Subdural Haematoma

Synonym: subdural haemorrhage

Anatomy

The meninges are the connective tissue membranes that line the skull and vertebral canal. They enclose the brain and spinal cord.

- The outermost layer is the dura mater.
- The middle layer is the arachnoid mater.
- The inner layer is the pia mater.
- The epidural space is the space between the vertebral column and the dura mater. There is only a 'potential' epidural space in the skull.
- The subdural space is the space between the dura mater and the arachnoid mater.
- The subarachnoid space is the space between the arachnoid mater and pia mater.

Definition

- A subdural haematoma (SDH) is a collection of clotting blood that forms in the subdural space. This may be:
  - An acute SDH.
  - A subacute SDH (this phase begins 3-7 days after the initial injury).
  - A chronic SDH (this phase begins 2-3 weeks after the initial injury).
- A simple SDH is when there is no associated parenchymal injury.
- A complicated SDH is when there is associated underlying parenchymal injury, such as contusion.
Pathophysiology

An acute SDH is usually caused by either:

- Tearing of bridging veins from the cortex to one of the draining venous sinuses – typically occurring when bridging veins are sheared during rapid acceleration-deceleration of the head.
- Bleeding from a damaged cortical artery.

Blunt head trauma is the usual mechanism of injury but spontaneous SDH can arise as a consequence of clotting disorder, arteriovenous malformations/aneurysms or other conditions.

In the subacute phase the collection of clotted blood liquifies. In the chronic phase it becomes a collection of serous fluid in the subdural space.

At-risk groups

Infants

- In the infant brain, SDHs are caused by tearing of the bridging veins in the subdural space and may result in significant brain injury. Some SDHs are due to physical abuse, so suspicion should be raised[1] but SDH should not be assumed to be always due to this cause in children.
- The so-called ‘shaken baby syndrome’ remains controversial and may have other potential aetiologies than ‘shaking’. [2] It may also be seen in older children. [3]

The elderly

- Cerebral atrophy can occur in people over the age of 60, causing tension on the veins, which may also be weaker and more susceptible to injury as a consequence of age.
- Chronic SDH is more common in this age group. [4]

Those with alcoholism

- Alcohol misuse leads to a risk of thrombocytopenia, prolonged bleeding times and blunt head trauma and is a risk factor for SDH. [5]
- Alcoholism also causes cerebral atrophy which can put tension on the bridging veins.
- People on anticoagulation treatment: [6]
  - Anticoagulation treatment (including with aspirin or warfarin) is another risk factor. [7]

Epidemiology

- SDH can occur in about one third of people with a severe head injury. [8]
- It is more common with increasing age, as described above. One study found a prevalence of 7.35 cases per 100,000 population in those aged 70-79 years. [9]
- A UK-based epidemiological study found that the annual incidence of SDH/effusion in infants is approximately 12.5 cases per 100,000 population in 0- to 2-year-olds and approximately 24 cases per 100,000 in 0- to 1-year-olds. [10] The majority of cases were deemed to be due to non-accidental injury (57%). Other causes included:
  - Perinatal complications.
  - Meningitis.
  - Undetermined cause.
  - Accidental head injury.
  - Non-traumatic medical conditions.
- Spontaneous intracranial hypotension has also been reported as a rare cause. [11]

Presentation [12]

Acute SDH

- Usually presents shortly after a moderate-to-severe head injury.
- Loss of consciousness may occur but not always.
- There may be a ‘lucid interval’ of a few hours after the injury where the patient appears relatively well and normal but subsequently deteriorates and loses consciousness as the haematoma forms.

Chronic SDH

- Usually presents about 2-3 weeks following the provoking trauma.
- The initial injury may be relatively trivial (or forgotten), particularly in an older patient on anticoagulants, or in the context of alcohol misuse.
- Symptoms tend to be gradually progressive.
- There is often a history of anorexia, nausea and/or vomiting.
There may be a gradually evolving neurological deficit such as focal limb weakness, speech difficulties, increasing drowsiness/confusion or personality changes.

If there is accompanying and progressive headache, this should raise suspicion of the diagnosis.

This is especially so in the context of coagulopathy, anticoagulant use or suspected alcohol misuse.

**Examination**

- Assess consciousness level using the [Glasgow Coma Scale](https://en.wikipedia.org/wiki/Glasgow_Coma_Scale).
- Check vital signs, looking for evidence of bradycardia and hypertension associated with raised intracranial pressure.
- Perform a full neurological examination, including examination for pupil size and reactivity and papilloedema (which can indicate raised intracranial pressure).
- Look for evidence of external trauma to the head or elsewhere.
- It is important to survey for other injuries in children with suspected SDH, as there may be evidence of non-accidental injury.
- In babies, the fontanelles may be tense due to raised intracranial pressure.
- Look for evidence of bruising or purpura, indicating a bleeding diathesis or meningitis.
- There is a wide variety of possible neurological signs depending on the site and severity of the accumulated haemorrhage and the rapidity with which it has developed.

**Differential diagnosis**

**NB**: remember the possibility of **non-accidental injury** in children or the elderly.

- Epidural haematoma.
- Subarachnoid haemorrhage.
- Intracerebral haemorrhage or infarction.
- Meningitis or encephalitis.
- Cerebral tumour (especially if associated with acute haemorrhage).
- Evolving stroke.
- Metabolic derangement causing confusion and impaired consciousness (encephalopathy) - eg, diabetic ketoacidosis, sepsis, hepatic encephalopathy due to alcohol abuse, chronic kidney disease.
- Decompensation of dementia.
- Any other cause of confusion in an older patient.
- Any other growing space-occupying lesion - eg, cerebral toxoplasmosis in an immunocompromised patient, cerebral tumour.

**Investigations**

**Blood tests**

- FBC, U&Es and LFTs may reveal alternative causes of impaired consciousness.
- Thrombocytopenia may indicate a bleeding diathesis.
- Coagulation screen should be checked to screen for coagulopathy.
- Take blood for group and save/cross-match if SDH seems likely, in anticipation of operative intervention.

**Imaging**

- In patients with impaired consciousness, confusion, focal neurology or signs of possible raised intracranial pressure, that cannot be otherwise explained, urgent neuroimaging is mandatory.
- CT scan of the head is good for detecting acute SDH and mandatory in children with significant head injury.[13]
- Subacute SDH may be more difficult to detect, so CT with contrast or MRI is preferred.[4]
- Chronic SDH is usually detectable on non-contrast CT and is a quicker examination.[4]
- In cases of severe trauma, it is wise to image the cervical spine in case of fracture and consider a radiological survey for secondary injuries.

**Management**

- In cases of severe trauma, immobilise the cervical spine and alert the trauma team.
- Assess and manage 'Airway, Breathing and Circulation'.
- Intubation and assisted ventilation may be needed depending on the level of consciousness. Obtain senior A&E, anaesthetic or neurosurgical advice.
- Priority should also be given to obtaining imaging of the head.
- Stabilise the patient before transfer for any imaging and send an appropriately experienced member of staff to accompany them during investigations, in case of deterioration.
- If the condition is strongly suspected or confirmed by investigation, refer urgently to the neurosurgical team.
- Hypertonic saline or mannitol may be considered if there is raised intracranial pressure.
- Burr holes may be considered if there is rapid deterioration.
- Any coagulopathy also needs treating.
- If transfer to another site for surgery is necessary, ensure that the patient's condition is optimised and stable before transfer and send an appropriately experienced member of staff, who has the ability to intubate and safely manage the patient in transfer, in case of deterioration.
- If there is a small, asymptomatic, acute SDH, this can be managed by observation, serial examinations, and serial CT scanning.[14]
- Surgery is needed if there are focal signs, deterioration, a large haematoma, raised intracranial pressure or midline shift.[15]
- SDH is treated by emergency craniotomy and clot evacuation.
- Recurrence is found in 5-30% of patients, which can be reduced with the use of a drain.[16]

### Complications

- Death due to cerebellar herniation.
- Raised intracranial pressure.
- Cerebral oedema.
- Recurrent haematoma formation during recovery.
- Seizures.
- Wound infection, subdural empyema, meningitis.
- Permanent neurological or cognitive deficit due to pressure effects on the brain.
- Coma/persistent vegetative state.

### Prognosis

- In cases of complicated acute SDH, where there has been concurrent parenchymal brain injury, the mortality rate can be up to 50%. SDH is a significant predictor of outcome at six months. In uncomplicated acute SDH, the mortality rate is around 20%. Chronic SDH that is treated surgically has a mortality rate of around 5%. In infants, the mortality rate in the UK/Republic of Ireland was found to be ~19% in an epidemiological study.

### Prevention

- Avoidance of over-anticoagulation in patients taking warfarin.
- Avoidance of falls in older people, especially if on anticoagulants.
- Treatment for alcoholism.

### Further Reading & References

9. Meagher RJ et al; Subdural Hematoma, Medscape, Mar 2013

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