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Neurogenic Unilateral Leg Edema

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Unilateral leg edema is almost always caused by a disease process that obstructs the venous or lymphatic drainage in the leg or pelvis but can also occur in chronic regional pain syndrome (CRPS).[1] We report unilateral leg edema that was a consequence of CRPS induced by an S1 radiculopathy (type 2 CRPS). We show that the etiology of the edema was neurogenic because a posterior tibial nerve block abolished the edema distal to the block.

One of the authors (H.F., a 78-year-old neurologist) developed left-sided buttock pain with numbness in the S1 distribution and moderate weakness in the corresponding muscles. Magnetic resonance imaging showed a disc herniation on the left at L5/S1 with compression of the S1 nerve root. He underwent a foraminotomy and discectomy. There was initial marked reduction in the pain and improvement in the sensation and muscle strength. Four weeks after the surgery, increased numbness in the S1 distribution occurred that now included waves of painful paresthesia. The weakness also increased. The skin temperature of both feet felt equal and there was no allodynia. The left leg developed pitting edema from the knee to the toes. Left lower-extremity venous duplex imaging and computerized tomography of the abdomen and pelvis were normal. The edema was controlled with a compression stocking and elevation of the foot. The pain was controlled with pregabalin and amitriptyline.

Magnetic resonance imaging of the lumbar spine 56 days after the surgery showed inflammation of the S1 nerve root and entrapment of the S1 nerve root in the foramen by inflammation and scar tissue. To control the painful paresthesia, a left posterior tibial nerve block using bupivicaine was performed on postoperative day 52. The block produced dense anesthesia of the foot for about 72 hours and eliminated the edema in the foot with no change in the edema between the knee and the ankle (**Figure**). After the anesthesia produced by the nerve block resolved, the edema and pain in the foot returned and resolved gradually over the next 90 days. Six months after the surgery the muscle strength was much improved but a painless dense numbness in the S1 distribution continued.

The symptomatology was consistent with CRPS caused by an S1 radiculopathy. The diagnostic criteria for CRPS[1] that were present included pain, hyperesthesia, edema, and weakness. However, there was no allodynia, temperature, or trophic skin changes.

The reversal of the edema in the foot by the posterior tibial nerve block implies that some type of peripheral nerve activity was controlling the lymphatic or microvascular system but does not distinguish which one. There is evidence for autonomic control of both the microvascular and lymphatic systems.[2] The S1 nerve root injury resulted in both loss of activity (numbness and weakness) and increase in activity (painful paresthesia). Thus, it is not possible to conclude whether the edema was the result of a loss or gain in activity in the autonomic system. We suggest that the differential diagnosis of unilateral lower extremity peripheral edema includes neurogenic etiologies. In CRPS that occurs without overt nerve injury such as after fracture or stroke (type 1 CRPS), the peripheral edema is also postulated to be neurogenic. This hypothesis could be tested by observing the effect of the appropriate nerve block on the edema in CRPS that occurs without overt nerve injury.

Figure. Left Foot After the Posterior Tibial Nerve Block

The site of the nerve block is indicated by the small bruise. The edema was abolished distal to the block but remained unchanged proximal to the block.

ARTICLE INFORMATION

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