e-ISSN (online): 2745-9497

Journal of Anesthesiology and Clinical Research (JACR)

Journal website: https://hmpublisher.com/index.php/jacr

A Rare Complication of Vasovagal Syncope Induced by Pulsed Radiofrequency in a Patient with Cervical Spondylosis and Occipital Neuralgia: A Case Report

Fajar Ristranda[1*], Buyung Hartiyo Laksono[2], Taufiq Agus Siswagama[2]

[1]Specialized Residency Training Program, Anesthesiology and Intensive Therapy Study Program, Faculty of Medicine, Universitas Brawijaya/Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia

[2]Consultant of Pain Management Division, Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya/Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia

ARTICLE INFO

Keywords:
Cervical radiculopathy
Neuromodulation
Occipital neuralgia
Pulsed radiofrequency
Vasovagal syncope

***Corresponding author:**
Fajar Ristranda

E-mail address:
Fajar.ristranda@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/jacr.v7i1.869

A B S T R A C T

Introduction: Pulsed radiofrequency is widely utilized as a minimally invasive neuromodulation technique for managing chronic neuropathic pain, including cervical radicular pain and occipital neuralgia. While pulsed radiofrequency is generally celebrated for its robust safety profile and absence of thermal tissue destruction, unexpected autonomic complications remain poorly characterized in the literature. **Case presentation:** A 41-year-old female with a six-month history of chronic cervical root syndrome (C3-C6) and refractory occipital neuralgia presented for interventional pain management. Following a comprehensive clinical and radiological evaluation, the patient underwent fluoroscopy-guided pulsed radiofrequency of the bilateral C3 and C4 dorsal root ganglia and the greater and lesser occipital nerves. The procedure was technically successful and uneventful. However, approximately 24 hours post-procedure, the patient experienced a sudden, profound episode of vasovagal syncope, characterized by acute hypotension, bradycardia, and a precipitous drop in consciousness (Glasgow Coma Scale: E3V3M6). Immediate resuscitation, including intravenous fluid boluses and continuous hemodynamic monitoring, led to a full neurological recovery. At follow-up, the patient reported significant attenuation of both radicular and occipital pain scores. **Conclusion:** This report documents a rare and severe episode of delayed vasovagal syncope following upper cervical and occipital pulsed radiofrequency neuromodulation. The temporal association suggests a complex neuro-autonomic reflex, potentially mediated by the trigeminocervical complex and sudden withdrawal of chronic sympathetic tone. Clinicians performing cervical pulsed radiofrequency must remain vigilant regarding delayed autonomic dysregulation, necessitating extended postoperative observation protocols in susceptible individuals.

1. Introduction

Pain is an intrinsically universal human experience serving as a fundamental biological warning mechanism; yet, when it transitions from an acute physiological response into a persistent, pathological state, it transforms into one of the most pervasive and debilitating complaints encountered in outpatient clinical settings worldwide.[1] Chronic pain conditions, formally defined within medical literature as persistent or recurrent pain lasting for a duration longer than three consecutive months, inflict a profound toll on the global healthcare infrastructure and function as a primary driver for medical utilization, lost workplace productivity, and long-term disability. The deleterious

impact of such conditions extends far beyond mere localized physical discomfort. Chronic pain initiates a complex cascade of maladaptive neuroplastic changes within the central nervous system, known as central sensitization, which significantly impairs the cognitive function, emotional stability, sleep architecture, and overall social well-being of afflicted individuals. Epidemiological reviews and sweeping meta-analyses consistently highlight a concerning therapeutic gap in current clinical practice, suggesting that up to fifty percent of patients suffering from chronic pain do not achieve optimal, sustained analgesic management through standard conservative pharmacotherapy. This inadequacy underscores the pressing clinical imperative for the development and utilization of advanced, targeted interventional therapeutic modalities.[2]

Among the myriad and diverse presentations of chronic neuropathic pain, chronic radicular cervical pain stands out as particularly prevalent, recalcitrant, and challenging to manage.[3] With an estimated epidemiological incidence of approximately eighty-three cases per one hundred thousand individuals, this specific condition represents a major health burden. The underlying pathophysiology is classically driven by the mechanical compression, chemical irritation, or intense inflammatory sensitization of the delicate cervical nerve roots or their corresponding dorsal root ganglia. This anatomical compromise clinically manifests as cervical root syndrome. Patients suffering from this syndrome typically endure a distressing constellation of symptoms, including sharp, shooting pain, severe paresthesia, sensory deficits, and varying degrees of motor weakness extending along highly specific dermatomal and myotomal distributions of the upper extremities and shoulder girdle. Furthermore, the clinical picture of cervical root syndrome is frequently complicated by the presence of comorbid regional pain disorders, most notably occipital neuralgia. Occipital neuralgia is a severely debilitating neurological condition characterized by paroxysmal, piercing, and electric shock-like pain originating in the suboccipital region. This intense pain radiates unilaterally or bilaterally upwards and forwards throughout the specific dermatomal territories of the greater occipital nerve and the lesser occipital nerve. The convergence of cervical radiculopathy and occipital neuralgia creates a highly complex, overlapping pain matrix that is notoriously resistant to conventional oral analgesics, physical therapy, and targeted rehabilitative exercises.[4]

When conventional, non-invasive modalities fail to provide adequate symptomatic relief and functional restoration, interventional pain management techniques become an indispensable component of the treatment algorithm. Over the past two decades, the precise application of pulsed radiofrequency has emerged as a cornerstone therapy for refractory neuropathic pain syndromes.[5] Pulsed radiofrequency represents a monumental paradigm shift from traditional ablative neurological procedures. It is a highly sophisticated, non-destructive, and minimally invasive neuromodulation technique that delivers short, high-voltage bursts of high-frequency electrical fields directly to targeted peripheral nerves and neural tissues, which are immediately followed by relatively long, silent resting phases. This unique pulsatile delivery mechanism allows for the rapid and efficient dissipation of thermal energy into the surrounding tissues, thereby ensuring that the targeted neural tissue temperatures strictly remain within a safe, physiological range, typically not exceeding forty-two degrees Celsius to forty-five degrees Celsius. By rigorously maintaining this sub-lethal temperature threshold, the procedure effectively prevents the Wallerian degeneration, permanent structural nerve damage, and indiscriminate thermal ablation that are the functional hallmarks of conventional continuous radiofrequency treatments.[6]

Instead of permanently destroying the nerve fiber to halt the transmission of pain signals, the intense electrical fields generated during the pulsed procedure fundamentally alter the synaptic signaling pathways and the microenvironment of the nerve. The precise analgesic efficacy of pulsed radiofrequency is hypothesized to stem from a complex, multi-tiered cascade of neuromodulatory and immunomodulatory mechanisms acting at both the peripheral nociceptor and the spinal cord levels.[7] Current neurobiological research indicates that these high-intensity electric

fields induce immediate and profound alterations in cellular gene expression. Specifically, the electrical stimulation downregulates the expression of c-Fos in the dorsal horn of the spinal cord, which serves as a critical biological marker of active nociceptive pathways and central sensitization. Additionally, the intervention significantly modulates the local biochemical environment by decreasing the synaptic release of excitatory neurotransmitters, such as glutamate and aspartate, while simultaneously upregulating endogenous inhibitory neural pathways. Beyond the direct modulation of neurotransmitters, the electrical bursts exert a profound and sustained anti-inflammatory effect by actively inhibiting microglial overactivity and drastically reducing the local concentration of pro-inflammatory cytokines around the chemically irritated neural structures. Furthermore, the intermittent, rhythmic nature of the electrical stimulation is widely believed to promote the induction of long-term depression within the spinal synaptic pathways, effectively resetting and quieting the hyperactive, aberrant pain circuits that strictly characterize chronic neuropathic conditions.[8]

The clinical utilization of this advanced neuromodulatory technique for the treatment of both cervical root syndrome and occipital neuralgia has consistently demonstrated robust and sustained improvements in patient-reported pain scores, daily functional capacity, and overall quality of life metrics. Numerous clinical trials and extensive observational studies continually highlight the procedure's high therapeutic efficacy, paired seamlessly with an exceptionally favorable safety profile and a strikingly low incidence of severe, systemic, or permanent complications. When adverse effects are reported in the clinical literature, they are almost exclusively mild, highly localized, and transient in nature, presenting typically as localized hyperesthesia, temporary exacerbation of baseline pain at the needle insertion site, or mild procedural site edema. The distinct absence of motor deficits, severe deafferentation pain, or significant permanent sensory loss makes pulsed radiofrequency a highly attractive and preferred option for interventions involving the anatomically dense cervical spine and cranial nerves.[9]

However, the application of high-voltage electrical fields in the superior cervical region necessitates a profound and cautious understanding of the intricate regional neuroanatomy. The upper cervical spinal nerves, specifically those originating from the C1 through C3 levels, and their associated sensory dorsal root ganglia, share a highly complex anatomical, structural, and functional relationship with the trigeminocervical complex and the central autonomic nervous system pathways. Afferent sensory nociceptive fibers from the upper cervical roots converge extensively with the descending spinal tract of the trigeminal nerve within the upper segments of the cervical spinal cord. This vital convergence creates a dense neural highway where nociceptive, proprioceptive, and neuromodulatory signals can easily cross-communicate with adjacent critical brainstem structures. Furthermore, these specific neural pathways possess intimate, direct neuroanatomical connections with primary autonomic regulatory centers, including the nucleus tractus solitarius located deep within the medulla oblongata, which serves as the principal integration site for systemic cardiovascular and visceral autonomic control. Consequently, the application of localized, intense electrical stimulation in this highly sensitive and connected anatomical neighborhood presents a compelling theoretical risk for profound neuro-autonomic dysregulation.

Vasovagal syncope, a specific and potent form of reflex syncope, represents a prime and dangerous clinical example of such sudden autonomic dysregulation. It is classically triggered by transient, global cerebral hypoperfusion secondary to a sudden, systemic failure of the autonomic nervous system to maintain adequate vascular tone and sufficient cardiac output.[10] The complex physiological cascade underlying a vasovagal event involves an initial precipitating trigger—most commonly acute visceral pain, severe emotional distress, extreme orthostatic stress, or sudden, uncompensated shifts in background autonomic tone—that rapidly stimulates specific afferent neural pathways. This sudden overstimulation paradoxically triggers an aggressive, largely uncompensated surge in parasympathetic efferent activity traveling via the vagus nerve, coupled directly

with a simultaneous, precipitous withdrawal of peripheral sympathetic vascular tone. The overt clinical manifestation of this autonomic imbalance is a life-threatening triad of profound cardioinhibition, manifested as severe symptomatic bradycardia or even transient asystole, alongside marked vasodepression, leading directly to profound systemic hypotension and subsequent immediate loss of consciousness.

While vasovagal syncope is a relatively common occurrence in the general population, frequently manifesting during minor medical procedures, venous blood draws, or instances of acute minor trauma, its presentation as a delayed, severe, and acute medical complication following interventional neuromodulation is exceptionally rare. In the specific context of cervical and occipital nerve interventions, the occurrence of such a severe, unprovoked autonomic collapse occurring hours or even days after the initial electrical stimulus is virtually absent from contemporary, peer-reviewed pain medicine literature. The highly unexpected nature of a delayed, severe autonomic crisis poses a significant diagnostic and therapeutic challenge for managing clinicians, highlighting a critical gap in the current medical understanding of the broader systemic side effects and autonomic reflexes associated with targeted peripheral nerve modulation.

Therefore, the fundamental aim of this study is to report and analyze a highly unusual clinical case of acute, severe vasovagal syncope that presented with a sudden, precipitous drop in the patient's level of consciousness twenty-four hours following the successful application of pulsed radiofrequency to the cervical dorsal root ganglia and occipital nerves. By systematically detailing the patient's preoperative state, the precise interventional parameters utilized, and the subsequent clinical trajectory, and by deeply investigating the potential neurophysiological links between upper cervical neuromodulation, the trigeminocervical complex, and sudden autonomic cardiovascular collapse, this case report aims to provide a highly novel and vital addition to the existing medical literature. The novelty of this work lies in its documentation of a delayed, severe systemic reflex to a localized, non-ablative therapy, thereby expanding the known safety profile of this widely utilized intervention and strongly underscoring the critical necessity for enhanced postoperative observation requirements, extended hemodynamic monitoring, and refined clinical risk stratification protocols to ensure the absolute optimization of patient safety in interventional pain medicine.

2. Case Presentation

Ethical consideration

This case report was written in strict accordance with the ethical principles established by the Declaration of Helsinki for medical research involving human subjects. Prior to the initiation of the therapeutic intervention and the subsequent drafting of this manuscript, comprehensive written informed consent was obtained directly from the patient. The patient received a detailed explanation regarding the proposed interventional procedures, including all potential risks and expected benefits. Furthermore, the patient explicitly consented to the publication of her anonymized clinical data, radiological imaging, and procedural documentation for academic and scientific purposes. The patient was assured that all personally identifiable information would be entirely redacted to guarantee strict confidentiality and preserve anonymity throughout the publication process. Formal approval from the institutional review board was waived due to the retrospective, single-case nature of this observational report, which strictly documents standard clinical care without the introduction of novel experimental protocols.

Patient history and clinical evaluation

A 41-year-old female patient presented to the interventional pain management clinic with a primary complaint of severe, intractable neck and head pain lasting for six months. The pain was described as a persistent, throbbing ache exacerbated by cervical movement, radiating from the posterior neck to the bilateral shoulders and the occipital region. The patient reported a baseline Numeric Rating Scale pain score of 3-4 at rest, escalating to 5-6 during exacerbations (Table 1a).

In addition to the radicular symptoms, she detailed a history of intermittent cluster-like headaches

originating in November 2023. These headaches were described as severe, unilateral right-sided periorbital pain lasting approximately three hours, occurring monthly, and accompanied by ipsilateral conjunctival injection, lacrimation, and nasal congestion. Her medical history was notable for chronic spontaneous vertigo and right-sided tinnitus diagnosed in May 2024 with associated mild hearing loss. She was recently diagnosed with primary hypertension in October 2024. She had no history of diabetes mellitus, structural cardiac disease, or prior surgical interventions. Her current pharmacological regimen included betahistine three times daily, diclofenac sodium twice daily, amitriptyline 25 mg, diazepam 5 mg, and amlodipine 10 mg daily.

Table 1a. General Assessment and Physical Examination

PARAMETER	CLINICAL FINDINGS
GENERAL ASSESSMENT & ANTHROPOMETRY	
General Appearance	Moderate distress
Glasgow Coma Scale (GCS)	15 (E4V5M6)
Vital Signs	**Blood Pressure:** 163/80 mmHg **Heart Rate:** 80 beats per minute **Respiratory Rate:** 16 breaths per minute **Oxygen Saturation:** 98% on room air **Temperature:** 36.5 Celsius
Anthropometry	**Weight:** 50 kg | **Height:** 155 cm **BMI:** 22.9 (Normal weight)
SYSTEMIC PHYSICAL EXAMINATION	
Head and Neck	Pale conjunctiva (-), icteric sclera (-), erythema (-), edema (-)
Cardiopulmonary	Symmetrical expansion. Vesicular breath sounds, rhonchi (-), wheezing (-). S1/S2 single and regular, murmurs (-), gallops (-)
Abdomen & Extremities	Abdomen supple, normal bowel sounds. Extremities warm, dry, red; capillary refill time < 2 seconds; cyanosis (-), edema (-)

Physical and neurological examination

On admission, the patient appeared in moderate distress. Vital signs revealed a blood pressure of 163/80 mmHg, heart rate of 80 beats per minute, respiratory rate of 16 breaths per minute, and a peripheral capillary oxygen saturation of 98% on room air. Her body mass index was normal at 22.9 kilograms per square meter. A comprehensive neurological examination was performed. The patient was fully alert with a Glasgow Coma Scale of 15 (E4V5M6). Cranial nerve testing confirmed right-sided tinnitus and a mild hearing deficit, but was otherwise unremarkable. Motor strength was 5/5 in all upper extremity myotomes (C5-T1), though testing elicited marked pain. Deep tendon reflexes were symmetric and normal (+2), with absent pathological reflexes. Provocative cervical testing was highly positive for Lhermitte's sign and Spurling's test bilaterally, indicating cervical nerve root irritation. Marked pericranial tenderness was noted upon palpation of the suboccipital musculature (Table 1b).

Table 1b. Neurological Examination

PARAMETER	CLINICAL FINDINGS
CENTRAL AND PERIPHERAL NERVOUS SYSTEM	
Meningeal Signs	Nuchal rigidity (-), Kernig (-), Brudzinski I-IV (-)
Cranial Nerves (I-XII)	**CN VIII:** Right-sided tinnitus (+), mild right-sided hearing loss (+). **Other Cranial Nerves:** Intact and within normal limits. Pupil light reflex (+/+), eye movement normal, jaw reflex normal, gag reflex (+).
Motor System	Normal muscle tone. Power 5/5 in bilateral C5-T1 myotomes (accompanied by pain during testing). Involuntary movements (-).
Sensory System	Within normal limits. No hyperalgesia or allodynia noted in trigeminal distribution.
REFLEXES AND PROVOCATIVE TESTS	
Physiological Reflexes	Biceps (+2), Triceps (+2), Patellar (+2), Achilles (+2) bilaterally.
Pathological Reflexes	Hoffman (-), Tromner (-), Babinski (-), Chaddock (-), Oppenheim (-), Gordon (-), Schaffer (-) bilaterally.
Provocative Cervical Tests	**Lhermitte's Sign:** Positive bilaterally (+/+) **Spurling's Test:** Positive bilaterally (+/+) **Pericranial Tenderness:** Positive (+) **Valsalva Maneuver:** Negative (-)

Imaging and diagnosis

Magnetic Resonance Imaging of the cervical spine, conducted on November 4th, 2024, revealed multilevel degenerative changes. Notable findings included severe spinal canal stenosis at the C5-C6 level and mild stenosis at C4-C5 secondary to disc bulging. Mild bilateral foraminal stenosis was confirmed at C5-C6. Endplate and disc degeneration were classified as Modic Type II and Pfirrmann Grade III, respectively. Based on the clinical and radiological data, the formal diagnoses established were: (1) Chronic radicular cervical pain secondary to cervical herniated nucleus pulposus; (2) Primary tension-type headache with pericranial tenderness; (3) History of cluster headaches; and (4) Chronic spontaneous vertigo.

Interventional procedure

Given the refractory nature of her symptoms to conservative pharmacotherapy, the patient was scheduled for interventional pulsed radiofrequency targeting the lesser and greater occipital nerves, as well as the C3 and C4 dorsal root ganglia. The procedure was performed in the operating theater on November 19th, 2024. Standard monitoring, including electrocardiography, non-invasive blood pressure, and pulse oximetry, was applied by the attending anesthesiologist. Mild conscious sedation was achieved using intravenous propofol 0.5 mg/kg and midazolam 0.01 mg/kg to ensure patient comfort while maintaining the ability to communicate for sensory testing.

Following sterile preparation and local infiltration with 2% lidocaine, a 22-gauge, 10-cm radiofrequency cannula with a 10-mm active tip was advanced under continuous fluoroscopic guidance toward the C3 and C4 neural foramina. Proper positioning near the dorsal root ganglia was confirmed radiologically. Electrical impedance was registered between 320 and 410 ohms, confirming an appropriate tissue interface. Sensory stimulation at 50 Hz elicited concordant paresthesia in the corresponding dermatomes at low voltage thresholds between 0.4 and 0.6 V, without motor

fasciculations at 2.0 Hz up to 1.5 V, confirming optimal proximity to the sensory ganglia and a safe distance from the anterior motor root.

Subsequently, 1 mL of 1% lidocaine was injected to provide local anesthesia. Pulsed radiofrequency was then delivered for 120 seconds using a pulse frequency of 2 Hz, a pulse width of 20 milliseconds, and a peak voltage of 45 V. The generator's auto-temperature control strictly limited the electrode tip temperature to a maximum of 42 degrees Celsius. This protocol was successfully repeated for the bilateral greater and lesser occipital nerves under ultrasound and fluoroscopic guidance. The patient tolerated the procedure well, remaining hemodynamically stable throughout.

Clinical course and complication

The patient was transferred to the inpatient ward for standard 24-hour observation. The immediate postoperative course was uneventful. However, on November 20th, 2024, approximately 24 hours post-procedure, the patient experienced a sudden and dramatic clinical deterioration. At 16:00, the patient suffered an acute decrease in the level of consciousness, with her Glasgow Coma Scale rapidly dropping to 12 (E3V3M6). This neurological decline was accompanied by profound autonomic collapse. Her blood pressure plummeted to 75/40 mmHg, and continuous telemetry revealed marked sinus bradycardia dropping to 42 beats per minute. The patient exhibited classic prodromal signs immediately prior to the event, including severe diaphoresis, pallor, and nausea. A clinical diagnosis of severe, acute vasovagal syncope with marked cardioinhibitory and vasodepressor components was established.

Immediate resuscitative measures were instituted. The patient was placed in the Trendelenburg position. Intravenous fluid resuscitation with isotonic crystalloids was administered rapidly. Due to the persistent symptomatic bradycardia, a single dose of intravenous atropine 0.5 mg was administered, which successfully restored the heart rate to 78 beats per minute and stabilized the blood pressure at 110/70 mmHg. Following hemodynamic stabilization, the patient's neurological status gradually improved. By 23:00, her Glasgow Coma Scale improved to 14 (E3V4M6), and by the following morning, she had fully regained her baseline consciousness.

A comprehensive post-procedural evaluation revealed substantial clinical success regarding the primary indications. The chronic radicular cervical pain and occipital tightness diminished significantly from a Numeric Rating Scale of 5-6 to 4-5, with the character of the pain transitioning from sharp radicular shooting to a dull, non-radicular localized soreness at the injection sites. Notably, provocative testing converted to negative bilaterally, and her vertigo and tinnitus demonstrated subjective improvement.

3. Discussion

This manuscript details a highly unusual and clinically profound case of acute, severe vasovagal syncope resulting in a significant, albeit transient, drop in consciousness occurring exactly twenty-four hours following pulsed radiofrequency neuromodulation of the cervical dorsal root ganglia and the occipital nerves. While the application of pulsed radiofrequency is widely heralded across interventional pain medicine as a remarkably safe therapeutic modality with an inherently low risk profile, this specific case highlights a critical, poorly understood, and potentially life-threatening systemic autonomic vulnerability. The unexpected nature of a delayed autonomic crisis following a localized, non-ablative intervention challenges the current understanding of the systemic effects of neuromodulation and necessitates a rigorous reevaluation of post-procedural monitoring protocols for high-risk patients.

To understand the severity of the complication observed in this patient, it is essential to first delineate the complex physiological mechanisms underlying vasovagal syncope. Vasovagal syncope represents a profound failure of the autonomic cardiovascular regulatory system, ultimately resulting in transient global cerebral hypoperfusion and subsequent loss of consciousness.[11] The pathophysiology is typically initiated by a specific trigger—often acute somatic or visceral pain, severe emotional distress, or sudden shifts in orthostatic pooling—that vigorously stimulates afferent neural pathways (Figure 1).

Table 2. Diagnosis, Treatment, Follow-up, and Outcome

CLINICAL PHASE	DETAILED DESCRIPTION
PRE-PROCEDURAL DIAGNOSES	
Primary & Secondary Diagnoses	**Diagnosis 1** Chronic radicular cervical pain secondary to Herniated Nucleus Pulposus (HNP) Cervicalis **Diagnosis 2** Primary Cephalgia secondary to Tension-Type Headache (TTH) with Pericranial Tenderness **Diagnosis 3** History of Primary Cephalgia secondary to Cluster Headache **Diagnosis 4** Chronic Spontaneous Vertigo secondary to suspected Meniere's Disease
INTERVENTIONAL TREATMENT (NOVEMBER 19, 2024)	
Procedure Targets	**Target 1** Pulsed Radiofrequency of bilateral Lesser and Greater Occipital Nerves **Target 2** Pulsed Radiofrequency of Cervical Dorsal Root Ganglia (C3 and C4)
Neuromodulation Parameters	**Duration:** 120 seconds per target **Frequency:** 2 Hz **Pulse Width:** 20 milliseconds **Voltage:** 45 V **Temperature Control:** Maximum 42 Celsius
FOLLOW-UP & ACUTE COMPLICATIONS (NOVEMBER 20, 2024)	
Adverse Event (16:00)	**Critical Event** Acute decrease in consciousness secondary to suspected severe Vasovagal Syncope. **Hemodynamic Collapse:** Blood pressure dropped to 75/40 mmHg; profound sinus bradycardia at 42 beats per minute. **Neurological Decline:** Glasgow Coma Scale dropped to 12 (E3V3M6).
Resuscitation & Recovery (16:00 - 23:00)	**Intervention:** Trendelenburg positioning, rapid intravenous isotonic crystalloids, and intravenous atropine 0.5 mg. **Trajectory:** GCS gradually improved from 12 (E3V3M6) to 13 (E3V4M6), reaching baseline 15 (E4V5M6) post-resuscitation. Heart rate stabilized at 78 beats per minute.
CLINICAL OUTCOMES POST-PROCEDURE	
Pain Scores & Symptoms	**Improved** **Cervical Radicular Pain:** Decreased from pre-procedural NRS 5-6 to post-procedural NRS 4-5. Pain character shifted from sharp/radicular to localized dull soreness. **Improved** **Paresthesia/Hypoesthesia:** Subjective improvement noted in C3-C6 dermatomes. **Improved** **Headaches:** Cluster headache symptoms absent. Intermittent tension headache stable at NRS 4-5. **Improved** **Vestibular Symptoms:** Subjective improvement in both vertigo and tinnitus.
Provocative Testing	**Lhermitte's Sign:** Converted to negative bilaterally (-/-) **Spurling's Test:** Remained positive bilaterally (+/+) **Pericranial Tenderness:** Remained positive (+)

These stimulated afferent fibers project directly to the nucleus tractus solitarii located deep within the medulla oblongata. The nucleus tractus solitarii serves as the central relay station and primary integration center for systemic cardiovascular and visceral autonomic control.[12] Under normal physiological

conditions, the nucleus tractus solitarii elegantly balances sympathetic and parasympathetic outflow to maintain hemodynamic stability. However, during a vasovagal event, a failure in compensatory mechanisms occurs. The central nervous system paradoxically orchestrates an abrupt and massive surge in parasympathetic efferent activity traveling via the vagus nerve, which is simultaneously coupled with a sudden, precipitous withdrawal of peripheral sympathetic vascular tone. This catastrophic autonomic dysregulation leads directly to the classic, life-threatening triad of severe cardioinhibition, manifesting as profound sinus bradycardia or transient asystole, alongside marked vasodepression, which manifests as severe systemic hypotension.[13]

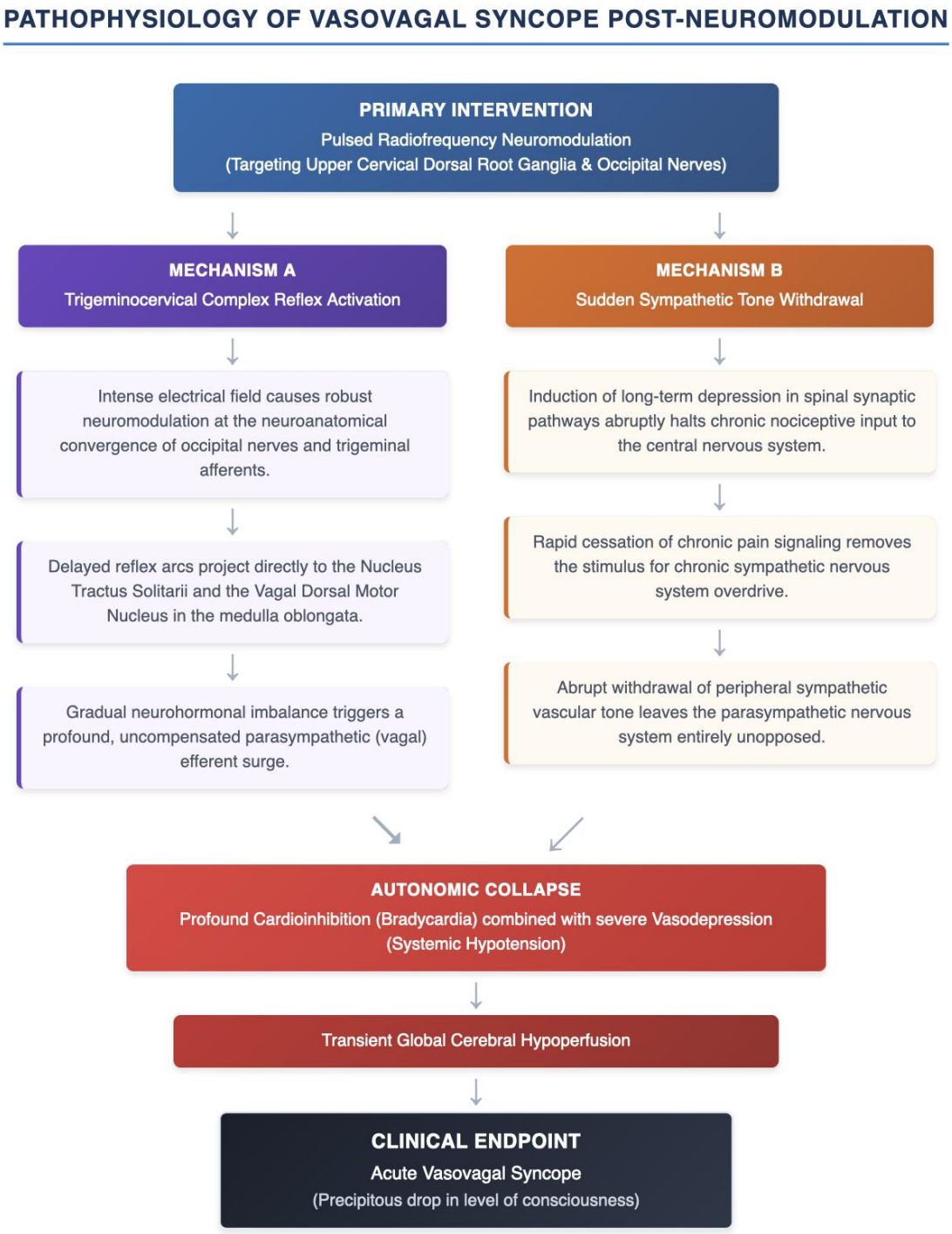

Figure 1. Pathophysiology of vasovagal syncope post-neuromodulation.

The application of high-voltage pulsed radiofrequency fields in the upper cervical region and the occipital nerve distribution uniquely positions this interventional procedure at the precise anatomical crossroads of the central autonomic network. The primary mechanism by which pulsed radiofrequency induces targeted analgesia involves the intense, localized modulation of synaptic transmission.[14] Specifically, the electrical bursts facilitate the downregulation of excitatory nociceptive neurotransmitters, such as glutamate and aspartate, while simultaneously driving the upregulation of inhibitory neurotransmitters, including gamma-aminobutyric acid. While these neuromodulatory effects are highly desired for pain relief, the intense electrical stimulation occurs in a region densely packed with autonomic pathways. Based on the temporal latency and the severity of the hemodynamic collapse observed in this patient, we hypothesize two primary, potentially overlapping physiological mechanisms responsible for the delayed vasovagal syncope.[15]

The first proposed mechanism centers on the intricate neuroanatomical convergence known as the trigeminocervical complex. The greater and lesser occipital nerves, which arise from the dorsal rami of the upper cervical spinal nerves, share direct and extensive neuroanatomical connections with the ophthalmic division of the trigeminal nerve within the upper segments of the cervical spinal cord and the lower brainstem. The robust, high-frequency electrical neuromodulation of these specific pathways via pulsed radiofrequency may trigger delayed, aberrant reflex arcs. These localized sensory and neuromodulatory signals can easily cross-communicate with adjacent critical brainstem structures, specifically sending continuous afferent traffic to the vagal dorsal motor nucleus and the nucleus tractus solitarii. A defining characteristic of this case is the twenty-four-hour latency period between the successful completion of the procedure and the onset of the syncopal event.[16] This significant delay strongly suggests a neurohormonal or inflammatory-mediated pathophysiological shift, rather than an acute electrical artifact or immediate reflex to the needle insertion. This delayed presentation aligns perfectly with contemporary physiological theories proposing that some forms of vasovagal syncope are delayed due to the gradual, insidious buildup and subsequent systemic imbalance of endogenous vasoactive hormones. The intervention may have initiated a slow cascade of neuroendocrine alterations that eventually breached the patient's autonomic threshold, culminating in sudden cardiovascular collapse.[17]

The second, and perhaps most compelling, hypothesis involves the systemic autonomic adaptations to chronic pain and the sudden physiological consequences of its abrupt alleviation. Chronic pain states, such as the severe cervical radiculopathy and occipital neuralgia experienced by this patient, force the body to maintain a state of chronic, relentless sympathetic overdrive. Over months or years, the cardiovascular system adapts to highly elevated circulating levels of endogenous catecholamines and increased baseline sympathetic vascular tone.

Pulsed radiofrequency is a highly efficacious intervention precisely because it effectively and abruptly blocks nociceptive input via the induction of long-term depression at the spinal synaptic level.[18] However, this sudden, highly successful cessation of chronic pain signaling removes the primary driving force behind the patient's elevated sympathetic baseline. We hypothesize that this intervention precipitated a rapid, uncompensated withdrawal of sympathetic tone. By abruptly silencing the nociceptive pathways, the parasympathetic nervous system was suddenly left unopposed. This rapid neurochemical shift effectively lowered the physiological threshold for the Bezold-Jarisch reflex—a potent cardioinhibitory reflex that responds to an underfilled ventricle or an imbalance in autonomic tone. Once triggered, this reflex leads directly to profound systemic vasodilation, severe symptomatic bradycardia, and the subsequent syncopal event observed in the ward.

The standard, long-term management of vasovagal syncope remains largely conservative. It relies heavily on patient education regarding the recognition of prodromal symptoms—such as diaphoresis, pallor, and nausea—to initiate physical counter-maneuvers like isometric muscle tensing. It also requires maintaining

adequate systemic volume by increasing fluid intake to at least 2.5 liters per day and optimizing dietary salt intake. However, in this specific acute clinical scenario, the sheer severity of the hypotensive and bradycardic shock rendered conservative measures entirely insufficient. The precipitous drop in the Glasgow Coma Scale necessitated immediate, aggressive pharmacological and physical intervention. The prompt administration of intravenous atropine was critical to reverse the severe vagally mediated cardioinhibition, while aggressive isotonic fluid loading and Trendelenburg positioning were required to restore central venous return and cerebral perfusion pressures.[19]

Furthermore, a rigorous analysis of this case reveals that the patient possessed several intersecting, synergistic risk factors that likely predisposed her to this catastrophic event. Most notably, she was recently diagnosed with primary hypertension and had been initiated on Amlodipine, a potent dihydropyridine calcium channel blocker. Amlodipine exerts its antihypertensive effects through significant peripheral arterial vasodilation. The combination of newly prescribed systemic vasodilatory pharmacotherapy, the inherent physiological stress and anxiety associated with a spinal intervention, and the profound, neuromodulatory-induced shifts in cervical sympathetic tone created a perfect physiological storm. These factors undoubtedly synergized to overwhelm the patient's baroreceptor compensatory mechanisms, directly precipitating the severe syncopal event.

While this manuscript provides a thorough analysis of a rare complication, the study is inherently limited by its retrospective, single-case observational design. Because the syncopal event occurred completely unexpectedly twenty-four hours after the procedure, the clinical team lacked continuous, minute-to-minute pre-syncopal neurohormonal data. The absence of targeted catecholamine assays, continuous invasive arterial blood pressure monitoring, or formal tilt-table testing diagnostics prior to the syncopal episode prevents the definitive confirmation of the exact pathophysiological mechanism.[20] Future research within the field of interventional pain medicine must urgently address this knowledge gap. Prospective, multi-center observational trials are heavily warranted. These trials should focus on monitoring subtle autonomic tone variability in patients undergoing upper cervical and cranial nerve pulsed radiofrequency. The implementation of continuous heart rate variability indices in the perioperative period could serve as a non-invasive, highly sensitive biomarker for impending autonomic dysregulation. Determining the true incidence of subclinical vasovagal episodes or asymptomatic bradycardic events post-intervention will dictate whether extended inpatient telemetry should become the standard of care. Furthermore, clinical guidelines may need to be updated to include specific medication adjustments, such as the temporary cessation of peripheral vasodilators, for high-risk patients prior to cervical neuromodulation.

4. Conclusion

In conclusion, pulsed radiofrequency remains an overwhelmingly safe, non-destructive, and highly effective neuromodulatory intervention for the long-term management of chronic radicular cervical pain and severe occipital neuralgia. Its ability to provide profound analgesia without the tissue destruction associated with conventional thermal ablation solidifies its role as a cornerstone therapy in modern pain management. However, this highly unusual case serves as a vital clinical demonstration that severe, delayed vasovagal syncope is a rare but highly critical complication that can occur in the postoperative period. The complex, highly interconnected physiological interplay between upper cervical electrical neuromodulation, the sensitive trigeminocervical complex, and the sudden systemic cessation of chronic sympathetic pain responses likely mediates this potent reflex.

Pain physicians and interventional anesthesiologists must maintain a high index of clinical suspicion when treating this anatomical region. It is imperative to ensure thorough post-procedural hemodynamic monitoring, extending observation periods for patients exhibiting any signs of autonomic lability. Most importantly, clinicians must preoperatively assess all patients for underlying dysautonomia, states of chronic dehydration, or the concurrent use of vasodilatory

pharmacological regimens. By recognizing these synergistic risk factors and implementing proactive preventative strategies, practitioners can effectively mitigate the risks of catastrophic syncopal events and ensure the continued safety and efficacy of targeted neuromodulatory therapies.

5. References

1. Akhlaghdoust M, Shakeri M, Faraji M, Khanmirzaei A. Central nervous system neuromodulation techniques for management of phantom limb pain: a review of clinical trials. Interv Pain Med Neuromod. 2023; In Press(In Press).
2. Lan X, Wang Z, Huang Y, Ni Y, He Y, Wang X, et al. Clinical and radiological comparisons of percutaneous low-power laser discectomy and low-temperature plasma radiofrequency ablation for cervical radiculopathy: a prospective, multicenter, cohort study. Front Surg. 2021; 8: 779480.
3. Yildiz G, Perdecioglu GRG, Akkaya OT, Can E, Yuruk D. Comparison of selective nerve root pulsed radiofrequency vs paramedian interlaminar epidural steroid injection for the treatment of painful cervical radiculopathy. Pain Physician. 2024; 27(2): E221–9.
4. Handa K, Kanno H, Hoshikawa T, Murotani M, Ozawa H. Diaphragmatic paralysis due to cervical spondylotic radiculopathy: a case report and literature review. Eur Spine J. 2025.
5. Kwon K, Park S, Song MG, Park WS, Hwang CJ, Cho JH, et al. Is motor weakness in cervical radiculopathy an indication for surgery? Analysis of risk factors for poor recovery. Eur Spine J. 2025.
6. Baldelli I, Zaottini F, Picasso R, Pistoia F, Hamedani M, Raposio G, et al. Preoperative ultrasound evaluation for decompressive surgery for occipital neuralgia: Preliminary results of a case-control study. J Plast Reconstr Aesthet Surg. 2025; 107: 24–34.
7. Ji Q, Yang Y, Ou C. Short-term stimulation of spinal cord and occipital nerve for subacute postherpetic neuralgia: Report of two cases. Asian J Surg. 2026; 49(3): 1883–5.
8. Ravaillault S, Alipour H, Leger F, Labarre J, Nguyen J-M, Pluchon YM, et al. Occipital nerve stimulation for refractory occipital neuralgia: a multicenter, randomized, controlled trial [StimO study]. J Clin Med. 2026; 15(5): 1922.
9. Hamaguchi S, Shinozaki M. Complications associated with spinal cord stimulation, radiofrequency and pulsed radiofrequency. Masui. 2016; 65(7): 686–92.
10. Dong Q-P, Li S-J. Ultrasound combined with DSA-guided pulsed radiofrequency for perineal herpes zoster pain management: clinical outcomes and complications. Front Med (Lausanne). 2024; 11: 1442199.
11. Zhu M, Mao Y, Zhang Z, Ou C. Dual-target pulsed radiofrequency neuromodulation for trigeminal herpes zoster complicated with Ramsay Hunt syndrome: a case report and literature review. Asian J Surg. 2025.
12. Başarı A, Cansu Ç, Arslan İ, Özgencil GE, Ünal HA, Kılınç H, et al. Sensorimotor cortical integration remains unchanged after dorsal root ganglion pulsed radiofrequency. Pain Med. 2026; 27(3): 262–7.
13. Abed SM, Thabet TS, Ibrahim MA, Gad AF, Elshamy FH, Eltwab DA, et al. Pulsed radiofrequency of S2-4 nerve roots vs ganglion impar neurolysis for severe perineal cancer pain. Pain Rep. 2026; 11(2): e1393.
14. Aksu T, Davila A, Gupta D. The “heart brain” and neuromodulation for vasovagal syncope. Auton Neurosci. 2021; 236(102892): 102892.
15. Khan A, Huang HD, Aksu T. Neuromodulation for vasovagal syncope and bradyarrhythmias. Card Electrophysiol Clin. 2024; 16(3): 297–305.
16. Chen A, Tu B, Lai Z, Maimaitijiang P, Wang X, Zheng L, et al. Deceleration capacity as a predictor of vasovagal syncope subtypes. Auton Neurosci. 2026; 264(103401): 103401.
17. Basunlu MT, Akdeniz C, Sari G, Tuzcu V. Role of midodrine in children with recurrent vasovagal syncope: a retrospective examination of therapeutic outcomes. Cardiol Young. 2026;

1–4.

18. Neymark D, Seifer CM. Vasovagal syncope: distinct features and management strategies in women. Curr Opin Physiol. 2026; 47(100901): 100901.
19. Hou M, Wu J, Ren L, Liming C. Primary cerebral autoregulation dysfunction: a distinct subtype of vasovagal syncope. Acta Neurol Belg. 2026.
20. Tian L, Hou H, Zhang L, Gao Y. Index of cardiac electrophysiological balance in patients with vasovagal syncope confirmed by head-up tilt test. Front Cardiovasc Med. 2026; 13(1743842).