REVIEW

Sudden headache emergency department

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INTRODUCTION

Headache is a common symptom that most of us have experienced at some time in our lives. Fortunately for doctors, only a minority of people with headache seek a medical opinion, but even so, headache is a common presenting symptom in primary care, and perhaps the most common single symptom general neurologists expect to see in their clinics. Most of these patients present in a non-urgent setting, and are dealt with as outpatients. However, some present as an emergency; indeed, headache accounts for 1–2% of patients presenting to an emergency department (Barton 1994; Morgenstern *et al.* 2001; Ward *et al.* 2001).

When asked to see a patient in the emergency department with a headache, the goals for the physician are straightforward:

- Make a (correct) diagnosis.
- Investigate appropriately and at the appropriate time.
- Manage appropriately.

This does not mean expose everyone to a brain scan and lumbar puncture as a blanket policy to avoid missing serious diagnoses. It means identify who might need tests (and thus probably admission to hospital for at least a day), and who definitely does not (and might therefore be safely discharged, with appropriate reassurance and treatment if necessary). The decision depends crucially on the suspected diagnosis. This article is intended to provide the neurologist (and interested general and emergency physicians) with advice on how to best achieve these aims.

MAKING THE DIAGNOSIS

Neurologists do not (or at least should not) need to be reminded that the history is everything (Thrush 2002; Morrish & Blame 2003). This is never more true than when assessing headache. However, most of us are used to the quiet, calm environment of our outpatient clinic or rooms, with a relatively undistressed patient, and adequate time to conduct our history and examination. The environment of the emergency department is very different; it may well be rather alien to many of us, there is plenty of noise and activity, the patient (and relatives) are likely to be distressed, and there is the inevitable pressure to 'do something' and 'shift the patient'. Although neurologists cannot afford to be leisurely in such situations, they must still remember that only an adequate history will lead to the correct diagnosis and management. Resist the temptation to curtail the history in the

hope that a quick CT brain scan will answer your questions; even if it is an obvious subarachnoid haemorrhage on CT, there are many aspects you need to pursue which can only be found in the history and examination. It may well be appropriate to provide analgesia and anti-emetics as you start to take the history, but whoever sees the patient at that first presentation must ensure they have obtained the best possible history. Most misdiagnoses of acute headache that I see are made within the first hour, and most often because the history was inadequate.

WHAT ARE THE POSSIBLE CAUSES OF THIS ACUTE ONSET HEADACHE?

You need to quickly work out whether it is a primary (and thus non-life-threatening) headache syndrome that you can diagnose and treat, but not investigate (e.g. migraine), or whether it is a potentially life-threatening secondary headache syndrome, requiring urgent investigation. Table 1 includes some of the more common primary and secondary headache syndromes, but the most important diagnosis not to miss is subarachnoid haemorrhage (SAH). Important because it is the most common of the secondary syndromes, and patients who are not diagnosed at their first point of medical contact, and then rebleed, have a considerably worse prognosis. We should err on the side of over-diagnosis rather than under-diagnosis (other neurological conditions such as epilepsy are the other way around). So, how likely is it that a patient with an acute onset headache has had an SAH, and how does one avoid missing it?

HOW LIKELY IS SAH IN A PATIENT PRESENTING WITH ACUTE ONSET HEADACHE?

A prospective, hospital-based study of sudden onset headache (reaching maximum intensity within 10 s, minimum length not specified) recorded an annual incidence of 43 per 100 000, 11% of whom were due to SAH, an annual incidence for SAH of about 5 per 100 000 (Landtblom *et al.* 2002). A community-based, prospective study of acute headache (onset within 60 s, lasting at least an hour) identified 148 patients, 37 (25%) of whom had suffered an SAH, an annual incidence of about 4 per 100 000 (Linn *et al.* 1994). Therefore, somewhere between 1 in 10 and 1 in 4 patients presenting with an acute onset headache has had an SAH. In the Linn study, 12% of the

Table 1 Causes of acute onset headache

Primary headache syndromes
Migraine
Cluster headache and related syndromes (including paroxysmal hemicranias)
Thunderclap headache
Hypnic headaches
Benign exertional/sex headache
Cough headache
Secondary headache syndromes
Vascular disease
Subarachnoid haemorrhage
Unruptured aneurysms
Acute cerebral ischaemia (TIA or stroke)
Non-traumatic subdural/extradural/intracerebral haemorrhage
Dissection of carotid/vertebrobasilar arteries
Intracranial venous thrombosis
Vasculitis (including giant cell arteritis)
Traumatic (head injury)
CNS infection
Non-vascular intracranial disease
Intermittent hydrocephalus (e.g. colloid cyst)
Idiopathic intracranial hypertension
Intracranial hypotension (spontaneous or post lumbar puncture)
Intracranial tumour
Pituitary apoplexy
Arnold-Chiari malformation
Optic neuritis
Metabolic or toxic disturbances (e.g. pheochromocytoma)

patients had another serious neurological condition (the most common being some other form of intracranial haemorrhage or infectious meningoencephalitis) – SAH is not the only potentially life threatening cause of acute headache.

HOW NOT TO MISS SUBARACHNOID HAEMORRHAGE

There are three main reasons why SAH is missed (Edlow 2003): failure to appreciate the clinical presentation, failure to understand the limitations of CT imaging, and failure to perform or correctly interpret the results of CSF examination. Table 2 summarizes how not to miss SAH, and Box 1 describes a semi-fictional case to highlight some of the potential pitfalls encountered in real life.

Clinical presentation of subarachnoid haemorrhage

In patients who are able to provide a history, the principal SAH symptom is headache. In about half the patients, it arises within a split second, and in the remainder the headache very rapidly evolves, reaching a maximum within seconds to

Table 2 How not to miss subarachnoid haemorrhage

- Take the history carefully: this will identify all patients with true acute onset headache (maximal within seconds/minutes). All these patients require investigation.
- Brain CT can never completely exclude SAH, and its sensitivity falls rapidly after 48 h.
- All acute onset headache patients with a normal CT need a CSF examination, and you need to know how to interpret the results.

minutes. It is uncertain what the longest period of evolution of a headache might be and still be due to SAH (senior physicians always seem to ‘recall’ a patient who appeared to have had a rather gradual onset of headache due to their SAH). But the evidence suggests that almost all SAH patients present with very abrupt onset headache (most maximal within seconds, a few within minutes), and that the likelihood of SAH is inversely proportional to the length of time it takes for the headache to reach a maximum. SAH must be an exceedingly rare cause of a headache taking 10 min or more to evolve to maximal.

How short might a headache last and still be due to SAH? Typically an SAH headache lasts at least a few days, although more rapid resolution does occur. But what about a headache which lasts no more than 30 min? Or an hour – or two? Could this still be an SAH? Again there is no unequivocal answer, but the ‘expert’ view is that SAH headache lasts at the very least for an hour or two (Warlow *et al.* 2001).

The site and character (other than onset) of the headache is not helpful in distinguishing SAH from other causes of sudden headache, but the headache is usually (but not invariably) severe, leading to the concept of ‘first or worst’ headache. Think carefully before you dismiss the patient complaining of their worst headache ever.

In summary, headache is the most common presenting symptom of SAH, and arises within seconds to minutes, and lasts at least an hour or two. These aspects can *only* be assessed with an accurate history. Of course there are various other ways in which SAH can present including coma, acute confusional states, status epilepticus, etc., but this article is concerned with headache (Warlow *et al.* 2001; Kirkpatrick 2002).

Non-headache symptoms of subarachnoid haemorrhage

Table 3 lists other symptoms that may arise in SAH. None are exclusive to SAH, and about 20%

BOX 1: THE POTENTIAL PITFALLS IN ASSESSING ACUTE ONSET HEADACHE

A 40-year-old woman was seen by her primary care physician with her worst ever headache, which had started abruptly 8 h previously, associated with vomiting. There was no other relevant history, and apart from distress, she had no abnormal physical signs. The doctor diagnosed 'a virus' and prescribed analgesia. Twenty-four hours later her husband took her to the local emergency department because she was no better where the physicians suspected SAH, and did a brain CT scan, which was reported (correctly) as normal. They reassured and discharged her. 24 h later she was back with continuing headache. The CT was repeated, was still normal, and a lumbar puncture (LP) was attempted. After several attempts by junior staff, the procedure was successfully performed by an anaesthetist; the results revealed 800 red blood cells, 2 white cells, a protein of 0.8 g/L, but neither bilirubin nor oxyhaemoglobin was detected. She was referred to the neurosurgeons as an SAH. She was seen by junior staff who requested a CT angiogram, which was normal. A senior neurosurgeon then reviewed the history during a teaching round; her original headache had been replaced by a different pattern such that lying flat she was almost headache free, but incapacitated with a crushing headache within a minute of sitting up. He cancelled the planned catheter angiogram, and requested a neurological referral. Her presumed post LP headache was cured with a lumbar blood patch performed by a (different) anaesthetist, and after 48 h of pain free mobilization, she was finally discharged for good. The neurologist agreed with the neurosurgeon that the initial headache was a thunderclap headache not SAH, and that the CSF results were due to the traumatic LP procedure.

Errors made

- The primary care physician failed to recognize the potential implications of the initial symptoms, which were entirely compatible with SAH.
- Hospital staff recognized the symptoms on initial presentation, but failed to recognize the limitations of a normal CT brain scan.
- Repeating the CT within 24 h was rather pointless, and wasted time and resources; a review of the original CT, before proceeding to LP, would have been more appropriate.
- LP attempt by junior staff was inappropriate, and should have been performed by an experienced operator.
- Results of the LP incorrectly interpreted as SAH, but did at least lead her to a neuroscience unit.
- Decision to proceed to CT angiography was probably incorrect, as the CSF results did not suggest an SAH, and it was only the intervention of an experienced neurosurgeon which prevented her exposure to an invasive catheter angiogram.

Would this story have been different had she been seen at the initial presentation by an experienced neurologist/neurosurgeon?

of SAH presents with headache alone. Neck stiffness may take several hours to develop, and even not develop at all in comatose patients. So whilst these symptoms in the setting of an abrupt onset headache might heighten your confidence in the diagnosis, the symptoms alone cannot help distinguish between SAH and more benign forms of abrupt onset headache (Linn *et al.* 1998; Landtblom *et al.* 2002).

Other features of the history may also raise the likelihood of SAH, especially a family history of aneurysmal SAH (one or more first degree relatives), as well as other rather rare predisposing conditions to aneurysm formation such as polycystic kidney disease, Marfan's syndrome, etc.

Sentinel headaches and warning leaks: reality or neuromythology?

Many reviews of SAH refer to sentinel headaches, defined as a sudden onset headache with subsequent recovery in the weeks or months prior to a diagnosed aneurysmal SAH. A systematic review concluded that the frequency varies from 0 to 40%, which perhaps reflects the variability of the quality of the included studies as much as the true figure (Polmear 2003). These headaches were originally thought to be due to 'warning leaks', although non-haemorrhagic causes such as stretching or dissection of an aneurysmal wall have also been entertained. Therefore it has been suggested that sentinel headaches matter

Table 3 Non-headache features of subarachnoid haemorrhage

Nausea, vomiting
Pyrexia
Photophobia
Neck stiffness (meningism)
Visual disturbances (due to intraocular haemorrhage)
Transient loss of consciousness
Coma
Epileptic seizures
Focal neurological signs
Sudden death

because if recognized at the time they might be a way of identifying and treating patients early, and thus reducing morbidity and mortality (Demaerschalk & Dodick 2003). So do sentinel headaches exist, and if so, what are they due to and how common are they?

The best study design to answer this question would be prospective (to avoid recall bias), and community based (to avoid referral bias). The two studies which best fit this description concluded that sentinel headaches are rare. In the Dutch study, only two of 37 patients with SAH reported previous similar headache, one a month previously, the other 3 days prior to admission (Linn *et al.* 1994). The non-SAH patients in this study were followed up for a further 12 months, and none suffered SAH or other serious neurological disease. In the Swedish study only one out of 23 SAH reported a recent (time not specified) similar headache, whilst three other patients reported previous sudden onset headache with an interval of several years (Landtblom *et al.* 2002). In a further prospective study, the clinical and radiological features of a group of patients presenting to hospital with aneurysmal SAH were compared; there were no differences between those with preceding episodes of sudden headache and those without (Linn *et al.* 2000).

In summary, sentinel headaches are rare which is reassuring. If one really believed that sentinel headaches were common, and might not even represent a bleed, then one would be forced to conclude that every patient presenting with sudden onset headache needs not only a CT and lumbar puncture (LP), but also an angiogram of some kind to exclude that aneurysm with its 'stretched wall' or ever so small 'warning' leak undetectable on CT and LP – and then wonder how many of the aneurysms one identified would be entirely incidental, as the prevalence of asymptomatic aneurysms in the general population over the age of 30 is about 5% (Wardlaw & White 2000).

'Sentinel headaches' are rare and the term 'warning leak' should be abandoned; aneurysms do not leak, they rupture and cause SAH, which may then not be recognized by either the patient or their doctor – but that is a missed diagnosis of SAH, not a 'warning leak'. Rather than perpetuate the mythology of sentinel headaches (Horwitz 2003), we should redirect our energies into educating the public and physicians that sudden onset headache must not be ignored when it *first* occurs (Fridriksson *et al.* 2001). Grieving rela-

tives (and their lawyers) remind us that SAH is missed by doctors, and may prove costly in both human and financial terms.

Activity at onset

It is commonly said that SAH is more likely to start during physical activity, particularly sexual activity (the first fatal SAH I saw as a student occurred in precisely such circumstances). Once again, however, the evidence is not supportive. In the Swedish study, most had headache onset during 'resting or calm activity', 61% of the SAH group and 66% of the non-SAH group. Eleven patients (8%) experienced onset during sex, but only two (both men) had had an SAH (Landtblom *et al.* 2002). SAH arising from sleep is anecdotally rare, although there are few published data.

Examination

Clinical examination is rather little help in deciding whether the patient has had an SAH or not. Subhyaloid haemorrhage if present is helpful, but photophobia and distress may present technical problems, and this sign may be difficult even for the specialist, and can never replace the history. The examination is more helpful at suggesting alternative diagnoses such as infection (fever, skin rash), intracranial venous thrombosis (papilloedema), etc. It is also of vital importance to accurately assess the patient's haemodynamic state, conscious level, and for SAH to grade the patient (Table 4).

Summary

An accurate history will identify who has had a true acute onset headache (maximal within seconds to minutes), but it will *not* allow you to identify accurately the benign primary syndromes within this group, even though they are the majority. Thus all patients with a true acute onset headache require investigation to exclude SAH. If the history is not suggestive of SAH (i.e. it is not an acute onset headache), then investigation for SAH is inappropriate (of course other suggested diagnoses may require investigation). So the history is of vital importance in the decision making process.

INVESTIGATIONS

Having identified who needs investigating, the next issue is what tests to do and when. All patients who are admitted even for a few hours merit simple blood tests, including a full blood count (raised white cell count suggesting infection,

platelet disturbance an explanation for intracranial haemorrhage, etc.), urea and electrolytes (hyponatraemia due to salt wasting is common in SAH and should be corrected), perhaps a simple coagulation screen (but not a full thrombophilia screen), definitely in patients on anticoagulants, and a 12-lead electrocardiogram (ischaemic-looking ECG changes in SAH are very common although their significance is uncertain).

Brain CT in suspected subarachnoid haemorrhage

The neurological investigation of first choice is a non-enhanced computed tomogram of the brain (CT). Whilst there is no need to delay CT (fresh blood will be immediately apparent), if the patient is conscious and stable, they do not need an out of hours scan unless this is easily available, and may safely wait a few hours overnight until the first slot the next morning. On the other hand, patients with a Glasgow Coma Score less than 15 or focal neurology *do* merit immediate scanning, to aid diagnosis and direct management (e.g. hydrocephalus requiring surgical intervention). A detailed discussion of radiological patterns of blood on CT is beyond the scope of this article, but the distribution can help distinguish aneurysmal from perimesencephalic haemorrhage (van Gijn *et al.* 1985), and the likely site of the aneurysm, the most common being anterior and

Table 4 World Federation of Neurological Surgeons grading of subarachnoid haemorrhage

WFNS grade	Glasgow Coma Score (sum)	Focal neurological signs
I	15	No
II	13–14	No
III	13–14	Yes
IV	7–12	Yes/No
V	3–6	Yes/No

An accurate history will identify who has had a true acute onset headache, but it will not allow you to identify accurately the benign primary syndromes within this group, even though they are the majority

Figure 1 (a) Distribution of subarachnoid blood on unenhanced brain CT, mainly symmetrical but thickest around the suprasellar cistern with extension up the anterior interhemispheric fissure, a pattern suggestive of ruptured anterior communicating artery aneurysm (note, patient also has previously clipped left middle cerebral artery aneurysm causing scan artefact); anterior communicating artery aneurysm (arrow) confirmed on (b) catheter and (c) CT angiogram.

Figure 2 (a) Distribution of subarachnoid blood on brain CT mainly right sided, with thickest clot around the posterior communicating artery region with blood in both anterior & posterior basal cisterns, suggestive of a ruptured right posterior communicating artery aneurysm; right posterior communicating artery aneurysm (arrow) confirmed on (b) catheter and (c) CT angiogram.

Figure 3 Brain CT distribution of blood suggestive of a ruptured right middle cerebral artery aneurysm (arrows), later confirmed on angiography.

Figure 4 (a) Reconstructed 3-D image of a right posterior communicating artery aneurysm (arrow) and (b) catheter angiogram appearance.

posterior communicating, and middle cerebral arteries (Figs 1,2,3 and 4). The amount of blood can also be graded using Fisher's scale (Kirkpatrick 2002).

Provided the scan is done within 48 h of symptom onset, on a modern CT scanner, and interpreted by an experienced neuroradiologist, the sensitivity of CT imaging is very high, in excess of 95% (but crucially not 100%). In real life, however, not all of these criteria may be met, and my own experience is that SAH identified by CSF examination following a 'normal' CT is more often due to observer error than anything else (i.e. the subarachnoid blood was evident on the initial CT, but not identified at the time). This is especially true for non-neuroradiologists, who paradoxically (in the UK at least) report the majority of CTs requested in such circumstances. Telemedicine may allow for more specialist reporting in the future.

After the first 48 h the sensitivity of CT (even in expert hands) starts to fall rapidly as the blood is absorbed. Clearly there is nothing (other than education) that can be done about delays in patients presenting to medical attention, but once a patient does present to secondary care, the delay to CT imaging should be no more than a few hours if presenting out of hours, and none when presenting within hours. Accept no delay for CT other than a few hours overnight assuming the patient is stable. The 'late presenter' to medical care is discussed below.

CSF examination

It is evident from the above that a small number of SAH patients – even presenting early – really do have a truly normal CT brain scan, and they can only be diagnosed by CSF examination. However, the interpretation of the CSF findings is far more complicated than most reviews of SAH suggest (Williams 2004). Therefore I repeat my earlier plea that you think very carefully whether the history is suggestive of SAH before doing tests. Doing a 'quick CT, just in case', finding the scan is normal, but not then proceeding to look at the CSF is to misunderstand the role of these tests. On the other hand, undertaking a CT and CSF examination in all patients who happen to be admitted under your care with a headache of more-or-less any description is equally foolish. In such circumstances, tests are more likely to confuse than clarify, and even in specialist units patients are being inappropriately exposed to the hazards

Figure 5 Evolution of 'xanthochromia' in the CSF.

of catheter angiography, based on the dubious results of an inappropriate test, most often a lumbar puncture. Unfortunately things have usually developed a momentum of their own by that stage, making it difficult to reverse the process.

The 'rules' for lumbar puncture in CT – negative suspected subarachnoid haemorrhage

1. Ideally delay the LP for 12 h after symptom onset

The logic is straightforward (Fig. 5) and the biological explanation for xanthochromia has been discussed elsewhere in detail (UK NEQAS for Immunochemistry Working group 2003; Williams 2004). Although oxyhaemoglobin released from lysed red blood cells probably appears within 2 or 3 h, the development of bilirubin (which only occurs *within* the body, not a sample tube), takes longer, although no one is quite sure how long. Twelve hours is taken as the standard but this is based on clinical data from patients with CT-*positive* SAH (Vermeulen *et al.* 1989). However, we want to know the sensitivity and specificity of CSF examination at different time points after headache onset

in CT- *negative* patients, and these data simply do not exist. For this and other reasons, the policy of delaying LP has been vigorously challenged, the non-delayers claiming that the presence of red cells alone is the key observation (Gerber *et al.* 1998; Edlow 2003). I have sympathy with this view, and an acellular CSF within 48 h of symptom onset would exclude SAH in my own mind. The problem of course comes with a traumatic tap mimicking SAH. Various ways have been suggested of differentiating between traumatic tap and SAH (Shah & Edlow 2002), the most important being the appearance of bilirubin on spectrophotometric analysis (present in SAH, but not traumatic tap). However, this analysis is likely to be negative regardless of the diagnosis if the sample is analysed within 12 h of headache onset, thus not providing any help with the differentiation. Pragmatically therefore I do delay, as I do not think a patient is likely to come to harm by delaying, and I want to get the diagnosis right (and I know that with a true CT-negative patient within 12 h, the pre-test probability of an SAH is already very low). The only caveat to this delay is if there is any suggestion of an alternative serious diagnosis, the most obvious being CNS infection; however, in that case, you should have taken blood cultures and filled the patient with antibiotics long before you reached for the LP needle.

2. LP should be performed by an experienced operator

This is not the time for the junior resident, or worse a medical student, to practice his or her underdeveloped skills. Because of the problems interpreting the results, one needs to reduce the chance of a traumatic tap to an absolute minimum, which means someone who knows what they are doing does the LP. Of course even the most experienced and smoothest practitioner will on occasion produce a traumatic tap (let us dispel the notion that success at the first pass necessarily means a clean tap), but common sense dictates that it is less likely than with a novice. And if one observes the brain CT rule above, then there should be no need for LPs in the middle of the night by junior staff.

3. Undertake the procedure correctly

Non-neurologists frequently omit to measure the opening pressure, which should be routine in all LPs. A very raised pressure in a sudden

headache setting might alert you to intracranial venous thrombosis as an alternative diagnosis. You should also endeavour to collect four separate samples, and label the fourth sample clearly as such – the laboratory will use this one for spectrophotometric assessment.

4. Transport the sample to the laboratory correctly

Protect the specimen from light (e.g. wrap in silver foil) to prevent the *in vitro* degradation of bilirubin to deoxyhaemoglobin (Fig. 5), and avoid pneumatic delivery systems, which may cause haemolysis. The sample should be delivered to the laboratory as soon as possible (ideally within an hour of being taken), and you should warn the laboratory that the sample is coming, so they may anticipate its arrival and deal with it appropriately.

5. Know how your laboratory analyses the sample

Recent guidelines have indicated that spectrophotometric assessment is the gold standard (UK NEQAS for Immunochemistry Working Group 2003), and is preferred to visual inspection which may provide a false negative result (Cruickshank *et al.* 2005). Despite this, some laboratories do still rely on visual inspection. You should also understand what it is that the laboratory is looking for, which will help you understand that the result is not a simple 'yes or no', but based on the analysis of a waveform, which can sometimes be difficult (Williams 2004).

6. Know how to interpret the result

There is a surprising amount of controversy and uncertainty about how to interpret the CSF results, but recently published guidelines have helped clarify some areas of confusion (UK NEQAS for Immunochemistry Working group 2003). In brief:

- Spectrophotometry is the recommended method of analysis, and should be performed on the final sample of CSF collected.
- The presence of CSF bilirubin is the key result suggestive of SAH, and is usually accompanied by oxyhaemoglobin.
- Isolated oxyhaemoglobin is usually artefactual, but may *occasionally* occur in SAH.
- Absence of either pigment is not supportive of SAH.

Xanthochromia is rather a meaningless (diagnostically at least) term, simply referring to the colour of the CSF supernatant. It is clear

from the above that this is a rather simplified notion, and you should instead think in terms of pigments, oxyhaemoglobin and bilirubin.

Early on (within the first 48 h), the red cell count is also important, and SAH with less than a 1000 red cells per cumm is probably exceedingly rare (Edlow 2003). Beyond this time window, the key result is spectrophotometric analysis.

FURTHER INVESTIGATION OF SUBARACHNOID HAEMORRHAGE

Once you have completed the above investigations, you should have identified the patient with SAH who now needs further specialist investigations and management. If you do not work in a unit with interventional radiologists and neurosurgeons on hand, now is the time to pick up the telephone and transfer the patient, if indeed you have not already done so. If you have excluded SAH, it is likely that your patient has a benign diagnosis, such as thunderclap headache, or migraine. But before discharging them, consider the list in Table 1 and satisfy yourself that there are no features that suggest other investigations are required. If not, discharge the patient, with usually no requirement for further review.

About 85% of SAH is secondary to a ruptured berry aneurysm (10% are perimesencephalic haemorrhages with an excellent prognosis, the remaining 5% are a mixed group of rarities including arteriovenous malformations of various sorts). Therefore, the immediate priority is to identify the aneurysm, and secure it. In my own unit, the investigation of first choice is CT angiography (CTA), which is very sensitive for aneurysms as small as 3 mm, and it seems that observer error is a bigger source of false negatives than technical issues of CT resolution (Keston *et al.* 2005; White, pers. comm.). CTA is less invasive than a catheter angiogram (although it does require a large dose of intravenous contrast); fewer resources are required (it does not take up time in the angiography suite); and it allows identification and management planning for identified aneurysms. Perhaps surprisingly, it does not free up much radiologist time, as adequate interpretation requires analysis of the base images on a workstation to produce 3-D reconstructions, which is time consuming (Fig. 4).

Some patients who have a negative CTA will still require a catheter angiogram, but not all;

the decision will be based on a number of factors, most importantly the distribution of blood on the presenting CT, which may suggest certain patterns of bleeding (e.g. aneurysmal vs. perimesencephalic).

WHO SHOULD ASSESS AND ADMIT PATIENTS WITH SUDDEN ONSET HEADACHE?

In the UK, most patients presenting to hospital with a sudden onset headache never see a neurologist or neurosurgeon, and are dealt with by emergency and general physicians. What few data there are suggest that the quality of care can be much improved (Webb *et al.* 2003; Schofield *et al.* 2004). Certainly all patients who have a diagnosis of SAH should be transferred to a neurosciences unit as soon as possible, for further investigations and management (Kirkpatrick 2002; Molyneux *et al.* 2002; Sellar 2005). Although my own view is that ideally all patients presenting with acute onset headache should be initially assessed by a neurologist, I appreciate that, in the UK at least (Carroll & Zajicek 2004), and perhaps even in the US (Edlow 2003), such a goal is at present impossible to achieve in many areas, due to lack of neurologists. In which case, emergency and admitting medical physicians need to be aware of all the potential pitfalls in diagnosis, in particular the need for, and interpretation of CSF analysis.

THE LATE PRESENTATION OF ACUTE ONSET HEADACHE

All neurologists will be familiar with the difficult scenario of the patient who gives a history of an acute onset headache that occurred a few weeks ago. We are then faced with the dilemma of whether we should investigate (and if so how) for a possible SAH, or whether to reassure and discharge the patient, and hope we are right!

It is impossible to provide any clear protocols, and each case will require assessment on its own merits. Clearly some factors such as a family history of SAH will persuade one to investigate, as will the attitude of the patient. If one does decide to investigate, which tests should one opt for? This really depends on how recent the onset of the headache was. Within the previous 2 weeks, I would undertake a CT brain scan (likely to be normal, unless there is a large visible aneurysm), followed by an LP. Absence of pigments (especially bilirubin) would probably reassure me. Much beyond 3 weeks, and I would be increas-

The presence of CSF bilirubin is the key result suggestive of SAH, and is usually accompanied by oxyhaemoglobin

BOX 2: A DIFFICULT CASE

A 33-year-old man presented to the on-call neurologist. Ten days earlier, he had been awoken at 2 am by a headache. He had been able to doze on and off for the rest of the night. The headache had persisted, although definitely improved. There had been no associated focal neurological or migrainous symptoms. He had suffered occasional headaches in the past without seeking medical attention, but none had persisted this long. He was otherwise well, and on no medication. Nine months earlier, his brother (aged 40) had died of a ruptured basilar aneurysm; there was a suggestion that the diagnosis had initially been missed, and that he had died of a rebleed, but no other details were known. Not surprisingly this patient knew a lot about aneurysms, but had not previously sought any screening. Examination was normal, other than a rather tense patient.

Options

- Reassure and discharge with no investigations (the history is not suggestive of SAH, onset during sleep is exceptional).
- CT brain and CSF examination, and only proceed further if tests suggest SAH.
- CT or catheter angiogram.
- Others?

Outcome

We did not think the story was suggestive of an SAH, but we did not think we could easily ignore the family history. We elected to exclude an SAH, and thus undertook a CT brain and CSF examination, both of which were entirely normal. We did not proceed to further imaging, as we felt this would be 'screening', which was not the immediate clinical problem we faced. We discussed the results and implications with the patient, who understood that we had excluded an SAH, but not an unruptured asymptomatic aneurysm. However, he was invited to return to the clinic at some stage if he wished to discuss screening for an unruptured aneurysm.

What would you have done?

ingly concerned that a normal CSF might not adequately exclude SAH. I appreciate this 2-week 'cut off' is quite arbitrary, and we really do not have much adequate data on the persistence of bilirubin after SAH. Beyond 2 weeks, I would ask for a CT angiogram, accepting (and explaining

Ideally all patients presenting with acute onset headache should be initially assessed by a neurologist

CONCLUSIONS

- Acute onset headache is a common problem in the emergency department.
- Most patients do not have a serious underlying cause, but between 10 and 25% do.
- Subarachnoid haemorrhage (SAH) is the key diagnosis not to miss, although it is not the only potentially serious diagnosis that requires consideration.
- Accurate diagnosis is based on a careful history, and judicious use of the appropriate tests, with an understanding of their strengths and weaknesses.
- A normal CT brain scan does not exclude SAH, particularly if delayed by more than a day or two after headache onset.
- CSF results may be more difficult to interpret than many imagine, and you should know what you and your laboratory are looking for.
- Neurologists should be involved early in the management of acute onset headache.

to the patient in advance) that the risk of finding an incidental unruptured aneurysm is perhaps as high as 5% (Wardlaw & White 2000), and that there was small chance that non-invasive imaging might miss a very small aneurysm. Others suggest that any patient presenting late with an acute onset headache should have catheter angiography. I offer my own approach, but with no persuasive evidence that it is unequivocally the right thing to do – Box 2 illustrates the possible problems with a case admitted recently.

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