COLLEGE OF HEALTH SCIENCES
SCHOOL OF MEDICINE
DEPARTMENT OF RADIOLOGY
POST GRADUATE PROGRAM

# Retrospective Analysis of Brain MRI Findings in Patients with Clinical Diagnosis of Trigeminal Neuralgia in Tikur Anbessa Specialized Hospital and MyungSung Christian Medical Center

By: Dr Kebron Atnaw

A THESIS TO BE SUBMITTED TO ADDIS ABABA UNIVERSITY, COLLEGE HEALTH SCIENCE, SCOOL OF MEDICINE IN PARTIAL FULFILLMENT FOR THE REQUIREMENT OF NEURORADIOLOGY FELLOWSHIP PROGRAM.

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# Declaration

By signing below, I declare that this thesis is entirely my original work. I have adhered to all ethical principles throughout its preparation, including during data collection, analysis, and completion. All scholarly material included in this thesis has been properly acknowledged through citation; I have cited and referenced every source used. Every effort has been made to avoid plagiarism.

This thesis is submitted in partial fulfillment of the requirements for a graduate degree from Addis Ababa University, College of Health Sciences, School of Medicine, Department of Radiology, and Neuroradiology Unit. It has not been presented or submitted, in whole or in part, to this or any other university for the award of a degree, diploma, or other qualification.

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**Abbreviations**

MVD: Microvascular decompression

TASH: Tikur Anbessa Specialized Hospital

MCM: MyungSung Christian Medical Center

CPA: Cerebellopontine Angle

MS: Multiple Sclerosis

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## Abstract

**Background:** Trigeminal neuralgia (TN) is a debilitating facial pain disorder most commonly caused by neurovascular compression (NVC) of the trigeminal nerve, as well as secondary and idiopathic causes**.** Magnetic resonance imaging (MRI) plays a central role in identifying etiologies and guiding management, particularly in distinguishing clinically relevant neurovascular conflict from incidental contact.

**Objective:** To describe MRI patterns in patients with clinically diagnosed trigeminal neuralgia and to compare neurovascular relationships between symptomatic and asymptomatic side.

**Methods:** A retrospective multicenter cross-sectional study was conducted at Tikur Anbessa Specialized Hospital (TASH) and Myung Sung Christian Medical Center (MCM), Ethiopia, including patients aged ≥18 years with a clinical diagnosis of TN who underwent MRI between January 2024 and December 2025. MRI scans were reviewed for neurovascular contact or conflict, secondary structural causes, and incidental findings. Paired comparisons between symptomatic and asymptomatic sides were performed using McNemar's test.

**Results:** Thirty-four patients were included (mean age 53.2 ± 17.7 years), with equal gender distribution. Trigeminal neuralgia was predominantly unilateral (97.1%), more frequently affecting the right side (60.6%). Neurovascular compression was identified in 25 patients (73.5%), constituting the most common etiology. Secondary causes were found in 5 patients (14.7%), including multiple sclerosis and tumors, while 4 patients (11.8%) had normal MRI findings consistent with idiopathic TN. Neurovascular contact or conflict was significantly more frequent on the symptomatic side compared with the asymptomatic side ($p < 0.001$), and true neurovascular conflict was strongly associated with symptoms ($p = 0.012$). The superior cerebellar artery was the most commonly implicated vessel 21 (84%). Most patients were managed medically, while microvascular decompression was performed in 6 patients (17.6%) with MRI-confirmed NVC.

**Conclusion:** MRI is a valuable tool in evaluating trigeminal neuralgia, demonstrating a high prevalence of neurovascular compression, particularly on the symptomatic side. Differentiating neurovascular conflict from simple contact is essential for etiological classification and surgical decision-making. Routine use of standardized high-resolution MRI protocols is recommended to optimize diagnosis and management of trigeminal neuralgia, especially in resource-limited settings.



**Keywords:** Magnetic resonance imaging, Trigeminal Neuralgia, Trigeminal Neuralgia Classification. Neurovascular Contact, Neurovascular Conflict

# Background and Literature Review

**Definition:**

Trigeminal neuralgia (TN) is a debilitating condition characterized by severe, episodic facial pain. The underlying causes of TN can vary, with neurovascular compression (NVC) being the most common cause of primary TN, while secondary TN may be caused by multiple sclerosis (MS), tumors, perineural spread of tumors, or vascular malformations.

A classification of TN into three diagnostic categories has been suggested. Classical TN requires demonstration of morphologic changes in the trigeminal nerve root from vascular compression. Secondary TN is due to an identifiable underlying neurologic disease. TN of unknown etiology is labeled idiopathic(1).

The prevalence of primary and secondary causes are variable in multiple literatures varying from 60% to 90 % of TN, most common being primary trigeminal neuralgia. The most common cause of Trigeminal neuralgia is neurovascular compression by offending vessel, most commonly superior cerebellar artery as demonstrated by multiple studies.

Trigeminal neuralgia mainly affects individuals in middle and older age groups, and it occurs more frequently in females. Prior imaging-based studies have consistently demonstrated a female predominance, with female-to-male ratios ranging from approximately 1.5:1 to 2:1. The condition most commonly presents after the fifth decade of life, although younger patients may be affected, particularly in cases associated with secondary causes. MRI-focused studies evaluating neurovascular conflict and trigeminal nerve pathology have reported similar age and sex distributions, supporting the demographic trends observed in clinical cohorts.

The most common cause for trigeminal neuralgia is contact of the trigeminal nerve with an offending vessel which is also observed routinely in many asymptomatic patients. Prospective as well as retrospective studies demonstrated severe degree of compression where more readily identified on preoperative MRI.

Neurovascular conflict is the most common cause of trigeminal neuralgia, most frequently involving the superior cerebellar artery.

**Role of MRI in patients with trigeminal neuralgia.**

MRI is the primary imaging modality for evaluating trigeminal neuralgia, specifically **heavily T2-weighted sequences.(2)** The combination of time-of-flight (TOF) MR angiography and three-dimensional (3D) T2-weighted imaging demonstrates higher sensitivity and specificity than either sequence used alone. Notably, 3D T2-weighted imaging outperforms TOF MRA when used as a standalone sequence.

The role of MRI is not only to identify the secondary pathologies but also to characterize the neurovascular relation in primary or classic causes of TN. Both high-resolution T2-weighted imaging and MR angiography have been used in multiple prior studies. In the present study, high-resolution T2-weighted imaging was the primary imaging sequence, as MRA was not performed in all patients"

The MRI protocol used in this study included 3-dimensional (3D) T1-weighted imaging, T2-weighted imaging, FLAIR, 3D T2 DRIVE, Gradient echo (GRE), diffusion-weighted imaging (DWI) with apparent diffusion coefficient (ADC) maps, and time-of-flight (TOF) MR angiography. Misdiagnosis as dental illness and delayed diagnosis were common as demonstrated in Ayele et al which demonstrated higher proportion of tooth extraction in TN patients. (3)

**Degree of compression Contact vs Conflict (4) (5) . Where is the contact?**

Beyond mere neurovascular contact, true neurovascular conflict must be demonstrated to confidently diagnose trigeminal nerve compression on MRI. Neurovascular contact—defined as loss of the cerebrospinal fluid (CSF) cleft between the trigeminal nerve and an adjacent vessel without associated nerve displacement, atrophy, or intrinsic signal abnormality—may also be observed on the asymptomatic side. Counterintuitively, trigeminal neuralgia may also be idiopathic, with no identifiable neurovascular conflict on imaging.

Trigeminal NVC occurs in asymptomatic patients but is more severe and more proximal in patients with TN (6).

Murya and Vinya et al. demonstrated neurovascular contact alone is not enough for diagnosis of conflict as it is also present in some asymptomatic individuals, sign of conflict including thinning of nerve, arterial imprint or grooving and distortion in course of nerve, are reliable signs of a conflict between the vessel and the nerve, and these cases are best treated surgically by Micro Vascular Decompression (MVD).(7)

Medical management is 1st-line therapy and if conservative therapy fails, Micro vascular decompression or focused radiotherapy (Gamma knife) are possible treatment options. MVD is available in our setup making imaging characterization very essential prior to the surgery. Imaging prior to surgery has crucial role to identify those patients who will benefit from surgery. It is possible to further characterize simple contact versus conflict or compression, this study characterized neurovascular relationships comparing the symptomatic vs the asymptomatic side (8, 9).

Where access to advanced imaging, particularly MRI, is limited, making imaging correlation of trigeminal neuralgia challenging. This study assessed MRI patterns in Ethiopian patients diagnosed with trigeminal neuralgia clinically, exploring both primary and secondary causes.

# Objectives

## General:

To describe the Brain MRI patterns observed in patients with clinical diagnosis of trigeminal neuralgia and trigeminal nerve relationship with adjacent structures.

## Specific:

Prevalence of trigeminal neuralgia by age and gender.

Prevalence of primary and secondary causes of trigeminal neuralgia and classify based on MRI findings.

To identify the prevalence of neurovascular compression in patients with primary TN.

To compare and contrast MRI findings between symptomatic and asymptomatic sides in primary TN.

To investigate secondary causes of TN, such as MS, tumors, or vascular anomalies, based on MRI findings.

To identify the treatments given specifically medical or micro vascular surgery-MVD.

# Methods

## Study area and period

This study was carried out at two tertiary hospitals in Ethiopia, Tikur Anbessa Specialized Hospital and MyungSung Christian Medical Center from January 2024 to December 2025. Patients diagnosed with trigeminal neuralgia who have undergone MRI from January 2024 to December 2025 were included.

## Study design

The study is a retrospective cross-section observational study to study MRI finding patterns in patients with trigeminal neuralgia

## Study population

Patients above the age of 18 years for whom MRI was done after the diagnosis of trigeminal neuralgia clinically.

## Inclusion Criteria

- Patients with clinical diagnosis of trigeminal neuralgia with MRI imaging.
- Availability of MRI scans showing high-resolution imaging of the brain and cranial nerves for primary TN and MR scans without High resolution for secondary TN are included in the study.

## Exclusion Criteria:

- Patients with Clinical diagnosis of Trigeminal neuralgia with incomplete MRI imaging or poor-quality scans.
- Patients without sufficient clinical documentation for correlating symptoms with MRI findings.
- Patient with only post-operative scans.

## Operational definition

**Trigeminal neuropathy**: Post-herpetic, traumatic suspect if pain is not paroxysmal and/or if there is sensory loss.

**Trigeminal Neuralgia.**

**Normal Trigeminal nerve:** Normal cisternal part of the trigeminal nerve is readily identified extending from pons to Meckel's cave, surrounded by CSF signal. **Trigeminal Nerve identified in the High T2 resolution scan. T2 3D DRIVE (**Image 1)

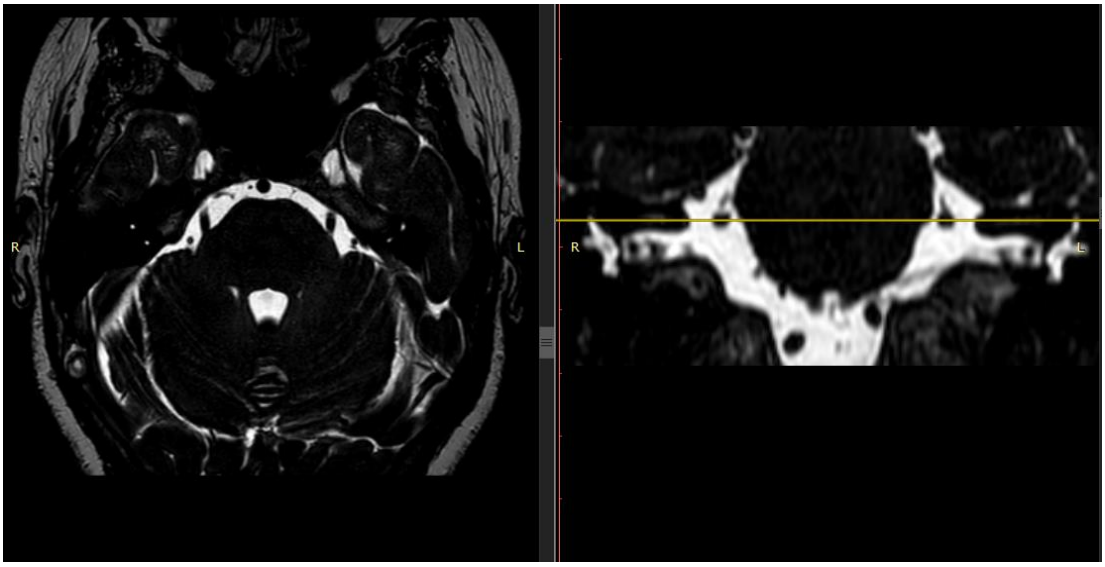


*Image 1: 30 year old female Normal T2 DRIVE and No evidence of contact despite the clinical diagnosis of Trigeminal neuralgia.*

**Vascular contact vs vascular conflict**

**Contact:** CSF space between the nerve and vessel is lost. No evidence of nerves displacement, atrophy or signal change within the nerve.

**Conflict:** Beyond loss of CSF space between the nerve and vessel, there are additional features including nerves displacement, atrophy or signal change within the nerve.

**Location of contact** Classified as Proximal vs mid vs distal portion of the Trigeminal nerve.

**Symptomatic vs asymptomatic side:** Based on the clinical history given for patients with unilateral trigeminal neuralgia, the nerve ipsilateral to the clinical symptoms is referred as the symptomatic side.

## Sample Size:

Based on the hospitals data, 34 patients with TN and MRI findings which passed the inclusion criteria were included in the analysis.

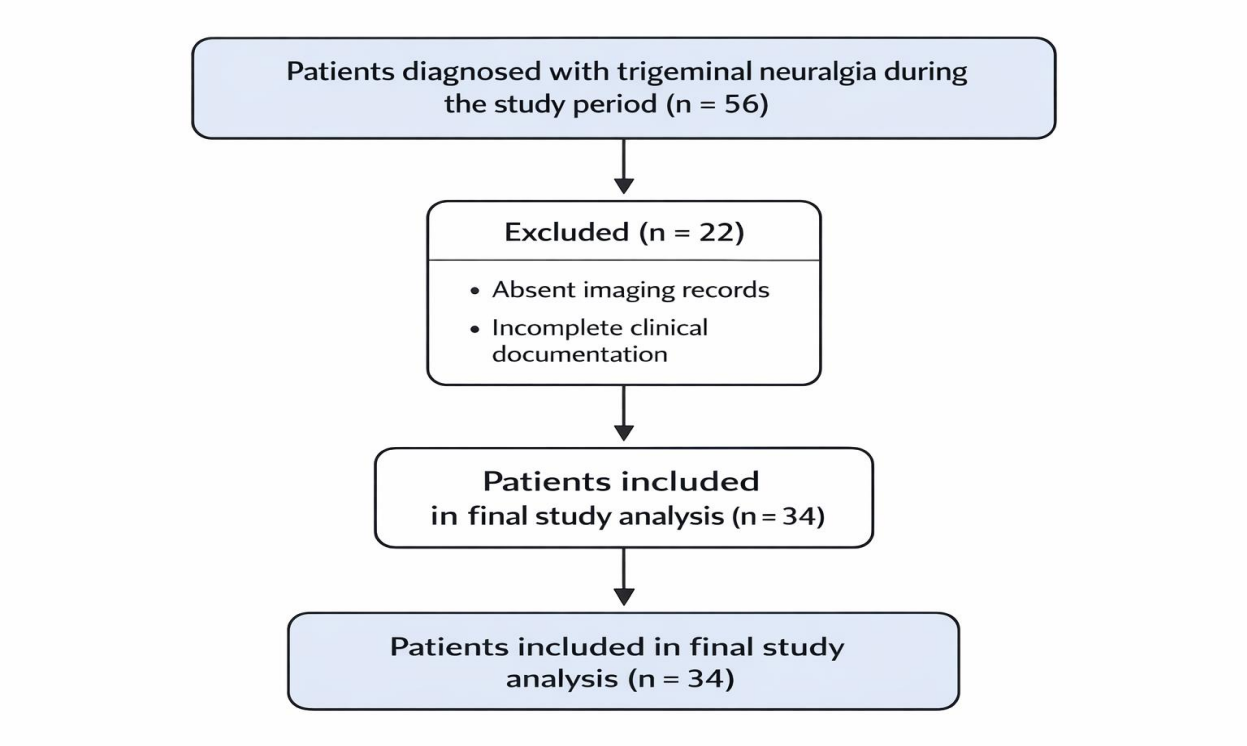


*Fig 1: Flow Chart of included patients*

## Data collection

We described and characterized the patterns of Brain MRI findings in patients with clinical diagnosis of trigeminal neuralgia, using high-resolution magnetic resonance imaging (MRI).

**Imaging Review:** All available MRI scans of TN patients were retrospectively reviewed by neuroradiologist and/ or neuroradiology fellow.

**Trigeminal Nerve identified in the High T2 resolution scan. Imaging is further characterized based on the operational definitions.**

**For primary TN:** Imaging focused on identifying neurovascular compression, assessing whether an artery or vein is in contact with or compressing the trigeminal nerve root, and determining the severity (contact, displacement, or distortion of the nerve).

**For secondary TN**: Imaging was reviewed for structural abnormalities such as:

Multiple sclerosis (MS) plaques in the brainstem or periventricular areas.

Tumors (e.g., meningiomas, schwannomas) compressing the trigeminal nerve.

Vascular malformations or other mass lesions affecting the trigeminal nerve.

Radiological reports categorized the causes into primary and secondary TN.

**Clinical Data Collection:** Clinical data was extracted from patient records and imaging requests, focusing on:

Clinical history: Side of involvement

Treatment type (e.g., medications, micro vascular decompression, tumor resection, or MS therapy).

Imaging reports was retrieved to identify primary and secondary causes.

For those with primary TN and having high resolution T2 WI MRI Neurovascular contact vs conflict will be assessed comparing and contrasting the symptomatic and the asymptomatic side imaging findings.

## Data Analysis

Frequency of neurovascular compression and secondary causes (tumors, MS, vascular anomalies) was calculated. Subgroup analysis will compare patients with primary and secondary TN. Subgroup analysis in comparison between symptomatic and asymptomatic side for the primary causes. Descriptive analysis of prevalence of secondary causes. Descriptive statistics will be used to summarize demographic, clinical, and MRI findings. Chi-square or Fisher's exact tests will be used to compare categorical variables between independent groups (primary vs secondary trigeminal neuralgia). McNemar's test will be used to compare paired MRI findings between symptomatic and asymptomatic sides in patients with unilateral primary trigeminal neuralgia. A p-value $< 0.05$ will be considered statistically significant.

## Ethical Considerations

Ethical clearance was obtained from the institutional review boards (IRBs) of the participating hospitals. Patient confidentiality was preserved by collecting data in an anonymized manner.

# Results

## Study Population, Hospital Distribution, and Socio-Demographic Characteristics

A total of **34 patients** with a clinical diagnosis of trigeminal neuralgia who underwent MRI were included in the study, 26 patients were imaged at MCM **(**76.5**%**), while 8 patients (23.5%) were from Tikur Anbessa Specialized Hospital (TASH)**.**

The age of participants ranged from **27 to 89 years** (mean **53.2 ± 17.7 years**). The most represented age group was **41–60 years** (**38.2%**), followed by **18–40 years** (**29.4%**), **61–80 years** (**23.5%**), and **81–90 years** (**8.8%**). Gender distribution was equal, with **17 males (50.0%)** and **17 females (50.0%)**.

A hospital-wise comparison of age group distribution was performed. Although patients imaged at TASH showed a higher proportion in the **41–60** year age group (50.0%) compared to MCM (34.6%), no statistically significant difference in age group distribution was observed between the two hospitals (Fisher–Freeman–Halton exact test, p = 0.667).

A hospital-wise comparison of gender distribution was performed. At MCM, females constituted **14(53.8%)** of patients, while males accounted for **12(46.2%).** In contrast, patients imaged at TASH were predominantly male **5(62.5%),** with females representing **3(37.5%)** of cases. However, no statistically significant difference in gender distribution was observed between the two hospitals (Fisher's exact test, p = 0.688).

*Table 1. Socio-demographic characteristics of the study population (N = 34)*

| Characteristic | Category | n (%) |
|---|---|---|
| **Hospital** | MCM | 26 (76.5) |
| | TASH | 8 (23.5) |
| **Age (years)** | Mean ± SD | 53.2 ± 17.7 |
| | Range | 27–89 |
| **Age group (years)** | 18–40 | 10 (29.4) |
| | 41–60 | 13 (38.2) |
| | 61–80 | 8 (23.5) |
| | 81–90 | 3 (8.8) |
| **Gender** | Male | 17 (50.0) |
| | Female | 17 (50.0) |

## Clinical Characteristics

Trigeminal neuralgia was **unilateral**, observed in **33 patients (97.1%)**, while **1 patient (2.9%)** had **bilateral** involvement. Among patients with unilateral disease, the **right side** accounting for **20 cases (60.6%)**, compared to **13 cases (39.4%)** involving the **left side**.

Based on MRI findings, patients were classified as having **classical trigeminal neuralgia**, with **neurovascular compression (NVC)** identified in **25 patients (73.5%)**. **Secondary trigeminal neuralgia** was identified in **5 patients (14.7%)**, including **multiple sclerosis** in **2 patients (5.9%)**, **trigeminal schwannoma** in **1 patient (2.9%)**, **vestibular schwannoma** in **1 patient (2.9%)**, and **brain infarction** involving relevant neural pathways in **1 patient (2.9%)**.

In **4 patients (11.8%)**, MRI showed no structural or vascular abnormality to explain symptoms, and these cases were classified as **idiopathic trigeminal neuralgia with normal brain MRI**.

*Table 2. Clinical characteristics and MRI-based classification of trigeminal neuralgia (N = 34)*

| Characteristic | Category | n (%) |
|---|---|---|
| **Laterality of trigeminal neuralgia** | Unilateral | 33 (97.1) |
| | Bilateral | 1 (2.9) |
| **Side involved (unilateral cases, n = 33)** | Right | 20 (60.6) |
| | Left | 13 (39.4) |
| **MRI-based classification / cause** | Classical TN (Neurovascular compression) | 25 (73.5) |
| | Secondary TN – Multiple sclerosis | 2 (5.9) |
| | Secondary TN – Trigeminal schwannoma | 1 (2.9) |
| | Secondary TN – Vestibular schwannoma | 1 (2.9) |
| | Secondary TN – Infarction | 1 (2.9) |
| | Idiopathic TN (normal brain MRI) | 4 (11.8) |

## Overall MRI Findings

34 patients included in the study, 30 patients (88.2%) showed abnormalities on imaging, while only 4 patients (11.8%) had normal MRI findings. This indicates that MRI was highly sensitive in detecting structural changes among patients with clinically diagnosed trigeminal neuralgia.

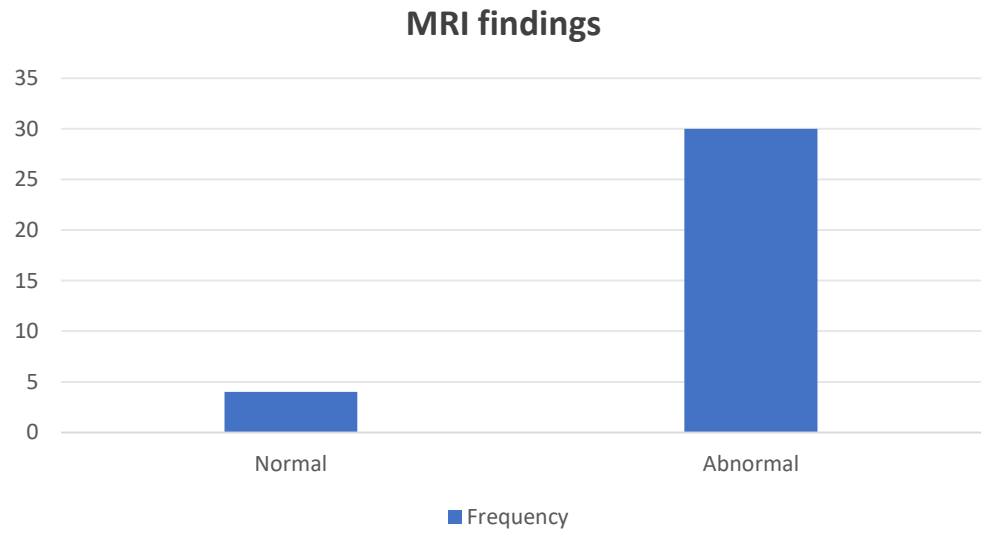


*Fig 2: Overall MRI Findings*

## Neurovascular Relationship of the Trigeminal Nerve

### Neurovascular Relationship on the Symptomatic Side

Assessment of the symptomatic trigeminal nerve was possible in **29 patients** with unilateral trigeminal neuralgia and adequate high-resolution MRI. **Neurovascular contact or conflict** was identified in the majority of cases. **Simple vascular contact** was observed in **15 patients (51.7%)**, while **neurovascular conflict**—defined by nerve displacement, atrophy, or signal change—was present in **10 patients (34.5%)**. **No neurovascular contact** was identified in **4 patients (13.8%)**.

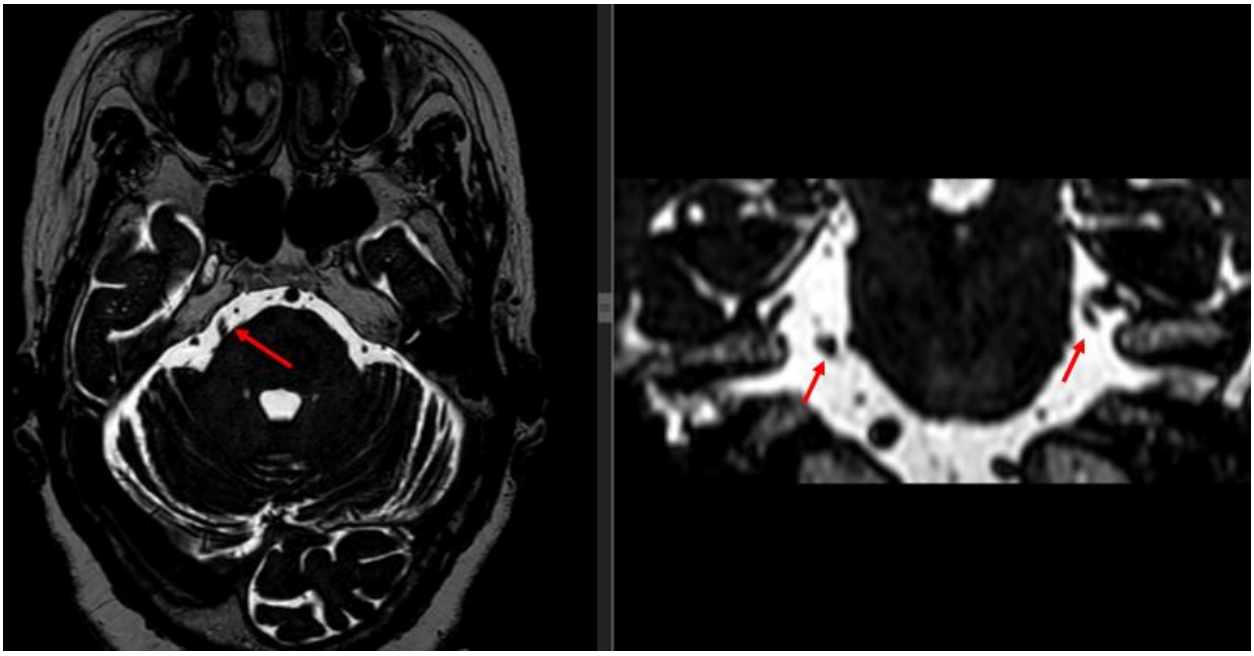

*Image 2: 84 year old female patient with Trigeminal neuroralgia T2 DRIVE showed Right Trigeminal Nerve: Conflict evidenced by decreased caliber/atrophy and deviation of the nerve at the/ Proximal contact. Left Trigeminal Nerve: No Contact.*

### Neurovascular Relationship on the Asymptomatic Side

On the asymptomatic side, **no neurovascular contact** was the most common finding, seen in **23 patients (79.3%)**. **Simple contact** was identified in **5 patients (17.2%)**, while **neurovascular conflict** was rare, observed in only **1 patient (3.4%)**.

**Comparison between Symptomatic and Asymptomatic Sides**

Paired comparison between symptomatic and asymptomatic sides demonstrated a significant asymmetry in neurovascular relationships.

When **neurovascular contact (contact + conflict)** was compared with **no contact**, contact was significantly more frequent on the symptomatic side (**McNemar's test, $p < 0.001$**).

When the analysis was restricted to **true neurovascular conflict** versus **no conflict**, conflict was significantly more common on the symptomatic side compared with the asymptomatic side (**McNemar's test, $p = 0.012$**).

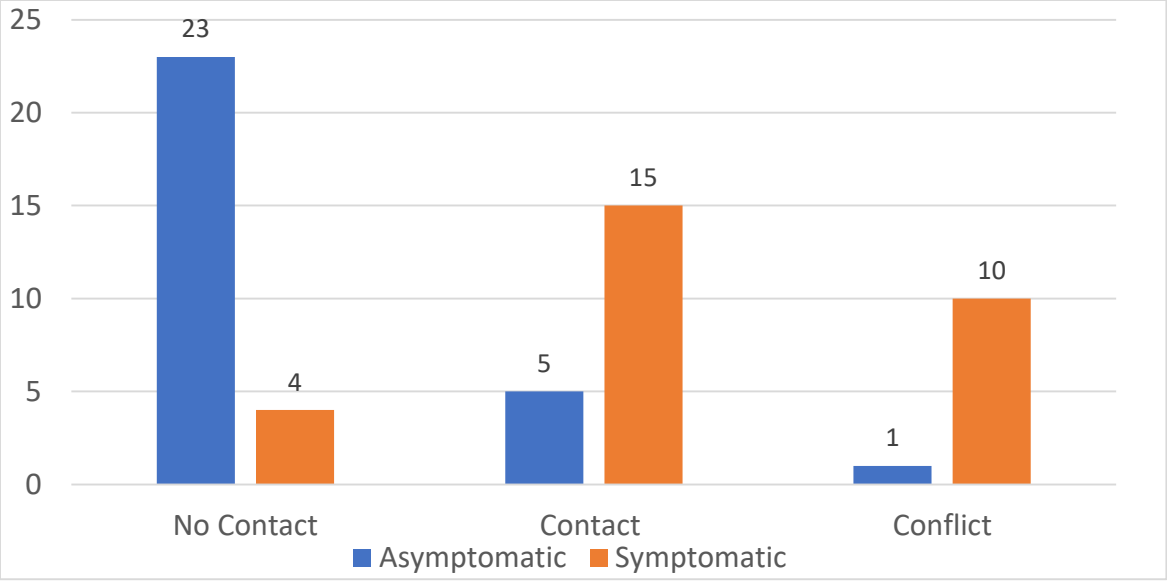


*Fig 3: Comparison of neurovascular relationship between symptomatic and asymptomatic trigeminal nerve sides*

*Table 3: Comparison of neurovascular relationship between symptomatic and asymptomatic trigeminal nerve sides (n = 29)*

| Neurovascular relationship | Symptomatic side n (%) | Asymptomatic side n (%) |
|---|---|---|
| No contact | 4(13.8) | 23 (79.3) |
| Contact | 15 (51.7) | 5 (17.2) |
| Conflict | 10 (34.5) | 1 (3.4) |

*Table 4: Paired binary comparisons (McNemar's test)*

| Comparison | Symptomatic side | Asymptomatic side | p-value |
|---|---|---|---|
| Contact or conflict vs no contact | More frequent | Less frequent | <0.001 |
| Conflict vs no conflict | More frequent | Less frequent | 0.012 |

## Vascular Findings

Among patients with **MRI-identified neurovascular contact or conflict** (**n = 25**), the **superior cerebellar artery (SCA)** was the most frequently implicated vessel, identified in **21 patients (84.0%)**. The **antero-inferior cerebellar artery (AICA)** was involved in **3 patients (12.0%)**, while a **persistent trigeminal artery** was identified in **1 patient (4.0%)**.

All cases demonstrated **single-vessel involvement**, and no patient showed evidence of **multiple offending vessels**.

No additional vascular abnormalities, such as **vertebrobasilar dolichoectasia** or **intracranial aneurysms**, were identified in this cohort.

### Secondary Structural MRI Findings

Mass lesions with direct compressive effect on the trigeminal nerve were identified in **2 patients (5.9%)**, both of which were **schwannomas**. No mass lesion was detected in the remaining **32 patients (94.1%)**.

The **Meckel's cave** appeared **symmetrical and normal** in **33 patients (97.1%)**. **Asymmetry** of Meckel's cave was observed in **1 patient (2.9%)**.

MRI evidence of **demyelination** was identified in **2 patients (5.9%)**, while **32 patients (94.1%)** showed no demyelinating lesions. The demyelinating lesion were features consistent with multiple sclerosis. Signal intensity change within the trigeminal nerve secondary to neurovascular compression are excluded from primary demyelinating lesions.

### Other Intracranial Findings

Additional intracranial findings were identified in a subset of patients. **Chronic small vessel disease** was the most common incidental finding, observed in **14 patients (41.2%)**. **Paranasal sinusitis** was noted in **2 patients (5.9%)**. No other intracranial abnormalities were identified in **18 patients (52.9%)**. No other abnormal head and Neck Findings including tumor and perineural spread of tumors in the study population.

*Table 5. Secondary structural and incidental MRI findings in patients with trigeminal neuralgia (N = 34)*

| MRI finding category | Specific finding | n (%) |
|---|---|---|
| **Mass adjacent to trigeminal nerve** | Schwannoma | 2 (5.9) |
| | No mass detected | 32 (94.1) |
| **Meckel's cave** | Symmetrical and normal | 33 (97.1) |
| | Asymmetrical | 1 (2.9) |
| **Demyelination** | Present | 2 (5.9) |
| | Absent | 32 (94.1) |
| **Other intracranial findings** | Chronic small vessel disease | 14 (41.2) |
| | Sinusitis | 2 (5.9) |
| | No other intracranial findings | 18 (52.9) |

## Etiology of Trigeminal Neuralgia Based on MRI

Based on MRI findings, **primary (classical) trigeminal neuralgia** due to **neurovascular compression** was the most common etiology, identified in **25 patients (73.5%)**. **Secondary trigeminal neuralgia** was identified in **5 patients (14.7%)**, while **4 patients (11.8%)** had **idiopathic trigeminal neuralgia** with no identifiable abnormality on brain MRI.

Among the specific MRI-defined causes, **neurovascular compression (NVC)** accounted for **25 patients (73.5%)**. Secondary causes included **multiple sclerosis** in **2 patients (5.9%)**, **trigeminal schwannoma** in **1 patient (2.9%)**, **vestibular schwannoma** in **1 patient (2.9%)**, and **brain infarction** affecting relevant neural pathways in **1 patient (2.9%)**. In **4 patients (11.8%)**, MRI was normal, and these cases were classified as **idiopathic trigeminal neuralgia**.

MRI-based causes showed complete concordance with TN classification, with neurovascular compression corresponding to classical TN, structural or demyelinating lesions corresponding to secondary TN, and normal MRI findings corresponding to idiopathic TN.

*Table 6. Causes of trigeminal neuralgia based on MRI findings (N = 34)*

| MRI-based classification | Specific cause | n (%) |
|---|---|---|
| **Primary (Classical TN)** | Neurovascular compression | 25 (73.5) |
| **Secondary TN** | Multiple sclerosis | 2 (5.9) |
| | Trigeminal schwannoma | 1 (2.9) |
| | Vestibular schwannoma | 1 (2.9) |
| | Infarction | 1 (2.9) |
| **Idiopathic TN** | Normal brain MRI | 4 (11.8) |

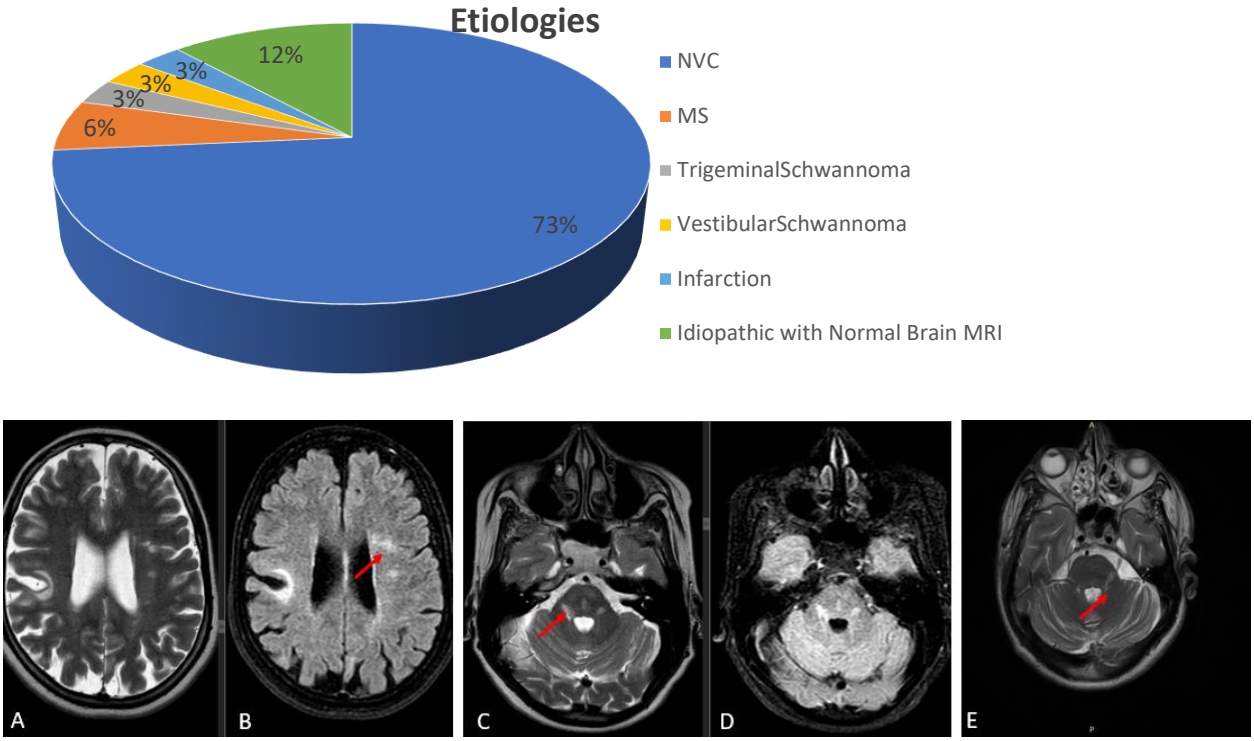


*Image 3 : A to D, 36 year old female with diagnosis of Multiple sclerosis,supratentorial and Brain stem demyelinating lesions demonstrated along the nerve at the level of the exiting CN V. (E) second patient demyelination along the left CN V.*

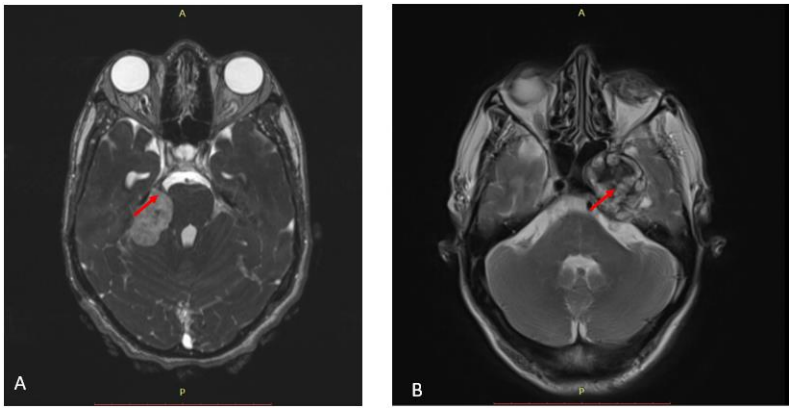


*Image 4:A. High resolutions T2 wieghted image demonstrates right CPA mass vestibular schwannoma compressing the trigeminal nerve. B. T2 weighted image showed trigeminal schwannoma*

## Treatment Modalities

The majority of patients were managed with **medical therapy including carbamazepine**, which was administered in **27 patients (79.4%)**. **Microvascular decompression (MVD)** was performed in **6 patients (17.6%)**, all of whom had MRI evidence of neurovascular compression. **Surgical removal of mass lesions** was performed in **1 patient (2.9%)** with a tumor-related secondary cause.

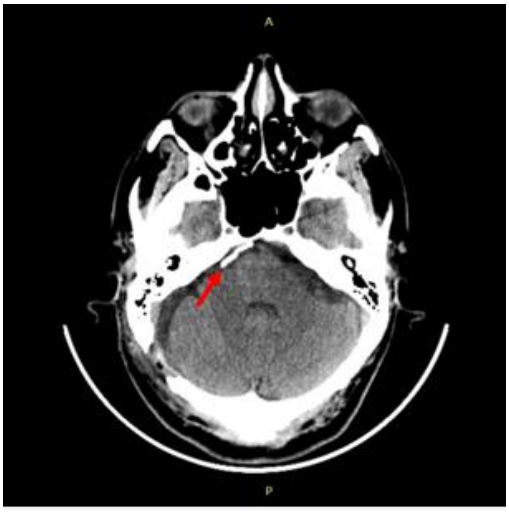

*Image 5: Axial non contrast CT showed hyper dense structure (Teflon) at the right CPA along anatomic course of right trigeminal nerve* in *53 year old male status post Microvascular Decompression-MVD surgery.*

# Discussion

This study investigated MRI characteristics of trigeminal neuralgia (TN) patients and highlighted the diagnostic utility of neuroimaging in differentiating symptomatic from asymptomatic findings. Our results reinforce that **neurovascular compression (NVC)** is a predominant imaging finding in TN, particularly when morphological changes such as nerve displacement or atrophy are present, supporting the long-held hypothesis of vascular conflict as a key etiological mechanism in classical TN. Several recent imaging studies corroborate these findings and extend the understanding of MRI features associated with symptomatic pain.

**Neurovascular Compression and Symptom Correlation**

Our findings align closely with observations by Maurya et al. from India, who emphasized that simple neurovascular contact is frequently observed in both symptomatic and asymptomatic individuals, and that only contacts associated with morphological nerve changes, such as nerve thinning, distortion, or arterial imprint, represent true neurovascular conflict. In their cohort, nerve deformation was significantly associated with symptomatic trigeminal neuralgia, reinforcing our observation that compression severity rather than mere contact is clinically relevant.(7)

Consistent with prior work, our analysis showed that **NVC was significantly more common on the symptomatic side** than the asymptomatic side in patients with unilateral TN. A comprehensive meta-analysis found that NVC at the root entry zone is substantially more prevalent in symptomatic nerves compared to asymptomatic nerves, indicating a strong association between imaging evidence of compression and clinical symptoms (10). As typical MRI sequences at 3 T facilitate high-resolution visualization of vascular loops adjacent to the trigeminal nerve, the presence of severe compression with nerve distortion contributes to symptom generation.(11) Early studies also demonstrated that the proximity of the offending vessel to the nerve root correlates with symptomatic pain, with closer vessel-nerve distances significantly associated with clinical manifestations.(8)

However, while NVC is a key imaging marker, **simple neurovascular contact (absence of morphological nerve changes)** can also be present in asymptomatic individuals. Historic autopsy and imaging work indicates neurovascular contact even in healthy persons, emphasizing that

contact alone is not sufficient for symptomatic TN without accompanying structural changes.(12) Advanced imaging approaches, such as diffusion tensor imaging (DTI), have been explored to distinguish true pathological compression from incidental contacts by revealing microstructural nerve alterations, though standardized thresholds remain under investigation.(11)

Our findings are comparable to reports from other low- and middle-income countries. A study by Bora et al. in India similarly demonstrated a higher prevalence of neurovascular compression on the symptomatic side, with arterial loops being the most frequent offending vessels. Likewise, Elakhder et al. from Egypt reported that MRI identified neurovascular compression as the predominant finding in patients with classical trigeminal neuralgia, while also emphasizing the importance of detecting secondary causes such as tumors and demyelinating lesions. The consistency of these findings across different populations reinforces the robustness of MRI in characterizing trigeminal neuralgia and supports the generalizability of our results.(2, 13)

**Secondary Etiologies and Imaging Beyond NVC**

While classical TN is frequently related to vascular conflict, secondary causes remain important considerations, particularly in atypical clinical presentations or younger patients. MRI reliably identifies structural abnormalities such as demyelinating plaques in multiple sclerosis or space-occupying lesions in the posterior fossa, which were present in a minority of cases in our cohort and similar observational studies.(11) The robust detection of these secondary lesions on MRI underscores its value not only in confirming NVC but also in excluding alternative pathological causes of facial pain.(14)

Additionally, research has begun to explore **nonvascular anatomical patterns** contributing to TN symptoms. For instance, changes in the angles of the petrous ridge and trigeminal nerve have been reported in TN without evident neurovascular compression, suggesting that anatomical configuration may play a role in symptomatic onset.(15) These findings highlight that subtle anatomical variation can contribute to nerve irritation and pain independent of vascular loops.

## Prognostic Implications of MRI Findings

The clinical relevance of preoperative MRI findings extends to treatment planning and prognostication. Studies suggest that **the presence of significant NVC is associated with surgical outcomes**; for example, patients with preoperative compression may experience different patterns of pain recurrence following percutaneous interventions compared to those without clear MRI evidence of compression.(16) In surgical cohorts, microvascular decompression shows high efficacy, particularly when MRI demonstrates significant morphological nerve changes, with sustained pain relief in most treated patients.(17) These data support the premise that advanced imaging not only aids in diagnosis but also enhances prognostic stratification.

From a surgical planning perspective, Hughes et al. highlighted that preoperative MRI plays a critical role in identifying features most relevant to microvascular decompression, including the site of compression, degree of nerve deformation, and relationship to the root entry zone. These imaging features have been shown to correlate with favorable surgical outcomes, supporting our observation that patients with clear morphological evidence of neurovascular conflict represent optimal candidates for MVD.(5)

## Limitations and Future Directions

In our study, detailed clinicoradiological correlation was limited by the retrospective design, which restricted control over clinical documentation and imaging techniques and provided insufficient data on pain intensity, duration of symptoms, and response to treatment.

The relatively small sample size from two tertiary centers may limit generalizability, particularly to patients managed at lower levels of care who may not undergo MRI. Follow up patient without MR imaging was also important limitation as these patients was not included in this study.

Some studies lacked advanced vascular imaging –MRA, potentially affecting detection of subtle neurovascular relationships. Classification of neurovascular contact versus conflict was based on qualitative criteria without formal inter-observer agreement assessment, introducing potential variability. Advanced imaging techniques such as diffusion tensor imaging and magnetic resonance angiography were unavailable and may have identified microstructural nerve abnormalities in cases classified as idiopathic.

Long-term clinical outcomes were not systematically assessed, limiting evaluation of the prognostic value of MRI findings, particularly in relation to surgical outcomes following microvascular decompression. Despite these limitations, the study provides valuable region-specific data and reflects real-world imaging practice in a resource-limited setting.

Despite its integral role, MRI has inherent limitations. Interobserver variability in detection and interpretation of NVC remains a documented challenge, and some studies suggest moderate diagnostic accuracy, particularly when distinguishing high-grade compression from incidental contact.(18) This underscores the need for standardized imaging protocols and objective criteria for pathological compression. The emergence of high-field MRI, refined sequences (e.g., 3D CISS, FIESTA), and multimodal fusion approaches aims to improve spatial resolution and diagnostic confidence.(11, 19) Furthermore, evolving quantitative imaging biomarkers such as nerve microstructure metrics warrant further research.

A recent network meta-analysis by Chen Liang et al. compared different MRI-based techniques for detecting neurovascular compression in trigeminal neuralgia and hemifacial spasm, demonstrating that high-resolution T2-weighted imaging alone provides acceptable diagnostic performance, while combined multimodal approaches (HR T2WI with 3D TOF MRA or image fusion) yield the highest accuracy. These findings support our use of high-resolution T2WI as a practical diagnostic tool, particularly in resource-limited settings, while also highlighting the potential value of advanced multimodal protocols where available.(14)

**Implications for Practice**

In our setting, where clinical misdiagnosis and delayed referral for imaging are common, these findings emphasize the importance of **incorporating dedicated MRI protocols specifically highly T2WI sequences** early in the diagnostic pathway for TN. Facilitating accurate identification of both classical and secondary causes not only guides appropriate management strategies but can also reduce unnecessary interventions and improve patient outcomes. The demonstrated utility of high-resolution T2-weighted imaging as a standalone sequence, as supported by recent comparative meta-analyses, makes it especially suitable for routine clinical use in low- and middle-income settings.(14)

# Conclusion and recommendation

## Conclusion

This multicenter retrospective study demonstrates that MRI is a valuable diagnostic tool in patients with clinically diagnosed trigeminal neuralgia, with the majority showing identifiable imaging abnormalities. Neurovascular compression was the most common MRI finding, particularly on the symptomatic side, and true neurovascular conflict was significantly associated with clinical symptoms. The superior cerebellar artery was the most frequently implicated vessel. MRI also effectively identified secondary causes, including demyelinating disease and tumors, while a minority of patients had normal imaging consistent with idiopathic trigeminal neuralgia. These findings highlight the importance of MRI in etiological classification, treatment planning, and surgical decision-making in patients with trigeminal neuralgia.

## Recommendations

Dedicated High-resolution T2 MRI of the trigeminal nerve should be routinely obtained in patients with suspected trigeminal neuralgia to help distinguish classical, secondary, and idiopathic types. Standardized MRI protocols with consistent high-resolution T2-weighted sequences and vascular imaging are recommended to improve detection and characterization of neurovascular conflict. MRI findings should be interpreted in conjunction with clinical features to guide management, particularly in selecting candidates for microvascular decompression. Future prospective studies with larger sample sizes, standardized imaging protocols, and correlation with long-term clinical outcomes are recommended to further refine imaging criteria and optimize patient care in resource-limited settings.

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# Annex

Questionnaire

PART I: Socio Demographic Data

1. Medical Record Number / Imaging Accession Number / Where MRI done
2. Age
3. Gender A. Male B. Female

PART II: Trigeminal Neuralgia imaging findings

a. Trigeminal Nerve:
    a. Laterality: Unilateral vs Bilateral
        i. If unilateral Right Vs Left (Symptomatic side)
    b. Symptomatic side vs Asymptomatic side if unilateral
        i. Contact vs conflict (displacement, and /or distortion of the nerve).
        ii. Displacement:
        iii. Atrophy:
        iv. Signal Change:
        v. Site of contact measurement / Zone of contact
        vi. Thickening:
        vii. Enhancement: No enhancement vs Enhancement vs Not assessed

Neurovascular compression characteristics T2 3D Drive

| | Symptomatic side | Asymptomatic side |
|---|---|---|
| i. Contact vs conflict | | |
| ii. Displacement: | | |
| iii. Atrophy: | | |
| iv. Signal Change: | | |
| vi. Enhancement: | | |
| vii. Site of contact measurement/ Zone of contact | Proximal vs Mid vs Distal portion of the nerve | Proximal vs Mid vs Distal portion of the nerve |

b. Vascular findings /Vessel patterns involved in the neurovascular compression
    a. Vessel identified:
        i. Superior cerebellar artery (SCA)
        ii. Antero-inferior cerebellar artery (AICA)
        iii. Basilar artery (BA)
        iv. Vertebral artery (VA)

v. Veins/ variant vein

vi. Vascular structure Not specifically Reported

vii. Vertebrobasilar dolichoectsia

viii. Multiple contacts

ix. No contract

b. Vessel contact vs Vessel conflict:

i. Simple contact: [CSF space between the nerve and vessel is lost. No evidence of nerves displacement, atrophy or signal change within the nerve.] NB: Axial and coronal views of T2 3D Drive.

ii. Conflict Definition: [Beyond loss of CSF space between the nerve and vessel, there are additional features including nerves displacement, atrophy or signal change within the nerve.]

c. Other vascular findings:

i. Aneurysms

c. Mass adjacent to the Trigeminal Nerve:

Specify

d. Meckel's Cave:

a. Visualized

b. Symmetrical

c. Enlarged

e. Demyelination:

f. Other Head and Neck Findings:

a. Tumor

b. Perineural spread of Tumor- PNS

c. Skull base abnormality:

g. Other intracranial Findings:

a. Sinusitis

b. Small vessel disease, infarcts

c. Brain stem vascular anomalies

h. No abnormality detected despite the clinical diagnosis of TN

a. Normal MRI

b. Abnormal MRI

i. Second Reading

   a. Specify which findings are new:

j. Imaging report: Findings and impression:

PART III: Clinical Documentation

1. Symptoms
   a. Duration and which side involved
   b. Right side or Left side or both [Symptom laterality]
2. Classification of TN Post MRI imaging A. Classical TN B. Secondary TN C. Idiopathic
3. Follow up:
   a. 1 yrs , 5 yrs , > 5yrs
4. Treatment:

   Medical: Specify

MVD: Microvascular Decompression / Intraoperative Findings