Cerebral Herniation Syndromes

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Cerebral Herniation Syndromes

- Cerebral herniation occurs when the brain shifts across structures within the skull such as the falx cerebri, the tentorium cerebelli and the foramen magnum.
Cerebral Herniation Syndromes

Cerebral herniation is caused by a number of factors that cause a mass effect within the skull and increase the intracranial pressure including:

- Cerebral edema
- Hematoma
- Stroke
- Tumor
- Infection
Cerebral Herniation Syndromes

- There are four main types of brain herniation syndromes:
  - Subfalcine
  - Central or downward transtentorial
  - Temporal transtentorial or uncal
  - Cerebellar tonsillar
Subfalcine Herniation

- Subfalcine herniations occur as the brain extends under the falx cerebri.
- Imaging characteristics include a shift of the septum pellucidum, effacement of the anterior horn of the lateral ventricle, and compression of the anterior cerebral artery against the falx.
Subfalcine Herniation

- The most common form of herniation
- Presence does not necessarily lead to severe clinical symptomatology or harm
- Shift of the septum pellucidum from midline can be measured in millimeters and compared over time to determine any change
- Present clinically as headache and as the herniation progresses, contralateral leg weakness
Uncal Herniation

- Subset of transtentorial herniations
- The uncus, the medial part of the temporal lobe, is displaced into the suprasellar cistern
- As the herniation progresses the uncus puts pressure on the midbrain
Uncal Herniation

- As the uncus herniates it squeezes the third cranial nerve affecting the parasympathetic input to the eye causing and pupillary dilation and a lack of pupillary constriction to light
Uncal Herniation

- Contralateral hemiparesis occurs with compression of the ipsilateral cerebral peduncle of the midbrain.

- Since the corticospinal tracts decussate below the midbrain, the hemiparesis is contralateral.
In some cases of uncal herniation the lateral translation of the brainstem is so severe that the midbrain is pushed against the opposite edge of the tentorium.
Kernohan’s Notch

- A false localizing sign occurs as the shift of the midbrain causes compression of the contra-lateral cortico-spinal tract and less frequently, the contra-lateral third nerve.

- The side of the dilated pupil is a much more reliable sign (90%) of the side of the lesion than the side of the hemiparesis.
Uncal Herniation

- In addition to pupillary dilatation, a second key feature of uncal herniation is a decreasing level of consciousness (LOC) due to distortion of the ascending arousal systems as they pass through the midbrain.
- A dilated pupil from in the absence of a LOC is not due to uncal herniation.
Central Herniation

- In the first phase of central herniation, the diencephalon (the thalamus and hypothalamus) and the medial parts of both temporal lobes are forced through a notch in the tentorioum cerebelli.
Central Herniation

- Caused by diffuse cerebral edema as seen in patients with severe traumatic brain injury
- CT Scan shows effacement of the perimesencephalic cisterns and loss of gray-white matter differentiation
Central Herniation

- Early diencephalic stage (reversible)
  - Decreasing level of consciousness with difficulty concentrating, agitation and drowsiness
  - Pupils are small (1-3 mm) but reactive
  - Pupils dilate briskly in response to a pinch of the skin on the neck (ciliospinal reflex)
  - Oculocephalic reflexes are intact (Doll’s eyes)
  - Plantar responses are flexor
  - Respirations contain deep sighs, yawns and occasional pauses then progress to Cheyne-Stokes
Central Herniation

- Late diencephalic stage
  - Patient becomes more difficult to arouse
  - Localizing motor responses to pain disappear and decorticate posturing appears with eventual progression to decerebrate posturing
Central Herniation

- Progressive diencephalic impairment is thought to be the result of stretching of the small penetrating vessels of the posterior cerebral and communicating arteries which supply the hypothalamus and thalamus.
As herniation progresses to the midbrain stage signs of oculomotor failure appear

- The pupils become irregular and then fixed at midposition
- Oculocephalic movements become more difficult to elicit
- Extensor posturing appears spontaneously
- Motor tone is increased and plantar responses are extensor
Central Herniation

- The progression of symptoms indicates irreversible ischemia and therefore intervention must occur before the midbrain stage to prevent permanent deficits from central herniation.
Tonsillar Herniation

- The cerebellar tonsils move downward through the foramen magnum causing compression of the medulla oblongata and upper cervical spinal cord
- May cause cardiac and respiratory dysfunction
Treatment of Cerebral Herniation

- Treat the underlying cause of the raised intracranial pressure that is causing the brain to herniate from one intracranial compartment into another
Monro Kellie Doctrine

- The Monro Kellie doctrine states that the intracranial compartment is incompressible and the volume inside the cranium is a fixed volume.
- The intracranial volume constituents are brain tissue, blood and cerebrospinal fluid (CSF).
Monro Kellie Doctrine

- Changes in ICP may result from an increase in volume of brain tissue, blood or CSF
- Compensatory mechanisms maintain a normal ICP for any increase in volume of 100-120 ml
- For example, the mass effect of a hematoma causes a decrease in the volume of CSF and venous blood within the brain to maintain a normal ICP
Treatment of Cerebral Herniation

- When the lesion volume increases beyond the point of compensation, the ICP increases, which can lead to cerebral herniation.

- The first treatment of raised ICP is to remove the lesion causing mass effect within the brain, such as a tumor, hematoma, or abscess.
Treatment of Cerebral Herniation

- Preop subfalcine herniation from a subdural hematoma (L). Postop CT shows resolution of the midline shift. Also note the presence of a craniectomy to treat increased ICP from cerebral edema.
Treatment of Cerebral Herniation

- Hydrocephalus caused by a mass lesion or intraventricular blood should be aggressively treated by removing the mass lesion and/or placing a ventriculostomy.
Treatment of Cerebral Herniation

Methods to decrease cerebral edema
- Maintain adequate cerebral oxygenation to minimize vasodilatation
- Maintain CPP (MAP-ICP) greater than or equal to 60 mm Hg to increase vasoconstriction
- Mild hyperventilation to increase vasoconstriction
- Intubation as required to avoid hypercapnia which leads to vasodilatation
- HOB 30 degrees to increase venous drainage
- Sedation to decrease cerebral metabolism
- Seizure control
Treatment of Cerebral Herniation

- Place ventriculostomy to drain CSF
- Use osmotic therapy (mannitol, lasix, hypertonic saline) to pull fluid out of the brain tissue
Treatment of Cerebral Herniation

- Decompressive craniectomy allows for the control of increased ICP from cerebral edema caused by trauma or stroke.
- In this case, cerebral herniation through the defect is desired.
- Efficacy is controversial.
In summary, cerebral herniation is the result of increased intracranial pressure which exceeds the body’s compensatory mechanisms.

Understanding the types of cerebral herniation is essential to making the diagnosis and determining the best course of treatment.
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Thank you