TENSION-TYPE HEADACHE

*Messoud Ashina, MD, PhD, Dr.Med.Sci.¹ and Sait Ashina, MD²

¹*Danish Headache Center and Department of Neurology, Faculty of Health Sciences, University of Copenhagen, Glostrup Hospital, Glostrup, Copenhagen, Denmark*

²*Headache Program, Department of Pain Medicine and Palliative Care, and Department of Neurology, Albert Einstein College of Medicine, Beth Israel Medical Center, New York, NY, USA*

Corresponding author:

Messoud Ashina, MD, PhD, Dr.Med.Sci.; Danish Headache Center and Department of Neurology, Faculty of Health Sciences, University of Copenhagen, Glostrup Hospital, DK-2600 Glostrup, Copenhagen, Denmark

ABSTRACT

Tension-type headache is the most common primary headache disorder. The life-time prevalence of tension-type headache in general population is between 30 to 78%. Tension-type has the tremendous socio-economic impact on the individual and the society. Unfortunately, it is the least studied primary headache. The pathophysiology of this headache disorder is not fully understood. The diagnosis of tension-type headache is based on the history, and general and neurological examinations. Abnormalities in peripheral and central nociceptive nervous systems in combination with environmental and genetic factors may play a role in the pathophysiology of tension-type headache. The pharmacotherapy of episodic tension-type headache is non-specific and includes simple analgesics and nonsteroidal anti-inflammatory drugs. Tricyclic antidepressants are the mainstay in the prophylactic treatment of chronic tension-type headache.

Key words: Tension-type headache, classification, epidemiology, pathophysiology.

INTRODUCTION

Tension-type headache is the most common primary headache disorder [1, 2] with enormous socioeconomic impact [3, 4]. Tension-type headache used to be called tension headache, muscle contraction headache, psychomyogenic headache, stress headache, ordinary headache, essential headache, idiopathic headache or psychogenic headache. In 1988 the International Headache Society (IHS) introduced a new term, tension-type headache, to avoid terminology with a specific pathophysiological implication [5]. The pathophysiology of tension-type headache is far from understood but recent progress in basic and clinical research have increased our understanding of the mechanisms of tension-type headache [6, 7].

CLASSIFICATION

The second and latest edition of The

International Classification of Headache Disorders (ICHD-II criteria) [8] subdivides tension-type headache into three forms:

- Infrequent episodic tension-type headache
- Frequent episodic tension-type headache
- Chronic tension-type headache

All forms are subdivided further into headache associated or not associated with pericranial tenderness. ICHD-II diagnostic criteria for tension type headache are presented in Table 1.

CLINICAL CHARACTERISTICS

Tension-type headache is diagnosed on the basis of the history and general and neurological examinations. The secondary headache disorder must be ruled out in imaging studies (magnetic resonance or computer tomography depending on availability) if the individual presents risk factors or warning signs (Table 2). Patients

*e-mail: ashina@dadlnet.dk

Table 1. Diagnostic criteria for tension-type headache.

2.1 Infrequent episodic tension-type headache
A. At least 10 episodes occurring on <1 day per month on average (<12 days per year) and fulfilling criteria B–D   B. Headache lasting from 30 minutes to 7 days   C. Headache has at least two of the following characteristics: 1. Bilateral location   2. Pressing/tightening (non-pulsating) quality   3. Mild or moderate intensity   4. Not aggravated by routine physical activity such as walking or climbing stairs   D. Both of the following: 1. No nausea or vomiting (anorexia may occur)   2. No more than one of photophobia or phonophobia   E. Not attributed to another disorder
2.2 Frequent episodic tension-type headache
A. At least 10 episodes occurring on ≥ 1 but <15 days per month for at least 3 months (≥ 12 and <180 days per year) and fulfilling criteria B–D   B. Headache lasting from 30 minutes to 7 days   C. Headache has at least two of the following characteristics: 1. Bilateral location   2. Pressing/tightening (non-pulsating) quality   3. Mild or moderate intensity   4. Not aggravated by routine physical activity such as walking or climbing stairs   D. Both of the following: 1. No nausea or vomiting (anorexia may occur)   2. No more than one of photophobia or phonophobia   E. Not attributed to another disorder
2.3 Chronic tension-type headache
A. Headache occurring on ≥ 15 days per month on average for >3 months (≥ 180 days per year) and fulfilling criteria B–D   B. Headache lasts hours or may be continuous   C. Headache has at least two of the following characteristics: 1. Bilateral location   2. pressing/tightening (non-pulsating) quality   3. Mild or moderate intensity   4. Not aggravated by routine physical activity such as walking or climbing stairs   D. 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 attributed to another disorder

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Table 2. Indications for neuroimaging.

- Acute severe de novo or subacute persistent headache
- Headache beginning after 50 years of age
- Focal neurological signs and symptoms
- Change of pattern or worsening headaches
- Pattern of headache different from primary headache
- Seizures associated with headache
- Personality changes
- Headaches unresponsive to therapy
- History of significant trauma
- New headache in patient with cancer, immunosuppressive state or pregnancy

usually have mild to moderate pain of variable duration in episodic form or constant mild or moderate pain in chronic form. The headache is bilateral pressing or tightening pain, often felt like “a pressure bandage around the head” and is not associated with typical migraine characteristics such as aggravation by routine physical activity, severe nausea, vomiting, and severe photophobia or phonophobia. According to ICHD-II criteria (Table 1) patients with episodic tension-type headache are allowed to have no more than one of the following associated symptoms: photo- or phonophobia. Mild nausea, photo- or phonophobia (no more than one of symptoms) are allowed in chronic tension-type headache.

EPIDEMIOLOGY

Tension-type headache is the most common type of primary headache disorder with the life time prevalence from 30 to 78% [1, 2]. The majority of patients (59%) reported tension-type headache 1 day each month or less, 24% to 37% had headache several times each month, 10% had it weekly, and 2% to 3% of the population had chronic tension-type headache (15 days per month) [9]. The global prevalence of chronic tension-type headache is uniform, i.e., 2% to 3%. Unlike migraine headache women are only slightly more affected than men with male:female ratio of 4:5 [1, 2]. The average age of onset of TTH is 25 to 30 years as demonstrated in cross-sectional epidemiologic studies. The infrequent subtype has very little impact on the individual and does not represent a clinical or treatment

challenge. However, frequent and chronic subtypes are always associated with considerable disability and high personal and socioeconomic costs [3, 4].

PATHOPHYSIOLOGY AND GENETICS

Despite the considerable progress in basic and clinical tension-type headache research in recent years [6, 7], the origin of pain in this primary headache disorder is unknown. It has been suggested that both peripheral (nociception from pericranial myofascial tissue) and central (increased excitability of CNS) factors play a major role in pathophysiology of tension-type headache. Epidemiological studies reported an increased familial risk in tension-type headache [10, 11].

PERIPHERAL NOCICEPTION

It has long been known that if individual has been exposed to static or repetitive work for a long period he or she may develop tender areas in the pericranial (head, neck and shoulder) muscles and tension-type headache. For this reason the research on mechanisms leading to tension-type headache has always been focused on muscular factors. A large number of studies have consistently shown that the pericranial myofascial tissues are considerably more tender in patients with tension-type headache than in control healthy subjects, and that the tenderness to manual palpation is positively associated with both the intensity and the frequency of tension-type headache [12-14]. Painful impulses from

pericranial myofascial tissues may be referred to the head and perceived as headache. Myofascial mechanisms may, therefore, play a major role in the pathophysiology of tension-type headache [15]. The origin of pain in TTH traditionally has been attributed to excessive muscle contraction, ischemia and inflammation of head and neck muscles. The possible causes have been extensively studied in tension-type headache. However, surface electromyography (EMG) electrodes failed to demonstrate significantly increased activity in tension-type headache [16-18]. A microdialysis study reported altered blood flow regulation in tender skeletal pericranial muscle during static work in patients with chronic tension-type headache [19]. The authors found no difference in local increase of interstitial lactate between patients and controls. This seems to rule out the presence of ischemia in the tender point of patients with chronic tension-type headache during static exercise. It was hypothesized that increased excitability of neurons in the CNS may affect the regulation of muscle blood flow during static work [19]. The microdialysis study also demonstrated that the interstitial concentration of inflammatory mediators in tender muscle did not differ between patients with chronic tension-type headache and healthy subjects [20]. These data indicate that tender points are not sites of ongoing inflammation.

In summary, pericranial myofascial pain sensitivity is increased in patients with TTH and peripheral mechanisms may play a role in the pathophysiology of TTH. However, a firm evidence for peripheral muscle pathology as a cause of muscle pain and chronic headache is still lacking in tension-type headache.

CENTRAL SENSITIZATION

The increased myofascial pain sensitivity in tension-type headache could also be caused by central factors such as sensitization of second-order neurons at the level of the spinal dorsal horn/trigeminal nucleus, sensitization of supraspinal neurons, and decreased antinociceptive activity from supraspinal structures [6]. In the last ten years there has been increasing interest on the role of central factors in tension-type headache.

It has been shown that pressure pain detection and tolerance thresholds to mechanical stimuli, thermal and electrical were decreased in chronic tension-type headache sufferers [21-24]. In addition, Bendtsen et al. [25] demonstrated that patients with chronic tension-type headache had a qualitatively altered pain perception. On the basis of these findings and data from basic pain research [26] it has been suggested that the central sensitization and thereby the chronic pain state in patients with chronic tension-type headache may be due to sensitization at the level of the spinal dorsal horn or trigeminal nucleus, or both, induced by prolonged nociceptive input from pericranial myofascial tissues [6]. The hypothesis of central sensitization in tension-type headache is supported by experimental pharmacological studies. Animal studies have shown that sensitization of pain pathways may be caused by or associated with activation of nitric oxide synthase (NOS) and the generation of nitric oxide (NO) [27]. To test the hypothesis of central sensitization in tension-type headache the antinociceptive effect of NOS inhibitors [28, 29] and the nociceptive effect of NO donor [30] was investigated in patients with chronic tension-type headache. The NOS inhibitor NG-monomethyl-L-arginine hydrochloride reduced headache [29] and pericranial myofascial tenderness and hardness [28] in patients. Furthermore, the NO donor glyceryl trinitrate induced tension-type headache in these patients [30]. These data suggest that NO plays an important role in the pathophysiology of in chronic tension-type headache [7] and that inhibition of NOS may become a novel principle in the treatment of this disorder.

ALTERED DESCENDING INHIBITORY CONTROL

Decreased antinociceptive activity from supraspinal structures, i.e. deficient descending inhibition, may contribute to the increased pain sensitivity in chronic tension-type headache [31-33]. A recent high-density brain electroencephalogram (EEG) mapping study found impaired inhibition of nociceptive input in CTTH patients [34]. Cathcart et al. [35] recently

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reported deficient inhibition of repeated noxious mechanical stimulation in patients with chronic TTH. Impaired descending inhibition could be the primary abnormality or it could contribute to or be a consequence of central sensitization [6]. However, longitudinal studies are needed to clarify this finding.

ENVIRONMENTAL AND PSYCHOLOGICAL FACTORS

Stress and mental tension are the most frequently reported precipitating factors for tension-type headache [36]. However, these factors may therefore not be of specific importance for the pathophysiology of tension-type headache. It has recently been demonstrated that stress induces more headache in patients with chronic tension-type headache than in healthy controls probably through hyperalgesic effects on already sensitized nociceptive pathways [37]. However, stress did not aggravate increased temporal summation or abnormal diffuse noxious inhibitory control mechanisms in individuals with tension-type headache [35]. Previous studies demonstrate that there is no increase in anxiety or depression in patients with infrequent tension-type headache, while frequent tension-type headache is associated with higher rates of anxiety and depression [38]. It has been shown that depression increases vulnerability to tension-type headache in patients with frequent headaches during and following a laboratory stress test [39]. Furthermore, the induced headache was associated with elevated pericranial muscle tenderness. It was suggested that depression may aggravate existing central sensitization (discussed later) in patients with frequent headaches [39]. Therefore there may be a bidirectional relationship between depression and frequent TTH.

GENETICS

A single genetic epidemiologic study has investigated the familial aggregation of chronic tension-type headache in 122 probands recruited from a tertiary headache clinic [10]. Compared to the general population, first degree relatives had a 3.1- fold significantly increased risk of chronic tension-type headache, whereas spouses had no

increased risk of chronic tension-type headache. Thus, the increased familial risk of tension-type headache can be caused by both genetic and environmental factors [11]. Currently, we think that the majority of the population has the potential to develop tension-type headache if exposed to sufficiently strong environmental factors.

In summary, we are beginning to understand some of the complex mechanisms leading to tension-type headache. This will hopefully lead to the development of more effective and specific treatment modalities for tension-type headache.

TREATMENT

The pharmacotherapy of tension-type headache is non-specific and includes simple analgesics and NSAIDs for the episodic form and antidepressants for prophylaxis in the chronic form. Establishment of an accurate diagnosis of tension-type headache and its subtypes is very important before initiation of any pharmacologic or non-pharmacologic therapy. It is highly recommended that patients complete a diagnostic headache diary [40] during a 4-week period to confirm the diagnosis and to exclude possible medication overuse headache in patients with frequent episodic or chronic tension-type headache.

ACUTE THERAPY

Acute pharmacotherapy includes treatment of each episode of headache in individuals with infrequent and frequent episodic tension-type headache). Simple analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) are the mainstays in the acute therapy of tension-type headache (Table 3 and Fig. 1). Aspirin and acetaminophen are the most commonly used analgesics. Randomized, controlled trials demonstrated that acetaminophen, aspirin and NSAIDs are effective in the treatment of single episodes of tension-type headache and should be included in the treatment of mild or moderate episodes [41]. Acetaminophen may be recommended as the first choice because of a better gastric side-effect profile compared to NSAIDs. Combinations analgesics have been

Table 3. Recommended dosages for acute (< 15 headache days per month) and preventive therapy of tension-type headache (Modified from Ashina and Ashina (2003)).

Acute therapy	Preventive therapy
500 mg of aspirin	10-75 mg of amitriptyline daily
1000 mg of aspirin or 1000 mg of acetaminophen	15-30 mg of mirtazapine
200 mg of ibuprofen	
25 mg of ketoprofen	
400 mg of ibuprofen or 50 mg of ketoprofen	

used in the treatment of tension-type headache but the evidence is limited. More randomized, controlled, and comparative studies are needed to evaluate the efficacy and safety of combination analgesics in the treatment of infrequent and frequent episodic tension-type headache [41]. Muscle relaxants are not considered to be effective because of insufficient studies and the risk of habituation [42] and generally are not recommended. Moreover, physicians should be aware of the risk of developing medication overuse headache as a result of frequent and excessive use of analgesics used in acute therapy. Use of analgesics on more than 2-3 days per week increases the risk of medication overuse headache in individuals with tension-type headache. Recommended dosages of simple analgesics are shown in Table 3.

PREVENTIVE THERAPY

Preventive or prophylactic treatment is considered if the individual experiences a headache on 15 days each month or more, which is consistent with diagnosis of chronic tension-type headache. Controlled randomized trials have demonstrated that tricyclic antidepressant, amitriptyline has a statistically significant and clinically relevant effect in the prophylactic treatment of chronic tension-type headache [43-46]. In addition, amitriptyline reduced secondary variables such as headache duration and frequency, intake of analgesics and myofascial tenderness in individuals with chronic tension-type headache [47]. Amitriptyline should be considered as the drug of first choice in the preventive treatment of chronic tension-type headache (Table 3). In a recent study, Bendtsen and Jensen [48] evaluated

the efficacy of the antidepressant mirtazapine in non-depressed patients with chronic tension-type headache in a randomized, double-blind, placebo-controlled, crossover trial. Mirtazapine was shown to reduce area-under-the-headache curve (duration x intensity of the headache) by 34% more than placebo, and headache frequency, duration and intensity in difficult to treat patients including patients who had not responded to amitriptyline. The efficacy was comparable to that of amitriptyline reported in a previous study using same methodology but with considerably fewer side effects and better tolerability. Mirtazapine should be considered as the drug of second choice for the prophylactic treatment of CTH. In a randomized, double-blind, placebo-controlled, crossover trial the selective serotonin reuptake inhibitor (SSRI) citalopram had no effect on patients with chronic tension-type headache [46]. In a randomized, double-blind, placebo-controlled, crossover trial tizanidine was more effective than placebo at the end of the treatment period and it was suggested that tizanidine was effective in the preventive treatment of chronic tension-type headache in women [49]. However, in a recent study a modified-release formulation of tizanidine in dosages up to 12 mg daily did not differ from placebo [50]. In summary, more placebo-controlled trials are needed to demonstrate the possible efficacy of SSRIs and muscle relaxants in the preventive treatment of chronic tension-type headache. Data on the efficacy of botulinum toxin in the treatment of tension-type headache are based on a limited number of studies with several methodological reservations. The results from the recent trial demonstrated that botulinum toxin was not

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effective in the preventive treatment of chronic tension-type headache [51].

NON-PHARMACOLOGIC THERAPY

Behavioral approaches and physical therapy are widely used as adjunct therapies in tension-type headache. The main goal of these non-pharmacological approaches is to eliminate factors responsible for an increase in frequency and severity of tension-type headache. Few studies have studied these treatment modalities in properly classified patients with tension-type headache using a controlled design. Holroyd et al. [52] reported that cognitive-behavioral (stress management) therapy had comparable effectiveness to amitriptyline in individuals with recurrent tension-type headache. In a large randomized, controlled trial tricyclic antidepressant medication and stress management therapy each produced larger reductions in headache activity, analgesic medication use, and headache-related disability compared to placebo, but antidepressant medication resulted in more rapid improvements in headache [53]. Combined therapy was more likely to produce clinically significant reductions in headache index scores (64% of participants) than antidepressant medication (38% of participants), stress management therapy (35%), or placebo (29%). The authors of the study concluded that combined therapy might improve to higher degree outcome relative to monotherapy [53]. Torelli et al. [54] investigated the therapeutic effect of physical therapy in patients with frequent episodic tension-type headache or chronic tension-type headache. While the average number of days with headache per 4-week period was significantly reduced

by physical therapy compared to waiting list, severity and duration of headache as well as drug consumption did not differ. Interestingly, the number of responders was higher among patients with chronic tension-type headache than among patients with frequent episodic tension-type headache [54].

In summary, verification of the effectiveness of non-pharmacological treatment strategies necessitates more well-designed randomized controlled trials, which should be reported according to the Consolidated Standards of Reporting Trials (CONSORT) guidelines [55].

CONCLUSION

Tension-type headache is the most common primary headache disorder. Frequent episodic and chronic subtypes are associated with considerable disability and represent a therapeutic challenge. It is recommended that patients with frequent episodic and chronic tension-type headache complete a diagnostic headache diary to exclude possible medication overuse headache. Simple analgesics and NSAIDs are the mainstays in the acute therapy of episodic tension-type headache. Amitriptyline should be considered as the drug of first choice in the preventive treatment of chronic tension-type headache. If the patient does not respond to amitriptyline, mirtazapine can be attempted. This may also be drug of first choice, if side effects are a major concern. The SSRIs may be considered if amitriptyline or mirtazapine are not tolerated in patients with concomitant depression. The psychological and behavioral approaches and physiotherapy may be used as adjuncts to standard medication.

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ГОЛОВНАЯ БОЛЬ НАПРЯЖЕНИЯ

Месуд Ашина, ДФ по Мед., Др. Мед. Наук.¹, Саит Ашина²

¹Датский Центр Головной Боли и Отделение Неврологии, Факультет Медицинских Наук, Копенгагенский Университет, Больница Глоструп, Копенгаген, Дания.

²Программа Головной Боли, Отделение Медицины Боли и Паллиативного лечения и Отделение Неврологии, Медицинский Колледж Альберта Эйнштейна, Медицинский Центр Бет Израэль, Нью Йорк, США.

Головные боли напряжения являются наиболее часто встречающимся видом цефалгий. Распространенность данного вида головных болей в общей популяции находится в пределах между 30 и 78%. Головная боль напряжения имеет огромное социально-экономическое влияние, как на индивидуума, так и в целом на общество. К сожалению, это наименее изученный вид первичной головной боли. Патопфизиология головных болей напряжения также достаточно не изучена. Диагностика данной патологии основывается на данных анамнеза, неврологической и инструментальной диагностике. Возможно, в патопфизиологии могут играть роль нарушения в периферической и центральной ноцицептивной системе в сочетании с природными и генетическими факторами. Фармакотерапия эпизодических головных болей напряжения неспецифична и включает анальгетики и нестероидные противовоспалительные средства. Применение трициклических антидепрессантов является основой профилактики хронических головных болей напряжения.

Ключевые слова: головная боль напряжения, классификация, эпидемиология, патопфизиология.

XÜLASƏ

GƏRGİNLİK BAŞ AĞRILARI

Məsud Aşina, TÜFD, TED¹, Sait Aşina²

¹Danimarka Baş Ağrıları Mərkəzi və Nevrologiya kafedrası, Tibb Elmləri Fakültəsi, Kopenhagen Universiteti, Qlostrup xəstəxanası, Kopenhagen, Danimarka.

²Baş Ağrıları Programı, Ağrı Təbabəti və Palliativ müalicə şöbəsi və Nevrologiya şöbəsi, Albert Eynşteyn Tibb Kolleci, Bet İzrael Tibb Mərkəzi, Nyu York, ABŞ.

Gərginlik baş ağrıları sefalgiyaların ən çox rast gəlinən növüdür. Ümumi populyasiyada bu növün təsadüf olunması 30-78% arasındadır. Gərginlik baş ağrıları həm individuuma həm də ümumi cəmiyyətə geniş sosial-iqtisadi təsirə malikdir. Əfsuslar olsun ki, birincili baş ağrılarının bu növü nisbətən az öyrənilmişdir. Gərginlik baş ağrılarının patofiziologiyası da kifayət qədər öyrənilməmişdir. Bu patologiyanın diaqnostikası anamnezin, nevroloji və instrumental diaqnostikanın nəticələrinə əsaslanır. Patofiziologiyanın əsasında periferik və mərkəzi nosiseptiv sistemin pozulmaları ilə yanaşı ətraf mühitin və genetik faktorların rolu da ola bilər. Epizodik gərginlik baş ağrılarının farmakoterapiyası qeyri-spesifik olaraq ağrıkəsici və qeyri steroid iltihab əleyhinə preparatlardan ibarətdir. Gərginlik baş ağrılarının profilaktikasının bünövrəsini trisiklik antidepressantların istifadəsi təşkil edir.

Açar sözlər: Gərginlik baş ağrıları, təsnifat, epidemiologiya, patofiziologiya.