BRITISH SOCIETY OF NEURORADIOLOGISTS STANDARDS SUBCOMMITTEE

GUIDELINES FOR NEUROIMAGING IN HEADACHE

Dr Catriona Good
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1. INTRODUCTION

This neuroimaging guideline should be considered in combination with clinical headache guidelines (for example., NICE guidelines, Scottish Intercollegiate Guidelines network (SIGN) National Clinical Guideline on the Diagnosis and Management of Headaches in Adults (2008)[1] and Guideline for primary healthcare management of headaches., 2015- referred to within Appendix 7.2).

1.1. The Need for a Guideline

Headache is one of the most common neurological conditions with a lifetime prevalence of over 90% of the United Kingdom (UK) population[1,2,3,4,5] and many patients with headache are investigated with imaging.

Headache disorders are generally classified as primary or secondary, and subclassified into specific headache types. Primary headache disorders are not associated with an underlying pathology and include migraine, tension-type, and cluster headache. Secondary headache disorders are attributed to an underlying pathological condition and include any head pain of infectious, neoplastic, vascular, or drug-induced origin.[6]

Migraine is the most common severe form of primary headache affecting about six million people in the UK in the age range 16-65, and can cause significant disability.[7] The World Health Organisation (WHO) ranks migraine in its top 20 disabling conditions for women aged 15 to 44.[8] Migraine costs the UK almost £2 billion a year in direct and indirect costs[9] with over 100,000 people absent from work or school because of migraine every working day.[10]

Tension-type headache affects over 40% of the population at any one time. Although less of a burden to the individual sufferer than migraine, its higher prevalence results in a greater

burden to society.[11] Chronic headache, defined as headache on 15 or more days per month, affects approximately 3% of people worldwide.[11]

The diagnosis of headache can be challenging and both doctors and patients worry about serious rare causes of headaches such as brain tumours[3,12.] General practitioners (GPs) are often uncertain about when to refer patients for diagnostic tests and secondary care.[3] Most primary headaches are managed within primary care and imaging investigations are usually not required.

1.2. Remit of the Guideline

This guideline provides recommendations based on evidence for best practice in the imaging diagnosis of headache in adults and is an adjunct to the SIGN National Clinical Guideline on the Diagnosis and Management of Headaches in Adults (2008)[1].

This guideline considers primary and secondary headaches and focuses on the types of headache that require further investigation with imaging. "Red flags" (Appendix 7.2.1) for secondary headache are highlighted and tailored scanning protocols are provided.

This guideline will be of interest to healthcare professionals in primary and secondary care, including Neuroradiologists, general radiologists, neurologists, hospital physicians, general practitioners, community pharmacists, opticians and dental practitioners, and patients with headache.

1.3. Definitions

The guideline uses the definitions given in the International Headache Society International Classification of Headache Disorders, 3rd edition (see Appendix 7.1)[32].

Please see also the Scottish Intercollegiate Guidelines network (SIGN) National Clinical Guideline on the Diagnosis and Management of Headaches in Adults (2008).

1.4. Statement of intent

This guideline is not intended to be construed or to serve as a standard of care. Standards of care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge and technology advance and patterns of care evolve. Adherence to guideline recommendations will not ensure a successful diagnosis and/or outcome in every case, nor should they be construed as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgement must be made by the appropriate healthcare professional(s) responsible for clinical decisions regarding investigations .

It is advised, however, that significant departures from the national guideline or any local guidelines derived from it should be fully documented in the patient's case notes at the time the relevant decision is taken.

1.5. Key to evidence statements and grades of recommendations

1.5.1. Levels of evidence

1++	High quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias.
1+	Well conducted meta-analyses, systematic reviews, or RCTs with a low risk of bias.
1-	Meta-analyses, systematic reviews, or RCTs with a high risk of bias.
2++	High quality systematic reviews of case control or cohort studies High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal

2+	Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
2-	Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal.
3	Non-analytic studies, e.g. case reports, case series.
4	Expert opinion.

1.5.2. Grades of recommendation

Note: The grade of recommendation relates to the strength of the evidence on which the recommendation is based. It does not reflect the clinical importance of the recommendation.

A	At least one meta-analysis, systematic review, or RCT rated as 1++, and directly applicable to the target population; or A body of evidence consisting principally of studies rated as 1+, directly applicable to the target population, and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2++, directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+
C	A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++
D	Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+

1.5.3. Good practice points

☑	Recommended best practice based on the clinical /radiological experience of the guideline development group.

2. KEY RECOMMENDATIONS FOR NEUROIMAGING

D ☑	Neuroimaging is not indicated in patients with a clear history of uncomplicated migraine without red flag features for potential secondary headache and a normal neurological examination.
D ☑	Neuroimaging, sinus or cervical spine x-ray scans are not recommended for the routine assessment of patients with headache: history and physical and neurologic examination findings are usually sufficient to make a diagnosis of migraine or tension-type headache.
D ☑	In the acute setting of suspected subarachnoid haemorrhage, focal neurology or raised intracranial pressure, CT is the first line investigation, according to NICE guidelines. Further imaging with CTA, CTV, MR or MRA may be required depending upon the CT findings and the clinical presentation.
D ☑	In the non-acute setting, patients with primary or secondary headaches who are considered by clinicians to require further imaging investigation, should have MRI as the first line, if available.
D ☑	Specific headache types such as low pressure headache, positional or cough headache with possible CSF flow obstruction (Chiari malformation or colloid cyst), meningitic features, vasculitic features or cranial nerve neuropathies require specialist MRI protocols and should ideally be reviewed by a Neuroradiologist.
D ☑	Clinicians requesting neuroimaging should be aware that both MRI and CT can identify incidental neurological abnormalities which may result in patient anxiety as well as practical and ethical dilemmas with regard to management.

3. HEADACHE TYPES

This is an abbreviated list pertinent to imaging. Please see Appendix 6.1 for a full sub-classification of the each of the major types.

A. The Primary Headaches

1. Migraine

- 1.1 Migraine without aura
- 1.2 Migraine with aura
  - 1.2.2 Migraine with brainstem aura
  - 1.2.3 Hemiplegic migraine
    - 1.2.3.1 Familial hemiplegic migraine (FHM)
- 1.2.4 Retinal migraine
- 1.3 Chronic migraine
- 1.4 Complications of migraine
  - 1.4.1 Status migrainosus
  - 1.4.2 Persistent aura without infarction
  - 1.4.3 Migrainous infarction
  - 1.4.4 Migraine aura-triggered seizure
- 1.6 Episodic syndromes that may be associated with migraine
  - 1.6.1 Recurrent gastrointestinal disturbance
  - 1.6.2 Benign paroxysmal vertigo
  - 1.6.3 Benign paroxysmal torticollis

2. Tension-type headache

- 2.1 Infrequent episodic tension-type headache
- 2.2 Frequent episodic tension-type headache
- 2.3 Chronic tension-type headache

3. Trigeminal autonomic cephalalgias

- 3.1 Cluster headache
- 3.2 Paroxysmal hemicrania
- 3.3 Short-lasting unilateral neuralgiform headache attacks
  - 3.3.1 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)

3.3.2 Short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)

3.4 Hemicrania continua

3.5 Probable trigeminal autonomic cephalalgia

4. Other primary headache disorders

4.1 Primary cough headache

4.2 Primary exercise headache

4.3 Primary headache associated with sexual activity

4.4 Primary thunderclap headache

4.5 Cold-stimulus headache

4.6 External-pressure headache

4.7 Primary stabbing headache

4.8 Nummular headache

4.9 Hypnic headache

4.10 New daily persistent headache (NDPH)

B. The Secondary Headaches

1. Headache attributed to trauma or injury to the head and/or neck

2. Headache attributed to cranial and/or cervical vascular disorder

2.1 Headache attributed to cerebral ischaemic event

2.2 Headache attributed to non-traumatic intracranial haemorrhage

2.3 Headache attributed to unruptured vascular malformation

2.3.1 Headache attributed to unruptured saccular aneurysm

2.3.2 Headache attributed to arteriovenous malformation (AVM)

2.3.3 Headache attributed to dural arteriovenous fistula (DAVF)

2.3.4 Headache attributed to cavernous angioma

2.3.5 Headache attributed to encephalotrigeminal or leptomeningeal angiomatosis (Sturge Weber syndrome)

2.4 Headache attributed to arteritis

2.4.1 Headache attributed to giant cell arteritis (GCA)

2.5 Headache attributed to cervical carotid or vertebral artery disorder

2.5.1 Headache or facial or neck pain attributed to cervical carotid or vertebral artery dissection

2.6 Headache attributed to cranial venous disorder

2.6.1 Headache attributed to cerebral venous thrombosis (CVT)

2.7 Headache attributable to other intracranial arterial disorder

2.7.1 Headache attributed to reversible cerebral vasoconstriction syndrome (RCVS)

2.7.2 Headache attributed to intracranial artery dissection.

2.8 Headache and/or migraine-like aura attributed to chronic intracranial vasculopathy

2.8.1 Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL)

2.8.2 Headache attributed to mitochondrial encephalopathy, lactic acidosis and stroke-like episodes (MELAS)

6.8.3 Headache attributed to Moyamoya angiopathy (MMA)

6.8.4 Migraine-like aura attributed to cerebral amyloid angiopathy (CAA)

6.8.5 Headache attributed to syndrome of retinal vasculopathy with cerebral leukoencephalopathy and systemic manifestations (RVCLSM)

2.9 Headache attributed to pituitary apoplexy

3 Headache attributed to non-vascular intracranial disorder

3.1 Headache attributed to increased cerebrospinal fluid (CSF) pressure

3.1.1 Headache attributed to idiopathic intracranial hypertension (IIH)

3.1.2. Headache attributed to intracranial hypertension secondary to hydrocephalus

3.2. Headache attributed to low cerebrospinal fluid (CSF) pressure

3.2.1 Post-dural puncture headache

3.2.2 Cerebrospinal fluid (CSF) leak headache

3.2.3 Headache attributed to spontaneous intracranial hypotension

3.3 Headache attributed to non-infectious inflammatory intracranial disease

3.3.1 Headache attributed to neurosarcoidosis and Lyme disease

3.3.2 Headache attributed to aseptic (non-infectious) meningitis

3.3.3 Headache attributed to other non-infectious inflammatory intracranial disease

3.3.4 Headache attributed to lymphocytic hypophysitis

3.3.5 Syndrome of transient headache and neurological deficits with cerebrospinal fluid lymphocytosis (HaNDL)

3.4 Headache attributed to intracranial neoplasia

3.4.1 Headache attributed to intracranial neoplasm

3.4.1.1 Headache attributed to colloid cyst of the third ventricle

3.4.2 Headache attributed to carcinomatous meningitis

3.4.3 Headache attributed to hypothalamic or pituitary hyper- or hyposecretion

3.5 Headache attributed to intrathecal injection

3.6 Headache attributed to epileptic seizure

3.7 Headache attributed to Chiari malformation type I (CM1)

3.8 Headache attributed to other non-vascular intracranial disorder

4 Headache attributed to a substance or its withdrawal

5.1. Medication overuse headache

5. Headache attributed to infection

6. Headache attributed to disorder of homoeostasis

7. Headache or facial pain attributed to disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cervical structure

8. Headache attributed to psychiatric disorder

9. Painful lesions of the cranial nerves and other facial pain

9.1 Trigeminal neuralgia
9.2. Optic neuritis
9.3 Facial neuralgia
9.3.1. Post herpetic
9.4. Glossopharyngeal neuralgia
9.4. Occipital neuralgia
9.5 Other

4. NEUROIMAGING FOR HEADACHES

4.1. When is neuroimaging required?	
In the acute setting when a patient presents with headache with red flag features (see Appendix 6.2.2) or fulfils NICE guidelines (Appendix 6.2.1), urgent CT imaging is the first line imaging investigation. Further imaging with MRI and/or vascular imaging may be required (see paragraphs 4.5 and 4.6)	2+
Investigation should be avoided in principle if it does not lead to a change in management or it is unlikely to reveal a relevant abnormality. Occasionally, neuroimaging may be required on an individual basis if a patient is disabled by fear of serious pathology.[17,25]	4

The vast majority of primary headaches do not require neuroimaging. In a prospective study where patients with headache for more than four weeks underwent neuroimaging (CT, MRI or both), significant intracranial abnormalities were found in 0.4% of patients with migraine, 0.8% of patients with tension-type headache, and in 5% (one out of 20 patients) of those with cluster headache. In a subgroup of 188 patients without clearly defined headache type, significant intracranial abnormality was found in 3.7%.[18]	2+
A meta-analysis of neuroimaging studies estimated a 0.2% prevalence of significant intracranial abnormalities in patients with migraine and a normal neurological examination.12 In a retrospective review of 402 patients with chronic headache without neurological symptoms or signs, relevant abnormalities on MRI were found in 0.6% of patients with migraine, 1.4% of patients with tension-type headache and 14.1% of patients with "atypical headache".[19] In another retrospective review MRI revealed relevant intracranial abnormalities in 0.7% of patients with chronic or recurrent headache and a normal neurological examination.[20]	4 3
Neuroimaging in patients with headache and an abnormal neurological examination is significantly more likely to reveal an underlying cause.[13,18,,21,22] Further investigation is required to exclude secondary aetiology when headache is accompanied by red flags (see Appendix 7.3).	4
4.2. Incidental abnormalities	
Both MRI and CT can identify neurological abnormalities incidental to the patient's presenting complaint and which may result in heightened patient anxiety and clinician uncertainty.[23,24] Cranial MRI in 2,536 healthy young males revealed incidental abnormalities in 6.55%.[23,24] A prospective cohort study of 2,000 volunteers (mean age, 63.3 years; age range 45-96 years) who received brain MRI revealed incidental abnormalities in 13.5%.[23]	3 2+
4.3. Patient reassurance	
A randomised controlled trial of 150 patients with chronic daily headache in a specialist clinic found that patients who received MRI had a decrease in anxiety levels at three months, but that the reduction in anxiety was not maintained at one year. Patients with high scores on the hospital anxiety and depression scale	1+

who did not receive a scan had significantly higher health service costs overall due to a greater use of healthcare resources such as psychiatric and psychology services than comparable patients who received a scan.[25]	
4.4. CT versus MRI	
CT is readily available and is often used as the first line, particularly in the acute setting for suspected haemorrhage or raised intracranial pressure or mass effect, according to NICE guidelines. In ruling out secondary causes of headache MRI is more sensitive than CT in identifying white matter lesions and developmental venous anomalies.[13] The European Federation of Neurological Societies guidelines suggest that MRI is the imaging modality of choice because of this greater sensitivity.[27] The US headache consortium concluded that MRI may be more sensitive than CT in identifying clinically insignificant abnormalities, but not more sensitive in identifying clinically significant pathology relevant to the cause of the headache.[26] Considering the detection rate for significant intracranial abnormalities using CT and MRI. In a cohort of 1876 persons with a non- acute headache defined as any type of headache experienced for at least 4 weeks, the rate of detection was 19/1432 (1.3%) using CT and 4/444 (0.9%) using MRI. Of 119 normal CT scans 2 (1.7%) had significant intracranial abnormality on MRI. [18]	3

4.5. Acute Thunderclap headache

- Thunderclap headache is a severe headache that peaks within 60 seconds of onset.
- Neurovascular disorders often present with thunderclap headache.
- Infectious disorders, intracranial hypertension, and hypotension syndromes occasionally present with thunderclap headache.
- Subarachnoid haemorrhage is the most common cause; diagnosis is based on plain brain computed tomography (CT) and, if normal, on lumbar puncture.

- Suspect reversible cerebral vasoconstriction syndrome when thunderclap headaches recur over a few days.
- Cervical artery dissection, cerebral venous thrombosis, reversible cerebral vasoconstriction syndrome pituitary apoplexy and intracranial hypotension may present with isolated thunderclap headache and normal physical examination, CT, and cerebrospinal fluid.

CT is widely available in the acute and outpatient setting. CT is the first line imaging modality of choice for detecting haemorrhage and should be performed as soon as possible and preferably within 12 hours of onset. When subarachnoid haemorrhage (SAH) is suspected, CT brain scan should be carried out as soon as possible to maximise sensitivity. Sensitivity of CT for subarachnoid haemorrhage is 98% at 12 hours dropping to 93% by 24 hours.[28]

Despite the high sensitivity of CT, considering uncertainties related to timing of ictus and clinical expertise in the emergency setting, a normal CT brain scan is insufficient to rule out SAH and a lumbar puncture is required in patients with normal CT scans.

If negative results are obtained from both brain CT and lumbar puncture with cerebrospinal fluid analysis within two weeks of onset of thunderclap headache, then SAH can be excluded from diagnosis.[28,29,30]

When CT and cerebrospinal fluid are normal, other investigations are needed, including cervical and cerebral vascular imaging and brain magnetic resonance imaging [33]

Sudden severe headaches precipitated by sexual activity can be diagnosed as primary if they cannot be attributed to another disorder according to ICHD-II classification.[17]

On first onset of this headache it is essential to exclude SAH with CT and/or LP and arterial dissection with CTA or MRI plus MRA[34.] Some centres advocate MRI of the spine to exclude

spinal vascular lesions, although there is no robust evidence for this and the yield is extremely low.

4.6. Indications for Magnetic Resonance Imaging (MRI)

MRI provides more comprehensive imaging assessment of the brain in multiple plains, but is less available than CT in a timely fashion.

4.6.1. Cluster headache, paroxysmal hemicrania or SUNCT

Expert opinion suggests that MRI should be considered in patients with cluster headache, paroxysmal hemicrania or Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT), in order to exclude the wide variety of secondary causes.[33,34,35,36]

In a review of 31 cases in which a trigeminal autonomic cephalalgias (TAC) or TAC like syndrome was associated with a structural lesion and the treatment of the lesion resulted in significant clinical improvement, 11 out of 31 had a pituitary adenoma. Only 10 out of the 31 cases had atypical presenting features.78 In a prospective study of 43 patients with SUNCT and nine patients with short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA), cranial imaging was carried out in 36 with SUNCT and eight with SUNA. Twelve patients (11 with SUNCT, 1 with SUNA) of the 44 who received cranial imaging had significant intracranial abnormalities.[35]

4.6.2. Headache which is precipitated, rather than aggravated, by cough

In a retrospective study in patients with cough-induced headaches (n=30), exertional headaches (n=28) and sexual headaches (n=14); 17 out of 30 patients with cough-induced

headache had Chiari type 1 malformation; 10 out of 28 patients with headache induced by exertion had SAH, 1 had sinusitis and 1 had brain metastases; and 1 out of 13 patients with explosive orgasmic headache had SAH.[33,36]

Primary cough headache (valsalva manoeuvre headache) which is precipitated rather than aggravated by coughing, laughing or straining may be diagnosed only after structural lesions are excluded by neuroimaging.[16] Patients with cough headache should have an MRI brain scan to rule out posterior fossa lesion including Chiari malformation.

If Chiari malformation is identified, additional spinal imaging should be performed to look for additional signs of dysraphism including syringohydromyelia.

4.6.3. Hydrocephalus

The aim of imaging is to differentiate between enlarged ventricles without obstruction (communicating hydrocephalus) and enlarged ventricles with obstruction (obstructive hydrocephalus).

The high resolution heavily T2 weighted sequence (CISS or 3D-SPACE) is used to identify the CSF flow void in the aqueduct or ventriculostomy to confirm patency and CSF flow and is considered to be most efficient technique for evaluation of hydrocephalus and ventriculostomy[37]. In complex cases, high resolution contrast enhanced MRI and CSF flow studies may use, however, techniques are tailored to individual Neuroscience Centres.

The sagittal CISS sequence allows optimal assessment of the cerebral aqueduct and post-operative assessment of third ventriculostomy. Cerebral CSF flow studies may be used, however this depends upon individual neuroscience centre preferences.

4.6.4. Exertional or sexual headache (see also cough headache)

MRI should be carried out to exclude a structural cause or vascular abnormality in patients with exertional headache.

Benign exertional headache which is precipitated rather than aggravated by exertion can be diagnosed as primary if it is not associated with any other disorder (*see Annex 2*).[16] ICHD-II classification states that on first occurrence subarachnoid haemorrhage or arterial dissection need to be excluded.[16] If headaches are prolonged beyond a few hours, are accompanied by focal neurological symptoms or vomiting, or appear de novo after the age of 40 the chance of finding a relevant abnormality is increased.[33]

Further investigation should be considered in patients with headaches which are precipitated, rather than aggravated, by exercise.

4.6.5. Low pressure headache

All patients with suspected low pressure headache should be referred to a specialist for consideration of the most appropriate investigation with MRI of the brain and spine including Gd.

Patients with spontaneous intracranial hypotension often exhibit low CSF pressure and changes on brain MR imaging and/or evidence of CSF leak on myelography. A retrospective, cross-sectional study of 99 subjects with spontaneous intracranial hypotension showed dural enhancement, brain sagging, and venous distension sign in 83%, 61%, and 75% of subjects, respectively, and myelographic evidence of CSF leak was seen in 55%. [38,39,40] Several case series show an association between spontaneous intracranial hypotension and diffuse pacchymeningeal gadolinium enhancement on brain MRI [38,39,40]

4.6.6. Papilloedema and suspected Idiopathic intracranial hypertension (IIH)

For optimal investigation of patients with papilloedema, there must be clear communication between clinicians for seamless joint investigation between the various specialities.

The aims of investigations of papilloedema are to:

1. find any underlying treatable cause in a timely manner,
2. protect the vision and ensure timely re-examination when vision is at risk,
3. enable onward care of the patient with the input from the most appropriate experienced clinician.

Neuroimaging should include urgent MRI brain within 24 hours; if unavailable within 24 hours, then urgent CT brain with subsequent MRI brain if no lesion identified.

If IIH is suspected further imaging should be performed with MRI with MRV. Typical imaging features include a predominantly CSF filled expanded pituitary fossa (empty sella sign), dilated optic nerve sheaths, flattening of the posterior aspects of the optic globes and there may be venous sinus thrombosis or attenuation.[41,42,43,44]

There should be no evidence of hydrocephalus, mass lesion, structural, vascular lesion and no abnormal meningeal enhancement. [42,42,43,44.]

If venous thrombosis is present, there needs to be clinical assessment of venous thrombosis as part of the IIH phenotype and venous thrombosis from other causes.

4.6.7. Suspected vasculitis

Neuroimaging should include MRI with diffusion weighted imaging to look for acute ischaemia and Gd to look for active enhancement. Vascular studies with CTA or MRA are usually required.

5. IMAGING PROTOCOLS FOR SPECIFIC HEADACHES

Acute headache with red flags / fulfilling NICE guidelines	Urgent CT head within 12 hours. If CT shows SAH, perform CTA. If hydrocephalus, consider MRI with sagittal CISS. If tumour/mass, perform MRI including Gd.
Low pressure features	MRI brain and spine with Gd. You are looking for venous distension sign, distended pituitary gland, sagging of hindbrain, extra axial proteinaceous collections and diffuse meningeal enhancement. Spinal imaging is performed to try and identify extra axial collections and CSF leak.
Cough headache	MRI with sagittal sequences to look for Chiari malformation or posterior fossa mass or colloid cyst.
Coital headache	Exclude SAH with CT/LP. MRI and vascular imaging. Look for vasoconstriction or dissection.
Hydrocephalus or post ventriculostomy headache	MRI with sagittal CISS (high res T2 and consider flow studies.
Exertional headache	MRI to look for underlying vascular lesion such as AVM or dural AV fistula (DAVF).
Papilloedema/Idiopathic intracranial Hypertension	CT may be first step if NICE guidelines or red flags. MRI and MRV or CTV.

Suspected venous thrombosis	MRI and MRV or CTV.
Trigeminal neuralgia	Standard brain MRI plus high resolution T2 CISS, MRA and post Gd fat saturated high resolution images in axial and coronal planes. You are looking to exclude vascular compression of the trigeminal root entry zone, or intrinsic trigeminal nerve or branch pathology.
Suspected infection, inflammation or malignancy	MRI with Gd. Further spinal imaging may be required.
Suspected vasculitis	Standard MRI head (which includes DWI) and Gd and MRA.

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7. APPENDICES

7.1. The International Classification Of Headache Disorders 3rd Edition

Cephalalgia 2018, Vol. 38(1) 1–211International Headache Society 2018

Classification

ICHD-3 code Diagnosis

1. Migraine
1.1 Migraine without aura

1.2 Migraine with aura
1.2.1 Migraine with typical aura
1.2.1.1 Typical aura with headache
1.2.1.2 Typical aura without headache
1.2.2 Migraine with brainstem aura
1.2.3 Hemiplegic migraine
1.2.3.1 Familial hemiplegic migraine (FHM)
1.2.3.1.1 Familial hemiplegic migraine type 1 (FHM1)
1.2.3.1.2 Familial hemiplegic migraine type 2 (FHM2)
1.2.3.1.3 Familial hemiplegic migraine type 3 (FHM3)
1.2.3.1.4 Familial hemiplegic migraine, other loci
1.2.3.2 Sporadic hemiplegic migraine (SHM)
1.2.4 Retinal migraine
1.3 Chronic migraine
1.4 Complications of migraine
1.4.1 Status migrainosus
1.4.2 Persistent aura without infarction
1.4.3 Migrainous infarction
1.4.4 Migraine aura-triggered seizure
1.5 Probable migraine
1.5.1 Probable migraine without aura
1.5.2 Probable migraine with aura
1.6 Episodic syndromes that may be associated with migraine
1.6.1 Recurrent gastrointestinal disturbance
1.6.1.1 Cyclical vomiting syndrome
1.6.1.2 Abdominal migraine
1.6.2 Benign paroxysmal vertigo
1.6.3 Benign paroxysmal torticollis
2. Tension-type headache (TTH)
2.1 Infrequent episodic tension-type headache
2.1.1 Infrequent episodic tension-type headache associated with pericranial tenderness
2.1.2 Infrequent episodic tension-type headache not associated with pericranial tenderness
2.2 Frequent episodic tension-type headache
2.2.1 Frequent episodic tension-type headache associated with pericranial tenderness
2.2.2 Frequent episodic tension-type headache not associated with pericranial tenderness
2.3 Chronic tension-type headache
2.3.1 Chronic tension-type headache associated with pericranial tenderness
2.3.2 Chronic tension-type headache not associated with pericranial tenderness
2.4 Probable tension-type headache
2.4.1 Probable infrequent episodic tension-type headache
2.4.2 Probable frequent episodic tension-type headache
2.4.3 Probable chronic tension-type headache
3. Trigeminal autonomic cephalalgias (TACs)
3.1 Cluster headache
3.1.1 Episodic cluster headache
3.1.2 Chronic cluster headache

3.2 Paroxysmal hemicrania
3.2.1 Episodic paroxysmal hemicrania
3.2.2 Chronic paroxysmal hemicrania
3.3 Short-lasting unilateral neuralgiform headache attacks
3.3.1 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)
3.3.1.1 Episodic SUNCT
3.3.1.2 Chronic SUNCT
3.3.2 Short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)
3.3.2.1 Episodic SUNA
3.3.2.2 Chronic SUNA
3.4 Hemicrania continua
3.4.1 Hemicrania continua, remitting subtype
3.4.2 Hemicrania continua, unremitting subtype
3.5 Probable trigeminal autonomic cephalalgia
3.5.1 Probable cluster headache
3.5.2 Probable paroxysmal hemicrania
3.5.3 Probable short-lasting unilateral neuralgiform headache attacks
3.5.4 Probable hemicrania continua
4. Other primary headache disorders
4.1 Primary cough headache
4.1.1 Probable primary cough headache
4.2 Primary exercise headache
4.2.1 Probable primary exercise headache
4.3 Primary headache associated with sexual activity
4.3.1 Probable primary headache associated with sexual activity
4.4 Primary thunderclap headache
4.5 Cold-stimulus headache
4.5.1 Headache attributed to external application of a cold stimulus
4.5.2 Headache attributed to ingestion or inhalation of a cold stimulus
4.5.3 Probable cold-stimulus headache
4.5.3.1 Headache probably attributed to external application of a cold stimulus
4.5.3.2 Headache probably attributed to ingestion or inhalation of a cold stimulus
4.6 External-pressure headache
4.6.1 External-compression headache
4.6.2 External-traction headache
4.6.3 Probable external-pressure headache
4.6.3.1 Probable external-compression headache
4.6.3.2 Probable external-traction headache
4.7 Primary stabbing headache
4.7.1 Probable primary stabbing headache
4.8 Nummular headache
4.8.1 Probable nummular headache
4.9 Hypnic headache
4.9.1 Probable hypnic headache
4.10 New daily persistent headache (NDPH)

4.10.1 Probable new daily persistent headache
5. Headache attributed to trauma or injury to the head and/or neck
5.1 Acute headache attributed to traumatic injury to the head
5.1.1 Acute headache attributed to moderate or severe traumatic injury to the head
5.1.2 Acute headache attributed to mild traumatic injury to the head
5.2 Persistent headache attributed to traumatic injury to the head
5.2.1 Persistent headache attributed to moderate or severe traumatic injury to the head
5.2.2 Persistent headache attributed to mild traumatic injury to the head
5.3 Acute headache attributed to whiplash
5.4 Persistent headache attributed to whiplash
5.5 Acute headache attributed to craniotomy
5.6 Persistent headache attributed to craniotomy
6. Headache attributed to cranial and/or cervical vascular disorder
6.1 Headache attributed to cerebral ischaemic event
6.1.1 Headache attributed to ischaemic stroke (cerebral infarction)
6.1.1.1 Acute headache attributed to ischaemic stroke (cerebral infarction)
6.1.1.2 Persistent headache attributed to past ischaemic stroke (cerebral infarction)
6.1.2 Headache attributed to transient ischaemic attack (TIA)
6.2 Headache attributed to non-traumatic intracranial haemorrhage
6.2.1 Acute headache attributed to non-traumatic intracerebral haemorrhage
6.2.2 Acute headache attributed to non-traumatic subarachnoid haemorrhage (SAH)
6.2.3 Acute headache attributed to non-traumatic acute subdural haemorrhage (ASDH)
6.2.4 Persistent headache attributed to past non-traumatic intracranial haemorrhage
6.2.4.1 Persistent headache attributed to past non-traumatic intracerebral haemorrhage
6.2.4.2 Persistent headache attributed to past non-traumatic subarachnoid haemorrhage
6.2.4.3 Persistent headache attributed to past non-traumatic acute subdural haemorrhage
6.3 Headache attributed to unruptured vascular malformation
6.3.1 Headache attributed to unruptured saccular aneurysm
6.3.2 Headache attributed to arteriovenous malformation (AVM)
6.3.3 Headache attributed to dural arteriovenous fistula (DAVF)
6.3.4 Headache attributed to cavernous angioma
6.3.5 Headache attributed to encephalotrigeminal or leptomeningeal angiomatosis (Sturge Weber syndrome)
6.4 Headache attributed to arteritis
6.4.1 Headache attributed to giant cell arteritis (GCA)
6.4.2 Headache attributed to primary angiitis of the central nervous system (PACNS)
6.4.3 Headache attributed to secondary angiitis of the central nervous system (SACNS)
6.5 Headache attributed to cervical carotid or vertebral artery disorder
6.5.1 Headache or facial or neck pain attributed to cervical carotid or vertebral artery dissection
6.5.1.1 Acute headache or facial or neck pain attributed to cervical carotid or vertebral artery dissection
6.5.1.2 Persistent headache or facial or neck pain attributed to past cervical carotid or vertebral artery dissection

6.5.2 Post-endarterectomy headache
6.5.3 Headache attributed to carotid or vertebral angioplasty or stenting
6.6 Headache attributed to cranial venous disorder
6.6.1 Headache attributed to cerebral venous thrombosis (CVT)
6.6.2 Headache attributed to cranial venous sinus stenting
6.7 Headache attributed to other acute intracranial arterial disorder
6.7.1 Headache attributed to an intracranial endarterial procedure
6.7.2 Angiography headache
6.7.3 Headache attributed to reversible cerebral vasoconstriction syndrome (RCVS)
6.7.3.1 Acute headache attributed to reversible cerebral vasoconstriction syndrome (RCVS)
6.7.3.2 Acute headache probably attributed to reversible cerebral vasoconstriction syndrome (RCVS)
6.7.3.3 Persistent headache attributed to past reversible cerebral vasoconstriction syndrome (RCVS)
6.7.4 Headache attributed to intracranial artery dissection
6.8 Headache and/or migraine-like aura attributed to chronic intracranial vasculopathy
6.8.1 Headache attributed to Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL)
6.8.2 Headache attributed to mitochondrial encephalopathy, lactic acidosis and stroke-like episodes (MELAS)
6.8.3 Headache attributed to Moyamoya angiopathy (MMA)
6.8.4 Migraine-like aura attributed to cerebral amyloid angiopathy (CAA)
6.8.5 Headache attributed to syndrome of retinal vasculopathy with cerebral leukoencephalopathy and systemic manifestations (RVCLSM)
6.8.6 Headache attributed to other chronic intracranial vasculopathy
6.9 Headache attributed to pituitary apoplexy
7. Headache attributed to non-vascular intracranial disorder
7.1 Headache attributed to increased cerebrospinal fluid (CSF) pressure
7.1.1 Headache attributed to idiopathic intracranial hypertension (IIH)
7.1.2 Headache attributed to intracranial hypertension secondary to metabolic, toxic or hormonal cause
7.1.3 Headache attributed to intracranial hypertension secondary to chromosomal disorder
7.1.4 Headache attributed to intracranial hypertension secondary to hydrocephalus
7.2 Headache attributed to low cerebrospinal fluid (CSF) pressure
7.2.1 Post-dural puncture headache
7.2.2 Cerebrospinal fluid (CSF) fistula headache
7.2.3 Headache attributed to spontaneous intracranial hypotension
7.3 Headache attributed to non-infectious inflammatory intracranial disease
7.3.1 Headache attributed to neurosarcoidosis
7.3.2 Headache attributed to aseptic (non-infectious) meningitis
7.3.3 Headache attributed to other non-infectious inflammatory intracranial disease
7.3.4 Headache attributed to lymphocytic hypophysitis
7.3.5 Syndrome of transient headache and neurological deficits with cerebrospinal fluid lymphocytosis (HaNDL)
7.4 Headache attributed to intracranial neoplasia
7.4.1 Headache attributed to intracranial neoplasm

7.4.1.1 Headache attributed to colloid cyst of the third ventricle
7.4.2 Headache attributed to carcinomatous meningitis
7.4.3 Headache attributed to hypothalamic or pituitary hyper- or hyposecretion
7.5 Headache attributed to intrathecal injection
7.6 Headache attributed to epileptic seizure
7.6.1 Ictal epileptic headache
7.6.2 Post-ictal headache
7.7 Headache attributed to Chiari malformation type I (CM1)
7.8 Headache attributed to other non-vascular intracranial disorder
8. Headache attributed to a substance or its withdrawal
8.1 Headache attributed to use of or exposure to a substance
8.1.1 Nitric oxide (NO) donor-induced headache
8.1.1.1 Immediate NO donor-induced headache
8.1.1.2 Delayed NO donor-induced headache
8.1.2 Phosphodiesterase (PDE) inhibitor-induced headache
8.1.3 Carbon monoxide (CO)-induced headache
8.1.4 Alcohol-induced headache
8.1.4.1 Immediate alcohol-induced headache
8.1.4.2 Delayed alcohol-induced headache
8.1.5 Cocaine-induced headache
8.1.6 Histamine-induced headache
8.1.6.1 Immediate histamine-induced headache
8.1.6.2 Delayed histamine-induced headache
8.1.7 Calcitonin gene-related peptide (CGRP)-induced headache
8.1.7.1 Immediate CGRP-induced headache
8.1.7.2 Delayed CGRP-induced headache
8.1.8 Headache attributed to exogenous acute pressor agent
8.1.9 Headache attributed to occasional use of non-headache medication
8.1.10 Headache attributed to long-term use of non-headache medication
8.1.11 Headache attributed to use of or exposure to other substance
8.2 Medication-overuse headache (MOH)
8.2.1 Ergotamine-overuse headache
8.2.2 Triptan-overuse headache
8.2.3 Non-opioid analgesic-overuse headache
8.2.3.1 Paracetamol (acetaminophen)-overuse headache
8.2.3.2 Non-steroidal anti-inflammatory drug (NSAID)-overuse headache
8.2.3.2.1 Acetylsalicylic acid-overuse headache
8.2.3.3 Other non-opioid analgesic-overuse headache
8.2.4 Opioid-overuse headache
8.2.5 Combination-analgesic-overuse headache
8.2.6 Medication-overuse headache attributed to multiple drug classes not individually overused
8.2.7 Medication-overuse headache attributed to unspecified or unverified overuse of multiple drug classes
8.2.8 Medication-overuse headache attributed to other medication
8.3 Headache attributed to substance withdrawal
8.3.1 Caffeine-withdrawal headache

8.3.2 Opioid-withdrawal headache
8.3.3 Oestrogen-withdrawal headache
8.3.4 Headache attributed to withdrawal from chronic use of other substance
9. Headache attributed to infection
9.1 Headache attributed to intracranial infection
9.1.1 Headache attributed to bacterial meningitis or meningoencephalitis
9.1.1.1 Acute headache attributed to bacterial meningitis or meningoencephalitis
9.1.1.2 Chronic headache attributed to bacterial meningitis or meningoencephalitis
9.1.1.3 Persistent headache attributed to past bacterial meningitis or meningoencephalitis
9.1.2 Headache attributed to viral meningitis or encephalitis
9.1.2.1 Headache attributed to viral meningitis
9.1.2.2 Headache attributed to viral encephalitis
9.1.3 Headache attributed to intracranial fungal or other parasitic infection
9.1.3.1 Acute headache attributed to intracranial fungal or other parasitic infection
9.1.3.2 Chronic headache attributed to intracranial fungal or other parasitic infection
9.1.4 Headache attributed to localized brain infection
9.2 Headache attributed to systemic infection
9.2.1 Headache attributed to systemic bacterial infection
9.2.1.1 Acute headache attributed to systemic bacterial infection
9.2.1.2 Chronic headache attributed to systemic bacterial infection
9.2.2 Headache attributed to systemic viral infection
9.2.2.1 Acute headache attributed to systemic viral infection
9.2.2.2 Chronic headache attributed to systemic viral infection
9.2.3 Headache attributed to other systemic infection
9.2.3.1 Acute headache attributed to other systemic infection
9.2.3.2 Chronic headache attributed to other systemic infection
10. Headache attributed to disorder of homoeostasis
10.1 Headache attributed to hypoxia and/or hypercapnia
10.1.1 High-altitude headache
10.1.2 Headache attributed to aeroplane travel
10.1.3 Diving headache
10.1.4 Sleep apnoea headache
10.2 Dialysis headache
10.3 Headache attributed to arterial hypertension
10.3.1 Headache attributed to phaeochromocytoma
10.3.2 Headache attributed to hypertensive crisis without hypertensive encephalopathy
10.3.3 Headache attributed to hypertensive encephalopathy
10.3.4 Headache attributed to pre-eclampsia or eclampsia
10.3.5 Headache attributed to autonomic dysreflexia
10.4 Headache attributed to hypothyroidism
10.5 Headache attributed to fasting
10.6 Cardiac cephalalgia
10.7 Headache attributed to other disorder of homoeostasis
11. Headache or facial pain attributed to disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cervical structure
11.1 Headache attributed to disorder of cranial bone

11.2 Headache attributed to disorder of the neck
11.2.1 Cervicogenic headache
11.2.2 Headache attributed to retropharyngeal tendonitis
11.2.3 Headache attributed to craniocervical dystonia
11.3 Headache attributed to disorder of the eyes
11.3.1 Headache attributed to acute angle-closure glaucoma
11.3.2 Headache attributed to refractive error
11.3.3 Headache attributed to ocular inflammatory disorder
11.3.4 Trochlear headache
11.4 Headache attributed to disorder of the ears
11.5 Headache attributed to disorder of the nose or paranasal sinuses
11.5.1 Headache attributed to acute rhinosinusitis
11.5.2 Headache attributed to chronic or recurring rhinosinusitis
11.6 Headache attributed to disorder of the teeth
11.7 Headache attributed to temporomandibular disorder (TMD)
11.8 Head or facial pain attributed to inflammation of the stylohyoid ligament
11.9 Headache or facial pain attributed to other disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cervical structure
12. Headache attributed to psychiatric disorder
12.1 Headache attributed to somatization disorder
12.2 Headache attributed to psychotic disorder
13. Painful lesions of the cranial nerves and other facial pain
13.1 Pain attributed to a lesion or disease of the trigeminal nerve
13.1.1 Trigeminal neuralgia
13.1.1.1 Classical trigeminal neuralgia
13.1.1.1.1 Classical trigeminal neuralgia, purely paroxysmal
13.1.1.1.2 Classical trigeminal neuralgia with concomitant continuous pain
13.1.1.2 Secondary trigeminal neuralgia
13.1.1.2.1 Trigeminal neuralgia attributed to multiple sclerosis
13.1.1.2.2 Trigeminal neuralgia attributed to space-occupying lesion
13.1.1.2.3 Trigeminal neuralgia attributed to other cause
13.1.1.3 Idiopathic trigeminal neuralgia
13.1.1.3.1 Idiopathic trigeminal neuralgia, purely paroxysmal
13.1.1.3.2 Idiopathic trigeminal neuralgia with concomitant continuous pain
13.1.2 Painful trigeminal neuropathy
13.1.2.1 Painful trigeminal neuropathy attributed to herpes zoster
13.1.2.2 Trigeminal post-herpetic neuralgia
13.1.2.3 Painful post-traumatic trigeminal neuropathy
13.1.2.4 Painful trigeminal neuropathy attributed to other disorder
13.1.2.5 Idiopathic painful trigeminal neuropathy
13.2 Pain attributed to a lesion or disease of the glossopharyngeal nerve
13.2.1 Glossopharyngeal neuralgia
13.2.1.1 Classical glossopharyngeal neuralgia
13.2.1.2 Secondary glossopharyngeal neuralgia
13.2.1.3 Idiopathic glossopharyngeal neuralgia
13.2.2 Painful glossopharyngeal neuropathy
13.2.2.1 Painful glossopharyngeal neuropathy attributed to a known cause

13.2.2.2 Idiopathic painful glossopharyngeal neuropathy
13.3 Pain attributed to a lesion or disease of nervus intermedius
13.3.1 Nervus intermedius neuralgia
13.3.1.1 Classical nervus intermedius neuralgia
13.3.1.2 Secondary nervus intermedius neuralgia
13.3.1.3 Idiopathic nervus intermedius neuralgia
13.3.2 Painful nervus intermedius neuropathy
13.3.2.1 Painful nervus intermedius neuropathy attributed to herpes zoster
13.3.2.2 Post-herpetic neuralgia of nervus intermedius
13.3.2.3 Painful nervus intermedius neuropathy attributed to other disorder
13.3.2.4 Idiopathic painful nervus intermedius neuropathy
13.4 Occipital neuralgia
13.5 Neck-tongue syndrome
13.6 Painful optic neuritis
13.7 Headache attributed to ischaemic ocular motor nerve palsy
13.8 Tolosa–Hunt syndrome
13.9 Paratrigeminal oculosympathetic (Raeder's) syndrome
13.10 Recurrent painful ophthalmoplegic neuropathy
13.11 Burning mouth syndrome (BMS)
13.12 Persistent idiopathic facial pain (PIFP)
13.13 Central neuropathic pain
13.13.1 Central neuropathic pain attributed to multiple sclerosis (MS)
13.13.2 Central post-stroke pain (CPSP)
14. Other headache disorders
14.1 Headache not elsewhere classified
14.2 Headache unspecified

7.2. Clinical Guidelines

7.2.1. NICE guidelines

Assessment

Evaluate people who present with headache and any of the following features, and consider the need for further investigations and/or referral

- worsening headache with fever
- sudden-onset headache reaching maximum intensity within 5 minutes
- new-onset neurological deficit
- new-onset cognitive dysfunction

- change in personality
- impaired level of consciousness
- recent (typically within the past 3 months) head trauma
- headache triggered by cough, valsalva (trying to breathe out with nose and mouth blocked) or sneeze
- headache triggered by exercise
- orthostatic headache (headache that changes with posture)
- symptoms suggestive of giant cell arteritis
- symptoms and signs of acute narrow angle glaucoma
- a substantial change in the characteristics of their headache. [2012]

Consider further investigations and/or referral for people who present with new-onset headache and any of the following:

- compromised immunity, caused, for example, by HIV or immunosuppressive drugs
- age under 20 years and a history of malignancy
- a history of malignancy known to metastasise to the brain
- vomiting without other obvious cause. [2012]

7.2.2. American Academy of Neurology recommendations

CT Head indications:

Acute trauma, SAH, MRI Contraindicated

MR angiography (MRA):

Thunderclap headaches,

Family history (FH) (two first degree relatives) of aneurysms,

Headaches that are continuously ipsilateral or progressive in nature.

GRE sequence: Consider if history of head trauma, thunderclap headache, FH of vascular malformations or aneurysms

Gadolinium:

Abnormal neurological examination

Positional headaches

Exertional or valsalva maneourvre exacerbated headaches

Cluster or neuralgic type headaches or facial pain.

Known history of cancer, HIV, infectious disease

Contraindicated during pregnancy.

Red flags:

- The “first or worst” thunderclap
- Subacute headaches with increasing frequency and severity
- Progressive or new daily persistent headache
- Chronic daily headache
- Headaches always on the same side
- Headaches resistant to treatment
- New onset of headaches in high risk population

  Cancer, HIV +ve, Dementia, taking an anticoagulant, Neurocutaneous syndrome
- New onset of headaches after aged 50
- Associated seizures
- Associated with fever, neck stiffness, nausea and vomiting
- Focal neurological deficits (not meeting the criteria for migraines with aura)

- Associated with papilloedema, cognitive impairment or personality changes.
- Headaches precipitated by exertion, valsalva manoeuvre or positional change.
- Atypical cranial neuralgias poorly responsive to treatment.

7.2.3. Imaging patients with suspected brain tumour: guidance for primary care.

Br J Gen Practice 2008 Vol 58 (557):880-885

Red flags — presentations where the probability of an underlying tumour is likely to be greater than 1%. These warrant urgent investigation.

- Papilloedema
- Significant alterations in consciousness, memory, confusion, or coordination
- New epileptic seizure
- New-onset cluster headache (imaging, particularly of the region of the pituitary fossa, required but non-urgent)
- Headache with a history of cancer elsewhere particularly breast and lung
- Headache with abnormal findings on neurological examination or other neurological symptoms (although evidence base suggests orange flag)

Orange flags — presentations where the probability of an underlying tumour is likely to be between 0.1 and 1%. These need careful monitoring and a low threshold for investigation.

- New headache where a diagnostic pattern has not emerged after 8 weeks from presentation
- Headache aggravated by exertion or Valsalva-like manoeuvre
- Headaches associated with vomiting

- Headaches that have been present for some time but have changed significantly, particularly a rapid increase in frequency
- New headache in a patient over 50 years
- Headaches that wake the patient from sleep
- Confusion

Yellow flags — presentations where the probability of an underlying tumour is likely to be less than 0.1% but above the population rate of 0.01%. These require appropriate management, and the need for follow-up is not excluded.

- Diagnosis of migraine or tension-type headache
- Weakness or motor loss
- Memory loss
- Personality change

7.2.4. Guideline for primary healthcare management of headaches

Becker WJ, Findlay T, Moga C, Scott NA, Harstall C, Taenzer P. Guideline for primary healthcare management of headaches. Canadian Family Physician. Le Médecin de famille canadien . Vol 61: Aug 2015

7.2.4.1. General practice points for managing primary headache in adults

The following are general practice points for the management of primary headache in adults:

• Rule out secondary headache when diagnosing a primary headache disorder

• Neuroimaging is not indicated in patients with recurrent headache with the clinical features of migraine, normal neurologic examination findings, and no red flags

• Neuroimaging, sinus or cervical spine x-ray scans, and electroencephalograms are not recommended for the routine assessment of patients with headache: history and physical and neurologic examination findings are usually sufficient to make a diagnosis of migraine or tension-type headache

• Migraine is by far the most common headache type in patients seeking help for headache from physicians

• Migraine is historically underdiagnosed and undertreated; many patients with migraine are not diagnosed with migraine when they consult a physician

• Migraine should be considered in patients with recurrent moderate or severe headaches and normal neurologic examination findings

• Patients consulting for bilateral headaches that interfere with their activities are likely to have migraine rather than tension type headache and might require migraine-specific medication

• Consider a diagnosis of migraine in patients with a previous diagnosis of recurring “sinus” headache

7.3. Red flags

Potential indicators of secondary headache: Appropriate referral or investigation should be considered.

(Based on the Scottish Intercollegiate Guidelines Network guideline 29 and expert opinion of the Guideline Development Group).

Red flags: emergent (address immediately)

- Thunderclap onset
- Fever and meningismus

- Papilledema with focal signs or reduced level of consciousness
- Acute glaucoma

Red flags: urgent (address within hours to days)

- Temporal arteritis
- Papilledema without focal signs or reduced level of consciousness
- Relevant systemic illness
- Elderly patient: new headache with cognitive change

Other possible indicators of secondary headache (less urgent)

- Unexplained focal signs
- Atypical headaches (not consistent with migraine or tension-type
- headache)
- Unusual headache precipitants
- Unusual aura symptoms
- Onset after age 50 y
- Aggravation by neck movement; abnormal neck examination
- findings (consider cervicogenic headache)
- Jaw symptoms; abnormal jaw examination findings (consider
- temporomandibular joint disorder)