Brief Communication

Spontaneous Muscle Contraction with Extreme Pain after Thoracotomy Treated by Pulsed Radiofrequency

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Chronic post thoracotomy pain (CPTP) is a common complication of thoracotomy, which often causes refractory pain and decreases patients' quality of life. CPTP remains a stubborn problem for pain physicians. However, spontaneous muscle contraction (SMC) is a very rare complication of thoracotomy. Here we present a case of extreme pain with SMC after thoracotomy. The patient was treated with pulsed radiofrequency (PRF) targeting the intercostal nerves through the angulus costae.

The patient, a 57-year-old man, underwent lung cancer resection surgery in 2009. The patient suffered consistent severe pain immediately after the surgery. Two years after the surgery the patient still felt throbbing, prickling, and numbing pain around the surgical scar. Allodynia existed in these areas, and the pain intensity was 8 points assessed by visual analogue scale (VAS). In addition, SMC around the surgical scar developed in this patient, the frequency of which was approximately 15 times per minute, in 5-minute cycles, with 50 – 60 cycles each day. Gabapendin (1800 mg/day) and amitriptyline (100 mg/day) showed poor efficacy in pain intensity and SMC.

After the predictive intercostals never block (INB), we performed PRF on the intercostal nerves through the angulus costae 2 weeks after the INB. The specialized medical instruments required for the intervention was a radiofrequency (RF) generator (Baylis PM230) with a 15 cm RF cannula with a 5 mm exposed electrode tip. During the intervention the patient was placed in a prone position. The skin projection position of the angulus costae was marked under guidance from a C-arm x-ray. The RF cannula was vertically inserted at the lower edge of the angulus costae of T5 until the cannula tip touched the rib. (Fig. 1). After the positive response to the testing mode (50HZ, 0.3V), the mode was then turned to the therapy mode (42°C, 120s for 2 cycles). The same treatment was then applied in T6. The patient received these PRF treatments 3 times, at 2-week intervals.

The patient reported pain relief of 50% a few days after the intervention. The frequency and amplitude of SMC was reduced by approximately 70% and 40%, respectively. The patient's pain was reduced to 25% and the frequency of SMC was ultimately reduced to 10%. In 2 years of follow-up, the pain intensity and SMC was roughly stable.

Discussion

CPTP is defined as pain occurring or persisting in the area of the thoracotomy incision for more than 2 months (1). Neuropathic pain after thoracotomy is pain lasting for more than 6 months after the surgery, presenting symptoms such as spontaneous pain or evoked pain (e.g., allodynia). Neuropathic pain carries an incidence of ap-

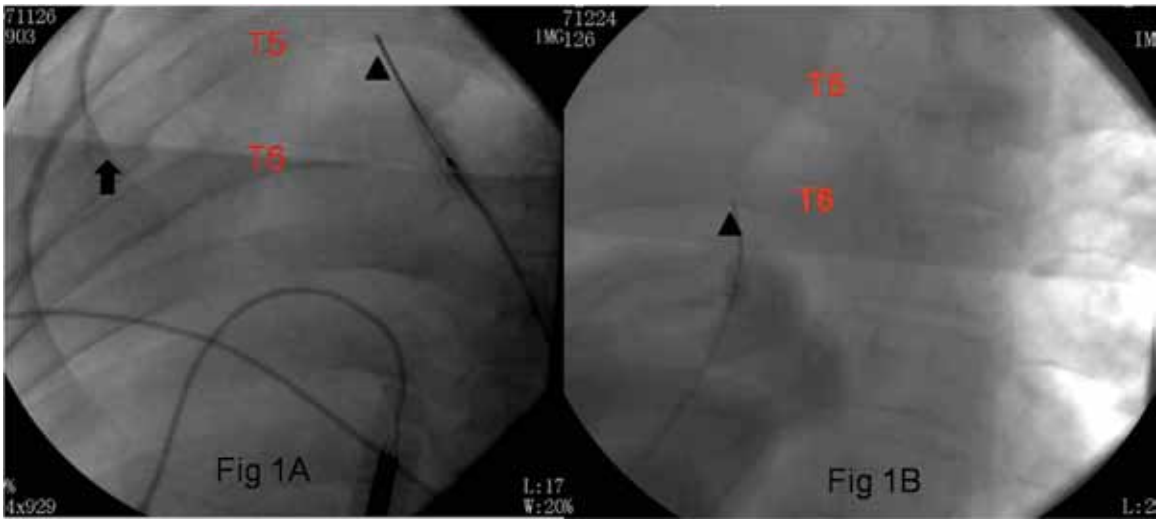

Fig 1. *The digital figures captured during the intervention: The old rib fracture after the thoracotomy (arrow in Fig. 1A), the exposed tip on the intercostal nerve through angulus costae of T5 (arrowhead in Fig. 1A) and T6 (arrowhead in Fig. 1B).*

proximately 29% (2). In other studies, 32.5% – 50% of patients suffering from CPTP were also diagnosed with neuropathic pain (3,4). Patients with the neuropathic pain component suffered more severe pain.

In this patient, the pain was greater during the SMC. SMC is a very rare complication after thoracotomy. It has been known that peripheral nerve injury could lead to muscle spasm (5). In the pain-spasm-pain model, pain would lead to muscular hyperactivity such as spasm, which in turn would cause pain. The possible mechanism of SMC was that nociceptors affect the output of muscle spindles via direct excitatory projection on the gamma motor neurons, and then the increased muscle spindles output will cause the hyperexcitability of the alpha motorneuron pool. During the muscle contraction, accumulations in the muscle of bradykinin, potassium, and lactate could cause pain. In this case, the improvement of both pain and SMC may be because the PRF treatment led to pain reduction by relieving the peripheral sensitization, and then improved the vicious cycle of pain-spasm-pain.

It has been accepted that early treatment of pain could reduce the incidence of CPTP (6,7). Aggressive analgesia such as thoracic epidural analgesia, paravertebral blocks, and intercostal nerve blocks were effective in the early phase (7). However, these therapies provided limited long-term analgesia in CPTP (8). A single case report showed that botulinum toxin relived pain in CPTP (9). Peripheral nerve and spinal cord stimulation for the treatment of CPTP have also been reported in a series of case reports (1,10-12). As for PRF, both intercostal nerves and the dorsal root ganglion (DRG) have been targeted in the treatment of CPTP in several studies and case series (13,14).

The peripheral nervous system plays an important role in central sensitization (15). A functional neuroimaging study showed that peripheral nerve stimulation led to brain activity in chronic migraine (16). In addition, in animal studies, PRF on a peripheral nerve induced the change of numerous pain-related molecules including TNF-a, IL-6, GABAB-R1, and met-enkephalin in the spinal cord (17,18). Treatments on the peripheral nervous system have been applied in many neuropathic pain and chronic migraine cases (19,20). Subcutaneous peripheral nerve stimulation for the treatment of neuropathic pain, such as CPTP and thoracic PHN, also showed good results (10). In a clinical trial conducted by our research group, PRF on intercostal nerves through the angulus costae provided significant pain relief in PHN patients (21).

PRF has been regarded as a safe and effective treatment for various types of postoperative and non-postoperative pain (22). PRF on intercostal nerves in treating CPTP has been reported in a few studies (13,14). An earlier study suggested that PRF targeting the DRG is superior to targeting intercostal nerves in the treatment of CPTP (13). However, in this retrospective study, the accurate puncture point was not mentioned. In an-

other case series, the puncture point was 8 cm lateral to the spinous processes (14). In this case, the angulus costae were accurately targeted under fluoroscopy, because in this manner, PRF could modulate the entire axis of the intercostal nerves including the dermal, lateral, and anterior nerve branches to maximize the range of analgesia. In addition, compared with the DRG, PRF on intercostal nerves is easier to manipulate, and carries less risk of pneumothorax (21).

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Brief Communication

Sudden Discontinuation of Chronic High Dose Intrathecal Hydromorphone and Its Withdrawal Implications

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Intrathecal (IT) therapies have become increasingly utilized since their inception in the 1980s. Clinical research into their effectiveness has been ongoing since that time. Morphine has had the most robust evidence with multiple clinical trials. However, many IT agents are used clinically despite the lack of US Food and Drug Administration approval. Use of these agents is based upon personal clinical experience and consensus recommendations. Dosages for these IT agents also vary greatly and can make instances in which they are quickly stopped challenging. The optimal dose range for hydromorphone has a wide range, with current dose range recommendations up to a maximum of 10 mg/d (1). This is an increase compared to a previous recommendation made in 2007, in which the maximum dose range was 4 mg/d (2). The increase in dose range occurred despite new randomized clinical trials. This case report describes a 67-year-old patient on high dose IT hydromorphone that was emergently stopped due to wound dehiscence and infection. The patient was additionally on IT bupivacaine and clonidine. No time was available to wean down any of the IT medications.

The patient is a 67-year-old female with an IT pump infusing 9.4 mg/d hydromorphone, 1.6 mg/d bupivacaine, and 47 mcg/d clonidine for back pain. Despite this, the patient continued to take oxycodone controlled-release (CR) 40 mg twice daily with 5-10 mg oxycodone immediate-release 4 times a day as needed. The patient presented to the emergency department with an infected pocket site and the decision was made by the surgical team to explant the pump and catheter that night. Preoperatively, a 150 mcg fentanyl patch was started and immediately postop hydromorphone patient-controlled analgesia (PCA) was started using a continuous infusion of 5 mg/h with a bolus dose of 1 mg every 6 minutes with no lockout. The patient was monitored in the intensive care unit (ICU) for signs and symptoms of withdrawal. During the 24 hour time frame from the evening on postoperative day (POD) #0 and POD #1, the patient used 104.7 mg of intravenous (IV) hydromorphone with no signs and symptoms of withdrawal. The continuous infusion of hydromorphone was decreased to 3 mg/h during POD #1; this subsequently decreased the total hydromorphone dose to 46.1 mg for the 24 hour period for POD #2. The PCA continued to be weaned in an aggressive fashion such that the total dose of IV hydromorphone was 12.5 mg for POD #4. One episode of hypertension with chest pain occurred POD #5 requiring readmission to the ICU, which was not thought to be secondary to opioid withdrawal. The patient had a rash caused by the fentanyl patch and was started on oxycodone CR/oxycodone immediate-release as needed that was gradually increased as PCA was weaned off. The patient was discharged on 60 mg oxycodone CR 3 times a day and oxycodone 15 mg every 6 hours as needed.

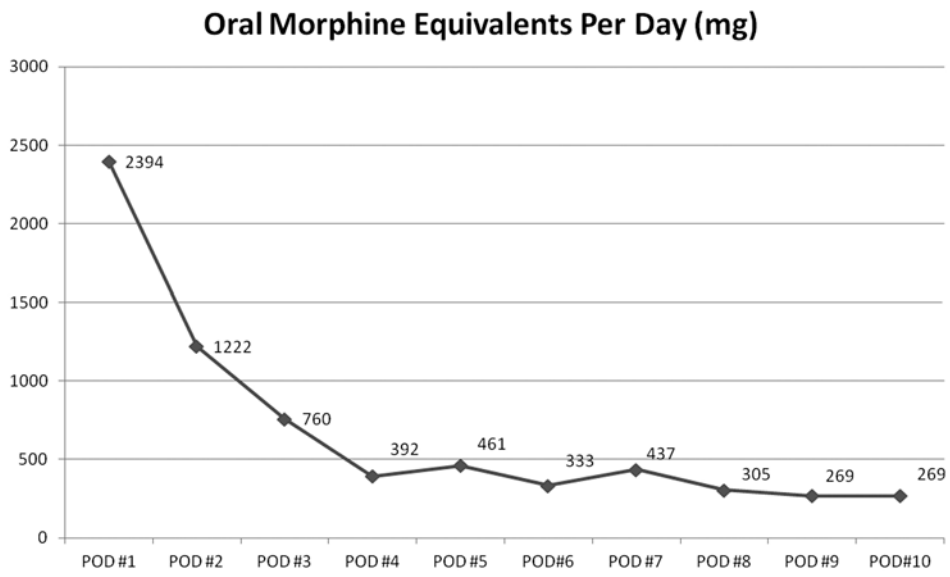

Fig. 1. *The daily use of oral morphine equivalents in milligrams.*

The conversion of IT hydromorphone can be challenging. Although it is debated, the approximate conversion of IT hydromorphone to IT morphine is 5:1 (3). Additionally, the conversion of intrathecal morphine to its oral form is challenging as well, with ratios of IT to oral morphine ranging from 300:1 to 12:1 (4,5). Some of these conversion factors are extrapolated from animal data. This wide conversion factor range creates clinical situations in which they are used troubling since they differ by such a large factor. For the sake of simplicity, a commonly accepted conversion factor of 100:1 IT morphine to IV morphine is used here. Intravenous morphine can then be converted to oral morphine at a value of 3:1 (9.4 mg of IT hydromorphone/d x 5 x 100 x 3). This yields an approximate value of 14,000 mg oral morphine equivalents (OME) per day. This commonly accepted dosing conversion may be more accurate for opioid naïve patients than for patients with opioid tolerance on chronic therapy.

Starting a replacement regimen to prevent withdrawal given such a high dosing requirement presents obvious challenges as there is no evidence in the literature of how to wean down from such high doses. Opioid withdrawal after cessation of intrathecal opioid therapy is a known clinical entity with one case report demonstrating withdrawal after a one-time dose of IT morphine (6). Case reports exist in which high dose IT therapies were stopped due to complications from pump failure or granuloma formation, but these reports did not examine the regimen to prevent withdrawal (7, 8). The initial combination of hydromorphone PCA and fentanyl patch could provide a theoretical maximum of 7,360 mg of OME per day if the patient is able to hit the button every 6 minutes during a 24-hour period. The patient used less than 20% of the initial PCA settings and still nursing reported that the patient had mental status changes at night and the PCA button was removed with no side effects of withdrawal. This allowed for a very aggressive weaning schedule. Common thinking is patients must be weaned down slowly in order to prevent withdrawal, but this patient weaned very quickly. The authors suspect that there is a certain threshold of opioid consumption that can prevent any withdrawal regardless of starting dose. This suspicion is based on the patient not withdrawing on a fraction of the previous dose but still receiving a large dose of opioids. This may also be a reason to question the conversion factor of IT to IV opioids.

This patient also demonstrates a problem with using high dose IT opioids despite the lack of good randomized clinical data. The patient continued to use oral opioids that were but a small fraction of the large amounts of IT therapies calling into question the true efficacy of using such a high dosing regimen of IT opioids. Interestingly, the patient reports that her pain is better now without the IT pump. The authors believe

that the use of oral high dose opioids for chronic non-malignant pain does not have good evidence and perhaps it is time to call into question the use of high dose IT opioids for chronic non-malignant pain as well (9).

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Brief Communication

Newly Diagnosed Lumbar Nerve Root Intradural Mass in the Setting of Chronic Lumbar Radicular Pain Refractory to Conservative Management

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A 31-year-old male, full-time, office worker presented to the outpatient clinic with worsening sciatica of roughly 3 year duration in consultation for left lower leg radicular symptoms refractory to conservative therapy. Although he complained of left buttock pain and leg pain, he denied any constitutional symptoms. On examination, he had negative straight-leg-raise and slump tests and was without motor-sensory deficits. His history and physical exam suggested left lumbar radiculitis. A year-old lumbar spine magnetic resonance image (MRI) without contrast revealed a central disc extrusion at L5-S1 in close proximity to the left S1 nerve root (Fig. 1), consistent with his clinical presentation. Yet, the patient had been treated conservatively with physical therapy, non-opioid medications, and epidural steroid injections for 18 months with gradually worsening symptoms. Given only temporary and minimal benefit from conservative treatment, a follow-up lumbar spine MRI with contrast was deemed necessary to rule out any intramedullary process for his enduring and worsening symptoms. The MRI revealed an intradural enhancing mass with central necrosis creating a thecal sac stenosis with compression of exiting L5 and traversing S1 nerve roots (Fig. 2). The main differential considerations included schwannoma, neurofibroma, and ependymoma as the location of the mass was roughly consistent with the patient's dermatomal distribution of dysesthesias. He was immediately referred to spine surgery for evaluation for tumor excision with the goal of improving his suspected left leg radiculitis.

After discussion with 2 independent spine surgeons, each of whom recommended elective surgical intervention for symptom relief, the patient consented to and underwent L4-L5 laminectomy and resection of the intradural tumor. A surgical microscope was used to circumferentially dissect the mass and debulk the lesion. The intraoperative frozen section was consistent with a nerve sheath tumor (later confirmed upon histopathological examination). The fascicle of origin was separated from the remainder of the nerve roots and stimulated at supraphysiologic levels. Subsequently, the involved fascicle was cauterized and incised, with tumor and capsule being removed prior to closure. There were no complications postoperatively and the patient was discharged to home on postoperative day 2.

At the one-month postoperative follow-up appointment with neurosurgery, the patient reported good pain control and subjective "moderate back stiffness" without radicular symptoms. He was cleared for further physical therapy by neurosurgery and continued to work from his home computer. The patient suffered no further lower extremity radicular symptoms upon subsequent phone follow-up at 3 months.

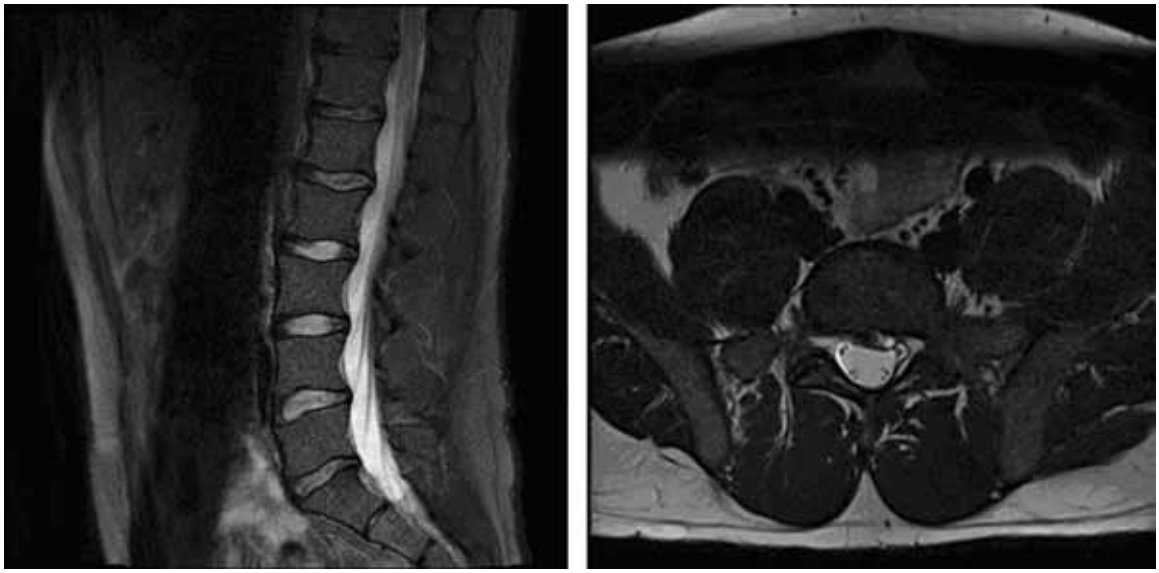

Fig. 1. *Initial T2 weighted lumbar MRI without contrast taken 12 months before initial consultation exhibiting L5-S1 central disc extrusion impinging on the left S1 nerve root.*

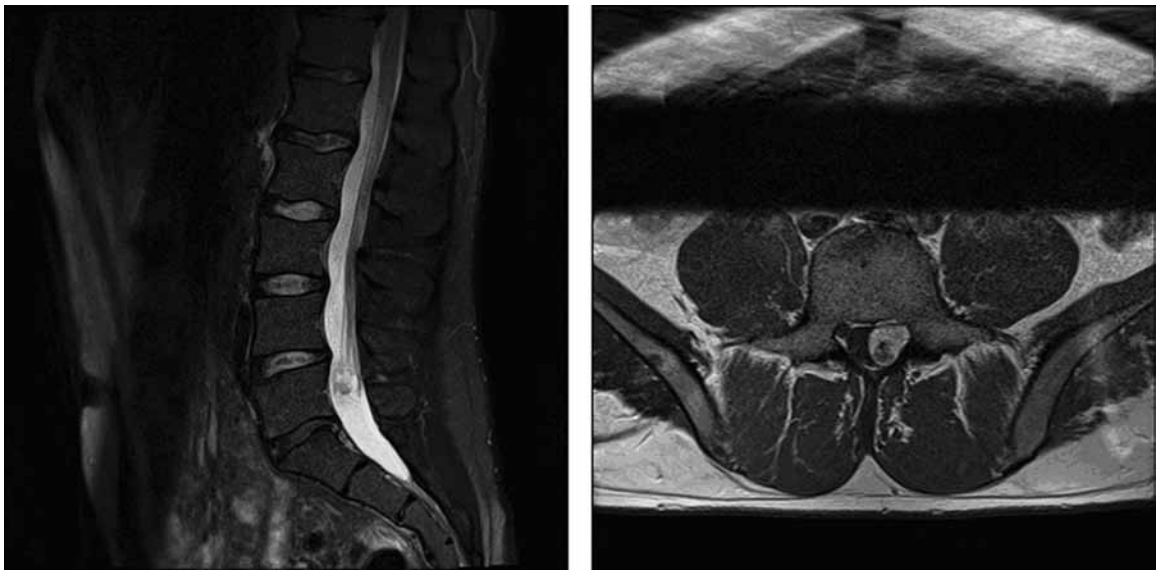

Fig. 2. *Follow-up T2 weighted lumbar MRI with contrast exhibiting an intradural, extramedullary mass with central necrosis causing thecal sac stenosis with compression of exiting L5 and traversing S1 nerve roots.*

Discussion

Diagnosis of a degenerative herniated intervertebral disc is a typical occurrence in patients presenting with neck or lower back pain with radiation to the limbs. Although much rarer, spinal nerve root tumors can manifest with an identical clinical presentation (1-4). Considering the sheer number of cases of chronic low back pain with radiculopathy that physicians assess,

whereby the predominant underlying pathology will classically be degenerative disc disease or spondylosis, it is hardly surprising that clinicians will infrequently consider an intraspinal neoplasm as cause of lumbosacral radiculopathy. Further confounding matters, by their very nature and anatomical location, certain intraspinal neoplasms lend themselves to mimicry of herniated intervertebral discs (1-4). As our case illustrates, it may be imperative to still consider an occult neoplasm at the lumbar intervertebral foramen or lateral recess that may clinically mimic disc pathology on non-contrast MRI, especially in cases failing to respond to conservative treatments.

Of the tumors responsible for such misdiagnoses, nerve sheath tumors such as schwannomas or neurofibromas represent the majority, with schwannomas comprising about 60% (1). Spinal nerve sheath tumors can occur at any level of the spine and are usually classified as intradural, extradural, or intradural-extradural. They arise from the spinal nerve root and grow concentrically along its length with 2 possible sites of growth restriction: the dural aperture for the spinal nerve root and the intervertebral foramen (5). Schwannomas predominately present with intradural localization (1,5,6) and with pain (6,7), as in our patient. In a retrospective study by Gelabert-Gonzalez et al (7), including 68 patients treated surgically for spinal schwannomas, 80.8% presented with local or radicular pain and 66.2% of the tumors were situated in the lumbosacral region. In a separate retrospective study by Safavi-Abbasi et al (6), including 128 patients without neurofibromatosis who underwent resection of their spinal schwannomas, 45.8% were situated in the lumbosacral region and the majority presented initially with local or radicular pain. Conflicting evidence supporting a predilection for cervical, lumbosacral, and thoracic spine has been reported for schwannomas (6), but the relationship of the tumor to the dura mater and intervertebral foramen at an individual vertebral level must be emphasized above all in pathologic symptomatology.

Schwannomas, formerly called neurilemomas in the literature, are benign, slow-growing tumors, arising from the myelin-producing Schwann cells of neural crest origin. Although erroneously used interchangeably when discussing nerve sheath tumors, schwannomas and neurofibromas have distinct histological, biological, and clinical characteristics that merit separate consideration (4,6). Both are associated with neurofibromatosis (NF), with neurofibromas predominating in NF type-1, while schwannomas are more common in NF type-2. Neurofibromas aggressively invade the nerve root, making surgical excision impossible without consequent neurologic deficit. NF type-2 is associated with a biological variant of schwannomas that are aggressive and behaviorally distinct from sporadically occurring, isolated spinal schwannomas. Non-NF spinal schwannomas tend to merely encapsulate the nerve, allowing successful excision enmass without neurologic deficit for the vast majority of patients, as also in the present case. However, following complete resection of isolated, non-NF, spinal schwannomas, clinical follow-up and radiological examination should be performed for at least 5 years after resection, as some studies have shown a recurrence rate of 10% at a mean of 4.1 – 4.3 years after surgery (6).

Schwannomas of the cauda equina are insidious and present non-specifically with back and leg pain, yet attempts have been made to identify specific clinical characteristics differentiating the presentation of neural tumors around the spine and disc herniation (1,8). In general, tumors tend to have a longer average duration of symptoms and can be refractory to conservative treatment. In a retrospective review of 744 surgical procedures performed on patients with symptoms of disc disorders who had failed conservative therapy, 1.2% were found to have intraspinal tumors (none of which were malignant) (8). On the other hand, in advanced cases the pain might become very severe, unresponsive to treatment, and disproportionate to that normally expected with disc herniation (1). To aid in the decision-making process, a 2013 Cochrane review of 8 cohort studies, evaluating red flags to screen for malignancy in patients with low back pain, found insufficient evidence to provide recommendations for the diagnostic accuracy of isolated red flags, such as night pain as reliable indicators for malignancy (9). In our experience, isolated night pain in recumbency is ubiquitous in degenerative spondylosis and does not warrant further work-up for occult tumor. However, a constellation of factors including 2 or more red flags, or atypical behavior and chronicity (insidious onset / chronic persistence) of symptomatology and refractoriness to conservative treatments, should bring consideration of an intraspinal neoplastic process to the fore. MRI is the modality of choice in examining cases of suspected spinal tumors. On T2-weighted MRI, a peripheral hyperintense rim with a central low intensity, called a target pattern, is characteristic of, but not specific to, schwannomas (10). Contrast enhanced MRI aid the differentiation from lumbar disc herniations or sequestrated disc fragments (3,10) in the setting of radiculopathy.

In this patient, the occult tumor noted on re-eval-

uation by MRI with contrast was subsequently found to be a schwannoma. This schwannoma was found at the same level as the chronic L5-S1 herniated disc with noted impingement of left S1 nerve root on previous non-contrast MRI. As such, chronic low back pain with radiculopathy refractory to conservative therapies may require a more comprehensive reassessment despite initial diagnostic imaging suggestive of degenerative spondylosis or discogenic pathology for fear of missing an occult tumor.

Disclosure

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Brief Communication

Perioperative Pain Management in a Patient with Anaphylaxis to Full Mu-agonists Presenting for Head and Neck Salvage Surgery

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Anaphylaxis during the perioperative period is one of the most feared complications for anesthesiologists who care for surgical patients. While muscle relaxants account for the majority of perioperative anaphylactic reactions, opioids are a rare, yet known, cause for anaphylaxis with a perioperative incidence of 1.4% (1). We present the management of a patient with documented anaphylaxis to phenanthrene derivatives (hydromorphone, morphine, oxycodone, hydrocodone), phenylheptylamine derivatives (methadone), and phenylpiperidine derivatives (fentanyl, alfentanil, remifentanil, sufentanil, meperidine).

A 56 year-old ASA class III male presented to the pain management clinic for evaluation and treatment recommendations for his upcoming head and neck salvage surgery. He had a past medical history significant for laryngeal carcinoma status-post radiation therapy, radiation-related pharyngocutaneous fistula, tracheostomy, and multiple documented allergies to opioids. Prior to presenting to our institution, he was previously administered morphine, fentanyl, hydrocodone, oxycodone, hydromorphone, and methadone. These medications all independently resulted in allergic reactions associated with angioedema. He was ultimately referred to Allergy & Immunology for sensitivity testing to various opioid classes including phenanthrenes, phenylheptylamines, and phenylpiperidine derivatives. However, since opioids cause direct mast cell degranulation, it was determined by the allergist that skin sensitivity testing was not recommended. She was unable to determine an opioid that could be used peri-operatively for pain management besides tramadol; he was able to tolerate tramadol without any allergic symptoms. Given his extensive allergy list and upcoming surgery, it was decided that the safest option would be to combine neuraxial anesthesia with non-opioid adjuncts. A previously documented study by Merquiol et al (2) demonstrated the use of cervical epidural anesthesia for laryngeal and hypopharyngeal cancer surgery. In their single-center retrospective cohort study, perioperative cervical epidural analgesia was associated with significantly increased cancer-free survival as compared with patients treated with general anesthesia alone (2). For our patient, our goals were to utilize fluoroscopically guided cervical epidural analgesia to manage his pain perioperatively given his significant allergy history to multiple parenteral and enteral opioids.

After written consent was obtained, the patient was placed in the prone position on the fluoroscopy table. Standard ASA monitors were applied and a peripheral intravenous line was placed. The upper back was prepped with chlorhexidine gluconate and draped in the usual sterile fashion. Initially, fluo-

roscopic anterior-posterior views were obtained of the T1-T2 interspace. The skin and subcutaneous tissues overlying this were anesthetized with bicarbonated 1% lidocaine through a 27-gauge 1.25-inch needle. Then, using an 18-gauge Tuohy epidural needle, we advanced in a coaxial fashion until the ligamentum flavum was engaged. A loss-of-resistance syringe filled with air was applied and a firm loss of resistance was noted at 6 cm. After negative aspiration was confirmed, a Pajunk catheter (Sonolong Nanoline Kit, Pajunk, Geisingen, Germany, 521185-31C) with a radiopaque stylet was threaded under fluoroscopic guidance until the tip was located at the inferior border of C6. After negative aspiration, 1 mL of omnipaque was injected to confirm proper epidural catheter placement on an anterior-posterior as well as lateral fluoroscopic view. The needle was withdrawn and the catheter was secured to the skin. A sterile cap was placed on the epidural catheter. Due to the several allergies to Tegaderm and certain types of adhesive, we confirmed that Hypafix would be safe for catheter securement. A Stat-Lock and Hypafix with a series of 1/4" steristrips were used to secure the epidural catheter. It was draped over his left shoulder. A test dose consisting of 3 mL of 1.5% lidocaine with 1:200,000 epinephrine was injected to ensure that there was no vascular or intrathecal spread. After 15 minutes, we checked the dermatomal blockade with pin prick testing which demonstrated bilateral dermatomal blockade from C4 to T2 without any respiratory compromise. The patient was advised not to contaminate the epidural site, not to take a shower, or to inject anything through the epidural catheter. He was then discharged from the clinic to home.

The following day, the patient underwent laryngopharyngectomy with tubed left anterolateral thigh free flap, anastomosis to the internal mammary artery in his right chest, right pectoralis flap, and split-thickness skin-graft from the left thigh over the pectoralis flap. After discussions with the surgeons, preoperatively he received the following medication through his percutaneous endoscopic gastrostomy (PEG): gabapentin 1,200 mg, acetaminophen 1,000 mg, and diclofenac 100 mg. Intra-operatively he was started on intravenous ketamine 2 mcg/kg/min and his cervical epidural infusion of ropivacaine 0.1% was started at 6 mL/hour without a bolus. Acetaminophen was continued with 1,000 mg IV every 6 hours and ketorolac 15 mg IV every 6 hours for the first 48 hours. Postoperatively his pain was controlled with an IV ketamine infusion at 2 mcg/kg/min, acetaminophen 1,000 mg was given via the PEG every 6 hours, diclofenac 50 mg given via the PEG every 8 hours, gabapentin 1,200 mg given via the PEG every 8 hours, and a cervical epidural infusion of ropivacaine 0.1% at 6 mL up to 9 mL per hour without any demand dose. Tramadol 100 mg was given via the PEG every 6 hours as needed for moderate to severe pain. His pain scores ranged from 0 to 6 postoperatively and his cervical epidural catheter was eventually removed on postoperative day 9. The remainder of his postoperative course was uneventful and he was eventually discharged from the hospital.

Discussion

Patients presenting with opioid allergies pose a particular challenge to the anesthesiologist. During the preoperative visit, it is imperative to determine the exact nature of the reaction and what workup has been performed to confirm which medications are safe and which produce cross-reactivity. During cross-reactivity, medications such as opioids may have similar epitopes such that known anaphylaxis to one opioid may trigger anaphylactic/anaphylactoid reactions to other opioids with similar structures (3). Opioids induce direct mast cell degranulation and histamine release, making skin sensitivity testing extremely difficult. Additionally, mast cell degranulation is different depending on the mast cell anatomical location. Skin mast cells have been shown to release histamine while mast cells located in other organs of the body show little to no degranulation when exposed to opioids (4,5).

In our case, the patient presented with known anaphylaxis to the following classes of opioids: phenanthrenes, phenylpiperidines, and phenylheptylamines (6). He did not have any known clinical reactions to morphinans or benzomorphans. It was suggested that all opioids were potentially capable of an adverse reaction and testing was inaccurate because of cutaneous mast cell degranulation associated with all opioids. Based on his previous experience, he was able to utilize tramadol without adverse effect. Tramadol is unique in that it is an atypical opioid with partial mu agonist activity in addition to central GABAergic, serotonergic, and noradrenergic activity (6). For our patient, his perioperative pain management plan involved a multimodal approach with sub anesthetic doses of ketamine (Glutamate N-methyl-D-Aspartate receptor antagonist), acetaminophen, diclofenac/ketorolac (cyclooxygenase inhibitors), and gabapentin. This was combined with continuous cervical epidural analgesia with local anes-

thetic, sodium-channel blockade, only. Given the extent and nature of the surgery, we anticipated that his pain would not have been adequately treated with only tramadol and intravenous non-opioid analgesics. Tramadol at doses up to 400 milligrams/day provided suboptimal pain control. While it may be difficult to determine the relative efficacy and contribution of the cervical epidural infusion compared with other components of the multi-modal analgesic regimen, the patient noted significant benefit from the epidural infusion.

For these difficult patients, pain physicians are optimally positioned to assist in the perioperative management of their pain. The use of fluoroscopy allows the safe placement of cervical epidural catheters as well as ensuring the optimal position of the catheter such that the appropriate dermatomal segments will be anesthetized. In this case our test dose of 3 mL of lidocaine 1.5% produced a 7 level dermatomal blockade, which one might not normally expect. Without fluoroscopic confirmation of epidural placement, the anesthesiologist might attribute this finding to intrathecal placement. We hypothesize that this happened in our case due to undiagnosed spinal canal stenosis at multiple levels making this patient's epidural volume smaller, as well as the heterogenous nature (fat and blood vessels) of the epidural space that can result in quite variable dermatomal spread. It is possible that the rate of injection may have also interacted with the above variables as well. This unexpected result perhaps best exemplifies the value of fluoroscopic guidance to confirm proper epidural catheter placement.

Conclusion

Patients presenting with opioid allergies from multiple opioid classes present a unique challenge to anesthesiologists. Opioids cause direct mast cell degranulation and histamine release independent of the opioid receptor or IgE specific antibodies. Combining neuraxial or regional anesthesia with multimodal non-opioid regimens is an alternative option to improve pain control not only during surgery but also postoperatively. Pain physicians are in a unique position to assist in both the preoperative evaluation as well as the perioperative pain management of patients in whom optimal pain control may be difficult. The use of fluoroscopy for preoperative placement of difficult epidural catheters ensures not only epidural placement but also optimal positioning of the catheter to ensure the appropriate dermatomal segments are covered.

Disclosure

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Brief Communication

Medial Sesamoid Bone Avulsion but not Plantar Fasciitis: Ultrasonographic Diagnosis Using Sonopalpation

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A 21-year-old female was referred for an ultrasound (US) examination with a likely diagnosis of plantar fasciitis. On questioning, she declared that she had intermittent right medial heel pain which worsened after walks. She added that the pain gradually developed after she had stumbled over a stone half a year ago. On US, plantar fasciae appeared normal in echogenicity and thickness. Meanwhile, she also pointed another painful area at her medial fore foot and the US evaluation was extended accordingly. With sonopalpation, a disrupted medial sesamoid bone and a thickened, hypervascular deep intersesamoid ligament were uncovered (Fig. 1A). A bony fragment was observed at the insertion of the medial tendon slip of the flexor hallucis brevis muscle as well (Fig. 1B). As such, the patient was diagnosed with a medial sesamoid bone avulsion fracture and we considered that her heel pain originated from overstrain of the plantar fascia during the push off phase of the gait cycle to decrease irritation on her medial forefoot.

Sesamoid bone fracture is not a common cause of metatarsalgia (1,2). Since the tendon slips of the flexor hallucis brevis muscle attach on the sesamoid bones, like in our case, an abrupt hyperextension force on the forefoot may result in avulsion injury of the hallux. Herewith, partition of the sesamoid is a normal variant that should be differentiated from a fractured sesamoid (3). The former is less likely to cause adjacent hypervascularity and overlap of the detached bony fragment on the main body. The above scenario highlights the importance of further scrutinizing in patients with symptoms of a particular diagnosis where US findings are inconsistent. Last but not least, it should always be kept in mind that sonopalpation is definitely paramount for prompt diagnosis especially for small bony cortical lesions which radiographs fail to capture (4).

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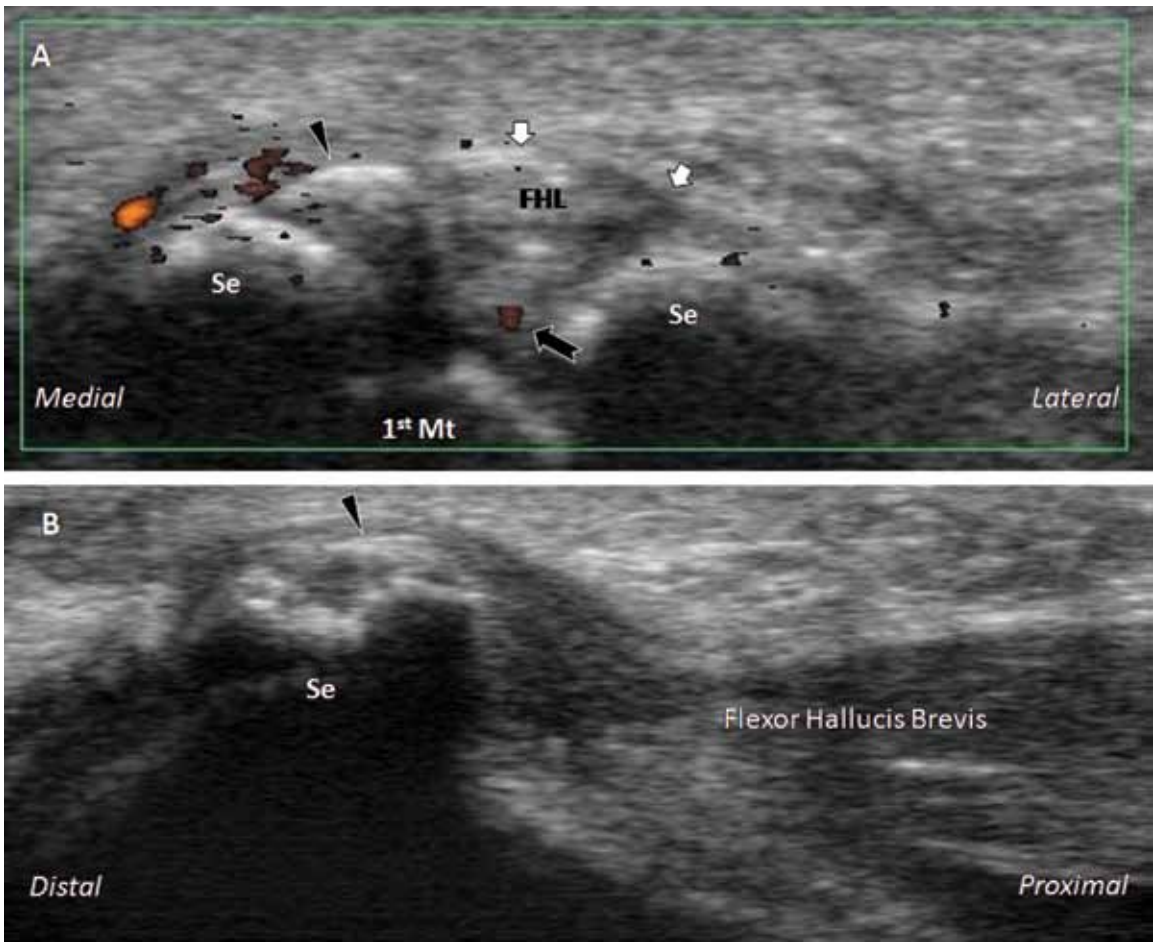

Fig. 1. *(A) US imaging (axial view) of the plantar aspect of the first metatarsal head (1st Mt) shows a separate bony fragment (arrowhead) overlapping the medial sesamoid bone (Se). Also note hypervascularity surrounding the fractured sesamoid and inside the deep intersesamoid ligament (black arrow) underlying the flexor hallucis longus tendon (FHL) and the superficial intersesamoid ligament (white arrows). (B) US imaging (longitudinal view) shows the sesamoid (Se) fragment (arrowhead) connecting to the medial tendon slip of the flexor hallucis brevis muscle.*

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Brief Communication

Onset of Spontaneous Lower Extremity Pain After Lumbar Sympathetic Block

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Lumbar sympathetic nerve blocks (LSBs) can be performed to determine whether or not a patient's pain is sympathetically mediated. They can be used as prognostic injections to determine the response to future more permanent sympathectomy or as therapeutic interventions on their own. Common presentations of sympathetically mediated pain include vascular insufficiency and peripheral nerve injuries suffered in trauma or limb amputation. Such injuries play a prominent role in complex regional pain syndrome (CRPS). The Budapest Criteria detailed in the Table 1 describe the conditions under which a diagnosis of CRPS can be made (1). CRPS is characterized by severe pain, pseudomotor, and vasomotor symptoms affecting a specific area of the body that is sometimes associated with injury or nerve damage. Pain originates from multiple sources including neurogenic inflammation, vasomotor dysfunction, and changes in central pain processing. It is the result of the body's abnormal response to tissue injury with varying clinical presentations including hyperalgesia, allodynia, swelling and skin discoloration. LSBs are one of the early interventions used to treat CRPS because they are minimally invasive, have a long safety record, and can help determine what component of the pain is sympathetically mediated.

Case Report

The patient discussed herein consented to the use of this case for educational purposes.

A 50-year-old woman presented with chronic right lower extremity (RLE) pain. Her medical history included morbid obesity, status-post bariatric surgery, diabetes, hypertension, anxiety, transient ischemic attack, and bilateral carotid artery stenosis. She complained of severe right lower extremity pain as if her "leg was on fire." The pain was perceived to originate from the ankle and radiate towards the knee. Exam demonstrated significant tenderness to palpation diffusely in the RLE in the same distribution. She also endorsed generalized weakness in the RLE, and the right calf was visibly atrophied versus the left. Range of motion exam resulted in severe pain in the knee and ankle. The patient showed decreased ability to discern light touch from pinprick sensation from knee to ankle on the RLE. The RLE was about 1 degree Celsius warmer than the left from toes to knees. Recent electromyography and nerve conduction studies (EMG) were negative for large fiber neuropathy in the affected limb. Lower extremity magnetic resonance imaging (MRI) and vascular consultation were also negative and she failed medication therapy with gabapentin 300 mg 3 times per day. A differential diagnosis of peripheral neuropathy versus CRPS was given and the patient was scheduled for a right lumbar sympathetic block.

Table 1. *Budapest Criteria for Complex Regional Pain Syndrome.*

Proposed clinical diagnostic criteria for CRPS

To make the *clinical* diagnosis, the following criteria must be met:

1. Continuing pain, which is disproportionate to any inciting event
2. Must report at least one symptom in *3 of the 4* following categories:

Sensory:
Reports of hyperesthesia and/or allodynia

Vasomotor:
Reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry

Sudomotor/Edema:
Reports of edema and/or sweating changes and/or sweating asymmetry

Motor/Trophic:
Reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

3. Must display at least one sign *at time of evaluation in two or more* of the following categories:

Sensory:
Evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or temperature sensation and/or deep somatic pressure and/or joint movement)

Vasomotor:
Evidence of temperature asymmetry (>1°C) and/or skin color changes and/or asymmetry

Sudomotor/Edema:
Evidence of edema and/or sweating changes and/or sweating asymmetry

Motor/Trophic:
Evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

4. There is no other diagnosis that better explains the signs and symptoms

From Harden RN, Bruehl S, Stanton-Hicks M, Wilson PR. Proposed new diagnostic criteria for complex regional pain syndrome. *Pain Med* 2007; 8:326-831(1). © 2007 by John Wiley & Sons, Inc. Reprinted by permission of John Wiley & Sons, Inc.

For the procedure, the patient was placed in the prone position with EKG, oxygen saturation, noninvasive blood pressure and temperature monitoring. No sedation was administered. Pre-procedure lower extremity temperature measured 22.97°C on the right foot and 23.03°C on her left foot. Skin and subcutaneous tissue was anesthetized using 2% lidocaine. Next, a 22 gauge 6-inch needle was advanced in an oblique approach under fluoroscopic visualization so that the tip of the needle rested at the right anterolateral aspect of the L4 vertebral body. 3 milliliters of Iohexal was subsequently injected and appropriate contrast spread was demonstrated. A total of 15 milliliters of 1:1 mixture of 2% lidocaine and 0.5% bupivacaine was injected in 3 milliliter increments without significant changes in vital signs or other signs of complications. Immediately after the procedure the patient complained of new onset "stabbing" pain over the affected leg that was significantly worse than her preprocedure pain. She showed no deficits in strength, but displayed marked guarding of her right leg with allodynia. Ten minutes after the procedure, right foot temp was noted to be 33.78°C, a 10-degree change, while the left foot temperature had remained almost unchanged at 24.31°C.

After an hour of observation the patient was released to the care of her family with instructions to proceed to the emergency room should she experience new onset weakness or changes in bowel or bladder function. She was also provided with a prescription for tramadol. Over the next 48 hours, the patient noted continued extreme pain followed by resolution of the pain over the next 2-3 days. The patient returned to clinic one week later for reevaluation. Her lower extremity temperatures were noted to be 24°C bilaterally at the feet and 27.8°C at the ankles. Her pain level was 0/10 and she denied having filled her tramadol prescription or taken any pain medication.

Discussion

Though many complications of sympathetic blockade are represented in medical literature, it has thus far provided an incomplete picture of spontaneous onset of pain following sympathetic blockade. Some complications include bleeding, hypotension, genitofemoral nerve block or neuralgia, intravascular injection, ureteral/kidney damage, and psoas muscle injection (2-6). Transient increases in pain have been noted lasting up to a week in a significant proportion of patients after sympathetic block according to van Ejis et al (7).

Surgical literature has described post sympathectomy neuralgia including aching discomfort in the dermatome distribution immediately proximal to that of sympathetic denervation beginning between 5 and 10 days postoperatively with an overall mean duration of 5.0 weeks. In one study the incidence of this post-surgical pain was found to be as high as 41% (8).

Postsympathectomy limb pain, also named sympathalgia, has been described as early as the 1920s according to reports (9). One common theme has been the presence of a post procedural pain-free interval between 1-24 days and the abrupt onset of a severe deep, boring, dull ache. Pain is generally considered to be worst at night and usually localized to the anterior and anterolateral aspect of the thigh (9). It remains unknown whether a post-surgical sympathetic pain interval may derive from the same mechanisms that result in a post-sympathetic block neuralgia. Specific mechanisms for post surgical sympathectomy neuralgia that have been proposed include direct axon surgical damage and nociception-induced sensitization of spinal nociceptive neurons (10).

The typical "pain free interval" was not present in our case, making sympathalgia an unlikely cause of the post procedural pain in our patient. We feel direct axonal injury is also an unlikely cause because the placement of the needle, appropriate spread of contrast, lack of pain during injection, lack of objective neurological findings, and spontaneous resolution of symptoms within 5 days are factors that do not fit the typical picture of patients with axonal nerve injuries.

It is our opinion that the sudden onset of excruciating pain that the patient experienced may have been due to the sudden and immediate revascularization of the patient's lower extremity, such as muscles, subcutaneous tissues, and skin. This may have resulted in a "steal phenomenon," decreasing the blood supply to deeper structures such as the bone and deep muscles, which may have ultimately led to the patient's pain reaction. Vascular steal phenomena are well-documented in vascular surgery literature; pain at rest is their defining characteristic (11-12).

Changes in central pain processing may also have been responsible; patients undergoing mirror therapy for phantom limb pain, which may share central pain processing features with CRPS, sometimes experience initial pain increase with initial treatment (13).

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