Cranial-entry Electrical Burns with Neurological Sequelae: A Case Report

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Summary
Cranial-entry high-voltage electrical burn injury rarely occurs. Hence, early presentation is rare. We herein present a case of a teenage male with mainly full-thickness electrical burn injury covering 9% of the total body surface area, with gangrenous mid-fronto-parietal scalp and calvarium, moderate head injury with bi-hemispheric deficits, and left frontoparietotemporal intracerebral hematoma and exit wounds (right thumb, thigh, and toes). He underwent initial outer-strip and, later, inner-strip corticectomy, covered with split-thickness skin graft (STSG) and bilateral posterior-auricular artery-based flap, respectively, as well as tangential burn wound excisions with STSG and louvre flap cover for the digits. He also underwent right upper eyelid contracture release and tarsorrhaphy. He developed late post-traumatic seizures, which were managed with anti-epileptic drugs. Definitive calvarial reconstruction is underway. Cranial-entry electrical injury had high morbidity and mortality risks. The spectrum of nervous damage causation mimics nervous syndromes and their presentations. Although elaborate surgical and medical care offers the possibility of survival, the neurological and neuropsychological syndromes in the post-acute and rehabilitative phases of care should be taken care of.

Keywords: Electrical burns, cranial entry, neurological sequelae, scalp reconstruction, electrical burn injury rehabilitation

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Introduction

High-voltage electrical burn injury is rare and potentially life-threatening, due to widespread coagulative necrosis of deep tissues from generated heat (1, 2). Ohm's law had described the relationship among current, voltage, and tissue resistance when electricity passes through a tissue; while Joule's heat equation calculated the heat energy produced from electricity, the Joules' effect posited that the heat generated in a tissue by current is directly and inversely proportional to the electrical potential and tissue resistance, respectively (2–6). Tissues with excellent conductive properties (e.g., nerves, arteries) can deliver electricity to other tissues; meanwhile, in some of the deeper, poor-conductive tissues, electricity causes more injury, resulting in rhabdomyolysis, myoglobinemia, myoglobinuria, and potential interstitial nephritis causing acute kidney injury (2, 4, 6). The extent of damage from electrical injury is grossly underestimated when using standardized burns surface area estimation methods;

hence, the fluid resuscitation is titrated with the desired urine output (>1.5 mL/kg/h) (7).

Tracking of unduly large electric currents through excitable tissues (especially nerves, cardiac tissues) may offset the premorbid electrical rhythmicity of these tissues. The result is a cascade of short- and long-term conditions such as cardiac arrhythmias, altered sensorium, cognitive deficits, seizure disorder, focal brain lesions (e.g., such as infarcts, hematoma), diffuse brain lesions (e.g., cerebral edema), spinal cord or nerve lesions (presenting with varying paresis or plegia, in addition to motor or sensory unit losses), retinal detachment, etc. (3, 4, 8).

Cranial-entry electrical burns, which are very rare, causes significant damage to the scalp, calvarium, meninges, and brain (1, 9). Scalp defect could pose a challenge for scalp reconstruction (9). Early presentation for care is a rare occurrence in Ibadan, Nigeria. This case report presents a stepwise follow-up of a high-voltage electrical burn survivor, which provided the patient a greater chance at survival from the acute and chronic sequelae.

Case presentation

A 16-year-old right-hand dominant male, presented 2 hours post-electrical burns that were sustained while attempting to turn off power following a neighborhood power surge. He was thrown onto a nearby energized iron rod, impacting with his head, right hand, and toes. He was rescued after approximately 2 minutes with a dry wooden staff, suffering multiple burn injuries to the head, fingers, thighs, and toes as well as transient loss of consciousness, which was partially regained within 30 minutes. There were no seizures, craniofacial effluxes, bisphincteric dysfunction, or vomiting. He was able to actively move all his limbs. He had no dyspnea or limb deformity. On presentation, there was marked craniofacial edema, and his Glasgow Coma Scale score was at least 9 (best eye response, not testable; best verbal response, 2; best motor response, 6); he had marked bilateral periorbital edema, was making incomprehensible sounds, and can obey two-phased motor commands. The mid-frontoparietal scalp was charred, with exposed, denuded parietal calvarium (Figure 1). He had full-thickness burns on the exit wounds to the right thumb, index, and ring fingers, right thigh, and toes.

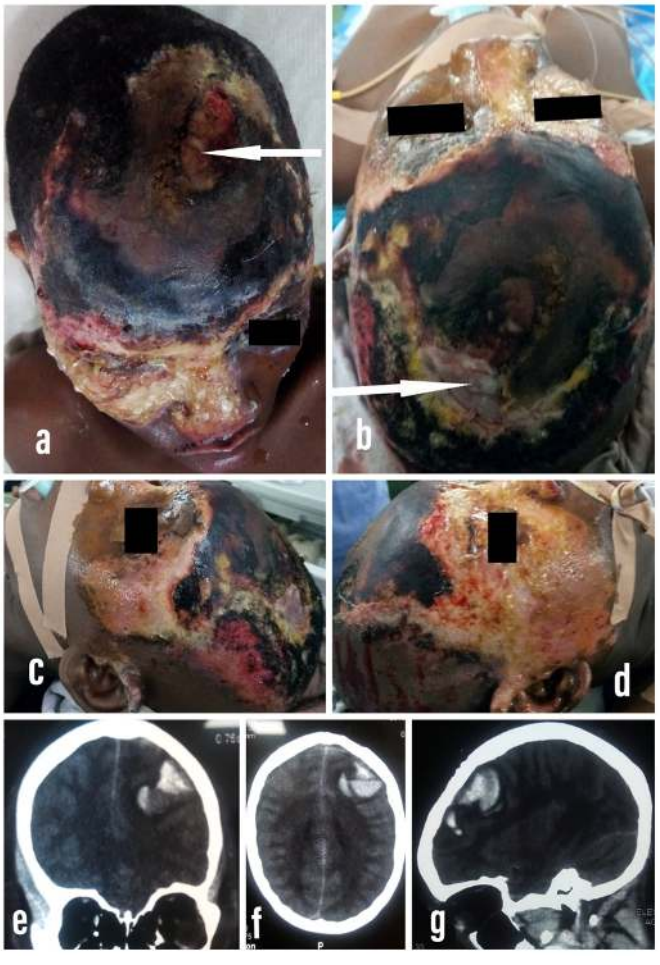

Figure 1. Showing areas of denuded scalp and cranial computed tomography scan images. Charred fronto- parietal scalp images are shown in 'a-d', with partial thickness burns on the superior face. White arrows in 'a' and 'by show exposed calvarium. Intracerebral haematoma (areas of complex pattern of hypodensities in the left fronto-parietal cerebrum) is shown in 'e', 'f' and 'g', representing the coronal, axial and sagittal slices respectively. 975x1411mm (72 x 72 DPI)

The diagnoses were mainly full-thickness electrical burn injury on 9% of the total body surface area, gangrenous mid-frontoparietal scalp and calvarium, and moderate head injury with bihemispheric deficits and left frontoparietotemporal intracerebral hematoma. His complete blood count showed leukocytosis (22,000 cells/mm3) with relative neutrophilia. The serum electrolytes, urea and creatinine, urine analysis, and

electrocardiogram were normal. A cranial computed tomographic scan showed cerebral edema, left frontoparietotemporal intracerebral hematoma with a midline shift (2mm) toward the left (Figure 1e-g). He was resuscitated with the standard burn unit protocol. His neurological status on the 5th post-operative day was that of a defective Mini Mental State Examination (defects in short-term memory, calculation, sluggish speech, and neutral affect), but he had intact motor and sensory levels. The Glasgow Coma Scale scores were 14 and 15 on the 2nd and 5th days post-burn, respectively. His first surgical intervention was on the 6th day post-burn and involved both the plastic and neurological surgery services, wherein he had debridement of the necrotic scalp, outer-table strip corticectomy + split thickness skin graft (STSG) of the mid-frontoparietal inner-table because the patient had an on-table cardiac arrest from which he was successfully resuscitated (Figure 2). The burns to the digits were debrided and resurfaced with flaps. On the 9th post-operative day, necrosis of some of the inner-table strip of the calvarium with necrosis of the overlying STSG was observed, necessitating a second surgical procedure on the 10th post-operative day (17th day post-burn). He had debridement of the STSG and inner-strip corticectomy, and the defect was reconstructed with two transposition flaps based on both posterior auricular arteries. A planned STSG cover of secondary scalp defect was not done because of intraoperative cardiac arrest from which he was successfully resuscitated. On the 27th day post-burn, he had a STSG to resurface the residual scalp defect as the third surgical procedure. Definitive calvarium reconstruction was deferred because a satisfactory definitive calvarium prosthesis was unavailable. He developed right upper eyelid ectropion with the onset of a right-sided forehead contracture, necessitating a right upper eyelid tarsorrhahpy (prophylactic for corneal xerophthalmia), and on the 50th day post-burn, he had a fifth surgical procedure for release of forehead and right upper eyelid contractures, with FTSG and STSG cover of the defects on the upper eyelid and forehead, respectively. He was discharged from inpatient care on the 73rd day post-burn with a satisfactory neurological status; his GCS at discharge was 15.

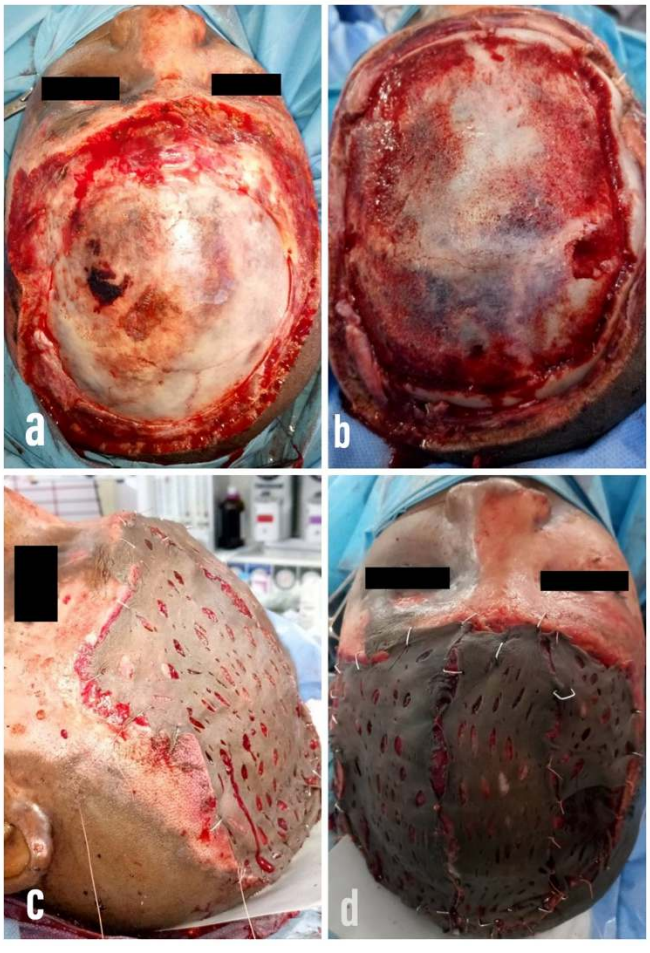

Figure 2. Showing the patient's peri-operative images after the first surgical procedure. Immediate post-debridement of denuded calvarium in 'a', with 'b' showing immediate post outer-strip corticectomy of the fronto-parietal calvarium, 'c' showing the meshed STSG cover post outer-strip corticectomy, while 'd' shows the STSG with dark areas of non-take. 529x765mm (72 x 72 DPI)

He had regular, uneventful out-patient clinic visits; however, by 4 months post-burn, there were two episodes of seizures followed by loss of consciousness. These started with about three episodes of snapping of the lips (absence seizures), each lasting approximately 10 minutes. He had several other episodes; thus, he was commenced on anti-epileptic medications (initially parenteral phenytoin boluses and infusion, and later on, daily phenytoin capsules). He was not prophylactically placed on anti-epileptic drugs. These necessitated his

readmission on the 5th and 7th months post-burn. At 12 months post-burn, his neurological and ophthalmological reviews were satisfactory. The patient sought overseas plans for definitive calvarium reconstruction. The deidentified images of the craniofacial state at 12 months post-burn are shown in Figure 3.

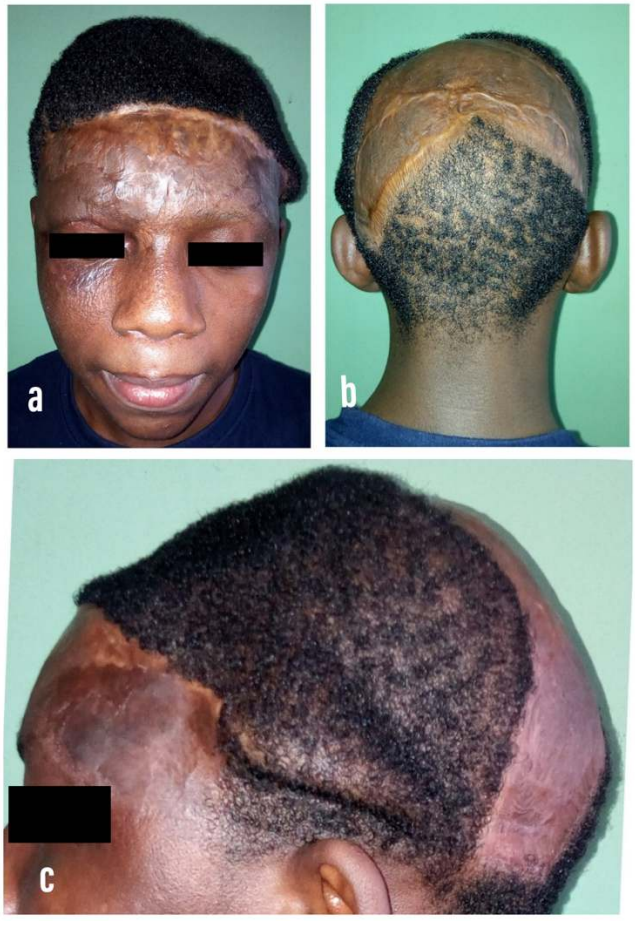

Figure 3. Shows the patient post-operatively after bilateral posterior-auricular artery-based flaps at twelve months post-surgery, with healed flaps and healed STSG cover of the secondary defect.

The professional responsibilities to the patient were not breached or compromised, directly or otherwise, as a result of the documentation of this case report. The confidentiality of the patient was always maintained, as agreed upon with the patient at the time of acquisition of informed consent. No specific identifying information and markers were used, and the patient was appropriately de-identified. Informed consent was obtained from the parent of the patient and from the patient at every stage of the documentation and acquisition of information for this report.

Discussion

The neurological sequelae post-electrical burns originate from either one or a combination of the following syndromes: central (cerebral, cerebellar, cranial nerves), spinal, or peripheral nerve (3, 8). The temporal onset of these neurological symptoms have also been used to categorize them as acute or chronic (long term) (4, 5). This patient presented with an acute-onset altered sensorium and intracerebral hematoma; he also later developed seizures at 4 months post-burn, necessitating the use of anti-epileptic drugs for control. The neurological sequelae post-electrical burns are described as being from direct electrical damage (from electrostatic separation of tissues, electroporation of cell membranes, etc.), thermal damage (from the Joules effect), vascular-related damage (as current transits through blood vessels, evoking vasospasm and distal ischemic changes; free radical-associated vascular endothelial damage, thrombogenesis, and worsening ischemic changes also occur) (4, 5), elaboration of circulating neurohumoral mediators (passage of current is reported to elaborate mediators such as cortisol, free radicals, nitric oxide, and glutamate) (3). The most plausible etiology of this patient's neurological features were thermal and vascular, albeit electrical damage cannot be dismissed. The nervous tissue damage can thus be considered as being direct neuronal and/or neuroglial injuries (thermal-, ischemic-, and tissue mediator-related), indirect local/regional injures (vascular-related and those ensuing from contiguous action of adjacent inflammatory mediators), and, indeed, indirect distant injuries (usually following humoral mediator activities); a combination of these may be the usual occurrence (3–5), as in this patient. The various causative theories have varying degrees of postulated causation of either acute or chronic neurological sequelae. The neurohumoral mediators have been linked with capacity to elicit neurological deficits in sites distant from the primary electrical injury (3).

This patient had two intraoperative cardiac arrests. The

first occurred on the 6th day post-burn, but the exact cause could not be established, as the previous electrocardiogram recordings and electrolyte levels were normal and within the reference ranges, respectively, but it was postulated to be related to the putative electrical and thermal damage to the cardiac myocytes following transit of high-voltage electricity through the patient. The second cardiac arrest occurred after significant primary hemorrhage and intraoperative hypotension. The patient was appropriately resuscitated following each event.

Conservative treatment was selected for the intracerebral hematoma because the clinical and neurological situation significantly improved by the 2nd day post-burn. If the clinical scenario were more severe, as evidenced by larger-volume hematoma, with pressure effects in the presence of worsening clinical and neurological status, evacuation would have been necessary. As in this case, with full-thickness scalp and calvarium injuries, the debridement would ultimately result in a skull defect and exposed dura. Hence, proceeding with evacuation will involve a dichotomy and brain cannulation to evacuate the hematoma. The patient's available options for reconstruction of the calvarium defect includes a customized polymethyl methacrylate implant, use of autogenous split-thickness corticocancellous calvarial bone graft, allopathic three-dimensional dynamic titanium mesh cranioplasty, and, as a temporary measure, the use of metallic hats. The timing of calvarial reconstruction draws from the previously advocated 6- to 12-month delay, which is believed to be the duration required for brain to recover from the trauma and revascularization of the surrounding skull flap. Recently, the recommended timing is <3 months from the initial procedure, with 6 weeks believed to be the ideal time for cranioplasty (9). When treating patients with post-acute cranial-entry electrical injury, the possible ongoing pathological events, especially those that, if uninterrupted, would evoke long-term neurological syndromes, should be considered. The use of antioxidants (e.g., flavonoids, which would remove free radicals), nitric oxide antagonists, glutamate antagonists, and cortisol antagonists, among other agents in different phases of research, is putatively expected to be useful (3). The role of psychotherapy and neurocognitive rehabilitation to address the neuropsychological syndromes should be emphasized (3). This patient, being a teenager with active interaction with his peers, may face social stigma from the burn scars, and he received care from the clinical psychologists and occupational therapists to enable him to reintegrate smoothly within the community. The elaborate ablative and reconstructive surgical techniques that offer immediate resuscitation, alongside medical and intensive care modalities, characterize the acute phase care (1, 4, 10). When done satisfactorily, these offer patients a chance at survival.

Conclusion

High risks for morbidity (neurological risks such as seizures, cardiovascular risks such as cardiac arrests, etc.) and mortality follow cranial-entry, high-voltage electrical injury. The spectrum of nervous damage causation mimics an array of nervous syndromes in their acute and chronic presentations. Although elaborate surgical and medical care now offers survivors a true chance at survival, attendant neuropsychological and other long-term syndromes in the post-acute and rehabilitative phases of care should be addressed. There are no financial interests to disclose for this article.

References

1. Onah I, Onumaegbu O. Extensive calvarial exposure and full thickness sequestration following electrical burn injury presenting after one year: case report. Niger J Plast Surg. 2010; 6(2).
2. Kurt A, Yildirim K, Yagmur C, et al. Electrical burns: highlights from a 5-year retrospective analysis. Ulus Travma Acil Cerrahi Derg. 2016; 22(3): 278-282.
3. Andrews CJ, Reisner AD. Neurological and neuropsychological consequences of electrical and lightning shock: review and theories of causation. Neural Regen Res. 2017; 12(5): 677-686.
4. Kim HM, Ko Y, Kim JS, et al. Neurological complication after low-voltage electrical injury: a case report. Ann Rehabil Med. 2014; 38(2): 277-281.
5. Schaefer NR, Yaxley JP, Jeyarajan E, et al. Electrical burn causing a unique pattern of neurological injury. Plast Reconstr Surg Glob Open. 2015; (3): e378.
6. Axayacalt GA, Alejandro CE, Marcos RA, et al. Brain haemorrhage after electrical burn injury: case report and

probable mechanism. Surg Neurol Int. 2016; (7): S759-S762.
7. Sharma VP. Re: Electrical injury. Br Med J. 2017; (357): j1418.
8. Deveci M, Bozkurt M, Sengezer M. Clonus: an unusual delayed neurological complication in electrical burn injury. Burns. 2001; (27): 647-651.
9. Jeyaray P. Cranial vault defects and deformities resulting from combat related gunshot, blast and splinter injuries: how best to deal with them. J Maxillofac Oral Surg. 2019; (19): 184-207.
10. Kotera S, Narayan N, Mahesh M, et al. Reconstruction of post electrical burns scalp defect. Int J Res Med Sci. 2016;4(6): 2497-2499.