

Hemifacial Spasm After Ultrasound-Guided Left Stellate Ganglion Block for Post-traumatic Stress Disorder (PTSD): A Case Report

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Abstract

Stellate ganglion block (SGB) is an ultrasound-guided cervical sympathetic block used for pain syndromes and adjunctively for post-traumatic stress disorder (PTSD). Hemifacial spasm (HFS) is a unilateral facial movement disorder usually due to neurovascular compression of cranial nerve VII. A 48-year-old woman with chronic migraine, fibromyalgia, and PTSD underwent an uncomplicated left SGB (C7 level, 6 mL 2% lidocaine). Over 2-3 weeks, she developed new left-sided facial spasms including clusters of orbicularis oculi and lower facial muscle contractions. Neurologic workup included magnetic resonance imaging (MRI) of the brain showing no infarct/mass/lesion and routine electrodiagnostic testing, facial nerve conduction, and electromyography (EMG) of trigeminal/facial muscles, all normal. Botulinum toxin was injected into the left orbicularis oculi with partial transient relief. We propose that SGB-induced head/neck vasodilation and altered sympathetic tone may have converted an asymptomatic neurovascular contact into symptomatic HFS, though causality is unproven. Prior reports note SGB can increase cerebral and cranial perfusion and that sympathetic fibers may bridge facial nerve ephaptic transmission. Clinicians should be aware that new neurologic movement symptoms like HFS, though rare, might follow SGB. Thorough reporting (imaging, labs, consent) and multidisciplinary management are indicated.

Categories: Pain Management, Physical Medicine & Rehabilitation, Anesthesiology

Keywords: botulinum toxin, ephaptic transmission, facial nerve, hemifacial spasm, neurologic complication, neurovascular compression, post-traumatic stress disorder, stellate ganglion block, sympathetic nervous system, ultrasound-guided intervention

Introduction

Ultrasound-guided stellate ganglion block (SGB) is widely used to block cervical sympathetic fibers for conditions like complex regional pain syndrome and increasingly as an adjunctive treatment for post-traumatic stress disorder (PTSD) [1,2]. Ultrasound-guided SGB is typically performed at C6, targeting the cervical sympathetic chain, although the stellate ganglion itself is anatomically located at C7-T1 [3]. SGB is thought to exert therapeutic effects through the modulation of sympathetic outflow and autonomic signaling, although the mechanisms underlying its effects in PTSD remain incompletely understood. Reported complications are generally uncommon (e.g., transient Horner's syndrome, hoarseness), with serious adverse events being rare [3,4]. Hemifacial spasm (HFS) is characterized by involuntary unilateral contractions of muscles innervated by the facial nerve [5]. It is most often attributed to vascular compression at the facial nerve root exit zone, leading to ephaptic excitation [5,6]. Ephaptic excitation refers to abnormal electrical cross-talk between adjacent demyelinated nerve fibers, allowing the propagation of action potentials without synaptic transmission and resulting in hyperexcitability and involuntary muscle contractions. Other proposed mechanisms include ectopic impulse generation and hyperexcitability of the facial motor nucleus. Treatment of HFS is primarily symptomatic with botulinum toxin injections, while definitive management involves microvascular decompression in cases with confirmed neurovascular conflict [7]. This report describes a paradoxical new-onset HFS occurring shortly after an SGB, to our knowledge not previously documented, and explores potential mechanisms.

Case Presentation

A 48-year-old right-handed woman (retired Marine with combat-related PTSD) had chronic migraine, fibromyalgia, mild cervical degenerative changes, a history of cervical fusion at C5-C6 and C7-T1, and no prior facial movement disorders. On day 0, she underwent an outpatient procedure for PTSD: ultrasound-guided left SGB at the C7 level. Written consent was obtained prior to any intervention. Under sterile technique, the C7 vertebral body was identified, and the carotid artery and left longus colli muscle were visualized. Skin was anesthetized with 1% lidocaine. A 25-gauge 3.5" needle was advanced from lateral to medial in a transverse plane to lie anterior to the longus colli and posterior to the carotid. After confirming negative aspiration, 6 mL of 2% lidocaine was injected (no epinephrine added). The patient tolerated the procedure well, and her lungs remained clear on exam. The patient was discharged after 30 minutes of

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monitoring (standard post-SGB observation). No immediate complications such as hoarseness of voice or dyspnea were noted.

On the same day, she also received onabotulinumtoxinA injections for chronic migraine (155 units total across 31 sites, including the procerus, corrugators, frontalis, temporalis, occipitalis, cervical paraspinals, and trapezius), an ultrasound-guided left ankle sinus tarsi injection, and thoracic trigger-point injections. Standard post-procedure instructions were provided, including brief rest, resumption of activities and medications the following day, and instructions to report any new neurologic or motor deficits.

The patient reported developing new involuntary twitching of the left facial muscles approximately 2-3 weeks after the procedure, which was first documented at the day 32 follow-up. She described intermittent clusters of spasm, initially affecting the lower left eyelid and nasolabial region, later involving the cheek and mouth. The spasms were more pronounced in the evening, disrupting sleep, and seemed exacerbated by stress (the patient noted PTSD-related hypervigilance). There were no obvious triggers and no new medications except a brief trial of trazodone which she discontinued due to headache, and she later started nightly tizanidine. She had normal facial sensation and no photophobia or aura.

At a follow-up pain clinic visit on day 38, physical examination revealed intermittent twitching of the left lower eyelid, with a clinically estimated frequency of approximately 0.5 Hz. The rest of the neurologic exam, including cranial nerves, was unremarkable. Reflexes, strength, and coordination were normal. Given persistent spasms, a neurology referral was made. On day 64, the neurology exam was non-focal (no spasms observed then), but the clinical impression was "likely left hemifacial spasm". Laboratory studies, including a thyroid panel, antinuclear antibody (ANA), rapid plasma reagin (RPR), folate, and basic metabolic panel (BMP)/comprehensive metabolic panel (CMP), were ordered and ultimately returned within normal limits. Magnetic resonance imaging (MRI) of the brain with and without contrast performed on day 81 revealed no infarct, hemorrhage, mass lesion, or abnormal enhancement. However, dedicated high-resolution sequences (fast imaging employing steady-state acquisition (FIESTA)/constructive interference in steady state (CISS)) and magnetic resonance (MR) angiography were not obtained, limiting assessment for subtle neurovascular compression of the facial nerve. On day 135, electrodiagnostic testing was performed: left upper extremity motor/sensory nerve conduction studies were normal. Facial nerve conduction to the nasalis bilaterally was symmetric. Needle electromyography (EMG) of the left masseter (V) and mentalis (VII) and select limb muscles was normal. The interpreting neurologist concluded a normal electrodiagnostic study.

In the interim, to address symptoms, the patient received botulinum toxin injections to the left orbicularis oculi (total 20 units, divided among four injections) on day 74. She reported a subjective reduction in twitch intensity of approximately 50% lasting about two weeks, although this response was not assessed using a standardized severity scale. At the last documented follow-up, approximately six months after SGB, spasms persisted intermittently; the patient and providers planned continued symptomatic botulinum therapy and consideration of baclofen as needed.

The clinical course is summarized in Table [1](#).

Timepoint	Event/intervention	Details and outcome
~23 months before day 0	Right SGB (for migraine/PTSD)	10 mL 1% lidocaine; no complications reported
~22 months before day 0	Left SGB (for scapular pain, migraines)	5 mL 2% lidocaine + 10 mg dexamethasone; no immediate issues
Day 0	Left SGB (for PTSD) + migraine Botox	Left SGB (6 mL 2% lidocaine at C7) and 155 U OnabotA for migraine; all well
Day 32	Patient-reported symptoms	New left facial twitching (eye/cheek/mouth regions)
Day 38	Pain clinic visit	Exam: intermittent left lower eyelid spasms; advised caffeine/sleep hygiene; started trazodone
Day 64	Neurology consultation	Impression: likely left hemifacial spasm; ordered MRI and labs
Day 74	Eyelid Botox injection	20 U into the left orbicularis oculi; reported ~50% transient relief
Day 81	MRI of the brain w/w/o contrast	No acute lesion; only mild non-specific sinus changes
Day 135	Electrodiagnostic study	L UE NCS normal; facial nerve conduction normal; needle EMG (masseter, mentalis) normal
Ongoing	Follow-up	Continue botulinum toxin as needed; consider baclofen/microvascular decompression

TABLE 1: Timeline of key events

SGB: stellate ganglion block; PTSD: post-traumatic stress disorder; OnabotA: onabotulinumtoxinA; MRI: magnetic resonance imaging; L UE NCS: left upper extremity nerve conduction studies; EMG: electromyography

Discussion

This patient’s presentation was clinically suggestive of HFS, based on patient-reported unilateral periocular and lower facial contractions [5]. Most commonly, HFS affects women in middle age, fitting the profile of this patient [5,6]. MRI did not identify a structural intracranial lesion, and electrodiagnostic testing showed no facial nerve abnormality; however, these findings do not exclude primary HFS or subtle neurovascular conflict. The paradox is that her HFS began only after a left SGB, a sympathetic blockade normally not associated with eliciting such spasms. In fact, prior reports have explored SGB (or pulsed radiofrequency) as a treatment for HFS, not a cause [8]. One Korean case report noted two failed SGB attempts before successful pulsed radiofrequency (RF) to the facial nerve for intractable HFS [8]. To our knowledge, there are no prior reports of new HFS triggered by SGB, making this an unusual adverse event.

What mechanisms could plausibly link SGB to HFS? One potential mechanism involves the vascular effects of cervical sympathetic blockade. SGB has been associated with acute changes in cerebral perfusion-related measures; Park et al. demonstrated increased regional cerebral oxygen saturation on the ipsilateral side following SGB [9]. We hypothesize that in this patient, enhanced pulsatility or caliber of a small arterial loop near the facial nerve root (previously asymptomatic) could have precipitated ephaptic transmission. In other words, SGB-induced vasodilation may have converted a subclinical neurovascular contact into symptomatic compression over time (Figure 1). However, there is no direct evidence that the vascular effects of a single SGB persist for weeks or produce sustained changes in vessels near the facial nerve. Although sympathetic involvement in HFS has been proposed [6,10], prior reports of symptom improvement following sympathetic blockade [8,10] highlight the uncertainty surrounding our proposed mechanism.

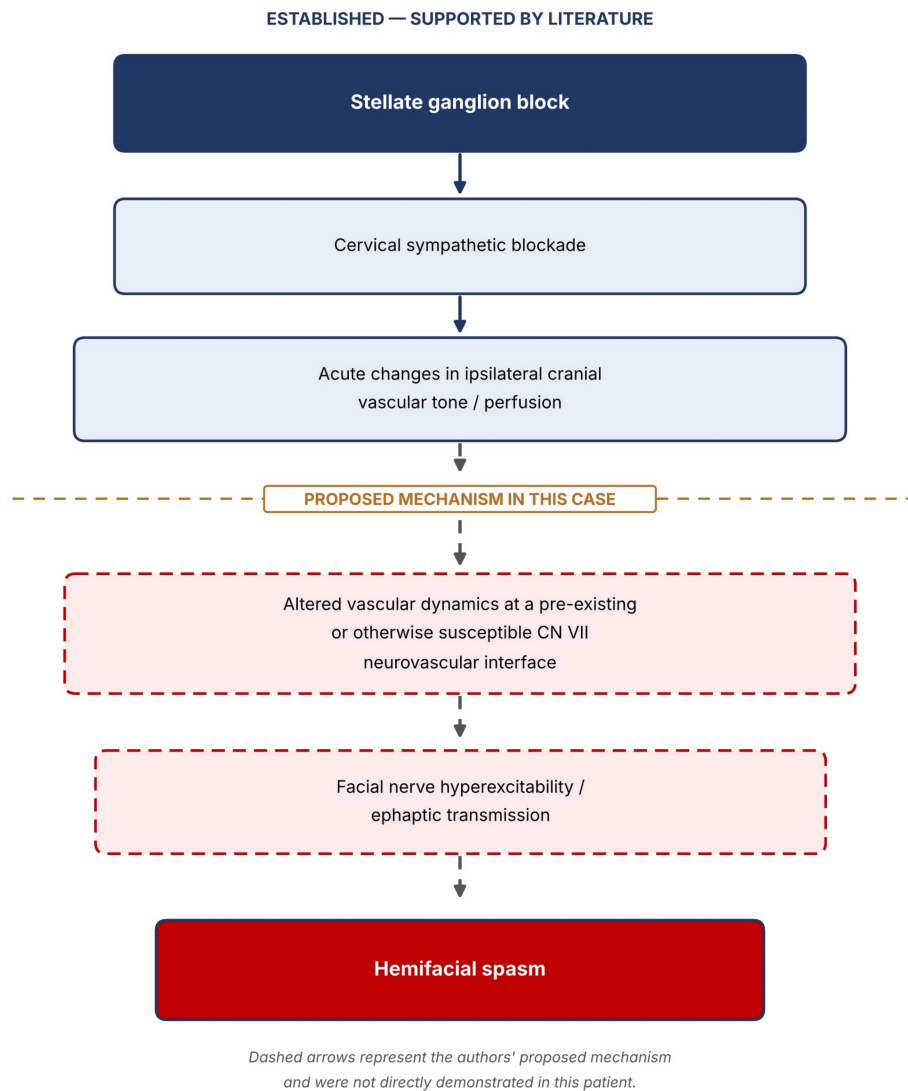


FIGURE 1: Proposed mechanistic hypothesis linking stellate ganglion block to new-onset hemifacial spasm

Solid arrows denote steps supported by prior physiologic literature; dashed arrows denote the authors' proposed, unproven extension of that physiology to this patient.

Alternative explanations should also be considered. A direct local anesthetic effect is unlikely given the delayed symptom onset. Although the patient received onabotulinumtoxinA for chronic migraine on the same day, including injections into muscles adjacent to the orbicularis oculi, these injections followed a migraine protocol and did not directly target the orbicularis oculi. Given the timing of symptom onset, a potential contribution from botulinum toxin cannot be excluded, although it more commonly produces focal weakness than new-onset spasm. Eyelid myokymia related to stress, fatigue, or sleep disturbance was also considered, although the patient-reported progression from periocular twitching to involvement of the cheek and mouth was considered more suggestive of HFS [11]. Other diagnostic considerations include facial myokymia, functional facial spasm, and tics. Finally, coincidental onset of primary HFS cannot be excluded, particularly given the patient's demographic and clinical phenotype. The temporal relationship to SGB therefore suggests an association but does not establish causality.

No definitive evidence confirms the mechanism. Cervical vascular injury was not specifically evaluated with dedicated neck vascular imaging, representing an additional limitation of the diagnostic workup. MRI did not reveal a clear neurovascular conflict; however, standard brain MRI (non-FIESTA sequences) may miss small compressive loops [6]. Further evaluation with high-resolution MRI/MR angiography or dedicated vascular sequences would be necessary to assess the proposed neurovascular mechanism, which remains unconfirmed.

Management-wise, our approach aligns with HFS guidelines: we trialed botulinum toxin injections, which typically achieve significant relief in 73–98.4% of HFS patients [7]. However, the patient's partial and short-lived response was atypical and further limits diagnostic certainty. If spasms remained disabling, surgical microvascular decompression would be considered after confirming a vascular loop.

Limitations

This report represents a single case, and causality between SGB and the onset of HFS cannot be definitively established. The diagnosis of HFS was clinically suspected but not definitively confirmed through direct observation of the broader facial contractions or specialized electrophysiologic testing. Additionally, the patient's previous uncomplicated SGBs, concurrent facial botulinum toxin injections, and lack of evidence for sustained vascular changes following SGB further limit causal interpretation. Objective measures of spasm frequency, severity, and functional impact were not systematically recorded, limiting the quantitative assessment of symptom progression and treatment response. Immediate post-procedural findings, including documentation of expected sympathetic blockade (e.g., Horner's syndrome), were not consistently captured, which may have provided additional insight into the physiologic response to the block. Additionally, MRI did not include high-resolution vascular sequences (e.g., FIESTA/CISS), which may be more sensitive for detecting subtle neurovascular compression. Finally, changes in PTSD symptom severity following SGB were not formally assessed, limiting the interpretation of the broader autonomic and neuropsychiatric response in this patient.

Clinical implications

As SGB use broadens, pain physicians should be alert to new neurologic symptoms and report unusual events. A thorough neurologic evaluation, including appropriate imaging and electrodiagnostic testing, should be considered when new-onset HFS occurs. Key takeaways from this case include the following: (1) HFS developed temporally following SGB in this patient, although causality cannot be established; (2) vigilance for new focal neurologic symptoms following sympathetic blocks is prudent; and (3) multidisciplinary evaluation may be valuable when uncommon neurologic symptoms arise.

Conclusions

We describe a patient who developed left HFS shortly after an ultrasound-guided SGB for PTSD. Although causality cannot be established, this case highlights a potential previously unreported neurologic event temporally associated with SGB. Evaluation did not identify a structural lesion or electrodiagnostic abnormality. Clinicians performing SGB should remain vigilant for unexpected neurologic symptoms following the procedure. Further investigation is needed to determine whether the observed temporal association represents a reproducible phenomenon.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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