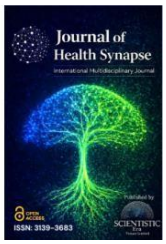
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Trigeminal Neuralgia: A Comprehensive Review of Neuropathic Facial Pain Pathophysiology, Diagnosis, Pharmacotherapy, Surgical Interventions, and Clinical Pharmacy Care

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A B S T R A C T

Trigeminal neuralgia (TN), historically termed tic douloureux, is a severe, chronic neuropathic facial pain disorder characterized by recurrent, abrupt, electric shock-like paroxysms restricted to one or more divisions of the trigeminal nerve (cranial nerve V). Recognized as one of the most excruciating neurological conditions, TN profoundly impairs mastication, speech, oral hygiene, sleep, and psychosocial well-being. Etiologically, the International Classification of Headache Disorders (ICHD-3) categorizes TN into classical, secondary, and idiopathic subtypes. Classical TN is predominantly driven by pulsatile neurovascular compression—most commonly by the superior cerebellar artery—at the trigeminal root entry zone, inducing focal demyelination, voltage-gated sodium channel clustering, ectopic pacemaker discharges, and ephaptic cross-talk between low-threshold mechanoreceptive and nociceptive fibers. Secondary TN arises from identifiable structural or demyelinating pathologies such as multiple sclerosis plaques or cerebellopontine angle tumors. Diagnosis relies on characteristic clinical features corroborated by high-resolution magnetic resonance imaging (MRI) to confirm neurovascular contact and exclude secondary lesions. First-line pharmacological management centers on voltage-gated sodium channel blockers—specifically carbamazepine and oxcarbazepine—supported by second-line agents such as lamotrigine, baclofen, gabapentin, and pregabalin. For medically refractory cases, microvascular decompression (MVD) and ablative procedures (radiofrequency thermocoagulation, glycerol rhizolysis, balloon compression, and stereotactic radiosurgery) provide durable pain relief. This review synthesizes the epidemiology, pathophysiology, diagnostic workup, pharmacological and surgical management, and multidisciplinary clinical pharmacist responsibilities in optimizing patient safety and therapeutic outcomes.

1. Introduction

Trigeminal neuralgia (TN), historically known as tic douloureux, is a severe, chronic neuropathic pain disorder characterized by recurrent paroxysms of sharp, unilateral facial pain localized within the somatosensory distribution of one or more divisions of the trigeminal nerve (cranial nerve V) [1–4,19]. The trigeminal nerve provides general somatic

afferent innervation to the face, scalp, conjunctiva, cornea, nasal cavity, paranasal sinuses, oral mucosa, tongue, and teeth via three major branches: the ophthalmic (V1), maxillary (V2), and mandibular (V3) divisions [8,20]. Pain attacks are characteristically described as electric shock-like, stabbing, shooting, or lancinating, predominantly affecting the maxillary (V2) and mandibular (V3) territories [2,3,20]. Individual paroxysms typically last from a fraction of a second up to two minutes, occurring either as isolated stabs or repetitive volleys throughout the day [1, 4, 19].

A hallmark of TN is the presence of cutaneous or intraoral trigger zones—commonly around the nasolabial fold, upper lip, chin, gingiva, or alveolar ridge—where innocuous tactile or mechanical stimuli such as light facial touch, washing, shaving, applying cosmetics, toothbrushing, chewing, speaking, or exposure to a cool breeze precipitate intense pain discharges [2,3,8,20]. Spontaneous attacks without identifiable triggers also occur, and while patients traditionally experience pain-free refractory intervals between paroxysms, a subset develops concomitant continuous or near-continuous dull aching background pain within the affected territory [1, 13, 15]. Because attacks are unpredictable and excruciating, patients frequently restrict mastication, fluid intake, oral hygiene, and social interaction, leading to dehydration, malnutrition, anxiety, depression, and marked deterioration in health-related quality of life [17, 54, 55].

Diagnosis is primarily clinical, anchored in pain distribution, quality, paroxysmal duration, and triggerability [1,4,19]. However, symptom overlap with odontogenic infections, temporomandibular joint (TMJ) disorders, acute or chronic rhinosinusitis, postherpetic neuralgia, and trigeminal autonomic cephalalgias frequently delays accurate diagnosis and exposes patients to unnecessary irreversible dental extractions or endodontic procedures [2,3,6,53]. High-resolution magnetic resonance imaging (MRI) is indispensable for confirming neurovascular contact and ruling out secondary structural etiologies [4,21,22]. This review provides an updated synthesis of the etiology, epidemiology, pathophysiology, clinical presentation, diagnostic workup, pharmacological and surgical management, and clinical pharmacy perspectives in trigeminal neuralgia.

2. Etiology, Classification, and Epidemiology

In accordance with the International Classification of Headache Disorders, 3rd edition (ICHD-3), and the European Academy of Neurology (EAN) diagnostic grading system, trigeminal neuralgia is stratified into three etiological categories summarized in Table 1 [1,4,19]. Classical TN accounts for the majority of cases and is defined by the demonstration of neurovascular compression—most frequently by an elongated or tortuous superior cerebellar artery (SCA), and less commonly by the anterior inferior cerebellar artery (AICA) or pontine veins—producing morphological changes such as atrophy, displacement, or grooving of the trigeminal nerve at its root entry zone (REZ) [5,9,21,22].

Table 1. Etiological Classification, Underlying Pathology, and Neuroimaging Features of Trigeminal Neuralgia (ICHD-3 / EAN Criteria)

TN Classification	Primary Etiological Mechanism	Characteristic MRI & Clinical Findings	Epidemiological & Prognostic Notes
Classical TN	Pulsatile neurovascular compression (typically superior cerebellar artery) at trigeminal root entry zone	High-resolution 3D T2/TOF-MRA shows vascular contact with nerve atrophy, distortion, or displacement	Most prevalent subtype (~75–80%); responds well to Na+ channel blockers and microvascular decompression [1,21,22]
Secondary TN	Identifiable structural or demyelinating CNS pathology (e.g., multiple sclerosis plaque, CPA tumor, AVM, cyst)	MRI demonstrates pontine demyelinating lesion or space-occupying mass; higher rate of sensory deficits	Presents at younger age; higher incidence of bilateral involvement in multiple sclerosis [4,27,28]
Idiopathic TN	No demonstrable neurovascular compression with morphological nerve change or secondary lesion	Normal high-resolution brain MRI and electrophysiological testing despite classic paroxysmal phenotype	Accounts for ~10–15% of cases; managed initially with standard anticonvulsant pharmacotherapy [1,4,19]

Epidemiologically, the annual incidence of trigeminal neuralgia is estimated at 4 to 13 cases per 100,000 individuals, with a lifetime prevalence of approximately 0.16% to 0.3% [16,18]. TN exhibits a clear female predominance (female-to-male ratio of approximately 1.5:1 to 2:1) and increases substantially in incidence after the fifth decade of life, peaking

between 50 and 70 years of age [16,18,20]. The right side of the face is affected slightly more often than the left (~60% vs. 40%), whereas bilateral TN occurs in fewer than 5% of patients and strongly warrants investigation for underlying multiple sclerosis or familial neuropathy [2, 20, 27].

3. Pathophysiology

The pathogenesis of trigeminal neuralgia integrates peripheral trigeminal nerve injury with secondary central pain sensitization [5, 15, 23, 56]. As illustrated in Figure 1, chronic pulsatile compression by an aberrant blood vessel at the trigeminal root entry zone—an anatomical transition zone where central oligodendrocyte myelin shifts to peripheral Schwann cell myelin—induces focal demyelination, axonal dystrophy, and loss of Schwann cell insulation [5, 24].

According to the 'ignition hypothesis' proposed by Devor et al., focal demyelination exposes bare axonal membranes, triggering dysregulated clustering of voltage-gated sodium channels (notably Nav1.3, Nav1.7, and Nav1.8) and hyperexcitable ectopic pacemaker activity [23,25,56]. Close apposition of demyelinated axons permits ephaptic cross-talk (non-synaptic electrical short-circuiting) between large-diameter, low-threshold myelinated Aβ mechanoreceptive fibers and thinly myelinated Aδ or unmyelinated C nociceptive fibers [5,15,23]. Consequently, physiological tactile signals generated by light touch or chewing cross-excite adjacent pain fibers, producing synchronized, self-sustaining paroxysmal discharges perceived as sudden electric shocks [15,23].

Figure 1. Pathophysiological Mechanism of Trigeminal Neuralgia: Root Entry Zone Demyelination and Ephaptic Cross-Talk

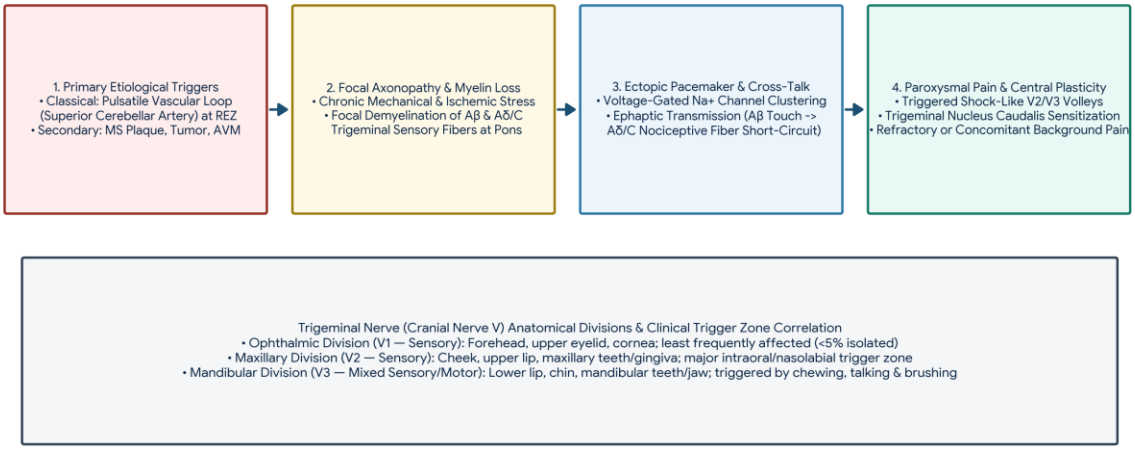


Figure 1. *Pathophysiological mechanism of trigeminal neuralgia illustrating root entry zone focal demyelination, ephaptic cross-talk between Aβ and nociceptive fibers, and trigeminal nerve anatomical divisions.*

In secondary TN, analogous axonal demyelination and hyperexcitability arise from neuroinflammatory plaques in the pontine trigeminal root entry zone or spinal trigeminal nucleus in multiple sclerosis, or from mechanical stretching and ischemia caused by cerebellopontine angle meningiomas, schwannomas, or epidermoid cysts [27,28]. Over time, repetitive high-frequency nociceptive bombardment induces central sensitization within the trigeminal nucleus caudalis and thalamocortical pain pathways, contributing to concomitant continuous background pain and progressive pharmacoresistance [15, 26, 56].

4. Clinical Features, Diagnostic Workup, and Differential Diagnosis

Clinically, TN presents with strictly unilateral, stereotyped paroxysms lasting from 1 second to 2 minutes, separated by refractory periods during which triggers temporarily fail to evoke pain [1,20]. Involvement is most frequent in V2 and/or V3 (>90% of patients), whereas isolated ophthalmic (V1) pain occurs in less than 5% of cases [20]. Routine neurological examination in classical or idiopathic TN is typically normal; however, subtle sensory hypoesthesia or corneal reflex diminution on quantitative sensory testing warrants thorough neuroimaging [4, 6, 22].

Magnetic resonance imaging of the brain using high-resolution 3D T2-weighted cisternography (e.g., CISS or FIESTA sequences), 3D time-of-flight magnetic resonance angiography (TOF-MRA), and contrast-enhanced T1-weighted sequences is the gold standard to exclude secondary tumors or demyelinating plaques and to grade neurovascular compression [4,6,21,22]. Accurate differentiation from overlapping craniofacial pain syndromes—outlined in Table 2—is essential to prevent iatrogenic dental interventions and diagnostic delays [2, 3, 6, 53].

Table 2. Differential Diagnosis of Trigeminal Neuralgia and Distinguishing Clinical and Diagnostic Features

Condition	Pain Quality & Temporal Pattern	Anatomical Distribution & Triggers	Key Distinguishing Diagnostic Features
Trigeminal Neuralgia (TN)	Sudden, electric shock-like, stabbing paroxysms (seconds to 2 min) with refractory periods	Unilateral V2/V3 > V1; triggered by light touch, chewing, talking, brushing teeth	Normal neurological exam (classical); high-res MRI shows neurovascular compression [1,4,19]
Odontogenic Pain (Pulpitis / Abscess)	Continuous, throbbing, dull-to-severe ache worsening with thermal or percussion stimuli	Localized to affected tooth/jaw; thermal (hot/cold) and biting triggers	Abnormal dental exam, percussion tenderness, periapical radiograph pathology [2,53]
Temporomandibular Disorder (TMD)	Dull, aching, myofascial or preauricular pain exacerbated by jaw opening/mastication	Preauricular region, masseter/temporalis muscles; jaw movement triggers	TMJ clicking/crepitus, masticatory muscle tenderness, restricted mandibular opening [2,3]
Postherpetic Neuralgia (PHN)	Constant burning, aching, or lancinating pain with prominent mechanical allodynia	Unilateral dermatomal (commonly V1 ophthalmic); gentle touch triggers	History of acute herpes zoster vesicular rash, cutaneous scarring, sensory loss [2,6]
Glossopharyngeal Neuralgia	Severe, brief, stabbing paroxysms identical in quality to TN	Posterior tongue, tonsillar fossa, pharynx, ear; triggered by swallowing/coughing	Anesthetizing tonsillar pillar relieves pain; MRI evaluates IX/X nerve compression [3,19]
SUNCT / SUNA & Cluster Headache	Orbital/supraorbital stabbing (SUNCT: 1–600 s) or severe boring pain (Cluster: 15–180 min)	Strictly unilateral V1 orbital/temporal distribution	Prominent ipsilateral cranial autonomic signs (lacrimation, conjunctival injection, ptosis) [3,19]
Persistent Idiopathic Facial Pain	Continuous, poorly localized dull, nagging, or burning ache without paroxysms	Deep facial pain not conforming to peripheral nerve anatomy; no trigger zones	Normal neurological exam and MRI; diagnosis of exclusion [2,19,53]

5. Therapeutic Management: Pharmacological and Surgical Strategies

5.1. First-Line and Second-Line Pharmacotherapy

Pharmacotherapy remains the initial treatment of choice for classical and idiopathic trigeminal neuralgia [4,6,7]. As detailed in Table 3, voltage-gated sodium channel blockers stabilize hyperexcitable neuronal membranes, prolonging the inactivated state of sodium channels and suppressing ectopic high-frequency discharges [7,12,29–32]. Carbamazepine (200–1200 mg/day) is the established gold standard, achieving initial pain relief in 70–90% of patients, while oxcarbazepine (300–1800 mg/day) offers comparable efficacy with fewer cytochrome P450 drug interactions and improved tolerability [4,7,31,32]. Both agents require gradual dose titration and baseline/periodic monitoring of complete blood counts, hepatic transaminases, and serum sodium (due to syndrome of inappropriate antidiuretic hormone secretion-mediated hyponatremia, particularly with oxcarbazepine) [42–44]. Screening for the HLA-B*1502 allele is recommended in patients of East and South Asian descent prior to initiating carbamazepine or oxcarbazepine to prevent Stevens–Johnson syndrome and toxic epidermal necrolysis [40,41].

Table 3. Pharmacological Management Options, Dosing Regimens, Adverse Effects, and Clinical Monitoring in Trigeminal Neuralgia

Drug & Line of Therapy	Mechanism of Action	Typical Adult Titration & Maintenance Dose	Major Adverse Effects & Monitoring Requirements
Carbamazepine (First-Line)	Blocks voltage-gated Na+ channels in inactivated state; inhibits ectopic firing	Start 100 mg BID; titrate by 100–200 mg every 3–7 days to 400–1200 mg/day	Dizziness, ataxia, diplopia, somnolence, CYP3A4 autoinduction,

			hyponatremia, leukopenia, SJS/TEN (HLA-B*1502) [4,12,40,42]
Oxcarbazepine (First-Line)	MHD metabolite blocks voltage-gated Na+ channels and modulates Ca2+ channels	Start 150 mg BID; titrate by 300 mg/week to 600–1800 mg/day	Somnolence, dizziness, headache, clinically significant hyponatremia (monitor serum Na+); fewer CYP interactions [4,31,32,44]
Lamotrigine (Second-Line / Add-on)	Inhibits voltage-sensitive Na+ channels and suppresses glutamate release	Start 25 mg/day for 2 weeks; slow titration over 5–6 weeks to 200–400 mg/day	Maculopapular rash (risk of SJS with rapid titration or valproate co-therapy), dizziness, diplopia [4,33]
Baclofen (Second-Line / Add-on)	GABAB receptor agonist facilitating segmental inhibition in trigeminal nucleus	Start 5–10 mg TID; titrate gradually to 30–80 mg/day in divided doses	Sedation, muscle weakness, dizziness, nausea; avoid abrupt withdrawal (rebound seizures/hallucinations) [4,34]
Gabapentin / Pregabalin (Second-Line)	Binds α2δ subunit of voltage-gated Ca2+ channels; reduces neurotransmitter release	Gabapentin: 300–3600 mg/day; Pregabalin: 150–600 mg/day	Peripheral edema, weight gain, somnolence, dizziness; requires renal dose adjustment (CrCl) [4,35,36]
Botulinum Toxin Type A (Refractory)	Inhibits vesicular release of CGRP, substance P, and glutamate at nerve terminals	25–100 Units intradermal/submucosal across facial trigger zones	Transient facial asymmetry, local injection-site edema or hematoma; relief lasts 3–6 months [4,37]

When first-line sodium channel blockers fail due to inadequate efficacy or dose-limiting neurotoxicity, specialist-directed second-line monotherapy or combination regimens incorporating lamotrigine, baclofen, gabapentin, pregabalin, or targeted subcutaneous botulinum toxin type A injections are employed [4,7,33–37]. During severe acute pain exacerbations ('TN crisis') impairing hydration or oral intake, inpatient intravenous fosphenytoin or lidocaine infusions may be used as short-term bridge therapy [38,39].

5.2. Surgical and Procedural Interventions

Approximately 25–40% of patients eventually develop pharmacoresistance or intolerable medication side effects, warranting neurosurgical evaluation [4,11,14]. As illustrated in Figure 2, procedural selection balances durability of pain relief against surgical invasiveness and sensory risk [4,10,11]:

- **Microvascular Decompression (MVD):** The gold-standard non-destructive posterior fossa craniotomy procedure for medically fit patients with classical TN and confirmed neurovascular compression. By mobilizing the offending vessel away from the trigeminal REZ and interposing a Teflon felt pledget, MVD achieves immediate pain relief in >85–90% of patients and long-term pain-free rates of 70–80% at 5–10 years, though carrying small risks of cerebrospinal fluid leak, ipsilateral hearing loss, facial numbness, or brainstem stroke [9,10,45,46].
- **Percutaneous Rhizotomy Techniques:** Minimally invasive ablative procedures targeting the Gasserian ganglion via the foramen ovale—including percutaneous radiofrequency thermocoagulation (RFT), percutaneous glycerol rhizolysis (PGR), and percutaneous balloon compression (PBC)—offer rapid pain relief for elderly or surgically high-risk patients and those with multiple sclerosis, with trade-offs of higher recurrence rates and intentional facial hypoesthesia or masseter weakness [4,47–49].
- **Stereotactic Radiosurgery (Gamma Knife / CyberKnife):** Delivers focused high-dose ionizing radiation (70–90 Gy) to the cisternal trigeminal root without incision or general anesthesia. Pain relief develops gradually over 4–8 weeks, making radiosurgery well suited for frail patients unable to undergo craniotomy [50,51].

Figure 2. Evidence-Based Diagnostic, Pharmacological, Surgical, and Clinical Pharmacy Care Pathway for Trigeminal Neuralgia

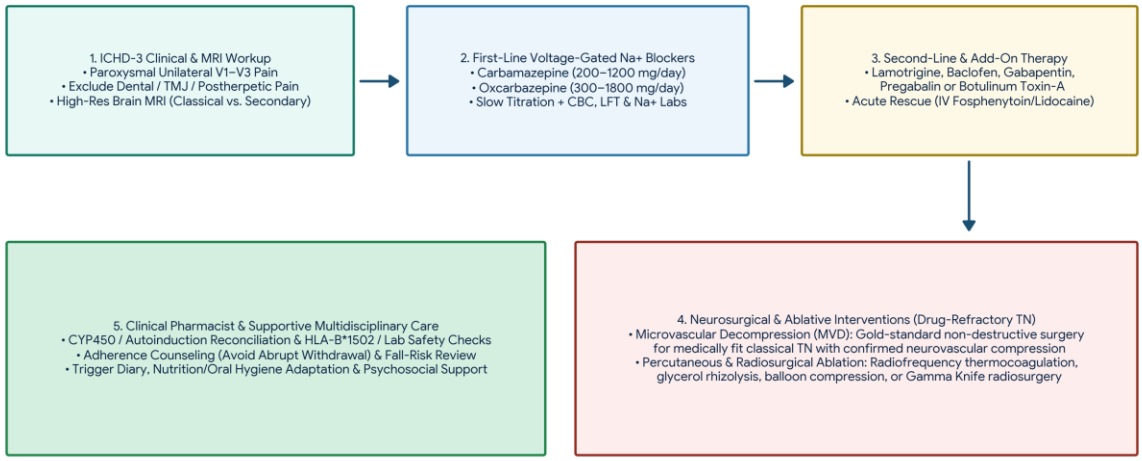


Figure 2. *Evidence-based diagnostic, pharmacological, neurosurgical, and clinical pharmacy care pathway for trigeminal neuralgia.*

6. Supportive Care and Clinical Pharmacy Practice Perspective

Comprehensive care extends beyond pharmacotherapy and surgery to address nutritional hydration, modified oral hygiene (e.g., soft-bristled brushes, lukewarm water rinses), identification of avoidable mechanical triggers without inducing severe behavioral avoidance, structured pain diaries, and screening for comorbid anxiety, depression, and sleep disturbance [17,54,55,74-76].

Within the multidisciplinary healthcare team, the clinical pharmacist plays an indispensable role in safeguarding medication efficacy and tolerability [4,7,32]:

- **Medication Reconciliation and CYP450 Interaction Management:** Carbamazepine is a potent inducer of CYP3A4, CYP1A2, CYP2C9, and UDP-glucuronosyltransferases—and exhibits autoinduction over the first 3–5 weeks of therapy—necessitating systematic review of concomitant anticoagulants, antihypertensives, statins, oral contraceptives, and antidepressants [42,69-73].
- **Pharmacogenomic and Laboratory Safety Surveillance:** Coordinating baseline and follow-up complete blood counts, hepatic panels, serum sodium levels (to detect oxcarbazepine- or carbamazepine-induced hyponatremia), and HLA-B*1502 screening in at-risk populations [40,41,44].
- **Adherence Counseling and Titration Support:** Educating patients on gradual upward dose titration to minimize initial vestibular side effects (dizziness, ataxia, diplopia) and warning strongly against abrupt discontinuation of anticonvulsants or baclofen [12,32,34].
- **Geriatric and Renal Dosing Optimization:** Adjusting gabapentin and pregabalin dosages according to creatinine clearance and evaluating cumulative central nervous system sedative burden to mitigate fall risk in older adults [35,36, 63-68].

7. Discussion and Future Perspectives

Despite decades of clinical experience, trigeminal neuralgia remains a major diagnostic and therapeutic challenge due to its excruciating intensity, episodic unpredictability, and propensity for late relapse [2,3,17]. Early recognition by primary care clinicians and dentists is paramount to curtail unnecessary dental procedures and initiate targeted sodium channel blockade promptly [16,53]. High-resolution MRI has transformed preoperative stratification by distinguishing classical neurovascular compression—amenable to curative MVD—from secondary demyelinating or neoplastic etiologies [21,22].

Future translational research is advancing selective voltage-gated sodium channel blockers (such as Nav1.7 and Nav1.8 subtype-selective inhibitors like vixotrigine) engineered to suppress ectopic trigeminal firing without central nervous system sedation or hyponatremia, alongside advanced diffusion tensor imaging (DTI) tractography to quantify microstructural nerve recovery and optimize personalized surgical timing [7,15,56-62].

8. Conclusion

Trigeminal neuralgia is a debilitating neuropathic pain condition that severely compromises physical functioning and emotional well-being. Timely ICHD-3 clinical diagnosis corroborated by high-resolution MRI enables accurate classification into classical, secondary, or idiopathic subtypes. Carbamazepine and oxcarbazepine remain foundational first-line therapies, while second-line anticonvulsants, botulinum toxin type A, microvascular decompression, and percutaneous or radiosurgical procedures provide vital options for refractory

disease. Integrating neurologists, neurosurgeons, dentists, pain specialists, and clinical pharmacists within a coordinated, patient-centered care model is essential for maximizing long-term pain control, medication safety, and quality of life.

References (56)

1. Maarbjerg S, Gozalov A, Olesen J, Bendtsen L. Trigeminal neuralgia — a prospective systematic clinical study of 158 patients. Headache. 2014;54(10):1574-1582. doi:10.1111/head.12441.
2. Maarbjerg S, Wolfram F, Gozalov A, Olesen J, Bendtsen L. Significance of neurovascular contact in classical trigeminal neuralgia. Brain. 2015;138(2):311-319. doi:10.1093/brain/awu349.
3. Antonini G, Di Pasquale A, Cruccu G, Truini A, Morino S, Saltelli G, et al. Magnetic resonance imaging contribution for diagnosing symptomatic neurovascular contact in classical trigeminal neuralgia: a blinded case-control study and meta-analysis. Pain. 2014;155(8):1464-1471. doi:10.1016/j.pain.2014.04.020.
4. Cruccu G, Biasiotta A, Di Rezze S, Fiorelli M, Galeotti F, Innocenti P, et al. Trigeminal neuralgia and pain related to multiple sclerosis. Pain. 2009;143(3):186-191. doi:10.1016/j.pain.2008.12.026.
5. Truini A, Prosperini L, Calistri V, Fiorelli M, Pozzilli C, Millefiorini E, et al. A dual concurrent mechanism explains trigeminal neuralgia in patients with multiple sclerosis. Neurology. 2016;86(22):2094-2099. doi:10.1212/WNL.0000000000002720.
6. Blom S. Trigeminal neuralgia: its treatment with a new anticonvulsant drug (G-32883). Lancet. 1962;1(7234):839-840. doi:10.1016/S0140-6736(62)91847-0.
7. Gandhi MY, Prasad SB, Kumar V, Soni H, Rawat H, Mishra SK, et al. Quantification of phytochemicals and metal ions as well as the determination of volatile compounds, antioxidant, antimicrobial and antacid activities of the *Mimosa pudica* L. leaf: Exploration of neglected and under-utilized part. Chem Biodivers. 2023;20(10):e202301049.
8. Shamim, Ali S, Ali T, Sharma H, Kishor BN, Jha SK. Recent advances in monodisperse gold nanoparticle delivery, synthesis, and emerging applications in cancer therapy. Plasmonics. 2025;20(9):7121–41.
9. Jha SK, Singh N, Shanker OR, Antil I, Baghel JS, Huddar V, et al. A review on integrative approaches in oncology: Bridging Ayurvedic medicine and modern cancer therapeutics. Front Nat Prod. 2025;4:1635197.
10. Sharma S, Kumar C, Kushwaha H, Jha SK, Chawla S, Sharma A, et al. Advancing anticancer drug development: Overcoming challenges and exploring new therapeutic strategies. Ayush J Integr Oncol. 2025;2(1):8–27.
11. Gandhi Y, Kumar V, Singh G, Prasad SB, Mishra SK, Soni H, et al. Chemoprofiling and medicinal potential of underutilized leaves of *Cyperus scariosus*. Sci Rep. 2024;14(1):7263.
12. Singh A, Jha SK, Dwivedi PC, Srivastava U, Pal A. Revolutionizing healthcare: Integrating electronics, AI, traditional, and conventional methods. World J Adv Res Rev. 2024;23:2426–34.
13. Jha SK, Islam M, Kumar R, Rana L, Saifi MA, Ali S, et al. Evaluation of *Vernonia amygdalina* Del. containing phytoconstituents as potential inhibitors of monkeypox virus using molecular docking analysis. World J Adv Res Rev. 2023;17(1):1112–22.
14. Jha SK, Mishra AK, Kumar V, Dane G, Kumari S, Charde V, et al. Ecological and behavioral impacts of COVID-19 on human existence and potential preventive measures through traditional and alternative medicine: A narrative review. Pharmacol Res Nat Prod. 2024;3:100042.
15. Sonawane SM, Lohiya GV, Rao AK, Beniwal M, Chavan SU, Aparadh VM, et al. Conjunctivitis in unusual populations: A review of rare cases and challenges in diagnosis and management. World J Adv Res Rev. 2023;19(3):1326–36.
16. Khan R, Maheshwari D, Chauhan S, Lohiya GV, Kumar C, Antil I, et al. Exploring the potential therapeutic value of *Solanum lycopersicum* L. phytoconstituents for Parkinson's disease through molecular docking analysis. World J Adv Res Rev. 2023;20:488–501.
17. Sonkar V, Jha SK, Tiwari S, Akarsh A, Priya S, Sharan P, et al. Characterization and explanation of the soil fertility state of Sakaldiha block in the Chandauli District of Uttar Pradesh's industrial area. World J Adv Res Rev. 2023;18:63–72.
18. Singh NK, Sengar AS, Jha SK. A review of Ayurvedic measures for preventing COVID-19 and promoting health during pregnancy. J Indian Syst Med. 2024;12(2):61–5.
19. Trivedi LM, Saxena S, Sharma S, Beniwal M, Jha SK, Rao AK, et al. Characterization, profiling, and molecular docking analysis of phytochemicals derived from *Daucus carota* for evaluating their potential role in cardiovascular disease assessment. World J Adv Res Rev. 2023;20:159–75.
20. Mehta V, Zargar AA, Attri P, Jha SK. Bagging the role of herbal drugs in the management of cervical cancer. Ayush J Integr Oncol. 2025;2(1):35–43.
21. Chauhan S, Jha SK, Tamta A, Sharma V, Kumar C, Lohiya G, et al. Descriptive analytical study based on profiling, morphological, pomological and pharmacological traits to identify the genotypes of the

promising mango *Mangifera indica* L. World J Adv Res Rev. 2023;19:1544–53.
22. Tamta, Arnima, and Sudhanshu Kumar Jha. "Screening of Germplasms and Population Dynamics of Major Insect Pests of Spring Green Gram Vigna radiata (Linn.)." PhD diss., Doctoral Dissertation, Doctoral Dissertation, GB Pant University of Agriculture and Technology, Pantnagar-263145 (Uttarakhand).
23. Kumar R, Verma H, Ali M, Midha T, Kumar D, Jha SK. Review of mitigating cancer risk through Ayurvedic practices: A holistic approach to combating sedentarism. Ayush J Integr Oncol. 2025;2(2):86–90.
24. Patel S, Singh S, Gupta AK, Dalimbe AY, Muthoju SM, Pawar AR, et al. Ayurveda and common Indian spices: A natural alternative for cancer therapy. Ayush J Integr Oncol. 2025;2(2):91–102.
25. Jha KT. Stereoisomerism in chemistry and drug development: Optical, geometrical, and conformational isomers. 2025.
26. Jha SK, Gupta A, Huddar VG. Botanical breakthroughs: The growing impact of plant-derived compounds in cancer treatment. Ayush J Integr Oncol. 2025;2(2):62–7.
27. Sharma A, Jha SK, Huddar VG. Integrative role of Ayurvedic phytochemicals in cancer treatment: Targeting signaling pathways, boosting chemosensitivity, and utilizing traditional therapeutics. Ayush J Integr Oncol. 2025;2(2):77–85.
28. Jha SK, Charde V, Kumar V, Narasimhaji CV. Vitiligo treatment with natural bioactive: A narrative review. Nat Prod J. 2026;16(3):e22103155337960.
29. Zargar AA, Mehta V, Gupta R, Bhandari K, Sharma MC, Balaji P, et al. Incretin hormones: Mechanisms, therapeutic implications, and future directions in glucose regulation and diabetes management. Curr Proteom. 2025;22:100014.
30. Doshi R, Lohiya G. Synthesis, in-silico design and spectral characterization, elucidation of *Cannabis sativa* L. (Cannabaceae) containing phytoconstituents demonstrating novel therapeutic efficacy against epilepsy.
31. Shrivastav S, Tyagi R, Singh M, Jha S. The effectiveness of curcumin on dysmenorrhea. Int J Med Sci Pharma Res. 2022;8(4):8–12.
32. Sharma S, Yadav C, Chawla S, Jha SK. Beyond chemotherapy: Exploring the potential of phytomedicine in oncology. Ayush J Integr Oncol. 2025;2(3):133–45.
33. Badal R, Ranjan S, Jha SK, Kumar L, Patel AK, Yadav P, et al. GC-MS/MS and HR-LCMS-QTOF analysis of various extracts of Saraswata Ghrita: A comprehensive dataset on phytochemical compounds. Data Brief. 2025;61:111675.
34. Ahlawat A, Chauhan S, Antil I, Jha SK, Huddar VG. Examining the impact of the microbiome on cancer immunotherapy outcomes: A review. Ayush J Integr Oncol. 2025;2(3):118–24.
35. Gupta AJ, Huddar VG, Jha SK, Soni U. Traditional wisdom in modern oncology: Ayurvedic pain relief and supportive care. Ayush J Integr Oncol. 2025;2(3):178–87.
36. Sandrepogu J, Khandelwal P, Tiwari SK, Jha SK, Chauhan S, Sharma H, et al. A holistic paradigm for cancer care: Merging Ayurveda with contemporary oncology. Ayush J Integr Oncol. 2025;2(4):254–61.
37. Antil I, Bangarwa S, Jha SK, Gupta AJ, Soni U, Huddar VG, et al. Exploring the chemopreventive and anticancer effects of green tea. Ayush J Integr Oncol. 2025;2(3):158–68.
38. Attri P, Zargar AA, Mehta V, Mushtaq SU, Jha SK, Soni U. Genetic basis of cancer: Harnessing natural remedies for therapeutic potential. Ayush J Integr Oncol. 2025;2(3):188–94.
39. Chauhan S, Jha SK, Huddar VG, Ahlawat A, Sharma S, Tripathi A, et al. Targeting cancer at the molecular level: A biochemical and medicinal chemistry approach. Ayush J Integr Oncol. 2025;2(3):169–77.
40. Pandey JK, Verma SK, Kumar J, Perwej Y, Jha SK, Panchal BY, et al. Transformative role of advanced neural computation in clinical image diagnostics: A review of key concepts and applications. Semin Ultrasound CT MR. 2026;47(5):374–90.
41. Sharma H, Mahto RR, Jha SK, Chauhan S, Khandelwal P, Tiwari SK. Rasayana-based adjuvant strategies in cancer care: An Ayurvedic-oncologic perspective. Iran J Blood Cancer. 2025;17(4):35–68.
42. Raj H, Sharma S, Tripathi A, Huddar VG, Jha SK. Herbal medicine in cancer therapy: A comprehensive review of phytoconstituents, mechanisms, and clinical applications. Ayush J Integr Oncol. 2025;2(3):146–57.
43. Shamim PT, Khatri N, Yadav AK, Mishra B, Khan MR, Behera SK, et al. Role of human chorionic gonadotropin in frozen-thawed embryo transfer cycles for secretory transformation: A narrative review. Eur Chem Bull. 2023;12(8):5477–93.
44. Mandal P, Kumar J, Bora D, Hashmi SA, Tiwari M, Gupta R, et al. Literature review on forensic implications of illicit drug metabolism: A medicinal chemistry perspective. J Forensic Leg Med. 2026;103158.
45. Kumar C, Chauhan P, Chauhan S, Jha SK, Lohiya G. Phyto-pharmacognostic experimental study of *Epimedium sagittatum* and *Gloriosa superba* L. for the treatment of hypogonadism. Int J Innov Sci Res Technol. 2023;8(6):254–60.
46. Raj H, Jha SK, Negi M, Khandelwal P, Chauhan S, Sharma H, et al. Integrative oncology: A comprehensive comparative analysis of herbal and allopathic medicine in cancer treatment. Curr Pharm Res. 2025;1:427–35.

47. Dhiman S, Thakur N, Dwivedi PC, Pande P, Jha SK, Lohiya GV. Phytochemicals: Investigating bioactive compounds for antifungal uses and quality issues in functional foods and therapeutics. 2024:258–68.
48. Singh V, Mandal P, Kaur N, Bora D, Gupta R, Kumar A, et al. Synthesis, characterization, and binding interactions of rhodanine-3-acetic acid-based compound for latent fingerprint development. J Forensic Sci. 2026;71(1):584–92.
49. Jha SK, Chaturvedi SK, Singh SK, Chauhan S, Chauhan P, Singh H, et al. Synthesis and in-silico design of a novel silver metal ciprofloxacin compound. World J Adv Res Rev. 2023;18(1):885–92.
50. Mahto RR, Bansal KS, Soni H, Tiwari SK, Sandrepogu J, Bhatted SK. Efficacy of Vidangadi Loha and Phalatrikadi Ghana Vati with and without the prior administration of Virechana Karma in Madhumeha (obese diabetic): A randomized controlled trial. Int J Drug Deliv Technol. 2026;16(41s):1275–81.
51. Shivani, Thakur N, Lohiya GV, Gupta R, Dhiman S, Bharadwaj S, et al. Natural origins, bioavailability and therapeutic potential of astaxanthin: A systematic review across diverse health applications. 2024:5929–39.
52. Tamta A, Jha SK. Implications of green technology for climate change mitigation minimizing waste and pollution. TIDEE. 2023;22(4):476.
53. Kaur AK, Venkatasubramanian H, Sarma S, Gupta R, Singh RG, Chaudhary B, et al. Targeting cancer pathways with phytochemicals: Molecular mechanisms and clinical potential. Curr Proteom. 2026;23(6):100182.
54. Patel S, Paliwal A, Jha SK, Singh R, Mishra AK. Environmental neurotoxicants as accelerators of brain ageing: Exposomic mechanisms linking pollution to amyloidogenesis and Alzheimer's disease. Int J Environ Health Res. 2026:1–21.
55. Dhiman S, Thakur N, Jha SK, Karkare P, Bora D, Sahu RK, et al. Pyrazole derivatives: A comprehensive review of synthesis, antimicrobial activity, structure-activity relationships, and therapeutic potential. Curr Proteom. 2026;23(5):100154.
56. Mundada AB, Mundada PA, Kathirvel S, Kumar PR, Krishnamoorthy P, Ezzaoui N, et al. Ion channels as emerging drug targets: Pharmacological insights and therapeutic advances. Curr Proteom. 2026;23(4):100141.
57. Midha T, Kumar D, Kumar A, Jha SK. Rasayana therapy in Ayurveda: A promising approach for cancer support and immunomodulation. [Publication details required].
58. Garg N, Sharma R, Bhojani MK, Rai S, More AB, Jha SK. Bridging traditional wisdom and modern science: An integrative approach to breast cancer care. Ayush J Integr Oncol. 2026;3(2):81–9.
59. Jha SK, Negi M, Singh K, Shukla D, Sharma H, Khandelwal P, et al. Harmonizing traditional Ayurvedic knowledge with modern science: A comprehensive review. Curr Pharm Res. 2026:393–410.
60. Antil I, Jha SK, Negi M, Sharma H, Tiwari SK. Baicalein from *Scutellaria baicalensis* as natural therapeutic agent for glioblastoma. Curr Pharm Res. 2025;1:411–26.
61. Mahto RR, Sharma H, Bhatted SK, Jha SK, Tiwari SK, Chauhan S, et al. Diabetes and erectile dysfunction: Exploring the Ayurvedic understanding of Madhumehaj Klaibya. Curr Pharm Res. 2026:446–57.
62. Gangurde RB, Goel P. Approach towards type 2 diabetes mellitus-induced erectile dysfunction (DMED): A case study.
63. Campbell FG, Graham JG, Zilkha KJ. Clinical trial of carbazepine (tegretol) in trigeminal neuralgia. J Neurol Neurosurg Psychiatry. 1966;29(3):265-267. doi:10.1136/jnnp.29.3.265.
64. Beydoun A, Schmidt D, D'Souza J. Oxcarbazepine versus carbamazepine in trigeminal neuralgia: a meta-analysis of three double-blind comparative trials. Neurology. 2002;58(Suppl 3):A81.
65. Chung WH, Hung SI, Hong HS, Hsih MS, Yang LC, Ho HC, et al. Medical genetics: a marker for Stevens-Johnson syndrome. Nature. 2004;428(6982):486. doi:10.1038/428486a.
66. Phillips EJ, Sukasem C, Whirl-Carrillo M, Muller DJ, Dunnenberger HM, Chantratita W, et al. Clinical Pharmacogenetics Implementation Consortium guideline for HLA genotype and use of carbamazepine and oxcarbazepine: 2017 update. Clin Pharmacol Ther. 2018;103(4):574-581. doi:10.1002/cpt.1004.
67. Bertilsson L. Clinical pharmacokinetics of carbamazepine. Clin Pharmacokinet. 1978;3(2):128-143. doi:10.2165/00003088-197803020-00003.
68. May TW, Korn-Merker E, Rambeck B. Clinical pharmacokinetics of oxcarbazepine. Clin Pharmacokinet. 2003;42(12):1023-1042. doi:10.2165/00003088-200342120-00002.
69. Dong X, Leppik IE, White J, Rarick J. Hyponatremia from oxcarbazepine and carbamazepine. Neurology. 2005;65(12):1976-1978. doi:10.1212/01.wnl.0000188819.45330.90.
70. Broggi G, Ferroli P, Franzini A, Servello D, Dones I. Microvascular decompression for trigeminal neuralgia: comments on a series of 250 cases, including 10 patients with multiple sclerosis. J Neurol Neurosurg Psychiatry. 2000;68(1):59-64. doi:10.1136/jnnp.68.1.59.
71. Sarsam Z, Garcia-Finana M, Nurmikko TJ, Varma TR, Eldridge P. The long-term outcome of microvascular decompression for trigeminal neuralgia. Br J Neurosurg. 2010;24(1):18-25. doi:10.3109/02688690903370289.

72. Kanpolat Y, Savas A, Bekar A, Berk C. Percutaneous controlled radiofrequency trigeminal rhizotomy for the treatment of idiopathic trigeminal neuralgia: 25-year experience with 1,600 patients. Neurosurgery. 2001;48(3):524-532. doi:10.1097/00006123-200103000-00013.
73. Brown JA, McDaniel MD, Weaver MT. Percutaneous trigeminal nerve compression for treatment of trigeminal neuralgia: results in 50 patients. Neurosurgery. 1993;32(4):570-573. doi:10.1227/00006123-199304000-00012.
74. Macianskyte D, Januzis G, Kubilius R, Adomaitiene V, Sciupokas A. Associations between chronic pain and depressive symptoms in patients with trigeminal neuralgia. Medicina (Kaunas). 2011;47(7):386-392.
75. Allsop MJ, Twiddy M, Grant H, Czoski-Murray C, Mon-Williams M, Mushtaq F, et al. Diagnosis, medication, and surgical management for patients with trigeminal neuralgia: a qualitative study. Acta Neurochir (Wien). 2015;157(11):1925-1933. doi:10.1007/s00701-015-2515-4.
76. Gambeta E, Chichorro JG, Zamponi GW. Trigeminal neuralgia: an overview from pathophysiology to pharmacological treatments. Mol Pain. 2020;16:1744806920901890. doi:10.1177/1744806920901890.