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Cerebral Infarction in Scalp Electrical Burns

Shiva Reddy[1], Ravi Kumar Chittoria[2], Barath Kumar Singh. P[3]

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Abstract

Electrical burns are a commonly encountered emergency more often seen in males, especially near major industries. Contact electrical burns can cause external skin injury and also affect the internal organs. They lead to various complications that involve nervous system, cardiovascular system and musculoskeletal system. They are known to cause brain haemorrhage, brain infarct, neuropathy, cardiac arrythmias, rhabdomyolysis, soft tissue injuries. This case report describes a case of frontal cerebral infarct in electrical injury.

Keywords: Cerebral; Infarction; Electrical; Burns; Scalp; Management; Wound; Electrocution.

INTRODUCTION

Electrical burns are common burn injuries in India. Different organs and tissues affected in electrical burns like local tissue damage, musculoskeletal damage, cardiovascular injury and neurological deficits. Electrical burns can cause full thickness burns to the scalp involving bone damage which can be diagnosed by CT Skull and MRI and technetium bone scan. Electrical injury may manifest as peripheral nerve injury, spinal cord damage, cerebellar ataxia, hypoxic encephalopathy or intracerebral haemorrhage.[1] The Severity of electric injury depends on type of current, voltage, tissue resistance, pathway of the current, and duration of contact with the body.[2] However, the severity of the electrical injury is not proportional to the source voltage, visible burns, loss of consciousness, or neuroimaging findings. While most neurologic after effects due to electric injuries varies from immediate, transient, delayed and permanent manifestations are known. This case report describes a case of frontal cerebral infarct in electrical burn injury.

MATERIALS AND METHODS

This study was conducted in the Department of Plastic Surgery in a tertiary care institute. Department scientific committee approval was obtained. In this case report a 45 year old male sustained electrical burn injuries while working at construction building. He sustained electrocution by contact with electric wire and initiated sparks and electrical wire fell on patient head. During the incident he was wearing rubber slippers and polyester clothes and Patient initially went to local hospital, then arrived to emergency department of Jawaharlal Institute of Postgraduate Medical

Author Affiliation: [1]Junior Resident, [3]Senior Resident, Department of Plastic Surgery, [2]Professor & Registrar (Academic), Head of IT Wing and Telemedicine, Department of Plastic Surgery & Telemedicine, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry 605006, India.

Corresponding Author: Ravi Kumar Chittoria, Professor & Registrar (Academic), Head of IT Wing and Telemedicine, Department of Plastic Surgery & Telemedicine, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry 605006, India.

E-mail: drchittoria@yahoo.com

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Education and Research (JIPMER) with an electrical burn in the frontoparietal area (entry zone) and the left leg (exit zone). The other external skin injury to scalp, chest wall, abdomen and both thighs and left foot. At the time of admission his Glasgow Coma Scale score was 12. The patient was disoriented and unconscious at the time of admission and patient was intubated. Multiple second degree superficial burns involving face, neck, chest and abdomen (anterior aspect), bilateral arms (anterior aspect), bilateral thighs, multiple blisters over thigh, legs (Fig. 1) and second degree burns involving frontoparietal region of scalp at the vertex (Fig. 2). He was able to actively move all his limbs. The mid-frontoparietal scalp was charred. CT skull showed small ill defined hypodense area with loss of grey white differentiation noted in the left frontal region suggestive of left frontal infarct (Fig. 3). The serum electrolytes, urea and creatinine, urine analysis, and electrocardiogram were normal, urine myoglobin negative. He was resuscitated with the standard burn unit protocol. Patient was asymptomatic (no seizures, syncope, focal neurological deficits). He was managed conservatively with prophylactic antiepileptic Phenytoin. The scalp had a contact with a 220 V of alternating current. It was presumed that the current entered his skull and exited through his left foot. The patient was extubated after three days of intensive care. The patient perceived weakness in both upper and lower extremities. The strength of all extremities increased gradually. According to the manual muscle test, both upper and lower extremities were normal. Sensory function was intact, muscle stretch reflexes were normoactive, no pathological reflexes were identified, and all the other cranial nerve and cerebellar functions were normal.

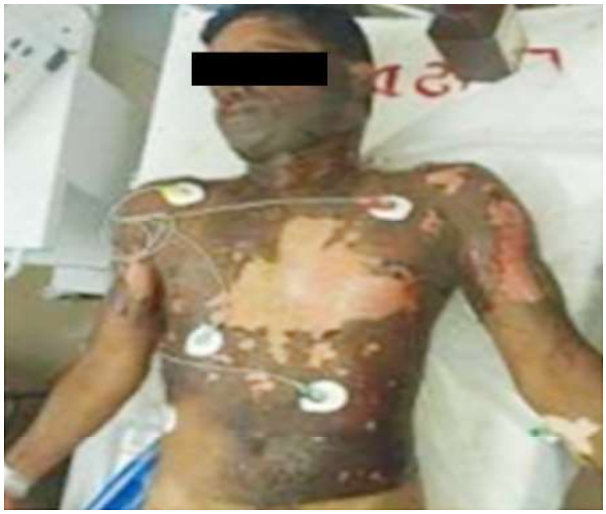

Fig. 1: Patient with electrical burns at admission

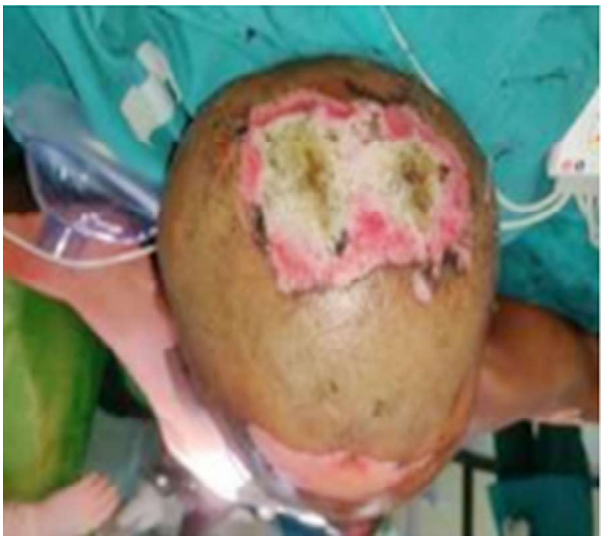

Fig. 2: Electrical burn with entry wound at scalp

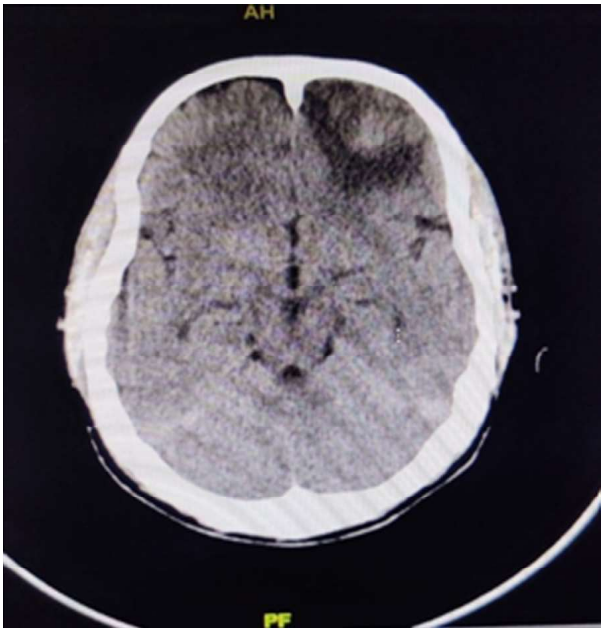

Fig. 3: CT brain showing cerebral infarction in left frontal region

RESULTS

Electrical burns can affect various tissues and organs causing cardiac or neurological complications like stroke, focal neurological deficits, seizures. This case reports presents a rare complication frontal infarct following electrical burns which was be managed conservatively with oral anti-epileptics. Patient is doing well with all wounds in the face, trunk and other areas healed well. The scalp wound is waiting for reconstruction as wound bed is not ready due to presence of osteomyelitis. Rest of the wounds have healed (Fig. 4).

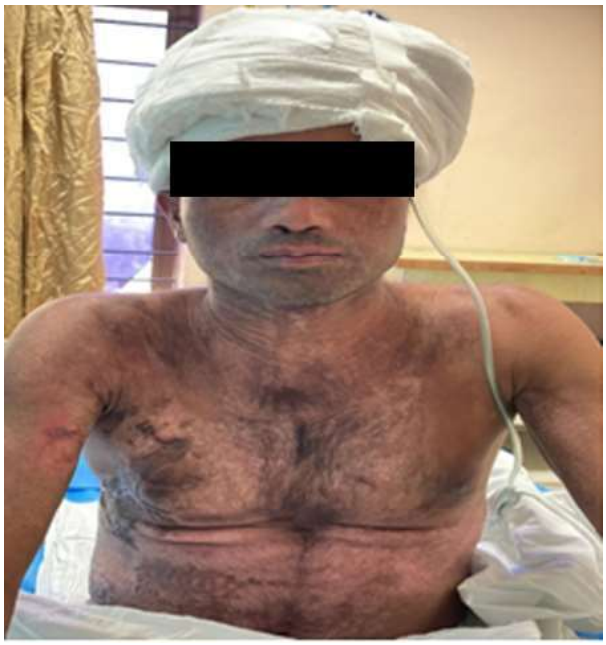

Fig. 4: Healed electrical burn wounds at 3 weeks

DISCUSSION

When an electrical source is touched, a high energy current flows through the body, causing the body to convert the electricity into heat, resulting in a thermal burn. The path with the least amount of resistance is the one taken by electricity, which results in more tissue damage.[3] Bone is the next most resistant tissue in the human body, followed by skin. Blood, muscle, and nerves offer the least resistance. Muscle has substantially lower resistance than dry tissues since it is moist (skin). Skin burns that are more diffuse are caused by higher skin resistance. The highly resistant skin tissue allows electricity to get through, and then the least resistant tissues beneath it allow it to spread. The external appearance of an electrical burn does not adequately reflect the exact amount of the injuries since internal organs and tissues are severely harmed even though the skin damage may be less.[4,5] The Joules effect causes thermal damage, vascular damage when current travels through blood arteries, causing vasospasm and distal ischemia changes, as well as damage to the vascular endothelium caused by free radicals, thrombosis, and increasing ischemic changes.[6]

Due to the widespread use of AC currents in homes and other buildings, AC injuries are far more prevalent than DC injuries. More extensive tissue damage is brought on by low frequency alternating current (AC) than by high frequency AC or direct current (DC).[6] In contrast to DC, which causes a single strong muscle contraction and frequently throws its victim away from the energy source, low frequency AC causes continuous local muscle contraction (flexor muscles greater than extensor muscles) at the site of contact with the electrical source, making the victim unable to let go of the offending source of electricity. Injuries from DC typically result from lightning strikes and contact with automobile batteries.[7] Due to its short distance of travel, high voltage current causes severe soft tissue damage and widespread skin necrosis at the contact site. Because they pass through low resistance tissue, low currents can produce lethal injuries like ventricular fibrillation and cardiac collapse without causing skin lesions.

The cell membrane of a cell contains amino-acids and lipids that carry charge when come into contact with electric current, and proteins undergo denaturation, causing disruption of the integrity of the cell membrane and cell death.[8] Tissue damage in electrical injury occurs as a result of both the direct effect of electrical current and the transformation of electrical energy to heat. The course of the current, the amount of time in contact with the source of electricity, the amperage, and the voltage all affect how severe an electrical burn.

Following electrical burns, neurological complications may result from direct electrical damage (such as electrostatic tissue separation and electroporation of cell membranes), thermal damage (from the Joules effect), or vascular related damage (as current transits through blood vessels, evoking vasospasm and distal ischemic changes; free radical associated vascular endothelial damage and worsening ischemic changes also occur) developing circulating mediators (passage of current is reported to elaborate mediators such as cortisol, free radicals, nitric oxide, and glutamate).[9] According to a retrospective study, the delayed neuropathies that occurred in 17% of people with high voltage electric injuries may have been caused by thermal damage to the peri-neural tissue, which led to progressive peri-neural fibrosis and delayed onset neuropathies. Cerebral infarction is a relatively uncommon occurrence, but it can occur in watershed regions that are susceptible to ischemia when there is cardiopulmonary arrest and low voltage current passes through tissues with the least amount of resistance, leading to endothelial damage, myelin degeneration, and thrombus formation. Blood flow to the nervous system is hampered by cerebral vasospasm/ thrombosis, which results in intra-neural fibrosis.[10] Considering that electricity favours the path of least resistance, and that nerves have a lower resistance than bone and muscle, a victim who has suffered an electrical injury is at a significant risk of

having neurological issues. Hemiplegia, cognitive impairment, focal neurological impairment, limb paralysis, seizures, and peripheral motor sensory neuropathies are some of the victim's possible symptoms. The neurological issues may go away right away or steadily improve over the course of a few days to weeks, but occasionally the deficiencies develop and become permanent.[11] It is thought that neuronal tract collateralization facilitates recovery. Despite the lack of a clear mechanism, electrical shock induced delayed neurological injury is a well known phenomenon. Clinicians should carefully perform a neurological examination and consider various causes if a patient with an electric injury complains of weakness. They should also use electro-physiological techniques like EEG or imaging techniques like CT scans to rule out cerebral damage and nerve conduction studies to rule out damage to peripheral neuropathy and myelopathy. Doctors must be aware of any potential delayed neurological issues that may arise after an electric injury, and patients must be monitored until they reach a stable state both physically and neurologically.[12]

In order to rule out further internal injuries, cardiac and neurological tests are required if the patient is stable. Cardiac problems can happen. At the time of the damage, one may have an arrhythmia, maybe a deadly arrhythmia. Arrhythmia can occur 24 to 48 hours after an injury in anyone who has had an arrhythmia, chest discomfort, or any other common cardiac symptoms.[13] Therefore, these patients should always be connected to a heart monitor. Any high voltage injury requires continuous cardiac monitoring with ECG, cardiac enzymes, CBC, and urine analysis to look for myoglobin due to rhabdomyolysis for a minimum of 8 hours and a maximum of 72 hours.

CONCLUSION

Electrical burn injuries associated with high risk of developing various neurological complications like stroke, seizures, neurological deficits etc. This case report of a patient with an electric burn injury causing brain cerebral infarction that can be useful to elucidate the behaviour of the electric current flow into the nervous system causing brain injury.

Conflicts of interest: None

Authors' contributions: All authors made contributions to the research, is putatively expected to be useful article.

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