

Case Report

A Case Report of Pain and its Treatment Following Nerve Damage Due to Electrical Injury

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Abstract

Background: Electrical injuries, particularly those caused by high voltage, are characterized by multi-organ damage, such as skin burns and cardiovascular, respiratory, nervous, and other system injury. In this case, due to precautions based on his professional experience and knowledge, an electrician could reduce the impact of an electrical injury, resulting in less skin injury, but could not avoid nervous system injury, presenting as visual disturbances, deafness and pain after high voltage electrification. We present his post-injury pain management using analgesic adjuvants.

Case presentation: A 39-year-old male electrician experienced a 5000-volt electrical injury while working. He was able to avoid fatal injuries such as respiratory and cardiac arrest, and sustained no skin burns, but presented with visual disturbances, deafness, pain and paresthesia in both his upper extremities. Considering that the pain and paresthesia were caused by direct electrical damage to nerves, we prescribed analgesic adjuvants (mirogabalin, amitriptyline, clonazepam) as for neuropathic pain, with subsequent improvement in the pain and paresthesia.

Conclusion: High-voltage injury is commonly associated with high morbidity and mortality. In this case, the electrical injury caused isolated nervous system injury. This report shows the efficacy of adjuvant analgesics for the treatment of nerve damage pain after electrical injury.

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Background

Electrical injuries occur when an electrical current passes over or through the body. They range from skin burns to life-threatening multisystem complications with significant morbidity and mortality. Electrical injury is classified divided into low voltage (<1,000-volt) and high voltage (>1,000-volt) types.¹ Electrical injuries in adult populations are most commonly seen in males, and are more commonly related to workplace accidents, with high voltage trauma typically occurring as a result of professional electrical work.¹

High voltage injury might result in severe skin burns, cardiac dysrhythmias, nervous system injuries, respiratory arrest, renal failure and other complications.²

Here, we describe the case of an electrical worker who experienced high voltage electrical injury that caused minimal cutaneous injuries and no lethal injuries due to precautions he took base on his professional experience and knowledge, but which caused nerve damage, including visual disturbances, deafness, and pain, which needed management.

Case presentation

A 39-year-old male electrician attempted to replace a device (load disconnecter) in a high voltage power receiving equipment called a cubicle. Anticipating the potential risk of electric shock, he approached the device with the back of his leather glove-covered hands, for cable work, after a direct current of 5,000-volt had been passed through it as a test. As he got within 2-3 cm of

the device, pain and paresthesia shot through his body, from his right arm, through his chest, to his left arm. Since he approached the device using the backs of his hands, he was thrown away from it by the force of muscular contraction of his upper extremities resulting from the electric current (Fig. 1). He felt a strong impact and fell down. He said it felt like he had received a strong punch to his chest. Although he had shortness of breath, he was able to breathe and went home with severe chest and upper extremity pain, paresthesia, and right-sided visual disturbances and deafness. Two days later, since these symptoms continued unabated, he visited the emergency department of our hospital.

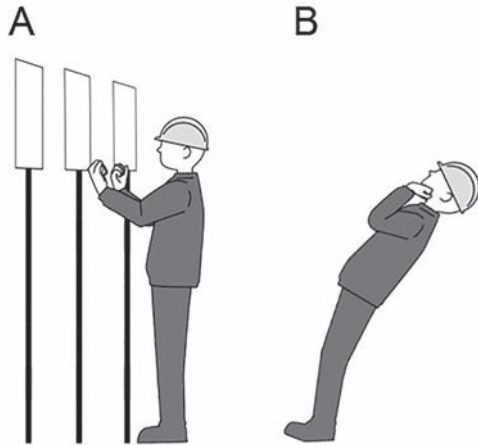


Fig. 1 Assuming the potential risk of electric shock, the patient approached the electrical device using the back of his hands (A). Even though he sustained electrical arc shock, tetanic muscular contraction of his upper extremities as a result of the electric current caused him to be thrown backwards, away from the current (B).

Physical examination revealed almost complete loss of vision in his right eye, which was reduced to only light perception, along with right-sided deafness (a subsequent hearing test showed that he could hear sounds of 6.3 decibels in his left ear but only 85 decibels in his right ear), although only slight redness was observed on the tips of the little fingers of both his hands, with no burn marks. He had bilateral muscle and bone pain, and paresthesia from his shoulders to his fingertips and in his chest. He showed an obvious decrease in grip strength, but no weakness in his biceps, triceps, or deltoids. His electrocardiogram and laboratory testing showed no obvious abnormalities. He subsequently underwent an echocardiogram, which showed no abnormal findings. A cranial MRI image showed no findings suggestive of deafness or visual disturbances, and there were no other notable findings. Chest/abdominal CT scan was also unremarkable. On the same day, he consulted an ophthalmologist, and a visual acuity test revealed vision of 0.1 in the right eye and 1.2 in the left eye, with concentric peripheral visual field constriction.

He was referred to a neurologist for further examinations and management of pain and paresthesia. Nerve conduction studies showed no evidence of neuropathy.

Cervical spine MRI showed cervical spondylosis and ossification of the yellow ligament with slight spinal stenosis. These findings were, however, unlikely to cause severe pain and paresthesia in the upper extremities. He was prescribed pregabalin 300 mg/day, which helped him sleep through the night, although the pain and paresthesia persisted (NRS 7/10). Hence, he presented to our pain clinic 62 days after the injury. Since he subsequently complained of drowsiness and dizziness, pregabalin was reduced to 150 mg/day, and amitriptyline (10 mg/day) and mexiletine (150 mg/day) were prescribed. One week later, he reported that the pain was relieved by amitriptyline (once a day) but not mexiletine (3 times a day), as determined by the fact that he felt pain relief only at night after taking amitriptyline, with the pain returning by the afternoon. Due to persistent drowsiness and dizziness, pregabalin was discontinued and mirogabalin (7.5 mg/day) was prescribed, to minimize the side-effects. Additionally, clonazepam (0.5 mg/day) was added to treat his occasional anxiety and insomnia. Since his pain and paresthesia were mostly controlled (NRS 3/10) with amitriptyline (10 mg/day), mirogabalin (7.5 mg/day) and clonazepam (0.5 mg/day), this prescription is still currently ongoing.

We explained to the patient that cognitive behavioral therapy will be the next step in his recovery.

Discussion

The electrical injury in this case was characterized by the absence of burns. Thermal injury, caused by conversion of electricity into heat as the current passes through body tissues, is an indirect effect of electrical injury.³ Heat from the electric current is determined by Joule's law, defined as the product of current, resistance and contact duration. According to Ohm's Law, the resultant current is calculated as voltage divided by tissue resistance, which varies significantly between tissues. Thus, exposure of different parts of the body to the same voltage generates different current and heat, resulting in different degrees and types of damage to different body parts.⁴ The skin has intermediate resistance, while bones, fat and tendons have the highest resistance.⁴ The weather when the patient was injured was very dry and windy, characteristic of Japan's winter weather. His hand skin, which was covered by a leather glove, offered higher resistance. Since tetanic contraction of the upper extremities threw him away from the current source, he could minimize the damage due to his professional experience and knowledge. Thus, the contact duration seemed to have been brief. Additionally, since according to Ohm's and Joule's law, high resistance reduces current flow through the body, we speculated that the lower current and brief contact duration resulted in almost no thermal damage in this case. However, due to the extremely low resistance in nerves,⁴ he suffered greater nervous system injury. These occurred probably via the direct effects of the current on body cells, since electric current can affect cell membranes, abruptly altering their electrical properties (cellular depolarization) and causing direct

cell injury by forming pores in cell membranes (cellular electroporation).³ These changes likely caused the long-lasting, visual disturbances, deafness, pain and paresthesia, which have not improved even after eight months. Even without direct contact with the electrical source, very high voltage currents can cause electrical injuries via an arc current through nominally high-resistance air.⁵ In this patient, since the current passed from the right hand to the left hand, the arcing current might have flowed through the right eye and ear when the right upper extremity was flexed by tetanic contraction. We also speculate that dyspnea immediately after the electric shock was due to thoracic muscle tetany. Besides indirect non-thermal injury, electrical injury can also cause injury to muscle or bone from tetanic contraction, because tetany can generate muscular contractions powerful enough to cause orthopedic trauma, rhabdomyolysis, and muscle necrosis.⁶ Since most electrical injuries result from grasping an electrical source with the hands, the resultant tetany leads to the inability to release the contact source,⁷ with the additional possibility of respiratory arrest due to thoracic muscle tetany.^{2,8} The average current that causes tetany and prevents release of the electrical source is termed the “let-go” threshold. A current of 20 to 50 mA causes respiratory muscle tetany, and 50-100 mA causes ventricular fibrillation (Table 1).^{4,9,10}

Table 1 Physical effects of different intensities of electrical current

Current (mA)	Physical effects
1 mA	Tingling sensation (nearly imperceptible)
3-5 mA	“Let-go” threshold in children
6-8 mA	“Let-go” threshold in women
7-9 mA	“Let-go” threshold in men
16-20 mA	Tetany (skeletal muscles)
20-50 mA	Tetany (respiratory muscles; respiratory arrest)
50-100 mA	Threshold of ventricular fibrillation
>2 A	Asystole

Based on data from *References 4, 9, 10*

In this case, since there were no burns to suggest thermal injury, his neurological damage was presumed to be caused by direct neuronal injury. We selected analgesic adjuvants to treat the nerve injury pain, as in neuropathic pain. Tricyclic antidepressants and pregabalin are highly recommended medications for neuropathic pain.¹¹ We chose amitriptyline, a tricyclic antidepressant, and instead of pregabalin, we used mirogabalin, an $\alpha 2\delta$ -1 calcium channel subunit ligand, which causes less side-effects, according to the guideline for the pharmacologic management of neuropathic pain by the Japan Society of Pain Clinicians.¹² Clonazepam was also prescribed to improve his symptoms and anxiety.

Some electrically injured patients experience chronic pain that requires a multimodal approach to pain management.¹³ The common medications include opioids and acetaminophen in the acute phase, and analgesic adjuvants and NSAIDs during rehabilitation and at discharge. During rehabilitation, opioids and other medications are often tapered and analgesic adjuvants are added.

The common non-pharmacologic modalities are heat treatment and massage therapy in the acute phase, and massage therapy and cognitive behavioral therapy during rehabilitation.¹² Since the patient consulted our clinic about 2 months after his injuries, we decided to use analgesic adjuvants to treat his neuropathic pain.

There are also several case reports of PTSD following electrical injury in the literature.¹⁴⁻¹⁶ PTSD is a significant problem leading to poor cognitive performance and decreased ability to return to work.¹⁶ Hence, patients should be given long-term psychological care in addition to treatment for chronic pain. In this case, the patient has been unable to return to work since his visual disturbances and deafness have not improved. He is still receiving cognitive behavioral therapy and analgesic adjuvants for controlling chronic pain.

Conclusion

In this electrical injury case, although the patient’s precautions stemming from his professional experience and knowledge contributed to avoiding lethal injury, long-term pain management will probably be necessary due to the direct and/or indirect nerve damage resulting from his injuries. We achieved effective pain management with analgesic adjuvants, which will be continued in combination with cognitive behavioral therapy.

Abbreviations

NRS: Numerical rating scale; PTSD: Post-traumatic stress disorder; NSAID: Non-steroidal anti-inflammatory drug.

Consent to Publish

Written informed consent was obtained from the patient for publication of this case report.

Authors’ contributions

MHY was in charge of the patient’s treatment, and drafted the manuscript. SS made critical revisions to the text. All authors approved the final manuscript.

Acknowledgement

Not applicable

Disclosure

The patient provided informed consent for this report. Ethical approval for this study was waived as it is not considered human medical research.

Conflicts of interest: The authors declare that they have no competing interests.

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