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A Multidimensional Clinical-Functional Model of Cervicogenic Headache: Neck Disability, Pain Topography, and Treatment Response in a Multicentre Cohort

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Abstract

Background

Cervicogenic headache is clinically challenging because cervical symptoms, imaging abnormalities, and migraine-like features may overlap. This multicentre study aimed to characterise cervicogenic headache as a multidimensional clinical-functional phenotype and to determine whether neck disability, pain topography, cervical provocation findings, migrainous symptom burden, and treatment response provide diagnostic information beyond cervical imaging alone.

Methods

This observational, cross-sectional study included 660 patients with ICHD-3-based cervicogenic headache evaluated at 15 neurology and headache clinics between 2023 and 2025. Headache burden, cervical imaging findings, Neck Disability Index (NDI) score, neck-to-head radiating pain, cervical provocation signs, pain localisation, migrainous accompaniments, lifestyle factors, and treatment response were analysed. The primary outcome was headache attack severity. Correlation, multivariable regression, phenotype, block-response, and exploratory mediation analyses were performed.

Results

Median age was 44 years, 72.9% were women, median monthly headache frequency was 8 days, and median attack severity was 7/10. Neck-to-head radiating pain was reported by 94.8% of patients, and clinically relevant cervical imaging abnormalities were present in 70.6%. Cervical straightening, radiculopathy, and clinically relevant imaging abnormalities were associated with greater headache burden in unadjusted analyses, but did not independently predict attack severity after adjustment. NDI was the strongest independent predictor of attack severity and correlated with monthly headache frequency and attack duration. Exploratory mediation analyses suggested that the associations between cervical radiculopathy and cervical straightening and attack severity were largely mediated by NDI. Occipital/craniocervical pain clustered with higher NDI, cervical provocation findings, and better block-related outcomes, whereas frontal/periorbital pain clustered with migrainous accompaniments. Migrainous symptom burden independently contributed to attack severity. Among treated patients, block response was better explained by dominant pain phenotype than by NDI or structural imaging findings alone.

Conclusion

This study moves cervicogenic headache beyond an imaging-based construct and supports a multidimensional clinical-functional model in which neck disability, pain radiation, posterior pain

topography, cervical provocation findings, migrainous overlap, and treatment response define clinically meaningful headache burden, thereby improving differential diagnosis and treatment selection.

INTRODUCTION

Cervicogenic headache is a secondary headache disorder attributed to nociceptive input from cervical spine structures, including osseous, discal, articular, muscular, and neural elements [1–3]. Its biological plausibility is supported by the convergence of upper cervical afferents with trigeminal pathways within the trigeminocervical complex, through which cervical nociception may be perceived as occipital, temporal, frontal, orbital, or diffuse head pain [2–4]. This anatomical and functional overlap explains why cervicogenic headache may closely resemble migraine or other headache phenotypes and why distinguishing a cervical pain generator from a primary headache disorder remains challenging in clinical practice [2, 3, 5].

Although cervicogenic headache has long been recognized, its clinical phenotype remains incompletely defined [3, 6]. The diagnosis often depends not on a single defining feature, but on the interpretation of several overlapping clinical, functional, radiological, and treatment-related findings [1, 2, 5].

The diagnostic challenge is particularly relevant in the differential diagnosis between cervicogenic headache and migraine [5, 7, 8]. Neck pain is highly prevalent in migraine and may occur before, during, or after the headache phase, whereas cervicogenic headache may present with nausea, photophobia, phonophobia, dizziness, and other symptoms traditionally considered migrainous [3, 9, 10]. Therefore, the presence of migraine-like symptoms does not exclude a cervical contribution, and the presence of neck pain alone does not establish a cervicogenic origin [2, 5, 7]. This creates a clinically important grey zone in which pain topography, neck-related disability, cervical provocation, imaging findings, and response to cervical interventions must be interpreted together [1–3, 5]. Cervical straightening, degenerative abnormalities, and radicular or other structural findings may reflect a clinically relevant pain generator in some patients, but may also be nonspecific or incidental in others [2, 11–13]. Overreliance on imaging may lead to misclassification, whereas underrecognition of functional cervical impairment may delay targeted treatment [5, 7, 11, 12].

Neck disability may represent a key clinical link between cervical pathology and headache burden [3, 7, 14]. It may be clinically meaningful to determine whether cervical straightening or other structural abnormalities translate into functional cervical impairment rather than assuming that imaging findings alone explain headache severity [5, 11–13]. The Neck Disability Index may help operationalize this pathway by capturing patient-reported pain-related functional impact in everyday activities rather than relying on structural findings alone [15, 16]. In this context, neck disability may function not merely as a coexisting cervical complaint, but as a marker of the extent to which cervical dysfunction contributes to headache burden [3, 7, 14].

Clinical examination and pain topography may further refine this distinction [2, 5, 7]. Pain beginning in the neck and radiating to the head, restricted neck movement, posture- or movement-triggered headache,

a positive flexion-rotation test, and pain provoked by upper cervical or occipital pressure may indicate a stronger cervical contribution [5, 17–20]. Similarly, occipital and craniocervical pain may suggest a cervicogenic-dominant phenotype, whereas more frontal or periorbital pain may overlap more closely with migraine-like presentations [3, 8, 21]. However, these features have only rarely been evaluated together with neck disability, cervical imaging findings, migrainous symptom burden, and treatment response within the same cohort [5, 6, 8]. A multidimensional approach may therefore be more useful than a binary imaging-based model [2, 3, 5, 7].

This multicentre study aimed to characterize cervicogenic headache as a multidimensional clinical-functional phenotype and to identify factors relevant to its differentiation from migraine-like presentations. We examined the associations of headache burden with cervical imaging abnormalities, cervical straightening, cervical radiculopathy, Neck Disability Index score, neck-to-head pain radiation, cervical provocation findings, pain topography, migrainous symptom burden, lifestyle-related factors, and response to cervical interventions. The primary outcome was headache attack severity; secondary outcomes included monthly headache frequency, attack duration, NDI score, pain localization, post-treatment neck pain course, and headache reduction after cervical structure or nerve block.

We also assessed whether NDI was associated with headache burden and whether it mediated the relationship between cervical pathology and headache severity. In addition, we compared occipital/craniocervical and frontal/periorbital pain phenotypes with respect to cervical findings, migrainous features, provocation signs, and treatment response. We hypothesized that neck-related disability, pain radiation, cervical provocation, pain topography, migrainous overlap, and response to cervical interventions would provide clinically meaningful diagnostic value beyond cervical imaging abnormalities alone.

METHODS

Study design and participants

This multicentre, observational, cross-sectional study included patients with cervicogenic headache who were evaluated at 15 participating neurology and headache clinics between 2023 and 2025. Diagnosis was established according to the International Classification of Headache Disorders, third edition (ICHD-3), based on headache history, cervical symptoms, clinical examination findings, and cervical imaging when available [1]. The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee of Bülent Ecevit University Application and Research Hospital (approval number: 2023/06). All participants provided written informed consent.

Eligible patients had an ICHD-3-based clinical diagnosis of cervicogenic headache and available data on headache severity and cervical clinical features. Patients with missing core clinical data or headache attributed to another major neurological, infectious, inflammatory, neoplastic, traumatic, or systemic disorder were excluded from the relevant analyses. The final analyzable cohort comprised 660 patients.

Demographic, headache-related, cervical, and lifestyle variables were collected using a standardized clinical dataset. Headache burden was characterized by attack severity, monthly headache days, attack duration, pain localization, pain character, and accompanying symptoms. Attack severity was rated on a 0–10 numerical/visual analogue scale, and attack duration was assessed using a predefined ordinal scale. Pain localization was classified as frontal, temporal, occipital, craniocervical, periorbital, or diffuse whole-head pain. Associated symptoms, neck-to-head pain radiation, NDI score, cervical provocation findings, cervical imaging variables, daily screen exposure, screen posture, and exercise habits were recorded. Provocation findings included headache triggered by neck movement, sustained posture, or routine neck-related activity; a positive flexion-rotation test; and headache induced by upper cervical or occipital pressure. Cervical imaging variables included cervical straightening, cervical radiculopathy, and clinically relevant cervical abnormalities, which were interpreted together with clinical and examination findings rather than as isolated diagnostic markers.

Migraine-like accompanying symptoms were systematically assessed and summarized using a 0–7 migrainous symptom score based on the presence of nausea, vomiting, photophobia, phonophobia, osmophobia, allodynia, and dizziness. Higher scores reflected greater migrainous symptom burden and were analyzed in relation to headache severity, pain topography, and treatment-related outcomes.

Treatment variables and outcomes

Treatment data included medication use, non-pharmacological therapies, and interventional procedures, together with their reported benefits. In patients who underwent cervical structure or nerve blocks, headache reduction after block and the reported benefit of greater occipital nerve block, lesser occipital nerve block, and trigger point injection were recorded. Block-response analyses were restricted to treated patients to avoid classifying untreated patients as non-responders.

Post-treatment neck pain course was categorized as complete improvement, 75% reduction, 50% reduction, 25% reduction, no change, or worsening. For binary analyses, complete improvement and 75% or 50% reduction were classified as good response, whereas 25% reduction, no change, or worsening were classified as poor response.

Outcomes and explanatory domains

The primary outcome was headache attack severity. Secondary outcomes included monthly headache frequency, attack duration, NDI score, pain localization, post-treatment neck pain course, and headache reduction after cervical structure or nerve block.

Explanatory domains included demographic characteristics, cervical imaging findings, neck disability, neck-to-head pain radiation, cervical provocation findings, pain localization, migrainous symptom burden, screen exposure, exercise habits, and treatment response. The analytical framework was designed to

evaluate cervicogenic headache as a multidimensional clinical-functional phenotype rather than an imaging-defined condition alone.

For subgroup analyses, patients were classified according to selected clinical phenotype markers. High- and low-NDI groups were defined using the cohort median value. Patients with NDI values at or below the median were classified as the low NDI group, whereas those with values above the median were classified as the high NDI group.

Pain topography phenotypes were defined by posterior/cervicogenic and anterior/migraine-like pain distributions. A cervicogenic-dominant pain phenotype was defined by occipital and/or craniocervical junction pain predominance. A migraine-like anterior pain phenotype was defined by frontal and/or periorbital pain predominance. When posterior and anterior pain features overlapped without clear predominance, patients were classified as having a mixed phenotype. These phenotype groups were compared for headache burden, NDI score, cervical imaging findings, provocation signs, migrainous symptoms, and treatment response.

Statistical analysis

Continuous and ordinal variables were summarized as median and interquartile range, and categorical variables as number and percentage. Missing data were reported for each variable, and analyses used available data without imputation. Group comparisons were performed with the Mann–Whitney U or Kruskal–Wallis test for continuous or ordinal variables and the chi-square or Fisher's exact test for categorical variables, as appropriate. All tests were two-sided, with $p < 0.05$ considered statistically significant.

Associations between NDI and headache burden were assessed using Spearman's rank correlation. Multivariable regression models were used to identify independent predictors of headache attack severity, with demographic variables, cervical imaging findings, NDI score, and neck-to-head radiating pain included in the primary model. A complementary model evaluated the added contribution of migrainous symptom burden. Multicollinearity was assessed before final interpretation.

Exploratory mediation analyses tested whether NDI statistically mediated the associations of cervical radiculopathy and cervical straightening with headache attack severity, using bootstrapped confidence intervals for indirect effects. Because of the cross-sectional design, these analyses were interpreted as indirect-pathway models rather than causal mediation. Block-response analyses were restricted to treated patients and evaluated clinical, topographic, imaging, provocation, and migrainous predictors of response using group comparisons and logistic regression. Post-treatment neck pain course was analyzed using ordinal and binary response definitions. Analyses were performed using on Statistical Package for Social Sciences (SPSS) for Windows version 22.0 software.

RESULTS

Study population

A total of 660 patients with cervicogenic headache were included in the final analysable cohort. The median age was 44 years (IQR 34–54), and 481 patients (72.9%) were women. Median disease duration was 3 years (IQR 1–5), median monthly headache frequency was 8 days (IQR 4–15), median attack severity was 7 (IQR 6–8), and median NDI score was 3 (IQR 2–3). Baseline demographic and clinical characteristics are summarized in Table 1.

Table 1
Demographic, headache, and baseline clinical characteristics of the cohort.

Domain	Variable	n	Value
Continuous variables	Age, years	660	44 (34–54)
	Disease duration, years	660	3 (1–5)
	Monthly headache days	660	8 (4–15)
	Headache attack severity, 0–10	660	7 (6–8)
	Attack duration category	660	3 (2–3)
	Neck Disability Index (NDI)	660	3 (2–3)
	Daily screen exposure, hours	560	3 (1–4)
Sex	Female	660	481 (72.9%)
	Male	660	179 (27.1%)
Pain radiation	Neck-to-head radiating pain	660	626 (94.8%)
Pain distribution	Side-locked pain	660	238 (36.1%)
	Unilateral radiating/spreading pain	660	320 (48.5%)
	Pain radiating to the neck	660	102 (15.5%)
Pain location	Frontal pain	660	151 (22.9%)
	Temporal pain	660	185 (28.0%)
	Occipital pain	660	408 (61.8%)
	Craniocervical junction pain	660	437 (66.2%)
	Periorbital pain	660	65 (9.8%)
	Diffuse whole-head pain	660	110 (16.7%)
Pain character	Pulsating pain	660	272 (41.2%)
	Pressing/tightening pain	660	321 (48.6%)
	Stabbing pain	660	109 (16.5%)
	Pressure-like pain	660	276 (41.8%)
	Explosive pain	660	30 (4.5%)
Associated symptoms	Shoulder pain	660	333 (50.5%)

Values are median (Q1-Q3) for continuous variables and n (%) for categorical variables. NDI: Neck Disability Index.

Domain	Variable	n	Value
	Restricted neck movement	660	368 (55.8%)
	Vertigo-like symptoms	660	168 (25.5%)
	Dizziness	660	103 (15.6%)
	Nausea	660	248 (37.6%)
	Vomiting	660	89 (13.5%)
	Photophobia	660	206 (31.2%)
	Phonophobia	660	210 (31.8%)
	Osmophobia	660	95 (14.4%)
	Allodynia	660	88 (13.3%)

Values are median (Q1-Q3) for continuous variables and n (%) for categorical variables. NDI: Neck Disability Index.

Neck-to-head radiating pain was reported by 626 patients (94.8%). The most frequent pain locations were the craniocervical junction (66.2%) and occipital region (61.8%), followed by temporal, frontal, diffuse whole-head, and periorbital pain. Shoulder pain and restricted neck movement were reported by 50.5% and 55.8% of patients, respectively. Migrainous accompaniments were also common, particularly nausea, photophobia, and phonophobia. Cervical provocation findings were frequent: headache was triggered by sustained neck posture in 76.2%, routine neck-related physical activity in 70.8%, and neck movement in 67.0%. The flexion-rotation test was positive in 64.1%, and headache was provoked by upper cervical or occipital pressure in approximately 60% of patients. Clinically relevant cervical imaging abnormalities were present in 70.6%, cervical straightening in 47.4%, and cervical radiculopathy in 38.5%.

Cervical imaging findings and headache burden

Cervical imaging findings were associated with greater headache burden in unadjusted analyses. Cervical straightening, cervical radiculopathy, and clinically relevant cervical imaging abnormalities were each associated with higher monthly headache frequency, greater attack severity, longer attack duration, and higher NDI scores. However, these structural variables were no longer independent predictors of headache attack severity in multivariable models after functional and clinical variables were included.

Neck disability and headache burden

NDI was positively associated with all major headache burden measures (Table 2). The strongest correlation was observed between NDI and headache attack severity (Spearman's $\rho = 0.352$, $p < 0.001$). NDI also correlated with monthly headache frequency ($\rho = 0.265$, $p < 0.001$) and attack duration category

(ρ = 0.263, p < 0.001), indicating that neck-related functional impairment was associated not only with cervical symptoms but also with headache intensity, frequency, and duration.

Table 2
Spearman correlations among NDI, headache burden, disease duration, age, and screen exposure.

Variable pair	Spearman rho	p value
NDI - headache attack severity	0.352	< 0.001
NDI - monthly headache days	0.265	< 0.001
NDI - attack duration category	0.263	< 0.001
NDI - disease duration	0.150	< 0.001
NDI - age	0.219	< 0.001
NDI - daily screen exposure	-0.174	< 0.001
Disease duration - headache attack severity	0.208	< 0.001
Disease duration - monthly headache days	0.021	0.583
Disease duration - attack duration category	0.186	< 0.001
Age - disease duration	0.173	< 0.001
Age - monthly headache days	0.102	0.008
Age - headache attack severity	0.016	0.682
Age - attack duration category	0.063	0.105
Daily screen exposure - attack severity	-0.138	0.001
Daily screen exposure - attack duration category	-0.124	0.003

NDI: Neck Disability Index. Correlations are Spearman rho coefficients.

Table 3
Dominant pain phenotypes, NDI differences, and regression model for attack severity.

Analysis	Variable / comparison	Migraine-like anterior	Cervicogenic-dominant	Statistic / difference	p value
Dominant phenotype comparison	NDI, mean	2.15	2.86	t=-6.04	< 0.001
	Headache attack severity, mean	6.21	6.83	t=-2.01	0.048
	Monthly headache days, mean	8.04	10.94	t=-2.80	0.006
	Attack duration category, mean	2.47	2.65	t=-1.53	0.130
Categorical comparison	Neck movement-triggered headache	-	-	chi2 = 17.84	< 0.001
	Occipital pain	-	-	chi2 = 158.81	< 0.001
	Treatment-related headache reduction	-	-	chi2 = 16.57	< 0.001
NDI across 3 phenotypes	Overall ANOVA	-	-	F = 24.49	< 0.001
Tukey post-hoc	Mixed vs migraine-like anterior	-	-	difference = 0.52	< 0.001
	Cervicogenic-dominant vs migraine-like anterior	-	-	difference = 0.72	< 0.001
	Cervicogenic-dominant vs mixed	-	-	difference = 0.19	0.042
Regression for attack severity	Cervicogenic-dominant phenotype	-	B = 0.121, SE = 0.223	t = 0.544	0.587
	NDI	-	B = 0.802, SE = 0.094	t = 8.554	< 0.001
	Age	-	B=-0.0069, SE = 0.0052	t=-1.329	0.184

Dominant phenotype comparison excluded the mixed phenotype. Occipital and craniocervical pain were part of the cervicogenic-dominant phenotype definition; therefore, occipital pain should not be interpreted as an independent validation marker. Regression dependent variable: headache attack severity; model R2 = 0.158, adjusted R2 = 0.152.

Analysis	Variable / comparison	Migraine-like anterior	Cervicogenic-dominant	Statistic / difference	p value
	Male sex	-	B=-0.433, SE = 0.159	t=-2.722	0.007

Dominant phenotype comparison excluded the mixed phenotype. Occipital and craniocervical pain were part of the cervicogenic-dominant phenotype definition; therefore, occipital pain should not be interpreted as an independent validation marker. Regression dependent variable: headache attack severity; model R2 = 0.158, adjusted R2 = 0.152.

Disease duration showed weak but significant correlations with attack severity, attack duration, and NDI, but not with monthly headache frequency. Thus, longer disease duration appeared to be more closely related to severity, duration, and neck disability than to headache frequency.

When patients were classified by the cohort median NDI value, the high-NDI group had greater attack severity, more monthly headache days, and longer attack durations than the low-NDI group. High NDI was also associated with neck movement-triggered headache and, more weakly, with occipital pain. Treatment response did not differ significantly between NDI groups. In an age- and sex-adjusted regression model, high NDI remained independently associated with greater attack severity.

Neck-to-head radiating pain

Neck-to-head radiating pain identified a high-burden clinical phenotype. Patients with radiating pain had more monthly headache days than those without radiating pain (median 8 vs. 2 days, $p < 0.001$), higher attack severity (median 7 vs. 4, $p < 0.001$), longer attack duration ($p < 0.001$), and higher NDI scores (median 3 vs. 1, $p < 0.001$). Radiating pain was also associated with occipital and craniocervical pain, restricted neck movement, shoulder pain, neck movement-triggered headache, posture-triggered headache, positive flexion-rotation test, upper cervical pressure-induced headache, and occipital pressure-induced headache. Radiating pain was also associated with recorded headache reduction after cervical structure or nerve block.

Pain localization and dominant pain phenotypes

Pain topography separated clinically meaningful phenotypes (Table 4). Occipital and craniocervical pain were associated with higher NDI scores, greater headache burden, cervical imaging abnormalities, cervical provocation findings, block application, and recorded headache reduction after block. Craniocervical pain showed the most consistent cervicogenic profile across imaging, disability, provocation, and treatment-response domains.

Table 4
Multivariable linear regression model for headache attack severity.

Predictor	B	SE	Beta	95% CI for B	t	p value
Constant	7.749	0.696	-	6.383 to 9.115	11.137	< 0.001
Age	-0.015	0.005	-0.117	-0.025 to -0.006	-3.104	0.002
Disease duration	0.046	0.011	0.147	0.024 to 0.069	4.094	< 0.001
NDI	0.682	0.090	0.304	0.504 to 0.859	7.542	< 0.001
Male sex	-0.310	0.147	-0.074	-0.598 to -0.022	-2.113	0.035
Neck-to-head radiating pain coding effect	-1.528	0.325	-0.182	-2.166 to -0.890	-4.705	< 0.001
Clinically relevant cervical imaging abnormality	-0.124	0.226	-0.030	-0.567 to 0.319	-0.550	0.583
Cervical straightening	-0.060	0.179	-0.016	-0.411 to 0.291	-0.335	0.738
Cervical radiculopathy	-0.133	0.180	-0.035	-0.485 to 0.220	-0.740	0.460

Dependent variable: headache attack severity. Model: R = 0.469, R2 = 0.220, adjusted R2 = 0.210, F = 22.890, p < 0.001. Sex was coded as 1 = female and 2 = male. Neck-to-head radiating pain was coded as 1 = yes and 2 = no; the negative coefficient therefore indicates lower severity in patients without radiating pain.

In contrast, frontal and periorbital pain were more closely linked to migraine-like features. Frontal pain was associated with nausea, vomiting, photophobia, phonophobia, and osmophobia, whereas periorbital pain was associated with multiple migrainous accompaniments, including allodynia and vertigo-like symptoms. These findings suggest that occipital/craniocervical pain reflects a more cervicogenic-dominant phenotype, whereas frontal/periorbital pain overlaps more closely with an anterior migraine-like phenotype.

When patients were classified by dominant pain topography, the cervicogenic-dominant phenotype had higher NDI scores than the migraine-like anterior phenotype (mean 2.86 vs 2.15, p < 0.001). The cervicogenic-dominant phenotype also had higher headache attack severity (mean 6.83 vs 6.21, p = 0.048) and more monthly headache days (mean 10.94 vs 8.04, p = 0.006). Attack duration did not differ significantly between the two dominant phenotypes (mean 2.65 vs. 2.47, p = 0.130).

NDI differed significantly across the three phenotype groups ($F = 24.49$, $p < 0.001$). Post-hoc comparisons showed that NDI was higher in the cervicogenic-dominant phenotype than in the migraine-like anterior phenotype (mean difference 0.72, $p < 0.001$) and the mixed phenotype (mean difference 0.19, $p = 0.042$). The mixed phenotype also had higher NDI scores than the migraine-like anterior phenotype (mean difference 0.52, $p < 0.001$).

In a regression model including phenotype, NDI, age, and sex, NDI remained strongly associated with headache attack severity ($B = 0.80$, $p < 0.001$), whereas cervicogenic-dominant phenotype was no longer independently associated with attack severity after adjustment for NDI ($B = 0.12$, $p = 0.587$). This suggests that although pain topography defines clinically useful phenotypes, neck-related functional impairment may better explain headache severity than phenotype classification alone.

Cervical provocation findings

Cervical provocation findings were consistently associated with headache burden. Neck movement-triggered headache, posture-triggered headache, positive flexion-rotation test, upper cervical pressure-induced headache, and occipital pressure-induced headache were each associated with higher attack severity, higher monthly headache frequency, longer attack duration, and higher NDI. These findings were also associated with block-related variables, suggesting that cervical provocation signs may help identify patients with a treatment-relevant cervical pain generator.

Migrainous accompaniments and migrainous symptom burden

Migrainous symptoms were common in this cohort of cervicogenic headache patients and were associated with greater headache burden. Nausea, vomiting, photophobia, phonophobia, osmophobia, and allodynia were associated with higher attack severity and longer attack duration, while photophobia, phonophobia, and osmophobia were also associated with higher monthly headache frequency. Several migrainous symptoms were additionally associated with higher NDI scores.

Migraine-like symptoms were strongly linked to anterior pain topography. Frontal and periorbital pain clustered with multiple migrainous accompaniments, supporting the concept that migraine-like symptoms may coexist with cervicogenic headache and should not be interpreted as excluding a cervical contribution.

In the multivariable model including migrainous symptom score, NDI, age, and sex, both NDI and migrainous symptom burden were independently associated with attack severity. NDI showed the strongest association ($B = 0.778$, $p < 0.001$), followed by the migrainous symptom score ($B = 0.318$, $p < 0.001$). Age and sex were not significant in this model, which explained 26.2% of the variance in attack severity.

High and low NDI groups

Patients were divided into low- and high-NDI groups based on the cohort median. Compared with the low NDI group, the high NDI group had higher headache attack severity (mean 7.89 vs 6.47, $p < 0.001$), more monthly headache days (mean 13.95 vs 9.84, $p < 0.001$), and longer attack duration (mean 3.00 vs 2.55, $p < 0.001$). High NDI was also associated with headache triggered by neck movement ($\chi^2=30.33$, $p < 0.001$) and, more weakly, with occipital pain ($\chi^2=4.52$, $p = 0.034$). Treatment response did not differ significantly between NDI groups ($\chi^2 = 1.71$, $p = 0.190$).

In a regression model adjusted for age and sex, high NDI remained independently associated with greater headache attack severity ($B = 1.38$, $p < 0.001$). Age was not associated with attack severity in this model, whereas male sex was associated with lower attack severity ($B = -0.45$, $p = 0.004$).

Multivariable predictors of headache attack severity

The main multivariable model for headache attack severity is shown in Table 4. The overall model was significant and explained 22.0% of the variance in attack severity. NDI was the strongest independent predictor of attack severity ($B = 0.682$, $\beta = 0.304$, 95% CI 0.504 to 0.859, $p < 0.001$). Disease duration and neck-to-head radiating pain were also independently associated with greater attack severity. Male sex and older age were associated with small negative associations with attack severity.

In contrast, cervical straightening, cervical radiculopathy, and clinically relevant cervical imaging abnormalities were not independently associated with attack severity after adjustment for clinical and functional variables. These findings indicate that imaging abnormalities alone do not explain headache severity when neck disability and radiation of pain are considered.

Mediation analyses

Exploratory mediation analyses were performed to evaluate whether NDI statistically mediated the relationship between cervical structural pathology and headache attack severity. In the cervical radiculopathy → NDI → attack severity model, the indirect effect through NDI was significant (ACME = 0.444, 95% CI 0.297 to 0.615, $p < 0.001$), whereas the direct effect of cervical radiculopathy on attack severity was not significant (ADE = 0.111, 95% CI -0.177 to 0.394, $p = 0.450$). The total effect was significant (0.555, 95% CI 0.265 to 0.848, $p < 0.001$), and the estimated proportion mediated was approximately 80%.

In the cervical straightening → NDI → attack severity model, the indirect effect through NDI was also significant (ACME = 0.232, 95% CI 0.119 to 0.362, $p < 0.001$), whereas the direct effect of cervical straightening on attack severity was not significant (ADE = 0.149, 95% CI -0.109 to 0.405, $p = 0.248$). The total effect was significant (0.381, 95% CI 0.099 to 0.668, $p = 0.009$), and the estimated proportion mediated was approximately 61%.

These findings support an indirect-pathway model in which the effects of cervical radiculopathy and cervical straightening on headache attack severity are mediated largely by neck-related functional

impairment rather than by a direct structural effect alone. Exploratory mediation analyses evaluating NDI as an indirect pathway between cervical pathology and attack severity are presented in Table 5.

Table 5
Exploratory mediation analyses evaluating NDI as an indirect pathway between cervical pathology and headache attack severity.

Pathway	Effect	Estimate	95% CI	p value
Cervical radiculopathy -> NDI -> attack severity	ACME (indirect effect)	0.444	0.297 to 0.615	< 0.001
	ADE (direct effect)	0.111	-0.177 to 0.394	0.450
	Total effect	0.555	0.265 to 0.848	< 0.001
	Proportion mediated	0.800	0.488 to 1.615	< 0.001
Cervical straightening -> NDI -> attack severity	ACME (indirect effect)	0.232	0.119 to 0.362	< 0.001
	ADE (direct effect)	0.149	-0.109 to 0.405	0.248
	Total effect	0.381	0.099 to 0.668	0.009
	Proportion mediated	0.610	0.303 to 1.821	0.010

ACME: average causal mediation effect; ADE: average direct effect; NDI: Neck Disability Index. Analyses should be interpreted as exploratory statistical mediation/indirect-pathway models because of the cross-sectional design.

Treatment response and cervical block

Cervical structure or nerve block was performed in 247 patients (37.4%) (Table 6). Patients who underwent block had higher headache burden and higher NDI scores, and block application was associated with occipital and craniocervical pain, clinically relevant cervical imaging abnormalities, and cervical provocation findings.

Table 6
Predictors and phenotype distribution of block response among treated patients.

Analysis	Variable / comparison	Estimate / distribution	95% CI / statistic	p value
Logistic regression	NDI	OR = 0.77	0.40 to 1.44	0.42
	Age	OR = 0.99	0.96 to 1.02	0.41
	Male sex	OR = 0.55	0.23 to 1.36	0.18
	Absence of cervical straightening	OR = 0.83	0.33 to 2.04	0.68
	Absence of cervical radiculopathy	OR = 1.08	0.42 to 2.78	0.87
NDI direct comparison	Non-responders vs responders	3.07 vs 2.95	t = 0.79	0.437
Mediation: cervical straightening -> NDI -> block response	ACME (indirect effect)	0.001	-0.008 to 0.011	0.84
	ADE (direct effect)	-0.018	-0.108 to 0.079	0.72
	Total effect	-0.017	-0.107 to 0.081	0.74
	Proportion mediated	-0.08	wide	0.99
Response by phenotype	Migraine-like anterior	7 no / 8 yes; response ~ 53%	-	-
	Mixed	7 no / 34 yes; response ~ 83%	-	-
	Cervicogenic-dominant	13 no / 178 yes; response ~ 93%	chi2 = 24.60	< 0.001

Analyses were restricted to patients who received a cervical structure or nerve block (n = 247). Response was defined as recorded headache reduction after block. OR: odds ratio; ACME: average causal mediation effect; ADE: average direct effect.

Definitive block-response analyses were restricted to treated patients. In logistic regression among treated patients, NDI, age, sex, cervical straightening, and cervical radiculopathy did not independently predict block response. NDI was also not associated with block response in direct group comparison, and mediation analysis showed no evidence that NDI mediated the relationship between cervical straightening and block response.

In contrast, the dominant pain phenotype strongly differentiated the block response. Response was observed in approximately 53% of patients with the migraine-like anterior phenotype, 83% with the mixed phenotype, and 93% with the cervicogenic-dominant phenotype, with a significant difference across

groups ($\chi^2 = 24.60$, $p < 0.001$). Thus, block response appeared to be better explained by clinical pain phenotype than by NDI or structural cervical findings alone.

Post-treatment neck pain course

Post-treatment neck pain data were available in 591 patients. Complete improvement was reported in 11.3%, 75% reduction in 27.6%, 50% reduction in 29.6%, 25% reduction in 14.0%, no change in 15.7%, and worsening in 1.7%. Overall, 68.5% reported complete improvement or at least 50% reduction. Post-treatment neck pain course was associated with clinically relevant cervical imaging abnormalities, occipital pain, craniocervical pain, selected anterior pain locations, cervical provocation findings, migrainous symptoms, and regular walking. Cervical straightening and cervical radiculopathy were not significantly associated with post-treatment neck pain course.

DISCUSSION

In this large multicentre cohort of 660 patients with cervicogenic headache, headache burden was more closely related to a multidimensional clinical-functional profile than to cervical imaging abnormalities alone [2, 3, 22, 23]. The most consistent pattern combined a higher NDI score, neck-to-head pain radiation, posterior pain topography, cervical provocation findings, migrainous symptom burden, and response to cervical interventions. Although cervical straightening, radiculopathy, and clinically relevant imaging abnormalities were associated with greater headache burden in unadjusted analyses, these structural variables did not independently predict attack severity after adjustment for functional and clinical markers. In contrast, NDI emerged as the strongest independent predictor of headache attack severity. These findings support the concept that cervical pathology becomes clinically meaningful primarily when it translates into neck-related disability, pain radiation, and provocation-sensitive headache [2, 3, 13, 22–24].

Diagnostic uncertainty and why a multidimensional model matters

The present findings are relevant in light of the persistent diagnostic uncertainty surrounding cervicogenic headache [6, 24]. Robust ICHD-based clinical data remain limited, and the clinical phenotype of cervicogenic headache is still insufficiently characterised [6]. Previous diagnostic studies and reviews have similarly emphasised that diagnosis should not rely on any single clinical or radiological marker, but on the integrated assessment of headache history, cervical examination, provocation findings, flexion-rotation testing, pain topography, functional impairment, and, when relevant, response to cervical interventions [14, 17, 18, 20, 25–28]. Our study directly addresses this gap by evaluating these domains together in a large real-world cohort.

This integrated approach may be particularly useful in patients with overlapping migraine-like presentations. Neck pain is common in migraine and tension-type headache, while cervicogenic headache may present with nausea, photophobia, phonophobia, dizziness, or other symptoms

traditionally considered migrainous [3, 9, 10]. Therefore, neither neck pain alone nor migraine-like symptoms alone can reliably define or exclude a cervical pain generator [2, 5, 7]. The present data suggest that the clinically relevant phenotype is better captured by the convergence of functional neck disability, neck-to-head radiation, posterior pain distribution, cervical provocation, migrainous symptom load, and treatment response.

NDI and mediation interpretation

The central role of NDI is one of the most important findings of this study. NDI correlated with headache attack severity, monthly headache frequency, and attack duration, with the strongest association observed for headache intensity. In multivariable models, NDI remained the most robust independent predictor of attack severity, whereas cervical straightening, radiculopathy, and clinically relevant imaging abnormalities were no longer significant. This indicates that NDI is not merely a parallel measure of neck discomfort, but a clinically meaningful marker of headache burden in cervicogenic headache.

The mediation analyses further support this interpretation. In both the cervical radiculopathy → NDI → attack severity model and the cervical straightening → NDI → attack severity model, the indirect effect through NDI was significant, whereas the direct structural effect was not. This pattern suggests that the relationship between cervical pathology and headache severity may be transmitted predominantly through neck-related functional impairment rather than through a direct imaging-defined effect alone [2, 14, 22]. Because of the cross-sectional design, these findings should be interpreted as statistical indirect-pathway models rather than proof of causal mediation. Nevertheless, they provide a clinically plausible explanation for why cervical imaging abnormalities do not always parallel headache severity and why functional assessment may be more informative than structural findings alone [2, 3, 23].

Pain topography and migraine-like overlap

Pain topography provided an additional layer of clinical differentiation. Occipital and craniocervical pain were associated with higher NDI scores, greater headache burden, cervical imaging abnormalities, provocation findings, block application, and recorded headache reduction after block. This posterior pattern is consistent with previous descriptions of cervicogenic headache as a disorder characterised by cervical onset, occipital or suboccipital pain, and trigeminal irradiation [3, 29–31]. In contrast, frontal and periorbital pain were more strongly associated with migrainous accompaniments, including nausea, vomiting, photophobia, phonophobia, osmophobia, allodynia, and vertigo-like symptoms. These findings support a topographic model in which occipital/craniocervical-dominant pain reflects a more cervicogenic phenotype, whereas frontal/periorbital pain represents an anterior migraine-like overlap phenotype [3, 8, 21].

Importantly, migraine-like symptoms were not rare in this cervicogenic headache cohort and should not be interpreted as excluding a cervical contribution. The migrainous symptom score independently predicted attack severity together with NDI, indicating that migraine-like symptom burden contributes to headache intensity within the cervicogenic headache spectrum. This overlap is biologically plausible

because cervical and trigeminal afferents converge within the trigeminocervical complex, allowing cervical nociceptive input to be perceived in trigeminal distributions and to coexist with migraine-like sensory symptoms [4, 9, 10, 32]. Our data therefore support a more nuanced view: posterior pain topography and cervical provocation may identify a cervicogenic-dominant pattern, whereas anterior pain and migrainous accompaniments may define an overlap phenotype rather than an alternative diagnosis [3, 8, 21].

The phenotype analysis also showed that the cervicogenic-dominant group had higher NDI, greater attack severity, and more monthly headache days than the migraine-like anterior phenotype. However, when phenotype and NDI were included in the same regression model, NDI remained strongly associated with attack severity, whereas phenotype was no longer independently significant. This suggests that pain topography is clinically useful for classification, but headache burden severity may be better explained by the degree of neck-related functional impairment than by pain location alone.

Provocation signs and block response

Cervical provocation findings were frequent and consistently associated with headache burden. Headache triggered by neck movement, sustained posture, or neck-related activity; a positive flexion-rotation test; and headache induced by upper cervical or occipital pressure were each associated with higher attack severity, higher monthly headache frequency, longer attack duration, and higher NDI. These findings support the diagnostic relevance of cervical examination in patients with suspected cervicogenic headache. They also align with prior evidence suggesting that musculoskeletal examination, manual cervical assessment, pressure-provoked pain, and the flexion-rotation test may help identify cervicogenic headache, although no single sign is sufficient in isolation [5, 18, 20, 26–28].

The block-response findings provide a clinically important distinction between markers of burden and markers of treatment response. NDI was a strong marker of headache severity, but it did not independently predict response to cervical structure or nerve block. Similarly, NDI did not mediate the relationship between cervical straightening and block response. In contrast, the dominant pain phenotype strongly differentiated block response, with the highest response rate in the cervicogenic-dominant phenotype and the lowest response rate in the migraine-like anterior phenotype. These findings suggest that NDI captures the global functional burden of cervicogenic headache, whereas block response may be more closely related to topographic and provocation-based evidence of a cervical pain generator [1, 2, 5, 22, 33].

This distinction has practical implications. A high NDI score may identify patients with greater disability and headache burden, but it may not be sufficient to select patients for cervical blocks. Conversely, occipital/craniocervical-dominant pain, neck movement provocation, upper cervical pressure sensitivity, and occipital pressure sensitivity may be more treatment-relevant markers when considering cervical interventions. Thus, treatment selection should integrate disability, pain topography, provocation findings, and clinical judgement rather than relying on imaging or NDI alone.

Imaging, screen exposure, and exercise

Our results reinforce the long-standing view that cervicogenic headache cannot be reliably defined by imaging findings alone [2, 23, 34]. Cervical straightening, radiculopathy, and clinically relevant cervical imaging abnormalities were associated with greater headache burden in unadjusted analyses, but they did not independently predict attack severity or block response after adjustment. This finding is consistent with previous concerns that cervical structural abnormalities may be nonspecific and should be interpreted in conjunction with clinical symptoms, examination findings, and functional impairment [2, 13, 23]. Imaging may provide supportive evidence, but it should not be treated as a stand-alone explanation for headache severity.

The findings related to screen exposure should be interpreted cautiously. Daily screen exposure was lower in patients with several cervical burden markers, including radiculopathy, clinically relevant imaging abnormalities, neck-to-head radiating pain, positive flexion-rotation test, and upper cervical pressure-induced headache. This inverse association may reflect behavioural restriction due to pain and disability rather than a protective effect of screen use. In contrast, previous work has linked excessive smart-device use and poor posture to the frequency of cervicogenic headache in student populations [35]. Differences in age, clinical setting, disease severity, and behavioural adaptation may explain these apparently divergent findings.

Exercise findings were exploratory but clinically suggestive. Regular walking showed the most consistent favourable association, with lower NDI scores and a better post-treatment neck pain course. This observation is consistent with the therapeutic literature suggesting that cervical and myofascial interventions may improve pain and disability in cervicogenic headache, although the quality of the evidence varies [2, 36]. These findings should be considered hypothesis-generating and require confirmation in prospective interventional studies.

Strengths and limitations

This study has several strengths. First, it included a large multicentre real-world cohort of patients with cervicogenic headache diagnosed within an ICHD-3-based clinical framework [1]. Second, it evaluated cervicogenic headache using an integrated model that included headache burden, NDI, cervical imaging, neck-to-head radiation, cervical provocation signs, pain topography, migrainous symptom burden, lifestyle factors, and treatment response. Third, the analysis identified NDI as the strongest independent predictor of attack severity and demonstrated that the associations between cervical radiculopathy and cervical straightening and headache severity were largely mediated by NDI. Fourth, the distinction between occipital/craniocervical-dominant and frontal/periorbital-dominant pain phenotypes helped separate a more cervicogenic pattern from a migraine-like overlap phenotype. Finally, block-response analyses were restricted to patients who actually underwent cervical structure or nerve block, reducing the risk of misclassifying untreated patients as non-responders [1, 2, 33].

Several limitations should also be acknowledged. The cross-sectional design prevents causal inference, and the mediation findings should be interpreted as statistical indirect pathways rather than definitive causal mechanisms. Diagnosis was based on real-world clinical assessment according to ICHD-3 criteria, but diagnostic practices may have varied across centres. Imaging availability and interpretation were not fully standardised. Treatment response variables, including block response and post-treatment neck pain course, were observational and may have been influenced by treatment selection, documentation patterns, and follow-up variability. The migrainous symptom score was pragmatic and not externally validated. Finally, because the study did not include a matched migraine control group, the differential diagnostic implications of pain topography and migrainous symptom burden should be interpreted as phenotype-generating findings within cervicogenic headache rather than definitive case-control discriminators.

Conclusion

This study supports a multidimensional clinical-functional model of cervicogenic headache and provides one of the most comprehensive real-world characterisations of ICHD-3-based cervicogenic headache to date [1, 6]. NDI was the most consistent marker of headache burden and the strongest independent predictor of attack severity, whereas cervical imaging abnormalities alone did not independently explain headache severity after adjustment for functional and clinical variables. The relationship between cervical radiculopathy or cervical straightening and headache severity appeared to be mediated largely by NDI, suggesting that structural findings become clinically meaningful mainly when accompanied by functional neck impairment. Occipital/craniocervical pain, neck-to-head radiation, cervical provocation findings, migrainous symptom burden, and block response further refined the clinical phenotype. By integrating imaging, disability, pain topography, provocation signs, migrainous overlap, and treatment response, this study moves beyond an imaging-based concept of cervicogenic headache and provides a clinically applicable framework for differential diagnosis and treatment selection [3, 5, 7, 8, 21, 23].

Declarations

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Author Contribution

All authors reviewed the manuscriptAll authors gathered the patientsDHS, BT, BA ran a statistical analysis

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