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Review Article

Tension - Type, the Forgotten Headache
A Common but under Treated Condition

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Abstract

Headache is a universal problem. Of all the types of headache, the commonest is Tension-type Headache. The word 'tension' implies that this type of headache can be attributed entirely to tension or stress, which may make people with this type of headache reluctant to consult a physician. Physicians misperceptions that tension-type headache are mild, benign, self-treatable and occur solely due to stress may further trivialize the problem. Thus this disease is often under-diagnosed and not treated properly. This article describes the disease and its management and is being published to increases awareness of the disease amongst the physicians.

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Introduction

Headache has troubled humankind from the dawn of civilization. Evidence of trepanation, an early form of neurosurgery, was found on skulls from 7000 BC. Migraine symptoms, including headache, aura, prodrome, nausea, vomiting, and familial tendency have been described for over 1000 years.

The pain sensitive structures in the head are blood vessels, meninges, cranial nerves, nerve roots, bones, scalp and muscles, sinus mucosa and teeth. Any disease process that distort or displace these structures can produce headache.

Up to 88% of women and 69% of men experience tension-type headache (TTH) during their lifetime [1]. The word 'tension' implies that this type of headache can be attributed entirely to tension or stress, which may make people with this type of headache reluctant to consult a physician. Physicians misperceptions that TTH are mild, benign, self-treatable and occur solely due to stress may further trivialize the problem.

According to the 1988 International Headache Society (HIS) classification of headache, TTH refers to a condition characterized by bilateral mild to moderate pain and pressure that often is described as similar to that of having "a vise around the head". However, some patients with TTH may experience severe pain and marked disability.

TTH usually is not associated with migraine like symptoms such as nausea, vomiting, increased pain with physical exertion, photophobia and phonophobia. Stress may be one of many precipitating factors, but the underlying cause is unknown. Research advances into this type of headache have not kept pace with exciting new migraine discoveries.

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Table-1. International Headache Society criteria for Tension Type Headache2

Primary diagnosis	**Subdivision diagnosis**
1. Headache has *at least* two of the following characteristics: * Bilateral pain * Pressure * Mild to moderate pain * No increased pain with physical exertion	1. Episodic (< 15 days/month) or chronic (> 15 days/month for > 6 months)
2. *And* no more than one of the following: * Sensitivity to light * Sensitivity to sound	2. Associated with or not associated with coexisting pericranial muscle tenderness (Diagnosed by manual palpation or EMG studies)
3. *And* neither of the following: * Nausea * Vomiting	
4. *And* duration of 30 minutes to 7 days	

Table-2. Differential Diagnosis of Headache

I. Primary diagnosis -	Nonvascular: Tension-type Vascular: Migraine, Cluster
II. Secondary (organic) diagnosis – Vascular:	Subarachnoid hemorrhage; Subdural hematoma; Unruptured arteriovenous malformation or aneurysm; Ischemic cerebovascular disease; Temporal arteritis; Arterial hypertension; Cerebral venous thrombosis;
Non Vascular Intracranial disorders:	Idiopathic intracranial hypertension; Intracranial hypotension following Lumbar puncture; Intracranial neoplasm; Intracranial infection or meningitis;
Substances that act as triggers:	Medications (e.g. nitrates, OTC drugs); Foods (e.g. Monosodium glutamate, alcohol); Rebound (e.g. Caffeine, analgesic, ergot); Exposures (e.g. Carbon monoxide);
Metabolic disorders:	Hypoxia (COPD, Sleep apnea); Hypercapnia; Hypoglycemia;
Abnormalities of extracranial structures:	Eye (e.g. glucoma, refractive errors); Ears & sinuses (e.g. sinusitis); Skull (e.g. Paget's disease, multiple myeloma); Neck (e.g. spondylosis);

Epidemiologic Factors

About 80% of the population will experience a TTH at some time during their life. An estimated prevalence of 86% in women and 63% in men means that it is more probable to have experienced a TTH than not.[1] Headache is one of the 10 chief complaints of patients seen in primary care practice, and 47% of headaches are TTH.[3] During childhood there is no male or female predominance for TTH, but during adulthood it is more commonly experienced by women (F: M ratio 5:4).[4] Onset of TTH is before age 20 in 40% of affected persons, between ages 20 and 40 in 40%, and between ages 40 & 50 in 18%.[3] Three percent of the general population (5% of women, 2% of men) experience chronic TTH, defined as more than 180 headache days per year.[1] Prevalence of chronic TTH increases with age; the inverse is true for episodic TTH.

Social Impact

TTH has greater socio-economic impact than any other headache type because of its high prevalence and wide spectrum of disability. About 820 annual workdays for every 1,000 persons are lost on

account of TTH (versus 270 days on account of migraine).[5] The reduced productivity of those who remain at work despite headache has an even greater impact. Overall, TTH have a negative effect on the emotional life of affected persons, resulting in marked reductions in quality of life and frequency of social and family activities. Despite this, less than 15% of people seek medical attention.[3] Proposed reasons for this disparity are lack of respect, empathy, and understanding by physicians, fear of not being taken seriously, trivialization of the disorder by media advertising and jokes, and the wide spread misperception that the headache has a purely psychological basis.

Precipitating Factors

TTH triggers are not always identifiable or consistent, and multiple triggers may have the additive effect of lowering the threshold of headache activation. In the past, studies have demonstrated few TTH triggers other then emotional stress; more recent studies have found similar headache precipitants for both TTH and migraine (Table 3).

TTH may be aggravated by barosinusitis from allergic or structural turbinate congestion or by functional disturbance of the masticatory muscles around the temporomandibular joint from clenching, bruxism or malocclusion. Head trauma, even a mild whiplash injury not associated with a blow to the head or loss of consciousness may initiate or exacerbate TTH, possibly on a chronic basis. That patients with first onset of TTH after trauma have a genetic predisposition for the disorder is suspected but unproven.[7]

Stress is undoubtedly the most common precipitant, triggering up to 80% of TTH. Studies have demonstrated that, compared with control group, patients with TTH have similar major stressful life events but perceive more events as "hassles" and have less effective coping strategies (e.g. avoidance, self-criticism).[8] The increased prevalence of co-morbid anxiety, depression, and somatoform disorders among people with TTH is still debatable. It is uncertain whether these disorders can initiate, contribute to, or maintain TTH; whether they are the result of a chronic pain condition; or whether they are due to a genetic susceptibility and serotonin abnormalities.

Table 3. Potential Triggers of Tension - Type Headache [6]

- Stress (e.g. everyday hassles, family crisis, heavy workload, unpleasant work etc)
- Change in sleep regimen (e.g. shift work, oversleeping)
- Skipping meals
- Certain foods (e.g. caffeine, alcohol, chocolate)
- Environmental factors (e.g. sun glare, odors, smoke, sustained posture)
- Female hormonal changes (e.g. menses, menopause, pregnancy, exogenous hormones)
- Medications used for co-morbid conditions (e.g. nitrates, SSRI, antihypertensive)
- Overuse of headache medications (e.g. analgesic and caffeine combinations, ergot, and opiates)

Pathophysiologic Mechanism

The cause of TTH is unknown, but most research has focused on a peripheral mechanism pertaining to pericranial muscles tenderness, thus explaining the previous term "muscle contraction headache". Electromyograhic readings are conflicting and indicate tenderness is usually greater in migraine patients than in patients with TTH during a headache.[9] Sustained muscle contraction from physical or emotional causes compress intraspinal arterioles, causing ischemia, accumulation of noxious metabolites, or both, which results in localized tenderness.[8]

Some physicians believe that migraine and TTH is part of the same underlying disorder and according to them TTH has a purely central mechanism.[10] Studies have shown that there are reductions in platelet and serum serotonin, epinephrine,

norepinephrine, and dopamine levels and these findings support the idea that an alteration in amine central system results in depressed pain suppression and activated pain pathways in TTH.[11]

However, the cause of TTH is most likely multifactorial and best described by Olesen's vascular-myogenic-supraspinal model.[12] It is the convergence of multiple pain pathways - vascular, myogenic, supraspinal, or all of these that enter the caudate nucleus of the trigeminovascular system. This in combination with other precipitating factors in predisposed persons, determine whether headache activation threshold is met.[9]

Diagnosis

There are no objective signs for TTH. An accurate diagnosis rests on comprehensive history taking, which only helps to eliminate secondary, potentially life-threatening diagnosis (table-2). The principal feature of TTH is mild to moderate pressure-type pain, usually on both sides of the head, often described as occurring in the frontal or occipital areas or as a band around the head. Associated symptoms may include fatigue, irritability, and difficulty concentrating, but migraine-like symptoms usually are absent. TTHs typically last 30 minutes to 1 week, but some people experience them daily.

Physical classification is rarely helpful but a detail neurological and fundoscopic evaluation is essential in every patient. Diagnostic studies are not required unless the history or physical examination reveals features that suggest an organic cause: atypical headache features not fulfilling IHS criteria, sudden onset of or change in characteristics of headache (>50 years of age), or abnormal findings on physical examination.

Generally, a severe headache of sudden onset is best evaluated by computed tomography of head, and, possibly, lumbar puncture to rule out subarachnoid hemorrhage. Magnetic resonance imagining (MRI) of the brain is preferred for evaluating chronic progressive headache because of its higher sensitivity for tumors, aneurysms, and posterior fossa lesions. Interestingly, non specific white matter abnormalities are an incidental finding on MRI scans in many patients with TTH as well as migraine (32% & 34% respectively, versus 7.4% in controls).[13] Laboratory studies may identify suspected secondary causes of headache (e.g. temporal arteritis, anemia, thyroid or metabolic abnormalities, Lyme disease) or may be done to monitor for toxic effects from headache medication. Electrocardiography may be performed before prescribing drugs known for altering conduction times (e.g. TCA) or cause vasoconstriction. Standardized psychometric testing may identify co-morbid psychiatric disorders, but these abnormalities may be the result of chronic headache pain.

Treatment

A recent study14 found that 60% of those reporting severe headaches used only over the counter (OTC) medications. Many get adequate relief with OTC abortive medications, but others experience a marked reduction in function and quality of life. Inappropriately frequent use (>2 days a week) of OTC abortive medications may cause or maintain a chronic daily headache pattern called analgesic rebound.[15] In analgesic rebound, headache frequency and severity typically increase, and patients require regular dosing of the offending medication to avoid becoming incapacitated by headache.

A comprehensive therapeutic approach, incorporating both nonpharmacologic and pharmacologic means, is successful for over 90% of patients with TTH. Nonpharmacologic approaches include regulation of sleep and meal schedules, avoidance of headache precipitants and relaxation techniques (table-4). Many patients seek alternative therapies as most headache therapies have a 40% placebo response, which is dependent on patient's faith on the treatment and the physician prescribing it.

Psychological stresses or comorbidities should be addressed for the best possible therapeutic outcome. Family or individual psychotherapy, or

both may help patients to a great extent. Generally, families should de-emphasize concerns regarding headaches and encourage the patients to maintain as normal a lifestyle as possible.

If pharmacologic agents are indicated, abortive medications to relieve headaches may be

used for infrequent headaches (occurring <2 days a week) or in an addition to a daily prophylactic medication for more frequent or severe headaches.

Prophylaxis should be considered if significant disability occurs with attacks, if abortive medications cannot be used because of co-morbidity or a history of substance abuse, or if preventive medications enhance the abortive medication effect. The mainstays of abortive therapy are analgesics and muscle relaxants (table-5). OTC analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) have been found to be effective in clinical trails. Little scientific evidence is available to support the effectiveness of muscle relaxants. Caffeine, used as an analgesic adjuvant in OTC and prescription barbiturate combinations, increases the absorption, peak concentration, and analgesic potency of medication as much as 40%. Interestingly, sumatriptan, used for migraine, has been found to b effective in subgroups of patients with TTH who experience frequent vascular headache qualities.[16]

Abortive migraine therapy has advanced from a stepped care to a stratified care approach, but no such schema has been proposed for TTH. A proposed stratified care approach is shown in table-6. The stepped care approach involves beginning treatment of all patients with the weakest medications and slowly advancing to stronger medications on the basis of response. In a stratified care model, more effective medications may be considered as first line therapy for patients experiencing more disabling headaches. This is true as long as the headache is not frequent (< 2 days a week) and the patient is at low risk for habituation and has no history of substance abuse.

Tricyclic antidepressant are the mainstay of prophylactic therapy for TTH.[17] Selection of a TCA is usually based on the presence of sleep disturbances, with poor sleepers receiving the more sedating drugs. The average maintenance dose of TCA is 50 to 75 mg daily. Despite their improved side effect profile SSRI antidepressants are less reliable for headache prophylaxis. Atypical antidepressant classes have potential but unproven benefits.

Physicians who feel that migraine and TTH are common entities propose that all migraine prophylactics are also effective for TTH. However few controlled studies of beta-blockers, calcium channel blockers or anticonvulsants used for TTH is available. Botulinum toxin injection into the frontalis muscle has been studied as a potential headache treatment, but further studies need to identify the group of patients who are best suited for this treatment.[18]

A collaborative relationship with patients, making them active participant in their care, is likely to achieve higher treatment success rate. Patients should be educated about realistic treatment expectations. Physician should explain that a 4 to 8 week drug trail might be required because of delayed effectiveness, that combinations of medications may be required, and that overuse of analgesics negates treatment effect.

Preventive medications are not cure; successful therapy is considered to be a 50% reduction in headache frequency, a reduction in headache intensity or duration, an enhancement of abortive medication effect, or all of these.

Treatment is a balance between medication efficacy and adverse effects. Headache is a dynamic condition, and regular follow up is required to reassess the need for medication adjustments and to reinforce nonpharmacologic regimens. When patients are doing well reduction of preventive medications should be attempted. The art of headache treatment is knowing when to maintain therapy and reassure patients, when to make minor adjustment in medications and when to change the entire treatment regimen.

Table 4. Non - Pharmacologic Approaches to Treat Tension - Type Headache

1. Regulation of lifestyle
    * Maintain regular sleep schedule * Eat regular meals
    * Avoid known dietary triggers * Get regular aerobic exercise
2. Minimization of emotional stresses
    * Plan ahead and avoid stressful situations * Learn biofeedback * Meditate
    * Increase undemanding leisure activities * Consider individual or family psychotherapy
3. Avoidance of environmental precipitants
    * Wear sunglasses * Avoid smoke, strong order and noisy areas
    * Maintain proper posture, limit sustained positions
4. Physical therapy techniques
    Heat, ice, ultrasound, transcutaneous electrical nerve stimulation
    Massage or cervical traction
    Stretching and strengthening exercise for cervical musculature
    Trigger point stretching, compression, Injection
5. Osteopathic or chiropractic manipulations
6. Alternative therapies
    Acupuncture, Acupressure
    Therapeutic touch, Aromatherapy
    Topical salves (e.g., salicylic acid, and piroxicam)

Table 5. Pharmacologic Treatment of Tension – Type Headache

A. Abortive therapy
    Simple analgesics without caffeine
    Simple analgesic combinations with caffeine
    Non-steroidal anti-inflammatory drugs
    Muscle relaxant with or without an analgesic combination
    Barbiturate or analgesic compound
    Narcotic analgesics

B. Prophylactic therapy
    Non-steroidal anti-inflammatory drugs
    Tricyclic antidepressant
    Sedating (e.g. Amitryptyline, triimipramine)

    Intermediate sedation (e.g. Nortryptyline, imipramine)

    Nonsedating (Desipramine, Protriptyline)
    Selective serotonin reuptake inhibitors (e.g. Fluoxetine, Sertraline, Citalopram)
    Other Antidepressant (e.g. Venlafaxine, Bupropion)
    Monoamine oxidase inhibitors (e.g., phenelzine)

Table 6. Stratified Care Approach To Tension – Type Headache
Management Based On Frequency And Degree Of Disability *

Infrequent

Low disability

- Non pharmacologic approach only
- OTC simple analgesics with or without caffeine
- Non-steroidal anti-inflammatory drugs
- No prophylaxis

High disability

- Narcotic analgesics
- Non-steroidal anti-inflammatory drugs with or without analgesic adjuvant
- Muscle relaxants and **Anxiolytics**
- Barbiturate and analgesic compounds
- Consider prophylaxis

Frequent

Low disability

- Non pharmacologic approach only
- OTC simple analgesics without caffeine
- Non-steroidal anti-inflammatory drugs
- Consider prophylaxis

High disability

- Muscle relaxants
- Non-steroidal anti-inflammatory drugs
- Prophylaxis indicated

Headache frequency - Infrequent: < 2 days per week.
Low disability means no to moderate impairment in function.
High disability means moderate to severe impairment.
High potential for analgesic overuse; avoid caffeine products, anxiolytics and narcotic analgesics.

Conclusion

Recent advances in the treatment and understanding of migraine have overshadowed concerns about TTH, the most prevalent headache disorder. Because of its high prevalence and wide spectrum of disability, TTH is the most important headache type in regard to reduction in work productivity, quality of life and socioeconomic impact.

Future research should address the pathophysiology of TTH. Well-controlled clinical trails are needed to clarify the best treatment approaches and determine whether standard migraine medications may be used for TTH. Patient education and more aggressive treatment regimens would likely increase use of the healthcare system and decrease the large indirect economic cost attributable to this often inadequately treated disorder.

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