Cervical Myelopathy after High-voltage Electrical Burn of the Head: Report of an Unusual Case

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Abstract

High-voltage electrical injuries are uncommonly reported and may predispose to both immediate and delayed neurologic complications. We present a case of 27-year-old male who experienced a high-voltage electrical burn of the head resulting in quadriparesis. High-voltage electrocution injuries are a serious problem with potential for immediate, delayed, and long-term neurologic sequelae. The existing literature regarding effective treatment of neurologic complications is limited. Multidisciplinary management and long-term follow up are required.

Keywords: Electric burns, electric injuries, high voltage

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Introduction

High-voltage electrical injuries are uncommon and may result in mortality or have debilitating neurologic sequel. Electrocution can occur while working and illegal handling of the lines. The true incidence and prevalence of these events are difficult to ascertain. These cases are underreported in literature, probably due to population unawareness and medically underserved communities. Children and young men are at the highest risk to receive an accidental electrical injury, with these subgroups being less likely to report the incident. Almost two-thirds of the fatalities occur in people between the ages of 15 and 40 years.[1] Electrical injury may produce an immediate or delayed myelopathy.

Quadriparesis is a severe disabling condition with long-lasting impact on victim's quality of life and life expectancy, and it also places a huge burden on the family and society with attendant long-term dependence on health-care personnel and resources.[2] Clinicians need to be aware of the neurological consequences of electrocution. We encountered an unusual case of high-voltage electrical burn of the head causing charring of scalp, bone, and dura with surrounding infected brain matter with cervical myelopathy.

Case Report

A 27-year-old male experienced an electrical burn after contact with a 440-volt line while working on a roof. On standing from squatted position, our patient's head came into contact with an exposed wire, resulting in brief loss of consciousness followed by recovery and a charred wound on vertex for which he was being treated at home. Since the wound did not heal and foul smell emanated from it, he was brought to a hospital to treat scalp injury; debridement of dehiscence and skin necrosis was performed. After 8 weeks of electrocution, he was transferred from local community hospital to our institution, he was fully conscious, and he had total amnesia about electrocution. The entry wound was through scalp, traversing through his neck and chest, into his groin, and exiting from his right thigh.

He had 15 cm × 10 cm scalp loss with charred area on the vertex bone with little smelling and fungation of brain matter through dura was merely visible.

Over the ensuing days, he was observed to move his legs less than his arms. The patient had neurologic deficits manifesting as spasticity, motor weakness, and sensory impairment along with bladder involvement. Spasticity of the lower extremities from hip to ankles was grade 2 on the Modified Ashworth scale. There were severe deficits in sensitivity to pain, light touch, and proprioception in the area below neck. Deep tendon reflexes were brisk and muscle weakness with medical research council grade of 3, and plantar responses were equivocal.

Investigation revealed normal hemogram and serum electrolytes. Plain computed tomography (CT) scan was done which showed vertex bone defect. No fracture or dislocation was seen by radiography. Cervical spine magnetic resonance imaging (MRI) and brain MRI done 1 week after injury did not show any specific abnormalities. Since the MRI findings did not match symptoms of patients, repeat MRI performed on admission demonstrated changes suggestive of cord edema and ischemic infarction at cervical levels 1–7. He was commenced on fluid resuscitation, nutritional support, wound care, and physiotherapy. He was transferred to surgery for duraplasty and transposition flap and skin graft. Postoperative period was uneventful and subsequently transferred to rehabilitation department. Till that time, no improvement was observed and unfortunately lost to follow-up thereafter.

Discussion

Electrical injuries have been reported to occur in low-voltage setting, such as with household use and high-voltage exposures from occupational hazards and lightning strikes.[1] Given the natures of these injuries, most of the literature has been reported as case studies with limited data regarding evaluation and treatment of neurological complications.

Several pathophysiological mechanisms of injury to the nervous system have been proposed, including thermal injury, electroporation, and vascular damage through direct injury as well as indirect injury.

Electrical current flows from an area of low resistance which includes muscle, nerve, and blood vessels to high resistance which includes skin, connective tissue, and bone, which suffer greater heat injury, typically at entry and exit sites, but damage can be caused in any structure along the path of the current.[3] Nervous tissues provide a low resistance route for electrical current. As the electrical current travels through tissue, neurons with larger surface area are more likely to be damaged by electroporation, in which the increase in cellular permeability and conductivity caused by permanent conformational change to membrane proteins ultimately leads to cell death.[4] The heat loss that occurs as current flows through tissues of increasing resistance causes damage to intima and adventitia of the vasculature, such as thrombosis, necrosis of the vascular wall, vasospasm, and spread into nearby tissue. Most patients survive the initial insult, limiting pathological workup with few postmortem studies available for review.[1] Morbidity of electrocution has related to electrothermal injury causing direct tissue damage with secondary ischemic changes from vascular insult. An accurate prognosis is challenging given the variation in duration and extent of injury, the frequency of current, and the anatomic site. There are relatively few reports that correlate the clinical, electrophysiological, and imaging changes that occur with electrocution injuries.[5]

Classification of injuries has been divided by onset of symptoms. Silversides divided the stages into immediate, secondary, and late effects. Immediately, after an electrical current passes through the human body, thermal injury occurs within nerve cells, manifesting effects such as altered sensorium and/or loss of consciousness, severe pain, hearing and vision changes, motor signs (including paralysis), respiratory compromise, or sensory complaints. Recovery occurs within

24 h. Secondary effects include temporary paralysis and autoimmune disturbances. The late effects are noted to start after 5 days, manifesting as hemiplegia, movement disorders, brainstem dysfunction, and cranial neuropathies.[6]

Electrocution can occur while working and illegal handling of the lines. Chances of accidentally coming in contact with high tension cables are there as these wires pass through the fields which are the place of work for most of our population. Spinal cord injuries have also been classified into transient and permanent disability. Motor deficits occur more often than sensory disturbances and are secondary to vascular damage incurred by the anterior spinal artery and its branches, which supply two-thirds of the spinal cord, including the lateral corticospinal tracts, spinothalamic tracts, and anterior horn cells along with the central gray matter of the spinal cord. This hypothesis may be supported by the susceptibility of smaller lumen vessels to injury, with less dissipation of heat to surrounding structures compared with their larger vessel counterparts.[6] The index case was a victim of high-voltage electrical injury. The patient showed a combination of features of immediate and delayed onset spinal cord injury. This is similar to report by Johl *et al.*[7] Several other case reports have also documented similar findings.[5] Cervical myelopathy with late-onset progressive motor neuron disease following electrical injury has also been described by Ghosh *et al*. Arévalo *et al*. reported two cases of neurologic symptoms immediately following electrical injury in which CT and MRI were normal.[1] However, the MRI done in our patient revealed signal intensity abnormalities. Ko *et al*. carried out a retrospective study of spinal cord injuries related to electrical burns. They reported that 11 out of 13 patients with entry wounds in the head and neck region were found to have quadriplegia with exit sites located in the upper extremities and paraplegia with exit sites located in the lower extremities. Most of these patients were noted to have hypotonia acutely in 2–10 days after injury in an ascending pattern. It was postulated that this pattern of injury was related to ischemic damage to the arterial blood supply and vulnerability in the spinal cord.[3] Our patient had exit site in the right thigh.

Early discovery of extensive myelopathy of the cervical spine is aided by neuroimaging. Most of the described cases used T2 hyperintensities to clinical deficits.[4] Initial imaging of our patient did not show any significant changes to explain his quadriparesis. However, repeat imaging performed roughly after 7 weeks of initial MRI of his brain and cervical showed extensive involvement of the pyramidal tracts as well as cord edema, which explained our patient's clinical findings. It was felt that the bilateral lesions involving the corticospinal tracts particularly contributed to our patient's paresis. As with our case, serial imaging may prove essential for identifying delayed sequelae associated with electrocution injuries. Repeat imaging assists in making correct diagnosis. Otherwise, differential diagnosis made with radiology may be wide and includes neoplasm, infarction, and electrocution injury. Exploring additional neuroimaging modalities may be beneficial, such as MR spectroscopy. Electrodiagnostic studies such as evoked potentials, nerve conduction studies, and electromyography may have prognostic value. The latter two modalities can help determine the severity of motor axonal loss, stemming from anterograde anterior horn cell degeneration in the ventral gray matter. Technical aspect limits the value of these studies secondary to the presence of skin injury and accessibility.[6]

No established guidelines are available in the literature regarding the treatment of high-voltage electrical injury. Each case has been treated with supportive care. Early treatment of electrical injury starts with initial fluid resuscitation, respiratory support, and prevention of infection. The lack of systematic guidelines makes clinical care for the patient with electrocution challenging. Workup of the neurological deficits with imaging or electrodiagnostic studies, treatment, and prognosis for recovery is dictated by the personal experience of the physician. There are no randomized double-blinded trials available on electrocution. The majority of information comes from isolated cases, case series, and animal experimental models, with fewer articles available in the radiology and neurological literature.[7,8]

No consensus exists regarding medication use. Neurological morbidity has been variable. Some case studies with delayed finding of severe myelopathy due to electrocution injury did not show full recovery as in our index case while other case reports showed near complete resolution of motor deficits with aggressive physical therapy.[3] Multidisciplinary management approaches involving neurologist, plastic surgeon, physical and occupational therapists, and psychologist, as well as a nutritionist, is necessary to achieve best possible outcomes. Long-term neurological follow-up is justified by the rich body of evidence of delayed complications cited in literature.

Conclusion

High-voltage electrocution is a serious problem with potential for immediate, delayed, and long-term neurologic sequelae. The mechanisms of damage to the central nervous system after high-voltage electrical injury are not fully elucidated. Multidisciplinary management and long-term follow-up are required. As a preventive measure, high voltage lines should be installed at a safe position and occupational safety practices should be employed among electrical workers.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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