Dangerous CT brain findings in head trauma warranting urgent intervention

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Authors: P. M. A. D. Mohamed Abouelhoda¹, A. H. A. H. Ibrahim¹, R. N. M. Abdalla², A. Mugahid¹, A. Khaled¹, A. daoud¹, M. A. M. Ayoub¹; ¹Cairo/EG, ²Cairo, ca/EG
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Learning objectives

1. To review the basic anatomy of CT brain.
2. To recognize different patterns of head trauma on CT brain.
3. To highlight the dangerous signs on CT brain requiring urgent intervention.
4. To recognize when to ask for other radiological modalities.
Background

Traumatic brain imaging is a common problem. Urgent proper management of patients suffering from traumatic brain injury can dramatically alters their clinical course, and therefore immediate diagnostic work up for these patients is mandatory in the acute setting of head trauma.

Non contrast enhanced CT brain in head trauma is the modality of choice, it is quick, feasible, and in most cases it can determine the presence and extent of injury and guide surgical planning and minimally invasive interventions, yet not all head trauma patients need neuro imaging.
Findings and procedure details

Studies have found that less than 10% of patients that are considered to have minor head injuries have positive findings on CT and less than 1% require neurosurgical intervention. That's why the National Institute for health and care clinical excellence (NICE), recommended new guidelines in 2014 stating the criteria needed for performing CT head scan in trauma patients, depending on the age and the clinical data, which implies the following:

**In Adults**

Who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:

- GCS less than 13 on initial assessment in the emergency department.
- GCS less than 15 at 2 hours after the injury on assessment in the emergency department.
- Suspected open or depressed skull fracture.
- Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
- Post-traumatic seizure.
- Focal neurological deficit.
- More than 1 episode of vomiting.

For adults with any of the following risk factors who have experienced some loss of consciousness or amnesia since the injury, perform a CT head scan within 8 hours of the head injury:

- Age 65 years or older.
- Any history of bleeding or clotting disorders.
- Dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 meter or 5 stairs).
- More than 30 minutes retrograde amnesia of events immediately before the head injury.

**In Children**

For children who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:
• Suspicion of non-accidental injury
• Post-traumatic seizure but no history of epilepsy.
• On initial emergency department assessment, GCS less than 14, or for children under 1 year GCS (paediatric) less than 15.
• At 2 hours after the injury, GCS less than 15.
• Suspected open or depressed skull fracture or tense fontanelle.
• Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
• Focal neurological deficit.
• For children under 1 year, presence of bruise, swelling or laceration of more than 5 cm on the head.

For children who have sustained a head injury and have more than 1 of the following risk factors (and none of those in the previous recommendations), perform a CT head scan within 1 hour of the risk factors being identified:

• Loss of consciousness lasting more than 5 minutes (witnessed).
• Abnormal drowsiness.
• Three or more discrete episodes of vomiting.
• Dangerous mechanism of injury (high-speed road traffic accident either as pedestrian, cyclist or vehicle occupant, fall from a height of greater than 3 metres, high-speed injury from a projectile or other object).
• Amnesia (antegrade or retrograde) lasting more than 5 minutes.

Children who have sustained a head injury and have only 1 of the risk factors in the previous recommendations, should be observed for a minimum of 4 hours after the head injury. If during observation any of the risk factors below are identified, perform a CT head scan within 1 hour:

• GCS less than 15.
• Further vomiting.
• A further episode of abnormal drowsiness.

**For Patients having warfarin treatment**

For patients (adults and children) who have sustained a head injury with no other indications for a CT head scan and who are having warfarin treatment, perform a CT head scan within 8 hours of the injury.

For accurate diagnosis of different pathologies, proper knowledge of brain anatomy is crucial, the brain parenchyma is divided into paired, bilateral anatomical areas or 'lobes'. The lobes are divided into frontal, parietal, temporal and occipital. (Figure 1)
The parenchyma consists of grey and white matter structures which are differentiated on CT by differences in density. White matter has a high content of myelinated axons (which contains fat). Grey matter contains higher number of cell bodies. Therefore, white matter appears darker than grey matter.

The cerebral cortex is a layer of grey matter formed in gyri over the entire brain surface, while the white matter of the brain lies deep to the cortical grey matter.

The folds of the cerebral cortex are known as gyri, and the furrows between them are known as sulci, which contain CSF. (Figure 2)

The posterior fossa accommodates the cerebellum and the brain stem.

The brain is surrounded by cerebrospinal fluid (CSF) within the sulci, fissures and basal cisterns. CSF is also found centrally within the ventricles. The sulci, fissures, basal cisterns and ventricles together form the 'CSF spaces', also known as the 'extra-axial spaces'.

The ventricles consists of two paired lateral ventricles located on either sides of the brain. The third ventricle is located centrally, which is communicated to the lateral ventricles via small holes (called foramina of Monro), The fourth ventricle is located in the posterior fossa between the brain stem and cerebellum , and it communicates with the third ventricle above via a very narrow canal, the aqueduct of Sylvius . (Figure 3)

The brain is covered by the meninges which is a thin layers of tissue found between the brain and the inner table of the skull. The meninges comprise the dura mater, the arachnoid, and the pia mater. (Figure 4)

Trauma to the head can cause injury to brain tissue by different mechanisms. It can be categorized to primary lesions that results due to the impact of trauma itself, and secondary lesions that occurs afterward, with or without association of primary injury.

**Primary lesions:**

- Skull injury: fracture
- Brain tissue injury: contusion and diffuse axonal injury
- Hemorrhage: which can be furtherly categorized to intra-axial and extra-axial hemorrhage.

**Secondary lesions:**
• Midline shift
• Brain herniation
• secondary or delayed hemorrhage
• ischemia and infarction
• diffuse hypoxic injury

Many classification have been used for pathoanatomic description in acute head injury studies including the Marshall score for CT findings and the Rotterdam score, however this pathoanatomic classification helps us to know where the lesion is, but it doesn't give us any clue about its severity.

Recent studies states that when assessing the severity of head trauma, different aspects should be considered, like the lesion type, site and size, also patient clinical data (usually by calculating Glasgow coma score) should be considered.

The combination of all these data together helps up to assess the severity of the lesion and guides us to proper management. Yet, clinical deterioration of the patient alone can warrant surgical intervention.

That's why we are going to discuss what are the dangerous signs that we should look for in CT brain of acute head trauma patients

**Skull Fracture:**

Skull fracture have different patterns, it ranges from simple linear fissure fracture to more severe comminuted depressed fracture.

**Linear fracture:**

a. Skull vault linear fracture; if it is an isolated finding, then it is treated conservatively and no further management is needed.

b. Skull base linear fracture; the management of the patient depends on the site.

- Sphenoidal fracture may involve the carotid canal and cause injury of internal carotid artery leading to pseudo aneurysm or carotid cavernous fistula and therefore it is mandatory to perform **CT angiography** for any patient suspecting carotid canal injury.
- Petrous part of temporal bone fracture may lead to ossicular dislocation (in case of longitudinal fracture), or vestibulo-cochlear nerve affection (in case
of transverse fracture), accordingly radiological findings should be correlated with patient clinical data, if fascial nerve palsy is present with continuous deterioration then surgical decompression to the nerve is recommended.

**Depressed skull fracture:**

Surgical intervention is indicated mainly in open (compound) fracture in which:

- The depressed fracture is more than the thickness of the calvaria
- The depression is more than 1 cm *(Figure 5)*
- If there is any other significant brain lesion
- If this criteria is not fulfilled, then conservative treatment is recommended *(Figure 6)*

**Epidural hematoma(EDH):**

It is the hemorrhage taking place between the skull and the dura usually due to arterial bleeding in 85% of cases.

Its commonest location is the temporo-parietal area due to disruption of middle meningeal artery as it exits its bony groove at the pterion.

On CT:

it appears bilenticular lesion, with high fresh blood density, it may cross the dural attachments but not sutures.

**Acute epidural hematoma:** It is mainly hyperdense with isodense areas (representing serum) and hypodense areas (representing active bleeding). *(Figure 7)*

**Subacute epidural hematoma:** homogenously heperdense, it contains solid blood clot.

**Chronic epidural hematoma:** hetrogenous or of hypodense attenuation.

It is treated surgically, yet conservative management with close follow up is recommended if:

- Its volume is less than $30\text{cm}^3$
- Thickness <$15\text{mm}$
- Midline shift <$5\text{mm}$
- The patient is intact neurologically without focal deficit.

**Subdural hematoma(SDH):**
It is bleeding between the dura and arachnoid, due to injury of the bridging vessels lying between them.

CSF hygroma can occur, in which the subarachnoid space is filled with CSF due to arachnoid tear.

**On CT:** it shows crescent extra axial collection with concave inner border

Its density varies according to the age of the hemorrhage

**Acute SDH:** mixed hyperdense and hypodense due to the presence of fresh blood, serum exudate and CSF.

**Subacute SDH:** isodense on CT, with effacement of the cortical sulci and midline shift. *(Figure 8)*

**Chronic SDH:** typically hypodense, it occurs usually in elderly patients. *(Figure 9)*

**Subduaral hygroma:** CSF density, more crescent and bilateral

*for better visualization of SDH and EDH its better to evaluate the brain in intermediate brain window

Surgical management is recommended if:

- Thickness <10mm.
- Midline shift >5mm.

**Subarachnoid hemorrhage (SAH):**

It is the presence of blood between the arachnoid and pia, it occurs due to damage of blood vessels on the pia, or it may be due to rupture of intraparenchymal hematoma in the ventricular space.

Communicating hydrocephalus can occur as a secondary complication

**On CT:** it will appear as hyperdense area in the sulci, sylvian fissure and basal cisterns. *(Figure 10)*

*Pseudo subarachnoid hemorrhage due to diffuse brain edema can be falsely diagnosed as subarachnoid hemorrhage.

Surgical intervention is needed in case of hydrocephalus
**Intraventricular hemorrhage:**

It is usually associated with other primary intra axial hemorrhage.

**On CT:** seen as high density blood inside the ventricles with or without fluid level. *(Figure 11)*

**Intraparenchymal Hematoma:**

It is bleeding inside the brain parenchyma, it is usually multiple and mostly associated with other types of hemorrhage as SDH and SAH.

**On CT:** its appearance varies according to the age

**Acute hematoma (<3 days):** high density with irregular border, surrounded by low density with positive mass effect. *(Figure 12)*

**Subacute hematoma (3-14 days):** gradual decrease in the density from the peripheral towards the center

**Chronic hematoma (14 days):** Hypodense lesion with later encephalomalacia transformation and ex-vacuo dilation of the ipsilateral ventricle.

*it is differentiated from hemorrhagic contusion by having sharp borders, perifocal hypodenisty and positive mass effect.

**Brain contusion:**

It is a common type of traumatic brain injury representing a region of primary neuronal and vascular injury, due to direct impact to the brain (coup mechanism) or impact of moving brain on stationary calvarium(counter coup mechanism)this will result in tissue necrosis, capillary disruption and then later on followed by brain edema.

It can be furtherly classified into hemorrhagic contusion and non-hemorrhagic contusion.

**On CT:** we can find single or multiple low attenuating areas with irregular outline, with or without tinny areas of increased density representing petechial hemorrhage. *(Figure 13)*
They will be isodense to the brain in 2-3 weeks.

Surgical treatment is usually indicated if:

- Its volume is more than 50 cm$^3$
- Its volume is more than 20 cm$^3$, and the patient is clinically deteriorating
- If there is signs of positive mass effect (midline shift, compression on the ventricle, brain herniation)

**Diffuse axonal injury:**

It is characterized by widespread axonal disruption occurring in response to acceleration or deceleration forces. Typically, patients are immediately unconscious after the injury.

CT is commonly negative, there may be foci of low density, with 20% of lesions contain sufficient hemorrhage to be visible. *(Figure 14)*

If suspecting diffuse axonal injury, **MRI is mandatory.**

Characteristic locations are:

- The frontal and temporal white matter near the grey - white matter junction
- Internal and external capsule
- Corpus callosum
- Brain stem involved in the most severe cases.

**Post traumatic cerebral edema:**

It is a secondary brain lesion which can occur immediately at the time of trauma or at delayed onset.

It is diagnosed by loss of differentiation between white and grey matter, and by effacement of sulci. *(Figure 15)*

Diffuse cerebral edema can occur following the impact of trauma either immediately or with delayed onset.

Usually brain edema is treated with medical agents, yet surgery in the form of bifrontal decompression craniotomy is preserved for patients with medically refractory cerebral edema or with those who have evidence for **impending trans-tentorial herniation.**
**Post Traumatic hydrocephalus:**

Hydrocephalus is dilatation of the ventricular system. It is found in 40% of cases with severe head injury, it may present early at time of injury, or later as a complication of a primary lesion, up to 3 months following SAH.

**On CT:**

- Dilated ventricular system, the first ventricle to dilate is of the temporal horns of the lateral ventricles *(Figure 16)*
- Ballooning of the third ventricle

If the patient is neurological intact, with no intra cranial pressure it can be treated conservatively with close monitoring, yet if there is any neurological deficit or increase in intracranial pressure surgical intervention is recommended.

**Cerebral herniation:**

It is a secondary brain lesion that occurs due to mass effect of another associated brain lesion.

It is furtherly categorized into many types according to its site, with the commonest ones are subfalcine and transtentorial herniation, while the others are rare to see. *(Figure 17)*

The presence of brain herniation is a sign of surgical intervention, as it can lead to infarction and death.

**Subfalcine herniation** occurs when the cingulate gyrus shifts beneath the falx, due to medially directed supratentorial mass effect. This may cause compression of the anterior cerebral artery (resulting in ipsilateral distal anterior cerebral infarction) and internal cerebral veins.

**On CT:** *(Figure 18)*

- The cingulate gyrus will be pushed to the other side under falx
- The ipsilateral ventricle will be compressed and displaced across midline.

**Transtentorial herniation** may be descending (towards the posterior fossa) or ascending (upward displacement of the cerebellum through the tentorial incisura). Descending transtentorial herniation causes shift of the temporal lobe over the tentorium,
which may compress the third cranial nerve, the posterior cerebral artery, and the midbrain. *(Figure 19)*

**On CT:**

- The uncus is displaced medially
- Ipsilateral effacement of suprasellar cistern
- Ipsilateral dilatation of prepontine and cerebellopontine cistern

**Sphenoid herniation** involves herniation of the frontal lobe posteriorly across the edge of the sphenoid ridge, and rarely produces significant clinical symptoms.

**Transforaminal herniation** results in herniation of the inferior cerebellum downward through the foramen magnum, which can result in obtundation and death.

**On CT:**

- The tonsils level will be >5 mm below foramen magnum (>7 mm in children)
- The inferior aspect of the tonsils becomes pointed.
- The tonsillar fossa become vertically oriented.
- The tonsils are impacted into foramen magnum with obliteration of CSF and displacement of Medulla oblongata.

*All these findings are more evident by MRI than CT.*

Although all of the previous findings affect the management and prognostic outcome of the patient, yet the status of the basal cistern, midline shift and subarachnoid hemorrhage in the basal cistern correlates with the outcome.

**The status of the basal cistern** is assessed at the midbrain level, as the CSF around the midbrain is divided into 3 limbs, one posteriorly and two laterally.

The limbs are assessed separately and the cistern is either:

- Open : 3 limbs are opened
- Partially closed: 1 or 2 limbs obliterated
- Closed : the 3 limbs are obliterated

**The midline shift** is assessed at the level of the foramen of monro, by drawing a ventricle line at the midline and the distance from septum pellucidum *(Figure 12)*

**The Subarachnoid hemorrhage** which was discussed earlier
Fig. 1: Diagrammatic representation of a sagittal section across the brain image showing the brain lobes.

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**Fig. 2:** normal CT brain axial view showing grey and white matter, with appearance of cortical sulci and gyri.

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Fig. 3: Diagram showing the brain ventricular system

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Fig. 4: Diagrammatic representation of a section across the top of the skull, showing the membranes of the meninges. The relationship between the dura (epidural above - subdural below), the subarchnoid space and the cerebral cortex (brain) is shown here. This is where a brain bleed will occur.

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Fig. 5: Left parietal depressed bone fracture with displacement more than 1 cm (1.2 cm), surgical intervention was recommended.

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Fig. 6: A case of frontal bone depressed fracture, the displacement is less than 1 cm, and so the decision in this patient was conservative treatment with follow up. Follow up 6 weeks later showed that the displacement had decreased to 4mm.

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**Fig. 7:** Right frontal lenticular area of mixed heterogeneity (hyperdense with hypodense areas) representing acute epidural hemorrhage.

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Fig. 8: Isodense crescent area is seen in right fronto-parital lobe with concave inner border, representing subacute SDH, with effacement of the sulci and midline shift.

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**Fig. 9:** Bilateral hypodense crescent areas are seen in right fronto-parietal and left parietal lobes, representing chronic Subdural hemorrhage more on the right side with positive mass effect and midline shift.

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**Fig. 10:** Fresh blood density seen within sylvian fissures and cortical sulci, representing acute Subarachnoid hemorrhage.

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Fig. 11: Left basal ganglionic intraparenchymal hematoma with extension to the ipsilateral lateral ventricle

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**Fig. 12:** Hyperdense area seen in right parietal lobe representing intraparenchymal hemorrhagic lesion with surrounding edema and positive mass effect in the form of midline shift and compressing the ipsilateral lateral ventricle.

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Fig. 13: Hyperdense area seen in left frontal lobe representing hemorrhagic contusion measuring 2.5 x 3 x 4 cm, with average volume 15 cm³, not requiring surgical intervention.

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Fig. 14: Small foci of hemorrhage seen at the cortico-medullary junction of the right occipital lobe representing diffuse axonal injury, with associated intraventricular hemorrhage noted in the left occipital horn of lateral ventricle.

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Fig. 15: Showing brain edema evident by loss of differentiation between white and grey matter, and by effacement of sulci.

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Fig. 16: There is dilatation of the temporal horns and the fourth ventricle representing communicating hydrocephalus.

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**Fig. 17:** The table showing different types of brain herniation, with the expected clinical and radiological findings and anticipated impending complications.


<table>
<thead>
<tr>
<th>Herniation</th>
<th>Clinical Findings</th>
<th>Imaging Findings</th>
<th>Where to Look?</th>
<th>Complications</th>
</tr>
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<tbody>
<tr>
<td>Descending transtentorial</td>
<td>- Ipsilateral dilated pupil</td>
<td>- Uncus extending into suprasellar cistern</td>
<td>Midbrain</td>
<td>Occipital infarct from PCA compression</td>
</tr>
<tr>
<td></td>
<td>- Contralateral hemiparesis</td>
<td>- Widening of ipsilateral ambient and preoptic cisterns</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(If Kemohar notch is present)</td>
<td>- Widening of contralateral temporal horn</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascending transtentorial</td>
<td>- Nausea</td>
<td>- Spinning top appearance of midbrain</td>
<td>Midbrain and associated cisterns</td>
<td>Hydrocephalus Rapid onset obtundation and possible death</td>
</tr>
<tr>
<td></td>
<td>- Vomiting</td>
<td>- Narrow bilateral ambient cisterns</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Obtundation</td>
<td>- Filling of quadrigeminal plate cistern</td>
<td></td>
<td></td>
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<tr>
<td>Alar (sphenoid)</td>
<td>- None</td>
<td>- Displacement of MCA on axial views</td>
<td>MCA</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Distorted insular cortex on sagittal views</td>
<td></td>
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<tr>
<td>Subfalcine</td>
<td>- Headache</td>
<td>- Asymmetric anterior falx</td>
<td>Septum pellicudum at level of foramen of Monro</td>
<td>Ipsilateral ACA infarction</td>
</tr>
<tr>
<td></td>
<td>- Contralateral leg weakness</td>
<td>- Obliterated ipsilateral frontal horn and atrium of lateral ventricle</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Septum pellicudum shift</td>
<td></td>
<td></td>
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<td>Tonsillar</td>
<td>- Bilateral arm dysesthesia</td>
<td>- Tonsils at level of dens on axial</td>
<td>Foramen magnum on axials and sagittals</td>
<td>Death</td>
</tr>
<tr>
<td></td>
<td>- Obtundation</td>
<td>- Tonsils on sagittal 5mm below foramen magnum (adult), 7mm below (children)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 18:** A. Coronal section of the brain through the midportion of the thalamus. There is right displacement of the cingulate gyrus of the left hemisphere (red arrow), representing subfalcine herniation. B. CT brain shows right frontal epidural hematoma with multiple hemorrhagic contusions in the right frontal lobe causing positive mass effect in the form of right displacement of the cingulate gyrus of the left hemisphere (white arrow), and midline shift, with underlying right frontal bone fracture.
**Fig. 19:** A. The mounted specimen of the brain shows that the left uncus is pushed medially with encroachment on the suprasellar cistern representing left uncal herniation. B. CT brain showing hyperdense area in right temporal lobe representing left intraparenchymal hematoma with positive mass effect in the form of medial displacement of the right uncus medially (white arrow), narrowing of suprasellar cistern and shifting of the midbrain to the opposite site, all representing uncal herniation. Intaventricualr and Subarachnoid hemorrhage are also noted in this patient.

© A. UTAS school of medicine, Discipline of pathology, viewed at: [https://secure.health.utas.edu.au/intranet/cds/pathprac/Files/Cases/CNS/Case85/Case85.htm#Logo](https://secure.health.utas.edu.au/intranet/cds/pathprac/Files/Cases/CNS/Case85/Case85.htm#Logo) B. cairo/EG(Ain shams university-demerdash hospital)
Conclusion

Traumatic brain injury is a catastrophic common event that affects the patient morbidity and mortality, therefore accurate diagnosis is mandatory for precise management of the patient.

Non-enhanced CT brain is the modality of choice, it has a significant role in guiding the management of the patient. Knowing brain anatomy and what we are searching for allows us to accurately detect urgent signs on CT brain, which can save our patients life.
References