This is a 5-year old female who is brought to the emergency department at 8:00 a.m. because she was poorly responsive when her mother awoke her in the morning. This prompted her mother to drive her to the E.D. There is a history of headache and vomiting during the evening and night. There is no history of trauma.

Exam: VS T36.7 (rectal), P92, R32, BP 137/97. She is minimally responsive. Pupils equal and reactive. There are no signs of external trauma. Within minutes of arrival, she exhibits extensor posturing. She is orally intubated using the rapid sequence induction method with atropine, thiopental, and vecuronium. She is hyperventilated. End-tidal CO2 monitoring is used to keep her pCO2 in the 25 mmHg range. A loading dose of phenytoin is administered.

An emergency CT scan is ordered.

There is obvious bilateral intraventricular hemorrhage and ventricular dilatation. Clinically, extensor posturing suggests the possibility of impending herniation. She sustains episodes of bradycardia which respond to doses of IV mannitol. A neurosurgeon decompresses her ventricles immediately. She recovers well without neurological deficits. Subsequent studies demonstrate the presence of a choroid plexus arteriovenous malformation. This is neurosurgically ablated.

Discussion

Increases in intracranial pressure (ICP) compress the brain within the rigid skull. This reduces cerebral blood flow prompting reflex hypertension to maintain cerebral perfusion. As intracranial pressure increases further, the contents of the skull can no longer remain in place. Focal increases in pressure, such as with tumors and acute hemorrhages, result in focal deviations in anatomy. While the term "herniation" is used loosely when intracranial pressure increases,
there are specific herniation syndromes with different mechanisms and outcomes. Identifying increases in intracranial pressure by clinical and radiographic means is important to intervene early to prevent herniation. Clinical signs and symptoms of acute increased intracranial pressure include, headache, vomiting, vision distortion, diminished sensorium, pupillary dysfunction, hypertension, bradycardia, flexor/extensor posturing, etc. Papilledema may not be present if ICP increases acutely.

When intracranial hypertension is suspected, an immediate CT scan should be obtained to assess the degree of ICP increase and to identify the cause of the this.

The following areas should be assessed on CT when attempting to determine the presence and severity of intracranial hypertension. These are discussed in more detail below.
1. Prominence of sulci/gyri.
2. Lateral ventricle size.
4. Suprasellar cistern.
5. Quadrigeminal cistern.

There are several brain herniation syndromes. These are discussed in more detail below.
1. Uncal herniation.
2. Transtentorial herniation.
3. Tonsillar herniation.
4. Subfalcine herniation.
5. Superior vermian herniation.

CT signs of intracranial hypertension:

1. Prominence of sulci/gyri:
   When intracranial pressure increases, this compresses the cerebral cortex against the calvarium. This attenuates the visibility of the sulci and gyri. Additionally, the space between the cortex and the calvarium is minimal when ICP increases.

View loss of sulci/gyri.

The image on the left is a high CT cut which should show the sulci and gyri well. Due to increased ICP, the cortex is compressed up against the calvarium losing the distinctness of the sulci and gyri. The space between the cortex and the calvarium is obliterated.
The sulci/gyri sign cannot be totally relied upon in some instances. In cases of external hydrocephalus or chronic (or subacute) subdural effusions, fluid collects over the cortex. The fluid space between the cortex and the calvarium appears to be increased and the sulci/gyri may appear prominent.

View prominent sulci/gyri.

![Image of a focal extra-axial hematoma. Note the prominent sulci and gyri despite intracranial hemorrhage.](image)

2. Lateral ventricle size:

   In acute hydrocephalus, due to obstruction in the outflow of CSF, the lateral ventricles will be enlarged. Similarly, in acute intraventricular hemorrhage, the lateral ventricles will be enlarged.

View dilated ventricles.

![Images of bilateral dilated lateral ventricles due to acute intraventricular hemorrhage.](image)

In other causes of intracranial hypertension, the lateral ventricles will be compressed (slit-like) or obliterated due to increases in pressure in compartments other than the lateral ventricles. This is the case in generalized cerebral edema, subdural hematoma, epidural hematoma, etc.

View compressed ventricles.
Shown here are two cuts showing subarachnoid hemorrhage. The ventricles are slit-like due to cerebral edema and acute hemorrhage resulting in intracranial hypertension.

3. Grey/White matter distinction:
   This is mostly a sign of cerebral edema in association with elevated ICP.

View good grey/white matter distinction.

View poor grey/white matter distinction.
4. Suprasellar cistern:

The suprasellar cistern is a fluid-filled space above the sella turcica. It contains the circle of Willis and the optic chiasm. On CT scan, it has a star-shaped appearance. Anteriorly, the top point of the star is formed by the interhemispheric fissure between the two frontal lobes. The lateral border of the suprasellar cistern is formed by the uncal portion of the temporal lobes. The posterior border is formed by the pons in lower cuts and the cerebral peduncles of the midbrain in higher cuts. In lower cuts where the pons forms the posterior border of the suprasellar cistern, the suprasellar cistern takes on the shape of a 5-pointed star. In cuts where the cerebral peduncles (which have a central cleft) form the posterior border of the suprasellar cistern, the suprasellar cistern takes on the shape of a 6-pointed star.

View the midline anatomic diagram of the brain.
Po - pons
P - cerebral peduncles (midbrain)
M - medulla
C - quadrigeminal plate (superior and inferior colliculi)
V - fourth ventricle
Q - quadrigeminal cistern

Note that the fourth ventricle is connected to the third ventricle by the cerebral aqueduct which is very thin and may not be visible on CT.

View the anatomic diagram of the suprasellar cistern.

The midline sagittal MRI scan shows the levels of the axial diagrams. Note that the fourth ventricle is at roughly the same level of the suprasellar cistern, but depending on the angle of the axial cut, the fourth ventricle may be seen in cuts above, below, or at the same level as the suprasellar cistern. The suprasellar cistern is seen in cuts 7 and 8.

In the lower cut (7), the suprasellar cistern (s) takes on the shape of a five pointed star. The frontal lobes (F) form the anterior border with the anterior interhemispheric fissure between the frontal lobes forming the apex of the star. The uncus (U) of the temporal lobes forms the lateral borders. The pons (Po) forms the posterior border. The fourth ventricle (V) is also seen in this cut.

In the higher cut (8), the suprasellar cistern (s) takes on the shape of a six pointed star. The only difference higher up is that the posterior border is formed by the cerebral peduncles (p) of the midbrain. The cleft between the cerebral peduncles forms the sixth point of the star. The inferior colliculi (c) can also be seen at this level of the suprasellar cistern.

View CT scan of suprasellar cistern.
The left and center images show the suprasellar cistern. Its anterior borders are formed by the frontal lobes (F). Its lateral borders are formed by the uncus (U) of the temporal lobes. The left image shows the 5-pointed star appearance of the suprasellar cistern where the posterior border is formed by the pons (Po). The black arrow points to the fourth ventricle. The center image shows a higher cut where the suprasellar cistern has a 6-pointed star appearance since the posterior border is formed by the cerebral peduncles (P) which have a central cleft.

When ICP increases, the suprasellar cistern space is compressed. The space may still be visible; however, with severe intracranial hypertension, the cistern is obliterated due to encroachment of brain tissue that normally forms the borders of the suprasellar cistern. Depending on the cause of the intracranial hypertension, the suprasellar cistern may be totally obliterated in global or severe ICP increase. In focal lesions, brain tissue may encroach into only one part of the suprasellar cistern. In early unilateral uncal herniation, the uncus of the temporal lobe (lateral border of the suprasellar cistern) will protrude into the suprasellar cistern.

5. Quadrigeminal cistern:

Also known as the quadrigeminal plate cistern, this fluid filled space is located cephalad to the fourth ventricle.

View the anatomic diagram of the quadrigeminal cistern.
The midline sagittal MRI scan shows the levels of the axial diagrams. The quadrigeminal cistern is located above (anterior to) the "Q" in the highest cut shown (number 9). The anterior border of the quadrigeminal cistern is formed by the superior colliculi (c). Image 8 (lower cut) also shows the quadrigeminal cistern. In this case, its anterior border is formed by the inferior colliculi (c). This gives the anterior border of the quadrigeminal cistern the appearance of a "baby's bottom". The quadrigeminal plate is comprised of the superior and inferior colliculi. The quadrigeminal cistern is posterior to this quadrigeminal plate, thus its anterior border may be formed by the inferior or superior colliculi.

View CT scan of quadrigeminal cistern.

The right image shows the quadrigeminal cistern (black arrow). Note the "baby's bottom" appearance of its anterior border. When ICP is increased, the quadrigeminal cistern space is compressed or obliterated.
Identify the suprasellar and quadrigeminal cisterns in the following examples.

View moderately increased ICP.

The suprasellar cistern is slightly smaller than its normal size (the right uncus is pushing into the suprasellar cistern) and the quadrigeminal cistern is compressed. An epidural hematoma is noted.

View severe ICP increase.

The suprasellar cistern (left image) is tissue-filled, indicating the presence of brain tissue herniating into this space. The quadrigeminal cistern is very
compressed and pushed posteriorly (center image). The suprasellar cistern is located just above the base of the skull (above the sella). It should be visible in the cuts near the base of the brain. If it is not visible, it suggests that the suprasellar cistern is obliterated. Similarly, the quadrigeminal cistern should be located in the cut above the suprasellar cistern. A subdural hematoma is noted with a midline shift.

Brain Herniation Syndromes:

1. Uncal herniation:
    When mass effects within or adjacent to the temporal lobe occur, the medial portion of the temporal lobe (uncus) is forced medially and downward over the tentorium. There is ipsilateral pupillary dilation. The uncus is pushed medially into the suprasellar cistern.

View uncal herniation.

There is bilateral uncal herniation. The suprasellar cistern is obliterated.

View early uncal herniation.
2. Transtentorial herniation:
   It should be noted that this term is somewhat vague. It is used rather loosely and it may sometimes be used similarly to the terms temporal lobe herniation and uncal herniation. The uncus may herniate over the tentorium as described above. Supratentorial lesions on one side may initially result in uncal herniation. As ICP increases further, bilateral temporal lobe herniation occurs transtentorially. Early unilateral uncal herniation is more accurately called uncal herniation. The terms cranial-caudal transtentorial herniation, rostro-caudal transtentorial herniation, or central transtentorial herniation more accurately describe what is generally meant by "transtentorial herniation". Thus, uncal herniation is described separately above.

   In transtentorial herniation the medial portions of the temporal lobes (uncus) and the brainstem herniate downward from supratentorial to the infratentorial compartment. The clinical signs include headache, decreasing level of consciousness and ipsilateral fixed dilated pupil (from compression of the third cranial nerve on the ipsilateral side). As herniation worsens, decerebrate (extensor) posturing, contralateral (i.e., bilateral) pupillary dilation and Cushing's triad occur. Cushing's triad includes alteration in respiration, bradycardia, and systemic hypertension. It is rare to have all three present in children. Often there is just bradycardia alone. Children tolerate brainstem compression produced by herniation better than adults.
Immediate early intervention can result in recovery. Intervention at the stage of unilateral pupillary dysfunction is likely to have a better prognosis than intervention at the stage of bilateral pupillary dysfunction, decerebrate posturing and bradycardia. CT scan shows obliteration of the suprasellar and quadrigeminal cisterns. Later findings include infarcts andbrainstem hemorrhage.

View transtentorial herniation.

The suprasellar cistern (left image) is obliterated. The quadrigeminal cistern is very compressed andpushed posteriorly (center image). A subdural hematoma with a midline shift is noted. There is central transtentorial and subfalcine herniation.

3. Tonsillar herniation:
   In tonsillar herniation (rare), a mass effect in the posterior fossa causes the cerebellar tonsils to herniate inferiorly through the foramen magnum compressing the medulla and upper cervical spinal cord. Conscious patients complain of neck pain and vomiting. They may have nystagmus, pupillary dilatation, bradycardia, hypertension and respiratory depression. Early tonsillar herniation is difficult to recognize in an unconscious patient. It may not be evident on CT scan since axial views cannot see the pathology well. It is best seen on sagittal MRI. Clinically changes in vital signs may be the only clinical clue in an unconscious patient.

4. Subfalcine herniation (cingulate herniation):
   A unilateral supratentorial mass or hemorrhage results in a midline shift. If the pressure pushing the brain to one side is great enough, one of the hemispheres is pushed under the falx (subfalcine). This may compress the anterior cerebral artery. There is ipsilateral lateral ventricle compression and contralateral lateral ventricle dilation (due to obstruction of the foramen of Monroe).

View subfalcine herniation.
The suprasellar cistern (left image) is obliterated. The quadrigeminal cistern is very compressed and pushed posteriorly (center image). A subdural hematoma with a midline shift is noted. There is central transtentorial and subfalcine herniation.

5. Superior vermian herniation:
   Also called ascending transtentorial herniation, this involves upward herniation of the vermis and cerebellar hemispheres through the tentorial incisura due to a mass effect in the posterior fossa. There is effacement of the quadrigeminal cistern. There is hydrocephalus due to compression of the aqueduct of Sylvius.

References


