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Management of chronic headache

Background

Headache remains the most common cause of neurological consultation in clinical practice for which correct diagnosis and treatment are essential.

Objective

This article provides a review of headache presentation and management, with an emphasis on chronic headaches and the differentiation between migraine and tension-type headache (TTH).

Discussion

By far the most important diagnostic tool for proper headache diagnosis is the taking of a concise and representative history of the headaches. Migraine and TTH exist along a continuum and identification of the patient's position on this continuum has important implications for management.

Keywords

headache disorders; tension-type headache; migraine; therapeutics

CPD

Headache remains the most common cause of neurological consultation in clinical practice[1,2] for which correct diagnosis and treatment are mandatory. The International Headache Society (IHS) produced an International Classification of Headache Disorders of which the third edition was published this year.[3] This process involved working groups investigating primary headache disorders and headaches attributed to trauma or injury, vascular and non-vascular disorders, psychiatric disorders, substance abuse and several other contributing factors.[3] Olesen reinforced such classifications, stating that they enhanced '…better recognition (and therefore management)…' while also contributing to headache research.[4,5]

Although such classifications first appeared in the 1960s, there remain those who consider primary headaches to reflect a continuum in which the nature, character and quality of the headaches change with time.[2,6] In a study of almost 100 patients with chronic headache history of 20 years median duration, there was a significant change in headache diagnoses, which moved from migrainous to tension-type headache (TTH) with the passage of time.[6] The purpose of this article is to review the presentation and management of headaches, with an emphasis on chronic headaches and the differentiation between migraines and TTH.

Definitions

Diagnosis remains the cornerstone of headache management but may also be subject to external factors. In the United States, insurance rebates for TTH may be either much lower than that for migraine or there may be no remuneration at all for TTH.[7] This creates bias that results in under-representation of the true extent of TTH, which is the most prevalent type of headache.[8] The 2013 IHS classification divided primary headaches into migraines, TTH, trigeminal autonomic cephalalgias and other primary headache disorders.[3]

Researchers define 'chronic headache' on the basis of frequency (≥15 days per month) and duration (≥4 hours per headache day)[3,6] over the preceding 6 months and it may include either TTH or migraine. This necessitates at least half a year of headache history, a concept that is often unacceptable to the patient and family physician. Patients and family physicians are often concerned about headaches lasting less than a month and consider these as 'chronic' even if they do not adhere to

the formal definition. The initial imperative for the family physician is to differentiate primary headache types, particularly TTH and migraine, from secondary headaches, which may require emergency intervention.

Symptomatic/secondary headaches

There are symptoms that should raise red flags to alert clinicians to consider more serious diagnoses rather than primary headaches. Sudden onset, severe headache (often referred to as thunderclap headache)[9] may herald subarachnoid haemorrhage or intracranial haemorrhage, vertebral artery dissection, cerebral venous thrombosis or reversible cerebral vasoconstriction syndrome.[8,9] Headaches exacerbated by coughing, straining or sneezing raise concerns of raised intracranial pressure.[10] If headaches are provoked by posture, such as stooping, imaging is required to exclude some of these headaches, which require emergency intervention. Associated neurological features, such as sensory changes, weakness, diplopia (including sixth cranial nerve palsy), Horner's Syndrome or visual field defects necessitate further investigation.[2,8] Exacerbation with eye movement and impaired vision may suggest retrobulbar neuritis.[2] Enlarged blind spot suggests papilledema or raised intracranial pressure.[2]

Headache with stiff neck, nausea and vomiting, recent onset of confusion, altered consciousness and/or fever raises concerns of infection, such as meningitis or encephalitis, and requires hospital admission and lumbar puncture.[8] If in doubt, the family physician, when faced with a red flag (*Table 1*), should seek further advice as soon as is practicable.

Differentiating between tension-type headache and migraine

Most patients call all bad headaches 'migraines'. This is not unexpected, particularly as there may be a skew in the epidemiological approach to migraine, as discussed above. For those who ascribe to the continuum model of headaches (*Figure 1*),[2] patients can have features of both TTH and migraine. Even those strictly adhering to the IHS classification[3] acknowledge the potential for coexistence of both headache types. Where it is difficult to differentiate between TTH and migraine, some have adopted the term 'tension-vascular headaches' (a term not included in the IHS classification)[3] to denote a headache type that has features of both TTH and migraine but has therapeutic ramifications (*Figure 1*).[2]

The most important diagnostic tool for proper headache diagnosis is the taking of a concise and representative history of the headaches. It is important to question all reported symptoms. The history and description of headache can change with time.[6] The history should include:

- how long the patient has had headaches
- the nature of the pain (eg. tight and gripping, pulsating and throbbing, or stabbing and lancinating)
- the site of the pain (eg. frontal and/or occipital and vertex, band-like, temporal or retro-orbital, unilateral or bilateral)
- possible association with visual symptoms, which may include teichopsia, fortification spectra or blurring of vision
- gastrointestinal symptoms, such as nausea and/or vomiting
- accompanying features, such as photophobia, phonophobia and/or osmophobia
- the frequency and duration of the headache, and precipitating and relieving factors.

Once these data have been obtained, it should be simple to differentiate between the two most common types of chronic headache, namely TTH or migraine (*Table 2*). Where such differentiation proves difficult, the term 'tension-vascular headache' may be acceptable.

Table 1. Red flags, which should alert the family physician to seek further investigation

- Headache exacerbated by coughing, sneezing or straining
- Headache provoked by postural change (stooping or bending)
- Headache associated with eye movement and blurred vision
- Headaches of sudden, severe onset (thunderclap) – worse than previous headache
- Headaches with new-onset neurological signs (sensory changes, weakness, diplopia, Horner's Syndrome, visual field defects)
- Headaches associated with stiff neck, generalised aches/pains, rash, malaise, altered consciousness or confusion
- Headaches that have changed dramatically in quality, nature or site
- Headaches failing to respond to appropriate therapy

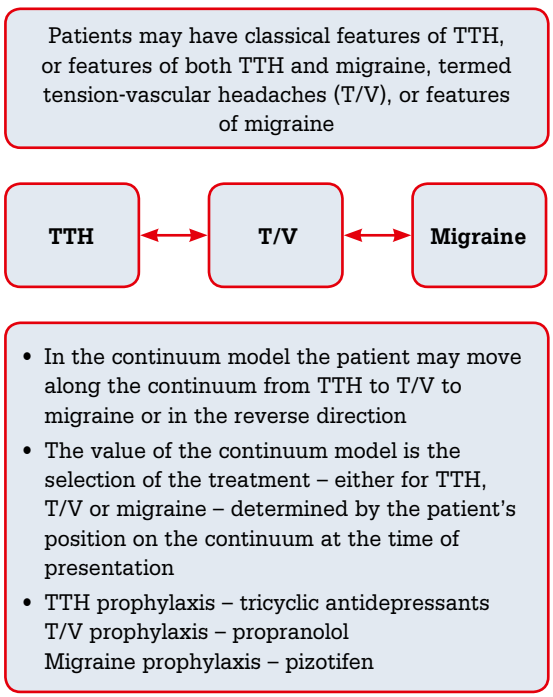

Figure 1. Continuum of headache model

Although a concise history is the backbone of all neurological diagnoses, one should not ignore the need for a comprehensive physical examination. The use of an ophthalmoscope may be imperative in the examination of the patient who presents with headache.[2] It is important to exclude raised intracranial pressure as this may identify a medical emergency. The identification of localising neurological signs has diagnostic relevance. One should not ignore features such as stiff neck, skin rash or altered states of consciousness, as infection is important and may reflect a need for emergency intervention.

Depending on the jurisdiction, migraine is often represented as the most common headache type (especially where remuneration for TTH is negligible) but epidemiology suggests a prevalence of 5% for true migraine and 27% for TTH.[11] It must be acknowledged that TTH can be severe and intrusive[12] and that such severity alone does not differentiate between TTH and migraine. Patients may have a headache, largely indistinguishable from TTH, caused by analgesic overuse and it is imperative, when taking a history, to explore the use of medications. It may be necessary to restrict the use of such agents and to warn patients that this may result in short-term exacerbation of their headache.[13]

The American Academy of Neurology developed practice parameters for migraine,[14] which are also applicable for TTH as the border between these types of headache may be blurred. Treatment should commence according to headache type, rather than chronicity,[2,6,8,15] but where traditional intervention fails, additional agents, such as gabapentin (and hence also pregabalin) and levetiracetam, may offer additional benefit.[8,15]

Other headache types

Having reviewed TTH and migraine as the most common primary headaches, it must be acknowledged that there are many more headache types included in the IHS classification.[3] Most of these are the precinct of the specialist and of research interest but some deserve special consideration when discussing the role of family physicians in managing chronic headache.

Cluster headache

Cluster headache does not merely mean headaches that occur close together and is often overdiagnosed by family physicians. It is a relatively rare and distinctive type of headache.[2,3,16,17] Such headaches may have an identifiable precursor, such as occurring in the morning after a 'big night out' or they may be associated with significant alcohol consumption. They often occur at the same time of day for a finite period, often in the early morning, possibly waking the patient at the same time each day. They are often accompanied by suffused red eye with ipsilateral conjunctival injection, lacrimation and stuffy nose with ipsilateral nasal congestion or rhinorrhea, accompanying unilateral pulsating pain in the ipsilateral forehead, possibly associated with eyelid oedema, meiosis and ptosis. Diagnosis dictates at least five attacks, which can occur from one every other day to eight per day, devoid of an alternative aetiology. This type of headache should initiate specialist consultation and may be treated with agents such as verapamil or lithium.[8]

Table 2. Differentiating between TTH and migraine

	TTH	Migraine
Nature of pain	Tight gripping pressure, constant	Throbbing, pulsating
Site of pain	Bitemporal, occipital or generalised (may be retro-orbital, may be band-like)	Unilateral (often in temple or retro-orbital)
Associated features	Possible blurred vision May have nausea (rarely vomits) Usually no: • photophobia • phonophobia • osmophobia May be associated with sleep disturbance.	Teichopsia (zigzag, bright, shimmering lights) Fortification spectra (like top of fortress) Rainbow effect Photons of bright light in visual field Nausea and vomiting Photophobia Phonophobia Osmophobia
Precipitating factors	Often at times of stress	Often after stress has passed Smells Foods (eg. chocolate) Alcohol Hormonal changes
Acute intervention	Rest Simple analgesia Alcohol may reduce symptoms	Triptans Ergots Occasional early use analgesics
Prophylaxis	Tricyclic antidepressants: • amitriptyline if difficulty sleeping • imipramine if no problem sleeping	Pizotifen

Cervicogenic headache

Cervicogenic headache is another type of headache, often so labelled at the time of referral. It is thought to emanate from the spinal trigeminal nucleus[3,18] but is often clinically indistinguishable from TTH,[2] although trigeminal autonomic cephalalgias were the subject of a special focus group during the development of the IHS Classification.[3] These headaches will often respond to the treatment paradigm for TTH and can often be effectively managed by the family physician.

Treatment of chronic headaches

Where headaches occur less frequently than once per fortnight, it seems acceptable to use pulse therapy, such as simple analgesia with aspirin, diclofenac, ibuprofen, naproxen, paracetamol or similar agents. These may be all that is required for either TTH or migraine if treated early.[8] Such intervention should be complemented by a healthy dose of reassurance, as is best provided by the family physician.

Where such agents have been suboptimal for the treatment of migraine, triptans (sumatriptan, zolmitriptan, rizatriptan, naratriptan or elmatriptan) may be more efficacious for acute intervention.[8] Ergot derivatives may offer a cheaper alternative with equal efficacy.[2] Opioid analgesics should be considered as a last resort for acute headache management and if required should provoke a red flag consideration. Where headaches occur more frequently than once per fortnight, prophylaxis should be offered and the choice determined by the headache type, as set out within the continuum model (*Figure 1).*

TTH is best managed with tricyclic antidepressants (amitriptyline where sleep disturbance is a prominent feature and imipramine where sleep does not pose a problem). Tension-vascular headache can be managed with beta-blockers (propranolol), and migraine is treated with pizotifen.[2,8] The operative rule with all these agents is to 'start low and go slow' but to escalate treatment until achieving either efficacy or intolerable side effects.

Tricyclic antidepressants can be started with as little as 10–25 mg nocte up to a maximum of 75 mg[8] but are often abandoned if they are ineffective at those doses, hence discarding a potentially effective remedy. Doses as high as 200 mg or 250 mg nocte may be required for unremitting headaches[2] but escalation to such high levels necessitates close observation and evaluation. It is important to warn patients of potential unwanted effects, such as 'drying out', which is caused by the anticholinergic properties of the tricyclics. Patients often interpret the dry mouth as hunger, because eating produces salivation, but this may lead to weight gain and thus irrigating a thirst, rather than feeding it, should be advocated. Issues such as possible fatigue, impairment of driving capacity or gastrointestinal disturbance should be discussed. Palpitations may occur and the patient should report when experiencing these.

Tension-vascular headaches are often responsive to beta-blockers, such as propranolol. Again the aim is to 'start low and go slow', starting at 10–40 mg twice daily and titrating up as needed. It is important to ask about the patient's potential for asthma or heart disease and to monitor heart rate and possibly blood pressure. As discussed above for tricyclic antidepressants, many advocate stopping propranolol at too low a dosage (160 mg daily).[8] Doses as high as 160 mg four times daily may be required for unremitting headaches,[2] but again such high doses necessitate close supervision and detailed monitoring. It cannot be overemphasised that treatment should be tailored to the patient's needs and the patient should not be on either too little or too much medication; however, abandoning a potentially efficacious treatment at too low a dosage may deny the patients their best option for a satisfactory outcome.

Pizotifen is the agent of choice for prophylaxis of migraine. Again the 'start low and go slow' adage prevails. Dosage starts at 0.5 mg twice daily but again too low a dosage may be suggested as the maximal acceptable (3 mg daily).[8] Doses as high as 4.5 mg daily may be necessary to achieve adequate prophylaxis.[2] Fatigue and hunger are the two most common adverse effects associated with pizotifen, although all medications may cause nausea, vomiting, diarrhoea, constipation and skin rash.

Agents such as sodium valproate, topiramate and verapamil have been advocated for migraine prophylaxis.[8] Other antiepileptic medications, such as gabapentin,[15] pregabalin (now approved for neuropathic pain) or even levetiracetam[6] may have a role in chronic headache but are the domain of the specialist and will not be discussed further in this article. Similarly, the role of botulinum toxin may be considered for headache management[19] but will not be discussed in this paper as it is usually the domain of the specialist.

Should the headache not respond to treatment as would have been expected, or the quality or site of the pain changes, specialist advice should be sought. Although chronic headache may pose a diagnostic dilemma, it is better to overreact and to seek assistance for those with longstanding, refractory headaches because complacency may have dangerous consequences for the patient.

Non-pharmacological intervention

There are a number of important issues to consider regarding non-pharmacological treatment of headaches but perhaps the most important is to remind family physicians of the very serious consequences that may ensue following neck manipulation, which can cause dissection, stroke or even death.[20]

TTH and migraines are often worse within the context of stress[21] and it behoves a family physician to explore factors that exacerbate the patient's stress and seek ways to relieve the stress. The family physician is often well-placed to delve into such private domains and to provide intimate counselling. Lifestyle issues, sleep pattern and other possible contributing factors, such as sleep apnoea, should not be ignored. If sleep apnoea is considered a contributing factor, polysomnography is an appropriate intervention.[22] This will necessitate referral to a sleep physician and will not be discussed further in this paper.

Accompanying features, such as arthritis, hypertension, obesity and other associated diagnoses also require attention within the context of headache management. Family physicians are best placed to treat the whole patient, rather than focusing on just one aspect, as may occur within specialist practice.

Conclusion

Chronic headache is the most common neurological complaint to present to the family physician. This overview has offered a practical approach to the management of chronic headache, provided clues to differentiate between TTH and migraine (the two most common primary headache types to present to the family physician), and discussed treatment options, red flags, which necessitate more detailed consideration, and referral for specialist opinion.

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References

1. O'Flynn N, Risdale L. Headache in primary care: how important is diagnosis to management? Br J Gen Pract 2002;52:569–73.
2. Beran RG. Headache. In: Neurology for General Practitioners. Sydney: Elsevier, 2012;45–55.
3. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders. Cephalalgia 2013;339:629–808.
4. Olesen J, Steiner TJ. The International Classification of Headache Disorders. 2nd edn (ICDH–II) J Neurol, Neurosurg Psychiatry 2004;75:811–12.
5. Olesen J. The International Classification of Headache Disorders. Headache 2008;48:691–93.
6. Beran RG, Spira PJ. Levetiracetam in chronic daily headache: a double-blind, randomised placebo-controlled study (The Australian Keppra Headache trial [AUS-KHT]). Cephalagia 2011;31:530–36.
7. Levin M. Making a headache practice work: the elements of diagnosis and coding in headache medicine. Headache 2008;48:491–96.
8. Therapeutic Guidelines. Headache. In: Neurology, Version 4, 2011;73–99.
9. Schwedt T, Matharu M, Dodick D.Thunderclap headache. Lancet Neurol 2006;5:621–31.
10. Dodick DW. Clinical clues and clinical rules: primary vs secondary headache. Adv Stud Med 2003;3:550–55.
11. Fong KJ. Recent advances in the diagnosis and management of primary headache disorders. Medical Session, June 2002. Available at www.fmshk.org/database/articles/821.pdf [Accessed 6 February 2014].
12. Loder E, Rizzoli P. Tension-type headache. BMJ 2008;336:88–92.
13. Paemeleire K, Crevits L, Goadsley PJ, Kaube H. Practical management of medication overuse headache. Acta Nuerologica Belgica 2006;106:43–51.
14. Silberstein SD. Practice parameter: evidence-based guidelines for migraine headache (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2000;55:754–62.
15. Spira PJ, Beran RG. Gabapentin in the prophylaxis of chronic daily headache – a randomised, placebo-controlled study. Neurology 2003;61:1753–59.
16. May A. Cluster headache: pathogenesis, diagnosis and management. Lancet 2005;366:843–55.
17. Beck E, Sieber W, Trejo R. Management of cluster headache. Am Fam Physician 2005;71:717–24.
18. Biondi DM. Cervicogenic headache: a review of diagnostic and treatment strategies. J Am Osteopathic Assoc 2005;105:16S–22S.
19. Jackson JL, Kuriyama A, Hayashino Y. Botulinum toxin A for prophylactic treatment of migraine and tension headaches in adults: a meta-analysis. JAMA 2012, 307:1736–45.
20. Beran RG, Schaeffer A, Sachinwalla T. Serious complications with neck manipulation and informed consent. Med J Aust 2000;173:213–14.
21. Cathcart S, Winefield AH, Lushington K, Rolan P. Stress and tension-type headache mechanisms. Cephalalgia 2010;30:1250–67.
22. Rains JC, Poceta JS. Headache and sleep disorders: review and clinical implications for headache management. Headache 2006;46:1344–61.

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