Traumatic Brain Injury Part II: Secondary Brain Injury

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First Edition Update 2022

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Intended Learning Outcomes

TBI Part II: Secondary Brain Injury

1. Define secondary brain injury
2. Describe the difference between primary to secondary brain injury
3. Recognise the extra-cranial and intra-cranial causes of secondary brain insults
4. Describe image findings of intra-cerebral lesions and consequences in TBI patients including: Mass effect, Focal lesions, Skull fractures, tSAH, Epidural hematoma, Subdural hematoma, and Traumatic contusion/laceration and intracerebral hematoma

eModule Information

Relevant Competencies from CoBaTrICE

TBI Part II:Secondary Brain Injury

- **2.2** Undertakes timely and appropriate investigations
- **2.8** Liaises with radiologists to organise and interpret clinical imaging
- **2.9** Monitors and responds to trends in physiological variables
- **2.10** Integrates clinical findings with laboratory investigations to form a differential diagnosis
- **3.6** Recognises and manages the patient with neurological impairment
- **4.4** Uses fluids and vasoactive / inotropic drugs to support the circulation
- **6.3** Manages the care of the patient following craniotomy under supervision

1. Definition And Detection Of Secondary Brain Injury

Mortality and morbidity after a head injury are influenced by both primary and secondary damage. While there is no effective treatment for primary brain damage, prevention and/or effective treatment of secondary insults to the brain are the key issues in treatment.

Having ascertained the severity of brain injury at the earliest possible opportunity, this is the best guide to the extent of primary brain injury which has resulted from the direct mechanical trauma at the scene of the accident. We have discussed how this may be achieved clinically using the Glasgow Coma Scale and we shall examine additional methods later in this Task. Changes in these parameters with time may assist you in detecting secondary brain injury, determining the effects of treatment and predicting outcome.

Primary and secondary brain injury often cannot however be clearly distinguished from each other.

Task

Test your knowledge

Another clinical description of head injury is 'open' or 'closed'. How would you define an open head injury and how may it be caused?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

An open injury involves direct communication between intra- and extracranial spaces. The integrity of the dura mater may be disrupted by bullets, sharp instruments or skull fractures.

What is the relevance of the distinction between the 'open' vs 'closed' mode of head injury?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Patients with open head injuries are more likely to develop intracranial infection (meningitis, subdural empyema, brain abscess) or post-traumatic epilepsy and therefore often require neurosurgical intervention (best done within the first eight hours).

1. 1. Primary and secondary brain injury

Primary brain injury includes disruption of brain vessels, haemorrhagic contusion and traumatic axonal injury (TAI, also know as diffuse axonal injury, DAI).

Secondary brain injury may have extra- or intracranial causes. Extracranial causes include systemic hypotension, hypoxaemia, hypercarbia, disturbances of blood coagulation and infection.

At macroscopic level intracranial causes include intracranial haematoma, brain swelling and cerebral oedema, intracranial infection and uncontrolled fits. At cellular level, a cascade of events contributes to secondary injury, and the interrelationship between primary and secondary brain insults and their potential consequences may result in ultimate cerebral cell ischaemia and cell death.

Note

Secondary insults are more commonly associated with cardiorespiratory disturbances and are independent predictors of poor outcome. They are often avoidable.

Challenge

Identify the presence, severity and duration of secondary insults in the next ten patients with TBI. Determine to what extent more effective prevention and treatment of these secondary insults might have been achieved. Discuss these findings with your colleagues.

In text References

(Maas, Stocchetti and Bullock. 2008; McHugh et al. 2007)

References

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- McHugh GS, Engel DC, Butcher I, Steyerberg EW, Lu J, Mushkudiani N, Hernández AV, Marmarou A, Maas AI, Murray GD., Prognostic value of secondary insults in traumatic brain injury: results from the IMPACT study., 2007, PMID:17375993

1. 2. Extra-cranial causes

Extracranial causes oof secondary insults to the damaged brain, e.g. hypotension and hypoxia, require prompt and efficient management. Following on from the section on resuscitation in the previous course, subsequent maintenance of optimal cerebral oxygen delivery remains of paramount importance.

The more severe the extracranial injury the higher the risk of mortality. This association may be due to several factors beyond hypotension, hypoxia and anaemia, including multiorgan failure, infections and medical complications. However from a practical standpoint, the initial management should be directed to minimise any source of bleeding due to extracranial injury.

Note

One of the problems with the concept of optimising cerebral oxygen delivery is that traumatised tissue may not respond physiologically.

1. 3. Intra-cranial physiological causes

Multi-modal monitoring may be used to monitor for and trigger treatment for secondary causes of brain injury. The specifics of such monitoring will be discussed later in this series of ACE Courses.

One of the major aims of the intensive care management of severe TBI is to control intracranial hypertension and provide haemodynamic support to achieve an “adequate” cerebral perfusion pressure (CPP). Raised ICP is well described to be associated with poor outcomes. In addition, impaired cerebral autoregulation and cerebrovascular reactivity are also associated with increased mortality and poor functional outcome after TBI. The most widely described index to measure this is the pressure reactivity index (PRx) which is a moving correlation coefficient between slow waves of ICP and the mean arterial blood pressure. There is growing evidence that a single CPP target is not sufficient given the diversity of patients and that an optimal target individualised for patients may be of benefit.

Cerebral microdialysis (CMD) may be used to provide insights into cellular metabolism and injury by monitoring for metabolic distress which may be used to guide therapy. Commonly measured analytics include glucose, lactate, pyruvate (allowing for calculation of the lactate:pyruvate ratio (LPR)), glutamate and glycerol. Elevated CMD measured mean LPR, glutamate and glycerol has been associated with elevated ICP and/or decreased CPP. Elevated LPR has also been associated with frontal lobe atrophy at 6 months.

Monitoring of brain oxygenation levels either focally using brain tissue oxygenation monitors or globally using a jugular venous catheter can be used to titrate oxygenation and avoid tissue hypoxia/ischemia. This has been the focus of a Phase-II trial which showed a trend towards lower mortality and more favourable outcomes. A definitive RCT is awaited.

In text References

(Aries et al. 2012; Le Roux et al. 2014; Okonkwo et al. 2017; Sorrentino et al. 2012; Wright et al. 2013; Zeiler et al. 2017)

References

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1. 4. Overview of Imaging after TBI

Intracranial causes of secondary brain insult will be the focus of the remainder of this course.

Traumatic brain injury is a heterogeneous disease but the traditional classification, based on GCS, considers only the neurological consequences of trauma. CT is the primary imaging modality for TBI. Its fast-scanning times, and ease of availability mean it is used to drive the key decisions for the need for surgical interventions and can be useful to help define and investigate the heterogeneity of TBI. Standard clinical MRI provides greater sensitivity than CT for parenchymal lesions, especially in the posterior fossa, brainstem, and superficial cortical areas potentially providing a more refined stratification of injury.

There are several CT classification systems for TBI including the Marshall Grade, Helsinki, Stockholm and Rotterdam Scores. The most used, the Marshall Score distinguishes focal from diffuse lesions and comprises part of the IMPACT Score for prognostication.

Before defining these lesions in more detail, you may wish to refer to the following summary of the main elements of CT and MRI interpretation.

Task

Review the paper of (Maas et al. 2007)

In text References

(Marshall et al. 1992; Thelin et al. 2017)

1. 4. 1. Interpretation of CT images

You should start by reminding yourself of the normal anatomy as presented on a CT scan. Useful web addresses in this connection are:

- Radiopaedia
- Whole Brain Atlas

Note

CT interpretation is a valuable skill and at least a basic understanding is recommended. Help is available. Search and you shall find!

Before examining any CT scan always check the patient's details, the date and time, and the anatomical orientation (i.e. the patient's right is on the left side of the image as you examine it and vice versa).

According to their density, the various structures of the head absorb radiation to a different degree:
CT.

Figure 1: Relationship between radiation absorption and CT appearance of different brain components.

Interpretation of a CT scan involves two tasks: firstly to detect (what do I see?), ... and secondly to analyse (what does this mean?). Things to look for are shown below in Table 1:

Table 1: Interpretation of CT images and their related injuries

Scalp	Swelling		
Skull	Fracture (CT better for skull base and midface)		
Intracranial	Hyperdense	Blood	Extradural Intradural
	Mixed density	Blood and cerebral oedema	Contusion
	Hypodense	Cerebral oedema	Contusion General or prolonged cerebral ischaemia
	Foreign bodies metal/bone hyperdense; wood/glass etc. hypodense		

The intensivist should be able to rapidly determine the following:

- Is there a focal lesion?
- Is it associated with a mass effect (see 'mass effect' heading below)?
- Would the patient benefit from an urgent evacuation or would it be more appropriate to monitor neurological status and/or ICP?
- Is there a diffuse lesion?
- Is it associated with brain swelling, and probably intracranial hypertension?
- Is it associated with petechiae, and where are they located?
- Are there both diffuse and focal lesions?
- Are there lesions warning of potential evolution toward a new mass or the enlargement of a previous detected mass?

The Marshall classification is extremely useful in that following these priorities, when looking at the CT, will automatically generate the CT score:

1. If there is a large mass (roughly above 25 mL), it is helpful to picture and determine whether there is ‘mass effect’
2. Does this mass object require an urgent evacuation? This indirectly suggests that the mass has a ‘mass effect’
3. Is it associated with a midline shift toward the opposite side where it is located? This is a quantitative indicator of a ‘mass effect’
4. Are the basal cisterns distorted or compressed on the same side of the mass lesion; this is a further sign of ‘mass effect’
5. Are the basal cisterns bilaterally compressed or absent? This is a sign of intracranial hypertension and coning due to the ‘mass effect’ or a sign of diffuse brain swelling.

A look to these five points focuses on intracranial emergencies and can assist in estimating the ICP before its measurement.

Secondarily it is important to evaluate:

1. The nature of the mass lesion: the physiology, evolution, management and prognosis are substantially different between the different lesions. Isolated extradural haematoma (EDH) needs emergent management and may have a relatively good outcome.
2. A subdural haematoma (SDH) will exert a mass effect, and most are associated with cortical laceration. Mass effect together with simultaneous primary brain damage means that SDH is the haematoma with the worst prognosis.
3. Intraparenchymal laceration/contusion may sometimes need immediate surgery (as a consequence of the volume, or the location) but most are observed over hours and days. They may enlarge in the core or more frequently result in perilesional oedema. Lesions with a dense and large haemorrhagic core are called intraparenchymal haematomas. They are less frequent but need urgent evacuation, except in unsalvageable patients.

Once intracranial emergencies have been excluded, it is important to anticipate the evolution of mass lesion in the next hours.

1. Is there thin layer EDH or SDH in temporal fossa or posterior fossa? These can enlarge over time and rapidly cause regional hypertension.
2. Is there vault or scissural subarachnoid haemorrhage? This may be suggestive of an evolving intraparenchymal CT lesion.
3. Are there intraparenchymal lesions? These can develop over days and result in peri-haemorrhagic oedema.

Finally, once the warning signs of potentially evolving lesions have been excluded, the focus should be on the severity of diffuse lesions:

1. Are there multiple petechiae, or are there gliding petechiae, on the corpus callosum, on thalami, or on the midbrain? If present, an MRI is needed to improve the diagnostic specificity.
2. If the CT is negative, is an MRI indicated?
3. Are there risk factors for an extracerebral arterial lesion, e.g. dissecting lesion of extracranial carotid? If so, a CT-Angiogram may be needed.
4. Carotid and Vertebral artery dissection can be easily missed and cause subacute brain infarction. Clinical suspicion is important based mechanism of trauma and list of injuries. Diagnosis can be difficult, requiring CT angiography or MRI to confirm and an expert neuroradiologist report.

1. 4. 2. Interpretation of Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) may offer important insights after TBI and it is clear that MRI is more sensitive to the location, extent and distribution of intracerebral lesions secondary to TBI than CT, especially for lesions in the posterior fossa, brainstem, and superficial cortical areas. Commonly used conventional sequences are summarised in Table 2.

It is clear that prognosis defining lesions, including brainstem lesions not detectable on CT may be found when a patient has MRI. This may add important prognostic information when making key decisions about patients including whether to insert a tracheostomy, whether a decompression may be effective or for whether it is more appropriate to continue or withdraw life sustaining therapies. It is important to note that MRI is only part of the prognostic picture.

T1-weighted imaging is commonly used as a volumetric sequence to help with anatomical localization. While lesions may be seen on this sequence they are often seen better on others following including Fluid-attenuated inversion recovery (FLAIR) and gradient-recalled echo (GRE)/ Susceptibility weighted imaging (SWI). Given its ability to delineate anatomical structures it may be particularly useful in the detection of atrophy.

FLAIR has a long inversion time that is adjusted to remove CSF from the resulting images. This makes it particularly useful to detect subtle changes in the periventricular region and periphery of the hemispheres, as well in the detection of vasogenic oedema. FLAIR has been found to be particularly sensitive at the detection of white matter lesions (especially hyper-intensities) and oedema, particularly in the context of TAI.

The detection of traumatic micro-haemorrhages (also known as petechial haemorrhages) are felt to be a surrogate biomarker of traumatic axonal injury and is best done with GRE or SWI. SWI uses a special GRE sequences that takes advantage of the susceptibility differences between tissues. The phase and magnitude data is combined to produce images with accentuated local susceptibility which are sensitive to venous blood, haemorrhage and iron. However, it should be remembered that they represent microvascular injury rather than direct axonal injury and so it is possible to have TAI detected using other methods including DTI when there are no microhaemorrhages present. SWI is more sensitive for the detection of microhaemorrhages than GE, and a higher number detected may be associated with an unfavourable outcome.

Diffusion weighted imaging (DWI) studies the molecular motion of water, a contrast that may be substantially altered by disease. The typical clinical diffusion imaging study acquires an image without diffusion weighting (b0), and three images with diffusion weighting along mutually orthogonal directions from which an apparent diffusion coefficient (ADC) can be calculated. Oedema is particularly well defined using this sequence, and is able to distinguish between cytotoxic and vasogenic oedema. In addition small lesions may be more conspicuous than T2 weighted imaging.

The addition of at least 3 more non-collinear diffusion-sensitizing gradient acquisitions allows for a tensor to be resolved in a technique known as Diffusion tensor imaging (DTI). This much more sophisticated approach allows for better insights into the microstructure of tissues, including anisotropy. This of the most promising techniques to provide a means of addressing the neuroanatomical substrate of such disease evolution is diffusion tensor imaging (DTI). However, unlike DWI and the conventional sequences it requires complex image processing and so it not yet widely used clinically.

Table 2: Overview of commonly used MRI sequences and the lesions best detected by each.

Sequence	Most relevant tissue contrasts for TBI imaging
T1	Normal grey-white contrast
T2	High signal in CSF, vasogenic oedema, gliosis, acute and subacute bleed (may be hypointense signal in hyperacute or chronic bleed)
FLAIR	Like T2, but CSF nulled, so good for superficial lesions

Gradient echo (SWI, SWAN)	Sensitised to blood – very useful for petechial haemorrhages associated with TAI. Most prone to artefacts (air in sinus, probes)
DWI, ADC, DTI	Early cytotoxic oedema, white matter shearing, tractography

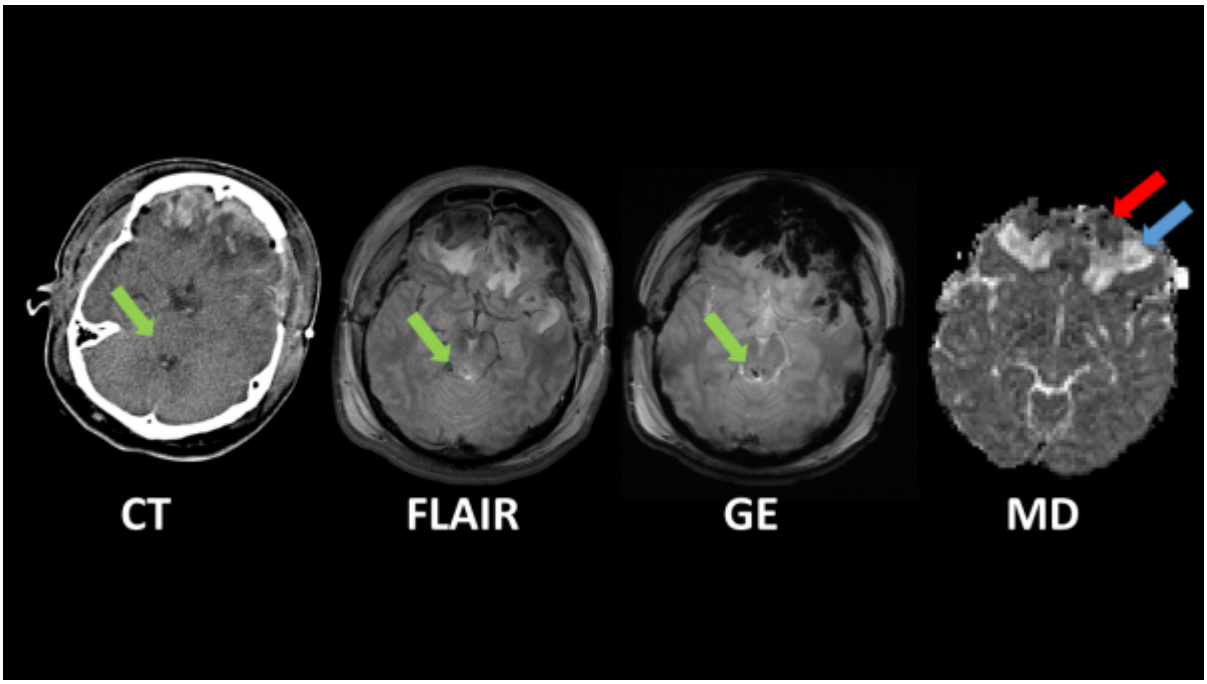

Figure 2: Detection of brain stem lesions and contusions after TBI with CT and MRI. Bifrontal contusions can be seen on CT and all MRI sequences. The MD (mean diffusivity) map calculated using DTI shows a bright area (blue arrow) before a darker contusion core (red arrow). The bright area represents an area of vasogenic oedema. A small lesion can be seen on the posterior brainstem in the region of the fourth ventricle (green arrow), which is most conspicuous on the GRE sequence (sensitised to blood products). The brainstem lesion is not able to be seen on the CT scan.

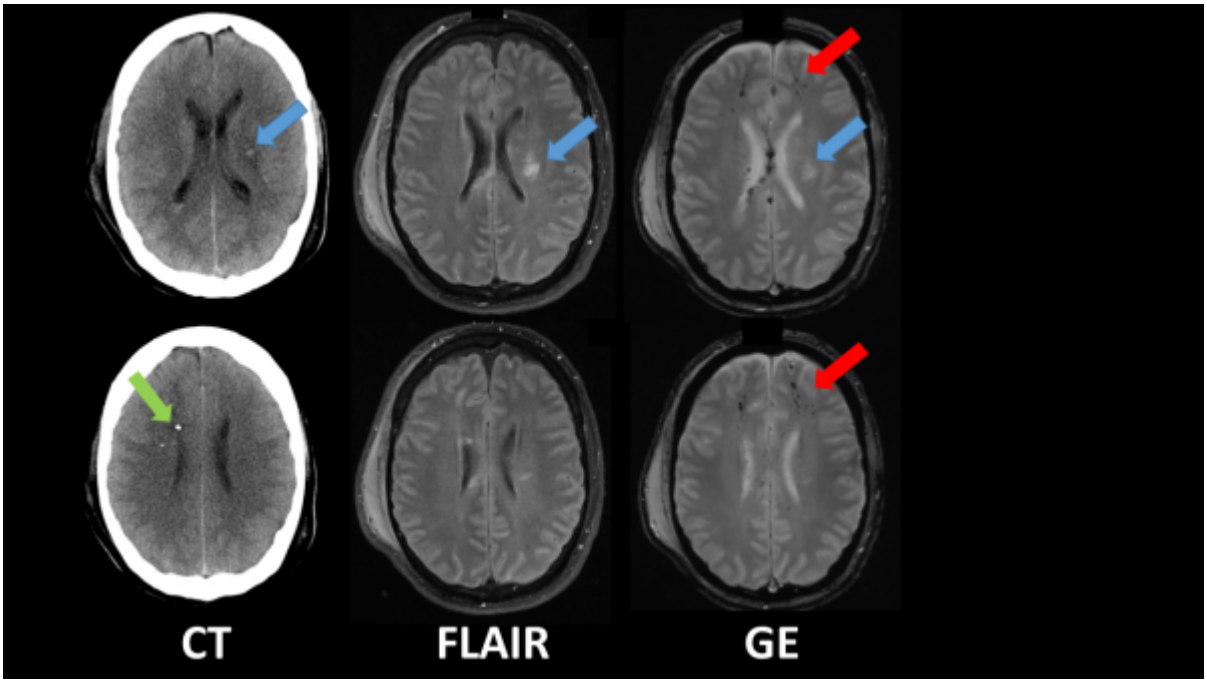

Figure 3: Detection of structural brain damage after TBI with CT and MRI. The green arrow points to a small hyperdensity which is the intraparenchymal probe, Blue arrow points to an area of oedema that

is only just visible on the CT, though it is very apparent on the FLAIR and the extent much larger on the FLAIR. The red arrow points to areas of pectechial or micohaemorrhages that are only visible on the gradient echo.

In text References

(Miller et al. 2004; Moen et al. 2014; Izzy et al. 2017; Newcombe et al. 2013; de Haan et al. 2017; Thanvi et al. 2005)

References

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1. 5. Intra-cerebral lesions and their effects

1. 5. 1. Mass effect

A developing haematoma requires space. It therefore compresses other intracranial structures. Ultimately, the brain itself is compressed and displaced – the so-called space-occupying or mass effect. The larger the haematoma the more pronounced the mass effect. Patients with atrophic brains (alcoholics and the elderly) tolerate haematomas better than younger patients – they have more CSF.

In the case of supratentorial haematoma, the following sequence occurs:

- narrowing of the ipsilateral subarachnoid space and the ipsilateral ventricle
- shifting of the ventricle to the opposite side
- compression and displacement of the third ventricle
- transtentorial herniation of the medial portion of the temporal lobe
- compression of the paramesencephalic cisterns (pupillary dilation on the side of the haematoma).

A series of helpful diagrams illustrating various forms of herniation is available in the reference below. Although these mass effects are closely correlated with raised intracranial pressure, CT scans are not a substitute for continuous monitoring of ICP.

In text References
(Trinidad and Milhorat. 1999)

1. 5. 2. Focal lesions (intracranial mass lesions)

Approximately a quarter of all patients with severe head injury have an ‘operable’ intracranial haematoma.

Extra-axial lesions are found outside the brain parenchyma and include subdural and epidural haematoma. Intra-axial lesions include haemorrhagic contusions and traumatic intracerebral haematoma.

The localisation of a focal lesion in the posterior fossa deserves special and more urgent consideration.

The final decision as to whether and when to operate on an intracranial mass rests with the neurosurgeon and is based on a variety of factors in the individual patient (including space-occupying effect, neurological state and general condition of the patient). Over time, however, and with tuition in CT interpretation, the critical care doctor will become increasingly familiar with the haematomas for which immediate evacuation is indicated and for those which are not so urgent.

Challenge

You should take every opportunity to participate in the case conferences and more acute decision-making processes. As your experience grows your input will be increasingly valued. Note where decisions are based on ‘solid information’ and where on ‘clinical judgment’. Ask questions about the latter and remember a previous example we noted in Neurological examination.

In text References

(Bullock et al. 2006)

1. 5. 3. Skull fractures

You may be confused about the significance of skull fractures which are a common finding in head injury. If the fracture is closed and not depressed, specific treatment is rarely required and healing occurs spontaneously. Open and depressed fractures usually need neurosurgical intervention.

Normally, skull fractures are not palpable through the intact galea. Bruises to the skin may indicate an underlying fracture; periorbital (raccoon eyes), and retroauricular haematomas (Battle’s sign) may indicate basal skull fractures (look for CSF fistula). Patients with skull fractures have a high incidence of intracranial haematomas (Table 3).

Table 3: Incidence of intracranial haematomas depending on the severity, and the presence of a skull fracture in 3802 patients (Miller JD, 1995).

Severity of Head Injury		**Haematoma on CT (%)**	**No Haematoma (%)**
Severe (GCS 3–8)	with fracture	44	56
	without fracture	32	68
Moderate (GCS 9–12)	with fracture	29	71
	without fracture	8	92
Mild (GCS 13–15)	with fracture	10	90
	without fracture	1	99

Detection of a skull fracture is important and is usually detected by means of emergent CT.

1. 5. 4. Traumatic subarachnoid haemorrhage (tSAH)

This is the presence of blood in the subarachnoid space. Its distribution varies greatly, but it may be located

- in the cortical vault and/or within cortical scissurae, and/or
- at the base of the brain, in the basal cisterns, over the tentorium, and/or
- in the interpeduncle space.

Most evidence suggests that subarachnoid blood in TBI is not associated with vasospasm, but is a landmark of CT severity. Furthermore traumatic subarachnoid haemorrhage (tSAH), especially if located above the cortex or within scissurae could be a warning for potentially evolving cortical lesions which bleed into the subarchnoid space.

In text References

(Servadei et al. 2002; Mata-Mbemba et al. 2018)

1. 5. 5. Epidural haematoma

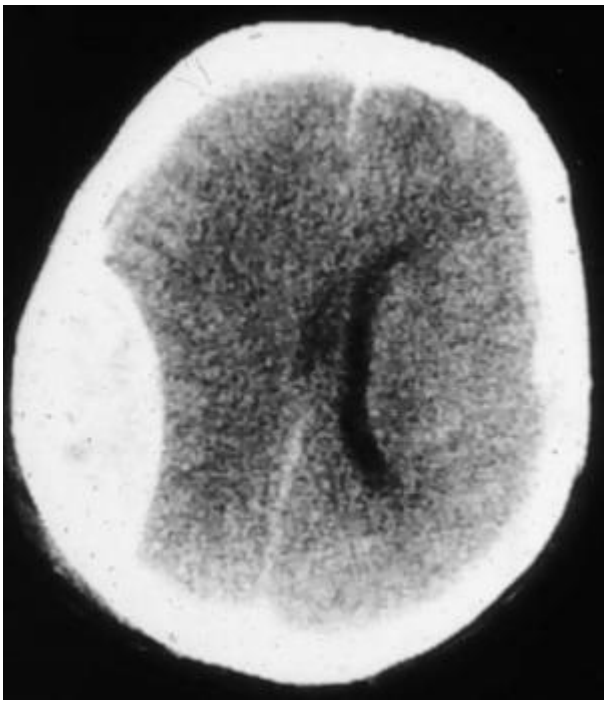

Figure 4: CT appearance of epidural haematoma

Normal neurological status on admission does not rule out significant brain pathology. An epidural haematoma (EDH) is an accumulation of blood in the epidural (also known as ‘extradural’) space between the inner side of the skull and the dura mater. In most cases the cause is a skull fracture crossing the middle meningeal artery or its branches in the fronto-temporal region. Rarely, a fracture may be associated with tearing of large veins at the vertex of the skull or of the venous sinuses of the brain itself.

Patients with a skull fracture may be neurologically intact on admission and later deteriorate as the EDH develops. More often, however, primary brain damage has caused some disturbance of consciousness and the developing haematoma results in

rapid neurological deterioration. This sequence of events occurs in the first of the patients in the ‘Patient Challenges’.

Task

Test your knowledge

Epidural (extradural) haematoma (EDH) is quite uncommon in the young (<5 years) and the elderly (>65 years). Why do you think this might be?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

The dura is tightly adherent to the skull in these age groups and does not tear easily.

The management is described by ABC guidelines. EDH is a medical emergency and the final outcome is definitively affected by a prompt critical care and surgical intervention. Once the patient is declining neurologically and surgery has been decided, especially for patients requiring transfer from a hospital which is far from the neurosurgical centre, it is preferable to intubate, sedate and ventilate the patient to transfer him/her to OR in the best condition possible – having taken and titrated concurrent, acute, temporising measures to reduce the elevated intracranial pressure.

1. 5. 6. Subdural haematoma

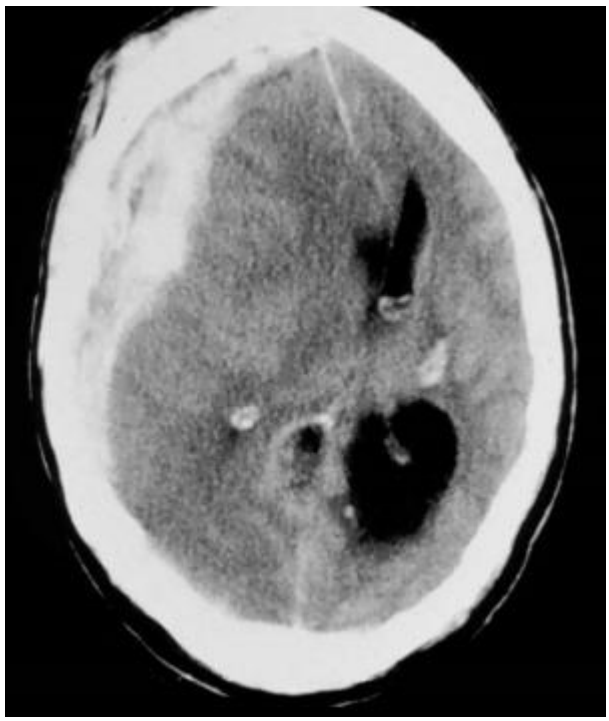

Figure 5: CT appearance of subdural haematoma, associated with mid-line shift

Subdural haematoma (SDH) is an accumulation of blood between the inner side of the dura and the arachnoid layer of the brain. It occurs if a cortical vessel is torn. In most cases a large contusion at the frontal or temporal surface of the brain is found. Subdural haematomas are called acute if they develop during the first 24 hours, subacute if they develop between 1–7 days, and chronic thereafter.

As most patients with acute SDHs have some kind of accompanying brain injury, their prognosis is worse than that of patients with EDHs.

Most patients harbouring an acute SDH are unconscious immediately after the trauma. The expanding haematoma then causes additional neurological deterioration. You will find further images in the appendix in the interactive version.

Subacute subdural haematoma is more common in patients with an atrophic brain. Most of them have minor or moderate disturbances of consciousness level at first and deteriorate during the first two to four days.

The management is described by ABC guidelines. Intensivists and anaesthetists should consider that before SDH evacuation, ICP can be extremely elevated and every effort should be done to minimise intracranial hypertension even if not measured.

Challenge

In the forthcoming period, analyse patients that are diagnosed with either epidural or subdural haematomata. List how you differentiate between these two conditions. Compare your answer with the table below.

Table 4: Essential differences between epidural and subdural haematomas

	Epidural	Subdural
Incidence	Approximately 10% of all patients with severe head injuries.	Most frequent extra-axial haematomas in patients with TBI.
Onset	Most develop during first 8 hours after the injury.	Occur either during the first 24 hours (acute) or during day two to four (subacute) after the injury.
Clinical features	Risk of rapid deterioration of conscious level (GCS) with focal neurological signs (ipsilateral dilating pupil, contralateral hemiparesis).	Rapid neurological deterioration (GCS) and focal neurological signs (ipsilateral dilating pupil, contra lateral hemiparesis) in an already deeply comatose patient in acute cases. More gradual neurological deterioration and focal neurological signs in subacute or chronic cases.

1. 5. 7. Traumatic contusion/laceration and intracerebral haematoma

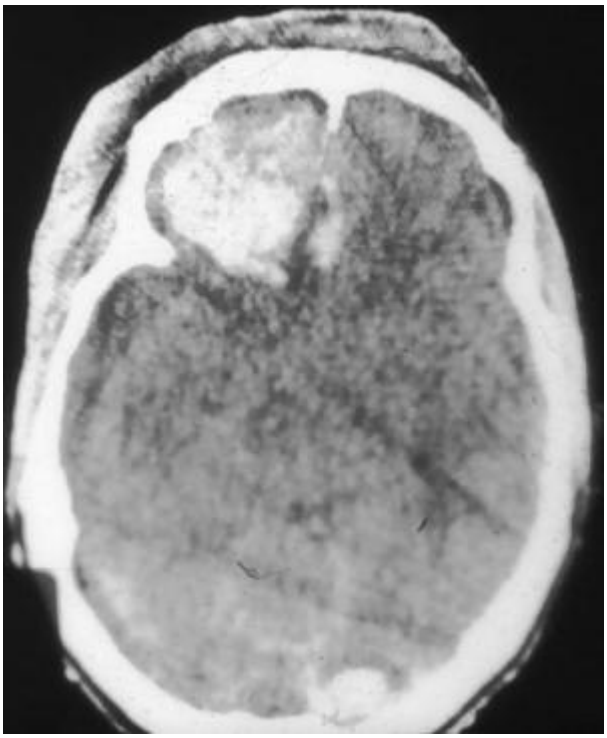

Figure 6:CT appearance of intracerebral haematoma

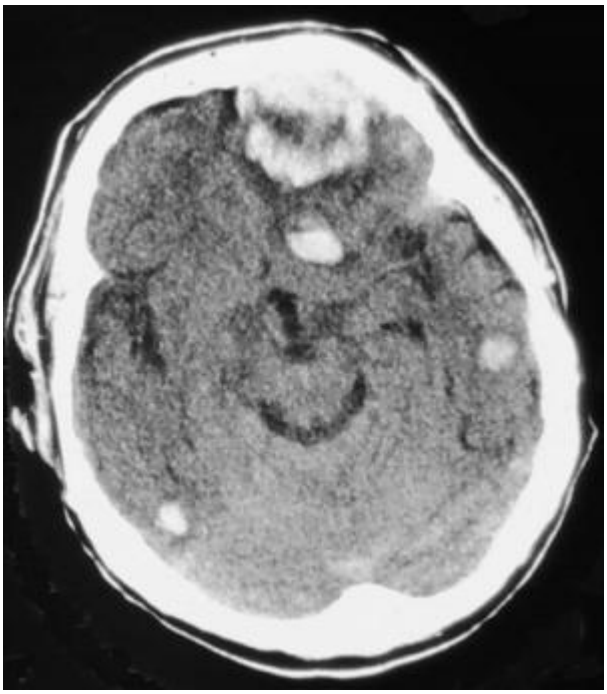

Figure 7: CT appearance of cerebral contusions

Parenchymal lesions are by far the most frequent lesion in TBI. They cover a wide range, moving from hypo-attenuated lesions without bleeding to lesions with a dense haemorrhagic core.

Traumatic contusion core is affected by an irreversible damage of the tissue. Bleeding can occur within this area in a variable manner, from none to ‘salt and pepper’ appearance on CT, to larger lesions. In the case of larger lesions they are more

appropriately defined as a traumatic haematoma. In such lesions, a core bleeding (hyperdense lesion) predominates at CT. Traumatic haematomata are frequently larger than traumatic contusions but there is no clear cut-off defined. The initial CT scan often does not represent the definitive size of the contusion or haematoma since these lesions can enlarge even days after the original trauma. The enlargement of the core is unlikely after the first days.

However, a further evolution can involve an increase of the intraparenchymal lesion via growth of perilesional oedema. This usually peaks at the end of the first week post injury.

Space-occupying (mass) effect, neurological state and general condition of the patient are all variables influencing the management. Relevant for surgical decision is also the location of mass lesion in an eloquent area (e.g. left temporal lobe). A conservative approach, applying a higher level of medical therapy, is suggested in such cases. The previous general medical condition of the patient and complications during ICU stay may argue in favour of surgery to decrease the risk of further functional impairment.

Task

Test your knowledge

In relation to head injury, arrange intra- and extra-axial haematoma e.g. intracerebral, subdural and extradural haematomata in order of frequency?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Intracerebral, subdural, extradural.

Remain vigilant particularly in the early stages of head injury. Do not consider ICP a surrogate for CT at least in the first phases. Any progressive increase of ICP would indicate a need to repeat the CT to disclose any potential mass evolution.

Task

Test your knowledge

We have emphasised the importance of early assessment in TBI. Is a negative CT scan on admission sufficient to rule out intracranial haemorrhage?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Most are diagnosed on the first CT, but they can arise or enlarge even days after the injury. Therefore a repeat CT scan is suggested in all patients with a severe or moderate injury within 6 hours after the initial CT scan.

In patients in coma, with traumatic subarachnoid haemorrhage (tSAH), fractures or a known history of antiplatelet or anticoagulant therapy, there should be a low threshold to repeat the CT brain to assess for progression.

Although at a poor grade of evidence, surgical guidelines suggest some strong recommendations.

In text References

(Bullock et al. 2006; Bullock et al. 2006; Verweij, Muizelaar and Vinas. 2001)

1. 5. 8. Penetrating injuries

Penetrating injuries are caused by gunshots, knives, and other objects that penetrate the skull and the brain tissue. External evidence is a scalp wound (sometimes quite minor).

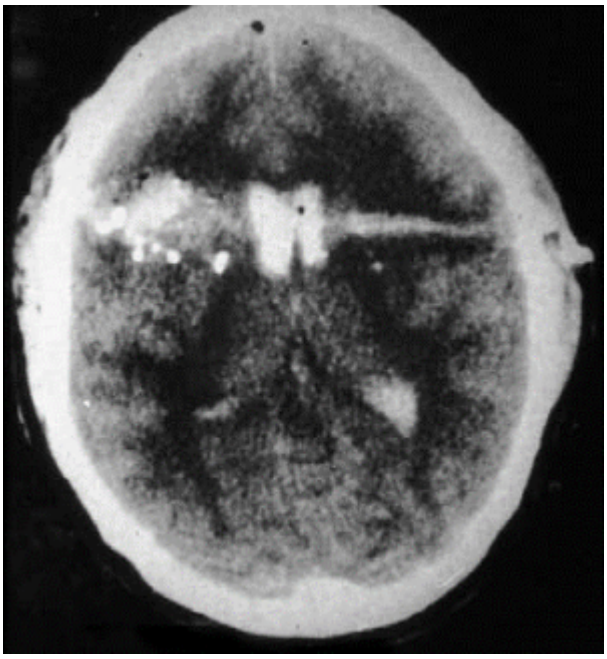

Figure 8: CT appearance of gunshot trajectory in the brain tissue

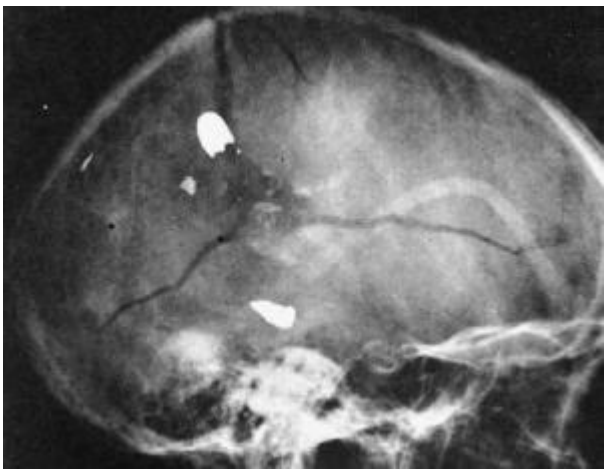

Figure 9: Skull X-Ray showing gunshot point of entry and associated fractures

1. 5. 9. Traumatic axonal injury

Note

Dissociation of clinical and CT findings are often a feature of TAI

Traumatic axonal injury (TAI) without an intracranial mass occurs in almost half of patients with severe head injury. Neuropathologically severe TAI is a microscopically widespread damage to axons, often associated with scattered small haemorrhages and mainly located along or near the midline. It is classified in three subtypes. It may predominate:

- At the junction between the cortex and the white matter and be suspected by the presence of small petecchiae – gliding contusion.(grade 1) or
- In the corpus callosum, often associated with traumatic intraventricular haemorrhage (grade 2) or
- In the thalamus and brain-stem (grade 3).

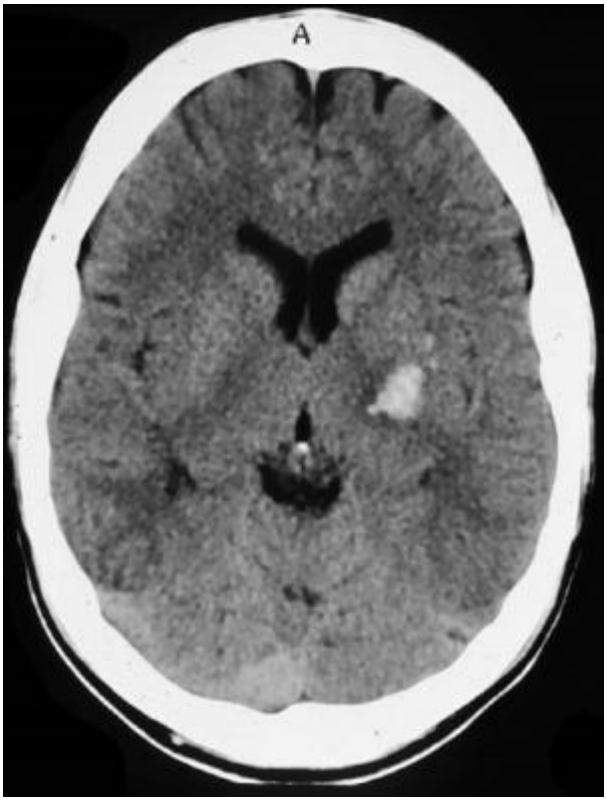

Figure 10: Haemorrhage in posteriorly to the thalamus

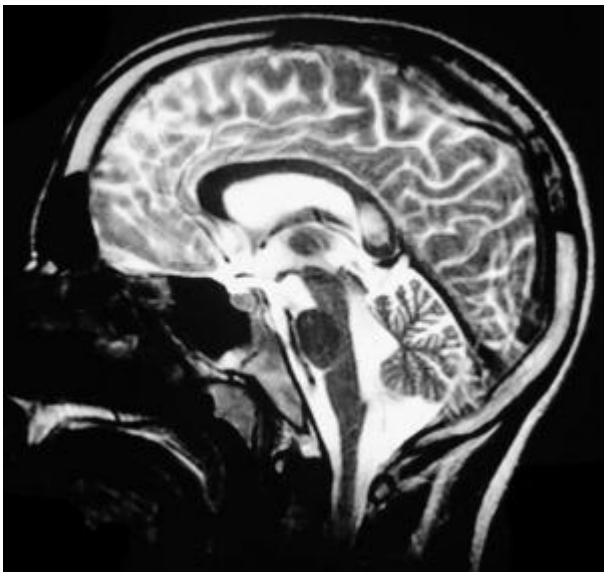

Figure 11: T2-weighted image showing haemorrhagic injury in the mid-brain and pons

Patients with severe TAI are often in deep coma in contrast to a CT scan that often looks quite normal. In such cases but even in those with overt CT findings, an MRI during the acute phase is suggested. T2-weighted or FLAIR MR sequences will reveal the cerebral damage and oedema to a greater extent than CT. Sequences tuned for susceptibility artefacts, for example Gradient Echo or Susceptibility Weighted Imaging can emphasise haemorrhagic lesions more clearly. The extent and distribution of lesions has been associated with prognosis.

The morphologic pictures are complex and a tight relationship between clinical patterns in acute phase and long-term outcome have not been yet established. However the early MRI may help in the decision to monitor ICP, to estimate the expected duration of coma and the need for mechanical ventilation and tracheostomy. Furthermore it could ultimately be of help in planning the most appropriate rehabilitation care.

In text References

(Adams et al. 1989; Graham. 1996; Moen et al. 2014)

1. 5. 10. Cerebral oedema

Cerebral oedema is a consistent reaction of the brain to a variety of insults. It usually develops during the first three to five days and causes an increase in intracranial pressure, thus creating a potential vicious cycle (see diagram below).

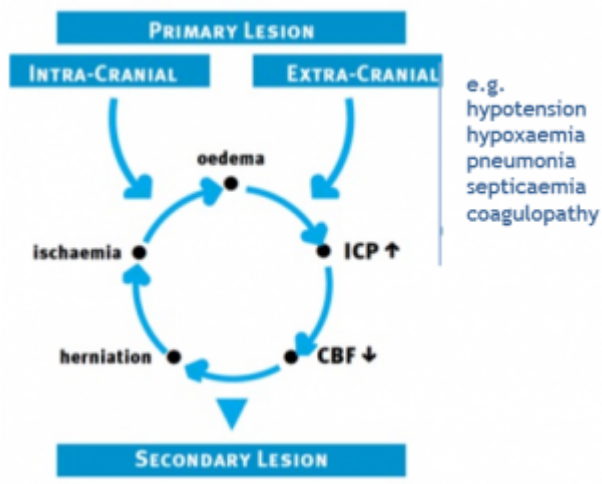

Figure 12 : Cascade of effects of cerebral oedema

Cerebral oedema signifies an increase in the brain water content. There are three different types of cerebral oedema – vasogenic, cytotoxic, and interstitial.

Cerebral oedema should be distinguished from brain swelling, which a more generic term describing just a homogeneous increase of brain volume independently from the cause. It may be associated with specific intraparenchymal lesions.

Patients with head injuries usually have a mixed type of oedema: vasogenic and cytotoxic.

1. 5. 11. Diffuse cerebral oedema

The most common imaging applied to TBI patients is CT but it is unable to detect diffuse cerebral oedema. The reduction of the difference between the density of the grey matter toward that of the white matter is a subjective, operator dependent, index of cortical oedema (hypoattenuation of the grey matter).

CT scan can suggest an increase of global cerebral volume and this picture is called ‘diffuse brain swelling’ to avoid any evaluation of the nature of this volume increase. Magnetic Resonance Imaging has the potentiality to quantify diffuse oedema.

In text References
(Marmarou et al. 2006)

1. 5. 12. Focal cerebral oedema

Unlike diffuse cerebral oedema, CT can easily detect focal oedema. In trauma it is commonly appreciated in cases of traumatic contusion/laceration. It may appear immediately or evolve over days. Initial and central oedema is probably the expression of tissue disruption and cytotoxic oedema.

In contrast, the oedema that enlarges concentrically from the core over days is predominantly vasogenic oedema. A further subtype of oedema has been described, osmotic oedema. This type of oedema is located in the central area of tissue disruption where water accumulates driven by the high osmotic forces due to concentration of cellular debris.

In the acute phase after injury DTI may be used to characterize cytotoxic and vasogenic oedema around contusions, providing insights into a potentially reversible “traumatic penumbra,” a potential target for prevention of secondary injury.

Further oedematous lesions are those affecting post-traumatic infarction (predominantly cytotoxic), the non-contused cortex behind subdural haematoma, or the oedematous area of cerebral venous infarction (predominantly vasogenic).

The estimation of the subtype of the oedema by means of just CT is not possible and its interpretation is simply conjectural on the basis of clinico-anatomic knowledge. MRI can define case by case the respective relevance of vasogenic or cytotoxic oedema. This evaluation is not academic because vasogenic oedema is potentially reversible and should be spared by surgery. Futhermore in the near future the use of medical therapies may be guided by this knowledge.

Task

Test your knowledge

What is the quantitative relationship between the volume of cerebral oedema and the increase in ICP?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

The ICP increases with increasing cerebral oedema but there is no direct quantitative relationship.

Is the duration of the pathology a major determining factor in this relationship?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

The duration of oedema/space-occupying lesion is an important determinant of how much the ICP rises – in more chronic lesions there is a greater volume reserve than in acute conditions.

In a patient who has deteriorated neurologically, what are the CT scan features which raise the suspicion of increased ICP and how would you confirm this?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

The third ventricle becomes obliterated and the basal cisterns compressed. In the case of mass lesion an enlargement of the lesion, an increase of shift, distortion of the ipsilateral perimesencephalic cisterns, compression of ventricular horns, or the presence of unilateral hydrocephalus.

How good are these CT features in predicting elevated ICP. How would an elevated ICP be confirmed?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

While these radiological parameters are associated with elevated ICP, their predictivity remains poor. In younger and paediatric patients, high ICP can be overestimated. Suspicion of raised ICP can be confirmed by measuring it.

In text References
(Miller et al. 2004; Graham and Ward. 1999; Newcombe et al. 2013; Hirsch et al. 2002)

1. 5. 13. Brain-stem lesions

Brain-stem lesions have been traditionally believed to be invariably associated with a poor outcome. Their pathogenesis is however heterogeneous and the ability to detect and understand this is easier now due to MRI. Recently a clinico-radiological classification which is MRI based has been proposed:

- Secondary to supratentorial herniation (mass lesion with shift)
- As a part of severe diffuse brain injury (the grey-white matter junction, corpus callosum, basal ganglia/internal capsule/thalamus)
- Isolated/remote brain-stem injury (without supratentorial lesion due to diffuse injury).
- There is also evidence that dorsal brainstem TAI, especially involving the ascending arousal network nuclei, may have greater prognostic utility than the total number of lesions in the brain or brainstem.

In text References
(Izzy et al. 2017; Mannion et al. 2007)

1. 5. 14. Post-traumatic cerebral ischaemia and infarction

Post-traumatic cerebral ischaemia (PTCI) includes functionally impaired yet still viable tissue, so-called ischaemic penumbra, and irreversible cerebral infarction. It is recognisable on CT as a new hypoattenuation area, without a perilesional distribution. Post-traumatic cerebral infarction can be present with three different patterns:

- Territorial cerebral infarction (complete or incomplete): well circumscribed hypodense lesions within a defined cerebral vascular territory, involving the entire territory (complete) or only part of it (incomplete).
- Watershed cerebral infarction: well circumscribed hypodense lesions located in boundary zones between the territories of anterior, middle and posterior cerebral artery (superficial or leptomeningeal border zones) or situated in terminal zones of perforating arteries within the deep white matter (deep or medullary border zones).

- Non-territorial non-watershed cerebral infarction: single or multiple hypodense lesions, unilateral, bilateral, or multifocal with marked borders without a precise localisation in a vascular territory.

Post-traumatic cerebral infarction is associated with more intracranial hypertension and with a poorer prognosis.

In text References

(Marino et al. 2006)

1. 5. 15. Post-traumatic venous infarction

Thrombosis of a major intracerebral sinus and veins may occur frequently in association with fractures of the vault. It can be suspected in the presence of atypical haemorrhage with large volume oedema which evolves over the first hours.

Diagnosis should be confirmed by means of CT-Angiogram or MR-Angiogram, which is the preferred modality.

MRI is of specific interest for its capability to detect oedema in the territory of the occluded veins.

This condition is frequently associated with seizures, which may aggravate intracranial hypertension.

The treatment may be surgical, if secondary decompression seems appropriate, or medical. Treatment with heparin is usual.

In text References

(Einhäupl et al. 2006)

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1. 6. Blast injury

In recent years, patients more frequently present with injuries not due to blunt or penetrating trauma. The current Iraq conflict and the prominent role of improvised explosive devices (IED) dramatically increased the fraction of war-associated TBI. This perhaps led to the well publicised view that blast-induced traumatic brain injury (bTBI) is the signature brain injury for combat troops in today's military. The Centers for Disease Control and Prevention (CDC) defines blast injury in four phases. However, the bulk of bTBI occurs in the first three phases: the primary injury phase is comprised of the response of brain tissue to the blast wave (an intense overpressurisation impulse component of the blast). The secondary injury phase results from shrapnel penetration into the head. The tertiary injury phase results from head contact/acceleration forces as the body is moved by the 'blast wind' (a forced super-heated air flow). The quaternary injury phase incorporates any injury not covered in the other three phases such as some of the extracranial injuries or 'polytrauma' including haemorrhagic shock and chemical or thermal burn injuries that can occur. This quaternary phase of bTBI can significantly alter the timing and consequences of the primary damage occurring in the first three phases, and therefore can be a major contributor to overall brain pathology. This may be particularly true in mild bTBI, where there are either minor or no complicating factors.

In text References

(Chen and Huang. 2011)

References

- Chen Y, Huang W., Non-impact, blast-induced mild TBI and PTSD: concepts and caveats., 2011, PMID:21604927
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1. 7. Cellular and molecular events

Recent research has shed light on changes at cellular and molecular level in TBI. Glutamate and other 'excitotoxic' transmitters released from damaged neurones generate unnecessary action potentials and squander cellular energy resources. Failing tissue perfusion can lead to the build up of metabolites such as lactate and highly reactive free radicals, which damage the membranes of neurones and their organelles. Compromise of structure and function can lead to distortions in transmembrane ion gradients, a catastrophic rise in the cytoplasmic calcium concentration, and cell death. These various changes are summarised in the diagram below.

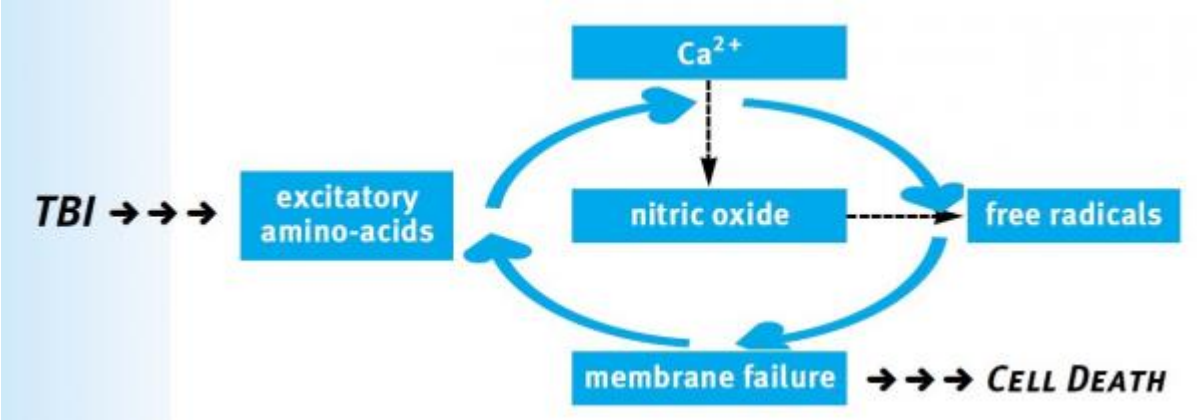

Figure 13: Effects of injury at a cellular and molecular level.

We have discussed the various pathophysiological categories of secondary brain injury and considered how the condition manifests itself clinically and radiologically. The vicious cycle of cerebral ischaemia, cerebral oedema and raised ICP has been noted as a constant threat in TBI. Advances in neurobiology form the basis for future therapeutic strategies. In the next two sections we shall examine this issue further in relation to the assessment and treatment of patients with severe head injury.

Note

As always, prevention is better than cure.

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