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Association Between Trigeminal Neuralgia and Degenerative Cervical Myelopathy: A Cross-Sectional Study Using US Data

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Association between trigeminal neuralgia and degenerative cervical myelopathy: A cross-sectional study using US data

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Abstract

Background: Limited research has suggested that trigeminal neuralgia (TN), an often-idiopathic pain disorder affecting the face and head, may arise from compression of the cervical spinal cord due to involvement of the spinal trigeminal tract. We hypothesized that adults with TN would have a greater likelihood of concurrent degenerative cervical myelopathy (DCM) compared to matched adults without TN.

Methods: We retrieved de-identified data from a US network (TriNetX, Inc.) including medical records of >113 million patients, with a query date of October 1, 2023, and data spanning the previous 20 years. We created two groups of adults (aged ≥18 years): Those with (1) TN and (2) No TN, excluding individuals with predisposing conditions for TN (e.g., multiple sclerosis, ophthalmic and oral/maxillofacial surgery) and propensity matched for confounders (e.g., age, sex, body mass index, diabetes mellitus, hypertensive diseases, migraine, osteoporosis). We calculated the point prevalence and odds ratio (OR) of DCM with 95% confidence intervals (CI).

Results: After matching there were 37,163 patients per group. The mean point prevalence of DCM in the TN group was 0.55% (95% CI: 0.47–0.63%) compared with 0.04% (0.03–0.06%) in the no TN group, yielding an OR of 12.94 (95% CI: 7.78–21.53; $p < 0.0001$).

Conclusions: Adults with TN had more than 12 times greater odds of concurrent DCM compared to those without TN. These findings suggest that DCM may be a risk factor for TN, yet causality should be further examined using case–control or cohort designs.

KEYWORDS
cranial nerve disease, facial neuralgia, myelopathy, spinal cord disease, trigeminal nerve

1 | INTRODUCTION

The trigeminal nerve provides sensory innervation to the face, nose, mouth, scalp, eyes, and ears. According to the International Classification of Headache Disorders (ICHD), trigeminal neuralgia (TN) is a paroxysm of facial pain in one or more divisions of the trigeminal nerve.[1] TN typically occurs in middle to older aged adults and may be exacerbated when stimuli are applied to regions innervated by the trigeminal pathway.[2] This condition has a lifetime prevalence of 0.03% to 0.3%.[3] The prevailing hypothesis regarding the etiology of TN is a

neurovascular compression of the intracranial portion of the trigeminal nerve.[4] However, studies have shown that neurovascular compression is absent in 4% to 89% of patients,[4] prompting the question of whether there may be other unrecognized risk factors for TN.

Given the uncertainties regarding the source of TN, there has been an increasing focus on pathology of the spinal trigeminal nucleus, also called the trigeminal tract, as a potential etiology.[5,6] Several recent case reports have highlighted a potential link between degenerative conditions affecting the cervical spine and development of TN.[7–11] Some of these cases reported that patients undergoing cervical spine surgery for degenerative cervical myelopathy (DCM), a relatively common condition involving spinal cord compression, experienced remission of TN postoperatively.[8–11]

To our knowledge, the potential association between TN and DCM has only been explored by one study larger than a case report or series.[5] This recent case–control study, which included 20 individuals, found a significant association between cervical disc displacement causing indentation on the cervical spinal cord superior to the C4 vertebra and symptoms of TN.[5] While this preliminary finding from a relatively small sample remains unconfirmed, neuroanatomical evidence suggests that a link between cervical cord compression and TN is plausible. The spinal trigeminal tract, which contributes to the sensory components of the trigeminal nerve, extends as far caudal as the fourth cervical segment with the cervical component termed the pars caudalis.[12]

Considering the limited research suggesting that compression of the cervical spinal cord may result in TN, we conducted a cross-sectional study that examined the potential association between these conditions. We hypothesized that adults with DCM would have a greater likelihood of TN compared to matched controls without TN.

2 | MATERIALS AND METHODS

2.1 | Study design

This study implemented a cross-sectional design, following an a priori protocol.[13] With a query date of October 1, 2023, we included data spanning the prior 20 years to maximize sample size. Study reporting follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline.[14] The University Hospitals Institutional Review Board (IRB; Cleveland, OH, USA) considers the current study methods, which use de-identified data from TriNetX (Cambridge, MA, USA) obtained through the hospital's Clinical Research Center Honest Broker, to meet criteria for "not human subjects research" and not require IRB review or consent to participate.

2.2 | Setting and data source

We used data from a US research network (TriNetX), which aggregates de-identified medical records from over 113 million patients attending 79 healthcare institutions.[15] Users can query the dataset using the standardized nomenclature such as International Classification of Diseases, 10th Edition (ICD-10) codes. We also used natural language processing within TriNetX (Averbis, Freiburg im Breisgau, DE) to strengthen our selection criteria and ascertainment of cases of TN. This processing service includes mechanisms to account for intent and negation when screening free text appearing in medical notes and imaging reports.

2.3 | Participants

We included adults at least age 18 who had a recorded healthcare visit and blood pressure measurement available in the dataset anywhere from 1 month to 2 years prior, a requirement intended to maximize completeness of data in both groups. We excluded patients with specific known causes of TN or conditions that mimic TN. These were multiple sclerosis (ICD-10: G35) or prior cerebral infarction (ICD-10; 163)[16] appearing on the index date or at any time in preceding records; ophthalmic, oral, maxillofacial surgery (via Current Procedural Terminology codes 1009727 and D7111-D7999, respectively), appearing on the index date or within the preceding 6 months.

We divided patients into two groups based on the presence or absence of TN (ICD-10: G50.0). Patients with TN were included at the index date of TN diagnosis while those without TN (No TN) were included at the first instance that our other selection criteria were met (i.e., healthcare visit, preceding blood pressure measurement).

2.4 | Variables

We used propensity score matching to reduce selection bias, matching key confounding variables present within a year preceding index date of inclusion and having a known association with TN, including: body mass index,[17] demographics (age, sex), diabetes mellitus (ICD-10: E08-E13),[18] hypertensive diseases (ICD-10: I10-I16), and antihypertensive medications (Anatomical Therapeutic Chemical classification [ATC] C02]),[19] migraine (ICD-10: G43),[20] and osteoporosis (ICD-10: M81).[21]

We queried for any diagnosis of DCM at the time of index date appearing in the patients' medical records (ICD-10: M48.02, M47.12, M50.0).[22] These diagnoses were used as a single composite outcome, such that patients with any one of these codes were considered to have DCM.

2.5 | Statistical methods

Statistical analysis was conducted within the TriNetX software, and baseline group characteristics were compared using an independent samples *t*-test or Pearson chi-squared test, using standardized mean difference (SMD) to evaluate residual imbalance (<0.1 indicating

successful balance). To provide markers of data completeness we described the mean number of data points per group and calculated the percentages of unknown demographic variables. Propensity scores were calculated using logistic regression, and matching was conducted with a 1:1 greedy nearest-neighbor algorithm with a caliper of 0.1 pooled standard deviations. We graphed the propensity score densities per group before and after matching as a visual indicator of covariate balance. Odds ratios were calculated by dividing odds of DCM in the TN group by odds in the no TN group with statistical significance evaluated at $p < 0.05$. No imputations were made for missing data points. We calculated 95% confidence intervals for mean point prevalence using R and R studio (version 2022.12.0 Build 353, Posit Software, Public Benefit Corporation) and created prevalence plot and propensity score graphs via this interface using the ggplot2 package.

We calculated a total sample size of 24,024 using G*Power (version 3.1.9.7, Universitat Kiel, DE) via the z-test function to power a comparison of difference in proportion (0.06% and 0.24%), with an alpha error of 0.05, power of 0.95, and allocation ratio of one.

3 | RESULTS

Included patients were identified from a total of 79 healthcare organizations within the TriNetX database, which came from 57 unique healthcare organizations for the TN group and 10 for the No TN group. Before propensity matching there were 42,529 patients in the TN group and 304,102 in the No TN group. After matching there were 37,163 patients per group. Prior to matching, a greater proportion of patients in the TN group had hypertensive diseases, diabetes mellitus, migraine, age-related osteoporosis, and use of antihypertensive medications compared with the no TN group (SMD > 0.1; Table 1). After matching, most variables were optimally balanced (SMD < 0.1) while the similarity in age was adequate (SMD = 0.184; Table 1).

3.1 | Descriptive data

Both groups had a high mean number of data points per patient (TN: 3726, No TN: 3859). After matching, the frequency of "unknown" variables was identical between groups as age and sex were each present in 100% of patients. A propensity score density graph suggested that groups were well balanced after matching (Figure 1). Together, these findings suggested that there were no relevant between-group differences regarding data density, completeness, or covariate balance.

3.2 | Key results

DCM was more prevalent in the TN group both before and after propensity matching (Table 2). For our primary outcome, after matching, the mean point prevalence of DCM in the TN group was 0.55% (95% CI: 0.47–0.63%) compared with 0.04% (0.03–0.06%) in the no TN group (Figure 2), yielding an OR of 12.94 (95% CI: 7.78–21.53; $p < 0.0001$). Confidence intervals for prevalence were narrow, which was expected given the large group sizes.

4 | DISCUSSION

Consistent with our hypothesis, the present study identified a significant increase in odds of DCM in adults with TN compared to matched adults with No TN in a large US population. To our knowledge, this is the largest study to examine the association between TN and DCM. Our results have clinical implications which may benefit diagnosis and treatment of both conditions.

Our findings suggest that DCM may be an under-recognized etiology of TN in certain patients, especially given the often-idiopathic nature of TN. These results align with and build upon prior case reports and a small case–control study that first noted a potential

TABLE 1 Baseline characteristics before and after propensity matching.

	Before matching			After matching[a]		
Variable	TN $N = 42,529$	No TN $N = 304,102$	SMD	TN $N = 37,163$	No TN $N = 37,163$	SMD
Age at index	56.4 (15.8)	44.3 (14.6)	0.798	55.1 (15.7)	52.4 (13.1)	0.184
Age (min-max)	(18–90)	(18–72)	0.180	(18–90)	(18–72)	0.057
Female	29,987 (71%)	182,902 (62%)	0.217	26,173 (70%)	27,124 (73%)	0.056
Male	11,627 (27%)	110,243 (38%)	0.465	10,960 (29%)	10,031 (27%)	0.002
Hypertensive diseases	16,275 (38%)	52,923 (18%)	0.270	13,076 (35%)	13,118 (35%)	0.062
Diabetes mellitus	6652 (16%)	21,076 (7%)	0.464	5195 (14%)	6018 (16%)	0.103
Migraine	6599 (16%)	7607 (3%)	0.211	3958 (11%)	5221 (14%)	0.031
Age-related osteoporosis	2066 (5%)	3694 (1%)	0.265	1389 (4%)	1614 (4%)	0.106
Antihypertensives	3004 (7%)	5019 (2%)	0.061	1972 (5%)	2953 (8%)	0.053
Body mass index	29.4 (6.8)	29.0 (6.5)	0.587	29.5 (6.9)	29.5 (6.9)	0.086

Abbreviations: SMD, Standardized mean difference; TN, trigeminal neuralgia.

[a]Used for our primary outcome.

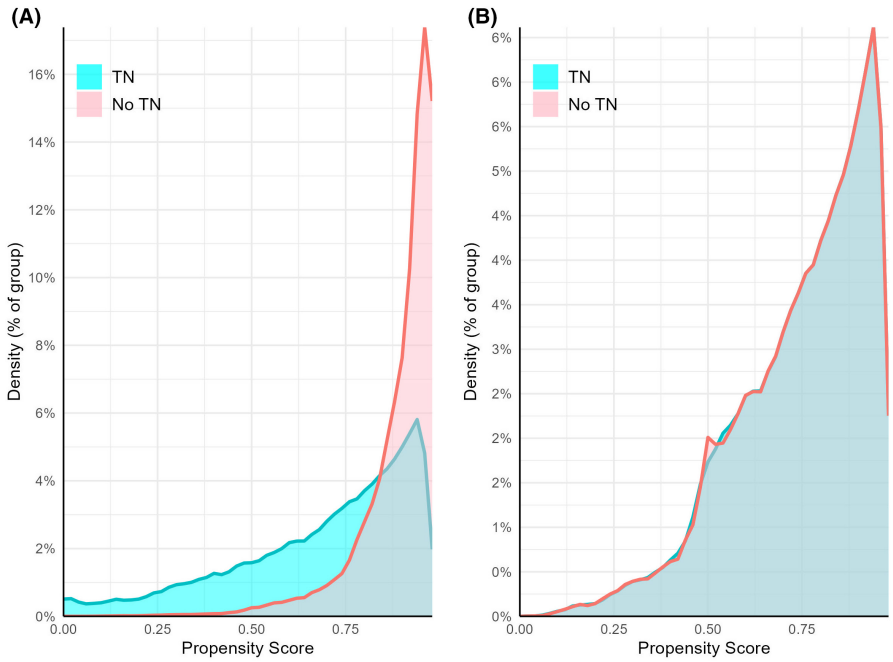

FIGURE 1 Propensity score densities per group before (A) and after (B) matching. The trigeminal neuralgia group (TN) group is cyan while the No TN group is pink. After matching, the densities closely overlapped, suggesting that there were no meaningful between-group differences in propensity score.

TABLE 2 Key results before and after propensity score matching.

	Before matching		After matching[a]	
	TN $n=42,529$	No TN $n=304,102$	TN $n=37,163$	No TN $n=37,163$
DCM No. (%)	264 (0.62)	116 (0.04)	206 (0.55)	16 (0.04)
DCM % CI	0.53–0.71		0.47–0.63	0.03–0.06
OR (95% CI)	15.78 (12.68–19.64; $p<0.0001$)	(reference)	12.94 (7.78–21.53; $p<0.0001$)	(reference)

Abbreviations: %, percentage of patients with DCM; 95% CI, 95% confidence intervals; DCM, degenerative cervical myelopathy; No., number; OR, odds ratio.

[a]Used for our primary outcome.

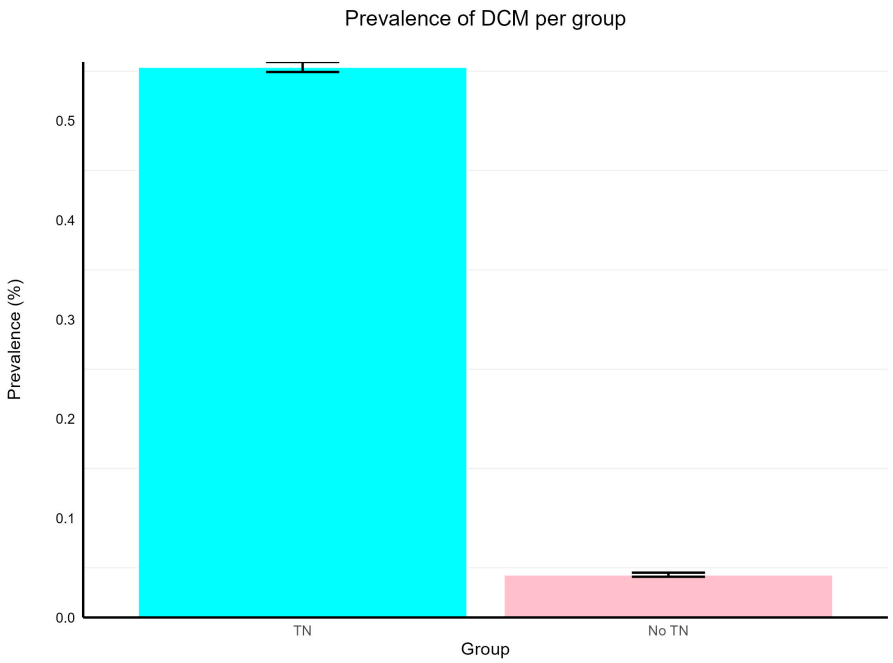

FIGURE 2 Mean point prevalence of degenerative cervical myelopathy (DCM) per group after propensity matching. The trigeminal neuralgia (TN) group is indicated in cyan, while the no trigeminal neuralgia (No TN) group is pink. 95% confidence intervals are indicated, which are narrow given the large group sizes.

relationship between TN and DCM.[7–11] While our point prevalence of DCM was low for both TN and no TN groups (0.55% and 0.04%, respectively), compared to a previous systematic review (2.3%; 95% CI: 1.4–3.1%),[23] this discrepancy may stem from our study methods including not requiring prior cervical spine imaging for inclusion. Nonetheless, we suggest the large magnitude of odds increase of

DCM in TN patients bears clinical relevance in underscoring the potential role of cervical pathology in this pain syndrome.

Although the prevailing hypothesis regarding TN is neurovascular compression of the trigeminal nerve, this mechanism only explains some cases and TN often remains idiopathic.[4] Our findings raise the possibility that in certain patients TN could arise from compression or inflammation of the spinal trigeminal tract in the cervical spinal cord as a result of DCM. As the spinal trigeminal nucleus extends caudally to the C4 spinal segment[12] (Figure 3), pathology affecting this region could contribute to aberrant signaling and pain in the trigeminal pathways, manifesting as TN. While DCM is most prevalent at the C5/6 spinal segments, it also frequently affects the cord at C3/4 and C4/5.[24] Possible mechanisms whereby DCM may contribute to TN include[25]: (1) direct compression and/or distortion of the spinal trigeminal tract from cervical stenosis, myelomalacia, or disc herniation; (2) inflammatory cytokines released by the affected cervical cord; (3) vascular compression causing ischemia or hypoxia of the tract. More research is needed to determine if DCM-related compression and inflammation of the cervical cord and spinal trigeminal tract represent a causal pathogenesis of TN.

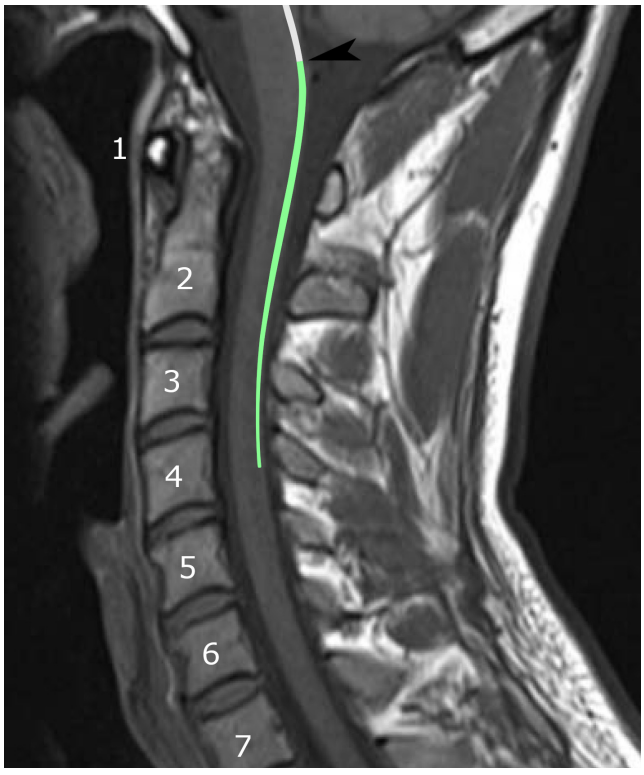

FIGURE 3 Spinal trigeminal tract and relationship to cervical spinal segments. This tract (highlighted gray and green) extends from the brainstem through the dorsal spinal cord to approximately the fourth cervical spinal segment (4).[12,26] The pars caudalis (green), the most inferior part of the spinal trigeminal tract, begins at the obex (arrowhead) and continues caudally.[12,26] T2 weighted MRI of a normal cervical spine adapted by Robert Trager to include the trigeminal tract, dotted line, and labels, from Alghamdi et al.[27] CC-BY-4.0 License.

Our study is consistent with a previous systematic review that identified that a female predominance of approximately 3:1 (female: male) in TN.[28] In our study, the baseline TN cohort had a predominance of females (2.5:1 female: male ratio), which remained after matching (2.4:1 ratio). The female predominance in TN may result from sex differences in genetics (e.g., serotonin signaling, pain processing, and susceptibility to mood disturbances) or simply stem from reporting bias.[28] Conversely, DCM is more prevalent and extensive in males, rather than females.[24,29] Therefore, the opposing sex predominance of these conditions strengthens our observed association between DCM and TN. Further research could examine if sex acts as an effect modifier on the relationship between DCM and TN through interaction testing or subgroup analyses.

Migraine is a condition with a known association with TN, possibly due to activation of the trigeminovascular system.[20] In the present study, migraine was common in the TN cohort before matching (16%), slightly greater than the expected population prevalence of 12%.[30] However, the prevalence of migraine reduced after matching (11%), with adequate between-group balance. Accordingly, the confounding effect of migraine on our findings was minimized, yet it cannot be ruled out entirely. Future studies could examine the relationships between TN, migraine, and DCM, such as whether these conditions form a common co-occurrence or have a shared biological mechanism.

Currently, promising treatments for TN are available to manage symptoms, including neuropathic pain medications and surgical procedures or microvascular decompression targeting the peripheral components or ganglion of the trigeminal nerve or nerve root.[31] However, our finding that individuals with TN have greater odds of concomitant DCM raises the possibility that treatments addressing cervical spine pathology may benefit some TN patients. For example, a handful of case reports have shown that cervical decompression and fusion can alleviate DCM as well as symptoms of TN including facial pain and paresthesia.[8,32]

Our research highlights the variability of symptoms associated with DCM. Patients with DCM may present with a range of symptoms that can be subtle or transient, which contributes to diagnostic delay of this condition.[33] Accordingly, clinicians should remain vigilant in detecting potential DCM and referring for surgical assessment. Our findings suggest that in the appropriate clinical context, TN could serve as an additional indicator of DCM. Recognizing TN as a potential marker for DCM might prompt more thorough investigation, facilitating early diagnosis and intervention.

Additional studies are needed to corroborate our findings. A case–control design comparing the frequency of TN amongst those with or without DCM could examine the association between these conditions using methods different from ours. A longitudinal study design, which follows patients with TN or without TN over time, could better determine the incidence of DCM in amongst adults affected by TN compared to controls. A synthesis of case reports describing TN in the context of DCM may better clarify which spinal levels of cervical stenosis or myelopathy

contribute to TN (e.g., upper, mid, or lower cervical region). Finally, retrospective analyses or registries of patients undergoing cervical spine decompression and fusion may help determine if TN or other facial symptoms improve postoperatively. While sample size may be prohibitive, a cross-sectional study wherein patients with or without TN undergo magnetic resonance imaging and clinical examination could be used to calculate the sensitivity and specificity of TN as a marker of DCM.

4.1 | Strengths and limitations

The strengths of this study include the use of a national US dataset, large sample of over 70,000 propensity matched patients, use of natural language processing, and adherence to a registered protocol. However, as cross-sectional study, there are several limitations including the inability infer causality between DCM and TN. Due to the de-identified nature of the dataset, we could not validate diagnoses of DCM and TN using chart review or any specific criteria. We were unable to include potentially relevant data items regarding DCM. If available, diameter measurements of the spinal canal could have been used to define inclusion criteria for DCM diagnosis.[34] Family history of TN was poorly represented in the TriNetX data and could not be propensity matched. Finally, results obtained from this study are based on US data and may not be generalizable to other countries.

5 | CONCLUSIONS

In this large cross-sectional study, adults with TN had over 12 times greater odds of concurrent DCM relative to matched controls, suggesting DCM may be a risk factor for TN. Clinicians should be vigilant to detect potential DCM among patients with TN, as addressing this condition could supplement standard treatments for TN. Overall, this study identifies a significant association between TN and DCM which may facilitate earlier diagnosis and better management of both conditions.

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CONFLICT OF INTEREST STATEMENT

The authors have declared no competing interests.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in Figshare at https://doi.org/10.6084/m9.figshare.24230332.v1, https://doi.org/10.6084/m9.figshare.24230326.v1, and https://doi.org/10.6084/m9.figshare.24230323.v1.

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