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CASE REPORT

Bradycardic Episodes of Trigeminocardiac Reflex During Fluoroscopy-Guided Ganglion Gasseri Radiofrequency Ablation in a Trigeminal Neuralgia Patient: A Case Report

Muhammad Syarif Nurdianto✉[*], Mahmud[*], Djayanti Sari[*], Dhanty Dwita Sari[*]

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Authors' affiliations :
[*]Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Public Health, and Nursing, University of Gadjah Mada, D.I Yogyakarta, Indonesia
Correspondence:
✉syarifnurdianto@gmail.com

ABSTRACT

Background: Trigeminocardiac reflex (TCR) is a known but infrequently reported complication of Ganglion Gasseri interventions, manifesting as profound bradycardia and hemodynamic instability.

Case Illustration: A 55-year-old woman with left-sided trigeminal neuralgia, hypertension, and type 2 diabetes underwent fluoroscopy-guided Ganglion Gasseri block with radiofrequency ablation. At the 25th minute of needle manipulation, her heart rate dropped acutely to <30 bpm, followed by a second episode to 30–35 bpm ten minutes later. Both episodes were successfully managed by immediate cessation of manipulation and deepening of sedation using intravenous midazolam (2 mg) and propofol (20 mg), without the need for atropine. Post-procedural pain scores decreased from 7–8 to 2–3 on the Numeric Rating Scale.

Conclusion: This case highlights the necessity of vigilant hemodynamic monitoring and the potential role of adequate sedation depth in mitigating vagal responses during trigeminal procedures, particularly in patients with pharmacologically altered autonomic tone

Keywords: Ganglion gasseri block; Pain management ; Radiofrequency ablation; Sedation; Trigeminal neuralgia; Trigeminocardiac reflex (TCR).

Medical Faculty of Universitas Sebelas Maret - PERDATIN Solo.

INTRODUCTION

Trigeminal neuralgia is identified by brief episodes of sharp pain in one or more trigeminal nerve divisions. This pain should be triggered by light touch, speaking, chewing, nose cleaning, or wind. Both pain and its triggers must be present for a clear diagnosis. Trigger zones don't always match the pain area; pain may be in one division while the trigger is in another. Commonly, pain in the first division is triggered by zones in the second or third division. Typically, pain episodes alternate with pain-free periods, but 42% of long-term sufferers report residual pain. This combination of continuous pain and trigeminal neuralgia is termed "trigeminal neuropathy." This mixed pain presentation is often refractory to standard pharmacotherapy.[1]

Individuals with trigeminal neuralgia are often ***treated*** with ganglion Gasseri block. It is an interventional pain procedure to treat the chronic pain caused by trigeminal nerve. Patients who undergo ganglion gasseri procedures may experience trigeminocardiac reflex, which is marked by a sudden onset of parasympathetic dysrhythmias. This includes hemodynamic changes, breathing irregularities, and increased stomach motility, as well as episodes of extreme slow heart rate and transient low blood pressure.[2]

The Trigeminocardiac Reflex (TCR) is a recognized brainstem reflex that characterized by the abrupt onset of parasympathetic dysrhythmias, including irregular heart rates, breathing difficulties, and enhanced gastric motility, following the stimulation of the sensory branches of the trigeminal nerve.

A systematic review of 221 patients undergoing neurointerventional procedures found that the incidence of the trigeminocardiac reflex (TCR) was 14.5%. In patients with trigeminal neuralgia, the trigeminocardiac reflex can present as severe bradycardia and temporary hypotension, particularly during procedures involving the ganglion gasseri.[2]

This paper is reporting a patient with left trigeminal neuralgia who had experienced trigeminocardiac reflex in the form of an episode of extreme bradycardia during a ganglion gasseri procedure, its intraoperative management, and the potential influence of pre-existing antihypertensive medications on TCR susceptibility.

CASE ILLUSTRATION

A 55-year-old female admitted to the pain clinic with a primary complaint of mouth pain radiating from the left jaw to the left lip, persisting for about three months. The pain, which is triggered by activities such as eating, drinking, talking, and brushing teeth, is localized to the left jaw. It lasts from a few seconds to minutes, disappears, and then reoccurs. During episodes of intense pain, the patient sometimes bursts into tears. The NRS score was 7-8 at the moment. She has been taking carbamazepine, but the pain returns once the medication's effect wears off. After a thorough evaluation and detailed examination, the patient was diagnosed with left-sided trigeminal neuralgia. Additionally, she has hypertension and type 2 diabetes mellitus, for which she regularly takes medical treatment.

The patient's medical history includes hypertension and type 2 diabetes mellitus, which she manages with irbesartan and metformin. Her medications also included bisoprolol, clonidine, pregabalin, tramadol, amitriptyline, mecobalamin, and ranitidine. She reports no food or medication allergies and does not use alcohol, tobacco, or recreational drugs. Although she uses carbamazepine to alleviate her pain, the relief is short-lived, and the pain returns once the medication's effects wear off. The intensity of the pain often leads to emotional distress, causing her to cry frequently during episodes.

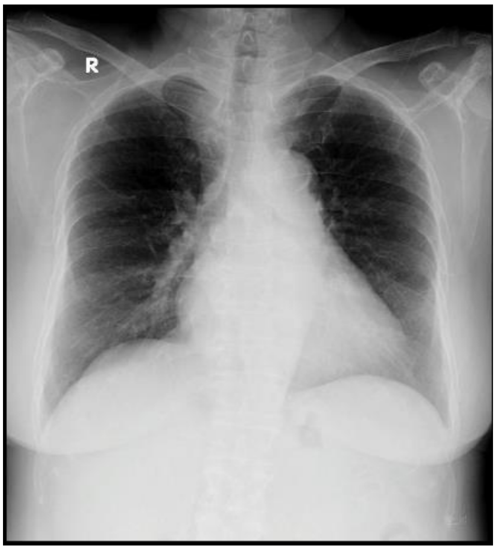

Figure 1. Patient’s Chest X-Ray Examination

A baseline electrocardiogram (ECG) indicated a sinus rhythm with a heart rate of 78 bpm and no abnormalities in chest X-Ray (Figure 1). Complete hematologic test showed that patient in anemic condition with 9.3 in haemoglobin, other laboratory tests including renal function tests, serum electrolytes, and coagulation profile were normal (Table 1). Despite the various medications including carbamazepine, the patient continues to experience significant pain, highlighting the need for further medical evaluation and adjustment of her pain management regiment.

Table 1. Lab results on admission

	Result	Normal range
Leukocyte	8.0	4.50-11.50
Erythrocyte	3.0	4.60-6.00
Haemoglobin	9.3	12.0-15.0
Hematocrit	29.9	40.0-54.0
Platelets	314	150-450
PTT	10.2	9.4-12.5
APTT	27.2	25.1-36.5
INR	0.93	0.90-1.10
BUN	22	5-18
Creatinine	1.26	0.67-1.17
Glucose	198	74-106
Sodium	135	136-145
Potassium	4.6	3.5-5.1
Chloride	103	98-107
HbsAg	NR	NR

BUN = blood urea nitrogen, Lab = laboratory, HBsAg = surface antigen of hepatitis B virus, PTT = partial thromboplastin time; APTT = activated partial thromboplastin time

The patient underwent a comprehensive evaluation after patient admitted to in-patient ward, during which their physical status was determined to be ASA class 2, indicating mild systemic disease. The assessment revealed that the patient had anaemia with a haemoglobin level of 9.3 g/dL, and was also being treated for hypertension (HT) and diabetes mellitus (DM), both of which were under therapeutic management.

Following this thorough assessment, the patient was subsequently scheduled for a pain interventional procedure. This procedure involved radiofrequency ablation; a technique aimed at disrupting nerve function to reduce pain. Additionally, a ganglion gasseri block was to be performed, guided by fluoroscopy to ensure precise targeting and effective treatment. The multidisciplinary medical team carefully planned the procedure, taking into account the patient's overall health status and existing medical conditions. This planning aimed to optimize the outcomes of the radiofrequency ablation and ganglion gasseri block, thereby providing significant pain relief and improving the patient's quality of life. The patient adhered to a preoperative protocol that included an eight-hour fasting period, in preparation for the upcoming procedure, which was precisely planned to involve the administration of sedation.

Subsequently, the patient was comprehensively informed about the details of the interventional procedure, including radiofrequency ablation and ganglion gasseri block, as well as the anesthesia plan. Additionally, general consent was properly obtained. In the operating theatre, standard monitoring equipment, including an electrocardiogram (ECG), non-invasive blood pressure monitor, and pulse oximetry probe, was carefully applied. A 20-gauge intravenous line was successfully inserted to ensure reliable venous access. Prior to the start of the intervention, the patient received sedation with a carefully adjusted dosage of midazolam (3 mg) and fentanyl (25 mcg) to ensure optimal comfort and compliance throughout the procedure.

After an interval of three minutes, the patient reached a state of comfortable sedation, allowing the procedure to be initiated. Oxygen was administered at a rate of 3 litres per minute through a nasal cannula to ensure adequate oxygenation throughout the duration of the procedure.

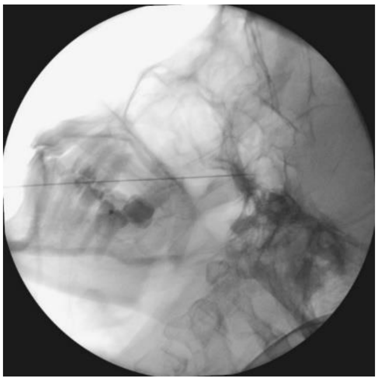

Figure 2. Lateral view of fluoroscopic image showing the needle tip of ganglion gasseri block procedure

The patient's vital signs were closely monitored, and they remained hemodynamically stable initially. This procedure also included a fluoroscopy-guided ganglion Gasseri block to ensure accurate targeting for effective treatment (Figure 2 and 3). However, at the 25th minute, the patient's heart rate suddenly dropped below 30 bpm during the needle insertion targeting the gasserian ganglion. The heart rate ultimately returned to within 20% of the anesthetic baseline upon cessation of manipulation. Once the hemodynamics stabilized, the procedure was continued.

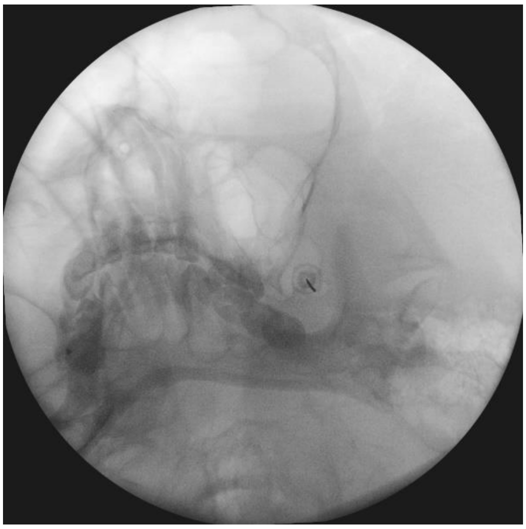

Figure 3. Submental view of fluoroscopic image showing the foramen ovale and the needle tip going toward the foramen ovale

Ten minutes later, the patient's heart rate dropped again to 30-35 bpm. We promptly stopped the procedure and administered an additional 2 mg of Midazolam and 20 mg of Propofol, which effectively restored the heart rate to normal. The patient exhibited a significant manifestation of the trigeminocardiac reflex (TCR), successfully managed by increasing the depth of sedation or anesthesia. Importantly, the drop in the patient's heart rate was not accompanied by hypotension, allowing her blood pressure to remain stable throughout the episode. The intervention was then continued until the procedure was successfully completed.

Within 25–30 minutes, the patient's heart rate and respiration rate stabilized and returned to normal limits, with her blood pressure recorded at 135/80 mmHg. The total duration of the procedure was one hour. Following the procedure, the patient was transferred to the recovery room where she remained completely conscious and oriented. She was then given 4 mg of intravenous ondansetron for postoperative antiemetic treatment. Afterward, she was moved to the inpatient unit for further observation. We evaluated the pain score, and the patient reported a decrease to NRS 2-3. After requesting discharge, the patient was prescribed 500 mg of paracetamol three times a day and 12.5 mg of amitriptyline once daily. She was then sent home in fair condition following a 24-hour observation period in the inpatient unit.

The patient was followed up at the pain clinic three weeks after the procedure. She reported a significant decrease in pain, stating that she no longer felt the pain as she did before the intervention. Due to her low NRS score, the daily pain medications were discontinued. The routine follow-up appointments at the pain clinic were also stopped.

The patient no longer experienced the complaints she had previously reported. She expressed satisfaction with the outcome of the procedure. Overall, the patient's condition had improved markedly since the intervention.

DISCUSSION

Trigeminal neuralgia is characterized by brief, intense episodes of unilateral facial pain, often triggered by specific sensory stimuli like light touch, chewing, or speaking. Patients describe the pain as sharp, stabbing, and similar to an electric shock, localized to one or more divisions of the trigeminal nerve, most commonly the mandibular division. The condition usually appears in otherwise healthy individuals during late middle age, with a higher prevalence in women, though earlier onset may indicate underlying conditions such as multiple sclerosis. According to the International Headache Society, trigeminal neuralgia involves paroxysmal pain lasting from a split second to two minutes, described as intense, sharp, superficial, or stabbing, and is often triggered by specific factors, with patients reporting attacks in a stereotyped manner and no signs of other neurological disorders.[1,3]

For the last three months, this patient has experienced trigeminal neuralgia, a condition characterized by intense, often unbearable pain similar to a sudden gunshot. This severe pain can be triggered by everyday activities such as brushing teeth, eating, shaving, or exposure to temperature changes. The pain episodes are sudden and can be debilitating. The condition significantly affects the patient's daily life and routine. Treatment options were being explored to manage and alleviate the pain.

The exact cause of trigeminal neuralgia pain is uncertain, but it is often linked to compression of the trigeminal nerve root by a blood vessel, typically the superior cerebellar artery, which can cause demyelination of nerve fibers and result in pain. This pain usually affects the maxillary and mandibular branches of the trigeminal nerve, rarely involving the ophthalmic branch; when it does, differential diagnosis is essential to rule out autonomic trigeminal cephalgias. The severe pain can lead to depression in patients, who live in constant fear of the next attack. [2, 3]

This patient experiences brief, recurrent pain episodes lasting seconds to minutes. During severe attacks, the pain is so intense it brings her to tears. The pain is rated 7-8 on the NRS, showing significant discomfort. She also has hypertension and type 2 diabetes, which complicate her trigeminal neuralgia treatment. These conditions might affect her pain perception and the effectiveness of treatments. We were exploring alternative treatments to manage her pain better while also monitoring her other health issues.

Management of trigeminal neuralgia typically starts with antiepileptic drugs such as carbamazepine and gabapentin, with baclofen, a muscle relaxant, also being useful in some cases. If pain is resistant to medication, surgical interventions like selective radiofrequency destruction of trigeminal nerve fibers, transection of the sensory root of the trigeminal nerve, and microsurgical decompression of the trigeminal nerve root may be necessary to reduce or eliminate pain. Long-term management often involves carbamazepine and oxcarbazepine, with alternatives like lamotrigine, gabapentin, botulinum toxin type A, pregabalin, baclofen, and phenytoin if initial treatments become ineffective. Dosages must be adjusted gradually, and doctors should inform patients about neurosurgical options for informed decision-making. About 10% of patients do not respond to medication and may need interventional treatments if no underlying cause is found.[1,3]

The patient's medication regimen includes bisoprolol, clonidine, pregabalin, tramadol, amitriptyline, mecobalamin, and ranitidine. She has no known allergies to food or medication and abstains from alcohol, tobacco, and recreational drugs. Despite taking carbamazepine for pain relief, its effects are temporary, and the pain resurfaces once the drug wears off. The recurring pain episodes are so severe that they cause her significant emotional distress, often resulting in tears. The pain management challenge is further complicated by the need for a comprehensive treatment strategy. Balancing pain control with the management of her other health issues is a priority.

When medical management fails or causes significant side effects, interventional treatments for trigeminal neuralgia are considered. The main procedures include Surgical Microvascular Decompression (MVD), stereotactic radiation therapy (Gamma Knife), percutaneous balloon microcompression, percutaneous glycerol rhizolysis, and percutaneous radiofrequency (RF) treatment of the Gasserian ganglion, all aiming to alleviate pain by addressing nerve compression or disrupting pain pathways. Managing trigeminal neuralgia requires a combination of pharmacological and interventional strategies tailored to each patient’s response and tolerance. Doctors must guide patients through their options, ensuring they understand the potential benefits and risks associated with each treatment, aiming to improve the quality of life for those suffering from this debilitating condition.[3,4]

After a comprehensive evaluation, the patient was scheduled for a pain intervention of gasserian ganglion block involving radiofrequency ablation, a method designed to disrupt nerve function and alleviate pain. This procedure also included a fluoroscopy-guided ganglion Gasseri block to ensure accurate targeting for effective treatment. The multidisciplinary medical team carefully planned the intervention, considering the patient's overall health and existing medical conditions to enhance the procedure's success. The goal was to optimize the outcomes of the radiofrequency ablation of the ganglion Gasseri block, aiming to provide substantial pain relief and improve the patient's quality of life.

Trigeminal cardiac reflex (TCR) is characterized by the sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, apnea, or gastric hypermotility during stimulation of any sensory branches of the trigeminal nerve. A TCR episode should include a decrease in heart rate and mean arterial blood pressure of more than 20% compared to baseline values, coinciding with surgical manipulation around the trigeminal nerve.[5,6]

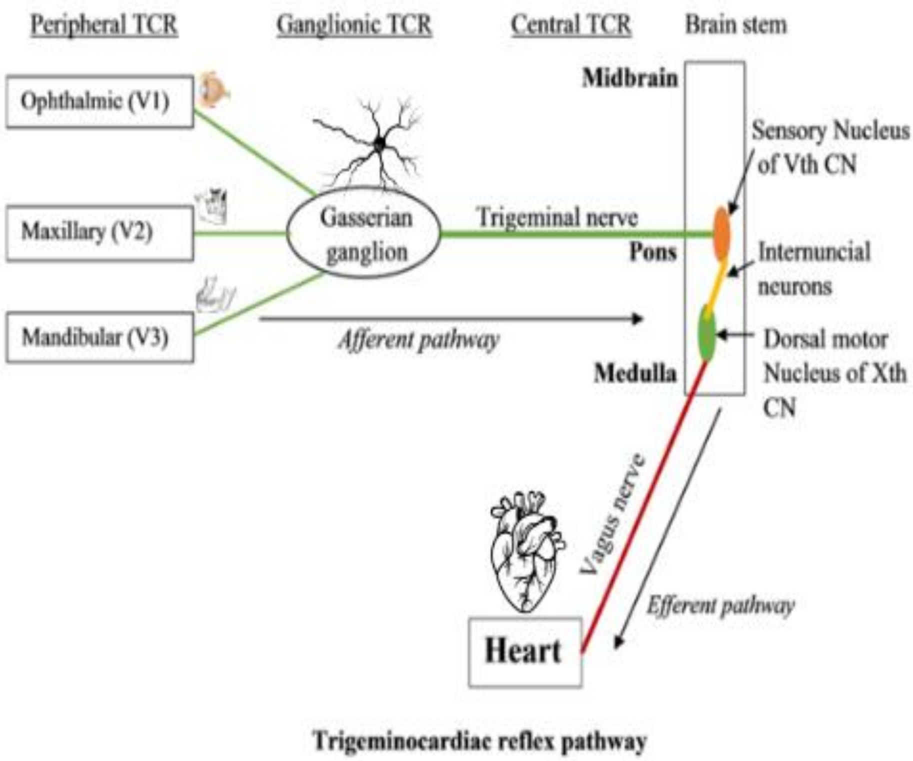

Figure 4. Trigeminocardiac Reflex Pathway.[8]

Peripheral TCR includes the oculocardiac reflex (OCR) and maxilla-mandibular cardiac reflex (MCR), triggered by stimulation of the fifth nerve pathway at or beyond the Gasserian ganglion. Central TCR involves episodes due to stimulation of the trigeminal nerve pathway beyond the Gasserian ganglion. These distinctions help in understanding the different reflex arcs and specific pathways involved in each type of TCR, aiding in effective diagnosis and management of the condition [Figure 4].[6,8]

The trigeminocardiac reflex (TCR) is activated by physical or chemical stimulation of the nerve endings in the branches of the fifth cranial nerve. Sensory fibers transmit the afferent signal through the Gasserian ganglion to the sensory nucleus of the trigeminal nerve, located on the floor of the fourth ventricle. This signal then travels via small internuncial fibers in the reticular formation, linking the sensory nucleus of cranial nerve V with the efferent premotor neurons found mainly in the nucleus ambiguus and the dorsal motor nucleus of the vagus nerve. The activation of cardioinhibitory parasympathetic vagal neurons results in the clinical symptoms of TCR [Figure 5].[7]

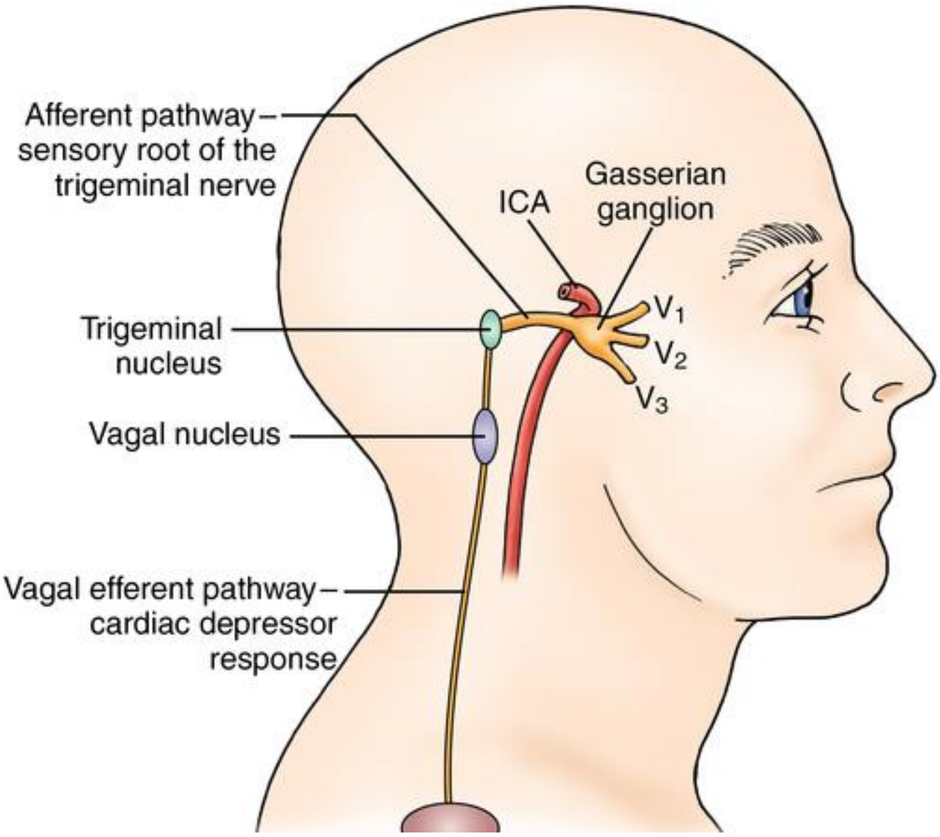

Figure 5: The schematic view of reflex pathway of the trigeminocardiac reflex[7]

Clinically, TCR has gained enormous attention and importance due to its potentially life-threatening complications which may include sudden onset of severe bradycardia culminating in asystole, asystole without preceding bradycardia, or apnea.[6]

This patient experienced a sudden drop of heart rate to below 30 bpm during the needle insertion aimed at the Gasserian ganglion. The heart rate eventually returned to within 20% of the anesthetic baseline once the manipulation stopped. After the hemodynamics were stabilized, the procedure was able to proceed.

Trigeminocardiac reflex (TCR) has been classified into various subtypes, including central, peripheral, and ganglionic TCR. The central TCR is triggered by the stimulation of the intracranial part of the trigeminal nerve proximal to the Gasserian ganglion. On the other hand, the peripheral TCR is triggered by the stimulation of the ophthalmic, maxillary, or mandibular branches of the trigeminal nerve. This distinction helps in understanding the different pathways and mechanisms involved in the reflex, aiding in accurate diagnosis and appropriate management of the condition.[8,9]

This patient is classified under ganglionic TCR. This type of TCR is triggered by the stimulation of the Gasserian ganglion due to the intervention. Unlike centrally stimulated TCR, which typically presents as bradycardia and hypotension due to the activation of a cardioinhibitory vagal response, ganglionic TCR can manifest in a variety of forms. Clinically, it may present as either an increase or decrease in heart rate, showing bradycardia or tachycardia. Additionally, blood pressure can vary, resulting in either hypotension or hypertension. These diverse presentations arise from the simultaneous activation of both the parasympathetic and sympathetic nervous systems. The dual activation leads to complex cardiovascular responses. Consequently, managing ganglionic TCR requires a thorough understanding of these physiological mechanisms to develop appropriate treatment strategies.[9]

Stimulation of trigeminal nerve leads to consecutive reflex bradycardia, hypotension, apnea, and gastric hypermotility, commonly known as the trigeminocardiac reflex (TCR). This reflex is most often transient, but sometimes may be pronounced and sustained, particularly, in infants. The diving reflex (DR) (a subtype of TCR) is triggered as a result of stimulation of one of the sensory branches of the trigeminal nerve and leads to inhibition of cardiorespiratory center, thereby causes bradycardia and apnea.[10] In managing TCR, the initial and crucial step involves identifying bradycardia and immediately halting the procedure to remove the stimulus. This intervention typically reverses the phenomenon in most cases.

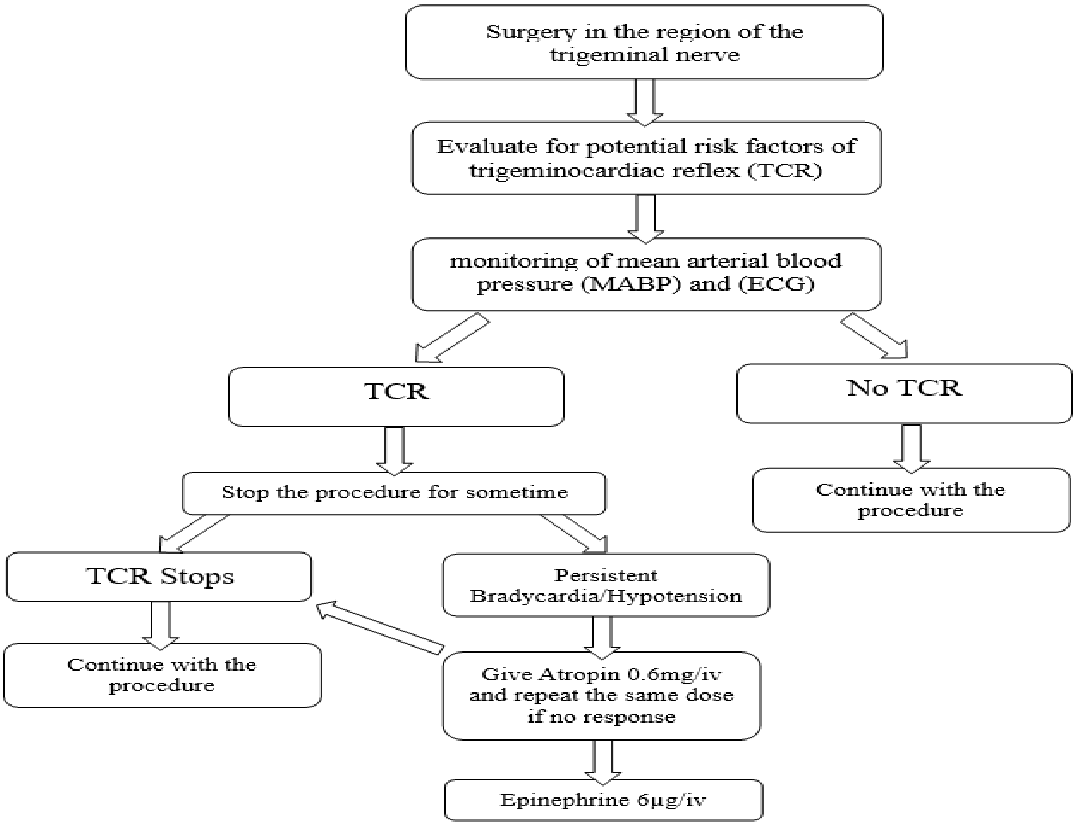

Figure 6. Algorithm to treat the patient with trigeminocardiac reflex[11]

However, in instances where bradycardia is severe or persists even after stopping the stimulus, the administration of anticholinergics like atropine or glycopyrrolate may be necessary to stabilize the patient's heart rate. The autonomic nervous system's response can vary depending on the type of sedation used. Conscious sedation with midazolam and deep sedation with a combination of midazolam and propofol affect autonomic tone differently. In this specific patient, the profound bradycardia was not accompanied by hypotension, suggesting a pure cardioinhibitory reflex with preserved sympathetic vasomotor tone. Given the transient nature of the bradycardia upon stimulus withdrawal and the patient's pre-existing hypertension, we opted to deepen sedation to increase central GABAergic inhibition rather than administer atropine, which could precipitate undesirable rebound tachycardia. The use of both midazolam and propofol can shift the sympathovagal balance towards sympathetic predominance, potentially blunting the vagal reflex during procedures like the ganglion Gasseri block.[11, 12, 13] This highlights the distinct neuromodulatory effects observed during radiofrequency applications.

The American Society of Anesthesiologists classifies sedation into four levels: minimal, moderate, deep, and general anesthesia. Minimal sedation allows the patient to stay awake and responsive to verbal prompts and is typically induced with oral medications or nitrous oxide. Moderate sedation, also known as "conscious" sedation, makes the patient feel sleepy and they may drift off but can be roused by verbal or physical stimulation, often using intravenous drugs. Deep sedation keeps the patient asleep during the procedure, but they can be woken by painful stimuli. General anesthesia renders the patient completely unconscious and unresponsive even to pain. There is evidence that moderate sedation reduces the risk of a vasovagal reaction.[13]

In our cases, we observed a negative alteration in vitals exceeding 20%, consistent with the expected stimulation of the trigeminal nerve during procedures like the ganglion Gasseri block. Fortunately, the condition reversed once the stimulus was removed, and no additional management was required during the first episode. In the second episode, we administered an additional 2 mg of midazolam and 20 mg of propofol, which effectively restored the heart rate to normal levels.

CONCLUSION

This case illustrates the clinical complexity of managing trigeminocardiac reflex (TCR) during interventional pain procedures. The patient, with trigeminal neuralgia and a history of hypertension and diabetes, experienced severe bradycardia during a ganglion Gasseri block. Immediate cessation of the procedure and additional sedation were required to stabilize the heart rate. This emphasizes the importance of careful monitoring and rapid intervention to manage sudden cardiovascular responses effectively.

The successful management of TCR in this case demonstrates the importance of understanding the reflex's physiological mechanisms and the impact of various sedation protocols on autonomic nervous system responses. Rapid identification and management of TCR episodes ensured the safety and efficacy of the pain management intervention, resulting in significant pain relief and an improved quality of life for the patient. This case also highlights the critical role of adequate sedation during the procedure to prevent vasovagal episodes and underscores the necessity of a collaborative approach and comprehensive pre-procedural planning, particularly for patients with complex medical histories, to optimize outcomes in interventional pain management.

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