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Types of Cerebral Herniation and Their Imaging Features

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Abbreviations: CSF = cerebrospinal fluid, DTH = descending transtentorial hernia, ICP = intracranial pressure

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Identify the basic intracranial anatomy necessary for understanding the clinical and radiologic features of brain herniation syndromes.
- Discuss use of a systematic approach to cases of cerebral herniation to make an accurate diagnosis.
- Recognize the main imaging findings in cerebral herniation and integrate them with the clinical manifestations.

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Cerebral herniation, defined as a shift of cerebral tissue from its normal location into an adjacent space, is a life-threatening condition that requires prompt diagnosis. The imaging spectrum can range from subtle changes to clear displacement of brain structures. For radiologists, it is fundamental to be familiar with the different imaging findings of the various subtypes of brain herniation. Brain herniation syndromes are commonly classified on the basis of their location as intracranial and extracranial hernias. Intracranial hernias can be further divided into three types: *(a)* subfalcine hernia; *(b)* transtentorial hernia, which can be ascending or descending (lateral and central); and *(c)* tonsillar hernia. Brain herniation may produce brain damage, compress cranial nerves and vessels causing hemorrhage or ischemia, or obstruct the normal circulation of cerebrospinal fluid, producing hydrocephalus. Owing to its location, each type of hernia may be associated with a specific neurologic syndrome. Knowledge of the clinical manifestations ensures a focused imaging analysis. To make an accurate diagnosis, the authors suggest a six-key-point approach: comprehensive analysis of a detailed history of the patient and results of clinical examination, knowledge of anatomic landmarks, direction of mass effect, recognition of displaced structures, presence of indirect radiologic findings, and possible complications. CT and MRI are the imaging modalities of choice used for establishing a correct diagnosis and guiding therapeutic decisions. They also have important prognostic implications. The preferred imaging modality is CT: the acquisition time is shorter and it is less expensive and more widely available. Patients with brain herniation are generally in critical clinical condition. Making a prompt diagnosis is fundamental for the patient's safety.

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Introduction

Cerebral herniation is a potentially life-threatening condition that needs to be diagnosed promptly. The imaging spectrum can range from subtle changes to clear displacement of brain structures. The radiologist should be able to identify the main imaging features of the brain herniation subtypes.

The skull is a rigid vault-shaped structure containing three main components: brain, cerebrospinal fluid (CSF), and blood. It is compartmentalized by bony landmarks and inelastic dural reflections (1). Given the inflexible nature of the skull, the intracranial volume

TEACHING POINTS

- Cerebral herniation is a potentially life-threatening condition that needs to be diagnosed promptly. The imaging spectrum can range from subtle changes to clear displacement of brain structures.
- Brain edema, tumors, or hemorrhage are causes of cerebral herniation secondary to an increase in volume and intracranial pressure (ICP). A decrease in ICP can also produce herniation, as in paradoxical herniation.
- Brain herniation may cause brain pressure necrosis, compress cranial nerves and vessels causing hemorrhage or ischemia, and obstruct the normal circulation of CSF, producing hydrocephalus. Therefore, each type of hernia may be associated with a specific neurologic syndrome.
- The basal cisterns are spaces filled with CSF and located in the subarachnoid space. They contain the proximal portions of some cranial nerves and basal cerebral arteries. They are in close contact with the main intracranial structures. Basal cisterns are involved in almost any hernia type, making them a key anatomic landmark.
- In subfalcine hernias, the degree of midline shift correlates with the prognosis; less than 5-mm deviation has a good prognosis, whereas a shift of more than 15 mm is related to a poor outcome.

is fixed and there is little room for expansion. As the Monro-Kellie hypothesis states, the sum of volumes of the brain, CSF, and intracranial blood is constant. An increase in the volume of one component will result in a decrease in the volume of one or both of the other components (2).

When there is a change in the intracranial volume that exceeds these compensation mechanisms, brain tissue will be displaced from one compartment into another. It can be through anatomic or acquired spaces. Brain edema, tumors, or hemorrhage are causes of cerebral herniation secondary to an increase in volume and intracranial pressure (ICP). A decrease in ICP can also produce herniation, as in paradoxical herniation (1,3).

Brain herniation can be classified into two broad categories: intracranial and extracranial. Furthermore, intracranial hernias can be subdivided into three basic types: *(a)* subfalcine hernia; *(b)* transtentorial hernia, which can be ascending or descending (lateral and central); and *(c)* tonsillar hernia (Table, Fig 1) (4,5).

Brain herniation may cause brain pressure necrosis, compress cranial nerves and vessels causing hemorrhage or ischemia, and obstruct the normal circulation of CSF, producing hydrocephalus. Therefore, each type of hernia may be associated with a specific neurologic syndrome. Knowledge of the clinical manifestations ensures a focused imaging analysis.

The most useful imaging modalities are CT and MRI. In the emergency setting, CT is regularly performed to readily identify a condition that may require surgical intervention (6). MRI findings are analogous to those on CT scans but with better tissue characterization, especially in posterior fossa disease. Anatomic MRI sequences (T1-weighted, T2-weighted, and contrast-enhanced T1-weighted) are the best for evaluation; coronal and sagittal acquisitions are encouraged (7). Historically, angiography was an important diagnostic tool, but this technique is now obsolete (8).

Approach to Diagnosing Brain Herniation Syndromes

We suggest a six-key-point approach to analyze all the clinical and imaging information to make a prompt and accurate diagnosis (Fig 2).

Clinical Information

Clinical information is useful for guiding a detailed analysis of the potentially involved anatomic structures. The patient's history, current clinical scenario, and specific neurologic syndromes should be considered when available.

Anatomic Landmarks

Anatomic landmarks are boundaries used as a reference for the different brain compartments that help determine if a specific brain structure is displaced.

Direction of Mass Effect

If there is any disease that causes mass effect, it is important to establish its location and determine the direction of the vector force it creates. This will point out the brain structures that may be displaced or involved.

Displaced Structure

Identifying the displaced structure is necessary to classify the type of hernia. Knowledge and adequate evaluation of specific anatomic regions that can herniate are fundamental.

Indirect Signs

Sometimes the herniation can be subtle and difficult to identify at first glance. Aside from looking at the specific brain structure that might be displaced, evaluating other potentially involved structures can provide valuable information by showing indirect signs of the herniation.

Herniation-related Complications

Brain herniation may cause different complications secondary to compression of vessels, nerves, and the ventricular system. Stroke of the anterior cerebral artery, posterior cerebral artery, or posterior inferior cerebellar artery occurs owing to vascular compression. Hydrocephalus manifests when

General Features of Main Intracranial Hernias

Type of Hernia	Clinical Information and Neurologic Syndromes	Anatomic Landmarks	Direction of Mass Effect	Displaced Structure(s)	Indirect Signs
Subfalcine	Anterior cerebral artery syndrome	Midline, falx cerebri, cingulate gyrus, CC, and Monro foramen	Medial and anterior, beneath falx	Cingulate gyrus and CC	Dilatation of contralateral ventricle due to compression of contralateral foramen of Monro
Transtentorial descending	Paralysis of third nerve, compression of PCA and choroidal arteries (occipital and medial temporal infarction)	Tentorium, perimesencephalic cisterns	Downward from supratentorial compartment, through tentorial incisura	Anterior: uncus Posterior: parahippocampal gyrus, isthmus of fornical gyrus, and anterior portion of lingual gyrus Central: diencephalon, midbrain, and pons	Displacement, rotation, and elongation of brainstem Anterior or posterior: widening of contralateral ventricular atrium and temporal horn Central: hydrocephalus
Transtentorial ascending	Manifestations of increased ICP, brainstem and cerebellar compression PCA and SCA compression (occipital cerebral and superior cerebellar infarction)	Tentorium, superior cerebellar and quadrigeminal cisterns	Upward from posterior fossa through tentorial incisura	Superior cerebellar hemispheres and vermis, superior and inferior colliculi, midbrain	Obliteration of ipsilateral perimesencephalic and contralateral crural cisterns Anterior displacement of brainstem, hydrocephalus
Tonsillar	Manifestations of brainstem and cerebellar compression PICA compression (posterior inferior cerebellum, inferior cerebellar vermis, and lateral medulla infarction)	Foramen magnum (McRae line) Cerebellar tonsil	Downward through foramen magnum	Cerebellar tonsils Pons Medulla	Effacement of perimedullary CSF through foramen magnum Obliteration of cisterna magna and fourth ventricle Vertical orientation of folia of tonsil

Note.—CC = corpus callosum, PCA = posterior cerebral artery, PICA = posterior inferior cerebellar artery, SCA = superior cerebellar artery.

there is involvement of the foramen of Monro or aqueduct of Sylvius. Cranial nerves may be affected when there is involvement of the brainstem and basal cisterns.

Relevant Anatomy

The cranial cavity is divided by bony landmarks and reflections of the dura mater. The main dural reflections are the falx cerebri and tentorium cerebelli, which divide the cranial cavity into right and left cerebral hemispheres and the posterior fossa, thus defining the supra- and infratentorial compartments (Fig 3) (7,9).

The falx cerebri has an anteroposterior orientation and is attached superiorly to the inside of the skull. Anteriorly, it is fixed to the crista galli; posteriorly, it widens and adheres to the tentorium. Immediately inferior to the free edge of the falx is the corpus callosum and cingulate gyrus. The pericallosal artery runs through the pericallosal sulcus (Fig 4a) (7,10).

The tentorium cerebelli extends inferiorly and laterally from its confluence with the falx (10). It has a U-shaped opening called the tentorial incisura, which provides communication between the supratentorial space and the posterior fossa, a potential herniation site. The midbrain and cerebral peduncles pass through the incisura. The uncus and hippocampus are located just superior to the medial edges of the incisura.

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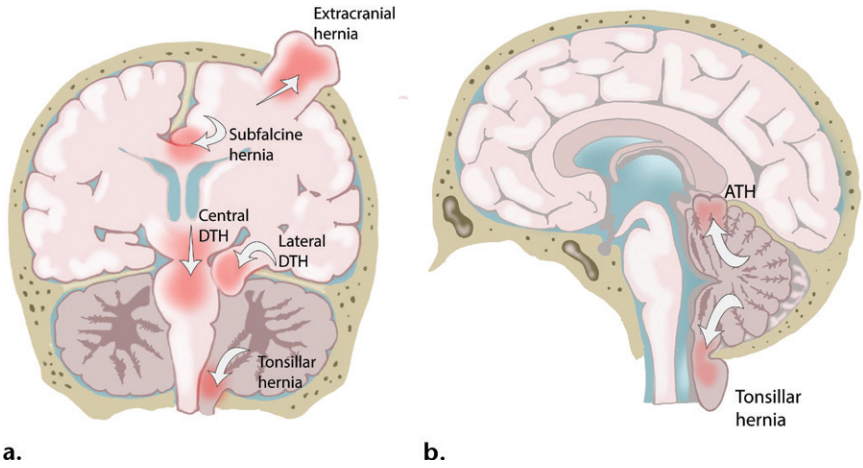

Figure 1. Drawings depict different kinds of brain herniation. *ATH* = ascending transtentorial hernia, *DTH* = descending transtentorial hernia.

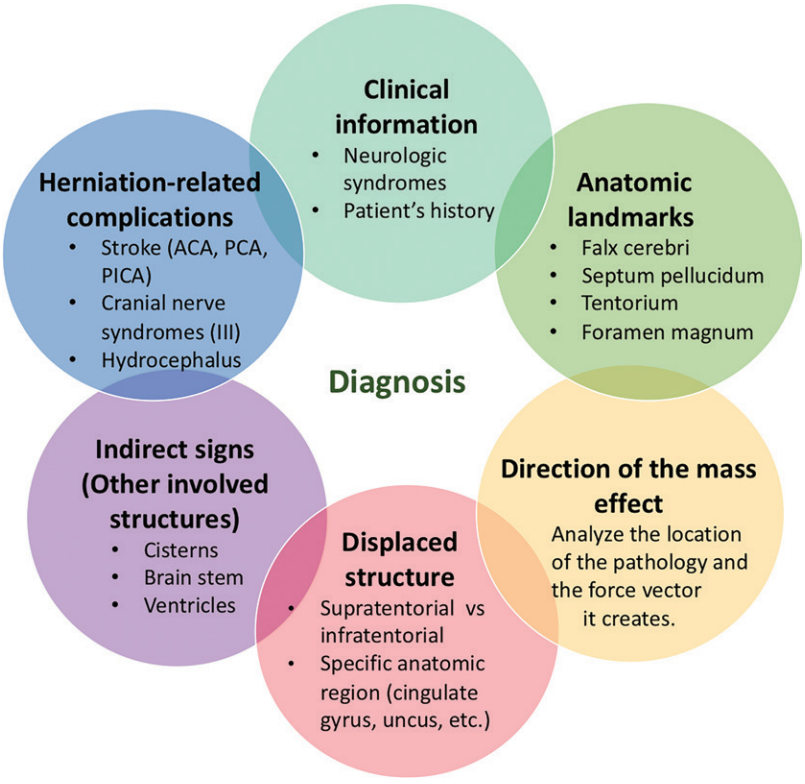

Figure 2. Approach to diagnosing brain herniation syndromes. Diagram shows the six-point guideline for analysis of cerebral herniation cases. *ACA* = anterior cerebral artery, *PCA* = posterior cerebral artery, *PICA* = posterior inferior cerebellar artery.

The basal cisterns are spaces filled with CSF and located in the subarachnoid space. They contain the proximal portions of some cranial nerves and basal cerebral arteries. They are in close contact with the main intracranial structures (Fig 4b, 4c) (11). Basal cisterns are involved in almost any hernia type, making them a key anatomic landmark.

The posterior cerebral arteries, anterior choroidal arteries, and basal veins of Rosenthal pass around the midbrain through the perimesencephalic cistern, close to the free edge of the tentorium. The oculomotor nerve exits the midbrain anteriorly and courses medially to the uncus on its way to the cavernous sinus. These structures are at risk of compression by the herniated tissue.

Finally, the ventricular system is a set of cavities that produce and circulate CSF through the central nervous system. It consists of two lateral ventricles divided by the septum pellucidum. They communicate with the third ventricle via

the foramen of Monro (interventricular foramen). The third ventricle is in communication with the fourth ventricle through the cerebral aqueduct, then empties into the subarachnoid space through the foramen of Luschka (side opening) and foramen of Magendie (median opening) (6).

Intracranial Hernias

Subfalcine Hernia

Subfalcine hernia, also known as midline shift or cingulate hernia, is the most common type of cerebral hernia. It is generally caused by unilateral frontal, parietal, or temporal lobe disease that creates a mass effect with medial direction, pushing the ipsilateral cingulate gyrus down and under the falx cerebri.

The anterior falx, although rigid, is displaced secondary to the mass effect. On the other hand, the posterior falx, wider and more rigid, will resist the displacement. This explains why subfalcine hernias occur anteriorly.

The septum pellucidum deviates at the level of the foramen of Monro, which serves as a landmark for quantification of the degree of midline shift (12). This shift can be measured on axial images by drawing a central line at the level of the foramen of Monro and measuring the distance between this line and the displaced septum pellucidum (Fig 5). In subfalcine hernias, the degree of midline shift correlates with the prognosis; less than 5-mm deviation has a good prognosis, whereas a shift of more than 15 mm is related to a poor outcome (13).

In more severe hernias, the displaced tissue may compress the corpus callosum and contralateral cingulate gyrus, as well as the ipsilateral ventricle and both foramina of Monro, causing dilatation of the contralateral ventricle (Fig 6). There may also be focal necrosis of the cingulate gyrus due to direct compression against the falx cerebri (7,12). Compromise of these structures manifests clinically as hypobulia, apathy, and indifference (14). Subfalcine hernias are best demonstrated at coronal MRI (Fig 7). Another potential complication is compression of the anterior cerebral artery, specifically the pericallosal artery, with consequent infarction of the corresponding vascular territory (4,7) (Fig 8). The most common clinical manifestation of anterior cerebral artery–territory infarction is contralateral leg weakness (14).

Descending Transtentorial Hernia

Descending transtentorial hernia (DTH) is the second most common type of cerebral hernia. It occurs when brain tissue is displaced downward through the tentorial notch (9).

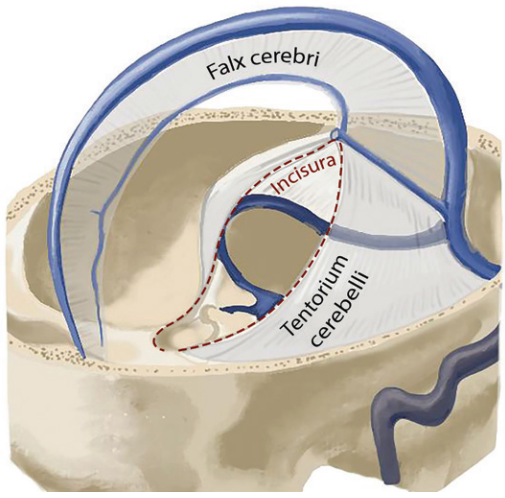

Figure 3. Drawing shows the main dural reflections.

DTH may be divided into two types: lateral (anterior and posterior) and central hernias. Lateral hernias involve the medial temporal lobe. In the anterior subtype, the uncus is herniated downward into the ipsilateral crural cistern. In the posterior subtype, the parahippocampal gyrus is displaced downward into the posterolateral part of the tentorial incisura (9). Finally, in central hernias, there is descent of the diencephalon, midbrain, and pons (12). This classification can be understood as a continuum representing the progression of DTH.

In this type of hernia, the pressure caused by the crowding of tissue within the incisura compromises the third cranial nerve, posterior cerebral artery, and midbrain. Hydrocephalus develops because of the compression of the cerebral aqueduct. In cases with severe and abrupt downward displacement of the brainstem, stretching and shearing of perforating branches of the basilar artery occur, resulting in ischemia and hemorrhage in the brainstem. Usually, these findings are located near the pontomesencephalic junction. However, the effect can be multiple or even extend into the cerebellar peduncles. This is called Duret hemorrhage; it is a late finding and portends a poor prognosis, usually death (12).

It is important to note that different types of cerebral hernias can be present at the same time. In DTH, if there is further descent of brain tissue, a tonsillar hernia might occur. Also, a subfalcine hernia may be present, depending on the location of the disease.

Lateral Hernia

Lateral hernias occur when the medial temporal lobe is displaced downward through the tentorium incisura. They can be divided into anterior and posterior hernias, depending on the portion that is displaced.

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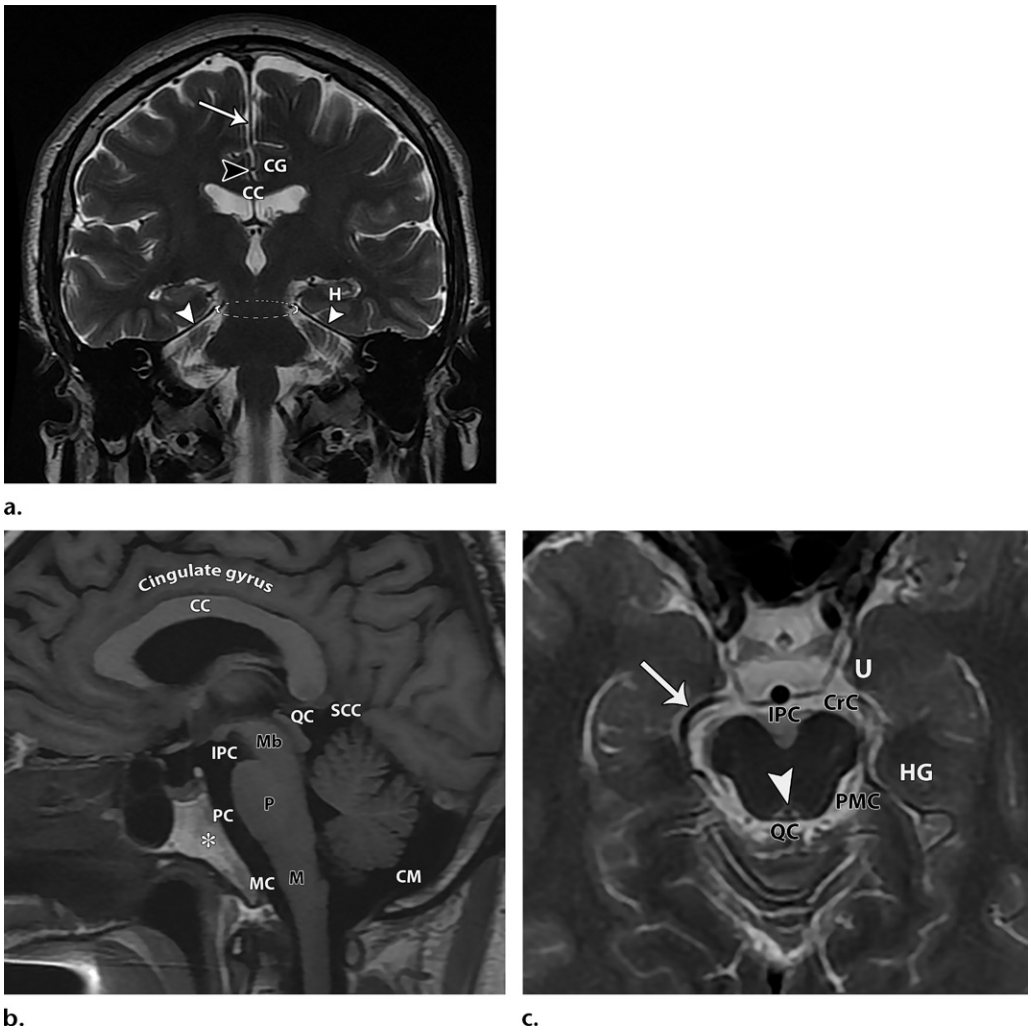

Figure 4. Relevant radiologic anatomy in cerebral hernias. **(a)** Coronal T2-weighted MR image shows the cerebral falx in the interhemispheric fissure (arrow), tentorium (white arrowheads), tentorial incisura (dashed oval), corpus callosum *(CC)*, cingulate gyrus *(CG)*, hippocampus *(H)*, and pericallosal sulcus with the pericallosal artery (black arrowhead). **(b)** Sagittal T1-weighted MR image at the midline of the cranial cavity depicts the cisterna magna *(CM)*, interpeduncular cistern *(IPC)*, medullary cistern *(MC)*, pontine cistern *(PC)*, quadrigeminal cistern *(QC)*, and supracerebellar cistern *(SCC)*. The corpus callosum *(CC)*, cingulate gyrus, and clivus (*) are also noted. The brainstem divisions are as follows: medulla *(M)*, midbrain *(Mb)*, and pons *(P)*. **(c)** Axial T2-weighted MR image shows the aqueduct (arrowhead), posterior cerebral artery (arrow), crural cistern *(CrC)*, hippocampal gyrus *(HG)*, interpeduncular cistern *(IPC)*, perimesencephalic cistern *(PMC)*, quadrigeminal cistern *(QC)*, and uncus *(U)*.

Anterior Hernia.—Anterior (or uncal) hernia is the better understood subtype of DTH (12). Usually, a unilateral supratentorial lesion (particularly in the middle cranial fossa) causes an inferior and medial mass effect that pushes the uncus over the free edge of the tentorium (7). It is the first event in most cases of DTH, usually followed by herniation of more posteriorly located brain tissue. However, the distribution and sequence of the DTH will also depend on certain factors, such as the location of the disease and the size and configuration of the incisura (9).

The initial displacement of the uncus results in effacement of the suprasellar cistern, the earliest finding in this type of hernia. Often, that is all it effaces. As the herniation progresses, there is widening of the ipsilateral perimesencephalic cistern, with displacement and rotation of the brainstem (Figs 9, 10). With more advanced herniation, the midbrain and opposite cerebral peduncle are compressed against the tentorial edge (Fig 9b) (7,12). Descending corticospinal and corticobulbar tracts may be affected above the medullary decussation, resulting in motor weakness on the same side as the lesion, known as the Kernohan notch phenomenon (false localizing sign) (15).

Compression of the posterior cerebral artery, third cranial nerve, and aqueduct of Sylvius may result in medial temporal and occipital lobe infarcts, blown pupil, hemiparesis, and hydrocephalus (Fig 11) (1).

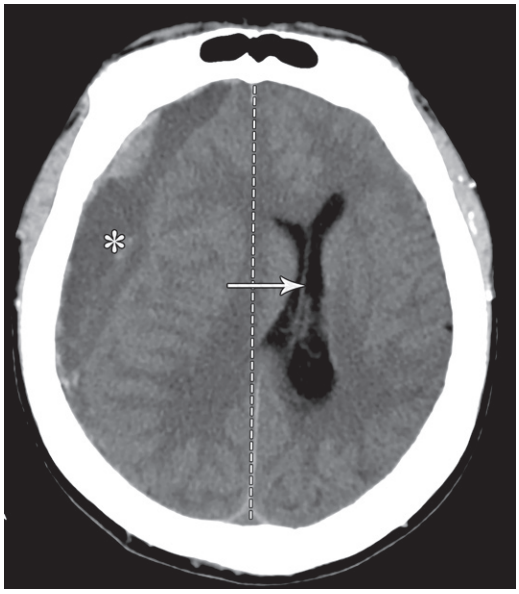

Figure 5. Subfalcine hernia. Axial nonenhanced CT image shows a right subdural hematoma with mixed attenuation that represents different blood stages (*). The hematoma displaces the septum pellucidum to the left (arrow) relative to the midline (dashed line). The right lateral ventricle is compressed, but the left lateral ventricle is dilated.

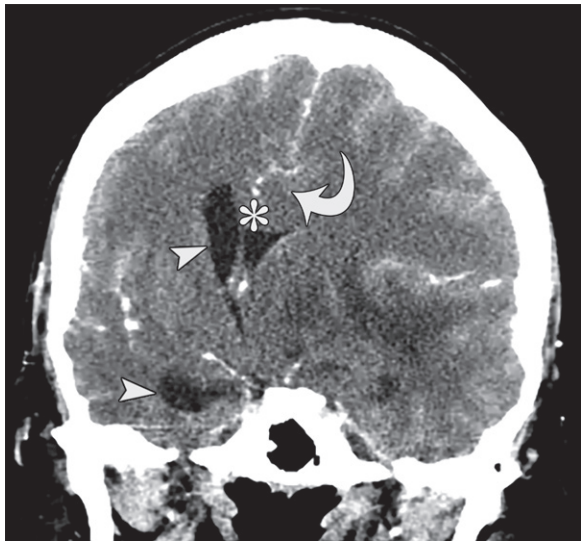

Figure 6. Subfalcine hernia in a 33-year-old man with a cerebral metastasis from a germinal tumor. Coronal contrast-enhanced CT image shows herniation of the cingulate gyrus beneath the free edge of the falx cerebri from left to right (arrow) and associated inferior and lateral displacement of the ipsilateral corpus callosum (*). Note the compression of the ipsilateral ventricle and dilatation of the contralateral ventricle (arrowheads).

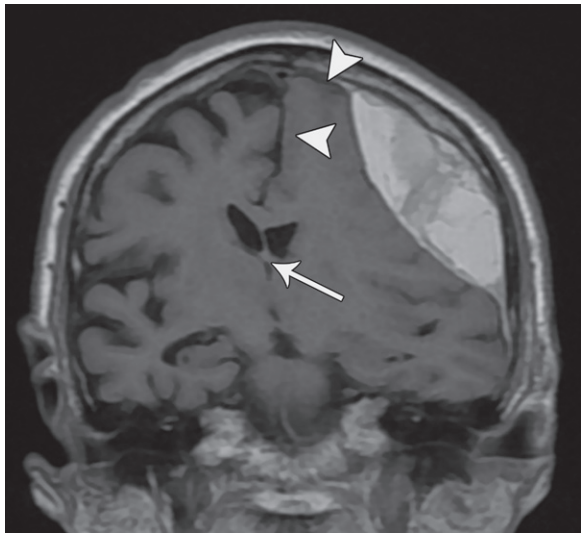

Figure 7. Coronal T1-weighted MR image shows a loculated subdural hematoma and subfalcine herniation. There is effacement of the subarachnoid space and compression of the left frontal lobe against the hard falx (arrowheads). Also note the mild displacement of the foramen of Monro to the right (arrow).

Posterior Hernia.—In patients with occipital and posterior temporal disease, the herniation of the medial temporal lobe occurs more posteriorly (12). The parahippocampal gyrus, behind the uncus, is displaced downward into the posterolateral part of the tentorial incisura. Larger posterior hernias may also include the isthmus of the fornical gyrus and the anterior part of the lingual gyrus. This brain tissue will impinge on the lateral part of the quadrigeminal plate cistern and cause displacement, rotation, and compression of the brainstem (Fig 12) (9). It may involve the tectum at the level of the superior colliculus, resulting in Parinaud syndrome, which is commonly present in this type of DTH. There is relatively less compression of the oculomotor nerve and posterior cerebral artery than in other types of DTH (12).

Central Hernia

In central hernia, there is descent of the diencephalon, midbrain, and pons. It usually manifests along with other types of DTH. Bilateral supratentorial disease causing mass effect, midline masses, severe brain edema, or supratentorial hydrocephalus may cause this type of hernia.

Effacement of the perimesencephalic cisterns is the most useful and consistent finding. Caudal displacement of the basilar artery and pineal gland, flattening of the pons against the clivus, and inferior and posterior displacement of the quadrigeminal plate are other useful indicators of this type of hernia (Fig 12). Hydrocephalus and infarction of the posterior cerebral artery territory are common complications (12). Progressive central herniation can lead to oculomotor palsy, progressive alteration of consciousness, decerebrate posturing, coma, and eventually death (12).

Ascending Transtentorial Hernia

Ascending transtentorial hernia occurs when a mass effect, coming from the posterior cranial

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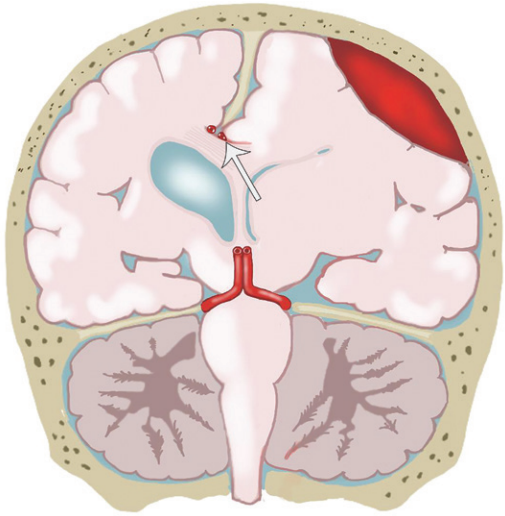
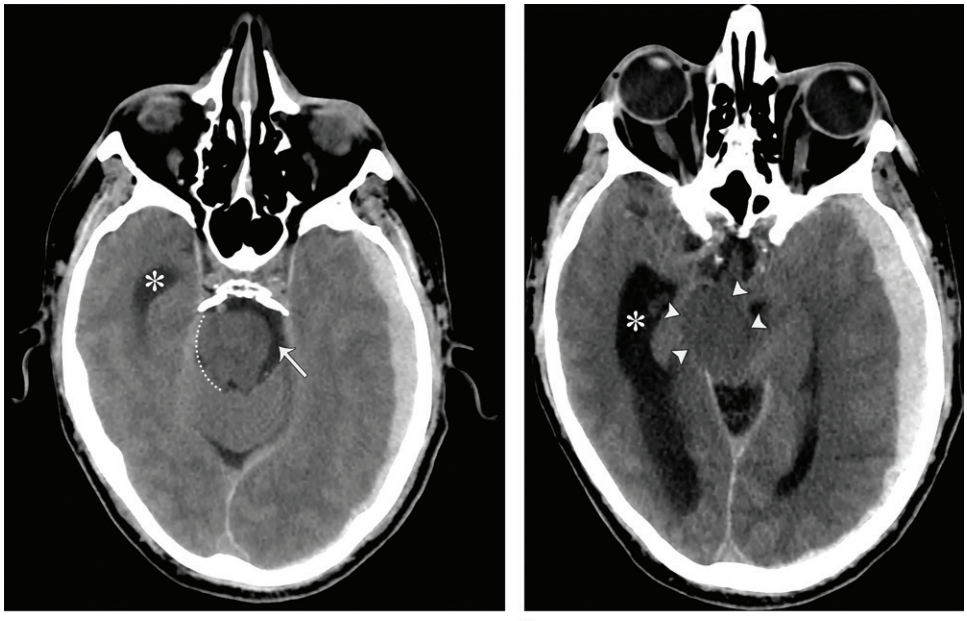


Figure 8. Drawing shows compression of the pericallosal artery (arrow) against the falx cerebri due to a subfalcine hernia.



a. **b.**

Figure 9. DTH in a 38-year-old man with an acute head injury and left subdural hematoma. There is right shift and mild rotation of the brainstem. **(a)** Axial nonenhanced CT image shows widening of the left basal cistern (arrow) and effacement of the right basal cistern (dashed line), as well as dilatation of the temporal horn of the right lateral ventricle (*). **(b)** CT image shows compression and rotation of the midbrain, which appears elongated (arrowheads). There is complete obliteration of the perimesencephalic cisterns. Note the widening of the opposite ventricular atrium and temporal horn (*).

fossa with an upward direction, displaces the cerebellar vermis and hemispheres superiorly through the tentorial incisura. It is more likely to occur when the mass originates near the incisura, like in the cerebellar vermis (10). Another possible cause is sudden relief of supratentorial intracranial hypertension (7).

The tentorial incisura size is variable and influences whether ascending transtentorial hernia or tonsillar hernia occurs. In the context of increased intracranial pressure, brain tissue is displaced toward the site that offers less resistance. When the tentorial incisura is small, cerebellar tissue will slide through the foramen magnum, causing tonsillar herniation. On the other hand, when the tentorial opening is large, upward herniation of the superior cerebellar vermis will occur before tonsillar herniation (10).

As there is upward herniation of the cerebellar vermis, anterior displacement of the midbrain and cerebral aqueduct takes place. The normal concave configuration of the quadrigeminal plate cistern is distorted, taking on a flat or convex morphology. If the posterolateral aspect of the midbrain is compressed bilaterally, the classic "spinning top" configuration will appear (16).

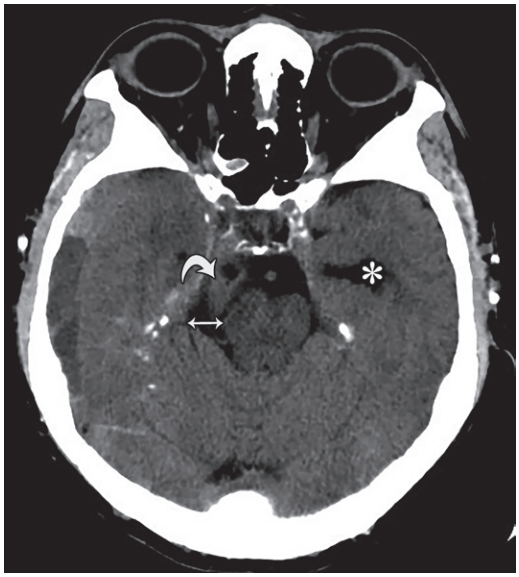


Figure 10. Subdural hematoma after a hemodialysis session in a 26-year-old man with a history of end-stage kidney disease. The right pupil was dilated and unresponsive to light. Axial CT image shows the uncus displaced downward across the tentorial incisura (curved arrow). The ipsilateral perimesencephalic cistern (double-headed arrow) is widened, and the contralateral cistern is compressed. Note the contralateral dilatation of the temporal horn of the lateral ventricle (*).

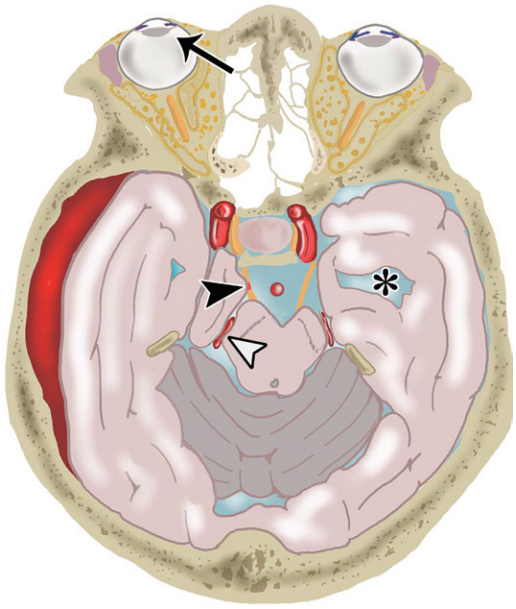


Figure 11. Drawing shows a lateral DTH with compression of the posterior cerebral artery (white arrowhead) and third cranial nerve (black arrowhead). Note the ipsilateral blown pupil (arrow) and contralateral temporal horn dilatation (*).

Hydrocephaly may be present due to compression of the aqueduct. The most relevant imaging findings are illustrated in Figure 13.

Hemispheric branches of the superior cerebellar arteries, as well as posterior cerebral arteries, may be compressed. This causes ischemic infarction of the superior portion of the cerebellar hemispheres and occipital cerebral lobe (10).

Clinically, signs of cerebellar and brainstem compression, as well as increased ICP, may be present (6,17).

Tonsillar Hernia

Tonsillar hernia is inferior displacement of the cerebellar tonsils through the foramen magnum into the cervical spinal canal. It may be congenital (Chiari spectrum) or acquired.

Normal tonsillar position relative to the foramen magnum varies with age. Mikulis et al (18) described the normal position of tonsils below the foramen magnum for different age groups. In the 1st decade of life, the presence of cerebellar tonsils more than 6 mm below the foramen magnum is considered abnormal. In the next 2 decades, the reference value is 5 mm; for the 4th to 8th decades, the threshold is greater than 4 mm; and at age 80 years or older, 3 mm is the limit (18,19).

The McRae line is used as a reference for this measurement. It is obtained by drawing a line from the basion to the opisthion. The degree of

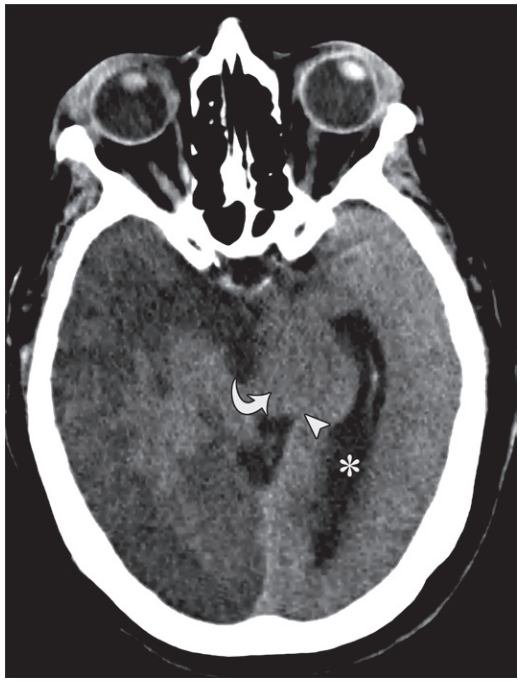
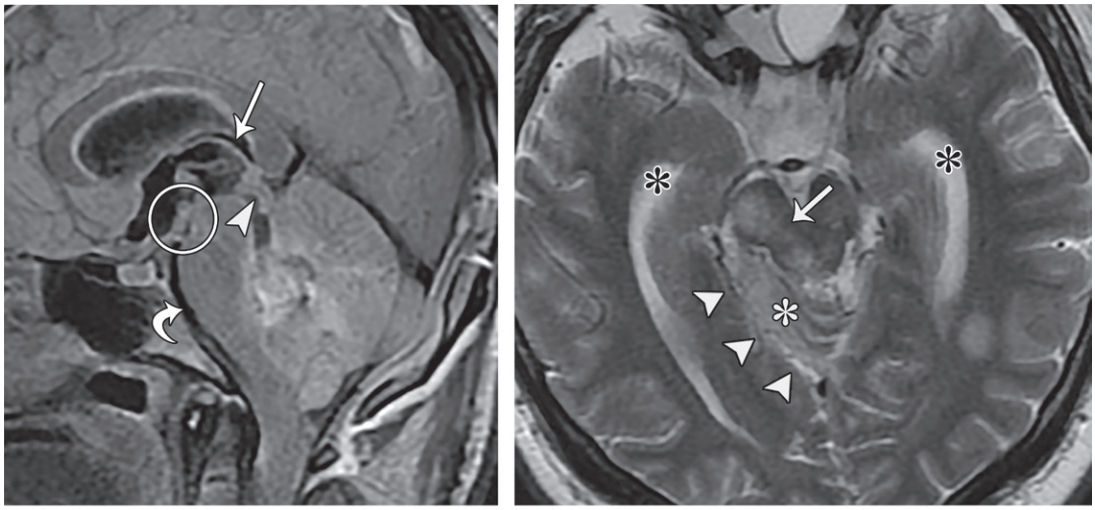


Figure 12. DTH in a 64-year-old man with a history of ischemic heart disease who presented with symptoms of sudden neurologic deterioration due to a stroke. DTH occurred secondary to brain edema. Axial nonenhanced CT image at the level of the midbrain shows left displacement and rotation of the mesencephalon (curved arrow), left displacement of the quadrigeminal plate (arrowhead), and obliteration of the perimesencephalic cisterns. In addition, dilatation of the atrium and temporal horn of the left lateral ventricle is present (*).

RadioGraphics



a. b.

Figure 13. **(a)** Ascending transtentorial hernia in a 26-year-old man after resection of a medulloblastoma. Sagittal gadolinium-enhanced T1-weighted MR image shows obliteration of the quadrigeminal, superior cerebellar, and interpeduncular cisterns. There is associated folding of the inferior colliculi (arrowhead) under the superior colliculi, and both structures have shifted upward. Anterior displacement of the brainstem reduces the space of the pontine and medullary cisterns (curved arrow). Note the superior displacement of the third ventricle roof (straight arrow) and the anterior displacement of the mamillary bodies and tuber cinereum (circle), which are in close contact with the midbrain. **(b)** Ascending transtentorial hernia in a 33-year-old man suspected of having intracranial lesions. Axial T2-weighted MR image shows the cerebellum (white ∗) ascending through the right side of the incisura (arrowheads), causing obliteration of the right perimesencephalic and left crural cisterns, diminished space of the quadrigeminal cistern, enlargement of the temporal horns of the lateral ventricles (black ∗), and edema in the tectum and right cerebral peduncle (arrow).

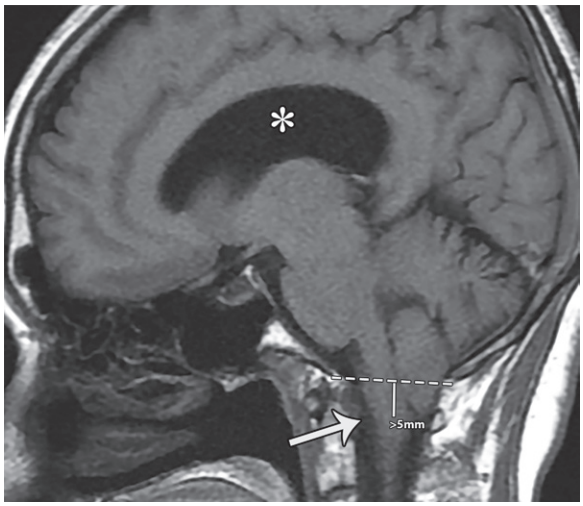


Figure 14. Sagittal T1-weighted MR image shows downward displacement of the cerebellar tonsils (>5 mm) relative to the McRae line (dashed line). Note the obliteration of the cisterna magna, anterior displacement of the medulla (arrow), and hydrocephalus (∗).

tonsillar herniation is the perpendicular length from the McRae line to the tip of the displaced tonsil (Fig 14) (20).

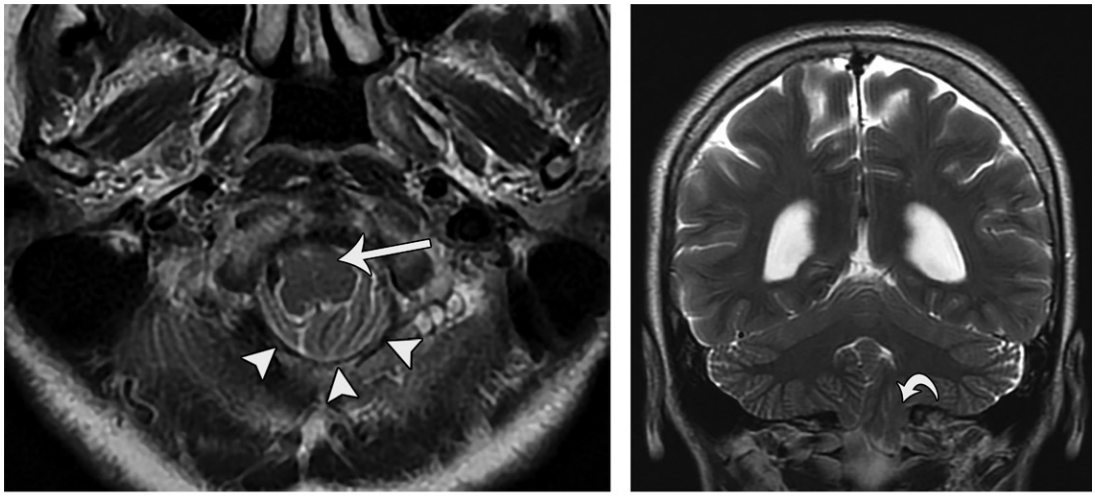
The most common cause is an infratentorial mass creating a downward mass effect. It may also be secondary to a supratentorial mass, in which case it is usually associated with a DTH. It can cause severe neurologic damage followed by sudden respiratory arrest (12,19).

Visualization of tonsils extending below the foramen magnum, anterior brainstem displacement, and loss of CSF surrounding it are common features (Figs 15, 16). The fourth ventricle may be compressed, producing obstructive supratentorial hydrocephalus (4,12). Compression of the posterior inferior cerebellar artery by the herniated tonsils can lead to cerebellar infarcts (7).

Transalar Hernia

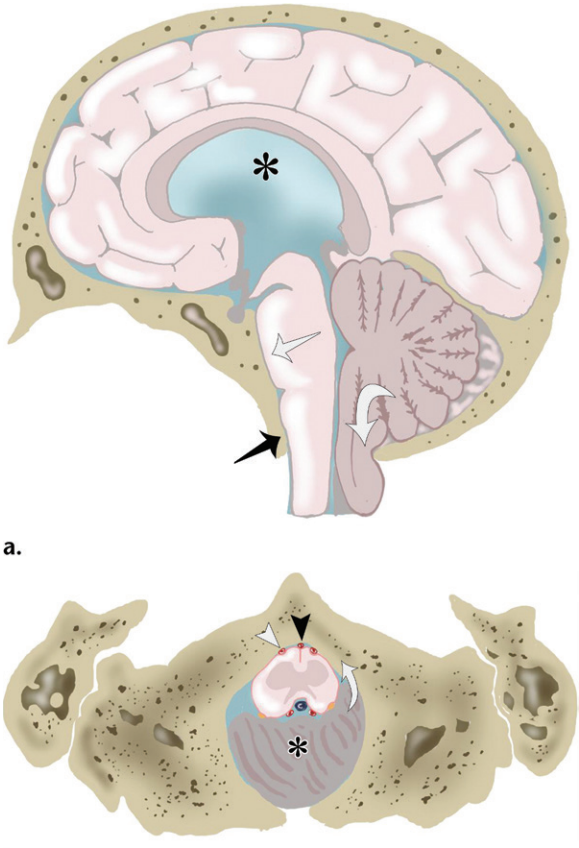
Transalar hernia is an uncommon and less described type of hernia. It is usually associated with subfalcine and transtentorial hernias (21). It can be divided into descending and ascending transalar hernias. In the descending type, the frontal lobe is displaced posteriorly and inferiorly over the sphenoid wing. It manifests secondary to frontal lobe disease. It can cause compression of the middle cerebral artery against the sphenoid ridge with a middle cerebral artery infarction.

With ascending transalar hernia, the temporal lobe is displaced superiorly and anteriorly across the sphenoid ridge owing to a middle cranial fossa mass effect. This displacement can compress the supraclinoid internal carotid artery against the anterior clinoid process with infarction of the anterior cerebral artery and middle cerebral artery



a. b.

Figure 15. Chiari I malformation. Axial **(a)** and coronal **(b)** T2-weighted MR images show tonsillar descent (arrow in **b**) with anterior brainstem displacement (arrow in **a**) and effacement of the CSF in the foramen magnum (arrowheads in **a**).



a.

b.

Figure 16. Drawings show tonsillar hernia. **(a)** Sagittal view demonstrates tonsils extending below the foramen magnum (curved arrow), brainstem compression against the clivus (straight white arrow), obliteration of the medullary cistern (black arrow), and obstructive hydrocephalus (*). **(b)** Axial view at the level of the foramen magnum. The displaced tonsils (*) cause obliteration of the surrounding CSF, anterior displacement of the medulla (arrow), and compression of the spinal (black arrowhead) and vertebral (white arrowhead) arteries.

territories (7). Identifying anterior or posterior displacement of the middle cerebral artery is useful for suspecting this type of hernia (21).

Extracranial Hernia

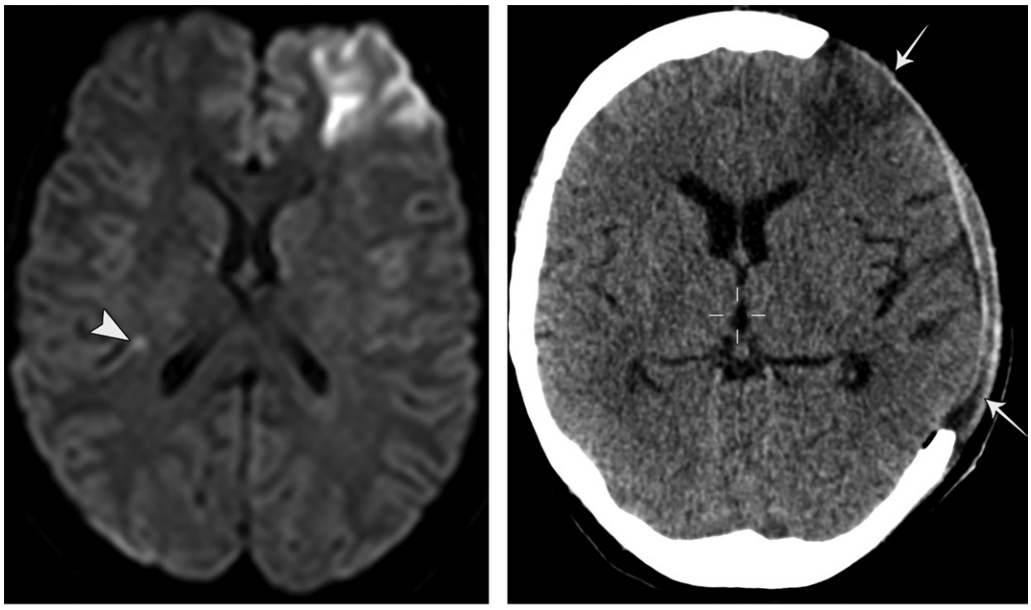
External hernias are less common than other types of hernias. They are most frequently caused by postsurgical and posttraumatic cranial defects that allow brain tissue to pass through.

Craniectomy may be performed to decompress intracranial contents in patients with intracranial hypertension after medical management fails (22). Brain edema is common in the 1st week after decompressive craniectomy. It may correspond to hyperperfusion and loss of resistance in brain tissue, causing a higher hydrostatic pressure gradient that favors transcapillary leakage of fluid (23).

A large craniectomy defect allows the brain to expand without constriction. If the defect is too small, swollen brain may herniate with a "mushroom cap" appearance. This can result in compression of cortical veins and lead to venous infarction and contusion of the brain at the craniectomy margins. Both CT and MRI are effective in depicting this hernia (3,12) (Fig 17).

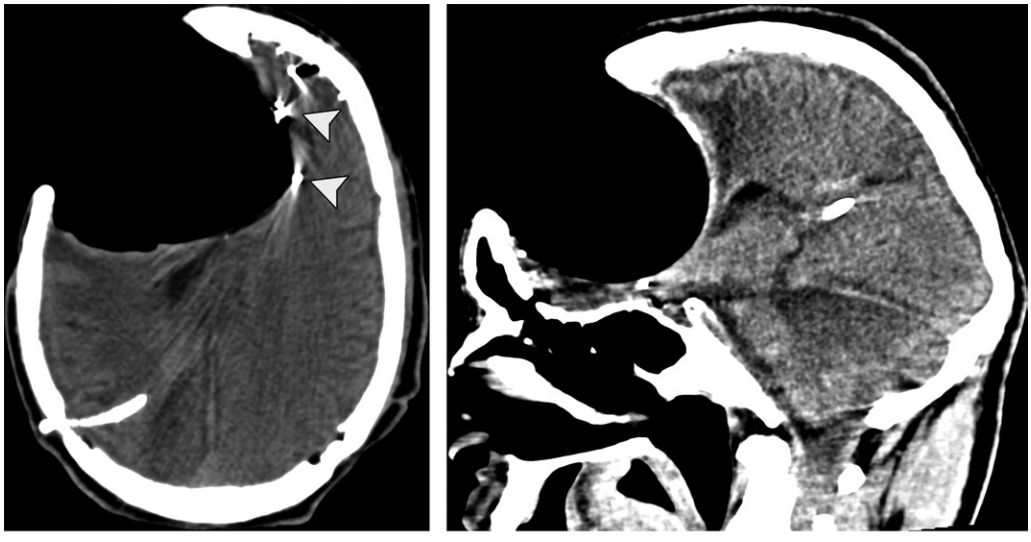
Paradoxical hernia is a rare and potentially fatal complication of decompressive craniectomy. It is a neurosurgical emergency. Atmospheric pressure exceeding ICP at the site of the craniectomy causes a pressure imbalance and consequent subfalcine and/or transtentorial hernia. The brain tissue is displaced from the craniectomy defect (Fig 18). It is often triggered by an acute imbalance of ICP secondary to CSF drainage or lumbar puncture (3).

Symptoms include a depressed level of consciousness, autonomic instability, signs of brainstem release, and focal neurologic deficits (3).



a. b.

Figure 17. Extracranial hernia. **(a)** Axial diffusion-weighted image shows an acute infarction in the left frontal lobe. Also note the small focus on the right (arrowhead). A few days later, the patient developed vasogenic brain edema, and decompressive craniectomy was performed. **(b)** Axial CT image after craniectomy shows brain parenchyma herniating through the defect of the frontal and temporal lobes (arrows). The sylvian fissure is enlarged, and the frontal and occipital horns of the left lateral ventricle are retracted.



a. b.

Figure 18. Paradoxical hernia in a 32-year-old man who was shot in the head three times. He underwent decompressive craniectomy and developed sinking flap syndrome. **(a)** Axial nonenhanced CT image shows left midline displacement and right ventricle compression with associated edema. There is metal artifact in the left frontal lobe from remnants of the bullet (arrowheads). **(b)** Sagittal CT image demonstrates the frontal and temporal lobe displacement.

Intracranial Hypotension

Intracranial hypotension is another cause of cerebral herniation that should be considered. It must be suspected in patients without an intracranial mass or edema or when the degree of herniation is out of proportion to the degree of mass effect (12). It is caused by iatrogenic or spontaneous CSF leak. The loss of CSF volume results in downward descent of the brain secondary to a negative pressure gradient between the cranial and spinal compartments. DTH and tonsillar hernia may manifest (23). Also, blood volume will increase to maintain the intracranial volume. This results in pachymeningeal hyperemia and edema, which can be identified at MRI as diffuse pachymeningeal enhancement (12).

Communicating Critical Results

Effective communication between interpreting physicians and ordering health care providers is a critical component of diagnostic imaging. Those responsible for making treatment decisions should be notified of imaging results in a timely fashion. In 2014, the American College of Radiology published the "ACR Practice Parameter for Communication of Diagnostic Imaging Findings" as an educational tool designed to assist practitioners in providing appropriate radiologic care for patients (24). In this guideline, different methods of communication are mentioned depending on the clinical scenario.

The guideline discusses the imaging final report, the preliminary report, and nonroutine communications. Nonroutine communications are used in emergent clinical situations. Many institutions have established policies and guidelines for communicating critical results. Specifically, in neuroradiology, there are multiple time-sensitive pathologic conditions that need urgent treatment once diagnosed. A list of critical findings for which immediate notification is required should be established (25), as well as the policy for communication and proper documentation.

Conclusion

Cerebral herniation syndromes represent a common life-threatening neurologic emergency. They manifest as complications of multiple different pathologic conditions. The patient history and clinical scenario are key for making an accurate diagnosis. Having a thorough understanding of cerebral herniation syndromes and their imaging spectrum is essential. The radiologist should keep in mind that many of these patterns of herniation may overlap and that common complications include compression of cranial nerves, CSF spaces, and blood vessels. Misdiagnosis can lead to increased patient morbidity and even death. CT and MRI are the imaging modalities of choice for establishing the diagnosis, guiding the therapeutic decisions, and determining the prognosis.

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