Diagnosing acute headache

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Headache is a universal symptom and a common reason for adults to seek emergency medical care.[1–3] Most patients will have benign and self-limiting causes for their headaches, but a few will have serious intracranial disease. There is particular concern for the patient presenting with an acute onset headache, defined for the purposes of this article as a new spontaneous headache which has developed within the preceding few hours. The physician's task is apparently simple: to identify who needs further investigation and who needs reassurance. This article outlines the differential diagnosis, discusses how to investigate such patients and provides a brief management scheme. It is targeted at all physicians who see headache presenting acutely. It is not intended to be applied to children. Interested readers may wish to read more detailed articles on the subject.[4,5]

Making the diagnosis

Diagnosing headache syndromes depends upon extracting as accurate a history as possible, though this may be difficult in an emergency setting. Some factors such as HIV status substantially change the differential diagnosis, but this will not be discussed further here. General examination, particularly vital signs, can provide important clues (see below). Neurological examination is important as the presence of abnormal neurological signs changes the problem from being one of headache alone; generally speaking, a neurological abnormality will determine the direction of further investigations.

There is a large differential diagnostic list (Tables 1–3). Many of these conditions do not require specific urgent treatment, and failing to diagnose them immediately may not affect outcome, but this is definitely not true for subarachnoid haemorrhage (SAH) or meningitis – the diagnoses that most alarm doctors when faced with an 'acute headache'. The flow diagram (Fig 1) outlines the approach to making sense of the acute headaches.

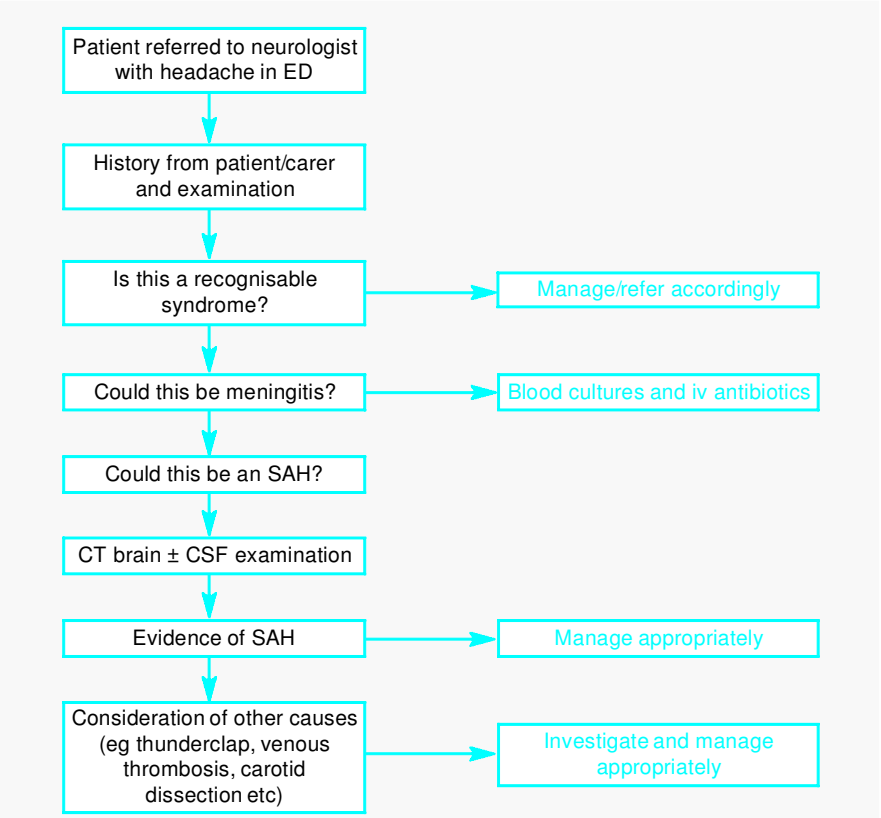

Fig 1. Flow diagram indicating the approach to a patient with acute headache (CSF = cerebrospinal fluid; CT = computed tomography; ED = emergency department; iv = intravenous; SAH = subarachnoid haemorrhage).

Table 1. Differential diagnosis of acute headache: primary headache syndromes.

Primary headache syndrome	Comment
Migraine	Diagnosis usually apparent from history of prior headaches
Cluster headache and related syndromes (including paroxysmal hemicranias)	Recurrent attacks of severe pain around eye lasting 15 min to 3 hours, with autonomic disturbance: tearing, conjunctival injection, ptosis, miosis
Thunderclap headache	A diagnosis of exclusion Headache has features of SAH but with negative investigations
Hypnic headaches	Generalised headaches that wake from sleep, brief (5–180 min), may occur 2–3 times a night
Benign exertional/sex headache	Repeated attacks of short-lived pain associated with activity Diagnosis cannot be made after single attack without investigation

SAH = subarachnoid haemorrhage.

Is it a recognisable syndrome?

A prior history of similar or closely related headaches may allow the diagnosis of a primary headache syndrome such as migraine or cluster headache (Table 1). The history of recent events may allow diagnosis of post-traumatic or post-ictal headaches (in someone with established epilepsy). The headache may clearly be part of a larger pathological process, for example in a patient with stroke and most of the conditions listed in Table 3.

Could this be meningitis?

The diagnosis of meningitis is often straightforward when a patient is unwell and febrile with a headache and neck stiffness. The headache is usually progressive over a few hours or days, but very occasionally may be very abrupt in onset, mimicking SAH. Focal neurological symptoms and disturbances of consciousness or confusion are common. Clinical examination is important to look for features of infection, including skin rashes, although a systematic assessment of individual items of clinical assessment found them to have a low accuracy.[6]

The implications of a diagnosis of meningitis are such that, if it is suspected, treatment should be commenced and investigations initiated. Blood cultures should be taken, intravenous antibiotics given and a lumbar puncture (LP) undertaken unless there is an indication for the need for imaging (see Box 1).

How not to miss subarachnoid haemorrhage

Most SAH patients present with sudden onset headache, and many have additional features such as vomiting, loss of consciousness or epileptic seizures, and focal neurological symptoms. However, about 20% of SAH present with headache alone.[2] The myth of 'warning' or 'sentinel'

Table 2. Differential diagnosis of acute headache: secondary headache syndromes (main differential diagnosis).

Secondary headache syndrome	Comment	Investigations		
		CT	LP	Other
Vascular disease				
Subarachnoid haemorrhage		++	+	
Non-traumatic subdural/ extradural/ intracerebral haemorrhage		++	N/A	
Dissection of carotid artery	Pain often unilateral; look out for Horner's syndrome; may be symptoms of transient ischaemia	%	%	MRI/MRA
Dissection of vertebrobasilar arteries	Pain often focal and sudden onset; may be symptoms of transient ischaemia	%	%	Angiography (MRA or catheter)
Cerebral venous thrombosis	Usually insidious onset headache; may be sudden; may have impaired consciousness, focal signs or seizures *NB*: increased risk post partum	–	–	MRV or CTV
Vasculitis (including giant cell arteritis)	Headache usually insidious onset; high level of suspicion in older patients	%	%	ESR, plasma viscosity
CNS infection				
Meningo-encephalitis (bacterial, viral, fungal)	Rarely acute onset headache; headache is usually progressive (see text)	%	++	
Cerebral abscess		++	N/A	May need MRI
Non-vascular intercranial disease				
Idiopathic intracranial hypertension	Occurs in obese young women; headache insidious; associated with visual obscurations; papilloedema	%	++	MRV or CTV to exclude venous sinus thrombosis
Intracranial hypotension (spontaneous or post-LP)	Posturally related generalised headache – relieved by lying down	%	++	MRI brain with contrast
Intracranial tumour		++	N/A	
Other causes				
Dental, ENT or ophthalmicdisease (eg sinusitis, acute glaucoma)	Usually apparent from the history	–	%	
Secondary to general medical conditions (eg infection other thancranial, epilepsy)	Usually apparent from the history; may need investigation for meningitis	%	%	

CNS = central nervous system; CT = computed tomography; CTV = computed tomography venogram; ENT = ear, nose and throat; ESR = erythrocyte sedimentation rate; LP = lumbar puncture; MRA = magnetic resonance angiogram; MRI = magnetic resonance imaging; MRV = magnetic resonance venogram.

Investigations: the entries indicate the likelihood of finding an abnormality: ++ = highly likely; + = likely; – = possible non-diagnostic abnormality; % = no abnormality usually found; N/A = not applicable as the test would not be done.

Table 3. Differential diagnosis of acute headache: secondary headache syndromes (other differential diagnosis).

		Investigations		
Secondary headache syndrome	**Comment**	**CT**	**LP**	**Other**
Traumatic				
Simple closed head injury	Usually apparent from the history. Beware: an unexplained, unwitnessed head injury might have been a subarachnoid => collapse and head injury	%	%	
Complex with subdural/extradural/ subarachnoid/intracerebral haemorrhage	Usually apparent from history	++		
Vascular disease				
Unruptured aneurysms	Not normally associated with headache; posterior communicating artery aneurysms compressing third nerve painful	%	%	
Acute cerebral ischaemia (TIA or stroke)	Usually the symptoms and signs of the stroke lead to the diagnosis	+		
Non-vascular intracranial disease				
Intermittent hydrocephalus (eg colloid cyst)	Rare; may present with acute headache	+		
Arnold-Chiari malformations	Typically produce headache on coughing and straining	–		MRI for diagnosis
Optic neuritis	Visual loss usually makes diagnosis clear	%	%	
Metabolic or toxic disturbances				
Phaeochromocytoma, thyroid disease, hypercarbia	Usually other features of history and examination lead to diagnosis	%	%	
Hypertensive encephalopathy		%	%	
Drug induced or withdrawal syndromes		%	%	
Other causes				
Cervical spine disease		%	%	

CT = computed tomography; LP = lumbar puncture; MRI = magnetic resonance imaging; TIA = transient ischaemic attack.

bleeds should be dispelled – the evidence indicates that these are most likely to be due to methodological flaws of previous studies (most commonly recall bias in retrospective studies) rather than a true phenomenon.[8]

The crucial step in the history is to establish how *quickly* the headache reached its maximal intensity. For exactly how many minutes a headache may crescendo and still be due to SAH is unknown, but a headache still increasing in severity after 15 minutes is unlikely to be due to SAH (although this does not exclude alternative serious pathology such as meningitis). Also unknown is how brief an SAH headache may be, although most physicians would think SAH exceedingly unlikely in any patient whose headache completely resolves within an hour. The history will allow the physician to identify those patients with a true acute onset headache (ie those who require investigation). Unfortunately, it will not allow confident clinical differentiation between serious pathology, such as SAH, and benign syndromes such as thunderclap headache).[9]

In summary, therefore, any patient presenting with a spontaneous new onset headache maximising immediately or within minutes requires further investigations to exclude an SAH – it is as simple as that.

BOX 1. Recommendations of the American College of Emergency Physicians (evidence at level of expert opinion.)[7]

Adult patients with headache exhibiting signs of increased intracranial pressure including papilloedema, absent venous pulsations on funduscopic examination, altered mental status or focal neurologic deficits should undergo a neuroimaging study before having an LP.

Alternative diagnoses

The main differential diagnoses are given in Table 2. If investigation directed towards the diagnosis of a possible SAH (ie computed tomography (CT) brain scan followed by LP) is negative, it will have clarified most differential diagnoses. The few that have not been, and

are likely to cause diagnostic confusion, include carotid and vertebral dissections (Box 2), cerebral venous thrombosis and non-neurological causes, particularly sinus disease and acute glaucoma. For all these, an awareness of the diagnosis and review of the patient following initial assessment are likely to direct further investigation. The need for other specialist investigations (eg catheter or non-invasive angiography/venography) will be dictated by the situation and should definitely involve specialists if they are not already responsible for the patient.

Management

A detailed description of the management of the many different types of headache is beyond the scope of this article; a 'generic' approach is taken in Table 4. The first step is to provide an accurate differential diagnosis, initiate any immediate treatment required (including analgesia) and organise appropriate investigations. All 'possible SAH' patients (ie anyone with a sudden onset headache as defined above) require admission for cerebral imaging and possibly LP. CT remains the investigation of choice for patients presenting with an acute onset headache soon after the ictus (Fig 3), although identification of blood on CT becomes increasingly difficult after a few days. An LP is still required conclusively to exclude an SAH in patients who have a 'normal' CT. Spectrophotometry should be used rather than simply visual inspection for xanthochromia.[10]

Conclusions

Acute onset headache is a common presenting symptom which alarms doctors and patients, yet most are benign. The diagnosis is based upon obtaining an accurate history, the most important part of which is separating the truly sudden onset (maximal immediately or within minutes) from the rest. All the former require investigations to exclude SAH before a benign diagnosis can be made. Patients should ideally be managed by either specialists or physicians experienced in headache management.

BOX 2. Case study.

A previously well 26-year-old man developed a severe headache of immediate maximal severity whilst preparing for a night out. He vomited and lay down for 30 minutes, during which time his headache improved a little, enough to allow him to continue showering and dressing. It then became suddenly worse again, he vomited once more and was admitted to a neuroscience centre within two hours of headache onset. When assessed, he was in considerable distress, sweating and becoming rather drowsy as the examination proceeded. There was no fever, rash, neck stiffness or focal neurological signs, and his vital signs were stable except for a mild tachycardia and hypertension thought to be a physiological response to pain. The (experienced) specialist registrar and (relatively inexperienced) consultant confidently diagnosed an SAH and both were surprised by normal results from routine blood tests, CT brain scan and LP. Within a few hours, his headache had almost resolved and he felt much better, but when he tried to get out of bed it became clear that he had developed hemi-ataxia. A magnetic resonance brain scan (Fig 2) revealed the explanation – an ischaemic stroke due to vertebral artery dissection.

Fig 2. Magnetic resonance axial brain (T2-weighted) showing a left cerebellar infarct, which initially presented with headache and vomiting alone.

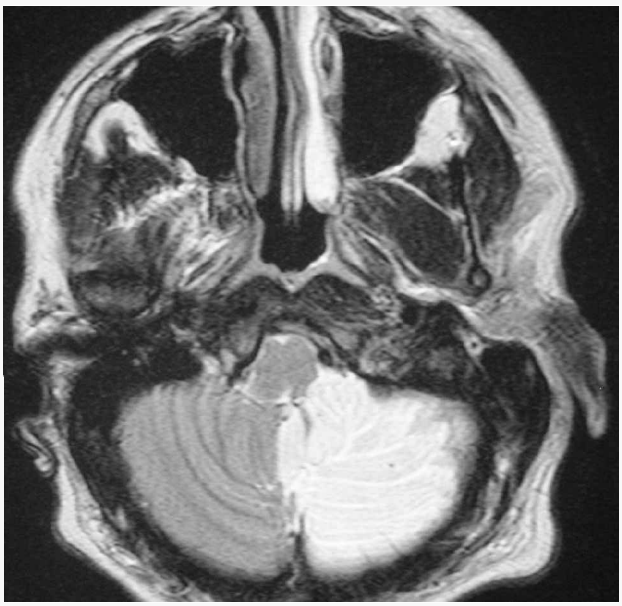

Table 4. Initial management of acute headache.

Drug therapy	Analgesia (paracetamol/aspirin, NSAID, codeine) Anti-emetic Nimodipine 60 mg 4-hourly po (all suspected SAH) Antibiotics iv (all suspected meningitis)
Fluid management	3 litre N saline iv over 24 hours for suspected SAH
Explanation to patient/family	
Investigations	Bloods (including blood cultures in suspected meningitis) 12-lead ECG (suspected SAH) CT brain (unenhanced) Lumbar puncture
Referral to neurosciences unit (if not already there)	

CT = computed tomography; iv = intravenous; NSAID = non-steroidal anti-inflammatory drug; po = by mouth; SAH = subarachnoid haemorrhage.

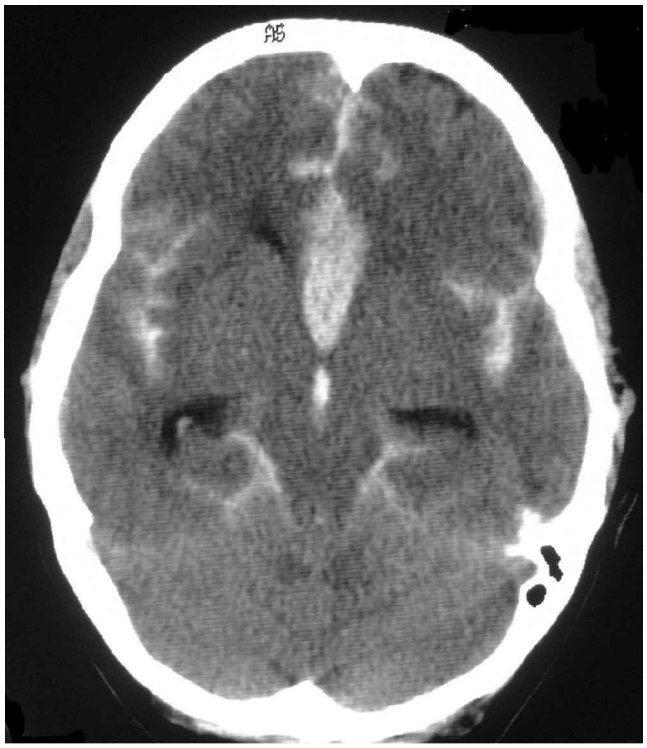

Fig 3. Unenhanced computed tomography brain scan revealing widespread subarachnoid blood with early hydrocephalus. The scan was performed within 24 hours of headache onset. Subsequent investigations revealed an anterior communicating artery aneurysm.

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Key Points

Acute onset headache is common

Most such patients do not have a sinister underlying cause, but some do

The key to deciding who requires investigation and who does not lies in the history

All patients presenting with headache maximal immediately or within a few minutes and lasting longer than an hour, require investigations (computed tomography brain scan – lumbar puncture) to exclude subarachnoid haemorrhage

KEY WORDS: acute headache, diagnosis, intracranial aneurysm, subarachnoid haemorrhage