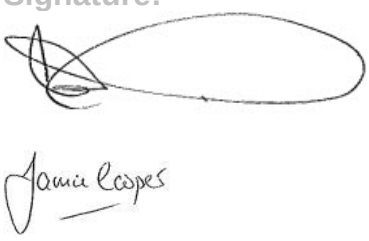
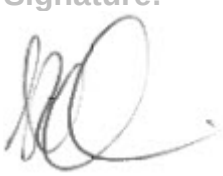
NHS Grampian

Treatment of Acute Non-traumatic Headache Protocol

Lead Author/Co-ordinator:	Reviewer:	Approver:
Callum Duncan, Consultant Neurologist and Jamie Cooper, Consultant in Emergency Medicine	Linda Gerrie, Consultant Neurologist	Steve Wilkinson, Consultant Physician
Signature: 	Signature: 	Signature: 
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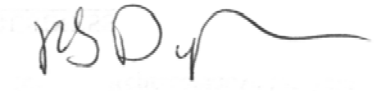
Signature: 

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Policy statement:
It is the responsibility of all staff to ensure that they are working to the most up to date and relevant clinical process documents.

Responsibilities for implementation:

Organisational:	Operational Management Team and Chief Executive
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CONTENTS

1. Need for a protocol

Acute headache is a common presentation to emergency departments and acute medical admission units.[1-4] Documentation is often inadequate[1,3,4] and patients are discharged without a specific diagnosis in up to 50% of cases.[3] The differential diagnosis in patients presenting with headache to emergency departments is broad. In the study by Locker et al[2] 60% of patients had a primary headache such as migraine and 40% of patients had a headache attributed to a secondary cause, with 13% of patients having a sinister cause for headache.

2. Red flags for secondary headache[5]

Warning features "red flags" indicate a higher probability of a patient having a sinister headache[2,3,5,6] and can be used to help identify patients that need to be admitted and investigated:

- New or changed headache aged >50
- New headache in a patient with a history of cancer or immunosupression
- Change in headache pattern or frequency
- Thunderclap onset
- New focal neurological symptoms
- New non-focal neurological symptoms
- New abnormal neurological examination
- Headache wakening the patient up
- Headache precipitated by exertion or valsalva
- Headache that changes with posture
- Neck stiffness or fever
- Jaw claudication or visual disturbance

3. Acute non-traumatic headache protocol

A checklist and protocol have been devised to aid the management of patients with acute non-traumatic headache. The protocol is primarily intended for use in patients presenting with headache to Accident and Emergency and the Acute Medical Initial Assessment Unit, but may also benefit doctors working in General Medicine, Neurology, Neurosurgery, Ophthalmology and the Infection Unit. It is intended to facilitate a thorough history and examination and appropriate investigation.

3.1 Initial assessment

The first priority in all patients who present acutely with non-traumatic headache is to ensure that there is not an immediately life threatening cause for their headache. If there is significant concern then the priority is to stabilise the patient in the resuscitation room whilst ascertaining the likely cause of their headache and arranging appropriate specialist review and investigation. In a poorly responsive patient a prompt and comprehensive history from a relative or other witness is vital. If the patient is stable then a comprehensive history and examination is required with the following considerations in mind:

3.2 Is there a metabolic or toxic cause?

Carbon Monoxide in particular, though other toxins too, present with headache. Often the diagnosis is not immediately apparent and the clinical presentation may be very vague. This step is put in, in order that the diagnosis is considered. Carbon Monoxide poisoning can be diagnosed on a blood gas and easily ruled in or out as a cause. The main problem is that the possibility may not be considered and hence the importance of this step.

3.3 Is there evidence of CNS infection?

Central nervous system infection should be considered in any patient presenting with headache and fever. Many of these patients will have headache associated with a systemic infection. There should, however, be a low threshold for considering CNS infection.

In bacterial meningitis[7] the headache is usually progressive and is associated with fever, neck stiffness and altered mental status. The headache onset can occasionally be sudden, mimicking Subarachnoid Haemorrhage. A non-blanching petechial rash should be looked for. Not all symptoms may be present in the initial stages and a high index of suspicion is required. Assessment and management of suspected bacterial meningitis and meningococcal septicaemia is detailed in the British Infection Society protocol which is easily accessible in A&E and in AMIA. Unless contraindicated a lumbar puncture should be performed without delay. A CT brain scan is required first **only** if there are associated seizures, papilloedema, focal neurological symptoms/signs or moderately to severely reduced conscious level (suspicion of focal lesion or raised intracranial pressure) or if the patient is immunocompromised. Isolated cranial nerve signs are not a contraindication to lumbar puncture. If there is going to be a significant delay before a lumbar puncture can be performed, then appropriate empirical antibiotics should be given.

There is usually a flu like prodrome in viral encephalitis[8], followed by progressive headache, fever and altered mental status. Seizures, focal symptoms/signs, neck stiffness and altered conscious level may be present. Any patient presenting with fever and a seizure or a seizure (or series of seizures) from which they do not recover should be investigated for encephalitis. A CT brain scan is more likely to be required before lumbar puncture, but if none of the contraindications listed above for bacterial meningitis are present then it is safe to proceed directly to lumbar puncture. The most common cause of viral encephalitis is Herpes Simplex. Acyclovir should be started immediately after lumbar puncture if viral encephalitis is suspected. If there is going to be a delay before a lumbar puncture is carried out, then it should be given immediately.

Patients with suspected meningitis should be referred to infectious diseases and patients with suspected encephalitis should be referred to infectious diseases or neurology.

3.4 Is there evidence of a focal lesion or raised intracranial pressure?

An underlying focal brain abnormality should be considered in any patient presenting with headache associated with new focal symptoms.[5] Sudden onset headache associated with new neurological symptoms and signs usually indicates a vascular event (haemorrhage or ischaemic stroke). Progressive headache associated with progressive signs may be caused by a number of pathologies, including primary or secondary brain tumours, CNS infection (encephalitis, abscess), cerebral venous sinus thrombosis and hydrocephalus. If there is a depressed conscious level then an urgent CT brain scan should arranged and an urgent neurosurgery or neurology opinion obtained. This includes patients presenting out of hours. Patients with a normal conscious level may need to be admitted for urgent investigation. An early neurology or neurosurgery opinion should be sought in these patients depending on the level of concern.

Red flags for raised intracranial pressure include: headache wakening the patient from sleep, headache worse lying flat, rapidly increasing headache frequency and severity, headache precipitated by valsalva (coughing, sneezing, laughing, straining, lifting), new focal symptoms and new abnormal neurological examination.

A normal CT brain scan does not exclude raised intracranial pressure and if the CT brain scan is normal a lumbar puncture should be considered looking for raised CSF pressure. A CSF opening pressure > 25cm CSF is abnormal. Visual obscurations (transient altered or reduced vision on change in posture) and pulsatile tinnitus are useful symptoms, although their absence does not exclude intracranial hypertension. Idiopathic intracranial hypertension is most commonly seen in overweight patients. Other causes to consider are drug induced (tetracyclines, retinoids), pregnancy, venous sinus thrombosis and any process that impedes CSF drainage (infection, inflammation or malignancy). Raised CSF pressure can also be seen after subarachnoid haemorrhage due to poor CSF drainage because of blood. Usually hydrocephalus is seen on CT, but the absence of hydrocephalus does not exclude raised CSF pressure and the appearances on the CT brain scan can be similar to that seen in intracranial hypertension.

3.5 Is this a sudden onset headache?

Approximately 1 in 10 patients with a sudden severe headache will have a subarachnoid haemorrhage (SAH).[9] The headache usually peaks instantaneously, although may progress over a few minutes (<5 minutes) in some.[10] It is not known how long a headache due to subarachnoid haemorrhage should be. Expert opinion suggests that it should last at least an hour or 2. There are no reliable features that distinguish SAH from benign thunderclap headache[9] and all patients with sudden severe headache that presents over a few minutes and lasts at least 1 hour should be admitted for investigation of SAH.[11] Despite improvement in investigation and treatment there is still a 50% mortality rate for SAH. There is also a high early re-bleed rate (4-6% in the first 24 hours, 40% in the first month). Investigation of potential SAH should therefore be done without delay.

All patients should have a CT brain scan as soon as possible (blood rapidly degrades, up to 7% of CT scans will be negative at 24 hours). If the patient presents overnight, has a normal conscious level and no focal signs then it is reasonable for the scan to wait till the next morning. If the patient presents during the day or in the early evening then ideally the scan should be on the same day. There should be no delay if there are focal signs or a reduced conscious level.

If the CT scan is negative then a lumbar puncture should be performed. This should be delayed for 12 hours after headache onset to allow bilirubin to form, unless there is a concern about bacterial meningitis. If subarachnoid haemorrhage is considered then the lumbar puncture should be performed by someone experienced in lumbar punctures to avoid a traumatic tap. If there is a traumatic tap then the first tube should be discarded and the samples taken to the lab without delay and centrifuged. The 3 bottle rule is unreliable. An opening pressure should always be measured. CSF should be sent for cell count, protein, glucose and xanthochromia and cytology. The biochemistry sample should be protected from light to ensure bilirubin does not degrade in transit.

A normal CT brain scan and normal lumbar puncture within 2 weeks of the headache onset excludes SAH. After 2 weeks this is unreliable and an angiogram (initially a CTA) is required. If SAH is confirmed Nimodipine 60mg (2am, 6am, 10am, 2pm, 6pm, 10pm) should be started, a CTA requested (if this has not already been done) and a neurosurgical opinion obtained. If the headache onset is more than 2 weeks ago, neurology or neurosurgery advice should be sought prior to investigation.

Some patients present after several thunderclap headaches. There is no reliable number of thunderclap headaches a patient can have before SAH is unlikely and all patients are likely to require investigation as detailed above to exclude SAH. A neurology opinion should be obtained in such patients.

Most patients with sudden severe headache, negative CT brain scan and negative lumbar puncture have benign headache. Investigation can often stop at that point. There is however a wide differential diagnosis and a careful history should be obtained in all patients looking for other causes of thunderclap headache. Further investigation including MRI and angiography may be required on a case by case basis.

Differential Diagnosis of Thunderclap Headache

Primary Headaches	Secondary Headaches
Primary Thunderclap Headache	Subarachnoid Haemorrhage
Migraine	Reversible Cerebral Vasoconstriction Syndrome
Cluster Headache	Intracerebral, Intraventricular, Subdural or Extradural Haemorrhage
Primary Exertional Headache	Carotid or Vertebral Artery Dissection
Primary Orgasmic Headache	Pituitary Apoplexy
	Cerebral Venous Sinus Thrombosis
	Meningitis
	Acute Hydrocephalus e.g. colloid cyst
	Acute severe hypertension e.g. Phaeochromocytoma
	Spontaneous Intracranial Hypotension

3.6 Are there any other red flags?

Older patients are more likely to have a sinister cause for their headache. Subarachnoid haemorrhage, strokes, cancer, angle closure glaucoma and giant cell arteritis are all more common in older patients and there should be a lower threshold for investigation in older patients. A history of immune suppression increases the risk of CNS infection and metastases should be considered in patients with a PMH of cancer presenting with new headache.

All patients over the age of 50 with new persistent headache or change in headache should have an ESR to screen for giant cell arteritis. A CRP and platelet count may also be elevated. Scalp tenderness, jaw claudication (jaw and temple pain that develops during chewing and resolves with rest), visual disturbance and prominent beaded temporal arteries are helpful, but there absence does not exclude the diagnosis.[12] If giant cell arteritis is considered likely then high dose prednisolone should be started immediately and a temporal artery biopsy arranged via Ophthalmology. Ideally a temporal artery biopsy should be performed within 2 weeks of starting prednisolone. An ophthalmological or neurology opinion should be sought depending on the patient's symptoms.

Headache that develops once the patient is upright suggests low CSF pressure.[13] Patients are usually pain free on awakening and develop headache shortly after assuming an upright posture, although in some patients the headache may come on slowly (2nd half day headache). The headache improves or may resolve on lying down. This needs to be differentiated from headache aggravated by movement which is most likely to represent migraine. Most low pressure headache is due to a diagnostic lumbar puncture, but may be spontaneous. 15% of spontaneous low pressure headaches have a thunderclap onset. Post LP headaches usually settle by 7 days with conservative treatment (fluids, bed rest, oral caffeine). If not settling, an epidural blood patch may be required. If spontaneous intracranial hypotension is considered, a neurology opinion should be sought. MRI is the investigation of choice to demonstrate the typical features seen with low CSF pressure.

3.7 Is this a recognisable primary headache syndrome?

Primary headache disorders such as migraine are not serious, but can be very disabling. The majority of headache presenting to an emergency department (60%) is primary.[2] A concerning cause is unlikely in longstanding episodic headache without red flag features.[5] Incidental findings (imaging findings that are not due to the headache under investigation) are common with both MRI[14] and to a lesser extent with CT. They may cause anxiety and lead to unnecessary investigation.

Tension type headache is a mild featureless band like headache. It is common in the general population but rarely presents to doctors and is frequently over-diagnosed. In a prospective multicentre general practice study most patients diagnosed with tension type headache had the diagnosis changed to migraine on the basis of a longitudinal diary.[15]

The majority of patients with primary headache presenting to A&E are likely to have migraine. Migraine headache can be unilateral or bilateral. It typically escalates over minutes to hours, but can occasionally be thunderclap. The most useful features in making a diagnosis are associated nausea, sensitivity to light (photophobia) and sound (phonophobia), and aggravation by routine physical activity. Aura is present in a third of patients. It can involve vision, sensation and speech, and has both positive (e.g. zig zags in visual field) and negative (e.g. numbness) features. It typically slowly evolves, distinguishing it from a TIA.

In a proportion of migraineurs their headaches transform from episodic to chronic migraine. A history of a gradual increase in headache frequency over weeks, months or years to become daily headache with a mixture of migraine and tension type headache is typical. This is frequently associated with medication overuse (analgesics, opioids or triptans used more than 10 days per month).

Cluster headache is the most common of a group of headache disorders known as Trigeminal Autonomic Cephalalgias. These are brief, excruciatingly severe, side locked headaches associated with restlessness and ipsilateral autonomic features.

If a patient presents with a severe headache typical of their usual primary headache syndrome and no red flag features, they can usually be discharged with treatment and reassurance. Patients with frequent headache or with a Trigeminal Autonomic Cephalalgia may benefit from a Headache Clinic referral. If there is immediate concern, a neurology opinion should be obtained.

References

1. Locker T, Mason S, Rigby A. Headache management – Are we doing enough? An observational study of patients presenting with headache to the emergency department. Emerg Med J. 2004;21:327-32.
2. Locker TE, Thompson C, Rylance J, Mason SM. The utility of clinical features in patients presenting with nontraumatic headache: An investigation of adult patients attending an emergency department. Headache 2006;46:954-61.
3. Breen DP, Duncan CW, Pope AE, Gray AJ, Al-Shahi Salman R. Emergency department evaluation of sudden severe headache. Q J Med. 2008;101:435-43.
4. Hewett R, Counsell C. Documentation of cerebrospinal fluid opening pressure and other important aspects of lumbar puncture in acute headache. Int J Clin Pract. 2010;64:930-35.
5. Scottish Intercollegiate Guidelines Network (SIGN). Diagnosis and management of headache in adults. 2008. Guideline No. 107:www.sign.ac.uk.
6. Kernick DP, Ahmed F, Bahra A, Dowson A, Elrington G, Fontebasso M, et al. Imaging patients with suspected brain tumour: guidance for primary care. Br J Gen Pract. 2008;58:880-85.
7. Schut ES, de Gans J, van de Beek D. Community-aquired bacterial meningitis in adults. Practical Neurology. 2008;8:8-23.
8. Solomon T, Hart IJ, Beeching NJ. Viral encephalitis: a clinicians guide. Practical Neurology. 2007;7:288-305.
9. Landtblom AM, Fridriksson S, Boivie J, Hillman J, Johansson J, Johansson I. Sudden onset headache: A prospective study of features, incidence and causes. Cephalalgia. 2002;22:354-60.
10. Linn FHH, Rinkel GJE, Algra A, van Gijn J. Headache characteristics in subarachnoid haemorrhage and benign thunderclap headache. J Neurol Neurosurg Psychiatry. 1998;65:791-3.
11. Al-Shahi R, White PM, Davenport RJ, Lindsay KW. Subarachnoid Haemorrhage. BMJ. 2006;333:235-40.
12. Smetana GW, Shmerling RH. Does this patient have temporal arteritis? JAMA. 2002;287:92-101
13. Schievink WI. Missdiagnosis of spontaneous intracranial hypotension. Arch Neurol. 2003;60:1713-8.
14. Morris Z, Whitely WN, Longstreth Jr WT, Weber F, Lee YC, Tsushima Y, et al. Incidental findings on brain magnetic resonance imaging: systematic review and meta-analysis. BMJ. 2009;339:b3106.
15. Tepper SJ, Dahlof CG, Dowson A, Newman L, Mansbach H, Jones M, et al. Prevalence and diagnosis of migraine in patients consulting their physician with a complaint of headache: data from the Landmark Study. Headache 2004;44:856-64.

Appendix 1

Acute Non-Traumatic Headache – Clinical Pathway

Patient Details

Sex: Male ☐ Female ☐

Place of presentation: A&E ☐ AMIA ☐ Other ☐

Date of presentation: _____

Date of headache onset: _____

Time of headache onset: _____

History

Onset: sudden (instantaneous – few minutes) ☐ or gradual ☐ **Time to peak**: _____

Duration of headache: _____ Headache free ☐ or headache persisting ☐

Periodicity : episodic ☐ persistent ☐ **Severity (1-10)**: _____

Focal symptoms: Yes ☐ _____ No ☐ **Non-focal symptoms**: Yes ☐ _____ No ☐

Medication use (including over the counter analgesics) _____

NB medication use on more than 10 days per month may cause medication overuse headache

Past headache history:

Is there a pre-existing primary headache syndrome? Yes ☐ _____ No ☐

Is this headache the same or different to the patient's usual headache? Same ☐ Different ☐

Has there been a recent change in the headache frequency or type? Yes ☐ No ☐

Examination

Temp: _____ Pulse: _____ BP: _____ Rash: _____ Temporal arteries: _____

GCS: _____ Fundoscopy: _____ Neurological examination: Normal ☐ Abnormal ☐

General medical examination as prompted by history: ☐

Red flags?

New onset or change in headache in patient aged >50 ☐, with immunosuppression or HIV ☐, with cancer ☐

Change in headache frequency, characteristics or associated symptoms ☐

Fever ☐, rash ☐, neck stiffness ☐

Thunderclap: sudden onset (instantaneous to a few minutes) ☐

New onset seizures ☐

New focal neurological symptoms (sudden or gradual onset, aura <5mins or >60mins) ☐

New non-focal neurological symptoms (eg cognitive disturbance) ☐

Headache initiated by exertion or valsalva (coughing, laughing, straining, stooping) ☐

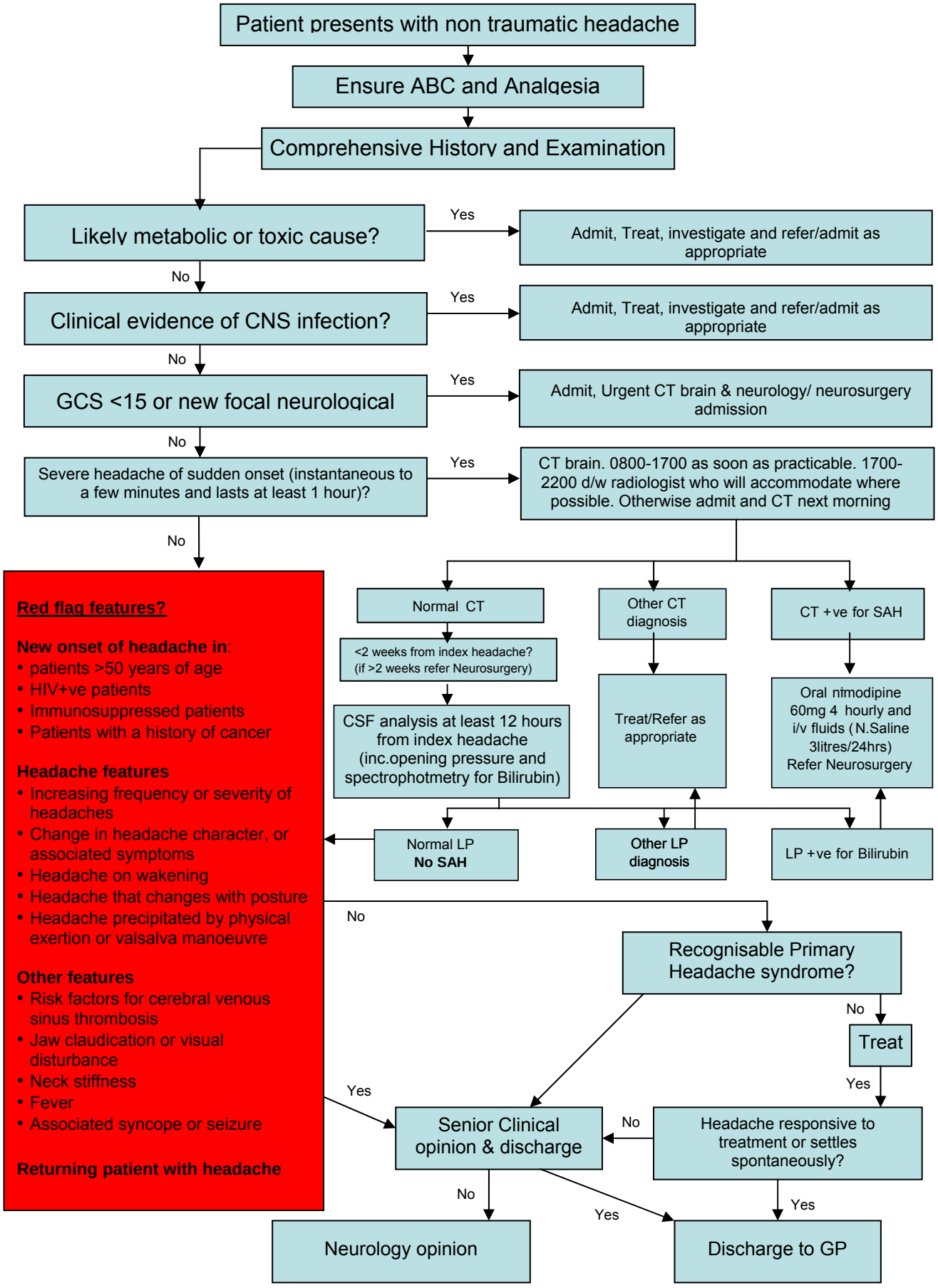
Headache wakening the patient up ☐

New headache in a patient with risk factors for venous thrombosis ☐

Headache brought on or aggravated by sitting or standing ☐

Jaw claudication or visual disturbance ☐

New abnormality on neurological examination ☐


Patient presents with non traumatic headache
Ensure ABC and Analgesia
Comprehensive History and Examination
Likely metabolic or toxic cause?
Yes
Admit, Treat, investigate and refer/admit as appropriate
No
Clinical evidence of CNS infection?
Yes
Admit, Treat, investigate and refer/admit as appropriate
No
GCS <15 or new focal neurological
Yes
Admit, Urgent CT brain & neurology/ neurosurgery admission
No
Severe headache of sudden onset (instantaneous to a few minutes and lasts at least 1 hour)?
Yes
CT brain. 0800-1700 as soon as practicable. 1700-2200 d/w radiologist who will accommodate where possible. Otherwise admit and CT next morning
No
Red flag features?
New onset of headache in:
• patients >50 years of age
• HIV+ve patients
• Immunosuppressed patients
• Patients with a history of cancer
Headache features
• Increasing frequency or severity of headaches
• Change in headache character, or associated symptoms
• Headache on wakening
• Headache that changes with posture
• Headache precipitated by physical exertion or valsalva manoeuvre
Other features
• Risk factors for cerebral venous sinus thrombosis
• Jaw claudication or visual disturbance
• Neck stiffness
• Fever
• Associated syncope or seizure
Returning patient with headache
Normal CT
Other CT diagnosis
CT +ve for SAH
<2 weeks from index headache? (if >2 weeks refer Neurosurgery)
CSF analysis at least 12 hours from index headache (inc.opening pressure and spectrophotmetry for Bilirubin)
Treat/Refer as appropriate
Oral nimodipine 60mg 4 hourly and i/v fluids (N.Saline 3litres/24hrs) Refer Neurosurgery
Normal LP No SAH
Other LP diagnosis
LP +ve for Bilirubin
No
Recognisable Primary Headache syndrome?
No
Treat
Yes
Yes
Senior Clinical opinion & discharge
No
Headache responsive to treatment or settles spontaneously?
No
Yes
Yes
Neurology opinion
Discharge to GP