PAIN MEDICINE CASE REPORTS

ISSN 2768-5152

Volume 7, Number 7, pp. 341-344

A Series of Stellate Ganglion Blocks for the Management of Post-Traumatic Stress Disorder: A Case Report

Eric Xu, DO[1], Michael Downing, DO[2], and Maria De La Pena, MD[1]

Background: An overactive sympathetic nervous system plays a role in the pathophysiology of post-traumatic stress disorder. The cluster of sympathetic nerves located in the inferior cervical column, known as the stellate ganglion, is an optimal target for anesthetic delivery in an effort to calm this sympathetic overdrive. Recent studies have shown that stellate ganglion blocks may be beneficial for patients with post-traumatic stress disorder (PTSD).

Case Report: The authors present a 40-year-old man who is a military veteran with an existing diagnosis of PTSD. He experiences frequent symptoms such as flashbacks, anxiety attacks, and insomnia that have not improved with standard treatment modalities including antidepressants, anxiolytics, group therapy, and psychotherapy. The patient experienced symptomatic relief immediately after the stellate ganglion block and continued relief after the third block.

Conclusion: Stellate ganglion blocks are an effective alternative therapy for PTSD.

Key words: Stellate ganglion block, post-traumatic stress disorder (PTSD), case study

BACKGROUND

Post-traumatic stress disorder (PTSD) is a psychiatric diagnosis precipitated by traumatic events, such as sexual violence (most common), physical violence, accidents, witnessed death, and combat exposure. In the setting of war, the duration of combat exposure is directly proportional to the risk of an individual developing PTSD (1). PTSD has a prevalence of approximately 6-9% and is more commonly seen in men than women. Risk factors for the development of PTSD include psychiatric comorbidities such as depression, substance use disorders, lack of social support, lower socioeconomic status, and a younger age at the time of the experienced trauma (2).

The pathophysiology of PTSD involves the sympathetic nervous system. Broadly, neurohormonal and neurotransmitter dysregulation likely causes a sympathetic nervous system overdrive that accounts for many of the behavioral and physical symptoms demonstrated. Most common is its association with dissociative symptoms such as flashbacks or intrusion symptoms such as vivid nightmares. However, PTSD can present with many symptoms that overlap with other psychiatric disorders, such as major depressive disorder and generalized anxiety disorder. PTSD can also present with an array of physiologic symptoms such as tachycardia, palpitations, tachypnea, perspiration, and tremors. Specific diagnostic criteria are located within the Diagnostic and Statistical Manual of Mental Illness, or DSM-5 (2).

From: [1]Holy Cross Hospital, Fort Lauderdale, FL; [2]Department of Medicine, Albany Medical Center, Albany, NY

Corresponding Author: Michael Downing, DO, E-mail: Mikedowning1515@gmail.com
Disclaimer: There was no external funding in the preparation of this manuscript.
Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.
Patient consent for publication: Institutional policy excempts the need for consent since all patient information has been deidentified.
Authors adhere to the CARE Guidelines for writing case reports and have provided the CARE Checklist to the journal editor.
Accepted: 2023-08-22, Published: 2023-11-30

Assessment tools such as Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) is utilized in the diagnosis and management of PTSD. The PTSD Checklist for DSM-5 (PCL-5) is also utilized for provisional diagnosis and allows for monitoring of symptom changes before and after treatment (3,4).

The DSM-5 criteria for PTSD pertains to children, adolescents, and adults. Of note, there is different criteria for patients 6 years and younger. The criteria includes specific terms for each of the following umbrella categories: the traumatic exposure, intrusion symptoms that occurred after and associate with the traumatic event, avoidance of stimuli associated with the traumatic event, negatively affected mood and cognition associated with the traumatic event, altered reactivity or arousal associated with the traumatic event, a duration of symptoms greater than one month, impairment of daily living, and the symptoms cannot be explained by other medical conditions or substance use disorders (5).

First-line treatment for PTSD is psychotherapy with or without adjunctive pharmacotherapy. Specifically, trauma-focused psychotherapy that includes exposure therapy (e.g., showing pictures of battlefields to war veterans) and/or cognitive processing therapy has shown to be beneficial in patients with PTSD. In patients who do not have access to or refuse psychotherapy, pharmacotherapy alone is recommended. Selective serotonin reuptake inhibitors (SSRIs) such as sertraline, fluoxetine, paroxetine or selective norepinephrine reuptake inhibitors (SNRIs) such as venlafaxine are mainstay medicinal therapies for patients with PTSD. The alpha-adrenergic receptor antagonist, prazosin, is useful for the treatment of nightmares in PTSD, but should not be used as monotherapy (6).

Fluoroscopic or ultrasound-guided stellate ganglion blocks (SGBs) provide an alternative therapy option for patients with PTSD. The stellate ganglion is a group of sympathetic nerves that result from a fusion of the inferior cervical and first thoracic sympathetic ganglions. Thus, the stellate ganglion is a prime target for medication delivery in an effort to calm the aforementioned sympathetic overdrive in PTSD patients. Promising literature, described later, demonstrates the therapeutic benefits and safety of SGBs in patients with PTSD. However, the recommended amount of SGBs, optimal duration between blocks, duration of therapeutic effect, and the specific PTSD-population (i.e., mild, moderate, severe-PTSD) that would most benefit from this intervention requires further investigation. This case report discusses a 40-year-old man who is a military veteran with treatment-refractory symptomatic PTSD whose condition improved after a series of 3 fluoroscopically guided SGB, each 2-weeks apart.

CASE PRESENTATION

The patient is a 40-year-old man who is a military veteran who was diagnosed with PTSD in 2013 after completing multiple tours in the Middle East. He experiences flashbacks, anxiety attacks, and insomnia daily. As a result, he has difficulty maintaining jobs, personal relationships, and his psychiatric condition. The patient followed with psychiatry, who prescribed him sertraline 50 mg daily and recommended psychotherapy. He enrolled in both psychotherapy and group therapy on the advice of his psychiatrist. Unfortunately, the patient was still experiencing symptoms daily and referred to our clinic for additional management.

After an initial encounter and benign physical examination, a follow-up procedural appointment was made. Each SGB was completed under fluoroscopic guidance and administered on the right side of the C6 transverse process. The injectate for each shot was a combination of 10 mg dexamethasone and 2 mL 1% lidocaine. Each SGB was carried out under fluoroscopic guidance. In the fluoroscopy suite, the patient was placed supine, and an anteroposterior view was initially obtained for identification of C6 vertebral body. The target, Chassaignac's tubercle, is located at the anterior position of the C6 transverse process. Under an oblique view, from a more anteromedial position (medial to the carotid, lateral to the trachea), the needle was guided with a slight lateral to medial trajectory. After contacting the bony target, the needle tip was drawn back from the bone. Positioning was confirmed with new anteroposterior and lateral views. Aspiration followed by an injection of 0.5 mL of contrast was completed for localization. Slow delivery of the injectate combination was then administered (7,8).

After the initial SGB, the patient reported improvement in almost all his daily symptoms. In the first week, he reported feeling less emotionally labile, a 50% reduction in daily anxiety, and being able to fall asleep within the first hour of lying down in bed. Given his initial benefit, the patient requested a second SGB and was scheduled 2-weeks later. After the second injection, he reported improvements such as being able to perform his duties at work without feeling restless and

improved sleep quality. Even with these remarkable improvements, he still reported feeling more generalized anxiety than he would like to live with. Thus, a third and final SGB was scheduled 2-weeks after the second. After the third shot, the patient was seen in the office 2 weeks later and stated he was symptom-free. He was advised to follow-up as needed for additional treatments, but has not returned to our clinic since then. No adverse events were reported from any of the 3 procedures.

DISCUSSION

This case study further expands on the evolving literature of stellate ganglion blocks for the treatment of PTSD. Similar to existing literature, the SGB demonstrated an effective adjunct treatment for our patient's PTSD. Unique to this case study, are the 3-SGBs the patient received 2-weeks apart, as most of the literature demonstrates one to 2 SGBs in the management of PTSD. The patient experienced immediate benefits after each injection, as well as prolonged symptomatic relief after the third and final SGB. This likely reflects acute and chronic therapeutic benefits of multiple SGBs. Supporting this is a published quality assurance project examining 250 SGBs in patients with PTSD. Of 110 surveys to participants, 95% were willing to undergo as many repeat procedures as possible due to its efficacy, safety, and tolerability (9). Thus, this case study warrants further discussion of the pathophysiology of PTSD, mechanism of action of SGB, and review of existing literature in an effort to further the conversation of creating optimal treatment guidelines for the use of SGB in patients with PTSD.

The pathophysiology of PTSD can, in part, be attributed to neurotransmitter and/or neurohormonal dysfunction. This dysfunction results in a sympathetic nervous system overdrive that results in the psychological and physical symptoms described in PTSD. Corticotropin-releasing hormone (CRH) has been found elevated in patients with PTSD. CRH is released from the hypothalamus in response to stress and stimulates the release of adrenocorticotropic hormone (ACTH) from the pituitary. ACTH subsequently targets the adrenal glands for synthesis of cortisol, a key hormone involved with the fight-or-flight sympathetic response. CRH also increases the release of norepinephrine and influences the amygdala, which functions as the fear-response center of the brain and communicates this information with the hippocampus. The amygdala is thought to be hyperactive in patients with PTSD, and its inhibitory control center (located in the prefrontal cortex of the brain) does not adequately regulate the amygdala (2,10).

While the exact mechanism of SGB is unknown, delivery of local anesthetic to the stellate ganglion is theorized to travel throughout the sympathetic chain and reduce or reset sympathetic tone. This sympathetic reset is caused by reduction in both nerve growth factor, a neuropeptide that maintains sympathetic neurons, and norepinephrine (11). It is also speculated that despite peripheral delivery, SGBs have central effects on the hypothalamus, hippocampus, and amygdala (11). Other theories suggest the closely associated vagus nerve is affected by SGBs and plays a part in its therapeutic effects (12). The vagus nerve regulates the parasympathetic nervous system and provides afferent information from the periphery to the nucleus of solitary tract in the medulla oblongota. As such, stimulation of this nerve would promote parasympathetic drive and its communication with the limbic system is hypothesized to reduce fear-related activity in the brain (12). A majority of SGBs are completed targeting the right stellate ganglion rather than the left, though successful results from left-sided SGBs have been reported (13). Psychoneurological models theorize that the right stellate ganglion has a sympathetic predominance with communication to the right insula, perhaps contributing to the predilection for right-sided SGBs. However, both left and right insular subdivisions have shown to exhibit reduced functional connectivity in patients with PTSD, leading to abnormal emotional and somatosensory processing (14).

Multiple case studies and case series have shown the safety and efficacy of SGBs in patients suffering from PTSD. A 2010 case study involving 2 patients with PTSD demonstrated promising effects of SGBs, which inspired a 2014 large multi-site case series involving 166 patients with PTSD who received SGB (15,16). This large case series demonstrated that over 70% of patients experienced symptomatic relief and improvement in PTSD-checklist scores (PCL-5, higher scores indicating greater severity of disease) (16). A similar case series in 2016 examined 30 patients from the same clinical site and displayed a similar reduction in PCL-5 scores, as well as reduction in PTSD-related symptoms such as hyperarousal, insomnia, and avoidance characteristics (17). A randomized control trial published in JAMA in 2019 displayed

statistically significant improvement in the CAPS-5 in the SGB-group (n = 74) vs sham (n = 39). This study administered 2 SGBs at 0 and 2-weeks apart (18). A 2021 non-randomized control trial examined 12 patients undergoing exposure therapy, in addition to a SGB, displayed an average reduction of 32 points in PCL-5 scores (19).

SGB is a safe, effective, and well-tolerated procedure. A 2015 quality assurance assessment of 250 SGBs over the span of 1.5 years demonstrated that there were no immediate or delayed complications per patient reported data (9). Although a safe and routine procedure, SGB is not without risk. Surrounding vasculature such as the carotid, vertebral, subclavian arteries and jugular veins pose risk for vascular puncture, bleed, or injection of anesthetic into the vasculature. The lung pleura remains in close vicinity; thus pneumothorax is a risk. Brachial plexus and radicular nerve injury are also possible. These risks are mitigated by the procedural approach mentioned earlier and targeting C6 rather than the C7 (7).

CONCLUSION

PTSD is difficult to treat given its complicated pathophysiology and the various symptoms that may manifest. The first line treatment for PTSD is psychotherapy with or without pharmacotherapy. Recent studies have demonstrated that SGBs are a safe and effective form of therapy for patients with PTSD. This case report supports existing literature that SGB is an effective alternative treatment in the management of PTSD. Unique to this case study is the frequency of intervention, specifically a series of 3 SGBs each delivered 2-weeks apart. Our patient's immediate relief after each block, in addition to the sustained benefits after the third block, demonstrates the versatility of SGB for acute and chronic management in patients with PTSD. Despite recent studies, there is no standardization of SGB and its optimal procedural frequency, duration of its effects, and the specific subpopulation of PTSD-patients that may benefit. Further clinical investigation is necessary to cultivate SGB treatment guidelines and its general utility in specific PTSD-populations.

REFERENCES

1. Combat Exposure Scale (CES) - PTSD: National Center for PTSD.www.ptsd.va.gov/professional/assessment/te-measures/ces.asp
2. Vieweg WVR, Julius DA, Fernandez A, Beatty-Brooks M, Hettema JM, Pandurangi AK. Posttraumatic stress disorder: Clinical features, pathophysiology, and treatment. *Am J Med* 2006; 119:383-390.
3. Weather FW, Blake DD, Schnurr PP, Kaloupek DG, Marx BP, Keane TM. The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5). www.ptsd.va.gov/professional/assessment/adult-int/caps.asp
4. Blevins CA, Weathers FW, Davis MT, Witte TK, Domino JL. The posttraumatic stress disorder checklist for DSM-5 (PCL-5): Development and initial psychometric evaluation. *J Traumatic Stress* 2015; 28:489-498.
5. Center for Substance Abuse Treatment (US). Exhibit 1.3-4, DSM-5 Diagnostic Criteria for PTSD. Nih.gov. Published 2014. www.ncbi.nlm.nih.gov/books/NBK207191/box/part1_ch3.box16/
6. VA/DOD. VA/DOD Clinical practice guideline for the management of posttraumatic stress disorder and acute stress disorder: Clinician summary. *Focus (Am Psychiatric Publ)* 2018; 16:430-448.
7. Fluoroscopic guided stellate ganglion block – Technique and overview. https://theprocedureguide.com/fluoroscopic-guided-stellate-ganglion-block-technique-and-overview/
8. Piraccini E, Munakomi S, Chang KV. Stellate ganglion blocks. [Updated 2022 Aug 9]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: www.ncbi.nlm.nih.gov/books/NBK507798/
9. McLean B. Safety and patient acceptability of stellate ganglion blockade as a treatment adjunct for combat-related post-traumatic stress disorder: A quality assurance initiative. *Cureus* 2015; 7:e320.
10. Mann SK, Marwaha R. Posttraumatic stress disorder. [Updated 2022 Feb 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan. Available from: www.ncbi.nlm.nih.gov/books/NBK559129/
11. Lipov E, Kelzenberg B, Rothfeld C, Abdi S. Modulation of NGF by cortisol and the stellate ganglion block – Is this the missing link between memory consolidation and PTSD? *Med Hypotheses* 2012; 79:750-753.
12. Kim SY, Cohen SP, Rodriguez SE, McCabe J, Choi KH. Central effects of stellate ganglion block mediated by the vagus nerve? An alternate hypothesis for treating PTSD. *Med Hypotheses* 2022; 162:110833.
13. Mulvaney SW, Lynch JH, MD, Curtis KE, MS, Ibrahim TS. The successful use of left-sided stellate ganglion block in patients that fail to respond to right-sided stellate ganglion block for the treatment of posttraumatic stress disorder symptoms: A retrospective analysis of 205 patients. *Mil Med* 2022; 187:e826-e829.
14. Zhang Y, Xie B, Chen H, Li M, Guo X, Chen H. Disrupted resting-state insular subregions functional connectivity in post-traumatic stress disorder. *Medicine (Baltimore)* 2016; 95:e4083.
15. Mulvaney SW, McLean B, de Leeuw J. The use of stellate ganglion block in the treatment of panic/anxiety symptoms with combat-related post-traumatic stress disorder; preliminary results of long-term follow-up: A case series. *Pain Practice* 2010; 10:359-365.
16. Mulvaney SW, Lynch JH, Hickey MJ, et al. Stellate ganglion block used to treat symptoms associated with combat-related post-traumatic stress disorder: A case series of 166 patients. *Mil Med* 2014; 179:1133-1140.
17. Lynch JH, Mulvaney SW, Kim EH, de Leeuw JB, Schroeder MJ, Kane SF. Effect of stellate ganglion block on specific symptom clusters for treatment of post-traumatic stress disorder. *Mil Med* 2016; 181:1135-1141.
18. Rae Olmsted KL, Bartoszek M, Mulvaney S, et al. Effect of stellate ganglion block treatment on posttraumatic stress disorder symptoms: A randomized clinical trial. *JAMA Psychiatry* 2020; 77:130-138.
19. Peterson AL, Straud CL, Young-McCaughan S, et al; STRONG STAR Consortium. Combining a stellate ganglion block with prolonged exposure therapy for posttraumatic stress disorder: A nonrandomized clinical trial. *J Trauma Stress* 2022; 35:1801-1809.