A Consequence of an Electrical-Burn Injury: Atrial Fibrillation

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ABSTRACT

Cardiac arrhythmias following electrocution injuries can accompany high-voltage or high- intensity currents. Contributing factors to electrical hazard are the type of current, voltage, resistance, and duration of contact and pathway through the body. It is important to monitor for delayed arrhythmias in patients with an electrical injury. We describe a case of a 52-year-old man who presented after an electrical shock injury while grabbing a 5,000-voltage wire at work. In this case report, we discuss the presentation, management, and follow-up recommendations for this type of injury.

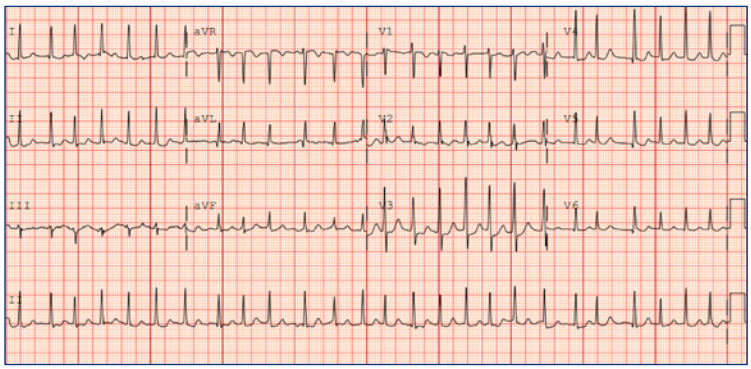
KEYWORDS: arrhythmia, electrocution injuries, trauma

CASE PRESENTATION AND SUMMARY

A 52-year-old male electrician with a history of uncomplicated rheumatic fever and no cardiac medical history presented to the Emergency Department after sustaining an electrical shock injury while grabbing a high-voltage electrical wire. The patient was working at the local airport and arrived to the Emergency Department approximately 20 minutes after making contact with a wire at work with both hands and experiencing 5,000 V that traveled from his left hand to his right hand. He reported holding on to the wire for approximately 15 seconds and then letting go and falling directly onto his face. There was no reported loss of consciousness. He stated he felt his heart racing. The patient denied any associated symptoms of chest pain, diaphoresis, nausea, or vomiting prior to arrival. His social history was significant for drinking several beers per day, but he had no history of a withdrawal. He is a non-smoker. Given his history of childhood rheumatic fever without carditis, he had undergone multiple surveillance echocardiograms, the last of which was eight years ago.

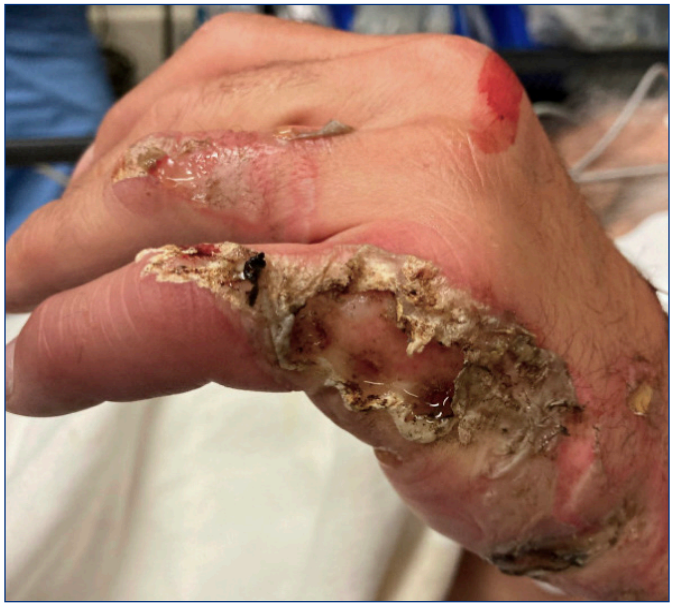
Lab work performed included complete blood count (CBC), basic metabolic panel (BMP), liver function tests (LFTs), all of which were within normal limits. The patient had a normal creatine kinase (CK), and his urinalysis (UA) did not show myoglobinuria. His initial high sensitivity troponin was < 3 ng/L (ref: 3–57 ng/L), and his initial EKG revealed atrial fibrillation with a rapid ventricular rate (RVR) in the 150s–160s

Image 1. Patient's presenting EKG (atrial fibrillation with rapid ventricular response)



(**Image 1**). Point-of-care ultrasound (POCUS) echocardiography performed by the ED provider was limited, but a grossly normal ejection fraction was appreciated, and no pericardial effusion was detected. The patient sustained bilateral hand burns with deep burn at the first webspace of the left hand (**Image 2**). The patient had several abrasions and lacerations to his face that were repaired.

Image 2. Patient's electrocution burns on presentation to the Emergency Department



A computed tomography (CT) scan of his head, cervical spine, face, chest, abdomen and pelvis was performed given the significant mechanism of injury. CT imaging was remarkable only for a nasal bone fracture. Atrial fibrillation with RVR was treated with two doses of metoprolol 5mg intravenously (IV), followed by the initiation of a diltiazem drip with a heart rate goal of <120 BPM. He was admitted to the trauma intensive care unit (TICU) due to the severity of his burns and the persistent rapid ventricular rate. He was further treated with amiodarone 150mg IV in the TICU, as the inpatient team aimed to obtain rhythm control in addition to rate control.

DISCUSSION

Electrical burns account for 4–5% of all burns treated in a medical setting.[1] While there has been improvement in injury prevention and implementation of safety protocols at the workplace, electrical injuries in the adult population are most often work-related and are the fourth leading cause of traumatic work-related death in the United States, with approximately 1,000 work-related deaths occurring annually.[2,3] Accidental high-voltage electrical injuries comprise approximately 400 of these cases.[1] Despite the rarity of electrical injuries, the morbidity and mortality are high – ranging from 10–30% – and it is imperative to obtain the voltage of injury from the patient or their workplace upon arrival to the Emergency Department.[4]

The magnitude of electrical hazard is dependent on multiple factors, including voltage (V), resistance (R), type of current (alternating or direct), duration of contact, and the path that the electricity takes throughout the body.[5,6] Current intensity (I), which is measured in Amperes (A), contributes to the severity of the electrical injury. This considers both voltage and resistance as described by Ohm's law [I = V/R]. While there are variable definitions of voltage magnitude in the literature, it is generally accepted that <600 V is low and >1000 V is high.[2,7,8] The difference between Alternating Current (AC) and Direct Current (DC) is important to consider as well. In AC the direction of flow of electrons changes on a cyclical basis, whereas in DC the direction of flow remains constant. Common household breakers range between 15–20 A, and the threshold for ventricular fibrillation is as little as 50–100mA, demonstrating the possible danger of domestic electrical injury. AC is thought to be about 3-5 times more damaging than DC. This is because AC can induce muscle tetany due to strong muscle contractions, which does not allow a person to easily let go of an electrical source.[2] The patient in this case report described this sensation on presentation. He felt that his hands were "stuck" holding onto the wire. Electrical energy, which is converted into thermal energy, lead to the burns on our patient's hand (**Image 2**). This occurs, in part, due to the body becoming part of the circuit.[2]

High-voltage injuries, as seen in our patient, should be treated as multi-system trauma. High-voltage electrical injuries generally have significantly greater morbidity as seen by larger numbers of surgical procedures required, higher rates of medical complications, and multiple post-traumatic injuries secondary to additional falls in these injuries.[8] While low voltage is associated with fewer fatalities, low-voltage AC injuries are particularly arrhythmogenic and can cause ventricular dysrhythmias.[8]

The exact rate of dysrhythmias following an electrical injury is unknown.[4] The most common arrhythmias described in the literature include sinus tachycardia, premature ventricular contractions (PVCs), premature atrial contractions (PACs), and sinus bradycardia.[4] However, cases of supraventricular tachycardia, atrial fibrillation, ventricular tachycardia, ventricular fibrillation, and asystole have been documented as possible sequelae of electrical injury. The majority of arrhythmias occur shortly after the electrical shock and, more rarely, there are delayed ventricular arrhythmias. The most common fatal dysrhythmia is ventricular fibrillation. Asystole is associated with lightning or high-voltage injuries.[4,8-13] Other arrhythmias that occur include first- and second-degree AV block and bundle branch blocks.[13]

While the pathophysiology of these arrhythmias is not well understood, it is thought that the development of an arrhythmia may occur due to changes in extracellular potassium concentration, an increased Na+/K+ pump concentration, and may be associated with transient and localized changes in sodium and potassium transport, potassium concentration, and membrane potential. This is proposed to lead to enhanced automaticity of the myocardium.[9]

Although most arrhythmias can be diagnosed on initial EKG, there have also been case reports of delayed arrhythmias reported in the literature.[12,13,14] A striking case reported by Sharma et al described a 24-year-old male who sustained a 220–240V electrical injury in his home. On arrival at the hospital, he was found to have first-degree heart block which, over the next 24 hours, deteriorated into Mobitz type 1 heart block, complete heart block, and then eventually ventricular fibrillation requiring electrical cardioversion.[14] Karatas et al reported a patient who developed pulseless ventricular tachycardia within 24 hours of hospitalization, which ultimately was terminated with DC shock.[15] More recent systematic reviews suggest that clinically relevant arrythmias not noted on initial presentation to the hospital are rare in patients after an electrical accident, and that clinically relevant arrhythmias are likely to be diagnosed on presenting EKG.[10,16-18]

Due to the high incidence of dysrhythmia, autonomic dysfunction, and delay in presentation of a fatal arrhythmia, current recommendations suggest continuous cardiac monitoring for >24 hours for all patients except those who sustain low-voltage shocks, are asymptomatic, have no loss of

consciousness, and have a completely normal physical exam, including a normal skin exam without burns. For patients with high-voltage injuries, and who have EKG abnormalities, loss of consciousness, or have cardiac risk factors, 24 hours of cardiac monitoring is indicated.[17] Because troponin is specific to the cardiac muscle, it seems reasonable to monitor patients with an elevated troponin, though there have not been specific studies looking at troponin increase in these settings.[1] Elevations in creatinine kinase on the other hand may be confounded with peripheral skeletal muscle injury, which are common in electrocution.

Atrial fibrillation after an electrocution injury is uncommon.[19] The management of atrial fibrillation with RVR secondary to electrical injury follows standard treatment guidelines and requires a combination of rhythm and rate control. Our patient's rhythm converted into normal sinus rhythm 12 hours after he arrived to the hospital. An inpatient echocardiogram demonstrated mild pulmonary hypertension and elevated right-sided filling pressures, but demonstrated no cardiac wall motion abnormalities or pericardial effusion. He did not have recurrence of his atrial fibrillation during his hospitalization and was discharged several days later. He was not started on any rate or rhythm controlling medications on discharge, nor was he started on anticoagulation medications. During a follow-up cardiology appointment one month after discharge, he was noted to be in normal sinus rhythm and was without any cardiac sequelae.

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