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CASE REPORTS

From burn to paralysis: delayed spinal cord injury in high-voltage electrical burns

Sabo G[1,*], Skunca A[1], Banić M[1], Magdić Turković T[1], Vučić M[1]

[1]Department of Anesthesiology and ICU, Sisters of Charity Hospital, Zagreb, Croatia

*Corresponding author: Goran Sabo, Department of Anesthesiology and ICU, Sisters of Charity Hospital, Zagreb, Croatia
E-mail: goran.sabo@kbcsm.hr

ABSTRACT

Electrical burn injuries, though uncommon, can cause severe neurological complications, with sequelae observed in up to 67% of high-voltage cases. This report presents two cases of spinal cord injuries following high-voltage electrical burns in male patients aged 29 and 53. The first patient, a 29-year-old male, sustained 9% total body surface area (TBSA) burns. His initial neurological assessment was unremarkable, but post-injury he developed quadriplegia with preserved sensation. Imaging was inconclusive, and he required prolonged intensive care unit care, showing only modest neurological improvement over time. The second patient, a 53-year-old male, suffered 25% TBSA burns, initially presenting unconscious and requiring resuscitation. He developed delayed paraplegia, with electromyoneurography confirming severe bilateral lumbosacral plexus injury despite normal spinal imaging. Both patients underwent supportive treatment, surgical interventions, and rehabilitation, showing varying degrees of motor recovery. Neurological sequelae in electrical injuries may result from direct electrical damage, vascular injury, or ischemic changes. Despite no clear spinal abnormalities on imaging, significant neurological deficits were present. No specific treatment exists, and corticosteroid use remains controversial. These cases underscore the need for early neurological evaluation, awareness of long-term consequences, and further research to develop standardized treatment protocols.

Keywords: electrical injury; spinal cord injury; quadriplegia; paraplegia; neurologic deficit

Introduction

Electrical burn injuries, though relatively uncommon, can be devastating. They account for approximately 0.04% to 5% of burn unit admissions in developed countries and up to 27% in developing countries [1,2]. In adults, these injuries primarily affect men and are most often work-related [3,4]. Recent research from the World Health Organization's Global Burn Registry indicates that 8.8% of electrical burn injuries patients have a median age of 28 years, with 89.2% being male, and the majority sustaining injuries in the workplace [5]. At the burn Intensive Care Unit (ICU) of the Croatian Burn Unit, data from the past five years show that out of 130 total admissions, 9 (6.92%) were due to electrical burns, with a mean age of 32.22 ± 13.45 years—all of whom were male. This pattern aligns with trends observed in the literature. While electrical burn injuries are relatively uncommon, they pose a significant concern due to their impact on young, working-age individuals in high-risk industries. These injuries can result in severe consequences and impose a substantial socioeconomic burden on this demographic. Morbidity and mortality rates associated with electrical injuries are relatively high, with both short- and long-term physical and psychological consequences [6].

Electrical injuries can be defined as sequels due to accidental contact with electrical power and typically are categorized as low-voltage (LV, < 1000 volts) or high-voltage (HV, > 1000 volts). Injury occurs when electrical current travels through the body upon contact with an electrical source, causing damage through multiple

mechanisms. These include direct tissue injury due to electrical forces and indirect injury through heat generation. Resistance is an important concept in those injuries. Electricity tends to take the path of the least resistance, and when electrical current encounters resistance, heat is generated. Different tissues have varying resistance levels; nerves and blood vessels exhibit lower resistance than bones and fat, making them more susceptible to injury [7]. Significantly, the external appearance of an electrical burn usually does not correlate with the severity of internal damage, as deeper tissues and organs may be considerably more affected than the skin [4,8]. Numerous systems, including cardiac, respiratory, ocular, renal, and nervous systems, within the individual's body can be affected [7]. Neurological complications are observed in 67% of high-voltage and 50% of low-voltage injury cases [9]. These complications include cerebral, spinal, and peripheral nerve syndromes [9,10], with potential outcomes such as hemiplegia, aphasia, epilepsy, cerebellar dysfunction, and delayed spinal cord injury. Here, we present two cases of high-voltage electrical burns in male patients aged 29 (Figure 1) and 53 who developed neurological sequelae due to spinal cord injury.

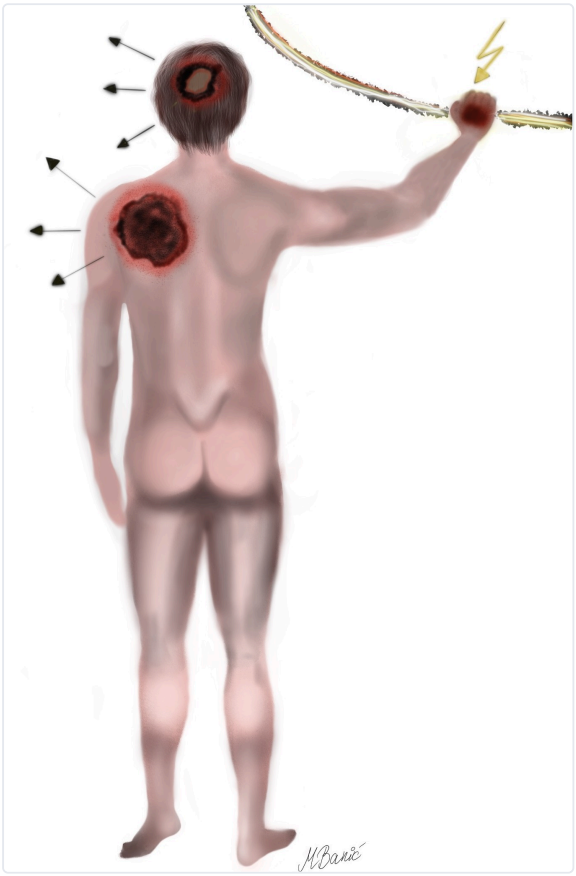

Figure 1. High-voltage electrical burn in a 29-year-old male.

Case 1

A 29-year-old male sustained a work-related high-voltage electrical injury while working on a powerline. His medical history was significant for a prior thyroidectomy, and he was on levothyroxine replacement therapy. On the initial examination, the patient was conscious but highly agitated. He was spontaneously breathing with a SpO2 of 89%, blood pressure of 115/80 mmHg, and a pulse rate of 112 bpm. Neurological assessments revealed no gross neurological deficits. His pupils were isochoric, but the eye bulbs were deviated to the right. Physical examination identified a full-thickness burn measuring 20 × 10 cm on his right upper back region (Figure 2a), a full-thickness burn measuring 10 × 10 cm on his head (Figure 2b), and a second-degree burn on the palm of his right hand. The total body surface area (TBSA) affected was estimated at approxim-

ately 9% (8% full-thickness burns, 1% partial-thickness burns). He was subsequently admitted to the ICU at Zadar General Hospital.

Upon admission, data regarding the fall was unknown, so a computed tomography (CT) scan of the brain, cervical, thoracic, and lumbar spine, as well as the abdomen and pelvis, was performed per the trauma protocol. Since the patient was extremely agitated, sedation was administered, and the patient was intubated and placed on mechanical ventilation for diagnostic purposes. The only notable findings included an interstitial pulmonary pattern suggestive of either pulmonary edema or contusion and a minor pneumocephalus. Later, it was revealed that the patient had not hit his head or fallen during the electric shock. On the second day after the injury, a repeat brain CT scan, performed due to pneumocephalus observed in previous imaging, revealed a hypodense lamellar extra-axial collection in the left frontal region. On the fifth post-injury day, the patient was transferred to the Burn Unit at Sestre Milosrdnice University Hospital Center for further management. Sedation was discontinued on days seven and fourteen, and the patient was extubated on both occasions. However, both extubating attempts were unsuccessful due to respiratory fatigue, necessitating reintubation on the same day. On post-injury day twenty, a tracheostomy was performed, and mechanical ventilation was ultimately discontinued on post-injury day twenty-two. Thereafter, the patient maintained spontaneous respiration. On post-injury day fourteen, following sedation discontinuation and extubation, it was noted that the left pupil was slightly larger than the right. A neurological deficit was also observed, characterized by an inability to move both upper and lower extremities. Sensory examination revealed preserved sensation in all four limbs; however, assessment was limited due to light sedation with dexmedetomidine and sufentanyl. Further neurological workup included a brain CT, cervical spine CT, CT angiography of the head and neck, and magnetic resonance imaging (MR) of the brain and cervical spine, none of which revealed significant pathological findings. The previously noted extra-axial collection on the initial CT scan had been resolved entirely. The laboratory workup for thyroid hormones revealed a TSH level of 94.69 mIU/L, FT4 of 11.5 pmol/L, and FT3 of 1.9 pmol/L, indicating severe hypothyroidism. Treatment was initiated with higher doses of levothyroxine to correct thyroid function, along with vitamin B1, B6, and B12 supplementation.

Over time, the patient demonstrated modest neurological improvement. During his hospital stay, he underwent ten surgical procedures, including debridement, and skin grafting. On post-injury day eighty-six, he was discharged from the ICU to the general ward for further rehabilitation and management and is currently awaiting transfer to the stationary rehabilitation program. Four months after the injury, on the day this report is written, his neurological status has improved. Electromyoneurography (EMNG) shows a right brachial plexus lesion, peripheral polyneuropathy, and myopathy. The presence or level of a spinal cord lesion could not be confirmed or excluded due to inconclusive results. A neurological consultation suggested a probable combination of spinal cord injury and peripheral polyneuropathy with a right brachial plexus lesion. The hand grip in both of his hands has slightly improved. He is able to flex both arms at the elbows and dorsiflex his feet but is unable to lift his legs from the bed. Subjectively reports improvement in stability while sitting.

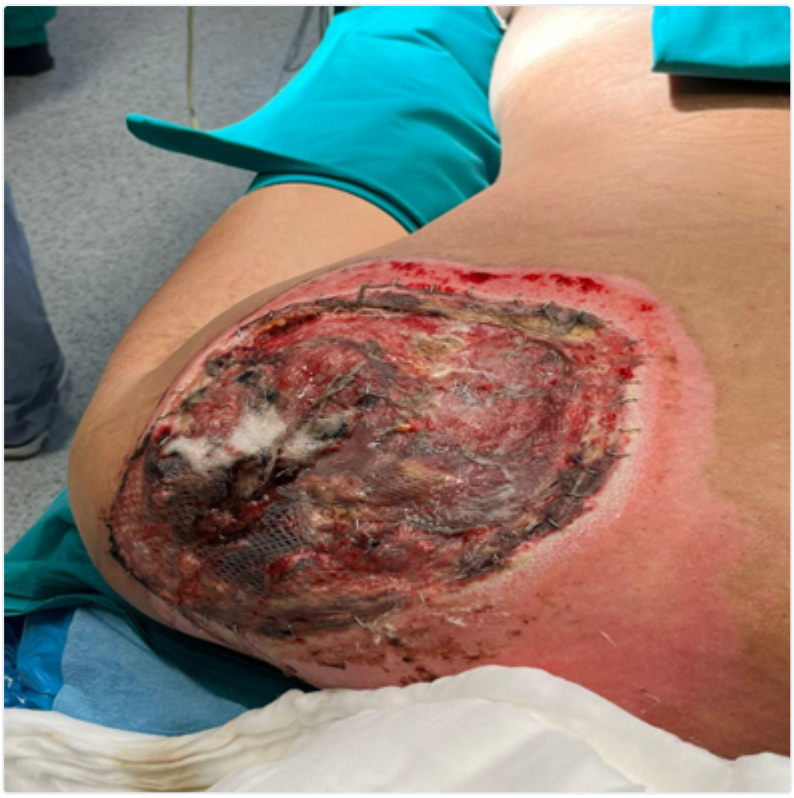

Figure 2a. A 29-year-old male with electrical burn injury: a full-thickness burn on his right upper back region.

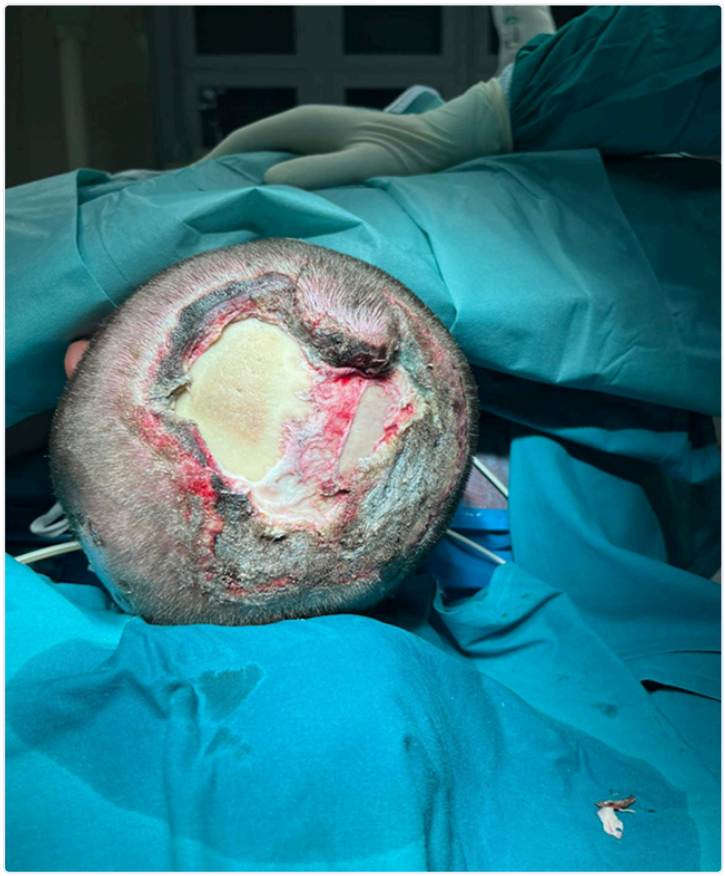

Figure 2b. A 29-year-old male with electrical burn injury: a full-thickness burn on his head.

Case 2

A 53-year-old male sustained a work-related high-voltage electrical injury while working on a powerline (Figure 3). It was unclear whether he fell during the incident. He was initially unconscious and required resuscitation by emergency medical services. Spontaneous breathing and circulation were restored. His past medical history was unremarkable. He sustained partial thickness, and full-thickness burn injuries affecting 25% TBSA, including the face, anterior side of the neck, left anterior thoracic wall, anteromedial side of the right thigh, anterior side of the lower leg, small areas on the right arm, and lower back.

He was admitted to the ICU of Vinkovci General Hospital. Upon admission, he was conscious, spontaneously breathing, and hemodynamically stable. A trauma computed tomography (CT) scan of the head, spine, thorax, abdomen, and pelvis was performed. The only pathological finding was the contusion of the right lung. On the fifth post-injury day, he was transferred to the Burn Unit at Sestre Milosrdnice University Hospital Center, for further treatment. Upon admission, paraplegia with dissociated sensory impairment for temperature sensation was noted. The only significant laboratory finding was severe hypokalemia. A nephrology consultation suspected hypokalemic transient paralysis, but no neurological improvement was observed after potassium levels correction. He received supportive treatment along with vitamin B1, B6, and B12 supplementation to address the neurological deficits and promote recovery. CT scans of the cervical, thoracic, and lumbar spine and MRI of the brain, cervical, thoracic, and lumbar spine showed no demyelination or significant injuries. EMNG revealed severe neurogenic muscle lesions in the right leg, with severe denervation of the peroneal musculature. The conclusion was a severe lesion of the lumbosacral plexus of the right leg. The left leg could not be examined initially due to burns, but after the resolution of burn injuries, an EMNG performed before hospital discharge revealed a severe bilateral lumbosacral plexus lesion.

During hospitalization, the patient exhibited significant improvement in neurological function over time. He underwent four surgeries, including three skin grafting procedures and plastic reconstruction of the hand. He spent 17 days in the ICU, for a total hospital stay of 145 days. After discharge, he was referred to inpatient medical rehabilitation. He continued to be followed for 10 months post-injury after returning home. During this period, he reported significant improvement, started verticalizing and walking, but was still unable to return to work.

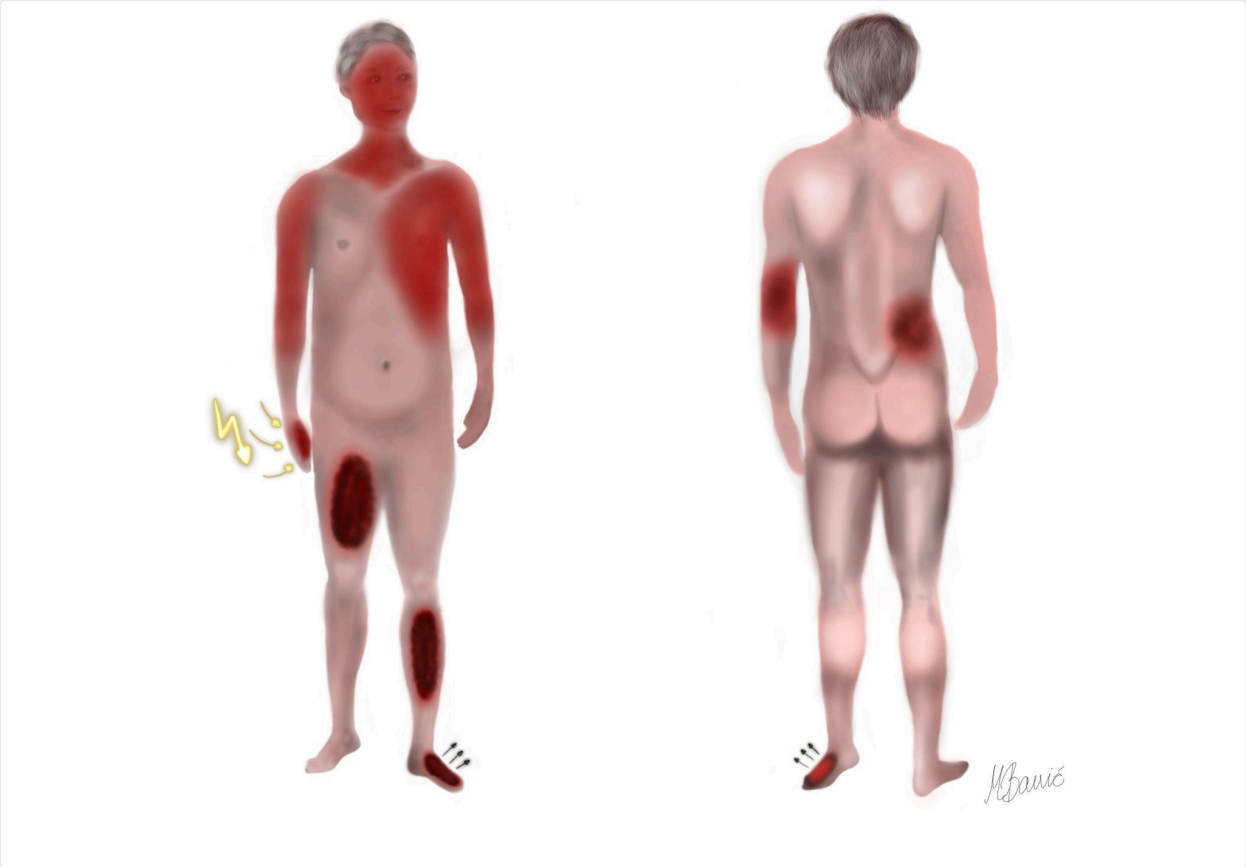

Figure 3. High-voltage electrical burn in a 53-year-old male.

Discussion

Neurological sequelae associated with electrical injuries occur at a high rate, likely due to the low resistance of nerves and blood vessels, which allows electrical current to flow along these structures. The pathophysiology of neurological sequelae following electrical injury remains poorly understood, and several mechanisms have been proposed. Injury may result from direct mechanical or thermal damage to nervous tissue or indirect effects. Commonly suggested mechanisms include vascular damage, pure electrical damage causing electrostatic separation of tissues, electroporation, and alterations in proteins, DNA, and lipids—particularly in myelin cells, leading to demyelination. Neurohumoral influences involving circulating substances such as cortisol have also been implicated [9]. Delayed spinal cord injury has been proposed to result from the degeneration of tiny blood vessels. Specifically, injury to the small branches of the anterior spinal artery, which supplies the anterior two-thirds of the spinal cord, has been suggested as an etiologic factor for delayed spinal cord injury. This hypothesis is supported by findings that motor nerve dysfunction predominantly characterizes delayed spinal cord injury [11]. Consistent with this, our patients presented with more significant motor deficits than sensory deficits.

In patients presenting with neurological sequelae after electrical injury, other possible treatable causes of neurological deficits should be excluded. Both of our patients underwent CT and MRI of the brain and spine, which ruled out vertebral fractures or spinal cord compression. The first patient was diagnosed with a combined central and peripheral neurologic deficit, reminding us that electrical current can damage both the central and peripheral nervous systems, and that such injuries can be combined. The right brachial plexus lesion was attributed to the current entry point, which could have directly damaged the brachial plexus. Additionally, the patient had severe hypothyroidism, raising suspicion of hypothyroid myopathy. Hypothyroid myopathy can occur in both congenital and acquired cases, presenting symptoms such as generalized myalgias, musc-

le weakness, and muscle pain or stiffness [12]. In this case, despite the correction of thyroid function, there was no improvement in neurological status; however, thyroid dysfunction may have contributed to motor weakness. The second patient presented severe hypokalemia upon admission, raising suspicion of hypokalemic periodic paralysis [13]. However, this was promptly excluded, as the patient had no history of muscle weakness and exhibited no resolution of paraplegia after potassium correction. After excluding other potential diagnoses, we concluded that both cases represented delayed-onset spinal cord injury due to electrical current exposure. In the first patient, neurological impairment was noticed on the fourteenth day after discontinuation of sedation and extubating, while in the second patient, it was observed on the fifth day, upon transfer to a specialized burn center. Due to prior sedation, the exact onset of the first patient's neurological deficit could not be determined. This highlights the importance of daily sedation breaks for neurological status examination, as sedation may mask neurological deficits.

Currently, there is no standardized treatment for electrical injury-induced spinal cord damage, and most management strategies are supportive. Corticosteroids are sometimes administrated therapy in periprocedural nerve damage (for example, during anesthesia or pain medicine procedures) [14]. High doses of corticosteroids are rescue therapy in acute traumatic spinal cord injury in selected cases due to their anti-inflammatory effect and reduction of membrane peroxidation. Although recent recommendations suggest that the potential risks may outweigh advantages without significant neurological improvement [15]. Corticosteroids increase the risk of infections, gastrointestinal bleeding, metabolic disturbances like hyperglycemia, and delay wound healing. When discussing electrical injuries, some reports in the literature describe neurological improvement following corticosteroid therapy [11,16]. The exact dosage, duration, and protocol in such cases of electrical injury remain undefined. Additionally, prostaglandin E1 has been mentioned as a potential treatment to improve ischemic tissue injury [11]. Since there is no specific treatment protocol for such injuries, given the absence of visible edema or ischemia on MRI in both patients, and considering potential complications, we decided against corticosteroid therapy.

The only specific therapy, aside from supportive treatment, that was initiated in both patients was vitamin B1, B6, and B12 supplementation after the diagnosis of neurological sequelae, due to their well-known neurotropic effects. Vitamin B plays a significant role in nerve repair and function; however, no research currently addresses its role in electrical injuries. Vitamin B1 (thiamine) contributes to nerve regeneration, vitamin B6 (pyridoxine) is involved in neurotransmitter synthesis, and vitamin B12 (cobalamin) plays a role in nerve regeneration, cell survival, and remyelination [17]. Due to its application, positive effect, and recommendation in other neuropathies and neurological diseases [18], vitamin supplementation was initiated. However, the actual effect of the treatment could not be fully evaluated, as the observed improvement may have been due to the natural course of the disease or rehabilitation therapy.

Outcomes for spinal cord injuries from electrical burns vary, with some patients achieving full recovery, while others experience permanent deficits. At the time of this article, the first patient was in the process of being transferred to inpatient rehabilitation, showing some improvement in motor function of the arms and legs. The second patient had completed inpatient rehabilitation with significant improvement in leg motor function but remained unable to return to work. These cases highlight the severe neurological impact of electrical injuries, emphasizing the need for comprehensive neurological evaluations upon admission, daily assessments for new-onset neurological impairment, greater awareness of long-term consequences, the impact on patients' quality of life after discharge, and further research into treatment protocols. We must always keep in mind the possibility of multiple injuries at both the central and peripheral levels, resulting from various mechanisms, and, when possible, rely on objective EMNG diagnostics.

Conclusions

Electrical injuries can lead to significant neurological complications, including delayed-onset spinal cord injury. A thorough neurological examination is essential in the acute phase, and patients should be monitored for delayed neurological deterioration. Further research is needed to establish standardized treatment protocols for electrical injury-induced spinal cord damage.

Informed consent

Written informed consent has been obtained from the patients to publish this paper.

Abbreviations

CT — Computerized tomography

EMNG — Electromyoneurography

ICU — Intensive care unit

MR — Magnetic resonance imaging

TBSA — Total body surface area

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Data availability. Data is available on request due to ethical and legal reasons.

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