Botulinum toxin type A (BTX-A) is a recognized treatment for the temporary relief of refractory palmar hyperhidrosis. Unfortunately, intradermal injection of BTX-A can be painful. Several modalities to reduce the pain of injection have consequently been devised. Peripheral nerve blockade with lidocaine before BTX-A treatment of palmar hyperhidrosis is a safe and effective method of pain relief. Anesthesia of the palm can be achieved by injecting 4 to 6 mL of 2% lidocaine without epinephrine subcutaneously around the superficial branches of the radial, median, and ulnar nerves. Use of a short, bevelled needle is recommended to reduce the risk of iatrogenic nerve injury. The risk of nerve injury when using a 30-gauge needle in a conscious patient who can provide feedback concerning nerve puncture is remote.

Complex regional pain syndromes (CRPS, formerly reflex sympathetic dystrophy and causalgia) are neuropathic pain conditions of an extremity after a peripheral nerve trauma or central nervous system lesion. CRPS is characterized clinically, although there is no diagnostic criterion standard. Accepted criteria include sensory, autonomic, and motor disturbances. These symptoms are most often described as deep, “tearing” pain at rest and hyperalgesia. Secondary peripheral changes include edema, decreased temperature, decreased sweating, and trophic changes of the skin and soft tissues. Autonomic changes are frequent and often change with the duration of CRPS. Skin temperatures are warmer in acute and colder in chronic stages. Edema has a higher incidence in acute stages. Motor dysfunction is often present, including weakness, tremor, exaggerated tendon reflexes, dystonia, and myoclonic jerks.

We report a case of bilateral CRPS of the upper extremities after local nerve block anesthesia for treatment of palmar hyperhidrosis. Although pain reduction using local nerve blocks is widely recommended for BTX-A treatment, we have not found published cases of CRPS in this setting.

Case Report

A 24-year-old healthy man presented to our office with a 10-year history of previously diagnosed idiopathic palmar hyperhidrosis for treatment with intradermal BTX-A injections. The patient was not interested in other treatment options. He had no history of sensor or motor nerve abnormalities, overuse syndromes, or traumatic nerve injuries to either of his upper extremities and was taking no medications. On physical examination, the patient was anxious, but otherwise well. His palms were visibly moist and dripping.

With informed consent, the patient was treated with the following protocol. His wrists and palms were cleansed with chlorhexidine solution. Bilateral median and ulnar nerve blocks were achieved using a 0.5 30-gauge needle inserted just to the radial side of the palmaris longus tendon under the flexor retina-
culum at the proximal wrist crease and just radial
to the flexor carpi ulnaris tendon, respectively.
Two milliliters of 2% lidocaine without epinephrine
were injected into each site (two sites per wrist).
The patient complained of “tingling” pain in his
hands during injection of the anesthetic. The
nerve blocks provided effective but not complete
anesthesia and the patient tolerated the procedure
well. One hundred units of botulinum exotoxin
A (Botox Cosmetic, Allergan Inc., Irvine, CA,
reconstituted in 2.5 mL of nonpreserved normal
saline) were injected intradermally in each palm
over 25 injection sites. When injections were
complete, the palms were cleansed with hydrogen
peroxide and the patient discharged with
instructions as to onset of BTX-A effect and to
call with any concerns.

Five days after treatment, the patient returned com-
plaining of a “pins and needles” sensation in both
palms. On further examination, this dysesthesia ex-
tended in the approximate sensory distribution of the
median nerve. Touch sensation and muscle power
were normal. Two-point discrimination was not
assessed. The volar wrist did not exhibit erythema
or swelling. The skin exhibited no vascular changes,
but sweating was noticeably reduced. A tentative
diagnosis of neuritis involving the median nerve was
given, and treatment with reduced wrist movement
and ibuprofen (200 mg by mouth twice daily)
was recommended. A return visit was scheduled for
5 days.

Ten days after treatment, the patient returned ur-
gently, complaining of “painful electrical sensations”
and persistent “tingling” throughout both palms. He
entered the examination room holding his arms
flexed 90° at the elbows, with the palms facing up-
ward (Figure 1). Slight edema of both wrists was
noted. No abnormalities in skin temperature, sen-
sation, or trophic changes were noted in the patient’s
hands, wrists, or arms. Bilateral upper extremity
motor nerve function examinations were within
normal limits. Sweating was markedly reduced, as
expected.

A diagnosis of bilateral CRPS was made in the
clinical setting of postural changes of the forearm,
pain markedly out of proportion to the physical
findings, hyperalgesia of the palms, and a normal
neurologic examination. Intensive physical hand
therapy was recommended and instituted the fol-
lowing day. The patient attended eight to 10 phys-
iotherapy sessions over 2 weeks. An 80% to 90%
 improvement in symptoms was evident after this
2-week period. The patient reported complete
resolution of symptoms at 2 months.

Discussion

Peripheral nerve blockade with lidocaine is regularly
performed for management of pain with BTX-A
treatment of palmar hyperhidrosis. Documented
risks of nerve blocks of the wrist include pain,
ecchymosis, hematoma formation, infection, tendon
injury, and nerve laceration. Classically, nerve lac-
eration is immediately recognized because of exces-
sive pain during needle insertion. In this report, we
describe a novel case of bilateral CRPS after local
nerve block anesthesia for treatment of palmar
hyperhidrosis.

CRPS is a disproportionate response to a provoking
event. A literature review of the pathophysiology of
CRPS yields multiple proposed mechanisms, but no
single hypothesis explains all features of this syndrome. Some authors suggest that injury to central neural tissue is a common mechanism. Others maintain that the primary abnormality is in the peripheral nervous system. A personality predisposing toward depression is noted in some CRPS literature, although many studies have shown that most patients become depressed as a result of the pain caused by CRPS. In the United States, Type I CRPS (formerly reflex sympathetic dystrophy) occurs in 1% to 15% of peripheral nerve injury cases, usually secondary to fractures, sprains, and trivial soft tissue injuries. Many cases are not associated with an identifiable nerve injury. The upper extremities are more likely to be involved than the lower, and women are noted to predominate in a range of 60% to 80% of cases. People of all ages are affected.

Diagnosis of CRPS is clinical, as previously outlined. Patients with CRPS may benefit from a multidisciplinary therapeutic approach, including consultations with anesthesiology, physical therapy, and hand surgery. Failure to recognize and treat CRPS early and aggressively may result in progressive pain and worsening physical changes. These can include induration and livedo reticularis or cyanosis of the skin of the affected limb, hypotrichosis, atrophy of subcutaneous tissues, stiffness and joint contractures, and osteoporosis or marked demineralization of underlying bone. Late changes in untreated CRPS may be irreversible.

In this report, a young man undergoing treatment for palmar hyperhidrosis developed symptoms of complex regional pain syndrome in his hands. Anxiety and pain intolerance in this patient may have contributed to a progressively heightened interpretation of pain as it related to any limb movement, thus leading to the neutral postural changes he exhibited at the time of CRPS diagnosis. Investigators have demonstrated bilateral reduction of intracortical inhibition in patients with CRPS involving the hand, and experts have noted that the temperature changes observed with CRPS are invariably bilateral, because temperature regulation at the level of the spinal cord is modulated at the central grey matter of the spinal cord. Perhaps a central cortical mechanism can explain our patient’s bilateral manifestation of CRPS. Fortunately, his symptoms resolved completely within a relatively short time with intensive physical therapy.

Data on this medically and economically significant condition suggest that a universal etiology does not exist; instead, authors describe central and peripheral nervous system abnormalities and differences in personality characteristics and interpretations of pain. Although the pathophysiology of CRPS remains unclear, the early recognition and aggressive treatment of this debilitating syndrome is essential in avoiding irreversible physical injury.

References