TriNeural Injury to the Right Hand After Domestic Electrocution

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Electrocution injuries account for a significant amount of burns unit admissions each year, and can be fatal. These injuries are divided into high-voltage (over 1000 volts) and low-voltage (less than 1000 volts) injuries, with lightning strikes (greater than 100 million volts) considered separately. Although the majority of electrocution injuries are of low voltage, most of the published reports concern industrial/high-voltage and lightning injuries. This disparity may trivialize low-voltage injuries in the minds of clinicians. We report a rare case of trineural (median, ulnar, and radial) injury in an upper limb after a low-voltage electrocution, and discuss the pathogenesis, investigation, and treatment of these injuries. (J Burn Care Res 2013;XXX:00–00)

Electrocution injuries account for between 3 and 7% of burns unit admissions each year[1,2] and 1% of accidental household deaths in the United States.[2] Conventionally, these injuries are divided into high voltage (over 1000 volts) and low voltage (less than 1000 volts). Lightning strikes, which exceed 100 million volts, are in a separate category. The majority of published reports on electrocution injuries concern high-voltage electrocution, and the effects of these injuries are often more obvious and injurious than those of lower voltage. However, low-voltage injuries can have a significant morbidity both physically[3,4] and psychologically,[5–7] and in fact account for 60 to 70% of electrocution admissions to burns units.[2] Therefore, they should not be underestimated by clinicians. We present the second reported case of a domestic, low-voltage electrocution causing a "trineural" injury to the patient's upper limb—that is, involving the median, ulnar, and radial nerves.

CASE REPORT

A left-hand dominant 54-year-old woman was referred to our burns unit 3 days after an electrocution injury sustained when changing a domestic light bulb with wet hands while standing on a chair. She felt an electric current pass through her hand, and fell off the chair. At the time of the injury she suffered amnesia of the event, and was unable to accurately relate the events to her regional emergency department. The cutaneous burns sustained on her hands were diagnosed as friction burns from the fall, and treated with dressings. There is no documentation of any parasthesia of either upper limb at that time, and no blood tests or other investigations were performed. After 3 days, she had regained full memory of the events and was able to relate them to our service on referral from the emergency department. Her medical history was unremarkable except for a diagnosed anxiety disorder—she did not have diabetes and did not suffer from any neurological disorders. As the patient was alone at the time of the injury, any third-party corroboration of the event was not possible.

On examining the patient it was found that she had sustained a partial-thickness burn to the dorsum of the little, middle, and ring fingers of her right hand, and also to her left little finger. She complained of parasthesia of the entire palmar aspect of the right hand, as well as of the radial aspect of its dorsum. Sensation was normal in the left hand. She had significant weakness in flexion and extension of her interphalangeal and metacarpophalangeal joints of the right hand, but normal power in the wrist, elbow, and shoulder. She had no motor deficit of the left upper limb. Both upper limbs were well perfused, and there was no clinical evidence of compartment syndrome.

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Hematological investigations showed a mildly increased level of creatine kinase (474 U/L, normal <400 U/L) and a normal Troponin T. She had no ECG changes suggestive of cardiac injury or dysrhythmia, and a CT of her brain was normal. She remained an inpatient for 1 night and was referred to Occupational and Physiotherapy department for assessment of hand function and commenced on hand therapy. She was then discharged home with hydrocolloid dressings for her burns and a static volar resting splint in the position of function. At this point the patient had a full passive range of motion of all hand joints in the affected limb, but no active range of motion was observed.

Nerve conduction studies performed at 2 weeks postinjury showed absent sensory and motor responses of the median, ulnar, and radial nerves on the right side, and reduced motor amplitude of the left ulnar nerve, with absent sensory responses. The detailed results of these tests are reproduced in Tables 1 (motor) and 2 (sensory). The motor responses were measured from wrist to abductor pollacis brevis for the median nerve, and wrist to adductor digit minimi for the ulnar nerve. The sensory responses were measured from wrist to second digit for the median nerve, wrist to ulnar palm for the ulnar nerve, and forearm to thumb for the superficial branch of the radial nerve. These findings are consistent with peripheral nerve damage distal to the dorsal root ganglion and direct electrical injury to the nerves, perhaps together with a superimposed compression neuropraxia. On review at 3 months postinjury, the patient still suffered from a right-sided "glove" neuropathy, which has improved. Hand mobility and function was improving slightly with hand therapy.

Table 1. Nerve conduction study results demonstrating absent motor nerve responses of the median and ulnar nerves on the right side, and a reduced motor amplitude of the left ulnar nerve

Motor Nerves	Latency (msec)	Amplitude (mV)
Right median nerve (wrist to abductor pollacis brevis)	Absent	Absent
Left median nerve (wrist to abductor pollacis brevis)	2.8*	8.0†
Right ulnar nerve (wrist to adductor digit minimi)	Absent	Absent
Left ulnar nerve (wrist to adductor digit minimi)	1.50‡	2.5§

*Mean ± standard deviation 2.78 ± 0.41, normal limit 3.60.
†Mean ± standard deviation 14.62 ± 8.45, normal limit 500.[19]
‡Mean ± standard deviation 2.03 ± 0.24, normal limit 2.52.
§Mean ± standard deviation 11.49 ± 2.51, normal limit 5.00.[19]

Further nerve conduction studies were performed at 6 months postinjury, and these demonstrated absent sensory responses and almost unrecordable motor responses at the median and ulnar nerves. Needle electromyelogram at this time showed no recordable motor unit action potentials in the median nerve distribution, and only one or two units for the ulnar nerve—the neurophysiologists stated "findings suggest severe functional disturbance of all nerves in the right hand." The two sets of nerve conduction studies were performed in the same laboratory at the same settings.At 14 months after the electrocution injury, the patient had suffered considerable wasting of the hand muscles, and the hand was consistently cold. Again the patient was referred for electrophysiological testing but the neurophysiologist did not perform them given the clinical findings, and was of the opinion that that at this point of the patient's clinical course "functional recovery of the hand is unlikely." Again at this point the patient was referred to Occupational and Physiotherapy department, but no further improvements were seen in hand function, with minimal active range of motion elicited.

DISCUSSION

The complications of electrocution injuries can be legion, and range in severity from transient to lethal. Although cardiac or respiratory arrest can occur at injury, later complications can include rhabdomyolysis and renal injury, compartment syndrome and limb injury, cardiac dysrhythmia, pulmonary edema, cholelithiasis, nerve injuries, cataracts, uveitis, and retinal lesions.[4,8–12] Skeletal muscle and soft tissue injuries frequently need repeat debridement, although this is more common in high-voltage injuries,[13] and this may be because of a small vessel vasoconstriction

Table 2. Nerve conduction study results demonstrating absent sensory responses of the median, ulnar, and radial nerves on the right side, with absent left-sided ulnar nerve sensory response

Sensory Nerves	Latency (msec)	Amplitude (mV)
Right radial nerve (forearm)	Absent*	Absent†
Right CTS – second finger–wrist	Absent	Absent
Right CTS – Palm (U)–wrist	Absent	Absent
Left CTS – second finger–wrist	2.6‡	6.3§
Left CTS – palm (U)–wrist	Absent	Absent

*Mean ± standard deviation 1.19 ± 0.14, Normal limit 1.47.
†Mean ± standard deviation 31.06 ± 8.66, normal limit 10.00.[18]
‡Mean ± standard deviation 1.57 ± 0.39, normal limit 2.35.
§Mean ± standard deviation 30.93 ± 12.07, normal limit 10.00.[18]
CTS,

Figure 1. The patient's injured right hand at 3 months past the injury. Note the healed burn to the dorsum of the index finger, the shiny atrophic skin, and the absence of any single clinically appreciable motor nerve palsy.

leading to continuing ischemia without an obvious or reversible cause.[3] There is also a subset of severe injuries in children who make oral contact with household sockets and wiring.[14]

Both the voltage and current can affect the injury, partly by altering the duration of contact. Lee et al[14] postulate that high-voltage electrocutions generate an explosive arc, which repulses the patient from the power source, in contrast to a tetanic contraction "no let go" phenomenon in low-voltage injuries, which leads to longer contact and deeper burns. In contrast, other authors suggest that this difference is because of the nature of the current employed—alternating current causing contracture, and direct current ejecting the injured party from contact.[9]

Furthermore, patients who experience low-voltage injuries may also experience neurological (numbness, weakness, pain) and psychological (anxiety, insomnia, nightmares, flashbacks) problems, which may not develop for up over 2 years after the injury.[7] Beyond the abovementioned injuries, there can also be an economic legacy. Noble et al[5] found that in a survey of patients admitted to a burns unit with electrocution injuries, only 23% returned to the same job as they were previously employed in and 32% did not return to work at all. Theman et al[6] found a 25 to 30% return to work rate in 40 similar patients. It has been suggested that a fear of a repeat injury may be a "roadblock" to return to work,[15] and as these injuries are commonly occupational and often occur in young men,[13] the lifelong repercussions are clear.

Neuropathies such as those described by us are a rare but reported complication of low-voltage electrocution injuries—including facial nerve,[8] hypoglossal nerve,[10] and the nerves of the upper limb.[1] A classification of peripheral nerve injuries possible in electrocution injuries is suggested by Smith et al[1]—1) Delayed Reversible, caused by heating of perineural tissues with subsequent fibrosis and compression; 2) Immediate Transient, because of reversible histological and electrophysiological changes; 3) Immediate Irreversible, from a direct thermal injury to the nerve causing necrosis. The leading cause of type 2 injuries is likely to be "electroporation," which is a loss of cell membrane continence because of the development of pores that allow intracellular contents to leak out, and can lead to findings such as rhabdomyolysis in macroscopically normal tissue.[16,17] This is usually self-limiting, but in serious injuries this may not be the case.[16] Smith et al advocate early release of affected nerves through surgery at the classical points of compression, but as our patient's symptoms improved with conservative measures alone this was not undertaken. A conservative course was also followed in other published reports of neuropathies.[8,10] However, given the deterioration in hand function over the year after our patient's injury, we feel that this may dissuade us from following a similar course in similar patients in the future.

We would advocate nerve conduction studies as an adjunct to the investigation of these patients, after a suitable period (2–3 weeks). However, the limitations of any investigation must be borne in mind. In a recent article, Fish et al[18] reviewed the use of investigations in patients attending a burns outpatient service who had suffered a low-voltage electrical injury. They found that of a wide variety of tests performed (ie, hematological, electrophysiological, radiological) many gave negative results and did not inform patient care: in their study of 37 patients, 13 had an electromyelogram and only four were positive (30.8%), and nine had NCS of which only one was positive (11.1%). Further, they advise against arranging tests of uncertain value because "negative" results may affect the patient's psyche.

The severity of the injury that we present is unusual, as is its localized nature and the seemingly innocuous insult that leads to it. This case highlights one of the many complications of these injuries and emphasizes the importance of complete examination and, where indicated, investigation and appropriate referral of these patients—to limit the long-term physical, psychological, and economic impact of the injury on the patient. The examining physician, whether a first contact or in a referral center, must examine the whole patient thoroughly, and be wary of the "distracting injury." Careful attention should be paid to neurological examination of both the injured region and of the patient as a whole with accurate documentation, and this should be used as a guide to investigations. This careful examination not

a single event, but should be repeated at intervals over the length of follow-up. Further, the patient must be made aware of the potential for late-evolving psychological and neurological complications. It is, however, difficult to find something unless one knows to look for it.

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