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Case Report

Spinal segmental myoclonus following ultrasound-guided suprascapular nerve block: A case report

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ABSTRACT

Herein, we reported a case of spinal myoclonus following an ultrasound-guided suprascapular nerve block. A 58-year-old male patient who had a diagnosis of calcific tendinitis inside the right supraspinatus tendon received a right suprascapular nerve block (3 mL of 2% prilocaine mixed with 2 mL of 1% lidocaine) under the guidance of ultrasound. Sudden, painless, and involuntary movements began in the upper extremity 10 min after the intervention. Three months after the injection, the involuntary movements were entirely stopped. Involuntary, painless, and brief muscle jerks may be suggestive of myoclonus after interventions with local anesthetics.

Keywords: Local anesthetics, myoclonus, suprascapular block, ultrasound.

Myoclonus is described as a hyperkinetic movement disorder characterized by sudden, brief jerks arising from involuntary movements of the muscles. Jerks may be repetitive and rhythmical or irregular and may be present during movement, at rest, or on sustaining a posture. The clinical features and electrophysiological results of myoclonus vary based on its site of origin in the nervous system.[1]

Myoclonus may be produced from spinal, subcortical, or cortical circuits and includes the somatotopic organized dorsal column/superimposed lemniscal and corticospinal systems, as well as spinoreticular and reticulospinal circuits.[2] Many neurologic conditions and a wide variety of drugs and toxins can induce myoclonus by affecting these pathways. The use of antidepressants, anticonvulsants, opiates, antipsychotics, and anesthetics may generate myoclonus. There are a limited number of studies reporting local anesthetics-induced spinal myoclonus, and most of the cases are related to spinal interventional procedures. Herein, we reported a case of spinal myoclonus following an ultrasound-guided suprascapular nerve block.

CASE REPORT

A 54-year-old male patient was admitted to the physical medicine and rehabilitation clinic with a complaint of right shoulder pain for two to three months. He had a history of trauma to the left shoulder from an in-car traffic accident 18 years ago. At that time, the patient had attended a rehabilitation program for left shoulder rotator cuff rupture, and the pain resolved after the rehabilitation program. He did not describe pain in the right shoulder until two to three months ago and denied any recent history of trauma or strenuous exercise that might initiate the right shoulder pain. There was no personal or family history of movement disorders.

The pain was aggravated by overhead activities. The patient had taken paracetamol, nonsteroidal anti-inflammatory drugs, and tramadol before visiting

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the clinic, but the pain had not improved. The patient's shoulder radiography and computed tomography were compatible with calcific tendinitis inside the right supraspinatus tendon. Hemogram, acute phase reactants, and biochemical tests, including thyroid function tests and electrolytes, were unremarkable. The patient had chronic obstructive pulmonary disease and did not have any history of psychological neurological, renal, or metabolic disorders. The patient did not smoke or drink alcohol.

In the patient's examination, the right shoulder abduction and flexion were 90°, external rotation was 30°, and internal rotation was 20°. The patient had been prescribed tramadol, paracetamol, and nonsteroidal anti-inflammatory drugs for two weeks and recommended to avoid overhead activities, but the pain did not alleviate, and the range of motion in the right shoulder did not improve. Due to the persistent shoulder pain, the patient consented to a right suprascapular nerve block under ultrasound guidance. At the suprascapular notch, 3 mL of 2% prilocaine mixed with 2 mL of 1% lidocaine was injected with a 26-gauge needle. The patient did not complain of injection pain or severe paresthesia during the injection.

The patient developed an involuntary and rhythmic muscle jerk of the right shoulder, arm, forearm, and hand 10 min after the injection. The painless muscle jerks were rhythmic, unilateral, and occurred at a rate of approximately 1 Hz. No muscle contractions were observed in the other parts of the body. The movements could not be suppressed intentionally, and the patient remained conscious and communicated appropriately. There were no relieving factors, and the effort to move increased the contractions. External triggers, such as light or noise, did not affect the jerks. The muscle jerks did not change during rest or while maintaining posture but accelerated during action. Auditory or tactile stimuli did not aggravate the movements.

The jerks were accompanied by high blood pressure and dizziness; therefore, the patient was followed in the emergency room. The patient’s vitals, except for hypertension, were stable. On examination, motor and sensory functions were intact in all muscle groups, except for the numbness in the distribution of the right suprascapular nerve. Pain, tactile sensation, and position-vibration sensations were normal. Cerebellar tests were normal on the left side. Full cooperation could not be achieved due to involuntary movement on the right. Bilateral Hofmann's tests were negative, and bilateral plantar skin reflexes revealed downgoing toes. Laboratory findings, including electrolytes, were unremarkable.

The patient was evaluated by a neurology specialist. Biperiden 5 mg was administered intramuscularly as per the advice of the consultant since the movements lasted longer than 30 min. However, there was no change after administration. The movements persisted during the night and interrupted the patient's sleep as noted by a physician and the patient's spouse. Electroencephalography and contrast-enhanced cranial magnetic resonance imaging (MRI) performed the next day were normal. Contrast-enhanced cervical MRI revealed degenerative changes; however, any changes that could be associated with the movements were not observed. Electromyography (EMG) showed that sensory and motor conduction studies and motor unit morphology were normal. Movement analysis was performed with multichannel recording for voluntary movements in the right upper extremity. Needle EMG showed rhythmic, repetitive, and synchronized discharges of motor units with a rate of 1 to 2 Hz in the muscles of the right arm and forearm. Surface EMG agonist and antagonist muscles had simultaneous, rhythmic, synchronous burst discharges. Discharge duration was between 100 and 500 msec, and discharges were unaffected by reflex stimuli. Abnormal motor unit discharges were not observed in the other three extremities.

As a result of clinical, laboratory, radiological, and electrophysiological evaluations, the patient was diagnosed with spinal myoclonus. Valproate 500 mg orally twice a day was advised by the neurologist. A decrease in the amplitude of involuntary movements and an increase in frequency were observed. As the patient's uncontrolled hypertension continued, the patient was evaluated by a cardiologist, antihypertensive treatment was initiated, and valproate discontinuation was recommended. No increase in involuntary movements was observed after discontinuation.

The patient's involuntary movements decreased gradually. After one week, the movements ceased at rest, the amplitude of muscle jerks decreased, and the frequency increased at the voluntary movements of the right upper extremity. One month after the injection, the involuntary movements almost entirely ceased. Muscle jerks were observed only at the end of active movement. Three months later, myoclonus completely stopped despite no other treatment being applied.

A written informed consent was obtained from the patient to write and publish the present case report.

DISCUSSION

Myoclonus may arise from the cortical system (cortical myoclonus), brainstem (brainstem myoclonus), and spinal cord (spinal myoclonus) and present due to lesions in spinal roots, plexus, or peripheral nerves (peripheral myoclonus). The most common causes of peripheral myoclonus are peripheral nerve injury related to trauma, surgery, and degenerative radiculopathy.[3] In our ultrasound-guided suprascapular nerve block injection, the needle was oriented along the suprascapular region with the in-plane technique. Both the orientation of the needle body and the needle tip, which was in the suprascapular notch, were visible. The combination of drugs was injected around the suprascapular nerve, the position of the needle was observed, and the patient did not feel any sharp sense or tingling during the procedure. Nevertheless, the suprascapular nerve could not be detected. Irritation of the needle or neurotoxicity of local anesthetics could result in right suprascapular nerve injury that could cause peripheral myoclonus in our case, but the movements and EMG results were not limited to the muscles innervated to the right suprascapular nerve.

The affected muscle groups may suggest that myoclonus arises as a result of a lesion in a particular part of the nervous system, such as the peripheral nerve, plexus, spinal root, spinal cord, or cranial. The myoclonus in our case was widespread and not limited to muscles innervated from the right suprascapular nerve. Electromyography also did not reveal any peripheral nerve or root damage. Since the myoclonus was not associated with only one nerve or root and movements were restricted to the right upper extremity, it was considered a spinal myoclonus. Two types of spinal myoclonus are described: spinal segmental myoclonus and propriospinal myoclonus. Spinal segmental myoclonus involves a restrained body part, and the propriospinal myoclonus generates axial jerks.[1] Our patient was diagnosed with spinal segmental myoclonus.

The cervical spinal MRI performed after the procedure revealed no significant pathologies that could explain involuntary movements. The injection site in the case (suprascapular notch) was also not close enough for drugs to diffuse into the spinal canal or for the needle to irritate the spinal cord. Although there are scarce cases of spinal myoclonus following peripheral nerve damage, it is still the subject of debate.[3,4] It has been revealed that peripheral nerve injury increases activity in the spinal cord by reorganization of circuits.[5] Adjacent intact nerves are responsible for increased response at dorsal horn nociceptive neurons.[6] Various pathological mechanisms have been described for the contribution of the spinal cord to the myoclonus. Campos et al.[7] stated the following mechanisms: loss of local inhibitory spinal interneuronal function, suppression of dorsal horn interneurons, and expression of spontaneous spinal neuronal discharge. It is believed that future experimental studies may shed light on the contribution of the spinal cord to the myoclonus, which occurs due to peripheral causes.

Spinal myoclonus following neuraxial anesthesia is rare, the neurotoxic effect of local anesthetics and neuronal irritation by needle may precipitate spinal myoclonus.[8] Neurotoxicity or irritation may cause increased impulse transmission and sympathetic or anterior horn cell hyperexcitability.[8] There are few reports of spinal myoclonus secondary to local anesthetics, with bupivacaine used as a local anesthetic in most cases.[7-12] In this case, prilocaine and lidocaine were used as local anesthetics. Although it is known that lidocaine reduces glycine, which is an inhibitory neurotransmitter, and decreased levels may result in hyperactivity,[13] there has been no reported case of lidocaine-induced myoclonus. Lidocaine can also be used to reduce the number and severity of etomidate-related myoclonus.[14] Novales and Celorrio[10] reported the first case of prilocaine-induced spinal myoclonus. In this case, myoclonus started 60 min after epidural 5% hyperbaric prilocaine injection and persisted for 1 h until resolution with full recovery. The myoclonus in our case started 10 min after injection and persisted for more than one month. In an experimental study, local anesthetics decreased glycine and gamma-aminobutyric acid (GABA) induced currents in the hippocampal neurons of the rats. Depression of the inhibitory neurotransmitter in neurons may cause local anesthetic-induced convulsions.[13] The usage of a combination of local anesthetics may result in the persistence of myoclonus, as in our patient.

This is the first case of spinal myoclonus following suprascapular nerve block to our knowledge. Hudson et al.[15] reported a myoclonus case after a regional peripheral nerve block. Left sciatic and femoral nerve blocks with bupivacaine and mepivacaine were administered in their case. Involuntary movements were restricted to the left ankle of the

patient. The myoclonus started the day after the procedure, persisted for 6 to 10 h, and resolved spontaneously with no sequela. Hudson et al.[15] could not achieve a definitive diagnosis due to the lack of electrophysiological studies; nonetheless, their patient exhibited clinical features suggestive of both peripheral and spinal myoclonus. Electrophysiological studies are the first step in evaluating a patient with myoclonus, and it can be shown that agonist and antagonist muscles usually fire synchronously in myoclonus. Although most of the jerks generate short bursts of 10 to 50 msec, spinal myoclonus may present with 100 msec jerks.[1] Our case of spinal myoclonus following peripheral nerve block is also valuable, as it was demonstrated by EMG.

Spinal myoclonus is reported to be resistant, while the underlying reason for peripheral myoclonus is generally reversible and treatable.[4] Anticonvulsant drugs, which enhance GABA inhibitory activity, are the first treatment choice for myoclonus. The most used antiepileptic drugs are clonazepam and valproate. Although initial treatment with clonazepam is recommended for patients with subcortical nonsegmental myoclonus, clonazepam treatment was not preferred in our patient since it could worsen cognitive status or movement coordination, considering the patient's age.[16] In addition, the benefit of clonazepam in spinal segmental myoclonus can often be limited. Controlled studies on symptomatic treatment of segmental myoclonus are limited. Valproate can suppress myoclonus through several mechanisms, including GABA synthesis in nerve terminals and increased sodium and potassium conductance.[17] Initiating the treatment with sodium valproate, followed by clonazepam if necessary, was also suggested.[1] It was thought to be myoclonus and valproate was administered to the patient. Valproate has to be discontinued two days later because of adverse effects and two days is a short time to evaluate its effectivenesS.

In conclusion, peripheral nerve blocks are widely performed for pain relief or regional anesthesia. Neurotoxicity from local anesthetics or direct nerve irritation by the needle may lead to myoclonus. Involuntary, painless, and brief muscle jerks following the procedure should raise suspicion of myoclonus. Accurate diagnosis can be achieved through clinical evaluation and electromyographic studies. Although myoclonus after interventional procedures generally resolves spontaneously without sequela, it may persist for up to a month.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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