Review Article

Tension type headache

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Abstract:

Tension type headaches are common in clinical practice. Earlier known by various names, the diagnosis has had psychological connotations. Recent evidence has helped clarify the neurobiological basis and the disorder is increasingly considered more in the preview of neurologists. The classification, clinical features, differential diagnosis and treatment of tension type headache are discussed in this paper.

Key words:

Episodic tension type headache, Chronic tension type headache

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Introduction

Tension type headaches (TTH) are recurrent episodes of headache lasting minutes to weeks. The pain is typically pressing or tightening in quality, of mild to moderate intensity, and bilateral in location, and does not worsen with the routine physical activity. Nausea and vomiting is usually absent, but photophobia *or* phonophobia may be present.[1] These headaches were previously known by many terms such as psychogenic headache, stress headache, psychomyogenic headache, muscle contraction headache etc. However, the term "tension type headache" (TTH) has been chosen by the International Classification Headache Diagnosis I (ICHD I)[2] in 1988 and have been retained by ICHD II[1] in 2004 [Table 1]. The words "tension" and "type" underscore its uncertain pathogenesis and indicate that some kind of mental or muscular tension may play a causative role. However, a large number of clinical and neurophysiological studies leave little doubt about its neurobiological basis and takes it away from the realms of psychological diseases.[3-5]

Tension type headache is the most common form of headache. However, most subjects with acute tension type headache never consult a doctor. If necessary they treat themselves with over-the-counter analgesic drugs. Sometimes, however, these can be frequent or chronic (as well as severe) thereby causing major health problems and great suffering to the affected individuals.

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Classification and Nomenclature

In International headache Society classification (ICHD II), tension type headaches have been divided into two forms, episodic (ETTH) and chronic (CTTH) {first digit classification} [Table 1]. The episodic tension type headache has been divided into two groups, namely infrequent and frequent (second digit classification). All the three types of tension type headaches share similar clinical features except for frequency [Table 2]. It is also suggested that on clinical examination, the clinicians should look for pericranial tenderness in patients with tension type headaches and sub-classify them as those associated or not associated with pericranial tenderness (third digit classification).

Uncertain areas

Probable TTH

Clinically, it is difficult at times to distinguish TTH from early phase of migraine attack. Further, many chronic

Table 1: Classification of tension type headache

Tension type headache
Infrequent episodic tension type headache
Associated with pericranial tenderness
Not associated with pericranial tenderness
Frequent episodic tension type headache
Associated with pericranial tenderness
Not associated with pericranial tenderness
Chronic tension type headache
Associated with pericranial tenderness
Not associated with pericranial tenderness

patients of TTH overuse medications and in this setting differentiating medication overuse headache from CTTH becomes difficult. ICHD II has introduced the diagnostic category of probable tension type headache to address this situation.[1] Patients meeting all but one criterion of TTH are listed here [Table 3]. For probable episodic tension type headache, migraine without aura needs to be excluded. For probable CTTH, medication overuse headache (MOH) needs to be ruled out in addition. It is to be noted that ICHD II version requires withdrawal of medication for 2 months and disappearance of headache following the withdrawal to diagnose MOH. This is paradoxical as majority of CTTH patients overuse medications. Hence in 2006, appendix criteria were published to address this problem.[6]

Epidemiology

Prevalence

In a population-based study in Denmark, the lifetime prevalence of TTH was high (78%), but the majority had episodic infrequent TTH (1 day a month or less) without specific need for medical attention.[7] About 24% to 37% had TTH several times a month, 10% had it weekly, and 2% to 3% of the population had chronic TTH, usually lasting for the greater part of a lifetime.[7,8]

Incidence

Incidence of TTH (tension type headaches developing de novo) is difficult to measure. However, in a Danish epidemiologic follow-up study, the annual incidence for TTH was 14.2 per 1000 person years for frequent TTH (female-to-male 3:1), decreasing with age. [9] Risk factors for developing TTH were poor self-rated health, inability to relax after work, and sleeping few hours per night.[9]

Age and sex

In contrast to migraine, in TTH, women are only slightly more affected than male (the female-to-male ratio of TTH is 5:4) and the average age of onset (25 to 30 years) is delayed. The peak prevalence occurs between ages 30 to 39 and decreases slightly with age.[10,11]

Disability

Few studies from Europe[8,12] and the United States[13] have shown that absenteeism resulting from TTH is considerable and can be as high as three times more than that seen in migraine. Indirect costs of non-migraine headaches (of which TTH is the major contributor) are also higher than that of migraine.[14] The disability is higher in patients with psychiatric co-morbidities.[15]

Pathophysiology

Despite numerous clinical and neurophysiological studies, the exact cause of tension headache remains elusive. Detailed discussion on these studies is beyond the scope of this article. A distillate of the currently available data does however suggest that pericranial myofascial mechanisms probably are of importance in episodic TTH, whereas sensitization of pain pathways in the central nervous system resulting from prolonged nociceptive stimuli from pericranial myofascial tissues seems to be responsible for the conversion of episodic to chronic TTH.[4] The model proposed by Bendtsen *et al*.[4] [Figure 1], is attractive for its simplicity but needs to be worked upon further. Clearly, source of this peripheral nociception, remains to be identified and ways to prevent the central sensitization may provide the tools to manage these difficult to treat patients.

Table 2: Frequency characterization of tension type headaches

Feature	Infrequent ETTH	Frequent ETTH	Chronic TTH
Frequency	Less than 12 days / year	More than 12 days and less than 180 days /year	More than 180 days / year
		At least 10 episodes occurring more than 1 day and less than 15 days /month for at least 3 months	More than 15 days / month for at least 3 months

Table 3: Probable tension type headache

Probable tension type headache
Probable infrequent episodic tension type headache
Episodes fulfilling all but one of criteria a-d for 2.1 infrequent episodic tension type headache
Episodes do not fulfill criteria for 1.1 migraine without aura
Not attributed to another disorder
Probable frequent episodic tension type headache
Episodes fulfilling all but one of criteria a-d for 2.2 frequent episodic tension type headache
Episodes do not fulfill criteria for 1.1 migraine without aura
Not attributed to another disorder
Probable chronic tension type headache
As in 2.3 except e Not attributed to another disorder but there is or has been within the last 2 months, medication overuse fulfilling criterion b for any of subforms of 8.2 medication overuse headache

Table 4: Core clinical features of tension type headache

Duration	30 minutes to 7 days
2 out of 4 following headache characters	Bilateral location
	Pressing/ tightening quality (non-pulsating)
	Mild to moderate intensity
	Not aggravated by routine physical activity
Both of the associated symptoms	No nausea and vomiting (anorexia may occur)
	No more than one of photophobia or phonophobia
Not attributed by another disorder	Excluded by clinical history and examination or by suitable investigation if necessary

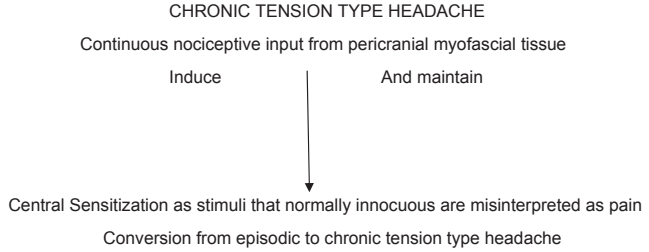

Figure 1: Proposed pathophysiologic model of CTTH. Source: Bendtsen L. Central sensitization in tension-type headache—possible pathophysiological mechanisms. Cephalalgia 2000; 20(5):486–508

Diagnosis

The diagnosis of tension type headache is essentially clinical and relies only on symptoms [Table 4]. Thus a detailed history taking through examination (to rule out any secondary cause) is mandatory. No laboratory tests can make the diagnosis.

Clinical features

Pain

The pain of TTH is usually described as dull, pressure like, constricting or giving a sense of fullness in the head. Quite frequently patients describe their pain as like wearing a tight hat or a tight band around the head, or bearing a heavy burden on the head. Physical activity has no influence on headache intensity in majority of patients. This is in sharp contrast to migraine where the pain worsens on routine physical activity and thus is considered to be one of the best criteria to distinguish between migraine and TTH.[16-18] In the TTH the pain location is usually bilateral in 90% of the patients.[19] Location of pain however varies considerably and can be either interior or posterior.

Accompanying symptoms

Nausea and vomiting if present, rules out the diagnosis of tension type headache. However, about 20% patients of TTH can complain mild-to-moderate anorexia (as per ICHD I criteria) which needs to be distinguished from nausea.[17] Photophobia or phonophobia may be present while presence of both symptoms is not allowed.

Precipitating and aggravating factors

Stress, lack of sleep, and not eating on time are among the most common headache precipitants reported by both migraine patients and those with tension type headache.[18,20] Occasionally alcohol and menstruation have also been reported as headache precipitants by some patients with ETTH.[20] Further, conditions that are known to commonly aggravate established headache attacks in patients with migraine are also reported as aggravating factors by a significant number of ETTH patients. Thus these features are not very helpful to distinguish TTH from migraine although they may have a bearing on management.

Diurnal variations

TTH is often reported to start at some time during the day and to increase slowly. Then the headache remains throughout the day, and is often unaltered during widely varying activities although some people may have an aggravation by late evening.

Physical examination

As the diagnosis of TTH requires exclusion of other causative disorders, good and detailed clinical history and careful physical and neurological examinations are mandatory. The physical examination should include manual palpation of the pericranial muscles to identify tender points and trigger points. Tender points are areas where manual pressure induces local pain, and trigger points at areas of localized deep tenderness where sustained pressure also induces referred pain in another area in the region.

Table 5: Differential diagnosis of chronic tension type headaches

Medication overuse headache
Chronic posttraumatic headache
Sinus/ eye disease
Temporomandibular joint disorder
Idiopathic intracranial hypertension
Brain tumor
Psychiatric disorder
Cervical spondylosis

Differential Diagnosis

Although tension type headache is present in up to 78% of headache patients in population-based studies, it is the least distinct of all headache types. Its clinical diagnosis is based chiefly on negative features (that is, absence of symptoms that characterize other primary or secondary headaches). For example, the absence of unilaterality, absence of pulsatile and throbbing character, lack of aggravation by physical activity, lack of nausea and vomiting, lack of photo and photophobia etc.

However, it is to be understood that a minority of TTH patients can have some of these features. For example 18% may have pulsatile headache, 10% unilateral pain, 28% aggravation on routine physical activity, 18% anorexia, 4% nausea, and 11% photophobia.[21] Further, many of the secondary headache types may mimic TTH at some stage of their clinical evolution [the common secondary causes mimicking TTH are listed in the Table 5]. Therefore, as a general dictum, atypical history or abnormal clinical examination in patients of suspected TTH indicate the need for further investigations by computed tomography or magnetic resonance imaging. However, the vast majority with typical history and normal clinical examination will have a very low likelihood of significant intracranial disease and therefore do not need further investigations.

Treatment

Making the correct diagnosis

The importance of thorough history and examination in patients with headache has already been emphasized. It is very important to exclude secondary headaches, to recognize comorbid conditions and finally to establish whether TTH coexist with migraine. It is also extremely important to detect whether the headaches are being aggravated by overuse of medications. In many patients with long history of typical headaches with normal examination, the diagnosis of TTH can be made without special investigations; at the same time, if felt necessary the investigations like neuro-imaging should not be withheld to exclude a secondary cause.

Talking to the patient

Many patients of frequent ETTH and CTTH have grave concerns about possibility of a serious disease such a brain tumor. Many patients also fail to understand how it is possible to have headaches with normal clinical examination and a normal scan. However, once these patients get to know that the physician is taking interest in their problems, they feel reassured. Further, correct explanations can allay these

concerns. Brief explanation about the brain's pain-modulating mechanism may be helpful. The physician should refrain from using such terms like "psychosomatic" or "depression" which might be perceived negatively by the patient. They should be told that the control of their headache is possible and they should not lose hope.

Use of a headache diary

The patient can keep a written record of the frequency and severity of attacks and the medications used in headache diaries. This often is very helpful for the physicians. They can measure the progression of the frequency and severity. Also sometimes specific headache triggers and true estimation of medication overuse can be unearthed through these diaries.

Treatment options

The treatment of tension type headaches can be divided into two broad groups Pharmacologic and non-pharmacologic. Both these strategies can be applied for acute treatment as well as for prevention.

Treatment of episodic tension type headache

As discussed at the outset, most of the patients of infrequent episodic tension type headache will not report to the physicians and usually take over-the-counter analgesics. For patients with frequent episodic tension type headache, simple analgesics and NSAIDSs are the mainstays in the acute therapy. Aspirin (500 mg and 1000 mg), acetaminophen (1000 mg) are effective in the acute therapy for TTH as shown by randomized trials.[22] There is no consistent difference in efficacy between aspirin and acetaminophen.[22] Other non-steroidal anti-inflammatory drugs (NSAIDs), ibuprofen (200–400 mg), naproxen sodium (375–550 mg), ketoprofen (25–50 mg), and diclofenac potassium (50–100 mg) all have been demonstrated more effective than placebo in acute TTH.[23] These NSAIDs are probably more effective than acetaminophen and aspirin as shown in many studies although the results were not always unequivocal.[23] Caffeine, codeine, sedatives, or tranquilizers have often been combined to increase the efficacy of NSAIDs which however, should be avoided because of the risk for dependency, abuse, and chronification of the headache.[23] Care should be taken to avoid their over use. Opiates are to be avoided. Evidence for efficacy of muscle relaxants is weak and there is risk for habituation. Hence these are not to be recommended. Some patients with ETTH who also have migraine may respond to triptans. However, these patients should be clearly taught how to recognize and differentiate between the symptoms of migraine and ETTH so that for a given attack they can take the appropriate drug. Nonpharmacologic treatment in the form of relaxation training can be of benefit in recurrent ETTH.

Prevention of recurrent attacks of ETTH first should take into account two issues. First, known triggers should be avoided. For example, missing a meal can trigger an attack of ETTH just like in migraine and therefore should be avoided. There is some new evidence that estrogens can trigger ETTH similar to migraine and therefore consideration should be given for their withdrawal if indicated. Secondly, drug overuse should be identified and stopped as just like migraine it renders prophylactic therapy refractory.

The most efficacious drug for preventing recurrent ETTH is amitriptyline.[24] It should be started on low dose (10 mg to 25 mg per day) and gradually increased if needed. Its side effects are to be explained to the patients and closely monitored (discussed below). Nonpharmacologic treatment in the form of relaxation therapy and biofeedback have been found to be equally good but need trained and skilled personnel to administer and available mostly in large centers (see below).

Treatment of chronic tension type headache

Acute therapy has little role in CTTH since this is a chronic condition by definition and most patients have headaches for most of the days in a month. On the contrary, a large majority of these patients have already tried a number of analgesics and NSAIDs and some of them may even be addicted to opiods. Further, many of patients suffer for a long time, changing physicians frequently and finally resign to the idea that their headaches can not be treated. Thus, these patients are very difficult to treat. The first thing is to stop the overuse of these medications when present. Many patients with TTH have significant comorbidities like depression and anxiety which need proper evaluation and management. In general, pharmacotherapy, behavioral modalities and physical medicine are effective for prevention and should be used in combination to achieve optimum results.[25]

Pharmacotherapy

Amitriptyline

Tricyclic antidepressant amitriptyline has been most extensive studied and has been found to most effective for the treatment of CTTH.[24] Way back in 1964, Lance *et al*. conducted the first controlled cross over trial and demonstrated the superiority of amitriptyline over placebo in patients with CTTH.[26] Since then, a number of studies tested the various doses and compared amitriptyline with other antidepressants like citalopram.[27-30] By and large, doses upto 75 mg of amitriptyline was found to be useful. Mechanism of action of amitriptyline in CTTH is uncertain. Possible explanations include serotonin reuptake inhibition, potentiation of endogenous opioids, NMDA receptor antagonism and blockade of ion channels.[30]

Amitriptyline should be started on low dose (10 mg to 25 mg per day) and titrated by 10-25 mg weekly till the therapeutic effect or the side effects appear. Significant clinical effect of Amtriptlyline is usually seen by the end of one week and should be apparent by 3-4 weeks.[30] If the patient does not show an improvement by 4 weeks of treatment, serious consideration should be given for alternatives. It is also important to clarify to the patients that this drug is being given for pain (and not as antidepressant) to improve compliance. The common side effects of the drug are dry mouth and drowsiness. Serious side effects like cardiac arrhythmias, precipitation of glaucoma and urinary retention can occur in predisposed, especially elderly subjects. Nortriptyline, a drug closely related to amitriptyline has also been found to be useful in CTTH patients in one study. Usually, amitriptyline is continued for 6 months following which withdrawal is attempted. Upon withdrawal, some patients continue to remain headache free while others start to have headaches again. These patients usually require long term treatment.

Other antidepressants

Other antidepressants like SSRIs and tetracyclic have been found to be not so useful. Although studies have found modest effect on prevention of CTTH by drugs like citalopram,[30] sertaline,[31]

mianserine,[32] fluvoxamine,[32] paroxetine,[33] velnaflaxine (extended release)[34] and a D2 antagonist sulpiride,[33] there are no robust data for recommending these agents yet. A new drug, mirtazapine,[35] a noradrenergic and serotonergic antidepressant however has been found to be efficacious and can be given in situations where amitriptyline is either ineffective or contraindicated. At a dose of 30 mg/day, it reduced headache index by 34% more than placebo in difficult-to-treat patients, including patients who had not responded to amitriptyline.

Muscle relaxants

The role of muscle relaxants in prevention of CTTH is debatable. Centrally acting muscle relaxant like tizanidine may have some benefit but is not recommended routinely. Peripherally acting muscle relaxants have no role. At least 3 studies have tested tizanidine in CTTH and while two studies[36,37] showed modest benefit, one failed to show any.[38]

Botulinum toxin type A

Following an open labeled study in which Botulinum Toxin Type A injection was shown to be efficacious in CTTH patients,[39] few controlled studies have been undertaken.[40-43] The results have been conflicting and largely negative. Hence, Botulinum Toxin Type A is not recommended for CTTH prevention.

Nonpharmacologic therapy

Non-pharmacologic management includes physical therapy and psychologic treatment. Ideally these should be tried in all patients as adjuncts to pharmacotherapy. These may however be more attractive to patients reluctant to use drugs.

(i) Physical therapy: It is the most commonly used non-pharmacologic treatment of TTH. Its components include improvement of posture, relaxation, exercise programs, hot and cold packs, ultrasound, and electrical stimulation.[44] Active treatment strategies generally are recommended. A controlled study combining various techniques, such as massage, relaxation, and home-based exercises found a modest effect.[45] Adding craniocervical training to classical physiotherapy may be better than physiotherapy alone.[46]

(ii) Psychologic therapy: This includes relaxation training, EMG biofeedback and cognitive-behavioral therapy.[47] During relaxation training, the patients consciously reduce muscle tension and autonomic arousal that can precipitate and result from headaches. Thus, it is a strategy for training in self-regulation. EMG biofeedback helps the patients to develop control over pericranial muscle tension. The patients use the feedbacks that are presented with an auditory or visual display of electrical activity of the muscles in the face, neck, or shoulders. It is uncertain whether reductions in muscle tension or cognitive changes of self efficacy account for improvement. The latter is more likely. In cognitive-behavioral therapy, patients are taught to identify thoughts and beliefs that generate stress and aggravate headaches. Although treatment outcome of psychologic therapies is difficult to measure, there seems to be reasonable scientific support for their effectiveness.

(iii) Miscellaneous treatments: Oromandibular treatment with occlusal splints is an attractive option but lack scientific data and hence not recommended for routine use.[48] Similarly for acupuncture, there are conflicting results regarding its efficacy for the treatment of TTH.[49-51] Spinal manipulation has shown no effect on the treatment of episodic TTH.[52]

Prognosis

Data on long term natural course of the disease are sparse. In a 12-year longitudinal epidemiologic study from Denmark,[7] out of 549 persons who were followed up, 146 subjects had frequent episodic TTH and 15 had chronic TTH at baseline. Of these, 45% experienced remission, 39% had unchanged frequent episodic TTH, and 16% had unchanged or newly developed chronic TTH at follow-up. Patients who fared poorly include those with baseline chronic TTH, coexisting migraine, unmarried, and had insomnia.

Conclusion

Vast majority of TTH patients do not seek medical attention. Only when ETTH attacks become frequent or the headache changes into chronic type (CTTH), patients seek treatment. Diagnosis of TTH is difficult as headache characteristics are non specific. Diagnosis is essentially clinical and based on negative associations and by exclusion. A typical case or those with abnormal neurological examination must be investigated thoroughly to exclude secondary headaches. Coexistent migraine or medication abuse must be identified. CTTH is difficult to treat but with correct diagnosis and approach, these patients can be treated and their headaches controlled. Significant psychological comorbidities when present need physician's attention. Medication abuse (analgesics, opiods and triptans) must be identified and corrected to get optimum therapeutic results. The best preventive drug for frequent ETTH and CTTH is amitriptyline. Non-pharmacologic treatment should be combined with drugs to get optimum results.

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