Chronic daily headaches

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Abstract

Chronic Daily Headache is a descriptive term that includes disorders with headaches on more days than not and affects 4% of the general population. The condition has a debilitating effect on individuals and society through direct cost to healthcare and indirectly to the economy in general. To successfully manage chronic daily headache syndromes it is important to exclude secondary causes with comprehensive history and relevant investigations; identify risk factors that predict its development and recognise its sub-types to appropriately manage the condition. Chronic migraine, chronic tension-type headache, new daily persistent headache and medication overuse headache accounts for the vast majority of chronic daily headaches. The scope of this article is to review the primary headache disorders. Secondary headaches are not discussed except medication overuse headache that often accompanies primary headache disorders. The article critically reviews the literature on the current understanding of daily headache disorders focusing in particular on recent developments in the treatment of frequent headaches.

Key Words

Chronic daily headache, chronic migraine, chronic tension type headache, hemicrania continua, new daily persistent headache

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Introduction

Chronic Daily Headache (CDH) is a descriptive term and not a diagnosis per se. It is commonly defined as headaches occurring on 15 or more days in a month for at least three months and affects around 4% of the general population.[1] It causes significant distress with substantial impact on the quality of life of an individual and huge economic cost to the society through occupational disability and healthcare consultations. In comparison to episodic headache disorders, CDH is less responsive to acute and preventive treatments.

The term CDH is mainly referred to the primary headache disorder, although secondary CDH must be excluded. Common secondary causes are given in Table 1. The primary CDH is divided into short and long duration [Tables 2 and 3]. The short duration i.e. lasting < 4 hours include various trigeminal autonomic Cephalalgias (TAC) including cluster headaches (CH), paroxysmal hemicrania (PH) and others such as hypnic headaches, primary stabbing headaches etc. Those that last > 4 hours include chronic migraine (CM), chronic tension-type headaches (CTTH), hemicrania continua (HC) and new daily persistent headaches (NDPH) although CM and CTTH account for the vast majority.[2] Medication overuse headaches (MOH), essentially a secondary headache disorder commonly accompany the primary headache disorders and are described with them.[3-5]

CDH may evolve from episodic headache through gradual transformation over months to years. An estimated 3-6% of patients move from episodic to chronic and vice versa each year.[6] Many risk factors have been identified that predict the development of CDH,[3] in particular CM, some of which are modifiable [Table 4]. It is imperative to identify sub-types of CDH to appropriately manage the condition.

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Chronic Migraine

Headaches account for 1 in every 10 consultations with the general practitioner and around 30% of out-patient neurology referrals in the UK. Migraine is the most common headache to require consultation and is ranked by the World Health Organization as 19th among all causes of years lived with disability.[7] An estimated 20 million work days are lost in England every year due to migraine that cost 20 billion to the economy in both direct and indirect cost.[8] In 75% of patients, a migraine attack causes functional impairment and in 50% of the attacks, patients seek help from others and/or have significant impact on social activities.[9] The episodic variety is characterised by headaches that are unilateral, throbbing,

Table 1: Common secondary CDH

Medication overuse
Post-traumatic headaches
Temporal arteritis
Idiopathic intracranial hypertension
Spontaneous intracranial hypotension
Others
Degenerative disease of the cervical spine, temporomandibular joint dysfunction, errors of refraction and chronic sinusitis rarely cause chronic daily headache but simple to treat.

Table 2: Common short duration primary CDH

Trigeminal autonomic cephalalgia (TAC)
Cluster headaches
Paroxysmal hemicrania
Short-lasting Unilateral Neuralgiform headaches with Conjunctival Tearing (SUNCT)
Other short duration primary CDH
Trigeminal neuralgia
Idiopathic stabbing headaches
Hypnic headaches

Table 3: Long duration orimary CDH*

Chronic migraine
Chronic tension type headaches
New daily persistent headache
Hemicrania continua

* All CDH may be associated with medication overuse

Table 4: Risk factors for CM

Modifiable risk factors
Medication overuse
Obesity
Snoring and sleep disturbance
Smoking
Caffeine consumption
Psychiatric co-morbidity (depression, anxiety)
Non-modifiable Risk factors
Female
Caucasians
Unmarried
Low socioeconomic status
Previous head or neck injury

and moderate to severe, aggravated by physical activity and associated with nausea, vomiting and/or photophobia, phonophobia that last between 4-72 hours with complete freedom of symptoms between attacks.

The concept of high frequency migraine has been in the literature for a long time with terms such as transformed migraine, mixed headache syndrome and evolutive migraine. The term CM was introduced in the International Classification of Headache Disorders – second edition (ICHD-II) and defined as headaches occurring on 15 or more days per month for at least 3 months of which at least 8 days with headaches fulfilling the criteria for migraine[1,10] [Table 5].

The definition of CM is not universally accepted and there is lack of consensus among professionals on its application in clinical practice. As a result it is difficult to estimate the true prevalence although between 1.3 – 2.4% of the overall population may have the condition.[2] Progression from episodic to chronic migraine is seen in 6% in the population-based and 14% in clinic-based studies.[11,12] However, the total number of CM remains static at 2.5 – 4.6% of the general population.[13]

CM represents the most disabling form of the headache disorder and its impact on Health Related Quality of Life (HRQoL) is greater than episodic migraine (EM) as measured with Migraine Specific Questionnaire (MSQ);[14] patients with CM are more likely to miss work or activities of daily living, more likely to be unemployed and have relationship difficulties and family problems.[15] Co-morbidities such as anxiety, depression and co-existent pain are more common and 76% - 95% of patients report recognisable triggers. Patients with sleep disturbance, obesity, frequent migraine attacks and analgesic overuse are more prone to develop CM.[16,17] Opiates and combination analgesics with barbiturates and caffeine are particularly associated with progression to CM than with triptans..[11] Non-steroidal anti-inflammatory drugs (NSAID) are protective in those with low frequency headaches but not in those with high levels of monthly headache days.[18]

The recognition and management of CM poses considerable challenge to primary care physicians. The disorder generates more referrals to secondary or tertiary care and accounts for more emergency care visits than the episodic form.[19] In addition a large number of patients with CM overuse analgesics and the jury is out as to whether analgesic overuse

Table 5: Chronic migraine (Olesen *et al.* 2006)

Headache (tension-type and/or migraine) on 15 or more days per month for at least 3 months.
Occurring in a patient who has had at least 5 attacks fulfilling criteria for migraine without aura.
On 8 or more days per month for at least 3 months, headache has fulfilled criteria for pain and associated symptoms of migraine without aura (Criteria a and b below) or was treated and relieved by triptan (s) or ergot before the expected development of symptoms listed in criteria a and b.
Has at least two of the following
Unilateral location
Pulsating quality
Moderate or severe pain intensity
Aggravation by or causing avoidance of routine physical activity.
Has at least one of the following
Nausea and/or vomiting
Photophobia and phonophobia
No medication overuse and not attributed to another causative disorder.

is a complication or a cause of CM. Those taking painkillers for other conditions such as arthritis or back pain only develop MOH if they had previously suffered from migraine or have a strong family history of primary headache disorder.[20,21] Similarly patients with TAC who overuse painkillers also have co-existing migraine.[22] There are patients with analgesic overuse with a better outcome on withdrawal than others raising the question of genetic susceptibility.[23]

It is postulated that patients with CM have a high and more persistent cortical excitability than those with EM.[24] A PET study in 10 patients with CM has shown increased metabolism in pons and right temporal cortex with reduced metabolism in several other areas bilaterally such as medial frontal, parietal, and somatosensory cortices and caudate nuclei suggesting impaired normal inhibitory capacity.[25] There is suggestion that change in the nociceptor threshold and pain pathways occur as progression occurs in EM.[26] Patients with CM have significantly more allodynia.[27] Cutaneous allodynia is believed to represent sensitization of 2nd order brainstem trigeminal neurons.[28] It has been shown that presence of cutaneous allodynia is associated with poor response to triptans[29] and unlike EM, allodynia remains on headache free days in those with CM.[30] A high CSF concentration of vasoactive neuropeptides suggests persistent trigeminovascular activation.[31] Central sensitization due to neurogenic inflammation may lead to chronicity.[32] Functional imaging has shown activation of dorsal rostral pons in CM.[33] There is also increased iron deposition in the periaqueductal grey, red nucleus, globus pallidus and putamen of patients with CM possibly a result of frequent migraine attacks.[34] Despite the fact that some patients with EM evolve into CM, the above inferences suggests that CM may well be a unique condition distinct from EM. It is likely that patients with CM have a low pain threshold and abnormal cortical processing of cutaneous nociceptive input.[35]

Patients with CM, mostly female with a history of EM without aura (in the vast majority) for many years, gradually progress to daily or near daily headaches. The associated migraine symptoms of nausea, photophobia and phonophobia get less frequent and milder [36,37] and the clinical picture appears to be a mixture of migraine and tension-type headache. The history of transformation is not always present and hence the term CM is preferred by the IHS. Progression to CM may occur without medication overuse although medication overuse accompanies headache in up to 80% of patients seen in the specialist headache clinics.[38] Due to the high prevalence of medication overuse in CM the IHS in their guidelines for CM trials have allowed these subjects to be included provided they are stratified accordingly[39] and a revision of the International Committee on Headache Disorders (ICHD-IIR) is expected based on the proposal by the experts that inclusion of medication overuse patients should be allowed within the classification of CM to accurately reflect the patient population seen in actual clinical practice.[1] In essence medication overuse may be a complication of CM rather than a separate entity. A well maintained headache diary would address issues of medication overuse, confirm the number of days of headaches and migraine and may help identify triggers.[40]

For effective management of CM, analgesic overuse must be addressed. There is lack of consensus on whether preventive treatment is introduced before or after detoxification.[41,42] The fact that early intervention may reduce efficacy of preventive drugs is questioned in some studies.[43] The only controlled trial evidence comes from a small study of topiramate in CM in which patients with medication overuse responded equally to those without overuse[44] although the absolute response rate was far less than seen in other clinical trials with Topiramate.[45] It is also debated as to whether patients with CM should treat the acute attacks earlier as critics argue that such approach may lead to medication overuse.[46] As a matter of fact these patients should be given preventive treatment to make acute treatment effective and reduce attack frequency.

The preventive treatments used for EM such as tricyclic antidepressants, beta-blockers, anti-convulsants, calcium-channel blockers have not been evaluated in CM. These drugs may well be effective in CM although majority of them have been in the market for decades; are generic and cheap to prescribe, and hence unlikely that any data in CM prophylaxis will emerge. Topiramate has the best available evidence in two studies[44,47] one of which included patients with medication overuse.[44] There is also evidence that its use in EM may prevent transformation to CM.[45] The discontinuation rate for topiramate is around 25% in clinical trials mostly due to adverse events.[47] Paraesthesia is the most common adverse event although cognitive impairment is the main reason for discontinuation. Most of the adverse events occur in the first 6 weeks of titration period.[48,49] Topiramate works through modulation of trigeminovascular system with reduced nociceptive transmission to the CNS and by inhibiting cortical spreading depression.[50]

Some other drugs mainly studied in patients with CDH and transformed migraine show promising results. In an open-label study on 30 patients with transformed migraine, valproate was found to be effective in reducing headache days and disability[51] while a review of 138 CDH patient diaries treated with valproate monotherapy showed a 65% reduction in migraine frequency.[52] Other drugs with some benefit in CDH and transformed migraine include Gabapentin,[53] Tizanidine,[54] Fluoxetine,[55] amitriptyline[56] and Levatiracetam.[57] Memantine[58] (an NMDA antagonist) has recently been reported to induce remission in CM.

A considerable number of patients with CM remain refractory to the abovementioned preventive treatment or are unable to continue due to intolerable side effects. Goadsby *et al*.[59] recommend that patients who fail to respond to at least four classes of preventive treatment should be considered for more invasive treatment options such as greater-occipital nerve block (GON) and occipital nerve stimulator (ONS). There is also the option of treatment with OnabotulinumtoxinA before resorting to these invasive and expensive options.

OnabotulinumtoxinA is obtained from bacteria Clostridium Botulinum. The exact mechanism of action in preventing headaches in CM is uncertain, although it is postulated that reduction in central sensitisation through inhibition of peripheral nociceptive fibres may be responsible for pain relief. Such inhibition prevents the release of neuromediators such as substance P, glutamate and CGRP. The efficacy of

Botulinum Toxin A in headache date back to early 90's when patients' receiving cosmetic treatment reported improvement in migraines. Various trials using different doses and injections sites in early studies did not show any evidence of its use in episodic headache disorders including EM. The evidence for its efficacy in CM came from two pivotal studies (PREEMPT 1, 2)[60,61] and the pooled analysis of the two trials.[62] A total of 1384 patients were studied; two third of whom were overusing acute medications. The primary outcome measure of reduction in headache episodes (defined as headaches lasting at least 4 hours) from the baseline was not met in PREEMPT 1 and was switched to headache days in PREEMPT 2 before completion of the study. This was based on the results of the PREEMPT 1 and recommendation from the Food and Drug Administration (FDA). The PREEMPT 2 achieved the primary outcome measure and the pooled analysis showed that the trials achieved both primary outcome measures i.e. headache episodes and headache days. Those receiving Botox had two less headache days than placebo which was statistically significant although critics argue whether such response is clinically meaningful considering potential cost of such treatment. There was a high placebo response of 30% compared to 40% in the active group with no difference between groups in the reduction of intake of acute medicines. Critics feel an unimpressive 10% net response in the active group could be due to lack of blinding as a result of the anti-wrinkling effect of Botox, although a very high placebo response is against this critique. It is argued that two third of patients overusing medication did not fulfil the criteria for CM and were in fact suffering from MOH and could have responded to appropriate withdrawal of the overused medication.

The evidence was adequate for the Medicine and Healthcare Regulatory Agency (MHRA) in the UK and FDA in the USA to approve the drug, although their decision was met with strong criticism from some leading headache experts.[63,64] The criticism was further strengthened by Drugs and Therapeutic Bulletin (DTB) who concluded that the evidence was limited and it was difficult to see the place of Botox in CM prophylaxis.[65] The proposal for NHS funding was rejected by the Scottish Medicine Consortium (SMC) last year, although NICE has just published their final appraisal determination through single technology appraisal recommending the treatment on the NHS to those who fail to respond to three preventive treatments. The treatment be stopped if there is <30% response on two consecutive treatments (negative stopping rule) and in those who respond once they revert to EM (positive stopping rule).[66]

GON blockade with local anaesthetic and steroids such as betamethasone, triamcinolone or methylprednisolone[67-69] has been used for CH and other TAC for some years. A double-blind placebo-controlled study has shown its efficacy in CH with 80% improvement and the effect lasting 4 weeks.[68] Its use in intractable CM remains a choice for various headache experts in the absence of robust evidence from trials. Focal alopecia and increased pain at the injection site is reported in some patients.

ONS through peripheral neuro-stimulation has shown promising results in intractable CM with medication overuse[33,70] with 84% showing 50% improvement. A prospective, multi-centre, single-blind randomised feasibility study (ONSTIM) showed a 39% responder rate (50% reduction of headache days) compared to 8% in the control group.[71] The procedure is safe in experienced hands and so far no neurological deficit reported post-operatively. MR imaging and Indomethacin challenge is mandatory before the procedure is considered. Most patients would have the procedure again and recommended it for others.[72]

Medication Overuse Headache

The IHS used the term medication overuse headache (MOH) in their 2nd edition of classification (ICHD-II, 2004), although this was revised with details of various sub-types by Silberstein in 2005[73] [Table 6]. The revised definition used the term "probable MOH" until the headaches improved on medication withdrawal. As this definition was retrospective and less sensitive, an appendix criterion was published in 2006 [Table 7].

MOH is the most common headache to present in the specialist headache clinic and an estimated 1-1.5% of the population suffers from this condition and accounts for 50-80% of patients presenting to a tertiary headache clinic.[74] It is 3.5 times more common in females[75] and vast majority (90%) take more than one painkiller.[76] A meta-analysis of 29 studies indicate that nearly two third (65%) of patients with MOH suffer from migraine, 27% have tension-type headache and 8% report other primary headache disorders.[75]

Any analgesic can cause MOH although combination analgesics are the most common (39-42%) followed by simple analgesic (29-38%), triptans (12-20%), opioids (6%) and ergotamine (4-11%).[77] The risk is higher in smokers, obese patients and those with previous substance overuse including alcohol.[78,79] The risk of progressing to CM is higher in those taking barbiturates (OR 1.73) followed by opiates (OR 1.4), triptans (OR 1.07) and NSAID (OR 0.97). NSAIDs like triptans induce progression in those with more than 10-14 days headache days per month.[80] However, combined with barbiturates or caffeine, NSAIDs can induce headache after a short period and with use of lower doses.[81] MOH develops faster and in a much lower dose with triptans than ergot or simple analgesic alone. Similarly withdrawal symptoms are much milder and shorter with triptans than others.[82,83]

Physical dependence with opioids and barbiturates can occur[84] while psychological mechanism may operate among those taking painkillers in anticipation of headache or fear of missing a social occasion. Some experts suggest that early treatment of a migraine attack may increase the risk of MOH; therefore, such advice is better placed for those with pure migraine or those who can differentiate migraines from other headaches. Patients must be warned about the risk of development of MOH. It is postulated that repeated exposure to the same substance sensitises the central receptors or reduce threshold of activation. Frequent intake of triptan may lead to down-regulation of 5-HT receptors and change central inhibitory pathways.[85] The periaqueductal gray matter is thought to be the potential site for such action. PET studies have shown significant but reversible changes in thalamus, anterior cingulated gyrus, inferior parietal lobe although irreversible changes are seen in the orbitofrontal cortex.[86]

Table 6: Revised ICHD-II diagnostic criteria for MOH (Silberstein 2005)

Medication overuse headache
Headache presents on ≥ 15 days per month fulfilling criteria c and d
Regular overuse for ≥ 3 months of one or more drugs that can be taken for acute or symptomatic treatment of headache
Headache has developed or markedly worsened during medication overuse
Headache resolves or reverts to its previous pattern within 2 months after discontinuation of overused medication
Sub-types of medication-overuse headache
Ergotamine-overuse headache
Ergotamine intake on ≥ 10 days per month on a regular basis for > 3 months
Triptan-overuse headache
Triptan intake (any formulation) on ≥ 10 days per month on a regular basis for > 3 months
Analgesic-overuse headache
Intake of simple analgesics on ≥ 15 days per month on a regular basis for > 3 months
Opioid-overuse headache
Opioid intake on ≥ 10 days per month on a regular basis for > 3 months
Combination analgesic-overuse headache
Intake of combination analgesic medications on ≥ 10 days per month on a regular basis for > 3 months
Medication overuse headache attributed to the combination of acute medications
Intake of any combination of ergotamine, triptans, analgesics, and/or opioids on ≥ 10 days per month on a regular basis for > 3 months without overuse of any single class alone.
Headache attributed to other medication overuse
Regular overuse for > 3 months of a medication other than those described above
Probable medication-overuse headache
Headache fulfilling criteria a, c and d for 8.2
Medication overuse fulfilling criterion b for any one of the sub forms 8.2.1 -8.2.7
One or other of the following:
Overused medication has not yet been withdrawn
Medication overuse has ceased within the last 2 months but headache has
Not yet resolved or reverted to its previous pattern.

Table 7: Appendix criteria for medication-overuse headache (Headache classification committee 2006)

Medication-overuse headache
Diagnostic Criteria
Headache present on ≥ 15 days per month
Regular overuse for > 3 months of one or more acute/symptomatic treatment drugs as defined under subforms of 8.2
Ergotamine, triptans, opioids or combination analgesicsmedications on ≥ 10 days per month on a regular basis for > 3 months
Simple analgesics or any combination of ergotamine, triptans, analgesics, or opioids on ≥ 15 days per month on a regular basis for > 3 months without overuse of any single class alone
Headache has developed or markedly worsened during medication overuse.

The headache experts agree that withdrawal of the overused medication should be the first priority in all cases as preventive treatment can only be fully effective after the overused medication is stopped.[43] The withdrawal also limits progression to chronic state, positively impact on pain coping behaviour and maximise response to acute treatment.[87,88] There is lack of consensus as to how (abrupt or gradual), where (in-patient or out-patient) and when (before or after preventive treatment) to detoxify. Opiates and those with barbiturates and caffeine must be withdrawn gradually due to unpleasant withdrawal effects[79] while other analgesics can be stopped abruptly. The duration of rebound symptoms is shortest with triptans (4 days) longer with ergot (6.7 days) and longest with NSAIDs (9.5 days).[89] An out-patient withdrawal with explanation and reassurance suffice in the vast majority of patients,[90] although a brief period of in-patient (usually a week) may be necessary for those who are not motivated or where out-patient attempts have failed. Cognitive behaviour therapy in such patient may be helpful. In the absence of robust evidence, the choice of preventive treatment before or after withdrawal remains with the treating physician. Topiramate has the best evidence for prevention in MOH although amitriptyline,[91] nortriptyline[92] and botulinum toxin may be effective.[93]

There is no clear guideline or consensus for treatment given for symptomatic relief during withdrawal. A brief period of prophylaxis with Naproxen 500 mg bd for 10-20 days has been recommended based on experience;[94] others use different dose for different duration.[95,96] The role of steroids remain controversial with some reporting benefits[97,98] although a bigger study has shown no role as a bridging treatment.[99] IV dihydroergotamine (DHE) with metoclopramide intermittently or as a continuous infusion in those not misusing ergot or triptan has shown good results.[95,100-102] Intravenous valproate,[103] prochlorperazine, injectable aspirin and oral tizanidine have been advocated by some experts.[104]

The success rate defined by reduction of headache days by 50% or more was 60-73% at 6 months[23,105] and 53% at 12 months.[43] The relapse rate was 38-42% over 5 years of which the highest risk was in the first year.[89,106] The risk of relapse is higher for tension-type headache (73%) than migraine (22%) and higher for combination analgesics (58%) than ergot (22%) and triptans (19%). The duration of drug overuse or primary headache disorder does not affect the relapse rate.

Patient education plays an important role in both preventing MOH and success of withdrawal treatment. The amount of painkillers is restricted to no more than 2 doses per week and those who overuse need to understand that as long as they continue to use the analgesics they will not improve.[95]

New Daily Persistent Headache

NDPH is a primary headache disorder with a prevalence between 0.03 – 0.1%[2,107] and defined as a new onset headache which becomes unremitting and persistent within three days of onset and lasts for at least 3 months [Table 8]. It is commonly seen in the fourth decade although any age may be affected; females are affected more where the disorder occurs at a younger age than males.[108-110] A previous headache history was present in 38% of patients and majority (82%) were able to recall the day of onset, a principal requirement in making this diagnosis.[108] Although a limit on the number of migrainous features were defined in the ICH II criteria, more migrainous symptoms are increasingly recognised and there is suggestion that this patient sub- group is more difficult to manage.[10,111] The NDPH are typically mild to moderate, non-pulsatile and bilateral that is not aggravated by exertion. A secondary cause for the headache should be ruled out.

Table 8: Diagnostic criteria for NDPH (ICHD-II, 2004)

Headache that, within 3 days of onset,[145] fulfils criteria B-D
Headache is present daily, and is unremitting, for >3 months
At least two of the following pain characteristics:
bilateral location
pressing/tightening (non-pulsating) quality
mild or moderate intensity
not aggravated by routine physical activity such as walking or climbing
Both of the following:
no more than one of photophobia, phonophobia or mild nausea
neither moderate or severe nausea nor vomiting
Not attributed to another disorder[146]

Headache may be unremitting from the moment of onset or very rapidly build up to a continuous and unremitting pain. Such onset or rapid development must be clearly recalled and unambiguously described by the patient.; History and physical and neurological examinations do not suggest any other disorder, or history and/or physical and/or neurological examinations do suggest such disorder but it is ruled out by appropriate investigations, or such disorder is present but headache does not occur for the first time in close temporal relation to the disorder.

The exact aetiology and pathophysiology of primary NDPH remains unclear. A variety of infectious agents have been identified as likely triggers. Flu like illness at the onset of the headache is present in a third and serological evidence for current or past EBV infection in two thirds of the patients.[108,112] Herpes Simplex and CMV are other viruses identified in 42% and 11% respectively.[113] Stressful life event, extracranial surgery and panic attacks are recognised non-infective triggers although a considerable proportion has no identifiable inciting factor.[114-116]

NDPH is highly refractory to currently available treatments particularly one with pronounced migrainous features;[117] there is an unmet need for research in to better treatments.[116] Medications that have been tried in NDPH include gabapentin, topiramate, venlafaxine, nortriptyline, mexiletine and botulinum toxin.[118,119]

Chronic Tension Type Headache

Chronic tension type headache (CTTH) is a featureless bilateral headache occurring on 15 days or more in a month for more than three months[10] [Table 9]. The condition has a prevalence between 0.9 -2.2%[120] and is more common in females[121,122] and Caucasians with a mean age above 50.[121]

Unlike the episodic variety, CTTH is thought to be secondary to a central sensitization process[12,124] whereby repeated painful stimuli over years causes dorsal horn neuron sensitisation, and future painless stimuli would, in turn, lead to nociceptive input sensitising central pain pathways and reduction in pain inhibition.

The pain of CTTH is described as tight band-like or pressing with bilateral frontal, temporal, or frontotemporal areas affected predominantly that does not prevent affected individuals from carrying out their daily chores, nonetheless they feel restrained.[10,125,126] There may be pericranial tenderness

Table 9: ICHD-II diagnostic criteria for CTTH

Headache occurring on ≥15 days per month on average for >3 months (≥180 days per year) and fulfilling criteria 2-4
Headache lasts hours or may be continuous
Headache has at least two of the following characteristics:
bilateral location
pressing/tightening (non-pulsating) quality
mild or moderate intensity
not aggravated by routine physical activity such as walking or climbing stairs
Both of the following:
no more than one of photophobia, phonophobia or mild nausea
neither moderate or severe nausea nor vomiting
Not attributed to another disorder

in some.[127] . Like other daily headache a secondary cause need to be excluded and the condition be differentiated from other primary daily headaches.

Hemicrania Continua

Hemicrania continua (HC) described in 1984 as "a unilateral, moderate, fluctuating continuous headaches, absolutely responsive to indomethacin".[128]. The true prevalence remains unknown although commoner than it was originally thought. Females are affected twice as frequently as males. The disorder can affect any age, although most common in the third decade.[129] The diagnostic criteria, defined by the ICHD-II, are given in Table 10.

The pathophysiology of HC is not well identified although a vascular aetiology,[130] pupillometric changes,[131] and pain pressure threshold hypothesis[132] have been investigated. PET studies have shown activation in the pons and hypothalamus, which was reversed with indomethacin treatment.[133] It is recommended to image all patients to rule out secondary causes such as cervical root irritation, mesenchymal tumours, head injury and HIV infection.[134,135]

As the name suggest the pain is commonly unilateral, frontal or peri-orbital, although a side shift and bilaterality have been reported.[136,137] The headache is usually dull and continuous with intermittent exacerbations lasting from a few minutes to several days and with features of migraine or autonomic symptoms such as lacrimation, nasal congestion and conjunctival injection.[129,138] Those with autonomic features are difficult to differentiate from TAC, although the pain is less intense and autonomic features in HC are relatively mild. Unlike TAC the pain is not precipitated by sensory stimuli or alcohol.

The condition responds dramatically to indomethacin and hence a challenge must be given to all primary unilateral chronic daily headaches. An oral dose between 25 – 75 mg three times a day may induce a response within 8 hours[129] and an intramuscular administration (10-50 mg) within 2 hours (Indo Test).[139] Other headaches that responds well to indomethacin include primary stabbing headaches, hypnic headaches, paroxysmal hemicranias and some exertional headache such as primary cough headache. In recent study, response to indomethacin has been reported in less than half of individuals with phenotypically suspected hemicrania continua, but those resistant cases could potentially be a unique headache different from HC.[140] CM is another differential diagnosis to be considered.

Table 10: Diagnostic criteria for hemicrania continua[10]

Headache for more than 3 months fulfilling other 3 criteria:
All of the following characteristics:
Unilateral pain without side-shift
Daily and continuous, without pain-free periods
Moderate intensity, but with exacerbations of severe pain
At least one of the following autonomic features occurs during exacerbations and ipsilateral to the side of pain:
Conjunctival injection and/or lacrimation
Nasal congestion and/or rhinorrhea
Ptosis and/or miosis
Complete response to therapeutic doses of indomethacin
No other attributed aetiology identified

The most common side effect of indomethacin is gastric irritation with risk of bleeding. It is available as suppositories and given to those unable to tolerate orally. Patients who require long term indomethacin should be given concomitant proton pump inhibitors. Patients are advised to discontinue the treatment once every 6 months to look for any remission of the condition which has been reported in a few cases.[141] Loss of indomethacin efficacy or the need to increase the dose may be due to poor compliance or an alternate diagnosis.

There are anecdotal reports on some other pharmacological agents such as topiramate, gabapentin, verapamil and other NSAIDs although their efficacy is yet to be established.[142]

Some people argue as to whether the condition should be classified under TAC, although the continuous nature of pain and lack of autonomic features in a significant proportion of patients makes it difficult to define under the TAC. However, the condition must be suspected in any refractory unilateral headache and an indomethacin challenge is given before invasive treatments are considered.

Trigeminal Neuralgia

Trigeminal neuralgia (TN) is a unilateral, severe and brief pain of stabbing nature that commonly affects the maxillary and mandibular division and less often the ophthalmic division of the trigeminal nerve. The condition may be primary or idiopathic or secondary to a structural lesion other than a vascular compression. Bilateral TN is occasionally seen in Multiple Sclerosis. The condition is more common in women (3:2) and right side is affected more often (3:2).[143] The condition affects 12-27 per 100,000 and the incidence increases from 16/100,000 in the fourth to 30/100, 00 in the 9th decade.

The pain of TN is like an electric shock, paroxysmal, brief lasting a few seconds and precipitated with cutaneous or mucosal stimulation such as talking, eating or swallowing, brushing teeth or touching part of the face. The pain may accompany small twitching movement of the face or tics. Autonomic symptoms are uncommon and mild; prominence of such symptoms should raise suspicion of TAC. There are periods of remissions although with time the relapses get frequent and their duration gets longer. The condition is effectively treated with carbamazepine or oxcarbazepine. There is limited evidence of efficacy for other agents like gabapentin, lamotrigine and baclofen.[144]

Trigeminal Autonomic Cephalalgias

A detailed description of these disorders is covered elsewhere in the supplement and is beyond the scope of this article. As Trigeminal autonomic cephalalgias (TAC) is fairly uncommon, diagnosis in primary care is often significantly delayed. CH is the commonest TAC commonly seen in smokers and three times more common in men. The diagnosis must be considered in those with unilateral headaches mainly in the

first (ophthalmic) division of the trigeminal. The pain in CH is excruciating, shorter in duration (15 minutes to 2 hours) occurring between 1-8 times a day often at the same time with autonomic features and precipitated by alcohol.

Conclusions

Daily headache disorders have been the major focus of attention among headache experts since the ICHD- I and although the term CDH has been used for some time, the ICHD-II have generated considerable discussion on its classification and sub-type. There is still lack of consensus on the definition of CM due to its frequent accompaniment with medication overuse that makes it difficult to interpret clinical trials with pharmacological agents in this condition. The cause and effect of the two remains controversial. The access to electronic information through mobile devices and internet has changed patient perception. They are more likely to accept the fact that analgesic may be a cause of their daily headaches and show better compliance to treatment and advice. The development of neuro-stimulation will see a significant change in therapeutic options on various sub-types of CDH. There is still much to understand regarding pathophysiology of frequent headache disorders, particularly CM and MOH and further research in this field remain interesting.

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