

Surgical pathology

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Fractures: skull vault Closed linear fractures of the skull vault are managed conservatively. Open or comminuted fractures should be considered for debridement and prophylactic antibiotic therapy. Depressed skull fractures involve inward displacement of a bone fragment by at least the thickness of the skull (Figures 28.4 and 28.5). They occur when small objects hit the skull at high velocity. They are usually compound (open) fractures, and are associated with a high incidence of infection, neurological deficit and late-onset epilepsy. These fractures require exploration and elevation, especially where intracranial air is present, pointing to the dura mater. Fractures that involve the air to a breach of the sinuses should generally be managed as open fractures, using broad-spectrum antibiotics with or without exploration.

Fractures: skull base Clinical signs of skull base fracture include bleeding or CSF leak from the ears (otorrhoea) or nose (rhinorrhoea) and bruising behind the ear (Figure 28.3) or around the eyes. Skull base fractures may be complicated by pituitary dysfunction, arterial dissection or cranial nerve deficits, with anosmia, facial palsy or hearing loss typical. CSF leak will generally resolve spontaneously but persistent leak can result in meningitis so repair may be required. Blind nasogastric tube placement is contraindicated in these patients.

Extradural haematoma Extradural haematoma (Figure 28.6) is a neurosurgical emergency. It results from rupture of an artery, vein or venous sinus, in association with a skull fracture. The classical injury is a fracture to the thin squamous temporal bone, with associated damage to the middle meningeal artery. Transient loss of consciousness is typical, and the patient may then present in the subsequent lucid interval with headache but without any neurological deficit. As the haematoma expands, compensation is exhausted (see The Monro-Kellie doctrine and herniation syndromes) with rapid deterioration. There is hemiparesis, a reduced conscious level and ipsilateral pupillary dilatation, the cardinal signs of brain compression and herniation. Although this 'talk and die' pattern of deterioration occurs in only one-third of cases, it is critically important to recognise the potential for rapid avoidable secondary brain injury in patients who present neurologically intact.

On CT, extradural haematomas appear as a lentiform (lens-shaped or biconvex) hyperdense lesion between the skull and the brain, constrained by the adherence of the dura to the inner table of the skull. A mass effect may be evident, with compression of the sulci of mixed density surrounding brain and midline shift. Air suggests active bleeding. A skull fracture will usually be evident.

Significant extradural haematoma mandates urgent transfer to the most accessible neurosurgical facility for immediate evacuation in deteriorating or comatose patients or those with large bleeds and for close observation with serial imaging in

Figure 28.4 A right frontal comminuted depressed skull fracture, with a linear undisplaced fracture of the right parietal bone visible posteriorly. (b) (c) Figure 28.5 (a-c) A small depressed skull fracture of the parietal bone visible on an axial bone window (a), visualised on bone vault reconstructions (b) with an underlying breach of the dura (c).

haematoma, without associated primary brain injury, is excellent. Extradural haemorrhage /uni25CF /uni25CF /uni25CF /uni25CF Acute subdural haematoma Acute subdural haematoma (Figure 28.7) is encountered in two broadly distinct contexts. First, high-energy injury mechanisms can result in the rupture of cortical surface vessels with significant associated primary brain injury. This results in an expanding haematoma with rapid deterioration and

(a) (b) (c) Figure 28.6 (a) A large left extradural haematoma (note the biconvex shape) exerts a mass effect; a smaller right acute subdural haematoma is also evident. (b) Right frontal intracerebral haematoma extending into the lateral ventricle is evident. There is a small right posterior extradural haematoma and traumatic subarachnoid bleeding in the sulci of the right hemisphere. (c) A surgical temporal bone exposure showing a linear skull fracture with underlying extradural haematoma visible through a burr hole. Can follow relatively minor trauma with brief loss of consciousness Followed by a lucid interval and then sudden deterioration Lentiform lesion on CT Require immediate transfer to a neurosurgical unit for decision on evacuation (a) (b) Figure 28.7 (a) Right-sided acute subdural haematoma (hyperdense). The substantial midline shift re /f_ l_ects brain swelling as well as bleeding – this is a high-energy injury. (b) Bilateral subdural haematomas: the left is mixed density, the hypodense material representing old blood and the higher density indicating more recent bleeding, probably loculated so requiring a craniotomy to evacuate. The bleed on the right is isodense, indicating intermediate age.

haematoma, without the lucid interval. These collections require prompt evacuation, typically by craniotomy or craniectomy. In a second group of patients, older and often anticoagulated, a lower energy injury leads to venous bleeding around the brain. Depending on the total volume of bleeding, the resulting haematoma may present early as acute subdural haematoma, after delay and osmotic expansion as chronic subdural haematoma or may even remain clinically silent. This last group may present much later with a further ‘acute-on chronic’ subdural haematoma. On diagnosis, clotting function should be corrected wherever possible. Bleeds of significant size, with significant associated midline shift or with deteriorating neurology, require urgent evacuation. Smaller bleeds in neurologically stable patients may be managed conservatively, at least initially: liquefaction of the clot over 7–10 days after the bleed may allow for a much less invasive evacuation through burr holes. Since the dura is not as adherent to the brain as it is to the skull, subdural blood is free to expand across the brain surface, giving a diffuse concave appearance. Summary box 28.7 Acute subdural haemorrhage /uni25CF /uni25CF Chronic subdural haematoma Chronic subdural haematoma (Figure 28.7) is a common cause of acute neurological deterioration in older adults. Cerebral atrophy in this age group results in stretching of the cortical-dural bridging veins, which are then vulnerable to rupture. The resulting haematoma can expand over days or weeks by osmosis, ultimately producing symptoms of raised ICP or focal deficits. There is usually a history of recent injury, but, especially in the context of antiplatelet or anticoagulant medication, even apparently trivial impacts may be responsible. On presentation it is important to exclude coexisting electrolyte disturbance and infections, which may contribute to clinical impairment. Imaging reveals diffuse hypodensity overlying the brain surface. Recent bleeding may be isodense or hyperdense, and mixed density can indicate an acute-on chronic subdural haematoma. Anticoagulation should be reversed, either by administration of vitamin K or urgently by transfusion of recombinant clotting factors in patients who have deteriorated acutely. Conservative management, sometimes with administration of corticosteroids, can be considered for small bleeds without symptoms or with headache alone. For the majority drainage is performed

using burr holes. Urgency is dictated by the clinical condition of the patient and imaging evidence of mass effect. If clinically stable, a delay of 7–10 days to allow platelet function to normalise after withdrawal of aspirin/clopidogrel may be considered. Chronic subdural haemorrhage - Traumatic subarachnoid haemorrhage Trauma is the commonest cause of subarachnoid haemorrhage (Figures 28.6b and 28.8), and this is managed conservatively . - It is not usually associated with significant vasospasm, which characterises aneurysmal subarachnoid haemorrhage. The possibility of spontaneous subarachnoid haemorrhage actually leading to collapse and so causing a head injury needs to be borne in mind, and formal or CT angiography may be required to exclude this. Cerebral contusions Contusions are common and are found predominantly where the brain is in contact with the irregularly ridged inside of the skull, i.e. at the inferior frontal lobes and temporal poles. ‘ Coup contre-coup ’ contusions refer to brain injury both at the site of impact and distant to this, where the brain impacts on the inside of the skull as the skull and brain accelerate and then decelerate out of synchrony with each other. Contusions appear heterogeneous on CT , reflecting their composition of injured brain matter interspersed with acute blood (Figure 28.8). Contusions rarely require surgical intervention but may warrant delayed evacuation to reduce a mass effect. - - -

High-energy injuries, or elderly/anticoagulated Generally require urgent evacuation by craniotomy/craniectomy Common in the elderly, especially those on anticoagulants Clinical deficits result from osmotic expansion of a degrading clot over days/weeks Diffuse hypodense lesion on CT Burr hole drainage is usually preferred Figure 28.8 A large right extradural haematoma is evident. There are widespread cerebral contusions most prominent in the left frontal lobe. There is traumatic subarachnoid blood in the third and lateral ventricles.

This is a form of primary brain injury seen in high-energy accidents that usually renders the patient comatose; it is associated with poor outcomes. It is strictly a pathological diagnosis made at postmortem, but haemorrhagic foci in the corpus callosum and dorsolateral rostral brainstem on CT may be suggestive. Magnetic resonance imaging is more sensitive and is employed to evaluate for diffuse axonal injury in patients who fail to improve neurologically . Arterial dissection Cerebral arterial dissection occurs spontaneously or in the context of trauma. In the hours after significant trauma, dissection of the carotid extracranially , or at the skull base in association with fractures, is most common. It presents with headache, neck pain and focal ischaemic deficits due to occlusion by mural haematoma, thrombus and thromboembolism. Intracranial dissection often affects the vertebral artery and may result in subarachnoid bleeding. Summary box 28.9 Specific injuries

Traumatic versus primary subarachnoid haemorrhage is an important distinction Cerebral contusions arise adjacent to rough bone surfaces Diffuse axonal injury results from extreme accelerations of the skull contents Arterial dissection is associated with fractures of the skull base

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Fractures: skull vault Closed linear fractures of the skull vault are managed conservatively . Open or comminuted fractures should be considered for debridement and prophylactic antibiotic therapy . Depressed skull fractures involve inward displacement of a bone fragment by at least the thickness of the skull (Figures 28.4 and 28.5 They occur when small objects hit the skull at high

velocity . They are usually compound (open) fractures, and are associated with a high incidence of infection, neurological deficit and late-onset epilepsy . These fractures require exploration and elevation, especially where intracranial air is present, pointing into the dura mater. Fractures that involve the air to a breach sinuses should generally be managed as open fractures, using broad-spectrum antibiotics with or without exploration. Fractures: skull base Clinical signs of skull base fracture include bleeding or CSF leak from the ears (otorrhoea) or nose (rhinorrhoea) and bruising behind the ear (Figure 28.3) or around the eyes. Skull base fractures may be complicated by pituitary dysfunction, arterial dissection or cranial nerve deficits, with anosmia, facial palsy or hearing loss typical. CSF leak will generally resolve spontaneously but persistent leak can result in meningitis so repair may be required. Blind nasogastric tube placement is contraindicated in these patients. Extradural haematoma Extradural haematoma (Figure 28.6) is a neurosurgical emergency . It results from rupture of an artery , vein or venous sinus, in association with a skull fracture. The classical injury is a fracture to the thin squamous temporal bone, with associated damage to the middle meningeal artery . Transient loss of consciousness is typical, and the patient may then present in the subsequent lucid interval with headache but without any neurological deficit. As the haematoma expands, compensation is exhausted (see The Monroe-Kellie doctrine and herniation is contralateral syndromes) with rapid deterioration. There hemiparesis, a reduced conscious level and ipsilateral pupillary) . - dilatation, the cardinal signs of brain compression and herniation. Although this ' talk and die ' pattern of deterioration occurs in only one-third of cases, it is critically important to recognise the potential for rapid avoidable secondary brain injury in patients who present neurologically intact. - On CT , extradural haematomas appear as a lentiform (lens-shaped or biconvex) hyperdense lesion between the skull and the brain, constrained by the adherence of the dura to the table skull. A mass effect may be evident, with compression of the edges of mixed density surrounding brain and midline shift. Air suggest active bleeding. A skull fracture will usually be evident. - Significant extradural haematoma mandates urgent transfer to the most accessible neurosurgical facility for immediate evacuation in deteriorating or comatose patients or those with large bleeds and for close observation with serial imaging in

Figure 28.4 A right frontal comminuted depressed skull fracture, with a linear undisplaced fracture of the right parietal bone visible posteriorly. (b) (c) Figure 28.5 (a-c) A small depressed skull fracture of the parietal bone visible on an axial bone window (a) , visualised on bone vault reconstructions (b) with an underlying breach of the dura (c) .

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on CT Require immediate transfer to a neurosurgical unit for decision on evacuation (a) (b) Figure 28.7 (a) Right-sided acute subdural haematoma (hyperdense). The substantial midline shift reflects brain swelling as well as bleeding – this is a high-energy injury. (b) Bilateral subdural haematomas: the left is mixed density, the hypodense material representing old blood and the higher density indicating more recent bleeding, probably loculated so requiring a craniotomy to evacuate. The bleed on the right is isodense, indicating intermediate age.

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Summary box 28.7 Acute subdural haemorrhage Chronic subdural haematoma Chronic subdural haematoma (Figure 28.7) is a common cause of acute neurological deterioration in older adults. Cerebral atrophy in this age group results in stretching of the cortical-dural bridging veins, which are then vulnerable to rupture. The resulting haematoma can expand over days or weeks by osmosis, ultimately producing symptoms of raised ICP or focal deficits. There is usually a history of recent injury, but, especially in the context of antiplatelet or anticoagulant medication, even apparently trivial impacts may be responsible. On presentation it is important to exclude coexisting electrolyte disturbance and infections, which may contribute to clinical impairment. Imaging reveals diffuse hypodensity overlying the brain surface. Recent bleeding may be isodense or hyperdense, and mixed density can indicate an acute-on chronic subdural haematoma. Anticoagulation should be reversed, either by administration of vitamin K or urgently by transfusion of recombinant clotting factors in patients who have deteriorated acutely. Conservative management, sometimes with administration of corticosteroids, can be considered for small bleeds without symptoms or with headache alone. For the majority drainage is performed using burr holes. Urgency is dictated by the clinical condition of the patient and imaging evidence of mass effect. If clinically stable, a delay of 7–10 days to allow platelet function to normalise after withdrawal of aspirin/clopidogrel may be considered.

Chronic subdural haemorrhage - Traumatic subarachnoid haemorrhage Trauma is the commonest cause of subarachnoid haemorrhage (Figures 28.6b and 28.8), and this is managed conservatively. - It is not usually associated with significant vasospasm, which characterises aneurysmal subarachnoid haemorrhage. The possibility of spontaneous subarachnoid haemorrhage actually leading to collapse and so causing a head injury needs to be borne in mind, and formal or CT angiography may be required to exclude this.

Cerebral contusions Contusions are common and are found predominantly where the brain is in contact with the irregularly ridged inside of the skull, i.e. at the inferior frontal lobes and temporal poles. ‘Coup contre-coup’ contusions refer to brain injury both at the site of impact and distant to this, where the brain impacts on the inside of the skull as the skull and brain accelerate and then decelerate out of synchrony with each other. Contusions

appear heterogeneous on CT, reflecting their composition of injured brain matter interspersed with acute blood (Figure 28.8). Contusions rarely require surgical intervention but may warrant delayed evacuation to reduce a mass effect. - - -

High-energy injuries, or elderly/anticoagulated Generally require urgent evacuation by craniotomy/craniectomy Common in the elderly, especially those on anticoagulants Clinical deficits result from osmotic expansion of a degrading clot over days/weeks Diffuse hypodense lesion on CT Burr hole drainage is usually preferred Figure 28.8 A large right extradural haematoma is evident. There are widespread cerebral contusions most prominent in the left frontal lobe. There is traumatic subarachnoid blood in the third and lateral ventricles.

This is a form of primary brain injury seen in high-energy accidents that usually renders the patient comatose; it is associated with poor outcomes. It is strictly a pathological diagnosis made at postmortem, but haemorrhagic foci in the corpus callosum and dorsolateral rostral brainstem on CT may be suggestive. Magnetic resonance imaging is more sensitive and is employed to evaluate for diffuse axonal injury in patients who fail to improve neurologically. Arterial dissection Cerebral arterial dissection occurs spontaneously or in the context of trauma. In the hours after significant trauma, dissection of the carotid extracranially, or at the skull base in association with fractures, is most common. It presents with headache, neck pain and focal ischaemic deficits due to occlusion by mural haematoma, thrombus and thromboembolism. Intracranial dissection often affects the vertebral artery and may result in subarachnoid bleeding. Summary box 28.9 Specific injuries

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Summary box 28.7 Acute subdural haemorrhage /uni25CF /uni25CF **Chronic subdural haematoma**

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Chronic subdural haemorrhage /uni25CF - /uni25CF /uni25CF /uni25CF - **Traumatic subarachnoid haemorrhage**

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