Radiología 67 (2025) 331–342

RADIOLOGÍA

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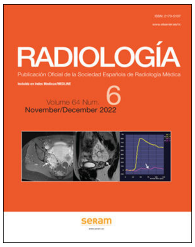

UPDATE IN RADIOLOGY

Imaging findings for severe traumatic brain injury

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Received 22 March 2024; accepted 6 May 2024
Available online 6 May 2025

KEYWORDS
Traumatic brain haemorrhage;
Subarachnoid haemorrhage;
Diffuse axonal injury;
Prognosis;
Brain contusion;
Computed tomography

Abstract Traumatic brain injury (TBI) is the leading cause of morbidity and mortality in young patients. The Marshall classification predicts six-month mortality and divides severe TBI patients into six groups based on CT findings in the acute phase of trauma. MRI also has prognostic value because it detects 30% more traumatic lesions, especially brainstem injury and diffuse axonal injury. Diffuse axonal injury occurs in three different anatomical areas, graded according to severity, and the greater the trauma, the deeper the brain involvement extends. Traumatic brainstem injuries with the worst prognosis are those of posterior location, with bilateral or haemorrhagic involvement. This article analyses the prognostic value of CT and MRI in the assessment of severe TBI and describes the main intracranial traumatic injuries.

PALABRAS CLAVE
Hemorragia cerebral traumática;
Hemorragia subaracnoidea;
Lesión axonal difusa;
Pronóstico;
Contusiones;
Tomografía computarizada

Hallazgos de imagen en el traumatismo craneoencefálico grave

Resumen El traumatismo craneoencefálico (TCE) es la principal causa de morbimortalidad en jóvenes. La clasificación de Marshall predice mortalidad a 6 meses y divide a los pacientes con TCE grave en 6 grupos en función de los hallazgos de la TC en la fase aguda del trauma. La RM tiene valor pronóstico ya que detecta un 30% más de lesiones traumáticas, sobre todo lesiones del tronco cerebral y lesión axonal difusa. La lesión axonal difusa asienta en 3 áreas anatómicas diferentes, su gradación varía en función de la severidad, y la afectación cerebral se extiende de manera progresiva, en zonas más profundas cuanto mayor es el trauma. Las lesiones traumáticas del tronco cerebral que asocian peor pronóstico son aquellas de localización posterior, con afectación bilateral o hemorrágicas. Este artículo analiza el valor pronóstico de la TC y RM en la valoración del TCE grave, y describe las principales lesiones traumáticas intracraneales.

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Introduction

Traumatic brain injury (TBI) is especially prevalent among young males and continues to be a major contributor to morbidity and mortality worldwide.

Trauma severity is classified using the Glasgow Coma Scale (GCS) which evaluates a patient's condition based on eye opening, verbal and motor responses. The combined score of those responses provides a scale ranging from 3 to 15.

Severe TBI makes up approximately 10% of all cases and is characterised by a GCS score below 8.[1,2]

Injury mechanisms vary depending on patient age and head trauma severity. In our setting, road traffic accidents constitute the most common cause of severe TBI, followed by falls from varying heights.[3]

TBI is a dynamic process that is characterised by progressive brain damage and evolving pathophysiology over several hours. The first phase refers to the initial brain injury which results directly from the mechanical impact of the trauma. The second phase can last from several days to weeks and is driven by a combination of metabolic, molecular, inflammatory and vascular processes that act synergistically.[1,3]

The aim of this paper is to review imaging indications in severe TBI, describe the main primary and secondary traumatic lesions, and analyse their prognostic significance.

The role of imaging in initial assessment of severe TBI

Non-contrast computed tomography (CT) is the preferred imaging technique for the initial assessment of TBI and it should be performed in all patients with a GCS score below 13 (Class I recommendation).[4–6] It is highly sensitive in detecting traumatic cerebral haemorrhage but it has certain limitations as regards:[7]

- the diagnosis of early-stage or small parenchymal contusions;
- the diagnosis of diffuse axonal injury (DAI) and non-haemorrhagic lesions; and
- the assessment of ischaemic changes secondary to oedema and intracranial hypertension.

Several authors have discussed the prognostic value of initial CT findings from imaging performed in the acute phase of trauma, particularly within the first 24 h.

In the 1980–90s, Marshall et al. published the Traumatic Coma Data Bank (TCDB), proposing a universally accepted classification of CT findings in severe trauma in order to divide patients into the following six groups[8,9] (Fig. 1):

- *Diffuse injury I*: No visible lesions.
- *Diffuse injury II*: High/mixed density lesion with a volume < 25 cc but no compressed cisterns or midline shift > 5 mm.
- *Diffuse injury III or swelling*: Cisterns compressed or absent. No midline shift > 5 mm or high/mixed density lesion > 25 cc.
- *Diffuse injury IV or swelling with shift*: Cisterns compressed, midline shift > 5 mm but no high/mixed density lesion > 25 cc.
- *Injury V or evacuated mass lesion*: Any haemorrhagic lesion surgically evacuated.
- *Injury VI or non-evacuated mass lesion*: High/mixed density lesion > 25 cc, not surgically evacuated.

The Marshall classification identifies patients at higher risk of intracranial hypertension and estimates the risk of death, as well as the likelihood of a favourable or unfavourable outcome. However, it does not provide a more specific prognostic assessment. This classification predicts six-month mortality but does not describe the type of lesion, assess subarachnoid haemorrhage (SAH) or provide clear indications for surgery.

The Rotterdam classification, introduced in 2005, builds on the Marshall classification by adding subarachnoid and intraventricular haemorrhage to the previously established prognostic factors of mass effect and midline shift.[10] However, it does not measure haemorrhage volume, it makes no reference to contusions and within mass-effect haemorrhagic lesions, it only refers to epidural haematomas (Table 1).

Both the Marshall and Rotterdam classifications are effective tools for predicting mortality in severe TBI. The mortality risk associated with each type of lesion varies depending on different series but, in general, it increases proportionally with the severity grade. Thus, higher scores in both classifications are associated with increased mortality in adults with severe TBI.[11] An exception to this is that Marshall type V lesions can have a better prognosis than type VI lesions due to the surgical evacuation of the haematoma.

In patients with severe TBI, serial follow-up CT scans have been shown to provide better prognostic correlation than a single initial CT scan.[12] This is because CT findings will evolve for more than 50% of patients, either spontaneously or after surgical interventions. These changes are particularly important in the first 48 h.

The main risk factors associated with the growth of traumatic intracerebral haemorrhage are: advanced age, decompressive craniectomy, cisternal compression, falling as the mechanism of injury, multiple haemorrhages, lesion volume < 5 cc and hypoxia.[13]

Follow-up CT images reveal changes in approximately 20% of patients with diffuse injury types II, III and IV of the Marshall classification. An injury usually progresses between six and nine hours after the trauma and more significant change is observed when the initial CT scan is performed within the first two hours.[14]

If the first CT scan is performed very early, new traumatic lesions will appear in later imaging for 50% of patients. Therefore, regardless of whether intracranial traumatic lesions are detected on initial imaging, the recommendation is to repeat the CT scan within six hours, especially if coagulation abnormalities are present.

Additionally, a CT angiography should be performed if there is suspicion of vascular injury and one of the following conditions is present: 1) a fracture line that crosses the dural sinuses or the carotid canal, 2) penetrating trauma, 3) post-traumatic ischemic lesions, 4) a neurological deficit not explained by conventional imaging tests, or 5) an atypical haemorrhage pattern for TBI (for example, cisternal haem-

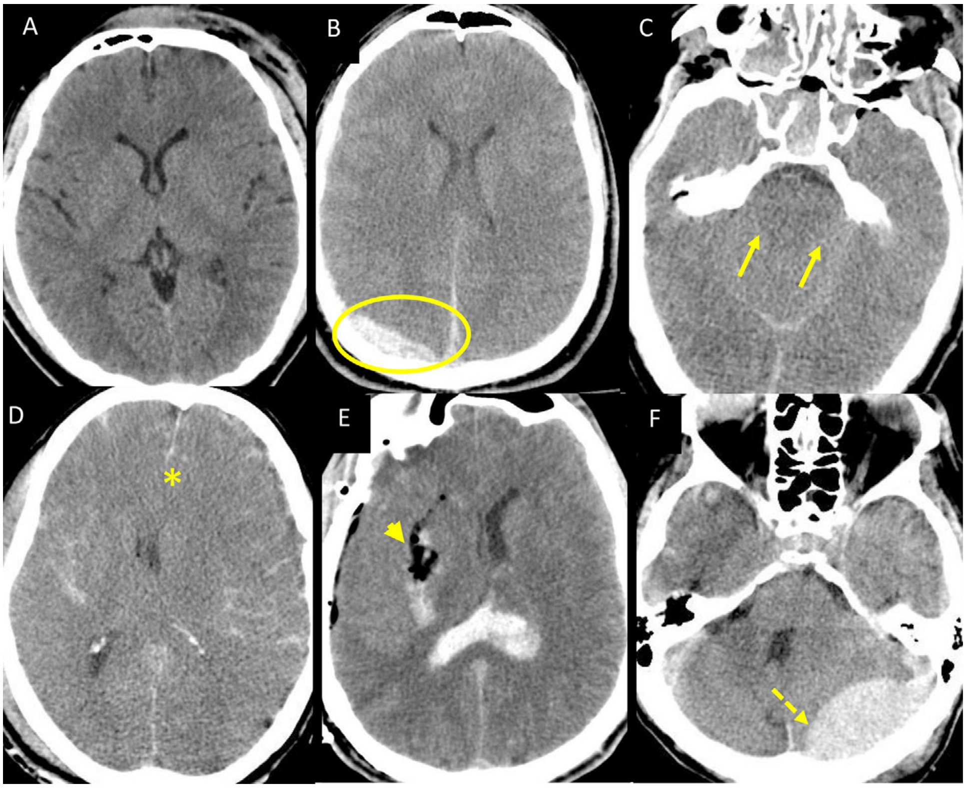

Figure 1 Marshall classification or Traumatic Coma Data Bank of computed tomography (CT) findings in the acute phase in patients with severe traumatic brain injury (TBI). Diffuse injury type I (A): soft tissue haematoma with no visible intracranial traumatic lesion. Diffuse injury type II (B): epidural haematoma < 25 cc (circle) with no cisterns compressed or midline shift. Diffuse injury type III (C): subarachnoid haemorrhage (SAH) with cisterns compressed (arrows). Diffuse injury type IV (D): SAH with midline shift > 5 mm (asterisk). Diffuse injury type V (E): surgically treated cerebral laceration in the right basal ganglia (arrowhead). Diffuse injury type VI (F): epidural haematoma in the posterior fossa > 25 cc, not surgically drained (dashed arrow).

orrhage surrounding the vascular structures of the circle of Willis).[4,15,16]

Magnetic resonance Imaging in traumatic brain injury Imaging indications and protocol

Magnetic resonance imaging (MRI) is not widely used in the study of TBI. Except in research studies, MRI is not indicated in mild TBI (Class IIb recommendation).[6] In the acute phase, it can detect small extra-axial haematomas and small intra-axial haemorrhagic lesions that can go unnoticed on CT. However, these lesions are managed conservatively and generally do not affect a patient's final prognosis.

In patients with moderate or severe TBI, cranial MRI is indicated (Class I recommendation) when CT findings do not justify the patient's clinical condition (such as focal neurological deficit or prolonged states of unconsciousness) and when there are indirect signs of DAI, such as intraventricular haemorrhage.[6,17,18]

MRI has prognostic significance, revealing up to 30% more traumatic lesions—especially brainstem injuries and DAI—which are associated with patient outcomes, though their impact is usually observed beyond the acute phase.

Ideally, MRI should be performed during the subacute phase of trauma, within the first two weeks, as this is when oedema associated with areas of axonal disruption is at its peak.

The majority of traumatic lesions can be identified when imaging is acquired in at least two spatial planes using T1-weighted, T2-weighted, FLAIR, diffusion (DWI), and susceptibility-weighted (SWI) or T2*-weighted sequences.[5,19]

DAI is often associated with small tissue tears accompanied by microhaemorrhages. Therefore, SWI sequences—which are more sensitive than T2*[17,20]—are essential for the assessment of cranial trauma.

DWI sequences are particularly useful for diagnosing non-haemorrhagic DAI in the acute phase, as it appears as focal areas of diffusion restriction.

T2-weighted sequences have high prognostic value due to their greater sensitivity in detecting posterior fossa lesions.

FLAIR sequences are more sensitive for the detection of small cortical contusions, laminar subdural haematomas, hemispheric DAI at the subcortical level, and SAHs.[19,21]

Diffusion tensor imaging (DTI) has high sensitivity for detecting microstructural injury associated with cranial trauma. In TBI-related white matter injury, axonal disruption and demyelination lead to elevated mean diffusivity and reduced fractional anisotropy.[22–24] However, DTI is currently limited to research studies due to its considerable variability. Furthermore, there are no defined criteria for normality

Table 1 Marshall and Rotterdam classifications of CT findings in severe trauma.

Marshall classification	Rotterdam classification
Diffuse injury type I	Basal cisterns
	0: normal
No visible lesions	1: compressed
	2: absent
Diffuse injury type II	Midline shift
High/mixed density lesion < 25 cc	
No cisterns compressed	0: ≤ 5 mm
No midline shift > 5 mm	1: > 5 mm
Diffuse injury type III or swelling	Epidural haematomas
High/mixed density lesion < 25 cc	
Cisterns compressed or absent	0: present
No midline shift > 5 mm	1: absent
Diffuse injury type IV or swelling with shift	Intraventricular haemorrhage or traumatic subarachnoid haemorrhage (SAH)
High/mixed density lesion < 25 cc	
Cisterns compressed or absent	0: absent
Midline shift > 5 mm	1: present
Evacuated mass lesion 5	
Any lesion evacuated surgically	
Non-evacuated mass lesion 6	
High/mixed density lesion> 25 cc not surgically evacuated	

With the Rotterdam scale, the final score is the sum of all the items + 1. Both classifications predict mortality, and in general, the risk of death associated with severe traumatic brain injury increases with higher grades.
CT: computed tomography.

and abnormality; quantitative analysis is constrained by MRI technique and post-processing; and it is a sequence that is difficult to interpret on an individual basis.[7]

Perfusion CT and MRI sequences (including T2* and arterial spin labelling techniques) enable the calculation of cerebral blood flow; they allow the identification of global hypoperfusion or areas at risk of hypoperfusion, which are associated with poorer prognosis; and they detect cortical contusions at early stages with greater sensitivity than non-contrast CT. However, they are not used in routine practice.[7,25]

Cranial MRI findings in the subacute phase of trauma contribute to enhancing the value of existing prognostic models for moderate and severe TBI.[26] These models are useful for identifying patients at higher risk of poor long-term outcomes, as they incorporate MRI and laboratory findings alongside traditional prognostic predictors such as age, pupillary reactivity and motor response. Two of the main prognostic models in TBI are the IMPACT model, which incorporates the location and extent of DAI on MRI,[27,28] and the TRACK-TBI model, which provides an integrated diagnosis of trauma by including blood biomarkers of inflammation, coagulation, homeostasis, and structural proteins.[29,30]

Primary traumatic lesions

Epidural haematomas

Epidural haematomas are frequently associated with fractures (>90%)[18] and are classified as either arterial (90–95%) or venous (5–10%), depending on the source of bleeding[5] (Fig. 2).

Arterial epidural haematomas result from injury to the middle meningeal artery and are most commonly located supratentorially in the region of the pterion and the middle cranial fossa.[5,19]

Venous epidural haematomas result from injury to a dural venous sinus or a cortical vein. They may cross sutures and their typical locations are:[19]

- *Anterior temporal,* due to injury to the sphenoparietal venous plexus.
- *Vertex,* due to injury to the superior sagittal sinus.
- Posterior fossa.
- *Retroclival*, less common, resulting from injury to the basilar venous plexus in clivus fractures or from tearing of the tectorial membrane due to flexion-extension mechanisms in paediatric cervical trauma.

Subdural haematomas

These extra-axial haematomas are caused by the rupture of bridging veins in the subdural space.[17,19] If the arachnoid is involved in the traumatic injury, cerebrospinal fluid (CSF) accumulates in the subdural space, resulting in a hygroma which typically appears around nine days after the TBI.[5,17]

It is not uncommon for traumatic injury to involve both the bridging veins and the arachnoid. As a result, these extra-axial collections may have a mixed component due

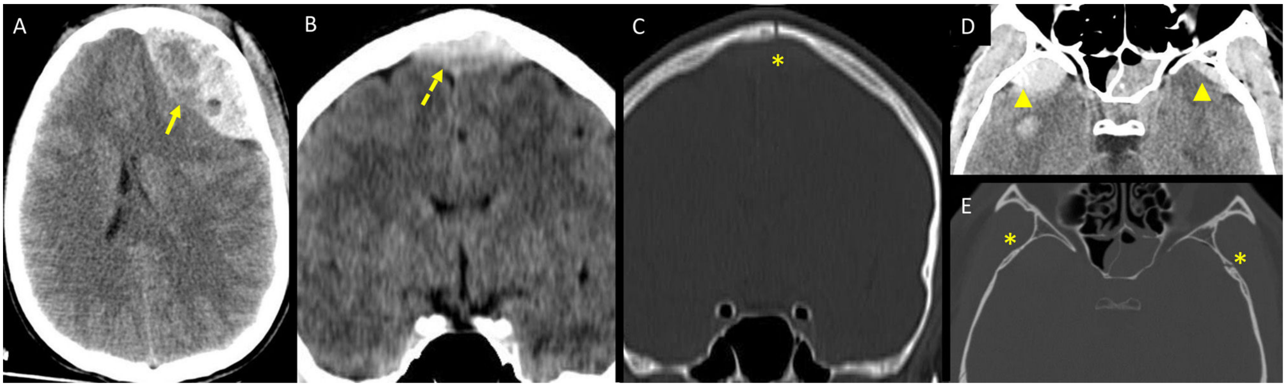

Figure 2 Epidural haematoma of arterial origin (A) (arrow) caused by injury to the middle meningeal artery, and of venous origin (dashed arrow) due to damage to the superior sagittal sinus (B and C) and the sphenoparietal plexus (arrowhead) (D and E), associated with a fracture (asterisks in C and E).

to the combination of CSF and haemorrhage within the subdural space.[4,17,19]

In young patients, there is sometimes notable swelling linked to small subdural haemorrhages and this can be explained by the involvement of the trigeminovascular system. The nerve fibres that originate from the ganglion cells of the trigeminal nerve are the initial sensors in cranial trauma. Through collateral innervation of intracranial and dural vessels, they can trigger a cascade of vascular responses with cerebral swelling[31] (Fig. 3).

Some extra-axial haematomas—including both subdural and epidural types—may exhibit a haematocrit level commonly observed in coagulopathies or a central hypodensity. This is also known as the 'swirl sign' and is indicative of active bleeding.[17]

Subarachnoid and intraventricular haemorrhage

The most common location for subarachnoid haemorrhage is in the convexity sulci. This may result from the rupture of cortical veins, be secondary to the extension of haemorrhagic contusions or occur due to the redistribution of intraventricular haemorrhage.[4,32]

Intraventricular haemorrhage may result from the rupture of subependymal veins. It is an indirect sign of traumatic injury to the corpus callosum and a marker of DAI.[4] On the initial CT scan, midline SAH affecting the interhemispheric fissure or perimesencephalic region is also a marker of DAI.[5]

A key location to consider is the posterior fossa, as SAH in the interpeduncular or perimesencephalic cisterns is associated with traumatic injury to the brainstem.

While it is less common than in aneurysmal SAH, traumatic SAH is also associated with vasospasm. In severe TBI, the risk of post-traumatic vasospasm can be as high as 40% and it is a late complication that typically occurs 7–10 days after the trauma.[5,17]

Cortical contusions

Cortical contusions are the most common intra-axial traumatic injury and are secondary to the impact of the cerebral parenchyma against the diploë, falx, and tentorium.[19] They are mostly haemorrhagic and primarily affect the cortical surface of the temporal lobes, perisylvian region, and orbitofrontal area.[4,19]

Research has shown a link between the site of impact and the location of intracranial haemorrhage in patients with moderate to severe TBI. In coup injuries, frontal impacts result in haemorrhages located in the frontal region. In contrecoup injuries, posterior impacts are associated with haematomas located in the temporal region. These contrecoup injuries are an independent risk factor for the progression of traumatic intracerebral haemorrhage[33] (Fig. 4).

In the early stages, contusions appear as hypodense areas (due to oedema), with or without patchy areas of haemorrhage. Up to 75% of cortical contusions will show radiological progression within the first 24–48 h. This progression is characterised by an increase in, or the development of, new areas of oedema and haemorrhage, along with worsening mass effect.[5]

Other cerebral contusions associated with high-energy trauma include: (Fig. 5)

- *Mixed contusions* involving the deep white matter and basal ganglia.[19]
- *Gliding contusions,* which are caused by acceleration–deceleration mechanisms and are characterised by the involvement of the superior cortical surface and parasagittal regions of the cerebral hemispheres.

Diffuse axonal injury (DAI)

Whereas focal lesions such as cortical contusions or extra-axial haematomas can be adequately assessed with CT, diffuse lesions, which are associated with higher morbidity and mortality, are better characterised with MRI.

DAI may be haemorrhagic, non-haemorrhagic or a combination of both.[5]

It typically involves three anatomical areas and is graded based on severity. The injury extends progressively into deeper brain structures as trauma severity increases.[4]

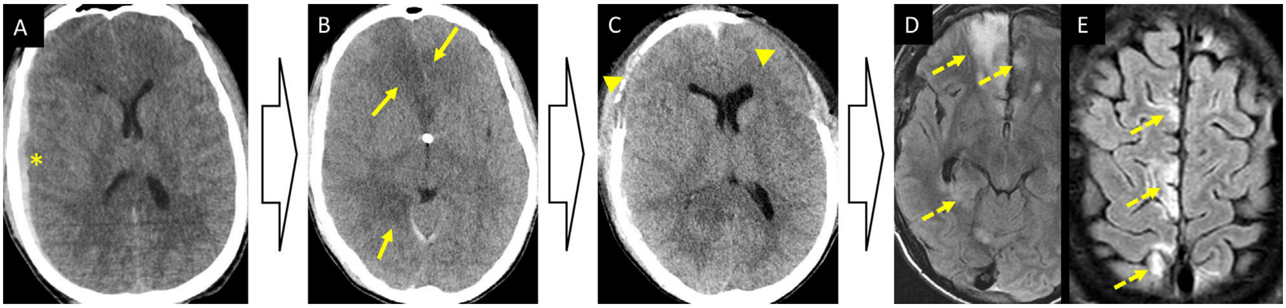

Figure 3 Swelling associated with subdural haematomas in young patients. Sixteen-year-old male with multiple trauma, showing a right convexity subdural haematoma on the initial CT scan (asterisk) (A). Follow-up CT performed seven days after the trauma due to increased intracranial pressure (B) shows diffuse swelling and hypodensities suggestive of ischaemic lesions in the regions of both anterior cerebral arteries and the right posterior cerebral artery (arrows). Follow-up CT after bifrontal decompressive craniectomy (arrowheads) (C) shows improvement in cerebral swelling, while MRI performed during the subacute phase of trauma (D and E) better delineates the extent of ischaemic lesions secondary to swelling and intracranial hypertension (dashed arrows in D and E).

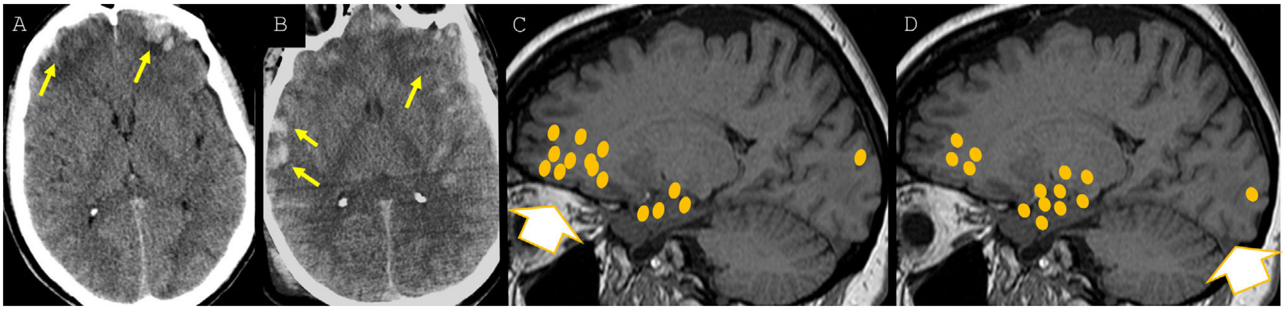

Figure 4 Typical frontotemporal location of haemorrhagic cortical contusions (arrows in A and B). Predominant frontal pattern in coup injuries (yellow dots in C) and temporal pattern in contrecoup injuries (yellow dots in D).

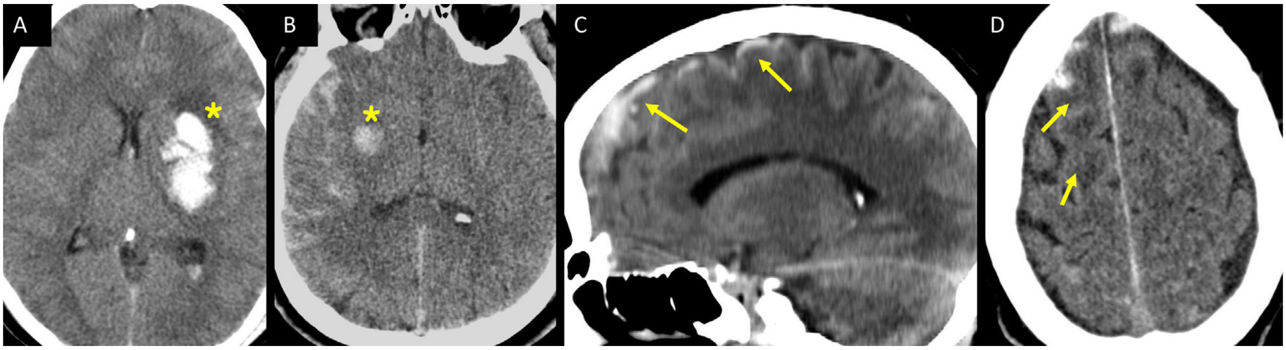

Figure 5 Mixed contusions with deep haematomas associated with high-energy trauma (asterisks in A and B). Patient with multiple trauma and gliding contusions located on the parasagittal and superior cortical surface of both frontal lobes (arrows in C and D).

DAI may be visualised on CT through direct signs such as petechial haemorrhages or indirectly via findings such as intraventricular or perimesencephalic haemorrhage.

Based on findings described by Adams et al.,[34] and later adapted by Gentry et al.,[35,36] DAI is classified into the following types (Fig. 6):

- *DAI type 1:* involves the white matter of the cerebellum and the lobar hemispheric white matter, predominantly in the anterior regions but particularly in the frontal and temporal lobes.
- *DAI type 2:* involves the corpus callosum, particularly the splenium and posterior body, as well as the paraventricular regions (fornix). Subcallosal haemorrhage has been described as a marker of DAI.[38]
- *DAI type 3:* involves the cerebellar peduncles and the dorsolateral region of the pontomesencephalic junction.

The number of DAI-associated microhaemorrhages has been found to correlate with the initial GCS score. In addition, a clear association has been reported between depth of intracranial traumatic brain injury and six-month prognosis, as determined by various clinical scales.[39,40] Ultimately,

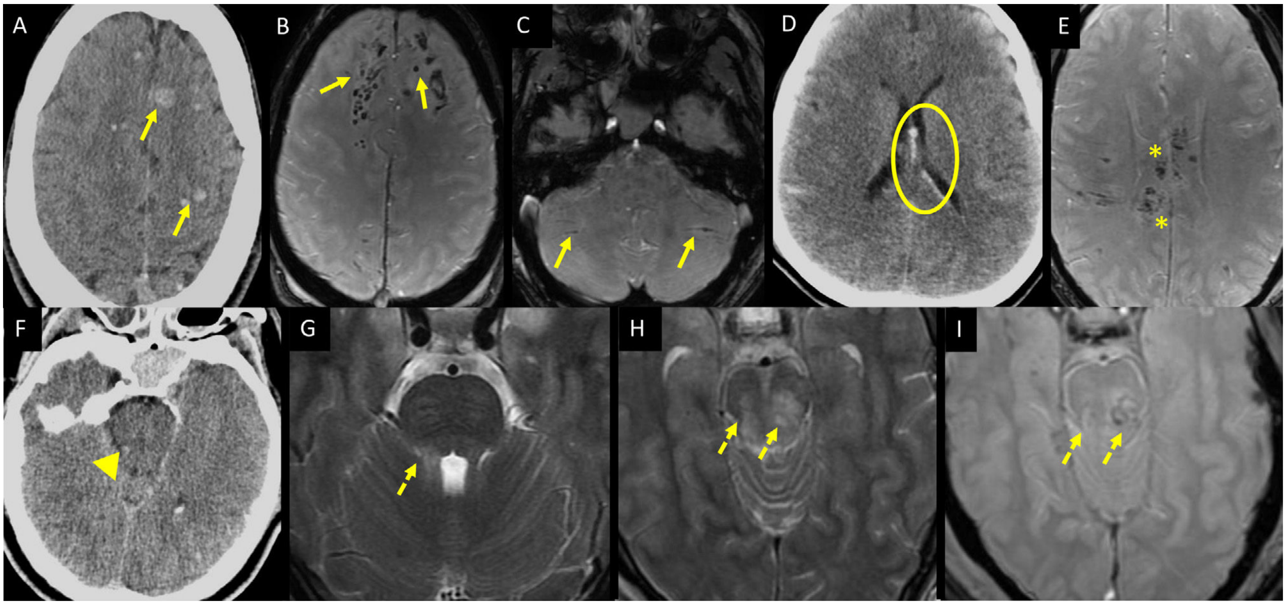

Figure 6 Gentry & Adams classification of diffuse axonal injury (DAI); CT and MRI findings. DAI type 1: CT shows petechial haemorrhages (A), while SWI reveals microhaemorrhages in the lobar regions and cerebellar white matter (arrows) (B and C). DAI type 2: CT shows intraventricular and corpus callosum haemorrhage (circle) (D), while MRI reveals microhaemorrhages in the corpus callosum and paraventricular structures (asterisks) (E). DAI type 3: CT shows microhaemorrhages (arrowhead) (F), while MRI demonstrates microhaemorrhages in the cerebellar peduncles (dashed arrow) (G) and the dorsolateral midbrain (dashed arrows) (H and I).

greater injury depth is linked to poorer outcomes. Although lesion volume and the location of DAI may influence neurological outcome, patients with TBI and type 3 DAI generally have a poorer prognosis and higher mortality.

Corpus callosum involvement in DAI is generally associated with high-energy trauma and indicates a worse prognosis.[37] However, not all patients with type 2 DAI have an unfavourable outcome. The prognosis will depend on both the location and volume of the lesion. As the lesion increases in size, the prognosis becomes less favourable. Within the corpus callosum, the most common site of involvement is the splenium, which tends to indicate a worse prognosis due to its association with dorsal brainstem injury.[41]

Technical advances in MRI equipment now enable the diagnosis of axonal disruption injuries that are smaller and associated with milder trauma. In relation to these technical improvements, small focal hyperintense lesions on T2 or FLAIR should be considered non-specific, unless they show diffusion restriction or microhaemorrhage, which supports the diagnosis of DAI over chronic ischaemic microangiopathy.[17]

Traumatic brainstem injuries

Due to its prognostic significance, one of the main indications for MRI in TBI patients is the assessment of brainstem injuries.

Primary Traumatic brainstem injuries are classified as those which occur as a direct consequence of the trauma, otherwise they are classified as secondary.

Primary lesions are classified into the following categories:[42,43] (Fig. 7)

- *Direct superficial lacerations or contusions*: resulting from the brainstem impacting against the lateral tentorial notch. They originate at the surface of the brainstem and are mostly non-haemorrhagic.
- Multiple petechial haemorrhages.
- *Diffuse axonal injury* located in the posterior and lateral regions of the pontomesencephalic junction, near the region of the tectum and periaqueductal grey matter.
- *Pontomedullary rent*, rarely seen on imaging as complete injury typically results in immediate death at the time of trauma.[44] In cases of incomplete injury, MRI may show focal hyperintense lesions with diffusion restriction involving the pontomedullary junction.

It has traditionally been thought that traumatic brainstem injuries almost always lead to a poor prognosis and a reduced chance of recovery from a persistent vegetative state. These conclusions support the Ommaya-Genarelli model which links the severity of head trauma with morbidity and mortality.[45–47]

However, subsequent studies have shown that not all patients with traumatic brainstem injuries have an unfavourable outcome and that prognosis depends on the type of injury.[48] Therefore, brainstem injuries associated with a worse prognosis are those that are located posteriorly, involve both sides, or are haemorrhagic.[49]

Traumatic vascular injuries

Traumatic vascular injuries are less common, with an incidence of approximately 0.8–1.7%.[50]

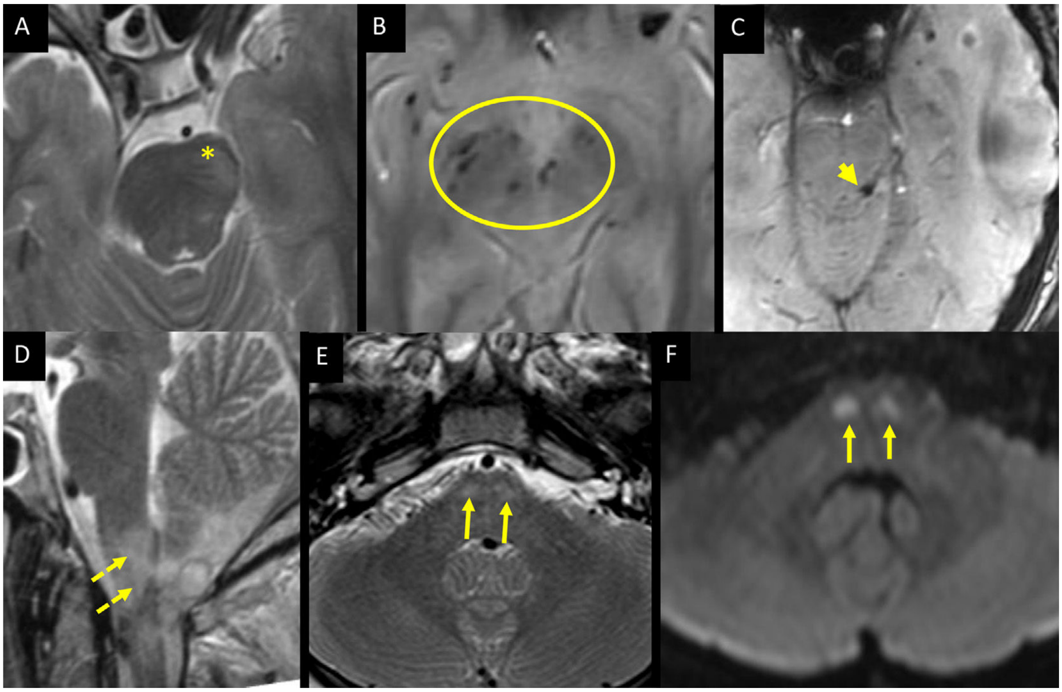

Figure 7 Primary traumatic brainstem injuries: direct superficial laceration or contusion (asterisk) (A), multiple petechial haemorrhages (circle) (B), diffuse axonal injury (arrowhead) (C) and pontomedullary rent which may be complete (dashed arrows) (D) or incomplete (arrows) (E and F).

The main risk factor for carotid injury is a GCS score of 6 or below, while the primary risk factor for vertebral artery injury is the presence of cervical trauma.[16,32]

According to the Biffl classification,[51] which is based on angiographic findings, traumatic cerebrovascular injuries are divided into the following categories:

Grade 1: mild intimal injuries with no flap, and with intramural haematoma with < 25% luminal stenosis.

Grade 2: intimal injury with flap, intraluminal thrombus and subintimal haematoma with ≥ 25% luminal stenosis.

Craniocervical arterial dissection (Biffl grades 1 and 2) occurs when the fracture line crosses the skull base. It may be intra- or extracranial and it causes stenosis, occlusion or pseudoaneurysms.[19] In decreasing order of frequency, it affects the cervical segment of the internal carotid artery just below the petrous canal, the V3 segment of the vertebral arteries at the level of C1–C2, and the V4 segment of the vertebral arteries in cases of intracranial traumatic dissection.[16,43] (Fig. 8)

Grade 3: pseudoaneurysm with or without stenosis. The two main locations are the vertebral artery and the anterior cerebral artery. The anterior cerebral artery bifurcation into the pericallosal and callosomarginal arteries is particularly prone to vascular injury due to its proximity to the falx cerebri.[43]

Grade 4: traumatic occlusion.

Grade 5: vascular transection with active bleeding. Some authors include arteriovenous fistulas within this grade, with the most common location being the cavernous sinus (carotid–cavernous fistula, frequently associated with skull base fractures).[16] (Fig. 9)

Venous thrombosis may also occur as a consequence of TBI when the fracture crosses a dural sinus or the jugular bulb.

Secondary traumatic lesions

Diffuse cerebral oedema

Diffuse cerebral oedema or swelling may be focal or diffuse and it occurs 24–48 h after head trauma in approximately 10–20% of patients.[4] While the underlying mechanisms remain unclear, it is thought to result from a combination of cytotoxic and vasogenic oedema, accompanied by a secondary increase in vascular permeability.[4,5] Diffuse cerebral oedema also leads to increased intracranial pressure, decreased perfusion, and ischaemia or infarction of the brain parenchyma.[5]

Lesions secondary to brain herniation

Brain herniation is the displacement of brain parenchyma from one cranial compartment to another due to mass effect caused by the primary traumatic injury.[4] (Fig. 10)

Subfalcine herniation is the most common type of herniation and occurs as a result of displacement of the anterior cingulate gyrus beneath the falx cerebri. In severe cases, it is associated with infarction of the anterior cerebral artery due to compression of the pericallosal arteries and contralateral ventricular dilatation caused by obstruction of the foramen of Monro.[43]

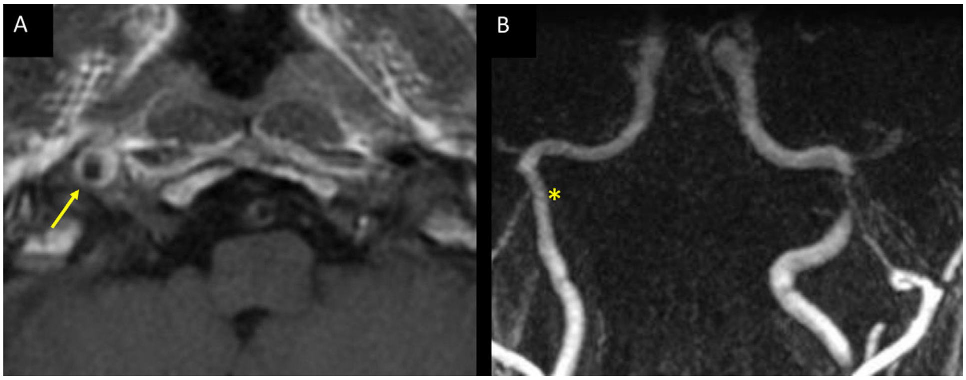

Figure 8 Craniocervical arterial dissection. Axial T1-weighted sequence (A) showing a subintimal haematoma (hyperintense, subacute phase) in the arterial wall of the cervical segment of the right internal carotid artery (arrow), causing luminal irregularity and narrowing (asterisk) on MR angiography of the supra-aortic trunks (B).

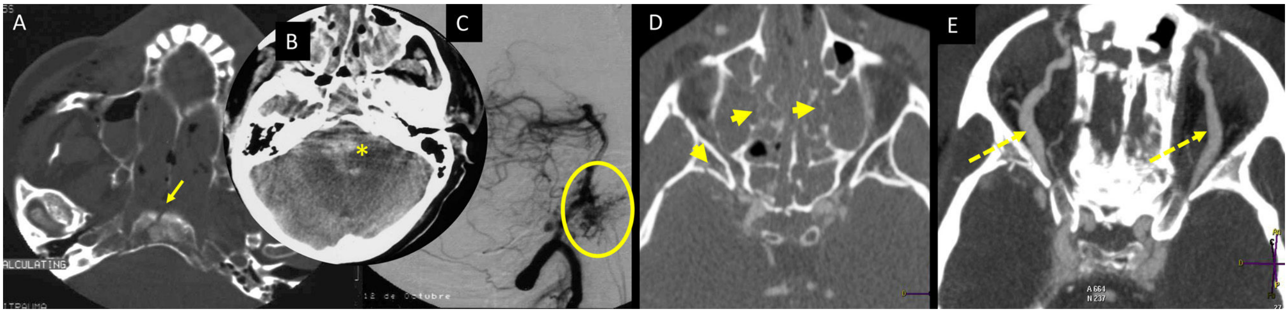

Figure 9 Biffl grade 5 traumatic cerebrovascular injuries. Facial and skull base fracture (arrow) (A) with SAH (asterisk) (B) and contrast extravasation on conventional angiography (circle) (C) due to traumatic rupture of the right vertebral artery. Bilateral traumatic carotid–cavernous fistula (D and E) with dilatation of both ophthalmic veins (dashed arrows), secondary to complex craniofacial fracture (arrowheads).

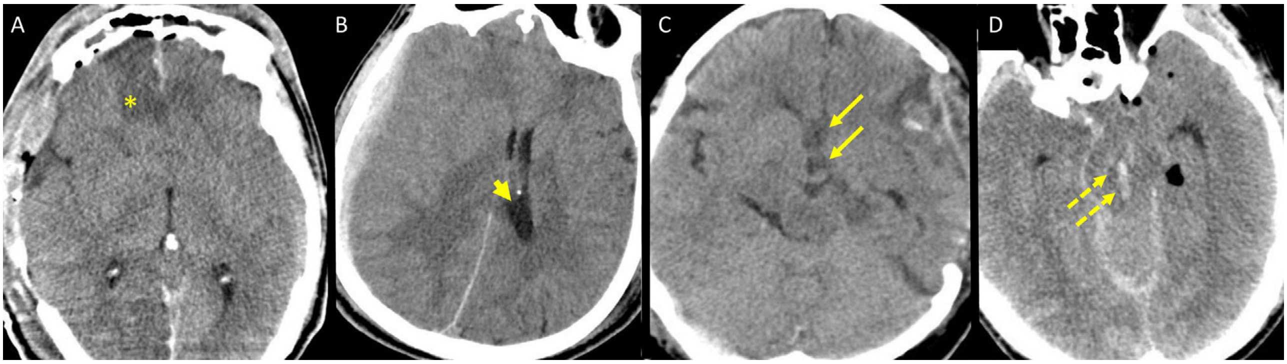

Figure 10 Traumatic injuries secondary to brain herniation. Anterior cerebral artery infarction (asterisk) (A) and unilateral hydrocephalus (arrowhead) (B) due to obstruction of the foramen of Monro, secondary to subfalcine herniation. Hypothalamic infarctions (arrows) (C) and central pontine haemorrhage or Duret haemorrhage (dashed arrows) (D), secondary to bilateral descending transtentorial herniation.

Descending transtentorial herniation is the second most common type and occurs due to downward displacement of brain parenchyma through the tentorial notch. When asymmetric or unilateral, it is associated with infarction in the territory of the posterior cerebral artery. Bilateral herniation leads to hypothalamic and basal ganglia infarctions due to the involvement of the perforating arteries of the circle of Willis. Secondary to herniation, non-communicating hydrocephalus may also occur as a result of compression of the quadrigeminal cistern and obliteration of the cerebral aqueduct.[32]

Tonsillar herniation involves the downward displacement of the cerebellar tonsils and the medial region of the cerebellar hemispheres through the foramen magnum. Notable complications include obstructive hydrocephalus due to involvement at the level of the fourth ventricle and cerebellar infarctions in the region of the posterior inferior cerebellar artery.[4,17,19,43]

Secondary traumatic injuries of the brainstem

Secondary traumatic brainstem injury may present with a range of characteristic findings including:[42,43]

- *Duret haemorrhage or central pontine haemorrhage*: due to increased intracranial pressure or descending transtentorial herniation. It is caused by stretching and/or rupture of pontine perforating branches resulting from brain herniation.[52]
- Focal infarctions due to vascular involvement.
- Compression, displacement and deformity of the brainstem due to mass effect.
- Necrosis secondary to transtentorial herniation.
- Diffuse hypoxic-ischaemic brain injury.

Hydrocephalus secondary to decompressive craniectomy

Following a decompressive craniectomy, TBI can lead to changes in intracranial pressure dynamics and disruption of CSF circulation. The condition is believed to progress in two phases. The first is marked by increased intracranial pressure on the falx due to mass effect, resulting in contralateral displacement of the cerebral parenchyma. The second occurs after decompressive craniectomy, when the midline shift resolves but a suction effect occurs, giving rise to an interhemispheric hygroma. This interhemispheric hygroma reflects distortion of CSF circulation and the subsequent development of hydrocephalus.

Although intraventricular haemorrhage could explain the pathophysiology of post-traumatic hydrocephalus, decompressive craniectomy is an independent risk factor in the development of communicating hydrocephalus.[53]

Conclusion

In patients with severe TBI, CT performed during the acute phase of trauma guides surgical management by identifying the type of lesion and its potential complications. MRI provides prognostic information and is typically performed in the subacute phase of trauma in patients with a GCS score below 13. It enables detailed characterisation of diffuse injuries and brainstem involvement. With the ongoing advancement of MRI technology, the future of head trauma lies in a new DAI classification that is based on MRI findings and offers improved prognostic correlation, particularly for DAI types 1 and 2.

CRediT authorship contribution statement

1 Study concept and design, data acquisition and data analysis and interpretation: not applicable.
2 Drafting of the article and critical revision of the intellectual content: Amaya Hilario, Elena Salvador and Ana Ramos.
3 Final approval of the manuscript: Amaya Hilario, Elena Salvador, Zhao Hui Chen, Agustín Cárdenas, Juan Romero and Ana Ramos.

Funding

This research has not received funding support from public sector agencies, the business sector or any non-profit organisations.

Declaration of competing interest

The authors declare there are no conflicts of interest.

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