Review Article

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Update on Tension-type Headache

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Abstract

Tension-type headache (TTH) is the most common type of headache, characterized by mild to moderate intensity, bilateral, with a pressing or tightening (non-pulsating) quality. Migraine and TTH can occur in the same person, and their risk factors and treatments can overlap. However, TTH receives less attention than migraine. Furthermore, despite the expanding market for migraine treatments targeting calcitonin gene-related peptide (CGRP) mechanisms, the lack of evidence regarding mechanisms related to CGRP-related mechanisms in TTH continues to be neglected. There remains a need to develop effective preventive treatments for chronic TTH, which imposes a very high burden of disease. From this perspective, this review aims to provide the latest evidence on TTH.

Keywords: Headache, Tension-type headache, Headache disorders, Primary, Migraine

INTRODUCTION

Tension-type headache (TTH) is the most common headache disorder. It is characterized by mild to moderate intensity, bilateral, pressing, or tightening pain quality in the forehead, occiput, and neck. The term "tension" emphasizes the role of muscle contraction and emotional tension, leading to various treatments that focus on muscle relaxation and stress management.

Despite knowing the nature of how TTH and migraine can co-occur and having similar medical treatments, such as non-steroidal anti-inflammatory drugs (NSAIDs) or amitriptyline, TTH remains less researched, poorly diagnosed and treated than migraine.[1] For example, a PubMed search for the word "migraine" yields to almost 50,000 hits, compared to less than 5,000 hits for the word "tension-type headache." It is also interesting to note that while migraine and TTH can become interchangeable over time and share similar risk factors, triggers, and comorbidities in individuals, they have distinctly different headache characteristics.[2] The distinctive features of migraine, as opposed to TTH, appear to be influenced in part by the calcitonin gene-related peptide (CGRP) mechanism, which has recently received a great deal of attention. In contrast, TTH

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has been neglected as a research topic due to the lack of a clear biomarker and the absence of randomized controlled trial-based effective preventive medications for frequent episodic or chronic TTH.

This review aims to provide an overview of recent findings on the epidemiology, pathophysiology, diagnosis, and treatment of TTH.

EPIDEMIOLOGY

The global prevalence of TTH is approximately 26% of adults, with wide variation between studies and ethnicities, but it is generally estimated that 30% to 80% of the adult population will be affected at some point in their lives.[3-6]

The incidence of TTH is higher in women than in men, and this gender difference may be due to hormonal factors, stress, and psychosocial influences. TTH can occur at any age, but the greatest burden in terms of years of life lived with disability is in the between 15 and 49 age group.[7] The incidence of TTH tends to decrease with age, although it remains a significant health problem in the elderly population.[7]

Lower socioeconomic status is often associated with higher levels of stress, poorer access to healthcare, and lower levels of education, all of which may contribute to the prevalence and severity of TTH.[8] Cultural attitudes toward pain and healthcare-seeking behaviors may influence how individuals report and manage their headache symptoms. In some cultures, headache may be underreported due to stigma or misconceptions about headache. In addition, infrequent episodic TTH may be under-reported in epidemiological studies.[9]

CLINICAL PRESENTATION

TTH is characterized by a diffuse, mild to moderate, bilateral headache that is often described as a tightening sensation. Unlike migraine, the headache is not typically pulsating and does not worsen with routine physical activity. Also, TTH is also not usually associated with nausea or vomiting, although mild photophobia or phonophobia may be present in some cases. The clinical presentation of TTH is highly variable between individuals, with some experiencing infrequent episodic headaches and others suffering from chronic daily headaches.[10]

The frequency of TTH episodes can vary widely, ranging from infrequent episodic TTH (occurring less than 1 day per month) to chronic TTH (occurring more than 15 days per month).

Factors that contribute to the chronicity of TTH include high levels of stress, co-existing migraine, fatigue, anxiety, and depression, and an inability to relax after work.[11]

DIAGNOSIS

The diagnosis of TTH is primarily clinical and based on the patient's history and symptoms according to the criteria of the International Classification of Headache Disorders, 3rd edition (ICHD-3) (Table 1).[12] However, due to the non-specific nature of TTH symptoms, diagnosis can be challenging because of overlap with other headache disorders and medical conditions. This diagnostic ambiguity can lead to misdiagnosis, inappropriate treatment, and unmet patient needs. Therefore, it is important to rule out other diseases on the basis of the headache history.

Headache diaries are the best assessment tool for diagnosis and classification. However, additional diagnostic tests such as neuroimaging, blood sampling, and lumbar puncture may be necessary if any red flags are present.[13]

Without strict criteria are applied, both migraine and TTH may coexist and sometimes overlap, further complicating the diagnostic process. Migraine and TTH may have some overlap in their clinical features. Photophobia and phonophobia are more common in migraine than in TTH, also nausea and vomiting are more common in migraine than in TTH, although mild nausea may be present in chronic TTH according to the ICHD-3.[14,15] This can lead to clinicians misdiagnosing a patient as having migraine when they are actually have TTH, and vice versa. In children and adolescents, the transition from migraine to TTH or from TTH to migraine occurs within a few years, supporting the continuum theory of headache in this subgroup of individuals. Not only are mixed presentations and diagnostic shifts common at younger ages, but the challenges associated with distinguishing TTH from migraine in clinical practice, clinical research, and epidemiologic studies have been widely recognized.[16,17] TTH with migraine comorbidity is associated with genetic factors.[18] Recently, machine learning models have demonstrated high

Table 1. Diagnostic criteria of tension-type headache according to the International Classification of Headache Disorders, 3rd edition (ICHD-3)[1]

	2.1. Infrequent episodic tension-type headache	2.2. Frequent episodic tension-type headache	2.3. Chronic tension-type headache	2.4. Probable tension-type headache		
				2.4.1. Probable infrequent episodic tension-type headache	2.4.2. Probable frequent episodic tension-type headache	2.4.3. Probable chronic tension-type headache
A	At least 10 episodes of headache occurring on <1 day/mo on average (<12 days/yr) and fulfilling criteria B–D	At least 10 episodes of headache occurring on 1–14 day/mo on average for >3 months (≥12 and <180 day/yr) and fulfilling criteria B–D	Headache occurring on ≥15 day/mo on average for >3 months (≥180 day/yr), fulfilling criteria B–D	One or more episodes of headache fulfilling all but one of criteria A–D for 2.1. Infrequent episodic tension-type headache	Episodes of headache fulfilling all but one of criteria A–D for 2.2. Frequent episodic tension-type headache	Headache fulfilling all but one of criteria A–D for 2.3. Chronic episodic tension-type headache
B	Lasting from 30 minutes to 7 days		Lasting hours to days, or unremitting	Not fulfilling ICHD-3 criteria for any other headache disorder		
C	At least two of the following four characteristics: 1. bilateral location 2. pressing or tightening (non-pulsating) quality 3. mild or moderate intensity 4. not aggravated by routine physical activity such as walking or climbing stairs			Not better accounted for by another ICHD-3 diagnosis		
D	Both of the following: 1. no nausea or vomiting 2. no more than one of photophobia or phonophobia		Both of the following: 1. no more than one of photophobia, phonophobia or mild nausea 2. neither moderate or severe nausea nor vomiting			
E	Not better accounted for by another ICHD-3 diagnosis[1]					

diagnostic accuracy in migraine from electronic health records or questionnaires.[19-21] However, these are not yet sufficient for application to TTH.[21]

Premonitory or prodromal symptoms are characteristic of migraine and include yawning, mood changes, fatigue, and neck pain. These symptoms typically occur within 2–48 hours of the onset of migraine headache.[22] There are no of premonitory symptoms in patients with TTH. Migraine headaches may also be associated with menstrual periods, with the drop in estrogen levels affecting the frequency of migraine headaches. Migraine attacks are common during the perimenstrual period and usually improve during pregnancy.[23]

And the selective 5HT1B/1D agonist is thought to relieve migraine by stimulating the 5HT1B receptor on cranial vascular smooth muscle to reduce the pain-inducing vasodilation that may be responsible for the headache.[24] However, it is not effective for the treatment of TTH, except in people who also have migraine.[25] The healthcare provider caring for patients with headache should be aware of these overlaps and their implications for the management of patients with headache.

PATHOPHYSIOLOGY

The pathophysiology of TTH is complex, multifactorial, and not fully understood, involving both peripheral and central mechanisms. The peripheral mechanisms are primarily related to myofascial tissues and nociception, while the central mechanisms of chronification are related to pain processing in the central nervous system.[26,27] There has been some research into the mechanisms of nitric oxide-induced TTH and drug development is currently underway, but to date there have been no significant results.[20,28,29] The role of CGRP in the progression and remission of chronic TTH is becoming a subject of interest, although treatment response to anti-CGRP monoclonal antibodies is poor.[14,30]

1. Peripheral mechanisms

The peripheral mechanisms of TTH are mainly related to pericranial muscle tenderness during acute headache attacks and myofascial trigger points.[31-33] The most common method used to assess tenderness is manual palpation of the pericranial muscles and calculation of the total tenderness score.[34] And muscle hardness can be measured using the hardness meter, a quantitative method.[35] Pericranial tenderness is exacerbated during the acute headache phase and increases with the severity and frequency of TTH attacks, supporting the presence of more severe tenderness in individuals with chronic TTH than in those with episodic TTH.[36]

Myofascial tissues, which include muscles and connective tissues, can develop localized areas of tenderness called trigger points. These trigger points can cause pain in other areas, such as the neck or shoulder, which may contribute to the headache pain experienced in TTH.[37] Active myofascial trigger points are common in TTH consistent with the hypothesis that peripheral mechanisms are involved in the pathophysiology.[27] However, the relationship between myofascial trigger points and the severity of TTH varies between studies.

Electromyography studies have shown increased muscle activity and tension in individuals with TTH, suggesting that sustained muscle contraction and tension play a role in the development of headache pain.[38]

2. Central mechanisms

Central sensitization of second-order neurons in the spinal cord or the spinal trigeminal nucleus is a key mechanism in the pathophysiology of transformation from episodic to chronic TTH.[39,40] Patients with chronic TTH had higher pain sensitivity and lower tolerance to pressure stimulation of cranial and extracranial structures than patients with episodic TTH patients.[26,41]

Comorbidities, such as back pain, fibromyalgia, and sleep disorders, may alter pain sensitivity in patients with chronic TTH and increase central sensitization compared to patients with transient TTH, suggesting shared central mechanisms between the two groups. Anxiety and depression are in patients with TTH and are associated with worsening symptoms.[42,43]

Functional magnetic resonance imaging studies have provided insight into the central mechanisms of TTH by examining dynamic brain changes between pain and pain-free periods in patients with episodic TTH.[44,45] These studies have shown changes in activation in pain-processing regions of the brain, including the anterior cingulate cortex, insula, and prefrontal cortex. These findings suggest that individuals with TTH have abnormal pain processing and modulation, which may contribute to the perception of headache pain.

In addition, neurotransmitters such as serotonin and norepinephrine have been found to be associated with TTH, although several studies have yielded conflicting results.

TREATMENT

There are significant gaps in the management of TTH, and many patients are not receiving adequate treatment. A multidisciplinary approach tailored to each individual patient. For example, patients with infrequent episodes of TTH can be managed with acute medications and non-pharmacological treatments such as lifestyle modifications, while patients with frequent episodes of TTH or chronic TTH may require preventive pharmacological treatments with behavioral interventions (Figure 1).

1. Acute pharmacological treatments

Acute treatment aims to provide rapid relief of headache attacks quickly and is typically used at the onset of a headache episode. Simple analgesics have evidence-based efficacy and are widely accepted as the first-line treatment for the acute treatment of patients with TTH.[46]

NSAIDs and acetaminophen are commonly used for acute symptom management. Opioids, triptans, and muscle relaxants are not generally recommended for symptomatic TTH.[47,48]

• NSAIDs

NSAIDs inhibit the enzyme cyclooxygenase, which reduces the production of prostaglandins that which mediate inflammation and pain. Initial treatments for acute TTH include ibuprofen, ketoprofen, naproxen, and diclofenac.[49,50] Side effects include gastrointestinal discomfort, ulcers, and cardiovascular risks with long-term use.

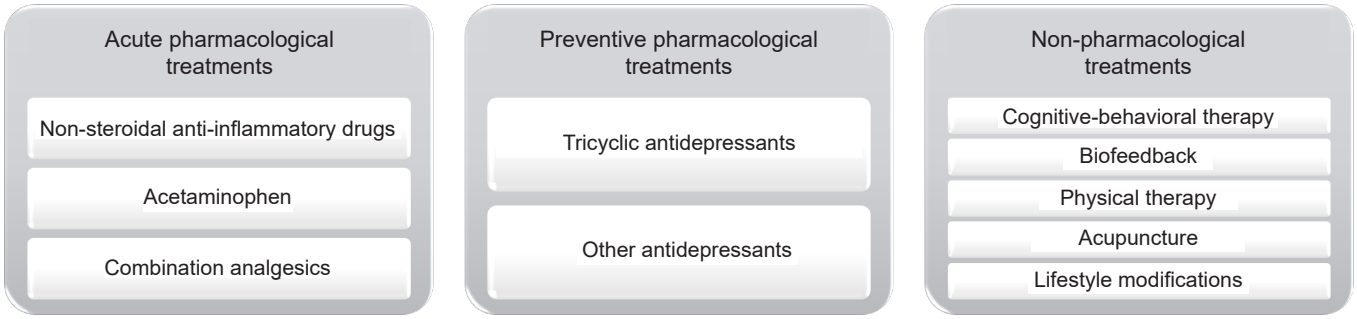

Figure 1. Current treatments for tension-type headache.

• *Acetaminophen*

Acetaminophen is the preferred initial therapy for patients with TTH who are intolerant of or contraindicated for NSAIDs for pregnant patients. Acetaminophen is typically used in a single oral dose of 500 to 1,000 mg. It is generally well tolerated, but overdose may cause liver toxicity.[51]

• *Combination Analgesics*

Combination of caffeine with acetaminophen, aspirin, or ibuprofen improves the efficacy for the acute treatment of TTH.[52] Caffeine may enhance the analgesic effects by promoting their gastric absorption.[53] However, frequent use may increase gastrointestinal discomfort and increase the risk of medication overuse headache.

2. Preventive pharmacological treatments

Preventive treatments have been shown to help reduce headache frequency and severity in patients with frequent episodic TTH (1 to 14 headache days per month) or chronic TTH (≥15 headache days per month). Preventive treatment is also indicated in patients with infrequent TTH when simple analgesics are ineffective, poorly tolerated, or contraindicated.

The goal of treatment is to reduce the frequency, severity, and duration of attacks and to improve the response to treatment of acute attacks. It is important to understand patient expectations and consider patient preferences when deciding which of the various preventive therapies to use. For patients who respond well (over 50% reduction in headache days per month), an adjunctive approach is to discontinue treatment after 3 or 6 months and monitor for headache recurrence, unless there are other comorbidities, such as depression or anxiety disorders.

• *Tricyclic Antidepressants (TCAs)*

TCAs have moderate to high potency for TTH, and amitriptyline controls pain through its inhibitory effects on serotonin and norepinephrine reuptake.

Amitriptyline also reduces pericranial muscle tenderness, resulting in peripheral antinociception and inhibition of central sensitization. It is common for clinicians to start amitriptyline at 2.5–10 mg nightly and increase by 5–10 mg per week to a maximum of 70–80 mg. Common side effects include sedation, weight gain, dry mouth, and constipation.

• *Other Antidepressants*

Mirtazapine (noradrenergic and specific serotonergic antidepressant) is comparable to amitriptyline and has a better tolerability profile than amitriptyline. Evidence for the effectiveness of venlafaxine (serotonin-norepinephrine reuptake inhibitor) in preventing TTH is weak and supports a level B rating by the EFNS-TF.[46]

3. Non-pharmacological treatments

Non-pharmacological treatments such as cognitive behavioral therapy (CBT), biofeedback, and relaxation therapy are often recommended as first-line interventions. In addition, integrative medicine (acupuncture and massage) and lifestyle modifications (sleep management, healthy diet, hydration, and exercise) may be considered to reduce headache triggers. However, the evidence for non-pharmacological approaches in TTH are very limited.[54]

• *CBT*

CBT is a psychological intervention that helps patients identify and modify negative thought patterns and behaviors that cause stress and muscle tension, both of which

are important factors in TTH.[55] CBT techniques for TTH include cognitive restructuring, behavioral activation, and relaxation techniques, among others. Stress management therapy has demonstrated efficacy in randomized and placebo-controlled trials and has been shown to be equivalent to amitriptyline in preventing chronic TTH.[56] Long-term group behavioral therapy has been shown to be effective in reducing headache frequency and intensity, improving coping strategies, and improving overall mental health.[57,58] CBT has been shown to improve quality of life and reduce comorbid symptoms of anxiety and depression.

• *Biofeedback*

Biofeedback is a technique that teaches individuals how to regulate physiological processes such as muscle tension, heart rate, and skin temperature through real-time feedback. Biofeedback helps patients become aware of and voluntarily control these processes, which can help reduce the frequency, duration, and intensity of headaches in patients with TTH.

• *Physical Therapy*

Physical therapy involves the use of massage, cervical spine manipulation, and exercise to improve muscle function, reduce tension, and promote relaxation.[59] It is particularly useful for TTH patients with severe musculoskeletal problems, such as poor posture, muscle imbalances, and trigger points.[60] However, there is no standardized protocol for treating TTH, and a combination of techniques appears to be more effective.[61]

• *Acupuncture*

The exact mechanism by which acupuncture relieves TTH is not fully understood, but it is believed to involve the modulation of pain pathways, release of endogenous opioids, and reduction of muscle tension and inflammation.[62] As the efficacy of greater occipital nerve block in various headache disorders has been confirmed, attempts have been made to use it as a treatment for TTH.[63-65] One systematic meta-analysis found acupuncture to be effective and safe for frequent episodic TTH and chronic TTH.[66]

• *Lifestyle Modifications*

Adopting healthy lifestyle habits can play an important role in the management and prevention of TTH. Important lifestyle changes include regular physical activity, healthy sleep patterns, a balanced diet, and effective stress management.[67,68]

Regular exercise, such as aerobic exercise, yoga, and stretching, has been shown to help reduce stress, improve sleep quality, and relieve muscle tension, all of which can help prevent TTH.

Strategies such as maintaining a consistent sleep schedule, avoiding caffeine and electronic devices before bedtime, and creating a comfortable sleep environment can help prevent headaches caused by sleep deprivation.

Eating a balanced diet that includes a variety of nutrients can help prevent and manage TTH.

FUTURE RESEARCH DIRECTIONS

Future research should focus on addressing the diagnostic challenges and improving our understanding and treatment of TTH. The co-occurrence of migraine and TTH may be coincidental, but more research is needed to determine whether there is a causal mechanistic relationship between the two disorders.[69]

One of the major challenges in diagnosing these headaches is the lack of reliable biomarkers, with diagnosis largely based on clinical criteria and patient self-report. More research is needed to improve diagnostic accuracy. While both migraine and TTH are associated with genetic factors, the specific genes responsible for the heritability of TTH remain unknown, in contrast to the multiple risk loci identified for migraine.

Pharmacological provocation studies have provided valuable insights into the pathophysiology of migraine, leading to the discovery of important therapeutic targets. However, similar studies have not been thoroughly performed for TTH. Unfortunately, in the absence of identified therapeutic targets, this approach is not currently feasible for TTH patients.

In addition, the evidence supporting the use of botulinum toxin and anti-CGRP monoclonal antibodies in the treatment of TTH is limited. We believe additional studies are needed to evaluate the utility of botulinum toxin and other emerging therapies for this common and debilitating condition.

CONCLUSION

TTH remains a common and often debilitating headache disorder. Despite its high prevalence, TTH remains under-recognized and under-treated, with significant public health implications. A comprehensive understanding of its epidemiology, pathophysiology, and clinical management is essential to improve patient outcomes. Continued research into the underlying mechanisms and public health efforts are needed to address the diagnostic and treatment gaps and ultimately improve the quality of life for individuals affected by TTH.

AVAILABILITY OF DATA AND MATERIAL

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

AUTHOR CONTRIBUTIONS

Conceptualization: HJL, SJC, JGS; Data curation: JGS, HWS; Formal analysis: JGS, HWS; Investigation: SJC; Methodology: HJL, SJC; Project administration: SJC, HWS; Resources: JGS; Software: JGS; Supervision: SJC, HWS; Validation: HJL, SJC, JGS, HWS; Visualization: HJL; Writing–original draft: HJL, SJC; Writing–review & editing: JGS, SJC, HWS.

CONFLICT OF INTEREST

Soo-Jin Cho is the Editor-in-Chief of *Headache and Pain Research* and was not involved in the review process of this article.
Hye Jeong Lee is the Editor of *Headache and Pain Research* and was not involved in the review process of this article.
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