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EMERGENCY RADIOLOGY SPECIAL FEATURE: REVIEW ARTICLE

MDCT imaging of traumatic brain injury

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ABSTRACT

The aim of emergency imaging is to detect treatable lesions before secondary neurological damage occurs. CT plays a primary role in the acute setting of head trauma, allowing accurate detection of lesions requiring immediate neurosurgical treatment. CT is also accurate in detecting secondary injuries and is therefore essential in follow-up. This review discusses the main characteristics of primary and secondary brain injuries.

INTRODUCTION

Traumatic brain injury (TBI) is a major health issue responsible for considerable mortality and long-term morbidity worldwide, especially among subjects under the age of 44 years.¹ The incidence of traumatic craniocerebral injuries is approximately 1.6 million per year in the USA, resulting in >50,000 deaths and >70,000 patients with permanent disability.^{2,3}

The aim of emergency imaging is to detect treatable lesions before secondary neurological damage occurs.⁴ There is evidence that prompt neurosurgical management of TBI can significantly improve outcome, especially if decompression is performed within 48 h of injury.^{5–7}

CT is the imaging modality of choice for evaluation of the acute head-injured patient. It is quick, non-invasive and widely available and has few contraindications. CT advantages for assessment of TBI include its sensitivity for demonstrating acute intra-axial and extra-axial haemorrhage, mass effect, ventricular size and bone fractures. Limitations include low sensitivity in detecting small non-haemorrhagic lesions such as cortical contusions and diffuse axonal injuries (DAIs), as well as in early demonstration of hypoxic–ischaemic encephalopathy.⁸

This review discusses the role of imaging in TBI with special focus on CT and illustrates the main characteristics of primary and secondary brain injuries.

INDICATIONS FOR CT IN PATIENTS WITH HEAD TRAUMA

The major appropriateness criteria for imaging of acute head trauma include the American College of Radiology

Appropriateness Criteria, the New Orleans Criteria and the Canadian Head CT rules.^{9–11} There is general agreement that patients considered at high or moderate risk for craniocerebral injury should undergo non-enhanced CT (NECT) early on admission. Whether patients with minor head injury should be imaged remains controversial. Clinical risk factors considered as predictive of need for neurosurgical intervention or of clinically important brain damage in patients with mild or minor TBI include failure to reach a Glasgow Coma Scale (GCS) score of 15 within 2 h, the presence of a suspected open skull fracture or a basal skull fracture, vomiting, an age over 60 or 65 years, drug or alcohol intoxication, deficits in short-term memory and seizure.¹² In all children aged less than 2 years, imaging deems to be “very appropriate”, given the difficulties with neurological assessment in this age group.

TBI is an evolving process with biochemical changes occurring within the injured brain that may result in progression of primary lesions or eventually lead to secondary injury. Therefore, the presence of intra- or extra-axial lesions on the initial CT scan warrants close observation and imaging follow-up.¹³ Approximately 25–45% of parenchymal contusions increase in size and 16% of diffuse injuries demonstrate significant evolution with evidence of new mass lesions on subsequent CT (Figure 1).^{14,15} Progression of primary lesions typically occurs within the first 24 h after the baseline CT.¹⁶ CT is also essential in detecting secondary injuries, such as cerebral oedema, ischaemia and herniation.^{17,18}

MRI is seldom performed in the acute setting of TBI because of the complex logistics of patient transport and monitoring, long imaging times and sensitivity to patient

Figure 1. Delayed post-traumatic haemorrhage in a 76-year-old female. (a) Non-enhanced CT (NECT) shows left parietal soft-tissue swelling in the site of impact (arrow) and contrecoup right frontal haemorrhagic contusions (arrows). A small right hemispheric isodense subdural haematoma is also noted (arrowheads). (b) NECT obtained 4 h later depicts a large right frontal intraparenchymal haematoma with severe mass effect and subfalcine herniation (arrow). The patient died shortly after the scan was obtained.

motion. However, MRI is more sensitive than CT for detection of small post-traumatic focal brain lesions.¹⁹ MRI is considered the modality of choice for patients with subacute and chronic TBI and is recommended for patients with acute head trauma when CT fails to explain the neurological findings.

MULTIDETECTOR CT IN TRAUMATIC BRAIN INJURY

In recent years, there have been notable advances in CT technology. Multidetector CT allows near-isotropic voxel acquisition that can be used to generate high-quality two-dimensional multiplanar reformation and three-dimensional (3D) images. Similarly, major advances have been made in image-processing software and hardware.

NECT scans of the brain should be obtained from just below the foramen magnum through the vertex. CT images are usually obtained at 120 kV and 200–400 mA in adults. Rotation time, slice collimation and pitch vary depending on the type of the scanner. CT images can be reconstructed at different thicknesses, using different algorithms. Two series of axial data sets should be obtained, one using soft tissue and one with bone-reconstruction algorithms, which both can be used for interpretation or to create multiplanar or 3D images.²⁰ Transverse reconstructed images are usually 1 mm thick with a reconstruction interval of 0.4 mm.

With the increasingly widespread use of picture archiving and communication system, imaging interpretation is now performed on computer workstations, allowing for optimal window setting. A narrow window width (80–100 HU) is used to view the brain, whereas a slightly wider window (150–200 HU) is recommended to increase contrast between extra-axial collections and the adjacent skull.²¹

In patients with head trauma, multiplanar reformation is recommended to better evaluate fracture lines and orientation of

displaced fragments, especially in patients with complex facial and temporal bone fractures, thus facilitating surgical planning.²² Multiplanar images can be thickened into slabs by means of projectional techniques including average, maximum and minimum intensity projection; surface-shaded display; and volume rendering.²³ 3D reconstructions of the skull are generally limited to difficult cases. The two main techniques for 3D reconstruction of bones are surface-shaded display and volume rendering, which both significantly improve fracture detection when compared with transverse sections alone.^{24–26} 3D angiographic maximum-intensity projections and volume rendering imaging are also essential in vessel imaging interpretation, providing detailed characterization of arterial narrowing or occlusion, and in planning surgical or endovascular intervention.²⁷

These capabilities have considerably enhanced the utility of CT, and CT usage has correspondingly increased. Given the health risk associated with ionizing radiation, new technologies have been developed, aimed at reducing radiation exposure. Currently, it is common practice to adapt radiation level using weight- or size-based protocols, which is also a requirement for American College of Radiology accreditation of paediatric imaging centres.^{28,29} Scanning techniques that depend on patient size include one or more of the following elements: size-dependent beam-shaping filters, manual tube current technique charts, automatic exposure control and optimal tube potential.³⁰ These strategies have proven particularly useful in children.

MULTIDETECTOR CT ANGIOGRAPHY

Craniocervical CT angiography (CTA) is often obtained as part of a whole-body work-up for trauma patients. CTA provides high spatial resolution and allows rapid assessment of penetrating and blunt cerebrovascular injuries (BCVIs). The spectrum of traumatic vascular injuries includes arterial dissection, pseudoaneurysm formation, carotid-cavernous fistula (CCF), dural venous sinus injury or active haemorrhage.³¹

An appropriate screening algorithm for BCVIs allows early detection and treatment, ultimately reducing the associated morbidity and mortality rates.^{32–34} Craniocervical CTA should be obtained in trauma patients meeting the “Modified Denver Criteria”, which can be classified into risk factors, and signs and symptoms. Signs and symptoms include arterial haemorrhage, cervical bruits, expanding neck haematomas, focal neurological deficits, major discrepancies between neurological and imaging findings, and ischaemic stroke on follow-up CT.^{35–37} Risk factors considered as predictors of BCVI include cervical spine fractures, mid-face Lefort II and III fractures, DAI with a GCS score of <6, skull base fractures with involvement of the carotid canal, and near hanging with anoxic brain injury.

Sensitivity of integrated craniocervical CTA protocols in detecting BCVIs has been proven comparable to that of dedicated neck CTA despite venous contamination due to reflux of contrast material and a low signal-to-noise ratio from arm elevation.³⁸

Moreover, the introduction of advanced CT techniques such as dual-energy CT for cervical CTA has allowed more accurate and faster bone subtraction as compared with threshold-based bone subtraction.³⁹ Dual-energy CT allows better visualization of the neck vessels, especially in their intraosseous segments, namely the petrous portions of the carotid arteries and vertebral arteries coursing through the foramina transversaria.⁴⁰

Digital subtraction angiography remains the definitive diagnostic tool for vascular injury because of its high sensitivity and specificity. However, because of invasiveness with a 1.3% complication rate, which includes arterial dissection, thrombosis and stroke, and owing to its limited accessibility, digital subtraction angiography is usually reserved to unresolved cases with dubious imaging findings and high clinical suspicion.^{41,42}

CT FOR PREDICTION OF OUTCOME IN TRAUMATIC BRAIN INJURY

Outcome prediction in TBI has been notably difficult. Mortality correlates with the severity of injury based on the GCS score.^{43,44} TBI has also been classified according to damage severity as demonstrated by neuroimaging. The Marshall CT score was published in 1992 and proved to correlate the presence of intracranial abnormalities on CT with intracranial pressure (ICP) and outcome.⁴⁵ The Marshall CT classification identifies six groups of patients with head trauma based on the following parameters on CT: the presence of a focal mass lesion (evacuated or non-evacuated) and/or diffuse intracranial abnormalities including brain swelling and midline shift. Some authors claim that the Marshall classification, broadly classifying injuries into diffuse and focal categories, fails to distinguish between subtypes of intracranial lesions and suggest that it may be preferable to use combinations of individual CT predictors.^{46,47} The Rotterdam score is a more recent classification system which allows single CT abnormalities to be scored separately and includes two additional parameters: traumatic subarachnoid haemorrhage and intraventricular haemorrhage.⁴⁶ Another classification scheme is the Helsinki CT score, which considers bleeding type and size, intraventricular haemorrhage and suprasellar

cisterns.⁴⁷ Because neurological examination may be unreliable following sedation in patients with severe TBI, these CT scoring systems play a primary role in the early management of brain lesions and in predicting outcome.

CLASSIFICATION OF HEAD INJURY

Craniocerebral lesions can be classified into primary and secondary. Primary lesions occur as a direct result of a trauma to the head and include scalp injuries, skull fractures, extra-axial haemorrhage and intra-axial lesions. Secondary brain injury occurs as a complication of primary lesions and includes ischaemic and hypoxic damage, cerebral oedema and brain herniations. Extra-axial haemorrhage includes epidural haematoma (EDH), subdural haematoma (SDH), and subarachnoid (SAH) and intraventricular (IVH) haemorrhage. Primary intra-axial injuries include DAI, cortical contusions, intraparenchymal haematomas (IPHs) and vascular lesions.

The main imaging characteristics of primary and secondary TBIs are presented below.

PRIMARY INJURY

Fractures

Fractures of the skull are seen as calvarial or skull base disruption on bone algorithm CT images. Skull fractures can be closed or open, simple or comminuted, linear, depressed, elevated or diastatic. Fractures can involve the calvaria, the base of the skull or both.

Major complications that can occur with fractures of the skull base include cerebrospinal fluid (CSF) leakage and infection, cranial nerves damage and injury to the dural sinuses, jugular vein or internal carotid artery (ICA).⁴⁸ The highest incidence of carotid injury has been associated with fractures involving the petrous segment of the carotid canal, whereas the most frequently fractured portion of the carotid canal is represented by the lacerum-cavernous junction.⁴⁹

Thin-section (1-mm) multidetector CT with bone algorithm and 3D reconstructions is recommended for the evaluation of fractures in the skull base, orbit and facial bones.⁵⁰

The presence of skull fracture does not correlate with the severity of TBI. Skull fractures are observed in only 25% of fatal head injury at autopsy.⁵¹ However, the incidence of contusion and/or haematoma is significantly higher in patients with a skull fracture than in those with no fractures.⁵²

Temporal bone fractures

Fractures of the temporal bone have been traditionally classified into longitudinal, transverse or mixed depending on their orientation relative to the long axis of the petrous bone. A newly proposed classification system divides temporal bone fractures into petrous (involving the otic capsule and/or petrous apex) and non-petrous (NPF) types (Figure 2).⁵³ NPFs are further subdivided based on the presence of middle ear and/or mastoid involvement.

This more recent classification scheme demonstrated better correlation with clinical complications of temporal bone

Figure 2. Temporal bone fractures. (a) Petrous fracture of the right temporal bone. Axial CT shows fracture line (arrows) involving the vestibule, labyrinthine segment of the facial nerve canal and cochlea, resulting in sensorineural hearing loss and facial nerve palsy. (b) Non-petrous fracture of the right temporal bone. Axial CT demonstrates fracture of the temporal squama (arrowheads) and dislocation of the incus (arrows), responsible for conductive hearing loss. Haemorrhage in the mastoid air cells and middle ear is also noted.

fractures such as facial nerve weakness, CSF leakage, sensorineural and conductive hearing loss. Petrous fractures appear to be significantly associated with an increased risk of facial nerve injury, carotid injury, CSF leaks and sensorineural hearing loss because of involvement of the inner ear structures. Likewise, the “middle ear” subcategory of NPFs carries an increased risk of conductive hearing loss secondary to ossicular chain damage. “Mastoid” NPFs seem to appear clinically less relevant.^{54,55}

Extra-axial haemorrhage

Extra-axial haemorrhage includes EDH, SDH, and SAH and IVH.

Epidural haematoma

EDHs occur in 0.2–12% of acute head-injured patients, and the overall mortality is 5%.^{56–60} EDHs generally occur at the site of impact, and a fracture is associated in >90% of cases.⁶¹ In contrast to SDHs, EDHs rarely cross the cranial sutures but can extend across the dural reflections. Hence, EDHs may be seen extending above and below the tentorium cerebelli or crossing the midline (Figure 3). 95% of EDHs are supratentorial. Most are temporal or parietal and usually result from disruption of the middle meningeal artery or one of its branches. Conversely, the vast majority of extra-axial haemorrhages in the cerebral posterior fossa are venous in origin, from disruption of dural

Figure 3. Venous epidural haematoma from superior longitudinal sinus disruption. (a) Coronal reconstruction CT shows subgaleal haemorrhage (arrows) and a large biconvex hyperattenuating extra-axial collection (large arrows), extending across the midline. The vertex epidural haematoma (EDH) exerts moderate mass effect on the underlying frontal lobes. At the vertex, the periosteal layer of the dura, which forms the external wall of the superior longitudinal sinus, is scarcely adherent to the sagittal suture. Therefore, EDHs of the vertex can cross the midline. Some foci of subarachnoid haemorrhage are noted on the medial surface of the frontal lobes (curved arrows). There is also right frontal encephalomalacia following previous injury (arrowheads). (b) Three-dimensional CT reconstruction viewed from above shows a sagittal fracture line (arrows) involving the left frontal and bilateral parietal bones, crossing the coronal and sagittal sutures.

veins and sinuses, which are especially abundant in this anatomic compartment.

Acute EDHs typically present as biconvex, hyperattenuating extra-axial collections on NECT. Occasionally, an acute EDH can appear heterogeneous because of areas of low attenuation, representing actively extravasating unclotted blood, also referred to as the swirl sign (Figure 4).⁶¹

According to previous observations, up to 8% of EDHs are not present on the initial CT scan but rather appear on follow-up.^{60,62,63} Delayed EDH formation is a rare but well-described complication following evacuation of cerebral haematomas (Figure 4). Contralateral intracranial haematoma formation has been reported in up to 7.4% of cases after decompressive surgery, which is responsible for decreasing the original mass effect supposed to tamponade the disrupted vessels.⁶⁴ Delayed EDH has also been associated with medical treatment aimed at re-establishing normal blood pressure or ICP in patients presenting with other intracranial lesions.⁶⁵

Subdural haematoma

SDHs are seen in 12–29% of patients with severe TBI.⁶⁶ As well as EDHs, the major causes of SDHs are motor vehicle accidents,

falls and assaults. SDHs usually arise from rupture of cortical bridging veins because of angular acceleration or deceleration of the head in the sagittal plane.⁶⁷

On axial CT, acute SDHs appear as crescent-shaped hyperdense collections between the cerebral hemisphere and inner table of the skull, frequently extending along the entire hemispheric convexity. Most SDHs are supratentorial in location and are frequently seen along the falx and tentorium as well. Unlike EDHs, SDHs may cross suture lines, but not dural reflections, and are rarely associated with skull fracture. There is commonly diffuse swelling of the underlying cerebral hemisphere, with midline shift and subfalcine herniation.

According to the literature, mortality rates for acute SDHs approach 60%.⁶⁸

Rarely, acute SDHs may be isodense or hypodense because of severe anaemia (Figure 5). A heterogeneous CT appearance may occasionally be noted because of active extravasation or mixture of fresh blood and CSF from associated arachnoid tears. A sediment level or haematocrit effect may be observed in patients with clotting disorders (Figure 6).⁶⁹

Subarachnoid haemorrhage

SAH is demonstrated in approximately 40% of patients with moderate to severe TBI.⁷⁰ It may result from injury to small subarachnoid vessels or from rupture of haemorrhagic contusions or haematomas into the subarachnoid space. SAH appears as curvilinear foci of increased attenuation within sulci and cisterns on NECT (Figure 7).

Intraventricular haemorrhage

The incidence of traumatic IVH varies between 1.5% and 3%, with rates approaching 10% in patients with severe TBI.⁷¹ IVH may result from stretch injury to the subependymal vessels that line the walls of the lateral and third ventricles or from bleeding of the choroid plexuses. Recent research demonstrated that the presence of IVH on CT was associated with corpus callosum injury on MRI in patients with blunt TBI, suggesting the role of IVH as a predictor for this disease.⁷¹ IVH may also result from direct extension of a parenchymal haematoma into the ventricular system or from retrograde flow of SAH.

On CT, IVH is seen as hyperdense material, tending to pool dependently within the ventricular system (Figure 8a).

Parenchymal injuries

Cerebral contusions

Cortical contusions are bruises of the brain surface.⁵¹ They occur in 43% of patients with blunt or non-penetrating head injuries and account for approximately half of all intra-axial lesions.⁷² Cerebral contusions involve the crowns of gyri, causing full-thickness necrosis and haemorrhage of the cortex and leptomeninges.⁵¹

In severe injuries, lesions may extend into subcortical white matter.

Figure 4. Delayed epidural haematoma following decompressive craniectomy (courtesy of Dr MC Valentini). Non-enhanced CT demonstrates a large temporoparietal biconvex epidural haematoma, with low-density areas suggesting active bleeding (swirl sign) (arrow). There is severe mass effect, with left-to-right midline shift, and external cerebral herniation through the craniectomy defect. A small left frontal subdural haematoma with fluid–fluid levels is also seen (arrows). The development of a delayed epidural haematoma as a result of decompressive craniectomy represents a life-threatening complication and warrants immediate neurosurgical intervention.

Figure 5. Acute isodense subdural haematoma in a 74-year-old female. (a) Non-enhanced CT shows effacement of the right lateral ventricle, mild dilation of the contralateral ventricle and moderate right-to-left midline shift. The haematoma is poorly seen. (b) Contrast-enhanced CT demonstrates enhancement of the cortical veins along the brain surface, allowing easier detection of left frontoparietal isodense subdural haematoma, which displaces the grey-white matter junction medially (arrows).

Contusions can occur in coup or contrecoup sites. They are typically focal or multifocal and are most often haemorrhagic. Coup contusions occur at the site of cranial impact; contrecoup contusions develop opposite or distant from the site of initial impact and are usually larger in size.

Figure 6. Subdural haematoma (SDH) with haematocrit level in a 77-year-old male. Non-enhanced CT shows bilateral hemispheric SDH with fluid-fluid levels, also referred to as the haematocrit effect. SDH with haematocrit levels are frequently encountered in the setting of anticoagulation therapy or coagulopathy. There is no midline shift because of bilateral mass effect with sulcal and ventricular effacement.

The most common locations for cerebral contusions are the inferior frontal and anterior temporal lobes. Cerebral contusions are seen on NECT as small foci of high attenuation within gyral crests; the association with ill-defined hypodense areas of vasogenic oedema is common.

Approximately 25–45% of contusions evolve, increasing in size over time, sometimes coalescing into larger intracerebral haematomas.⁷³ Delayed haemorrhages in areas of the brain that had previously appeared normal occur in approximately 15% of cases.⁷⁴

Compared with DAI, cerebral contusions are rarely associated with severe impairment of consciousness and are also associated with a better prognosis than DAI.

Intraparenchymal haematoma

Intracerebral haematomas and haemorrhagic contusions are part of the same spectrum of injuries. IPHs are less common than contusions and are observed in approximately 15% of acute head-injured patients.⁷⁵ As for cerebral contusions, traumatic IPHs are often multiple and show predilection for the frontal and temporal lobes.⁷⁵

Rarely, IPHs are unrelated to cerebral contusions, resulting from penetrating trauma such as gunshot or stab wounds.

Diffuse axonal injury

DAI is caused by acceleration–deceleration forces in the coronal or sagittal plane usually resulting from non-impact trauma and is also referred to as “axonal stretch injury”.⁷⁶ Association with skull fracture is rare. DAI is one of the most common parenchymal lesion in patients involved in high-velocity road accidents and is characterized by multiple small haemorrhagic and non-haemorrhagic lesions at the grey–white matter junction, corpus callosum, deep periventricular white matter, dorsolateral aspect of the mid-brain and upper pons. The basal ganglia, internal capsule and hippocampi are also sites of involvement.⁷⁷

Figure 7. Traumatic subarachnoid haemorrhage in a 73-year-old female. (a, b) Non-enhanced CT depicts increased density material in the basal cisterns, fronto-orbital sulci and left sylvian fissure (arrows). Subdural haematomas are noted overlying the tentorium (curved arrows). There is soft-tissue swelling in the right fronto-orbital region (arrowheads).

Loss of consciousness typically starts at the moment of impact. DAI rarely causes death but represents the most frequent cause of persistent vegetative state following trauma.

Anatomic distribution of DAI correlates with disease severity, with progressively deeper structural injury occurring with the increase of the acceleration–deceleration forces.⁷⁸

CT is known to have low sensitivity in detecting DAI, with only a minority of lesions demonstrated on NECT. Only 19% of non-haemorrhagic DAIs are demonstrated on CT and only 20% of

haemorrhagic DAIs contain sufficient haemorrhage to be detectable on CT as hyperdense foci.^{21,79} NECT may show focal areas of decreased attenuation secondary to oedema or punctate foci of increased density at the grey–white matter junction, corpus callosum and brain stem consistent with haemorrhagic DAI (Figure 9).

MRI is superior to CT for demonstrating DAI and is performed to search for DAI in unconscious patients with no evidence of structural brain damage on CT. Gradient-recalled-echo sequences are highly sensitive for detecting small haemorrhages, which are

Figure 8. Intraventricular haemorrhage and diffuse axonal injury (DAI) of the corpus callosum in a 30-year-old male with closed head trauma from high-speed motor vehicle accident. (a) Axial non-enhanced CT (NECT) shows hyperattenuation in the trigone of the right lateral ventricle consistent with haemorrhage (curved arrow). NECT also demonstrates mixed density subdural collection over the right convexity, with low attenuation areas indicating active bleed (arrows). There is moderate swelling of the underlying hemisphere and right to left midline shift. A small hyperattenuating left frontal subdural haematoma is also noted. CT also shows a large right parietal subgaleal haematoma (arrowheads). (b) Diffusion-weighted imaging in the same patient demonstrates restricted diffusion in the genu and splenium of corpus callosum (arrows) consistent with DAI.

seen as foci of decreased signal intensity.⁸⁰ Diffusion-weighted (DW) MRI has been proven to be more sensitive than any other sequence in acute stages of non-haemorrhagic DAI and to highly correlate with initial GCS score as well as modified Rankin outcome scale score.⁸¹ In the acute stage, DAIs appear as foci of increased signal intensity on DW MRI because of cytotoxic oedema resulting from axonal necrosis (Figure 8b). In the chronic stages of TBI, increased diffusivity and decreased fractional anisotropy are frequent findings, reflecting diffuse white matter abnormalities. Commonly damaged white matter tracts include the superior and inferior longitudinal fasciculus, anterior corona radiata, and frontal and temporal lobes.⁸² Microstructural white matter injury on diffusion tensor imaging in patients with mild TBI syndrome has been significantly associated with poor cognitive outcome as opposed to traumatic microhaemorrhages on conventional 3-T MRI.⁸³

Diffuse vascular injury

Previous research has shown that diffuse vascular injury is significantly associated with Grade 2 and 3 DAI, suggesting that the two entities depend on the same mechanism and are part of a pathological continuum.⁸⁴ Autopsy series of patients with diffuse vascular injury show innumerable punctate haemorrhages in the subcortical and deep white matter, as well as in the basal ganglia and thalami, because of disruption of microvasculature by high tensile forces.⁷⁶ CT findings may be subtle or absent. Susceptibility-weighted MR sequences show uncountable punctate and linear fan-shaped, convergent-type hypointensities that predominate in the subcortical and deep white matter and are classically oriented perpendicularly to the ventricles. The corpus callosum, deep grey nuclei, brain stem and cerebellum are often involved.⁸⁵ The major

differential diagnosis is DAI. The number and anatomic distribution of the haemorrhages allow differentiation from DAI (Figure 10).

Vascular injuries

Traumatic vascular injury includes arterial occlusion, dissection, pseudoaneurysm formation, arteriovenous fistula and dural venous sinus injury.³¹ Although uncommon, traumatic vascular injuries represent the most common cause of stroke in subjects younger than 45 years of age.⁸⁶ At present, the reported incidence of BCVIs varies between 0.3% and 1.6% among all patients with blunt trauma.³⁴ Associated mortality and morbidity rates are 24% and 58%, respectively.^{87,88}

Extracranial vascular trauma

Blunt extracranial vascular injuries typically occur as the result of motor vehicle accidents. The most common mechanism of injury is stretching of the artery from rapid deceleration.⁸⁹ The ICA is assumed to stretch over the lateral masses of the third and fourth cervical vertebrae, with formation of an intimal tear, which can progress into a dissection in the wall of the artery for a variable distance.⁹⁰ In most cases, cervical ICA affects the ICA distal to the carotid bulb and does not extend intracranially.⁹¹ Other mechanisms involved in ICA injury include direct blows to the head, neck or face; skull base fractures; blunt intraoral trauma; and hyperflexion of the neck. Bilateral dissections have been documented in as many as 45% of patients.⁹²

Extracranial vertebral artery dissection has a more varied aetiology and has been associated with chiropractic manipulation, tennis, seat belt use, yoga, head banging and kickboxing.⁹³

Figure 9. Diffuse axonal injury. (a) Non-enhanced CT (NECT) in a 12-year-old female with severe closed head trauma shows small haemorrhagic foci in the subcortical white matter consistent with grade I diffuse axonal injury (DAI) (arrows). A large left parietal subgaleal haematoma is also noted (arrowheads). (b) NECT in a 32-year-old male with severe non-impact head trauma shows low attenuation in the body of the corpus callosum consistent with non-haemorrhagic grade II DAI (arrows). (c) NECT in a patient with a Glasgow Coma Scale score of 3 shows haemorrhage in the dorsolateral aspect of the brain stem consistent with grade III DAI (arrow). According to the Adams and Gennarelli classification, in grade I DAI, lesions involve the grey-white matter interfaces of the frontal and temporal lobes. Grade II DAI involves the corpus callosum in addition to grade I lesions. In Grade III DAI, the dorsolateral mid-brain and upper pons are also affected.⁷⁰

Figure 10. Diffuse vascular injury in a 27-year-old male with severe closed head trauma. (a) Follow-up non-enhanced CT obtained 10 days after admission shows marked hypoattenuation of the deep white matter without mass effect (arrowheads). (b) T_2^* GRE image shows innumerable punctate hypointensities in the subcortical and deep white matter consistent with diffuse vascular injury (arrows). Brain stem and cerebellum are also affected.

In both carotid and vertebral dissection, delayed onset is common, with symptoms developing after hours or even weeks from the initial injury. Therefore, screening protocols to detect BCVIs are essential in order to decrease mortality and morbidity.

Cervical CTA is the modality of choice for the diagnosis of BCVIs. Pseudoaneurysms appear as contrast material-filled sacs outside the wall of the injured artery. With dissections, CTA typically shows severe narrowing and irregularity of the vessel lumen, creating a “string-like” appearance (Figure 11). The external diameter of the artery is typically increased.⁹⁴ In acute dissection, NECT may depict a crescent-shaped hyperattenuating area corresponding to a mural haematoma at the upper portion of the cervical ICA.⁹⁵

Cerebrovascular injuries are more common with penetrating wounds than with blunt trauma, with rates of approximately 25%.⁹⁶ Blunt trauma and penetrating trauma cause similar lesions,

resulting in the formation of pseudoaneurysms, arteriovenous fistulae, arterial transection and dissection.⁸⁰

Intracranial vascular trauma

Intracranial arterial injuries are generally associated with penetrating trauma or skull base fractures. The most commonly injured artery is the ICA, especially at its entrance to the carotid canal at the base of petrous bone or at its exit from the cavernous sinus beneath the anterior clinoid process. Traumatic injuries of the intracranial arteries include dissection, pseudoaneurysm and CCF.⁹⁶

Traumatic CCF is a direct high-flow arteriovenous shunt that develops within the cavernous sinus as a result of a tear of the cavernous portion of the ICA. The consequent increase in venous pressure causes dilatation of the cavernous sinus and retrograde venous outflow through the superior and inferior ophthalmic veins. Traumatic CCF may present weeks or even months after the initial trauma.⁹⁷

Figure 11. Post-traumatic right carotid-cavernous fistula following fracture of the carotid canal. (a, b) CT angiography shows early opacification of the cavernous sinuses (arrow in a) and a markedly enlarged right superior ophthalmic vein (arrow in b).

Figure 12. Left carotid artery dissection following motor vehicle accident. (a) Axial image from CT angiography (CTA) shows that the left internal carotid is markedly narrowed (arrow). The intramural haematoma cannot be differentiated from the surrounding muscles. (b) Sagittal reformatted image from CTA demonstrates marked narrowing and irregularity of the left carotid lumen (arrows).

In patients with CCF, contrast-enhanced CT may demonstrate an enlarged superior ophthalmic vein and cavernous sinus. Other CT features include extraocular muscles enlargement, proptosis and preseptal soft-tissue swelling (Figure 12). Rarely, a unilateral

CCF may present with bilateral symptoms, because of venous intercavernous communication.

SECONDARY INJURY

Post-traumatic brain swelling

Massive brain swelling with severe intracranial hypertension is the most severe of all secondary lesions. Post-traumatic brain swelling is an increase in brain volume resulting from an increase in tissue water content.⁷⁵ The underlying mechanism is unclear: brain swelling appears to be the result of cerebral oedema, both intracellular (cytotoxic) and extracellular (vasogenic). Brain swelling may be focal or diffuse and occurs in 10–20% of patients with TBI. Children and young adults are especially at risk to develop this condition. Delayed onset is typical, with cerebral oedema appearing 24–48 hours after the initial trauma.⁹⁷

Decompressive craniectomy is the treatment of choice but prognosis remains poor. Complications include herniation of the cortex through the bone defect, subdural effusions, hydrocephalus, seizures and syndrome of the “trephined” or “sinking skin flap syndrome”.⁹⁸

CT findings include ill-defined mass effect and effacement of sulci. In early stages of brain swelling, there is normal attenuation of the brain, and grey–white matter differentiation is relatively preserved. As oedema increases, all grey–white matter interfaces fade. The falx and circulating blood into cerebral vessels appear hyperdense relative to the swollen, hypodense cerebral hemispheres. The cerebellum and brain stem are typically spared and also show increased attenuation.⁹⁷

Brain herniation

Cerebral herniation is the displacement of brain tissue from one cranial compartment to another due to mass effect produced by primary intracranial injury.⁹⁹

Subfalcine herniation is the most common type of cerebral herniation and is caused by displacement of the anterior cingulate gyrus beneath the falx cerebri. Subfalcine herniation is also known as midline shift and is frequently associated with downward displacement of the corpus callosum. Compression of the ipsilateral lateral ventricle due to mass effect and dilatation of the contralateral ventricle due to obstruction of the foramen of Monro are common CT findings. In severe cases, compression of the pericallosal arteries may result in anterior cerebral artery (ACA) infarction.

Descending transtentorial herniation (DTH) is the second most common type of brain herniation and consists of caudal descent of brain tissue through the tentorial incisura, secondary to mass effect in the frontal, parietal and occipital lobes.¹⁰⁰ In DTH, the uncus of the temporal lobe is displaced over the free margin of the tentorium into the ipsilateral suprasellar cistern. As the mass effect increases, the hippocampus also herniates medially over the edge of the tentorium into the ipsilateral quadrigeminal cistern, pushing the mid-brain against the contralateral tentorial edge. As herniation progresses, both the uncus and hippocampus are displaced inferiorly through the tentorial incisura. Displacement of the mid-brain can result in compression of the contralateral cerebral peduncle against the opposite side of the incisura (Kernohan’s notch).¹⁰¹ Complications of DTH include damage to the corticospinal and corticobulbar tracts,

compression of the posterior cerebral artery and oculomotor nerve, resulting in hemiparesis, infarction and ipsilateral third nerve palsy.¹⁰⁰ Compression or stretching of the perforating branches of the basilar artery may occur, resulting in a secondary haemorrhagic mid-brain infarction, known as a Duret haemorrhage.¹⁰² Hydrocephalus may develop following distortion and obstruction of the mesencephalic aqueduct.

Axial CT shows effacement of the ambient and lateral suprasellar cisterns from medial displacement of the uncus and hippocampus.

Tonsillar herniation consists in downward displacement of cerebellar tonsils and medial aspect of cerebellar hemispheres through the foramen magnum into the cervical spinal canal. Up to 50% of cases of tonsillar herniation occur in association with descending transtentorial herniation.⁹⁹ Complications of tonsillar herniation include obstructive hydrocephalus and cerebellar infarction from occlusion of the posterior inferior cerebellar artery. Compression of the lower brain stem may lead to impairment of consciousness secondary to mass effect on the ascending reticular activating system. As herniation progresses, involvement of the cardiac and respiratory centres of the brain stem may occur, eventually leading to death.⁹⁹ Tonsillar herniation may be difficult to diagnose on CT. CT may show effacement of the cisterna magna and CSF spaces surrounding the medulla oblongata.

Tonsillar herniation has also been related to intracranial hypotension, the tonsils being pulled downward because of decreased intraspinal CSF pressure.

In *ascending transtentorial herniation*, the superior vermis and cerebellar hemispheres are displaced superiorly through the tentorial incisura into the supratentorial compartment. Ascending transtentorial herniation occurs rarely in TBI and is generally the result of large posterior fossa haematomas.

Axial CT demonstrates effacement of the supravermian and quadrigeminal cisterns along with mass effect and distortion of the mid-brain.

Post-traumatic cerebral infarction

Post-traumatic cerebral infarction is a rare but severe complication of TBI. In addition to cerebral herniation, other potential causes of infarction include vasospasm, increased ICP, global hypoxia or hypoperfusion, and compressive ischaemia from intracerebral haematomas.¹⁰³

SUMMARY

Imaging plays a primary role in the management of patients with TBI. CT is the imaging technique of choice in the setting of acute head trauma, allowing accurate detection, and thereby treatment, of extra- and intra-axial haemorrhage, hydrocephalus, mass effect and vascular injuries. CT is also accurate in detecting secondary injuries and is therefore essential in follow-up. In the acute setting, MRI is reserved to patients with severe neurological impairment despite the absence of structural brain damage on CT. MRI is the imaging modality of choice in subacute and chronic TBI and appears to be more reliable in CT in predicting outcome.

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