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Intrathecal Bupivacaine and Clonidine for Refractory Hyperhidrosis in Spinal Cord Injury

Soun Sheen
University of Rochester, ssheen2017@gmail.com

hemant kalia
InvisionHealth; C.R.I.S.P (Center for Research & Innovation in Spine & Pain), drhemantkalia@gmail.com

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Intrathecal Bupivacaine and Clonidine for Refractory Hyperhidrosis in Spinal Cord Injury

Abstract

Introduction: Autonomic dysreflexia (AD) in spinal cord injury (SCI) can present as hyperhidrosis due to sudomotor dysfunction. There are no standard treatment guidelines for refractory hyperhidrosis due to persistent noxious stimulation.

Case: A 58-year-old female with C7 ASIA-A quadriplegia was admitted to the hospital in 2018 for T7-8 discitis. Discitis acted as a noxious stimulus manifesting clinically as autonomic dysreflexia. On average, she experienced 50-60 sweating episodes every day. She underwent serial stellate ganglion blocks with >75% improvement which provided 9 months of continued relief. Patient transitioned to intrathecal bupivacaine 2.5mg/ml + Clonidine 200mcg/ml with the catheter tip located at C4-5 in 2019. She is maintained on Bupivacaine 0.2mg + Clonidine 20mcg/24hrs.

Results: Patient reports near complete resolution of hyperhidrosis following implantation of intrathecal pump. She is back to her baseline, independent with ADL"s using manual wheelchair.

Conclusion: To our knowledge, this is a first reported case of successful resolution of hyperhidrosis using intrathecal pump. Bupivacaine and clonidine may be a viable treatment option for not only refractory hyperhidrosis, but also AD in SCI patients.

Keywords

spinal cord injury, refractory hyperhidrosis, intrathecal therapy

Conflict of Interest Statement

No relevant conflict of interest.

CASE REPORT

Intrathecal Bupivacaine and Clonidine for Refractory Hyperhidrosis in Spinal Cord Injury

Soun Sheen [a,*], Hemant Kalia [b]

[a] University of Rochester, United States
[b] InvisionHealth, C.R.I.S.P (Center for Research & Innovation in Spine & Pain), United States

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1. Introduction

Hyperhidrosis is a common consequence of spinal cord injury (SCI), affecting approximately 25% of the population.[1,2] Although some aspects of hyperhidrosis in SCI remain unclear, its pathophysiology is thought to largely overlap with autonomic dysreflexia (AD).[3] In fact, hyperhidrosis is often associated with AD.[1,2] Patients with injuries at T6 or higher, above the splanchnic sympathetic outflow, lack descending control of all sympathetic preganglionic neurons.[4] Therefore, a noxious stimulus below the level of injury triggers an exaggerated sympathetic response due to intrinsic hypersensitivity of the receptors below the lesion and lack of descending sympathetic control.[4,5] This exaggerated sympathetic innervation of the eccrine glands results in hyperhidrosis. Although hyperhidrosis commonly presents above the level of injury, it can also present exclusively below the injury or diffusely.[1]

As with other autonomic dysfunctions in SCI, hyperhidrosis episodes are also insidious and self-limiting with resolution of the underlying stimulus. However, some cases can be idiopathic or persistent in the setting of noxious stimuli that are difficult to identify or control.[2] Although many forms of treatments for hyperhidrosis have been reported, there is no standard treatment guideline for patients with SCI. We describe a case of successful use of intrathecal bupivacaine and clonidine for refractory hyperhidrosis in a patient with complete cervical SCI due to pain associated with thoracic discitis.

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* Corresponding author.
E-mail addresses: ssheen2017@gmail.com (S. Sheen), drhemantkalia@gmail.com (H. Kalia).

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2. Case description

A 58-year-old female with a history of C7 ASIA A (American Spinal Injury Association classification) SCI from a traumatic C6−C7 spinal fracture in 1974 and prior autonomic dysreflexia presented to the hospital with profuse sweating episodes without reported antecedent illness, trauma, or other new signs and symptoms. The patient's medical, allergy, and social history were otherwise unremarkable. All vital signs were within normal ranges, and she did not report any pain. The initial lab was notable for white blood cell count of 11.5 $\times$ $10^3/\mu L$. Further imaging work-up revealed T7-T8 discitis and osteomyelitis (Fig. 1). Despite appropriate treatment with antibiotics, the patient continued to experience multiple disabling episodes of profuse sweating. Due to these episodes, the patient was no longer able to self-transfer and perform self-care. She failed multiple medications including propranolol, tizanidine, and clonidine patch. The patient then underwent a stellate ganglion block under ultrasound guidance with a greater than 75% reduction in the intensity and frequency of her symptoms. However, the effects were short lasting, and the patient required multiple repeat injections within 9 months. Targeted drug delivery using intrathecal clonidine and bupivacaine was considered as a long-term chemical sympathectomy option. The patient underwent implantation of the intrathecal catheter with a programmable pump in 2019. The catheter tip was placed at C4−C5 level, above the level of innervation to the stellate ganglion (Fig. 2). The patient noted immediate relief in her symptoms on low dose intrathecal bupivacaine and clonidine. The patient continues to report near complete resolution of her symptoms without needing significant dose adjustments. She is successfully maintained on a simple continuous dose of bupivacaine 0.2 mg/24-hour and clonidine 20 mcg/24-hour.

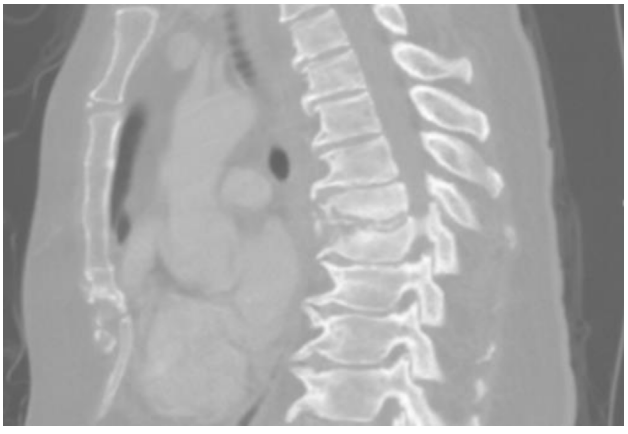

Fig. 1. CT image demonstrating T7-8 discitis and osteomyelitis.

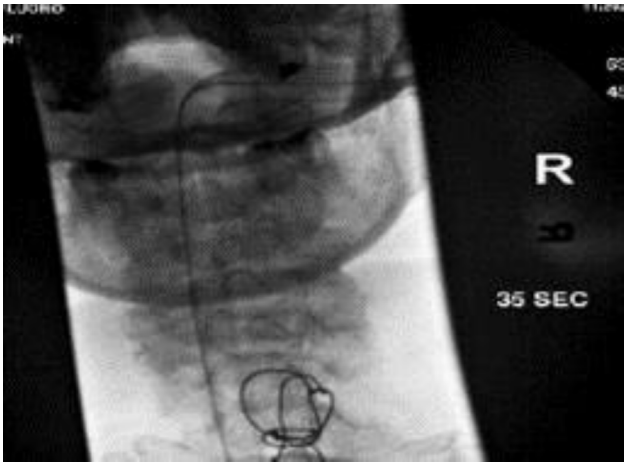

Fig. 2. Fluoroscopic view of the catheter tip placement at C4−C5. Sympathetic blockade at this level was targeted to block innervation to stellate ganglion given patient's response to stellate ganglion blocks.

3. Discussion

As hyperhidrosis in SCI is often self-limiting with the resolution of the underlying stimulus, treatment options are typically focused on the underlying cause. Therefore, there is a lack of standard treatment guideline for hyperhidrosis in patients with SCI. Oral medications to reduce sympathetic tone, including beta blockers and alpha-2 agonists, are often used; however, there is limited literature evidence to support their use in the SCI population. There is also a gap in literature regarding the use of interventional treatment options for hyperhidrosis in the SCI population, especially as it is often considered self-limiting.

This case presents a unique treatment approach to refractory reflexive hyperhidrosis in a complete cervical SCI patient utilizing intrathecal bupivacaine and clonidine. Bupivacaine, an amino-amide local anesthetic agent that prevents cellular depolarization by sodium channel blockade, has been widely used intrathecally for chronic pain management.[6,7] At low doses, bupivacaine has shown to preferentially block small diameter preganglionic sympathetic neurons, reducing discharge activity of splanchnic nerves.[6,8] Intrathecal clonidine, an alpha-2 adrenoreceptor agonist in the dorsal horn, has also been shown to have depressant effect on the sympathetic outflow.[9] Utilizing the sympathetic blockade effects of these two agents allowed for a successful chemical sympathectomy for this patient suffering from a refractory reflexive hyperhidrosis.

The preferential sympathetic effects of intrathecal bupivacaine and clonidine may have further role in

the treatment of other cases of refractory autonomic dysfunctions in SCI patients.

Conflict of interest

None.

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