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Neurovascular Compression at the Root Entry Zone Correlates with Trigeminal Neuralgia and Early Microvascular Decompression Outcome

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Abstract

Background—Trigeminal neurovascular contact (NVC) is hypothesized to be the etiology of classical trigeminal neuralgia (TGN). We aimed to seek a correlation between types of NVCs and the presence of TGN as well as early surgical outcome in TGN patients treated with trigeminal microvascular decompression (MVD).

Methods—We blindly analyzed preoperative high-resolution MRIs with respect to the degree ('none', 'touch', or 'compression') and location of bilateral NVC in 57 retrospectively identified Burchiel Type 1 TGN patients treated by MVD. Location of NVC was noted as either at the root entry zone (REZ) or distal to it. Using a logistic regression model, the degree and location of trigeminal NVC was assessed for correlation with the symptomatic side. Further, the NVC characteristics on the symptomatic side were correlated with early postoperative pain relief.

Results—While the degree and location of NVC were not statistically correlative independently, a combined interaction analysis of both statistically correlated with the symptomatic side and with early postoperative pain relief.

Conclusions—We conclude that in TGN patients treated with MVD, MRI identified neurovascular compression at the REZ correlates with the affected side and early post-operative pain relief.

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Keywords

Microvascular Decompression; Root Entry Zone; Trigeminal Neuralgia

Introduction

Classical trigeminal neuralgia (TGN) is a facial pain syndrome with a prevalence of 1-2 per 10,000 individuals.¹ It is usually characterized by unilateral, paroxysmal sharp pains that recur in one or more areas of the trigeminal nerve distribution with innocuous triggers.² While the exact pathophysiology of trigeminal neuralgia is incompletely understood, the leading hypothesis for its etiology is vascular contact at the cisternal segment of the trigeminal nerve.^{3,4} Microvascular decompression (MVD), where the offending vessel is dissected away from the trigeminal nerve and generally kept separated by synthetic padding, is highly effective for many cases of TGN^{5,6} and lends support to this hypothesis.

The primary diagnostic test in medically refractory TGN patients being evaluated for an MVD is a brain magnetic resonance image (MRI). It is obtained primarily to rule out other etiologies of TGN, such as a compressive mass or multiple sclerosis.⁷ Numerous studies have focused on the value of MRI in identifying trigeminal neurovascular contact (NVC) in TGN patients and its correlation to trigeminal neuralgia.⁷ With the advent of high-resolution MRIs, the NVC hypothesis as an etiology of TGN has been further challenged. MRI studies have demonstrated NVC in both non-TGN patients and on the asymptomatic side of TGN patients.^{8,9} Therefore, recent investigative efforts have been made to refine the NVC hypothesis by studying the degree of NVC and its location with respect to the root entry zone (REZ). Although the definition of REZ is variable in literature, it is commonly defined as the length of the centrally myelinated segment of the cranial nerve before it transitions into a peripheral nerve segment.^{10,11} It has been proposed that only vascular contact at the REZ would lead to symptoms.¹⁰ A recent meta-analysis of blinded studies focusing on the value of MRI in evaluating these characteristics of NVC in TGN patients demonstrated that NVC, NVC at REZ, and anatomical trigeminal nerve changes (implying not merely neurovascular 'contact' but *compression* and/or nerve atrophy) correlate with TGN.⁸ Furthermore, a recent study, where MRIs were blindly evaluated by a single neuroradiologist, argued that only neurovascular compression correlates with TGN.¹² However, the relationship of degree and location of NVC to surgical outcome has been largely unaddressed.

This study examines how the degree and location of NVC influence surgical outcome. We retrospectively identified 57 TGN patients treated with an MVD who underwent a preoperative high-resolution MRI. The MRIs were blindly analyzed with respect to the degree and location of bilateral NVC with two objectives: to correlate these characteristics 1) to the symptomatic side and 2) to early surgical outcome. We hypothesize that neurovascular compression at the REZ correlates with TGN symptomatology and early surgical outcome following MVD.

Material and methods

Clinical Data Collection

This study was conducted in accordance with the ethics and regulations of Vanderbilt University Medical Center and was approved by the Institutional Review Board. We retrospectively identified adult patients with trigeminal neuralgia (TGN) who underwent unilateral trigeminal microvascular decompression (MVD) from 2004 to 2010 at a single large academic, tertiary medical center (Vanderbilt University Medical Center, Nashville, TN), and who had undergone a preoperative Fast Imaging Employing Steady-state Acquisition (FIESTA) brain MRI with a 3.0 Tesla machine. The cases were identified by searching medical records with Current Procedural Terminology (CPT) codes (61450 and 61458). Only patients with unilateral classical Burchiel Type 1 trigeminal neuralgia with lancinating, paroxysmal or triggerable pain with pain-free intervals, who had been previously managed non-surgically were identified. MVDs were performed by one of the two senior neurosurgeons (J.S.N. and P.E.K.). The electronic medical record was retrospectively searched for the first postoperative clinic follow-up (around 1 – 2 months). Whether the patient experienced pain relief or not was recorded as well as the use of trigeminal neuralgia medications. Patients were grouped into the following four categories: complete pain relief and off pain medications, significant pain relief with continuation of some of the medications, less than or moderate pain relief, and no pain relief.

Assessing Trigeminal Neurovascular Contact

Bilateral trigeminal nerves identified on the preoperative FIESTA brain MRI of each patient were blindly assessed—without knowledge of the patients' identity, symptomatic side of TGN, or surgical outcome—by a single neurosurgeon (J.S.N.) for neurovascular contact (NVC) with regards to two characteristics: the 'degree' of vascular contact and if present, its location with respect to the nerve root entry zone (REZ).

The degree of NVC was classified as none, 'touch', or compression. None signified that no vascular structure was visualized in contact with the trigeminal nerve. Compression was defined as vascular contact visualized as causing deviation of the trigeminal nerve from an imaginary line from the mid trigeminal-pontine junction to the midpoint of Meckel's cave. 'Touch' was defined as any degree of NVC visualized between these two extremes.

If present, location of the NVC was recorded either at the REZ or distal to the REZ. While some may debate over the exact definition of REZ,¹¹ in this study we define it as the first 3mm segment of the trigeminal nerve as it exits the pons based on the upper limit of histological studies.^{10,11} Hence, if the NVC was noted to be within 3mm of pons on the trigeminal nerve, it was recorded as contact at the REZ; otherwise, it was deemed distal to the REZ.

Insets in Figure 1 provide examples of the degree and location of trigeminal NVC identified on FIESTA brain MRIs of patients in this study.

Data Analysis

The differences between the observed degree and location of trigeminal NVC noted on the symptomatic side versus the asymptomatic side, which we used as an internal comparison, were assessed using a two-tailed McNemar's test given that these data sets are paired or matched. Pain relief was analyzed as a binary outcome (complete or significant pain relief versus some or no pain relief) given the sample size. Correlation between postoperative pain relief from TGN after undergoing an MVD and the degree and location of the symptomatic trigeminal NVC noted on the FIESTA MRI was assessed using a two-tailed Fisher's exact test. GraphPad Prism version 6.00 for Windows (GraphPad Software, San Diego, CA, USA) was used for these statistical calculations. To assess for any correlation between the degree and location of NVC and symptomatic TGN side as well as post-operative pain relief, a multivariate logistic regression analysis was conducted using R version 3.3 (R Foundation for Statistical Computing, Vienna, Austria). Further, we included an interaction term between the degree and location in this analysis. Significance was set at a p-value less than or equal to 0.05 in all statistical analyses.

Results

We retrospectively studied 57 patients with unilateral classical Burchiel Type 1 trigeminal neuralgia (TGN) who had undergone a preoperative Fast Imaging Employing Steady-state Acquisition (FIESTA) brain MRI prior to a unilateral trigeminal microvascular decompression (MVD). Patient demographics together with frequency of laterality are summarized in Table 1. The predominance of Caucasian race and female sex with a slight preponderance of right-sided TGN are consistent with the epidemiology of TGN patients referred for surgical management.^{1,13} None displayed bilateral TGN.

Bilateral trigeminal nerves on the preoperative FIESTA MRIs of each TGN patient were blindly assessed—without knowledge of the symptomatic side—for the degree of neurovascular contact (NVC) classified as none, touch, or compression, and its location relative to the root entry zone (REZ). The degree and location of trigeminal NVC on the symptomatic and asymptomatic sides in each of the 57 TGN patients are displayed in Table 2.

Neurovascular Compression at the Root Entry Zone Correlates with Trigeminal Neuralgia

No statistical difference was noted in the presence or absence of NVC between the symptomatic and asymptomatic sides (odds ratio, OR = 1.33 [0.41-4.66]; $p = 0.79$). Furthermore, the frequency of neurovascular compression between the groups did not statistically differ (OR = 2.00 [0.85-5.05]; $p = 0.12$). When NVC was present, its location (REZ vs. distal to REZ) was not statistically different between the symptomatic and asymptomatic sides (OR = 0.80 [0.27-2.25]; $p = 0.81$). However, a statistically greater proportion of neurovascular compression at the REZ was noted on the symptomatic side compared to asymptomatic side (OR = 2.83 [1.07-8.78]; $p = 0.04$).

These findings were analyzed and confirmed using a multivariate logistic regression model (Table 3). Indeed, the degree of NVC (OR = 0.88 [0.38-2.00]; $p = 0.76$) and location (OR =

0.21 [0.03-1.24]; $p = 0.09$) did not statistically correlate with the symptomatic side. However, when evaluating the interaction between the degree and location of NVC, we found the interaction term to statistically correlate with the symptomatic side (OR = 4.17 [1.07-17.1]; $p = 0.04$) in our logistic regression model. This indicates that if the location of NVC was at the REZ on a particular side, then the odds of having trigeminal neuralgia on that side in our study patients statistically increase with greater degree of NVC.

Neurovascular Compression Correlates with Early Outcome Following Microvascular Decompression

The degree and location of trigeminal NVC on the symptomatic side in TGN patients with and without early postoperative pain relief following MVD are displayed in Table 4. Presence of NVC did not statistically associate with postoperative pain relief ($p = 1.00$). However, the frequency of trigeminal neurovascular compression in those who experienced pain relief was statistically greater ($p = 0.02$) than those who did not experience pain relief. When NVC was present, its location (REZ vs. distal to REZ) was not statistically different between those who experienced pain relief and those who did not ($p = 0.29$). However, statistically greater instances of REZ compression were observed in patients who had postoperative pain relief compared to those who did not ($p = 0.01$).

These findings were analyzed and confirmed using a multivariate logistic regression model (Table 3). While the degree of NVC did not statistically correlate with postoperative pain relief (OR = 0.67 [0.16-2.56]; $p = 0.55$), the location of the NVC (OR = 0.04 [0.00-0.94]; $p = 0.06$) demonstrated a strong correlation with postoperative pain relief. However, when evaluating the interaction between the degree and location of NVC, we found the interaction term statistically correlated with postoperative pain relief (OR = 19.1 [1.72- 492]; $p = 0.03$) in our logistic regression model. This signifies, for instance, that if trigeminal neurovascular compression at REZ was noted on the symptomatic side, then there was a strong likelihood of having early pain relief from TGN following MVD in our study patients.

Operative findings revealed neurovascular compression in all patients, except nine. Simple contact was noted in seven patients. MRI findings on the symptomatic side in these patients showed 'touch' in three patients, compression in two, and no NVC in two. In two patients, no NVC was noted intraoperatively. One of these patients had no demonstrable NVC on MRI and another had a simple 'touch'.

Discussion

In this study, high-resolution preoperative FIESTA sequence brain MRIs of 57 trigeminal neuralgia (TGN) patients treated with microvascular decompression (MVD) were blindly analyzed with respect to the degree and location with relative to root entry zone (REZ) of bilateral neurovascular contact (NVC). These characteristics were statistically analyzed to reveal a correlation with the symptomatic nerve and postoperative pain relief. Our data demonstrates that neither the degree nor the location of NVC, whether at the REZ or distal to it, was associated with the symptomatic nerve or early postoperative pain relief. However, when the location of NVC was taken into account with the degree, the combined interaction

of two factors, specifically compression at the REZ, was highly associated with a symptomatic trigeminal nerve and early pain relief following MVD.

Trigeminal nerve demyelination due to NVC is hypothesized as a pathophysiological mechanism behind the neurovascular contact theory of TGN.⁴ This focal demyelination renders the nerve hyperexcitable and turns that site into an ectopic impulse generator. It is further thought that REZ is the site of demyelination.¹⁴ It remains unclear whether the REZ is particularly susceptible to this demyelination caused by NVC or that it is a consequence of the observation that NVC commonly occurs at the REZ.¹² A limitation of this hypothesis is that it does not clearly explain the rapid clinical recovery after MVD, since re-myelination is much more gradual. Interestingly, electrophysiological studies of the TGN demonstrates almost an immediate recovery of conduction through the compressed TGN after MVD.¹⁵ Therefore, it has been suggested that demyelination and the resulting conduction impairment may both contribute to the TGN pathophysiology.¹⁶

Studies have reached variable conclusions regarding the significance of NVC at REZ and its prediction for trigeminal neuralgia. Some demonstrate strong correlation,^{8,17} while others conclude no association.¹² It is possible that the variable definition of REZ in literature¹¹ as well its variable reported length from 3-7mm,¹² may partially contribute to the heterogeneity of the reported results. Our study was conducted with a more specific and stringent REZ length criteria—3 mm—supported by histological studies,^{10,11} to classify the location of NVC with respect to the REZ, as opposed to the higher lengths commonly used in other studies. Our data show that NVC at REZ does not predict trigeminal neuralgia; and while it displayed a trend with postoperative pain relief, it did not meet statistical significance.

Our study demonstrates that simple neurovascular contact or ‘touching’ is prevalent in both symptomatic and asymptomatic sides of TGN patients; however, severe NVC, i.e., compression, especially at the REZ, is predictive of trigeminal neuralgia. In accordance, other studies conducted with high-resolution MRIs have also demonstrated that simple neurovascular contact or ‘touching’ can be a normal anatomical variant and/or be found on asymptomatic sides of TGN patients^{9,12,17} and that neurovascular compression, especially at REZ, is highly associated with trigeminal neuralgia.^{8,10,17} Presumably, the pathogenesis of TGN is more likely to ensue as a consequence of neurovascular compression, rather than a mere contact, and this seems to be especially true at the REZ, which is hypothesized to be the site of demyelination.¹⁴ Radiographically neurovascular compression is appreciated as distortion or deviation of the trigeminal nerve contour. Splaying of the nerve fibers, thinning and/or atrophy of the nerve can also be noted.^{8,18}

Limitations

This is a retrospective study, and as such, it has inherent limitations related to this study design, such as lack of healthy, non-TGN controls. Therefore, the knowledge that TGN is rarely bilateral could be a source of further bias when evaluating the sides given the lack of non-TGN controls. However, overall, similar number of non-NVC and NVC were noted on both sides in our sample. All patients in this study were surgically treated, having failed medical therapy, which may bias towards a high prevalence of NVC on the symptomatic

side.⁸ Lastly, long-term post-operative follow up would further strengthen our conclusions. We recognize these as a future direction of our study.

Conclusions

Trigeminal neurovascular contact is hypothesized to be the etiology of classical trigeminal neuralgia and can be commonly noted on high-resolution MRIs. We aimed to shed light on the correlation between characteristics of neurovascular contact (degree and location with respect to root entry zone) and the presence of trigeminal neuralgia as well as early surgical outcome after trigeminal microvascular decompression. Our results demonstrate that neurovascular ‘compression’ (not merely a ‘contact’) at the root entry zone was associated with the side of trigeminal neuralgia and early post-operative pain relief in patients treated with microvascular decompression.

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Abbreviations

FIESTA	Fast Imaging Employing Steady-state Acquisition
MVD	Microvascular Decompression
NVC	Neurovascular Contact
REZ	Root Entry Zone
TGN	Trigeminal Neuralgia

Highlights

Neurovascular contact (NVC) on the cisternal segment of the trigeminal nerve was evaluated with respect to the root entry zone (REZ) and the degree as simple contact or compression in patients with trigeminal neuralgia (TGN) treated with microvascular decompression (MVD).

While the degree and location with respect to root entry zone of NVC, as identified by preoperative MRI, did not statistically correlate independently, a combined interaction analysis of both parameters, i.e., compression at REZ, correlated with the symptomatic side and with early postoperative pain relief.

Figure 1. Examples of various degrees and locations of trigeminal neurovascular contact
 The four insets herein are of FIESTA brain MRIs with sections focused on the trigeminal neurovascular contact, with the nerve (N) and the vessel (V) indicated by arrows. A vessel is seen in (A) ‘touching’ the nerve at the root entry zone (REZ), while in (B) the vessel ‘touches’ the nerve distal to the REZ. In (C), a vessel perpendicular to the nerve is noted to compress it medially, causing an obvious deviation of the nerve contour laterally at the REZ, while in (D) this compression by a medial vessel is distal to the REZ.

Table 1
Patient demographics and clinical characteristics

Age at Surgery (Mean $\pm$ SD years)	55 $\pm$ 14
Gender (M : F)	13 : 45
Race (Caucasian : African American)	52 : 5
Symptomatic side (Right : Left)	30 : 27

SD = standard deviation

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Table 2

Distribution of the degree (none, touch, compression) and location (at root entry zone vs. distal to it) of symptomatic (columns) and asymptomatic (rows) trigeminal neurovascular contact in 57 trigeminal neuralgia patients. Each number represents the number of patients with a particular neurovascular contact characteristic on the symptomatic side (columns) and asymptomatic side (rows).

	Symptomatic Side						Total	
	None	Touch		Compression				
		REZ	Distal	REZ	Distal			
Asymptomatic Side	None	4	2	1	5	0	12	
	Touch	REZ	3	4	5	8	0	20
		Distal	1	4	4	4	1	14
	Compression	REZ	1	2	2	1	1	7
		Distal	1	2	1	0	0	4
	Total	10	14	13	18	2	57	

REZ = Root Entry Zone

Table 3

Results of multivariate logistic regression. Correlation of the degree and location of neurovascular contact with trigeminal neuralgia symptoms and pain relief after microvascular decompression.

	Symptoms	
	Odds Ratio [95% CI]	p-value
Location (REZ vs. distal to REZ)	0.21 [0.03-1.24]	0.09
Degree (none, 'touch', compression)	0.88 [0.38-2.00]	0.76
Interaction between location and degree of NVC	4.17 [1.07-17.1]	0.04
	Post-op Pain Relief	
	Odds Ratio [95% CI]	p-value
Location (REZ vs. distal to REZ)	0.04 [0.00-0.94]	0.06
Degree (none, 'touch', compression)	0.67 [0.16-2.56]	0.55
Interaction between location and degree of NVC	19.1 [1.72- 492]	0.03

CI = confidence intervals; NVC = neurovascular compression; REZ = root entry zone

Table 4

Trigeminal neurovascular contact degree (none, touch, compression) and its location (at root entry zone vs. distal to it) in patients with and without pain relief after microvascular decompression.

	None		Touch		Compression	
	REZ	Distal	REZ	Distal	REZ	Distal
None (n = 2)	0	0	2	0	0	0
Moderate or Less (n = 14)	3	6	3	1	1	1
Substantial (n = 10)	1	2	1	6	0	0
Complete (n = 31)	6	6	7	11	1	1

REZ = Root Entry Zone