Head injury

Extradural haematoma

An extradural haematoma (EDH) is a neurosurgical emergency.

An EDH is nearly always associated with a skull fracture and is more common in young male patients. The skull fracture is associated with tearing of a meningeal artery and a haematoma accumulates in the space between bone and dura. The most common site is temporal, as the pterion is not only the thinnest part of the skull but also overlies the largest meningeal artery – the middle meningeal. An EDH may also occur in other regions such as frontal as well as in the posterior fossa. They are not always arterial: disruption of a major dural venous sinus can result in an EDH.

The classical presentation of an EDH, occurring in less than one-third of cases, is initial injury followed by a lucid interval when the patient complains of a headache but is fully alert and orientated with no focal deficit. After minutes or hours a rapid deterioration occurs, with contralateral hemiparesis, reduced conscious level and ipsilateral pupillary dilatation as a result of brain compression and herniation. Early recognition and treatment of the condition is likely to result in full recovery whereas delay in diagnosis and treatment can result in death from secondary brain injury. Of course, a patient with an EDH may also sustain a primary brain injury and have a reduced conscious level from the time of injury.

The features of an EDH on a CT scan are a lentiform (lens-shaped or biconvex) hyperdense lesion between the skull and brain. There may be an associated mass effect on the underlying brain, with or without a midline shift.

The treatment of an EDH is immediate surgical evacuation via a craniotomy. The overall mortality for all cases of EDH is about 18% but for isolated EDH it is about 2%.

Acute subdural haematoma

Acute subdural haematoma (ASDH) differs from EDH in terms of pathophysiology, presentation and prognosis.

An ASDH accumulates in the space between the dura and the arachnoid. To produce a subdural haematoma there has usually been some disruption of a cortical vessel or brain laceration, and ASDH is nearly always associated with a significant primary brain injury. Patients with ASDH usually present with an impaired conscious level from the time of injury, but further deterioration can occur as the haematoma expands.

The CT appearance of an ASDH is also hyperdense (acute blood) but the haematoma spreads across the surface of the brain giving it a rather diffuse and concave appearance. This occurs because there is less resistance to blood moving through the subdural space than through the extradural space.

The treatment of an ASDH is usually evacuation via a craniotomy. Small haematomas with little mass effect may be managed conservatively in neurosurgical centres.The mortality rate from ASDH is much higher than for EDH and is as high as 40% in some series.

Chronic subdural haematoma

Chronic subdural haematomas (CSDH) usually occur in the elderly and are more common in those on anti-coagulant or antiplatelet agents. There is usually but not always a history of minor head injury in the weeks or months prior to presentation. It is thought that small bridging veins tear and cause a small ASDH which is clinically silent. As the haematoma breaks down it increases in volume, leading to a mass effect on the underlying brain.

Clinical features of CSDH include headache, cognitive decline, focal neurological deficits and seizures. It is important to exclude hypoxic, metabolic and endocrine disorders in this group of patients.

Subarachnoid haemorrhage

Although aneurysms are the most common cause of spontaneous subarachnoid haemorrhage, trauma is by far the commonest cause of subarachnoid haemorrhage overall. In rare cases, a spontaneous aneurysmal haemorrhage immediately precedes a head injury. Traumatic subarachnoid haemorrhage is managed conservatively **(The blood is distributed in the fissures in gyrii)**.

Cerebral contusions

Cerebral contusions are common in head injury and result from the brain being damaged by impacting against the skull either at the point of impact (the ‘coup’) or on the other side of the head (‘contre-coup’) or as the brain slides forwards and backwards over the ridged cranial fossa floor (most often affecting the inferior frontal lobes and temporal poles). Cerebral contusions on CT appear heterogeneous with mixed areas of high and low density. There may be an associated mass effect. A contusion may be described as an intracerebral haematoma if the lesion contains a large amount of fresh haemorrhage and therefore appears uniformly hyperdense.

Cerebral contusions rarely require immediate surgical treatment. A head-injured patient with cerebral contusions must be admitted for observation as these lesions will tend to

mature and expand for 48–72 hours following injury. A small proportion of cerebral contusions will require delayed surgical evacuation to reduce the mass effect.

Intracranial pressure monitoring

ICP monitoring is a useful adjunct in the management of unconscious patients with head injury. A sustained ICP of > 20 mmHg is associated with a worse outcome. The ICP can also be used to calculate the cerebral perfusion pressure. An ICP monitor may be parenchymal or ventricular.

Decompressive craniectomy

Controversy still surrounds the use of decompressive craniectomy. The purpose of a decompressive craniectomy is to control the ICP in patients without a focal intracerebral haematoma in whom the ICP is refractory to maximal medical therapy. The operation involves removing a large section of skull and opening the dura, allowing the swollen brain to expand underneath the scalp. The bone flap is stored and can be replaced 3–6 months later when the patient has made a good neurological recovery and the brain swelling has resolved.

Medical management of severe head injury

Environment

Severe head injury is best managed in a neuro-intensive care setting. The patient should be positioned with the head up 30 degree if spinal clearance allows. It is important to ensure that the cervical immobilisation collar does not obstruct venous return from the head.

Surgical management of raised intracranial pressure

■ Early evacuation of focal haematomas: EDH, ASDH

■ Cerebrospinal fluid drainage via ventriculostomy

■ Delayed evacuation of swollen contusions

■ Decompressive craniectomy

Airway and ventilation

A definitive airway is required in cases of severe head injury for several reasons: the patient in traumatic coma is unable to protect their airway and is at risk of aspiration; gas exchange may be impaired. Normal gas exchange in the head-injured patient must be confirmed and maintained. Hypoxia and hypercapnia will result in increased brain

ischaemia and secondary brain injury. A single episode of hypoxia with a PO2 <8 kPa is associated with a worse outcome in traumatic coma.

In the intubated patient, normocapnia is maintained: PCO2 4.5–5.0 kPa. A rise in PCO2 in a head-injured patient with an abnormally high ICP will result in generalised cerebral vasodilatation; an increase in cerebral blood volume, further raised ICP and reduced cerebral perfusion. Conversely, a fall in PCO2 will result in generalized vasoconstriction, a fall in cerebral blood volume and a drop in ICP. Although this global effect on ICP may seem beneficial it occurs at the expense of perfusion of ischaemic areas of the brain and therefore will result in a higher likelihood of cerebral infarction than recovery from ischaemia. It is for this reason that hyperventilation to induce hypocapnia is used only by anaesthetists experienced in the care of head-injured patients as an emergency measure, to temporarily reduce ICP while definitive care or urgent investigation is being organised.

Circulation and cerebral perfusion pressure

Hypotension sits alongside hypoxia as a major cause of secondary brain injury. A single episode of hypotension with a systolic blood pressure of < 90 mmHg is associated with a worse outcome in traumatic coma. Cerebral perfusion pressure should be maintained at > 65 mmHg in severely head-injured patients. This is an independent prognostic factor in neurological outcome. Cerebral perfusion can be estimated by the equation:

Cerebral perfusion pressure = mean arterial pressure − intracranial pressure

CPP = MAP − ICP

If the ICP is 20 mmHg, it follows that the MAP should be ≥ 85 mmHg.

MAP = [(2 x diastolic) + systolic] divided by 3

A MAP of at least 60 is necessary to perfuse the coronary arteries, brain, and kidneys. Normal range is around 70 – 110 mmHg.

Control of intracranial pressure

Normal ICP is 8–12 mmHg. A sustained ICP of > 20 mmHg is associated with poor outcome. ***As well as the environmental, respiratory and circulation measures outlined above, measures to reduce ICP include:***

• ***Sedation*****,** with or without muscle relaxants.

• ***Use of diuretics***. Judicious use of diuretics such as furosemide and mannitol will temporarily reduce cerebral swelling and ICP.

• ***Thermoregulation***. Pyrexia will increase the brain metabolic rate and should be avoided. Active cooling to reduce the metabolic rate is of questionable overall benefit but is used in some centres.

• ***Maintaining fluid and electrolyte balance.*** Severely brain-injured patients are susceptible to disturbances of sodium homeostasis such as diabetes insipidus and syndrome of inappropriate antidiuretic hormone (SIADH).

• ***Seizure control*.** Seizures increase the brain metabolic rate and should be controlled. Prophylactic anticonvulsants may reduce seizures in the first week. Steroids in severe head injury are associated with increased mortality and should not be used.

Medical management of raised intracranial pressure

■ Position head up 30º

■ Avoid obstruction of venous drainage from head

■ Sedation +/– muscle relaxant

■ Normocapnia 4.5–5.0 kPa

■ Diuretics: furosemide, mannitol

■ Seizure control

■ Normothermia

■ Sodium balance

SKULL FRACTURES

Skull vault fractures

Indications for surgery for skull fracture include:

1) Elevation of significantly depressed fragments
2) Wound debridement for compound fractures, particularly if there is evidence of underlying dural injury such as contusion or CSF leak.

Base of skull fractures

Base of skull fractures may be associated with seventh or eighth nerve palsies. CSF otorrhoea or rhinorrhoea often resolves spontaneously. Antibiotics are not required

prophylactically unless for concomitant facial fractures. A delayed craniotomy and anterior fossa dural repair is occasionally required for persistent CSF leak to prevent meningitis.

LONG-TERM SEQUELAE OF HEAD INJURY

Neuro-rehabilitation

Long-term management of the brain-injured patient requires the concerted efforts of medical, nursing, physiotherapy and speech and occupational therapy teams.

Neuropsychology

Neuropsychological sequalae are common after head injury and sometimes occur after relatively minor head injury. Post-concussional symptoms include headache, dizziness, impaired short-term memory and concentration, easy fatigability, emotional disinhibition and depression.

Seizures

Long-term epilepsy affects < 5% of patients admitted with head injury. The use of prophylactic anticonvulsants does not seem to change the long-term incidence of epilepsy.

Delayed CSF leak

Patients with delayed or on-going CSF leak should be investigated with CT cisternography or CSF isotope studies prior to surgical repair.

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