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Risk Factors of Chronic Daily Headache or Chronic Migraine

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Abstract Chronic daily headache (CDH) is a common neurological condition that affects 1–4 % of the general population. Most individuals with CDH originally suffered from episodic headaches, but over time, this developed into CDH. Although the pathophysiology of CDH is not fully understood, recent clinical and epidemiological studies suggest some risk factors that are associated with an increased risk of transformation from episodic headaches. If risk factors can be identified, they could provide a base for aggressive preventive intervention and thus decrease the transformation from episodic headaches to eventual CDH. In this article, we review and summarize the current data on risk factors for CDH.

Keywords Chronic daily headache · Chronic migraine · Headache · Migraine · Risk factor

Abbreviations

AMPP	American Migraine Prevalence and Prevention
CDH	Chronic daily headache
CM	Chronic migraine
CGRP	Calcium-gene-related peptide
CTTH	Chronic tension-type headache
EM	Episodic migraine
HR	Hazard ratio
ICHD-2	The second edition of the International Classification of Headache Disorder
ICHD-3 beta	The third beta edition of the International Classification of Headache Disorder
ICD-9-CM	The International Classification of Diseases, Ninth Revision, Clinical Modification
OR	Odds ratio
SES	Socioeconomic status
TM	Transformed migraine
TMD	Temporomandibular disorders

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Introduction

While most headache sufferers experience headaches only once or twice a month, approximately 1–4 % of the population experiences an attack on a daily or near daily basis [1–5]. Studies on such patients in hospital-based and population-based settings have generally reported high rates of medication overuse, psychiatric comorbidities, and disability [6–11]. Most of these patients have a history of episodic migraine (EM) with a gradual increase in headache frequency. Mathew et al. coined the term "transformed migraine" (TM), in which EM may progress to daily or near daily headaches [9].

Chronic daily headache (CDH) is usually defined as headache of any type that occur ≥15 days per month for longer than 3 months [12]. Silberstein et al. described subtypes of CDH including TM, chronic tension-type headache (CTTH), new daily persistent headache, and hemicranias continua [13]. Most population- and hospital-based studies reported that TM was the most common form of CDH [1, 2, 4, 5, 14]. The second edition of the International Classification of Headache Disorder (ICHD-2) incorporated the concept of TM and included chronic migraine (CM) as a complication of migraine

[15]. After proving too restrictive, the diagnostic criteria for CM were revised for the third beta edition of the International Classification of Headache Disorder (ICHD-3 beta). The criteria are now a headache on ≥15 days per month, including a migraine with/without an aura or relieved by a triptan or ergot derivatives on at least 8 days per month, with a duration of at least 3 months with or without medication overuse [16].

Annually, approximately 2.5 % of individuals with EM see them transform into CM [8, 14, 17]. Consequently, it is important for public health to identify risk factors for migraine chronification. If risk factors can be identified, they might provide a base for aggressive preventive intervention. Fortunately, patients with CDH often return to episodic headaches (Table 1) [2, 4, 18, 19, 20•].

Although the biological mechanisms underlying CM are currently unknown, epidemiological reports have provided some insights into risk factors. Risk factors for migraine chronification can be divided into those that may be modifiable through medical or behavioral intervention and those that are not [21]. Modifiable risk factors include psychiatric problems such as anxiety and depression, sleep-related breathing disorders, insomnia, temporomandibular disorders, obesity, excessive caffeine intake, frequent use of abortive medications, high frequency of headaches, and comorbid pain disorders. Examples of nonmodifiable risk factors are gender, age, low socioeconomic status (SES), genetic background of chronic headaches, medication overuse, a history of head or neck injury, and life events such as move, marriage, divorce, or change of employment (Table 2). The purpose of this review is to summarize evidence related to risk factors for CDH, in order to help understand the natural history of CDH and to identify potential targets for intervention.

Methods

To identify studies reporting on CDH or CM, a systematic search using PubMed (incorporating MEDLINE) was performed. The search included full papers and abstracts written in English before April 1, 2014. If the main body of the paper was written in a language other than English and the English abstract was adequate for our review, we included the paper.

Searches were performed on April 1, 2014, using combinations of the keywords "risk factors," "chronic migraine," and "chronic daily headache." Search strings were entered into PubMed as free text with no limits to minimize the possibility of omitting relevant records. We retrieved 2561 records from the PubMed search and then two authors (SJC and MKC) reviewed these records for relevance to this review. We selected 76 original articles and 23 review articles. Additional searches were performed using fewer or wider search terms to ensure that all potentially relevant studies had been identified. An additional 61 articles were added to this review (Fig. 1).

Results

Modifiable Risk Factors

Psychiatric Problems

Psychiatric disorders are very common comorbidities of migraine patients, and the association between psychiatric disorders and CM has recently been studied [22, 23, 24•]. In a national database in Taiwan, CDH patients had three times the risk of anxiety disorder, depression, bipolar disorder, and

Table 1 Prognosis of chronic daily headache in population-based studies

	Age (years)	Headache type	CDH prevalence (%)	Sample number	Follow-up duration	Follow-up results
Wang et al. [4]	≥65	≥15 headache days per month for ≥6 months	3.9	Total—1533 CDH—60	2 and 4 years	CDH after 2 years—61 % CDH after 4 years—73 %
Lu et al. [2]	≥15	>15 days per month	3.2	Total—3377 CDH—108	2 years	≤15 days per month—65 %
Scher et al. [17]	18–65	≥180 days per year	N/A	1134 CDH subjects 798 non-CDH subjects	1 year	<1 headache days per week—14 % <180 headache days per year—57 %
Wiendels et al. [18]	25–55	>14 headache days per month for ≥3 months (CFH)	3.7 (CFH)	Total—21,440 CFH—177	5 months	<7 days per month—12 % ≤14 headache days per month—35 %
Wang et al. [19]	12–14	≥15 days per month for >3 months	6.1	Total—2000 CDH—122	2 years	CDH after 1 year—40 % CDH after 2 years—25 %
Manack et al. [20•]	N/A	CM	4.6	Total—8219 CM—383	2 years	130 (34 %) persisted CM 100 (26 %) remitted CM 153 (33.9 %) transitioning CM

CDH chronic daily headache, *CFH* chronic frequent headache, *CM* chronic migraine, *N/A* not available

Table 2 Suggested risk factors for chronic daily headache or chronic migraine

Modifiable risk factors
Psychiatric problems: anxiety, depression, somatization disorders
Sleep-related problems: insomnia, habitual snoring, sleep bruxism, daytime sleepiness
Temporomandibular disorder
Obesity
Caffeine overuse
Medication overuse
Migraine features: high frequency of headache, allodynia or increase pain sensitivity, nausea, prolonged duration of headache
Nonmodifiable risk factors
Women gender
Old age
Low socioeconomic status
Genetic background
Life events
Head or neck injury

panic disorder diagnosed using the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) than nonmigraineurs and had about double the risk of psychiatric disorders than other migraineurs [24•]. Prevalences of depression (30.19 vs. 17.24 %) and anxiety (30.23 vs. 18.79 %) were higher in patients with CM compared to those with EM in the American Migraine Prevalence and Prevention (AMPP) study, a large population-based study for headache [22]. A web-based survey study in nine countries also demonstrated a more prominent association of CM with depression and anxiety than that of EM [23]. The association between CDH and anxiety, depression, and physical expression of psychological problems (somatization) is evident in pediatric and elderly CDH patients in Taiwan [4, 25].

Longitudinal data from the AMPP study showed that the risk of developing CM among EM patients increased with the severity of depression. Severe depression increased the risk of CM (odds ratio [OR]=3.19, 95 % confidence interval [CI]=1.26–8.09) more than moderate depression (OR=2.53, 95 % CI=1.06–6.05) [26••]. Some clinic-based studies showed an increased risk of CDH or medication overuse headache (MOH) among patients with a higher depressive severity [27, 28].

The association between CM and anxiety disorders is similar to the association with depression. Among patients with CM, the prevalence of anxiety disorders as diagnosed using the ICD-9-CM was 12.9–15.6 % [22, 24•]. Anxiety disorder was the significant predictor of chronification of migraine (OR=1.53, 95 % CI=1.15–2.04) [26••].

In summary, depression and anxiety are more common in patients with CM, and the severity of psychiatric disorders is associated with headache frequency and the future risk of CM, so psychiatric comorbidities are an important risk factor of CM/CDH. It is possible that a shared mechanism underlies the association between psychiatric factors and chronic headaches [29].

Sleep-Related Problems

People with CDH/CM frequently exhibit sleep-related problems. Insomnia, habitual snoring, sleep bruxism, and daytime sleepiness are all suspected risk factors for CDH/CM. Insomnia, multiple arousals during sleep, and the use of hypnotics are all associated with increased risk of CDH after adjusting for psychiatric illness [30]. Habitual snoring was more frequently present in CDH patients (24 vs. 14 %, $p<0.05$) than in people without CDH [31]. Sleep bruxism was related to CM (OR=3.8, 95 % CI=1.83–7.84) in a clinic-based cross-sectional study [32•]. Excessive daytime sleepiness (Epworth

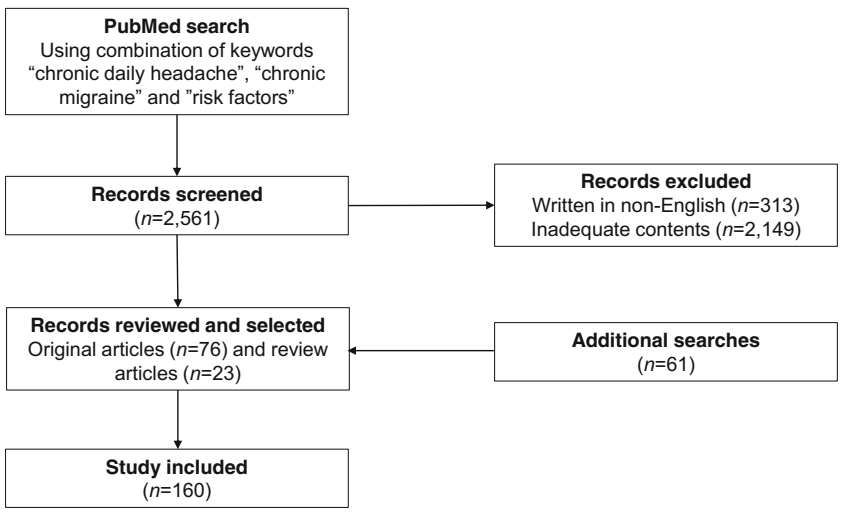

Fig. 1 Process of study selection

Sleepiness Scale scores ≥10) was more common in patients with CDH than in controls (20 vs. 6 %, OR=3.92, 95 % CI= 1.50–10.22) [33]. Although there is no strong evidence of a causal relationship between sleep issues and the onset of CDH/CM, any problem that can disturb sound sleep could aggravate headache status.

Sleep deprivation can cause headaches and both sleep and melatonin have been shown to have a therapeutic or analgesic effect for some headache patients. According to one study that used a prospective headache/sleep diary, occurrence of headache was associated with two prior days of either high stress or inadequate sleep in CDH patients; thus, sleep issues have a role in increasing the frequency of headaches [27].

Temporomandibular Disorders

Compared to controls, CM patients have more frequent tenderness in their masticatory muscles or temporomandibular joints (73 vs. 23 %, 61 vs. 25 %) [34]. Myofascial temporomandibular disorders (TMD) increased the risk of CDH (relative risk=7.8, 95 % CI=3.1–19.6) and the severity and pain of TMD was correlated with the frequency of headaches [35]. This association may be due to the central facilitation of nociceptive input from TMD. Indeed, migraine patients with TMD have lower threshold for heat and mechanical nociception, so an association between TMD and allodynia is evident in such patients [36].

Obesity

Being overweight or clinical obesity is a strong risk factor for cardiovascular and/or metabolic disease and results from a complex interaction between genetic, emotional, environmental, and behavioral factors. Compared to people of normal weight, the prevalence of CDH was increased in people whose body mass indexes (BMI) classified them as obese or morbidly obese (3.8 vs. 4.9 vs. 6.8 %, OR for morbidly obese contrasted to normal weight=1.8, 95 % CI=1.4–2.2). The prevalence of TM increased with increasing BMI (0.9 vs. 1.6 vs. 2.5 %, OR for morbidly obese in contrast to normal weight=2.2; 95 % CI=1.5–3.2) [37]. BMI was also associated with the severity, disability caused by, and the frequency of headaches. In a longitudinal population-based study, the influence of obesity was more evident for new onset CDH/CM, and the relative odds of CDH were three to five times higher in overweight or obese people than in people of normal weight in the USA [17]. In Taiwanese pediatric patients, obesity was a significant risk factor for new onset CDH (hazard ratio [HR]= 1.96; 95 % CI, 1.04–3.69) and CM (HR=2.41, 95 % CI= 1.13–5.14) [38•]. Obese people may be vulnerable to frequent headaches due to mechanisms which may include increased inflammatory markers, increased plasma calcium-gene-related peptide (CGRP) levels, and dismodulation of the orexin pathway in obese people [39–41]. Alternatively, disturbed leptin (the so-called safety hormone which regulates the amount of fat stored in the body) and insulin resistance were reported in migraine patients [42]. Obesity may also aggravate psychiatric and sleep-related problems. About half of CDH patients were below or of normal weight, so obesity may only influence some CDH patients or some features of migraine [37, 43].

Caffeine Overuse

Caffeine can be both beneficial and detrimental for migraine patients. Caffeine is a potent vasoconstrictor so it is used as a component in some acute abortive medication for migraine. In contrast, a delay in intake of caffeine can cause weak headaches. Abrupt withdrawal of caffeine evokes headaches in more than half of people who usually consume low or moderate amount of caffeine [44]. Migraine patients frequently complain about daytime sleepiness, especially during the prodromal phase of the headache, so are prone to caffeine overuse. One population-based study reported that previous excessive exposure to dietary caffeine (311 mg/day, top quartile) or medication containing caffeine was associated with a higher risk of CDH (OR=1.6, p=0.02); however, another study did not show any association of headache with caffeine consumption [45]. In one clinic-based study, 36 pediatric CDH patients showed a reduced frequency of headaches when they gradually withdrew from a previous caffeine consumption of at least 1.5 L of cola (193 mg of caffeine) per day. Thus, caffeine appears to have a role in the onset and remission of CDH [46].

Medication Overuse

Symptomatic medication use itself is the mainstay of acute abortive therapy for EM; however, there is a lot of evidence demonstrating a causal relationship between medication overuse and the onset of CDH/CM. First, frequent use of analgesics or opiates for nonheadache purposes can transform some migraine patients to CDH/CM, but only in people with a history of migraine [47•]. In an experimental rat model, repeated or sustained exposure to triptan for just over 6 days caused decreased sensory thresholds, sensory allodynia, and increased the CGRP after exposure in nitric oxide donors [48]. After discontinuation of triptan exposure, sensory thresholds returned to baseline, but the CGRP response persisted; thus, triptans can transform EM to a state of central sensitization. Second, there is a dose-response relationship between the use frequency of symptomatic medication and the risk of CDH/CM [49]. The safe threshold for CDH/CM is uncertain, but usage of any symptomatic medication 10 or more times per month increased the risk of CDM/CM. Similarly, the use of barbiturates and opiates over 5 and 8 days, respectively, per

month also increased the risk of CDM/CM [50–53]. Interestingly, the use of nonsteroidal anti-inflammatory drugs (NSAIDs) fewer than 10 times per month may be protective against the development of MOH (OR=0.31, 95 % CI=0.27–0.34) [50]. The influence of medication overuse on migraines is also evident in pediatric and elderly patients [4, 25].

High Baseline Frequency of Headaches

After adjusting for obesity, the risk of new onset of CDH increased with increasing baseline frequency of headaches in people suffering from more than 2 headaches per month [17]. Baseline headache frequency of more than 10 days per month critically increased the risk of progression [54]. In a clinic-based 1-year follow-up study with EM patients, when compared to patients with a baseline headache frequency of less than 5 days per month, the odds ratios of new onset CDH were 20.1 (95 % CI=5.7–71.5) in patients with a baseline headache frequency of 10–14 days per month and 6.2 (95 % CI=1.7–26.6) in patients with 5–9 headache days per month [55]. A baseline frequency of more than 7 headache days per month was a risk factor for new onset CDH/CM in pediatric patients [38•]. Considering the increased risk of CDH in patients with a baseline frequency of more than 2 headache days per month, preventative medication might be considered a possible option to prevent CDH in patients suffering from high-frequency EM.

Allodynia or Increased Pain Sensitivity

In a 12-year longitudinal population-based study, pain threshold was shown to decrease in CTTH patients, and thus, increased pain sensitivity is a result of frequent headache, not a risk factor [56]. In a web-based longitudinal study of 1992 migraine patients, cutaneous allodynia was common (70 %) in migraine patients and was associated with an increased frequency of headache attacks over a median follow-up period of 103 weeks (OR=11.12, 95 % CI=5.87–16.37) [57]. Allodynia was associated with the duration, frequency, and severity of migraine, so it may be a marker for possible CM or a symptom of psychiatric comorbidities rather than a risk factor for CM [58].

Nausea or Prolonged Duration of Headache

Nausea is one marker that measures the disability of migraines and can cause treatment failure due to poor absorption of medication. As measured in a longitudinal, population-based study, nausea, prolonged duration of headache, and a pulsating headache are all associated with new or persistent CDH [52].

Nonmodifiable Risk Factors

Gender

Population-based, cross-sectional studies have shown that women have an increased prevalence of chronic daily headache or chronic migraine when compared to men by a factor ranging from 1.6 to 2.6 [1, 2, 59–64]. The higher prevalence of CDH in women over men persisted even above the average age of menopause. When compared to men, one longitudinal population-based study reported an increased risk for the prevalence of CDH in women (OR=1.69, 95 % CI=1.4–2.1), but failed to demonstrate a positive result for the onset of CDH (OR=0.9, 95 % CI=0.4–2.4) [17]. In childhood or adolescence, two cross-sectional population-based studies studied gender-stratified CDH prevalence [65, 66]. Girls had a higher prevalence of CDH than boys, with ratios of 2.6 and 3.0 in these studies.

In summary, higher CDH prevalence in women was consistently reported in cross-sectional studies. However, there was no current evidence for gender differences in the onset of CDH. Higher headache or migraine prevalence in women suggests that the higher prevalence of CDH in women may just be due to a higher headache or migraine prevalence when compared to men.

Age

Most population-based studies for CDH showed increasing prevalence of CDH with increasing age. There were two exceptions. One exception was a cross-sectional study in Brazil, which showed a peak CDH prevalence for people in their 30s [67] with decreasing prevalence of CDH after the 30s. The other exception was a population-based study carried out in the USA that revealed no difference in the incidence of CDH with age [17].

Socioeconomic Status

Population-based studies have reported an increased risk for CDH or frequent headache in people with low SES such as low education level, low income, poor marital status, and low social class by occupation [14, 17, 68]. Low SES was also associated with a higher risk of CDH and a reduced likelihood of remission for CDH [17].

Genetic Background

Several studies have shown an increased risk of CDH for individuals with a family history of CDH. A population-based study in Brazil showed that the risk of CDH in children increased 13-fold when a mother has CDH, but the risk of infrequent headaches was not increased [69]. Another

hospital-based study in Italy reported an increased risk for headache chronification among individuals with a family history of chronic headaches or medication overuse [70]. Chronic tension-type headaches, the second most common form of CDH, were reported to be familial occurrence. Parents, siblings, and children of individuals with CTTH had a 2.1- to 3.9-fold increase in risk for CTTH, but no increase in risk was found for the spouses of CTTH sufferers [71].

Two studies compared the risk of CDH among different racial groups in the USA. One study reported higher CDH prevalence in Caucasians compared to African Americans [14]. The other study showed no difference in prevalence and incidence of CDH between different racial groups [17].

Two genetic polymorphism association studies for MOH were available. One study linked a glutamate transporter protein gene *EAAT2* A allele with analgesic overuse, which was commonly associated with CDH [72•]. The other study revealed that two serotonin 5HT2A receptor gene polymorphisms (A1438G and C516T) were associated with medication overuse [73].

Life Events

Life events such as a move, the death of a family member or friend, a change in marital status, and ongoing stressful events [74] have been considered a precipitating factor for CDH in clinical samples [75–79]. Most clinical studies showed an increased risk of CDH among individuals experiencing significant life events. A population-based study compared antecedent and subsequent life events in CDH cases and controls [80]. Antecedent life events were significantly associated with CDH after adjusting for gender and headache type (OR=1.2, 95 % CI=1.1–1.3). Subsequent life events were not significantly different in CDH and control groups (OR=0.94, 95 % CI=0.8–1.1). Another population-based study reported increased numbers of major life events in patients with CDH compared to controls around the onset of CDH (2.7 events vs. 2.0 events) [74]. A population-based study from the Netherlands also reported a close association between the number of significant life events recently experienced and CDH [81].

For adolescents, one population-based study from Taiwan was available. Adolescents with CDH had higher levels of childhood adversity (as measured by lower scores on the Global Family Environment Scale) when compared to controls. Physical abuse and parental abuse were more common events in adolescents with CDH [82].

Head or Neck Injury

Headaches developed in 50–80 % of head- or neck-injured patients, and these headaches persist in 20–30 % of patients 1–2 years later [83–85]. Recent studies suggest that head or neck injury may be a risk factor for CDH [6, 86–90]. However, most studies were clinical studies rather than population-based studies. In a population-based study in the USA, men with CDH reported more head or neck injury in the same year or the year before CDH onset than episodic headaches compared to men without CDH (20.0 vs. 7.9 %, OR=3.3, 95 % CI=1.0–10.8) [91]. Over a lifetime, an increased risk of reporting head or neck injury persisted among CDH patients (64.5 vs. 38.9 %, OR=3.1, 95 % CI=1.3–7.2). Women with CDH, when compared to women without CDH, reported slightly more a lifetime head or neck injury (37.2 vs. 26.9 %, OR=1.5, 95 % CI=0.97–2.3) and injuries in the same year or the year before CDH onset (8.8 vs. 3.3 %, OR=2.4, 95 % CI=0.98–5.9). The odds of CDH increased with the number of lifetime head or neck injuries.

Conclusions

Chronic daily headache is a common neurological disorder. Chronic migraine is the most common subtype of CDH and evolves from EM. A number of risk factors for CDH and CM have been identified and might provide a base for preventive intervention. Modifiable risk factors include psychiatric problems, sleep-related disorders, TMD, obesity, caffeine overuse, medication overuse, and high baseline headache frequency. Nonmodifiable risk factors include old age, low SES, a family history of CDH, significant recent life events, and head injury. Clinicians treating headache should be aware of the risk factors for CDH to prevent headache progression and to help revert CDH to episodic headaches.

Compliance with Ethics Guidelines

Conflict of Interest Dr. Soo-Jin Cho and Dr. Min Kyung Chu each declare no potential conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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