1.1 General principles

A CT scan of the brain is the most commonly performed cross-sectional imaging during out-of-hours periods. See Fig. 1.1.

Indications

Altered Glasgow Coma Scale (GCS), acute confusional state, sudden onset severe headache, head injury, drowsiness, status epilepticus, post-epileptic with decreasing consciousness level or with focal neurology, anoxic–hypoxic injury to brain (post-cardiac arrest).

Technique

Axial CT brain.

Contrast

Intravenous (IV) contrast is usually not needed except in cases of suspected intracranial or meningeal infection, arteriovenous malformations (AVMs) and suspected tumors. Following a road traffic accident (RTA), an initial non-contrast head scan should be carried out to exclude extra-axial or intracranial hemorrhage before a dynamic contrast scan of chest or abdomen. A contrast-enhanced CT scan of the brain may be helpful in suspected isodense subdural hematoma.

Review areas in a “near normal” CT head scan:

1. Foramen magnum: High density blood around the brainstem – sub-arachnoid hemorrhage (SAH), tight foramen magnum–tonsillar herniation suggests raised intracranial pressure.
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2. Cerebello-pontine (CP) angle and pituitary fossa: Isodense soft tissue mass – give IV contrast.


5. Vessels: Dense middle cerebral (MCA) and basilar arteries may represent early thrombus – look for infarct.


7. Ventricles: High-density blood/CSF level in the dependent posterior horn ventricle – a sign of SAH, extending into the ventricle.

8. Interhemispheric fissure: High density in SAH, non-accidental injury (NAI).

9. Sulci and gyri: Asymmetry, mass effect, isolated high density within the sulci (traumatic SAH), effacement insular ribbon – sign of early infarction.

10. Temporal lobe: Unilateral hypodensity may be due to herpes simplex encephalitis. Can be bilateral (often difficult to differentiate from artefact). Suggests lumbar puncture and MRI.

11. Superior slices of the scan: Always ensure that the uppermost slices of the brain are obtained to look for hidden pathologies like parafalcine meningioma, traumatic SAH or a small subdural bleed.

12. Bones: Review mandibular condyles, mastoid bones, skull base, inferior orbits, posterior occipital bone, foramen magnum as well as C1 and C2 on bone windows.

Fig. 1.1. (a) Traumatic SAH. Axial non-contrast CT shows linear hyperdensities within the cerebral sulci (arrows), consistent with blood in the subarachnoid space. (b) Spontaneous SAH. Axial non-contrast CT shows hyperdensity anterior to the brainstem at the level of the foramen magnum (arrow), where only hypodense CSF should normally be present. Note the tight foramen magnum without free CSF space.
1.2 Acute subarachnoid hemorrhage

Blood in the subarachnoid spaces. See Fig. 1.2.

Clinical

Sudden onset of severe headache associated with nausea, vomiting or altered level of consciousness. Altered conscious level and/or focal neurological deficit warrants an urgent CT brain to identify hydrocephalus and/or hematoma, which may need urgent neurosurgical treatment. 75–80% of SAH is due to spontaneous rupture of cerebral aneurysms. The site of hematoma is not considered as a reliable indicator of the site of aneurysm.

Technique

Non-contrast CT brain. Computerized tomographic angiography (CTA) of the brain can be performed to look for ruptured intracranial aneurysm as the cause of SAH.

CT brain findings

1. High attenuation (blood) in the cisterns and sulci. Typically blood is present in the suprasellar cistern near the circle of Willis.
   - Sylvian fissure/temporal lobe hematoma – in middle cerebral artery aneurysm rupture.
   - Frontal horn and frontal lobe blood – anterior communicating artery aneurysm rupture.
   - Blood in the interpeduncular and CP cistern and extending to brainstem – basilar artery aneurysm rupture.
   - Blood in the 4th ventricle, foramen magnum and around the brainstem – internal carotid artery (ICA) rupture.
2. Blood–fluid levels in the posterior horn of the lateral ventricle suggest intraventricular extension of SAH.
3. Subtle signs of SAH: Blood in the interpeduncular cistern, foramen magnum and ventricle. High-density asymmetrical tentorium cerebelli, and high-density sulci in traumatic SAH.
4. Mass effect: Subfalcine/tentorial herniation. Hydrocephalus – earliest sign is temporal horn dilatation – results from decreased CSF resorption by the arachnoid granulations in the presence of blood. Tight foramen magnum and/or basal cistern effacement are signs of impending coning.

5. An aneurysm may be visible as a rounded soft tissue mass with or without rim calcification which enhances intensely following intravenous contrast.

### Pearls

- Suggest urgent neurosurgical opinion and make sure images are available to the neurosurgeon.
- CT of the brain is very sensitive in the first 48 hours and thereafter its sensitivity falls to 50%. Chronic anemia patients can have low-density blood. A negative CT brain requires lumbar puncture to exclude SAH.
- CT cerebral angiography with or without more invasive digital subtraction angiography is often required at a later stage to identify aneurysms.
- Perimesencephalic bleeding is due to venous hemorrhage with no aneurysm and usually carries a good prognosis.
- Normal cerebellar tentorium reflection is symmetrical and uniformly of high density with well-defined anterior concave margin towards the ventricles.
Suggested reading


### 1.3 Acute subdural hematoma

Extra-axial collection of blood between the dura mater and cortical surface of the brain is due to venous hemorrhage. See Fig. 1.3.

#### Clinical

Headache, drowsiness, focal neurology and unequal pupils.

Etiology: Trauma, surgery, shunts, anticoagulant therapy.

#### Technique

Unenhanced CT brain is the first procedure of choice. Intravenous contrast may be helpful to identify an isodense subdural hematoma. MR is particularly sensitive in its detection.

#### Findings

1. Concavo-convex high-density extra-axial collection of blood seen along the surface of the brain with concavity towards the midline.

2. Fluid level due to different stages of hematoma. High density in acute (<1 week), intermediate/isodensity recent (1–2 weeks) and low density/CSF density represents 2–3 weeks old. High-density acute blood is seen in the dependent posterior subdural space in a supine position.

3. Mass effect: Effacement of ipsilateral sulci and ventricles, sub-falcine or sub-tentorial herniation with contralateral ventricular hydrocephalus.

4. *Contre coup* parenchymal contusions or hematoma.

5. Associated skull vault and basal skull fractures and soft tissue hematoma.

#### Pearls

- Subdural hematoma (SDH) is crescent shaped (concavo-convex) with the concavity towards midline of the brain. Extradural hematoma (EDH) is biconvex or lens shaped with the convexity towards the midline. EDH is usually smaller in volume compared with SDH. EDH does not cross the suture line. Subdural hematomas cross the suture line of the skull vault but do not cross the interhemisphere fissure.

- Small SDH: can be easily missed. Look for apparent asymmetrical “thickening” of the skull vault, effacement of cortical sulci and lateral ventricle, and dilated temporal horns. You should always widen the window width to better evaluate for subtle bleeds along the calvarium (WL 40 WW 150).

- Isodense SDH is associated with a subacute presentation and has similar density to the adjacent brain tissue. Look for sulci buckling, and absence of sulci at the peripheries.
Fig. 1.3. (a, b) Acute SDH. (a) Non-contrast CT shows a subtle thin rim of hyperdensity (arrow) along the right inner table of the skull, with mild sulcal effacement. (b) The same patient’s study with "hematoma windows" (L40, W150) more easily demonstrates the hematoma (arrow). (c) Acute on chronic SDH. Non-contrast axial CT image shows fluid–fluid level (arrow), with higher-density acute blood layering dependently. Note the marked mass effect with effacement of the left lateral ventricle and midline shift to the right. (d) Isodense SDH. Non-contrast CT shows effacement of sulci on the left side (arrowhead) and a large subdural collection (arrow) that is isodense to cortical parenchyma.
1.4 Extradural hematoma

Extra-axial collection of blood between the dura mater and inner table of the skull is usually due to arterial injury as a result of head trauma. See Fig. 1.4.

Clinical

Refer to Section 1.3.

Technique

Unenhanced CT brain scan.

Findings

1. Biconvex shape of high-density blood located in the periphery of the cranial cavity.
2. Unlike SDH it crosses the dural reflection but not the vault sutures.
3. Mass effect: Subfalcine, tentorial, brainstem herniations and diffuse or localized cerebral edema.
4. Associated vault or base of skull fractures, pneumocephalus and soft tissue hematoma.

Pearls

- EDH represents arterial hemorrhage and it needs immediate neurosurgical attention. Hypodensity within the hematoma represents unclotted active extravasation of blood (the “swirl sign”).
- Alter the window to look for associated skull fractures, subdural hematoma.
- Look for associated signs of traumatic subarachnoid hemorrhage, coup and contre coup parenchymal contusions.

Suggested reading

Refer to Section 1.2.

1.5 Traumatic parenchymal brain injury

Brain parenchymal injury can occur with or without evidence of subdural or extradural hematoma, and is most likely due to traumatic acceleration and deceleration injury. See Fig. 1.5.
Clinical
Headache, altered level of consciousness, falling GCS level.

Technique
Unenhanced CT scan is sufficient.

Findings
Can be classified as contusions, diffuse axonal brain injury, diffuse cerebral edema and brainstem herniations.

Contusions
Focal small hemorrhagic foci are seen usually within the frontal and temporal lobes.
Focal hypodensity areas seen due to associated cerebral edema.

Fig. 1.4. Acute extradural (epidural) hematoma. Non-contrast CT shows a biconvex or lens-shaped hyperdense extradural hematoma (arrow). Note effacement of the right lateral ventricle (arrowhead) and midline shift to the left indicative of subfalcine herniation. Posteriorly it is limited by the lambdoid suture.

Fig. 1.5. (a) Axonal brain injury. Axial non-contrast CT shows gray–white matter interface focal hyperdensity (arrow) in the left frontal lobe. (b) Brain contusions. Axial non-contrast CT shows several hyperdense foci (arrows) along the anteroinferior left temporal lobe, consistent with small intraparenchymal foci of blood.
Diffuse axonal brain injury (DAI)
Small foci of hemorrhage are present at the gray–white matter junction, deep white matter, corpus callosum and brainstem. DAI may be associated with herniations and cerebral edema.

Diffuse cerebral edema
Either due to hyperemia or increased interstitial edema.
- Seen as mass effect causing effacement of cortical sulci, ventricles with associated dilated temporal horns.
- Diffuse low-density cerebral parenchyma due to cerebral edema causes an apparently high attenuation of cerebellum (white sign) relative to the rest of the brain. There is also increased attenuation of the arterial vessels and meninges and effacement of the cisterns (pseudo-SAH).

Brainstem herniations
Subfalcine: Common type with cingular gyri herniations. Distortion of anterior cerebral artery can cause secondary ischemia and infarction in the frontal lobe.
- Tentorial: Due to herniations of temporal lobe and brainstem. This is seen as effacement of basal cisterns, dilatation of temporal horns and midline shift of brain parenchyma. These are all signs of raised intracranial pressure.
- Tonsillar: herniation of the cerebellar tonsils causes a tight and effaced foramen magnum. This is a sign of imminent coning.

Pearls
- Contusions and DAI are seen well on a MR of the brain.
- DAI is associated with high morbidity and mortality.
- Always look for associated extra-axial hematoma, soft tissue injury and skull fracture.
- Coup injuries are smaller than contre coup.

Suggested reading
Refer to Section 1.2.

1.6 Intracerebral/cerebellar hemorrhage
Focal collection of blood in the cerebellum or cerebral hemispheres. See Fig. 1.6.

Clinical
Sudden onset headache, unexplained confusion, altered level of consciousness and focal motor weakness. Risk factors: Hypertension, blood disorders, anticoagulants and amyloid microangiopathy. The most common locations of hypertensive bleeds are the putamen, subcortical white matter, cerebellum, thalamus and pons.

Technique
Non-contrast CT brain.
**Fig. 1.6.** (a) Cerebellar hematomas. Axial non-contrast CT shows large regions of hyperdense hematomas with surrounding hypodensities (arrows) in bilateral cerebellar hemispheres. Note the age-related calcification in the region of the left globus pallidus (arrowhead). (b) Intracerebral hemorrhage. Axial non-contrast CT shows a large hyperdense hematoma in the left basal ganglia and thalamus (arrow) with surrounding hypodensity as well as hyperdensity (blood) within the left lateral ventricle (arrowheads). (c) Pontine hemorrhage. Axial non-contrast CT with abnormal hyperdensity in the pons (arrow) is consistent with hemorrhage into the pons. (d) Sylvian fissure hematoma. Axial non-contrast CT shows a large Sylvian fissure hematoma (arrow) with extension into the cerebral parenchyma, with effacement of the left lateral ventricle and midline shift to the right. There is subarachnoid blood in the right Sylvian fissure (arrowhead).