Carotid Rupture Following Electrical Injury: A Report of Two Cases

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Electrical injuries often result in extensive tissue damage where vascular damage may occur and result in thrombosis and spontaneous rupture of blood vessels. Rupture of the brachial, radial, ulnar, internal mammary, and obturator arteries has been reported in the literature. The authors present two cases of carotid artery rupture following high-voltage electrical injuries. The first case is a 21-year-old man who was climbing a fence near a high-voltage power line when a gold chain he was wearing around his neck caught on the power line, resulting in a 10% circumferential electrical injury to his neck. He presented with visible arterial bleeding from the large neck wound and was taken to the operating room, where a 1-cm laceration to the carotid artery was repaired with a vein patch. On the second postoperative day, the patch dislodged, and a spontaneous rupture of the common carotid artery occurred. The damaged artery was subsequently ligated. The patient recovered with no neurological sequelae. The second case is a 43-year-old man who suffered a high-voltage injury while working on an electrical panel, resulting in a 50% TBSA full-thickness burn to the face, scalp, trunk, and extremities. Four weeks after admission, a latissimus dorsi myocutaneous free flap was used for coverage of exposed outer table of the skull. Intraoperatively, the carotid artery spontaneously ruptured proximal to where the dissection was being carried out. The patient recovered with no neurological sequelae. High-voltage electrical injury results in significant damage to blood vessels via a number of mechanisms. Rupture of a major vessel is a rare, life-threatening sequelae of electrical injury. (J Burn Care Res 2012;33:e161–e166)

Electrical injuries result in significant soft tissue damage while producing relatively small TBSA burns.[1] The tissue damage is principally a result of heat production, but membrane permeabilization[2] and protein denaturation also play a role.[3] The amount of damage done is determined by various factors such as the type of electrical current, the path taken by the current, the resistance of the conductor, and the duration of contact with the source.[3–5] Management of the resulting wound comprises extensive early debridement and definitive coverage with flaps or grafts.[3,6]

In addition to the local tissue damage, electrical injuries may lead to neurologic deficits, musculoskeletal damage, and cardiovascular injury. Severe vascular damage may occur making the blood vessels involved prone to thrombosis and spontaneous rupture.[7,8] Rupture of brachial,[1] radial, ulnar,[4] internal mammary,[9] and obturator[10] arteries has been previously reported in the literature. Herein, we present two cases of common carotid arterial rupture following high-voltage electrical injuries.

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CASE PRESENTATION

Case 1

A 21-year-old male student was climbing a fence near a high-voltage power line. The gold chain he was wearing around his neck caught on the power line resulting in a 10% TBSA full-thickness circumferential electrical contact injury (Figure 1A, B). The patient

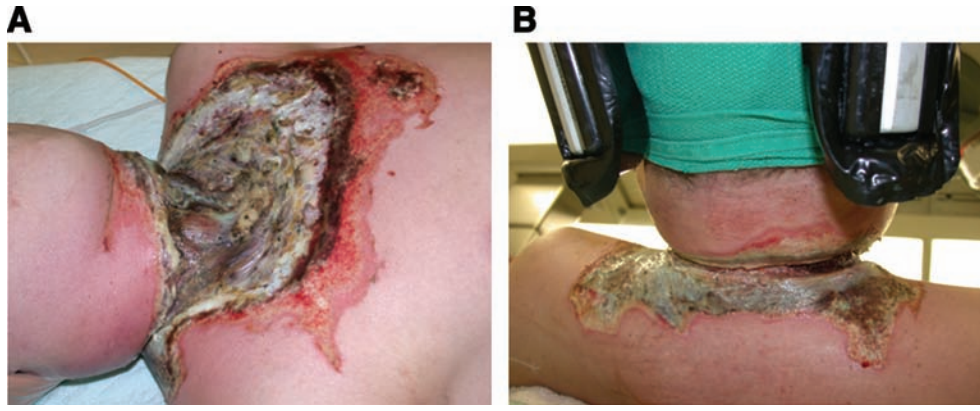

Figure 1. Case 1: Anterior (A) and posterior (B) views of circumferential high-voltage electrical injury to the neck.

then fell approximately 20 feet to the ground and was found unresponsive by friends. He was brought to the emergency department at the University of Alberta Hospital. On initial examination, the patient had a large neck wound with brown-black eschar and visible arterial bleeding. The initial systolic blood pressure was 60 mm Hg. His right pupil was slightly larger than the left, and the duration of the possible anoxic cerebral insult was unclear. The patient was spontaneously moving his legs. Chest and pelvic x-rays were normal. CT scans were performed of the head, spine, abdomen, and pelvis, all of which were normal aside from the soft tissue damage to the neck.

Primary management comprised intubation, fluid resuscitation, and an urgent surgical exploration of the neck wound. The internal jugular vein was completely coagulated and a 1-cm common carotid artery laceration was discovered. There was good back flow present in the damaged carotid artery. Neurosurgery placed a carotid shunt, and a saphenous vein patch was applied to the injured carotid. The wound was closed, and dressings were applied to the burn wounds on the patient's neck, chest, and scrotum.

Postoperatively, the patient was admitted to the general system intensive care unit with a pH of 6.88 and a hemoglobin of 11.0. An arch angiogram was performed, which demonstrated a normal right carotid artery (Figure 2A) and a pseudoaneurysm in the mid-left common carotid (Figure 2B). The patient was neurologically intact 36 hours after the injury. On the second postoperative day, bleeding was again noticed from the large neck wound. The

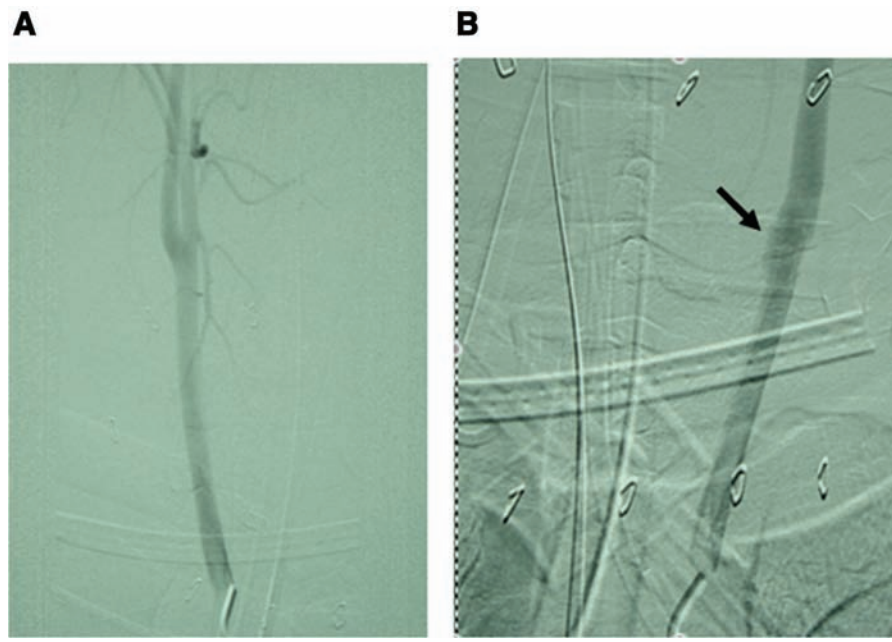

Figure 2. Angiogram of right common carotid artery demonstrating normal filling (A) and angiogram of left common carotid artery demonstrating pseudoaneurysm (arrow) (B).

patient returned to the operating room for reexploration. The saphenous vein patch had dislodged from the common carotid artery, and there was also a rupture proximal to the patch in an area of unhealthy looking vessel. The vessels had a region of white opaque color with minimal adherence of the adventitia around it. There was also no vascularity on the surface of the common carotid artery. Neurosurgery was consulted intraoperatively, and a decision was made to ligate the left common carotid. No further debridement was performed at this time although unhealthy injured vessels and necrotic tissue was present. The patient was hemodynamically stable throughout the operation, and plans were made for staged debridement.

On the fifth postoperative day, debridement was performed of the complex electrical burns to head and neck regions (Figure 3A). Electrical injuries on the scrotum and right lower extremity were also debrided. Three days later, the patient returned to the operating room. Direct laryngoscopy and bronchoscopy were performed, which demonstrated necrosis of the anterior trachea. A nasoendoscopic examination of the larynx was also performed, which demonstrated paralysis of the vocal cords. A tracheostomy was placed through the necrotic trachea wall. The neck wound was further debrided, and coverage was achieved with a pedicled pectoralis major muscle flap and autograft. The patient returned to the operating room again 2 weeks later for repeat debridement and coverage with autograft to the neck, chest, and scrotal wounds.

Esophagogastroduodenoscopy demonstrated a normal esophagus and stomach. Vocal cord edema and necrosis of the anterior trachea were noted on a repeat bronchoscopy.

On day 30 postadmission, the patient returned to the operating room for a third debridement and autografting of wounds on the neck and chest. Necrotic adductor tendon was debrided from the medial thigh. Necrotic fascia and testicular necrosis were debrided and covered with bilateral scrotal advancement flaps. The neck and groin wounds were once again debrided, and on day 47 postadmission, the groin wound was covered using a pedicled gracilis myocutaneous flap. The trachea was reconstructed on day 71 with a mucosal graft and right radial forearm free flap, and four days later the patient was extubated uneventfully. The patient's voice spontaneously recovered, and he had good swallowing function as demonstrated by a barium swallow. He required a suspension laryngoscopy and bronchoscopy to repair a small tracheal fistula. The patient was discharged soon after (Figure 3B) and tolerated the reconstructive procedures well, with no neurological sequelae and his voice having fully recovered.

Case 2

A 43-year-old man received a high-voltage (4000 V) injury while working on an electrical panel. The resulting flash electrical fire ignited his clothing, and the patient sustained a 50% TBSA full-thickness thermal burn. The patient was initially unresponsive, and assistance was delayed due to the electrical hazard posed by the live panel. He was transported via ambulance to the local hospital. On presentation to the emergency room, the patient was conscious with a Glasgow Coma Scale score of 7. He was somewhat combative and was moving all four limbs spontaneously. He was tachycardic and tachypnic, and no blood pressure could be obtained. Electrical contact sites were evident near both ears. The resulting burn injury involved the scalp, face, neck, anterior trunk, both upper extremities, and the anterior thighs (Figure 4). The patient also suffered from an inhalational injury.

Primary management comprised intubation and fluid resuscitation to maintain a urine output of 30

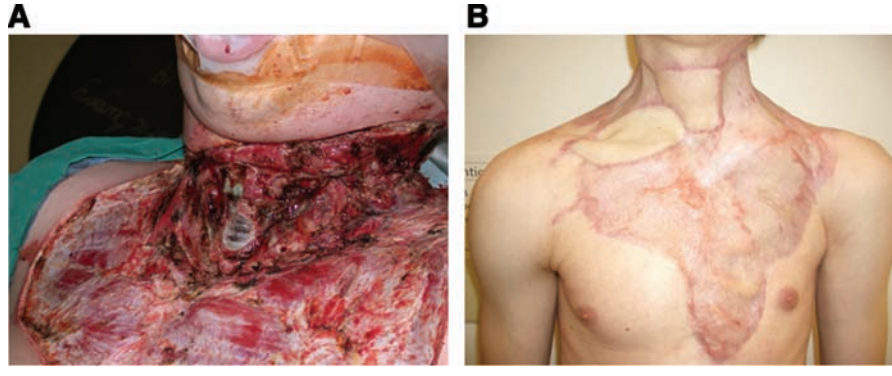

Figure 3. Neck wound following initial debridement (A) and the neck wound following reconstruction with pedicled pectoralis major muscle flap, autografting, and radial forearm free flap (B).

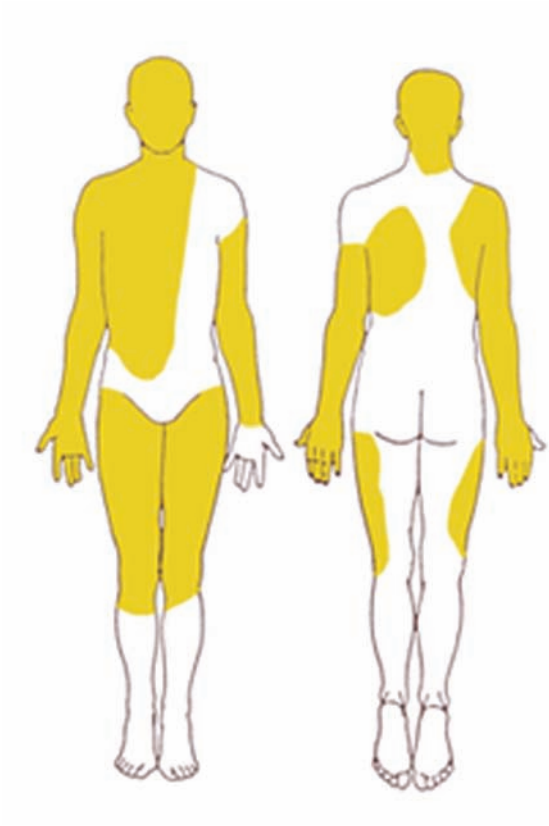

Figure 4. Flash electrical fire resulted in 50% TBSA thermal burns involving head and trunk, along with upper and lower extremities.

ml/hr. Polysporin and sterile gauze were initially applied to the wounds. The patient was then sedated and paralyzed for transfer and flown to the University of Alberta Hospital where he was immediately admitted to the burn unit.

Four days after admission, the patient went to the operating room for debridement and autografting of multiple burn wounds on his upper and lower extremities. Allograft was applied to his face. A week later, he returned to the operating room for further debridement and autografting of his trunk and shoulders. On postadmission day 18, another operation was undertaken in which a large area of necrotic bone was debrided from the outer table of the skull using a pneumatic burr, exposing the diploe in some areas. The inner table of the skull remained intact and appeared viable. A repeat debridement and autograft coverage was performed for wounds on the face (Figure 5A), upper extremities, and lower extremities.

Ten days later, on postadmission day 28, a latissimus dorsi muscle free flap was planned to cover the exposed inner table and diploe of the skull. During this procedure, a team exposed the recipient vessels in the neck for the free flap. The external and internal jugular veins were exposed, and then while dissecting the facial artery, a spontaneous rupture of the common carotid artery occurred approximately 8 cm proximally to where the dissection was being carried out. The artery appeared unhealthy at the time of dissection, likely from electrical-induced injury in the region. There was minimal adherence of the adventitia surrounding the vessel, and there was a generalized pallor of the vessel wall with very little vascularity on the surface. The defect was repaired with a saphenous vein graft and the bleeding was controlled. The flap was then inset, and the rest of the procedure went well (Figure 5B).

Two days later, the flap was showing signs of venous congestion, and the patient was returned to the operating room for reexploration of the flap. Necrotic muscle was debrided from the flap, and the outer

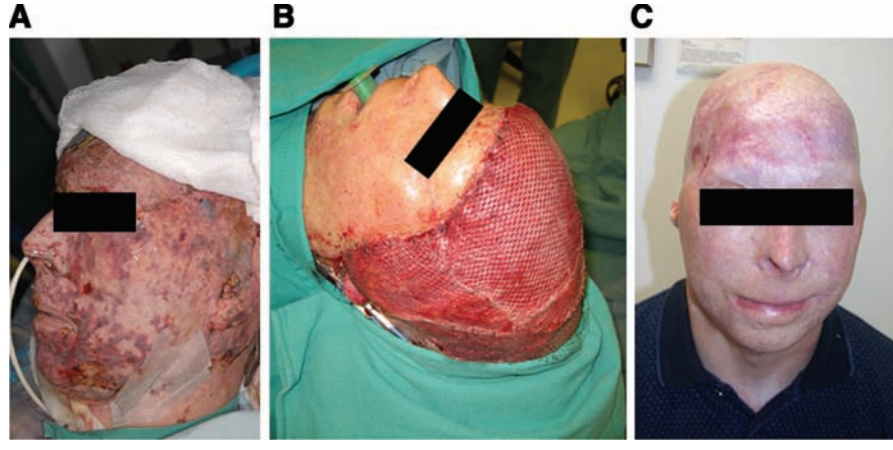

Figure 5. Facial burns following debridement and autografting (A); the spontaneous rupture of the common carotid artery occurred while dissecting recipient vessels for the latissimus dorsi free flap (B); and the patient 3 years after initial injury (C).

table of the skull was once again debrided to healthy viable tissue. Despite the reexploration, the flap failed and was removed from the skull 5 days later, on postadmission day 35. After the flap was removed, the scalp wound was covered with allograft. Autografting was performed for wounds on the face and upper extremities. The patient returned to the operating room 2 weeks later for a repeat debridement of the calvarium, in which small areas of dura matter were exposed. On postadmission day 53, the scalp was covered with Integra™ and a negative pressure dressing was applied. On postadmission day 91, the Integra™ was covered with split-thickness autograft.

Over the course of the following 2 years, the patient underwent further procedures for eyelid reconstruction. The scalp skin coverage was very unstable, and the patient experienced repeated breakdown of some of the grafted areas. The wound was redebrided and a second latissimus dorsi flap was applied which is now doing well. The patient is now in good condition and has returned to work (Figure 5C). He experienced no long-term sequelae to the electrical injury, and his cognitive function was normal upon discharge.

DISCUSSION

Electrical injury results in direct damage to tissue because of the electric current and subsequent heat generation. The amount of direct damage depends primarily on the intensity of the current. Ohm's law states that this current is proportional to the voltage and inversely proportional to the resistance of the tissue. Although one would expect the current density to be highest in nerves and blood vessels because of the low resistance, in reality the tissues act as a volume conductor, with the composite resistance of all the tissue components. The heat that is generated by the passage of the current can be quantified with Joule's law, which states that heat is proportional to the square of the voltage and to the resistance of the conductor.[3,5,8] Although the resistance and voltage are important for determining the current and the heat generated, other factors that determine the clinical injury are the duration of contact with the conductor and the pathway of the current flow.[3] Both the patients presented here sustained high-voltage injuries that resulted in a large amount of current flow and heat generation in the neck. In the first patient, the underlying damage to the vessels of the neck was not evident until they were exposed in the operating room. In the second patient, the large amount of soft tissue damage to the skin and muscle overlying the carotid arteries made the damage readily apparent.

High-voltage electric injury results in damage to blood vessels via a number of mechanisms. Heat generation due to the passing current causes direct damage to the tissues. Robson et al[11] demonstrated that electrical injuries result in elevated levels of thromoxane A_2. The high thromboxane A_2 levels contribute to progressive tissue necrosis by causing vasoconstriction and thrombosis in the microcirculation. Partial or complete vessel occlusion may occur at the time of the injury,[12] especially in smaller nutrient arteries,[13] and delayed arterial thrombosis may also occur.[7]

Electrical injury to blood vessels causes varying degrees of damage to different layers of the vessel. Wang et al[2] examined aorta and pulmonary artery endothelial cells from electrocution victims and found that cell membrane perforations were present within 24 hours of the electrical injury, whereas after 24 hours, the endothelium disintegrated. Jaffe et al[14] found that electrical injury caused a complete loss of endothelial cells. The muscle fibers of the media were most sensitive to electric current, whereas the adventitia showed little change. The vessels lost their elasticity, and fusiform aneurysms were common. Carmeliet et al[15] demonstrated similar findings using a perivascular electrical injury to femoral arteries in mice. Smooth muscle cells in the media were injured, the endothelial cells were completely denuded, and the vessel wall was infiltrated with polymorphonuclear cells. The denudation of endothelial cells resulted in nonocclusive mural thromboses caused by platelets adhering to the exposed basal lamina. Examination of blood vessels in amputated human extremities following electrical injuries demonstrates similar changes in blood vessels.[16] Electron microscope examination of blood vessels following electrical injury has shown that the loss of endothelial cells and smooth muscle cell damage is still present 2 months after the injury.[17]

These two cases also raise the question of how to manage carotid artery injuries and minimize postoperative neurological damage. In both cases, repair of the ruptured artery was attempted. Ledgerwood et al[18] and Pearce and Whitehill[19] found that neurological deficits following a carotid artery injury were well correlated with the neurological status on admission and not on whether repair or ligation is performed. The presence of shock on admission and occlusion of the injured vessel are also correlated with neurological impairment following surgery for carotid artery injury.[18] Patients who are neurologically normal on admission have better postoperative outcomes with repair than with ligation.[19,20] Repair of the injured vessel is recommended for patients who have stable vital signs, who are not comatose, and who have a technically repairable injury with good wound

coverage.[18,20] Ligation is recommended for patients with severe cerebral injury and for patients with trauma to the arteries in which a repair is not feasible and coverage of the repair cannot be readily achieved.[19]

It is important to note that the majority of patients in the reviews by Ledgerwood et al and Pearce and Whitehill suffered either gunshot wounds or stab wounds to the neck. In many of these patients, the injured portion of the carotid artery was likely surrounded by healthy, uninjured artery that would better support a repair. In a patient with electrical injury, the arterial damage is widespread, making repair of the vessel a less feasible option. In case 2, we were fortunate that the saphenous vein graft was successful in repairing the ruptured vessel. Despite the electrical injury to the vessel, it still had enough structural integrity to support the repair. In case 1, the damage to the common carotid was likely far more widespread than the 1-cm laceration that was evident. When the patient returned to the operating room following the venous patch dislodgement, the carotid arteries appeared unhealthy and there was necrotic tissue present. In this case, it may have been better to ligate the artery initially rather then attempting a repair on such an unhealthy artery.

It is highly likely that the failure of the venous patch in case 1 along with the rupture of the common carotid arteries in both cases were due to the damage caused by the electrical injury. The damage caused by electrical injuries not only affects the viability of muscle and soft tissue but major blood vessels as well. The electrical injury likely resulted in formation of mural thromboses, invasion of inflammatory cells into the vessel wall, and weakening of the vessel wall.[2,14] The weakness in the vessel wall made the carotid artery in case 1 unamenable to repair and caused a rupture of the carotid artery in both cases. Given the patient's medical history and the young age of the patient in case 1, it is unlikely that preexisting aneurysms or atherosclerotic damage played a role.

Both patients had successful outcomes, with no long-term neurological sequelae. These cases reaffirm the need to closely monitor the fluid and oxygenation status of patients who have sustained electrical injuries, especially when the injuries are near major vessels.

CONCLUSION

Two cases of common carotid artery rupture following electrical injury have been presented. One patient suffered a major circumferential neck burn that resulted in a common carotid laceration along with extensive soft tissue damage. The other patient suffered a burn that resulted in the loss of the majority of his scalp. The damage done to deep structures in his neck was not evident until the carotid artery ruptured while attempting a free flap for scalp coverage. Both patients were successfully treated. Close hemodynamic monitoring and the fact that the carotid ruptures were readily apparent both contributed to the successful outcomes in these two patients.

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