Emergency presentation of neurosurgical conditions

Many neurosurgical conditions present in an acute fashion, and optimal outcome often depends on prompt recognition and referral by general practitioners and emergency room personnel. The approach to neurosurgical emergencies is discussed in this article.

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Approach to a neurosurgical emergency begins with an appropriate history of surrounding events followed by general examination and focused neurological examination.[1] This should be performed while applying general supportive care. After outlining a general approach to the patient presenting with a neurosurgical emergency, the more common clinical scenarios will be discussed.

History

A detailed history of presenting events and general medical history should be obtained from the patient if possible, family members or emergency service personnel and should include the following:

Presenting events:

- rapidity of symptom onset or neurological deterioration
- seizures
- nausea or vomiting
- recent or previous trauma
- recent febrile illness.

Previous medical history:

- hypertension and compliance with treatment
- diabetes mellitus
- epilepsy
- known history of previous/underlying malignancy/pituitary tumour
- HIV and antiretroviral treatment
- history of anticoagulant use.

Surgical history:

- previous neurosurgery, e.g. ventriculo-peritoneal shunt.[1]

Examination

Clinical examination focuses on the principles of ABCD, and supportive management is instituted at the same time as problems are identified.

Elevated blood pressure may be a reactive phenomenon or reflect the underlying cause.

Vital signs

Airway and breathing

Ensure adequate airway and oxygen saturation above 95%. Cheyne Stokes respiration is the most common abnormal breathing pattern, characterised by periods of hyperventilation alternating with periods of apnoea. Consider intubation if Glasgow coma scale (GCS) <8.

Circulation

Elevated blood pressure may be a reactive phenomenon or reflect the underlying cause. With increasing intracranial pressure an acute rise in blood pressure may be accompanied by bradycardia and decreased respirations (Cushing's reflex) and is a sign of impending tonsillar herniation.

Temperature

Fever may indicate underlying CNS infection.

General examination

Evidence of trauma – scalp bruising or laceration, periorbital (raccoon eyes) or retro-auricular bruising (Battle sign) and CSF leak from the nose or ear.

Neurological examination

Level of consciousness

Level of consciousness is assessed using the GCS (Table 1).[2] The GCS is made up of the sum of the best eye opening, best verbal and best motor responses, with a total score ranging from 3 to 15 (normal).

Fundoscopy and pupillary response

Fundoscopic examination for papilloedema is an essential clinical skill. Pupils are checked for both size and response to

Table 1. Glasgow Coma Scale

Best motor response	6 Obeys commands 5 Localises painful stimulus 4 Abnormal flexion 3 Decorticate posturing 2 Decerebrate posturing 1 No response
Best verbal response	5 Orientated 4 Confused speech 3 Words 2 Incomprehensible sounds 1 No response
Best eye opening response	4 Opens eyes spontaneously 3 Opens eyes to speech 2 Opens eyes to painful stimulus 1 No response

light. A unilateral dilated pupil suggests the presence of a structural lesion with uncal herniation. Pontine lesions result in pinpoint pupils, while midbrain lesions are associated with midposition fixed pupils.

Eye movements

Evaluate spontaneous eye movements and the ocular reflexes – oculocephalic (doll's eye) and oculovestibular. Hemispheric lesions involving the frontal eye fields result in deviation of the eyes away from the side of the associated hemiplegia. Brainstem lesions involving the reticular formation in the pons result in conjugate deviation of the eyes toward the side of the associated hemiplegia. Nystagmus may indicate cerebellar lesions.[1]

Cranial nerve examination

Third and sixth nerve palsies are common indicators of raised intracranial pressure. Other cranial nerve deficits may also be present depending on the location of pathology.[1]

Meningism

Remember that neck stiffness has a number of causes and does not always mean meningitis – performing a lumbar puncture in a patient with tonsillar herniation will have disastrous consequences (Table 2).

Table 2. Causes of neck stiffness

Meningitis
Subarachnoid haemorrhage
Tonsillar herniation (raised intracranial pressure)
Cervical spine trauma

Focal motor deficits

Focal motor deficits include hemiparesis/plegia and monoparesis and should be documented during assessment of the motor component of the Glasgow coma score.

Common presentations

- Acute loss of consciousness**.** Loss of consciousness is a presentation common to a variety of medical conditions and is best assessed using the GCS, as described above. The causes of coma are divided into 2 major groups and common causes of each are shown in Table 3. Metabolic causes of acute loss of consciousness are more likely to produce symmetrical neurological deficits, compared with focal deficits in structural causes.[3]
- Sudden-onset severe headache.
- Subarachnoid haemorrhage (SAH) occurs when blood enters the subarachnoid space, in the majority of cases due to rupture of an intracranial saccular aneurysm. It occurs most commonly in the 5th - 6th decades.[4]

History

Sudden-onset severe headache, described as 'the worst headache of my life', occurs in 80% of patients. Headaches are commonly retro-orbital, radiate to the neck and often occur during periods of intense activity or straining. Vomiting is also common.[4] Twenty per cent of patients have a history of preceding symptoms, including headache, nausea, vomiting and dizziness, thought to arise from small haemorrhages from the aneurysm or acute expansion of the aneurysm sac. Within seconds or minutes following headache, patients may lose consciousness, develop seizures or die.

Table 3. Causes of acute loss of consciousness

Toxic/metabolic	Structural
Electrolyte imbalance	Vascular
Hypo/hypernatraemia	Bilateral cortical/subcortical infarcts
Endocrine	Bilateral carotid artery stenosis
Hypoglycaemia	Bilateral di-encephalic infarcts
Diabetic ketoacidosis	Infectious
Addison crisis	Abscess
Toxins	Empyema
Ethanol	Trauma
Drug overdose	Neoplastic
Organ failure	Primary
Uraemia	Secondary
Hypoxaemia	Increased intracranial pressure
Liver failure	Herniation from mass effect
Epilepsy	Acute lateral shift of the brain

All patients presenting with unusually sudden or severe headaches should be investigated for SAH.[5]

Examination

Findings include decreased level of consciousness, neck stiffness and focal neurological deficits. Focal deficits may be due to mass effect from the aneurysm (e.g. third nerve palsy in posterior communicating artery aneurysms) or intracerebral haematomas.

Investigation

Imaging

Uncontrasted CT scan of the brain is the investigation of choice in suspected SAH and may reveal blood in the cisterns at the base of the brain, intraventricular or intraparenchymal blood (Fig. 1). Hydrocephalus may also be present. If uncontrasted CT of the brain is negative, a lumbar puncture should be performed to establish the presence of xanthochromia, unless a contraindication to lumbar puncture is present.[6]

Laboratory

Full blood count (FBC), clotting profile, urea, electrolytes and creatinine (UEC).

Initial management

- Monitor arterial oxygen saturation and blood pressure
- Supplemental oxygen
- Intravenous access and fluid administration, correct hyponatraemia
- Stool softeners to prevent straining
- Anticonvulsants, especially if seizures occurred during the initial event.[6]

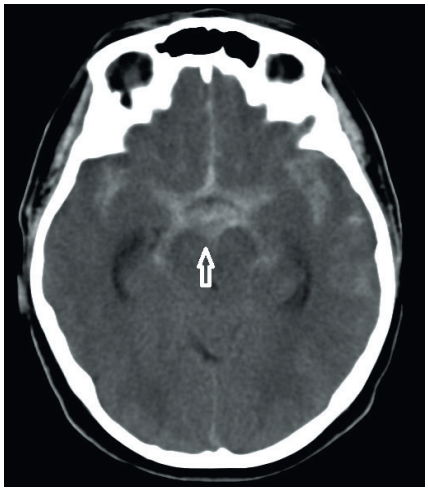

Fig. 1. Uncontrasted CT brain revealing extensive subarachnoid blood in the basal cisterns (arrow).

Pituitary apoplexy

Pituitary apoplexy results from haemor-rhage or infarction of a pituitary adenoma, resulting in acute tumour expansion with compression of surrounding structures in the sellar region, notably the optic nerves and hypothalamus.[7]

History

The patient may be known to have a pituitary tumour, but in the majority pituitary apoplexy is the first presentation. Symptoms include acute-onset headache, acute deterioration in visual acuity or visual fields. Vomiting often accompanies pituitary apoplexy. There may be a preceding history of symptoms suggestive of hypopituitarism (tiredness, weight gain, decreased libido, menstrual irregularities). Features of hormone excess may also be present, e.g. Cushing's disease, acromegaly or hyperthyroidism.[8]

Examination

Hypotension and hypothermia may indicate acute adrenal insufficiency secondary to low cortisol. Common findings are ophthalmoplegia, decreased visual acuity and visual field deficits – most commonly bitemporal hemianopia.[8]

Investigation

Imaging

A CT scan of the brain with and without contrast may reveal a sellar or suprasellar mass with evidence of haemorrhage, with or without subarachnoid haemorrhage. MRI is better for diagnosis and should be done in all cases of suspected pituitary apoplexy following an initial CT scan.[8]

Laboratory

FBC, UEC. Baseline endocrine bloods (T4, TSH, FSH, LH, prolactin, GH and cortisol).[7]

Initial management

- ABC
- IV access and immediate administration of hydrocortisone 100 mg
- Urinary catheter and monitoring of urine output to monitor for diabetes insipidus.[8]

Meningism with fever

Intracranial infections

Intracranial infections include brain abscess and subdural empyema. These conditions usually occur in the first 4 decades of life and result from spread from contiguous structures (oral cavity, sinuses, middle ear), either directly or by retrograde venous thrombosis, or haematogenous spread in patients with valvular heart disease (infective endocarditis), cyanotic congenital heart disease and chronic suppurative lung disease (bronchiectasis).[9]

History

Patients may have a history of recent febrile illness, including dental infections, otitis media, mastoiditis or sinusitis. Symptoms are usually present for 1 - 2 weeks before acute deterioration. Common symptoms are headache (often progressive and severe, localised to the side of the abscess), mental status changes, high- or low-grade fever, seizures, nausea and vomiting.[10]

Examination

Findings on physical examination include fever, neck stiffness and signs of raised intracranial pressure due to mass effect from the lesion or associated cerebral oedema. Note that some patients with brain abscess do not have a fever. Focal deficits are common and determined by the size and location of the pus collection. Cerebellar lesions result in cerebellar signs, while supratentorial lesions lead to hemiparesis, mental status changes and speech difficulties. A thorough ENT, oral, cardiovascular and chest examination should be performed to identify a possible source of infection.[9]

Investigation

Imaging

CT brain, with and without contrast, reveals a ring-enhancing lesion (location often depends on the underlying cause) with surrounding oedema in the case of an abscess

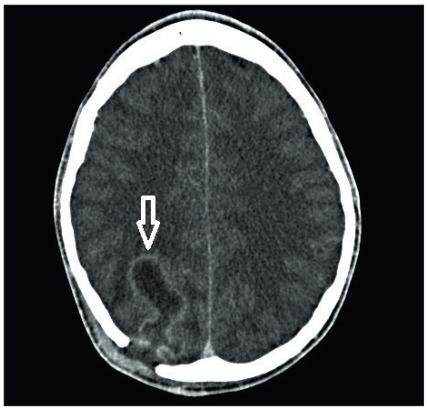

Fig. 2. Contrasted CT brain revealing ring enhancing cerebral abscess in the right parieto-occipital area (arrow).

(Fig. 2) or enhancing subdural collection in the presence of subdural empyema. Opacification or air-fluid levels in the sinuses may indicate sinusitis. Chest X-ray can reveal evidence of pneumonia or bronchiectasis.[10]

Laboratory

FBC, UEC, septic markers (CRP/ESR) and blood culture.[10] Lumbar puncture is contraindicated in the presence of decreased level of consciousness or focal neurological deficit.

Initial management

- ABCs
- Supplemental oxygen, intubation as required (GCS<8)
- IV access and broad-spectrum antibiotics
- Anti-seizure prophylaxis.[10]

Focal neurological deficits/ seizures

Acute-onset intracerebral haematoma (ICH)

History

Spontaneous ICH usually occurs in patients older than 55 years and is slightly more common in men. Pre-existing hypertension is commonly present and concurrent use of oral anticoagulants should be determined. In younger patients, structural lesions occur more frequently and a history of illicit drug use (e.g. cocaine) should be excluded. Common symptoms include headache, vomiting, acute-onset focal weakness and seizures.[11]

Examination

Elevated blood pressure may be an indication of the underlying cause in patients with chronic hypertension or a reactive phenomenon secondary to raised intracranial pressure. Excessive bruising or bleeding suggest anticoagulant toxicity. Focal neurological deficits are dependent on haematoma location and include hemiparesis, gaze palsy or other cranial nerve palsy.[11]

Investigation

Imaging

Uncontrasted CT scan of the brain shows hyperdense areas of acute haemorrhage. Hypertensive bleeds often occur in deep locations (basal ganglia, thalamus, cerebellum). Younger patients with more superficial haematomas or in atypical locations are more likely to have an

underlying vascular lesion, and further investigation is warranted, e.g. CT angiography.[11]

Laboratory
FBC, UEC, clotting profile.

Gradual progression

Progressive headache or neurological deficits are suggestive of an expanding mass lesion, most commonly intracranial neoplasms, which can be either primary or secondary.

History

A history of previous or current underlying cancer may be present, as well as systemic symptoms such as weight loss. Gradual progression of pre-existing weakness, progressive longstanding headache or seizures are suggestive of a progressively enlarging lesion or increasing cerebral oedema around the tumour. Acute deterioration may also occur due to haemorrhage into the tumour.

Examination

Altered level of consciousness, signs of raised intracranial pressure and focal neurological deficits are dependent on the location of the lesion. Systemic examination should also be performed to identify a possible underlying primary tumour.[12]

Investigation

Imaging

CT brain, with and without contrast, may reveal intrinsic or extrinsic mass lesions (Fig. 3) with surrounding oedema, with or without hydrocephalus. Further imaging is directed at identifying a possible underlying cause (CXR, ultrasound, etc.).[12]

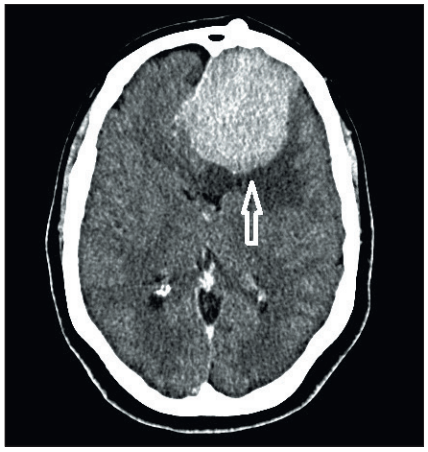

Fig. 3. Contrasted CT brain showing a homogenously enhancing extra-axial lesion in the interhemispheric fissure (parasaggital meningioma).

Initial management

- General supportive measures
- Correction of coagulopathies should be performed immediately.[11]
- Anti-seizure prophylaxis
- Steroids can be administered in the presence of oedema surrounding a suspected tumour.[12]

History of trauma

Extradural haematoma

Extradural haematomas are collections of blood outside the dura and are most commonly caused by skull fractures with an associated vascular tear, following a direct blow to the head. Clinically there may or may not be a period of loss of consciousness, often severe headache, vomiting and a history of seizures. About 20% of patients have the classic 'lucid interval' between the initial trauma and neurological deterioration.[13]

Subdural haematoma

Subdural haematomas are the most common intracranial haematomas following trauma and usually follow a high-speed impact to the skull, which causes brain tissue to decelerate relative to fixed dural structures, leading to tearing of blood vessels. Brain injury is often more severe and the patient is more likely to present with a decreased level of consciousness.[13]

Chronic subdural haematoma

In chronic subdural haematomas there is usually a history of minor head trauma some weeks prior to presentation. Patients complain of severe, progressive headache, seizures and decreased level of consciousness.[13]

History

- Mechanism of injury – blunt or penetrating assault, MVA
- Loss of consciousness or amnesia
- Seizures.

Examination

There is usually external evidence of trauma – lacerations, bruising, palpable fractures and CSF leak from the nose or ears suggestive of a base of skull fracture. Clinical findings include those of raised intracranial pressure, focal neurological deficits and signs of herniation, as discussed elsewhere in this issue.

Imaging

- Extradural haematoma – convex, hyperdense extra-axial lesion with or without midline shift (Fig. 4a). An overlying fracture is often identified.
- Subdural haematoma – crescent-shaped hyperdense lesion following the contour of the cerebral hemisphere (Fig. 4b). Parenchymal contusions are also common.

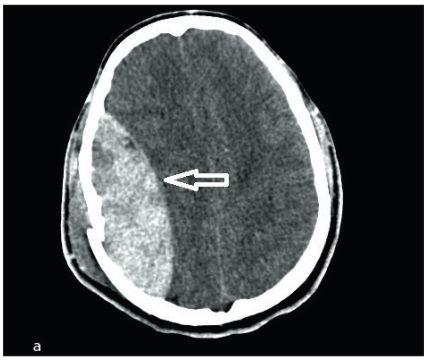

Fig. 4a. Acute extradural haematoma: biconvex hyperdense extra-axial lesion with mass effect on the underlying hemisphere.

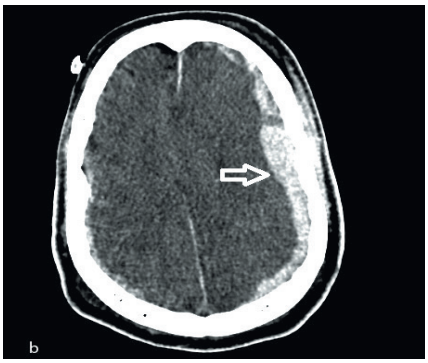

Fig. 4b. Subdural haematoma: hyperdense extra-axial lesion following the contour of the hemisphere.

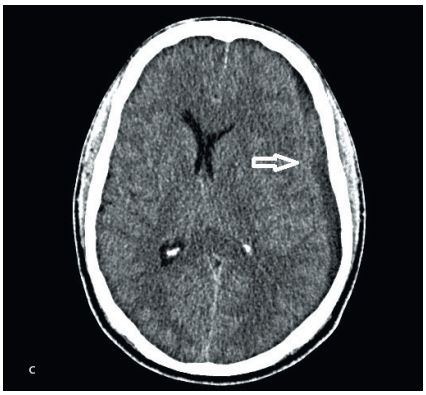

Fig. 4c. Chronic subdural haematoma. Hypodense extra-axial lesion with mass effect.

- Chronic subdural haematoma – iso- to hypodense collection over a hemisphere, often with a significant midline shift (Fig. 4c).[13]

Initial management

- Resuscitation according to ATLS principles, ensuring adequate blood pressure and oxygenation to prevent secondary injury
- Anti-seizure prophylaxis
- Urgent neurosurgical referral for surgical evacuation.

Previous neurosurgery

Ventriculoperitoneal shunt dysfunction

History

Progressive headache, vomiting or drowsiness are common complaints. Patients who have experienced shunt dysfunction before can often recognise a specific pattern when their shunt is not functioning, and very careful attention should be paid to this history. Endoscopic third ventriculostomy (ETV) has recently been accepted as a treatment for hydrocephalus. These patients do not have a shunt but the ETV can also block, leading to rapid clinical deterioration.

Various lesions may compress either the spinal cord or the roots of the cauda equina. This is one of the most commonly missed neurosurgical emergencies and an increasingly important cause of litigation.

Examination

Assess level of consciousness, presence of papilloedema and other signs of raised intracranial pressure. Document any signs of inflammation or swelling around the scalp or abdominal incisions suggestive of shunt infection or obstruction. The presence of neck stiffness is a sign of possible meningitis or shunt infection. Palpate the shunt tract for possible disconnection or fractures.[14]

Investigation

Imaging

CT reveals ventriculomegaly with or without periventricular lucency or generalised brain swelling. Shunt series X-ray studies (SXR, lateral neck, AP chest, AP abdomen) may reveal disconnection or fracture along the shunt tract.[14]

Laboratory

FBC, UEC and baseline septic markers for possible shunt infection.

Initial management

Provide initial supportive care and urgent neurosurgical consult.

Table 4. Causes of spinal cord compression

Degenerative	Cervical/thoracic stenosis Acute disc prolapse
Trauma	Fractures Fracture-dislocations
Infectious	Spinal epidural abscess/empyema
Vascular	Spinal epidural haematoma
Neoplastic	Extradural: vertebral tumours (primary or metastases) Intradural: extramedullary (e.g. meningioma or neurofibroma)

Spinal compression

Various lesions may compress either the spinal cord or the roots of the cauda equina. This is one of the most commonly missed neurosurgical emergencies and an increasingly important cause of litigation.

Spinal cord compression

The most common causes of spinal cord compression are listed in Table 4.[15]

Clinical presentation

- Paraplegia/paraparesis
- Quadriplegia/quadriparesis
- Urinary retention
- Impaired sensation below the level of compression
- Hyperactive reflexes
- Plantar reflexes may be upgoing
- Clinical findings may develop over hours or days.[15]

Investigation

- Plain film X-rays
- All patients need to be referred for urgent MRI to determine the level and cause of compression.[15]

Cauda equina compression (CES)

Aetiology

Possible causes include a massively herniated lumbar disc, tumour, trauma, spinal epidural haematoma and spinal epidural abscess,[16] resulting in dysfunction of multiple sacral nerve roots within the lumbar spinal canal.

Spontaneous ICH usually occurs in patients older than 55 and is slightly more common in men.

Clinical presentation

CES most commonly presents in an acute fashion. Clinical findings include lower back pain and sciatica (often bilateral), urine incontinence, decreased anal sphincter tone, saddle anaesthesia, motor weakness involving more than one motor nerve root and bilaterally decreased or absent Achilles tendon reflexes.[16]

Investigation

All patients require an urgent MRI scan and prompt referral to a neurosurgeon.

Conclusion

Neurosurgical conditions commonly present as emergencies. and a high index of suspicion should always be maintained when assessing these patients in order to ensure rapid diagnosis and prompt referral to a neurosurgeon.

References available at www.cmej.org.za

In a nutshell

- Neurosurgical conditions frequently present as emergencies.
- A good history, general examination and focused neurological examination provide vital diagnostic clues.
- Urgent imaging with CT or MRI is indicated in all neurosurgical emergencies.
- Basic supportive care should be provided to stabilise the patient.
- Urgent neurosurgical consult is required in all cases.