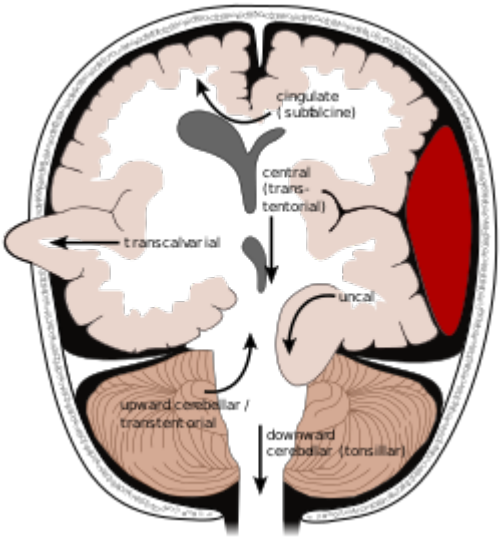
Uncal herniation



General information

Uncal herniation usually occurs in rapidly expanding traumatic intracranial hematomas, frequently in the lateral middle-fossa or temporal lobe pushing medial uncus and hippocampal gyrus over the edge of the tentorium, entrapping the third nerve and directly compressing midbrain. PCA may be occluded (as with central herniation).

Impaired consciousness is NOT a reliable early sign. Earliest consistent sign: unilaterally dilating pupil. However, it is unlikely that a patient undergoing early uncal herniation would be completely neurologically intact except for anisocoria (do not dismiss confusion, agitation, etc.). Once brainstem findings appear, deterioration may be rapid (deep coma may occur within hours).

Uncal herniation is a subtype of transtentorial herniation (downward brain herniation), usually related to cerebral mass effect increasing the intracranial pressure.

Etiology

Uncal herniation occurs secondary to large mass effect (that can occur from traumatic or non-traumatic haemorrhage, malignancy, etc.) that will lead to increased intracranial pressure and herniation.

Stages

Early third nerve stage

This is not a brainstem finding, it is due to 3rd nerve compression.

Pupils: Approach to the comatose patient

Oculomotor: Doll's eyes (oculocephalic reflex)=normal or dysconjugate. CWC (oculovestibular reflex) = slow ipsilateral deviation, impaired nystagmus, may be dysconjugate if external oculomotor ophthalmoplegia (EOO).

Respirations: Normal.

Motor: Appropriate response to nociceptive stimulus. Contralateral Babinski.

Late third nerve stage

Midbrain dysfunction occurs almost immediately after symptoms extend beyond those due to focal cerebral lesion (i.e., may skip diencephalic stage, due to lateral pressure on midbrain). Treatment delays may result in irreversible damage.

Pupils: Pupil fully dilates.

Oculomotor: Once pupil blown, then external oculomotor ophthalmoplegia (EOO). Consciousness: Once EOO occurs: stupor→coma

Respirations: Sustained hyperventilation, rarely Cheyne-Stokes.

Motor: Usually produces contralateral weakness. However, the contralateral cerebral peduncle may be compressed against the tentorial edge, causing ipsilateral hemiplegia (Kernohan's phenomenon, a false localizing sign). Then bilateral decerebration (decortication unusual).

Midbrain—upper pons stage

Contralateral pupil fixes in midposition or full dilation. Eventually, both midposition (5–6 mm) and fixed. Oculomotor: Impaired or absent. Respirations: Sustained hyperpnea. Motor: Bilateral decerebrate rigidity. Following the midbrain—upper pons stage

From this point onward, the uncal syndrome is indistinguishable from central herniation (see above).

Clinical presentation

Abnormal posture and poor GCS. There may be pupillary dilation and loss of light reflex due to direct compression of the oculomotor nerve.

Pathology

In uncal herniation, the uncus and adjacent part of the temporal lobe glide downward across the tentorial incisura compressing the brainstem and the posterior cerebral arteries in the ambient cistern. Uncal herniation may be unilateral or bilateral

Diagnosis

Uncal herniation diagnosis.

Clinical characteristics differentiating uncal from central herniation

- decreased consciousness occurs early in central herniation, late in uncal herniation
- uncal herniation syndrome rarely gives rise to decorticate posturing

Complications

extensive brainstem ischemia Duret haemorrhage contralateral midbrain compressed against tentorium, may cause Kernohan phenomenon compression of the ipsilateral posterior cerebral artery will result in ischemia of the visual cortex with resultant homonymous hemianopsia Practical points

if uncal herniation is diagnosed, the referring physician should be notified immediately, because of its life-threatening nature