



SURGICAL NEURECTOMY OF INFERIOR ALVEOLAR NERVE IN REFRACTORY TRIGEMINAL NEURALGIA: A REPORT OF CASE SERIES

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Abstract

Background: Trigeminal neuralgia is a commonly diagnosed neurosensory disease of head, neck and face region, involving 5th cranial nerve. “Tic Douloureux” and “Fothergill’s disease” are the synonyms used to describe the same disease. Peripheral neurectomy is one of the oldest surgical procedures for refractory trigeminal neuralgia (RTN). Moreover, Carbamazepine is usually the preferred first line of treatment for Trigeminal neuralgia, followed by some choice of drugs like Lamotrigine and Baclofen and anti-epileptics like Phenytoin, Gabapentin, Topiramate, Tocainide and Valproate. If there is a decrease in efficacy or development of tolerance to the drugs, some surgical modality needs to be considered. The purpose of this study was to present case reports of sixteen patients with the case of refractory trigeminal neuralgia who were suffering from chronic, debilitating trigeminal neuralgia and severe throbbing pain for 2-3 years and treated with Carbamazepine with no significant effect. The patients were treated and cured with neurectomy of Inferior Alveolar Nerve under general anaesthesia. The visual analogue scale (VAS) was used for pain assessment preoperatively and during the follow-up period. The outcome of surgery was graded as a marked, moderate, or mild improvement. Kaplan–Meier analysis was used for the time to recurrence to predict the probability of recurrence at any given time following the procedure. Fourteen patients had marked improvement of pain. There was recurrence of pain in 2 patients (12.5%) in an 18 month follow-up period in this study. The mean preoperative Hospital Anxiety and Depression Scale Anxiety and Depression scores significantly improved on the last follow-up visit following the procedure ($P < 0.001$).

Conclusion: Neurectomy of Inferior Alveolar Nerve is thus a safe and effective procedure for elderly patients, for those patients living in remote and rural places that cannot avail major neurosurgical facilities, and for those patients who are reluctant for major neurosurgical procedures. The preoperative severity of pain, anxiety, and depression levels also improved markedly after the procedure.

Key words: Neurectomy, trigeminal neuralgia, surgery, inferior alveolar nerve.

Introduction:

Trigeminal neuralgia (TN) is a rare neurosensory disease of the orofacial region involving the fifth cranial nerve. It is defined as “unilateral disorder characterized by brief electric shock-like pain, abrupt in onset and termination, and limited to the distribution of one or more divisions of the trigeminal nerve” [1]. The disease is mostly idiopathic. Common etiological factors include compression of the nerve trunk due to central pathology/compression by tortuous vessel or demyelination of the involved nerve [2-4]. Usually, there is the involvement of a single division of the trigeminal nerve, which may slowly spread to the other divisions, lasting from a fraction of a second to minutes and is triggered by trivial cutaneous or intraoral stimuli [4-6]. The estimated annual incidence of TN is about 13 per 100,000 persons/ year [7]. It most commonly occurs over the age of 40 years with a slight female predilection [8,9].

As a specific entity of facial pain, TN today is recognized by a variety of classification systems:

- International Classification of Headache Disorders (ICHD), created by the International Headache Society (IHS) [10] (table-1A & 1B)
- Classification of Chronic Pain, from the International Association for the Study of Pain (IASP) [11]
- International Classification of Diseases (ICD) coding, by the World Health Organization (WHO)[12]
- Classification from the American Association of Orofacial Pain (AAOP).
- Clinical classification of trigeminal pain by Burchiel and by Cruccu et al [13,14]

TN thus refers to a category of disorders affecting one or more branches of the trigeminal nerve that present with neuropathic pain. They are classified as follows:

Table-1A: International classification of headache disorders ICHD criteria for classical Trigeminal Neuralgia

A.	Paroxysmal attacks of pain, lasting from a fraction of a second to two minutes, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B and C.
B.	Pain has atleast one of the following characteristics. <ul style="list-style-type: none"> • Intense, sharp, superficial or stabbing. • Precipitated from trigger areas or by trigger factors.
C.	Attacks are stereotyped in the individual patient.
D.	There is no clinically evident neurological deficit.
E.	Not attributed to another disorder.

Table-1B: International classification of headache disorders ICHD criteria for symptomatic Trigeminal Neuralgia.

A.	Paroxysmal attacks of pain, lasting from a fraction of a second to two minutes, with or without persistence of aching between paroxysms, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B and C.
B.	Pain has atleast one of the following characteristics. <ul style="list-style-type: none"> • Intense, sharp, superficial or stabbing. • Precipitated from trigger areas or by trigger factors.
C.	Attacks are stereotyped in the individual patient.
D.	A causative lesion, other than vascular compression, has been demonstrated by special investigations and/or posterior fossa exploration.

Type I Trigeminal Neuralgia: It is also known as typical TN, type I TN is characterized by unilateral, severe, brief, paroxysms of sharp painful attacks in the distribution of one or more branches of the trigeminal nerve. These attacks are often described as electrical and shock-like. The pain is maximal at onset, lasts several seconds, and is triggered by nonpainful stimuli. Typical triggers include cold air, brushing teeth, chewing, or talking. Trigger zones are areas in the distribution of the affected nerve branch, close to the midline. Even light touch of these trigger zones can provoke paroxysms of pain. Between episodes of pain, there are refractory periods when previous triggers no longer produce pain. Patients may become dehydrated and experience weight loss due to avoidance of triggers. The pain may be bilateral. The Cruccu classification subdivides type I TN into idiopathic TN if no vascular compression is demonstrated on imaging, or classic TN if neurovascular compression is demonstrated [15-18].

Type II Trigeminal Neuralgia: Type II TN has continuous or near-continuous pain superimposed upon the sharp painful attacks seen in type I TN. Similar to type I, the Cruccu classification includes this syndrome into both idiopathic forms if no vascular compression is noted on imaging, or classic TN if neurovascular compression is seen[19].

Secondary Trigeminal Neuralgia:

Secondary TN is pain experienced in a trigeminal distribution caused by neurological disease other than neurovascular compression. Broadly, these etiologies may fall into inflammatory/demyelinating disease (eg, multiple sclerosis, sarcoidosis), tumours (eg, meningioma, vestibular schwannoma, trigeminal schwannoma, epidermoid, metastasis, glioma), other vascular lesions (eg, aneurysms, arteriovenous malformations, persistent trigeminal artery), connective tissue disorders (eg, scleroderma, mixed connective tissue disease), congenital diseases, and other systemic conditions (eg, Paget's disease, acromegaly, syphilis) which may affect the trigeminal nerve. TN should not be confused with other cranial nerve syndromes such as geniculate neuralgia versus nervus intermedius neuralgia, glossopharyngeal neuralgia,[13–16] superior laryngeal neuralgia, paratrigeminal neuralgia (Raeder's syndrome),[17–21] or occipital neuralgia[7] Trigeminal neuropathy due to herpes zoster, trigeminal postherpetic neuralgia[22–28] painful post-traumatic trigeminal neuropathy,[29–33] or trigeminal deafferentation pain (eg, anaesthesia dolorosa)[8,12,34] must be distinguished from TN. Pain related to the mouth such as dental pain, first bite syndrome,[35] or burning mouth syndrome should be separately elucidated. Other headache syndromes which should be diagnosed separately from TN include cluster headaches,[36] sphenopalatine neuralgia (Sluder's neuralgia), Short Lasting Unilateral Neuralgiform Pain with Conjunctival Injection and Tearing (SUNCT), and Short Lasting Unilateral Neuralgiform Pain with Cranial Autonomic Symptoms (SUNA). Rare other forms of facial pain syndromes such as persistent idiopathic facial pain, pain of psychological origin, and central neuropathic pain should be distinguished from TN. [37–39]

Epidemiology:

The prevalence rate of TN ranges between 0.03% and 0.3%.^{48–51} There is a female preponderance, with a male: female ratio between 1:1.5 and 1:1.7. Classic TN is generally diagnosed in elderly population with peak incidence between 50 and 60 years. V2 and V3 are the most commonly affected branches of TN. In less than 5% of the cases is the ophthalmic (V1) branch affected in isolation. Trigeminal symptoms are present in 2–4% of the patients with multiple sclerosis, and in 1–5%, it can be the presenting feature of the disease. Conversely, multiple sclerosis is detected in 2–14% of the patients with TN.[12,14] TN is more commonly seen in adults compared with pediatric TN, which comprises less than 1.5% of all cases.[20–22] . Paediatric TN is more likely to be bilateral (42%) compared with adult TN. It is associated with compression of multiple cranial nerves (46%) as a result of congenitally abnormal vessels, vascular malformations, tumors, cysts, aneurysms, or arachnoiditis.

Differential Diagnosis:

Even though they are less common than iatrogenic injury or trauma, other conditions can alter trigeminal nerve sensation and lead to clinical presentations similar to IAN injuries.[19] These include:

- Benign or malignant tumours involving the trigeminal nerve tract
- Autoimmune disorders: lupus erythematosus, dermatomyositis, progressive sclerosis, Sjögren's syndrome, rheumatoid arthritis, and other connective tissue diseases
- Infections: herpes zoster, herpes simplex virus, syphilis, leprosy
- Multiple sclerosis
- Vertebrobasilar disease
- Sarcoidosis
- Amyloidosis
- Sickle cell anemia

Treatment Strategies for RTN:

First-line treatment therapies for RTN involve medical management with anticonvulsant medications (table-1). Patients who fail medical management due to persistent pain or unacceptable side effects have transcutaneous, percutaneous, radiotherapy, and open surgical options available to them. Patient selection criteria, as well as the pros and cons of each procedure option are summarized in Table 2. In general, percutaneous, minimally invasive, and open surgical therapies for TN are most effective in patients with type 1 TN.

Table-2A: Medical Therapies for the Treatment of Trigeminal Neuralgia

Medication	Common Dosing	Side Effects	Monitoring
Carbamazepine	50 mg twice a day (elderly population) 100 mg twice a day (younger populations)	HLA-B*1502 variant patients, esp. in Asian patients, have increased risk of SJS/TEN Drug-drug interactions, by inducing CYP3A4	Monitor sodium, CBC, LFTs at baseline and periodically after HLA-B*1502 variant screening
Oxcarbazepine	150 mg twice a day (to start) 300–600 mg twice a day (goal; max 1800 mg per day)	Hyponatremia HLA-B*1502 variant patients have increased risk of SJS/TEN	Monitor sodium, HLA-B*1502 variant screening
Phenytoin and fosphenytoin	15–20mg/kg	Ataxia, dysarthria, nystagmus Significant incidence of recurrence of the neuralgia despite continuous use	If long-term dosing attempted, free and total phenytoin levels
Baclofen	15–80mg per day	Sedation, hypotonia, GI upset	NA
Lamotrigine	100bid	SJS/TEN, skin rash, sedation, nausea	NA
Pimozide	4–12 mg per day	Extrapyramidal symptoms, QT prolongation, neuroleptic malignant syndrome, hemolytic anemia, dry mouth, sedation, constipation	ECG, fasting glucose and lipids, CBC, CMP, monitoring for extrapyramidal symptoms
Levetiracetam	3000–5000 mg per day divided BID or TID	Agitation or worsening depression	NA
Gabapentin	300–1200 mg TID	Sedation, foggy thinking, lower extremity edema or weight gain	NA
Pregabalin	300–600 mg divided BID	Sedation, dizziness, lower extremity edema, blurry vision, possible thrombocytopenia	NA
Clonazepam	6–8 mg per day	Sedation, ataxia, memory impairment, withdrawal	NA
Valproate	500–1500 per day	Weight gain, hair loss, nausea, hepatotoxicity, pancreatitis, fetal malformations, thrombocytopenia	Total and free valproate level, LFTs, CBC, ammonia
Misoprostol	600 µg per day	Diarrhea, GI discomfort, menorrhagia	NA

Table-2B Drug therapy for Trigeminal Neuralgia

Drug	Initial dose	Maintenance dose
Gabapentin	300 mg tid	1800 mg
Baclofen	5 mg bid/tid	80 mg maximum dose
Clonazepam	0.5 mg tid	4 mg, maximum 20 mg
Lamotrigine	50 mg qd	300-500 mg
Oxcarbazepine	300 mg bid	1200 mgbid
Toprimate	50 mg qd	200 mg bid
Carbamazepine	100 mg bid	1200-2400 mg

Figure-1 Schematic diagram of treatment modalities for trigeminal neuralgia. (A) Treatment modalities for trigeminal neuralgia grouped by operative approach. (B) Treatment modalities for trigeminal neuralgia grouped by mechanism of treatment

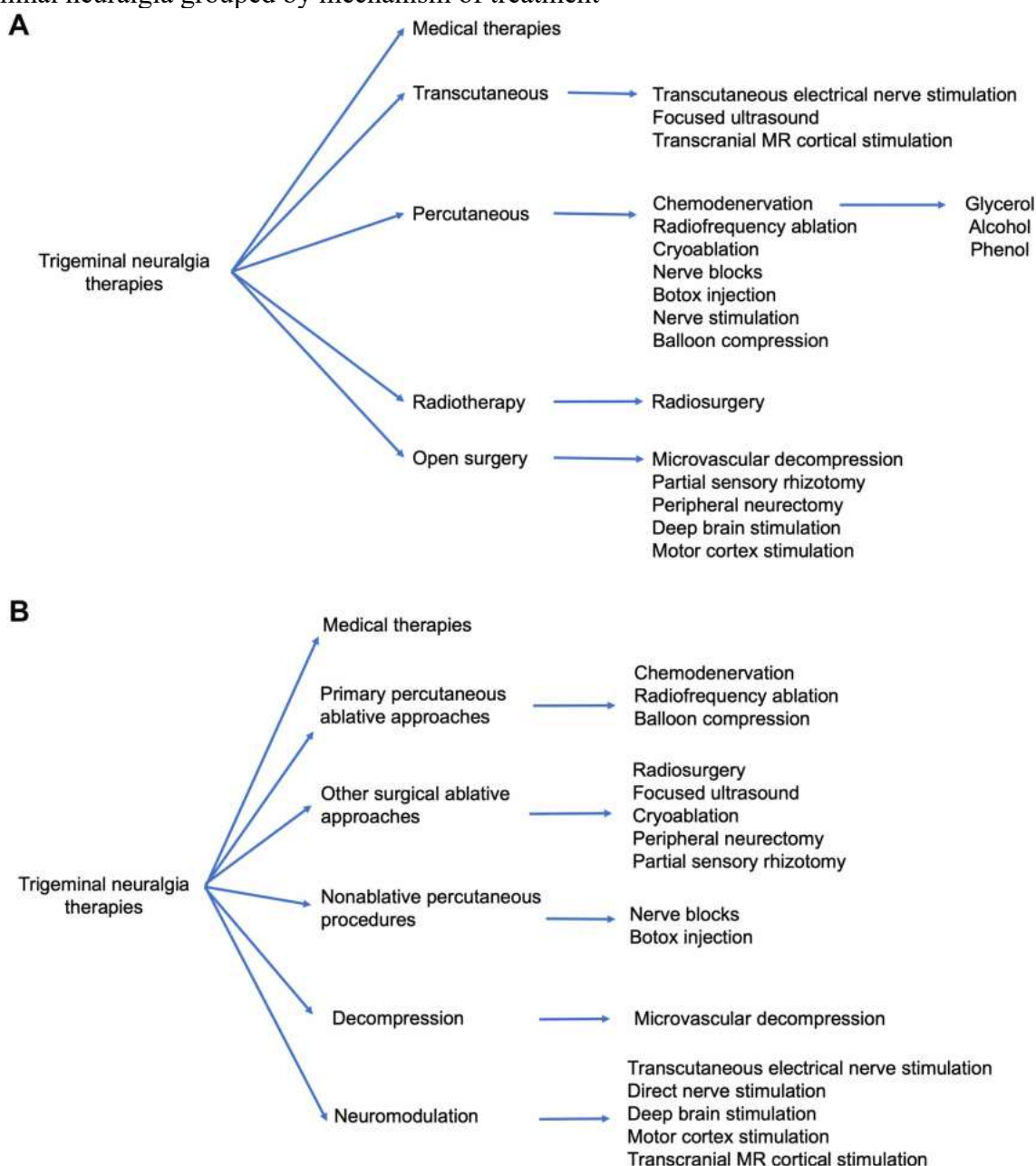


Table-3: Summary of Transcutaneous, Percutaneous, Radiotherapy, and Open Surgical Treatment Options for Patients with Trigeminal Neuralgia [43]

	Patient Selection Criteria	Initial Pain Relief Rates	Pain Recurrence
Transcutaneous			
TENS	Retractable disease without pain relief after medication	80–90% within 3 weeks	85% Reduction in pain after 3 months, but long term outcomes are not well studied
Focused ultrasound	Failed standard therapies, but further study is needed	Further study is needed	Further study is needed
Transcranial MR cortical stimulation	May be used as assessment method for cortical stimulation	50–60%	Long-term studies are lacking
Percutaneous			
Chemodenevation			
Glycerol	Failed medical management	70–90+%	20–40% Have pain relief
Alcohol	Failed medical management	80–90% success rate	>50% Require retreatment
Phenol	Failed medical management; end-stage cancer patients	80–90%	~40% Recurrence 1–2 years after procedure
Radiofrequency ablation	Failed medical management	75–95+%	25–50% Recurrence
Nerve blocks	Failed medical management	30–40%	Pain relief may last longer than expected based on local anesthetic's duration of action, 50–60% have sustained pain relief
Balloon compression	Failed medical management	80–90+%	15–50%
Cryoablation	Failed standard therapy	90+%	30–40%

	Patient Selection Criteria	Initial Pain Relief Rates	Pain Recurrence
Botox injection	Failed standard therapies	50–60%	50–60% Require second dose at 2 months, long term outcomes need further study
Nerve stimulation	Most commonly treating Type 2 TN in literature	40–50% but sample size is limited	Long-term outcomes need further study
Radiotherapy			
Radiosurgery	Patients who cannot tolerate general anesthesia or invasive procedures	Pain relief is not immediate; maximum time to pain relief is around 180 days after treatment	20–30%
Open surgery			
Microvascular decompression	Ability to tolerate general anesthesia and suboccipital craniectomy	>90%	~10% Underwent second operations; most recurrences within 2 years of surgery
Partial sensory rhizotomy	Absence of neurovascular contact on MRI	80–90%; Similar to slightly worse than MVD patients	Worse than MVD, 47% pain free at 5 years
Peripheral neurectomy	Failed medical therapy or severe medical comorbidities and unable to tolerate MVD suboccipital craniectomy	70–90+%	Up to 20%, Recurrence thought to be secondary to peripheral nerve regeneration
Deep brain stimulation	Refractory TN, excluding patients with psychogenic or factitious pain disorders, cognitive impairment, and psychiatric disease	>90%, but sample size is small	60% Require medication on follow-up, but long term outcomes are not well studied

	Patient Selection Criteria	Initial Pain Relief Rates	Pain Recurrence
Motor cortex stimulation	rTMS may be used as an initial assessment for cortical stimulation	60–80+%, but further studies are needed	>50%, but long-term outcomes are poorly studied

Inferior Alveolar Nerve:

The inferior alveolar nerve has a close relationship to the lingual nerve. After separating approximately 5 mm below the cranial base, the inferior alveolar nerve travels between the lateral and medial pterygoid muscles. The inferior alveolar nerve passes around the lower border of the lateral pterygoid muscle and then proceeds to the medial aspect of the ramus of the mandible to enter the mandibular foramen. It has been shown that in approximately 34% of mandibles, the neurovascular bundle divides soon after the beginning of the inferior alveolar canal.⁴⁴ Other studies have not shown as high an incidence, varying from 0.96% to 8%.³⁴ The superoinferior course of the nerve in the mandible is such that it descends to the lowest point near the first molar and then rises again. In a lateromedial position, the canal and bundle are closest to the lateral cortical plate in the third molar area, but the nerve remains constant in its relationship to the medial cortical plate throughout its course [34-36]. The mylohyoid nerve branches off the inferior alveolar nerve before entering the canal. This nerve runs downward and anteriorly in the mylohyoid groove before innervating the mylohyoid muscle, anterior belly of the digastric muscle, and sensory fibers of the chin area. Exiting the mental foramen, the inferior alveolar nerve divides into terminal mental nerves, which supply sensation to the chin, lower lip, and mucous membranes of the lower lip [42].

Causes of Inferior Alveolar Nerve Damage:

The inferior alveolar nerve can also be injured or damaged by certain acute (sudden, severe) or chronic (persistent medical conditions), including:

Iatrogenic Causes: A rare but serious complication associated with procedures like wisdom tooth extraction, lower-jaw dental implants, and orthognathic surgery[5, 42].

Trigeminal neuralgia: A painful condition that causes sudden and extreme shock-like pain on the side of the face [16]

Multiple sclerosis: A progressive neurological disease that often manifests with trigeminal neuralgia as the first symptom [27]

Oral cysts: Including radicular cysts in the pulp of the tooth, dentigerous cysts associated with impacted teeth, and aneurysmal bone cysts of the lower jawbone [38]

Ameloblastoma: A benign (non-cancerous) tumor that most often develops in the lower jaw near the molars[38]

Central giant cell granuloma: A benign condition of unknown origin that causes granular lesions, most often in the back part of the lower jaw [8]

Fibrous dysplasia: A condition in which bone is displaced by fibrous scar tissues, most commonly affecting long bones but occasionally the lower jaw as well [44].

Symptoms of Inferior Alveolar Nerve Damage: [44]

Inferior alveolar nerve damage can be permanent or short-lasting depending on the underlying cause. The impact of the damage can be substantial, affecting speech, eating or drinking, brushing your teeth, kissing, or performing everyday tasks like shaving or applying makeup.

Symptoms of damage to the inferior alveolar nerve include:

Pain

Abnormal sensations (tingling, electrical shock-like sensations)

Numbness in the chin, lower lip, or around the lower teeth

Drooling

Impaired speech

Difficulty opening the mouth

Inferior Alveolar Nerve Neurectomy:

The inferior alveolar nerve was accessed intraorally. A linear incision was placed over the anterior border of the ramus of the mandible. Soft tissue over the anterior and medial surface of the ramus was reflected in the subperiosteal plane. Careful blunt dissection was done medially at the level of the occlusal plane, to identify the inferior alveolar neurovascular bundle as it entered the mandibular foramen. The nerve was held in a haemostat. Two sutures were passed around the nerve, one above and one below the haemostat and tied. The nerve was resected between the two ligature sutures, and the distal segment of the nerve was avulsed holding it by the tip of the haemostat. Closure of mucosa was one using resorbable sutures [37-41].

This case report presents two cases of Refractory Trigeminal Neuralgia involving third division of the trigeminal nerve and the patient was treated by neurectomy of inferior alveolar nerve.

Materials and Methods:

Two RTN patients with inferior alveolar neuralgia, unwilling to consider neurosurgery options, were admitted to the surgical ward of the Department of Oral and Maxillofacial Surgery, Hind Institute of Medical Science, Sitapur, UP, India, between January 2024 and July 2025. There were one female, aged 55 years and one male patient aged 62 years. The right IAN was operated on for female patients, and the left IAN for male patients. The average length of complaint was 8.2 (range: 3–17) months. Male patient had a history of oral carbamazepine treatment, and female patient reported irregular administration of oral analgesics. Oral carbamazepine was initially effective in pain management, but lost effectiveness over time and could no longer manage sharp pain.

Surgical Procedures:

Routine preoperative examination was conducted, including blood count, urine routine, blood coagulation time, liver and renal function, HBV test, electrocardiogram, chest radiograph, etc. The patient, lying in supine position, was given general anaesthesia via nasal-tracheal intubation on the unaffected side. The patient's jaws were held wide open with a mouth gag; adrenaline saline was injected by the pterygomandibular ligament of the operated side to reduce surgical bleeding and keep the surgical field clear. A ~1cm longitudinal incision was created 1 cm from the pterygomandibular ligament (cold light source to keep the surgical view clear), and then the following steps were

performed:

- 1) Finding the IAN in the pterygomandibular space, by the mandibular lingual, via the medial of the mandibular ramus.
- 2) Hooking the IAN, to remove the lingula, expanding the mandibular foramen, and tracing and separating the nerve upward and backward.
- 3) Finding the IAN mandibular canal entrance with the nerve hook.
- 4) Exploring at different angles to enter the mandibular canal.
- 5) Dissecting and completely removing the IAN, followed by electrocoagulation of the mandibular canal entrance.
- 6) Rinsing the surgical site, followed by haemostasis, suture, and postoperative blood drainage.

Inferior alveolar neurectomy

- o Extra oral approach- Risdon's incision.
- o Intra oral approach- Dr Ginwalla's incision

A few possible adverse effects after this minimal invasive procedure, depending upon the branch of the nerve extirpated, are paraesthesia, facial sensory loss, weakness or paralysis of masseter muscles and, rarely, loss of the corneal reflex.

In view of above facts and to the best of knowledge, no studies have been reported on large number of patients with the case of refractory trigeminal neuralgia who were suffering from chronic, debilitating trigeminal neuralgia and severe throbbing pain for 2-3 years and treated with Carbamazepine with no significant effect in rural North Indian population. Hence, this study was aimed to evaluate the sixteen patients who were treated and cured with neurectomy of Inferior Alveolar Nerve under general anesthesia, also evaluated the clinical outcome and the recurrence rate following neurectomy for the management of RTN. To the best of knowledge, this was the first

study to use Kaplan–Meier analysis for the time to recurrence to predict the probability of recurrence at any given time following the procedure. The severity of the disease and the anxiety and depression status of the patients before and after the procedure were also evaluated.

Patients and Methods:

This was a prospective study of 16 patients, aged 30–70 years, both sexes with classical RTN treated by neurectomy of Inferior Alveolar Nerve under general anaesthesia, in the Department of Dentistry, Hind Institute of Medical Science, Sitapur, UP, India, from January 2024 to August 2025. The study was approved by the Institutional ethical committee. The classical RTN was diagnosed according to Sweet's criteria (the pain was paroxysmal, may be provoked by light touch to the face, confined to trigeminal distribution, unilateral and the clinical sensory examination was normal). The patients in this study included those who were resistant to medical treatment or developed intolerance to medications due to their side effects and those who were unfit because of either old age (above 70 Years) or comorbidity or reluctant to have invasive neurosurgical procedures. Patients younger than 30 years old, those with symptomatic RTN (a causative lesion, other than vascular compression like multiple sclerosis or tumour), those with an atypical presentation like bilateral involvement, and the patients with follow-up periods <1 year were excluded from the study.

The patients were assessed preoperatively including a detailed history, clinical general and neurological examination, and the response of pain to carbamazepine was detected. Diagnostic block with 2% lignocaine was used to confirm the involved division of the nerve in the patients after detecting the site of the pain by history and clinical examination when it gave complete relief from the symptoms. All patients underwent magnetic resonance imaging of the brain to rule out underlying structural lesions such as tumours or vascular malformations. Carbamazepine tablets 600–1200 mg daily were prescribed for all patients. It was used continuously or intermittently for an average of one to 3 years, sometimes alone and sometimes with other drugs to treat the condition. The visual analogue scale (VAS) was used for pain assessment preoperatively and during the follow-up period. Informed written consent was obtained from all the patients.

Antibiotics and anti-inflammatory drugs were prescribed for all patients postoperatively for 5–7 days. The initial postoperative relief of pain was assessed during the 1st week after surgery. The patients were followed up after surgery at 1 month, 3 months, 6 months then annually up to 18th months or if a change of pain severity appeared. VAS was used for pain assessment during the follow-up period. The outcome of surgery was graded as (A) Marked improvement (reduction of pain more than 90% of preoperative pain without medications for RTN); (B) Moderate improvement (50%–89.9% reduction of preoperative pain, treatment with low doses of Carbamazepine was allowed in this group); and (C) Mild improvement (reduction of pain <50% compared to preoperative pain, long-term medication was resumed, or an additional procedure was performed for RTN). Recurrence was defined as a transition from marked improvement group to either the moderate or mild improvement group during the follow-up period. Kaplan–Meier analysis was used for the time to recurrence to predict the probability of recurrence at any given time. Hospital Anxiety and Depression Scale (HADS) was used for the assessment of anxiety and depression status of the patients during the follow-up visits in comparison to the preoperative status. The degree of relief of pain, recurrence of pain, postoperative complications, the need for additional procedures in case of recurrent pain were recorded during the follow-up visits.

Statistical analysis:

Data were statistically described in terms of mean \pm standard deviation, median and range, or frequencies (number of cases), and percentages when appropriate. Numerical data were tested for the normal assumption using the Shapiro–Wilk test. Comparison between pre and postoperative data was done using the Wilcoxon signed rank test for paired (matched)

samples. Survival analysis for the time to recurrence was done using Kaplan–Meier statistics calculating the mean and median survival time for each group with their 95% confidence interval and the corresponding survival graphs. Two-sided $P < 0.05$ was considered statistically significant. All statistical calculations were done using the computer program IBM Statistical Package for the Social Science (SPSS) (SPSS; IBM Corp, Armonk, NY, USA) release 26.1 for Microsoft Windows.

Results:

The study included 16 patients who fulfilled the inclusion criteria. The mean age of the patients was 53.6 ± 10 (range, 30–67) years. The female/male ratio was 2.4:1. The characteristics of the patients in the study were shown in Table-4.

Table-4: The characteristics of the patients in the study (n=16)

Characteristic	Value (%)
Age (years)	
30-40	1 (6.25%)
41-50	3 (18.75%)
51-60	7 (43.75%)
61-70	5 (31.25%)
Sex	
Male	4 (25%)
Female	12 (75%)
Side of the face involved	
Right	13 (81.25%)
Left	3 (18.75%)
Preoperative duration of symptoms (years)	
Mean \pm SD	2.8 \pm 2.4
Range	1-4
Divisions of the trigeminal nerve involved	
V2 only	3 (18.75%)
V3 only	5 (31.25%)
V2 and V3	7 (43.75%)
V1, V2, and V3	1 (6.25%)

SD – Standard deviation

Table-5: Comparison of Visual Analogue Scale over time in the study patients (n=16)

VAS (years)	n (%)	Mean \pm SD	P
Preoperative	16 (100%)	8.18 \pm 0.760	
1.5	16 (100%)	0.53 \pm 0.624	<0.001*
2	15 (93.75%)	0.81 \pm 1.276	<0.001*
3	13 (81.25%)	1.07 \pm 0.730	0.001*

4	7 (43.75%)	1.43±0.787	0.017*
5	5 (31.25%)	1.60±0.894	0.042*
6	4 (25%)	1.25±0.500	0.066*
7	2 (12.5%)	1.50±0.707	0.180
7.5	2 (12.5%)	1.00±0.000	0.180

*P<0.05 was considered statistically significant. SD – Standard deviation, VAS – Visual Analogue Scale

Case Series- 1-12

A total of 12 female patients, aged 30-70 years, the mean age of the patients was 54.4 ± 9 (range, 30–68) years were reported to the Department of Oral and Maxillofacial Surgery, HIMS, Sitapur, UP, India with a chief complaint and a history of severe, shock like and throbbing pain in the left/right V3/ V2 & V3 region, lasting for few seconds. Aggravating factors were talking, chewing, smiling, with strong breeze and cold water while washing her face. Sleep was disturbed because of pain. Patient described trigger zones to be on left/right lower pre-molar region. Medical management with drugs like carbamazepine 200 mg (8 hourly) prescribed initially for two weeks then gradually increased up to 600 mg and was advised for 3 months which failed to alleviate her pain. Pre-operative radiographic investigation was done with OPG to rule-out any underlying pathology or any anatomical abnormalities. No abnormality was detected on OPG. The left inferior alveolar nerve was identified according to the site of pain and confirmed by diagnostic block with 2% lignocaine. After thorough examination and confirmation, the patient and her family were explained about the treatment options, its advantages and disadvantages. Informed and written consent obtained, inferior alveolar nerve neurectomy under local anaesthesia was planned as patient was reluctance to undergo invasive neurosurgical procedures. High mandibular nerve block by Gow-Gates technique given using 2% lignocaine with 1:2,00,000 adrenaline and Inferior alveolar nerve, lingual nerve, long buccal nerve and mental nerve were blocked. Intra oral supra periosteal dissection done in the retro molar area (Dr. Ginwalla's incision). Mandibular foramen was visualized and little above it the neurovascular bundle was identified stretched and held with haemostat. A few mm down another haemostat was used to hold neurovascular bundle. The second vestibular incision in the premolar region was done buccally on the same side. The mental nerve was identified, little stretched and held with another small artery forceps, dissected, ligated and transected. The left inferior alveolar nerve was dissected, ligated and transected between two hemostat just above the mandibular foramen. Avulsion of the inferior alveolar nerve was done from the distal segment from the mental foramen by pulling and rotating the artery forceps at the distal segment area. The mental foramen was closed with bone wax. Layer wise suture done with 3-0 black, non-absorbable silk at both the incision area. Haemostasis was achieved. Routine postoperative antibiotics and anti-inflammatory medications were prescribed for 5 days. Follow up was done to review the patient post-operatively on 2nd, and 7th day to review and rule out pain, infection, and bleeding or suture dehiscence at the surgical sites. Sutures were removed on the 7th post-operative day. Patient was reviewed periodically after 1, 3, 6, 12 and 18 months. In 18 months, follow-up period, patient remains symptom free.

Case Series- 13-16

A total of 4 male patients, aged 30-70 years, the mean age of the patients was 57.2 ± 10 (range, 30–69) years were reported to the Department of Oral and Maxillofacial Surgery with a chief complaint of acute bouts of pain on right/left side of face, which was lancinating and electric shock type lasting for few minutes, triggered on washing face, talking and eating food since 2 year. The patients were not responding to carbamazepine. A detailed history was taken, and comprehensive trigeminal nerve examination and cranial nerves examination was carried out. Diagnostic block in inferior alveolar nerve region with 2% lignocaine with 1:80,000 adrenaline was given, which has relieved the symptoms for 2 hours. There was recurrence of the symptoms on touching the trigger zones

when once the anaesthetic effect wore off. This confirmed the involvement of inferior alveolar nerve and was suggestive of trigeminal neuralgia involving inferior alveolar nerve. All the reports were in the normal limits. The peripheral neurectomy of inferior alveolar nerve was planned under General Anaesthesia. Inferior alveolar nerve was approached intraorally by Dr Ginwalla's incision; the nerve was identified and avulsed from the distal end. Vestibular incision in premolar region was made; the mental nerve was identified & avulsed from the mental foramen and from the soft tissues. The nerve was carefully separated from surrounding tissues and held with an artery forceps; the nerve was avulsed by winding around the artery forceps. The remaining nerve remnants were cauterized deeply. Wound closure was done in two layers using 3-0 vicryl. The patients were asymptomatic on regular follow ups of 18 months.

The patients were treated in a similar manner with an excellent outcome with total resolution of the neuralgic pain.

Table-6: Comparison of Visual Analogue Scale over time in the study patients (n=16)

VAS Score Months	Cases, N=16	VAS (Mean±SD)	P value
Preoperative	16	8.18±0.76	0.001
Post operative (Day 7)	16	1.17± 0.88	
1 month	16	0.86±0.69	
2months	16	0.53±0.79	
3months	16	1.05±0.45	0.631
6 months	16	0.89±0.44	
9 months	16	0.79±0.32	
12 months	16	0.56±0.69	
18 months	16	0.97±0.54	

*P<0.05 was considered statistically significant. SD – Standard deviation, VAS – Visual Analogue Scale

The mean preoperative VAS was 8.18 ± 0.76 . Initially, the pain was relieved in all patients following the procedure. The mean pain free interval was 1-18 months. The VAS had significantly improved during the follow-up period. At 1 and 18 months of follow-up period, the mean VAS was 0.86 ± 0.69 and 0.97 ± 0.54 . [Table 6]

Table-7: Comparison of preoperative anxiety and depression versus on the last follow-up visit in the study patients

Variables	Cases N=16	Mean±SD (HADS)	P value
Anxiety (preoperative)	16	11.48±2.13	0.001
Anxiety (on last follow-up visit)	16	6.59± 1.88	
Depression (preoperative)	16	12.07± 2.33	0.001
Depression (on last follow-up visit)	16	6.27±1.43	

*P<0.05 was considered statistically significant. SD – Standard deviation, HADS – Hospital Anxiety and Depression Scale

The mean preoperative HADS Anxiety score was 11.48 ± 2.13 , which significantly improved to 6.59 ± 1.88 on the last follow up visit following the procedure ($P < 0.001$). Likewise, the mean

HADS Depression score was 12.07 ± 2.33 which significantly improved to 6.27 ± 1.43 on the last follow-up visit ($P < 0.001$) [Table-7].

Long term follow up is required to establish the effectiveness of peripheral neurectomy on trigeminal neuralgia.

Figure-2: Intraoperative photographs of infraorbital nerve neurectomy showing the incision marking at lower buccal vestibule (Male)



Figure-3: Inferior alveolar nerve



Figure-4: Inferior alveolar nerve (Female)



Discussion:

The trigeminal nerve, or cranial nerve V (CN V), has three major divisions and is responsible for facial sensation as well as certain motor functions. The ophthalmic and maxillary divisions are purely sensory, while the mandibular division carries both sensory and motor components. Disorders affecting the trigeminal nerve can result in trigeminal neuralgia (TN) or in the loss of sensory and/or motor function within its distribution. RTN typically involves one or more branches of the trigeminal nerve and is characterized by episodes of severe, sharp, shock-like facial pain. It is the most commonly diagnosed form of neuralgia, with a mean incidence of 4 per 100,000 individuals. Although it most commonly presents in individuals over the age of 50, TN can occur at any age [12]. There is a female predominance ranging 1:2 to 2:3. The typical form results in episodes of intense pain, lasting for several seconds to a few minutes on one side of the face.

Patients may experience multiple daily attacks. While asymptomatic between episodes, they live in constant fear of recurrence, resulting in a significant psychological burden and reduced quality of life [13].

RTN is a clinical diagnosis based on a history of sudden, lancinating or stabbing pain with pain-free intervals between attacks and the absence of clinical findings. Injection of local anesthetic may aid diagnosis by confirming the trigger zone. A positive response to anticonvulsant therapy further supports the diagnosis, with drug treatment being convenient and generally well tolerated by most patients with TN [8]

The choice of surgical treatment for TN should be individualized. Peripheral neurectomy, a day-care procedure performed under local anesthesia with minimal instrumentation, is particularly useful for patients unwilling to undergo major neurosurgery and for those in rural areas lacking advanced neurosurgical facilities [9]. Owing to its low morbidity, this technique is often preferred by both patients and maxillofacial surgeons. The 'ignition hypothesis' has been proposed to explain the pathophysiology of TN [10], suggesting that injury to trigeminal axons, most often due to vascular compression at the root entry zone, precipitates the condition. Imaging studies have demonstrated evidence of demyelination and re-myelination in this region [11]. These damaged neurons become hyper excitable, producing 'after-discharge' bursts that may be triggered by external stimuli and persist beyond the stimulus duration. Through ephaptic crosstalk between demyelinated fibers, these discharges recruit adjacent neurons, resulting in the characteristic paroxysmal pain. The refractory period is attributed to post-burst potassium influx and subsequent neuronal hyperpolarization.

Peripheral neurectomy represents a minor, safe, minimally invasive surgical option that can be performed as an outpatient procedure under local anesthesia [13]. It involves surgically dividing or avulsing a peripheral branch of the trigeminal nerve to disrupt the afferent pain pathways. This approach may be favoured for patients who cannot undergo more invasive neurosurgical procedures due to financial or medical constraints. A study performed peripheral neurectomy on 14 patients with terminal branches of trigeminal nerve who were elderly and unfit for surgery. Postoperative pain was relieved after 15 to 24 months of follow-ups [29]. There were no intra-operative or post-operative complications. None of the patients had post-operative infection [29]. In a recent study on peripheral neurectomy of the infraorbital nerve, the reports shown the good post-operative results in the follow-up period between 12 to 15 months [18]. A study also reported a case of pain relief from peripheral neurectomies, for the infraorbital nerve after 24 months of follow-ups and that for the mental nerve was 26 months follow-up [21]. A similar study also reported a case series of 63 patients with 112 neurectomies, in which a follow-up period of 0–9 years was noted, and a pain relief period of 24–32 months was reported [20].

Surgical intervention is considered for medically refractory cases or when drugs produce intolerable side effects. Peripheral neurectomies offer a treatment option, particularly for select patient populations [34–38]. However, this approach has limitations compared to other definitive central neurosurgical procedures such as microvascular decompression (MVD) and gamma knife radiosurgery (GKS) [22–27]. Despite its historical limitations, peripheral neurectomy remains a valuable tool in trigeminal neuralgia treatment. Its advantages include the possibility of outpatient procedures under local anesthesia, making it suitable for elderly, debilitated, or geographically isolated patients who may not be ideal candidates for complex neurosurgery. Furthermore, some patients prefer this approach due to its potentially lower risk profile compared to more extensive surgeries. Key limitations of peripheral neurectomy include generally lower rates of complete and durable pain relief, paucity of high-quality evidence from large prospective studies, potential for procedural complications like persistent numbness or dysesthesias, and progressively diminishing efficacy with repeated neurectomies [14,15,37,41]. In contrast, central procedures like MVD and GKS that directly address the root causative mechanism of vascular compression demonstrate superior long-term outcomes and safety profiles [16,42,43]. For patients who fail to achieve adequate symptom control with medical management, early referral to neurosurgical evaluation may circumvent delays in definitive treatment and avoid risks associated with prolonged medication use or unsuccessful attempts at peripheral neurectomy. Careful patient selection, thorough counseling

regarding reasonable expectations, and judicious treatment sequencing are essential when considering peripheral neurectomy within the context of the full therapeutic armamentarium for trigeminal neuralgia [44].

Several studies [16,41,44] have reported retrospective case series of peripheral neurectomies with follow-up periods ranging from 0 to 9 years, demonstrating average pain relief lasting 24–32 months. Shah et al. [11] evaluated the role of peripheral neurectomies in the management of trigeminal neuralgia (TN) by analysing 50 patients and observed excellent pain relief for 2–5 years in 70% of cases. They concluded that peripheral neurectomy is particularly suitable for elderly patients. The present case reports are consistent with previous findings; at 18 months of follow-up, both patients remained symptom-free without recurrence of pain. Another study described an endoscopic microdissection technique of the infraorbital nerve in two patients with medically refractory V2 TN localized to the lateral midface and found it to be a safe and effective approach for symptomatic management while preserving sensation in unaffected areas of the dermatome [37]. Similarly, endoscope-assisted neurectomy and avulsion of the inferior alveolar nerve has been reported to provide effective pain relief in patients with TN involving the mandibular division, with the advantages of limited invasiveness and faster recovery [38].

A multicentric retrospective clinical trial for performing PN in cases of TGN of mandibular nerve was done. Patients who reporting at clinics in past 3 years, diagnosed as suffering from TGN of mandibular division, not responding to medicines, were considered for the surgery, Hashmukh's operation was performed for PN of inferior alveolar nerve (IAN), and results were obtained by recording post operative mouth opening, recurrence of symptoms and healing by visual analogue scale on subsequent follow up of at least 2 years. Hashmukh's Operation seems to be a novel approach, because of ease to perform and less time for complete healing and functioning. It was concluded that the Hashmukh's Operation was a better modality of PN for IAN in cases of TGN [39]. [Vanza B. IJAR, 2016; 6(7):].

A case series of six patients advocated that all six patients who were thereafter successfully treated by Peripheral Neurectomy of the involved nerve trunks, which resulted in a complete resolution of all symptoms with no recurrence even after a follow up period of three to five years. Histopathological and immunohistochemical examination of the extirpated nerve fibers revealed varying degrees of axonal degeneration and demyelination of the nerve sheaths at different locations, which could be a contributory or even an etiological factor of the condition, thus corroborating and confirming the relevance and efficacy of the mildly invasive surgical procedure of Peripheral Neurectomy in affording a permanent cure of the refractory neuralgic pain. In addition, an adjunctive measure had been proposed and elucidated, which was comprised of electrocauterization of the nerve canal and foramen following the nerve extirpation, which could prevented possible regeneration of remnants of the nerve fibers, in this way, further ensured a permanent resolution and cure, with nil recurrence [40]. A case report of a 40-year-old man, who suffered from chronic, debilitating trigeminal neuralgia. He underwent multiple tooth extractions and took carbamazepine, but neither provided improvement in his agonizing facial pain. He then had surgical neurectomies of the mental and inferior alveolar nerves, which provided only partial pain relief. The patient was not relieved, so he underwent a surgical neurectomy of the lingual nerve. The report advocated that, this third procedure successfully provided a significant reduction in his trigeminal neuralgia pain. Lingual neurectomy may be emerged as a potentially definitive treatment modality for trigeminal neuralgia refractory to medication or alternative neurectomy procedures. This minimally invasive surgical approach offered a valuable option for patients seeking sustained pain relief, particularly those who are not suitable candidates for or desire to avoid, more extensive or resource-intensive interventions [41].

A Retro prospective and prospective study was conducted on randomly selected 30 TN patients irrespective of age, gender and socioeconomic status. The branch of trigeminal nerve involved was identified according to the site of pain. Then the PN procedure was performed under local or general anesthesia. The follow up of each patient was done for next 18 months. Mean age of the TN patients 53.17 ± 13.84 years, with 66.7% of patients were within 60 years of age. Male to female ratio was

1:1.5. All patients showed unilateral TN. Mostly 26.7% trigger point was located in lower lip followed by 13.3% in upper lip. After 3-, 6- and 9-months follow-up, none of the TN patients treated with PN had pain and none had any effect on general activity. However, from 12 months till 18 months' follow up, 2 (6.7%) patients reported of pain. It was concluded that the PNs were viable treatment alternative for TN, although peripheral neurectomy has chances of reoccurrence but still offered better quality of life in patients for many years without relapses [42]. A case report highlighted the potential benefits of the Ginwala Approach for trigeminal neuralgia patients who have failed conventional treatments. The approach's minimally invasive nature and targeted intervention may be offered a promising alternative for managing this complex condition [43]. The preset case reports of 16 patients were also accordant with the similar studies, mentioned above.

Conclusion:

Misdiagnosis of trigeminal neuralgia (TN) remains common due to its atypical orofacial presentations, referral of pain to dental structures, low prevalence, and limited clinical experience among healthcare providers. A thorough clinical and radiographic assessment is essential to rule out odontogenic causes, as dental pathology can mimic or trigger TN. Irreversible dental interventions should be deferred until a definitive diagnosis is established. Further validation of treatment techniques requires large-scale, multicentre, long-term prospective studies.

There is no universally ideal surgical treatment for trigeminal neuralgia (TN); each case should be evaluated based on the individual patient's merits and limitations. Although peripheral neurectomy is one of the oldest techniques, it remains a highly suitable treatment option for patients who are refractory to medical therapy and for those who are not candidates for more invasive procedures under general anesthesia.

Dr. Ginwala's technique for peripheral neurectomy has demonstrated significant effectiveness and reliability, particularly in elderly and frail patients. This approach offers long-term pain relief, rapid recovery, and minimal postoperative complications, making it a safe and cost-effective solution.

The patient's 18-month pain-free period underscores the lasting success of Dr. Ginwala's technique, reinforcing its value as a viable treatment option for mandibular neuralgia. Furthermore, there was a marked improvement in preoperative pain severity, as well as in levels of anxiety and depression following the procedure.

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