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ORIGINAL RESEARCH

Symptomatic and Asymptomatic Neurovascular Compression of the Trigeminal Nerve: Correlation with Magnetic Resonance Imaging Findings

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Abstract

Purpose: Neurovascular compression (NVC) is a major cause of classic unilateral trigeminal neuralgia. However, all cases of NVC may not develop symptoms typical of trigeminal neuralgia. In this study, we aimed to compare the various anatomical and radiological features of NVC in symptomatic and asymptomatic individuals on high-resolution, heavily T2-weighted driven equilibrium (T2-DRIVE) sequence.

Materials and methods: This retrospective study included 31 patients in a symptomatic group (Group 1) and 31 patients in an asymptomatic control group (Group 2). T2-DRIVE imaging was evaluated in all patients for NVC of trigeminal nerve (CN V). Student's t test and Chi-square test were used as appropriate.

Results: In this study, NVC of cisternal segment of CN V was present on the symptomatic side in 100% of cases in Group 1, with 58.1% of cases on the opposite asymptomatic side. NVC was seen in Group 2 in 64.5% of cases. Symptomatic NVC was more common in the age group of 46-65 years with mean age of 50.2±12.9 years. The most common vessel associated with symptomatic NVC was SCA or its branches (67.7%), and venous loops were more associated with NVC in Group 2. Symptomatic NVC was associated with more proximal location, higher grades of NVC, smaller trigeminal-pontine angle and lesser nerve length as compared to Group 2.

Conclusion: The presence of neurovascular compression by itself may not be enough to cause neuralgic symptoms. Higher grades of neurovascular compression at proximal points by an arterial loop in older individuals, with characteristic clinical features on the same side, are more associated with trigeminal neuralgia.

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Introduction

Classic trigeminal neuralgia (TN) is characterized by intolerable, sharp, one-sided neuropathic pain along the branches of the trigeminal nerve (CN V) due to some specific sensory stimulus (1,2). It can occur due to compression of CN V by vessels, tumors, arachnoid cysts or due to inflammation, demyelination, vascular malformations and trauma among other causes (1-3). The cisternal segment of CN V is segregated into proximal and distal parts which are the posterior and anterior portions respectively. The proximal part is centrally myelinated (developed from oligodendroglia) and ends before the halfway point of the cisternal segment, while the distal part is peripherally myelinated (developed from Schwann cells) (4). The length of the proximal portion of CN V cisternal segment is known as the root entry zone (REZ) (5). The length of the REZ differs from 3-7mm. The medial part of the REZ has a shorter length of central myelination relative to its lateral part. The central myelination is lesser in thickness proportional to the nerve width, as compared to the peripheral myelination (5).

Neurovascular compression (NVC) causes considerable abrasion, inflammation and degeneration of central myelin of CN V associated with nerve demyelination. Chronic nerve compression causes Schwann cell injury with altered regulation and increased impulse activations of voltage-gated sodium channels. These aberrant discharges are further augmented and distributed by cross-talking among the demyelinated nerve fibers leading to recurring incidents of sharp, intense, electric pain along sensory supply of CN V (3). NVC of the proximal portion of CN V at the REZ is more prone to neuralgic symptoms (1,5,6).

The primary mode of treatment for TN due to NVC is antiepileptic medicines (like carbamazepine) and microvascular decompression is relied upon for refractory cases (1,2,7). Specific, heavily T2-weighted three dimensional (3D) magnetic resonance imaging (MRI) sequences of posterior cranial fossa with high spatial and contrast resolution are used for detection of NVC, as they markedly demonstrate the cranial nerves and vessels in cisterns. These sequences are driven equilibrium (T2-DRIVE; Philips Healthcare, Netherlands), constructive interference in steady-state (CISS; Siemens Healthcare, Erlangen, Germany) and fast imaging employing steady-state acquisition (FIESTA; GE Healthcare, Chicago, Illinois, USA) (7,8). Three-dimensional MR angiography and contrast-enhanced sequences have also been used for NVC in previous studies (9,10). These sequences can ascertain the grade of NVC and vessels involved. It is known that neurovascular contact or compression of CN V can occur at single or multiple locations and all cases of NVC may not develop typical symptoms of neuralgia (1,8). It is necessary to identify the factors which increase the risk of neuralgic symptoms. This study aimed to compare the various anatomical and radiological features of NVC in symptomatic and asymptomatic individuals on T2-DRIVE sequence. To the best of our knowledge, only a few studies based on T2-DRIVE imaging of NVC have been previously reported (11).

Methods

Study population

This retrospective observational study was conducted in the Department of Radiology of a tertiary care hospital from February 2020 to December 2022. We included patients who were referred from the Department of Neurology with symptoms and clinical diagnosis of unilateral classical TN, and underwent MRI brain in the department as a symptomatic group (Group 1). We randomly selected patients who presented with neurological complaints other than TN (such as headache, vertigo and hearing loss) and underwent brain MRI in the department as an asymptomatic/ control group (Group 2). Exclusion criteria were as follows: 1) unavailability of T2-DRIVE images of posterior cranial fossa; 2) history of prior intracranial surgery; 3) artifacts in MRI images hindering proper evaluation (motion artifacts, susceptibility artifacts etc.); 4) MRI images showing tumors or other lesions in prepontine cistern, skull base lesions and multiple sclerosis; and 5) herpes zoster of CN V. Ultimately, 31 patients who fulfilled the inclusion and exclusion criteria were included in each group.

Imaging

The MRI was performed using a 1.5 Tesla scanner (Ingenia, Philips Healthcare, Amsterdam, Netherlands) with head coil. Routine MRI brain protocol included multiplanar T2-weighted (T2W) images, axial T1-weighted (T1W) image, axial fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), and susceptibility-weighted imaging (SWI). The high-resolution 3D T2-DRIVE sequence was obtained with the following parameters: TR 1.5 sec, matrix 380x300, FOV 210 mm, TE 180 msec, NSA 2, thickness of slice 2.2 mm, flip angle 90°, inter-slice interval 1.1 mm. T2-DRIVE images were obtained on the axial plane, and multiplanar reconstruction was used to reformat the images in sagittal and coronal planes on a dedicated workstation supplied by the vendor.

Data collection

Firstly, the demographic characteristics of the study patients (age and sex) in both groups were noted. The patients in Group 1 were assessed on T2-DRIVE images for presence/ absence of NVC on the symptomatic side and asymptomatic sides. Patients in Group 2 were assessed for NVC on both sides. When NVC was present, data was collected for the side involved, the site of NVC (the point of NVC measured from the origin of CN V at the surface of the pons), the involved surface of the nerve, the grade of NVC, and the number and type of vessels. NVC was considered when a vascular loop was seen in contact with CN V in greater than one plane

without intervening cerebrospinal fluid. Nerve displacement was considered when there was nerve buckling at the site of NVC. When there was reduced volume or thinning of the nerve it was classified as nerve atrophy.

The grading of NVC was determined as follows: Grade 1: Only contact; Grade 2: Distortion/displacement/deviation of nerve without thinning; Grade 3: Atrophy / thinning of the nerve (8). The vascular loop causing NVC was evaluated based on its location and course on multiple planes. When there was more than one vessel causing NVC, all the vessels were subsequently evaluated. The length of the cisternal segment of CN V (distance from the surface of the pons to the point where the nerve enters Meckel's cave) was measured on both sides in all patients. The trigeminal-pontine angle (TPA) was measured on the axial plane (at the site of origin of CN V) between the anterior surface of the pons and medial surface of CN V on both sides.

Statistical analysis

We used Statistical Package for Social Sciences (SSPS) 20 (IBM, Armonk, NY, USA) for data analysis. We ascertained the mean and standard deviation (SD) for numerical variables such as age, site of NVC, length of CN V and TPA, and student's *t* test was used for analysis. Frequency with percentage were ascertained for sex, presence of NVC, side of NVC, grade of NVC, number and type of vessels causing NVC and the Chi-square test was used for data evaluation. A *P* value < 0.05 was considered statistically significant.

Ethical considerations

This study was approved by the Institutional Ethics Committee. Informed consent was not obtained, as this was a retrospective study.

Results

Patients in Group 1 and Group 2 had a mean age of 50.2±12.9 years (range 24-80 years) and 44.2±13.1 years (range 21-69 years), respectively (Figure 1). There were 14 females (45.2%) and 17 males (54.8%) in Group 1, and 12 females (38.7%) and 19 males (61.3%) in Group 2. There was no significant difference in age and gender distribution between the two groups ($P = 0.07$ and $P = 0.6$ respectively).

Group 1

NVC of CN V was present on the symptomatic side in all patients (100%) (Table 1). Right-side NVC was seen in 21 patients (67.7%) and left-side NVC in 10 patients (32.3%). In this group, 18 patients (58.1%) also had NVC on the asymptomatic side, with right-side NVC in 6 patients (33.3%) and left-side NVC in 12 patients (66.7%). The frequency of NVC was more common on the symptomatic side than the asymptomatic side ($P = 0.0001$) (Table 2). The mean site of NVC on the symptomatic side was more proximal (2.7±1.02 mm) than on the asymptomatic side (5.5±1.4 mm; $P < 0.0001$). The most common vessel associated with NVC was the superior cerebellar artery (SCA) or its branches (Figure 2) on both sides ($P = 0.51$), followed by a venous loop ($P = 0.94$). NVC at more than one site on the symptomatic nerve was observed in 8 patients (25.8%) with mixed NVC (due to artery and vein) in 6 of them; however, mixed NVC was not observed on the asymptomatic side. NVC due to only vein was seen in 3 patients (9.7%) on the symptomatic side. The basilar artery, anterior inferior cerebellar artery (AICA) and vertebral artery (2.6% each) were also found to be associated with symptomatic NVC.

The most common surface involved on both symptomatic and asymptomatic sides was the superior surface (48.4% and 71% respectively; $P = 0.09$) (Figure 3), followed by the

Figure 1. Age group distribution of both study groups.

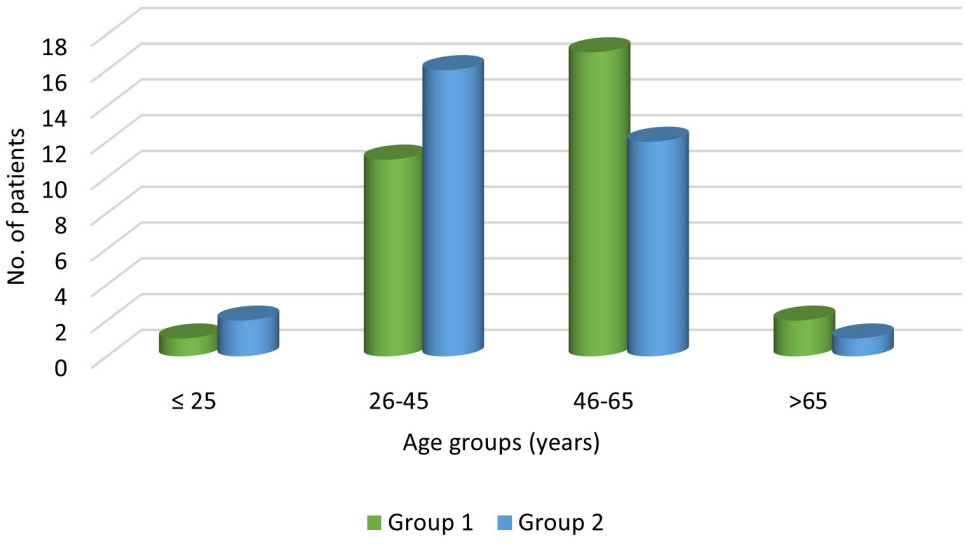

Table 1: MRI findings on T2-DRIVE in both study groups.

Parameters		Group 1 symptomatic side (n=31)	Group 1 asymptomatic side (n=31)	Group 2 (n=31)
NVC present		31 (100%)	18 (58.1%)	20 (64.5%)
NVC at 2 sites		8 (25.8%)	0 (0%)	1 (3.2%)
Grade of NVC	Grade 1	15 (48.4%)	31 (100%)	31 (100%)
	Grade 2	10 (32.3%)	0 (0%)	0 (0%)
	Grade 3	6 (19.3%)	0 (0%)	0 (0%)
Type of vessel	SCA	21 (67.7%)	24 (77.4%)	9 (29%)
	Vein	8 (25.8%)	7 (22.6%)	19 (61.3%)
	Others	2 (6.5%)	0 (0%)	3 (9.7%)
Site of NVC (mean±SD, mm)		2.7±1.02	5.5±1.4	5.2±1.9
Surface of NVC	Superior	15 (48.4%)	22 (71%)	18 (58.1%)
	Medial	9 (29%)	5 (16.1%)	4 (12.9%)
	Inferior	7 (22.6%)	4 (12.9%)	8 (25.8%)
	Lateral	0 (0%)	0 (0%)	1 (3.2%)
Mean length^ (mean±SD, mm)		8.4±0.82	8.8±0.95	9.9±1.4
TPA (mean±SD, º)		35.5±6.3	39.4±7.2	39.5±5.9

Group 1: symptomatic group (cases); Group 2: asymptomatic group (controls); NVC: neurovascular conflict; SCA: superior cerebellar artery; SD: standard deviation; Mean length^: mean length of cisternal segment of CN V; TPA: trigeminal-pontine angle; CN V: trigeminal nerve.

medial surface (29% and 16.1% respectively; $P = 0.26$) and inferior surface (22.6% and 12.9% respectively; $P = 0.38$). The most common grade on the symptomatic side was Grade 1 (48.4%) (Figure 4), followed by Grade 2 (32.3%) (Figure 5) and Grade 3 (19.3%) (Figure 6); however, all patients had Grade 1 NVC (100%) on the asymptomatic side ($P = 0.0002$). The mean length of the cisternal segment of CN V was comparable on both sides ($P = 0.08$). The mean TPA on the symptomatic side was considerably less than on the asymptomatic side (35.5±6.3º and 39.4±7.2º respectively; $P = 0.02$).

Group 2

Twenty patients (64.5%) had NVC in Group 2, with only right-side NVC in 8 cases (40%), only left-side NVC in 5 cases (25%), and bilateral NVC in 7 cases (35%) (Table 1). Frequency of NVC was higher on the symptomatic side in Group 1 as compared to Group 2 ($P = 0.0003$) (Table 3). The mean site of NVC was 5.2±1.9 mm, with significant discrepancy with symptomatic side of Group 1 ($P < 0.0001$) (Table 3). The most commonly associated vessel was vein (61.3%), followed by SCA or its branches (29%), showing significant disparity with Group 1 (p=0.001).

The most common surface involved was superior surface (58.1%), followed by inferior surface (25.8%), medial surface (12.9%) and lateral surface (3.2%) (Table 1) and there was no significant difference from Group 1 (Tables 3,4). All patients had Grade 1 NVC (100%) in Group 2. The mean length of cisternal segment of CN V was 9.9±1.4 mm, significantly more than Group 1 ($P < 0.0001$). The mean TPA was 39.5±5.9, significantly greater than the symptomatic side of Group 1 ($P = 0.003$).

Discussion

In this study, higher grades of NVC at proximal points with an arterial loop were more associated with TN. Symptomatic patients were more likely to have smaller trigeminal-pontine angle, smaller nerve length and nerve atrophy as compared to asymptomatic patients. NVC is a distinct cause of TN (2,8) originally described in 1959 by Gardner and Miklos (1). MRI is required for diagnosis to identify the site, vessels and grade of NVC and to rule out other causes of TN (5). The T2-DRIVE MRI sequence is similar to CISS and FIESTA in technique and applications providing better visualization of intracranial nerves and vessels in comparison to conventional MRI sequences. Imaging finding of NVC using these sequences have shown moderate agreement with surgical finding of NVC which is considered the gold standard for NVC assessment (6,12). Hence, they can be dependably used for diagnosis and treatment planning of TN (11,13).

There was no age or sex disparity between the study groups. Symptomatic NVC was more common in the age group of 46-65 years with mean age of 50.2±12.9 years analogous to findings of previous studies, since older age causes increased length and tortuosity of vertebral and basilar arteries and their branches along with a higher possibility of NVC (3,5). There was no sex predilection in NVC patients, in contrast to other studies which reported higher proportion of females having NVC (5,14). NVC was present in 100% of symptomatic patients on the ipsilateral side. NVC was seen in Group 2 in 64.5% of cases and in 58.1% of asymptomatic side in Group 1. Ruiz-Juretschke et al. (15) noticed asymptomatic NVC in 71% of individuals lacking acknowledged symptoms of TN, among which 75% had NVC on both sides. Likewise, asymptomatic NVC has been noted in other studies as well (4,8).

Table 2: Comparison of MRI findings on T2-DRIVE between symptomatic and asymptomatic sides in Group 1.

Parameters		*P* value
NVC present*		0.0001
NVC at 2 sites*		-
Grade of NVC*	Grade 1	0.0002
	Grade 2	-
	Grade 3	-
Type of vessel*	SCA	0.51
	Vein	0.94
	Others	-
Site of NVC#		< 0.0001
Surface of NVC*	Superior	0.09
	Medial	0.26
	Inferior	0.38
	Lateral	-
Mean length^#		0.08
TPA#		0.02

*Group 1: symptomatic group (cases); NVC: neurovascular conflict; SCA: superior cerebellar artery; Mean length^: mean length of cisternal segment of CN V; TPA: trigeminal-pontine angle; CN V: trigeminal nerve; * Chi-square test. # t-test.*

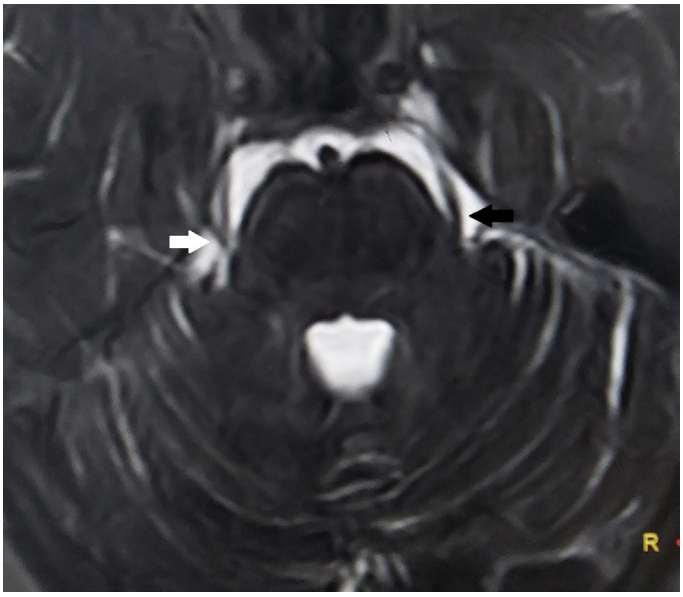

Figure 2. Axial T2-DRIVE image showing right superior cerebellar artery causing Grade 1 neurovascular compression of proximal right trigeminal nerve (white arrow) and normal left trigeminal nerve (black arrow).

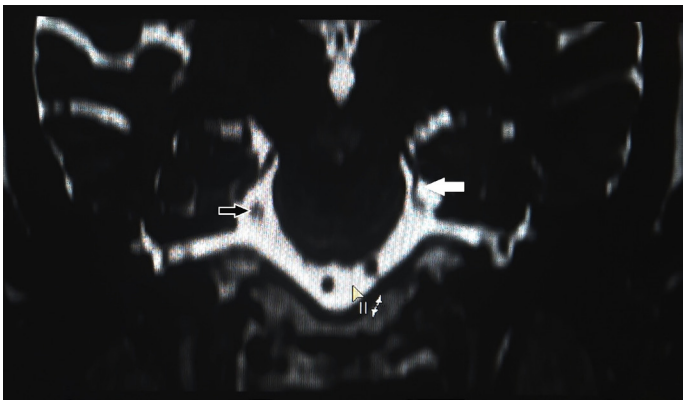

Figure 3. Coronal reconstructed image of T2-DRIVE sequence showing Grade 1 neurovascular compression of superior surface of left trigeminal nerve (white arrow) and normal right trigeminal nerve (black arrow).

The mean site of NVC was more proximal in location in symptomatic NVC as compared to asymptomatic NVC corresponding to previous studies (5,14,16). None of the TN cases in our study had NVC beyond 5.5 mm, which substantiates the concept that a distal site of vascular contact is less prone to demyelination and neuralgia (16). SCA or its branches were the more commonly associated vessel in Group 1, analogous to findings of previous reports (5,7,15,17). Venous loops were more frequently associated with the control group, comparable to previous studies (15). NVC due to a single vein was seen in 3 patients (9.7%) on the symptomatic side, consistent with previous studies (18). It was seen that more than one site of NVC and bilateral NVC were especially associated with symptomatic NVC. In one case of TN, both SCA and AICA were associated with NVC, comparable to previous studies (5,16).

TN was more associated with Grade 3 NVC (nerve thinning/atrophy) at the REZ in 19.3% of patients, as similarly reported by Anwar and colleagues (5) in 12% of cases. Nerve atrophy was not found in the distal part of cisternal segment of CN V, akin to previous studies (5). Nerve distortion, displacement and thinning were absent in asymptomatic NVC in this study. This corroborates with previous studies which have shown that risk of TN is greater in the presence of NVC in more proximal locations associated with an artery, nerve deviation/distortion, and higher grade of NVC (8,10,15,16). Patients with typical symptoms of TN, along

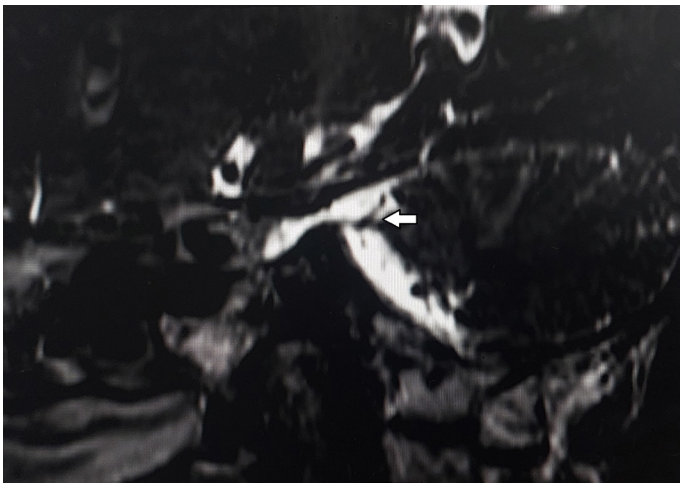

Figure 4. Sagittal reconstructed image of T2-DRIVE sequence showing Grade 1 neurovascular compression of superior surface of left trigeminal nerve (arrow).

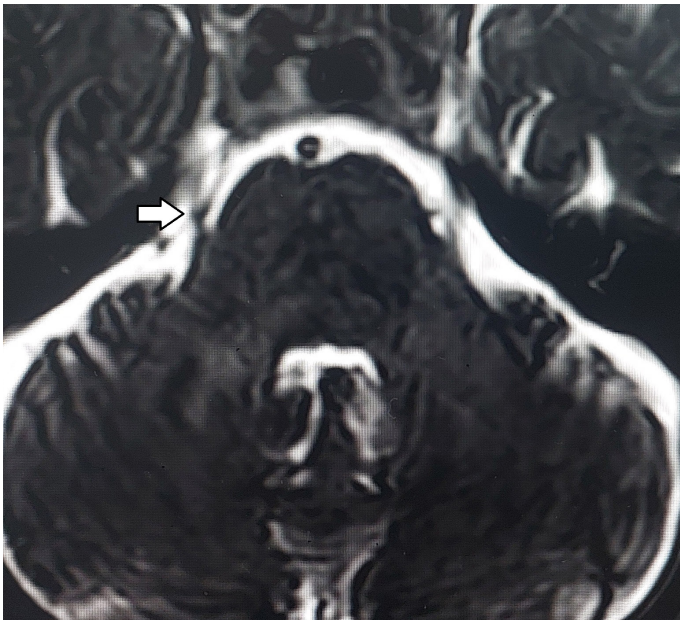

Figure 5. Axial T2-DRIVE image showing Grade 2 neurovascular compression of right trigeminal nerve (arrow).

with imaging confirmation of proximally located NVC, have better symptomatic improvement postoperatively (10). Additionally, TN related to nerve indentation and atrophy has a positive correlation with long-standing postoperative pain relief (19). Asymptomatic individuals with mild NVC may subsequently develop neuralgic symptoms due to hypertension and older age (15).

In this study, TN patients had notably smaller mean TPA in comparison to the unaffected side and control subjects. This finding is comparable to Pang et al. (20), who included 25 TN subjects and 25 asymptomatic subjects in their study and found significantly lesser mean TPA in TN patients as compared to the opposite asymptomatic side

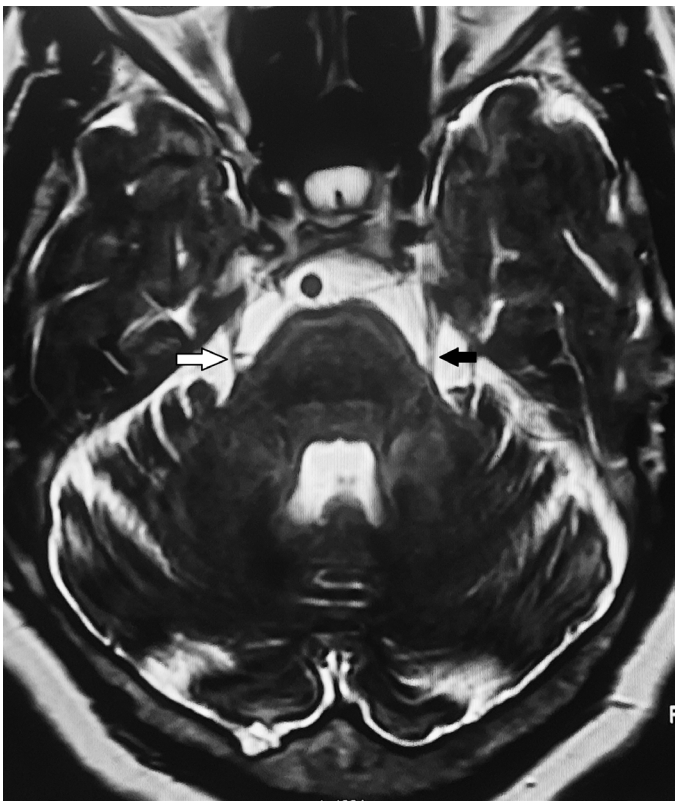

Figure 6. Axial T2-DRIVE image showing Grade 3 neurovascular compression of medial surface of right trigeminal nerve (white arrow) and normal left trigeminal nerve (black arrow).

and control group. They observed that a more acute TPA aggravates degeneration of the affected nerve and adds to the possibility of symptomatic NVC. Likewise, Cheng et al. (21) reported lower mean TPA on the symptomatic side in comparison to the opposite unaffected side and control subjects. They found no notable difference in mean TPA between the unaffected side of TN subjects and the control group, as in our study.

The mean length of the affected nerve was found to be significantly less than the control group in this study, which correlates with findings by Gunesli et al. (22). Gunesli et al. also noted that affected nerves are less thick than unaffected nerves, with smaller cerebellopontine cistern area on the symptomatic side. However, Pang et al. (20) and Cheng et al. (21) reported insignificant disparity in nerve length between the study groups. This could be due to disparity in age distribution of study subjects, imaging protocols, sample size and measurement techniques. Cheng et al. (21) also suggested that nerve atrophy occurs due to long-term nerve compression and is probably related to the development of TN. Additionally, Pang et al. (20) performed diffusion tensor imaging in these patients which revealed evident changes in diffusion parameters (fractional anisotropy and apparent diffusion coefficient) in the affected nerves, in contrast to unaffected nerves. Further studies using diffusion tensor imaging should be advocated for improved understanding of microstructural changes in TN.

Table 3. Comparison of MRI findings on T2-DRIVE between Group 1 symptomatic side and Group 2.

Parameters		*P* value
NVC present*		0.0003
NVC at 2 sites*		0.012
	Grade 1	0.0006
	Grade 2	0.02
Grade of NVC*	Grade 3	-
	SCA	0.001
	Vein	0.001
Type of vessel*	Others	-
Site of NVC#		<0.0001
	Superior	0.5
	Medial	0.6
	Inferior	0.12
Surface of NVC*	Lateral	-
Mean length^#		<0.0001
TPA#		0.003

*Group 1: symptomatic group (cases); Group 2: asymptomatic group (controls); NVC: neurovascular conflict; SCA: superior cerebellar artery; Mean length^: mean length of cisternal segment of CN V; TPA: trigeminal-pontine angle; CN V: trigeminal nerve; * Chi-square test. # t-test.*

Table 4. Comparison of MRI findings on T2-DRIVE between Group 1 asymptomatic side and Group 2.

Parameters		*P* value
NVC present*		0.61
NVC at 2 sites*		-
	Grade 1	0.34
	Grade 2	-
Grade of NVC*	Grade 3	-
	SCA	0.001
	Vein	0.011
Type of vessel*	Others	-
Site of NVC#		0.59
	Superior	0.3
	Medial	0.25
	Inferior	0.8
Surface of NVC*	Lateral	-
Mean length^#		0.0002
TPA#		0.94

*Group 1: symptomatic group (cases); Group 2: asymptomatic group (controls); NVC: neurovascular conflict; SCA: superior cerebellar artery; Mean length^: mean length of cisternal segment of CN V; TPA: trigeminal-pontine angle; CN V: trigeminal nerve; * Chi-square test. # t-test.*

Limitations

This was a retrospective study with a small sample size. A larger sample size would have increased the power of this study. Further correlation with improvement of clinical symptoms due to medications or microvascular decompression could not be done. Future prospective studies with increased sample size, advanced MRI sequences and clinical/surgical follow-up are recommended for improved understanding of underlying factors causing symptomatic NVC of the trigeminal nerve.

Conclusion

Neurovascular compression is a major cause of classic unilateral trigeminal neuralgia. However, the presence of neurovascular compression by itself may not be enough to cause neuralgic symptoms. Higher grades of neurovascular compression at proximal points associated with an arterial loop in older individuals, with characteristic clinical features on same side, are more associated with trigeminal neuralgia. Symptomatic patients are more likely to have a smaller trigeminal-pontine angle, nerve atrophy and smaller nerve length in comparison to asymptomatic patients. These morphological features augment the microstructural changes in compressed nerves and increase the risk of development of trigeminal neuralgia.

Abbreviations

AICA: Anterior inferior cerebellar artery; CN V: trigeminal nerve; mm: millimeter; MRI: magnetic resonance imaging; msec: milliseconds; NVC: neurovascular compression; REZ: root entry zone; SCA: Superior cerebellar artery; SD: standard deviation; sec: seconds; T2-DRIVE: Driven equilibrium; TN: trigeminal neuralgia; TPA: trigeminal-pontine angle; 3D: three dimensional.

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This study was approved by the Institutional Ethics Committee at Gajra Raja Medical College (IEC approval certificate number: 164/IEC-GRMC/2022).

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