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Sleep and Tension-Type Headache

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Abstract

Purpose of Review Tension-type headache (TTH) is a common neurological condition that is related to sleep dysfunction. This review discusses recent evidence for the association between TTH and sleep disturbances.

Recent Findings There is an increasing evidence for the association of TTH with sleep disturbances including insomnia, poor sleep quality, excessive daytime sleepiness, insufficient sleep, and shift working. Most studies have reported that sleep disturbances are more prevalent among subjects with TTH than among subjects without headaches. Clinical presentations of TTH are more exacerbated in TTH subjects with sleep disturbances than in those without sleep disturbances. Further, the close association of TTH with sleep disturbances is more robust in subjects with chronic TTH than in those with episodic TTH. Growing evidence highlights the association of TTH with psychiatric comorbidity, which is closely associated with sleep disturbances.

Summary Recent advances in our understanding of the association between sleep and TTH will help in improved diagnosis and treatment of TTH and sleep disturbances.

Keywords Headache · Insomnia · Obstructive sleep apnoea · Sleep · Tension-type · Sleep quality

Introduction

Tension-type headache (TTH) is a prevalent neurological disorder and was estimated to affect 1.89 billion people globally in 2016 [1, 2]. TTH is usually considered a mild and non-disabling disorder but can cause disability, especially in frequent episodic or chronic form. Owing to its high prevalence, the burden caused by TTH is substantial and causes 7.2 million years lived with disability worldwide [1].

The lifetime prevalence of TTH is as high as 89% [3]. The majority of individuals with TTH on 15 days or less per month are classified as having episodic tension-type headache (ETTH). Nevertheless, 0.5–3% of the population have chronic tension-type headache (CTTH) on 15 days or more per month for more than 3 months [4]. CTTH differs from ETTH not only in frequency but also with respect to pathophysiology, greater psychiatric comorbidities, less responsiveness to treatment, greater disability, and greater use of medications [5].

Sleep is the major modulator of precipitation, aggravation, chronification, and relief of primary headache disorder [6, 7••, 8, 9]. Sleep disturbances such as obstructive sleep apnea (OSA), insomnia, and bruxism are common in the general population [10–12]. TTH is the most common headache disorder that is often associated with sleep disturbances [9, 13, 14].

Both the third edition of the International Classification of Headache Disorders (ICHD-3) and the third edition of the International Classification of Headache Disorders (ICSD-3) included descriptions on the relatedness of headache and

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sleep. ICSD-3 described it in the Appendix as "sleep-related medical and neurological disorders". Sleep-related headache disorders included migraine, cluster headache, chronic paroxysmal hemicrania, hypnic headache, and secondary headaches. However, the most common sleep-related headache, TTH, was not listed [15]. ICHD-3 described the association of migraine, cluster headache, hypnic headache, primacy cough headache, sleep apnea headache, headache attributed to fasting, and high-altitude headache with sleep but did not include the association between sleep and TTH [4]. This article will provide an updated review on the association between sleep and TTH with the aim of improving the management of TTH and sleep disturbances.

Methods

We investigated articles regarding the relationship between TTH and sleep using a systematic search via the PubMed (incorporating MEDLINE) site. The search included full papers and abstracts published in English published until 2018. Papers published in languages other than English were excluded. Searches were performed on January 07, 2019, using a combination of the search terms "tension-type headache", "sleep", "sleep disturbance", and "insomnia". Search strings were entered into PubMed as free text with no limits to minimize the possibility of omitting relevant records. The search retrieved 224 records from the PubMed search. Three authors (SJC, TJS, and MKC) subsequently reviewed these records for relevance and selected 43 records. Additional searches were performed with fewer or more terms to ensure that all potentially relevant studies were identified. As a result, an additional 35 articles were used in this review (Fig. 1).

Results

Triggering TTH by Sleep Disturbances

Sleep disturbances are commonly encountered triggers of TTH. Sleep disturbances were the second most common trigger of TTH in a clinic-based study including 334 patients with TTH [16]. Both lack of sleep and excessive sleep are common triggers of TTH: lack of sleep was reported in 26 to 72% of TTH patients, and excessive sleep was reported in 13% [17, 18]. A prospective time-series analysis demonstrated a significant association of sleep and headache intensity. Short sleep (< 6 h) and long sleep (> 8 h) duration was associated with increased headache intensity over a 2-day period [8]. An actigraphic study reported that excessive sleep was associated with more severe headache intensity in TTH participants [19].

Sleep Disturbances and TTH

Insomnia

Insomnia is considered a major risk factor for increased headache frequency, particularly in TTH [20, 21•, 22]. In a Korean general population-based study, the prevalence of insomnia (Insomnia Severity Index [ISI] total score ≥ 10) among participants with TTH was significantly higher than that among participants without headache (13.2% vs. 5.8%, $p < 0.001$). In this study, factors contributing to insomnia among people with TTH were anxiety (odds ratio [OR] = 3.0, 95% confidence interval [CI] = 1.4–6.7), depression (OR = 5.8, 95% CI = 2.0–16.3), and poor sleep quality (Pittsburgh Sleep Quality Index [PSQI] ≥ 6) (OR = 9.9, 95% CI = 5.1–19.2) [23•]. Conversely, individuals with insomnia had an increased (OR = 2.3, 95% CI = 1.1–5.0) risk of having TTH after adjusting psychiatric comorbidity in a community-based study from Hong Kong [24]. The third Nord-Trøndelag Health Survey further identified that individuals with TTH had a 40% higher risk (relative risk [RR] = 1.4, 95% CI = 1.2–1.8) for developing insomnia after 11 years [25]. The study also found that individuals with non-migraine headache had an increased risk (OR = 1.7, 95% CI = 1.5–1.8) of developing insomnia after 11 years [26]. Considering that most non-migraineous headache in population-based setting can be classified as TTH, these findings suggest that insomnia and TTH has a bidirectional comorbidity [27]. Bidirectional comorbidity in epidemiological study suggests sharing pathophysiological mechanisms between two conditions [22]. Studies on the association between insomnia and TTH were summarized on Table 1.

OSA and Habitual Snoring

The relationship between obstructive sleep apnea (OSA) and TTH is controversial. Although the pain characteristics of sleep apnea headaches resemble those of TTH, headaches associated with sleep apnea are defined separately in the ICHD-3 and can be distinguished from TTH. To date, OSA does not influence the presence and frequency of TTH, and there is no definitive evidence for an association between TTH and OSA [28–30]. A cross-sectional study reported that there was an improvement in overall subtypes of headache after a continuous positive airway pressure (CPAP) trial but improvement in TTH subtype alone after CPAP was not identified [31]. A population-based study in Norway reported no significant relationship between TTH and OSA, with adjusted ORs for frequent TTH of 0.95 (95% CI = 0.55–1.62) and CTTH of 1.91 (95% CI = 0.37–9.85) [28]. In contrast, a Taiwanese population-based study found that the prevalence of TTH

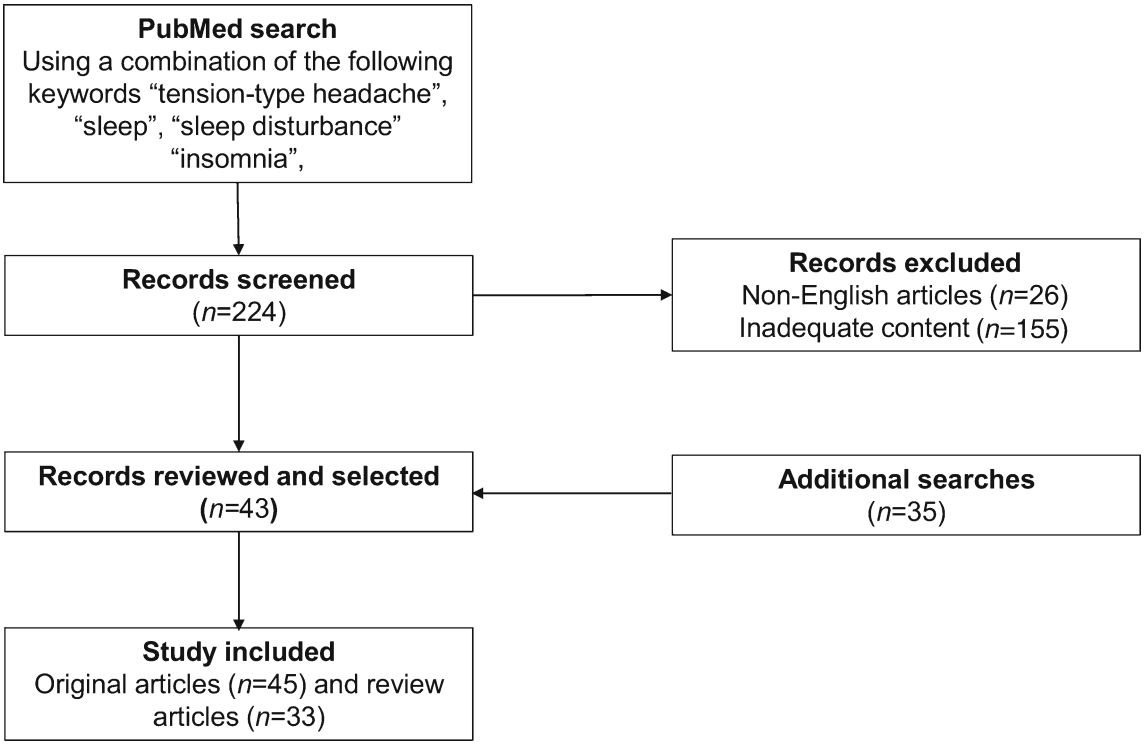

Fig. 1 Process of study selection

was higher among OSA patients than among non-OSA patients (10.2% vs. 7.7%, $p < 0.001$). This longitudinal study also indicated that patients with OSA were more likely to have TTH (hazard ratio [HR] = 1.18, 95% CI = 1.06–1.31, $p = 0.003$) than were patients in the non-OSA group [11]. Studies on the association between OSA and TTH were summarized on Table 2.

Considering habitual snoring, patients with CTTH had more frequent habitual snoring (OR = 4.9, $p < 0.005$). Furthermore, there was a significant association between chronic headache and snoring regardless of headache type, sex, age, and obesity [32].

Restless Leg Syndrome

Restless leg syndrome (RLS) is a sleep-related sensorimotor disorder characterized by unpleasant feelings in the legs, especially during bed time or rest [33, 34]. The prevalence of RLS was significantly higher in individuals with TTH than in non-headache individuals (8.0% vs. 3.6%, $p = 0.018$) in a general population-based study. Individuals with concurrent TTH and RLS had higher visual analogue scale scores for headache intensity (5.1 ± 2.0 vs. 4.3 ± 1.8, $p = 0.038$) than did TTH individuals without RLS [35]. A longitudinal study revealed that the TTH group exhibited an increased risk of developing RLS (HR = 1.57, 95% CI = 1.22–2.02) compared with the non-TTH group [36].

Temporomandibular Disorder and Bruxism

Temporomandibular disorder (TMD) and bruxism are disorders frequently associated with TTH and migraine [12, 37]. TMD itself is not a sleep disorder, but it is commonly associated with sleep disturbances including bruxism, sleep apnea, and poor sleep quality [38–40]. In TTH, clinical features for ETTH are similar to the characteristics of TMD related to bruxism. ETTH-associated pain may be similar to post-exercise muscle soreness from TMD related to bruxism [41]. Patients with TTH reported increased severity and chronicity of pain which accompanied TMD when compared with healthy controls [42]. Although the association between bruxism and ETTH was not significant, TTH was significantly related with bruxism (OR = 3.13, 95% CI = 1.25–7.70) [43]. When painful TMD and bruxism were combined, the risk of ETTH (OR = 3.8, 95% CI = 1.38–10.69, $p = 0.017$) was significantly elevated [44].

Shift Working

Shift working may increase the risk of sleep problems, metabolic disease, cardiovascular disease, and malignancy [45]. In a cross-sectional study in Norway, an increased frequency of headache was associated with shift work disorder (OR = 2.04, 95% CI = 1.62–2.59), but not with work schedule, number of night shifts, or number of quick

Table 1 Studies on the association between insomnia and TTH

Title	Authors, country, and year	Setting and participants	Main findings
Insomnia in tension-type headache: a population-based study [23•].	Kim J, Cho SJ, Kim WJ, Yang KI, Yun CH, Chu MK, Korea, 2016	Korean nation-wide survey on sleep and headache. 2695 participants were enrolled.	TTH and insomnia prevalence was 21.2% and 10.8%, respectively. The prevalence of insomnia among individuals with TTH was significantly higher than that among individuals without headache (13.2% vs. 5.8%, $p < 0.001$). Among individuals with TTH, headache frequency, visual analogue scale scores for headache intensity and depression prevalence (21.3% vs. 1.6%, $p < 0.001$) were significantly higher in those with insomnia than in those without insomnia.
Relationship between insomnia and headache in community-based middle-aged Hong Kong Chinese women [24].	Yeng WF, Chung KF, Wong CY, Hong Kong, 2010	All inhabitants aged ≥ 20 years in Nord-Trøndelag County of Norway.	The prevalence of TTH was 15.5%. Logistic regression analysis showed that women with insomnia disorder had 2.3-fold increased risk of TTH.
The impact of headache and chronic musculoskeletal complaints on the risk of insomnia: longitudinal data from the Nord-Trøndelag health study [25].	Ødegard SS, Sand T, Engstrøm M, Zwart JS, Hagen K, Norway, 2013	All inhabitants aged ≥ 20 years in Nord-Trøndelag County of Norway were invited to participate in two surveys ($n = 92,566$ and 93,860, respectively). 27,185 subjects participated in both surveys	Compared to subjects without headache, there was an increased risk of insomnia among those with migraine (OR = 1.9, 95% CI = 1.7–2.1) and non-migraineous headache (OR = 1.7, 95% CI = 1.5–1.8)
The Long-term effect of insomnia on primary headaches: a prospective population-based cohort study (HUNT-2 and HUNT-3) [26].	Ødegard SS, Sand T, Engstrøm M, Stovner, LJ, Zwart JS, Hagen K, Norway, 2011	Longitudinal cohort study included subjects who participated in 2 consecutive surveys of the Nord-Trøndelag Health Study (HUNT-2 and HUNT-3). Aged 20 years or more in HUNT-2 (n = 92,566) and HUNT-3 ($n = 94,194$).	The presence of baseline insomnia was associated with a 40% increased risk for TTH in HUNT-3 (RR = 1.5, 95% CI = 1.1–2.1).

CI confidence interval, *OR* odds ratio, *RR* relative risk, *TTH* tension-type headache

returns. Regarding TTH, headache was only associated with > 20 night shifts last year (OR = 1.41, 95% CI = 1.07–1.86) [46•].

Excessive Day Time Sleepiness

A polysomnographic study showed that TTH patients had a tendency for higher sleepiness (Epworth Sleepiness Scale [ESS], 7.5 ± 3.9 vs. 5.9 ± 3.2) when compared with controls. The difference was significant for the CTTH subgroup compared with controls ($p = 0.039$). The study found that TTH patients had more slow-wave sleep ($p = 0.002$) and less fast arousals ($p = 0.004$) in their polysomnography [47]. Another polysomnographic study reported that patients with CTTH had more excessive day time sleepiness (EDS, ESS ≥ 10) compared with those with chronic migraine (CM) (26.0% vs 3.0%. $p = 0.03$) [48•]. In contrast, a population-based study in Norway reported that the prevalence of EDS in CTTH was similar to that in CM (21.1% vs. 20.0%, $p = 0.782$) [49]. A population-based study in Korea demonstrated that the prevalence of EDS was elevated in CTTH but not ETTH. EDS was associated with increased headache frequency and intensity in subjects with TTH [50].

Psychiatric Comorbidities Associated with Sleep Disturbances and TTH

Headache and sleep disturbances are associated with psychiatric comorbidities. The association of TTH with sleep disturbances and mood disorders has been reported in cross-sectional and longitudinal studies [51]. The prevalence of anxiety and depression increased with an increase in TTH frequency [52–54]. The frequency of psychiatric comorbidity among CTTH patients ranged from 7 to 59%, which was similar to that among migraine patients in clinic-based studies [55]. Therefore, evaluation of anxiety and depression is important in the management of TTH patients with sleep disturbances, especially for CTTH.

Table 2 Studies on the association between OSA and TTH

Title	Authors, country, and year	Setting and participants	Main findings
Tension-type headache associated with obstructive sleep apnea: a nationwide population-based study [11].	Chiu YC, Hu HY, Lee FP, Huang HM, China, 2015	Taiwan Longitudinal Health Insurance Database, 4759 participants with OSA and 19,036 non-OSA patients	The prevalence of TTH was 10.2% among OSA patients and 7.7% among non-OSA patients ($p < 0.001$). The multivariate Cox proportional hazards model revealed patients with OSA were more likely to have TTH (hazard ratio, 1.18; 95% CI, 1.06–1.31) ($p = 0.003$) than patients in the non-OSA group.
Tension-type headache and sleep apnea in the general population [28].	Kristiansen HA, Kvaerner KJ, Akre H, Overland B, Russell MB, Norway, 2011	A random age and gender-stratified sample of 431 from 14,860 persons aged 20–80 years residing in Akershus, Norway. Polysomnography was conducted.	Logistic regression analyses showed no significant relationship between TTH and OSA, with adjusted odds ratios for frequent TTH of 0.95 (0.55–1.62) and CTTH of 1.91 (0.37–9.85). The results did not change when using cutoff of moderate and severe OSA.
Improvement in headaches with continuous positive airway pressure for obstructive sleep apnea: a retrospective analysis [31].	Johnson KG, Ziemba AM, Garb JL. USA, 2013	A retrospective chart review of all patients referred to adult neurology clinic for headaches and sent for polysomnography between January 2008 and December 2009. Follow-up ranged from 18 to 42 months.	Eighty-two headache patients (70 females, 12 males) were studied. Headache types included 17% chronic migraine without aura, 22% episodic migraine without aura, 32% migraine with aura, 21% TTH, and 6% chronic post-traumatic headache. Of the 33 patients who used CPAP, 13 reported improvement in headaches specifically due to CPAP therapy and 10 additional patients noted benefit in sleep symptoms.

CTTH chronic tension-type headache, *CPAP* continuous positive airway pressure, *OSA* obstructive sleep apnoea, *TTH* tension-type headache,

Possible Mechanisms Underlying the Association Between Sleep Disturbances and TTH

The constellation of sleep disturbances and headache disorders is likely underpinned by shared neuroanatomical structures and neurotransmitters. The brainstem and hypothalamus play important roles in both sleep and headache. The suprachiasmatic nucleus, which is the main center for circadian rhythmicity of the human body, has abundant connections to the pineal gland (circadian rhythm and pain modulation by melatonin), brainstem nuclei for sleep stage and motor control (locus coeruleus by norepinephrine), mood control (raphe nuclei by serotonin), and pain modulation (periaqueductal gray by enkephalin, beta endorphin, and dysnorphin) [56–59]. Peripheral (myofascial nociception) and central mechanisms (central sensitization) are believed to act in pathophysiologic mechanisms of TTH. Peripheral mechanisms are more predominant in ETTH whereas central mechanisms are more predominant in CTTH [60, 61]. Central sensitization is related to decreased pain threshold [62]. A polysomnographic study reported that TTH patients had greater arousal and tended to have lower pain thresholds than did healthy controls. The study also reported that cold pain threshold was inversely correlated with light sleep in TTH [47]. Higher prevalence of sleep disturbances or sleep disorders among individuals with CTTH than those with ETTH suggest that central mechanisms play important roles in the relationship between sleep and TTH.

Clinical Implications

TTH with Insomnia

TTH patients often have insomnia. Insomnia is primarily diagnosed by clinical evaluation through sleep, medical, substance, and psychiatric history. Self-administered questionnaires, at-home sleep logs, symptom check lists, psychological screening tests, and bed partner interviews are helpful in the evaluation and differential diagnosis of insomnia. Polysomnography and daytime multiple sleep latency tests are not indicated in the routine evaluation of chronic insomnia. If there is clinical suspicion of sleep disordered breathing, movement disorder, uncertain diagnosis, treatment failure, or precipitous arousals occurring with violent or injurious behavior, polysomnography is indicated. Actigraphy is indicated for circadian rhythm disorders [63].

Insomnia is often accompanied by psychiatric problems such as anxiety and depression, especially in CTTH [22, 64]. If a TTH patient has insomnia, psychiatric evaluation is an essential part of assessment. Interview, questionnaires for

psychiatric comorbidity such as anxiety and depression, or referral to psychiatry may be used for psychiatric evaluation.

If a TTH patient reports insomnia, sleep-prone prophylactic treatment medication may improve both TTH and insomnia. Amitriptyline is recommended as grade A evidence for the prophylactic treatment of TTH. TTH participants with insomnia may improve symptoms of TTH and insomnia by amitriptyline. Other sedative antidepressants including mirtazapine, venlafaxine, and clomiphene can be considered for the prophylactic treatment of TTH with insomnia [65]. Although SSRIs are less efficacious than tricyclic antidepressants, SSRIs may be considered for the treatment of accompanying depression [66]. Caffeine-containing combination analgesics are effective in acute treatment of TTH. Nevertheless, they may exacerbate insomnia symptoms and are more likely to induce medication overuse headaches than simple analgesics or NSAIDs [65]. Therefore, it may be advisable to use caffeine-containing combination analgesics after failure of simple analgesics or NSAIDs.

Cognitive behavioral therapy for insomnia (CBT-I) is recommended for primary and comorbid chronic insomnia [67]. A randomized trial for CBT-I showed efficacy in both improvement of headaches and insomnia symptoms among CM patients combined with chronic insomnia [68]. Although the clinical efficacy of CBT-I among individuals with TTH is currently lacking, CBT-I may be considered for an individual with TTH combined with insomnia.

TTH with EDS

CTTH patients have an increased chance of having EDS than do individuals without headaches [49]. EDS can be precipitated by various causes. Short sleep duration, poor sleep quality, sleep-disordered breathing, parasomnia, bad sleep habits, medications, narcolepsy, RLS, medical disorders, psychiatric disorders, shift working, and insomnia can cause EDS [69, 70]. EDS can be measured by clinical history and instruments. ESS is the most widely used instrument to assess EDS [71]. Short sleep duration, bad sleep habit, medications, medical disorders, psychiatric disorders, RLS, shift working, circadian rhythm disorder, and insomnia can be evaluated by clinical history, interviews, and sleep logs. Sleep-disordered breathing and narcolepsy can be objectively assessed by polysomnography. If the cause of EDS is identified, the cause for EDS should be properly treated. Short sleep duration and bad sleep habits can be improved by education of sleep hygiene. Circadian rhythm disorder, RLS, and insomnia can be treated by pharmacological and/or non-pharmacological treatments such as light therapy and melatonin for circadian rhythm disorder, CBT-I and hypnotics for insomnia, and pregabalin and dopamine agonists for RLS [67, 72–74].

TTH with OSA and Habitual Snoring

Although the relationship between OSA and TTH is controversial, OSA in individuals with TTH should be properly diagnosed and treated. A recent American Academy of Sleep Medicine guideline strongly recommends use of polysomnography or home sleep apnea testing rather than other clinical tools and prediction algorithms in the diagnosis of OSA [75]. After the diagnosis of OSA is confirmed, proper treatment of OSA should be started. If the patient has moderate-to-severe OSA (apnea hypopnea index > 15), continuous positive airway pressure (CPAP) is indicated. If the patient is intolerant to CPAP, bilevel positive airway pressure, pressure relief, or autotitrating airway pressure can be considered. In mild OSA ($5 \leq$ apnea hypopnea index < 15) cases with symptoms, behavioral treatment, oral appliances, and surgical treatment in addition to CPAP could be considered [76].

TTH with Other Sleep Disturbances

While there is no evidence of improvement in TTH by treatment of RLS, TMD, and bruxism at present, these sleep disturbances should be properly treated. RLS could be successfully treated by dopamine agonists, gabapentinoid drugs, iron, and physical measures [77]. TMD can be improved by education, behavioral modification, muscle relaxants, antidepressants, benzodiazepine, and occlusal splints [78]. Bruxism can be managed by improvement of sleep hygiene, splint therapy, and pharmacological treatment including amitriptyline, botulinum toxin, and dopamine agonists [79].

Conclusions

TTH is the most common primary headache type which shows a significant association with sleep in its onset, change in prevalence, and clinical presentations. In addition, TTH-like headaches can occur in association with sleep, sleep-apnea headaches, and hypnic headaches. Recent population-based studies have revealed bidirectional comorbidity between sleep-related factors and TTH, suggesting shared pathophysiologic mechanisms.

TTH is significantly associated with sleep apnea, insomnia, insufficient sleep, poor sleep quality, RLS, EDS, and bruxism in occurrence and symptom exacerbation, especially in CTTH. Therefore, more attention should be paid to the evaluation and treatment of these sleep disturbances during the treatment of TTH. Proper diagnosis and treatment of sleep disturbances and regulation of sleep/wake cycles may improve the management of TTH.

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Compliance with Ethical Standards

Conflict of Interest Soo-Jin Cho was involved as a site investigator of multicenter trial sponsored by the Otsuka Korea, Eli Lilly and company, and Novartis, worked as an advisory member for Teva, and received research support from the Hallym University Research Fund 2016 and a grant from the Korean Neurological Association (KNA-16-MI-09). Dr. Cho also received personal fees from Allergan Korea and Yuyu Pharmaceutical Company.

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

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