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The clinical features, risk factors, and surgical treatment of cervicogenic headache in patients with cervical spine disorders requiring surgery

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STROBE Statement—checklist of items that should be included in reports of observational studies

YOU MUST NOTE THE PAGE NUMBER WHERE EACH ITEM IS REPORTED INSIDE THE BRACKETS [ ]. IF NOT APPLICABLE WRITE N/A

	Item No	Recommendation
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract **[3]**
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found **[3-4]**
Introduction		
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported **[5-6]**
Objectives	3	State specific objectives, including any prespecified hypotheses **[6]**
Methods		
Study design	4	Present key elements of study design early in the paper **[7-8]**
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection **[7]**
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up **[N/A]** *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls **[N/A]** *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants **[7-8]**
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed **[N/A]** *Case-control study*—For matched studies, give matching criteria and the number of controls per case **[N/A]**
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable **[7-8]**
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group **[7-8]**
Bias	9	Describe any efforts to address potential sources of bias **[7-8]**
Study size	10	Explain how the study size was arrived at **[7]**
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why **[8-9]**
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding **[8-9]**
		(*b*) Describe any methods used to examine subgroups and interactions **[8-9]**
		(*c*) Explain how missing data were addressed **[N/A]**
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed **[N/A]** *Case-control study*—If applicable, explain how matching of cases and controls was addressed **[N/A]** *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy **[8-9]**
		(*e*) Describe any sensitivity analyses **[N/A]**

Continued on next page

Results		
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed **[10-11]**
		(b) Give reasons for non-participation at each stage **[N/A]**
		(c) Consider use of a flow diagram **[N/A]**
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders **[10-11]**
		(b) Indicate number of participants with missing data for each variable of interest **[N/A]**
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount) **[11-12]**
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time **[N/A]**
		Case-control study—Report numbers in each exposure category, or summary measures of exposure **[N/A]**
		Cross-sectional study—Report numbers of outcome events or summary measures **[10-12]**
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included **[N/A]**
		(*b*) Report category boundaries when continuous variables were categorized **[N/A]**
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period **[N/A]**
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses **[11]**
Discussion		
Key results	18	Summarise key results with reference to study objectives **[13-16]**
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias **[15]**
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence **[13-16]**
Generalisability	21	Discuss the generalisability (external validity) of the study results **[15]**
Other information		
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based **[N/A]**

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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ABSTRACT

Objective: To clarify the clinical features and risk factors of cervicogenic headache (CEH; as diagnosed according to the International Classification of Headache Disorders-Third Edition beta) in patients with cervical spine disorders requiring surgery.

Background: CEH is caused by cervical spine disorders. The pathogenic mechanism of CEH is hypothesized to involve a convergence of the upper cervical afferents from the C1, C2, and C3 spinal nerves and the trigeminal afferents in the trigeminocervical nucleus of the upper cervical cord. According to this hypothesis, functional convergence of the upper cervical and trigeminal sensory pathways allows the bidirectional (afferent and efferent) referral of pain to the occipital, frontal, temporal, and/or orbital regions. Previous prospective studies have reported an 86–88% prevalence of headache in patients with cervical myelopathy or radiculopathy requiring anterior cervical surgery; however, these studies did not diagnose headache according to the International Classification of Headache Disorders criteria. Therefore, a better understanding of the prevalence rate, clinical features, risk factors, and treatment responsiveness of CEH in patients with cervical spine disorders requiring surgery is necessary.

Methods: We performed a single hospital-based prospective cross-sectional study and enrolled 70 consecutive patients with cervical spine disorders such as cervical spondylotic myelopathy, ossification of the posterior longitudinal ligament, cervical spondylotic radiculopathy, and cervical spondylotic myeloradiculopathy who had been scheduled to undergo anterior cervical fusion or dorsal cervical laminoplasty between June 2014 and December 2015. Headache was diagnosed pre-operatively according to the International Classification of Headache Disorders-Third Edition beta. The Japanese Orthopaedic Association Cervical Myelopathy Evaluation Questionnaire, Neck Disability Index, and a 0–100 mm visual analog scale (VAS) were used to evaluate clinical features, and scores were compared between baseline (i.e. preoperatively) and 3, 6, and 12 months post-surgery.

Results: The prevalence of CEH in our population was 15/70 (21.4%, 95%CI: 11.8% to 31.0%). The main clinical features were dull and tightening/pressing headache sensations in the occipital region. Headache severity was mild (VAS, 32 ± 11 mm) and only 1 patient reported use of an oral analgesic. Compared to patients without CEH, patients with CEH had higher frequencies of neck pain (86.7% vs. 50.9%; $p = 0.017$), cervical range of motion limitation (ROM) (66.7% vs. 38.2%; $p = 0.049$), and higher Neck Disability Index scores (14 vs. 3; $p < 0.001$). Among the different cervical spine disorders, the prevalence of CEH was highest in cervical spondylotic myeloradiculopathy patients (60%), being ≤ 20% for all other disorders. Surgical treatments including cervical laminoplasty to relieve abnormal pressure on the spinal cord via a posterior approach, were associated with initial improvements in headache VAS that slightly diminished by 12 months post-surgery ($P < 0.001$).

Conclusions: We report a lower prevalence of CEH in patients with cervical spinal disorders requiring surgery than that reported previously. The main clinical features of CEH were mild, dull, and tightening/pressing headache sensations in the occipital region. Potential risk factors for CEH included neck pain, limited cervical ROM, high Neck Disability Index score, and a diagnosis of cervical spondylotic myeloradiculopathy. The further accumulation of patients in a multi-institutional study may be required in order to discuss the diagnostic criteria and pathophysiology of this condition.

INTRODUCTION

Cervicogenic headache (CEH) is caused by cervical spine disorders that were first identified by Sjaastad and colleagues in 1983[1]. CEH is non-throbbing with chronic pain, and can be provoked by neck movement and awkward head position[1,2]. It has been defined typically as a unilateral headache without sideshift, although patients with bilateral headaches may be considered as having CEH[1,2]. The prevalence of CEH in the general population has been reported to be 0.17–4.1%[3, 4]. Studies have characterized CEH as presenting with moderate-to-severe intensity in the occipital, frontal, temporal, and/or orbital regions[2, 4]. The clinical features of CEH are similar to those of tension-type headache and migraine, which make it difficult to distinguish CEH in clinical settings[2,4].

The pathogenic mechanism of CEH is hypothesized to involve convergence of the upper cervical afferents from the C1, C2, and C3 spinal nerves and trigeminal afferents in the trigeminocervical nucleus of the upper cervical cord[5, 6]. According to this hypothesis, functional convergence of the upper cervical and trigeminal sensory pathways allows the bidirectional (afferent and efferent) referral of pain to the occipital, frontal, temporal, and/or orbital regions[6]. However, several previous studies have reported that lower cervical spine diseases (below C4) can also cause headache[7-9]; to this end, it is not clear whether the middle-lower cervical roots also project into the trigeminocervical nucleus in humans.

The exact prevalence of and risk factors for CEH are poorly understood. Two previous prospective studies reported that 86–88% of patients with cervical myelopathy or radiculopathy requiring anterior cervical surgery presented with headache[10, 11]; however, these studies did not diagnose headache according to the International Classification of Headache Disorders 2nd edition (ICHD-2) criteria[12]. In 2013, publication of the ICHD-3beta updated the definition of CEH to include cervical spondylosis as an underlying cause of CEH[13]. Importantly, no study to date has reported the prevalence rate of or risk factors for CEH as diagnosed according to ICHD-3beta in patients with cervical spine disorders requiring surgery; additionally, it is unknown which types of cervical spine disorders most commonly cause CEH.

It is also important to note that a paucity of studies address the treatment of CEH. Treatment of CEH requires a multimodal approach that incorporates pharmacological and non-pharmacological interventions, the use of anesthetic blocks, and surgery[5, 6]. With regard to the surgical treatment of CEH, prospective studies of headache relief after anterior cervical surgery have been reported[10, 11]; yet, to the best of our knowledge, no prospective studies have addressed the therapeutic utility of dorsal cervical laminoplasty, which is the procedure of choice for elderly patients, patients with a narrow (≤ 13 mm diameter) anterior-posterior spinal canal, and patients with 3 or more cervical spinal lesions[14].

Therefore, the present study was therefore performed to clarify: 1) the clinical features of CEH as diagnosed according to the ICHD-3beta criteria in patients with cervical spine disorders requiring surgery; and 2) to identify the risk factors for CEH. We also investigate the therapeutic efficacy of surgical treatment for CEH at up to 12 months post-surgery.

METHODS

Participants

This study was approved by the Kameda-Daiichi Hospital Ethics Committee and written informed consent was obtained from all subjects prior to participation in the study. Based on previous experience, we prospectively enrolled 70 consecutive patients with cervical spine disorders such as cervical spondylotic myelopathy (CSM), ossification of the posterior longitudinal ligament (OPLL), cervical spondylotic radiculopathy (CSR), and cervical spondylotic myeloradiculopathy (CSMR) that were scheduled for cervical spine surgery at the Niigata Spine Surgery Center in Japan between June 2014 and December 2015. Patients were excluded if they were pregnant, had severe dementia, or had previous cervical spine surgery.

Study Design

All enrolled patients completed basic cervical radiography, magnetic resonance imaging (MRI), and CT myelography. The diagnosis of a cervical spine disorder, identification of the spinal lesion level(s), and identification of intramedullary high signal intensity on T2-weighted MRI was performed. The diagnosis of headache was performed pre-operatively by a single headache specialist according to the ICHD-3beta criteria. All patients were asked to attend a structured interview for the collection of clinical and demographic data as well as administration of the Japanese Orthopaedic Association Cervical Myelopathy Evaluation Questionnaire (JOACMEQ) and Neck Disability Index

(NDI). The JOACMEQ has been used in Japan as a patient-reported outcome measure to evaluate cervical, upper limb, lower limb, and bladder functions[15]. The NDI is another widely used questionnaire that evaluates disability due to neck pain and includes a headache severity index (headache NDI) graded on a scale from 0 to 5 (grade 0, no headache; grade 1, infrequent slight headaches; grade 2, infrequent moderate headaches; grade 3, frequent moderate headaches; grade 4, frequent severe headaches; and grade 5, persistent extreme headache)[16]. Higher NDI scores indicate greater degrees of self-related disability.

Headache and neck pain severities were evaluated preoperatively and at 3, 6, and 12 months after surgery using the headache NDI and a 0–100 mm visual analogue scale (VAS). The location and clinical features of headache, limited cervical range of motion (ROM), and prior oral analgesic use were also assessed. Limited cervical ROM referred to any limitations in flexion, extension, lateral flexion, or rotation. A definitive diagnosis of CEH was only confirmed after the observation of headache relief at either 3 or 6 months post-surgery on both the headache VAS and headache NDI.

Statistical analysis

Mann-Whitney rank sum tests were used to assess significant differences in NDI and JOACMEQ scores. Student's t-tests were used to compare age and quality of life (QOL) JOACMEQ subscores. Chi-squared tests were used to assess significant differences in neck pain VAS scores and limited cervical ROM. Finally, Fisher's exact tests were used for comparisons involving all other variables. Statistical comparisons were conducted using a repeated-measures one-way analysis of variance (ANOVA) or the Friedman non-parametric repeated-measures ANOVA followed by Dunnett's post hoc comparisons where appropriate to evaluate differences in headache VAS and NDI scores at pre-operative baseline and 3, 6, and 12 months after surgery. Two-tailed p values of < 0.05 were considered to be significant. SigmaStat version 12.0 (SYSTAT Software Inc, USA) was used for statistical analyses.

RESULTS

The prevalence of CEH and its clinical features

Table 1 summarizes the clinical and demographic features of the 70 patients included in the present study. There were no cases of missing data and no patients were lost to follow-up. The mean patient age was 65 years (range, 34–85 years) and 46 patients (65.7%) were male. With regard to causative cervical spine disorders, CSM was the most popular disorder, affecting 53 patients (75.7%); 7 patients (10%) had OPLL, 5 patients (7.1%) had CSR, and 5 patients (7.1%) had CSMR. Cervical MRI findings indicated that cervical lesions were upper cervical (C1/2, 2/3) in 1 patient and middle/lower cervical (C3/4, 4/5, 5/6, 6/7) in the remaining 69 patients. Intramedullary high signal intensity on T2-weighted MRI scans was detected in 46 patients (65.7%, 95%CI: 54.6% to 76.8%).

A total of 23 patients (32.9%) reported headache; of these, 15 were diagnosed with CEH and the prevalence of CEH in the included population was calculated to be 21.4% (95%CI: 11.8% to 31.0%). All patients with CEH had middle/lower cervical lesions. Of the 8 patients with non-CEH headache, 6 (8.6%) were diagnosed with migraine and 2 (2.9%) were diagnosed with migraine and tension-type headache. Of the 15 patients with CEH, 2 patients were diagnosed with both CEH and tension-type headache, and 1 patient was diagnosed with both CEH and headache due to psychiatric disease. In patients with CEH, the mean headache VAS rating was 30 ± 14 mm, and 12 patients (80%) reported headache NDI scores of grade 1 or grade 2. Only 1 patient required an analgesic for headache management.

The location of CEH was occipital in 12 patients (80%), occipital with temporal involvement in 1 patient (6.7%), occipital with frontal involvement in 1 patient (6.7%), and temporal in 1 patient (6.7%). All patients with CEH reported dull and tightening/pressing headache features, although 3 patients reported additional shooting features.

Risk factors for CEH

Clinical features were compared between patients with and without CEH (Table 2). No significant between-group differences were observed in age, sex, or frequency of intramedullary high intensity spinal lesion on T2-weighted MRI. Moreover, there were no significant between-group differences in neurological function of the neck as evaluated by the JOACMEQ. Significant differences were found for 3 variables; the CEH group showed a higher incidence of neck pain complications (86.7% vs. 50.9%; $p = 0.017$), a higher incidence of limited cervical ROM (66.7% vs. 38.2%; $p = 0.049$), and a higher median NDI score (14 vs. 3; $p < 0.001$) compared to the non-CEH group. CEH was most common among patients with CSMR, affecting 60% of CSMR patients; CEH was otherwise diagnosed in 20% of CSR patients, 18.9% of CSM patients, and 14.3% of OPLL patients.

Therapeutic effects of cervical spine surgery on CEH

Among 15 patients diagnosed with CEH, dorsal cervical laminectomy was performed in 12 patients and anterior cervical fusion was performed in 3 patients. Compared to the pre-operative baseline, headache VAS ratings were significantly reduced at 3, 6, and 12 months post-surgery ($p < 0.001$, $F = 9.728$; pre = 30±14; 3 months = 9±15; 6 months = 11±18; 12 months = 12±15) (Figure 1). That is, 13, 12, 12 out of 15 patients improved their CEH at 3, 6, and 12 months post-surgery, respectively. NDI scores were not significantly reduced compared to the baseline and at 3 months post-surgery, but were significantly reduced at 6 and 12 months post-surgery ($p = 0.030$; pre = 13 (median); 3 months = 9; 6 months = 8, 12 months = 8) (Figure 2). With regard to headache NDI scores, 66.7% of patients reported a grade 0 rating at 3 months post-surgery, but this statistic decreased to 60% at 6 months post-surgery and 40% at 12 months post-surgery (Figure 3). Alternatively, compared to the pre-operative baseline, neck pain VAS ratings were significantly reduced at 3, 6, and 12 months post-surgery ($p < 0.001$, $F = 6.694$; pre = 36±20; 3 months = 23±17; 6 months = 19±18; 12 months = 20±20).

Finally, we investigated the influence of surgery type of on therapeutic efficacy. In the 12 patients with CEH who underwent dorsal cervical laminoplasty, headache VAS ratings were significantly reduced at 3, 6, and 12 months post-surgery ($p < 0.001$, $F = 10.278$; pre = 29±14; 3 months = 10±17; 6 months = 7±14; 12 months = 8±10). NDI scores were not significantly reduced compared to the baseline at 3 and 12 months post-surgery, but were significantly reduced at 6 months post-surgery ($p = 0.021$; pre = 13 (median); 3 months = 9; 6 months, 7, 12 months = 8). With regard to headache NDI scores, 66.7% of patients reported a grade 0 rating at 3 and 6 months post-surgery, but this statistic decreased to 41.7% at 12 months post-surgery.

DISCUSSION

Here, we present novel findings regarding the prevalence and clinical nature of CEH. First, we found that the prevalence of CEH in patients with serious surgical spine lesions requiring surgery as diagnosed according to the ICHD-3beta criteria was low (21%) compared to that reported previously; Riina et al. reported CEH in 86% of 1003 patients and Schrot et al. reported CEH in 88% of 260 patients who underwent single-level anterior cervical surgery for the treatment of CSM or CSR accompanied by headache[10, 11]. The low prevalence in the present study might be attributable to differences in ethnicity (i.e., the use of a Japanese population) or the diagnostic criteria used, especially considering that headache was not diagnosed according to any ICHD criteria in the above-mentioned reports. Although ICHD-3 beta criteria has changed better than the previous criteria, the criteria are fairly nonspecific to diagnose CEH[17]. Because the diagnostic factors are too few compared to The Cervicogenic Headache International Study Groups (CHISG) criteria[2, 17], this might affect the result of low prevalence of CEH.

Second, we investigated the clinical features of CEH, and found that patients primarily reported dull and tightening/pressing headache sensations in the occipital region. According to headache VAS and NDI scores, severity of CEH was mild, and accordingly only 1 patient reported the use of an oral analgesic to manage headache pain. As CEH characteristics in our population are different from typical CEH, this condition did not require cervical spine surgery against headache itself. Furthermore, all patients with CEH had cervical lesions below C4 and reported symptom improvements after surgery. These findings suggest that lesions below C4 can be responsible for CEH in some patients. In agreement with this finding, several case reports and small case series have reported headache relief following surgical treatment of middle-lower cervical spine disorders[7-9, 18, 19].

Given that there is no known neuroanatomical link between middle-lower cervical afferents and the trigeminocervical nucleus[6], the mechanism by which lower cervical spine disorders cause headache remains unknown[7-9]. We can propose several possibilities; first, while most pain stimulation entering through the nerve root passes through the dorsal horn of the spinal cord and ascends via the contralateral anterior spinothalamic tract, some nociceptive input ascends via the ipsilateral spinocervicothalamic tract[20]. It is possible that the spinocervicothalamic tract and trigeminospinal complex communicate through anastomosis[18, 19]. Another possible mechanism is that excessive increases in ROM of the upper cervical spine occurring to compensate for kinesthetic impairment in the lower cervical spine cause overstimulation of the trigeminocervical nucleus[21], thereby triggering a CEH. Future research is required to determine the validity of these hypotheses. Further discussion is required to determine whether there is a need to expand the clinical concept of CEH and add the ICHD appendix diagnosis for headaches attributed to lower cervical spine disorders.

Third, we identified potential risk factors for CEH including neck pain, limited cervical ROM, high NDI score, and a diagnosis of CSMR. To confirm this results, the further accumulation of patients with a prospective multi-institutional study may be required.

Finally, this prospective study was the first of its kind to demonstrate that dorsal cervical laminoplasty might be effective for the treatment of CEH associated with multi-level cervical spine lesions; the 12 patients with CEH who had multi-segmental lesions reported headache relief after cervical laminoplasty. To date, one retrospective case series has reported improvements in CEH after dorsal cervical laminoplasty; however, this effect was observed in only 6 patients[22]. The ability of dorsal cervical laminoplasty to improve headache severity indices in the present study suggests that the dura mater of the spinal cord plays an important role in CEH, given that dorsal cervical laminectomy treats irritation of the dura mater[22]. It should be noted, however, that a large number of patients were candidates for dorsal cervical laminoplasty in the present study. This may be explained by the fact that the spinal canal in individuals of Japanese descent is narrower than that in individuals of European descent[23]. Moreover, given increasing elderly populations in industrialized countries, the number of dorsal cervical laminoplasty is expected to increase in the near future. From this perspective, this study provides meaningful information about the selection of surgical treatment for CEH. However, their CEH was moderate, and this condition did not require cervical spine surgeries against headache itself. Future studies will be needed to determine the efficacy of symptomatic treatment against this condition. In addition, we need to recognize that the therapeutic effect of surgery appeared to gradually diminish over time; therapeutic effect on headache was most favorable at 3 and 6 months post-surgery but tended to diminish by 12 months post-surgery. This may be explained by bed rest due to post-operative wound pain that lowered headache propensity in the early post-operative period.

The present study had some limitations. First, the generalizability of our study is limited by the fact that we only included Japanese adults recruited from a single center. Second, the population size itself was relatively small; future larger-scale studies are required to confirm our findings. Third, the convenience sampling limits generalization of the study participants as both patients at the clinic or the general population. Forth, the non-randomized nature of the treatment assignment precludes any causal statements about the potential benefits of the treatments applied to the patients. A better understanding of the mechanisms of CEH and future studies to validate our findings in other populations will better elucidate the clinical characteristics and optimal treatment approaches for CEH.

In conclusion, the present study demonstrates a relatively low prevalence of CEH in Japanese patients with serious cervical spine disorders requiring surgery. Our study also challenges previous clinical characterizations of CEH, as we found that the severity of CEH was mild in nearly all patients with mid/lower cervical lesions. Lastly, our data suggests that neck pain, limited cervical ROM, high NDI score, and a diagnosis of CSMR are risk factors for CEH, and that surgical treatment with dorsal cervical laminoplasty is an effective therapeutic option that warrants additional study. The further accumulation of patients in a multi-institutional study may be required in order to discuss the diagnostic criteria and pathophysiology of this condition.

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FIGURE LEGENDS

Table 1. Clinical and demographic characteristics.

Abbreviations: CSM, cervical spondylotic myelopathy; CSMR, cervical spondylotic myeloradiculopathy; CSR, cervical spondylotic radiculopathy; NDI, Neck Disability Index; OPLL, ossification of the posterior longitudinal ligament; ROM, range of motion.

Table 2. Prevalence of cervicogenic headache and its clinical presentation.

CSM, cervical spondylotic myelopathy; CSMR, cervical spondylotic myeloradiculopathy; CSR, cervical spondylotic radiculopathy; JOACMEQ, Japanese Orthopaedic Association Cervical Myelopathy Evaluation Questionnaire; MRI, magnetic resonance imaging; NDI, neck disability index; OPLL, ossification of the posterior longitudinal ligament; ROM, range of motion; QOL, quality of life.

Figure 1. Post-operative changes in headache visual analog scale (VAS) ratings. Average values and their standard deviations are shown. ** indicates $p < 0.01$.

Figure 2. Post-operative changes in headache Neck Disability Index scores. The boxes have lines at

the lower quartile, median and upper quartile values and the notches in boxes graphically show the 95% confidence interval. * indicates $p < 0.05$.

Figure 3. Detailed view of post-operative changes in headache Neck Disability Index scores. N0-N5 indicated headache severity index graded on a scale from 0 to 5 (grade 0, no headache; grade 1, infrequent slight headaches; grade 2, infrequent moderate headaches; grade 3, frequent moderate headaches; grade 4, frequent severe headaches; and grade 5, persistent extreme headache). Higher scores indicate greater degrees of self-related disability.

TABLES

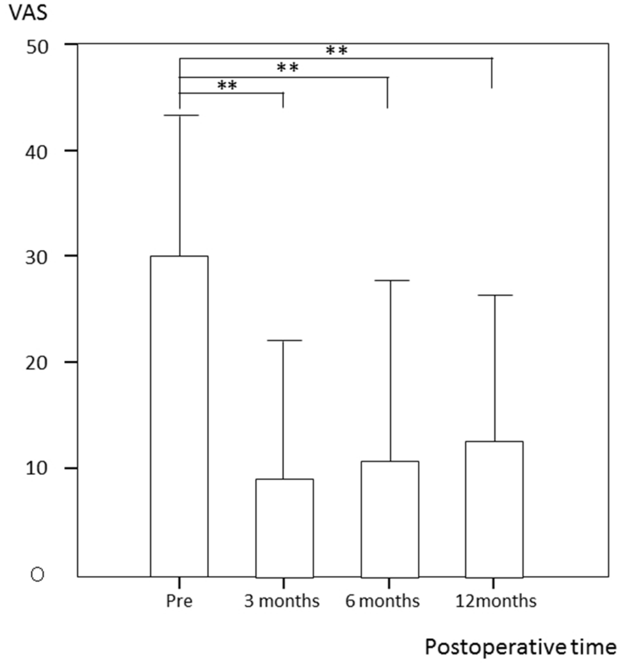
Table 1. Clinical and demographic characteristics.

Age (range)	65 ± 12 (34–85)
Sex (male:female)	46:24
Cervical disease	CSM 53/70 (75.7%) OPLL 7/70 (10%) CSR 5/70 (7.1%) CSMR 5/70 (7.1%)
Surgical treatment	Posterior cervical laminoplasty (multi-level) 55/70 (78.6%) Anterior cervical fusion (1 level) 10/70 (14.3%) Anterior cervical fusion (2 levels) 5/70 (7.1%)
Headache	23/70 (33%) Cervicogenic headache 15/70 (21.4%) Migraine 6/70 (8.6%) Migraine + tension-type headache 2/70 (2.9%)
Neck pain	41/70 (58.6%)
Limitation of neck ROM	32/70 (45.7%)
Headache NDI score	1 (I have slight headaches which come infrequently) 9/15 (60%) 2 (I have moderate headaches which come infrequently) 3/15 (20%) 3 (I have moderate headaches which come frequently) 2/15 (13.3%) 4 (I have severe headaches which come frequently) 0/15 (0%) 5 (I have headaches almost all the time) 1/15 (6.7%)

Table 2. Prevalence of cervicogenic headache and its clinical presentation.

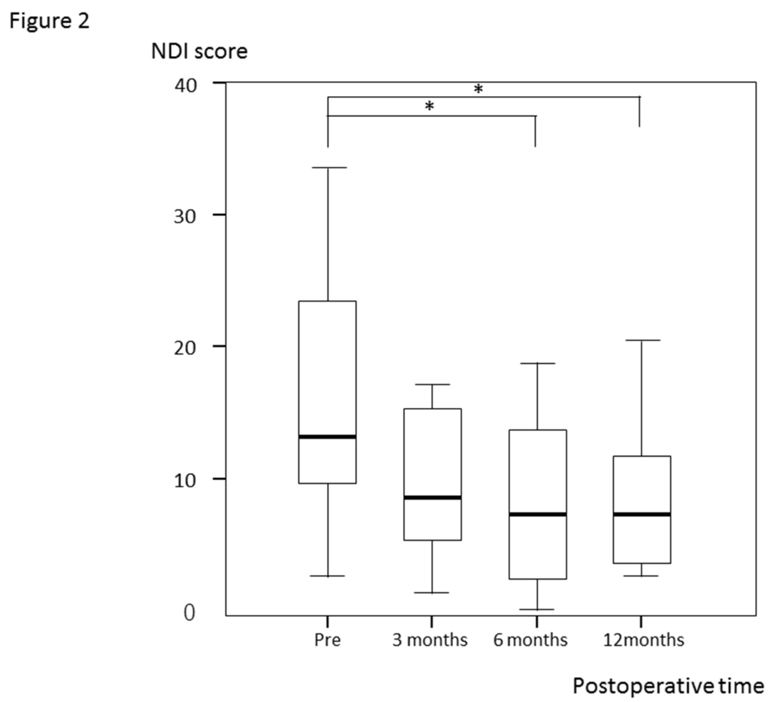
		Patients with cervicogenic headache	Patients without cervicogenic headache	P-value
Number of patients	70	15	55	
Age (years)		66 ± 12	64 ± 11	0.540
Sex (male:female)		10:5	36:19	0.930
Cervical disease	CSM OPLL CSR CSMR	10/53 (18.9%) 1/7 (14.3%) 1/5 (20%) 3/5 (60%)	43/53 (81.1%) 6/7 (85.7%) 4/5 (80%) 2/5 (40%)	
Neck pain		13/15 (86.7%)	28/55 (50.9%)	0.017
NDI (median)		14	3	< 0.001
JOACMEQ	Cervical Upper Lower Bladder QOL	70 95 73 88 41 ± 18	85 95 82 94 51 ± 21	0.239 0.946 0.388 0.525 0.119
Limited cervical ROM		10/15 (66.7%)	21/55 (38.2%)	0.049
Intramedullary high signal intensity on T2-weighted MRI		8/15 (53.3%)	38/55 (69.1%)	0.358
Surgery	Cervical laminoplasty Anterior cervical fusion	12 3	43 12	

Figure 1



Post-operative changes in headache visual analog scale (VAS) ratings. Average values and their standard deviations are shown. ** indicates $p < 0.01$.

254x190mm (96 x 96 DPI)



Post-operative changes in headache Neck Disability Index scores. The boxes have lines at the lower quartile, median and upper quartile values and the notches in boxes graphically show the 95% confidence interval. * indicates $p < 0.05$.

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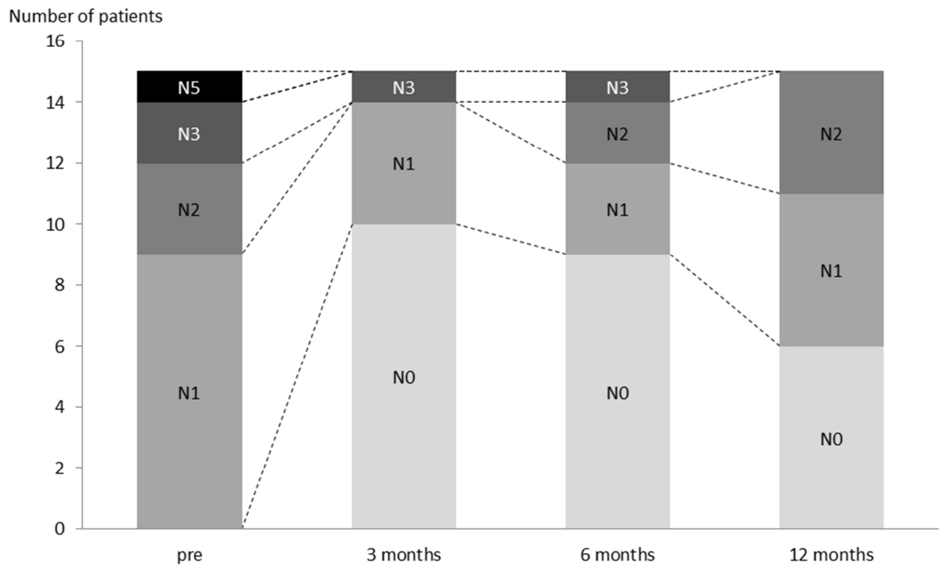
Figure 3



Detailed view of post-operative changes in headache Neck Disability Index scores.
N0-N5 indicated headache severity index graded on a scale from 0 to 5 (grade 0, no headache; grade 1, infrequent slight headaches; grade 2, infrequent moderate headaches; grade 3, frequent moderate headaches; grade 4, frequent severe headaches; and grade 5, persistent extreme headache). Higher scores indicate greater degrees of self-related disability.

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