Acute neurology

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Patients with acute neurological symptoms frequently present to the emergency department. The commonest presentations include headache and seizures. Careful clinical evaluation and prompt investigation are required to exclude sinister causes and initiate correct treatment.

Headache

Most patients presenting urgently with headache will not have sinister underlying pathology but the primary aim of acute assessment is the rapid identification of those with such pathology (Table 1). A careful history is critical, in particular determination of the rate of onset and location of the headache as well as any associated features.

Intracranial haemorrhage

Subarachnoid haemorrhage (SAH) (Fig 1) must be excluded in any patient with abrupt ('thunderclap') onset headache even in the absence of any neurological signs. The headache is instantaneous and reaches its peak rapidly (seconds to minutes).[1] Meningism, nausea and vomiting are common but not invariable. Seizures and loss of consciousness may occur at onset. If an urgent radiologist reported computed tomographic (CT) brain scan of the brain is normal, a lumbar puncture (LP) must be performed more than eight hours after ictus to assess cerebrospinal

Table 1. Causes to consider in emergency presentation of headache.

Headache type	Causes
Primary headache syndrome	eg migraine, cluster migraine headache, tension headache
Secondary headache	Intracranial haemorrhage: subarachnoid, subdural, intracerebral Meningitis or encephalitis Increased intracranial pressure: idiopathic or secondary Cerebral venous sinus thrombosis Arterial dissection, arteritis, arterial occlusion Intracranial hypotension Extracranial process: cervicogenic headache, sinusitis, glaucoma Malignant hypertension Temporal arteritis (usually over age 55 years)

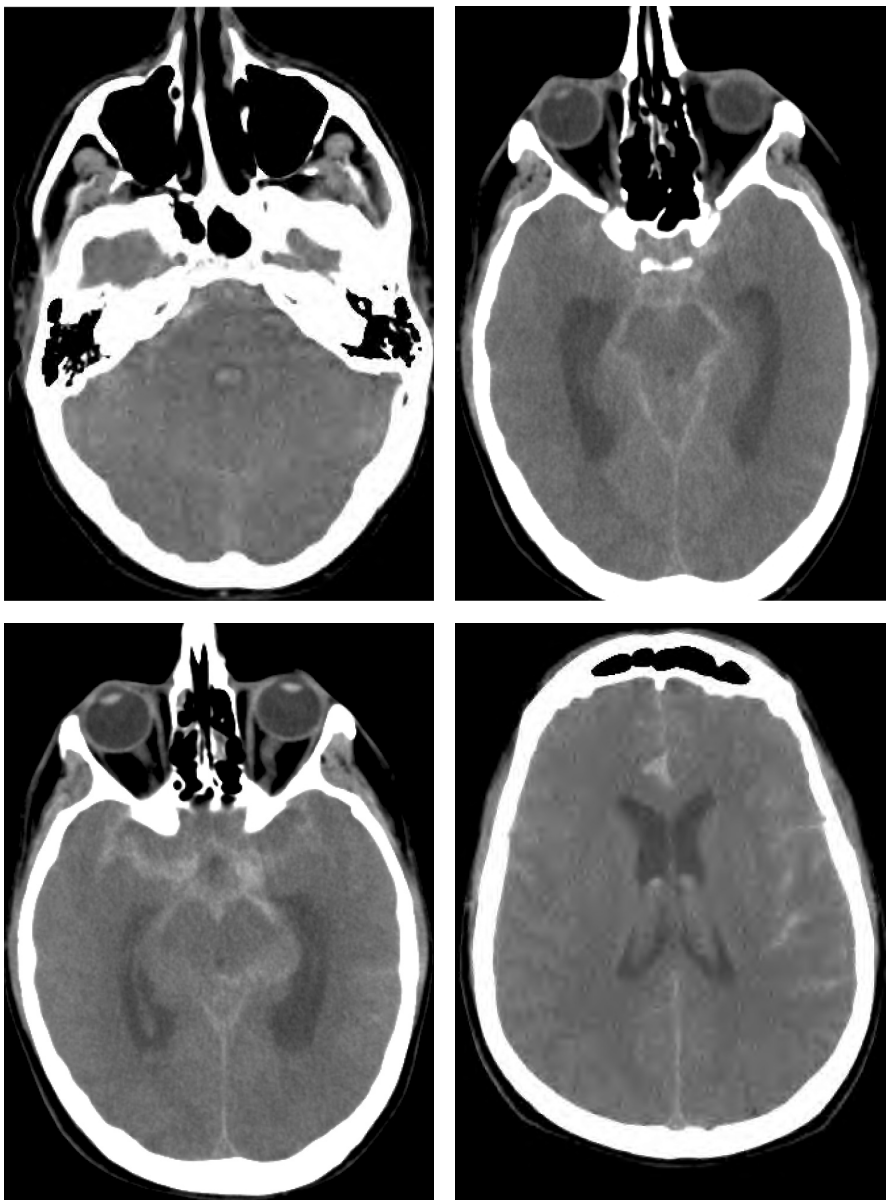

Fig 1. Non-contrast computed tomograph of the brain showing subarachnoid haemorrhage with hydrocephalus and blood extending into the lateral and fourth ventricles.

fluid (CSF) red cell count and xanthochromia. Arterial aneurysm is the commonest cause of SAH. Glasgow Coma Score (GCS) should be monitored since obstructive hydrocephalus and raised intracranial pressure (ICP) may occur. Urgent neurosurgical assessment is essential for definitive treatment to prevent rebleed which is associated with high mortality. Nimodipine may reduce cerebral vasospasm and should be administered when advised by the neurosurgical team.[2]

Intracerebral haemorrhage (Fig 2) usually causes acute onset focal neurological deficit 'stroke' and may be accompanied by headache, especially if accompanied by uncontrolled hypertension. Careful control of blood pressure and neurological monitoring for signs of raised pressure are important. Neurosurgical evacuation may be required in a small proportion of patients.

Subdural haemorrhage (SDH) (Fig 3) may be spontaneous or secondary to trauma (sometimes minor trauma in the elderly). SDH should always be thought of in an older patient with gradual onset headache, particularly if there is focal neurology. Once diagnosed, urgent neurosurgical advice should be sought regarding the need for evacuation versus monitoring.

Meningitis and encephalitis

Headache, fever, meningism and altered consciousness raise the suspicion of meningitis or encephalitis. The most common causes of community-acquired bacterial meningitis are *Streptococcus pneumoniae* and *Neisseria meningitidis*. Patients may become drowsy or haemodynamically unstable in a short period of time. If bacterial meningitis is suspected, blood cultures should be taken and antibiotics commenced immediately. Delayed antibiotic treatment may result in adverse outcome. Patients should undergo CT of the brain (or magnetic resonance imaging (MRI) if available) followed by LP. A recent review concluded that dexamethasone given from the time of administration of the first dose of antibiotics improves morbidity and mortality in adults with community-

Key Points

Patients with acute neurology commonly present in the emergency department; headache and seizures are among the most common presentations

Acute headache requires careful clinical evaluation. Rapid imaging may be required to exclude subarachnoid haemorrhage (SAH) or structural pathology. Further assessment of infection or haemorrhage with a lumbar puncture may be considered in the absence of structural pathology

A patient presenting with sudden onset of severe headache should be considered to have an SAH until proven otherwise

In acute seizures the immediate priority is to stop seizure activity, followed by rapid evaluation to determine the underlying cause

KEY WORDS: headache, meningitis, seizure, subarachnoid haemorrhage

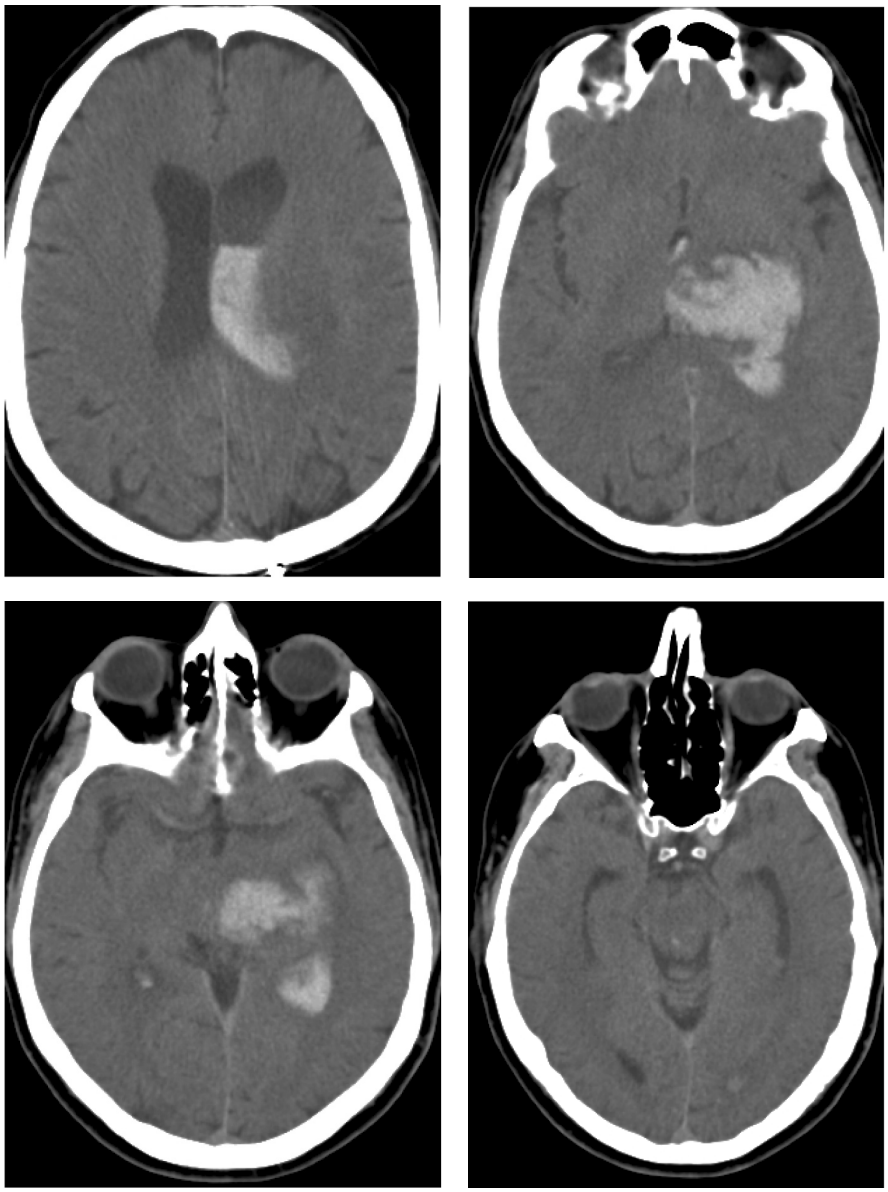

Fig 2. A large left-sided acute intraparenchymal haemorrhage adjacent to the left lateral ventricle. There is extension of haemorrhage into the lateral, third and fourth ventricles. The lateral ventricles appear dilated. There is some mass effect with minor midline shift. The basal cisterns are still visualised.

acquired bacterial meningitis.[3] LP should be deferred until after brain imaging if there are features of ICP, focal neurological deficits or the occurrence of seizures: in these situations the patient may be at risk of brain herniation.

Current international guidelines for empiric antibiotic treatment recommend vancomycin and a third-generation cephalosporin (ceftriaxone or cefotaxime) until organism identification and sensitivities are known.[4] Cephalosporin resistant *S. pneumoniae* is less prevalent in the UK than in other countries (eg USA, Australia, Spain). Local centres may advise initial treatment with cephalosporins alone, but with the recommendation that clinicians have a low threshold for addition of vancomycin if there is a delay to LP and/or if the patient has any risk factors for exposure to resistant *S. pneumoniae*.[5] Ampicillin should be added if the presence of *Listeria monocytogenes* is possible.

The risk of immunocompromise and history of recent travel should be documented. These features may prompt tailoring of initial antimicrobial treatment until microbial clarification is obtained.

The clinical presentations of bacterial meningitis, viral meningitis and viral encephalitis may overlap. Early seizures and confusion are more likely to occur in encephalitis. Brain MRI will often show temporal lobe changes in viral encephalitis. In patients with viral meningitis and encephalitis, CSF shows mononuclear pleocytosis and Gram stain is negative. In patients with bacterial meningitis there are predominantly polymorphs in the CSF. Viral polymerase chain reaction may assist in diagnosing the commoner viral causes of meningitis or encephalitis (eg herpes simplex). EEG may be helpful in viral encephalitis and show temporal lobe changes (eg slowing or 2–3 Hz spike/wave).[6]

Symptomatic management with analgesia, anti-emetics and adequate hydration is often adequate in uncomplicated viral meningitis. If there is clinical suspicion of encephalitis (seizure, delirium, obtundation), antiviral treatment should be commenced. Herpes simplex virus is the commonest cause and treatment is with aciclovir 10 mg/kg three times a day.

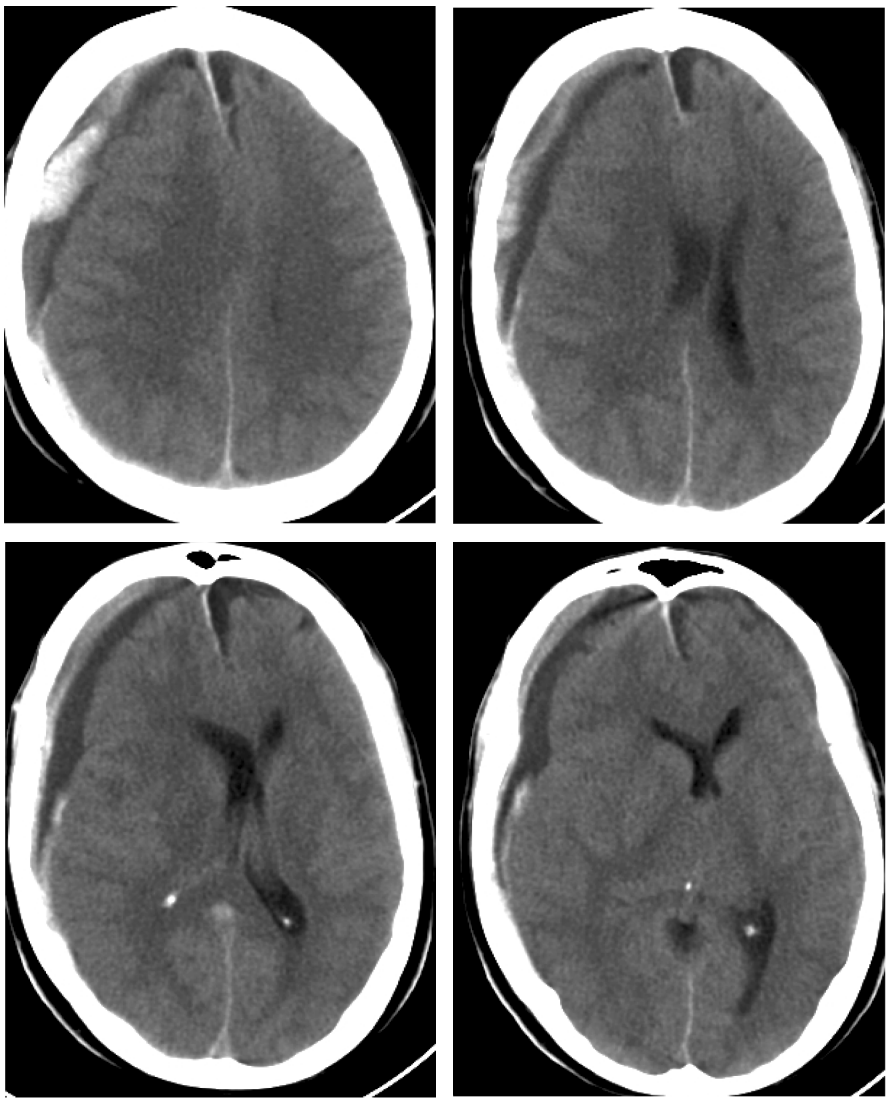

Fig 3. A large right acute-on-chronic right-sided subdural collection with significant mass effect and early subfalcine herniation.

Increased intracranial pressure

Headache which is worse in the morning, exacerbated by Valsalva manoeuvres and associated with nausea and vomiting may suggest increased ICP, but many acute headache patients without raised ICP may exhibit these clinical features. The finding of focal neurological signs with papilloedema strongly points to raised ICP. Imaging should be performed to exclude an underlying structural lesion or cerebral venous sinus thrombosis (CVST). LP should be considered only if a structural lesion is excluded on imaging. Intermittent severe headache, sometimes with episodes of loss of consciousness, may occasionally be due to intermittent obstruction of CSF flow, for example by a third ventricle colloid cyst.

Idiopathic intracranial hypertension is characterised by headache, often severe and pulsatile, with papilloedema. There may be transient visual obscurations or whooshing in the ears. This condition typically affects women of childbearing age and is associated with elevated body mass index. There may be a history of recent weight gain or use of certain medications (eg tetracyclines, vitamin A). LP confirms elevated opening pressure (>25 cm H_20). Other causes of increased ICP must be

excluded and CSF constituents and cerebral imaging be normal.[7] Untreated papilloedema may result in permanent visual loss. Treatment aims to lower ICP with acetazolamide, serial LP, shunting of CSF fluid or optic nerve sheath fenestration. Weight loss may help.[8]

Cerebral venous sinus thrombosis

Although uncommon, CVST is an important diagnosis to consider in patients presenting with severe headache. Predisposing factors include pregnancy, the postpartum period, prothrombotic states, dehydration and sepsis (especially intracranial infection). The headache is usually generalised and acute or subacute in onset. There may be features of raised ICP, meningism, drowsiness, seizures (often focal), papilloedema and focal neurological deficit due to stroke (venous infarct).[9] A non-contrast brain CT in the acute phase may show an 'empty delta' sign and hyperintensity in the sagittal, straight and transverse sinuses (Fig 4). Initial treatment is with therapeutic doses of heparin followed by oral warfarin. Secondary intracranial venous haemorrhage does not necessarily contraindicate anticoagulation but neurological advice should be sought.[10] Duration of warfarin therapy depends on persistence of underlying cause.

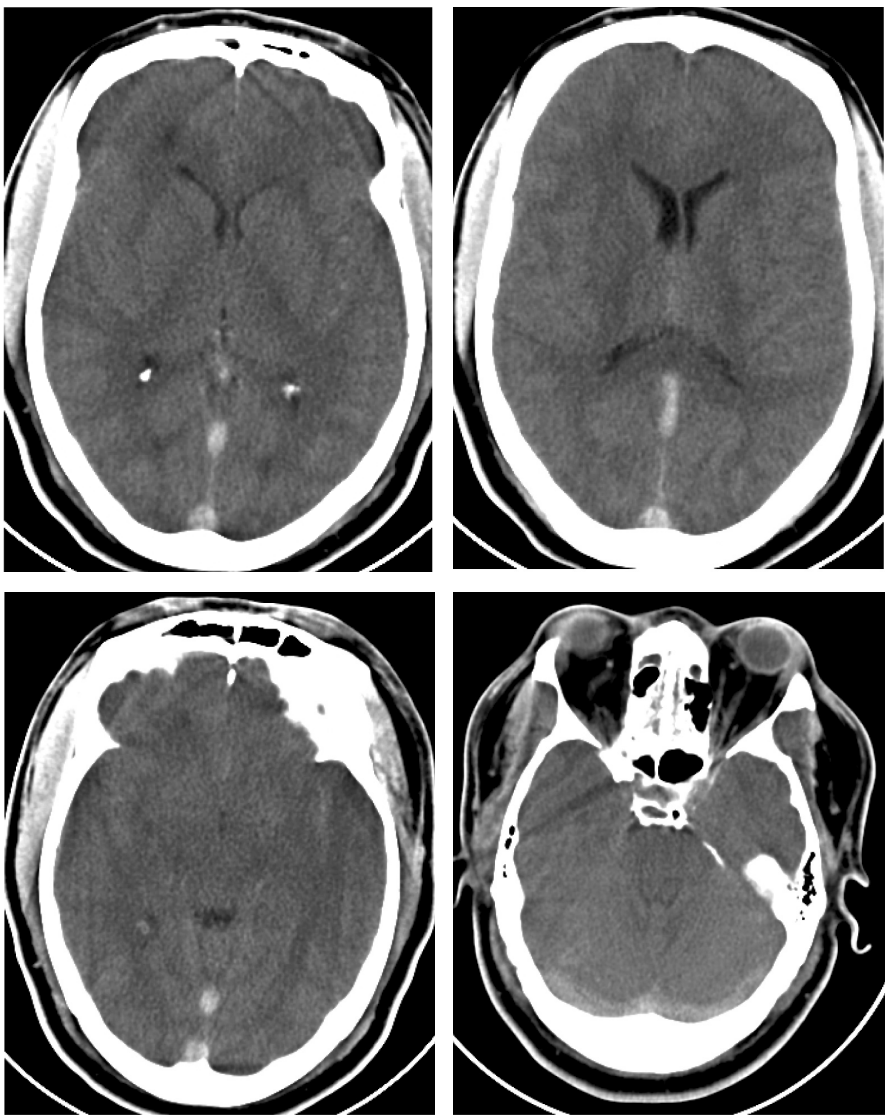

Fig 4. Hyperintensity within the straight sinus extends into the torcular, with further extension into the superior sagittal sinus. This is likely to represent acute thrombus. The suprasellar, ambient and quadrigeminal cisterns are effaced.

Migraine

Sudden onset of a severe headache, often unilateral, associated with nausea, vomiting and photophobia is typical of an acute migraine. However, this diagnosis should not be made in patients presenting with these symptoms for the first time unless all the above causes (intracranial haemorrhage, meningitis, encephalitis, ICP, CVST) have been considered and excluded by appropriate investigations. Patients known to have migraine may describe an antecedent prodrome or aura and may have a family history of migraine. Simple analgesia such as aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) in combination with anti-emetics may be adequate to control the acute event. 5-hydroxytryptamine-1 agonists (triptans) administered at the onset of a migraine may avert a full attack but should not be used in patients with cardiovascular disease.[11] After discharge, migraine patients may require ongoing management of their symptoms by their general practitioner or a neurologist. Opiate seeking patients sometimes present with apparent acute migraine to obtain pethidine or other opioids. The use of NSAIDs and triptans by acute physicians will help to reduce this behaviour.

Extracranial causes

Extracranial causes such as temporal arteritis, cervicogenic headache, sinusitis and glaucoma should be considered. History may point to the underlying diagnosis. Spontaneous or traumatic vertebrobasilar or carotid dissection can present with neck pain or headache and may be associated with stroke. MRI brain with MR angiogram may be required.

Seizure

A detailed history from the patient and, if possible, an eyewitness is important when assessing a patient with a blackout

suspected to be a generalised seizure. Syncope is the main differential; sometimes an initial syncopal event may be followed by a seizure if brain hypoxia is prolonged (eg if the patient is maintained in a vertical position). Features prior to the collapse should be asked about specifically (eg pallor, diaphoresis, palpitations, chest pain) which may point to syncope. In contrast, a specific aura (eg epigastric rising, olfactory symptoms, focal motor or sensory symptoms) is more suggestive of a seizure. Incontinence and tongue biting are more common in seizures but can sometimes accompany cardiac syncope. Recovery from syncope is usually more rapid than after a generalised seizure when a patient is often confused and may complain of headache and limb stiffness.

Seizures may be caused by idiopathic epilepsy, structural brain pathology or altered cerebral physiology (eg metabolic disturbance such as hypoglycaemia or alcohol withdrawal) (Table 2). Many patients presenting with seizure are known cases of epilepsy. Possible causes include general poor control of the condition (eg drug-resistant epilepsy), lack of drug compliance or other triggers such as lack of sleep. Patients presenting with a first seizure may represent a new case of idiopathic epilepsy or have underlying structural brain pathology, infection or other causes of altered cerebral physiology. Acute care includes assessing GCS, monitoring the airway in the postictal period and observing for recurrent events (Table 3).

Admission to hospital for observation and investigation is indicated for an unwitnessed or prolonged event, if there is serious injury, incomplete recovery of consciousness or mentation, or the presence of focal symptoms or signs. Full neurological examination including fundoscopy is essential. Ideally, EEG should be performed within the first 24 hours after the ictus to increase the chance of capturing a diagnostic abnormality.[12] Those presenting with a first idiopathic generalised seizure must be fast tracked for outpatient neurological assessment if they recover fully and have no focal neurological deficits.

Treatment should be directed at the underlying cause. Anticonvulsant therapy after a first seizure or in patients with infrequent seizures may reduce the seizure recurrence within the first 1–2 years. However, this does not affect long-term remission[13] so not all patients should be given anticonvulsant medication. Patients with known epilepsy already taking anticonvulsant medication at presentation should have serum anticonvulsant levels checked and a search for a precipitant for their seizure.

Table 2. Causes to consider in acute presentation with seizure.

Seizure type	Causes
Primary seizures	Idiopathic epilepsy syndrome
Secondary seizures	Medication, illicit drug or alcohol excess or withdrawal CNS process (eg tumour, infection, stroke, trauma) Metabolic disorder: glucose, calcium, sodium, mitochondrial disorder Cerebral venous sinus thrombosis Eclampsia Intracranial haemorrhage Convulsive syncope of cardiac or neurocardiogenic origin

CNS = central nervous system.

Table 3. Investigations to consider in acute presentation with seizure.

- Serum glucose
- Electrolytes (especially sodium and calcium)
- Arterial blood gas
- Blood alcohol
- Urinary drug screen
- Anticonvulsant levels
- ECG (cardiac rate and rhythm)
- MRI brain
- EEG
- Lumbar puncture and screen for infection

MRI = magnetic resonance imaging.

Status epilepticus

Status epilepticus is a medical emergency. Lorazepam 0.1 mg/kg can be administered for seizure termination, repeated in two minutes if ineffective. The patient should be given a loading dose of phenytoin 1.5 mg/kg to prevent further events. An underlying cause should be sought if the patient is not known to have epilepsy and a trigger sought in known epileptics. Occasionally, general anaesthesia is required to control status epilepticus.[14]

Safety concerns

It is important to discuss and document the regulations for driving and general safety behaviour in patients presenting with seizures.

Conclusions

Acute neurological problems frequently present to the emergency department. Accurate history taking and detailed clinical examination are paramount when assessing the patient presenting with neurological problems such as headache or seizure. This clinical assessment will direct further investigations to identify and treat serious underlying pathology.

References

1 Linn FH, Rinkel GJ, Algra A, van Gijn J. Headache characteristics in subarachnoid haemorrhage and benign thunderclap headache. *J Neurol Neurosurg Psychiatry* 1998;65:791–3.

2 van Gijn J, Rinkel GJ. Subarachnoid haemorrhage: diagnosis, causes and management. Review. *Brain* 2001; 124(Pt 2):249–78.

3 van de Beek D, de Gans J, McIntyre P, Prasad K. Steroids in adults with acute bacterial meningitis: a systematic review. *Lancet Infect Dis* 2004;4:139–43.

4 van de Beek D, de Gans J, Tunkel AR, Wijdicks EF. Community-acquired bacterial meningitis in adults. Review. *N Engl J Med* 2006;354:44–53.