Onset of symptomatic hydrocephalus requiring emergency cerebrospinal fluid diversion following high-voltage electrical burn injury

Case report

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High-voltage electrical injuries have been reported to cause a plethora of neurological complications including cognitive, motor, and sensory deficits in an immediate or delayed fashion. In this setting, new-onset symptomatic hydrocephalus requiring CSF shunt placement has not been described. The authors present the case of an 18-year-old man who sustained a high-voltage electrical injury to a calvarial contact point that required emergency CSF diversion within hours of injury and subsequently required placement of a lumboperitoneal shunt. Management of the open calvarial wound, which required rotational flap reconstruction, and the need for ongoing CSF diversion required care and a team approach. (DOI: 10.3171/2009.7.JNS09578)

KEY WORDS • hydrocephalus • high-voltage electrical injury • burn injury • calvarial burn

High-voltage electrical burn injury is an uncommon injury, particularly when the burns include the calvarium.19 Numerous sequelae including change in cognition2,9,14 and various motor and sensory deficits occurring in both an acute and delayed presentation have been previously described.1,3–6,10,11,13–16,18,20–23 In this report, we describe an 18-year-old man with no history of hydrocephalus or neurological deficits who subsequently sustained a high-voltage electrical injury to his head. Hours after the injury, he required CSF diversion due to elevated ICP and, ultimately, required CSF shunt surgery. To our knowledge, this is the first case of shunt-treated hydrocephalus following an electrical burn injury.

Case Report

Injury and Initial Treatment. This 18-year-old man working in a utility bucket made contact with a high-voltage line with the right parietal area of his head. He was thrown approximately 20 ft from the utility bucket to the ground. He was transported by ambulance to a local hospital where examination on arrival revealed a pulse of 137 bpm, respirations were 32 and grunting, and he had palpable carotid artery and femoral pulses. Pupils were 4 mm bilaterally reacting to 2 mm. The patient’s Glasgow Coma Scale score was 7 (eye score, 1; verbal score, 2; and motor score, 4). The entrance wound was in the right parietal area, with exit wounds at the feet bilaterally. There was 1.5 cm of exposed calvarium in the right parietal area, burns to the right ear and right side of the neck, and circumferential full-thickness burns of the right upper extremity. There were additional burn injuries to the chest and both legs (total 23% burns). The patient was intubated on arrival. He later suffered 2 episodes of tachydysrhythmias requiring lidocaine, adenosine, and defibrillation. He was transported by air to the University of Iowa, arriving approximately 3 hours after the initial injury.

Examination. The patient was pharmacologically paralyzed for the flight, and on arrival his Glasgow Coma Scale score was 3; his pupils were reactive bilaterally. Appropriate resuscitation ensued as well as a standard trauma workup, including acquisition of a FAST (focused abdominal sonography for trauma) scan of the abdomen and pericardium that was negative. Head, neck, pelvis, and abdomen CT scans were then obtained. While the neck, abdomen, and pelvis CT scans were essentially unremarkable, head CT scans showed markedly enlarged ventricles with open cisterns (Fig. 1). Repeated neuro-
logical examination revealed a 7-mm left pupil with trace reaction, and the right pupil was not visible due to the burn injury and subsequent swelling. The pharmacological paralysis had worn off, and there was trace flexion in all extremities. The patient lost pulses in his right arm at this point and was taken to the operating room for placement of a ventriculostomy and escharotomies.

Treatment. A decision was made to place the ventriculostomy in the left frontal area because of the exposed calvarium on the right. On insertion, the opening pressure was markedly elevated with loss of ~ 20 ml of fluid with the initial placement and an estimated opening pressure of > 30 mm Hg. The right parietal scalp wound was debrided back to the bleeding, viable tissue (Fig. 2). The underlying calvarium was discolored but intact. The wound was packed with bacitracin-soaked sponges and covered with a sterile dressing. The patient was returned to the intensive care unit.

Initial CSF laboratory values were unremarkable (glucose level 106 mg/dl [normal range 40–75 mg/dl], total protein 16 mg/dl [normal range 15–45 mg/dl], 1 white blood cell, and 3 red blood cells). Gram stain and culture were negative. The patient remained on prophylactic nafcillin for skin flora coverage. Immediately postoperative, there was the need for bone grafting of the calvarium. Fortunately, only the outer table was removed, as bleeding, healthy, viable-appearing bone was seen in the diploe, and the inner table was left intact, eliminating the need for bone grafting of the calvarium.

Unfortunately, surveillance CSF cultures obtained from the lumbar drain were positive for Pseudomonas 2 days after surgery. The patient was treated with appropriate antibiotics, and the Pseudomonas infection resolved and was replaced by a Candida infection. During this time, his lumbar drain became occluded and could not be replaced, even with fluoroscopic assistance. Within hours, the patient developed signs of symptomatic hydrocephalus and experienced nausea, vomiting, headache, upgaze paresis, and decreased level of consciousness. Head CT scanning demonstrated marked enlargement of the ventricles (Fig. 4). He was taken to the operating room and a right frontal ventriculostomy was placed while taking care not to compromise the vascular pedicle to the graft, and the patient’s symptoms resolved immediately with CSF drainage. Ultimately once the CSF was cleared, a lumbo-peritoneal shunt was placed without difficulty. A lumbo-peritoneal shunt was chosen because of the likelihood of further scalp and cranial reconstructive procedures in the future, and we wished to place the shunt out of the future operative field. The patient was doing well at the 4-month neurosurgical follow-up visit, and at the 6-month neuropsychiatric follow-up evaluation his cognition was, according to the report, “within expectations in all areas assessed, with no clear evidence of brain dysfunction.”
Discussion

To our knowledge, this is the first report in the literature of symptomatic hydrocephalus requiring neurosurgical treatment following an electrical injury. Cerebral venous thrombosis, brain hemorrhage, myelopathy, amyotrophic lateral sclerosis, altered sensory nerve function, and tetraplegia have all been previously reported in association with high-voltage electrical injuries. Delayed onset of peripheral neuropathies have been reported in as many as 17% of patients, with 64% resolving over time. Significant neurobehavioral sequelae—including cognitive dysfunction such as impairment of memory and attention, deficits in verbal learning, and delayed recall of verbal information—have also been reported. Not surprisingly, depression, anxiety, and irritability are also widespread in patients suffering from high-voltage electrical injury. Other known complications of electrical injury include peripheral nerve and spinal cord damage, as well as early development of cataracts, renal failure, and dermal burns. Cardiac arrhythmias also occur, as was the case in our patient, and the hypoxic insult incurring during arrhythmia and cardiac arrest may also cause secondary nervous system injury.

The mechanism of damage in these injuries is not well elucidated. Current theory holds that the injury is a multifactorial combination of thermal and nonthermal causes. In thermal injury, electricity generates heat as it passes through body tissues. The amount of heat generated is proportional to tissue resistance. Tissue resistance, ranked from highest to lowest, is bone, fat, tendon, skin, muscle, blood vessels, and nerve. Theoretically, the tissues with highest resistance should generate the most heat, but in animal models this theory is not supported, with the body acting as a volume conductor in which there is a single uniform resistance. Thermal injury may also result in vasoconstriction of cerebral blood vessels, with intimal injury and subsequent thrombosis a possible mechanism for delayed injury manifestations. In nonthermal injury, electrical destruction of cells may occur both directly and indirectly. This effect may be due to disruption of the sodium-potassium-ATPase pump, which normally operates at −90 mV of direct current, thereby rendering it vulnerable to high-voltage alternating current that may disrupt this pump. Breakdown of cell membranes and electroporation of cellular membranes are potential mechanisms for these injuries. Nonthermal mechanisms of injury appear to be especially important in nervous system injury, as the injury seen is not well explained by heat injury alone.

There is little information on electrical injury and its relationship to the CSF and ventricular system. Cerebrospinal fluid temperatures of 145°F have been recorded 5 hours after a legal electrocution. The authors of a 1988 case report hypothesized that a superior orbital roof blowout fracture in the setting of an electrical injury occurred as a result of heating of the tissues and CSF, with resultant steam production disrupting the weakest portion of the skull. A series of articles in the German literature have described the incidental finding of large ventricles on a pneumoencephalogram obtained in a 25-year-old crane operator who had sustained a high-voltage electrical injury.
Hydrocephalus following electrical burn injury

Details surrounding this man’s injury were not well described, and the authors admitted that the large ventricles may have been present prior to the injury. They subsequently studied animal models and demonstrated ventricular enlargement in a cat model that had been exposed to an electrical injury, which was not noted in control group. In a dog model, the animal’s cerebral cortex and meninges were directly observed while a 220-V electrical current was applied to the animal’s paws; cerebral cortex pallor lasting from 90–150 seconds was observed. The details of these experiments were not rigorously detailed, and it is unclear whether the “hydrocephalus” later observed was from white matter degeneration/volume loss such as is seen in an ex vacuo hydrocephalus or from an alteration in CSF production or absorption resulting in hydrocephalus with elevated ICP. Further information regarding the animal’s outcomes was not made available. In any case, these experiments were conducted prior to the advent of modern neuroimaging, which makes them more difficult to interpret.

We are unsure if, in our patient, there was an enlarged ventricular system prior to the injury. However, clearly, after the injury, the patient exhibited symptoms of elevated ICP, and he poorly tolerated situations in which CSF diversion was not functional. Attempts to wean the patient from CSF drainage in the days and weeks after the injury were unsuccessful, and a shunt was subsequently inserted. In this particular patient’s case, the hydrocephalus was noted early, because of CT scanning undertaken as part of a trauma workup performed because of the patient’s fall and the obvious cranial contact point. One could ask whether this phenomenon may occur more frequently, and whether untreated hydrocephalus could contribute to increased morbidity in patients suffering a high-voltage electrical injury. Certainly in a patient who went into cardiac arrest twice en route to the hospital, neurological changes might be attributable to the arrest, and not aggressively studied in a patient with a seemingly poor prognosis.

The mechanism by which this young man became symptomatic is unknown. His head CT scan did not demonstrate any evidence of hemorrhage or stroke. There was no suggestion of cerebral sinus thrombosis (for which, admittedly, MR imaging would have been a more sensitive screening study), but the enlarged ventricles are still not explained. Also, we observed no venous sinus thrombus sequelae, such as venous infarction and brain swelling with decreased ventricular size, which would be anticipated with sinus thrombosis. The ventricles were large, but we do not know the size of the ventricles prior to the injury. It is interesting to speculate whether the increased CSF temperature, such as has been recorded in electrocutions,15 may have altered the permeability of the arachnoid granulations that absorb CSF and diverted it into the venous system, resulting in increased CSF accumulation and hydrocephalus. This theory is potentially attractive, because of the low tissue resistance to current in neural and vascular structures. Alternatively, small clots that were not discernable on conventional radiographic studies may have occluded or otherwise impaired the permeability of the arachnoid granulations. Certainly, the arachnoid granulations are vulnerable to injury and impaired CSF absorption. This vulnerability has been shown in other situations such as inflammation (meningitis) or bleeding.

**Fig. 4.** Head CT scans demonstrating change in ventricular size before and after shunt placement. A: Scans obtained several hours after the patient’s lumbar drain stopping functioning, demonstrating ventricular enlargement compared with preoperative baseline status the night of admission (see Fig. 1). B: Scans acquired 3 months after lumboperitoneal shunt placement. The ventricular size is still generous but appreciably decreased from the previous scans. The patient was well, with good cognitive function and no signs of shunt failure at the time of this study.
(subarachnoid hemorrhage) in which a number of patients have required shunts for CSF diversion. Analysis of the CSF in our patient notably did not suggest either of these possibilities, as the initial studies demonstrated a normal total protein and only 1 white blood cell.

The management of electrical scalp burns has been described in the literature. Briefly, if the calvarium is intact and retains some overlying galea, the burned area is excised and the galea is directly covered with a skin graft. If the calvarium is burned, it is debrided until only healthy viable bone remains. Soft-tissue coverage is accomplished using a full-thickness rotational graft, and the adjacent donor region is covered with split-thickness skin grafts. Drilling multiple, closely spaced holes into the bleeding diploic bone to promote granulation tissue has also been described. Tissue expanders can be used to expand hair-bearing tissues once the grafts have matured. Very large areas may require free flaps with appropriate anastomosis to scalp or cervical vessel. Reconstruction of the scalp is an important topic when one considers CSF diversion strategies.

Conclusions

We have described a case report of new-onset symptomatic hydrocephalus occurring in the setting of high-voltage electrical injury. This phenomenon has not been previously reported. Mechanisms of the injury could hypothetically relate to altered permeability of the arachnoid granulations or changes in vascular structures that could not be observed on imaging. The need for continued CSF diversion to treatment this entity in the setting of a burn injury is challenging, but with careful planning and a team approach, hydrocephalus was successfully treated. This case report raises the question of whether new-onset symptomatic hydrocephalus could occur in other patients sustaining burn injuries. We encourage early neuroimaging, as it was key to the early diagnosis and treatment of this patient, whose outcome was good.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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