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A Unique Presentation of Complex Regional Pain Syndrome Type I Treated with a Continuous Sciatic Peripheral Nerve Block and Parenteral Ketamine Infusion: A Case Report

Adam Everett, MD, Brian Mclean, MD, Anthony Plunkett, MD, and Chester Buckenmaier, MD

Walter Reed Army Medical Center—Army Regional Anesthesia & Pain Medicine, Washington, DC, USA

ABSTRACT

Objective. To successfully treat a patient with complex regional pain syndrome, refractory to standard therapy, to enable a rapid and full return to professional duties.

Setting. This case report describes the rapid resolution of an unusual presentation of complex regional pain syndrome type I after four days of treatment with a continuous sciatic peripheral nerve block and a concomitant parenteral ketamine infusion. The patient was initially diagnosed with complex regional pain syndrome (CRPS) I of the right lower extremity following an ankle inversion injury. Oral medication with naproxen and gabapentin, as well as desensitization therapy, failed to provide any relief of her symptoms. She was referred to the interventional pain management clinic. A lumbar sympathetic block failed to provide any relief. The patient was diagnosed with CRPS I and was admitted for treatment with a continuous peripheral nerve block and parenteral ketamine.

Conclusion. This case suggests therapeutic benefit from aggressive treatment of both the peripheral and central components of CRPS.

Key Words. Ketamine; Peripheral Nerve Block; Chronic Pain

Introduction

Complex regional pain syndrome (CRPS) is a disorder that is often challenging to treat and can be associated with a prolonged course of severe pain and psychosocial dysfunction for the patient. Although the diagnostic criteria for CRPS have been both revised and criticized [1,2], the basic features of CRPS include presence of a noxious event or immobilization, pain disproportionate to the injury, vasomotor abnormalities, and absence of other conditions that could account for the pain [2,3]. The disease is more common in young to middle-aged women [4], and often occurs during or after stressful periods [4,5]. It has been suggested that early and aggressive therapy may lead to a better long term outcome [6].

Reprint requests to: Anthony Plunkett, MD, Walter Reed Army Medical Center, Anesthesia and Operative Services, 6900 Georgia Avenue, NW, Washington, DC, WA 20307, USA. Tel: 202-782-2929; Fax: 202-782-5066; E-mail: anthonyplunkett@comcast.net

The pathophysiology of CRPS has not been fully elucidated, however it is postulated that the pain response may be mediated though the peripheral, central, or sympathetic nervous system. Treatment modalities have focused on medical management with opioids, anticonvulsants, corticosteroids, and bisphosponates, as well as interventional management with sympathetic plexus blockade [3,7]. A review of the literature does not reveal a superior therapy [7] as current trials pertaining to the treatment of CRPS have been suboptimal due to lack of blinding, randomization, power, and appropriate controls [5,6]. Both peripheral nerve blockade alone and ketamine alone have been used to successfully treat CRPS in previous studies [8–11], however we have not found any literature describing the combined use of both peripheral nerve blockade and parenteral ketamine for the treatment of CRPS. We describe combining these two modalities to treat the peripheral and central nervous system components theorized to contribute to CRPS in a patient with recent onset of CRPS type I.

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Case Report

A 17-year-old female (American Society of Anesthesiology classification I, 170 cm, 80 kg) U.S. Military Academy cadet in otherwise good health was participating in physical training when she sustained a right-ankle sprain due to inversion of her foot, with immediate pain and swelling of her ankle. Radiographs of her ankle revealed no fractures, joint space abnormalities, or degenerative changes. Within 3 days of initial injury she began to complain of proximal radiation of the pain, paresthesias in the lateral lower leg and foot, and weakness of dorsiflexion and eversion of the foot.

One week later, she was evaluated by an orthopedic surgeon, who noted on exam allodynia, hyperesthesia, and vasomotor changes and diagnosed her with early CRPS. He placed her in a walking boot, prescribed aggressive physical therapy, and started her on naproxen 500 mg twice daily and gabapentin 100 mg at bedtime. An MRI revealed soft tissue edema anterior to the lateral malleolus and edema within the talus. Over the next 2 weeks her gabapentin dose was gradually increased to a total daily dose of 2,700 mg, with minimal improvement in symptoms. She was transferred to Walter Reed Army Medical Center (WRAMC) for further evaluation and treatment. Her physical examination was remarkable for right-foot warmth, edema, and erythema. Her foot and lower leg were sensitive to light touch, especially the lateral lower leg and dorsum of the foot. She exhibited allodynia in both the L5 and S1 dermatomes. She demonstrated color changes in the affected extremity. She was counseled for a diagnostic and therapeutic lumbar sympathetic block for presumed CRPS I.

The patient had a lumbar sympathetic block in the interventional pain clinic, and despite a temperature change of 5.4 degrees centigrade in the affected extremity, the patient experienced no improvement in her pain, and was unable to weight-bear. She was admitted to WRAMC for aggressive treatment of her pain syndrome. A treatment plan aimed at both the peripheral and central components of her pain was initiated. She was started on an infusion of parenteral ketamine at a dose of 0.1 mg/kg/h. She was also evaluated by the Acute Pain Service for placement of a sciatic continuous peripheral nerve block (CPNB) catheter. She was admitted to the surgical intensive care unit for continuous pulse oximetry and 1-to-1 nursing coverage. Her room was kept as quiet as possible and disturbances by visitors and staff were minimized to help reduce sensory stimulation.

Before placement of the catheter, the patient rated her pain at 8/10 on the verbal rating scale (VRS, zero = no pain, 10 = worst pain imagined). The sciatic nerve was identified in the popliteal fossa using the lateral approach to the sciatic nerve, as described by Barrington et al. [12] Using ultrasound guidance, the division of the sciatic nerve into the tibial and common peroneal nerves was identified and the catheter was placed proximal to the division. The CPNB needle was placed near the sciatic nerve, and 20 mL 1.5% mepivacaine with epinephrine 1:400,000 was administered within the nerve sheath. A CPNB catheter was then threaded and tunneled. A continuous infusion of 0.2% ropivacaine was started at 10 mL per hour, with a patient controlled bolus dose of 3 mL every 20 minutes. During the course of the infusion, the patient's leg was protected from compression injury with foam padding under her lower leg and her foot was kept elevated.

Approximately 15 minutes later, the patient had a VRS pain score of 1/10, and she was unable to plantar or dorsiflex her foot. The ropivacaine infusion was continued overnight along with the ketamine infusion. The following day motor function had returned to the foot and she had excellent pain relief over most of the initially painful area. Unfortunately, she continued to have burning pain and allodynia over the lateral lower leg, just distal to the knee. It was proposed that a cutaneous branch of the sciatic nerve likely was missed with the lateral sciatic approach. She was offered replacement of the CPNB catheter using the posterior approach to the sciatic nerve at the gluteal level with neurostimulation. Over the course of the day, the ketamine infusion was titrated up to a maximum dose of 0.6 mg/kg/h and the ropivacaine infusion was discontinued for 8 hours prior to the placement of the new block. During this time, her VRS pain score increased to 5/10. After catheter replacement, and bolus of 30 mL of 0.5% ropivacaine, the patient experienced complete pain relief with VRS of 0/10, including the lateral aspect of the lower leg.

On evaluation the next morning, the initial motor block had dissipated and she was able to move her foot without experiencing pain. She remained pain free for 3 days with an infusion of ketamine 0.6 mg/kg/h and a ropivacaine infusion of 10 mL/h with a patient controlled bolus dose of 3 mL every 20 minutes via sciatic nerve catheter. On day 4, both the ketamine and ropivacaine infu-

sions were discontinued and the patient continued to have complete resolution of her pain symptoms. She underwent physical therapy and was able to weight-bear on the affected ankle with 0/10 pain, she also had full range of motion of the ankle joint. She was discharged back to West Point to resume cadet basic training. Six months after discharge from WRAMC, the patient remained pain free and able to perform her duties as a cadet. She has recently begun training for a marathon and is able to run without pain.

Discussion

While this is an atypical treatment course for a patient with CRPS, the patient was highly motivated to obtain effective treatment that would allow her to continue training at the Military Academy. The patient had a finite period of time to allow more traditional modalities such as physical therapy and medical management to have an effect. As a first-year cadet, she needed to progress rapidly in order to fulfill her responsibilities, or risk being discharged from the Academy. The ability to provide intense inpatient therapeutic options may not easily extend to the civilian population.

The pathophysiology of CRPS remains unclear. Others have suggested that CRPS may include both sympathetic and nonsympathetic pathways, and both central and peripheral neuronal pathways [9]. While the diagnosis of CRPS may not always be straightforward, the patient in this case clearly meets all four diagnostic criteria [2]. She had a lumbar sympathetic block in the affected extremity without improvement of her pain, as well as 2 weeks of medical management and aggressive physical therapy.

It has also been proposed that overexcitation of the N-methyl-D-Aspartate (NMDA) receptor complex may be involved in the development of CRPS [10,11]. Ketamine is a potent NMDA receptor antagonist that has been used in the treatment of both chronic and acute pain states. Ketamine has been specifically studied in the treatment of CRPS in a wide range of doses, with no clear optimal therapeutic regimen [10]. Moderate to high-dose ketamine infusions can produce significant central nervous-system side effects, complicating its use. Hospital admission can provide a safe environment, facilitate adequate patient observation, and allows treatment of side effects.

Treatment of CRPS with CPNB catheters is a relatively recent development. Several case reports have described good efficacy with this form of analgesia [13–16]. To our knowledge, there have been no prospective, randomized controlled trials for the treatment of CRPS with CPNB catheters. The infusion of local anesthetic provides adequate analgesia for intensive physical therapy, which is a key component in the functional rehabilitation of CRPS [8]. The infusion was continued for 4 days in this case, further investigation may help delineate the optimal length of CPNB infusion.

In this case, the first CPNB catheter that was placed was not completely effective, as an area of the lateral lower leg, just distal to the knee, was not blocked. Because of the intense pain experience by the patient in this region, the lateral sciatic catheter was replaced with a posterior sciatic catheter. The area missed by the first catheter is normally innervated by cutaneous branches of the common peroneal nerve [17]. We hypothesize that the patient had a branch of the common peroneal nerve that split proximal to the insertion point of the catheter. With the more proximal posterior sciatic catheter, the patient experienced complete analgesia of the lower leg. We chose to perform the posterior sciatic nerve block under neurostimulation, as opposed to ultrasound guidance, because we have good success with this technique at our institution. Also, as the ultrasound wave penetrates more soft tissue in the posterior approach, the resolution of the sciatic nerve can become much more difficult to visualize compared to the more distal lateral sciatic approach.

Both central and peripheral pathways are likely involved in the pathogenesis of CRPS, as central hyperexcitability and peripheral autonomic dysregulation both seem to play a role [18]. In this case, both pathways were aggressively treated resulting in a quick return to a normal functional status. The combination of ketamine and the CPNB catheter may have had synergistic effects leading to a complete resolution of the patient's pain. While the use of ketamine alone [10] and of peripheral nerve blockade alone [13–16] in the treatment of CRPS have been previously described, to our knowledge, the treatment regimen presented in this case (combined parenteral ketamine and CPNB) has not been described in the literature. While the treatment of CRPS remains a dilemma with no clear superior therapy, the treatment strategy described here may provide a safe and effective therapeutic option for the rapid resolution of CRPS. Our patient

required aggressive diagnosis and treatment in order to return to full functional status as a cadet. It is possible that the aggressive treatment course taken prevented more permanent and more difficult-to-treat neurodegenerative changes often associated with chronic CRPS symptomology. Based on this case report, we feel further study into this treatment approach for CRPS is warranted.

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