

FACIAL PAIN - AN ENIGMA

Geeta Rajput¹, Sakshi Madhok², Anuj Bhargav³, Vasundhara Bhatt⁴

1- Prof & Head, 2- Asst. Prof 4- Intern , Dept of Prosthodontics, Dr Ziauddin Ahmad
Dental College, A.M.U. Aligarh ,
3- Reader, Deptt of Oral and Maxillofacial Surgery, Index Institute of Dental
Sciences, Index City., Indore. M.P.

ABSTRACT:

It is well known, pain is the most common reason why people seek healthcare. Pain is a personal, unpleasant sensory experience reflecting the physiologic, biologic, genetic and psychosocial factors. Some orofacial pain conditions are very complex and perplexing. With regular diagnosis, misdiagnosis and medical interventions which unfortunately later prove to be deceptive, such atypical orofacial pains are seemingly untreatable and puts the physician in a fix. In this article various facets of orofacial pain, peripheral mechanism, central sensitization, the absurd referral patterns and altered peripheral receptive field of chronic odontogenic pain are discussed with ample clinical evidences implicating the fact that all trigeminal pains are not trigeminal neuralgic pains. Treatment by classic drugs for trigeminal neuralgia in such cases cause more harm than any benefit to the patients. This article depicts cases showing how unremitting pain in trigeminally mediated areas secondary to dental causes is a unique challenge for the doctor and patient alike.

KEYWORDS. Atypical orofacial pain, Atypical odontogenic pain. Trigeminal neuralgia, Trigeminal pain.

INTRODUCTION:

Pain is considered as the 'Fifth vital sign'¹. It is an important health status indicator. The most widely used definition of pain is ' An unpleasant, sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'². Most commonly involved part with chronic pain in human body is the head and neck region³. Orofacial pain refers to a group of disorders including headaches, arthralgic and myalgic temporomandibular disorders, neuralgia, pain having odontogenic origin and idiopathic pain^{4,5}. The epidemiology, classification and diagnosis of orofacial pain presents challenges because of the complexity of anatomic structures involved, diverse pain referral patterns, varying causes and presenting symptoms and a lack of unanimity regarding the diagnostic criteria^{6,7}.

Chronic orofacial pain affects approximately 10% of adults and up to 50% of elderly⁵. Women are more likely to seek medical attention for pain as compared to men in the ratio of 2:1^{8,9}. Orofacial pain is modulated by various factors like the genetic and hormonal influences, the psychosocial factors, central sensitization mechanism, trigeminal deafferentation etc. Facial pain that does not come under any reasoning, existing category, or understanding is assigned as atypical facial pain. It is like a waste paper term for all the pains of the face which are medically unexplained or undiagnosed. It is the atypical orofacial pain that is most difficult to treat and diagnostically confusing to the oro-facial clinician. International headache Society has developed a classification of oro-facial pain¹⁰.

Trigeminal neuralgia (TN) also known as Tic douloureux or Fothergill's disease is a neuropathic orofacial pain originating in one or more branches of sensory distribution of fifth cranial nerve. It uncommonly expresses in ophthalmic division. It has an incidence of 2 - 27 per 100000 of general population¹¹. It can be due to trauma, secondary to demyelinating diseases (e.g. multiple sclerosis) or idiopathic. Pathology involves the neurons rather than the peripheral structures innervated by them. It affects women more frequently than men. It has a

predisposition for right side of the face. Common age of onset is after 50 and rarely before 30. TN can refer pain to teeth. Dentist is in a strategic position in recognizing TN in its early stages as this condition can mimic dental pain, prompting dental treatment before a definite diagnosis is made. Classic signs and symptoms of typical TN includes unilateral, sharp, shooting, lancinating or lightening bolt-like pain that lasts from a few seconds to 2 minutes and association with trigger zone that when stimulated triggers pain. Paroxysms may be continuous or occur at intervals with varying periods of remission. The attacks are often but not always precipitated by mild sensory stimulation of so called trigger zones which may be located anywhere in the territory of the affected trigeminal nerve. Triggers can be intraoral including teeth, mucosa, tongue or extraoral . Most common extraoral trigger zones are lateral to alae of nose and lip commissures¹¹. Commonly reported triggers are touch or light breeze on the face, talking, swallowing, brushing, applying make-up, chewing, washing face, chewing etc. TN could even be spontaneous without any identified trigger zone. Treatment of classical trigeminal neuralgia begins with drug therapy. Most frequently used drugs are also used to treat epilepsy, among which the gold standard remains carbamazepine. If drug therapy fails then surgical intervention may be done. These procedures result in pain relief for variable lengths of time.

As mentioned TN can be referred to teeth causing atypical odontalgia similarly chronic pulpal and periodontal pain as a result of central sensitization can be mediated in the trigeminal region mimicking atypical trigeminal neuralgia wherein the symptoms are not exactly but somewhat like that of classical trigeminal neuralgia. It is diagnostically confusing for the clinician. Such cases are refractory to conventional membrane -stabilizing anticonvulsant therapy for TN.

Failure to diagnose the orofacial pain results in unintended misdirected treatment and worsening of pain and suffering of the patient. Atypical Orofacial pain can be vexing for the physician and the patient. Even the most skilled physician is put to a test with unusual trigeminal pain presentations. Unremitting trigeminal pain motivates a search for relief which very often drains the patient . The case reports presented in this article are the quintessence of trigeminal pain secondary to pulpal and periodontal causes, which were misdiagnosed by the physician as trigeminal neuralgia due to ignorance of the fact that an odontogenic pain can have an absurd referral pattern in trigeminal region mimicking trigeminal neuralgia. Misdiagnosis was proved as these patients were refractory to anticonvulsant drug therapy.

CASE REPORTS:

Case 1: A 40 year old female patient presented herself in Deptt of Prosthodontics with chronic pain , moderate in intensity on the left side of the angle of mandible. She had been experiencing the pain for past five years. According to the patient pain was spontaneous and occurred at irregular intervals, was unilateral and of jolting nature. She was diagnosed with trigeminal neuralgia by a physician and was put on carbamazepine twice a day for a year, but to the patient's disappointment she had no relief. She complained that the problems of nausea and drowsiness appeared after starting these medicines. Her vision was reduced after starting the drug therapy. On clinical examination there was no trigger zone found and the pain was diffuse in nature. However, it was found that patient had grade III calculus and plaque deposition. The gingiva was severely inflamed with deep pockets (Fig1).



Fig 1.

Case 2: A 65 year old male patient was referred to the Department of Prosthodontics by a neurophysician. The presenting complain was severe pain in the mandible. He had this agonizing pain for past six to seven years, which was on and off. According to the patient the pain was severe in the morning and of throbbing type. The patient underwent multiple dental treatments like root canal therapy, extractions, etc. He did get some relief post treatment but only temporarily. He also complained of increased salivation. He was diagnosed with trigeminal neuralgia two years back and was put on carbamazepine and phenytoin and was on these medication for past two years. According to the patient there was some relief in the intensity of the pain for sometime after starting the medications but then the condition recurred. On clinical examination no trigger zones were found, pain was present unilaterally on the left side and in the lower anteriors. Grade III calculus and

plaque were present. Severe generalized gingival recession, cervical abrasion and furcation involvement were present. Radiographically lower left anterior tooth shows failed root canal treatment, and generalized bone loss(Fig 2).



Fig2

Case 3: A 60 year old female patient presented with severe constant throbbing pain in the anterior portion of lower jaw. The patient was diagnosed with trigeminal neuralgia and was on carbamazepine and gabapentin, for four months. There was no relief in the pain instead the patient complained of nausea and drowsiness after taking the medications .On clinical examination, the oral hygiene of the patient was found to be very poor. There was grade III calculus and plaque deposition and severe generalized gingival inflammation. Grade I mobility was found in the lower incisors (Fig 3,4).



Fig3



Fig4

Case 4: A 40 year old male patient presented with the pain in the mandible for six to seven months. According to the patient the pain was intense and it increased

tremendously while eating, drinking, talking and brushing the teeth. The patient was diagnosed with trigeminal neuralgia and was put on phenytoin for 4 months, consequently gingival hyperplasia was observed. The patient complained despite of regular medication there was no improvement in the severity or the episodes of pain .On clinical examination it was found that the oral hygiene of the patient was very poor. Plaque and calculus score was grade III. There was severe generalized gingival inflammation and bleeding on probing was present. The patient had severe sensitivity to cold which was confused with pain. No trigger zone was found (Fig 5,6).



Fig5



Fig6

Case 5: A 53 year old female patient presented with the complaint of pain in the right side of the cheek since last three years. According to the patient, the pain was of throbbing nature and it radiated towards the auricular region while chewing the food and talking. The patient was put on analgesics by some local practitioner for one month and as there was no permanent relief she was referred to another practitioner who diagnosed the pain as trigeminal neuralgia and put her on carbamazepine. Following the drug therapy she developed nausea and vomiting. On clinical examination, no trigger zone was found. Oral hygiene was very poor with grade I mobility in the lower anteriors and accumulation of plaque and calculus(Fig7, 8, 9,).



Fig7



Fig8



Fig9

TREATMENT:

None of the patients had any intraoral or extraoral trigger zone. Chronicity of facial pain, intermittent pain, elderly patients and trigeminal pain with unilateral involvement prompted the physicians towards the diagnosis of trigeminal neuralgia. But to their dismay this did not show any results. Emotionally and financially drained patients were finally referred to department of prosthodontics suspecting a temporomandibular disorder. Thorough history was taken, regarding the episodes, intensity, duration and nature of the pain. The medications prescribed earlier for trigeminal neuralgia were stopped. The side effects as in gingival enlargement, drowsiness, nausea gradually faded away with the cessation of anti convulsant drug therapy. Masticatory muscles palpation, and proper inspection of temporomandibular joint revealed no obvious pathology. Most of the patients had unstable occlusion, were partially edentulous and with a thorough oral hygiene neglect. Complete oral prophylaxis (scaling, curettage and root planning) was performed. Periodontal flap surgery was undertaken in the cases with the furcation involvement. Cervical abrasions were restored with composite. Teeth showing pulpal involvement were treated by root canal therapy. Hopeless teeth were extracted. The patients were put on multivitamins and antioxidants for a month and were instructed to maintain a good oral

hygiene. They were recalled after a week. It was found that there was tremendous relief in pain. Further, on regular follow up every 3 months and a proper maintenance phase no recurrence of symptoms were seen. Later their dentition was restored with artificial substitutes. Post treatment pictures of Case 1(Fig 10), Case 3(Fig 11,12), Case 4 (Fig 13,14), Case5 (Fig15,16,17) are shown.

Case 1:



Fig 10

Case 3:



Fig 11



Fig 12

Case 4:



Fig 13



Fig 14

Case 5:



Fig 15



Fig 16



Fig 17

DISCUSSION:

Goal of any orofacial clinician is to alleviate the pain and suffering of patients. Orofacial region is afflicted by a number of acute, chronic and recurrent painful maladies. One can achieve optimum management only by determining accurate and complete diagnosis and identifying all of the factors associated with underlying pathosis. No relief in pain with anti-convulsant medicaments of the suspected trigeminal neuralgia patients is an alarm bell for the physicians and neurophysicians . Refractory trigeminal pain poses a significant diagnostic dilemma. Trigeminal pain can be odontogenic, arthralgic, myalgic, neurovascular or neurologic in origin. A thorough knowledge of the epidemiologic, etiologic and pathologic aspects of various types of orofacial pain is mandatory for proper diagnosis of a given specific case.

One should not be dogmatic in concluding any pain in trigeminal region to be trigeminal neuralgia pain. Deafferentation mechanism; peripheral, central, or sympathetically maintained pain usually is present when patients are labelled refractory to treatment or atypical^{12,13}. The

trigeminal nerve is a final outlet of face, neck and head pain¹⁴. As a result of central connections there is a high possibility of referral between divisions¹⁵. It is known that pain signals from sites of tissue or nerve injury lead to long term changes in the CNS, in the amplification and persistence of pain. These nociceptor induced neuronal changes known as central sensitization have important implications in understanding and managing orofacial pain. Central sensitisation is responsible for the expansion of receptive field of pain as seen in the above cases. Main diagnostic challenge lies in distinguishing between atypical trigeminal neuralgia and atypical orofacial pain. Mixed symptom cases that do not fall in the category of classical TN are atypical TN. When the pain goes beyond the areas served by trigeminal nerve it gives an even murkier tag of atypical orofacial pain. This differentiation is very critical as the line of treatment of both are not alike. Whereas TN is treated mainly by anticonvulsant therapy, atypical orofacial pain responds to anti-inflammatory and antidepressant therapy.

Pain can be nociceptive, neuropathic or mixed. Nociceptive pain is due to tissue damaging stimuli via peripheral nerves to CNS¹⁶. Examples include frank dental pain, degenerative joint disorders and myofascial pain. It is typically described as diffuse, aching, stiff or tender. Neuropathic pain is caused by primary pathology in the nervous system¹⁶. Examples are trigeminal neuralgia, post-herpetic neuralgia, idiopathic oral burning etc. Aching, burning, stabbing, sharp and electric like, numbness or tingling projected to cutaneous areas are the typical pain description for this type of pain. Mixed pain is caused by combination of primary and secondary effects as is seen in this series of cases. It is described by numerous terms that are diagnostically perplexing. For optimum treatment each of these pain types should be targeted at.

Chronicity of pain is one of the factors which prompts the physicians towards the diagnosis of TN in the cases presented. One critical concept is chronic versus acute pain. The international Association for the study of pain has defined chronic pain as pain lasting more than six months. Acute pain refers to pain lasting less than six months. Pain pathway has two divisions. First is the discriminative system and the other is motivational or effective system. The discriminative system allows the brain to properly understand, locate and isolate the site source and duration of pain. The motivational or effective system involves the emotional component of painful experiences¹⁷. During the first 6 months, the discriminative system dominates however, as the time progresses expression of motivational system strengthens. At six months inversion of

expression of pain response system occurs. As a result, the description by a chronic pain patient has got more of psychologic than descriptive terms. As the pain continues unresolved, the pain language becomes more nondescript i.e. it becomes difficult for the practitioner to identify the source and site of pain. This is in consensus with the case reports present herein. Their history of pain is just giving a clue that is not easy to understand and manage. Many researches have shown that higher level of psychological issues, anxiety levels, depression, sleep disorders are associated with patients having chronic orofacial pain^{18,19}. Thus a practitioner should rule out the psychological distress, orofacial patient is in while making a diagnosis.

As discussed chronic orofacial pain patients give more of vague, nondescript and confusing signs and symptoms which a clinician needs to trace to reach to a proper diagnosis. As Lasagna stated 'The clinician studying pain is at the mercy of the patient upon whose ability and willingness to cooperate and communicate he is dependent'²⁰. One should not get swayed away by classical features of trigeminal pain (e.g. unilateral pain or shock -like pain) in a haste to make a diagnosis, thus misdiagnosing it as TN neither should one overlook the history of ineffective treatment response given by the patient. Doctor's patience, through knowledge of orofacial ailments and pain mechanism, a multidisciplinary approach and patient's cooperation are all involved in successful management of orofacial pain. As seen in the exemplified cases their pursuit for effective treatment was rewarded by mere routine dental procedures. The initially presumed refractory and idiopathic trigeminal neuralgic pain was basically trigeminal mediated pain secondary to chronic dental pathosis following central sensitization mechanism.

CONCLUSION:

Chronic pain is a disease in itself. These case presentations provide a broad overview of varying and diagnostically perplexing clinical course of chronic odontogenic pain which were confused with trigeminal neuralgic pain by the physician.. All the clinicians should understand the peripheral and central mechanisms of pain and other psychosocial issues attached to it. This understanding will lead to more effective treatment. Ultimate goal is to manage pain. When suffering continues, re- evaluation of the clinical condition is necessary. Many seemingly refractory atypical facial pain can be treated by competent dental therapy rather than any other aggressive treatment. Cases of chronic orofacial pain are best managed by a multidisciplinary team involving dentist, neurologist, psychologist and other health care disciplines.

BIBLIOGRAPHY:

1. Lanser P, Gesell S. Pain management: the fifth vital sign. *Health benchmarks* 2001; 8(6):68-70
2. International Association for the study of pain. Subcommittee on taxonomy of pain teams: a list with definitions and notes on usage. *Pain* 1979;6:249-52
3. Donaldson D, Kroening R. Recognition and treatment of patients with chronic orofacial pain. *J Am Dent Assoc* 1993; 124:115-21
4. Agostoni E, Frigerio R, Santoro P. Atypical facial pain: clinical consideration and differential diagnosis. *Neurol Sci* 2005; 26(Suppl2):S71-4
5. Madland G, Newton-John T, Feinmann C. Chronic idiopathic orofacial pain:I: What is the evidence Base? *Br Dent J* 2001;191(1):22-4
6. Esposito CJ. Considerations in the diagnosis of orofacial pain and headache. *J Ky med Assoc* 2001;99(10):430-6
7. Gremillion HA. Multidisciplinary diagnosis and management of orofacial pain. *Gen Dent* 2002;50(2):178-86
8. Dao TT, LeResche L. Gender differences in pain. *J Orofac Pain* 2000; 14(3):169-84
9. Fillingim RB. Sex, gender and pain: women and men really are different. *Curr Rev Pain* 2000;4:24-30
10. International Headache Society Classification Committee. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalgia* 1998;8(Suppl7):9-96
11. Spencer CJ, Gremillion HA. Neuropathic orofacial Pain: Proposed Mechanisms, Diagnosis, and Treatment considerations. *Dent Clin N Am* 51(2007)209-224
12. Graff-Radford SB, Solberg WK. Differential neural blockade in atypical odontalgia. *Cephalgia* 1991; Suppl 11(2):289-91
13. Graff- Radford SB. Facial pain. *Curr Opin Neurol* 2000;13:291-6
14. Piovesan EJ, Kowacs PA, Tatsui CE, et al. Referred pain after painful stimulation of the greater occipital nerve in humans: evidence of convergence of cervical afferents on trigeminal nuclei. *Cephalgia* 2001;21(2):107-9
15. Bartsch T, Goadsby PJ. Increased responses in trigeminocervical nociceptive neurons to cervical input after stimulation of duramater. *Brain* 2003;126:1801-13

16. Merskey H, Bogduk N. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. 2nd edition. Seattle(WA): IASP Press: 1994
17. Auvenshine RC. Temporomandibular Disorders: Associated Features. Dent Clin N Am 51(2007)105-127
18. Korszun A, Hinderstein B, Wong M. comorbidity of depression with chronic facial pain and temporomandibular disorders. Oral Surg Oral Med Oral Path Oral Radiol Endod 1996; 82:496-500
19. Kight M, Gatchel RJ, Wesley L. Temporomandibular disorders: evidence for significant overlap with psychopathology. Health Psychol 1999;18:177-82.
20. Lasagna L.. Clinical measurements of pain. Ann N Y Acad Sci 1960;86:28-37

Acknowledgement- None

Source of Funding- Nil

Conflict of Interest- None Declared

Ethical Approval- Not Required

Correspondence Addresses :

Dr. Sakshi Madhok
Asst. Prof, Dept. of Prosthodontics
Dr Ziauddin Ahmad Dental College
A.M.U. Aligarh

Email ID- Sakshi_madhok@yahoo.co.in