Acquired Intracranial Herniations: MR Imaging Findings

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Many of the pathologic processes that increase intracerebral mass may eventually cause brain herniation. It is important to recognize brain herniation, as it can often produce the presenting clinical signs and symptoms and is often the cause of serious neurologic sequelae or death.

Normal Anatomy

Patterns of herniation can be classified according to which anatomic boundary the herniating structure crosses. The midline falx cerebri divides the supratentorial compartment into right and left hemispheres (Fig. 1). It is a rigid structure that extends downward from the calvaria toward the corpus callosum and ends as a free margin. The falx extends from the crista galli anteriorly to the confluence of the tentorial leaves at the straight sinus posteriorly. The distance between the falx and the corpus callosum decreases posteriorly. Anteriorly, the free edge lies in front of the cingulate sulcus. In passing posteriorly, the free edge crosses the cingulate sulcus and gyrus to become closely related to the splenium of the corpus callosum. The pericallosal branch of the anterior cerebral artery courses in the pericallosal sulcus between the corpus callosum and the cingulate gyrus.

The tentorium cerebelli divides the intracranial contents into supratentorial and infratentorial compartments (Fig. 1). It attaches to the superior border of the petrous bone and to the anterior and posterior clinoids. Its free edge extends posteriorly and superiorly to end at the level of the splenium, where it meets the falx cerebri. The midbrain is located in the tentorial opening (incisura). The uncus and parahippocampal regions of the temporal lobe lie along the lateral margins of the tentorial incisura.

Subfalcine Herniation

With subfalcine herniation, the ipsilateral cingulate gyrus is pushed down and under the rigid midline falx, and the contralateral cingulate gyrus is compressed by the herniated tissue (Fig. 2). This process is accompanied by depression of the ipsilateral corpus callosum and elevation or compression of the contralateral corpus callosum [1]. With mild subfalcine herniation, the lateral ventricle is compressed and the anterior falx deviated. Severe herniation will push the ipsilateral lateral ventricle to the opposite side. CT and MR imaging can demonstrate the different degrees of subfalcine herniation (Fig. 3). Owing to arterial compression, there may be focal necrosis of the cingulate gyrus or extensive infarction involving the anterior cerebral artery territory.
Fig. 1.—Normal anatomy.
A, Coronal T2-weighted image at level of sella shows normal corpus callosum (asterisk) and midline faix (straight arrow). Cingulate gyrus (curved arrow) and flow void of pericallosal artery (arrowhead) are seen.
B, Coronal T2-weighted image at level of cerebellum shows normal appearance of tentorial leaves (arrows). Note lateral attachment along petrous ridges.
C, Axial T1-weighted image at level of midbrain. The uncus (small asterisk) and parahippocampal gyrus (large asterisk) are seen.

Fig. 2.—Subfalcine herniation.
A, Coronal pathologic specimen of brain of patient with right subdural hematoma (curved arrows). Ipsilateral cingulate gyrus is displaced beneath faix with depression of corpus callosum. There is compression of contralateral cingulate gyrus (straight arrow) and corpus callosum.
B, Coronal contrast-enhanced T1-weighted image of patient with metastatic breast cancer. Coronal MR imaging accurately shows ipsilateral cingulate gyrus (asterisk) displaced beneath faix with depression of corpus callosum. There is compression of contralateral cingulate gyrus (straight arrow) and corpus callosum. Pericallosal artery (curved arrow) is displaced beneath faix. Lateral and third ventricles are also displaced.

Fig. 3.—Degrees of subfalcine herniation.
A, Axial CT scan of right parietal posttraumatic hemorrhage and subdural hematoma (arrowheads). Some compression of right lateral ventricle is present, with shift to left associated with mild deviation of anterior faix (arrow).
B, Axial CT scan of massive right infarct. Right lateral ventricle is compressed and markedly displaced to opposite side. Note that posterior faix remains nondisplaced despite massive shift including anterior faix (arrow).
Transalar Herniation

Descending transalar herniation occurs from frontal lobe mass effect, which displaces the posterior aspect of the orbital surface of the frontal lobe posteriorly and inferiorly over the sphenoid wing (Fig. 4). Small herniations involve only the orbital gyri, whereas larger herniations may include the gyrus rectus. Ascending transalar herniation is produced by middle cranial fossa mass effect with displacement of the temporal lobe superiorly and anteriorly across the sphenoid ridge (Fig. 5). Posterior displacement of the frontal lobe can cause compression of the middle cerebral artery against the sphenoid ridge, resulting in a middle cerebral artery infarction. Superior displacement of the temporal lobe can compress the suprachiasmatic internal carotid artery against the anterior clinoid process and result in infarction of the anterior and middle cerebral artery territories.

Transtentorial Herniation

Similar to transalar herniation, transtentorial herniation may be descending or ascending.

Descending transtentorial herniation can be unilateral and involve only the uncus (anterior), in which case the mass effect is centered close to the tentorial notch. The herniating brain is forced medially and inferiorly beneath the tentorium, into the perimesencephalic cistern, and compresses the ipsilateral cerebral peduncle as it crosses the incisura [2] (Figs. 6 and 7). Descending transtentorial herniation can also be bilateral (or central) and involve the parahippocampal gyrus, lingual gyrus, and isthmus of the gyrus fimbriatus (posterior). In these cases, the mass effect is far removed from the tentorial notch (Fig. 8). In general, transtentorial herniation is most often complete (anterior and posterior).

The clinical and pathologic consequences of transtentorial herniation are important to recognize [1]. Paralysis of the third nerve can occur from its compression against the tentorial edge by the uncus, resulting in a lateralized fixed and dilated pupil. Focal necrosis or infarction of the uncus and parahippocampal gyrus may occur as a result of arterial compression (Fig. 9A). Temporal or occipital lobe infarction can occur by compression of the calcarine branch of the posterior cerebral artery against the free edge of the tentorium.

Fig. 4.—Descending transalar herniation. A, Coronal enhanced T1-weighted MR image shows left frontal lobe glioblastoma. When compared with normal right side, medial orbital gyrus of left frontal lobe is displaced over sphenoid ridge into middle cranial fossa (arrow). B, Sagittal enhanced T1-weighted MR image shows tumor and resultant transalar herniation (arrow). Left sylvian fissure is effaced. Compare this image with normal right side. C, Sagittal enhanced T1-weighted MR image shows normal position of right frontal lobe (asterisk) above sylvian fissure (arrow).

Fig. 5.—Ascending transalar herniation. Axial contrast-enhanced CT scan of patient with right temporal lobe glioblastoma shows contrast-filled right middle cerebral artery (arrow) displaced anteriorly. Also present is asymmetry and effacement of ipsilateral sylvian fissure. These changes indicate ascending transalar herniation.
Fig. 6.—Descending transtentorial herniation. 
A–C, Axial diagram (A), axial CT scan (B), and axial T2-weighted MR image (C) show components of left unilateral descending transtentorial herniation (straight arrows). Brainstem is rotated and displaced to opposite side and caudally, producing widening of ipsilateral ambient cistern (curved arrows). Compression of neck of contralateral temporal horn results in its dilatation (asterisks).

Fig. 7.—Descending transtentorial herniation. 
A and B, Coronal diagram (A) and coronal T1-weighted MR image (B) show components of left unilateral descending transtentorial herniation (curved arrows). MR image shows extent of herniation across tentorium and deviation of brainstem.

Obstructive hydrocephalus may result from compression of the aqueduct of Sylvius.

As the midbrain is displaced, the contralateral cerebral peduncle is forced against the tentorium, resulting in ischemia of the corticospinal and corticobulbar pathways [1]. This contralateral brainstem compression, involving the uncrossed descending tracts, results in paresis or paralysis ipsilateral to the mass effect. The notching of the midbrain produced by compression against the tentorium is known as Kernohan's notch [1] (Fig. 9B). As the midbrain continues to descend through the incisura, venous congestion occurs, and stretched perforating arterial channels within the midbrain and pons begin to shear and tear, resulting in hemorrhages, often referred to as Duret's hemorrhages [3] (Fig. 10). Irreversible coma or death is the usual result because of hemorrhage within the respiratory and cardiac centers of the reticular substance.

Ascending transtentorial herniation is caused by expanding lesions in the posterior fossa. In most cases, ascending
Fig. 9.—Complications of transtentorial herniation.
A, Uncal infarct. Coronal pathologic specimen shows presence of bilateral uncal necrosis due to pressure against tentorium (curved arrows).
B, Kernohan's notch. Gross coronal pathologic specimen demonstrates Kernohan's notch (arrow) resulting from compression along tentorium.

Fig. 10.—Complications of transtentorial herniation. Midbrain (Duret's) hemorrhage.
A, Axial CT scan of patient with head trauma reveals left transtentorial herniation with deformity of brainstem. Midline hemorrhage is seen within upper pons (arrow) caused by herniation.
B, Axial pathologic specimen of midbrain in different patient demonstrates typical appearance of Duret's hemorrhages. These are most often small and multiple, occurring in midline of midbrain or tegmentum and upper pons.

Transtentorial herniation is symmetric and results in symmetric obliteration of the perimesencephalic cisterns (Fig. 11). In unilateral ascending transtentorial herniation, the upper surface of the cerebellar hemisphere extends across the posterior edge of the tentorial notch [4] (Fig. 12). As with descending transtentorial herniation, ascending transtentorial herniation can compress the posterior cerebral or superior cerebellar arteries against the tentorium, resulting in infarctions, or it can compress the aqueduct of Sylvius, resulting in hydrocephalus.

Tonsillar Herniation
Caudal herniation of the cerebellar tonsils into the foramen magnum is most commonly caused by an infratentorial mass but may also occur as a result of an expanding supratentorial mass. Compressed cerebellar folia extend into the upper cervical spinal canal for a variable distance [4] (Fig. 13). In general, cerebellar tissue identified more than 5 mm below the
foramen magnum is considered abnormal [5]. Compression of the posterior inferior cerebellar artery by the herniated tonsils can lead to cerebellar infarcts.

**Postsurgical or Posttraumatic Defect Herniation (Fungus Cerebri)**

This is a rare form of herniation most commonly occurring in uncontrolled situations or with massive trauma. Brain tissue will move from an area of high pressure to an area of low pressure by following a path of least resistance. Surgery or trauma can create calvarial defects, providing an easy pathway through which swollen brain tissue may herniate [1] (Fig. 14).

MR imaging and CT easily demonstrate the defect and the amount of herniating brain tissue (Fig. 15). MR imaging has greatly advanced detection of small herniations, especially in postoperative or posttraumatic sites involving the orbit, sella, and middle ear canal [6] (Fig. 16).
Fungus cerebri.

A, Axial CT scan of patient with chronic head injury shows left hemispheric encephalomalacia and dilated lateral ventricle. Brain tissue is identified herniating through calvarial defect.

B, Axial T1-weighted MR image of patient with severe head trauma. Swollen brain is seen herniating through calvarial defect (arrows outline margins of defect). Hemorrhage is seen adjacent to defect’s anterior rim, which may be result of herniation or initial injury.

Summary

Acquired intracranial herniations can accompany many pathologic processes of the brain. Herniations are important to recognize because they are frequently responsible for the presenting clinical signs and symptoms and can often be the cause of serious neurologic sequelae or death.

REFERENCES


