A Case Report of Survival of Young Adult Following Asystole after Sustaining High Voltage Electric Current

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Abstract
A prolonged cardiac arrest is usually associated with high mortality and revival after prolonged CPR is usually associated with neurological sequelae. A case of cardiac arrest caused by electric shock, who recovered after resuscitation efforts, defibrillation without sequelae is reported by this document. The main reason was timely management by an Advanced Cardiac Life Support (ACLS) trained physician.

Keywords: electric current, cardiac arrest, CPR, neurologic deficit

Key Messages: Vigorous step wise timely ACLS protocol may result in better outcome, especially young patient after electric shock.

Introduction
Electrical injuries, a relatively common form of mechanical trauma and are often associated with a high morbidity and mortality. Adult electrical injuries typically occur in occupational settings, whereas children are mainly injured in the household setting and all are accidental thus preventable.

There are mainly two types of electric current: Direct current and alternating current. The direct current (DC) will throw away a person from the source after one shock, whereas Alternating current (AC) causes muscle spasms, thus prolong period of contact and increased the extent of an injury. The electrical current of any type may cause irregular heartbeat or stop the heart entirely.

A prolonged cardiac arrest is usually associated with high mortality and revival after prolonged CPR is usually associated with neurological sequelae. Here, we report a case of cardiac arrest caused by electric shock, who recovered after resuscitation efforts, defibrillation without sequelae.

Case History
A 24 years old male working on a construction site as a labourer, presented to our hospital in the emergency department in an unresponsive state at 4:50 pm of 9th July 2018 in a private vehicle. It took 12 minutes for them to reach the hospital after sustaining the electrical injury. With no history of seizures or abnormal body movements
after sustaining the shock. His past medical history and family history had nothing of significance.

On examination, his GCS (Glasgow Coma Scale) score was 03. He had no spontaneous respiration and heart rhythm, no response to verbal stimuli, and had fixed and dilated pupils. Immediately chest compression was started and the airway was secured with a cuffed endotracheal tube size 8 mm. The patient was managed as per the latest American Heart Association Cardiopulmonary Guidelines of 2018.

The first cardiac monitor rhythm was ventricular fibrillation. Defibrillator shock of 200J was performed twice. Injection adrenaline 01 mg was given along with a 02 minutes CPR in between, after the second shock, as per American Heart Association guidelines. Asystole was noticed after 10 minutes of resuscitation. Injection adrenaline 01 mg was given twice along with 02 minutes CPR in between.

The patient started gasping after 32 minutes of no traceable cardiac activity. He presented the successful return of circulation (ROSC) with the heart rate of 123bpm and blood pressure 112/70mm Hg after resuscitation. Cardiac monitor showed sinus rhythm. Pulse oxygen saturation was 99% at FiO2 50%.

Intra Venous lines were inserted, blood samples were collected for laboratory investigations and Intra Venous fluid bolus of 500 ml of normal saline was given. The ABG analysis (Arterial Blood Gas analysis) showed respiratory acidosis which was managed by 50 ml of Injection Soda bicarbonate.

After resuscitation, the Foleys catheter was inserted and 300 ml of urine output was noted initially. Continuous input/ output charting was done and fluid maintenance was done using normal saline in view of suspected muscle injury. Adequate urine output was observed.

Artificial mechanical ventilation with CMV mode was started. Injection fentanyl and Injection Rocuronium was given to sedate. All routine blood investigations along with myoglobin and CPK levels were sent to the laboratory. The 12 lead electrocardiogram revealed sinus tachycardia. Bedside echocardiography revealed an ejection fraction of 60% and no wall motion abnormality. A NCCT (non-contrast computed tomography) scan head was performed, which showed no evidence of intracranial haemorrhage, infarction or any cerebral oedema. The patient was later shifted in intensive care unit. The next 24 hours showed stable hemodynamic, blood reports, and imaging. Successful weaning was possible on day 3 of hospitalization. After weaning no neurological deficit except agitation and aggressive behaviour after withdrawal of sedation and muscle relaxant. It was controlled with injection midazolam as per requirement. The patient was discharged on request on day 05 in stable condition. On follow up at 2 weeks after discharge no neurological or intellectual deficits detected.

**Discussion**

Cardiac arrest due to any cause is a leading cause of premature death throughout the world, survival from which is often less than 5%.[1] The severity of injuries depend on voltage, duration of contact, tissue resistance, and the path of current.[2] There are several mechanisms that are involved in injury: direct tissue damage from electrical energy, thermal damage from heat created by tissue resistance, cardiac dysrhythmias and mechanical injury induced by a fall or tetanic muscle spasm.

Along the lines of managing a case of trauma, electric injury patients are managed. As a part of the primary survey ABC should be performed which includes: airway, breathing, circulation and inline immobilization of the spine should be performed. Hidden injuries should be evaluated in all. American Heart Association (AHA) promotes the concept of the chain of survival to improve outcomes of cardiac arrest.[3] Timely and trained in cardiopulmonary resuscitation, chest compressions and defibrillation improves the survival.[4] As a part of initial resuscitation fluid
replacement is the most important aspect. Since, electrical injuries cause massive fluid shifts with extensive tissue damage and acidosis as is the case with conventional thermal injuries. Therefore, monitoring a patient's hemodynamic is crucial. For this input output charting is very important; thus a Foley catheter. This charting helps in monitoring output and, therefore, tissue perfusion. The documented cardiovascular effects following electric injury are sudden death (ventricular fibrillation, asystole) chest pain, dysrhythmias, ST-T segment abnormalities, bundle branch block, myocardial damage, hypotension (volume depletion) and hypertension (catecholamine release). Death following high and low voltage electrical injuries is most commonly caused by ventricular fibrillation. It is recommended in literature that cardiac monitoring for as long as 24 hours after electrical injuries to prevent deaths from delayed arrhythmias especially in children. But cardiac monitoring is not recommended; even after 120 or 240 V injuries in asymptomatic patients or patients without any cutaneous thermal burns having normal ECG (Electrocardiogram).

Typically prolonged cardiopulmonary resuscitation is associated with poor neurological outcomes and reduced long-term survival. In our case, the patient had a cardiac arrest due to electric shock. High-quality CPR and early defibrillation by the emergency department team contributed to the successful resuscitation and favorable neurological outcome. The patient required ventilator support for respiration as his breathing was slow and shallow after resuscitation. This might be contributed to respiratory arrest after electric shock and hypoxia during transportation.

Ethics
Appropriate consent was taken from the patient and his caretakers.

References