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REVIEW

Neuroimaging in trigeminal neuralgia

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Trigeminal neuralgia is a debilitating craniofacial pain disorder characterized by recurrent, unilateral, electric shock-like facial pain. Magnetic resonance imaging plays a central role in the evaluation of patients with suspected trigeminal neuralgia by identifying clinically significant neurovascular compression, demonstrating morphological changes of the trigeminal nerve, and excluding secondary causes. This review provides an overview of the relevant anatomy of the trigeminal nerve and its transition zone and discusses imaging features that help distinguish symptomatic neurovascular conflict from incidental vascular contact. Particular emphasis is placed on high-resolution three-dimensional heavily T2-weighted sequences for visualization of the cisternal trigeminal nerve and its relationship with adjacent vessels, as well as three-dimensional time-of-flight magnetic resonance angiography and contrast-enhanced three-dimensional T1-weighted imaging for vascular characterization. The imaging spectrum of secondary trigeminal neuralgia, including tumors, demyelinating disease, vascular malformations, inflammatory conditions, and infectious disorders, is also reviewed. Advanced techniques, including diffusion tensor imaging and quantitative assessment of trigeminal nerve morphology, may provide additional information regarding microstructural abnormalities and treatment response. Vendor-specific three-dimensional sequences, including constructive interference in steady state, fast imaging employing steady-state acquisition, sampling perfection with application-optimized contrasts using different flip-angle evolutions, volume isotropic turbo spin-echo acquisition, and three-dimensional variable-flip-angle fast spin-echo imaging, are discussed along with their practical advantages and limitations. Post-treatment imaging following microvascular decompression and stereotactic radiosurgery is also addressed. A systematic high-resolution magnetic resonance imaging approach can improve diagnostic confidence, facilitate treatment planning, and aid assessment of postoperative or recurrent symptoms.

Keywords: trigeminal neuralgia, magnetic resonance imaging, neurovascular compression, trigeminal nerve, microvascular decompression

Introduction

Trigeminal neuralgia (TN) is a debilitating craniofacial pain disorder characterized by recurrent episodes of sudden, unilateral, electric shock-like pain within the sensory distribution of the trigeminal nerve. It is widely regarded as one of the most intense pain syndromes encountered in clinical practice. According to the current diagnostic classification, possible TN is diagnosed when paroxysmal pain is confined to one or more divisions of the trigeminal nerve, involving the facial or intraoral region (1). Once this diagnosis is considered, the presence of characteristic trigger factors should be assessed to establish clinically confirmed TN. In most patients, pain attacks are precipitated by innocuous mechanical stimuli affecting the trigeminal territory, including light touch, a gentle breeze, oral manipulation, facial movements, or other tactile stimulation (1, 2).

Based on clinical evaluation and imaging findings, TN is classified into three categories (1, 2):

- Classical TN – magnetic resonance imaging (MRI) demonstrates neurovascular compression associated with morphological changes of the trigeminal nerve root; this subtype accounts for approximately 75–80% of cases (3, 4).
- Secondary TN – Pain is attributable to an underlying neurological disorder identified on MRI or other diagnostic investigations and represents nearly 15% of cases (3, 4).
- Idiopathic TN – No structural cause can be identified despite appropriate clinical and radiological evaluation, accounting for approximately 5–10% of patients (3, 4).

The diagnosis of classical TN requires demonstration of neurovascular compression producing structural alterations of the trigeminal nerve root on MRI (1). In contrast, patients with vascular contact in the absence of nerve deformation or distortion are currently classified as having idiopathic TN. This represents a change from previous classifications, in which isolated neurovascular contact without morphological nerve changes was often included within the classical subtype (5).

Anatomy of the trigeminal nerve

The trigeminal nerve consists of three sensory nuclei—the mesencephalic, principal (main) sensory, and spinal trigeminal nuclei—and one motor nucleus. Sensory fibers arising from the ophthalmic (V1), maxillary (V2), and mandibular (V3) divisions converge at Meckel's cave, pass through the porus trigeminus, traverse the prepontine cistern, and enter the pons at the root entry region. The motor root originates from the anterolateral aspect of the pons and occupies a superomedial position relative to the sensory fibers along the cisternal segment.

As the nerve courses through the cerebellopontine cistern, its myelin covering changes from Schwann cell-derived peripheral myelin **to** oligodendrocyte-derived central myelin. This interface is known as the transition zone (TZ) or Obersteiner–Redlich zone, where the centrally myelinated portion extends distally within the core of the nerve root (5). The term "transition zone" should not be confused with the root entry zone (REZ). Although these terms are sometimes used interchangeably, their definitions differ across the literature. In several publications, the REZ specifically refers to a broader anatomical region encompassing the TZ, the centrally myelinated root segment, and the adjacent brainstem surface; therefore, they should not be regarded as synonymous (6, 7).

Neurovascular compression involving the proximal cisternal segment, where the nerve is invested by central myelin, is considered the most clinically significant. Although the exact mechanism remains uncertain, central myelin is believed to be more susceptible to injury because it is relatively thinner than peripheral myelin for a given axonal diameter and possesses a comparatively limited capacity for repair following damage (7, 8).

Anatomical studies have demonstrated slight variability in the location of the TZ. Peker et al. reported that it lies within 2.5 mm of the brainstem surface (9), whereas Guclu et al. measured its average distance at 4.19 ± 0.81 mm (6). To facilitate radiological assessment, Haller et al. subsequently recommended using 3 mm from the brainstem as a practical landmark for defining the TZ during evaluation of neurovascular compression on MRI (7).

Neurovascular conflict and morphological changes of the trigeminal nerve root

The concept that vascular compression may underlie TN was first proposed by Dandy in 1934. Subsequently, Jannetta et al. established that compression of the trigeminal nerve root by an adjacent vessel at the root entry region is the pathological basis of classical TN and introduced microvascular decompression (MVD) as an effective surgical treatment for this condition (10).

Among the offending vessels, the superior cerebellar artery (SCA) is encountered most frequently and usually courses superior to the trigeminal nerve, whereas the anterior inferior cerebellar artery (AICA) typically approaches the nerve from below (6, 7, 11). Less common vascular causes include the basilar artery, vertebral artery (12), posterior communicating artery aneurysms (13), persistent trigeminal artery (7, 14), arteriovenous malformations (15), and venous structures (16). Although venous neurovascular compression is relatively uncommon, several veins have been implicated, including the transverse pontine vein, pontotrigeminal vein, cerebellopontine fissure veins, middle cerebellar peduncle veins, and lateral mesencephalic vein (16, 17). A relatively small cerebellopontine angle cistern has also been proposed as a predisposing anatomical factor for neurovascular compression (7). However, vascular contact between a vessel and the trigeminal nerve is frequently observed in asymptomatic individuals, with reported rates ranging from 25% to 74% (18). Consequently, the presence of vascular contact on MRI should be interpreted in conjunction with the patient's clinical presentation and associated imaging findings rather than being considered diagnostic in isolation.

Several MRI features increase the likelihood that a neurovascular contact is clinically significant (12):

- Contact occurring at the TZ located between central and peripheral myelin
- Associated morphological alterations of the trigeminal nerve, including displacement or atrophy
- An offending vessel
- Perpendicular orientation of the vascular contact with the nerve

On MRI, neurovascular conflict is commonly categorized into three grades (17):

- Grade I: Simple vascular contact without alteration of the nerve.
- Grade II: Displacement or distortion of the trigeminal nerve root.
- Grade III: Marked indentation of the nerve associated with nerve atrophy.

Because vascular loops adjacent to the trigeminal nerve are frequently identified in healthy individuals as well as on the asymptomatic side in patients with TN, distinguishing neurovascular contact from clinically relevant neurovascular conflict is essential (19–22). Imaging findings that favor a symptomatic conflict include compression occurring within 3 mm of the REZ, associated nerve displacement, and trigeminal nerve atrophy (19–22).

Yoshino et al. demonstrated a somatotopic arrangement of trigeminal nerve fibers at the REZ and showed that the location of vascular compression correlates with the clinical distribution of pain. Compression along the medial aspect of the nerve root is more commonly associated with maxillary (V2) symptoms, whereas lateral compression tends to produce mandibular (V3) neuralgia (12, 23).

The principal structural abnormalities associated with neurovascular conflict are nerve displacement (or distortion) and trigeminal nerve atrophy. Although simple neurovascular contact may also be encountered in individuals without TN, distortion of the nerve by the offending vessel is considered highly specific for symptomatic disease (24–27).

MRI studies have consistently demonstrated that the affected trigeminal nerve exhibits a smaller diameter and reduced cross-sectional area compared with the contralateral asymptomatic nerve (18, 28–30). These changes are believed to reflect chronic demyelination and axonal loss resulting from persistent vascular compression (29). Accordingly, the presence of trigeminal nerve atrophy in conjunction with neurovascular compression substantially increases the likelihood that the neurovascular conflict is clinically significant (18). Representative examples of neurovascular conflict causing TN are illustrated in **Figure 1**.

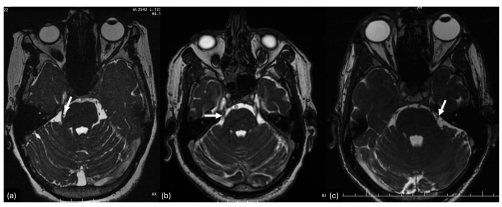

FIGURE 1 | (a) Axial FIESTA image shows the right superior cerebellar artery (SCA) indenting and displacing the cisternal segment of the right trigeminal nerve with kinking of the transitional zone (arrow). **(b)** Axial FIESTA image shows atrophy of the right trigeminal nerve cisternal segment (arrow). **(c)** Axial FIESTA image shows displacement and kinking of the left trigeminal nerve due to the left Dandy vein as well as the left SCA causing neurovascular conflict (arrow).

Secondary trigeminal neuralgia

Secondary TN refers to facial pain arising from an identifiable structural lesion affecting the trigeminal nerve or its central pathways, in contrast to classical TN, which is most often related to neurovascular compression. Neuroimaging is therefore essential in all patients with suspected atypical TN and in those presenting with red flag features such as younger age at onset, sensory deficits, bilateral symptoms, or poor response to medical therapy.

The spectrum of secondary causes is broad. Neoplastic lesions are among the most common etiologies and include cerebellopontine angle schwannomas, meningiomas, epidermoid cysts, and metastatic deposits (31). These lesions may cause TN through direct compression, displacement, or infiltration of the nerve. Imaging findings typically include a mass lesion with variable signal characteristics and contrast enhancement, often associated with distortion of the nerve REZ. Examples of these lesions are shown in **Figures 2** and **3**. Perineural tumor spread should be specifically sought, particularly in head and neck malignancies, and is characterized by nerve thickening, abnormal enhancement, and extension along the trigeminal divisions through skull base foramina.

Demyelinating disease, particularly multiple sclerosis, represents another important cause of secondary TN (31). In such cases, plaques involving the intrapontine trigeminal pathways or REZ are typically identified as T2/fluid-attenuated inversion recovery (FLAIR) hyperintense lesions, occasionally demonstrating contrast enhancement in active disease (**Figure 4**). TN may be the presenting manifestation in a subset of these patients.

Vascular causes other than classical neurovascular compression include arteriovenous malformations, aneurysms, and vertebrobasilar dolichoectasia, which may exert a mass effect on the trigeminal nerve. Examples of vascular malformations causing TN are shown in **Figures 5** and **6**.

Inflammatory and infectious processes, including granulomatous diseases and chronic meningitis, can also involve the trigeminal nerve, often manifesting as nerve enhancement and thickening on post-contrast imaging (31). An example of trigeminal neuritis is shown in **Figure 7**.

Imaging protocol

MRI is the imaging modality of choice for evaluating patients with TN because it provides excellent visualization

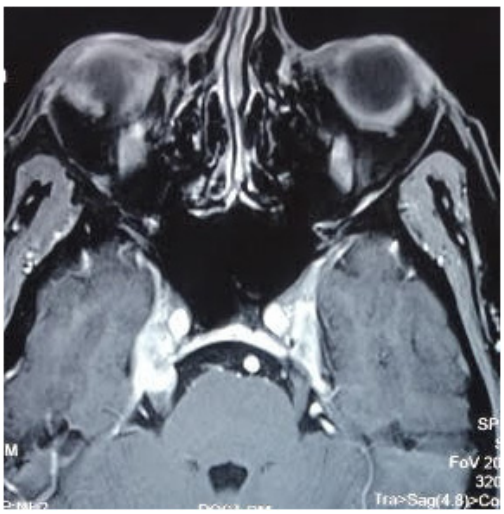

FIGURE 2 | Axial post-contrast T1 W image shows an enhancing extra-axial lesion involving the right cavernous sinus and extending along the right trigeminal nerve, suggestive of a right trigeminal schwannoma.

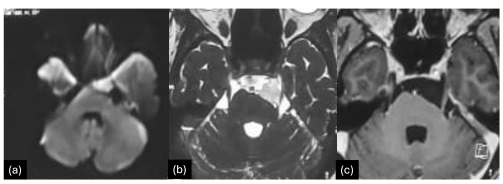

FIGURE 3 | (a) Axial diffusion-weighted imaging (DWI) image shows a lesion in the Pre-pontine cistern showing diffusion restriction. **(b)** Axial FIESTA image shows the lesion has a T2 hyperintense signal and is displacing and compressing the cisternal segment of the left trigeminal nerve. **(c)** Post-contrast axial T1W image shows the lesion does not show any contrast enhancement. Imaging features suggestive of an epidermoid cyst in the prepontine cistern.

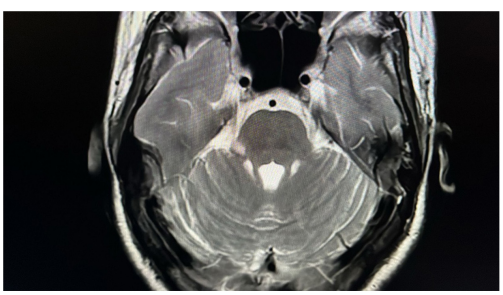

FIGURE 4 | Axial T2 W image shows T2 hyperintensity along the right trigeminal nerve nucleus. The patient was a known case of multiple sclerosis and presented with demyelination-related TN.

of the brainstem, skull base, trigeminal nerve, and associated intracranial pathologies.

Although several MRI techniques can demonstrate structural abnormalities of the trigeminal nerve and identify neurovascular compression, imaging should always be interpreted in the context of an established clinical diagnosis. MRI has the greatest diagnostic value when performed in patients with clinical features suggestive of TN rather than

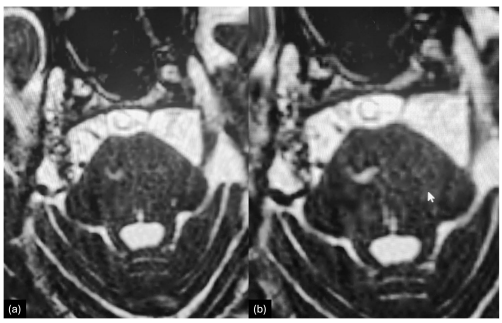

FIGURE 5 | (a, b) Axial FIESTA image shows flow voids along the right trigeminal nerve cisternal segment, suggestive of trigeminal nerve arteriovenous malformation. Image courtesy: Dr. Nirmalya Ray (DM-Neuroimaging and Interventional Neuroradiology).

as a screening investigation. For patients with suspected classical TN, the recommended imaging protocol combines high-resolution heavily T2-weighted three-dimensional (3D) sequences, 3D time-of-flight (TOF) MR angiography, and contrast-enhanced 3D T1-weighted imaging to comprehensively evaluate the trigeminal nerve and adjacent vascular structures (7, 17). In addition, advanced techniques such as diffusion tensor imaging (DTI) with tractography have shown promise for assessing microstructural nerve alterations and may serve as imaging biomarkers for disease severity and treatment response (7, 32).

Role of various MRI sequences in classical trigeminal neuralgia

Whenever available, 3-Tesla MRI should be preferred because its superior spatial resolution and signal-to-noise ratio allow more reliable visualization of the trigeminal nerve and adjacent neurovascular structures. Modern digitally transmitted 1.5-T systems may also provide excellent image quality, although 3T is particularly advantageous when evaluating subtle abnormalities associated with secondary TN, including perineural tumor spread (33).

Routine brain MRI sequences

A comprehensive MRI examination should include routine brain sequences comprising axial T1-weighted, T2-weighted, FLAIR, diffusion-weighted imaging (DWI), and susceptibility-weighted imaging (SWI) images, together with coronal and sagittal T2-weighted acquisitions. These sequences primarily serve to identify structural causes of secondary TN, such as demyelinating plaques, cavernous malformations, infarction, intracranial neoplasms, vascular malformations, or aneurysms. Whenever feasible, imaging should also extend inferiorly to approximately the C4 spinal level to assess the spinal trigeminal nucleus and tract for associated pathology.

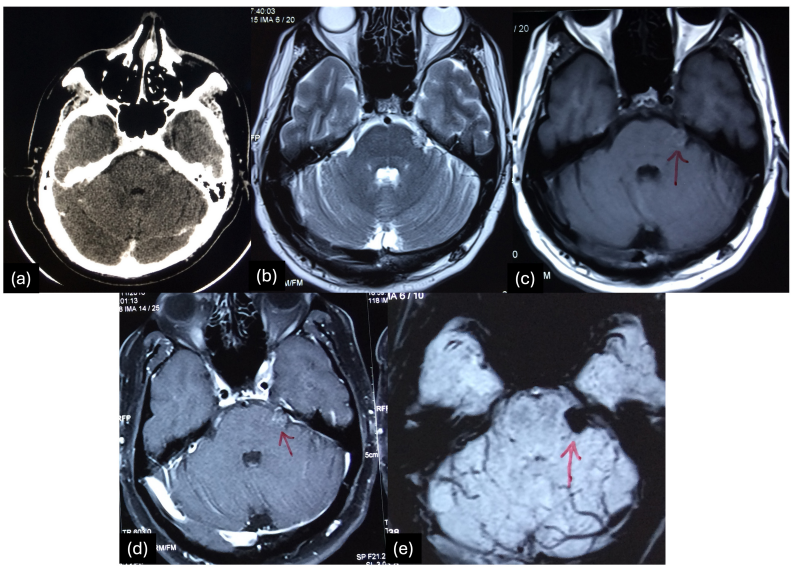

FIGURE 6 | (a) Axial contrast-enhanced CT shows a hyperdense lesion in the left cerebellopontine angle cistern in the path of the left trigeminal nerve cisternal segment. **(b)** T2W axial image shows a T2 heterogenous lesion with a T2 hypointense rim in the cisternal segment of the left trigeminal nerve. **(c)** An axial T1W image shows that the lesion has a mixed signal on T1-weighted sequence. **(d)** Axial post-contrast T1W image shows no significant enhancement. **(e)** The GRE axial image shows blooming within the lesion. Imaging features suggest a cavernoma along the left trigeminal nerve.

High-resolution heavily T2-weighted 3D sequences

High-resolution three-dimensional heavily T2-weighted imaging forms the cornerstone of MRI evaluation in classical TN because it provides a detailed depiction of the cisternal segment of the trigeminal nerve throughout its course. On these sequences, both the nerve and adjacent vessels appear hypointense against the bright cerebrospinal fluid, allowing precise assessment of neurovascular relationships.

These sequences typically achieve submillimeter isotropic resolution, with slice thickness and voxel dimensions of less than 1 mm, thereby enabling high-quality multiplanar reconstructions (34–36). They may be acquired using either fast gradient-echo or fast spin-echo (FSE) techniques (37).

Balanced steady-state free precession sequences, including True-fast imaging with steady state free precession (FISP), fast imaging employing steady state acquisition (FIESTA), and balanced fast-field echo (bFFE), provide excellent contrast between cerebrospinal fluid and cranial nerves but are susceptible to magnetic field inhomogeneity, which produces characteristic banding artifacts. These artifacts can be reduced by performing two acquisitions one with and the other without phase frequency alternations in the constructive interference into steady-state and fast imaging, employing steady-state acquisition with phase cycling sequences (constructive interference in steady state [CISS], FIESTA-C) (28–30, 34). However, these phase-cycled acquisitions increase scan time because two datasets are acquired and subsequently combined to reduce banding artifacts (38).

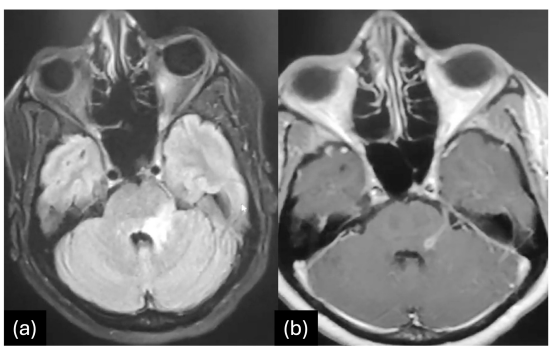

FIGURE 7 | (a) Axial fluid-attenuated inversion recovery (FLAIR) image shows FLAIR hyperintensity along the left trigeminal nerve nucleus in its pontine course. **(b)** An axial T1W image shows enhancement along the left trigeminal nerve and its nucleus. This patient had herpetic trigeminal neuritis and presented with neuralgia. Image courtesy: Dr. Safwan Ahmad (DM-Neurology).

Alternatively, high-resolution isotropic 3D FSE techniques, including sampling perfection with application-optimized contrasts using different flip-angle evolutions (SPACE), volume isotropic turbo spin echo acquisition (VISTA), and a 3D variable-flip-angle fast/turbo spin-echo sequence (vendor-specific terminology) (CUBE), provide excellent visualization of the cisternal cranial nerves (37). These sequences employ prolonged echo trains, variable refocusing flip angles, and non-selective radiofrequency pulses to reduce image blurring while minimizing flow-related and chemical shift artifacts. Consequently, they achieve isotropic spatial resolution approaching 0.3 mm together with excellent contrast between the nerve and surrounding cerebrospinal fluid (39, 40).

At present, high-resolution 3D cisternographic imaging represents the principal MRI technique for evaluating neurovascular compression. The isotropic volumetric datasets permit multiplanar reformation and comprehensive assessment of the trigeminal nerve, TZ, and adjacent vascular structures throughout their entire course (7, 41, 42).

Contrast-enhanced CISS imaging: Three-dimensional CISS sequences possess mixed T1/T2 weighting, allowing excellent visualization of the CSF-filled cisternal compartment while remaining compatible with intravenous gadolinium administration. Conventional balanced steady-state free precession (SSFP) techniques such as CISS and FIESTA demonstrate limited intrinsic contrast between nerves and vessels because both appear hypointense, making characterization of high-grade neurovascular compression difficult when cerebrospinal fluid is minimal or absent.

Administration of gadolinium improves delineation of adjacent vascular structures and facilitates identification of clinically significant neurovascular conflict (43). Previous studies have also shown that contrast-enhanced 3D-CISS improves prediction of the symptomatic side and correlates more closely with favorable surgical outcomes following MVD (43).

During interpretation of high-resolution 3D T2-weighted images, the following parameters should be systematically documented (43):

- Type of offending vessel (artery, vein, or both)
- Surface of the trigeminal nerve involved (superior, inferior, medial, or lateral)
- Grade of neurovascular conflict (Grade 0–3)
- Cross-sectional area of the trigeminal nerve at the compression site
- Orthogonal nerve dimensions to assess flattening
- Distance between the neurovascular conflict and the apparent pontine origin of the trigeminal nerve

MR angiography

Three-dimensional time-of-flight (3D TOF) MR angiography remains the preferred angiographic technique for evaluating arterial neurovascular compression. The sequence exploits flow-related enhancement so that flowing blood appears hyperintense, whereas stationary tissues demonstrate relatively low signal intensity. Consequently, intracranial arteries are well depicted on native TOF images, while the trigeminal nerve is less conspicuous because of its intermediate signal intensity (44).

Maximum intensity projection (MIP) reconstructions facilitate evaluation of the vertebrobasilar circulation, and post-processing techniques can suppress carotid circulation to improve visualization of posterior fossa vessels. Venous signal may also be reduced using presaturation bands (44). Nevertheless, interpretation may occasionally be complicated by intrinsic T1 hyperintensity of stationary tissues or signal loss resulting from blood flowing out of the imaging volume.

Despite its clinical utility, the specificity of TOF-MRA for detecting symptomatic neurovascular compression remains limited (45, 46). Small vessels measuring less than 1 mm may not be visualized because of slice thickness and partial-volume averaging. In addition, turbulent blood flow can generate phase-related signal loss in larger arteries, while vessel wall stiffness may reduce the apparent caliber of compressing arteries. These technical limitations contribute to false-negative examinations and explain why TOF-MRA alone cannot be considered the reference standard for identifying the offending vessel before surgery.

Contrast-enhanced 3D T1-weighted MRI

High-resolution contrast-enhanced 3D T1-weighted imaging complements TOF-MRA by providing a clear depiction of both arteries and veins following gadolinium administration. In contrast to TOF imaging, where predominantly arterial flow is visualized, gadolinium-enhanced T1-weighted sequences demonstrate enhancement of the entire vascular compartment while the trigeminal nerve maintains intermediate signal intensity against hypointense cerebrospinal fluid.

Accordingly, combining 3D TOF-MRA with contrast-enhanced 3D T1-weighted MRI improves differentiation between arterial and venous neurovascular compression. Korogi et al. further demonstrated that gadolinium-enhanced MR angiography can identify offending veins that may not be evident on non-contrast studies and therefore recommended contrast-enhanced angiography when vascular compression is not demonstrated on routine imaging (47).

Diffusion tensor imaging (DTI)

DTI with tractography has emerged as a valuable adjunct for evaluating patients with TN by providing insights into the microstructural integrity of the trigeminal nerve. Unlike conventional MRI, DTI characterizes tissue architecture by measuring the directionality and magnitude of water diffusion within nerve fibers, thereby allowing the detection of subtle abnormalities that may not be visible on routine anatomical sequences (7, 48, 49).

Patients with classical TN typically demonstrate reduced anisotropic diffusion within the affected trigeminal nerve. Quantitative DTI metrics reveal a decrease in fractional

anisotropy (FA) together with increased radial diffusivity, apparent diffusion coefficient (ADC), and mean diffusivity, reflecting microstructural alterations associated with neurovascular compression (7). Interestingly, FA values have been shown to improve following successful MVD, suggesting that these changes may be at least partially reversible. Furthermore, several studies have reported a correlation between DTI-derived parameters, disease duration, and pain severity measured using the visual analogue scale (VAS), highlighting the potential role of FA as an objective imaging biomarker for disease activity and therapeutic response (50). In one study, the symptomatic trigeminal nerve demonstrated significantly reduced FA with increased radial diffusivity, while axial diffusivity remained unchanged, supporting demyelination rather than primary axonal injury as the predominant pathological mechanism in TN (51).

The MRI sequences commonly employed in the evaluation of TN are summarized in **Table 1**.

TABLE 1 | Different magnetic resonance imaging (MRI) sequences useful in imaging of trigeminal neuralgia (TN).

Pulse sequences	Product sequences of different vendors
High-resolution heavily T2 weighted 3D sequence	
3D heavily T2-weighted fast gradient echo	3D fully refocussed balanced steady state free precession: True-FISP(Siemens), FIESTA (GE), bFFE (Philips) True-FISP acquisitions performed with alternating and non-alternating RF phase cycling CISS (Siemens) with and without contrast, FIESTA-C (GE)
3D heavily T2-weighted fast spin echo	Driven-equilibrium technique: longitudinal magnetization is rapidly restored by applying a 90° RF pulse at the end of the echo train RESTORE(Siemens), FRFSE(Fast recovery fast spin echo, GE), DRIVE (Philips) Special 3D FSE techniques sampling perfection with application-optimized contrasts using different flip-angle evolutions (SPACE) (Siemens), a 3D variable-flip-angle fast/turbo spin-echo sequence (vendor-specific terminology) (CUBE) (GE), volume isotropic turbo spin echo acquisition (VISTA) (Philips)
3D Time-of-flight (TOF) MR angiography	For assessment of arteries
3D Post contrast T1 weighted MRI	For assessment of arteries and veins
Diffusion tensor imaging (DTI)	Tractography, Assessment of Fractional Anisotrophy, Radial Diffusion coefficient, Axial Diffusion coefficient, Apparent Diffusion coefficient (ADC), Mean Diffusion coefficient

Quantitative imaging

Quantitative MRI techniques have demonstrated that patients with TN exhibit a reduction in trigeminal nerve volume and cross-sectional area on the symptomatic side compared with the contralateral nerve or healthy individuals (3, 52, 53).

Post-treatment imaging

The initial management of classical TN consists of pharmacological therapy. Patients who continue to experience disabling pain despite optimal medical treatment are considered to have refractory TN (54). For these patients, MVD remains the preferred surgical treatment, as it relieves neurovascular conflict by separating the offending vessel from the trigeminal nerve using a Teflon pledget or a vascular sling. Other treatment options include percutaneous procedures targeting the Gasserian ganglion and Gamma Knife radiosurgery (GKRS) directed at the affected trigeminal nerve root (55).

Post-microvascular decompression imaging

Postoperative imaging is primarily performed to assess the effectiveness of decompression and to identify early or delayed complications. On high-resolution CISS sequences, the Teflon pledget typically appears as a non-enhancing hypointense structure interposed between the trigeminal nerve and the offending vessel. Occasionally, displacement of the pledget may be identified. Early postoperative complications include hemorrhage and infection, whereas delayed complications include the development of a Teflon granuloma, which represents a foreign-body inflammatory reaction. Teflon granulomas often demonstrate contrast enhancement and may contain calcifications, producing recurrent mass effect on the trigeminal nerve (56). The identification of Teflon granuloma, recurrent neurovascular compression, or arachnoid adhesions on postoperative imaging may provide an anatomical explanation for persistent or recurrent TN and aid in determining the need for repeat MVD (57).

Other post-treatment imaging findings

Following GKRS, persistent contrast enhancement of the treated trigeminal nerve segment may be observed on follow-up MRI. This imaging finding has been associated with

ongoing facial pain as well as trigeminal sensory deficits in some patients (58).

Conclusion

Classical TN is characterized by recurrent episodes of sudden, severe, unilateral facial pain resulting from neurovascular compression of the trigeminal nerve. MRI plays a pivotal role in the evaluation of these patients by confirming neurovascular conflict, demonstrating associated morphological changes of the trigeminal nerve, excluding secondary causes, and guiding treatment planning. Advances in high-resolution MRI techniques and quantitative imaging have further improved the ability to characterize the site and severity of neurovascular compression and to assess post-treatment changes, thereby contributing to more accurate diagnosis and optimal patient management.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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