

A computed tomography pictorial review of head injury imaging

Computed tomography imaging of the head is commonly used in the investigation of traumatic brain injury. Emergency department personnel often need to interpret these images without expert radiological assistance. This article reviews the common computed tomography appearances of the head following traumatic brain injury.

An estimated 1 million patients per year attend hospital with head injuries of which 40% are the result of falls, 20% the result of assault and 13% the result of road traffic accidents (Kay and Teasdale, 2001). They are responsible for 1% of all deaths and 15–20% of deaths in 5–35-year-olds (Teasdale, 1995).

The National Institute for Clinical Excellence (NICE) issued head injury guidelines in June 2003. The implementation of these guidelines has seen a substantial increase in the number of computed tomography (CT) scans of the head undertaken at all hospitals with an emergency department. An understanding of the common CT appearances following traumatic brain injury is necessary.

Figure 1. Standard window settings for optimal brain imaging showing a depressed skull fracture.



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Basic points for interpreting CT scans of the head

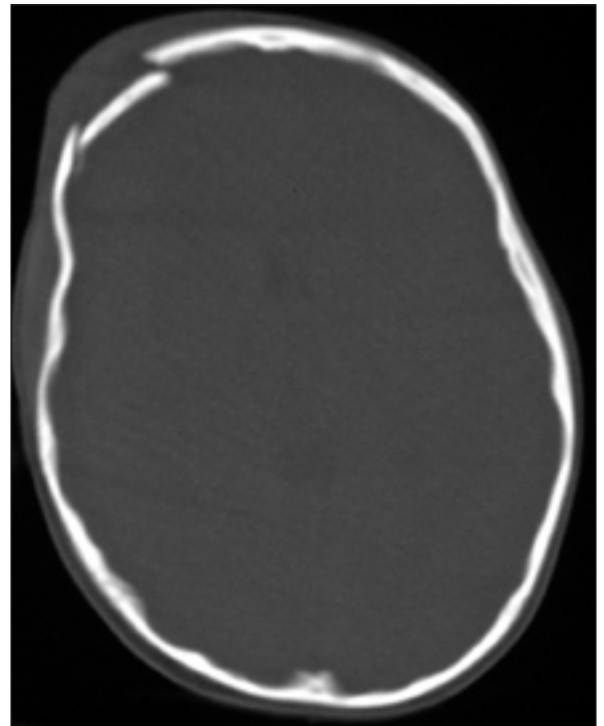
First, CT images are usually presented in a manner best suited for visualizing the brain (*Figure 1*), but in trauma cases the images should also be viewed in a way which allows study of the bones (a bone window setting) (*Figure 2*).

Second, before starting to analyse the main images, look at the scannogram which will be included in the majority of cases (*Figure 3*). This provides a crude overview of the skull bones in the lateral projection. Frequently, fractures will only be seen on this view.

Regarding fractures of the skull vault, depressed fractures are easily appreciated on CT, but non-depressed bone injuries are often difficult to see. Skull base fractures are notoriously difficult to detect.

On CT images, unclotted blood has the same density as brain (grey) while clotted blood (haematoma) appears hyperdense relative to brain, i.e. white. Even this simple

Figure 2. Bone window settings allow easier appreciation of the fracture but render brain detail more difficult to appreciate.



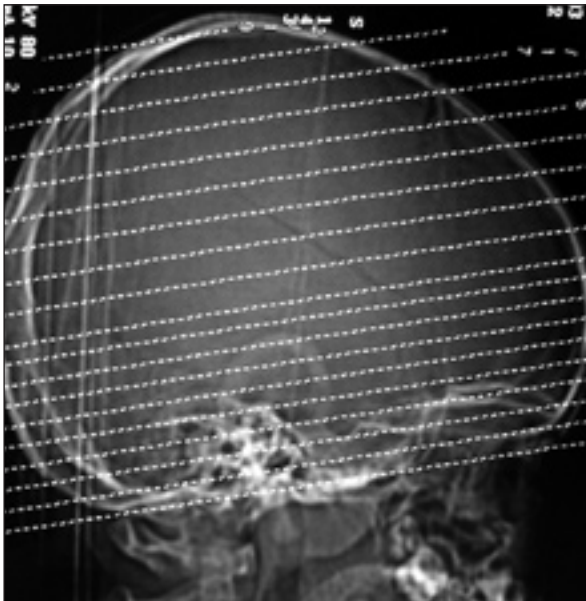


Figure 3. A lateral digital image of the skull, the superimposed lines representing the computed tomography slices. Note the linear fracture crossing the temporal and parietal bones.

statement needs qualification. The degree of whiteness depends on the patient's haemoglobin (Hb) concentration at the time of haemorrhage (strictly, the haematocrit). Therefore a haematoma in a young man with an Hb of 14 g/dl will appear much whiter (hyperdense) and will take longer to fade than one in an elderly female with an Hb of 9 g/dl. Initially the latter may seem only slightly hyperdense and will quickly fade to isodense and then hypodense (*Figures 4a and b*).

To facilitate understanding, this article will look at bleeding into each of the intracranial compartments in turn, but it should be remembered that an individual patient may have several coexisting traumatic lesions.

Extradural haemorrhage

Haemorrhage into the extradural space is usually caused by a tear of a meningeal vessel by a skull fracture. The accumulating haematoma strips the dura mater from the inner table of the skull vault producing a biconvex mass (*Figure 5*). When it consists entirely of clotted blood the CT appearance is homogeneously white. Owing to the absence of any tamponade, an extradural haemorrhage (EDH) is a surgical emergency requiring clot evacuation.

If the fracture is open or compound, air may enter the cranial cavity via the fracture. Air appears black on CT. (*Figure 6*).

Differing densities may be seen within an EDH because of the presence of clotted and unclotted blood (*Figure 7*). Whereas clotted blood appears white, the unclotted blood is grey, i.e. it is isodense with brain. The appearance is referred to as layering. The three main causes of an EDH with layering are:

1. Active haemorrhage occurring at the time of the scan

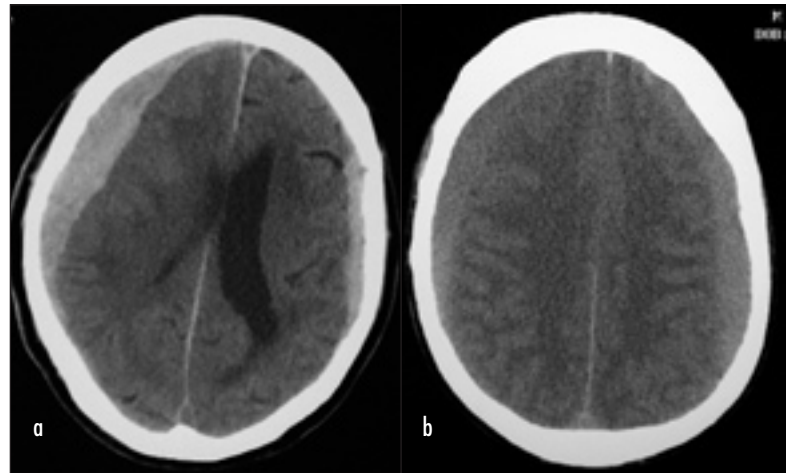


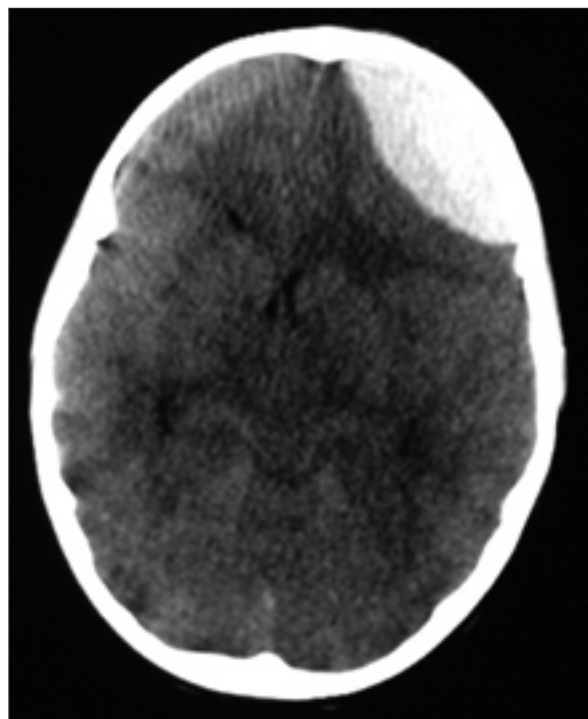
Figure 4. a. A biconcave hyperdense (white) subdural haematoma (SDH) is seen on the patient's right side with a very thin SDH on the opposite side. Haemoglobin concentration was 14 g/dl and the image was made 24 hours after injury. b. Bilateral SDH are seen in this elderly patient 36 hours after injury. Haemoglobin concentration was 9 g/dl and already the haematomata are almost the same density as brain.

2. Anticoagulant therapy causing a delay in the clotting time
3. Multiple injuries (polytrauma) which have consumed the available clotting factors – consumption coagulopathy.

Subdural haemorrhage

Bleeding into the subdural space between the dura mater and the arachnoid mater (subdural haemorrhage; SDH) is usually caused by rupture of a bridging vein(s) between the cortex and the venous sinuses. These veins are more

Figure 5. Extradural haematoma on patient's left side. Biconvex and uniformly hyperdense.



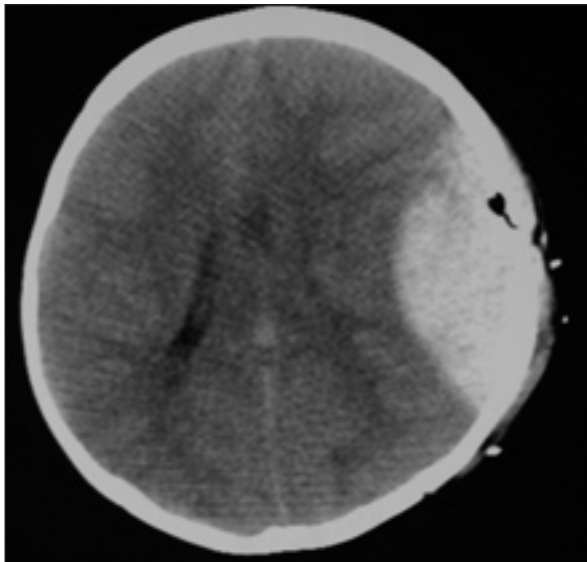


Figure 6. Extradural haematoma on patient's left side. Biconvex and uniformly hyperdense. Note compound fracture with air (black) tracking through it into the haematoma.

prominent in atrophic brains, such as one sees in the elderly and in alcoholics, where bleeding can often be the result of minimal trauma. The accumulating venous blood tracks between the meningeal layers producing a hyperdense crescentic mass (Figure 8). Accumulation of the haematoma is slower than in an EDH and limited by the tamponade effect of the dura mater.

With the widespread use of anticoagulants, particularly in the elderly, an SDH may also contain clotted and unclotted blood (Figure 9).

Figure 7. Right extradural haematoma of differing densities. Hyperdensity (white) represents clotted blood; isodensity (grey) is unclotted blood – active arterial bleeding during scan.

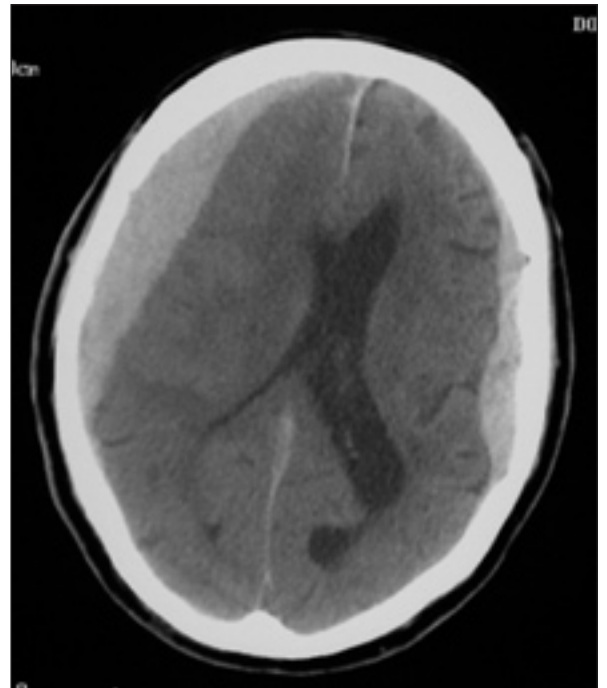
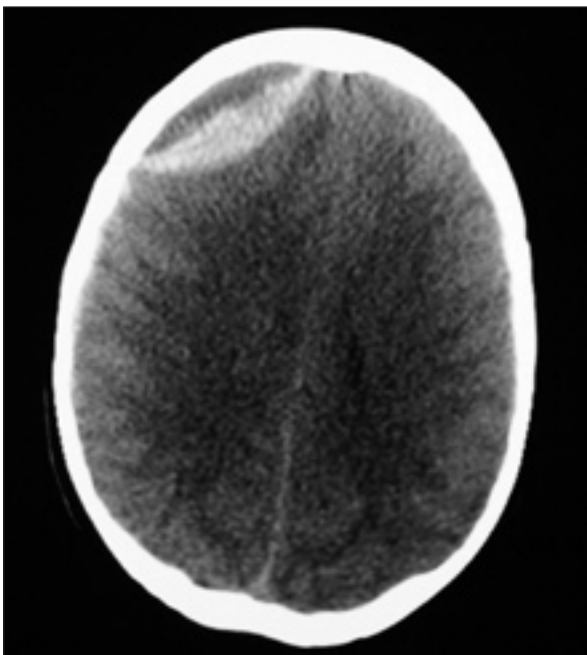
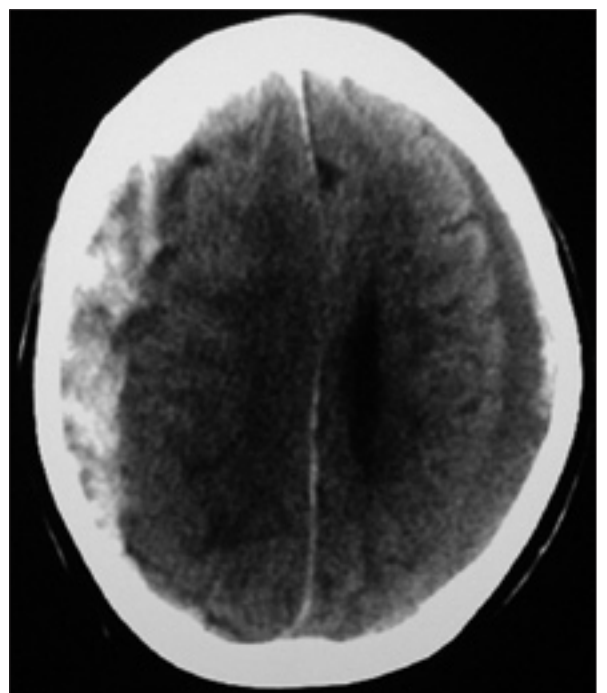


Figure 8. Bilateral subdural haematomata. Uniformly hyperdense and crescentic.

Subarachnoid haemorrhage

Post-traumatic subarachnoid haemorrhage (SAH) is rare in isolation. It is often not clear if a spontaneous SAH causes the patient to collapse and sustain a head injury or whether the SAH is the result of a primary head

Figure 9. Bilateral subdural haematomata (SDH). On the right, clotted and unclotted blood seen as white and grey respectively. On the left, SDH is darker than brain representing a chronic collection. Patient on anticoagulants.



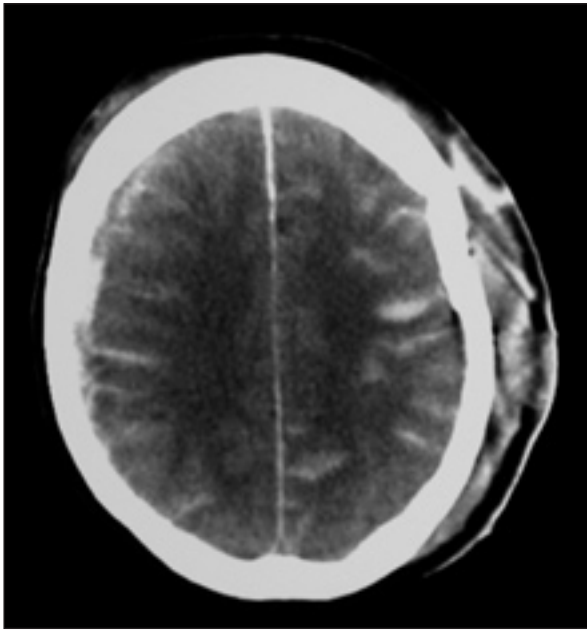


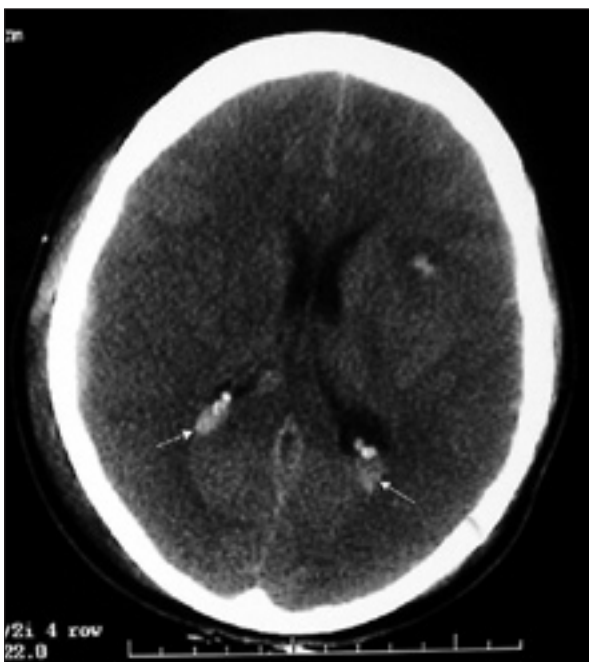
Figure 10. Clotted blood fills the cerebral sulci, an extension of the subarachnoid space at the vertex. An extracranial scalp haematoma is noted on the left.

injury. In the latter, acceleration-deceleration forces are the usual cause and in these circumstances the finding is associated with a poorer outcome. Clotted blood may fill the cerebral sulci (Figure 10).

Intraventricular haemorrhage

Post-traumatic intraventricular haemorrhage (IVH) is also a bad prognostic finding. In isolation it represents accel-

Figure 11. Clotted blood has settled in the dependent parts of both lateral ventricles producing blood/CSF levels (arrows). A tiny intracerebral haematoma is seen on the left.



eration-deceleration injury, but can result from rupture of an adjacent intracerebral haematoma (ICH) (Figure 11).

Intracerebral haemorrhage

Superficial ICH, also called cortical contusion or petechial haemorrhage, is common. Frequently the consequence of blunt impact to the adjacent bone, it is one cause of post-concussion syndrome. The patient usually makes a full recovery (Figure 12).

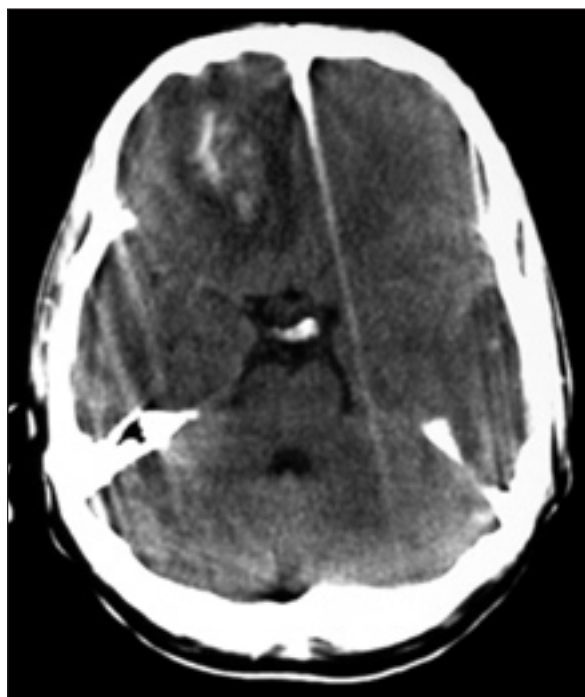
Deep ICH can destroy white matter tracts. The mechanism of injury is again direct impact, but of greater severity (Figure 13).

A haematoma may be seen on the side opposite the point of impact. The intervening brain tissue transmits a pressure wave which generates a contra lateral build up of energy with consequent brain injury and haematoma. This is referred to as a 'contra coup' lesion (Figure 14).

Diffuse axonal injury

Diffuse axonal injury (DAI) occurs as a result of shearing or acceleration/deceleration forces across the brain, from side to side or front to back. This results in a tear, typically in the white matter. Most of these are invisible to CT imaging, but pathologists have described them for many decades. They are the commonest cause of death in those surviving the immediate effects of traumatic brain injury. They are also a major cause of disability in the survivors. Occasionally, if the tear is large enough, it may be possible to see clotted blood within it on a CT image (Figure 15). Characteristically, they involve white matter tracts or fibres close to the midline, e.g. within the brainstem, vermis, corpus callosum or parafalcine

Figure 12. Superficial contusional haematoma right frontal cortex and subcortex.



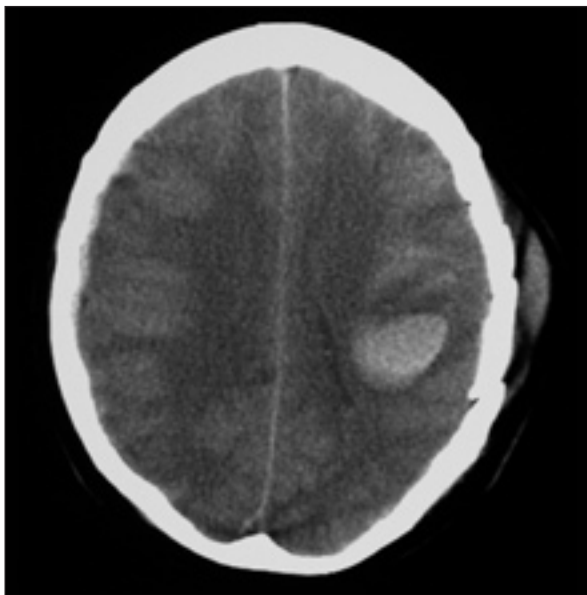


Figure 13. Extracranial soft tissue swelling, a fracture and a deep intracerebral haematoma on the left. On the opposite side there is a tiny subdural haematoma.

areas. They can also be seen within the subcortical white matter of the cerebral hemispheres where they present a radial pattern (*Figure 16*).

Pneumocephalus

Air can gain entry to the cranial cavity by a number of means: directly via a penetrating wound, via a compound vault fracture (as shown previously) or following a fracture through an air-containing sinus (e.g. frontal, mastoid or sphenoid) (*Figure 17*).

Figure 14. Direct impact, cortical haemorrhage on the right with contra coup intracerebral haematoma on the left. Diffuse brain swelling is present.

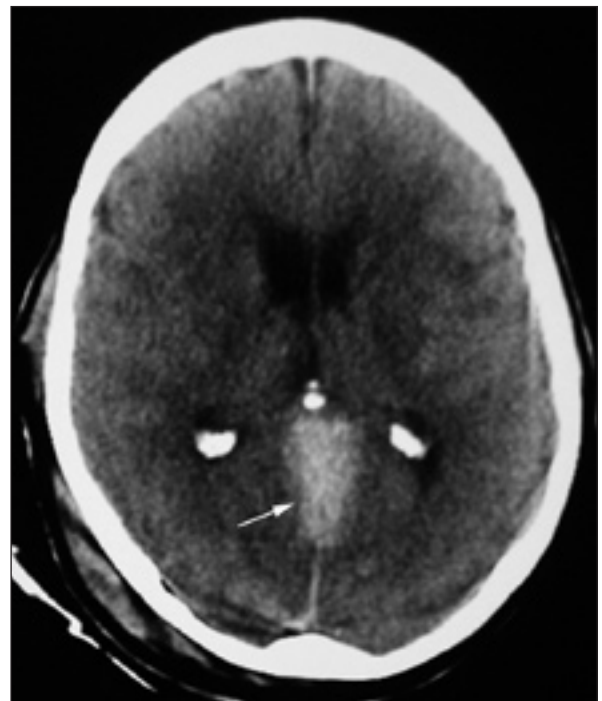
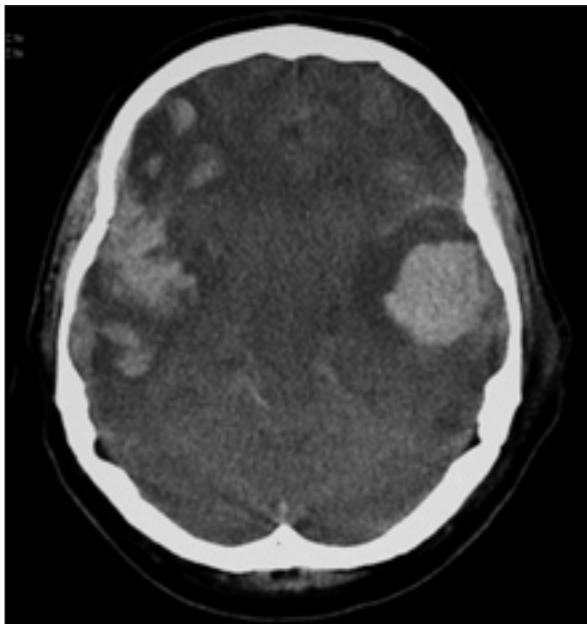


Figure 15. Diffuse axonal injury has caused bleeding within the superior vermis under the tentorium (arrow).

Depressed skull fracture

These result from point impact to the vault, often in a criminal setting. The first example followed an assault with a baseball bat (*Figure 18a*). In the second case a hammer was used (*Figure 18b*).

Figure 16. Diffuse axonal injury with haemorrhage into a radial tear extending from the cortex on the right (arrow), and bleeding alongside the falx anteriorly and at the back (arrowheads).





Figure 17. Subarachnoid haematoma (large arrow); haematoma in fourth ventricle (small arrow) causing hydrocephalus (dilated temporal horns); bubbles of air (arrowheads) – entry through midline compound frontal fracture.

A special category of depressed skull fracture is only encountered in children. It may be thought of as akin to a greenstick fracture of the vault in that the bone is depressed without a through-and-through break – in effect the bone buckles. It is known as a ping pong depression after the similar appearances found in table tennis balls (Figure 19).

Figure 19. Ping pong 'fracture'; buckling of vault on right.

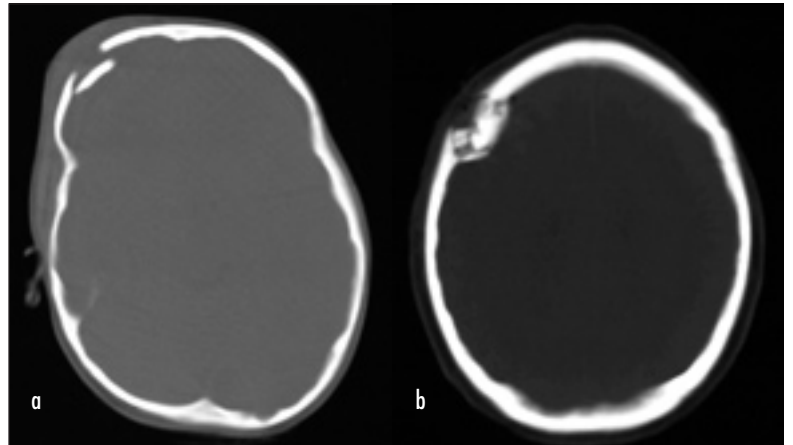
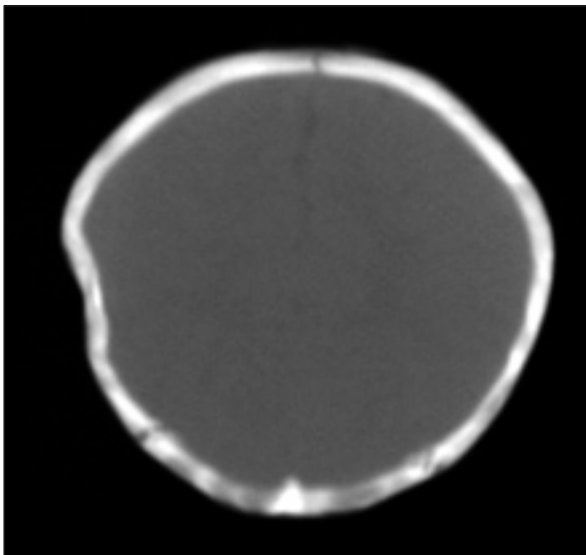


Figure 18 a and b. Depressed skull fractures on bone window settings.

Skull base and other fractures

If a fracture tears the meninges, CSF from the subarachnoid space mixes with fresh blood from torn vessels creating a haematoma where the denser blood sediments presenting a blood/CSF level (Figure 20).

Skull base fractures may be presumed if unilateral opacification of the petromastoid air cells is seen, especially if accompanied by CSF rhinorrhoea or bruising/swelling over the mastoid process (Battle's sign). Although a fracture may not be seen it should be presumed (Figure 21).

Sometimes the actual fracture line can be seen, but only if bone window images are used (Figures 22 and 23).

Figure 20. Fracture on left has torn the meninges. CSF from the subarachnoid space mixes with fresh blood from torn vessels creating a haematoma with a blood/CSF level.

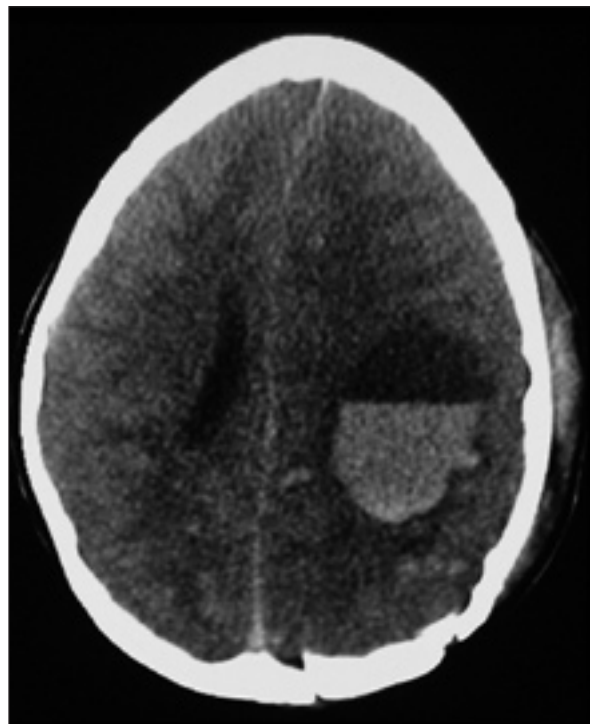




Figure 21. Clotted blood renders the mastoid region opaque on the right (arrow). Compare with the well-aerated mastoid cells on the left.

Conclusions

CT imaging of the head is the first investigation of choice where there is suspicion of traumatic brain injury. Understanding the anatomy of the meninges in relation to the brain and the cranial vault alongside an awareness of the common CT appearances of the brain post-traumatic brain injury will inform diagnosis and guide management. **BJHM**

Conflict of interest: none.

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KEY POINTS

- Traumatic brain injury is a significant cause of death.
- Computed tomography imaging of the head following traumatic brain injury is fundamental to accurate diagnosis.
- Do not forget to look at the scannogram, skull fractures may only be evident on this image.
- Request bone window images.
- Consider the patient's haemoglobin concentration/haematocrit and whether he/she is taking anticoagulants. Both can affect the appearance of a haematoma.
- A haematoma may be present on the side contralateral to the point of impact.
- Opacification of air sinuses suggest a skull base fracture which might otherwise not be evident.

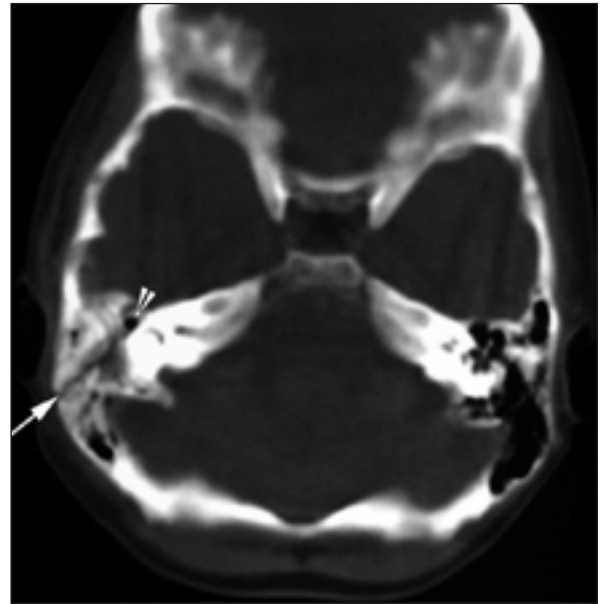


Figure 22. Compound fracture through petromastoid on right (arrow). Air within cochlea (arrowhead) indicates permanent hearing loss. Note opacification of the air cells by blood clot.

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Figure 23. Fracture extends across left petrous bone.

