



Pulsed Radiofrequency Therapy in Elderly People with Trigeminal Neuralgia: A Case Report

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Abstract. Introduction: A face nerve disorder known as trigeminal neuralgia is regarded as one of the most excruciating human ailments. Depending on the patient's medical comorbidities, trigeminal ganglion interventional techniques can lessen discomfort. The application of brief radiofrequency pulses via a needle tip that do not result in genuine thermal lesions is known as Pulsed Radiofrequency (PRF) therapy. **Case:** Elderly man complained of pain in the right upper and lower jaw. He was diagnosed atypical trigeminal neuralgia. The man got pulsed radiofrequency on the right trigeminal nerve with the help of C-arm, with temperature specifications of 42°C, voltage 45V, pulse width 20 ms, frequency 2 Hz, with a duration of 8 minutes. 1 week after the procedure, the VAS became 0. Oral therapy still continued. **Discussion:** The most prevalent cranial neuropathy is trigeminal neuralgia (TM). Traditional RF for TM is not without its problems. Research indicates that both early and late alterations in cellular activity, unaffected by temperature, take place in the cervical dorsal root ganglion (DRG) and rat dorsal horn following PRF exposure. According to meta-analysis, PRF is safer and more effective than conventional radiofrequency. By decreasing phosphorylated extracellular signal regulated kinase (a central sensitization biomarker), decreasing Ca²⁺ influx, which lowers nerve cell potential activity, and decreasing mitochondrial membrane potential, which lowers cytosolic ATP levels, PRF modifies sensory ganglion nerve cells. Reduced sensitivity to pain results from a decrease in nerve ganglion cell activity. **Conclusion** PRF for trigeminal neuralgia can be alternative treatment with better safety and efficacy.

Keyword: Manajemen Nyeri; Neuralgia Trigeminal; Nyeri neuropatik; Pulsed Radiofrequency (PRF); Saraf Trigeminal.

1. INTRODUCTION

Trigeminal neuralgia (TN) is a facial nerve problem that is regarded as one of the most painful conditions in humans. It is sometimes referred to as *tic doloureux* or *Fothergill's sickness*. About 50% of patients with trigeminal neuralgia do not benefit from continued use of oral medications. The pain has periods of remission, but it tends to become less frequent over time and occasionally develops into more persistent aches, pains, and burning sensations, which may or may not be accompanied by sharp attacks. Surgery is the most effective treatment, and interventional techniques on the trigeminal ganglion can lessen discomfort. Nonetheless, a patient's medical comorbidities, the risks they are willing to take, and, to a lesser extent, their age determine the treatments that can be administered (Turton, 2019).

Trigeminal neuralgia pain can be treated in a number of ways. from carbamazepine-based conservative therapy. Depending on the patient's ASA status, traditional treatment may involve radiofrequency intervention for elderly individuals or surgery for young people if pain ratings and quality of life do not improve (American Medical Association, 2018).

The application of brief radiofrequency pulses via a needle tip that do not result in genuine thermal lesions is known as *pulsed radiofrequency* (PRF) therapy. Divergent opinions exist regarding PRF in trigeminal neuralgia (TN). According to the author, one of the primary causes of these divergent opinions is the inadequate dosage of PRF employed in earlier research (Chua, 2012).

In a recent investigation into how PRF affected neuropathic pain caused by *resiniferatoxin* in an animal model, the anti-allodynic impact of PRF was noticeably stronger when exposure time was extended from two to six minutes (Chua, 2012). In this instance, the PRF exposure time is extended to eight minutes. With a 45volt voltage, a 2 Hz *pulse frequency*, and a 20 ms *pulse width*, the purpose of this case presentation is to observe the effects of PRF therapy on patients over the course of eight minutes.

2. CASE

Anamnesis

- a. Name : Mr. S
- b. Age : 72 years
- c. Gender : Male
- d. Occupation : Retired
- e. Address : Madiun Regency.
- f. Main Complaint : Right side facial pain.

The patient had been complaining of pain in the right upper and lower jaw for around seven months prior to visiting the pain clinic. When the patient visited the dentist, all of the teeth on the right side were extracted.

The patient complained of right-sided facial pain following tooth extraction. The pain is not above the eyes, but rather in the upper and lower jaws. Pain less than 1 minute per attack. The pain was so severe that the patient cried at every pain, sharp pain like a short electric shock in the right face. Pain recurs when talking, washing face, eating, when gargling. There are no watery eyes, facial twitching, and nausea and vomiting during attacks. There are no coughs and colds. Denied history of facial skin disorders. Accident history denied.

The patient only treated with medication given by the dentist, the pain still did not go away. The pain increases and the recurrence becomes more frequent. The patient went to the pain clinic at Soedono Madiun Regional Hospital. History of comorbidities: denied.

Physical Examination

Generalist Status

At normal status except Numeric Pain Scale: 8/10 during attack

Localist Status

No specific abnormalities were found. All the upper and lower right quadrant teeth have been removed. Mastication movements are within normal limits.

Supporting Examinations

Are not done.

Differential Diagnosis

- a. Atypical (idiopathic) trigeminal neuralgia
- b. Classical Trigeminal Neuralgia
- c. Secondary trigeminal neuralgia
- d. Sphenopalatine neuralgia
- e. Maxillary sinusitis
- f. Temporo mandibular arthritis
- g. Cluster headaches
- h. Migraine

Diagnosis

- a. Clinical Diagnosis : Trigeminal neuralgia
- b. Anatomical Diagnosis : Cranial Nerve V Neuropathy.
- c. Pathological Diagnosis : Atypical trigeminal neuralgia

Therapy

PRF was performed on the right trigeminal nerve at the level of V2 V3 with the help of Carm, with temperature specifications of 42°C, voltage 45V, *pulse width* 20 ms, frequency 2 Hz, with a duration of 8 minutes. The PRF needle enters the lesion target with the help of the C-arm. Home medication was given paracetamol 3x 500mg po and carbamazepine 2x200mg po.

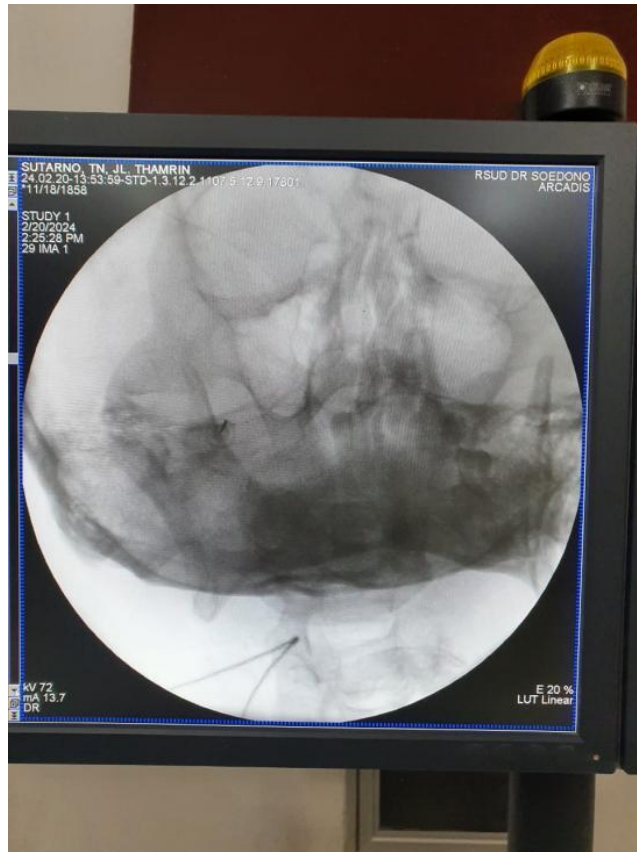


Figure 1. Submental obliq view PRF needle entering foramen ovale.



Figure 2. Lateral view needle through petrosus line.

Therapy Results and Follow Up.

Pain decreased after the procedure from VAS 8 to VAS 3. 1 week after the procedure, the VAS became 0. The patient was able to return to his normal activities without pain. Initiate carbamazepine dose reduction to 2x100mg p.o.

3. DISCUSSION

Trigeminal Neuralgia

The maxillary (V2), mandibular (V3), and ophthalmic (V1) are the three sensory branches of the trigeminal nerve. The ocular, supraciliary, frontal, and upper nasal areas are innervated by the V1 branch; the zygomatic area, upper teeth, lateral nose, lower eyelids, and upper lip are innervated by the V2 branch; and the mandibular area and temporomandibular joint are innervated by the V3 branch (Lambru, 2021).

Van Kleef (2008) state that V2 and V3 are the most commonly impacted branches, accounting for 32% of cases, with only V2 or all branches being involved at 17%. V1 was the branch that was least involved (4%). The most prevalent cranial neuropathy is trigeminal neuralgia, which affects 4.3 out of every 100,000 individuals annually. It is more common in women than in men (5.7 versus 2.5% per 100,000 population), and it is more common in older adults (11 instances per 100,000 over 75) (Lambru,2021).

According to the third edition of the International Classification of Headache Disorders (ICHD-3), TN is defined as recurrent paroxysmal unilateral facial pain that is limited to the trigeminal distribution, lasts anywhere from a few seconds to two minutes, is extremely intense, has a piercing or sharp quality, and is brought on by innocuous stimuli (Zakrzewska, 2021).

Based on the underlying etiology, trigeminal neuralgia is further divided into three categories: classic, secondary, and idiopathic. The most prevalent kind, known as the classic kind, is identified when trigeminal neurovascular compression with morphological (Turton, 2019).

During surgery or on MRI imaging using the proper trigeminal sequence, changes ipsilateral to the side of pain are shown. Despite being a typical neuroimaging finding, simple trigeminal contact without morphological alterations is insufficient to support such a diagnosis (Lambru, 2021).

Prospective trigeminal MRI imaging investigations in healthy individuals have demonstrated that on the symptomatic side, TN classical while these morphological alterations are uncommon in the unaffected side, trigeminal neuralgia is linked to

neurovascular compression with morphological abnormalities (distortion, indentation, atrophy). The secondary type, which makes up about 15% of cases, is brought on by a distinguishable underlying neurological condition that is known to cause trigeminal neuralgia, such as multiple sclerosis, cerebellopontine angle tumors, and arteriovenous malformations, with the exception of trigeminal neurovascular compression. Trigeminal neuralgia-like symptoms are seen in about 2% of people with multiple sclerosis. When there is no apparent explanation for trigeminal neuralgia, the idiopathic type which makes up around 10% of cases is diagnosed. Classic and idiopathic TN are further divided into groups with continuous or almost continuous interictal pain, pure paroxysmal pain, or concurrent chronic pain (depending on whether pain is present or absent) (Lambru, 2021).

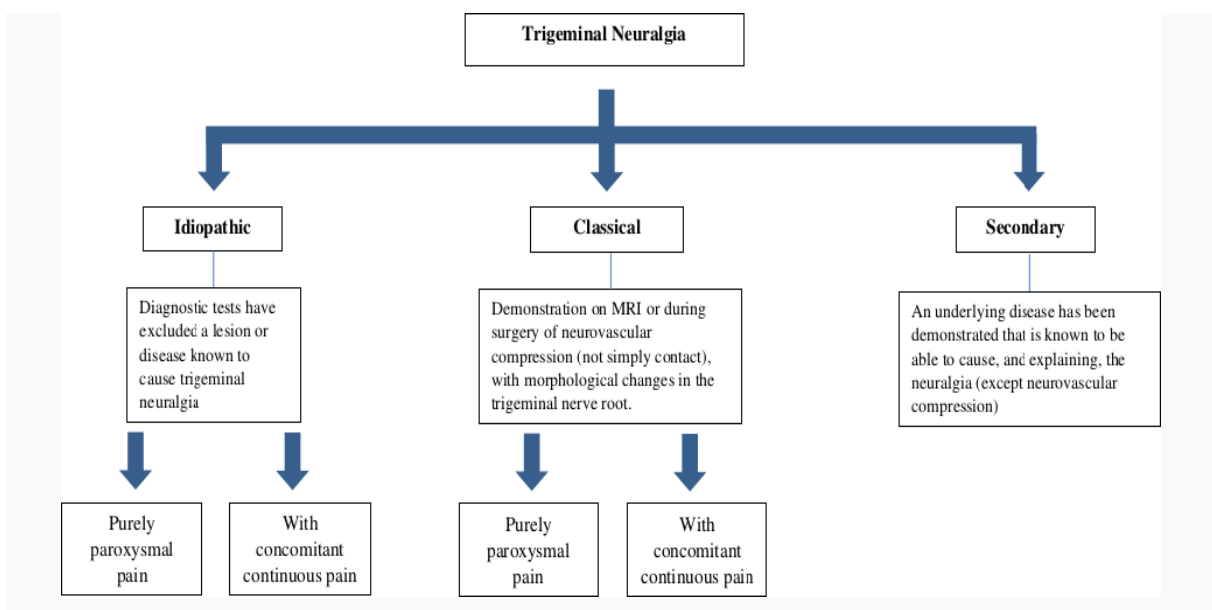


Figure 3. Classification of Trigeminal Neuralgia.

More damage is done to the right side of the face (60%) than the left. In patients with TN, unilateral pain bouts that alternate from side to side are more common than bilateral simultaneous pain, which is uncommon (1.7%–5%). Due to its infrequency, bilateral trigeminal paroxysmal pain that occurs simultaneously or alternately should arouse suspicions of a neurological or non-neurological condition affecting the skull. As a result, secondary pathology must be carefully ruled out. Temporomandibular joint dysfunction, persistent idiopathic facial pain, and infrequently migraine with facial pain are examples of persistent or long-lasting idiopathic bilateral trigeminal pain, assuming the tests are normal. Autonomic trigeminal cephalgia, such as short-term unilateral neuralgiform headache attacks (SUNHA), should be taken into consideration in cases of brief episodes of paroxysmal pain if the pain is accompanied by cranial autonomic symptoms, or idiopathic

stabbing headache if the pain primarily affects the distribution nerve trigeminal in the eye (V1)(Lambriu,2021).

The maxillary (V2) and mandibular (V3) divisions of the trigeminal nerve are most frequently affected by pain in trigeminal neuralgia, however the ophthalmic (V1) division is involved in roughly 25% of cases (Lambriu, 2021).

Attacks of trigeminal neuralgia vary widely in frequency and duration. A tiny percentage of people describe attacks lasting two to ten minutes, even though 74% of pain episodes last less than a second to two minutes. Furthermore, a series of paroxysms lasting up to an hour might occasionally occur in up to 70% of patients, which can lead to diagnostic uncertainty. It's critical to rule out other neuralgiform illnesses in people who experience attacks that last longer than two minutes but exhibit symptoms typical of trigeminal neuralgia. Even within a single patient, the frequency of attacks varies greatly, ranging from a few to hundreds each day; approximately 40% of patients report experiencing more than ten attacks every day. It can be difficult to get a solid history of the frequency and length of attacks in the transient painful disorder known as trigeminal neuralgia. Approximately two-thirds of people with trigeminal neuralgia experience relapses and remissions, while the other third experience persistent symptoms. Remission periods range greatly in frequency and length, lasting months in 37% of cases or years in 63% of cases (Zakrzewska, 2021).

Various triggers can trigger trigeminal neuralgia pain, including:

- a. Shave
- b. Touching the face
- c. Eat
- d. Drink
- e. Brushing teeth
- f. Speak
- g. Wearing makeup
- h. Facing the wind
- i. Smile
- j. Wash your face

Van Kleef (2008) proposed six questions that patients with suspected trigeminal neuralgia, particularly the atypical variety, should be asked.

- a. Does the discomfort happen during an attack?
- b. Are the majority of attacks brief, lasting only a few seconds to several minutes?
- c. Do you occasionally have brief outbursts of anger?

- d. Is attack unilateral?
- e. Did the attacks in the trigeminal nerve's innervation area happen simultaneously?
- f. Is there a history of trauma and are there unilateral autonomic symptoms?

Only the final question received a "no" response from the patient, whereas the first five questions had "yes" responses. The diagnosis of atypical trigeminal neuralgia is supported by Hata et al, where five questions have "yes" answers and one last question has "no" answers. From here, an MRI of the head may or may not be used to diagnose atypical trigeminal neuralgia.

Complete pain relief with a manageable amount of adverse effects should be the aim of treatment for those experiencing this severe pain. The patient needs to be free from both discomfort and the worry that the illness will return. The preferred first line of treatment is medical therapy. With great success rates, surgical therapy alternatives are available when medical treatment fails or is constrained by severe side effects. A patient's medical comorbidities, level of risk tolerance, and, to a lesser extent, age all influence the optimum surgical course of action for that patient (Zakrzewska, 2021).

According to Cheshire, there are numerous treatments available for trigeminal neuralgia. Traditional analgesics are inferior to antiepileptic medications, with carbamazepine being the preferred medication. Other drugs that have been proven to work include phenytoin, gabapentin, lamotrigine, baclofen, and oxcarbazepine. Many patients, nevertheless, eventually develop tachyphylaxis or are unable to withstand adequate dosages. Microvascular decompression, balloon compression, glycerol rhizotomy or RF thermocoagulation, and subcutaneous alcohol branch blockage are examples of surgical procedures. The next option is stereotactic gamma knife radiosurgery. Despite originally displaying encouraging results for trigeminal neuropathic pain, motor cortex stimulation and transcranial magnetic stimulation don't seem to work well for typical trigeminal neuralgia (Lambru, 2021).

Pulsed Radiofrequency

The components of radiofrequency ablation are electrodes and a generator. Generators often have two terminals. Using radiofrequency electrodes, the generator generates an output of 150–200 W of high frequency alternating current. The cutting tip of a metallic needle makes up the radiofrequency electrode. Isolators are mostly positioned on the active tip and electrode shaft (Goldberg, 2019).

The ablation zone extends just a few millimeters in front of the electrode tip, and its maximum length and width are roughly equal to the length of the exposed tip. Radio frequency electrodes used in modern technologies range in diameter from 16 to 20 G and 10

to 20 cm. As the length of Additionally, the electrode will make the ablation zone wider and longer. When biological tissue is subjected to RF current, a low energy, high frequency AC current (50–500 kHz), tissue molecules oscillate, creating heat and molecular friction (William, 2019).

The target tissue will coagulate, or sustain a thermal injury, if the current is delivered for a long enough period of time. The existence of an electric current surrounding the needle tip is the mechanism that underlies radiofrequency. Electrical ions migrate as a result of changes in current. Ion movement creates friction, which in turn generates heat around the needle's tip, damaging nerves. When temperatures reach 45°C, cells are harmed (Van Zundert, 2015).

There is now a lot of study being done on PRF's mode of action. Currently, the majority of research demonstrates alterations in synaptic transmission leading to neuromodulatory effects. Because PRF modifies the transmission of pain signals through pathways involving c-Fos, it is commonly believed that the electrofield changes quickly. Prior research has demonstrated that PRF enhances c-Fos expression in the dorsal horn and produces a sustained response for seven days following treatment. Additionally, these findings demonstrate sustained suppression of excitatory C fibers (Hatta, 2019).

Damage to cellular structures, neuronal activation, and changes in gene expression are some of the different ways that PRF works. Since the initial gene response triggers a transcription factor that alters the expression of the activated gene, these changes in gene expression can occur over time and are not just limited to the early stages. Through long-term depression, changes in synaptic field strength, and long-term potentiation, a worldwide decrease in evoked synaptic activity results in a decreased transmission of pain signals through C and A fibers. All things considered, it may lessen the transmission of pain signals (William, 2019).

High concentrations of RF electromagnetic waves and heat bursts from PRF can cause cell malfunction. Cosman showed that neuronal membranes might be altered by electrical waves coming from PRF. Neurobiological research demonstrates that upon exposure to PRF, the rat dorsal horn and cervical dorsal root ganglion (DRG) experience both early and late alterations in cellular activity that are unaffected by temperature. Numerous PRF experimental investigations have shown neural activity (Goldberg, 2019).

According to Erdine's analysis of ultrastructural changes after PRF, exposure to PRF caused comparatively minimal damage to C and A γ fibers as well as changes in mitochondrial morphology, including membrane alterations and disruption and disarray of

the microfilaments and axon. Deeper structures are more severely disrupted as a result of the waves' penetration of the C and A fiber axons' cell membrane. The degree and selectivity of ultrastructural damage are taken into account in deeper electrical wave calculations for each type of fiber. Key adenosine triphosphate-mediated cellular processes and cellular metabolism are disrupted when mitochondria are damaged due to membrane fragility, which affects pain signaling (Erdine, 2007).

Injuries to microtubules and microfilaments can prevent pain signals from being sent. The increase of ATF3, a hallmark of neuropathy and cellular stress, suggests that PRF has biological consequences separate from heat damage. Preprodynorphin, the second messenger RNA produced by the c-fos gene, promotes the synthesis of endorphins, which can regulate analgesia. Additionally, c-fos expression excites and inhibits neurons as well as the medulla's dorsal horn¹³. All things considered, PRF-induced alterations in gene expression might be a reflection of modifications in dorsal horn neuron components that decrease nociception (Hatta, 2019).

Pulsed Radiofrequency in Trigeminal Neuralgia

Carbamazepine is the first treatment choice (drug) before invasive therapy is used. Microvascular decompression may be the first invasive therapy option for younger trigeminal neuralgia sufferers. In the meanwhile, treatment for gasserian ganglion RF is advised for senior individuals (Santoso, 2020).

In this instance, the patient received PRF on the right trigeminal nerve at the V2 V3 level using a C-arm. The procedure lasted eight minutes and had the following parameters: temperature of 42 °C, voltage of 45 volts, pulse width of 20 ms, frequency of 2 Hz. Following the treatment, the patient's quality of life improved substantially, and their pain scores dropped from 8 to 0. This approach is, of course, grounded in earlier studies.

77-92% of patients who have conventional radiofrequency (RF) report a significant improvement in their quality of life and a decrease in their VAS. However, RF is not without its problems, such as facial hematoma, keratitis, hearing loss, masticatory muscle weakness, visual abnormalities, hypoesthesia, and temporal muscle atrophy. Facial hypoesthesia or numbness based on the mechanism of RF thermal cauterization of the sensory branch of the trigeminal nerve is the most likely adverse effect, according to the evaluated studies. An ordered logistic regression model in a study by Wang et al. (2018) found that fascial hyposthesia was linked to the incidence of atypical idiopathic TN (OR = 0.36, 95% CI = 0.18-0.71, p = 0.004) and prior RF procedures on the affected side (odds ratio (OR) = 2.33, 95%, confidence interval (CI) = 1.21-4.48, p = 0.011). Nineteen patients (9.1%) experienced

intra-buccal hematoma, five patients (2.39%) experienced masseter muscle weakness, and seven patients (3.35%) experienced corneal hypoesthesia or weakened corneal reflexes out of 209 patients treated with conventional trigeminal RF, according to Gunduz (2021) (Eskandar, 2023). According to a research by Smulders (2021), traditional RF results in trochlear nerve paresis, which causes diplopia (Smulders, 2021).

PRF therapy was given to this patient based on research conducted by Chua and Willy Halim (2012) showing a significant reduction in VAS (<50%) in patients who underwent PRF on the gasserian ganglion with a voltage of 45 V, 42° C, frequency 4 Hz and pulse width 10 ms in 6 minutes. This research has the weakness that it is only quantitative retrospective and the sample is small. However, a satisfactory output was obtained as seen from the reduction in pain scores with an average of 80% at the 2nd, 4th and 12th months after surgery. There were no complications found after PRF procedures in the sample (Chua, 2012).

PRF was delivered for 6 minutes at 45 V, 10 ms wavelength, 4 Hz frequency, and a cutoff temperature of 42 °C in another study. After a 6-month assessment in a randomized, prospective, double-blind research, it was discovered that the PRF group's pain scores significantly decreased (Erdine, 2017).

Existing research shows statistically significant pain improvement after PRF therapy. Navani *et al.* (2018) showed a continuous 60-70% improvement in pain after 4 months of initial therapy and 5 months after second therapy. Vanelderren *et al.* (2018) reported improvements in pain in around 50% of more than checks in the 2nd and 6th months found improvements in pain in VAS scores, improvements in sleep disturbances, mood disturbances, and disturbances in daily activities (Abd Elsayed, 2018). Meta analysis conducted by Zhang (2022) shows that *Pulse combined Conventional RF* shows better safety and efficacy than *Conventional RF* alone where $p < 0.00001$ is obtained (Zhang, 2022).

Providing PRF therapy to this patient is also supported by research conducted by Laksono Ristiawan *et al.* (2023) which examined the effect of *Pulsed Radiofrequency* on sensory ganglion cells in vitro. Neuronal cells were induced with *N-methyl-D-aspartate* (an excitatory neurotransmitter) which induces pain and given 2 Hz PRF therapy in vitro. It was found that PRF modulates sensory ganglion nerve cells by reducing *phosphorylated extracellular signal regulated kinase* (central sensitization biomarker), reducing Ca^{2+} influx which reduces potential activity of nerve cells, reducing mitochondrial membrane potential so that levels Cytosolic ATP decreases. Decreased nerve ganglion cell activity causes decreased sensitivity to pain (Laksono, 2023).

In contrast to study by Chua (2012) and Erdine (2017), which used a frequency of 4 Hz, this patient received PRF at a frequency of 2 Hz. In vitro research by Laksono Ristiawan (2023) revealed that the frequencies of 2 Hz and 4 Hz in activating nerve ganglion cell modulation did not significantly differ from one another. Compared to 4 Hz PRF, 2 Hz PRF better maintains the physiological characteristics of healthy neurons with the same energy. In therapeutic settings, a PRF frequency of 2 Hz may be used for neuron-targeting interventional therapy (Laksono, 2023).

4. CONCLUSION

Given the intricacy of the treatment and the potential for complications after the procedure, treating Trigeminal Neuralgia remains a challenge for pain interventionists. Patients who are unable to get conservative treatment for trigeminal neuralgia may benefit from PRF, which has a more favorable result in terms of lowering pain scores, has fewer post-operative problems, and preserves the function of the afflicted nerves. For PRF to be beneficial in treating trigeminal neuralgia and other persistent headaches, more samples, longer post-treatment monitoring, and clinical studies employing in vivo techniques are required.

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