The Journal of Headache and Pain

RESEARCH

Open Access

Pain beyond the head: clinical implications of body pain in migraine and tension-type headache in a population-based study

Woo-Seok Ha[1], Soomi Cho[1], Kyung Min Kim[1] and Min Kyung Chu[1*]

Abstract

Background Body pain significantly affects the quality of life. However, the relationship between headache and pain across broad body regions remains unclear. This population-based cross-sectional study aimed to investigate the prevalence and impact of body pain in migraine and tension-type headache (TTH).

Methods We analyzed baseline data from 2,548 participants in the Korean population-based Circannual Change in Headache and Sleep study. Participants were classified into migraine ($n=145$), TTH ($n=805$), and no-headache ($n=920$) groups. Body pain was assessed across 19 regions using the Widespread Pain Index (WPI). Multivariable analyses adjusted for age, sex, and psychiatric symptoms were performed.

Results Body pain was more widespread in the migraine and TTH than in the no-headache group, showing a gradient of migraine > TTH > no-headache ($P<0.001$). Migraine was most strongly associated with neck pain (odds ratio [OR] 2.84, $P=0.008$), whereas TTH showed the strongest association with upper back pain (OR 2.74, $P=0.008$). Higher WPI and body-pain intensity were associated with higher headache intensity, more monthly headache days, more monthly severe headache days, and higher HIT-6 scores, as well as poorer quality of life and greater depression, anxiety, and insomnia (all $P<0.001$). Axial body pain was associated with higher monthly headache days in migraine, whereas upper body pain was associated with higher monthly headache/severe headache days in TTH.

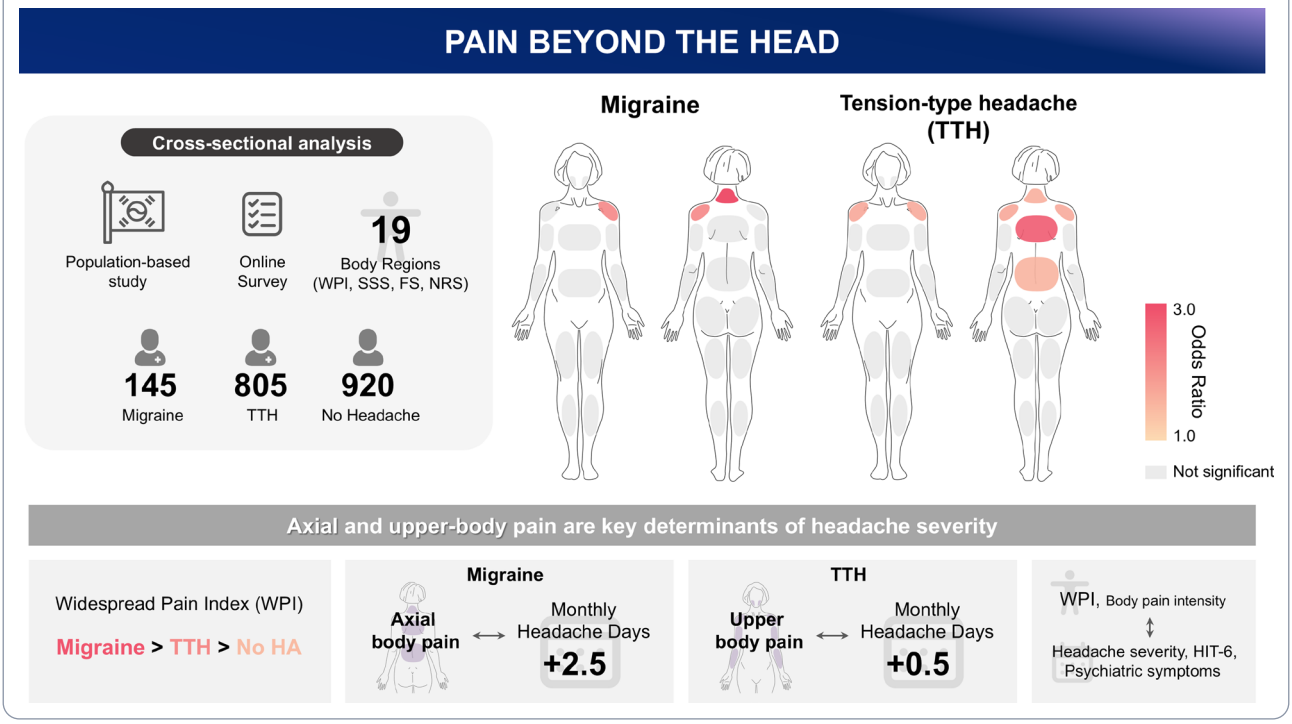
Conclusions Body pain is more prevalent and widespread in patients with migraine and TTH and is associated with a greater disease burden, potentially reflecting central sensitization. Distinct patterns of regional pain, particularly neck pain in migraine and upper back pain in TTH, may provide clinically relevant insights into underlying nociceptive mechanisms.

Keywords Migraine, Tension-type headache, Widespread pain, Body pain distribution, Headache impact

*Correspondence:
Min Kyung Chu
chumk@yonsei.ac.kr

Full list of author information is available at the end of the article

Graphical Abstract



Background

Migraine is a highly prevalent neurological disorder that imposes substantial personal and societal burdens through recurrent attacks and disability, significantly affecting the quality of life of patients. In the Global Burden of Disease framework, migraine consistently ranks among the leading causes of years lived with disability worldwide, significantly impacting working-age populations [1]. Tension-type headache (TTH) is the most common primary headache disorder and is often considered less disabling at the population level. However, some individuals experience frequent or severe attacks with considerable functional impairment [2]. Although migraine and TTH share important pathophysiological mechanisms, the relative contribution of peripheral nociceptive inputs may differ [3, 4]. In TTH, pericranial and myofascial inputs, often accompanied by pericranial tenderness, trigger points, and neck and shoulder girdle involvement, may contribute to headache initiation and maintenance via convergence in the upper cervical dorsal horn (C1 to C4) and the trigeminocervical complex [3, 5]. In contrast, migraine is typically linked to trigeminovascular nociception arising from the meningeal and vascular structures. C2 dorsal root ganglion afferents that traverse the suboccipital muscles and innervate the posterior dura provide an anatomical basis for upper cervical region involvement, especially at C1-C2, and for frequent neck pain or occipital hypersensitivity in migraine [6]. However, mechanisms beyond regional convergence within the trigeminocervical complex are likely required to explain pain extending beyond the head and neck. Both migraine and TTH involve altered central pain processing, including central sensitization and impaired descending inhibitory control [3, 7], which can facilitate the spread of pain beyond the cephalic region and contribute to widespread body pain [8].

An association between body pain and headache disorders has been consistently reported. Based on a US population-based chronic migraine epidemiology and outcome study, non-cephalic pain was reported more frequently as headache frequency increased, and this pattern was more pronounced in chronic migraine than in episodic migraine. Pain was most commonly reported in the neck and shoulder regions, followed by the back, whereas chest and abdominal pains were reported less frequently [9]. Lower back pain is also significantly associated with primary headache disorders. In a large population-based sample from the German Headache Consortium, both chronic migraine and chronic TTH were significantly associated with concomitant low-back pain, and the association was stronger with frequent low-back pain than with infrequent symptoms [10]. However, beyond neck and lower back pains, information remains limited regarding the relationship between headache disorders and pain across a broader range of body regions,

and the clinical significance of region-specific pain patterns has not been well established.

Therefore, we hypothesized that (1) body pain would be more prevalent and widespread in individuals with migraine and TTH than in those without headache, and (2) the anatomical distribution of body pain would differ between migraine and TTH. The aim of this study was to determine whether body pain is more prevalent and widespread in participants with migraine and TTH than in those without headache and to examine differences in the anatomical distribution of body pain between migraine and TTH.

Methods

Study design and participants

This study was a cross-sectional analysis using baseline data and 3-month follow-up data from the Circannual Change in Headache and Sleep (CHASE) Study, a nationwide, population-based, web-based cohort study conducted in South Korea. The CHASE study was launched in October 2020 and included longitudinal follow-ups every three months for one year. Detailed information on the CHASE study design has been previously reported [11–13]. Except for the assessment of pain chronicity over 3 months required for the diagnosis of fibromyalgia, which was based on the 3-month follow-up data, all other variables were derived from the baseline data.

The target population consisted of Korean adults aged 20–59 years, who were sampled using a two-stage stratified cluster random sampling method based on the 2015 Korean population and housing censuses. The sampling strategy was designed to be representative of the general Korean population (excluding Jeju Island), with a final target sample size of 3,000 and a sampling error margin of ± 1.8%. A total of 91,153 email invitations were distributed, and 3,030 participants were enrolled at baseline. Participants who suspended the survey by providing informed consent and initiating the questionnaire, but not completing all the required items or reaching the final submission page, were excluded to avoid potential bias due to missing data. At baseline, participants completed questionnaires assessing body pain across 19 anatomical regions, headache characteristics, psychiatric symptoms, and quality of life.

The study protocol was approved by the Institutional Review Board of the Yonsei University College of Medicine (2020-0034-001), and all participants provided informed consent. The study was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology guideline [14].

Headache classification

Headache types were classified using a validated web-based questionnaire based on the International Classification of Headache Disorders, 3rd edition (ICHD-3) [15]. Participants were categorized into three groups: migraine, TTH, and no headache (No HA). Migraine included both migraine without aura (code 1.1) and migraine with aura (code 1.2). TTH was diagnosed according to ICHD-3 criteria, including infrequent episodic TTH (code 2.1), frequent episodic TTH (code 2.2), and chronic TTH (code 2.3). Those who met criteria for probable migraine or probable TTH were excluded from the analysis to ensure diagnostic specificity. Participants who reported no headaches in the previous year were classified as not having headache. Participants were classified according to the characteristics of their most severe headache experienced during the previous year. Considering that migraine compared with TTH is generally associated with more severe symptoms and most individuals with migraine, in a previous study, had non-migraine headache including TTH, participants who were classified as having migraine were likely to have both migraine and TTH [3]. This classification approach is consistent with that used in previous population-based epidemiological studies [16, 17]. The questionnaire-based headache diagnostic algorithm used in this study has been previously validated, showing a sensitivity of 92.6% and a specificity of 94.8% for migraine, and a sensitivity of 78.4% and a specificity of 98.4% for TTH [18]. We excluded participants with headaches not attributable to migraine or TTH because migraine and TTH are major headache disorders in the general population, and the non-migraine/non-TTH headache group is heterogeneous [19]. Therefore, this study focused on comparisons among participants in the migraine, TTH, and No HA groups.

For subgroup analyses, chronic headache was operationally defined as experiencing headaches on ≥ 45 days during the past 90 days and on ≥ 15 days during the past 30 days. Participants with TTH who met this criterion were classified as having chronic TTH, while those with migraine meeting the same threshold were classified as having chronic migraine. In addition, migraine was further subclassified into migraine with aura and migraine without aura based on the self-reporting Visual Aura Rating Scale (VARS). This questionnaire has a sensitivity of 96.4% and a specificity of 79.5% for detecting visual aura in patients with migraine as a self-administered VARS score ≥ 3 [20].

Assessment of body pain

Body pain was assessed using a binary (yes/no) checklist of 19 specific anatomical regions corresponding exactly to the 19 regions used to calculate the Widespread Pain Index (WPI) as defined in the 2016 American College of Rheumatology (ACR) criteria for fibromyalgia [21]. At baseline, participants were asked to indicate whether

they had experienced pain in each region during the past 7 days. These regions included neck, chest, abdomen, upper back, lower back, bilateral jaw, shoulder girdle, upper arm, lower arm, hip (buttock), upper leg and lower leg. The WPI was calculated as the total number of regions with pain, ranging from 0 to 19. In addition, to assess fibromyalgia status, the Symptom Severity Scale (SSS) was administered, and the Fibromyalgia Severity (FS) score was derived as the sum of the WPI and SSS. For participants who reported pain in at least one region, the overall subjective body pain intensity was measured using an 11-point numerical rating scale (NRS). Fibromyalgia was diagnosed according to the 2016 ACR criteria, based on the combination of the WPI and SSS scores (WPI ≥ 7 and SSS ≥ 5, or WPI 4–6 and SSS ≥ 9), along with the presence of generalized pain (pain in at least 4 of 5 regions) [21]. To account for the chronicity requirement (symptoms persisting for at least 3 months), fibromyalgia was defined based on participants who met these criteria at both baseline and the 3-month follow-up assessment, while all other analyses were based on baseline data.

Assessment of headache symptoms and impact, psychiatric symptoms, and quality of life

Headache-related impacts were assessed using the Headache Impact Test-6 (HIT-6) [22]. In addition, participants reported headache intensity using the 11-point NRS (0–10), the number of monthly headache days, and the number of monthly severe headache days. Severe headache days were defined as days with headache that interfered with normal daily activities, including work, school, or household tasks. Psychiatric symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9) for depressive symptoms, the Generalized Anxiety Disorder-7 (GAD-7) for anxiety symptoms, and the Insomnia Severity Index (ISI) for sleep disturbance [23–25]. For each scale, higher scores indicated greater symptom severity, and validated cut-off scores were used to define clinical levels of depression (PHQ-9 ≥ 10), anxiety (GAD-7 ≥ 10), and insomnia (ISI ≥ 15). Health-related quality of life was assessed using the EuroQoL-5 Dimensions (EQ-5D) questionnaire, and scores were transformed into a single index using Korean population-based weights [26–28]. To facilitate parametric analyses, a log-transformed EQ-5D index [log(1 – EQ-5D)] was used as a continuous outcome variable. The Korean versions of the HIT-6, PHQ-9, ISI, and GAD-7 have been previously validated in Korean [29–32].

Statistical analysis

Categorical variables are presented as frequencies and percentages and were compared using the chi-squared test. Normality was assessed using the Kolmogorov–Smirnov test. If normality was confirmed, continuous variables were summarized as means with standard deviations and compared using the Student's *t*-test or analysis of variance. If normality was not confirmed, continuous variables were presented as medians and interquartile ranges (IQRs) and compared using the Mann–Whitney U test or the Kruskal–Wallis test for multiple group comparisons. For post-hoc pairwise comparisons between the headache groups, Bonferroni correction was applied to adjust for multiple comparisons. Population-based sampling weights were not applied, as the recruited sample showed demographic distributions comparable to the general Korean adult population in key characteristics.

Logistic regression analyses were performed separately for each of the 19 body regions to assess differences in regional pain by headache type, using the No HA group as the reference. Given that the analyses were conducted across two headache types (migraine and TTH), 38 region-wise comparisons were performed. All models were adjusted for age and sex. To account for multiple comparisons across regional analyses, false discovery rate (FDR) correction using the Benjamini–Hochberg method was applied. Adjusted *p*-values are presented alongside raw p-values to facilitate interpretation.

To examine the effects of overall body pain burden on quality of life and psychiatric symptoms, multivariable linear and logistic regression analyses were conducted. the WPI and body pain intensity (NRS) were each categorized into three groups based on distributional tertiles: WPI was classified as 0, 1–3, and ≥ 4, and body pain intensity as ≤ 3, 4–6, and ≥ 7. For quality of life, WPI and body pain intensity (NRS) were entered as predictors of the log-transformed EQ-5D index [log(1 – EQ-5D)]. For dichotomous psychiatric outcomes, including depression (PHQ-9 ≥ 10), anxiety (GAD-7 ≥ 10), and insomnia (ISI ≥ 15), both WPI and NRS were categorized into three groups based on distributional tertiles (WPI: 0, 1–3, ≥ 4, and body pain NRS: ≤ 3, 4–6, ≥ 7). All models were adjusted for age, sex, and headache type.

To further explore how body-wide pain influences the headache burden, multivariable linear regression analyses were conducted among participants with migraine and TTH. The dependent variables included headache intensity (NRS), monthly headache days, monthly severe headache days, and the HIT-6 score. Independent variables included age, sex, headache type, and either WPI or overall body pain intensity. To account for potential confounding by psychiatric symptoms, GAD-7 and PHQ-9 scores were additionally included in the models. An interaction term between migraine status and each measure (migraine × WPI and migraine × body pain intensity) were included to assess whether the association between body pain and headache burden differed according to headache type.

Finally, to evaluate the relative contribution of regional pain distribution on headache burden, multivariable linear regression analyses were performed with dichotomous indicators for axial, upper, and lower body pain (yes/no) as predictors of headache outcomes, adjusting for age, sex, headache type and GAD-7 and PHQ-9 scores. For this analysis, we categorized regions according to the ACR criteria: axial pain included the neck, chest, abdomen, upper back, and lower back; upper body pain included the bilateral shoulder, arm, and jaw; and lower body pain included the bilateral hip and leg. Each region was coded as "yes" if pain was reported in at least one of the corresponding regions. Individuals who reported no pain were assigned an NRS score of zero for headache or body pain.

Statistical significance was defined as a two-sided P value < 0.05. All analyses were performed using SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY). An a priori power analysis was not conducted because the sample size was determined by data availability. In addition, missing data were not recorded because the web-based survey platform was configured to prevent submission unless all the mandatory fields were completed.

Results

Study participants

Among the 91,153 individuals invited via email, 10,699 agreed to participate in the CHASE study. Of them, 3,030 completed the baseline assessment and were enrolled in the study. At the 3-month follow-up, 2,548 participants completed an additional 3-month survey module, which included assessments of body pain and headache (Fig. 1). These participants had distributions in sex, age, and educational level similar to those of the total Korean population (Supplementary Table 1). Among the 2,548 follow-up participants, 1,938 reported experiencing headaches in the past year. After excluding 678 individuals with non-migraine/non-TTH headaches, 145 participants with migraine and 805 participants with TTH were included in the final analysis, along with 920 participants without headache (No HA).

The sociodemographic characteristics of the study population are summarized in Table 1. Among the headache groups, women were more prevalent, particularly in the migraine group (73.8%) than in the No HA group (37.2%, $P < 0.001$). Participants with migraine also tended to be younger, with the highest proportion being in their 40s (36.6%), whereas the age distribution in the TTH group was more even ($P = 0.044$). The distributions of residential area ($P = 0.191$) and education level ($P = 0.783$) were comparable across study groups.

Body pain burden according to headache type

WPI score distribution varied substantially across study groups (Fig. 2). A clear gradient in WPI was observed from No HA to TTH and migraine groups. Compared to the No HA (median [IQR]: 2.0 [0.0–3.0]), participants with TTH (3.0 [1.0–5.0]) and migraine (4.0 [2.0–7.0]) reported progressively higher WPI (all pairwise comparisons, Bonferroni-adjusted $P < 0.001$ or $P = 0.001$). Similarly, SSS, FS score, and body pain intensity (NRS) also increased stepwise across the three groups, with the highest values consistently observed in the migraine

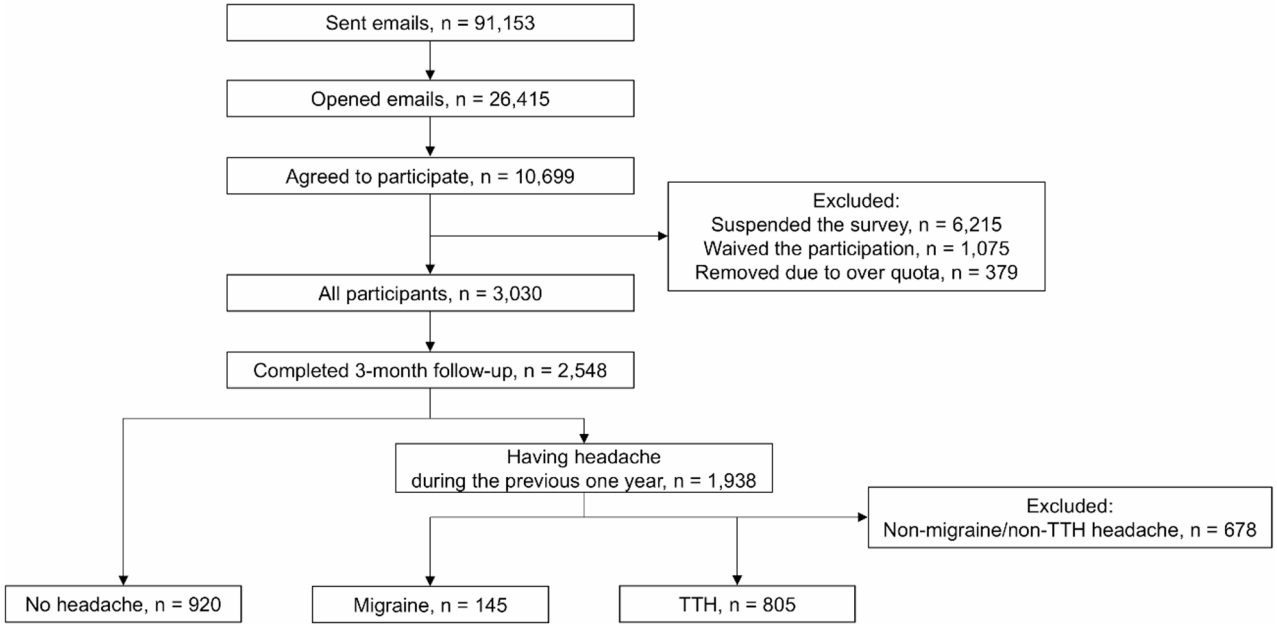


Fig. 1 Study flow diagram. TTH, Tension-type headache

Table 1 Demographic characteristics of the study participants

Variable	Migraine (*N* = 145)	TTH (*N* = 805)	No headache (*N* = 920)	*P*-value
Sex				
Men	38 (26.2)	409 (50.8)	578 (62.8)	< 0.001
Women	107 (73.8)	396 (49.2)	342 (37.2)	
Age				
20–29	30 (20.7)	165 (20.5)	207 (22.5)	0.044
30–39	33 (22.8)	177 (22.0)	188 (20.4)	
40–49	53 (36.6)	215 (26.7)	234 (25.4)	
50–59	29 (20.0)	248 (30.8)	291 (31.6)	
Residential area				
Large city	54 (37.2)	371 (46.1)	388 (42.2)	0.191
Medium/small city	74 (51.0)	362 (45.0)	431 (46.8)	
Rural area	17 (11.7)	72 (8.9)	101 (11.0)	
Education level				
≤ High school	55 (37.9)	330 (41.0)	375 (40.8)	0.783
≥ College	90 (62.1)	475 (59.0)	545 (59.2)	
WPI	4.0 (2.0–7.0)	3.0 (1.0–5.0)	2.0 (0.0–3.0)	< 0.001
SSS	7.0 (5.0–8.0)	5.0 (4.0–7.0)	3.5 (2.0–5.0)	< 0.001
FS	11.0 (8.0–14.0)	8.0 (6.0–11.0)	5.0 (3.0–8.0)	< 0.001
Overall body pain (NRS)	6.0 (5.0–8.0)	5.0 (3.0–6.0)	4.0 (2.0–6.0)	< 0.001

Values are presented as numbers (percentages) or medians (interquartile ranges). TTH, tension-type headache; NRS, numerical rating scale; WPI, Widespread Pain Index; SSS, Symptom Severity Scale; FS, Fibromyalgia Severity

group (all pairwise comparisons, Bonferroni-adjusted $P < 0.001$).

We further performed exploratory subgroup analyses according to headache chronicity and aura status (Supplementary Table 2). Among participants with TTH, those with chronic TTH showed higher SSS ($P = 0.004$), FS ($P = 0.005$), and body pain intensity ($P = 0.016$), compared to those with episodic TTH, with a similar trend observed for WPI ($P = 0.065$). In contrast, comparisons between episodic and chronic migraine did not show clear differences in pain-related measures (all P values > 0.05). Subgroup analyses according to aura status showed no consistent differences between migraine with and without aura, except for higher SSS scores in migraine with aura ($P = 0.024$). However, the number of participants with chronic headache, particularly chronic migraine ($n = 6$), was small, and these findings should be interpreted with caution.

In terms of anatomical distribution, pain in any body region was reported by 87.6% of the migraine group, 82.5% of the TTH group, and 66.5% of the No HA group (Supplementary Table 3, P < 0.001). The most commonly affected areas included the neck (migraine, 64.8%; TTH, 48.8%; No HA, 31.2% , $P < 0.001$) and lower back (migraine, 55.2%; TTH, 47.7%; No HA, 34.1%, $P < 0.001$).

Figure 3; Table 2 present the age- and sex-adjusted odds ratios (ORs) for the presence of each regional pain by headache type using the No HA group as a reference. After correction for multiple testing, participants with migraine were significantly more likely to have pain in the neck (OR: 2.84, 95% confidence interval [CI]: 1.91–4.23, adj. $P = 0.008$) and left shoulder girdle (OR: 2.16, 95% CI: 1.29–3.60, adj. $P = 0.016$). In TTH, significant associations were observed for neck pain (OR: 1.60, adj. $P = 0.008$), bilateral shoulder girdle (left OR: 1.68, adj. $P = 0.013$; right OR: 1.72, adj. $P = 0.008$), and axial regions, including the upper (OR: 2.74, adj. $P = 0.008$) and lower back (OR: 1.53, adj. $P = 0.008$). Further direct comparison between migraine and TTH (Supplementary Tables 4 and Supplementary Fig. 1) showed that only neck pain remained significantly more prevalent in the migraine group (OR: 1.73, 95% CI: 1.17–2.56, $P = 0.006$), while no other regional differences reached statistical significance.

Impact of WPI on comorbidities, quality of life and headache

In multivariable linear regressions, higher WPI showed linear association with poorer quality of life (EQ-5D,

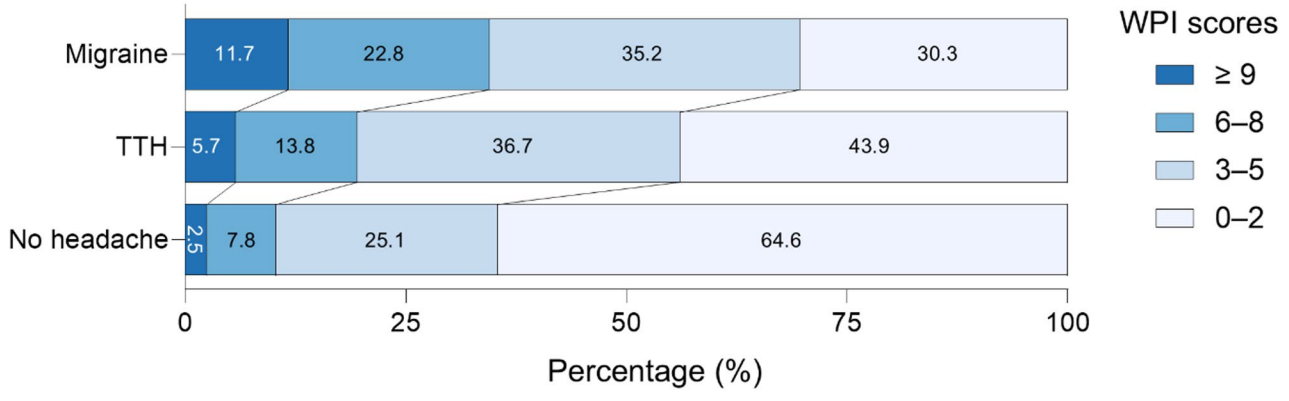


Fig. 2 Proportional distribution of widespread pain index scores across study groups. TTH: tension-type headache; WPI, widespread pain index

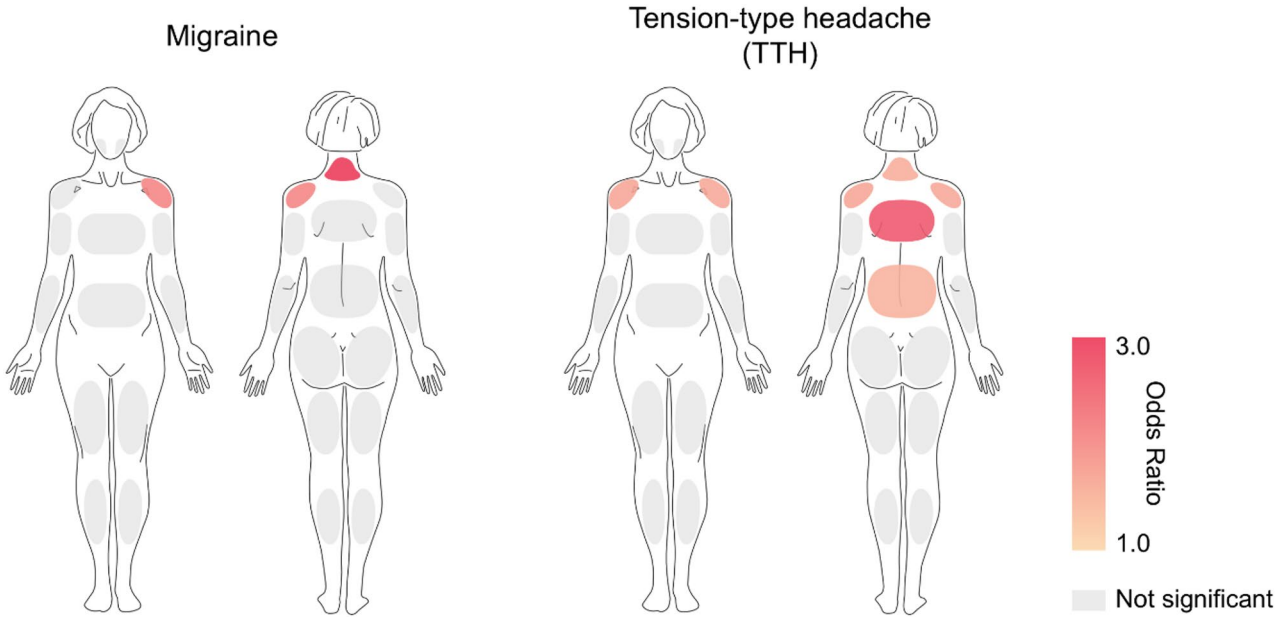


Fig. 3 Regional odds ratios of body pain in migraine and tension-type headache (versus no headache). Red-shaded regions indicate significantly elevated odds (adjusted $P < 0.05$) in each headache group compared to the no headache group

Table 2 Adjusted odds ratios for body pain by region, comparing each headache group to the no headache group

Body location	Migraine			Tension-type headache		
	OR (95% CI)	*P*-value	Adj. *P*-value	OR (95% CI)	*P*-value	Adj. *P*-value
Shoulder girdle, left	**2.16 (1.29–3.60)**	**0.003**	**0.016**	**1.68 (1.22–2.32)**	**0.002**	**0.013**
Shoulder girdle, right	1.82 (1.13–2.95)	0.015	0.071	**1.72 (1.28–2.30)**	**0.001**	**0.008**
Upper arm, left	2.01 (0.18–22.68)	0.574	0.797	3.37 (0.69–16.38)	0.132	0.314
Upper arm, right	5.44 (0.74–40.20)	0.097	0.288	5.96 (1.31–27.13)	0.021	0.089
Lower arm, left	2.35 (0.59–9.42)	0.227	0.507	0.70 (0.23–2.17)	0.537	0.797
Lower arm, right	3.11 (1.12–8.61)	0.029	0.110	1.33 (0.63–2.84)	0.454	0.797
Hip (buttock), left	1.93 (0.17–22.00)	0.595	0.797	1.43 (0.24–8.67)	0.695	0.846
Hip (buttock), right	1.00 (N/A)	0.996	0.996	0.70 (0.17–2.97)	0.629	0.797
Upper leg, left	1.00 (N/A)	0.996	0.996	0.60 (0.11–3.32)	0.557	0.797
Upper leg, right	1.00 (N/A)	0.996	0.996	1.09 (0.27–4.42)	0.907	0.985
Lower leg, left	1.61 (0.43–6.02)	0.481	0.797	1.41 (0.63–3.14)	0.406	0.771
Lower leg, right	1.71 (0.53–5.48)	0.370	0.758	1.41 (0.66–3.01)	0.379	0.758
Jaw, left	1.94 (0.19–19.72)	0.577	0.797	1.48 (0.33–6.69)	0.612	0.797
Jaw, right	1.52 (0.16–14.28)	0.712	0.846	1.43 (0.38–5.37)	0.598	0.797
Chest	1.16 (0.24–5.50)	0.855	0.956	1.93 (0.84–4.40)	0.120	0.304
Abdomen	2.08 (0.87–4.95)	0.099	0.288	1.08 (0.56–2.08)	0.820	0.944
Neck	**2.84 (1.91–4.23)**	**0.001**	**0.008**	**1.60 (1.24–2.06)**	**0.001**	**0.008**
Upper back	2.07 (0.90–4.79)	0.088	0.288	**2.74 (1.66–4.51)**	**0.001**	**0.008**
Lower back	1.42 (0.93–2.18)	0.106	0.288	**1.53 (1.20–1.94)**	**0.001**	**0.008**

All the models were adjusted for age and sex. Adjusted (Adj.) *P*-values were calculated using the Benjamini–Hochberg procedure to control the false discovery rate. N/A, Not available because of model convergence issues or zero counts; OR, odds ratio; CI, confidence interval

β: 0.253, $P < 0.001$). Compared with participants with WPI of 0, those with WPI scores of ≥ 4 showed higher odds of insomnia (ISI ≥ 15, OR: 3.939, $P < 0.001$), anxiety (GAD-7 ≥ 10, OR: 2.415, $P < 0.001$), and depression (PHQ-9 ≥ 10, OR: 2.397, $P < 0.001$) after adjusting for age, sex, and headache type (Table 3). Similar patterns were observed when using overall body pain intensity (NRS) as a predictor: body pain intensity showed linear association with poorer quality of life (EQ-5D, β: 0.353, $P < 0.001$), and those with NRS 4–6 or NRS ≥ 7 had progressively higher odds of insomnia, anxiety, and depression, all with $P < 0.001$.

Among participants with migraine and TTH, WPI was positively associated with headache intensity (β: 0.131,

Table 3 Association of widespread pain index and body pain with quality of life and psychiatric symptoms, adjusted for age, sex, and headache type

		EQ-5D (log)		ISI (≥ 15)		GAD-7 (≥ 10)		PHQ-9 (≥ 10)	
Variables		**Beta (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**
WPI (Ref. = 0)	1–3	0.253 (0.207–0.299)	< 0.001	2.045 (1.060–3.945)	0.033	1.540 (0.936–2.532)	0.089	1.515 (0.920–2.496)	0.103
	≥ 4			3.939 (2.067–7.506)	< 0.001	2.415 (1.466–3.977)	< 0.001	2.397 (1.453–3.954)	< 0.001
Body pain, NRS (Ref. ≤ 3)	4–6	0.353 (0.308–0.398)	< 0.001	3.383 (1.825–6.273)	< 0.001	6.520 (3.335–12.747)	< 0.001	5.610 (2.941–10.701)	< 0.001
	≥ 7			7.432 (3.892–14.193)	< 0.001	14.428 (7.194–28.939)	< 0.001	13.372 (6.840–26.141)	< 0.001

WPI, widespread pain index; NRS, numerical rating scale; Ref, reference group; OR, odds ratio; CI, confidence interval; EQ-5D, EuroQoL-5 Dimensions; ISI, Insomnia Severity Index; GAD-7, Generalized Anxiety Disorder-7; PHQ-9, Patient Health Questionnaire-9

Table 4 Association between the widespread pain index, body pain intensity and headache burden in participants with migraine and tension-type headache

Variables	**Headache intensity, NRS**		**Monthly headache days**		**Monthly severe headache days**		**HIT-6**	
	Beta (95% CI)	***P*-value**	**Beta (95% CI)**	***P*-value**	**Beta (95% CI)**	***P*-value**	**Beta (95% CI)**	***P*-value**
WPI	0.131 (0.071–0.190)	**< 0.001**	0.131 (0.063–0.199)	**< 0.001**	0.155 (0.086–0.223)	**< 0.001**	0.170 (0.114–0.226)	**< 0.001**
Migraine * WPI (interaction)	-0.093 (-0.186–0.000)	0.051	0.002 (-0.104–0.109)	0.965	-0.019 (-0.126–0.088)	0.724	-0.038 (-0.126–0.050)	0.399
Body pain (NRS)	0.186 (0.126–0.246)	**< 0.001**	0.147 (0.078–0.215)	**< 0.001**	0.181 (0.112–0.250)	**< 0.001**	0.32 (0.266–0.374)	**< 0.001**
Migraine * Body pain (interaction)	-0.111 (-0.295–0.073)	0.236	0.339 (0.129–0.549)	**0.002**	0.206 (-0.006–0.418)	0.057	-0.013 (-0.180–0.153)	0.875

Multivariable linear regression models included age, sex, headache type, GAD-7, PHQ-9, the WPI or body pain intensity (NRS), and the corresponding interaction terms with migraine. HIT-6, Headache Impact Test-6; CI, confidence interval; WPI, widespread pain index; NRS, numerical rating scale; GAD-7, Generalized Anxiety Disorder-7; PHQ-9, Patient Health Questionnaire-9

$P < 0.001$), monthly headache days (β: 0.131, $P < 0.001$), monthly severe headache days (β: 0.155, $P < 0.001$), and HIT-6 (β: 0.170, $P < 0.001$) after adjustment for age, sex, headache type, and psychiatric symptoms (Table 4). No significant interaction was observed between WPI and migraine status for any of the outcomes, suggesting that the relationship between body pain and headache burden was not specific to migraine. Similarly, overall body pain intensity (NRS) was positively associated with headache intensity (β: 0.186, $P < 0.001$), monthly headache days (β: 0.147, $P < 0.001$), monthly severe headache days (β: 0.181, $P < 0.001$), and HIT-6 (β: 0.320, $P < 0.001$).

Regional pain distribution and headache symptoms and impact

Among participants with migraine and TTH, multivariable linear regressions revealed that axial body pain was significantly associated with increased monthly headache days (β: 0.068, $P = 0.038$), and greater headache-related impact as measured by HIT-6 (β: 0.073, $P = 0.007$), after adjustment of age, sex, headache type, and psychiatric symptoms (Fig. 4). Upper body pain was also associated with increased monthly headache days (β: 0.071, $P = 0.033$), and monthly severe headache days (β: 0.081, $P = 0.016$). In contrast, lower body pain showed no significant association with headache outcomes.

Distinct patterns were observed when analyzed separately according to headache type (Table 5). In participants with migraine, those with axial body pain had, on average, 2.5 more monthly headache days than those without axial body pain (B = 2.449, $P = 0.010$). Among those with TTH, participants with upper body pain had 0.5 more monthly headache days (B = 0.498, $P = 0.027$), and 0.3 more monthly severe headache days (B = 0.302, $P = 0.040$) compared to those without upper body pain.

Prevalence of fibromyalgia and its association with migraine and TTH

Of the 1,870 participants, 35 (1.9%) met the diagnostic criteria for fibromyalgia, including eight (5.5%) with migraine, 24 (3.0%) with TTH, and three (0.3%) without headache. In participants with migraine, the clinical characteristics did not differ between those with and without fibromyalgia, with no significant differences in headache intensity, monthly headache days, monthly severe headache days, headache-related impact, depression (PHQ-9), anxiety (GAD-7), or insomnia (ISI) symptoms (Supplementary Table 5). In contrast, among participants with TTH, those with comorbid fibromyalgia had a greater disease burden, including higher headache intensity ($P = 0.008$), increased number of monthly severe headache days ($P = 0.005$), greater headache-related impact ($P = 0.006$)), and more severe depression ($P = 0.004$) and insomnia symptoms ($P < 0.001$) than those without fibromyalgia (Supplementary Table 6).

Discussion

The major findings of this study are as follows. (1) Body pain was more common and widespread in participants with migraine and TTH than in those without headaches,

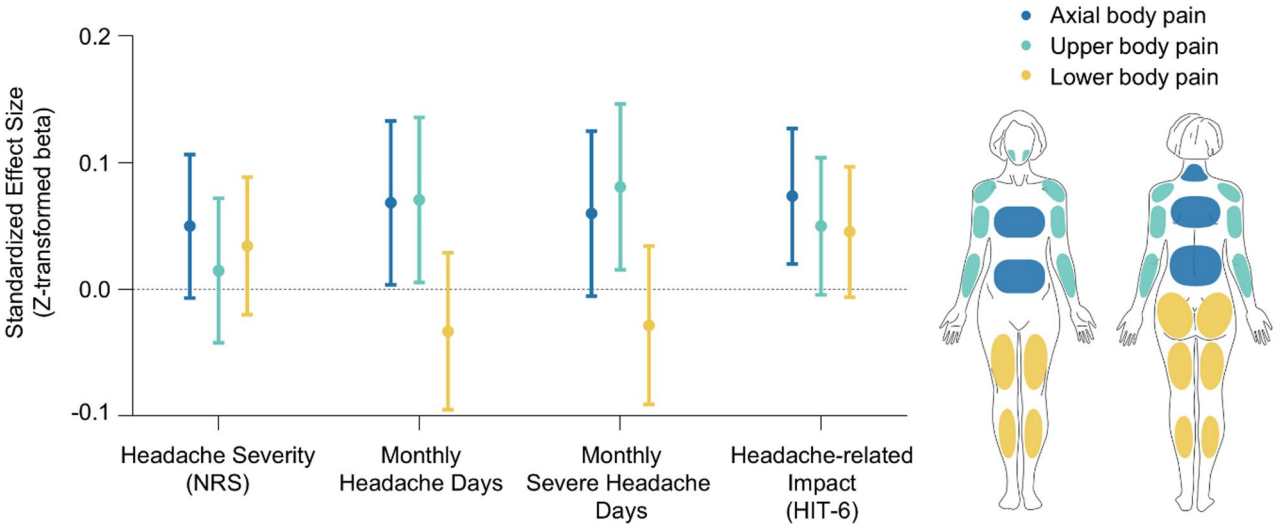


Fig. 4 Standardized effect sizes of axial, upper, and lower body pains on headache symptoms and the impact of headache among participants with migraine or tension-type headache. Effect sizes are derived from multivariable linear regression models adjusted for age, sex, headache type, GAD-7 and PHQ-9 scores

Table 5 Association of regional body pain with headache outcomes among participants with migraine and tension-type headache

		Headache intensity, NRS		Monthly headache days		Monthly severe headache days		HIT-6	
Headache	**Pain group**	**B (95% CI)**	***P*-value**	**B (95% CI)**	***P*-value**	**B (95% CI)**	***P*-value**	**B (95% CI)**	***P*-value**
Migraine	Axial body	-0.349 (-0.771–0.072)	0.104	2.449 (0.594–4.304)	**0.010**	0.914 (-0.385–2.213)	0.166	2.040 (-0.783–4.863)	0.155
	Upper body	0.282 (-0.124–0.688)	0.172	0.216 (-1.569–2.002)	0.811	0.765 (-0.484–2.015)	0.228	2.504 (-0.213–5.221)	0.071
	Lower body	0.333 (-0.033–0.699)	0.074	-0.493 (-2.103–1.117)	0.546	-0.682 (-1.809–0.445)	0.233	1.564 (-0.886–4.014)	0.209
Tension-type headache	Axial body	0.336 (0.009–0.663)	**0.044**	0.252 (-0.215–0.719)	0.290	0.202 (-0.103–0.507)	0.195	1.162 (0.153–2.171)	**0.024**
	Upper body	0.055 (-0.253–0.364)	0.725	0.498 (0.057–0.939)	**0.027**	0.302 (0.014–0.590)	**0.040**	0.690 (-0.262–1.642)	0.155
	Lower body	0.175 (-0.156–0.505)	0.300	-0.174 (-0.646–0.298)	0.469	-0.021 (-0.330–0.287)	0.892	0.790 (-0.229–1.809)	0.129

Values represent unstandardized regression coefficients (B) and 95% confidence intervals from multivariable linear regression models adjusted for age, sex, GAD-7 and PHQ-9. CI, confidence interval; NRS, numerical rating scale; HIT-6, Headache Impact Test-6; GAD-7, Generalized Anxiety Disorder-7; PHQ-9, Patient Health Questionnaire-9

showing a clear gradient of migraine > TTH > No HA. Neck pain showed the highest odds ratio in migraine. (2) Widespread body pain was associated with more severe headache symptoms, more psychiatric symptoms, and a poorer quality of life in both patients with migraine and TTH. (3) Axial and upper-body pains showed the strongest association with headache days and an impact on both migraine and TTH.

A greater extent and intensity of body pain were associated with higher headache intensity, more monthly headache days, and greater headache impact. Higher body pain was linked to more prominent depressive and anxiety symptoms and a lower quality of life, suggesting that non-cephalic pain meaningfully tracks the overall clinical burden of primary headache disorders. These findings are consistent with the concept of central sensitization, in which recurrent headache-related nociceptive input may increase excitability within central-pain processing pathways and weaken descending inhibitory control, thereby lowering pain thresholds and facilitating pain perception beyond the head [4, 33]. The close association between widespread pain and affective symptoms also supports a "bio-psychosocial framework," implying partially shared neurobiological circuits for pain and emotion and the potential for reciprocal amplification [34]. Finally, body pain may be a valuable clinical prognostic marker for identifying patients with a more burdensome and potentially sensitized headache phenotype who may benefit from earlier integrated management addressing pain and psychological comorbidities.

Neck pain was the most prevalent non-cephalic pain symptom among participants with migraine. An association between neck pain and headache disorders has been consistently reported. In a Danish population-based study, neck pain was reported by 76.2% of individuals with migraine, and its prevalence was higher in patients with chronic migraine (87%) than in those with episodic migraine (77%) [35]. Likewise, a meta-analysis of 24 studies found that 77.0% of patients with migraine reported neck pain compared to 23.2% of individuals without headache. Our findings are broadly concordant with those of previous studies. Neck pain was observed in 64.8% of participants with migraine and 34.1% of those without headache, thereby corroborating the established relationship [36]. Differences in the reported prevalence across studies may reflect variations in the study setting (population-based vs. hospital-based), ethnicity, and socioeconomic context.

Axial and upper body pains were significantly associated with monthly headache days, headache intensity, and the impact of headache, whereas lower body pain was not significantly associated with headache symptoms. Several mechanisms may explain these findings. First, the trigeminocervical complex (TCC) provides the preferential link between headache and upper-body pain. Trigeminal afferents and upper cervical nociceptive inputs converge on shared second-order neurons in the brainstem, enabling crosstalk and referred pain between the head and cervicothoracic regions [37]. Second, the anatomical and myofascial connectivity between cranial and cervical structure may further increase this association. Pericranial and neck-shoulder muscles commonly exhibit tenderness and myofascial trigger points in primary headache disorders, contributing to enhanced peripheral nociceptive input and symptom amplification [36, 38]. Third, central sensitization in headache disorders preferentially involve the trigeminal and upper cervical pathways, strengthening the coupling between head–neck and upper-body pain. In contrast, nociceptive inputs from the lower body ascend through more caudal spinal segments, which are less directly linked to these brainstem circuits [39, 40].

This study advances prior research on musculoskeletal pain in headache disorders by conducting a systematic, site-specific assessment of body pain that distinguishes neck pain from upper back pain, thereby moving beyond broad musculoskeletal pain constructs to delineate disorder-specific pain patterns. Although neck pain was the most prevalent site in both migraine and TTH, the biggest difference compared with the No HA group was observed for neck pain in migraine, whereas upper back pain showed the greatest contrast in TTH. Importantly, in the direct comparison between migraine and TTH, neck pain was the only body pain site that differed significantly, suggesting that neck pain may be a clinically relevant accompanying feature that could help distinguish these disorders in routine care, although it should not be considered a stand-alone diagnostic marker. From a pathophysiological standpoint, the prominence of neck pain in migraine is consistent with a closer coupling between migraine biology and TCC-based nociceptive processing and modulation, reflecting enhanced engagement of the trigemino-cervical interface [41, 42]. In contrast, the comparatively stronger association of upper back pain with TTH suggests that, beyond any shared TCC-related involvement, peripheral musculoskeletal mechanisms, including widespread shoulder girdle and upper thoracic muscle tension and myofascial trigger points, may contribute more substantially to the TTH phenotype [43]. Collectively, these site-specific patterns support the notion that migraine may be more strongly characterized by centrally driven pain amplification linked to TCC-mediated trigeminocervical integration, whereas TTH may be more influenced by broader musculoskeletal factors that extend into the upper back.

The prevalence of migraine in our study (5.7%) may appear lower than global estimates, which are often reported to be around 10–15% [44, 45]. However, this finding is consistent with previous population-based studies conducted in East Asian countries, where the 1-year prevalence of migraine has been reported to be lower, typically ranging from 5% to 10%. For example, previous studies reported a prevalence of approximately 6.0% in Japan [46], 9.1% in Taiwan [47], and 5–6% in Korea [48]. Notably, our estimate is comparable to that reported in a recent large-scale Korean study (5.2%) [48].

There are several limitations in the study. First, body pain was assessed using a web-based, self-reported questionnaire derived from the WPI in the 2016 ACR fibromyalgia diagnostic criteria [21] and was not corroborated by objective assessments, such as pressure algometry, quantitative sensory testing, and standardized tenderness measurements [49–51]. However, in large-scale epidemiological studies, including ours, device-based testing and individualized examinations are often impractical. Therefore, we used the WPI, which is widely used to quantify widespread pain in clinical and population-based studies. The WPI has demonstrated validity in diverse pain research settings [52]. Second, although our study used a large population-based sample, some subgroup analyses may have been underpowered due to small numbers within specific strata, limiting our ability to detect statistically significant associations. In particular, fibromyalgia was identified in only eight participants with migraine (5.5%) and 24 participants with TTH (3.0%). These small case counts likely reduced the statistical power to detect between-group differences in fibromyalgia prevalence between the migraine and TTH groups, as well as to

detect differences in headache characteristics according to fibromyalgia status within each headache group. Therefore, our null findings from these comparisons should be interpreted cautiously. Future studies with larger numbers of participants with comorbid fibromyalgia are warranted to estimate these associations more precisely. Third, this is a cross-sectional study. Therefore, a causal or temporal relationship between body pain and headache outcomes cannot be determined. Fourth, the use of acute or preventive medications was not considered. The regular use of analgesics or comorbidities related to medication overuse could potentially influence both headache and body pain sensitivity thresholds, acting as unmeasured confounders. Fifth, headache diagnoses were based on a structured questionnaire rather than face-to-face clinical interviews, which may introduce misclassification. In addition, body pain was assessed using the WPI, SSS and FS, which have been validated primarily in fibromyalgia and general pain populations, but not specifically in headache populations. Therefore, the applicability of these instruments to headache-related pain patterns may be limited. Sixth, the relatively low response rate may introduce selection bias. However, the sample showed similar demographic distributions to the general population, suggesting reasonable representativeness. Nevertheless, residual selection bias cannot be excluded. Seventh, we did not collect structured information on pain-related medical comorbidities (e.g., arthritis or inflammatory disorders) that could independently contribute to body pain. Although we accounted for psychiatric symptoms, residual confounding by unmeasured pain conditions may remain and should be considered when interpreting the observed associations between headache burden and non-cephalic pain. Eighth, headache frequency and severity were assessed using self-reported measures rather than a prospective headache diary, which may be subject to recall bias. Ninth, although we applied FDR correction using the Benjamini–Hochberg procedure for the multiple tests comparing demographic and clinical variables across chronotype groups, we may not have fully accounted for multiplicity across all analyses. Therefore, these findings carry an increased risk of false-positive results and should be interpreted as exploratory and hypothesis-generating, requiring confirmation in an independent sample. Tenth, we operationally defined chronic headache as having headaches on ≥ 45 days during the past 90 days and on ≥ 15 days during the past 30 days. However, the ICHD-3 defines chronic headache as occurring on ≥ 15 days per month for at least three consecutive months. Therefore, our definition differs slightly from the formal ICHD-3 criteria, which should be considered when comparing our findings with other clinical studies. Finally, this study was conducted exclusively on the Korean population, limiting the generalizability of the findings.

Conclusion

This nationwide population-based study showed that body pain was more frequent and widespread in participants with migraine and TTH than in those without these conditions. A greater widespread pain burden was associated with poorer quality of life and more prominent depression, anxiety, and insomnia symptoms, indicating that non-cephalic pain tracks the overall clinical burden of migraine and TTH. Axial and upper body pains showed the strongest association with headache burden, whereas lower body pain was largely unrelated to headache frequency and intensity. Neck pain was the only site that differed significantly between migraine and TTH, suggesting that neck pain may be a clinically relevant accompanying feature and reflect the potentially distinct contributions of trigeminocervical versus musculoskeletal mechanisms.

Abbreviations

ACR	American college of rheumatology
CHASE	Circannual Change in Headache and Sleep
EQ-5D	EuroQoL-5 Dimensions
FS	Fibromyalgia Severity
FDR	False discovery rate
GAD-7	Generalized Anxiety Disorder-7
HIT-6	Headache Impact Test-6
ICHD-3	International Classification of Headache Disorders, 3rd edition
ISI	Insomnia Severity Index
IQR	Interquartile range
NRS	Numerical rating scale
No HA	No headache
OR	Odds ratio
PHQ-9	Patient Health Questionnaire-9
SSS	Symptom severity scale
TCC	Trigeminocervical complex
TTH	Tension-type headache
WPI	Widespread Pain Index

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s10194-026-02334-x.

Supplementary Material 1

Acknowledgements

None.

Author contributions

WSH designed the study, analysed and interpreted the data, and drafted the manuscript. SC and KMK collected and interpreted the data, and reviewed the manuscript. MKC conceptualised and designed the study; collected, analysed, and interpreted the data; and reviewed the manuscript. All authors read and approved the final manuscript.

Funding

This work was supported by a Severance Hospital Research fund for Clinical excellence (SHRC) (C-2025-0017), and a National Research Foundation of Korea (NRF) grant from the Korean government (MSIT; 2022R1A2C1091767). The funders had no role in the study design, data collection and analysis, decision to publish, or manuscript preparation.

Data availability
The data are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The study was approved by the Institutional Review Board of the Yonsei University College of Medicine (2020-0034-001) and was conducted in accordance with the Declaration of Helsinki. All the participants provided written informed consent to participate in this study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Neurology, Yonsei University College of Medicine, 50-1 Yonsei-ro, Seodaemun-gu, Seoul 03722, Republic of Korea

Received: 22 January 2026 / Accepted: 13 March 2026
Published online: 19 March 2026

References

1. Steiner TJ, Husøy A, Stovner LJ (2024) GBD2021: headache disorders and global lost health - a focus on children, and a view forward. J Headache Pain 25(1):91. https://doi.org/10.1186/s10194-024-01795-2
2. Kim BK, Cho SJ, Kim CS, Sakai F, Dodick DW, Chu MK (2021) Disability and Economic Loss Caused by Headache among Information Technology Workers in Korea. J Clin Neurol 17(4):546–557. https://doi.org/10.3988/jcn.2021.17.4.546
3. Onan D, Younis S, Wellsgatnik WD, Farham F, Andruškevičius S, Abashidze A, Jusupova A, Romanenko Y, Grosu O, Moldokulova MZ, Mursalova U, Saidkhodjaeva S, Martelletti P, Ashina S (2023) Debate: differences and similarities between tension-type headache and migraine. J Headache Pain 24(1):92. https://doi.org/10.1186/s10194-023-01614-0
4. Sandrini G, Rossi P, Milanov I, Serrao M, Cecchini AP, Nappi G (2006) Abnormal modulatory influence of diffuse noxious inhibitory controls in migraine and chronic tension-type headache patients. Cephalalgia 26(7):782–789. https://doi.org/10.1111/j.1468-2982.2006.01130.x
5. Ashina S, Mitsikostas DD, Lee MJ, Yamani N, Wang SJ, Messina R, Ashina H, Buse DC, Pozo-Rosich P, Jensen RH, Diener HC, Lipton RB (2021) Tension-type headache. Nat Rev Dis Primers 7(1):24. https://doi.org/10.1038/s41572-021-00257-2
6. Bartsch T, Goadsby PJ (2003) The trigeminocervical complex and migraine: current concepts and synthesis. Curr Pain Headache Rep 7(5):371–376. https://doi.org/10.1007/s11916-003-0036-y
7. Zagami AS (1994) Pathophysiology of migraine and tension-type headache. Curr Opin Neurol 7(3):272–277. https://doi.org/10.1097/00019052-199406000-00016
8. Burstein R, Jakubowski M, Garcia-Nicas E, Kainz V, Bajwa Z, Hargreaves R, Becerra L, Borsook D (2010) Thalamic sensitization transforms localized pain into widespread allodynia. Ann Neurol 68(1):81–91. https://doi.org/10.1002/ana.21994
9. Scher AI, Buse DC, Fanning KM, Kelly AM, Franznick DA, Adams AM, Lipton RB (2017) Comorbid pain and migraine chronicity: The Chronic Migraine Epidemiology and Outcomes Study. Neurology 89(5):461–468. https://doi.org/10.1212/wnl.0000000000004177
10. Yoon MS, Manack A, Schramm S, Fritsche G, Obermann M, Diener HC, Moebus S, Katsarava Z (2013) Chronic migraine and chronic tension-type headache are associated with concomitant low back pain: results of the German Headache Consortium study. Pain 154(3):484–492. https://doi.org/10.1016/j.pain.2012.12.010
11. Kim KM, Kim BK, Lee W, Hwang H, Heo K, Chu MK (2022) Prevalence and impact of visual aura in migraine and probable migraine: a population study. Sci Rep 12(1):426. https://doi.org/10.1038/s41598-021-04250-3
12. Kim SJ, Lee HJ, Lee SH, Cho S, Kim KM, Chu MK (2023) Most bothersome symptom in migraine and probable migraine: A population-based study. PLoS ONE 18(11):e0289729. https://doi.org/10.1371/journal.pone.0289729
13. Cho S, Kim KM, Chu MK (2024) Coffee consumption and migraine: a population-based study. Sci Rep 14(1):6007. https://doi.org/10.1038/s41598-024-56728-5
14. Vandenbroucke JP, von Elm E, Altman DG, Gøtzsche PC, Mulrow CD, Pocock SJ, Poole C, Schlesselman JJ, Egger M (2007) Strengthening the Reporting of Observational Studies in Epidemiology (STROBE): explanation and elaboration. Ann Intern Med 147(8):W163–194. https://doi.org/10.7326/0003-4819-147-8-200710160-00010-w1
15. Headache Classification Committee of the International Headache Society (IHS) (2018) The international classification of headache disorders, 3rd edition. Cephalalgia 38(1):1–211. https://doi.org/10.1177/0333102417738202
16. Kim BK, Chu MK, Lee TG, Kim JM, Chung CS, Lee KS (2012) Prevalence and impact of migraine and tension-type headache in Korea. J Clin Neurol 8(3):204–211. https://doi.org/10.3988/jcn.2012.8.3.204
17. Zwart JA, Dyb G, Holmen TL, Stovner LJ, Sand T (2004) The prevalence of migraine and tension-type headaches among adolescents in Norway. The Nord-Trøndelag Health Study (Head-HUNT-Youth), a large population-based epidemiological study. Cephalalgia 24(5):373–379. https://doi.org/10.1111/j.1468-2982.2004.00680.x
18. Kim KM, Kim AR, Lee W, Jang BH, Heo K, Chu MK (2022) Development and validation of a web-based headache diagnosis questionnaire. Sci Rep 12(1):7032. https://doi.org/10.1038/s41598-022-11008-y
19. Kim SJ, Lee HJ, Lee SH, Cho S, Kim KM, Chu MK (2023) Visual Aura in Non-Migraine Headaches: A Population Study. J Korean Med Sci 38(31):e237. https://doi.org/10.3346/jkms.2023.38.e237
20. Eriksen MK, Thomsen LL, Olesen J (2005) The Visual Aura Rating Scale (VARS) for migraine aura diagnosis. Cephalalgia 25(10):801–810. https://doi.org/10.1111/j.1468-2982.2005.00955.x
21. Wolfe F, Clauw DJ, Fitzcharles MA, Goldenberg DL, Häuser W, Katz RL, Mease PJ, Russell AS, Russell IJ, Walitt B (2016) 2016 Revisions to the 2010/2011 fibromyalgia diagnostic criteria. Semin Arthritis Rheum 46(3):319–329. https://doi.org/10.1016/j.semarthrit.2016.08.012
22. Kosinski M, Bayliss MS, Bjorner JB, Ware JE Jr., Garber WH, Batenhorst A, Cady R, Dahlöf CG, Dowson A, Tepper S (2003) A six-item short-form survey for measuring headache impact: the HIT-6. Qual Life Res 12(8):963–974. https://doi.org/10.1023/a:1026119331193
23. Kroenke K, Spitzer RL, Williams JB (2001) The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med 16(9):606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x
24. Kroenke K, Spitzer RL, Williams JB, Monahan PO, Löwe B (2007) Anxiety disorders in primary care: prevalence, impairment, comorbidity, and detection. Ann Intern Med 146(5):317–325. https://doi.org/10.7326/0003-4819-146-5-200703060-00004
25. Bastien CH, Vallières A, Morin CM (2001) Validation of the Insomnia Severity Index as an outcome measure for insomnia research. Sleep Med 2(4):297–307. https://doi.org/10.1016/s1389-9457(00)00065-4
26. The EuroQol Group (1990) EuroQol–a new facility for the measurement of health-related quality of life. Health Policy 16(3):199–208. https://doi.org/10.1016/0168-8510(90)90421-9
27. Herdman M, Gudex C, Lloyd A, Janssen M, Kind P, Parkin D, Bonsel G, Badia X (2011) Development and preliminary testing of the new five-level version of EQ-5D (EQ-5D-5L). Qual Life Res 20(10):1727–1736. https://doi.org/10.1007/s11136-011-9903-x
28. Lee YK, Nam HS, Chuang LH, Kim KY, Yang HK, Kwon IS, Kind P, Kweon SS, Kim YT (2009) South Korean time trade-off values for EQ-5D health states: modeling with observed values for 101 health states. Value Health 12(8):1187–1193. https://doi.org/10.1111/j.1524-4733.2009.00579.x
29. Chu M, Im H-J, Ju Y-S, Ma H-I, Kim Y, Kim J, Lee K-J (2009) Validity and reliability assessment of Korean headache impact test 6 (HIT6). J Korean Neurol Assoc 27
30. Cho YW, Song ML, Morin CM (2014) Validation of a Korean version of the insomnia severity index. J Clin Neurol 10(3):210–215. https://doi.org/10.3988/jcn.2014.10.3.210
31. Choi HS, Choi JH, Park KH, Joo KJ, Ga H, Ko HJ, Kim SR (2007) Standardization of the Korean Version of Patient Health Questionnaire-9 as a screening instrument for major depressive disorder. J Korean Acad Fam Med 28:114–119
32. Ahn JK, Kim Y, Choi KH (2019) The Psychometric Properties and Clinical Utility of the Korean Version of GAD-7 and GAD-2. Front Psychiatry 10:127. https://doi.org/10.3389/fpsyt.2019.00127

33. Moulton EA, Burstein R, Tully S, Hargreaves R, Becerra L, Borsook D (2008) Interictal dysfunction of a brainstem descending modulatory center in migraine patients. PLoS ONE 3(11):e3799. https://doi.org/10.1371/journal.pone.0003799
34. Duan S, Ren Z, Xia H, Wang Z, Zheng T, Li G, Liu L, Liu Z (2023) Associations between anxiety, depression with migraine, and migraine-related burdens. Front Neurol 14:1090878. https://doi.org/10.3389/fneur.2023.1090878
35. Ashina S, Bendtsen L, Lyngberg AC, Lipton RB, Hajiyeva N, Jensen R (2015) Prevalence of neck pain in migraine and tension-type headache: a population study. Cephalalgia 35(3):211–219. https://doi.org/10.1177/0333102414535110
36. Al-Khazali HM, Younis S, Al-Sayegh Z, Ashina S, Ashina M, Schytz HW (2022) Prevalence of neck pain in migraine: A systematic review and meta-analysis. Cephalalgia 42(7):663–673. https://doi.org/10.1177/03331024211068073
37. Bogduk N (2004) The neck and headaches. Neurol Clin 22(1):151–171. https://doi.org/10.1016/s0733-8619(03)00100-2
38. Jensen R, Rasmussen BK, Pedersen B, Olesen J (1993) Muscle tenderness and pressure pain thresholds in headache. A population study. Pain 52(2):193–199. https://doi.org/10.1016/0304-3959(93)90131-8
39. Suzuki K, Suzuki S, Shiina T, Kobayashi S, Hirata K (2022) Central Sensitization in Migraine: A Narrative Review. J Pain Res 15:2673–2682. https://doi.org/10.2147/jpr.S329280
40. Bendtsen L (2000) Central sensitization in tension-type headache–possible pathophysiological mechanisms. Cephalalgia 20(5):486–508. https://doi.org/10.1046/j.1468-2982.2000.00070.x
41. Goadsby PJ, Holland PR, Martins-Oliveira M, Hoffmann J, Schankin C, Akerman S (2017) Pathophysiology of Migraine: A Disorder of Sensory Processing. Physiol Rev 97(2):553–622. https://doi.org/10.1152/physrev.00034.2015
42. Puledda F, Silva EM, Suwanlaong K, Goadsby PJ (2023) Migraine: from pathophysiology to treatment. J Neurol 270(7):3654–3666. https://doi.org/10.1007/s00415-023-11706-1
43. Fernández-de-las-Peñas C, Cuadrado ML, Arendt-Nielsen L, Simons DG, Pareja JA (2007) Myofascial trigger points and sensitization: an updated pain model for tension-type headache. Cephalalgia 27(5):383–393. https://doi.org/10.1111/j.1468-2982.2007.01295.x
44. Amiri P, Kazeminasab S, Nejadghaderi SA, Mohammadinasab R, Pourfathi H, Araj-Khodaei M, Sullman MJM, Kolahi AA, Safiri S (2021) Migraine: A Review on Its History, Global Epidemiology, Risk Factors, and Comorbidities. Front Neurol 12:800605. https://doi.org/10.3389/fneur.2021.800605
45. Li XY, Yang CH, Lv JJ, Liu H, Zhang LY, Yin MY, Guo ZL, Zhang RH (2023) Global, regional, and national epidemiology of migraine and tension-type headache in youths and young adults aged 15–39 years from 1990 to 2019: findings from the global burden of disease study 2019. J Headache Pain 24(1):126. https://doi.org/10.1186/s10194-023-01659-1
46. Takeshima T, Ishizaki K, Fukuhara Y, Ijiri T, Kusumi M, Wakutani Y, Mori M, Kawashima M, Kowa H, Adachi Y, Urakami K, Nakashima K (2004) Population-Based Door-to-Door Survey of Migraine in Japan: The Daisen Study. Headache: J Head Face Pain 44(1):8–19. https://doi.org/10.1111/j.1526-4610.2004.04004.x
47. Wang SJ, Fuh JL, Young YH, Lu SR, Shia BC (2000) Prevalence of migraine in Taipei, Taiwan: a population-based survey. Cephalalgia 20(6):566–572. https://doi.org/10.1046/j.1468-2982.2000.00085.x
48. Kim KM, Cho SJ, Shin HJ, Yang KI, Kim D, Yun CH, Chu MK (2021) Prevalence, Disability, and Management Patterns of Migraine in Korea: Nationwide Survey Data from 2009 and 2018. J Clin Neurol 17(1):77–85. https://doi.org/10.3988/jcn.2021.17.1.77
49. Rolke R, Magerl W, Campbell KA, Schalber C, Caspari S, Birklein F, Treede RD (2006) Quantitative sensory testing: a comprehensive protocol for clinical trials. Eur J Pain 10(1):77–88. https://doi.org/10.1016/j.ejpain.2005.02.003
50. Fischer AA (1987) Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. Pain 30(1):115–126. https://doi.org/10.1016/0304-3959(87)90089-3
51. Bendtsen L, Jensen R, Jensen NK, Olesen J (1995) Pressure-controlled palpation: a new technique which increases the reliability of manual palpation. Cephalalgia 15(3):205–210. https://doi.org/10.1046/j.1468-2982.1995.015003205.x
52. Kang JH, An M, Choi SE, Xu H, Park DJ, Lee JK, Lee SS (2019) Performance of the revised 2016 fibromyalgia diagnostic criteria in Korean patients with fibromyalgia. Int J Rheum Dis 22(9):1734–1740. https://doi.org/10.1111/1756-185x.13661

Publisher's note