

Case 11

A 37-year-old woman with sudden severe headache

You are telephoned by a GP about a 37-year-old female patient he has been called to see. This morning while at work Mrs Annika Jenski described a sudden onset of severe headache. Mrs Jenski has a past history of migraine but this headache is very different. Mrs Jenski has vomited twice and the pain has now continued for 2 h with no relief from paracetamol or ibuprofen. The GP is concerned about the possibility of subarachnoid haemorrhage (SAH).

What challenges will this patient present?

- Sudden severe headache is the characteristic symptom of a SAH, usually caused by spontaneous bleeding from a cerebral 'berry' aneurysm
- SAH can be catastrophic, resulting in raised intracranial pressure, hydrocephalus, permanent cerebral damage or death
- In many cases, however, a massive bleed is preceded by a sentinel haemorrhage, a small bleed providing clinicians with a warning of what may be around the corner
- Failure to recognise this condition can have devastating consequences for the patient, with a high probability of subsequent litigation against the clinical staff involved in the original assessment
- It is crucial for clinicians to maintain a high index of suspicion for the diagnosis of SAH when assessing patients presenting to hospital with sudden, severe headache

On arrival in hospital Mrs Jenski remains in considerable pain. She is clutching her head and prefers to keep her eyes shut. However, Mrs Jenski is able to answer questions appropriately, obeys commands and opens her eyes when requested. Mrs Jenski is holding a vomit bowl and has recently vomited. Her blood pressure is 150/90 with a pulse of 100 beats/min.

What immediate management is required?

Mrs Jenski is breathing spontaneously and has a Glasgow Coma Score of at least 14/15 (see p. 127). Mrs Jenski should therefore be able to maintain her own airway. Blood pressure is a little high, but this may relate to the pain and distress she is experiencing.

The following management is required.

- Ensure patency of the airway and calculate the Glasgow Coma Score (GCS)
- Connect to cardiac monitor and pulse oximeter
- Apply oxygen via a facemask
- Obtain intravenous access
- Send blood for full blood count (FBC), urea and electrolytes (U&E), liver function tests (LFT), clotting screen, and group and save
- Prescribe analgesia (see below) and an intravenous antiemetic (e.g. cyclizine 50 mg, 8 hourly)
- Ask the nursing staff to undertake neurological observations (neuro obs) every 30 min (see Fig. 37)

How should the choice of analgesia be determined?

- The choice of analgesic should be determined according to the severity of the pain
- Intramuscular (IM) injections of codeine phosphate (30–60 mg 2 hourly, maximum 240 mg in 24 h) are a common choice in this situation and frequently result in a significant improvement in pain
- Stronger opiate analgesia (e.g. intravenous morphine sulphate 5–10 mg) may be required; in such cases consideration should be given to the impact this may have had on pupillary responses and the conscious level when interpreting subsequent neurological observations

appears more comfortable. She still prefers to lie still with her eyes shut, but says the pain is easing. Mrs Jenski's blood pressure has improved to 130/70, pulse 90 beats/min. Mrs Jenski is now able to give a full history and consent to clinical examination.

Mrs Jenski describes that she felt well this morning and went to work as usual. While walking to the bathroom Mrs Jenski experienced a sudden and very severe headache at the back of her head. The pain was of such intensity that it made her fall to her knees. Mrs Jenski has never experienced pain like it before. Shortly after the onset of the pain she vomited. The pain continued unabated until the injection Mrs Jenski was given on the ward. Mrs Jenski continues to feel nauseated. She finds that movements of her head worsen the pain, as does bright light. The pain was also worse when Mrs Jenski vomited.

Mrs Jenski describes a previous history of migraine, but this has never started abruptly, and the pain is usually focused around the front of her head on the left side. Mrs Jenski normally experiences flashing lights in her vision with her migraine, which have not occurred this time. There is no other significant past history of note and no recent history of head trauma, although she had a car accident with whiplash to her neck three months ago.

On examination Mrs Jenski is in discomfort, worsened by head movement. Her neck is slightly stiff. There is mild

tenderness over the occiput and neck muscles. The remainder of her examination, including a full neurological assessment, is entirely normal.

Are the clinical features compatible with a diagnosis of subarachnoid haemorrhage?

The characteristic features of the headache of SAH are described in Table 46. Alternative diagnoses may need to be considered, but only after SAH has been excluded (see Table 47).

Mrs Jenski has mentioned a number of symptoms that should raise concern.

- Abrupt onset of pain
- Severity described as worst ever
- Worst pain at the onset
- Prolonged duration

Does the 'normal' clinical examination make SAH less likely?

Clinical examination of patients with SAH is usually entirely normal. It is unusual to find focal neurological signs on examination of patients with SAH. The history alone should be the trigger to proceed to further investigation. The following abnormalities may be found, but usually in association with a large bleed.

Table 46 Characteristic features of the headache in subarachnoid haemorrhage (SAH)

		Comments
Site	Often occipital or generalised	Localised/unilateral pain is unusual in SAH
Onset	Abrupt/explosive or rapid onset (over seconds/minutes)	Patient will usually remember what they were doing at the time of onset. Onset is usually during activity Patients classically describe pain as 'like being struck on the head'
Character	Continuous, unremitting	
Radiation	May radiate all over cranium/into neck	
Associated symptoms	Nausea/vomiting Syncope Drowsiness/irritability	Focal neurological symptoms/signs are unusual following SAH
Time course	Pain is worst at onset Usually persists over several hours	Transient headache (seconds or minutes)
Exacerbating/relieving factors	Often exacerbated by straining/vomiting, etc.	
Severity	Very severe	Often described as 'worst ever headache'

Table 47 Differential diagnosis of sudden severe headache

Condition	Clinical features/comments
Subarachnoid haemorrhage (SAH)	See text/Table 46; remember that no clinical feature will reliably distinguish SAH from any of the other conditions listed in this table; exclusion of SAH by CT and lumbar puncture (LP) is usually required before consideration of other diagnoses
Thunderclap migraine	May be clinically indistinguishable from SAH; usually young patients with past history of migraine; often unilateral; patient may describe previous episodes
Benign coital cephalgia	A form of 'thunderclap' headache occurring during sexual intercourse (often at the point of orgasm); remember that SAH during sexual intercourse is also well recognised
Cervicogenic pain	Pain originating from cervical spine may radiate to the occiput; often marked local tenderness and pain increased by movements of head; may be history of preceding neck pain/injury/whiplash
Cranial venous sinus thrombosis	Usually more gradual onset but sudden in up to 10% of patients May be history suggestive of thrombophilia (previous deep vein thrombosis/pulmonary embolism, etc.) or risk factors for thrombosis (see p. 40, Table 17) Often papilloedema and raised CSF opening pressure on LP. CT brain will often be normal unless intravenous contrast given (radiologists will usually not do this unless there is a specific request to do so)
Pituitary apoplexy	Results from infarction of, or haemorrhage into pituitary gland Often associated with a pituitary adenoma Examination may reveal hypotension, ophthalmoplegia and/or bitemporal hemianopia but may be entirely normal Often overlooked on CT. MRI scanning required for diagnosis
Carotid artery dissection	Pain is often unilateral ('hemicranial') with associated Horner's syndrome/other unilateral neurological signs May be preceding history of neck injury/trauma CT and CSF often normal: CT or MR angiography is usually required
Trigeminal neuralgia	Sudden, 'shooting' pains over face rather than scalp/head; usually transient and unilateral

- Drowsiness, agitation or confusion
- Evidence of raised intracranial pressure – hypertension, bradycardia, papilloedema
- Subhyaloid haemorrhages on fundoscopy

What further investigation is required to confirm/refute the diagnosis of SAH?

Further investigation usually requires two tests:

- CT brain scan
- Lumbar puncture (LP)

It is usual practice to undertake a CT scan of the brain for all patients where there is clinical suspicion of SAH.

The scan may show:

- Direct evidence of SAH – blood in subarachnoid spaces and/or ventricles
- Evidence of hydrocephalus – due to obstruction of cerebrospinal fluid (CSF) flow by the haematoma

- Alternative pathology to account for headache – cerebral tumours, other forms of intracranial bleeding, etc.

Mrs Jenski is transferred to the CT scanner and undergoes a CT brain scan; this is reported as normal by the on-call neuroradiologist and Mrs Jenski returns to the ward. Her headache is now much better controlled and she has not vomited again. Neurological observations have been stable and Mrs Jenski remains afebrile. She is keen to go home and asks if the normal scan has 'excluded' a brain haemorrhage.

Does a normal CT brain scan exclude the diagnosis of SAH?

The simple answer is *no*.

- CT brain scanning is normal in up to 10% of patients following SAH

- The incidence of a false-negative scan rises with longer delays in scanning following onset of pain, and is also dependent on the experience of the interpreting radiologist
- Whenever the CT brain scan is normal, patients with suspected SAH should undergo lumbar puncture

Why undertake a lumbar puncture in suspected SAH?

The aim of LP in suspected SAH is to show evidence of blood in the cerebrospinal fluid, which is in communication with the subarachnoid space.

Evidence of recent bleeding may be indicated by:

- Microscopy showing red blood cells within the CSF
- Photochemical analysis showing breakdown products of haemoglobin (haem pigments) in the CSF

What should be considered before undertaking lumbar puncture?

- **Has the CT scan already shown the diagnosis?** Ensure an experienced radiologist has examined the images – if SAH (or an alternative diagnosis) is confirmed on CT, lumbar puncture is usually not required
- **Are there contraindications to lumbar puncture?** (see Table 48)
- **What was the time of onset of headache?** Lumbar puncture should be delayed for at least 12 h following the onset of headache to enable accurate interpretation of the haem pigment levels (see Table 49)
- **Is equipment available to measure CSF opening pressure?** Raised pressure may be an indication of an alternative diagnosis if fluid analysis is normal (e.g. cerebral venous sinus thrombosis – see Table 46)
- **Have you contacted the laboratory** to ensure that they are able to analyse the fluid on arrival? This is particularly

important if the procedure is being undertaken outside normal working hours. Delayed analysis may affect the accuracy of the results, requiring the procedure to be repeated

What cerebrospinal fluid samples should be obtained?

- 0.5 mL in fluoride oxalate (or EDTA) bottle for protein/glucose measurement (not usually abnormal in case of SAH but may suggest alternative diagnosis)
- Two samples of 1 mL each (labelled 2 and 3) sent to microbiology department for cell counting and culture
- One sample (minimum 1 mL) for spectrophotometric analysis: cover immediately to protect from light and send to biochemistry department

Mrs Jensi is observed closely on the ward, with hourly neuro obs until 12 h from the onset of the symptoms, at which time your registrar undertakes a lumbar puncture. This proves to be a difficult procedure. CSF is obtained at the third attempt, and is noted to be bloodstained. The CSF opening pressure is measured at 20 cmH₂O. Samples are sent off as above and the results are phoned back to the ward 1 h later as follows:

- red cell count – bottle 2, 975 rbc/mm³; bottle 3, 450 rbc/mm³
- total oxy/methaemoglobin absorbancy: 0.25 (normal range <0.023)
- bilirubin absorbancy: 0.076 (normal range <0.007)

How should these CSF results be interpreted?

- A guide to interpretation of CSF results is shown in Table 49
- There are large numbers of red blood cells in bottle 2

Table 48 Contraindications to lumbar puncture (LP)

Clinical/CT evidence of raised intracranial pressure	Risk of 'coning' when cerebrospinal fluid (CSF) is removed – reduction of CSF pressure below foramen magnum causes herniation of brainstem through this foramen, resulting in brain damage/death
Reduced conscious level/cerebral irritability	Coning is more likely in this situation even in the absence of CT abnormalities
Coagulopathy/thrombocytopenia	Where LP is required, platelet/clotting abnormalities should be corrected first

Table 49 Interpretation of cerebrospinal fluid (CSF) results in suspected subarachnoid haemorrhage (SAH)

	Time from bleed to appearance in CSF	Persistence in CSF	Comments
Red blood cells	Immediate	No	Absence of red cells from CSF generally excludes SAH within previous 48 h May appear in CSF following 'traumatic' lumbar puncture – red cell count will be lower in bottle 3 than in bottle 2 in this case 'Uniform' blood staining (same number of blood cells in bottle 2 and 3) is more suggestive of SAH
Oxy/methaemoglobin	Immediate	No	May appear in CSF following traumatic LP Absence of these pigments from CSF excludes SAH within preceding 48 h
Bilirubin	12 hours	Yes	Delayed appearance in CSF means that if LP is delayed for 12 hours from onset of symptoms, presence of bilirubin in CSF indicates bleed rather than traumatic LP May persist in CSF for 2–3 weeks following bleed – therefore useful when presentation to hospital is delayed

with fewer in bottle 3, which may be a result of the 'traumatic' LP; trauma could also explain the raised level of oxy/methaemoglobin

- However, a traumatic LP could not explain the raised level of bilirubin as this takes >12 h to appear in the CSF
- The raised bilirubin level is strongly suggestive of SAH as a cause for Mrs Jenski's symptoms

If subarachnoid haemorrhage is confirmed, what further management/treatments should be instituted?

- Ensure symptoms remain adequately controlled
- Contact on-call regional neurosurgical team
- Commence nimodipine 60 mg orally every 4 h*
- Continue regular (at least hourly) neurological observations
- Careful fluid balance – reduction in plasma volume may increase cerebral ischaemia: ensure at least 3 L of fluid daily
- Compression stockings as deep vein thrombosis (DVT) prophylaxis (avoid anticoagulants until the aneurysm has been treated)

- Prescribe regular laxative – analgesia-induced constipation is common

*Nimodipine is a calcium antagonist, which has been shown to improve outcome following SAH, probably as a result of reduced cerebral vasospasm. If the patient is unable to swallow, a nasogastric tube should be sited. Intravenous nimodipine can only be administered via a central venous cannula, and so is generally avoided.

The neurosurgical team have been contacted and are planning to arrange transfer to the neurosurgical unit within the next 24 h to arrange magnetic resonance angiography with a view to treatment of the causative aneurysm if possible. Mrs Jenski has been stable on the ward for the past 18 h, receiving oral nimodipine (60 mg 4 hrly) and codeine phosphate with paracetamol when required for pain control.

You are called back to the ward because Mrs Jenski's neurological state has deteriorated. The nurses report that her conscious level has dropped. The Glasgow Coma Score is 8/15. Mrs Jenski's blood pressure has risen to 150/100 with a pulse of 50 beats/min.

What is the likely explanation for the deterioration in conscious level following SAH?

Possible causes include:

- Rebleeding: a further SAH
- Obstructive hydrocephalus: usually due to compression of the fourth ventricle
- Cerebral vasospasm: usually occurs later
- Seizure: unwitnessed seizure may be followed by a 'post-ictal' phase
- Drug-induced, e.g. following strong opiate analgesia

Which immediate actions should be taken?

- Assess the patency of Mrs Jenski's airway
- Apply oxygen and connect to cardiac monitor
- Recheck conscious level and pupillary responses
- Contact on-call anaesthetic/ITU team
- Inform the neurosurgical team of Mrs Jenski's deterioration

- Check Mrs Jenski's drug chart to ensure no culprit drug has been administered
- Arrange urgent repeat CT brain scan: Mrs Jenski is likely to require intubation and anaesthetic supervision during this procedure as she is drowsy and may not have the protective reflexes to prevent aspiration if she vomits

Mrs Jenski is intubated and undergoes a CT brain scan, which now reveals massive subarachnoid haemorrhage, extending into the fourth ventricle and resulting in early hydrocephalus. The neurosurgical team agree to immediate transfer to the neurosurgical intensive care unit following CT. Following urgent insertion of a CSF shunt Mrs Jenski's conscious level improves, enabling extubation. Magnetic resonance angiography the following day reveals an anterior communicating artery berry aneurysm that is suitable for surgical 'clipping'. This is undertaken the following day. Following a prolonged period of neurological rehabilitation Mrs Jenski is discharged from hospital three months later.

CASE REVIEW

A 37-year-old woman has been admitted with a history of sudden onset of severe headache associated with vomiting. This is improved with analgesia and a CT brain scan is normal. However, a subsequent lumbar puncture reveals raised levels of bilirubin, suggestive of a recent subarachnoid haemorrhage, which is the likely source of the patient's symptoms. Mrs Annika Jenski is observed closely on the ward, during which a sudden deterioration in her condition occurs. Mrs Jenski is intubated by the on-call anaesthetist and a repeat CT scan reveals that she has developed obstructive hydrocephalus which requires insertion of a shunt to lower the intracranial pressure. Following this, Mrs Jenski undergoes surgical clipping of an aneurysm that is the likely cause of her bleed. Mrs Jenski makes a full recovery after a prolonged period of neurological rehabilitation.

Recognition of the suggestive symptoms of subarachnoid haemorrhage in this case ensured appropriate investigation. A normal CT brain scan does not exclude the diagnosis, as illustrated by this case. Had Mrs Jenski been allowed home

following the normal scan her subsequent deterioration could not have been identified and managed as quickly. As a result the outcome might have been very different.

It should be noted that, in most cases presenting in this way, CT and LP are both normal, enabling SAH to be excluded. As indicated in Table 47, the differential diagnosis is quite wide. Many of the conditions listed are benign and would not normally require hospital treatment, so the patient may be discharged once the symptoms improve. However, care should be taken to ensure that a serious cause other than SAH has also been excluded. Where possible the patient should be given a 'positive' diagnosis before discharge from hospital care. If this is not possible, a careful discussion is required with the patient prior to discharge, particularly if the symptoms are ongoing. The patient should be reassured that SAH has been excluded, but give clear instructions both to the patient and their GP regarding further action if the symptoms continue or worsen.

KEY POINTS

- Sudden severe headache is the hallmark of SAH.
- All patients presenting with suspected SAH should undergo a CT brain scan as soon as possible after admission.
- A normal CT brain scan does not exclude SAH.
- When the CT is normal a LP should be undertaken.
- LP should be delayed for >12 h following the onset of symptoms.
- Following confirmation of SAH, early discussion with a neurosurgical team is required.
- Consider alternative explanations for the headache when SAH has been excluded.

Further reading

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