International Journal of Science and Research Archive
eISSN: 2582-8185
Cross Ref DOI: 10.30574/ijsra
Journal homepage: https://ijsra.net/

(CASE REPORT)

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Low level laser therapy in the management of trigeminal neuralgia: A rare case report

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International Journal of Science and Research Archive, 2024, 12(01), 2026–2030

Publication history: Received on 16 April 2024; revised on 26 May 2024; accepted on 29 May 2024

Article DOI: https://doi.org/10.30574/ijsra.2024.12.1.0938

Abstract

Trigeminal neuralgia (TN) is characterized by sudden, severe, brief, and stabbing recurrent episodes of facial pain in one or more branches of the trigeminal nerve. Pain attacks can occur spontaneously or can be triggered by non-noxious stimuli, such as talking, eating, washing the face, brushing teeth, shaving, a light touch or even a cool breeze. In addition to pain attacks, a proportion of the patients also experience persistent background pain, which along with autonomic signs and prolonged disease duration, represent predictors of worse treatment outcomes. Here, we present a classical case of trigeminal neuralgia affecting a 63yrs old male patient characterized by electric shock like pain lasting for the duration of 2 months.

Keywords: Trigeminal neuralgia; 5th Cranial nerve; Nerve compression; Low level laser therapy

1. Introduction

Trigeminal neuralgia is a rare condition marked by abrupt, recurring, and intense episodes of sharp, shock-like pain, lasting from a few seconds to two minutes [1]. It is defined as a disorder characterized by recurrent unilateral brief electric shock-like pain, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli by international headache society[2].TN is identified by recurring, unilateral bouts of brief pain affecting one or multiple branches of the trigeminal nerve. The pain is often described as sharp, piercing, electric-like, severe & sudden. Additionally, these pain episodes may coincide with tic-like spasms, characterized by involuntary facial muscle contractions, leading to the early label of TN as "tic douloureux." Pain episodes may arise without warning or be provoked by harmless activities like speaking, eating, washing the face, grooming, or even a gentle touch or breeze. Each attack lasts from a split second to two minutes, and individuals typically experience no symptoms between episodes[2]. Classical TN, formerly known as Idiopathic Trigeminal Neuralgia, refers to TN resulting solely from neurovascular compression. It is divided into two subtypes: 1) Classical TN, characterized by intermittent bouts of pain, and 2) Classical TN accompanied by ongoing facial pain. [4]. Trigeminal neuralgia-like pain that is related to an underlying disease, including tumors, trauma, viral infection, and multiple sclerosis is classified as Secondary TN[2]. TN usually affects the maxillary (V2) and mandibular (V3)trigeminal branches. The ophthalmic branch (V1) is afflicted less common [5]. It has been proposed that trigeminal neuralgia is caused by demyelination of the nerve, which leads to ephaptic impulse transmission[6]. various supportive treatment options have been developed to manage

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pain in TN. Carbamazepine is typically the initial medication of choice, although alternative medical and surgical strategies are employed for cases that do not respond to conventional therapy[7]. The objective of this case report is to discuss enticing response of TN to Diode laser therapy in a 63 year old man.

2. Case report

This is a 63 years old male patient, reported with the chief complaint of pain in his left side of face for the past 2 months. Patient gave history of intermittent, electric shock like pain, with each episode of pain lasted for 4 to 5 seconds, 7-8 episodes per day, aggravated by touching the face, talking, washing the face, gargling, breeze, during each episode of pain he holds the face with his hands until the pain subsided. Patient gave history of visiting a dental practitioner for the same before 2 years and underwent extraction of multiple teeth in the second quadrant, after which, patient was asymptomatic and pain again recurred, with increase in frequency and intensity over the last 1 month. Patient is known hypertensive for the past 3 years & is under medication for the same (Tab.Amlodipine 2.5mg 1-0-0). On Extra-oral examination, On palpation, Tenderness elicited on palpating the trigger zones in the Left malar region, upper lip, nasolabial fold on the left side. The pain was electric shock like, intense, lasting for a short duration of about 4-5 seconds. Patient holds the face and clenches due to pain until the episode subsides. On intraoral hard tissue examination, clinically missing: 15,18,23,24,25,26,27,28,36. On inspection, No marked abnormality detected. On palpation, tenderness elicited over the gingiva, vestibule and alveolar ridge in in relation to 24,25,26,27 region. Visual Analogue Scale(VAS) score was 9. Based on the history and clinical findings, provisionally Trigeminal neuralgia of Left maxillary division V2 was given. Patient underwent radiological investigations such as IOPA(Intra oral periapical radiograph) reveals normal trabecular bone pattern with completely missing 25, 26,27,28. Later, MRI(Magnetic Resonance Imaging) of brain was taken which gives impression of Small nodular lesion seen in the parietal falx region measuring – 1.2x0.9cm in size and Vertebro basilar dolichoectasia indenting over left anterior pons and left trigeminal Nerve Entry Zone region as shown in figure 1.

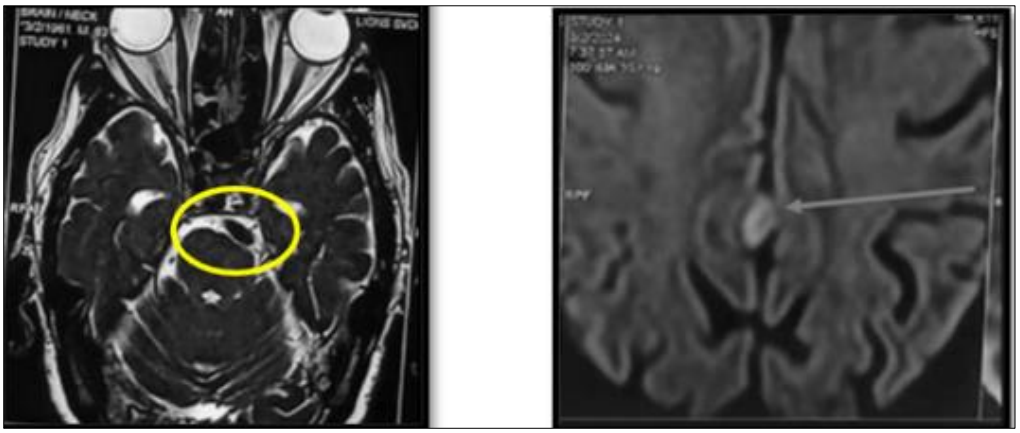

Figure 1 MRI shows Vertebro basilar dolichoectasia indenting over left anterior pons and left trigeminal Nerve Entry Zone region

On Correlation of radiological features with clinical findings, Trigeminal neuralgia of Left maxillary division of nerve V2 was given. At first visit patient was prescribed with medications (Tab. Carbamazepine 200mg 1-0-1,Tab. Baclofen 10mg 0-0-1 for 1 month after food) and asked to report back after 15 days. After 15 days, the pain did not subside on taking medications, so patient was subjected to low level laser therapy using diode Laser. Before the procedure, VAS score was 9, the laser therapy was performed under non-contact mode for 25 minutes duration placed in the trigger points for 3 minutes each as shown in figure 2, using continuous wave, 0.2Watts power,650nm wavelength. This was done for 5 days. During the laser therapy, patient had 3-4 pain episodes per day. At the end of fifth session, patient's VAS score reduced to 1. Patient was advised to continue the medication during the laser therapy. After the completion of therapy, Patient is asymptomatic and under regular follow-up.

Figure 2 LLLT placed in the trigger zones

3. Discussion

Trigeminal neuralgia manifests as intense facial pain marked by frequent bursts of electric shock-like sensations, typically triggered by neurovascular compression, often caused by the superior cerebellar artery [2]. Changes in voltage-gated sodium channels could explain heightened neuronal excitability and abnormal firing of incoming trigeminal nerve fibers [8]. Various levels of compression severity have been outlined, ranging from mere nerve contact to pronounced indentation, nerve degeneration, and distortion. The prevalence of V2 and V3 involvement is thought to stem from the somatotopic arrangement of sensory fibers within the trigeminal root, with vascular compression commonly located in a superior-lateral or inferior position relative to the nerve root's circumference[9]. Multiple studies have consistently shown that trigeminal neuralgia predominantly affects the right side of the face compared to the left[10]. One proposed theory suggests that the lateralization of pain in trigeminal neuralgia is attributable to a narrower foramen rotundum and foramen ovale on the right side[11]. In an effort to link the paroxysmal pain episodes with the structural changes observed in compressed trigeminal neurons, Devor and his team developed the ignition hypothesis, which has gained widespread acceptance in elucidating the pathophysiology of trigeminal neuralgia[12]. According to this hypothesis, sensory neurons that undergo partial damage become highly excitable and prone to cross-excitation due to their close proximity at the site of root compression. Consequently, the bursts of post-trigger neuronal activity recruit nearby neurons rapidly, resulting in a swift buildup of electrical activity. This activity can be further intensified by ephaptic interaction among neurons, facilitated by the compromised myelin sheath and the maintenance of close contact between nerve fibers. As a result, stimulation of a single sensory fiber may activate numerous others, and the neuronal activity triggered by an external stimulus may persist beyond the stimulus duration [12]. Over the last four decades, significant advancements have been made in the development of pharmacological and surgical treatments for trigeminal neuralgia. Prior to these developments, the most efficacious treatments involved either injecting caustic substances to destroy the affected nerve branch or surgically severing the sensory trigeminal root behind the gasserian ganglion[13]. Carbamazepine stands as the preferred medication for the initial management of trigeminal neuralgia[14]. Its primary mechanism involves blocking sodium channels in neuronal membranes, particularly during periods of high-frequency stimulation. This action diminishes the transmission of electrical signals and restricts the expansion of ectopic activity[15]. The administration of carbamazepine has been linked to diverse hypersensitivity reactions, spanning from mild maculopapular exanthemas to severe conditions such as hypersensitivity syndrome, drug reaction with eosinophilia and systemic symptoms, acute generalized exanthematous pustulosis, Stevens-Johnson syndrome, and toxic epidermal necrolysis[16]. Gabapentin, classified as a second-line medication, operates by disrupting nociceptive transmission within the central nervous system, targeting the α2δ subunit of voltage-dependent calcium channels [1]. Surgical interventions are recommended for patients experiencing severe symptoms of TN that significantly impair their daily functioning, those who do not respond to or experience recurrence of TN despite treatment, or those who cannot tolerate the adverse effects of medication[17]. Additionally, low-level laser therapy (LLLT) has been employed in the treatment of nerve injuries. It has been proposed that LLLT enhances nerve function, improves myelin production capacity, and facilitates axonal growth in damaged nerves. Its mechanism involves influencing prostaglandin synthesis, promoting the conversion of PGG2(Prostaglandin G2) and PGH2 (Prostaglandin H2) into PG12 (prostacyclin), which possesses vasodilatory and anti-inflammatory properties. One of the primary advantages of LLLT is its non-invasive nature, offering the capability to address nerve injuries without the need for surgical procedures, rendering it an attractive treatment choice[18].

4. Conclusion

Trigeminal neuralgia is diagnosed based on clinical assessment, with the primary cause often being neurovascular compression impacting the cisternal segment of the fifth cranial nerve. MRI serves as a useful tool to confirm this neurovascular contact and rule out alternative causes, which can range from the brainstem to areas beyond the skull. Apart from the regular pharmacological and surgical management, low level laser intervention is considered to be a boon in the treatment of trigeminal neuralgia which relieves the patient symptomatically and improves the quality of life.

Compliance with ethical standards

Statement of ethical approval

Since it is a non-invasive chair side procedure, ethical approval is not applicable for this case report.

Statement of informed consent

Informed consent was obtained from the patient.

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