Case Study

Carpal tunnel syndrome after an electrical injury: a case report and review of literature

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Abstract: Introduction: Carpal tunnel syndrome (CTS) is prevalent in workers who utilize hand-held vibration tools, engage in tasks involving repetitive wrist movements, and suffer from wrist overuse. Although electrical injuries involving the median nerve are a relatively rare but plausible cause of CTS, the related literature is limited. Here, we report a case of CTS in which the symptoms developed after an electrical injury, and review the related literature.

Case summary: The patient was a right-handed male electrician who often used hand tools but had no symptoms of CTS before the injury, with the left hand as the point of entry. Typical symptoms of CTS manifested after the electrical injury, and a nerve conduction velocity test confirmed the presence of severe CTS in the left hand. Therefore, we believe that the symptoms can be largely attributed to the electrical injury.

Conclusions: The available literature supports the occurrence of delayed compressive neuropathy caused by scarring from substantial cutaneous burns in patients with electrical injuries. This case shows that electrical injuries may cause CTS in the absence of severe scarring through other mechanisms such as direct injuries to the nerve. Therefore, patients with electrical burns should be routinely examined for peripheral nerve compression symptoms in follow-ups, even when there are minimal cutaneous burns.

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Key words: Burn, Carpal tunnel syndrome, Compressive neuropathy, Electrical injury, Nerve conduction velocity, Occupational medicine

Introduction

Carpal tunnel syndrome (CTS) is a peripheral mononeuropathy of the upper limb caused by the compression of the median nerve as it passes through the carpal tunnel and into the wrist. It is a common condition in working-age people, and occupational activities such as repeated and forceful movements of the hand and wrist and the use of hand-held vibration tools have been identified as risk factors\textsuperscript{1}. Typical symptoms of CTS consist of sensory and motor features such as gradual onset of numbness, tingling, and even pain in the median nerve distribution of the hand, together with evidence of delayed nerve conduction\textsuperscript{2}).

Damages to the median nerve induced by electrical injuries are a plausible cause of CTS. However, the literature regarding the association between electrical injuries and CTS is limited, and most of the previous reports have described cases with relatively severe burns which caused nerve compression due to scarring\textsuperscript{3,4). Here, we present the case of a patient with CTS who developed symptoms af-
ter an electrical injury with minimal burns, and we also review the relevant literature to discuss the possible mechanisms contributing to the case. We aim to demonstrate the plausibility of a low-voltage electrical injury causing CTS without significant burns that lead to scarring of peripheral tissue.

Case Report

A 49-year-old male was brought to the emergency department of a medical center after suffering from an electrical injury. The patient was right-handed and had been working as an electrician for more than 30 years, with 4 years at his current job at a resort. This work primarily involves weight-bearing, climbing up and down, and using hand tools to repair machines and electronics such as lights and air-conditioners. Tools used include screwdrivers, drills, wrenches, scissors, etc., and only a few are electrically powered. He works 8-9 hours per day and sometimes up to 12 hours with rotating shifts. The patient stated that he does not routinely work with his hands outside of his job, and had no systemic diseases that may involve the nerves such as diabetes and thyroid disease.

The patient was injured from a 220 V alternating current while repairing a machine on a rainy day. While holding a pair of scissors in his right hand, he accidentally touched a bare cable with his left hand. He wore rubber shoes but did not wear gloves. The patient lost consciousness and fell down backwards after being shocked, but regained consciousness after hitting his back on a cabinet. He was sent to the emergency department, where a first-degree burn approximately 1 cm in diameter was observed on his left hand, which was most likely caused by the electrical injury. His laboratory tests were generally within normal ranges, except for a slightly elevated myoglobin level, and imaging studies revealed normal findings. He was discharged on the same day after being diagnosed with an electrical injury. After discharge, he received follow-ups in the outpatient department as suggested.

A few days later, the patient developed bilateral lower leg soreness and pain and returned to the hospital. Mild rhabdomyolysis was suspected on the basis of these symptoms and the elevated level of myoglobin noted in the emergency department, and he rested for 2 months as suggested. However, during these 2 months he gradually developed numbness in both hands, causing him to wake up once or twice a night due to discomfort. After 2 months of rest, he returned to the same job, but the numbness in both hands progressed. He visited the occupational medicine outpatient department where the physical examination showed a positive Phalen’s test but not Tinel’s sign. A nerve conduction velocity (NCV) test showed bilateral CTS (Fig. 1) and bilateral cubital tunnel syndrome (Fig. 2), and the NCV test result was near the lower normal limit. Because of the severe numbness in both hands, he was treated with physical therapy and occupational therapy for about half a year. Following significant improvements in CTS symptoms, the patient was able to return to work.

Discussion

The individual predisposing factors of CTS include smoking, diabetes, obesity, hypothyroidism, and female sex. Occupational risk factors include repetitive use of the hand such as heavy manual work, working with vibrating tools, and highly repetitive tasks. Typical clinical symptoms of CTS include gradual onset of numbness, tingling, and even pain in the median nerve distribution of the hand, together with evidence of delayed nerve conduction. Our case demonstrated these typical clinical presentations.

The severity of an electrical injury depends on the pas-
sage of electric current through the body and other factors such as the type of circuit and current, the voltage of the circuit, the resistance of tissues, the amperage of the current, the pathway of the current, and the duration of contact\textsuperscript{6,7}. Charged particles collide with molecules in the tissue, losing energy in the form of heat. The amount of energy dissipated as heat by the electric current is given by the law \((\text{current})^2 \times \text{resistance} \times \text{time}\), and most of the tissue damage is caused by the disruption of cell membranes\textsuperscript{8}. Electrical injuries are generally classified as those caused by high voltage (>1000 V) or low voltage (<1000 V);\textsuperscript{9} our case was in the low-voltage range (220 V).

Compressive neuropathy, especially CTS, is caused by direct pressure on a nerve and is not uncommon after electrical burns\textsuperscript{10-12}. In a 17-year review of burn unit admissions, 22% of the patients with electrical injuries were found to have permanent nerve damage. The upper limb was the most frequently injured, and the median and ulnar nerves are usually affected\textsuperscript{9}. In a retrospective study aiming at identifying CTS following different types of burn injuries among burn center patients, 9 of the 28 patients with burns in the upper extremities had electrical burns\textsuperscript{9}. In another study on patients requiring nerve decompression secondary to thermal or electrical burns, CTS was the most common cause of compression, accounting for 46% of the cases\textsuperscript{9}. Four of the patients had electrical injuries, with three patients having high-voltage injuries and the fourth having a low-voltage injury. During the 4-year follow-up period, patients with electrical injuries had an average of 1.5 compressed nerves. CTS accounted for five (83\%) of all the nerve decompressions in electrical burn injuries, with the remaining one being attributed to radial tunnel syndrome. Therefore, some clinicians have recommended fasciotomies for patients who have high-voltage (or associated crush) injuries with entrance or exit wounds in the extremities, and release of the carpal tunnel is practiced routinely in some services for cases with electrical injuries\textsuperscript{9}.

A low-voltage electrical injury may also lead to neuropathy. In a study of 648 burn patients, 16 patients developed mononeuropathy after a low-voltage electrical injury\textsuperscript{10}. One patient with only a 2\% superficial partial-thickness burn on the right forearm developed median mononeuropathy on the wrist and a distal slowing of the ulnar nerve\textsuperscript{10}. Even without significant cutaneous burns, low-voltage electrical injuries may also lead to peripheral neuropathy\textsuperscript{10}. Therefore, in addition to the compression caused by scar tissue after burns, several mechanisms have been proposed to explain the injury to neuronal tissue after electrocution, such as thermal damage, sympathetic stimulation, vascular damage, histological and electrophysiological changes, and direct mechanical trauma\textsuperscript{14}. It has been shown by experiments that electrocution can lead to nerve dysfunction through both thermal and non-thermal mechanisms. The possible mechanisms that reflect the temporal relationship with the electrical injury include thermal injury to the perineural tissues, which leads to a progressive perineural fibrosis with compressive neuropathy, and direct thermal damage to the nerve resulting in necrosis\textsuperscript{10,15}.

In a study on electrical injuries with burns, compressive neuropathy was diagnosed 46 to 1,530 days after the burn injury, and CTS was the most common neuropathy, accounting for 46\% of all nerve decompressions\textsuperscript{9}. Our case developed numbness and was diagnosed with CTS within this time range.

Our patient suffered from electrical injuries when he touched a bare cable with his left hand while holding a pair of scissors with his right hand on a rainy day. Electricity passes through the path of least resistance, and the resistance of a nerve is lower than that of a muscle or bone\textsuperscript{10}. Accordingly, the current most likely entered through his left hand and exited through his right hand. Therefore, it is reasonable that both his hands were af-

**Fig. 2.** Bilateral cubital tunnel syndrome observed in the nerve conduction velocity test.

(A) Right ulnar nerve
(B) Left ulnar nerve
fected. When an electric current passes through a narrow pathway, such as the carpal tunnel or the cubital tunnel, the increased resistance and longer contact time may cause more energy to be transmitted as heat. Because more electrical energy is transformed to heat in high resistance areas, the damage caused by an electrical injury might be much more severe in the carpal and cubital tunnels than in a direct nerve injury. The heat energy generated when electricity passes through the narrow pathway such as the carpal tunnel or cubital tunnel may lead to perineural fibrosis and nerve damage. The NCV test showed damages to both the median and cubital nerves, and supports our interpretation. Because it is difficult to recover from such damage, the patient received regular follow-ups at our outpatient clinic.

Conclusion

Based on a review of the literature, we conclude that an electrical injury may cause neuropathies such as CTS, potentially through pathways such as perineural fibrosis and nerve necrosis. Moreover, a low-voltage electrical injury may still lead to CTS, even without cutaneous burns that cause sufficient scarring to induce remarkable nerve compression. Therefore, patients with electrical burns should be routinely examined for peripheral nerve symptoms during subsequent follow-ups.

Consent to Publish

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor of this journal.

Authors’ Contributions

Y-SW and H-RG were responsible for clinical management of the case and drafting the manuscript. Y-SW, C-TO, Y-YH, and T-WH contributed to the review of the literature and interpretation of data. All authors participated in the writing of the manuscript and have read and approved the final manuscript.

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Conflicts of interest: None declared.

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