Journal of Advanced Clinical & Research Insights (2017), 4, 72–75

CASE REPORT

Physical and psychological distress - trigeminal neuralgia: A case report

M. K. Navya, G. P. Sujatha, L. Ashok

Department of Oral Medicine and Radiology, Bapuji Dental College and Hospital, Davangere, Karnataka, India

Keywords
Carbamazepine, nerve demyelination, trigeminal neuralgia

Correspondence
M. K. Navya, Department of Oral Medicine and Radiology, Bapuji Dental College and Hospital, Davangere - 577 004, Karnataka, India. E-mail: navu_mk@yahoo.in

Received 02 March 2017;
Accepted 25 April 2017

doi: 10.15713/ins.jcri.161

Abstract

A 45-year-old female patient had reported with a complaint of pain for 8 months. On thorough history taking and clinical examination, we were able to confirm the final diagnosis as trigeminal neuralgia (TN). This article has a case report and review of literature on history, pathophysiology, clinical features, and therapeutics of TN.

Introduction

Trigeminal neuralgia (TN) also called as tic douloureux, meaning "painful tic" in French, was first explained by Arateus in the first century AD.[1] Later, Nicolaus Andre described the TN as a distinct clinical disease in 1756.[2] TN is the most common orofacial pain under neuralgia and is among the most painful conditions in orofacial pains.[3,4] TN causes physical distress to the patient, moreover, it causes psychological distress to the patient which compromises their quality of life. Causes for TN may be neoplasia, diseases of the connective tissue, infections, and demyelination of nerve or can be of idiopathic nature.[5] The occurrence of TN in men is 2.5 and women is 5.7/100,000/year which indicates females are more frequently affected than males.[2]

Case Report

A 45-year-old female patient reported to the Department of Oral Medicine and Radiology, Bapuji Dental College and Hospital, Davangere, with a chief complaint of pain on the upper right back and lower front teeth region for 8 months. Pain was sudden in onset, electric shock-like in nature. Starts when she has food or when she talks or washes her face. Pain was severe in intensity and was intermittent in nature. The patient gave a visual analog scale (VAS) of 8. The patient also gave a history of similar pain for 8 months and had visited a private dentist in Davangere and was referred to neurophysician who diagnosed it as TN and gave zeptol 200 mg twice daily and the patient was asked to continue. There was relief in pain after 1 month, after which the patient stopped taking the drug. After 2 months, pain again aggravated. medical history was noncontributory and dental history revealed that she had undergone uneventful extraction of the upper right back tooth 1 year back because of decay in a private dental clinic. Trigger zones were seen on the right ala of the nose, right upper lip, and lower chin region. Intraoral examination revealed, generalized mild gingival inflammation and shallow pockets. Except 16 and 17, complete compliments of teeth were present [Figure 1]. Root stump with respect to 37 was present; stainless steel crown with respect to 46, 47, and 48 and generalized attrition was evident. Based on the history and clinical findings, a provisional diagnosis of TN involving the V1 and V2 division of nerve on the right side was given. Differential diagnosis of crack tooth syndrome and idiopathic facial pain was considered. Radiographic examination was done by taking orthopantomogram which revealed missing 16 and 17, radiopaque crown with respect to 46, 47, and 48, and root stump with respect to 37. Furthermore, radiographic artifact was evident in the left ramus of the mandible [Figure 2]. In chairside investigation, infraorbital and mental nerve block was given and pain was relieved and the final diagnosis of TN was confirmed.

Figure 1: (a and b) Profile, and (c) hard tissue of the patient showing missing 16 and 17

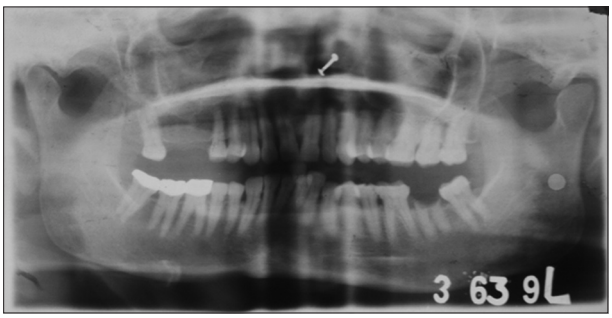

Figure 2: Orthopantomogram showing missing 16 and 17 and root stump with respect to 37. Radiographic artifact evident in the ramus of the mandible

The patient was then advised to take carbamazepine 200 mg t.i.d for 15 days. After 15 days of follow-up, VAS was 3 and the patient was told to continue the same drug regimen. The patient was evaluated every 1 month, and blood investigations were carried out to exclude drug-related side effects.

Discussion

TN has characteristic feature of lancinating, unilateral, paroxysmal pain which occurs in the distribution of the fifth cranial nerve. Patients usually describe the pain as sharp, stabbing, shooting, burning, or electric shock-like. The attacks are initiated by non-painful physical stimulation to specific "trigger points" which will be located same side to the pain. Stimuli may include day-to-day hygiene activities, mastication, and other functional movements. Noxious stimuli such as pinching over the area or pressure application generally does not trigger attacks. The main feature of TN is that the attacks usually last <2 min or sometimes a period of several hours. In our case also, the patient presented with unilateral pain and trigger points on the right side of the face. Clinical features of TN are shown in Table 1.[1]

At times, there will be persistent ache between paroxysms or mild sensory loss. Such disease is labeled as "atypical"[6] or "mixed" TN.[7] Patients with atypical disease are more likely to have symptomatic rather than idiopathic disease, and they are often more refractory to treatment than those with classic TN.[8] Atypical TN is usually confused with atypical facial pain. There are various diagnostic criteria for TN [Table 2].[9]

The examination of cranial nerves will be within normal limits because nerve damage rarely accompanies TN. In general, the patient history, a negative result in TN nerve examination, and pain relief to a course of carbamazepine are the keys to diagnosis of TN. Radiographic modalities should be taken into consideration if the diagnosis is uncertain.[10] VAS scale when noted on these patients will show a rate of 10, distributed unilaterally over the face, even in our patient, VAS was high around 8. Therefore, when there is no history of orofacial abnormalities and trauma to the head and neck region, then we should immediately suspect of TN. This patient gave a history of extraction of upper molars on the right side which can be considered as the cause.

Radiographic examinations in the form of routine conventional or advanced imaging techniques help to exclude lesions arising in the maxillary sinus and Meckel's cavum or arising from the pontocerebellar angle.[6] The apt advanced imaging modality to rule out any tumor compressing on the trigeminal ganglion or its pathway is head computed tomography (CT). Demyelination of nerve or soft tissue tumor can be detected using magnetic resonance imaging (MRI). Hutchins *et al.* suggested radiographic evaluation in patients with TN using CT and MRI to analyze the proximal and distal zone and the trajectories of the V cranial nerve.[7,11] Vascular compression is being demonstrated as one of the causes radiologically in patients with TN. Studies were conducted where radiologists were blinded to the affected side, and the results in terms of predicting subsequent surgical findings and response to surgery were excellent.[12]

The treatment modalities of TN can be divided into pharmacological and surgical intervention. In general, patients show a good response to drug therapy with over 80% using anticonvulsants. Carbamazepine (Tegretol, Summit) is generally used as the first drug of choice in the management of TN. Its mechanism of action is neuronal excitability depression, blocks Na + channels, thereby leading to a selective reduction in the ectopic neural discharges that are responsible for the pain.[11] This drug is used for therapeutic diagnosis of TN. Usually, we start with low dose and gradually increase the dosage. Maximum dosage of carbamazepine that can be given is 1200-2400 mg. Periodic follow-up and blood tests should be conducted to rule out neutropenia as it is an adverse effect of long-term use of carbamazepine.

Second-line therapeutics includes drugs such as phenytoin and baclofen, which has carbamazepine-like mechanism of action.[11] Other drugs such as pimozide, sodium valproate, and clonazepam are also used. Clonazepam 0.5 mg t.i.d is preferred dosage. However, the drawback is that these drugs have shown unclear efficacy. These drugs are associated with major side effects such as nausea, weakness, gingival enlargement, ataxia, and erythematous

Table 1: Trigeminal neuralgia: Clinical features

Average age of the patient with first symptoms	50-70 years
Sex distribution of patients/100,000/year	Women: 5.7 Men: 2.5
Potential causes	Injury or compression (vascular or from the posterior fossa tumor) of trigeminal root leading to demyelination; central nervous disease
Pain location	Unilateral along distribution of facial area innervated by trigeminal nerve
Pain duration	Brief attacks of seconds or minutes that may occur sequentially for minutes or hours
Pain quality	Sharp, stabbing, shooting, burning, cutting, shock-like, lancinating, paroxysmal
Affected trigeminal nerve branches	Maxillary (V2): 35% Mandibular (VS): 29% Ophthalmic (V1): 4% Maxillary and mandibular: 19% All branches: 1%
Trigger	Non-noxious light stimuli; trigger points or zones located ipsilaterally to pain
Chronic facial pain syndromes to exclude	Atypical odontalgia, "phantom" exclude tooth pain after exodontia, postherpetic TN, Temporomandibular joint disorders, glossopharyngeal neuralgia, short-lasting, unilateral neuralgiform headache attacks with conjunctival injection, and tearing syndrome

TN: Trigeminal neuralgia

Table 2: Diagnostic criteria for classic trigeminal neuralgia

Paroxysmal attacks of pain lasting from a fraction of a second to 2 min that affects one or more divisions of the trigeminal nerve
Pain has at least one of the following characteristics: Intense, sharp, superficial, or stabbing precipitated from trigger areas or by trigger factors
Attacks are similar in individual patients
No neurological deficit is clinically evident
Not attributed to another disorder

skin rashes.[13] For approximately 30% of patients with TN, pharmacological intervention fails due to unsuccessful pain control or because of the adverse reactions of the drugs, and 50% eventually become unresponsive due to the tolerability of medication. For these patients, surgical procedures are carried out.[11]

Cryotherapy, alcohol or anesthetic injection, or neurectomy will help in attaining peripheral block of trigeminal branches. Cryotherapy when performed leads to necrosis of the affected nerve branch. This method initially gives good results but has higher recurrence rate.[11,14] Peripheral blocks of the trigeminal ganglion are attained using alcohol injections. However, it can lead to adverse reactions such as dysesthesia and anesthesia dolorosa which are unpredictable, and the patient attains relief for short duration only. According to Türp and Gobetti, alcohol injections and neurectomy are less affective in the management of TN. Microvascular decompression of the trigeminal root and retrogasserian rhizotomy are interventional surgical procedures used in the treatment of TN. Microvascular decompression provides permanent pain relief with minor recurrences sometimes. It is not commonly used because of the expense and risks associated with this procedure.

Conclusion

TN is the most common neuralgia causing orofacial pain limited to the distribution of the trigeminal nerve branches. It has characteristic unilateral paroxysmal pain that is sudden in onset and last for few minutes to hours. The pain associated is described as sharp, stabbing, lancinating, and burning. The triggering factors are stimuli such as a light touch of the skin, daily hygiene habits, and mastication. The trigger points are usually found in the ipsilateral area supplied by the nerve. During the past several decades, major advances have occurred in the management of TN as the main aim is to reduce the physical and psychological distress, the patient is experiencing. Many pharmacological agents such as anticonvulsants have been highly effective for suppression of TN pain attacks; certain newer medications such as botulinum toxin have shown better results along with its combination of drugs have shown pain relief mainly a combination of carbamazepine and baclofen. Clearly, these patients need psychological approach also because the constant fear of pain makes their life dreadful. Therefore, along with the pharmacological and surgical intervention, psychology of the patient should also be considered. And also to better understand and manage TN, substantial studies or randomized controlled trials should be carried out.

References

1. Jackson EM, Bussard GM, Hoard MA, Edlich RF. Trigeminal neuralgia: A diagnostic challenge. Am J Emerg Med 1999;17:597-600.
2. Katusic S, Beard CM, Bergstralh E, Kurland LT. Incidence and clinical features of trigeminal neuralgia, Rochester, Minnesota, 1945-1984. Ann Neurol 1990;27:89-95.
3. Türp JC, Gobetti JP. Trigeminal neuralgia versus atypical facial

pain. A review of the literature and case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:424-32.
4. Fromm GO, Sessle BJ. Trigeminal Neuralgia: Current Concepts Regarding Pathogenesis and Treatment. Boston: Butterworth-Heinemann; 1991. p. 205-19.
5. Penarrocha M, Penarrocha MA, Soler F, Orozco M. Typical trigeminal neuralgia associated with posterior cerebellopontine angle. Report of two cases. Med Oral 1997;2:109-12.
6. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. 2nd edition. Cephalalgia 2004;24:1-150.
7. Zakrzewska JM, Jassim S, Bulman JS. A prospective, longitudinal study on patients with trigeminal neuralgia who underwent radiofrequency thermocoagulation of the Gasserian ganglion. Pain 1999;79:51-8.
8. Lopez BC, Hamlyn PJ, Zakrzewska JM. Systematic review of ablative neurosurgical techniques for the treatment of trigeminal neuralgia. Neurosurgery 2004;54:973-82.
9. Nurmikko TJ, Eldridge PR. Trigeminal neuralgia - Pathophysiology, diagnosis and current treatment. Br J Anaesth 2001;87:117-32.
10. Rappaport ZH, Devor M. Trigeminal neuralgia: The role of self-sustaining discharge in the trigeminal ganglion. Pain 1994;56:127-38.
11. Hutchins LG, Harnsberger HR, Hardin CW, Dillon WP, Smoker WR, Osborn AG. The radiologic assessment of trigeminal neuropathy. AJR Am J Roentgenol 1989;153:1275-82.
12. Patel NK, Aquilina K, Clarke Y, Renowden SA, Coakham HB. How accurate is magnetic resonance angiography in predicting neurovascular compression in patients with trigeminal neuralgia? A prospective, single-blinded comparative study. Br J Neurosurg 2003;17:60-4.
13. Ziccardi VB, Braun TW, Ochs MW. Trigeminal neuralgia: Review of etiologies and treatments. Compendium 1993;14:1256, 1258-62.
14. De Coster D, Bossuyt M, Fossion E. The value of cryotherapy in the management of trigeminal neuralgia. Acta Stomatol Belg 1993;90:87-93.

How to cite this article: Navya MK, Sujatha GP, Ashok L. Physical and psychological distress - trigeminal neuralgia: A case report. J Adv Clin Res Insights 2017;4:72-75.