Delayed Reversible Motor Neuronopathy Caused by Electrical Injury

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Electrical injury may cause central nervous system (CNS) complications and peripheral nerve disorders. Delayed neurologic complications are rarely reported. A case of delayed reversible motor neuronopathy caused by low-voltage electrical injury is reported due to its rarity. A 22-year-old female received an electric shock of 110 volts while pushing up a metal gate during a rainy morning on April 16, 2005. She initially suffered loss of consciousness for several hours, and then became quadriplegic, from which she completely recovered 10 days later. After return to work for 1 month, she developed weakness and numbness of bilateral upper limbs. Nerve conduction velocity study and bilateral median nerve somatosensory evoked potential were normal. Magnetic resonance imaging of the brain and cervical spine were also normal. Electromyography showed mild denervation, reduced interference and polyphasia over the upper arms, suggestive of anterior horn cell lesion. After rest and rehabilitation for 2 weeks, the patient completely recovered her muscle power over proximal upper limbs and partially over the distal upper limbs. Follow-up at the outpatient clinic 4 months later showed total recovery of muscle power. Low-voltage electrical current can cause acute transient quadriplegia and delayed motor neuronopathy. The mechanism of this patient's recovery from electric shock, followed by deterioration 1 month later, and then recovery after rest is unclear. We considered whether the mechanism of weakness after electric injury, with initial recovery followed later by the development of weakness, might be due to overuse, just like in post-poliomyelitis syndrome. This needs further investigation. [*J Chin Med Assoc* 2008;71(3):152–154]

Key Words: delayed neurologic complication, electrical injury, motor neuron disease

Introduction

Electrical injury has been divided into high (≥1,000 V) and low (<1,000 V) voltage injury. It is a relatively infrequent but potentially destructive cause of central nervous system (CNS) complications and peripheral nerve disorders. Several possible mechanisms of neuronal tissue damage after electrical injury have been proposed, including direct mechanical trauma, thermal damage, vascular damage and electrophysiologic changes. Delayed neurologic complications after low-voltage electrical injury have rarely been reported. We report a case of delayed, reversible motor neuronopathy 1 month after recovery from transient quadriplegia and discuss its possible pathophysiologic mechanisms.

Case Report

This 22-year-old female, a caregiver, suffered from electrical injury while pushing up a wet metal gate, which was driven by 110 volts current, during a rainy morning on April 16, 2005. She lost consciousness and was sent to the emergency room of a local hospital immediately. After resuscitation, her consciousness became clear hours later but quadriplegia developed. Neither obvious burn injury nor sites of entrance and exit of electricity were found over her hands and body. Computed tomography (CT) of the brain and nerve conduction velocity (NCV) study showed normal findings. The patient recovered from her weakness gradually within 10 days under regular physical therapy.

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She was discharged with full recovery of muscle power and was able to work as before.

The patient worked at a nursing home, caring for bedridden patients. She had no specific complaint except for soreness and easy fatigability of her limbs, especially of the upper limbs, which was relieved after rest but exacerbated after hard work. However, weakness of her upper limbs continued to develop gradually. She visited another local hospital for help on July 6, 2005, where bilateral upper limb paralysis and numbness was noted. NCV, somatosensory evoked potential study and magnetic resonance imaging (MRI) of the cervical spine were normal. She was referred to our hospital and was admitted on July 23, 2005.

On admission, physical examination was unremarkable. Neurologic examinations showed intact cranial nerve function. Muscle tests showed muscle power of grade 1 over both shoulders and of grade 0 over both upper limbs, with claw-hand posture but without muscle atrophy. Fasciculations were seen diffusely over the upper limbs. Lower limb muscle power was normal. Deep tendon reflexes were absent over the upper limbs, and normal in the lower limbs. The plantar reflex was of flexor type bilaterally. The sensation was intact, although subjective numbness below bilateral wrists was noted by the patient. Repeat NCV study of the 4 limbs, including F-waves and H-reflexes on July 25 was normal. Electromyography (EMG) via sampling the left biceps, abductor pollicis brevis (APB), and abductor digiti minimi (ADM) showed mild denervation, reduced interference pattern and polyphasia in all muscles sampled, with more prominence in the APB and ADM. The above studies suggested anterior horn cell lesion. Magnetic resonance angiography (MRA) of the head also showed normal findings. The findings of the above studies indicated cervical motor neuronopathy.

Upper limb weakness gradually improved with regular physical therapy and non-weight bearing condition. The muscle power of the patient's shoulders, upper arms and forearms recovered completely, and improved to grade 3 over both hands 16 days after admission. She was then discharged in a stable condition. Follow-up NCV study was normal, and EMG study showed mild impaired interference and polyphasia over the left bicep and APB on August 25, 2005. The patient came back to our outpatient clinic for follow-up on November 29, 2005, where muscle power over all 4 limbs was full. Follow-up NCV study was normal, and EMG study via sampling of the left APB and bicep showed mild polyphasia in the APB only on November 30, 2005.

Discussion

Electrical injury might cause CNS complications and peripheral nerve disorders.[1,2] Delayed neurologic complications after low-voltage electrical injury are rarely reported. From a literature review,[3–6] the interval between electrical injury and the onset of motor neuron disease is variable, from days to decades. Recovery, when it occurs, is usually slow and incomplete.[3]

The clinical course of our patient is unique. The initial quadriplegia recovered completely. After strenuous nursing work for 1 month, she developed painful sensation and progressive weakness of her upper limbs, which was improved after rest and rehabilitation. The delayed bilateral upper limb weakness could be due to CNS or peripheral nerve disorders in our patient. The possibility of polyneuropathy, plexopathy or mononeuropathy was excluded by normal findings of repeated NCV and sensory evoked potential studies. NCV showed slow results on plexopathy but normal results on anterior horn cell disorders. Normal MRI of the cervical spine and MRA of the head excluded myelopathy, cerebral infarction and hemorrhage. Abnormal EMG findings showed denervation pattern and suggested anterior horn cell lesion. The diagnosis of electrical injury-induced motor neuronopathy was based on the patient's history, although viral factors could not be excluded.

When an electrical current passes through the body, it may cause various neurologic complications, including cerebral arterial or venous thrombosis, intracranial hemorrhage, brain edema, myelopathy, and peripheral nerve disorders.[1,2,7] These complications could occur immediately and transiently, immediately and prolonged or permanently, or delayed and progressively. Several possible mechanisms of neuronal tissue damage after electrical injury have been proposed, including direct mechanical trauma, thermal damage, vascular damage and electrophysiologic changes.[8] The immediate and transient loss of consciousness and quadriparesis in our patient indicated that the electrical current passed through peripheral nerves and the cervical cord and induced related injury. Despite the input and output of electrical current and duration of contact not being clear in our patient, quadriplegia should result from non-thermal damage of the cervical spine because of the total recovery within 10 days.

The mechanism of recovery from electric shock, followed by deterioration 1 month later, and then recovery after rest is unclear. To determine whether it is similar to the mechanism of post-poliomyelitis syndrome needs further investigation. As the fatigability

and weakness developed after strenuous work and was improved by rest and physical therapy, overuse might have played a role in causing limb weakness in our patient. We hypothesized that overuse might have caused decompensation in previously damaged motor neurons and triggered motor unit degeneration, as is supposed to happen in patients with post-poliomyelitis syndrome.[9] Avoiding overuse and adequate rest might help to prevent or to rescue delayed motor neuronopathy in some electric shock-injured patients.

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