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Iatrogenic Posttraumatic Neuralgia Following Neuromuscular Electrical Stimulation: A Case Report

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Background: Posttraumatic neuralgia (PTN) is a complex and debilitating condition that can arise from physical injury to a peripheral nerve, resulting in chronic neuropathic pain that significantly affects the individual's quality of life.

Case Report: A 52-year-old woman with a recent history of incomplete spinal cord injury developed a common peroneal nerve injury from neuromuscular electrical stimulation during her acute inpatient rehabilitation. The patient was initially managed with oral corticosteroids and neuropathic medications without resolving neuropathic pain symptoms. Ultimately, the patient continued to have chronic neuropathic pain symptoms along the common peroneal nerve pathway leading to the diagnosis of PTN.

Conclusions: We hypothesize this unusual complication resulted from a prior neurological injury, making this patient more susceptible to nerve injury when intense electrical forces were transmitted to the common peroneal nerve, ultimately leading to PTN of the common peroneal nerve.

Key words: Posttraumatic neuralgia, common peroneal nerve injury, neuromuscular electrical stimulation, neuralgia

BACKGROUND

Posttraumatic neuralgia (PTN) is a chronic neuropathic pain condition that develops after an acute physical injury to the peripheral nervous system (1). Chronic neuropathic pain after a physical peripheral nerve injury is well recognized in clinical practice and can be broadly divided into iatrogenic and noniatrogenic causes. Iatrogenic PTN accounts for approximately 85% of all cases with orthopedic surgery, implantation of foreign objects, and body position of surgery are the most common risk factors (1). Peripheral nerve injuries are classified into 4 types of injuries: stretch, laceration, compression, and mechanical deformation, and their likelihood of neurological recovery depends on the degree of nerve damage, which is classified by the Seddon and Sunderland grading system (Table 1) (1-3).

Common peroneal neuropathy is the most common mononeuropathy of the lower extremity. It classically presents with acute and progressive onset ankle dorsiflexion and foot eversion weakness along with dysesthesias of the anterolateral lower leg and dorsum foot (4,5). The common peroneal nerve is the lateral branch of the sciatic nerve and travels across the fibular head, which branches into the superficial and deep peroneal nerve near the peroneus longus muscle and the fibula (6). The superficial peroneal nerve provides sensory innervation to the lateral leg and dorsum foot with the exception of the small web space between the first and second toes (6,7). The deep peroneal nerve provides motor innervation to the tibialis anterior, extensor digitorum longus, peroneus tertius, and extensor hallucis longus, as well as

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Table 1. Classification of nerve injury.

Seddon's Classification	Sunderland's Classification	Tissue Injury
Neurapraxia	Grade I	Focal demyelination
Axonotmesis	Grade II	Damaged myelin and axon
Axonotmesis	Grade III	Damaged myelin, axon, endoneurium
Axonotmesis	Grade IV	Damaged myelin, axon, endoneurium, and perineurium
Neurotmesis	Grade V	Complete nerve transection with disruption of myelin, axon, endoneurium, perineurium, and epineurium

the intertarsal joints along with sensory innervation to the web space between the first and second toes (6,7). There are numerous etiologies for peroneal nerve injury, but the 2 most common causes are extraneural compression and trauma (5). Nerve compression affecting the common peroneal nerve at the fibular head and neck level can result from repetitive movements, specific limb positions, accumulation of fluid, or soft tissue masses transmitting persistent forces on the neural tissue leading to neural ischemia (5,7). Often, the first common peroneal nerve injury symptoms involve sensory changes such as allodynia, paresthesias, or dysesthesias along the peroneal dermatome distribution (6). While diagnosing peroneal nerve neuropathy is clinical, electromyography, nerve conduction studies, diagnostic nerve block, and magnetic resonance imaging diagnostic can further assist in the diagnosis (4,8,9).

There have been many reports of common peroneal nerve injury throughout the literature; however, PTN of the common peroneal nerve following neuromuscular electrical stimulation (NMES) has not been reported. NMES is a widely used treatment modality primarily performed by physical and occupational therapists to stimulate weakened or paretic muscles in individuals who have suffered upper motor neuron injuries such as stroke, traumatic brain injury, and spinal cord injury (10). The overall goal is to facilitate motor recovery, and the current settings can be adjusted by manipulating pulse frequency, amplitude, cycle, and duration. Although recognized as a safe intervention, there can still be side effects, such as allergic skin reactions to electrode pads or gel, skin irritation, and discomfort during NMES (10). Despite the standard application of NMES, the literature lacks documented reports of peroneal nerve injury resulting from NMES. This report aims to present an unusual complication of common peroneal nerve injury leading to PTN following NMES therapy, discuss the potential mechanism of injury, and provide clinical points for future clinicians.

CASE DESCRIPTION

A 52-year-old woman was admitted to an acute inpatient rehabilitation facility due to a recent spinal cord injury located at T10 secondary to a thoracic disc herniation. The patient had no significant past medical history prior to this incident and now developed a right foot drop due to her spinal cord injury. On initial physical examination, she presented with a motor strength of hip flexion and knee extension at grade 2, ankle dorsiflexion and big toe extension at grade 2, plantarflexion at grade 2 on bilateral lower extremities, and decreased sensation throughout lower extremity dermatomes. The patient was receiving NMES from physical therapy as part of her rehabilitation course for approximately 15 minutes with 5 minutes of rest for a total of 60 minutes each session 5 days per week over the common fibular neck and anterior tibialis muscle belly with settings of 300 μs (pulse width), 1:5 cycle, 25 mA (amplitude), and 50 Hz (frequency). Two days before discharge, the patient received (NMES) for approximately 15 minutes with 5 minutes of rest for a total of 60 minutes at 10:00 over the common fibular neck and anterior tibialis muscle belly with settings of 450 μs (pulse width), 1:5 cycle, 100 mA (amplitude), and 50 Hz (frequency). The same session was repeated at 16:00 by another therapist. Shortly after her session, the patient developed acute onset constant burning pain with intermittent electric-shock sensations and intense spasms involving her right lower extremity. Physical examination of the skin was warm, dry, and allodynia with light touch along the lateral aspect of the knee just below the common fibular head to the anterolateral lower leg and dorsum foot. The motor strength of ankle dorsiflexion and big toe extension on the right side was reduced from 2/5 to 0/5, and no other changes of motor strength from baseline. Lower extremity pulses were normal. Decreased active range of motion in the right ankle secondary to pain. The patient underwent an urgent intra-compartmental pressure monitor to evaluate for possible compartment syndrome, and the result was within normal parameters.

Further diagnostic testing included a radiographic study of the right knee and tibia/fibula, demonstrating large soft tissues and no fractures. A doppler ultrasound was negative for deep vein thrombosis. There were no other remarkable findings, and common peroneal nerve injury was suspected.

The patient was started on 1,000 mg of acetaminophen as needed every 8 hours and gabapentin 300 mg twice daily with no relief in pain or improvement in function. Capsaicin 8% and lidocaine 5 % ointment were trialed, but she could not tolerate the light touch sensation with an electric shock-like sensation down the peroneal nerve distribution. After 3 days of initial insult and no improvements, pain medicine was consulted for further evaluation and proposed treating this peroneal neuralgia like the early stages of complex regional pain syndrome from a nerve injury. The patient was started on oral methylprednisolone beginning at 8 mg TID for the first 2 days, 8 mg BID and 4 mg in the evening for the third day, 4 mg BID and 8 mg once for the fourth day, 4 mg TID for the fifth day, 4 mg BID for the sixth day, and 4 mg daily for the last day. The patient regained her baseline motor strength (2 out of 5) with right ankle dorsiflexion and big toe extension; however, she continued to have symptoms of allodynia along the right common peroneal distribution with moderate improvement in the frequency of pain episodes. The patient was discharged home on 300 mg TID gabapentin and missed her 4-week follow-up appointment due to her symptoms being controlled by her current medical regimen. However, the patient demanded to be seen around the tenth-week mark due to more frequent and intense electric shock-like sensation down her lower leg. Gabapentin was increased to 600 mg TID, and a nerve conduction and electromyography study was attempted on the patient. However, she could not tolerate the study due to pain and was lost to follow-up. This case report is devoid of all patient-identifiable information, therefore; it is exempt from IRB review requirements and patient consent per institutional policy.

DISCUSSION

This case report describes a middle-aged individual with a recent spinal cord injury receiving rapid titration of NMES leading to a common peroneal nerve injury with progression to PTN. Neuralgia is characterized as a paroxysmal, intense shock-like pain within a specific nerve distribution and a symptom of an injury or disorder (11). The proposed mechanism of neuralgia is direct pressure on a specific nerve by nearby structures resulting in focal demyelination of afferent nerve fibers tend to become hyperexcitable and drive an upregulation of sodium channels (NAv 1.3,1.7, 1.8) capable of generating ectopic impulses manifesting as spontaneous pain (3,11). Another theorized mechanism for PTN is the involvement of a G protein-coupled receptor called CCR2, which was found that mechanical allodynia after peripheral nerve injury is prevented in CCR2-deficient animal models (12). Studies are still ongoing and yet inconclusive on human patients.

In this case study, the patient with impaired lower extremity sensation from her spinal cord injury received a dramatic up-titration of electrical stimulation (400 percent increased amplitude and 50 percent increased pulse width) within centimeters of direct contact to the common peroneal nerve from electrode placement. In one systematic review by Sachetti and his colleagues (13) looking at the safety profile of NMES among critically ill patients, the optimal dose for NMES training protocols remains unclear due to significant differences in the intensity, duration, repetition count, and application site. The most commonly recognized and researched application for foot drop is placing the cathode electrode on the skin just below the common fibular head site and the anode electrode on the anterior tibialis muscle belly (13). The lack of standard protocol for specific conditions makes it difficult for individuals to titrate the patient's treatment regimen appropriately.

Common peroneal nerve injury is most observed by direct trauma due to the superficial location of the nerve as it passes underneath the subcutaneous tissue and across the bone surface of the fibular head and neck, thus leaving it highly susceptible to injury (14). Yang et al (14) note there are 2 potential mechanisms for peripheral nerve injury related to the energy force on the common peroneal nerve: high energy versus low energy. The high-energy mechanism includes avulsion, transection, stretching, or contusion to the common peroneal nerve with external wounds. While low energy mechanisms primarily consist of compression forces leading to ischemic changes by applying excessive force over an extended period to the common peroneal nerve (14).

Over the last several decades, researchers have studied an electrophysiologic phenomenon related to peripheral nerve injuries called the double crush phenomenon. This is where a preexisting neurological condition precedes a new neurological insult to a peripheral nerve,

thus synergistically increasing symptom intensity (1,15). This is most often recognized with a prior compression injury like cervical radiculopathy followed by a new compression lesion along the course of a peripheral nerve like carpal tunnel syndrome; however, research suggests a wide range of etiologies may predispose to the double crush phenomenon, including metabolic, ischemic, toxic, autoimmune, or systemic processes (1,3,16). In this clinical scenario, we theorized that the patient's underlying neurological disease of the spinal cord injury limited the neurological reserve of the affected peripheral nerve, thus increasing the risk of new and more severe neurological deficit once intense electrical forces were directed transmitted to the common peroneal nerve, therefore, developing PTN.

One major limitation of this study was the lack of nerve conduction and electromyography study with this patient. This would localize and categorize the severity of peripheral nerve injury; however, the clinical presentation of acute onset ankle dorsiflexion and foot eversion weakness, along with dysesthesias of the anterolateral lower leg and dorsum foot, made the diagnosis of common peroneal neuropathy most probable. Other diagnoses were considered, such as complex regional pain syndrome (CRPS). However, the patient did not meet the Budapest criteria, and she continued having pain symptoms along a dermatomal distribution when most often, it is a nondermatomal distribution of the affected limb that characterizes CRPS.

CONCLUSION

Overall, we report a rare complication of PTN following a common peroneal nerve injury from improper use of NMES. Though chronic neuropathic pain following a prior acute peripheral nerve injury is well-recognized in clinical practice, understanding this symptomology as PTN is underappreciated. Through this case report, we hope to increase physician awareness of this painful neuropathic condition.

Author Contributions

RC: Principal Investigator, conceptualization of the presented idea, manuscript writing, and revisions.

YK: Manuscript writing, revision, and table construction.

EB: Manuscript review, revision, and table design.

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