ORIGINAL RESEARCH PAPER

Anatomy

KEY WORDS: Trigeminal Neuralgia; Neurovascular compression; Root entry zone; Superior Cerebellar Artery;

NEUROVASCULAR COMPRESSION IN TRIGEMINAL NEURALGIA: A CONCISE REVIEW

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ABSTRACT

Trigeminal Neuralgia is characterized by recurrent, paroxysmal shock-like facial pain, most commonly attributed to neurovascular compression at the trigeminal nerve root entry zone. Chronic pulsatile compression, typically by the Superior Cerebellar Artery, leads to focal demyelination, ephaptic transmission, and neuronal hyperexcitability.High-resolution MRI techniques, including 3D-CISS and FIESTA, have improved detection of neurovascular conflict; however, clinicoradiological correlation remains essential due to the presence of asymptomatic vascular contact. Management is guided by this pathophysiology, with Carbamazepine as first-line therapy and Microvascular Decompression providing definitive treatment in selected patients. This review summarizes the anatomical basis, pathophysiological mechanisms, imaging features, and therapeutic implications of neurovascular compression in trigeminal neuralgia.

INTRODUCTION:

Trigeminal Neuralgia (TN) is a debilitating craniofacial pain syndrome characterized by recurrent, unilateral, brief episodes of severe, electric shock-like pain distributed along one or more divisions of the trigeminal nerve. The condition has an estimated annual incidence of 4-13 per 100,000 individuals and predominantly affects individuals over the age of 50 years, with a slight female preponderance [1,2]. Clinically, TN is broadly classified into classical, secondary, and idiopathic forms. Among these, classical trigeminal neuralgia is most strongly associated with neurovascular compression (NVC) at the level of the trigeminal nerve root entry zone (REZ), a transitional region between central and peripheral myelin. This anatomical vulnerability renders the nerve particularly susceptible to focal demyelination when subjected to chronic pulsatile vascular contact [3,4]. The neurovascular compression hypothesis, first popularized by Peter J. Jannetta, remains the most widely accepted pathophysiological mechanism underlying classical TN. According to this model, compression of the trigeminal nerve by adjacent vascular structures, most commonly the Superior Cerebellar Artery, leads to focal demyelination, resulting in aberrant electrical transmission, ephaptic cross-talk, and hyperexcitability of trigeminal afferents [3,5].

Advances in high-resolution magnetic resonance imaging (MRI), particularly three-dimensional constructive interference in steady state (3D-CISS) and fast imaging employing steady-state acquisition (FIESTA) sequences, have significantly improved the visualization of neurovascular relationships in the cerebellopontine angle. These developments have not only strengthened the anatomical basis of the NVC theory but also enhanced preoperative planning for surgical interventions such as microvascular decompression [6,7]. Despite the strong association between NVC and TN, several unresolved questions persist. Notably, neurovascular contact can also be observed in asymptomatic individuals, raising important considerations regarding the clinical significance, severity, and morphological characteristics of compression required to produce symptoms. Furthermore, the role of venous compression, the contribution of central sensitization, and the variability in treatment response continue to be areas of ongoing investigation [8]. In this narrative review, we aim to provide a comprehensive and clinically integrated overview of neurovascular compression in trigeminal neuralgia, encompassing its anatomical basis, pathophysiological mechanisms, imaging correlates, and therapeutic implications. Particular emphasis is placed on bridging the gap between structural findings and clinical decision-making, with the goal of enhancing diagnostic accuracy and optimizing patient-specific management strategies.

SURGICAL NEUROANATOMY OF THE TRIGEMINAL NERVE AND NEUROVASCULAR RELATIONSHIPS:

The trigeminal nerve (cranial nerve V) emerges from the lateral aspect of the pons and traverses the cerebellopontine angle (CPA) cistern before entering Meckel's cave, where it divides into its three major branches. Of particular clinical relevance in Trigeminal Neuralgia is the proximal segment of the nerve, especially the root entry zone (REZ), which represents a transitional interface between central myelin, produced by oligodendrocytes, and peripheral myelin, produced by Schwann cells. This transition zone typically extends for a few millimeters from the pial surface and is structurally vulnerable to mechanical and pulsatile stress, making it the most common site implicated in neurovascular compression [9,10]. Within the CPA, the trigeminal nerve maintains close anatomical relationships with several vascular structures. The Superior Cerebellar Artery is most frequently identified as the offending vessel in cases of classical trigeminal neuralgia, typically looping superiorly or superomedially to the nerve and exerting compressive forces at or near the REZ. Less commonly, compression may arise from the anterior inferior cerebellar artery (AICA), basilar artery, or venous structures such as the petrosal vein complex. The anatomical configuration of these vessels, including their tortuosity, elongation, and proximity to the nerve, plays a critical role in determining the likelihood and severity of compression [4,10].

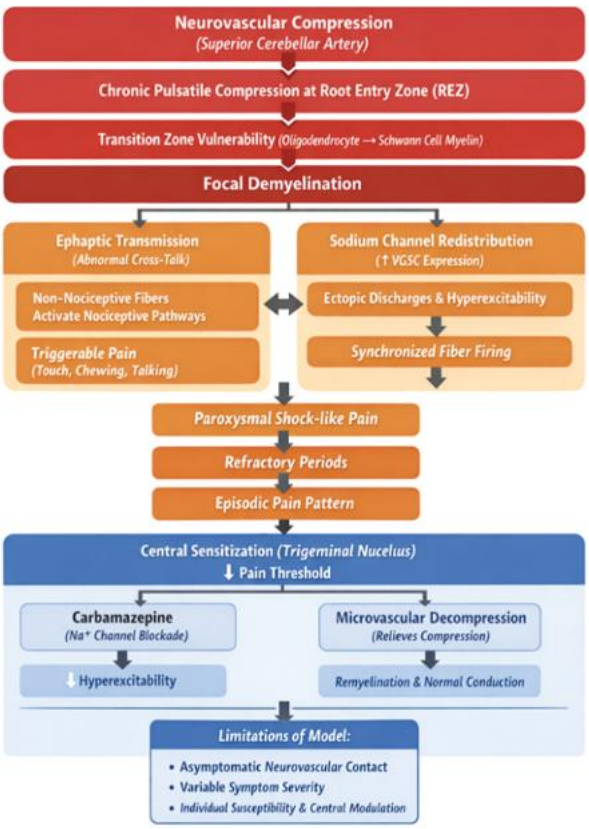
From a surgical perspective, the distinction between mere neurovascular contact and true compression is crucial. Compression is generally characterized by distortion, indentation, or displacement of the trigeminal nerve, whereas simple contact may be observed in asymptomatic individuals. The direction of vascular contact also holds significance; superior and superomedial compressions are more commonly symptomatic due to their proximity to the REZ. Additionally, age-related vascular changes such as arterial elongation and atherosclerotic rigidity may increase the propensity for sustained pulsatile compression [4]. Understanding this microsurgical anatomy is fundamental for procedures such as Microvascular Decompression, where

precise identification of the offending vessel and its relationship to the nerve determines surgical success. The goal of such intervention is not merely to separate the vessel from the nerve, but to achieve durable decompression while preserving neural integrity, thereby directly addressing the anatomical substrate of pain generation [11].

PATHOPHYSIOLOGICAL MECHANISMS OF NEUROVASCULAR COMPRESSION:

The pathophysiology of pain generation in Trigeminal Neuralgia is best explained by the neurovascular compression induced focal demyelination model, which integrates structural injury with functional neuronal hyperexcitability. Chronic pulsatile compression at the root entry zone (REZ), most commonly by the Superior Cerebellar Artery, leads to localized disruption of the myelin sheath. This segment is inherently susceptible due to the transition from oligodendrocyte-derived central myelin to Schwann cell derived peripheral myelin, resulting in reduced mechanical resilience and increased vulnerability to injury [3,5]. Focal demyelination at the REZ facilitates abnormal electrical interactions between adjacent nerve fibers, a phenomenon termed ephaptic transmission. In this state, action potentials can aberrantly "jump" between neighboring axons without synaptic mediation, allowing non-nociceptive fibers, such as those transmitting light touch, to activate nociceptive pathways. This mechanism explains the characteristic triggerability of pain in trigeminal neuralgia, where innocuous stimuli such as talking, chewing, or facial touch precipitate paroxysmal pain episodes. In addition to ephaptic cross-talk, demyelinated segments exhibit increased expression and redistribution of voltage-gated sodium channels, leading to ectopic impulse generation and heightened neuronal excitability. These hyperexcitable foci can generate spontaneous discharges or amplify incoming signals, contributing to the sudden, shock-like nature of pain. The paroxysmal pattern is likely due to synchronized discharges from clusters of hyperexcitable fibers, followed by refractory periods that transiently limit further firing [3].

Figure 1: Pathophysiology of Trigeminal Neutralgia.

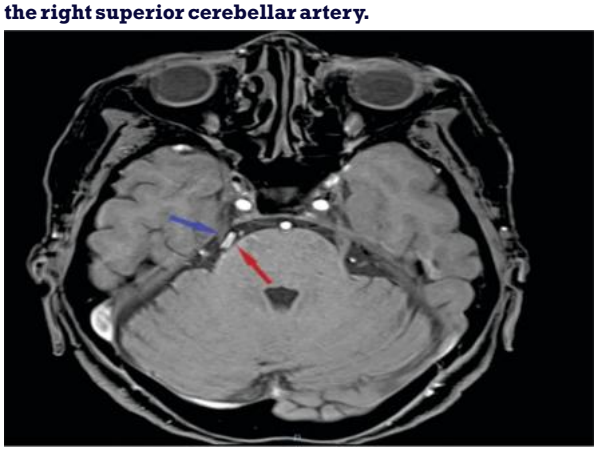


With disease progression, central mechanisms also become relevant. Persistent peripheral input may induce sensitization within the trigeminal nucleus in the brainstem, lowering activation thresholds and facilitating sustained pain perception. However, unlike other neuropathic pain syndromes, trigeminal neuralgia remains predominantly characterized by episodic rather than continuous pain, reflecting the primary role of peripheral ephaptic mechanisms rather than widespread central sensitization [2]. Importantly, this pathophysiological framework also explains the therapeutic efficacy of membrane-stabilizing agents such as Carbamazepine, which reduce neuronal hyperexcitability by inhibiting voltage-gated sodium channels. Furthermore, it provides the mechanistic basis for surgical interventions such as Microvascular Decompression, which directly eliminate the inciting compressive force, thereby allowing potential remyelination and restoration of normal impulse conduction [3]. Despite its broad acceptance, the neurovascular compression model does not fully account for all clinical observations, particularly the presence of asymptomatic neurovascular contact and variability in symptom severity. These discrepancies suggest that additional factors, including individual susceptibility, microstructural nerve changes, and central processing differences, likely modulate the clinical expression of the disease.

IMAGING EVALUATION OF NEUROVASCULAR COMPRESSION:

High-resolution magnetic resonance imaging (MRI) has become indispensable in the evaluation of Trigeminal Neuralgia, particularly for identifying neurovascular compression and excluding secondary causes. The primary objective of imaging is twofold: to demonstrate the anatomical relationship between the trigeminal nerve and adjacent vascular structures, and to assess morphological changes within the nerve that may correlate with symptomatology [6,7]. Advanced MRI sequences such as three-dimensional constructive interference in steady state (3D-CISS) and fast imaging employing steady-state acquisition (FIESTA) provide excellent spatial resolution and contrast between cerebrospinal fluid, cranial nerves, and vascular structures within the cerebellopontine angle. These heavily T2-weighted sequences allow precise visualization of the trigeminal nerve along its cisternal course, particularly at the root entry zone, where neurovascular conflict is most clinically relevant. When combined with MR angiography, these techniques enhance the identification of the offending vessel, most commonly the Superior Cerebellar Artery (Figure 2) [6,10].

Figure 2: Compression of the right trigeminal nerve by the right superior cerebellar artery.



Radiologically, neurovascular relationships are categorized based on the degree of interaction between the vessel and nerve. Simple contact refers to close apposition without distortion, whereas true compression is characterized by displacement, indentation, or thinning of the nerve.

Additional findings such as nerve atrophy or signal alteration may indicate chronicity and have been associated with more severe or long-standing disease. Importantly, the location and direction of compression, particularly superior or superomedial contact at the REZ are more strongly correlated with symptomatic trigeminal neuralgia [6,7]. However, a critical limitation in imaging interpretation lies in the relatively high prevalence of neurovascular contact in asymptomatic individuals. This underscores the necessity of correlating imaging findings with clinical presentation rather than relying solely on radiological evidence. The mere presence of vascular contact does not establish causation; rather, features such as nerve deformation, vascular loop configuration, and concordance with the side and distribution of pain increase diagnostic confidence [8]. From a management standpoint, imaging plays a pivotal role in patient selection for surgical intervention. Demonstration of clear arterial compression with corresponding clinical features strongly favors procedures such as Microvascular Decompression, which directly target the underlying pathology. Conversely, the absence of significant compression or the presence of alternative etiologies may guide clinicians toward medical therapy or ablative procedures. Emerging imaging approaches, including diffusion tensor imaging (DTI) and tractography, have shown promise in detecting microstructural changes within the trigeminal nerve, potentially improving diagnostic accuracy and prognostic stratification. Nevertheless, their routine clinical application remains limited, and further studies are required to validate their role in the standard evaluation of trigeminal neuralgia [7,8].

CLASSIFICATION OF NEUROVASCULAR COMPRESSION AND CLINICAL CORRELATION:

Neurovascular compression in Trigeminal Neuralgia can be broadly classified based on the nature and severity of the interaction between the vessel and the trigeminal nerve, as well as the type of offending vascular structure. From a morphological standpoint, the relationship ranges from simple neurovascular contact, where the vessel is in close proximity without causing structural distortion, to true compression, characterized by indentation, displacement, or thinning of the nerve. More severe forms may demonstrate nerve atrophy, suggesting chronic and sustained insult. This distinction is clinically significant, as symptomatic patients are more likely to exhibit definitive compression rather than mere contact, particularly at the root entry zone [4]. Based on the type of offending vessel, neurovascular compression is categorized into arterial and venous forms. Arterial compression, most commonly by the Superior Cerebellar Artery, is strongly associated with classical trigeminal neuralgia and is more likely to produce significant pulsatile injury leading to focal demyelination. In contrast, venous compression remains more controversial; while it has been implicated in some cases, its pathogenic significance is less consistent, and outcomes following decompression are comparatively variable [4,5]. Clinically, the severity and nature of neurovascular compression correlate with the phenotype of trigeminal neuralgia. Classical trigeminal neuralgia is typically associated with arterial compression at the REZ and presents with paroxysmal, shock-like pain, distinct trigger zones, and absence of objective sensory deficits. In contrast, secondary trigeminal neuralgia arises from identifiable structural or pathological causes such as tumors or demyelinating disorders like Multiple Sclerosis, where pain may be less stereotyped and often accompanied by sensory abnormalities. In such cases, the underlying mechanism is not primarily neurovascular compression but intrinsic nerve pathology [12].

An important clinical consideration is the presence of neurovascular contact in asymptomatic individuals, which challenges the specificity of imaging findings. Therefore, the diagnosis of clinically relevant neurovascular compression requires concordance between radiological evidence and the patient's symptom profile, including side, distribution, and characteristic triggers of pain. Furthermore, the degree of nerve deformation has been shown to correlate with surgical outcomes, with patients demonstrating clear compression being more likely to benefit from procedures such as Microvascular Decompression [12,13].

Overall, the classification of neurovascular compression is not merely descriptive but has direct implications for diagnosis, prognostication, and therapeutic decision-making. A nuanced understanding of these relationships allows for more precise patient selection and individualized management strategies.

MANAGEMENT IMPLICATIONS OF NEUROVASCULAR COMPRESSION:

The management of Trigeminal Neuralgia is fundamentally guided by the underlying pathophysiological mechanism, with neurovascular compression playing a central role in determining both therapeutic strategy and expected outcomes. Treatment approaches can be broadly categorized into medical therapy and procedural interventions, with the choice largely dependent on symptom severity, patient comorbidities, and the presence or absence of demonstrable neurovascular conflict on imaging [3]. Pharmacological therapy remains the first-line treatment, particularly in newly diagnosed cases. The cornerstone of medical management is the use of voltage-gated sodium channel blockers, most notably Carbamazepine, which reduces neuronal hyperexcitability and suppresses ectopic impulse generation. Oxcarbazepine is often preferred in clinical practice due to a more favorable side effect profile, although both agents are highly effective in controlling paroxysmal pain. The therapeutic response to these agents also carries diagnostic value, as classical trigeminal neuralgia typically demonstrates a robust initial response. However, long-term use is frequently limited by adverse effects, tachyphylaxis, or inadequate pain control, necessitating consideration of interventional options [3,13].

In patients with imaging-confirmed arterial compression and medically refractory symptoms, Microvascular Decompression (MVD) is regarded as the gold standard treatment. This procedure directly addresses the underlying etiology by relieving vascular contact at the root entry zone, typically through repositioning of the offending vessel and placement of a protective barrier such as a Teflon pad. MVD offers the highest rates of durable pain relief and the advantage of preserving nerve function, making it particularly suitable for younger, otherwise healthy patients. The success of MVD is closely linked to the presence of clear neurovascular compression, especially when caused by arterial loops such as the Superior Cerebellar Artery [11,14]. For patients who are poor surgical candidates or lack definitive neurovascular compression, ablative procedures provide effective alternatives. These include percutaneous techniques such as radiofrequency rhizotomy, glycerol rhizolysis, and balloon compression, all of which aim to disrupt pain transmission by selectively injuring trigeminal fibers. While these approaches offer rapid pain relief, they are associated with higher recurrence rates and a risk of sensory deficits, including facial numbness and dysesthesia. Stereotactic radiosurgery, such as Gamma Knife, represents a non-invasive option that delivers focused radiation to the trigeminal root, inducing delayed but sustained pain relief with a relatively favorable safety profile [13,14].

Importantly, the presence and characteristics of neurovascular compression significantly influence treatment selection. Patients with clear arterial compression and classical clinical features derive the greatest benefit from decompressive surgery, whereas those with equivocal imaging findings or secondary causes may be better

managed with medical therapy or ablative interventions. Thus, management of trigeminal neuralgia is inherently individualized, requiring integration of clinical presentation, imaging findings, and patient-specific factors.

Emerging strategies aim to refine patient selection and improve outcomes through advanced imaging, intraoperative monitoring, and a better understanding of disease heterogeneity. Nevertheless, the central principle remains that effective treatment hinges on accurately identifying and appropriately addressing the role of neurovascular compression in each patient.

CONCLUSION:

Trigeminal Neuralgia is most commonly driven by neurovascular compression at the root entry zone, leading to focal demyelination and neuronal hyperexcitability. Arterial compression, particularly by the Superior Cerebellar Artery, remains the principal mechanism in classical disease. While imaging has improved detection of neurovascular conflict, clinicoradiological correlation is essential given the presence of asymptomatic contact. Management is guided by this pathophysiology, with medical therapy as first-line and Microvascular Decompression offering definitive treatment in selected patients.

REFERENCES:

1. Gambeta, E., Chichorro, J. G., & Zamponi, G. W. (2020). Trigeminal neuralgia: An overview from pathophysiology to pharmacological treatments. Molecular pain, 16, 1744806920901890. https://doi.org/10.1177/1744806920901890
2. Yan, J., Wang, L., Pan, L., Ye, H., Zhu, X., Feng, Q., Ding, Z., Ge, X., & Shi, L. (2024). Analyzing the risk factors of unilateral trigeminal neuralgia under neurovascular compression. Frontiers in Human Neuroscience, 18, 1349186. https://doi.org/10.3389/fnhum.2024.1349186
3. Szmyd, B., So ek, J., B aszczyk, M., Jankowski, J., Liberski, P. P., Jaskólski, D. J., Wysiadecki, G., Karuga, F. F., Gabryelska, A., Sochal, M., Tubbs, R. S., & Radek, M. (2022). The Underlying Pathogenesis of Neurovascular Compression Syndromes: A Systematic Review. Frontiers in molecular neuroscience, 15, 923089. https://doi.org/10.3389/fnmol.2022.923089
4. Akkara, Y., Thakrar, P., Ahmed, M., Akosa, O., Saroa, A., Das, J. M., & Mendoza, N. (2026). Incidence, role, and imaging-based diagnostic accuracy of neurovascular compression in trigeminal neuralgia: a longitudinal cohort study. British journal of neurosurgery, 40(2), 313–320. https://doi.org/10.1080/02688697.2025.2538484
5. Liu, Y., & Tanaka, E. (2025). Pathogenesis, Diagnosis, and Management of Trigeminal Neuralgia: A Narrative Review. Journal of clinical medicine, 14(2), 528. https://doi.org/10.3390/jcm14020528
6. Henssen, D., van Grinsven, M., Vissers, K., & van Goethem, J. (2025). Magnetic resonance imaging in the diagnosis of trigeminal neuralgia: a systematic review of the imaging protocol and diagnostic accuracy. European radiology, 10.1007/s00330-025-12141-8. Advance online publication. https://doi.org/10.1007/s00330-025-12141-8
7. Petronis, Z., Golubevas, R., Rokicki, J. P., Guzeviciene, V., Sakavicius, D., & Lukosiunas, A. (2025). A systematic review and meta-analysis on trigeminal neuralgia linked to neurovascular compression using MRI analysis. Journal of Current Research in Oral Surgery, 5, 17–24. https://doi.org/10.51847/sptZWIrWeo
8. Kumar, A., Anwar, S. S. M., Bilal, K., Ahmed, A., Samnani, S., & Zafar, B. (2025). Frequency of trigeminal neurovascular contacts identified on 3D-fast imaging employing steady-state acquisition magnetic resonance imaging in asymptomatic adults. Surgical neurology international, x16, 31. https://doi.org/10.25259/SNI_1021_2024
9. Haller, S., Etienne, L., Kövari, E., Varoquaux, A. D., Urbach, H., & Becker, M. (2016). Imaging of Neurovascular Compression Syndromes: Trigeminal Neuralgia, Hemifacial Spasm, Vestibular Paroxysmia, and Glossopharyngeal Neuralgia. AJNR. American journal of neuroradiology, 37(8), 1384–1392. https://doi.org/10.3174/ajnr.A4683
10. Shi, X., Zhang, X., Xu, L., & Xu, Z. (2022). Neurovascular compression syndrome:Trigeminal neuralgia, hemifacial spasm, vestibular paroxysmia, glossopharyngeal neuralgia, four case reports and review of literature. Clinical neurology and neurosurgery, 221, 107401. https://doi.org/10.1016/j.clineuro.2022.107401
11. Myszkowska, B., Zawada-Wi niewska, P., Handzlik, I., & Krakowiak, M. (2025). Neurovascular compression: New evidence reveals better pain management protocols. Clinical Medicine and Health Research Journal, 5(5), 1397–1408. https://doi.org/10.18535/cmhrj.v5i05.505
12. Shankar Kikkeri, N., & Nagalli, S. (2024). Trigeminal Neuralgia. In StatPearls. StatPearls Publishing.
13. Chiotoroiu, A. L., Iordache, M. P., & Buliman, A. (2025). Therapeutic approaches in trigeminal neuralgia – a comprehensive review. Romanian Journal of Neurology/ Revista Romana de Neurologie. Amaltea Medical Publishing House. https://doi.org/10.37897/RJN.2025.3.11
14. Worm, J., Heinskou, T. B., Rochat, P., Springborg, J. B., Smilkov, E. A., Bendtsen, L., Schytz, H. W., & Maarbjerg, S. (2025). Five-year prospective outcomes of medical management and microvascular decompression in trigeminal neuralgia. Journal of neurology, 272(10), 701. https://doi.org/10.1007/s00415-025-13447-9