IMAGING OF ACQUIRED CEREBRAL HERNIATIONS

The cranium provides protection for the brain against external forces. The brain is suspended in the cerebrospinal fluid (CSF) and supported by the skull and dural septa, which divide the cranial cavity into compartments and prevent excessive movement of the brain [1]. The unyielding cranium is a closed system and hence there is a fixed volume for the brain, CSF, and blood vessels and very little room for expansion in the face of increased intracranial volume and pressure. The brain is essentially incompressible (its shape can be altered, but its volume is basically constant); therefore, the vascular and CSF spaces must compensate for alterations in intracranial volume because of mass lesions or cerebral edema [2,3]. Unfortunately, this capacity is very limited, and when it is exceeded, cerebral displacement and herniation result. Cerebral herniation is the displacement of brain tissue from one compartment to the other. Of course, there are numerous variables that influence the degree of herniation and its untoward neurologic effects, such as the location of the mass, the volume of the mass, the intracranial pressure, the rate at which alterations in pressure and volume develop, brain atrophy, and individual anatomical variations.

There are several different types of cerebral herniation and these are defined by the anatomic boundary through which the herniating tissue traverses [1,4]. Subfalcine, transtentorial, and tonsillar herniation are well known. Transtentorial herniation can be further classified as central (ascending and descending) and lateral (anterior and posterior).

Transphenoidal and external hernias are less well-known patterns of herniation. A thorough understanding of the anatomy of the brain, skull, and total septa is imperative to understanding cerebral displacement and herniation [5]. Recognition of cerebral displacement and herniation is critical, because the clinical consequences are devastating. Cerebral herniation causes compression of the brain, cranial nerves, and blood vessels and results in serious neurologic morbidity and even death.

ANATOMY

The cranial cavity is incompletely divided into fossae by bony ridges and further divided by dural septa. The base of the skull provides support for the brain and consists of three fossae: anterior, middle and posterior, which are separated by bony ridges. The foramen magnum is a large orifice in the floor of the posterior fossa that allows passage of the spinal cord, nerves and blood vessels. Although there are numerous skull base foramina, the foramen magnum is the only opening of any significant size.

The dural septa represent reflections of the dura mater that further divide the intracranial cavity. Dura is the Latin word for hard, an accurate description of this tough, fibrous membrane. The two main dural reflections are the falx cerebri, which separates the cerebral hemispheres, and the tentorium cerebelli, which separates the posterior fossa from the cerebral hemispheres defining the supratentorial and infratentorial compartments. The falx cerebella is a smaller dural reflection, which partially separates the cerebellar hemispheres.
Figure 1. Patterns of cerebral herniation. (A) Coronal, (B) axial, and (C) sagittal diagrams demonstrate the different types of cerebral herniation: (1) Subfalcine, (2) uncal, (3) descending transtentorial, (4) tonsillar, (5) ascending transtentorial, and (6) external herniation.

Figure 2. Patterns of cerebral herniation.

The falx cerebri is a sickle-shaped septum that attaches anteriorly to the crista galli, and posteriorly to the internal occipital protuberance and tentorium cerebella. The anterior part of the falx cerebri has a relatively narrow sagittal width, whereas the posterior part of the falx cerebri has a broad sagittal width where it attaches to the tentorium cerebella and contains the straight sinus [6,7]. Superiorly, the falx cerebri attaches to the inner table of the skull in the midline and contains the superior sagittal sinus, whereas the inferior free edge of the falx cerebri contains the inferior sagittal sinus [6]. The corpus callosum is located immediately inferior to the free edge of the falx and the distance between the two decreases posteriorly. The cingulate gyros is just lateral to the inferior aspect of the falx. The pericallosal artery, a branch of the anterior cerebral artery, runs in the pericallosal sulcus between the corpus callosum and cingulate gyrus [6].
The tentorium cerebella is a transverse dural septum that separates the posterior fossa from the cerebral hemispheres. The tentorium, as its name suggests, is shaped like a tent, with its apex suspended in the midline by the posterior inferior falx cerebri, and the base attached to the inner surface of the occipital bone and upper border of the petrous temporal bone [7]. The transverse sinuses and superior petrosal sinuses are contained within these basal dural attachments. The opening of the tentorium is the tentorial incisura, also known as the hiatus or notch. The incisura has a semioval shape and is attached at its base to the clinoid processes and at its apex to the falx cerebri [6,7]. Otherwise, the margins of the incisura are free, though quite rigid. The midbrain and cerebral peduncles pass through the incisura.

The anatomy of the tentorial incisura and adjacent structures is complex. The uncus and hippocampus of the medial temporal lobe slightly overhang the incisura. The posterior cerebral arteries, anterior choroidal arteries, and internal cerebral veins pass around the midbrain in the ambient cistern in close proximity to the free edge of the tentorium. The oculomotor nerve exits the midbrain and crosses the interpeduncular cistern between the posterior cerebral and superior cerebellar arteries, and courses just medial to the uncus on its way to the cavernous sinus. Both computed tomography (CT) and magnetic resonance (MR) imaging demonstrate the anatomy of the cranial cavity and enable diagnosis of cerebral herniation, although MR imaging provides superior anatomic detail.

**TYPES OF ACQUIRED HERNIATIONS**

- **Subfalcine herniation**
Subfalcine herniation, also known as midline shift or cingulate herniation, is the most common type of cerebral herniation and is easily recognized on CT and MR imaging. It is caused by unilateral frontal, parietal, or temporal lobe mass effect or edema [1]. When subfalcine herniation occurs, the anterior falx, although rigid, will tilt away from the mass effect. The posterior falx is wider, more rigid, and more resistant to displacement. This explains why subfalcine herniations occur anteriorly. As the mass effect increases, the ipsilateral cingulate gyros will be displaced beneath the free edge of the falx cerebri, pushing down the ipsilateral corpus callosum. With further progression, the contralateral cingulate gyrus and contralateral corpus callosum are compressed [2]. These findings are best demonstrated on coronal MR imaging. When subfalcine herniation becomes severe, both foramina of Monro become compressed and obstructed. This results in dilatation of the contralateral lateral ventricle, but the ipsilateral ventricle usually remains compressed because of mass effect. A wedge of pressure necrosis may occur where the cingulate gyros is squeezed against the falx [2]. Also, the pericallosal artery may be pressed against the unyielding falx, resulting in occlusion and brain infarction [8,9].

![Subfalcine herniation (arrows). Subfalcial herniation is displacement of the cingulate gyrus from one hemisphere to the other, under the falx cerebri. Subfalcial herniation can compress the pericallosal arteries, causing an infarct in their distribution.](image1)

![Subfalcine herniation. Coronal enhanced T1-weighted MR image demonstrates a large enhancing mass (large arrow) within the left frontal lobe causing subfalcine herniation. The cingulate gyrus (outline arrow) is displaced beneath the falx cerebri (small black arrow) and there is downward displacement of the corpus callosum (circle).](image2)

Quantification of the degree of midline shift is most easily done by measuring the degree of displacement of the septum pellucidum from the midline, on axial images with either CT or MRI. The midline is usually determined by halving the distance between the inner tables at the level of the septum pellucidum. The degree of septal shift, not surprisingly, is predictive of patient prognosis [10-12]. Ross et al [12] reported that greater midline shift on CT scans correlated with a significantly lower likelihood of recovery in patients with acute intracranial hematomas. All patients in their series with a septal shift over 15 mm had a poor outcome. Conversely, no patient with septal shift less than 5 mm shift had a poor outcome. Pineal shift and aqueductal shift appear to have less predictive value [12].
Figure 7. Subfalcine herniation. Axial CT scan reveals a large acute subdural hematoma (large arrow) causing subfalcine herniation. Note the displacement of the anterior falk cerebrei (small arrow) and extensive midline shift. The ipsilateral ventricle is compressed due to mass effect, but the atrium of the contralateral lateral ventricle (outlined arrow) is dilated due to obstruction of the foramen of monro.

Figure 8. This is a trauma case where a contusion of the inferior temporal lobe (blue arrow) has resulted in diffuse hemispheric edema. Note the compressed and flattened gyri on the right. This has resulted in subfalcine herniation of the cingulate gyrus, with a secondary hemorrhagic infarction above that (black arrow). A midline shift from right to left is also present, as is uncal herniation.

- **Transtentorial herniation**

  Transtentorial herniation occurs when brain tissue is displaced into the tentorial notch, and is perhaps the most important herniation phenomenon in terms of frequency and clinical consequences. Transtentorial herniation is a complex process that can be loosely classified into overlapping patterns. Lateral transtentorial herniation can be anterior (uncal) or posterior (parahippocampal); whereas central transtentorial herniation can be either ascending or descending. Only a few millimeters of space are present between the midbrain and rigid tentorial edge, and the oculomotor nerve and posterior cerebral artery are in close proximity [5,13]. Consequently, very little displacement can be tolerated, and a shift of a few millimeters will result in compromise of these vital structures, with serious neurologic sequelae.

- **Uncal herniation**
Uncal herniation represents anterior lateral transtentorial herniation. It is perhaps the best-known type of transtentorial herniation and is readily identified on both CT and MR. Uncal herniation typically occurs from a unilateral, expanding supratentorial lesion, especially in the middle cranial fossa. In this pattern of herniation, the uncus is displaced over the free edge of the tentorium [14]. Consequently, the midbrain is displaced and the opposite cerebral peduncle is squeezed against the contralateral tentorial edge. This damages the descending corticospinal and corticobulbar tracts, resulting in paralysis on the same side of the supratentorial mass. This "false-localizing" sign occasionally resulted in surgery on the wrong side of the brain in the days before CT [15]. The groove where the midbrain is compressed against the contralateral tentorial edge is known as Kemohan's notch after the author who first described it [16]. Mass effect on the ipsilateral cerebral peduncle can also lead to a contralateral hemiparesis. As herniation progresses, the midbrain is further compressed, narrowing it in the transverse dimension, which can affect the ascending reticular activating system and result in progressive loss of consciousness. Compression of the oculomotor nerve results in ipsilateral third nerve palsy ("blown pupil"). If the posterior cerebral artery is compressed, infarction of the occipital lobe ensues and produces homonymous hemianopsia, though by this time the patient is usually unconscious (2,17). Rarely, the anterior choroidal and superior cerebellar arteries may be compromised, resulting in infarction [5].
Uncal herniation is well demonstrated on both CT and MRI [18-20]. Early on there is displacement of the uncus, resulting in effacement of the lateral suprasellar cistern. As herniation progresses, there is widening of the ipsilateral ambient and lateral Pontine cistern, with displacement and rotation of the brainstem. With more advanced herniation, there is obliteration of the cisternal spaces, and the midbrain becomes compressed and elongated in the anteroposterior dimension.

- **Posterior (parahippocampal) tentorial herniation**

In patients with occipital and posterior temporal masses, the herniation of the medial temporal lobe occurs more posteriorly. Consequently, the herniating hippocampus impinges less upon the cerebral peduncle, but more on the tectum at the level of the superior colliculus [21]. This may result in Parinaud’s syndrome, with paralysis of upward conjugate gaze. There is also relatively less compression of the oculomotor nerve and posterior cerebral artery. However, as herniation becomes more severe, these structures will be compromised. Early on, MR and CT will demonstrate effacement of the ipsilateral quadrigeminal plate and ambient cisterns, while there is usually no effacement of the lateral suprasellar cistern. Compression of the tectum and rotation of the midbrain can be seen as herniation progresses, and finally the cisternal spaces will be obliterated and the midbrain displaced and deformed. Compared with uncal herniation, this pattern of herniation appears to be much less commonly recognized as an isolated clinical presentation. In many cases there is overlap of uncal and hippocampal herniation.

**Figure 12.** Uncal and hippocampal herniation. Axial T2-weighted MR image of the brain demonstrates uncal and hippocampal herniation due to multiple abscesses in the right temporal and occipital lobes. The largest abscess is in the occipital lobe resulting in parahippocampal herniation (black arrow) and compression of the right midbrain tegmentum (outlined arrow).

- **Descending transtentorial herniation**

Descending transtentorial herniation consists of caudal descent of brain tissue through the tentorial incisura, and occurs mainly in response to mass effect in the frontal, parietal, and occipital lobes [1]. Since intracranial masses are rarely in the midline, there is considerable overlap of horizontal and vertical shifts during transtentorial herniation; such that descent of the diencephalon, midbrain, and pons tends to occur simultaneously with uncal and hippocampal herniation. Furthermore, with unilateral lesions, subfalcine...
herniation commonly precedes transtentorial herniation; whereas midline masses, bilateral supratentorial masses, or supratentorial hydrocephalus cause transtentorial descent without midline shift [1].

As descending transtentorial herniation progresses, there is further downward displacement of brain tissue, with disastrous consequences. Damage is caused by local pressure from crowding of tissue within the incisura, which can lead to compromise of the third cranial nerve, posterior cerebral artery, and midbrain, and there are also traction forces wreaking havoc. The lower brainstem is less mobile because of the upper cervical dentate ligaments. The longitudinal compression of the upper brainstem against the unyielding medulla leads to buckling of the brainstem and further compression of the midbrain tegmentum [22]. Downward displacement of the brainstem also produces traction on the oculomotor nerve, causing third nerve palsy. Stretching and shearing of the perforating branches of the basilar artery occur, resulting in ischemia and hemorrhage within the brainstem, called Duret's hemorrhage [23,24]. This occurrence is a late finding and portends a poor prognosis, usually death [22,25]. Hydrocephalus develops because of distortion and obstruction of the cerebral aqueduct, further increasing the volume and pressure of supratentorial contents. Stovring pointed out that preferential dilatation of the contralateral temporal horn tends to occur, because it is relatively shielded within the middle cranial fossa from compression by the supratentorial mass [26]. There also is a propensity for dilatation of the contralateral occipital horn because of relative shielding by the rigid posterior falx cerebri. For the same reason, preferential dilatation of the contralateral temporal horn and occipital horn may also occur with subfalcine herniation, although the cause of the hydrocephalus is the result of obstruction of the foramen of Monro, rather than the aqueduct. Further descent can result in tonsillar herniation, with compression and distortion of the medulla oblongata causing cardiorespiratory compromise. It has been well established clinically that patients who decompensate from descending transtentorial herniation do so in an orderly rostral to caudal fashion of diencephalic and brainstem dysfunction [27]. The clinical consequences of progressive central herniation are oculomotor paresis, progressive alteration of consciousness, decerebrate rigidity, coma, and death.

Figure 13. Duret’s hemorrhage. Axial CT scan of the head in a patient who had a large traumatic acute subdural hematoma (not shown) and unilateral descending transtentorial herniation reveals a hemorrhage (arrowhead) within the dorsal brainstem consistent with a Duret's hemorrhage.

Figure 14. Postmortem specimens showing hemorrhage (arrowhead) within the dorsal brainstem consistent with a Duret's hemorrhage. The so-called Duret hemorrhages seen here in the pons are secondary to downward compression that leads to stretching, ischemia and rupture of perforating arterioles and brain stem hemorrhage.
Effacement of the cisternal spaces, caudal displacement of the basilar artery and pineal gland, deformity and displacement of the midbrain, hydrocephalus, and infarction of the posterior cerebral artery have all been described on CT as findings associated with descending transtentorial herniation [17,28-30]. Most of these findings are indirect, secondary, and late manifestations of descending transtentorial herniation. Effacement of the perimesencephalic cisterns, though qualitative, is probably the most useful and consistent finding on CT. With its better soft tissue definition and multiplanar imaging, MR provides superior anatomical detail of the tentorial incisura and adjacent structures. MRI findings of descending transtentorial herniation include downward descent of the brainstem, effacement of the basal cisterns, flattening of thepons against the clivus, and inferoposterior displacement of the quadrigeminal plate. As herniation progresses, deformity and buckling of the brainstem occur. Hydrocephalus develops because of aqueductal obstruction.

Numerous authors have attempted to quantitate the degree of downward shift on MR images [19,31,32]. Ropper et al measured horizontal and vertical components of brain displacement on coronal MR images in 10 patients with supratentorial masses [33]. Feldman et al compared vertical brainstem position on 50 images from normal patients and 21 images from 15 patients with large supratentorial masses [31]. The methods of both Ropper and Feldman are cumbersome to employ in the clinical setting, and neither study demonstrated significant correlation between measured vertical descent and neurologic signs. Reich et al measured the relationship of the iter (proximal opening of the aqueduct of Sylvius) to the incisural line on mid-sagittal MR images [19]. The incisural line was defined as a straight line from the anterior tuberculum sellae-to the inferior point of the confluent of the straight sinus, the vein of Galen, and the inferior sagittal sinus. In 123 normal patients, the iter was 0.2 ± .8 mm below the incisural line. Displacement of the iter by more than two standard deviations (1.8 mm) below the incisural line was considered as MRI criterion for descending transtentorial herniation. Eighteen patients with supratentorial masses demonstrated downward displacement of the iter ranging from 2.0 mm to 1.0 mm, which correlated well with clinical signs of acute brain herniation, but less so with chronic cases [19]; however, in clinical practice, qualitative assessment of downward shift on MRI scans is probably more widely applied, though measurement of displacement of the iter relative to the incisural line can be useful.

- **Ascending transtentorial herniation**
Just as a supratentorial masses may cause descent of brain tissue through the hiatus, an infratentorial mass may cause brain tissue to ascend through the hiatus [30,34,35]. Ascending transtentorial herniation has been most frequently reported in association with a cerebellar mass or a hydrocephalic ("trapped") fourth ventricle. Cerebellopontine angle masses and intrinsic masses of the pons have also been reported, albeit much less commonly [34,36,37]. The propensity for ascending tentorial herniation to occur is more likely when the mass is near the incisura (ie, superior cerebellar vermis), when shunting of the lateral ventricle reduces hydrocephalus and pressure above, and when the opening of the incisura is large [34]. MR and CT findings of ascending transtentorial herniation include effacement of the superior cerebellar cistern, superior displacement of the superior vermis through the incisura, compression of the midbrain, and forward displacement of the pons against the clivus [31,34]. Ascending tentorial herniation can compress the posterior cerebral artery or superior cerebellar arteries against the tentorium, resulting in infarctions, or it can compress the aqueduct of Sylvius, resulting in hydrocephalus. Obstruction of venous outflow by compression of the vein of Galen and basal vein of Rosenthal may occur and further increase intracranial pressure [38,39].

Figure 17. Ascending transtentorial herniation. (A) Axial T1-weighted MR image and (B) sagittal enhanced T1-weighted MR image demonstrate ascending transtentorial herniation in a patient with a cerebellar lung metastases. Note the upward displacement of the superior cerebellar vermis (black arrows) through the incisura, compression of the fourth ventricle, and anterior displacement of the pons (arrowhead) against the clivus.

- **Tonsillar (foramen magnum) herniation**

  Tonsillar herniation is defined as inferior displacement of the cerebellar tonsils through the foramen magnum into the cervical spinal canal. This is most commonly caused by a posterior fossa mass, but may also be caused by a supratentorial mass that causes downward transtentorial herniation [40]. In fact, tonsillar herniation occurs in association with descending transtentorial herniation anywhere from 20 to 50% of the time [14,19,41,42]. The propensity of tonsillar herniation to follow descending tentorial herniation is related to the size and shape of the incisura. If the incisura is small, the patient will be less likely to have tonsillar herniation.

Figure 18. Tonsillar (foramen magnum) herniation. B, Here a head injury has resulted in tonsillar herniation of the cerebellum. The arrows outline the necrotic cerebellar tissue which is overlying the medulla. There is also subarachnoid hemorrhage on the left cerebellar hemisphere.

When tonsillar herniation progresses, pressure necrosis may develop where the tonsil is squeezed against the unyielding foramen magnum. Compression of the medulla oblongata by the displaced cerebellar tonsils may lead to loss of consciousness secondary to involvement of the ascending reticular activating system. Further progression of tonsillar herniation may damage cardiac and respiratory centers of the brainstem and even result in death. Additionally, occlusion of the posterior inferior cerebellar artery between the displaced tonsils and foramen magnum may result in cerebellar infarction.

Obstruction of the outlet foramina of the fourth ventricle causes hydrocephalus, further increasing intracranial pressure within the posterior fossa and worsening the herniation.
Figure 19. Tonsillar herniation. Sagittal enhanced T1-weighted MR image demonstrates a large enhancing cerebellar mass causing both tonsillar herniation and ascending transtentorial herniation. Note the inferior displacement of the tonsils (arrow) below the foramen magnum and the effacement of the surrounding CSF spaces. The brainstem is compressed and displaced against the clivus and there is upward displacement of the superior cerebellar vermis (arrowhead) through the incisura.

There are large variations in the anatomy of the foramen magnum and the inferior cerebellum [2]. In a group of 200 normal patients, Barkovich et al demonstrated that the position of the tonsils ranged from 8 mm above the foramen magnum to 5 mm below the foramen magnum [43]. Also, Aboulez et al reported the average distance of the tonsillar tips from the foramen magnum was 2.9 ± 3.4 mm above the foramen magnum in 82 normal patients [44]. They concluded that extension of the tonsils below the foramen magnum is considered normal up to 3 mm, borderline between 3 mm and 5 mm, and clearly pathological when it exceeds 5 mm. Perhaps more important than strict quantitative criteria of tonsillar displacement are the morphologic appearance of the brainstem and the surrounding CSF spaces. When the tonsils extend below the foramen magnum, anterior displacement of the lower brainstem and loss of CSF space surrounding the brainstem are important in distinguishing tonsillar herniation from normal variations [45].

Tonsillar herniation can be difficult to assess on axial CT because of beam hardening artifacts at the skull base and partial volume average effects. MR imaging is the study of choice and sagittal images best delineate the relationship of the tonsils to the foramen magnum. The foramen magnum can be identified on sagittal MR images by drawing a line from the inferior tip of the clivus (basin) to the posterior lip of the foramen magnum (opisthion). Identification of the tonsils below the foramen magnum, anterior brainstem displacement, and loss of CSF surrounding the brainstem indicate tonsillar herniation [46]. Of course, chronic tonsillar herniation, such as occurs with Chiari malformations, must be distinguished from acquired tonsillar herniation. Fortunately, the clinical history and imaging are characteristic and rarely cause diagnostic difficulty. The exception may be the presence of a posterior fossa mass with a pre-existing Chiari I malformation.

- **Transphenoidal (transalar) herniation**

Transphenoidal herniation is less well known, but occurs when brain tissue is displaced across the superior sphenoid alae, which incompletely separate the anterior and middle cranial fossae [2]. Areas of necrosis and hemorrhage may develop where the brain is displaced against the bony sphenoid ridge. There are two types of transphenoidal herniation: descending and ascending. Descending transphenoidal herniation occurs when anterior cranial fossa mass effect causes displacement of the posterior frontal lobe over the sphenoid wing into the middle cranial fossa. Ascending transphenoidal herniation is produced by middle cranial fossa mass effect, which causes displacement of the anterior temporal lobe over the sphenoid ridge into the anterior cranial fossa. The middle cerebral artery can become compressed between the displaced brain and the sphenoid ridge, resulting in middle cerebral artery infarction [4] Although vascular compromise may occur with transphenoidal herniation, it is rare. Moreover, the clinical features of this type of herniation are poorly defined.

Figure 20. Transphenoidal herniation. Sagittal enhanced T1-weighted MR image demonstrates a large ring enhancing mass (arrowhead) in the right frontal lobe causing descending transphenoidal herniation (arrow)

- **External herniation**

External herniations, also known as fungus cerebri, are the rarest of acquired herniations [45]. They are most frequently caused by post surgical and posttraumatic defects that allow swollen or displaced brain to pass through. This type of herniation may be beneficial by relieving intracranial pressure and thereby preventing more devastating complications due to internal herniation; however, external
exposure of the brain can result in infectious complications, and many of these patients have a poor prognosis because of the severity of their underlying illness. Both CT and MR imaging are effective at demonstrating these herniations. MR imaging, however, is clearly superior at defining soft tissue, which can be critical in certain clinical scenarios. Distinguishing brain tissue from other tissues such as granulation tissue, cholesteatoma, tumor, or other lesions involving defects of the skull is essential, and MR imaging accomplishes this well [4,47]. MR imaging is also superior for evaluation of congenital encephaloceles, which are essentially congenital external hernias.

Figure 21. (A) Axial CT scan and (B) axial T1 weighted MR image of the head demonstrate a large right frontal external hernia in a patient who underwent a wide scalp excision, craniectomy, and free flap for an infiltrative squamous cell carcinoma. The flap became infected and failed, and the patient developed encephalitis resulting in external herniation.

- Intracranial hypotension

Intracranial hypotension is a cause of cerebral herniation that should be considered in patients who do not harbor an intracranial mass or edema, or in patients in whom the degree of herniation is out of proportion to the degree of mass effect. Intracranial hypotension is caused by a CSF leak, either iatrogenic or spontaneous. As previously stated, the intracranial cavity is essentially a closed system and the volume is constant. These principles were described as early as the 1780s by Scottish anatomists Alexander Monro and George Kellie in what is known as the Monro Kellie doctrine, which states that intracranial volume is constant and is represented by the summed volumes of brain substance, CSF, and blood [3]. Since brain volume is constant, the volumes of CSF and blood fluctuate reciprocally. This buffering capacity is very limited, but does explain the imaging findings in patients with intracranial hypotension. With a loss in CSF volume, there is a greater increase in blood volume. This results in dural venous hyperemia and pachymeningeal venous engorgement and edema, which can be identified on MR imaging as diffuse pachymeningeal enhancement. Furthermore, the decrease in volume of the suspending CSF results in downward descent of the brain and can cause descending central transtentorial herniation and tonsillar herniation. A blood patch or surgical repair of the dural defect is usually required.

Figure 22. Intracranial hypotension. (A) Coronal enhanced T1-weighted MR image demonstrates diffuse pachymeningeal enhancement (arrows) in a patient with severe postural headaches. (B) Sagittal enhanced T1-weighted MR image reveals tonsillar herniation (arrow), descending transtentorial herniation (note downward descent of the brainstem, loss of surrounding CSF spaces and flattening of the pons against the clivus) and pachymeningeal enhancement (arrowheads). (C) Postmyelogram CT scan of the thoracic spine shows an extradural accumulation of contrast (arrow) within the spinal canal consistent with a CSF leak. The extradural and intradural confluent medium outlines the dura (arrowhead) at the T10 level. A blood patch performed at this level resulted in resolution of the patient's symptoms.

SUMMARY

The consequences of cerebral herniation are compression of the brain, cranial nerves, and blood vessels that may result in serious neurologic morbidity, coma, and even death. A thorough understanding of the various patterns of cerebral herniation is essential, and it is important to remember that many of these patterns of herniation overlap. CT and MR imaging are effective at establishing the diagnosis of cerebral herniation, which will guide important decisions regarding therapeutic options and prognosis.

References


Addendum

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