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American Journal of  
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## Case Report

## Acute vertebrobasilar ischemic stroke due to electric injury ☆☆☆★★

## Abstract

Electrical injuries are most commonly due to household accidents. Various factors determine the severity of electric injury, including type of current, amperage, voltage, tissue resistance, pathway of current, and duration of contact with the body. Various types of neurologic damage due to electrical injury have been described in literature. It may manifest as peripheral nerve injury, spinal cord damage, seizures, cerebellar ataxia, hypoxic encephalopathy, and intracerebral hemorrhage. Acute ischemic stroke is an infrequent complication of electrical injury. Herein, we report a case of middle-aged man, who accidentally sustained high-voltage electrical injury followed by acute vertebrobasilar ischemic stroke. Magnetic resonance imaging of the brain showed acute infarct in bilateral cerebellar and medial occipital regions. Computed tomographic angiogram of the brain and neck vessels was normal. Possibly, in our patient, the mechanism could be related to direct vascular injury due to electric current.

Most electric injuries are seen in household accidents due to low-voltage alternating current, although injuries due to high-voltage current have also been reported [1,2]. The parts of the body, usually prone to injury are skin, muscles, heart, kidneys, blood vessels, and nerves.

Various types of neurologic damage due to electrical injury have been described in literature including peripheral nerve injury, spinal cord damage, movement disorders, amyotrophic lateral sclerosis, neuropsychological symptoms, cerebellar ataxia, seizures, hypoxic encephalopathy, and intracerebral hemorrhage [3–5]. We report a case of acute vertebrobasilar stroke due to electrical injury because of the infrequency of this neurologic complication.

A 40-year-old man while working accidentally touched an overhead high-tension wire carrying 11 000 V and sustained electric injury. The patient became unconscious but regained consciousness on the way to the hospital. In an emergency, quick initial resuscitation and assessment of the patient were done with special attention to the airway, breathing, and circulation. There was no previous history of transient ischemic attack, stroke, hypertension, or diabetes mellitus. The patient was then transferred to the burn unit. He had 18% burn injury composing of full and partial thickness burns on the right arm and axilla (entrance or source points), right thigh, and groin area (exit or ground points) (Fig. 1A–C). There were no signs of cardiovascular, respiratory, or abdominal involvement. His pulse and blood pressure were normal. The patient was alert, conscious, and oriented to place, time, and person. His speech was slurred. Fundi were normal. Visual field examination showed right homonymous

hemianopia. He had mild weakness in right upper and lower limbs (Medical Research Council grade 4/5). Nystagmus with the fast component toward left side was present. Cerebellar signs were present in form of limb ataxia, finger–nose incoordination, and dysdiadochokinesia on both sides (left more than right side). On the right side, deep tendon reflexes were exaggerated, and the Babinski sign was positive.

Complete hemogram, serum electrolytes, renal and liver function tests, and lipid profile were normal. Serum creatinine phosphokinase level was raised. Urine biochemical and microscopic analysis was normal. Urine examination for myoglobin was negative. Electrocardiogram, x-rays of chest, and cervical spine were normal. Computed tomographic scans of brain showed hypodensity in bilateral cerebellar and left posterior occipital regions (Fig. 2A–C). Magnetic resonance brain diffusion weighted imaging showed acute infarct in bilateral cerebellar hemisphere (left more than right), vermis, and left medial occipital areas (Figs. 3A–H and 4A–F). Computed tomographic angiography of the neck and brain vessels was normal (Fig. 5A–F). Two-dimensional echocardiography was normal. Coagulation profile, including proteins C and S; anticardiolipin antibody; and homocysteine level were normal.

Electric injury was self-evident from history. Radiographs of the chest and cervical spine and ultrasound abdomen were done to look for any associated injury, especially for spinal injury and blunt trauma. The patient came to us after window period; therefore, thrombolytic therapy could not be given. He received tetanus toxoid injection. Intravenous antibiotics were given to prevent secondary infection, and burn wounds were covered with antibiotic dressings (mafenide acetate/sulfadiazine silver). He was started on antiplatelet (aspirin) therapy and other supportive treatment along with rehabilitation services. After 15 days, he underwent cutaneous graft surgery. He gradually improved and was able to walk with support within a month.

The patient was discharged after 1 month. Rehabilitation services, including physical, occupational, and speech therapy, were continued; and at 6 months' follow-up, the patient was ambulatory without support. Neurologic examination still showed evidence of cerebellar signs including dysarthria, finger–nose incoordination, and gait ataxia.

Electric injuries are most commonly due to household accidents. Acute vertebrobasilar ischemic stroke is an infrequent neurologic complication of electric injury. Most effects due to electric injuries are immediate and transient; however, delayed and permanent manifestations are also known. Various factors determine the severity of electric injury including type of current, voltage, amperage, tissue resistance, pathway of current through the body, and duration of contact [6].

Although few case reports have been described in literature, there has been little advancement in the understanding of neurologic complication of electric injury [7]. As electric current passes through the body, thermal burn due to electric injury causes coagulation necrosis [8,9].

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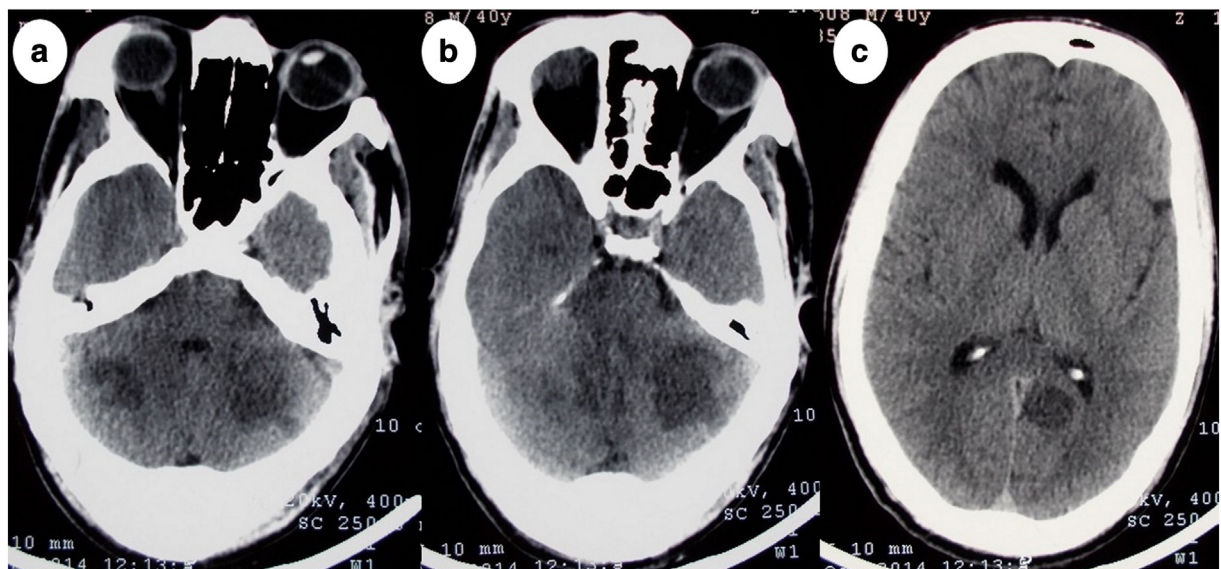


**Fig. 1.** Photographs of patient showing distribution of burn injury (18%). Right lateral view (a), right arm and axilla (b), and right side of groin and lower abdominal area (c).

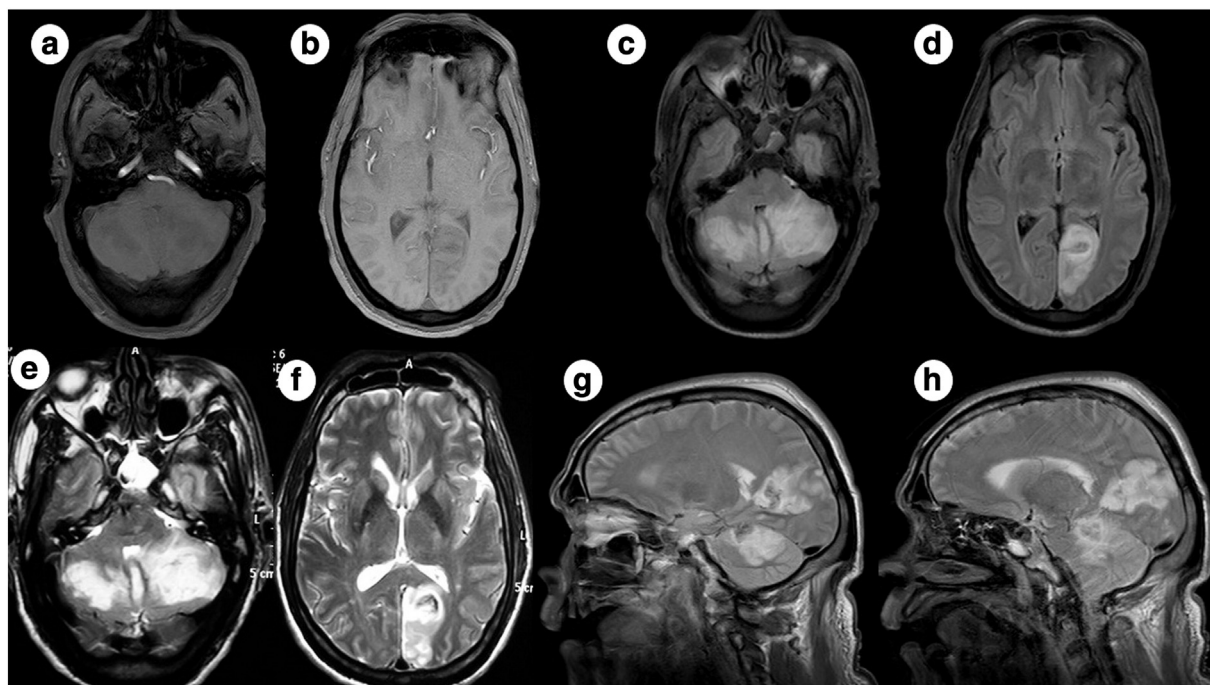
Ischemic stroke due to electric injury may occur from various mechanisms. One of the reasons may be mechanical injury due to prolonged muscle contraction. Electric injury causes intense muscle spasms and sympathetic hyperactivity, which may lead to vasospasm and thrombus formation or embolism. Another mechanism may be direct underlying cerebral damage due to thermal burns. Heat dissipated in intracranial vessels can cause protein conformation changes, cellular dysfunction, and tunica intima damage resulting in thrombosis with subsequent thrombosis or rupture [10,11]. Hemodynamic alterations and cardiac arrest leading to cranial hypoperfusion and watershed infarction can

be alternate mechanism. In our patient, acute vertebralbasilar infarct may possibly be related to direct vascular injury due to electric current.

All patients who sustain a high-voltage electric injury should undergo hemogram, biochemical tests, cardiac isoenzyme level, urine analysis for myoglobin, electrocardiogram, and radiographic studies to look for associated injuries [12]. To conclude, acute vertebralbasilar ischemia due to electric injury is an uncommon neurologic manifestation. Hospital stay and morbidity in our case were increased due to acute ischemic event. The exact mechanism of the damage still remains in doubt and therefore needs further research.



**Fig. 2.** Computed tomography of brain showing hypodensity in bilateral cerebellar left (a) more than right (b) and left medial occipital areas (c).



**Fig. 3.** a and b, Magnetic resonance imaging brain showing hypointensity in bilateral cerebellar and left medial occipital area on T1-weighted images. Hyperintensity seen in bilateral cerebellum (left more than right), vermis, and left medial occipital areas on fluid attenuation inversion recovery (c and d) and T2-weighted images (e–h).

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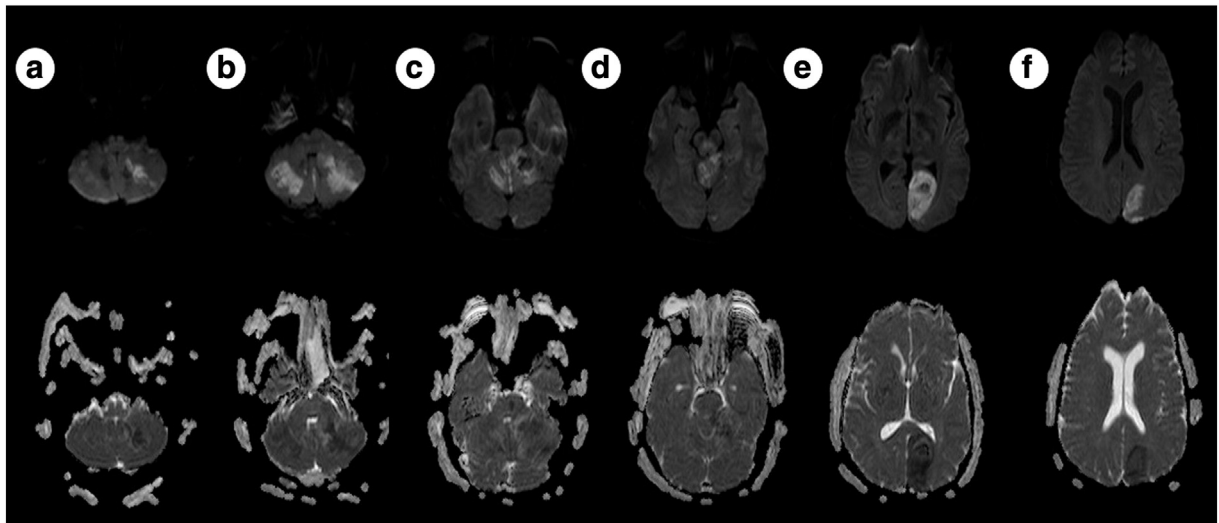
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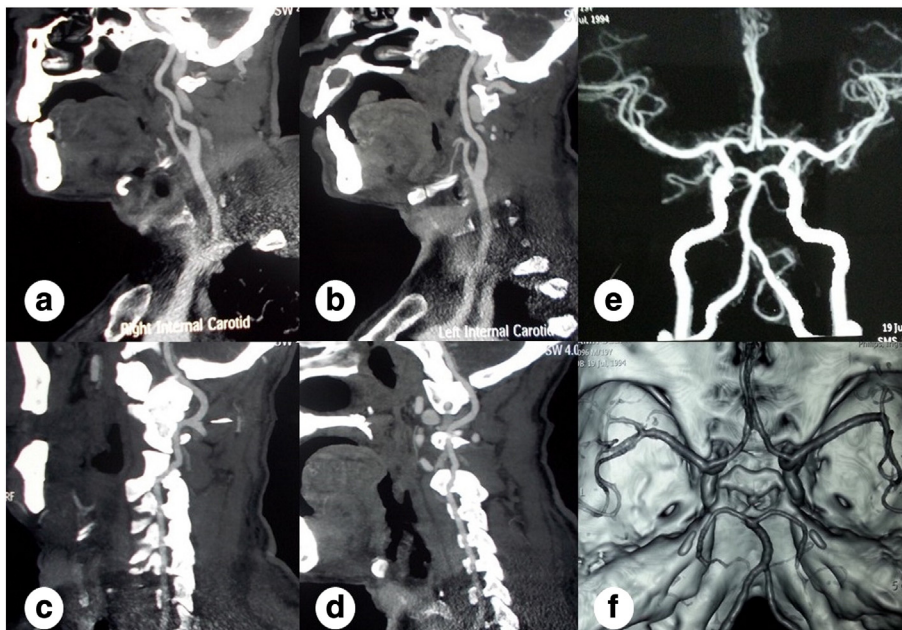
## References

- [1] Christensen JA, Herman RT, Balis GA, Wuamett JD. Delayed neurological injury secondary to high-voltage current, with recovery. *J Trauma* 1980;20:166–8.
- [2] Singerman J, Gomez M, Fish JS. Long-term sequelae of low voltage electrical injury. *J Burn Care Res* 2008;29(5):773–7.
- [3] Ramati A, Rubin LH, Wicklund A, Pliskin NH, Ammar AN, Fink JW, et al. Psychiatric morbidity following electrical injury and its effects on cognitive functioning. *Gen Hosp Psychiatry* 2009;31(4):360–6.
- [4] Morse JS, Morse MS. Diffuse electrical injury—comparison of physical and neuropsychological symptom presentation in males and females. *J Psychosom Res* 2005;58(1):51–4.
- [5] Chudasama S, Goverman J, Donaldson JH, van Aalst J, Cairns BA, Hultman CS. Does voltage predict return to work and neuropsychiatric sequelae following electrical burn injury? *Ann Plast Surg* 2010;64(5):522–5.
- [6] Baxter CR. Present concepts in the management of major electrical injury. *Surg Clin North Am* 1970;50:1401–18.
- [7] Cherington M, Yarnell P, Hallmark D. MRI in lightning encephalopathy. *Neurology* 1993;43:1437–9.
- [8] Childbert M, Maiman D, Sances A, Myklebust J, Prieto TE, Swiontek T, et al. Measure of tissue resistivity in experimental electric burns. *J Trauma* 1985;25:209–15.
- [9] Puschel K, Brinkman B, Lieske K. Ultrastructural alteration of skeletal muscles after electrical shock. *Am J Forensic Med Pathol* 1985;6:246–51.
- [10] Lee RC, Kolodney MS. Electrical injury mechanisms: electrical breakdown of cell membranes. *Plast Reconstr Surg* 1987;80(5):672–9.
- [11] Lee RC. Injury by electrical forces: pathophysiology, manifestation and therapy. *Curr Probl Surg* 1997;34(9):667–765.
- [12] Hussmann J, Kucan JO, Russell RC, Bradley T, Zamboni WA. Electrical injuries—morbidity, outcome and treatment rationale. *Burns* 1995;21(7):530–5.

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**Fig. 4.** a to f, Diffusion restriction seen in bilateral cerebellar (left more than right), vermis, and left medial occipital areas on diffusion weighted imaging brain (upper collum) and corresponding fall in apparent diffusion coefficient value (lower column).



**Fig. 5.** Computed tomographic angiography of neck vessels including right (a) and left (b) internal carotid and right (c) and left (d) vertebral arteries as well as brain vessels (e and f) were normal.