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Post-Traumatic Trigeminal Neuropathic Pain: - Case Series

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Abstract

Trigeminal Nerve- the largest cranial nerve has both sensory and motor functions which supplies the face, hence any injury either iatrogenic or accidental could result in deficit functioning of the nerve leading to a debilitating disease with various clinical symptoms which results in social, psychological burden for the patient, hampering health related quality of life. PTTN can result due to multiple etiologies affecting Inferior alveolar nerve, Lingual nerve and branches of maxillary nerve and is often misdiagnosed by the clinicians. Therefore, through this article we aim to present certain clinical features and diagnostic approach with managing strategies to help patients cope up better by presenting a case series of 5 patients who reported to the division of Oral Medicine and Dental Radiology at ITS Dental college, Muradnagar. The diagnosis of PTTN was made according to international classification of headache disorders ICHD-3rd edition, thorough clinical examination and relevant history of the patient. Treatment through multimodal analgesic protocols provides a favorable outcome however achieving complete resolution of the disease is a little difficult henceforth we also suggest a need for psychological support to be integrated into the management protocols to improve efficacy of the treatment for this chronic disorder.

Keywords: Trigeminal Neuropathy, Pttn, Orofacial Pain, Trigger Points

Introduction

The ICOP (International Classification of Orofacial Pain), 1ST edition describes post-traumatic trigeminal neuropathic pain (PTNP) as "a unilateral or bilateral facial or oral pain following and caused by trauma to the trigeminal nerve(s), with other symptoms and/or clinical signs of trigeminal nerve dysfunction, and persisting or recurring for more than 3 months" [1].

The trauma could be accidental or iatrogenic during the elective dental or oral and maxillofacial surgical procedures. Some patients may develop chronic pain following negligible trauma, such as root canal therapy, whereas the more common iatrogenic causes leading to this complicated condition are removal of impacted third molars, placement of the dental implant, any local anesthetic injection, orthognathic surgery, mandibular trauma, and pathology resection [2,3].

Such events lead to the injury of branches of the trigeminal root. The branch most commonly affected is the inferior alveolar nerve (IAN) and the lingual nerve (LN), with an average of 90% of all the PTNP cases [4].

The IAN supplies somatic sensory innervation to the chin, lower lip, lower vestibular gingiva, molars, premolars, and alveolar bone. The LN supplies somatic sensory innervation to the lingual oral gingiva and the anterior two-thirds of the tongue. Therefore, these territories experience loss of general sensation predominantly, followed by loss of special sensation to the anterior two-thirds of the tongue as well as loss of general sensation to the lingual mucosa and floor of mouth [5]. Depending on the mechanism and severity of injury, most injured IANs and LNs recover spontaneously (85-94% of the time) [6].

PTNP has a substantial impact on the quality of life of

the patient. It has been shown that these patients report moderate to severe pain, and that this pain correlates with lower quality of life and depression [3].

The patients who experience severe neuropathic pain endure considerable psychosocial effects and this can interfere with a range of social interactions and everyday activities such as eating and drinking, shaving and even tooth brushing [4].

The management of neuropathic pain should be early and multimodal in nature, considering therapeutic alternatives, pharmacological, non-pharmacological neuromodulatory, and behavioral. All interventions are targeted to improving quality of life through pain reduction, sensory improvement, functional recovery, the development of efficient coping strategies, or a combination of these outcomes [3,7].
The present study includes five patients who were evaluated for neurosensory deficit after injury to their nerve, with a view to identify the underlying causes, the clinical manifestations and the factors that influence recovery in PTNP.

Method

Five patients with sensory dysfunction involving the trigeminal nerve following dental treatments were diagnosed with painful post-traumatic neuropathy in the Oral medicine clinic of the ITS Dental College, Muradnagar. The patients were placed on Pregabalin, TCAs and Neurobion. Each patient was followed up for a minimum of 3.5 months for treatment outcomes.

Case 1

A 35-year-old woman reported with a chief complaint of pain on the right side of the face for 2 years which was spontaneous in onset with moderate to severe intensity and radiating and pulsating in nature which began almost 6 months after the atraumatic surgical extraction of the right lower third molar. She had associated headaches, heaviness in the eyes and nausea. She also had loss of sensation and taste on the right side of the tongue which started immediately after the surgical extraction. On intraoral examination, it was noted that the extraction site was clean and had healed satisfactorily. A provisional post-traumatic trigeminal neuropathy on the right side of face involving the mandibular branch of trigeminal nerve was established. On her first visit, she was prescribed Tablet Neurobion Forte (bd for a week) and Tablet Pregabalin (50mg- tds for a week). A one- week review revealed improving condition with less pain, however, there was still loss of sensation on the left side of the tongue. The dose of Pregabalin was reduced to two times a day and was continued for another one month. On a subsequent review, there was no pain but the patient still experienced loss of sensation and taste on the right side of the tongue. Patient was followed up for 3.5 months and the last review revealed slight improvement in the sensation and taste perception.

Case 2

A 29-year-old woman reported with a chief complaint of pain on the lower right back tooth region from 6 months. The pain was sharp, intermittent, radiating and pulsating in nature. The patient also reported tingling in the involved region. On taking thorough medical and dental history it was revealed that she had visited a dentist 6 months back for surgical extraction of her lower right third molar but even after several attempts the tooth wasn't extracted whereas the first and the second lower right molars were noted to be extracted 1.5years ago.

On intraoral examination there was no evidence of hyperemia, ulceration, or palpable tenderness other than precisely at the site in question i.e., mesial to the lower right third molar region in the edentulous ridge. The tender site appeared normal radiographically and there was no abnormal prominence of the bone beneath. Pain was readily elicited when the site was pressed with the finger.

Diagnostic injection was not given since the area was diffused, LA was sprayed around the area, which reduced the pain and brought down VAS from 8 to 4. A provisional of Post traumatic trigeminal neuropathy involving Right IAN was given. The patient was kept on Tab Pregabalin 75 mg OD for 7 days, Tab Amitriptyline (TCA) 10 mg HS for 7 days and a local anesthetic gel was given to apply in the concerned region. The patient was recalled on 7th, 14th and 21st day of the treatment. She reported 60% relief after 21 days from the commencement of the treatment. There is mild improvement noted after 4.2 months of follow up.

Case 3

A 40-year-old woman reported with a chief complaint of pain in the upper right back tooth region for 3.5 years. On taking thorough history, the patient revealed that she had undergone extraction 4 years ago for dental pain in her right upper back tooth region. The pain was relieved after extraction but recurred after 2-3 months in the same region.

Pain was dull, continuous, pulsating and radiating in the preauricular and right temporal region. Eating, chewing, opening mouth and even speaking were the aggravating factors. All this led to aggravation of pain which resulted in headaches. Patient had undergone various tests and imaging but did not get any relevant diagnosis.

On intraoral examination of the patient nothing relevant was observed. After excluding all the possible somatic causes of pain in the stomatognathic system she was evaluated for neuropathic pain and she was tested for the sensory component of the affected area with the help of the cue tip on both the sides of the face and it was revealed that the sensory supply on the affected side was altered as compared to the normal side. A diagnosis of post-traumatic trigeminal neuropathy in the upper right region was given. On her first visit she had a VAS score of 10 and was prescribed Tricyclic Antidepressant and Pregabalin(75mg) for 10 days. After 10 days the patient was recalled and the VAS improved to 7 and the prescription was repeated. The patient was recalled subsequently after 10 days for a period of 2.5 months with the patient reporting a significant decrease in her VAS. The patient showed complete remission after 6 months of treatment.

Case 4

A 57-year-old female with complaints of pain around the right ear, difficulty in chewing and restricted mouth opening all of one month duration reported. She was referred from the Conservative Department in between visits for a root canal treatment of her lower left first molar. The pain initiated about a week after the endodontic procedure was commenced. The pain started spontaneously and subsided on its own periodically with no relevant aggravating or relieving factors on the left side of her tongue. On examination, severe tenderness was noted on the left preauricular region, other findings were normal. The provisional diagnosis of myofascial pain following long surgical hours was given and the patient was advised muscle relaxants for a week. Subsequent review showed no improvement in the patient's condition. The tooth was not responsive to pulp vitality tests, and findings from the periapical digital radiographs were clinically normal. A diagnosis of painful post-traumatic neuropathy secondary to root canal therapy was made and the patient was immediately commenced on pregabalin 75mg twice daily for one week. The follow up visit revealed marked improvement in the pain sensation with better mouth opening. Patient was continued on pregabalin 75mg twice daily for 2 weeks and. Patient's condition was resolved within 4 weeks of treatment and was followed up for 4 months.

Case 5

A 55 years old female patient reported with a chief complaint of numbness and pain in her right lower back tooth region following extraction of her right lower third molar. The pain was dull, continuous and radiating in nature which subsided after one week of extraction. The patient also complained of burning sensation and loss of taste perception on her right half of the tongue. On intraoral examination it was observed that the surgical extraction site was clean and had healed spontaneously with no signs of hyperemia or ulcerations. The patient was evaluated for mechanoreceptive and nociceptive responses. After complete evaluation provisional PTTN was given and the patient was kept on Tab Pregabalin 75 mg (bd), Tab Amitriptyline 20mg (hs) and Gaba Fenac gel three times daily for a period of 7 days and was recalled. On the follow up visit it was noted that the patient had a substantial improvement. The medication was repeated and the patient was recalled for subsequent visits after every 15 days for 5 months. A follow up of 5 months was made and it was noted that the patient was completely symptom free.

Discussion

Post-traumatic trigeminal neuropathic pain reflects neuropathic pain of traumatic origin affecting the trigeminal nerve either unilaterally or bilaterally. It may result from an abnormal regeneration of the trigeminal nerve. It presents as allodynia, hyperpathia, hyperalgesia, and sympathetic mediated characteristic pain reactivity in and around the dermatome affected when a sensory or mixed nerve is injured [8].

It may occur at any age. Overall, approximately 3-5% of patients with trigeminal nerve injuries develop PTTN [9,10].

The most common underlying cause for PTTN is impacted lower third molar extraction. Most of the patients in this study suffered neuropathy after the removal of lower third molars, with the observation of localized sensory defects.

The territory of the inferior alveolar nerve and lingual nerve was affected in over two-thirds of all cases in this series. Damage to the inferior alveolar nerve is explained by the anatomical proximity between the apexes of the third

molar and the canal that houses the nerve. Loss of general neurosensory deficit in the areas innervated by IAN and damage to LN resulting altered taste and somatic sensation on the affected side were observed in all the cases.

In a study conducted it was observed that the sensory deficit of the inferior alveolar nerve appeared to be the most frequent complica- tion following orthognathic surgery [11]. 616 patients (19%) showed a sensory deficit in their study. Whereas in the present series, procedures like third molar extraction and endodontic therapy led to similar deficits in all the cases.

In their case series of 5 patients concluded that injuries to the trigeminal nerve can arise from different dental treatments and a proper pre-operative assessment and surgical skill is very necessary in reducing the risk of nerve injury [9]. They also stated that early diagnosis with prompt intervention is very crucial in the treatment outcome and psychosocial wellbeing of the patient.

Nerve injury secondary to trauma or disease initiates inflammatory processes, alters electrical activity of neurons, and allows for cross talk between neurons resulting in a phenomenon called peripheral sensitization. The substances released during tissue injury initiate a cascade of events that may lead to nociceptors' activation sometimes even in the absence of a noxious stimulus [12].

The Diagnostic Criteria for Ptnp as Given by International Classification of Headache Disorders is.

A.	Facial and/or oral pain in the distribution(s) of one or both trigeminal nerve(s) and fulfilling criterion C
B.	History of an identifiable traumatic event[1] to the trigeminal nerve(s), with clinically evident positive (hyperalgesia, allodynia) and/or negative (hypaesthesia, hypalgesia) signs of trigeminal nerve dysfunction
C.	Evidence of causation demonstrated by both of the following: 1. pain is localized to the distribution(s) of the trigeminal nerve(s) affected by the traumatic event 2. pain has developed <6 months after the traumatic event
D.	Not better accounted for by another ICHD-3 diagnosis.

Nerve damage is likely to result from a combination of poor risk assessment, poor technique and late recognition or management of intraoperative and post-operative signs of neuropathy. Risk assessment involves the patient selection, preoperative planning, both clinical and radiographic and suitable treatment protocol and follow up [13].

The main risk factors for the nerve(s) injury are the surgical skill/experience of the surgeon and the radiographic proximity of the tooth apexes to the IAN and/or LN respectively. Other factors that influence treatment outcome include duration, (time from injury), location, age of the patient, surgical reconstruction of the injured nerve [5,8].

The mainstay treatment for neuropathic pain is pharmacotherapy but the clinical outcome could be unpredictable and highly variable. In our study, we managed all the patients with pharmacological intervention and the results were overwhelming.

Complete recovery of the sensation was seen in the patient with extraction of upper right molar (case 3) after the follow up of 6 months. The patient who was diagnosed with PTNP secondary to endodontic treatment (case 4) experienced complete remission within 4 weeks of medical treatment. This was suggestive of the possibility of a minor nerve injury. Of the patients diagnosed with PTNP following surgical extractions of lower third molar, case 5 had marked improvement whereas case 1 and 2 reported with mild to moderate improvement.

All the patients were counseled on the unpredictable treatment outcomes before commencement of drug therapy. Prompt examination and diagnosis may be facilitated by early identification of this etiology [14].

As a result, the chronic issues associated with trigeminal

nerve dysfunction, such as facial pain, and pain or weakness when chewing, may be minimized. This may also speed up anatomic localization of the injury, prognostic information, and potential intervention to reverse or prevent further deterioration of trigeminal nerve function.

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