LATE COMPLICATIONS OF HIGH-VOLTAGE ELECTRICAL INJURY MIGHT INVOLVE MULTIPLE SYSTEMS AND BE RELATED TO CURRENT PATH

LES COMPLICATIONS TARDIVES DES ÉLECTRISATIONS PAR HAUT VOLTAGE PEUVENT ÊTRE MULTIFOCALES ET LIÉES AU TRAJET DU COURANT

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SUMMARY. Delayed complications of electrical burns are mostly unexpected, and the link between the injury and the symptoms often goes unrecognized. A possible relation between source-ground sites and late clinical manifestations was recently emphasized. We report a unique case of combined intestinal-spinal delayed complications following a high-voltage electrical injury, a possible explanation being a greater current flow through the right hemisoma. The potential for late complications is an additional feature that physicians must consider in managing electrical injuries. Manifestations are variable and presentation is confounding, but current flow path can constitute a precious source of information to predict complications in the late phase of management.

Keywords: electrical burn, current path, spinal cord injury, visceral injury

RÉSUMÉ. Les complications tardives des électrisations par haut voltage sont le plus souvent inattendues et le lien avec l’accident initial est fréquemment non reconnu. Une relation possible entre le trajet source-terre et les complications tardives a récemment été mise en évidence. Nous rapportons le cas de complication tardive touchant à la fois l’intestin et la moëlle épinière, pouvant être expliqué par le passage préférentiel du courant dans l’hémicorps droit. La possibilité de complications tardives doit être prise en compte des électrisations par haut voltage. Ces manifestations sont variables et la clinique peut être peu claire, mais le trajet supposé du courant peut être un argument permettant de relier l’atteinte initiale et les complications tardives.

Mots-clés: brûlure électrothermique, trajet du courant, lésion médullaire, lésion viscérale

Introduction

Electrical burns typically comprise only a small percentage of total admissions to major burn centres - about 3%.¹ However, they consume enormous amounts of resources as damage may extend far beyond the external lesion. They primarily affect young workmen, and are the most frequent cause of amputations on the Burn Service. Even in the best-case scenario, morbidity, length of hospital stay and number of operations are much higher than expected based on burn size alone.

We report the case of combined intestinal-spinal delayed complications following a high-voltage electrical injury. To the best of our knowledge this has never been reported in the literature.

Case description

A 34-year-old electrical worker was investigating a power failure when he made contact with a live power cable transmitting approximately 2000 volts a/c and sustained a 20% total body surface area (TBSA) burn. Past medical history included a previous traumatic cervical fracture treated surgically. He was not taking any medication at the time of the accident. He was acutely admitted to a national burn centre in Northeast Italy (Padova Burn Unit) with deep partial- and full-thickness burns on his anterior torso, upper extremities, and right thigh and knee (Figs. 1 and 2). The patient was fully awake and alert on admission. There was no concomitant trauma. A source lesion was noted on his right upper arm, and a ground lesion was visible on his right thigh. The patient was resuscitated according to the Parkland formula modified for electrical injuries, and did not have any episode of hypotension. He was started on a high-calorie diet (enteral feeding) and the following medications: lansoprazole, paracetamol, cefotaxime (for prophylactic coverage) and enoxaparin. Cardiac monitoring was done for 24 hours and no cardiac dysrhythmias were observed. Wounds were washed in normal saline and covered with dry sterile gauzes.

On day 2 he was operated on with debridement and donor skin application to all wounds. He made a rapid recovery and

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his clinical condition remained stable. On day 6, the patient complained of new-onset mild abdominal pain localized in the lower quadrants. The abdomen was non-tender but distended. He denied nausea and had no vomiting. He was passing flatus but bowel movements were quiet. An abdominal ultrasound performed the following day was disturbed by marked intestinal meteorism but noted dilated loops of small bowel in the left iliac fossa. The decision was made to manage conservatively with frequent nasogastric tube aspiration and stool evacuation. Over the following 48 hours the abdomen did not distend further but became tender to palpation, general diet was poorly tolerated and flatulence was reduced. An abdominal X-ray on day 8 revealed free air under the diaphragm (Fig. 3). A computed tomography (CT) scan of the abdomen the same day showed intraperitoneal free air with oral contrast leakage into the peritoneal cavity. By day 9 the patient’s clinical condition had worsened to acute abdomen with complete intestinal occlusion, and he underwent an urgent exploratory laparotomy. Intraoperative examination of abdominal content revealed a perforation associated with two small ulcerative lesions in the terminal ileum (20 cm from the ileocecal valve). No other evidence of ischemia or perforation was detected. Ileum resection was performed and clinical recovery was uneventful with extubation on post-admission day 11 (postoperative day 2). On the same day, microbiological wound culture results showed *Acinetobacter baumannii* and *Enterobacter cloacae*. The antibiotic regimen was switched to meropenem and colistin and maintained until culture negativization (day 24). The patient’s hemodynamic condition remained stable and he was scheduled for a further debridement and grafting.

On day 15, the patient started to complain of weakness in his lower limbs. Physical examination confirmed reduced strength (strength grade 2/5 on the L2 level). Sensory examination was normal. Laboratory investigations were non-contributory. The weakness continued to worsen throughout the following 60 hours, and clinical examination on day 18 showed lack of activation in segments L4, L5 and S1 (ankle dorsiflexion, dig I dorsiflexion and ankle plantar flexion) - 0/5 on the right, 2/5 on the left side. However, anal sphincter tone was intact, as were anal stretch, bladder function and bulbocavernosus reflex. Lumbar puncture and immunological studies were unremarkable. Serial MRI scan (contrast-enhanced) on day 19 did not detect any sign of a lesion. The patient was diagnosed with presumptive delayed myelopathy secondary to electric injury. He was treated with aspirin combined with dipyridamole and gabapentin. Neurological symptoms did not progress further.
Over the following 4 weeks his neurological status slowly improved, with partial recovery of strength in the lower limbs.
At 45 days after injury, motor function showed a grade 3/5 muscle strength in all neurological segments. His parameters remained normal and stable. The burns were further debrided and autografted, and all wounds healed without complication. He was discharged on day 58 to an inpatient rehabilitation facility. At a 12-month review the patient reported return to normal motion.

Discussion

Tissue damage due to electrical exposure is caused primarily by the conversion of electric energy to heat, resulting in thermal injury. Damage to internal tissues depends on current density and tissue resistance. If skin resistance is low, burns are less extensive, but more electrical energy is transmitted to internal structures with tissue destruction and organ dysfunction. Therefore, severity of injury is not reflected in the appearance of the external lesion.

Unlike early complications from organ damage (cardiac, renal, septic and early neurologic manifestations), which are well known and closely monitored, events that occur after the acute phase are mostly unexpected. Delayed complications of electrical burns might involve the gastrointestinal, neurological, visual and skeletal systems. Presentation might be subtle and pervasive, and the link between the injury and symptoms often goes unrecognized, leading to delayed treatment with increased morbidity and mortality.

Late-onset intestinal perforations are among the rare gastrointestinal complications of electrical injury, with only four other reports in the literature, manifesting between day 18 and 21. Our case differed from what has previously been reported in the time of onset (day 6). Pathophysiologic mechanisms include mucosal atrophy, change in digestive absorption, increased intestinal permeability and decreased gut blood flow. Mild symptomatology and delays in treatment have been found to increase morbidity and mortality.

Moore et al. reviewed existing reports of late caecum perforation in the literature, highlighting some similarities that were also supported by our case: burn injury of intermediate severity (less than 40%); presentation with a distended abdomen and free air under the diaphragm; absence of bowel ischemia, diverticular disease, foreign body, hypotensive episodes and systemic infections before or during the time of perforation.

Late neurological sequelae are classified as effects that are noted more than 5 days after electrical injury. Most of the symptoms are associated with an injury in the central, peripheral and autonomic nervous systems. Reports of delayed spinal injury have been sporadic, and severity varies from localized paresis to quadriplegia. Patients are at great risk of permanent disability as complete recovery is not the rule. Pathophysiology remains unclear, with authors debating between ischemia vs. inflammation interfering with the integrity of the spinal neurons on the microlevel. However, a recent review by Ko et al. emphasized the relation between source-ground sites with clinical manifestations. They hypothesized four different patterns of lesions according to the assumed current flow. Our case could be described as “pattern nr.1” with current flowing from the neck area to a lower extremity (Fig. 4). Due to the relatively poor blood supply at the level of T4-T8, this area is mostly affected. As in our case, patients with this pattern develop paraplegia but maintain bladder, bowel, and sensory functions. In our case, speculation on current path also applies well to a possible explanation for visceral damage: a source site at the right arm recurs in cases of late caecum perforation, and a ground site on the right thigh further suggests a greater current flow through the right hemisoma.

Conclusion

The potential for late complications is an additional feature that physicians must consider in managing electrical injuries, and they should always be suspected in poly-traumatized patients with electrical burns. Presentation confounds diagnosis, manifestations are variable, and onset time has no relation to the range of burn lesions. Combined intestinal and spinal se-
Quelea have never been reported. Current flow path can be suggested based on the detection of external lesions and can constitute a precious source of information. Attention should focus on this, especially in the late phase of management, and should be accompanied by a thorough physical examination combined with imaging methods and laboratory tests to permit early diagnosis of severe injuries, reducing morbidity and mortality by enabling early intervention.

BIBLIOGRAPHY