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Bilateral sciatic neuropathy following rhabdomyolysis : case-report

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Introduction

Rhabdomyolysis is a pathological condition caused by skeletal muscle cell damage that affects the integrity of the cell membrane, leading to the release of toxic intracellular components into plasma. The muscle damage is often due to trauma, extreme physical exercise, ischemic injury, drugs, alcohol, infections, metabolic disorder, and hyperthermia. The clinical features are often non-specific and may include muscle pain, fatigue, dark urine, and swelling of the affected muscles. In some cases, the swelling of the damaged muscle leads to local compression of a peripheral nerve, which may Result in neurological complications. Some studies have reported peripheral nerve injury following rhabdomyolysis. However, bilateral sciatic nerve injuries following rhabdomyolysis which led to bilateral sciatic neuropathy.

Case

A 42-year-old woman with no past medical history, who slept on an electric heating pad after drinking alcohol, visited the emergency department the next day with complaints of bilateral calf pain, redness, burning sensation, and weakness of the lower extremities. She denied ingesting any drug that can induce rhabdomyolysis and had no history of excessive exercise or trauma. Blood urea nitrogen, creatinine, and creatinine kinase were elevated in the blood test. She was diagnosed with acute kidney injury secondary to rhabdomyolysis and was admitted for treatment of rhabdomyolysis. After aggressive hydration, the rhabdomyolysis improved but the pain and weakness of the lower extremities persisted. Spine magnetic resonance imaging (MRI), cerebrospinal fluid (CSF) analysis, magnetic resonance angiography (MRA) of a lower extremity, and electrodiagnostic study were performed. There was no abnormal finding in spine MRI and CSF analysis. MRA showed multifocal edema and enhancement of the bilateral lower extremity muscles. Sensory nerve conduction study (NCS) revealed decreased amplitude of sensory nerve action potentials of both superficial peroneal and sural nerves. In the motor NCS, the right peroneal and both tibial nerves showed low compound motor action potential and the left peroneal nerve showed no response. The electromyography (EMG) test revealed abnormal spontaneous activities and decreased interference patterns in the biceps femoris, gastrocnemius, tibialis anterior, and peroneus longus muscles. The patient was diagnosed with bilateral sciatic neuropathy secondary to rhabdomyolysis.

Conclusion

Peripheral nerve damage is a rare complication of rhabdomyolysis. This is a rare case of bilateral sciatic neuropathy following rhabdomyolysis. Peripheral neuropathy could be caused by compression and inflammation associated with rhabdomyolysis. To minimize neurological complications, early diagnosis and intervention are needed.