CASE REPORT

Acute Ischaemic Stroke and Acute Myocardial Infarction Occurring Together in Domestic Low-Voltage (220-240V) Electrical Injury: A Rare Complication

Girish C Verma*, Gaurav Jain**, Abdul Wahid***, Chittora Saurabh***, Nirmal K Sharma****, AR Pathan*****

Introduction

Electrical injuries are relatively common in daily life, are accidentally incurred and few are fatal. The severity of electrical injury varies depending on the type of circuit (AC or DC), resistance, amperage, duration of contact, voltage and pathway of current. High voltage DC contact tend to cause a single muscle spasm often throwing the victim away from the source. This results in a shorter duration of exposure but increases the likelihood of traumatic blunt injury. AC exposure to the same voltage tend to be 2-3 times more dangerous than DC as it induces tetanic muscle contraction causing the victim to become locked to the point of contact. Many organs or tissues including the heart, muscles, kidneys, skin and vascular and nervous system are especially vulnerable to such injuries. The primary cause of death from electrical shock is cardiopulmonary arrest. Myocardial infarction is a potential though rare consequence of electrical shock.

Case Report

The patient is 30 yr old male, resident of Jhalawar, Rajasthan was referred to us from Jhalawar Medical College. Patient had accidental contact with an electricity power line while he was repairing the same. After shock, he had fallen to ground with electric burn injuries over the left side of forehead, left elbow (Figures 1, 2) and bruises over left leg and hip. Patient became unconscious and had weakness in left half of body. Patient was
immediately taken to Jhalawar Medical College. A CT scan head (Figure 3), ECG was done there and patient was referred to Govt. Medical College Kota. On admission, patient had pulse rate of 86/min; blood pressure of 110/70 mm Hg. Patient was unconscious, and had power 0/5 (left half of body); 5/5 (right half of body); deep tendon reflexes – brisk and plantar response was extensor on left side. Examinations of the cardiovascular system, respiratory system were normal clinically. CT scan head revealed – right middle cerebral artery territory large infarct with mass effect and midline shift (Figure 3). ECG showed anterior myocardial infarction (Figure 5). Troponin-T was positive. Serum electrolytes (Na+, K+) were normal. Patient was treated on line of ischemic stroke and myocardial infarction with antiplatelet, beta blocker, angiotensin receptor blocker, lipid stabilising agents, nitrates and mannitol. The course of the stay was uneventful. Patient regained consciousness. Subsequent CT scan showed reduction in mass effect (Figure 4). Neurologically there was minimal improvement and was discharged from hospital in satisfactory condition.

**Discussion**

The clinical effects of electric injury may be classified into immediate and late manifestations. The immediate manifestations include cardiac and respiratory arrest, loss of consciousness, confusion, and amnesia. Late manifestation may be focal; nonfocal deficits – occurred days to months after electrical injury. Focal deficits include cerebral – hemiplegia, aphasia; Spinal – transverse myelitis, ascending paralysis, amyotrophic lateral sclerosis; Peripheral nerve – Neuropathies, radiculopathies. Nonfocal symptoms such as psychoneurotic behaviour, personality changes, confusion, and headache are common. Several mechanisms have been proposed for myocardial injury after electrical injury. These includes – coronary artery spasm; direct thrombogenic effect on coronary arteries; direct thermal effect on myocardium; ischaemia secondary to arrhythmia – induced hypotension; coronary artery ischaemia as part of a generalised vascular injury. Also, hypoxic condition after respiratory arrest might possibly contribute to myocardial injury. Cardiac arrest, either from asystole or ventricular fibrillation, is a common presenting condition in electric accidents. Other ECG findings include sinus tachycardia, transient ST segment elevation, reversible QT segment prolongation, premature ventricular contractions,
Atrial fibrillations and bundle branch blocks. Damage to skeletal muscles may produce a rise in the CPK-MB fraction, leading to a spurious diagnosis of myocardial infarction in some settings. Thus troponins estimation should unquestionably be the preferred cardiac enzyme as in our case. Echocardiography can be beneficial in determining the presence of myocardial injury after electric shock. It may reveal diffuse hypokinesia or regional hypokinesia of myocardium, as in our case which showed anterior wall hypokinesia. Echocardiographic findings may markedly improve in the follow up. There was no history or evidence of cardiopulmonary resuscitation, hypoxic condition or arrhythmia induced hypotension in our case. Therefore, coronary artery spasm and direct thermal effect seemed the likeliest explanation for our case. Management of myocardial injury after electric shock is challenging as there is no consensus as to the best management of ST segment elevation MI after electric shock. Contraindications for fibrinolysis such as prolonged resuscitation, trauma or haematoma may unfortunately accompany electrical injury. So coronary angiography with subsequent percutaneous coronary intervention may be better option as an initial reperfusion strategy. Coronary angiography is of central importance and may clearly guide therapy.

The mechanism responsible for weakness in the extremities after electrical injury may be attributed to electrolyte imbalance, dehydration, rhabdomyolysis, thermal injury, hypoxic encephalopathy, keraunoparalysis, cerebral hypoperfusion and vascular injury causing electrical coagulation, vasospasm, dissection, aneurysm formation and rupture. In our case, electrolyte imbalance, rhabdomyolysis, dehydration were ruled out based on clinical evaluation and laboratory results. Keraunoparalysis, that is transient paralysis of one or more extremities after electrical injury was less likely to be the cause as weakness was persistent in our patient. The heat theory suggests thermal injury to brain tissue. The temperature of CSF may increase to as high as 145°F at 5 hours after electrocution. As the shortest current pathway passed through the brain in our patient, direct thermal injury may likely be the cause of ischaemic stroke in our patient. CT head may show watershed infarctions due to haemodynamic changes that occurs after electric shock. In our case, infarction was wedge shaped, in right frontoparietal region and was probably due to territory occlusion due to vasospasm, rather than systemic hypoperfusion and hypoxic encephalopathy.

**Conclusion**

Electric shock injury due to low voltage AC may cause ischaemic stroke as well as myocardial infarction. Vasospasm caused by the electrical injury as well as direct thermal injury may be the cause of concurrent phenomena. Troponin level estimation and echocardiography should be the primary consideration in order to detect myocardial injury after electric shock. Coronary angiography with subsequent percutaneous interventions may be better option as an initial reperfusion strategy.

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**References**
