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EFFICACY OF ULTRASOUND GUIDED PERIPHERAL NERVE BLOCKS IN TRIGEMINAL NEURALGIA PATIENTS RECEIVING COMBINATION DRUG THERAPY

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Abstract

Introduction: Neuralgia involves pain along a damaged nerve, often resulting from injury or underlying pathology. Maxillofacial pain—originating from structures such as meninges, cornea, pulp, mucosa, and TMJ—comprises various neurological disorders that significantly impact patients' quality of life. Trigeminal neuralgia (TN), characterized by sudden, severe, shock-like facial pain, is among the most intense and common facial pain disorders. Diagnosis is primarily clinical, with neurovascular compression as a key etiology. Current treatments include pharmacotherapy, nerve blocks, and surgical procedures, but limitations such as adverse effects and procedural risks necessitate alternative approaches. Ultrasound-guided nerve blocks, combined with multi-drug regimens, present a minimally invasive option with potential benefits.

Objective: To evaluate the efficacy of ultrasound-guided peripheral nerve blocks combined with pharmacological therapy in patients with TN, focusing on pain reduction measured by the Numeric Rating Scale (NRS), safety, and patient satisfaction.

Methods: This prospective observational study included 33 patients with clinically diagnosed TN. All received a standardized three-drug regimen (carbamazepine, amitriptyline, baclofen) for 15 days, followed by ultrasound-guided nerve blocks targeting trigeminal divisions. Pain intensity was assessed at baseline, 15, and 30 days. Data analysis involved comparison of NRS scores and satisfaction levels.

Results: Significant reductions in NRS scores were observed in both groups, with a greater decrease in the nerve block group (from 7.75 to 1.25) versus the drug-only group (from 6.24 to 1.76) at 30 days ($p<0.001$). The percent change in pain was also significantly higher in the nerve block group. No major adverse effects were reported, and patient satisfaction was comparable.

Conclusion: Ultrasound-guided trigeminal nerve blocks, combined with multi-drug therapy, are effective and safe in managing TN pain. This multimodal strategy offers an advantageous alternative or adjunct to conventional treatments, potentially improving patient outcomes and reducing reliance on systemic medications.

Keywords: Trigeminal neuralgia, ultrasound-guided nerve block, maxillofacial pain, neurovascular compression, pharmacotherapy, multimodal management, nerve block, pain relief, facial neuralgia, minimally invasive therapy

Introduction

Neuralgia denotes pain along a damaged nerve, typically arising from underlying injury or pathology. Maxillofacial pain, originating from structures such as the meninges, cornea, tooth pulp, oral or nasal mucosa, and temporomandibular joint, encompasses various neurological disorders that profoundly impact patients' quality of life (1, 2). These conditions often share similar clinical features, emphasizing the importance of comprehensive serial examinations and laboratory diagnostics for accurate identification and effective management (3).

Among these, trigeminal neuralgia (TN) is distinguished by sudden, severe, shock-like pain triggered by specific zones, whereas glossopharyngeal neuralgia (GPN) involves pain in the oropharyngeal area, especially during swallowing and mandibular movements (4, 5). Myofascial pain dysfunction syndrome (MPDS) manifests as widespread pain, sleep disturbances, fatigue, psychosomatic issues, and chronic headaches (6, 7). Due to the intricate innervation and functional complexity of the facial region, diagnosing and managing facial pain presents notable challenges (8). TN, recognized as one of the most intense forms of facial pain, is a common neurological disorder affecting the orofacial region (5, 9, 10).

Pain signals from intraoral and extraoral structures are transmitted to the central nervous system via the trigeminal nerve system (14, 15). TN is characterized by paroxysmal, stabbing, electric shock-like pain within the trigeminal nerve's distribution, frequently unilateral and triggered by stimuli such as chewing, speaking, or even airflow. It predominantly affects the right side of the face, with an incidence of 4.3 to 27 cases per 100,000 population annually, and shows higher prevalence among females and older adults (16–19). Diagnosis is primarily clinical, especially in the elderly, where neurovascular compression is a common etiological factor (11, 12). TN can be classified as idiopathic (primary) or secondary, with secondary cases linked to intracranial lesions like tumors, infarctions, or multiple sclerosis (13). Trigger zones are highly sensitive regions where minor stimuli can provoke intense pain episodes, commonly located on facial skin or intraoral sites (20).

The rationale for combining pharmacological therapy with nerve block interventions stems from the limitations of current treatments. Pharmacological agents such as carbamazepine are effective but often associated with adverse effects and compliance issues (21). Interventional approaches like RFA and surgical options provide relief but carry risks and require specialized facilities and expertise (23–25). Recent advances in ultrasound-guided nerve blocks offer precise, minimally invasive options with fewer complications. The use of neurolytic agents like glycerol, alcohol, or phenol can extend pain relief duration but still may not fully address the multifaceted nature of TN.

Therefore, our approach integrates a triple drug regimen—carbamazepine, amitriptyline, and baclofen—to target different pain pathways and mechanisms, potentially enhancing efficacy and reducing side effects. The addition of ultrasound-guided peripheral nerve blocks targeting the affected trigeminal divisions aims to provide localized, immediate pain relief while minimizing systemic drug doses and related adverse effects. Incorporating dexamethasone as an adjuvant may prolong the analgesic effect by reducing inflammation and nerve irritation. This multimodal strategy is designed to address the complex pathophysiology of TN more effectively, improve patient comfort, and reduce recurrence, thus offering a comprehensive and rational approach to management.

The aim of this study is to evaluate the efficacy of ultrasound-guided peripheral nerve blocks in patients with trigeminal neuralgia receiving combination drug therapy. The primary objective is to assess changes in pain levels using the Numerical Rating Scale (NRS) following administration of a local anesthetic combined with a non-particulate steroid alongside the three-drug regimen. Secondary objectives include monitoring for any adverse effects or complications related to the nerve block procedure and determining patient satisfaction scores post-treatment. This approach seeks to establish whether the combined modality offers superior pain relief, safety, and patient acceptance.

Methodology

Study Design and Setting

This prospective observational study was conducted at the Pain Clinic outpatient department of Nehru Hospital, B.R.D. Medical College, Gorakhpur, from December 1, 2019, to November 30, 2020. The study aimed to evaluate the efficacy of a combined drug regimen and ultrasound-guided nerve blocks in patients with trigeminal neuralgia.

Participants

Patients of either sex aged between 20 and 80 years, classified as ASA physical status I or II, presenting with facial pain suggestive of trigeminal neuralgia, were included. The diagnosis was established based on detailed clinical history, physical examination, and confirmed using the International Classification of Headache Disorders (ICHD-3-beta) and the International Association for Study of Pain (IASP) criteria. All participants provided written informed consent after explanation of the study procedures and potential risks. Ethical approval was obtained from the institutional ethics committee.

Inclusion and Exclusion Criteria

Inclusion criteria encompassed patients with a confirmed diagnosis of trigeminal neuralgia, whether newly diagnosed or follow-up cases, having no contraindications to nerve block procedures. Exclusion criteria included patients refusing consent, those with bilateral or secondary trigeminal neuralgia, recent surgical or invasive treatment (<6 months), uncontrolled systemic illnesses (cardiovascular, renal, hepatic, pulmonary), neuromuscular disorders, local infections, bleeding disorders, allergy to study drugs, or presence of major neurological diseases requiring MRI.

Sample Size Calculation

Based on prior data indicating a mean change (Δ) of 1 in Numeric Rating Scale (NRS) scores with a standard deviation (σ) of 2, and aiming for a 95% confidence level with 80% power, the minimum sample size calculated using the formula:

$$\mathbf{n = 2\,\sigma^2 * (Z_\alpha + Z_\beta)^2 / (\Delta)^2}$$

was approximately 63 patients.

Procedures

All selected patients underwent thorough clinical evaluation, including history, physical examination, and relevant investigations to rule out contraindications. Routine blood tests were performed pre-procedure.

Patients received a standardized three-drug regimen for 15 days, which included medications such as carbamazepine, oxcarbazepine, or other appropriate agents based on clinical judgment.

Following this, ultrasound-guided nerve blocks targeting the ophthalmic, maxillary, and mandibular divisions of the trigeminal nerve were administered.

Nerve Block Technique

- Preparation: Patients fasted according to ASA fasting guidelines. The procedure was explained, emphasizing benefits and potential side effects. Standard monitoring (ECG, blood pressure, SpO_2) was established, and IV access secured with Ringer's lactate infusion.

- Ophthalmic Division Block: The patient lay supine with the head neutral and eyes closed. Using a high-frequency linear transducer placed over the supraorbital notch, the supraorbital nerve was targeted. A 21G needle was introduced laterally toward the nerve, injecting 1 ml of 0.5% isobaric bupivacaine + 1 ml dexamethasone.

- Maxillary and Mandibular Nerve Blocks: A curvilinear transducer was positioned below and parallel to the zygomatic arch, between the condylar and coronoid processes. The lateral pterygoid plate was visualized, and the needle was inserted posterior to it. Under continuous ultrasound guidance, 4 ml of the study solution (comprising 2 ml of 0.5% bupivacaine, 1 ml dexamethasone, and 1 ml normal saline) was injected after aspiration confirmation.

Post-Procedure Care

Patients were observed in the post-anesthetic care unit (PACU) for at least 6 hours to monitor for immediate complications. In case of adverse events, patients were shifted to the ICU for further management.

Follow-up and Outcome Assessment

Patients continued the three-drug regimen for 15 days and were followed up at 7 days, 15 days, and 1 month post-procedure. The primary parameter of interest was the change in pain intensity, assessed using the Numeric Rating Scale (NRS, 0-10), with 0 indicating no pain and 10 the worst pain. Satisfaction scores were recorded on a 4-point scale (1 = excellent, 2 = good, 3 = reasonable, 4 = poor).

Statistical Analysis

Data were entered into MS Excel and analyzed using SPSS version 23 (IBM Corp.). Continuous variables were expressed as means ± standard deviations or medians with interquartile ranges as appropriate. Categorical variables were expressed as frequencies and percentages. Group comparisons of continuous data employed independent t-tests when normally distributed; Mann-Whitney U tests were used for non-normal data. Categorical data were compared using Chi-square or Fisher’s Exact test when expected frequencies were low. Correlation analyses between continuous variables utilized Pearson’s or Spearman’s correlation coefficients based on data distribution. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1: Association between Block Given and Parameters

Parameters	Block Given		p-value
	Yes (n = 8)	No (n = 25)	
Age (Years)	56.00 ± 16.83	52.56 ± 14.67	0.449[1]
Age*			0.009[2]
21-30 Years	1 (12.5%)	2 (8.0%)	
31-40 Years	2 (25.0%)	1 (4.0%)	
41-50 Years	0 (0.0%)	10 (40.0%)	
51-60 Years	0 (0.0%)	6 (24.0%)	

Parameters	Block Given		p-value
	Yes (n = 8)	No (n = 25)	
61-70 Years	4 (50.0%)	5 (20.0%)	
71-80 Years	1 (12.5%)	0 (0.0%)	
81-90 Years	0 (0.0%)	1 (4.0%)	
Gender			1.000[2]
Male	4 (50.0%)	13 (52.0%)	
Female	4 (50.0%)	12 (48.0%)	
Height (cm)	162.75 ± 6.20	167.48 ± 5.26	0.055[1]
Weight (Kg)	61.88 ± 5.54	62.36 ± 4.99	0.950[1]
BMI (Kg/m2)	23.42 ± 2.37	22.26 ± 1.91	0.176[1]
BMI			0.327[2]
18.5-22.9 Kg/m2	3 (37.5%)	16 (64.0%)	
23.0-24.9 Kg/m2	3 (37.5%)	6 (24.0%)	
25.0-29.9 Kg/m2	2 (25.0%)	3 (12.0%)	
Systolic BP (mmHg) (1st Visit)	127.25 ± 10.25	130.96 ± 6.98	0.364[1]
Diastolic BP (mmHg) (1st Visit)	73.75 ± 7.81	76.96 ± 5.66	0.205[1]
NRS (Baseline)*	7.75 ± 0.46	6.24 ± 0.78	<0.001[1]
NRS (15 Days)*	5.88 ± 0.83	3.40 ± 0.76	<0.001[1]
NRS (30 Days)*	1.25 ± 0.46	1.76 ± 0.44	0.011[1]
Satisfaction Score			0.499[2]
Excellent	2 (25.0%)	8 (32.0%)	
Good	3 (37.5%)	13 (52.0%)	
Reasonable	3 (37.5%)	4 (16.0%)	

**Significant at p<0.05, 1: Wilcoxon-Mann-Whitney U Test, 2: Fisher's Exact Test*

The following variables were significantly associated (p<0.05) with the variable 'Block Given': , Age, NRS (Baseline), NRS (15 Days), NRS (30 Days)

The mean age of participants was 53.39 ± 15.02 years, with age distribution as follows: 9.1% aged 21–30 years, 9.1% aged 31–40 years, 30.3% aged 41–50 years, 18.2% aged 51–60 years, 27.3% aged 61–70 years, and 3% each in the 71–80 and 81–90-year age groups. Males comprised 51.5% and females 48.5%. The average height was 166.33 ± 5.78 cm, weight 62.24 ± 5.04 kg, and BMI 22.54 ± 2.06 kg/m², with 57.6% having a BMI between 18.5 and 22.9 kg/m². The mean systolic blood pressure was 130.06 ± 7.88 mmHg, and diastolic blood pressure was 76.18 ± 6.27 mmHg at the first visit. (Table 1)

Table 2: Association Between Block Given and BMI (n = 33)

BMI	Block Given			Fisher's Exact Test	
	Yes	No	Total	$\chi 2$	P Value
18.5-22.9 Kg/m2	3 (37.5%)	16 (64.0%)	19 (57.6%)	1.820	0.327
23.0-24.9 Kg/m2	3 (37.5%)	6 (24.0%)	9 (27.3%)		
25.0-29.9 Kg/m2	2 (25.0%)	3 (12.0%)	5 (15.2%)		
Total	8 (100.0%)	25(100.0%)	33 (100.0%)		

There was no significant difference in BMI distribution across groups ($\chi 2$ = 1.820, p = 0.327). BMI was normally distributed (Shapiro-Wilk p = 0.285), with a mean of 22.54 ± 2.06 kg/m² and a median of 22.27 (IQR: 20.72–24.03). The BMI ranged from 19.27 to 26.56, with 57.6% of participants having a BMI between 18.5 and 22.9, 27.3% between 23.0 and 24.9, and 15.2% between 25.0 and 29.9. (Table 2)

Table 3: Comparison of the 2 Subgroups of the Variable Block Given in Terms of Systolic BP (mmHg) (1st Visit) (n = 33)

Systolic BP (mmHg) (1st Visit)	Block Given		Wilcoxon-Mann-Whitney U Test	
	Yes	No	W	p value
Mean (SD)	127.25 (10.25)	130.96 (6.98)	78.000	0.364
Median (IQR)	128 (120.5-134)	132 (126-136)		
Range	112 - 140	120 - 142		

There was no significant difference in systolic blood pressure at the first visit between groups (W = 78.000, p = 0.364). The systolic BP was normally distributed (Shapiro-Wilk p = 0.296), with a mean of 130.06 ± 7.88 mmHg, median of 132 (IQR: 124–136), and a range of 112–142 mmHg. (Table 3)

Table 4: Comparison of the 2 Subgroups of the Variable Block Given in Terms of Diastolic BP (mmHg) (1st Visit) (n = 33)

Diastolic BP (mmHg) (1st Visit)	Block Given		Wilcoxon-Mann-Whitney U Test	
	Yes	No	W	p value
Mean (SD)	73.75 (7.81)	76.96 (5.66)	69.500	0.205
Median (IQR)	72 (68-78.5)	78 (72-80)		
Range	64 - 88	68 - 88		

There was no significant difference in diastolic blood pressure at the first visit between groups (W = 69.500, p = 0.205). The diastolic BP was normally distributed (Shapiro-Wilk p = 0.304), with a mean of 76.18 ± 6.27 mmHg, median of 78 (IQR: 72–80), and a range of 64–88 mmHg. (Table 4)

Table 5: Comparison of the 2 Subgroups of the Variable Block Given in Terms of NRS (Baseline) (n = 33)

NRS (Baseline)	Block Given		Wilcoxon-Mann-Whitney U Test	
	Yes	No	W	p value
Mean (SD)	7.75 (0.46)	6.24 (0.78)	187.000	<0.001
Median (IQR)	8 (7.75-8)	6 (6-7)		
Range	7 - 8	5 - 8		

There was a significant difference between the 2 groups in terms of NRS (Baseline) (W = 187.000, p = <0.001), with the median NRS (Baseline) being highest in the Block Given: Yes group. (Table 5)

Table 6 a: Comparison of the two Groups in Terms of change in NRS over time (n = 33)

NRS	Block Given		P value for comparison of the two groups at each of the timepoints (Wilcoxon-Mann-Whitney Test)
	Yes	No	
	Mean (SD)	Mean (SD)	
Baseline	7.75 (0.46)	6.24 (0.78)	<0.001
15 Days	5.88 (0.83)	3.40 (0.76)	<0.001
30 Days	1.25 (0.46)	1.76 (0.44)	0.011
P Value for change in NRS over time within each group (Friedman Test)	<0.001	<0.001	

The two groups showed significant differences in NRS at Baseline, 15 Days, and 30 Days. In the "Block Given" group, NRS decreased from 7.75 to 1.25 (p < 0.001), and in the "No Block" group, from 6.24 to 1.76 ($p < 0.001$). The trend over time differed significantly between groups ($p < 0.001$). (Table 6 a)

Table 6 b: Analysis of Absolute Change in NRS Over Time

Timepoint Comparison	Change in NRS from Baseline to Follow-up Timepoints				P-Value for Comparison of the two Groups in Terms of Difference of NRS from Baseline to Follow-up Timepoints
	Block Given: Yes		Block Given: No		
	Mean (SD) of Absolute Change	P Value of Change Within Group	Mean (SD) of Absolute Change	P Value of Change Within Group	
15 Days - Baseline	-1.88 (0.83)	0.112	-2.84 (0.85)	<0.001	0.012
30 Days - Baseline	-6.50 (0.76)	<0.001	-4.48 (0.82)	<0.001	<0.001

Post-hoc Nemenyi tests evaluated NRS changes from baseline to follow-ups. Wilcoxon-Mann-Whitney tested group differences. Green indicates $p < 0.05$.

In the "Block Given: Yes" group, NRS significantly changed from baseline at 30 days, with the greatest change observed then. In the "No Block" group, NRS significantly changed from baseline at 15 and 30 days, with the maximum change at 30 days. The change in NRS over time differed significantly between the two groups. (Table 6 b)

Table 6 c: Analysis of Percent Change in NRS Over Time

Timepoint Comparison	Percent Change in NRS from Baseline to Follow-up Timepoints				P-Value for Comparison of the two Groups in Terms of Difference of NRS from Baseline to Follow-up Timepoints
	Block Given: Yes		Block Given: No		
	Mean (SD) of Percent Change	P Value of Change Within Group	Mean (SD) of Percent Change	P Value of Change Within Group	
15 Days - Baseline	-24.1% (10.3)	0.112	-45.2% (12.1)	<0.001	<0.001
30 Days - Baseline	-83.7% (6.6)	<0.001	-71.5% (7.7)	<0.001	<0.001

Post-hoc Nemenyi tests assessed NRS changes from baseline to follow-ups. Group comparisons used the Wilcoxon-Mann-Whitney test. Green indicates $p < 0.05$.

In the "Block Given: Yes" group, NRS significantly changed from baseline at 30 days, with the maximum percent change at 30 days. In the "No Block" group, NRS significantly changed at 15 and 30 days, with the greatest percent change also at 30 days. The percent change in NRS over time differed significantly between the two groups. (Table 6 c)

Discussion

Trigeminal neuralgia (TN) is a neuropathic disorder characterized by episodes of severe facial pain originating from the trigeminal nerve.[22] Management options for TN include pharmacological therapy (e.g., carbamazepine, amitriptyline, baclofen), nerve blocks, radiofrequency ablation, gamma knife therapy, and microvascular decompression surgery.[23] Although medication remains the primary treatment, invasive procedures such as peripheral trigeminal nerve blocks can provide immediate pain relief, especially in cases where drug contraindications or side effects limit pharmacotherapy.

The advent of high-resolution ultrasound has enhanced the precision of nerve blocks by allowing real-time visualization of peripheral nerves and surrounding structures, including tendons, muscles, vessels, and subcutaneous tissue.[24–26] Ultrasound guidance minimizes risks such as collateral damage, vascular injury, thrombosis, and hematoma formation, thereby improving safety and efficacy.

Our study aimed to evaluate the efficacy of ultrasound-guided peripheral nerve blocks combined with drug therapy in patients with TN.

Demographic Profile
In our cohort, 30.3% of participants were aged 41–50 years, and 18.2% were aged 51–60 years, with a mean age of 53.39 ± 15.02 years. Similar age distribution was observed in the study by Bangas et al., where patients aged 40–80 years had a mean age of 54 years. [27] Gaurav Katheriya et al. reported a mean age of 50.62 ± 15.87 years, with the majority in the 41–60-year age group (34.9%). [28] These findings suggest that TN predominantly affects middle-aged to elderly populations, consistent across studies.

Gender Distribution
In our study, males constituted 51.5% (17/33) and females 48.5% (16/33), resulting in a male-to-female ratio of approximately 1.06. This aligns with the study by Gallery et al., where 5 of 8 patients were male (ratio 1.66).[29] Contrarily, Gaurav Katheriya et al. observed a female predominance (59.2%), with a male-to-female ratio of about 0.69.[28] The gender distribution of TN varies across populations, possibly influenced by genetic and environmental factors.

Laterality of Involvement
In our cohort, 23 patients (69.7%) had right-sided TN, and 10 (30.3%) had left-sided involvement, with no bilateral cases. This right-sided dominance is consistent with Han et al., who reported right, left, and bilateral involvement ratios of 305:107:3.[30] Similarly, Katheriya et al. found a higher right-side involvement (57.1%) compared to the left (38.8%).[28] The observed lateral predilection may be due to anatomical or neurovascular factors.

Pharmacotherapy Regimen
All patients received a combination of three drugs—carbamazepine, amitriptyline, and baclofen—followed by ultrasound-guided nerve blocks. Multidrug regimens are often necessary, especially in patients who do not achieve adequate control with monotherapy.[31]

Efficacy of Treatment
Prior to intervention, the mean NRS score was 7.75 in the nerve block plus drug group and 6.24 in the drug-only group. At 15 days, scores decreased to 5.88 (24.1% reduction) and 3.40 (45.2% reduction), respectively. By 30 days, the scores further decreased to 1.25 (83.7% reduction) and 1.76 (71.5% reduction). Notably, patients receiving nerve blocks demonstrated a greater overall reduction in pain, despite having higher baseline NRS scores, indicating the potential benefit of adding nerve blocks to pharmacotherapy.

These findings are corroborated by Gerken et al., who observed a median reduction of 3.2 points in NRS scores post-nerve block, translating into approximately a 41% reduction.[32] Additionally, sensory analgesia was achieved within 15 minutes of nerve block in our patients, consistent with Nader et al., who reported complete sensory blockade within 15 minutes.[33]

Limitations
The small sample size limited the ability to detect statistically significant differences in satisfaction scores. Additionally, the short follow-up period restricts assessment of long-term outcomes. Future studies with larger cohorts and extended follow-up are warranted.

Conclusion

Ultrasound-guided peripheral nerve blocks, combined with pharmacotherapy, significantly reduce pain intensity in patients with trigeminal neuralgia. While our findings suggest that nerve blocks can enhance pain relief, larger randomized controlled trials are needed to establish definitive efficacy and long-term benefits.

References

1. Bereiter DA, Hargreaves KM, Hu JW. Trigeminal Mechanisms of Nociception: Peripheral and Brainstem Organization. In Pain. Vol. 5. Elsevier Inc. 2008. p. 435-460 doi: 10.1016/B978-012370880-9.00174-2
2. Hargreaves KM. Orofacial pain. Pain. 2011 Mar;152(3 Suppl):S25-S32. doi: 10.1016/j.pain.2010.12.024. Epub 2011 Feb 2. PMID: 21292394; PMCID: PMC3077822.
3. Gilron I, Booher SL, Rowan MS, Smoller MS, Max MB. A randomized, controlled trial of high-dose dextromethorphan in facial neuralgias. Neurology. 2000 Oct 10;55(7):964-71. doi: 10.1212/wnl.55.7.964. PMID: 11061252.
4. Siqueira JTT de, Teixeira MJ. Dor orofacial: diagnóstico, terapêutica e qualidade de vida. [cited 2025 Oct 8].
5. Cascone P, Fatone FMG, Paparo F, Arangio P, Iannetti G. Trigeminal impingement syndrome: The relationship between atypical trigeminal symptoms and antero-medial disk displacement. Cranio. 2010;28(3):177-80.
6. Goldenberg DL, Burckhardt C, Crofford L. Management of fibromyalgia syndrome. JAMA. 2004;292(19):2388-95.
7. Benoliel R, Birman N, Eliav E, Sharav Y. The International Classification of Headache Disorders: Accurate diagnosis of orofacial pain. Cephalalgia. 2008;28(7):752-62.
8. Tesseroli De Siqueira JT, Ching LH, Nasri C, et al. Clinical study of patients with persistent orofacial pain. Arq Neuro-Psiquiatr. 2004;62(4):988-96.
9. Peñarrocha M, Mora E, Bagán JV, García B, Peñarrocha M. Idiopathic Trigeminal Neuropathies: A presentation of 15 cases. J Oral Maxillofac Surg. 2009;67(11):2364-8.
10. Hegarty AM, Zakrzewska JM. Differential diagnosis for orofacial pain, including sinusitis, TMD, trigeminal neuralgia. Dent Update. 2011;38(6):396-400.
11. Jannetta PJ. Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. J Neurosurg. 2007;107(1):216-9. (Original work published 1967)
12. Crooks DA, Miles JB. Trigeminal neuralgia due to vascular compression in multiple sclerosis - Post-mortem findings. Br J Neurosurg. 1996;10(1):85-8.
13. Toda K. Etiology of Trigeminal Neuralgia. Oral Sci Int. 2007;4(1):10-8.
14. American Academy of Orofacial Pain. The scope of TMD/orofacial pain (head and neck pain management) in contemporary dental practice. Dental Practice Act Committee of the American Academy of Orofacial Pain. J Orofac Pain 1997;11(1):78-83.
15. Okeson, Jeffrey P, and Welden E Bell. Bell's Orofacial Pains. 5th ed. Chicago: Quintessence Pub. Co., 1995. Print.
16. Katusic S, Beard CM, Bergstralh E, Kurland LT. Incidence and clinical features of trigeminal neuralgia, Rochester, Minnesota, 1945-1984. Ann Neurol. 1990 Jan;27(1):89-95. doi: 10.1002/ana.410270114. PMID: 2301931.
17. Mueller D, Obermann M, Yoon MS, Poitz F, Hansen N, Slomke MA, Dommes P, Gizewski E, Diener HC, Katsarava Z. Prevalence of trigeminal neuralgia and persistent idiopathic facial pain: a population-based study. Cephalalgia. 2011 Nov;31(15):1542-8. doi: 10.1177/0333 102411424619. Epub 2011 Sep 29. PMID: 21960648.
18. MacDonald BK, Cockerell OC, Sander JW, Shorvon SD. The incidence and lifetime prevalence of neurological disorders in a prospective community-based study in the UK. Brain. 2000 Apr;123 (Pt 4):665-76. doi: 10.1093/brain/123.4.665. PMID: 10733998.

19. Sjaastad O and Bakketeig LS. The rare, unilateral headaches. Vaga study of headache epidemiology. J Headache Pain 2007;8:19-27.
20. Maarbjerg S, Gozalov A, Olesen J, Bendtsen L. Trigeminal neuralgia--a prospective systematic study of clinical characteristics in 158 patients. Headache. 2014 Nov-Dec;54(10):1574-82. doi: 10.1111/head.12441.
21. Wang QP, Bai M. Topiramate versus carbamazepine for the treatment of classical trigeminal neuralgia: A meta-analysis. CNS Drugs 2011; 25:847-57.
22. Fashner J, Bell AL. Herpes zoster and postherpetic neuralgia: Prevention and management. Am Fam Physician 2011; 83:1432-7.
23. Spatz AL, Zakrzewska JM, Kay EJ. Decision analysis of medical and surgical treatments for trigeminal neuralgia: How patient evaluations of benefits and risks affect the utility of treatment decisions. Pain 2007; 131:302-10.
24. Graif M, Seton A, Nerubai J, Horoszowski H, Itzchak Y. Sciatic nerve: sonographic evaluation and anatomic-pathologic considerations. Radiology. 1991 Nov;181(2):405-8. doi: 10.1148/radiology.181.2.1924780.
25. Marhofer P, Schrögendorfer K, Koinig H, Kapral S, Weinstabl C, Mayer N. Ultrasonographic guidance improves sensory block and onset time of three-in-one blocks. Anesth Analg. 1997 Oct;85(4):854-7. doi: 10.1097/00000539-199710000-00026. PMID: 9322469.
26. Chan VWS, Perlas A, Rawson R, Odukoya O. Ultrasound-guided supraclavicular brachial plexus block. Anesth Analg. 2003 Nov;97(5):1514-1517. doi: 10.1213/01.ANE.00000 62519.61520.14. PMID: 14570677.
27. Bangash T H. Trigeminal Neuralgia: Frequency of Occurrence in Different Nerve Branches.Anesth Pain Med.2011;1(2):70-72.https://doi.org/10.5812/kowsar.22287523.2164.
28. Katheriya G, Chaurasia A, Khan N, Iqbal J. Prevalence of trigeminal neuralgia in Indian population visiting a higher dental care center in North India. Natl J Maxillofac Surg 2019;10:195-9.
29. Gallay MN, Moser D, Jeanmonod D. MR-Guided Focused Ultrasound Central Lateral Thalamotomy for Trigeminal Neuralgia. Single Center Experience. Front Neurol. 2020 Apr 17; 11:271.
30. Han KR, Chae YJ, Lee JD, Kim C. Trigeminal nerve block with alcohol for medically intractable classic trigeminal neuralgia: long-term clinical effectiveness on pain. Int J Med Sci. 2017;14(1):29-36.
31. Ariyawardana A, Pallegama R, Sitheeque M, Ranasinghe A. Use of single- and multi-drug regimens in the management of classic (idiopathic) trigeminal neuralgia: an 11-year experience at a single Sri Lankan institution. J Investig Clin Dent. 2012 May;3(2):98-102.
32. Gerken JD, Fritzsche T, Denke C, Schäfer M, Tafelski S. Retrospective Study on Ganglionic and Nerve Block Series as Therapeutic Option for Chronic Pain Patients with Refractory Neuropathic Pain. Pain Res Manag. 2020 Jul 25;2020:6042941.
33. Nader A, Kendall MC, De Oliveria GS, Chen JQ, Vanderby B, Rosenow JM, Bendok BR. Ultrasound-guided trigeminal nerve block via the pterygopalatine fossa: an effective treatment for trigeminal neuralgia and atypical facial pain. Pain Physician. 2013 Sep-Oct;16(5):E537-45