Pathologies of postchiasmatic visual pathways and visual cortex
Optic radiation: anatomy
Pathologies of the postchiasmsatic visual pathways and visual cortex

Characterized by *homonymous hemianopsia*.

This visual field defect may be the consequence of lesions of the optic tract, optic radiation or visual cortex.
Optic tract
Rare by itself.

• Lesions involving the optic tract are generally large, and include the optic nerve and chiasm.

• **Etiology**
  • pituitary tumors,
  • aneurisms,
  • craniopharyngioma
  • demyelinating lesions.

• The presence of neurological symptoms is not determinant for the diagnosis.

• **Clinical characteristics** suggesting the involvement of the optic tract include:
  • homonymous hemianopsia, complete or incongruous,
  • relative afferent pupillary defect (RAPD) in the eye with the defect involving the temporal hemifield (contralateral to the lesion)
  • sectorial optic atrophy.
Optic tract field defect
The lateral geniculate nucleus represents the termination of nerve fibers which originate from the retina, and is generally involved in lesions of the temporal lobe.

- homonymous cone-shaped visual field defects

The fibers constituting the optic radiation originate from the superoposterior portion of the lateral geniculate nucleus. The first portion of the optic radiation, made up of inferior fibers (corresponding to the superior visual field), run anteriorly infront of the temporal horn of the lateral ventricle forming a loop known as «Meyer’s loop». In this site the visual fibers are adjacent to the internal capsule and the temporal isthmus. Lesions of the temporal optic radiations determine the onset of a homonymous defect prevalently involving the superior quadrants.
Optic radiation: anatomy

- lateral geniculate body
- visual cortex
- temporal horn of lateral ventricle
- Meyer's loop
• **Visual defects:** may involve a sector of the superior quadrant and have vertical margins which exactly correspond to the vertical meridian, while horizontal margins are rather irregular and undefined.

• The defect may obviously extend towards the inferior quadrant, causing a hemianopsia.

• **Associated neurological symptoms:**
  • hemiplegia
  • hemianaesthesia,
  • aphasia (when the dominant hemisphere is involved),
  • agnosia, hallucinations (sight, hearing, smell, taste),
  • epilepsy and behavioural changes.

• Pathologies causing lesions of the temporal lobe may have neoplastic (glioma, meningioma etc.), vascular and rarely inflammatory nature.
As the fibers proceed towards the calcarine sulcus they become more and more segregated.

Thus **lesions of the parietal lobe** cause a more congruous homonymous defect than temporal lobe lesions.

**Associated neurological symptoms:**

- severe hemianaesthesia
- hemiparesis
- alexia
- asomatognosia

These are generally so severe that in some cases do not allow the carrying out of the visual field examination.
• In addition it is possible to observe:
  • tonic deviation of the eyes to the opposite side of the parietal lesion, trying to reproduce Bell’s phenomenon.
  • reduction or absence of the optico-kinetic nystagmus when rotating the stimulus to the side of the lesion.

The most common causes of a parietal lobe syndrome are:
• tumors
• vascular lesions
• infectious pathologies (coccidiomycosis, candidiasis etc.).

In subjects with a parietal lobe syndrome, visual acuity, pupillary reactions and optic disc aspects are normal.
Temporal lobe defect
CT scan of parietal lobe tumour with loss of optokinetic nystagmus
• **Occipital lobe**
  
  • most of the visual cortex lies deeply in the medial portion of the occipital lobe;

  • macular projections have a posterio-lateral position in the occipital lobe;

  • the peripheral visual field has the most anterior projection along the interhemispheric fissure.
• Clinical characteristics of occipital lesions include:
• Congruity of the homonymous visual field defect.
• Defects of the visual field with occipital origin may present as:
  • a) congruous scotoma,
  • b) quadrantanopsia, complete hemianopsia or homonymous bilateral defects.
• c) Of particular interest, and typical of occipital lesions, is the phenomenon of macular sparing represented by the conservation of the central 5-10 degrees of the visual field on the side of the hemianopsia.
• This is explained by the double vascularization of the occipital pole by the *middle and posterior cerebral arteries* and by the possibility of a bilateral macular representation. Some patients affected by cortical lesions show a static-kinetic dissociation (*Riddoch’s phenomenon*), and perceive movements of a stimulus without perceiving its shape. This phenomenon is not considered pathognomonic of occipital lesions as it occurs in the presence of other pathologies of the visual pathways.
Circle of Willis
Blood supply of the optic radiations
Diagram of the visual cortex

- splenium of corpus callosum
- posterior pole of left cerebral hemisphere
Occipital lobe defect
CT scan of occipital infarct
Pathologies involving the occipital lobe are generally of vascular (adult age) or neoplastic (glioma and meningioma) origin

- Associated symptoms:
  - headache, nausea and vomiting, papilloedema and signs of cerebellar involvement

- Since the occipital lobe is connected to secondary associative areas and the two hemispheres are connected through the splenium of the corpus callosum, the involvement of these structures determines the onset of characteristic clinical manifestations. For example, the extension of the lesion towards the occipital lobe and splenium causes alexia without agraphia, associated to right homonymous hemianopsia.
• **Cortical blindness** is the consequence of bilateral lesions of the retrogeniculate visual pathways or visual cortex.

• Lesions generally develop simultaneously and must be anatomically symmetrical.
• **Etiology:**

  • vascular nature.
  • infectious nature,
  • toxic (eg. Carbon monoxide)
  • severe hypoxic or embolic phenomena during cardiac surgery.

• Clinical characteristics of cortical blindness include complete loss of visual function, normal pupillary reflexes, normal ocular fundus and normal ocular motility.

• Subjects with bilateral blindness may refer visual hallucinations.
• **Visual hallucinations** are defined as the visual perception of something that the subject is convinced he/she sees, but which is not visible to other people present in the same room.

• They are present in subjects with neurological and visual defects. Visual hallucinations may also be caused by:
  • drugs such as amphetamines,
  • anti-Parkinson drugs,
  • antidepressives,
  • cardiovascular drugs and numerous antibiotics
• Some forms of **migraine** may be responsible for visual hallucinations. The differential diagnosis is however facilitated by the presence of characteristic scintillating or fortifying scotomas in migraine.
- **Visual agnosia** is a rare disorder of the superior cortical functions in which patients are not able to recognize familiar objects, notwithstanding normal visual acuity. Only utilizing hearing and smelling are patients able to identify the object.

- The origin of this disorder is the presence of bilateral occipital or parieto-occipital lesions. Some forms of agnosia are characterized by the inability to recognize familiar faces (*prosopagnosia*) until the known person speaks. The latter seems to be the consequence of bilateral lesions of the inferior occipital-temporal junction.