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CASE REPORT

TRIGEMINAL NEURALGIA- CLINICAL PRESENTATION WITH REVIEW

***Dr. Abhishek Soni**

Department of Oral Medicine and Radiology, Modern Dental College and Research Center, Opp. Gandhi Nagar, Airport Road, Indore, Madhya Pradesh, India

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ABSTRACT

Trigeminal neuralgia or tic douloureux, because of its varied clinical presentation and distressing nature, can be especially challenging to the dental practitioner. It is characterized by a sharp shooting pain that is most often unilateral and ipsilateral to the trigger point. The pain episodes are short in duration and abruptly arise and subside. This case series is an attempt to highlight the various clinical presentations of trigeminal neuralgia along with the brief overview highlighting the various differential diagnosis of the condition.

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INTRODUCTION

Trigeminal Neuralgia (TN) is one of the most common neurological pains involving the orofacial region, which generally has the most intensive type of pain (Penarrocha *et al.*, 2009; Hegarty *et al*., 2011). It typically affects the elderly (1 in 25,000 of the population), with the most frequently reported cause being neurovascular compression (Jannetta PJ, 2007; Hamlyn *et al.,* 1992; Crooks *et al.*, 1996).It is characterized by a severe unilateral paroxysmal facial pain, often described by patients as sudden, severe, periodic, stabbing, lancinating, lightning-like, and shock-like. Attacks are most common in the second and third trigeminal divisions, and the right side of the face is more often involved than the left (Scrivani *et al.*, 2005). This case series of TN is an attempt to present different clinical presentations of the disease, which are successfully managed by the appropriate medical management. These cases also further highlights the various possible treatment options for managing TN resistant to standard treatment options.

CASE PRESENTATION

Case 1: A 67-year-old female reported with the complaint of severe pricking type of pain involving right side of the face since past two-year (Figure 1). Detailed history revealed that pain was severe, intermittent, shock-like and strictly unilateral in distribution; that radiated down the distribution of the second and third divisions of her right trigeminal nerve. It aggravates while eating, swallowing, washing her face, and even on touching; and relieved after 4-5 minutes. She denied of any pain between the episodes of lancinating pain, and also while sleeping. She also denied of any contralateral pain. Her past medical and dental history was unremarkable, and on examination she was neurologically intact. On extra oral examination, pain was triggered on touching the infraorbital area, zygomatic arch, nasolabial fold, upper and lower lip, and angle of mandible on the right side. On intraoral examination, completely edentulous maxillary arch and partially edentoulus mandibular arch were evident (Figure 2). Pain was elicited on palpation of maxillary and mandibular alveolar ridge on right side. Patient grimaces with pain and clutches her hand over the affected side of face while eliciting pain (Figure 3). Based on history and clinical examination, provisional diagnosis of trigeminal neuralgia involving the right maxillary and mandibular division was made. A panoramic radiograph revealed loss of all the teeth in maxilla with variable amount of bone loss; and multiple loss of teeth in mandibular arch (figure 4). Styloid process length was evaluated to rule out eagle's syndrome. A diagnostic block was given first for the mandibular division of the trigeminal nerve followed by the maxillary division and patient was immediately free of pain. Thus a definitive diagnosis of trigeminal neuralgia involving the maxillary and mandibular division on the right side was established. Vitals were normal at the time of examination. Complete heamogram was done before initiating the treatment. All the values come under the normal limits. Initially, the symptoms had been well controlled with carbamazepine 600 mg BID [Tab. Tegretal] and Pregabalin with Methylcobalmin 75mg HS [Tab. Mahagaba-M OD] given for ten days. To control her anxiety-related symptoms because of chronicity of

***Corresponding author:* Dr. Abhishek Soni,**
Department of Oral Medicine and Radiology, Modern Dental College and Research Center, Opp. Gandhi Nagar, Airport Road, Indore, Madhya Pradesh, India.

pain, a combination of Flupenthixol (0.5mg) with Melitracen (10mg) [Tab. Mankind's Placida], once in the morning, after breakfast was prescribed. After 10 days, the dose of carbamazepine has been decreased to 200 mg BID when reduction in the symptoms was evident. The patient responded well with the medications, and reported complete resolution of her symptoms at one-month follow-up (Figure 5).

Case 2: A 45-year-old female reported with the complaint of pain involving the left side of face for the past one year (Figure 6). History revealed that the pain was unilateral, sudden in onset, stabbing, lancinating type that aggravated on taking food, hot and cold beverages, and also while washing her face.

Figure 1. Case 1: Pre-treatment extraoral photograph complaining of pain involving right side of the face

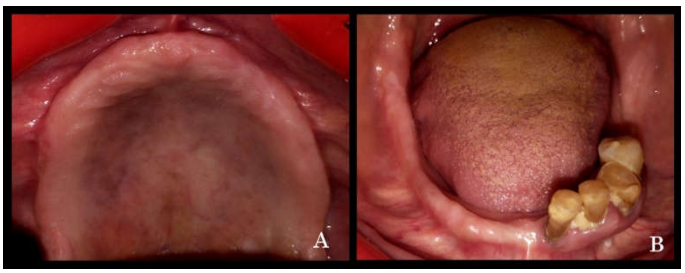

Figure 2. Case 1: Intraoral photograph of patient showing (A) completely edentulous maxillary arch; and (B) partially edentulous mandibular arch

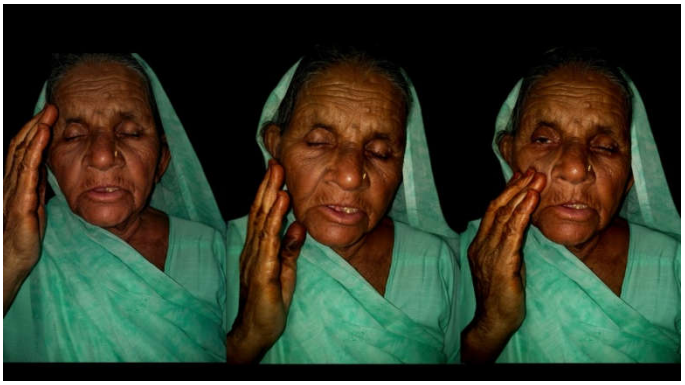

Figure 3. Case 1: Photograph of a patient taken during a right-sided painful attack

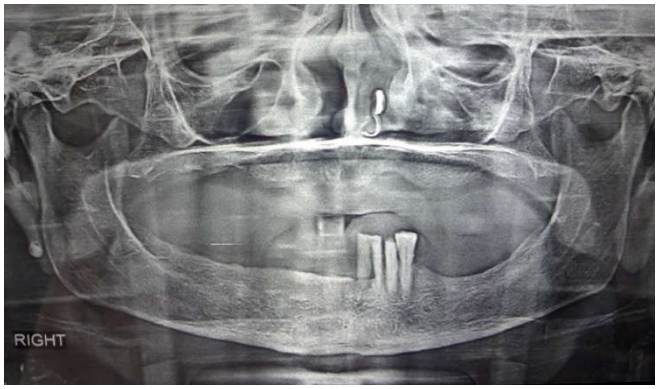

Figure 4- Case1: A panoramic radiograph showing loss of all teeth in maxilla and multiple loss of teeth in mandible with variable amount of bone loss

Figure 5. Case 1: Post-treatment extraoral photograph showing complete resolution of pain.

Figure 6. Case 2: Pre-treatment extraoral photograph complaining of pain involving left side of the face.

Table 1. Differential diagnosis of Trigeminal Neuralgia

	TN*	CH**	PH***	SUNCT****	AFP*****	PHN******
Age	Typically occur in middle aged women; around 50 years	Peak age of onset for CH is between 20 and 29 years	Around 30 years	Around 40-70 years	Around 40 years	It is more common as people gets older (risk of development directly related to patient's age)
Sex	Female preponderance (9:1)	Male preponderance (2.5:1 and 3.5:1)	Female preponderance (2:1)	High male predilection (2:1)	Predominantly women	Males-females equally affected
Pain						
Onset	Sudden in onset	Rapid in onset	Rapid in onset	Rapid in onset	Rapid in onset	Rapid in onset
Location	Unilateral •Only 3% to 10% of cases are bilateral Along distribution of branches of the trigeminal Usually will not follow anatomic pathways; nerve; Maxillary > Mandibular > Ophthalmic	CH is characteristically unilateral. •Side changes of presentation occurring in 97.2% of patients •78.5% of CH individuals reported always strictly unilateral pain •18.7% reported pain changing sides •2.8% reported bilateral pain Pain is localized deep in and around the orbit and temporal region and may radiate to the supraorbital region, maxilla, nostril, upper gingiva, and palate. In some cases the pain may spread and involve the entire ipsilateral head and neck	PH are characteristically unilateral without side shift Pain is localized and most intense in and around the orbit, temporal, maxilla, and frontal regions and may also involve the neck and occiput	Characteristically unilateral—without side shift in most patients (88%) Pain is localized and most intense in the periorbital, temporal, and frontal region and occasionally involves the neck, occiput, side and top of the head, ear, nose, cheek, palate, and throat 33% and 21% of SUNCT patients report pain localized to the maxillary branch of the trigeminal nerve and teeth, respectively	Usually will not follow anatomic pathways; unilateral, less likely bilateral	Forehead, eye, cheek (rarely)
Quality	Sharp, stabbing, electric shock-like, lancinating, flashing, burning	Constant, boring, tearing, screwing, burning, piercing, sharp, and "hot poker in the eye" or "eye is being pushed out." (pulsating and pressure-like)	Aching or throbbing initially and elevating to boring or stabbing at peak intensity	Typically neuralgic-like; stabbing, sharp, burning, pricking, piercing, shooting, lancinating, or electric shock–like. (pulsatile, throbbing, steady, spasmodic, and staccatolike)	Diffuse; burning, aching, dull	Constant, burning, throbbing or aching pain; extreme sensitivity to touch and temperature change
Intensity	Severe	Extreme and excruciating	Extreme and excruciating	Extreme and excruciating	Moderate to severe, may fluctuate	Extreme and excruciating
Duration	Persists for brief period (1-2 min)	Longer lasting attacks (15-180min)	Shorter lasting (2 to 30 minutes) and more frequent	5 and 250 seconds	Constant	Continues
Associated sign and symptoms	No autonomic signs	Almost all CH attacks exhibit ipsilateral cranial autonomic features such as miosis, ptosis, nasal congestion, rhinorrhea, and facial sweating, lacrimation	Almost all PH attacks exhibit ipsilateral cranial and facial autonomic features with lacrimation, conjunctival injection, nasal congestion, and rhinorrhea being the most frequently observed	Almost all SUNCT attacks exhibit ipsilateral cranial and facial autonomic features, namely conjunctival injection and tearing	Symptoms: allodynia, dysesthesia, paresthesia	Itching, numbness, headache, fatigue and sleeping difficulties
Nocturnal occurrence	Rarely nocturnal	CH is particularly noteworthy for its nocturnal appearance (approximately 50% of CH attacks take place at night)	No preponderance for nocturnal attacks	Attacks predominately occur during the day, typically with bimodal distribution in the morning and afternoon or evening. Only 1.2% of SUNCT attacks is nocturnal and usually occurs during severe periods	None	None
Treatment	Responsive to CBZ	Responsiveness to HBO and Sumatriptans	Responsiveness to oral indomethacin, 25 mg 3 times a day, may be used diagnostically	Resistance to classical anti-neuralgic therapy	Tricyclic antidepressants	Antidepressants- amitryptaline, desipramine Anticonvulsants- gabapantine, pregabaline Steroids- for PHN pt. with chronic pain (persistent long term pain)

*TN- Trigeminal Neuralgia; **CH- Cluster Headache; ***PH- Paroxysmal Hemicrania; ****SUNCT- Short-lasting, unilateral, neuralgiform headache attacks with, conjunctival injection and tearing; *****AFP- Atypical facial pain; ******PHN- Post-herpetic neuralgia

Pain was referred to left cheek, eye, and ipsilateral head and neck region; and lasts from few seconds to less than 2 min. There were two to three episodes of pain in a day from the last 6 months. There was no history of sleep disturbances. Her past medical and dental history was non-contributory, and on examination she was neurologically intact. On extra oral examination, touching the infraorbital area, zygomatic arch, and nasolabial fold on the left side produced a paroxysm of pain. On intraoral examination, partial edentulous maxillary and mandibular arches were evident along with the presence of multiple root pieces (Figure 7). Patient jerked from the dental chair while eliciting pain. A panoramic radiograph revealed no significant abnormality (Figure 8).

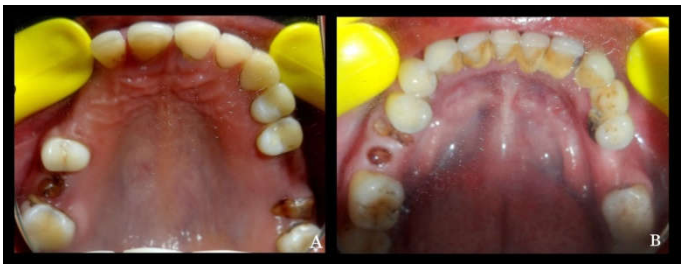

Figure 7. Case 2: Intraoral photograph of patient showing (A) maxilla; and (B) mandible revealed multiple root stumps and few missing teeth

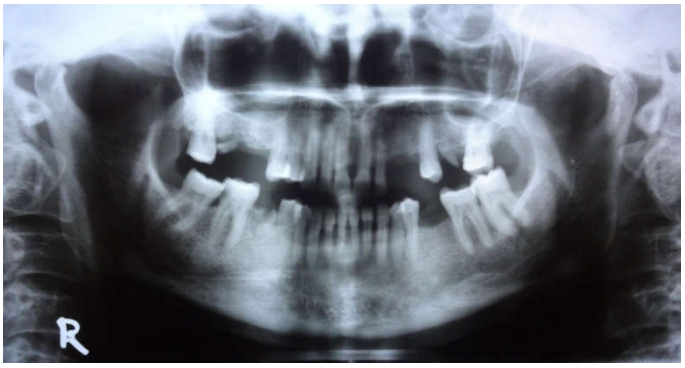

Figure 8. Case 2: A panoramic radiograph showing multiple root stumps and few missing teeth

Figure 9. Case 2: Post-treatment extraoral photograph showing complete resolution of pain

Thus, taking into account the clinical history as well as all the investigations and clinical examination findings, a definitive diagnosis of trigeminal neuralgia involving the left maxillary division of trigeminal nerve was established. Routine blood investigations were found to be normal. Root stumps were removed prophylactically from the affected area. Initially, carbamazepine [200 mg BID (Tab. Tegretal)] therapy was initiated for two weeks with good results; but pain recurred upon discontinuation of the therapy. Then, carbamazepine was restarted with gradual increase in the dose [600 mg BID (Tab. Tegretal)] along with Pregabalin with Methylcobalmin 75mg HS [Tab. Mahagaba-M OD] for ten days. However, debilitating pain persisted despite continuous treatment. Baclofen [Tab. Liofen 10 mg TDS] was added to the patient's regimen and increased to 60 mg daily with subsequent reductions in the carbamazepine dosages. To control her anxiety-related symptoms because of chronicity of pain, a combination of Flupenthixol (0.5mg) with Melitracen (10mg) [Tab. Mankind's Placida], once in the morning, after breakfast was prescribed. This resulted in effective pain relief and reduced the frequency of her facial pain. The patient responded well with the medications, and reported complete resolution of her symptoms at three-month follow-up (figure 9). Pregabalin and Baclofen were safely withdrawn gradually. At one-year follow-up, no recurrence was noticed.

DISCUSSION

TN is a clinical diagnosis. International Classification of Headache Disorders II (ICHD-II) establishes TN as a discrete clinical diagnosis under the general classification of ''Cranial neuralgias and central causes of facial pain'' (ICHD-II diagnostic code 13) (Scrivani *et al.*, 2005). The key feature is a sudden and severe lancinating pain, which usually lasts from a few seconds to two minutes, within the trigeminal nerve distribution, typically the maxillary or mandibular branches. The pain is often evoked by trivial stimulation of appropriately named "trigger zones" Bennetto *et al.*, 2007). ICHD-II further subdivides TN into classic TN and symptomatic TN. Classic TN, also known as primary TN, Idiopathic TN, or essential TN (13.1.1) is the most common idiopathic form of the disorder, which includes cases associated with vascular compression. The International Headache Society (ihs- classification.org) defines classical TN as- (A) Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B and C; (B) Pain has at least one of the following characteristics- intense, sharp, superficial or stabbing; and precipitated from trigger areas or by trigger factors; (C) Attacks are stereotyped in the individual patient; (D) There is no clinically evident neurological deficit; and (E) Not attributed to another disorder. Symptomatic TN (13.1.2) has the same key features of TN but results from another disease process (such as multiple sclerosis or a cerebellopontine angle tumor). Symptomatic TN is defined by IHS as ''pain indistinguishable from 13.1.1 classic TN but caused by a demonstrable structural lesion other than vascular compression (Scrivani *et al.*, 2005). The preferred theory of causation involves compression of the trigeminal root adjacent to the pons. When the pain is related to the maxillary or mandibular divisions of the trigeminal nerve, compression of the rostral and anterior portions of the nerve by the superior cerebellar artery is the most common finding. If the pain involves the ophthalmic division of the trigeminal nerve, then compression of the caudal and posterior portions by the anterior inferior cerebellar artery is common (Law *et al.*, 1995).

Table 2. Cases of TN reported in literature managed by pharmacotherapy

Author	Year	Age/Sex	Division of trigeminal nerve involved	Treatment	Recurrence on follow-up
Sadat et al.	2014	55/M	Left Mandibular division	Carbamazepine 600mg daily in three divide doses; along with an amitryptylene 10mg once daily	No
Ekici et al.	2013	10/F	Right Maxillary division	Carbamazepine 10 mg/kg/day which further increased to 15 mg/kg/day	No
Ando et al.	2012	39/M	Right Maxillary division	Carbamazepine 300 mg/day and additional Goreisan 7.5g/day	No
Moran et al.	2009	51/F	RightOpthalmic, Maxillary and Mandibular division	Oral sumatriptan 50 mg, along with a maintenance dose of propranolol 160 mg daily	No
Carciumaru et al.	2003	67/M	Left Opthalmic and Maxillary division	Carbamazepine 300 mg/day and gabapentin 900 mg/day	No
		31/F	Left Maxillary and Mandibular division	Gabapentin 900 mg/day	No

*TN- Trigeminal Neuralgia

Devor *et al.* (2002) proposed an ‘‘ignition hypothesis’’ to explain the principal signs and symptoms in TN. In this model, a trigeminal injury induces physiologic changes that result in a population of hyperexcitable and functionally linked primary sensory neurons. The discharge of any individual neuron in this group can quickly spread to activate the entire population. Such a sudden synchronous discharge could underlie the sudden jolt of pain characteristic of a TN pain attack. This model is attractive, not only because it explains many of the key features of TN, but also because it encourages specific testable hypotheses that should stimulate advances in both basic science and clinical investigation. It mainly affects middle aged women with a female predilection of 9:1. Pain is confined to one of the three division of trigeminal nerve, with maxillary division is predominantly involved; followed by mandibular and ophthalmic division. Only 3% to 10% of cases are bilateral. The formulation of accurate diagnosis begins with a very careful history taking. The major diagnostic clinical criteria summarized by International Headache Society (IHS) suggest at least four of the following parameters must be present to make the diagnosis (Filadora *et al.*, 1997; Debta *et al.*, 2010).

1. Character: Shooting, electric shock-like, sharp, and superficial.
2. Severity: Moderate to severe.
3. Duration: Each episode of pain lasts no more than 2 minutes, numerous episodes during the day.
4. Periodicity: Periods of weeks or months when there is no pain, also pain free periods between the attacks.
5. Site: Distribution of trigeminal nerve area. Mostly unilateral.
6. Radiation: Within trigeminal nerve area or beyond.
7. Provocating feature: Light touch such as eating, talking, washing.
8. Relieving factor: Often sleep, anticonvulsant drugs.
9. Associated features: Trigger zone, weight loss, poor quality of life, and depression.

To make a definitive diagnosis, it is often necessary to establish a list of possible differential diagnoses and then to systematically exclude each by a process of elimination through diagnostic tests and investigations. The list of differential diagnoses is long and most of these are easily ruled out after the thorough history taking and proper clinical examination (Table 1) (Balasubramaniam *et al.*, 2007; Balasubramaniam *et al.*, 2007; Benoliel *et al.*, 1998; Turp and Gobetti, 1996; Nurmikko *et al.*, 2001).TN is a well-recognized manifestation in patients with multiple sclerosis. Some 2% to 7% of patients with TN also have multiple sclerosis. Patient with multiple sclerosis associated with TN characterized by an earlier age of onset (50 years as opposed to 63 years of age in classical TN), bilateral symptoms (31% as opposed to 3% to 10% of cases of classical TN), and condition is frequently refractory to carbamazepine. Optic neuritis is commonly a precursor of multiple sclerosis, but a diagnosis of multiple sclerosis requires other clinical signs or abnormal investigations. Symptoms those are indicative of multiple sclerosis such as retrobulbar neuritis, diplopia, ataxia, and sensory disturbance or motor weakness (Gale *et al.*, 1995). Various case reports have been published in the literature showing the effectiveness of pharmacotherapy in managing trigeminal neuralgia (Table 2). Medical management for TN includes anticonvulsant (antiepileptic) drugs. Carbamazepine is the first drug of choice for TN. Alternative medications includes Oxcarbazepine, Baclofen, Gabapentin, Lamotrigine, and Pimozide. AED therapy routinely begins with a single agent, given in gradually increasing doses until pain attacks are either suppressed or satisfactorily reduced. When a patient only partially responds to single drug therapy at dosages that evoke side effects, adding a second AED may enhance the therapeutic response. Because AEDs have differing mechanisms of action as well as differing side effect patterns, combining agents is a reasonable approach (Scrivani *et al.*, 2005). For the management of acute exacerbation of TN pain attacks, standard local anesthetic block to the facial region containing the trigger zone often results in an immediate reduction in TN attacks. Han *et al.* (2008) described that trigeminal nerve block with high-concentration lidocaine (10%) was capable of achieving an intermediate period of pain relief, particularly in patients with lower pain intensity and shorter pain duration prior to the procedure. After anesthesia in the correct region has been obtained and pain relief monitored with local anesthetic, 0.5 to 1.0 mL 100% absolute alcohol is injected. The supraorbital nerve, infra-orbital nerve, mental nerve, maxillary nerve, mandibular nerve, and others are reported to be blocked with alcohol. In patients with a severe TN exacerbation, it is reasonable to consider initial treatment with a parenteral ‘‘loading’’ dose of an AED (Scrivani *et al.*, 2005; Toda, 2008).

Surgical therapy may be indicated when medication cannot control pain, when patients cannot tolerate the adverse effects of the medication, and in medically complex patients with polypharmacy for other conditions (Toda, 2008). According to Cohen J (2005), in general, surgical procedures for TN should be considered when the patient does not attain pain relief after adequate trials of 2 or 3 medications, or when pain relief is attained but the patient requires medication dosing at levels that result in significant drug toxicity. Various surgical options for the treatment of TN include microvascular decompression, partial sensory rhizotomy, percutaneous radiofrequency thermocoagulation of the trigeminal ganglion, percutaneous glycerol gangliolysis of the trigeminal ganglion, percutaneous balloon microcompression of the trigeminal ganglion, gamma

knife radiosurgery, cyberknife radiosurgery, cryotherapy, peripheral neurectomy (Cohen, 2005)

Conclusion

TN has long been recognized by healthcare professionals, but it is still an enigmatic condition and its management remains controversial. An early and accurate diagnosis of TN is important, because therapeutic interventions can reduce or eliminate pain attacks in the large majority of TN patients.

Key points

- Trigeminal neuralgia may sometime be associated with varied clinical presentation, which may pose diagnostic challenge to the dental practitioners. To accurately diagnose trigeminal neuralgia, we must perform a thorough clinical examination so as to rule out various differential diagnosis of the condition.
- Chronic pain condition may often be associated with anxiety-related symptoms, which can be best controlled by adding anxiolytic agents, preferably non-sedating, to the standard treatment regimen to manage trigeminal neuralgia. In all our cases, we prescribed a combination of Flupenthixol (0.5mg) with Melitracen (10mg) [Tab. Mankind's Placida], once in the morning, after breakfast, which shown a significant results in controlling such symptoms.

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Conflict of interest: None declared

Consent: A written informed consent was obtained from the patient for publication of this case report.

REFERENCES

Balasubramaniam R., Klasser GD. 2007.Trigeminal autonomic cephalalgias. Part 1: cluster headache. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.*, 104:345-58.

Balasubramaniam R., Klasser GD. 2007. Trigeminal autonomic cephalalgias. Part 2:Paroxysmal Hemicrania. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*., 104:640-6.

Bennetto L., Patel NK., Fuller G. 2007. Trigeminal neuralgia and its management. *BMJ*.,334:201-5.

Benoliel R., Sharav Y. 1998. SUNCT syndrome. Case report and literature review. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.*, 85:158-61.

Cohen J. 2005. Role of the neurologist in the evaluation and treatment of patients with trigeminal neuralgia. *Neurosurg Focus*., 18:E2.

Crooks DA., Miles JB. 1996. .Trigeminal neuralgia due to vascular compression in multiple sclerosis-Post-mortem findings. *British J Neurosurg*., 10:85-8.

Devor M., Amir R., Rappaport ZH. 2002. Pathophysiology of trigeminal neuralgia: the ignition hypothesis. *Clin J Pain.,* 18:4-13.

Debta FM., Ghom AG., Shah JS., Debta P. 2010. A Comparative Study between Oxcarbazepine and Gabapentin Regarding Therapeutic Efficiency and Tolerability in the Treatment of Trigeminal Neuralgia. *J Indian Acad Oral Med Radiol.*, 22:10-17.

Filadora V., Moner M., Lena M. 1997. Gabapentin for idiopathic TN: Report of two cases. *Neurology.*, 48:1467-71.

Gale D., Prime S., Campbell MJ. 1995. Trigeminal neuralgia and multiple sclerosis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.*, 79:398-40.

Hamlyn PJ., King TT. 1992. Neurovascular compression in trigeminal neuralgia: A clinical and anatomical study. *J Neurosurg.*, 76:948-54.

Han KR., Kim C., Chae YJ., Kim DW. 2008. Efficacy and safety of high concentration lidocaine for trigeminal nerve block in patients with trigeminal neuralgia. *Int J Clin Pract.*, 62:248-54.

Hegarty AM., Zakrzewska JM. 2011. Differential diagnosis for orofacial pain, including sinusitis, TMD, trigeminal neuralgia. *Dent Update*., 38:396-400.

Jannetta PJ. 2007. Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. J *Neurosurg*., 107:216-9.

Law AS., Lilly JP. 1995. Trigeminal neuralgia mimicking odontogenic pain. A report of two cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.,* 80:96-100.

Nurmikko TJ., Eldridge PR.2001. Trigeminal neuralgia-pathophysiology, diagnosis and current treatment. *Br J Anaesth.*, 87:117-32.

Penarrocha M., Mora E., Bagan JV., Garcia B., Penarrocha M. 2009. Idiopathic Trigeminal Neuropathies: A Presentation of 15 Cases. *J Oral Maxillofac Surg.*, 67:2364-8.

Scrivani SJ., Mathews ES., Maciewicz RJ., Boston., Mass. 2005. Trigeminal neuralgia. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.*, 100:527-38.

Toda K. 2008. Operative treatment of trigeminal neuralgia: review of current techniques. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.,* 106:788-805.

Turp and Gobetti. 1996. Trigeminal neuralgia versus atypical facial pain. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*., 81;424-32.

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