Headache and facial pain

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Abstract

Headache (any pain involving the neck and upwards, including the face) is the commonest neurological presentation, accounting for >25% of new outpatient neurology referrals. It makes up a significant proportion of GP and A&E attendances, and costs the health service more than Parkinson's disease, neuromuscular disease and other neurological conditions combined. Understanding the presentation and management of headache disorders is important in timely diagnosis, excluding worrying secondary causes, limiting morbidity and reducing unnecessary or lengthy hospital admissions. Facial pain is often regarded as separate from headache, with patients assessed in specialities such as ENT and maxillofacial surgery. Although dental and sinus pathology causes pain, migraine and other primary headaches can include, or exclusively involve, the face. Headache disorders are often incorrectly ascribed to a neck problem, but neck involvement in headache disorders is common and should not detract from the headache diagnosis. Migraine is the most common acute headache. It affects 20% of people, and many more experience some migraine symptoms during their life. It is one of the most disabling disorders according to the World Health Organization, and has significant socioeconomic impact for sufferers at the prime of their working lives. This article summarizes assessment, focusing on taking a complete headache history, to ensure correct and timely diagnosis.

Keywords Cluster headache; facial pain; headache; indometacin-sensitive headache; migraine

Key points

- Headache and facial pain is a common presenting medical symptom in different settings, including general practice, accident and emergency, acute medical wards and neurology clinics
- Primary headache disorders are much more common than secondary headaches. Migraine is the most common headache disorder encountered in primary and secondary care
- The key to differentiating these is a detailed history, with selected imaging and other tests used as needed, depending on phenotypic and 'red flag' features elicited in the history
- Acute treatment of migraine in a hospital setting involves subcutaneous sumatriptan, intravenous or high-dose oral aspirin and intravenous paracetamol where available and in the absence of contraindications; opioid use in general should be avoided
- Medication overuse should be screened for in every headache history as it alters phenotype, further management and prognosis

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The headache history

Conventional teaching is to take a headache history in the same way as a pain history, focusing on 'red flags' that may suggest a secondary disorder. A detailed and specific headache history guides diagnosis and effective treatment and ensures patient satisfaction. Examination and imaging are usually normal. Specific factors about the onset of pain, and the phenotype, are essential. Table 1 summarizes the key features in the history, with suggested questions; italic text represents the reasons for each question. In particular, it is vital to screen for medication overuse, which can aid diagnosis, alter typical phenotypes, influence treatment and have prognostic implications.

Examination and further tests

Although the neurological examination is usually normal in the case of headache, assessment should include blood pressure (malignant hypertension can cause headache, and hypertension can worsen headache), weight (for appropriate medication dosing and when idiopathic intracranial hypertension (IIH) is a possible diagnosis), and cranial nerve examination, including visual acuity and fields, fundoscopy and trigeminal sensation.

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The headache history

Feature in the history	Questions to ask
First ever headache *Is there migrainous biology?*	When do you first recall experiencing headache?[a] Do you get bad hangovers with headache? Do you get headaches with stress, dehydration, poor sleep, hunger or menses? Does caffeine withdrawal ever give you a headache? Are you prone to motion sickness?
Current frequency *How problematic is the current headache?*	How many days a month is there some discomfort in your head? How many days a month are you 'crystal clear'? How long have you been at this frequency? In daily and continuous pain, did the pain come on and never go away one day or gradually build up to this frequency?[b] Can you remember the exact day when this happened? What were the circumstances surrounding this worsening?[c]
Family history of headache disorder *Is there a genetic tendency to migraine?*	Is there a family history of headache problems? Do your mother, father or siblings experience headache?
Headache phenotype *What is the diagnosis?*	How did the headache come on? How quickly does it reach maximum intensity? Where is the headache? Is it on one side or both? What does it feel like? Does it throb? What do you feel like doing when you have the pain? How long does the pain last for without treatment? Is there aversion to light, sound and smell? Do you get nausea and vomiting?[d]
Triggers *What is the diagnosis?*	Does anything bring on or worsen the headache? Do you notice a worsening after alcohol, during or around menstruation, when you have not slept or eaten or when in certain environments? Is the headache worse in any particular position (lying versus sitting/standing)? Is there any variation in headache throughout the day? Does coughing, sneezing or straining worsen the headache or bring it on?[e]
Associated symptoms *What is the diagnosis?*	Does anything warn you that a headache is coming? Do you get other symptoms such as yawning, thirst, neck discomfort, concentration difficulty and fatigue? Do you feel dizzy with the headache, as if you are moving or the world around you is moving? Is it sensitive to touch any part of your head during the pain? How do you feel when a headache has settled?[f]
Cranial autonomic symptoms (unilateral/ bilateral) *What is the diagnosis? Is a trigeminal autonomic cephalalgia likely?*	When the headache is bad, do your eyes water or go red? Does your nose run or feel stuffy? Does your face flush or sweat or do you go pale? Does your eyelid droop? Has anyone commented that your facial appearance changes? Do you get discomfort behind your ears, like they are full or you are 'under water'?
Aura *What is the diagnosis?*	Do you experience visual, sensory or motor symptoms before, after or during headache? What does this look/feel like? Does it spread? Over how long? How long does it last for? What percentage of attacks are such symptoms present for?[g]
Medications *What has been tried and what has/has not worked? Is there medication overuse? Are there any potentially contributing drugs?*	What are you currently taking for headache, and at what dose? How many days a month do you take each drug? Do you take anything else over the counter, including any cannabinoid compounds or combined analgesic preparations? Are you taking any preventive treatment, and at what dose? How effective are your acute treatments (time to headache resolution)? How effective is your preventive treatment? Do you experience any side effects on your current therapy? What other medications are you on for other problems? Are you using any contraceptive agents? Do you have any allergies?
Previous medications *What has been tried and what has/has not worked? How difficult has the migraine been to treat in the past?*	What previous acute and preventive drugs have you tried for your headache? For each one, what dose did you get up to, how long did you take it for, did it work and why was it stopped? Have you ever used devices for headache such as the SpringTMS, GammaCore or Cefaly devices? Have you ever had a nerve block?
Other medical problems *Are there concurrent medical issues, contraindications to medication use, difficulties addressing medication overuse, etc.?*	Do you have any other medical conditions that you see your doctor for? In particular, do you have hypertension, cerebrovascular disease, cardiovascular disease, asthma or obstructive airways disease, kidney stones, mood disorders, cognitive impairment and renal or liver disease?
Pregnancies *Is there a history of recurrent miscarriage? If there is an active or planned pregnancy, this will impact on management*	Are you pregnant? How many pregnancies have you had? How many children do you have? Are you planning a pregnancy?
Menstrual history *Establishing a hormonal link*	Are you currently menstruating? What age was menarche? Is there a link of your headaches with menses? If so, how? Have you been through the menopause? How long have you had menopausal symptoms for? When did your menses stop? Has there been a headache worsening associated with the menopause?

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Table 1 (*continued*)

Feature in the history	Questions to ask
Social history *Are there contributing factors?*	Do you smoke? How much alcohol do you drink a week? Do you use cannabis or other recreational drugs? How often? How much caffeine would you drink in an average day (coffee, tea, colas, energy drinks)?
Previous brain imaging *Asking about this may prevent recurrent investigations for the same headache phenotype*	Have you ever had a brain scan? What type? Where? What did it show?
Ideas and concerns *Ensuring the patient's questions and concerns have been answered tends to encourage understanding and increase satisfaction with the consultation, compliance and emergency presentations*	What are your thoughts about what could be causing your headache? Are you worried about anything in particular?
Red flags *Exclude a secondary headache disorder*	See Table 2

[a] Many people deny a prior history, so it is worth specifically probing about even mild headache in the past.
[b] Headache that starts from one day to the next (with a memorable onset) and never remits is termed 'new daily persistent headache' (NPDH).(1) This is a descriptive rather than biological name, and it can encorporate migraine or hemicrania continua, or be featureless. NPDH needs a secondary cause, such as CSF hypotension, idiopathic intracranial hypotension or carotid dissection, to be considered.
[c] Specifically, systemic illness, head injury, cranial surgery, head or neck trauma, recreational drug use, physical exertion, emotional stress or hormonal change (menstruation, menopause, pregnancy).
[d] The laterality of the headache and the presence of migraine features (as per the International Classification of Headache Disorders 3),[1] such as sensory and movement sensitivities, nausea and vomiting, can be important in distinguishing migraine from other headaches. Localization can help in diagnosis, particularly in the trigeminal autonomic cephalalgias and trigeminal neuralgia.
[e] Diurnal and postural change helps in the diagnosis of cerebrospinal fluid disorders such as intracranial hypo- or hypertension.
[f] Migraine can be associated with non-painful prodromal symptoms including neck stiffness, yawning, concentration change, fatigue and thirst, among others such as allodynia, vertigo, tinnitus and visual complaints. Some symptoms persist after headache resolution and impair return to normal function; the most troubling are usually fatigue and cognitive dysfunction.
[g] Aura is seen in up to 30% of migraineurs but also uncommonly in other primary headaches such as cluster headache, and hence is not diagnostic of migraine. Aura can present without prominent headache. It is important to ask about focal neurological deficit associated with migraine attacks, to allow diagnosis and investigation as appropriate.

Table 1

Further examination can be tailored and can include looking for focal neurological deficits and greater occipital nerve tenderness, and temporal artery palpation.

All histories with red flag features warrant further investigations: for example, temporal arteritis indicates inflammatory markers and consideration of temporal artery biopsy, and subarachnoid haemorrhage a computed tomography scan, possibly followed by lumbar puncture and then arterial brain imaging (Table 2).

Investigations are tailored to assessment, and neuro-imaging is not always indicated. In the absence of red flag features, an incidental finding rather than a tumour is more likely on brain scan and may increase anxiety. Patients with trigeminal autonomic cephalalgia should have dedicated brain magnetic resonance imaging (MRI) with pituitary views, looking for cavernous sinus, orbital and pituitary pathologies.

Primary headache disorders

Migraine

Owing to its high prevalence and high level of disability, migraine is the most common primary headache disorder presenting to secondary care. Migraine has historically been seen as an episodic pain condition. Recent evidence regarding its neurobiology shows that the disorder involves more than just the pain pathways, sometimes manifesting with non-painful yet disabling symptoms contributing to the disability. The diagnosis is usually straightforward using the history structure to elicit canonical features as per the International Classification of Headache Disorders 3 classification.[1] However, an appreciation of the disability associated with each attack and the multifaceted symptomatic features of the condition are important in the doctor–patient alliance, and in helping patients communicate their disability to schools and employers. Management can be less than satisfactory, particularly in acute settings, because of a tendency to treat migraine as another painful condition, using opioids but with a lack of nausea control, follow-up analgesic plans or preventive treatment. Patients therefore often leave emergency departments still in pain and frustrated, and without long-term management options.[2]

Despite a recent wealth of headache research, the only effective and specific drugs available to treat migraine acutely on the UK NHS are currently the triptans. These are serotonin

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Headache 'red flags'

'Red flag' feature	Possible diagnosis	Investigations that can be considered
Constitutional symptoms (weight loss, fever, myalgia)	Malignancy, infection	Brain imaging, CSF analysis
Focal neurological deficit (aside from aura typical for the patient), change in mental status or personality	Mass lesion, stroke, viral encephalitis, arterial dissection	Brain imaging, arterial imaging, CSF analysis with viral studies
Thunderclap onset (peak intensity in <5 min)	Subarachnoid haemorrhage, intracerebral bleed, arterial dissection, reversible vasoconstriction syndrome, pituitary apoplexy	Brain imaging, arterial imaging, CSF analysis
Postural change in headache	Raised intracranial pressure (mass lesion, venous sinus thrombosis) Low intracranial pressure (CSF leak)	Brain imaging (with or without contrast), venous imaging, CSF analysis if history is suggestive of raised pressure and imaging normal? IIH, spinal imaging and myelography if CSF hypotension is likely
Age of onset >50 years	Temporal arteritis, acute angle-closure glaucoma, mass lesion	Blood inflammatory markers, temporal artery biopsy, ophthalmological review, brain imaging
Progressive headache or change in character of pre-existing headache	Worsening of primary headache disorder, presence of medication overuse, mass lesion, hypertension	Brain imaging, blood pressure monitoring
Accompanying seizures	Infection, venous sinus thrombosis, mass lesion	Brain imaging, venous imaging, CSF analysis and viral studies
HIV infection or malignancy	Infection, metastases, carcinomatous meningitis	Brain imaging, CSF analysis including infectious screening and cytology
Papilloedema	Intracranial hypertension from any cause, such as mass lesion, venous sinus thrombosis, IIH	Brain imaging, venous imaging, CSF analysis if imaging normal, ophthalmological assessment
Valsalva precipitation	Arnold-Chiari malformation, intracranial hypertension	Brain imaging
Pregnancy or postpartum	Reversible vasoconstriction syndrome, venous sinus thrombosis, pituitary apoplexy	Brain imaging, venous imaging

CSF, cerebrospinal fluid; IIH, idiopathic intracranial hypertension; min, minutes.

Table 2

($5HT_{1B/1D}$) receptor agonists, which have proven efficacy in migraine as tablets, nasal sprays and subcutaneous formulations. Variable effectiveness, contraindications to use and adverse effects limit their use for some. In individuals without contraindications such as cerebro- and cardiovascular disease and age >65 years, or previous adverse effects, a parenteral triptan, such as a nasal spray of sumatriptan or zolmitriptan, or a sumatriptan injection, should be offered to those presenting to the emergency department with severe migraine, as these agents have a high likelihood of offering at least some relief.

Other drugs that can be used in an emergency setting include 1 g paracetamol intravenously, 300–900 mg aspirin orally or 900–1000 mg intravenously, 50–75 mg diclofenac orally or rectally, 600–800 mg ibuprofen orally, or 250–500 mg naproxen orally.[3] Combining a non-steroidal anti-inflammatory drug with a triptan can prolong the triptan's effect and reduce headache recurrence. If there is renal disease or gastric ulceration, non-steroidal agents should be avoided.

Nausea should be managed alongside headache, as gastric motility slows in migraine, reducing effectiveness and speed of gastrointestinal absorption of oral medications. Metoclopramide, domperidone and prochlorperazine can be used orally or intravenously, and prochlorperazine is also available in suppository form.[4] All these drugs are in line with British National Formulary (BNF) recommendations for migraine.

In all settings, opioid analgesics should be avoided, given the risk of addiction, their poor effects in migraine and medication overuse effects.[5] Patients should be given a proper diagnosis when presenting acutely, and also a treatment plan. It is worth considering that the effect of triptans is not a class effect, so failure to respond to one agent does not predict response to another within the class; an alternative should always be offered.

Acute attack migraine therapeutics are evolving significantly with the emergence and licensing in the USA of two new classes of acute therapy. Ditans – serotonin 5-HT_{1F} receptor agonists – have been shown in Phase II and III trials to be effective for acute migraine without vasoconstriction. Their main adverse effect, in about 15% of patients, is dizziness. Gepants – calcitonin gene-related peptide (CGRP) receptor antagonists – are effective and well tolerated in acute migraine, with no vasoconstrictor actions. These agents remain in clinical trials and are currently not

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Some of the primary headache disorders

Headache type	Typical duration	Number of attacks a day (24 h)	Typical patient demographic	Phenotype	Estimated prevalence	Acute treatment	Preventive treatment
Migraine	4—72 h	Usually 1	Women > men (3:1), young, of child-bearing age	Unilateral or bilateral, throbbing, movement sensitive, nausea, vomiting, sensory sensitivities, can have cranial autonomic symptoms, 20—30% have aura	1:7	NSAIDs, triptans, paracetamol, antiemetics, fluids, intravenous dihydroergotamine, greater occipital nerve injection[a]	Propranolol, topiramate, candesartan, gabapentin*, amitriptyline, flunarizine*, single pulse transcranial magnetic stimulation
Cluster headache	30—180 min	Up to 8, occurring in 'bouts'	Young male smokers	Strictly unilateral, typically V_1 and orbital/retro-orbital, 'red hot poker', twisting sensation, severe, cranial autonomic symptoms[b]	1:1000	Subcutaneous or nasal triptan*, high-flow oxygen, intravenous dihydroergotamine, oral prednisolone, greater occipital nerve injection	Verapamil, lithium*, melatonin*, non-invasive vagal nerve stimulation, sphenopalatine ganglion stimulation
Paroxysmal hemicrania	2—45 min	Up to 10, occurring in bouts	Men and women equally affected, median age 40 years (range 3—81 years)	Strictly unilateral, cranial autonomic symptoms, absolute response to adequate dose of indometacin[c]	Rarer than cluster headache, but absolute prevalence unknown	Indometacin*, greater occipital nerve injection	Indometacin, celecoxib*, topiramate*, non-invasive vagal nerve stimulation*
Hemicrania continua	Continuous and daily	Can have worsenings, lasting minutes to days	Median age of onset in 30s	Unilateral continuous pain with superimposed worsenings, can have migrainous features, can have associated cranial autonomic symptoms. Absolute response to adequate dose of indometacin	Absolute prevalence unknown, but likely more common than initially thought	Indometacin*, greater occipital nerve injection	Indometacin*, celecoxib*, topiramate*, non-invasive vagal nerve stimulation*
SUNCT/SUNA	Seconds	Up to 300	Slightly more men (1.5:1), age >50 years	Strictly unilateral (typically V_1 region), with associated conjunctival injection and tearing (SUNCT) or other cranial autonomic symptoms (SUNA)	1:15,000	Greater occipital nerve injection	Lamotrigine*, topiramate*, gabapentin*

(*continued on next page*)

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Table 3 (*continued*)

Headache type	Typical duration	Number of attacks a day (24 h)	Typical patient demographic	Phenotype	Estimated prevalence	Acute treatment	Preventive treatment
Trigeminal neuralgia	Seconds, but can occur repetitively, up to 2 min	Determined by innocuous stimulation and refractory periods	More women than men, classical spontaneous form in over-50s	Unilateral, in V_2 and V_3 — electric shock-like sensations, triggered by cutaneous stimulation in the area, can have associated muscle spasms in the face, refractory period after cutaneous triggering for several minutes	4—13:100,000	—	Carbamazepine, oxcarbazepine*, amitriptyline*, gabapentin*

*Treatments used in headache practice that do not appear in the BNF for headache indications. Given the rarity of the trigeminal autonomic cephalalgias, many treatments that are used in clinical practice do not appear in the BNF for these disorders. Use is therefore based on clinical practice and available evidence.
NSAID, non-steroidal anti-inflammatory drug; V_{1-3}, branches 1—3 of the trigeminal nerve.
[a] A greater occipital nerve injection is a useful strategy to help manage any of the primary headache disorders, particularly those that are lateralized. The injection consists of corticosteroid and local anaesthetic, is administered around the greater occipital nerve at the back of the head on one side. It is usually well tolerated and can be effective as a traditional treatment in some headache types. Neuromodulation options such as transcranial magnetic stimulation, sphenopalatine ganglion stimulation and non-invasive vagal nerve stimulation do not appear in the BNF, although have been approved within the UK through NICE for specific indications. They have also been used for other headache disorders based on anecdotal evidence.
[b] Cranial autonomic symptoms include conjunctival tearing, injection, a sense of eye grittiness, nasal stuffiness, rhinorrhoea, facial flushing, facial sweating, aural discomfort, altered taste sensation, localized swelling and ptosis.
[c] An adequate dose of oral indometacin used for diagnosis of paroxysmal hemicrania and hemicrania continua (the indometacin-sensitive headache disorders) is, in our opinion, at least 150—225 mg taken for at least 14 days. A slow uptitration, starting at 25 mg three times a day orally and increasing every few days, is used. If the headache stops at a dose <150—225 mg, this dose can be continued for the 14-day period to ensure headache cessation. An indometacin trial can also be conducted using 50—200 mg intramuscularly in some centres for formal comparison to placebo in a randomized and double-blind fashion.

Table 3

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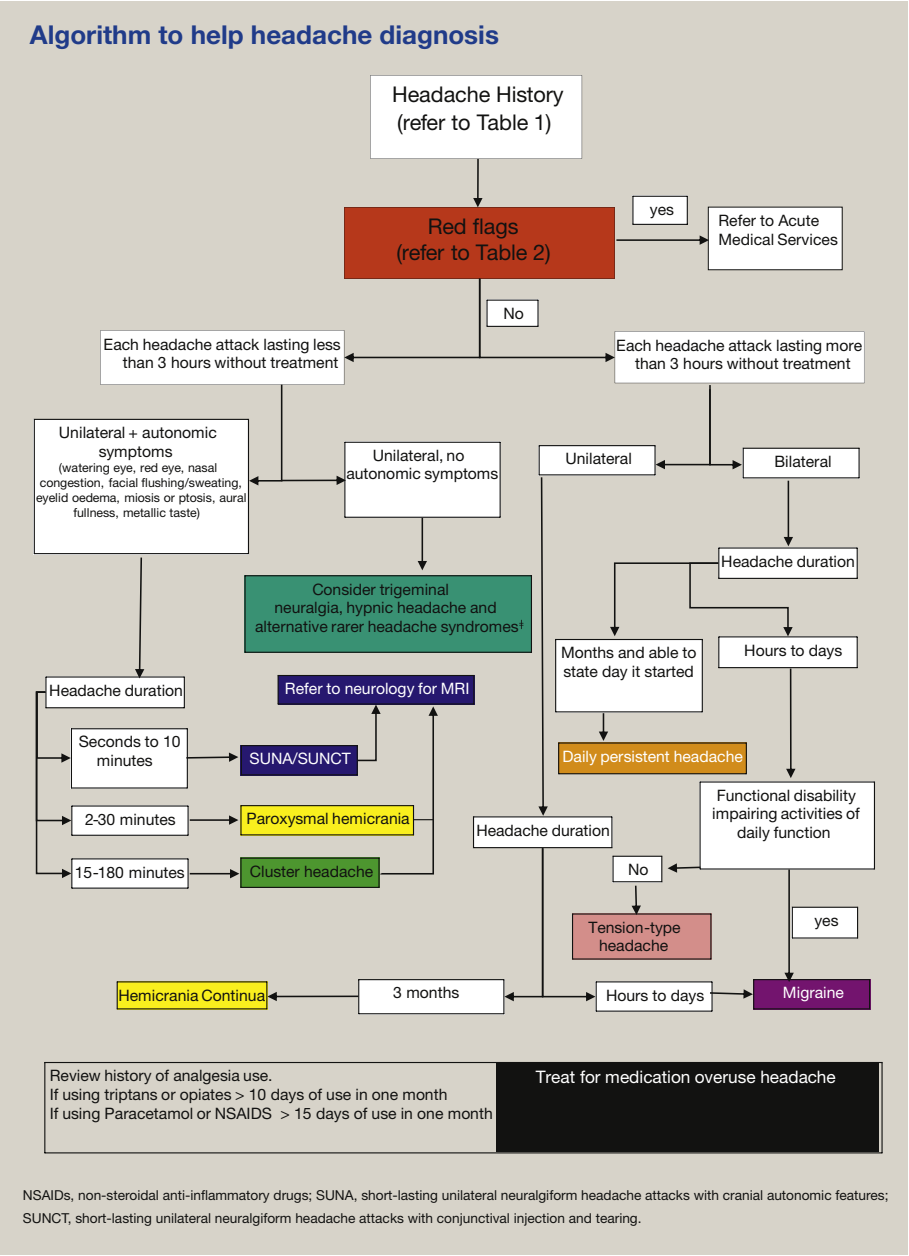

Figure 1 Clinical approach to headache diagnosis

available for clinical use within the UK, but may be promising options in the future.

Preventive treatment can be offered to patients who have >4 attacks a month. Unfortunately, most of the drugs available were developed for other indications and are therefore non-specific, often complicated by adverse effects and at best helpful for only around 50% of sufferers. These agents include antidepressants such as amitriptyline, anticonvulsants like topiramate, β-adrenoceptor blockers such as propranolol, and candesartan. All of these agents appear in the BNF as agents for migraine prevention.

Targeted acute and preventive options are currently in development or undergoing consideration for NHS funding. These include monoclonal antibodies against the CGRP receptor or protein, and small molecule CGRP antagonists (gepants), which should advance migraine therapeutics in the UK in the near future and have more favourable adverse effect profiles.

Other primary headache disorders

Other primary headache disorders are considerably rarer; they comprise largely unilateral syndromes of shorter lasting headaches known as trigeminal autonomic cephalalgias, which are accompanied by cranial autonomic symptoms. Trigeminal neuralgia, although rare, is experienced in clinical practice and can present acutely because of pain severity. These headache

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disorders are summarized phenotypically in Table 3. From the phenotypic descriptions of these headache disorders, it is clear that the trigeminal distribution in the face is involved in almost all, apart from migraine and indometacin-sensitive headaches, which can occur anywhere in the head. An algorithm to help guide management is shown in Figure 1. Although this figure is not for all the headache disorders that may be encountered, it provides a useful initial framework to aid diagnosis and appropriate next steps. Screening for medication overuse is vital and influences ongoing management.[5] ◆

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 28-year-old woman presents with a 4-day history of severe headache that had started 2 days before her menses, without any other clear provoking factor. She has a history of migraine with visual aura. The headache is lateralized to the right, similar to her usual attacks but more severe than before. She has had a visual aura, typical for her, daily for the previous 4 days. Sumatriptan unusually had no effect; the last dose was 2 hours ago. She has not used anything else for the headache. She is nauseated but not vomiting. She is otherwise well.

On examination, she is sensitive to light with some neck stiffness but no clear signs of meningeal irritation. She has a partial right-sided ptosis and some redness in the right eye. The pupils are equal and reactive, and the remainder of the neurological examination is normal. Her observations are stable. She is crying out in pain and clearly distressed.

What is the most appropriate next step in acute management?

A. Urgent CT scan of the head and CT angiography
B. Give anti-emetics and naproxen 500 mg by mouth
C. Administer 10 ml of Oromorph and 2 mg diazepam to calm her down
D. Discharge her from the department as she does not have a life-threatening problem, and advise her to see her GP the next day
E. Administer 6 mg subcutaneous sumatriptan and await its effect

Question 2

A 35-year-old man presented with a week's history of progressive 'all-over' headache, described as a dull ache that he said he 'just cannot shift' despite taking regular paracetamol. In the past day, he has felt disabled by the headache and has had to stay upright or seated, as it is worse lying down. There is some mild nausea but no vomiting. He has occasionally experienced a hangover headache but no regular headache in the past. He feels 'under the weather' with the headache, with some visual blurring and non-specific fatigue. He has HIV and had been stable on antiretroviral agents but had run out of medication 2 weeks previously. This coincided with moving to the UK from Mexico. He did not know the names of the drugs, and no records are available.

On examination, he appears thin. His temperature is 37.7°C, heart rate 100 beats/minute (sinus rhythm), and blood pressure 133/85 mmHg. He appears comfortable in the bright lights and loud sounds of the acute medical unit. There is no rash or signs of meningeal irritation, and neurological examination, including visual acuities and visual fields to confrontation, is normal. Fundi are not visualized.

What is the most appropriate next step in management?

A. Investigate further for HIV and arrange an urgent CT head scan
B. Discharge him with a trial of naproxen and metoclopramide for 5 days
C. Arrange an outpatient eye clinic review in due course and give analgesia in the interim for the headache
D. Organize a CT head and CT venogram for the following day with lumbar puncture afterwards
E. Administer subcutaneous sumatriptan and await the response

Question 3

A 70-year-old woman presented in general practice with muscle aches and pains and headache after a flu-like illness. The flu was accompanied by a runny nose, cough and fever. The headache is left-sided, around her temple, continuous but worse if she touches the area, or ties her scarf around her head and face. She

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finds it difficult to lie on that side of her head at night. She still feels a bit feverish. There is some pain on eating. She has twice had momentary loss of vision in the left eye. There is no past history of headache.

On examination, her temperature is 37.6°C, heart rate 105 beats/minute, and blood pressure 125/90 mmHg. She looks somewhat flushed and unwell. There is some tenderness to palpation at the left temple but no other abnormality.

What is the best immediate management in primary care at this stage?

A. Prescribe co-amoxiclav for 1 week
B. Reassure her that the symptoms will resolve
C. Refer her for hospital admission
D. Perform blood, throat and urine cultures and prescribe paracetamol and fluids
E. Prescribe prednisolone 1 mg/kg orally

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