hypothalamic-pituitary and chiasmal impairment is classic, by extension of the meningeal granulomas of the supracellar cisterns. The parenchymatous impairment may take the appearance of a pseudo-tumor by coalescence of the granuloma that appears in hyposignal T2, enhanced after injection. The coexistence of leptomeningeal and parenchymatous enhancement suggests the diagnosis (Fig. 6). The edema and mass effect are in general discrete. Hydrocephaly is often associated. Anomalies of the white matter, in the form of T2 hypersignals, are common. Non-specific, they predominate in the regions and ventricles, and may affect the deep regions (basal ganglia, brain stem), and are not enhanced after injection.

Degenerative: posterior cortical atrophy (Benson's syndrome)

Posterior cortical atrophy (PCA) is a neurodegenerative disease in the young subject, before 60 years of age, associating visual disorders of progressive evolution with atrophy of the posterior cortical regions. It is histologically related to Alzheimer's disease, with an atypical distribution sparing the hippocampus and predominating in the parieto-occipital regions. The structural imaging classically discovers focal, parieto-occipital cortical atrophy and the functional imaging (scintigraphy) demonstrates hypoperfusion and hypometabolism of the same areas (Fig. 7) [12–15].

Toxic and metabolic

Hypoglycemia

The brain lesions induced by hypoglycemia may affect the brain cortex, the hippocampus, the splenium of the corpus callosum, the inner capsules, and the white brain matter. In the MRI, they appear in the form of hyperintensities in diffusion imaging, in restriction on the mapping of the apparent diffusion coefficient (Fig. 8), and are sometimes reversible.

Status epilepticus

A prolonged state of epilepsy (status epilepticus) may be the cause of cortical (Fig. 9), or hippocampus impairment. The atypical forms involve the central grey nuclei (thalamus, striatum, cerebellum). The diffusion imaging is most often suggestive, revealing a non-systematized hypersignal at an arterial territory, where the evolution occurs towards restitution ad integrum.

Posterior reversible encephalopathy

Posterior reversible encephalopathy creates a clinical picture associating seizures, cortical blindness, headache,

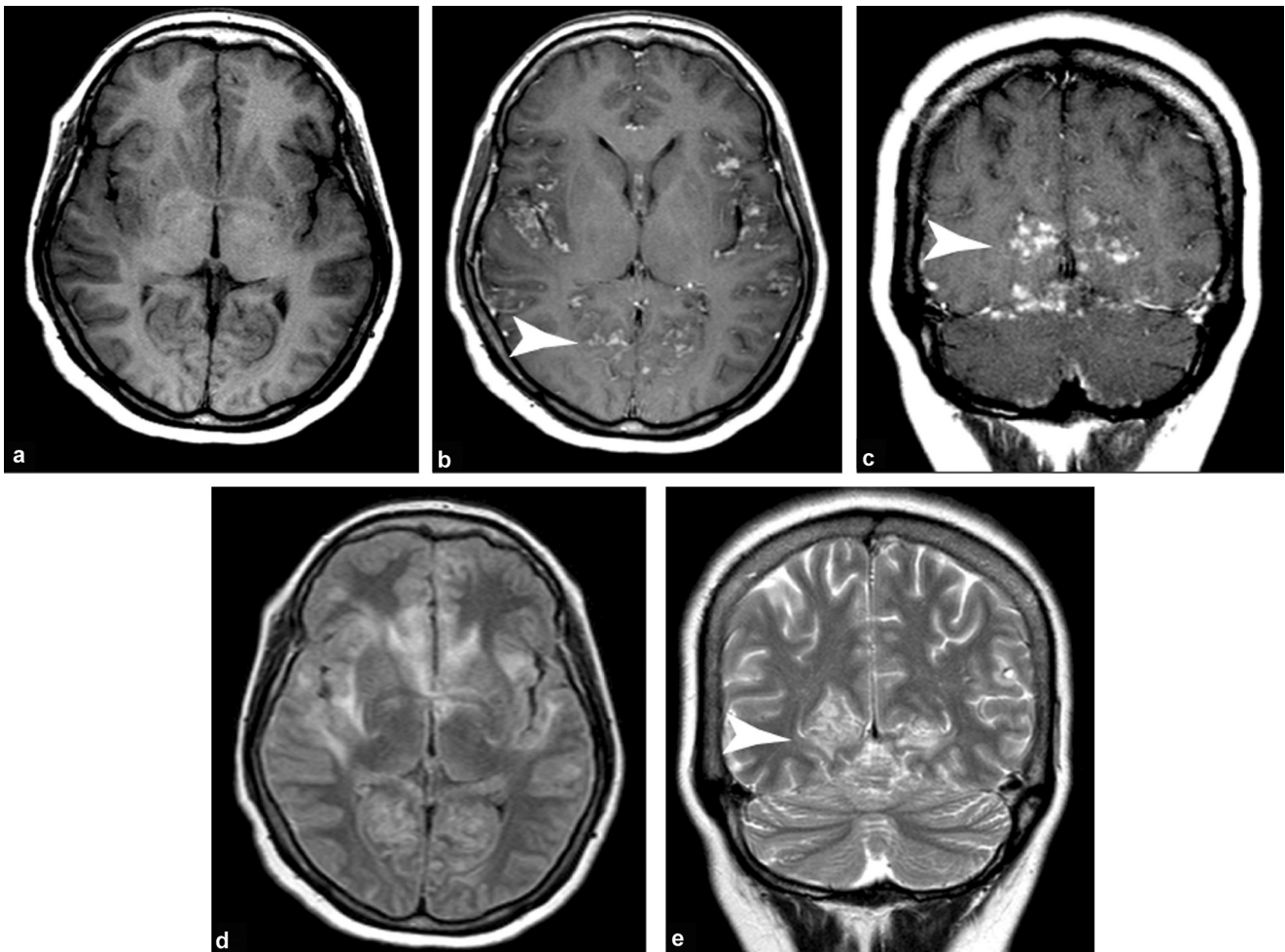


Figure 6. Neurosarcoidosis in a 47-year-old patient. Multiple nodular, confluent, cortico-pial enhancements of the Sylvian fissures as well as the edges of the calcarine fissure (tip of white arrows).

nausea and vomiting, awareness disorders, arterial hypertension and kidney failure. Most often a favouring circumstance is found (eclampsia, transfusion, drug toxicity). The signal modifications most often involve the posterior regions, in a bilateral and symmetrical manner with a vasogenic edema on the diffusion imaging (Fig. 10). They classically regress in ten to fifteen days without sequelae. The atypical forms involve the central grey nuclei or posterior fossa, and may be hemorrhagic.

Infectious: prion infections

Prion infections (sub-acute, transmissible spongiform encephalopathies or Creutzfeldt-Jakob's disease) may be

responsible for posterior cortical impairment resulting in visual symptomatology such as cortical blindness or visual agnosia. Certain types of cortical impairment are predominantly found in the sporadic form of the disease, often associated with bilateral striatal impairment. The topography of these cortical hypersignals is variable, fully visible in FLAIR [16], and on diffusion imaging (the ADC is most often reduced) [17,18], without mass effect or enhancement (Fig. 11). The Heidenhan form, also sporadic [19], involves an elective impairment of the parieto-occipital cortex.

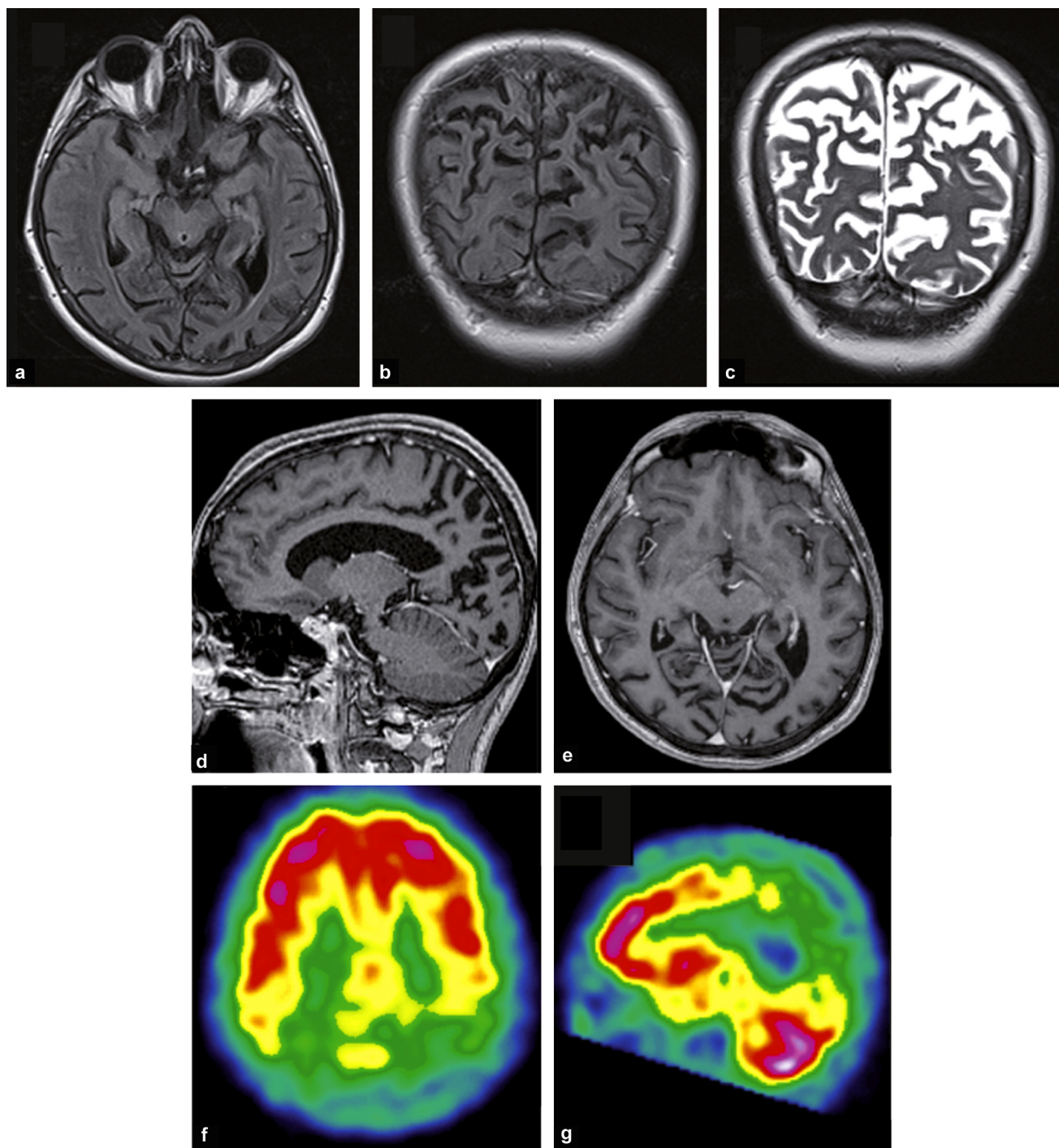


Figure 7. Visual and cognitive disorders in a 62-year-old patient revealing posterior cortical atrophy (Benson's syndrome). The MRI finds focal, parieto-occipital atrophy creating a bilateral sulcal enlargement (a to e). The perfusion CT-scan reveals parieto-occipital hypoperfusion (f and g).

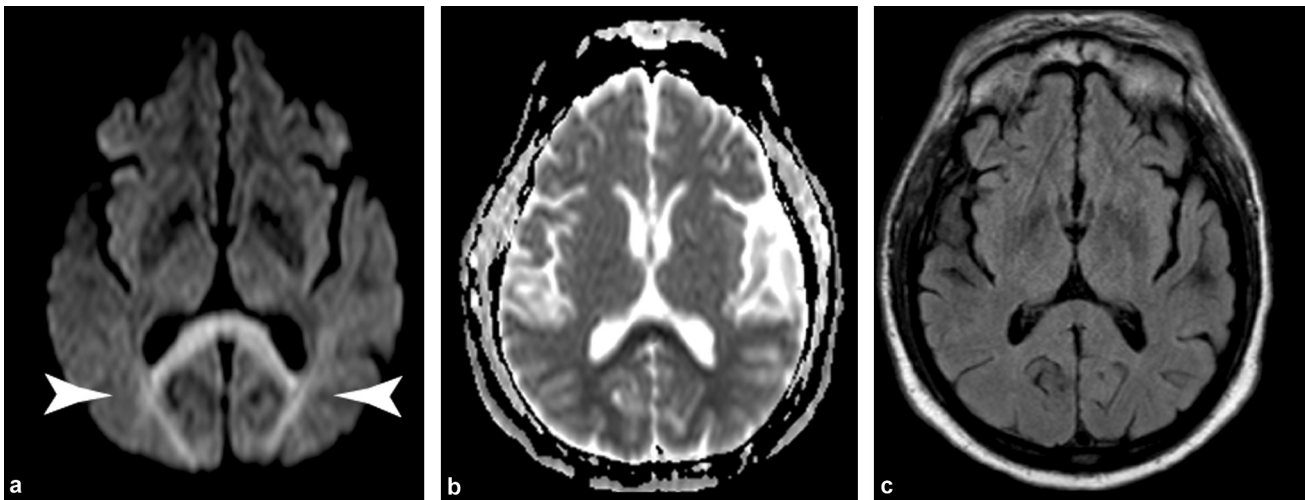


Figure 8. Sixty-eight-year-old, diabetic patient. The day before hospitalization, spontaneously regressive phasic disorder followed by the installation of a coma the following day. Upon care by the emergency medical aid, capillary glycaemia at 0.14 g/L. Signal anomalies of the occipital forceps and the bilateral occipital subcortical white matter related to severe hypoglycemia, in hypersignal on the diffusion imaging $b = 1000 \text{ s/mm}^2$ (a, tip of arrows), in restriction on the ADC mapping (b), without translation on the FLAIR sequence.

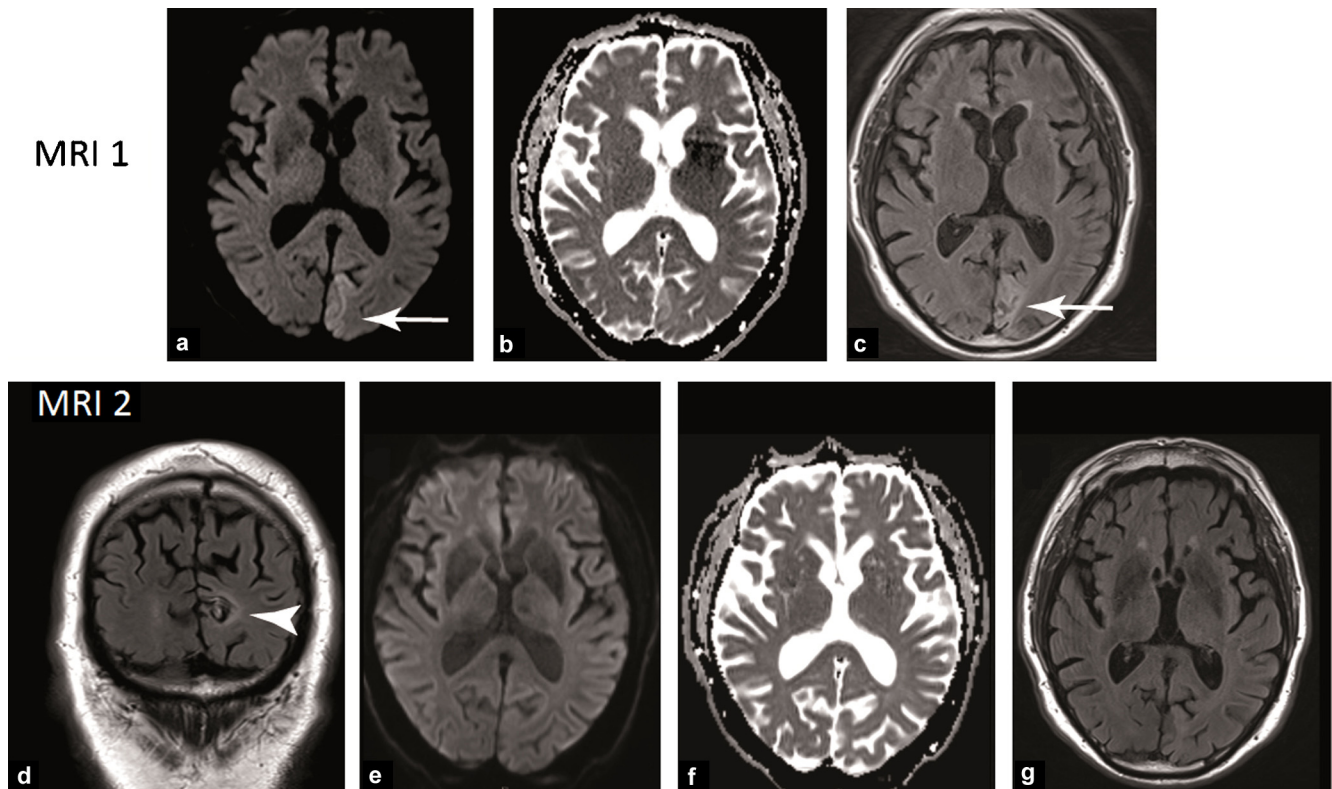


Figure 9. Seventy-six-year-old patient with a past history of left posterior ischemic stroke, hospitalized for partial status epilepticus. The initial MRI found the old ischemic sequence (tip of arrow, d) and identified the signal anomalies of the left occipital ribbon (white arrows), in hypersignal on the diffusion imaging $b = 1000 \text{ s/mm}^2$ (a), increase in the apparent diffusion coefficient (b), FLAIR hypersignal (c). The follow-up MRI carried out 10 days after the episode revealed a regression of the previously described lesions, in relation to the status epilepticus.

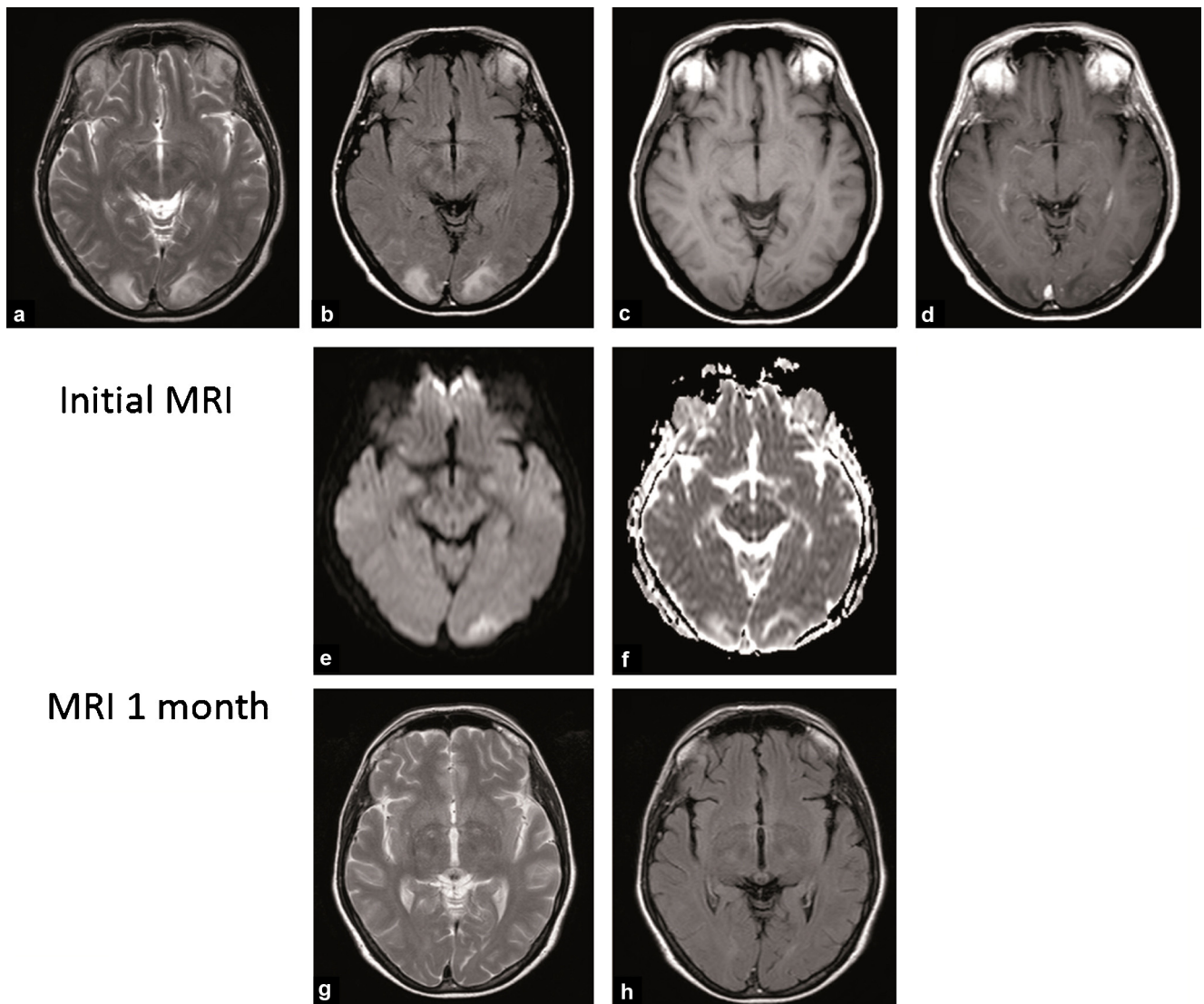


Figure 10. Sixty-five-year-old patient treated by chemotherapy for a pulmonary adenocarcinoma. Emergency hospitalization for generalized seizures and cortical blindness. An emergency MRI revealed posterior, bilateral and asymmetrical cortico-subcortical signal anomalies in T2 (a) and FLAIR (b), in hyposignal T1 (c) presenting several ruptures of the hemato-encephalic barrier after the injection of gadolinium salts (d), in hypersignal on the diffusion imaging (e), attesting to a vasogenic edema on the ADC map (f). The control MRI after 1 month reveals the ad integrum restoration on the T2 (g) and FLAIR (h) sequences.

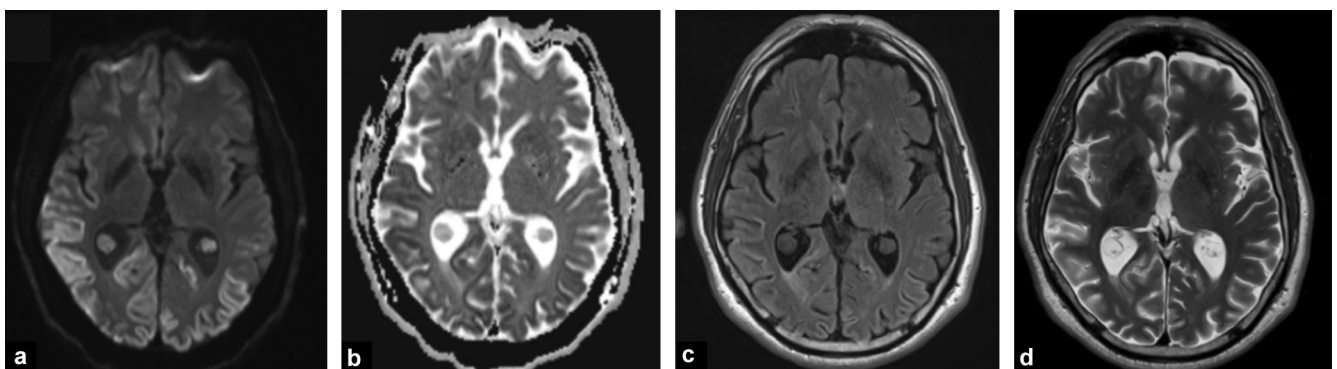


Figure 11. Seventy-two-year-old patient presenting visual disorders for the last 3 months, then progressive, rapidly installed ataxia, followed by a behaviour disorder and extrapyramidal impairment. Signal anomaly of the cortical ribbon in hypersignal on the diffusion imaging ($b = 1000 \text{ s/mm}^2$) (a), in restriction on the ADC mapping (b), in FLAIR hypersignal (c) and T2 (d).

Conclusion

The exploration of the chiasmal and retrochiasmal visual pathways is based on MRI. A bitemporal hemianopsia should call to mind a lesion of the optic chiasm while a homonymous lateral hemianopsia should lead to a search for a lesion of the retrochiasmal visual pathways. The causes of chiasmal impairment are mainly tumoral. In view of a seemingly retrochiasmal impairment, the tumoral or vascular origin should be suspected initially.

TAKE-HOME MESSAGES

- The exploration of the chiasmal and retrochiasmal visual pathways is based on magnetic resonance imaging.
- A bitemporal hemianopsia should suggest a lesion of the optic chiasm while a homonymous lateral hemianopsia should lead to a search for a lesion of the retrochiasmal visual pathways.
- The causes of chiasmal impairment are mainly tumoral.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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