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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

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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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