

Delayed Neurological Injury Following Electrical Injury/Burn: 2 Case Reports

Case 1: A 59 y/o M was cutting trees in a cherry-picker and made contact with a high-voltage wire, sustaining a combination of 2<sup>nd</sup>, 3<sup>rd</sup>, and 4<sup>th</sup> degree burns to his head, face, neck, chest, and both UE's, totaling 30% TBSA. He was conscious and able to move all 4 extremities. His initial hospital course was appropriate, consisting of airway management, sedation, fluid resuscitation, and multiple surgeries for excisions and grafting. He was weaned off sedation and extubated on HD#10, at which time he was noted to have full sensation, but no movement in any of his extremities. What happened? Wasn't he able to move his extremities initially? Did we miss something on our initial exam? Did we do something to cause this during his complicated hospital course?

Case 2: A 51 y/o M was changing light bulbs on a large sign when his L shoulder made contact with a socket, causing a small 1x1cm contact electrical burn to his L deltoid. At that exact moment, he had both of his hands weight-bearing through the edge of the large lift basket that he was working from and he was unable to release his weight or grip from his UEs for several seconds. He finally broke contact and was stable at that time. He presented to the ER 4 days later with a discomfort in his proximal RUE (contralateral to the contact point) and a "cramp" sensation which progressed over the next 48 hours to a full tetany of the R deltoid, biceps, triceps, and forearm flexors/extensors, unable to be relieved with large doses of muscle relaxers, anticonvulsants, and analgesics. This progressed further to weakness and incoordination of the RLE and vocal tics over the next 6 days of his hospital course. So what happened in this case? And why did it take so long to present?

A PubMed review of the literature using MeSH terms "Electric Injury" and "Delayed Neurologic Injury" resulted in only 35 papers. Of these, 16 papers were directly relevant to our patients above. 14 of those papers were case reports describing delayed neurological injury of variable severity following high-voltage electrical injury (not associated with lightning strikes). One of those papers specifically discussed a report of a patient with clonus following high-voltage electrical injury, but concluded that clonus was a sign of an upper motor neuron lesion rather than a specific neurological complication by itself. Of the 2 remaining papers, one described imaging findings of the neurological system after electrical injury, concluding that there are usually no specific MR findings (in the absence of trauma) to fully explain the degree of associated neurological injury. The final paper was a 7-year review of 13 patients who sustained high-voltage electrical injuries and developed delayed neurological injuries.

The review indicated that there was a pattern to the injury based on the entry and exit points. The authors determined that 11 of the patients had entry sites at the head/neck and the other 2 patients had entry sites at the hand. To summarize their findings, if the exit site was the lower extremity (with either entry site), the patient would have resulting paraplegia. If the exit site was the upper extremity (with either entry site), the patient would have resulting quadriplegia. In all of the patients, no complete loss of sensation

was noted. All of the cases presented **within 2-10 days post-injury, and all were characterized by ascending paralysis.**

The **suggested mechanism of these injuries was a disruption of vascular supply to the anterior spinal cord.** The anterior gray matter, especially the anterior horn cell, is particularly susceptible to ischemic injury because the blood supply is from the sulcal branch, the longest branch originating from the anterior spinal artery. The specific pathophysiology is likely due to possible thrombosis from endothelial injury or a diffuse degenerative change or necrosis of the vascular wall from the extreme heat. It was also suggested that vasospasm may play a role as well. It was suggested that the small-diameter vessels were more susceptible to these effects because they could not dissipate the heat as well as the large-diameter vessels. The authors also discussed how the path of current through the spinal cord plays a role in the pattern of injury. The cord at T4 to T8 is more vulnerable to ischemic injury due to poor collateral circulation. With relatively improved circulation above and below this region, any current that travels inferiorly through this portion of the cord results in paraplegia at these levels. When electrical current primarily exits through the middle or lower cervical cord and out through the upper extremities, this results in injury to this portion of the cord and a patient with quadriplegia.

The authors suggest the treatment to prevent the neurological sequelae be directed toward improvement of circulation. These included two specific interventions. They administered prostaglandin E1 to two patients under the hypothesis that vasospasm played an important role in the vascular injury. They also advocated for the administration of high-dose steroids to be systematically administered at an early stage. The authors did not report on whether either of these interventions actually improved their patients' outcomes.

In conclusion, it appears that delayed neurological injury following high-voltage injury is a **rare event** that is not well-described in the literature. In case reports where it is described, **complete recovery is not the rule, and when it does occur, it can take months to years for any neurological recovery.** In addition, **no specific interventions have been thoroughly investigated.** However, the suggested pathophysiology indicates a role for early administration of PGE1 and steroids. This remains an area where major advances can be made as further research is conducted.

#### References:

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3. Deveci M, Bozkurt M, Sengezer M. Clonus: an unusual delayed neurological complication in electrical burn injury. *Burns.* 2001 Sep;27(6):647-51.