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Peripheral nervous system damage following electrical injury (review)

Electrical injury can lead to neurological consequences in affected individuals. Retrospective cohort studies have shown that in approximately 40 % of cases of high-voltage electrical injury, peripheral nerve conduction parameters were abnormal.

Evidence collection. A retrospective literature search was conducted using a spatial vector descriptive model, supplemented by manual identification of relevant articles. A total of 37 recent sources were included, of which 91.9 % were published within the last 10 years and 78.4 % within the last 5 years.

Evidence synthesis. Disorders of the peripheral nervous system account for approximately 90 % of the neurological consequences of electrical injury and, according to reports from burn units, significantly exceed those observed after thermal injuries. The most common complication is mononeuropathy (including carpal tunnel syndrome), while polyneuropathy and polyradiculopathy occur less frequently. Electrical burns cause both direct nerve damage and indirect injury due to post-traumatic edema. Electrical injury may result in nerve paralysis caused by peripheral nerve damage. Partial or complete loss of peripheral nerve function, typically limited to the area of local contact, is usually temporary, and full recovery can be expected if the nerve is not affected by local tissue injury. Peripheral nervous system involvement is common and affects both sensory and motor functions. Immediate symptoms often resolve and have a more favorable prognosis than delayed manifestations. Sensory dysfunction may be associated with damage to sensory nerve endings. Autonomic disorders after electrical injury occur in approximately 60 % of patients with both high- and low-voltage injuries. Clinical manifestations may include tachycardia, significant fluctuations in blood pressure, headache, anxiety, hyperhidrosis, gastrointestinal disturbances, sleep disturbances, and dyspnea. Chronic neuropathic pain (allodynia) occurs in some patients, and accurate epidemiological data are currently lacking. Some patients may report intermittent discomfort or abnormal sensations without obvious physical injury, whereas others develop severe pain associated with evident tissue damage.

Conclusions. Complications affecting the peripheral nervous system may occur immediately after the injury or after a prolonged period. They are difficult to diagnose and significantly reduce patients' quality of life. Timely nerve decompression procedures improve treatment outcomes.

Keywords: electrical trauma, peripheral nervous system, autonomic nervous system, allodynia.

Electrical injuries (EI) can have neurological consequences for victims. Although the literature on the neurological complications of EI is limited to retrospective studies, case series, and studies of selected patient groups, preliminary findings provide some evidence for an association between EI and peripheral nervous system diseases and symptoms (e.g., sensory loss, neuropathy, and muscle weakness) [25]. Electrical injuries can cause long-term damage to both the central and peripheral nervous systems, which is less predictable [19, 30, 36].

Retrospective cohort studies have found that approximately 40 % of high-voltage electric shock cases had abnormal peripheral nerve conduction parameters such as decreased skin sensation, mononeuropathy, and polyneuropathy that persisted for 6 months or more after the injury [20, 30, 35].

Evidence collection

Selected literature sources were included in the study if they: (1) were published in Ukrainian or English; (2) reported on acute functional and mor-

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phological damage to the peripheral and autonomic nervous system in electrical trauma; (3) reported on the prevalence of neurological pathology in electrical trauma; (4) used an observational design (cohort or cross-sectional). A retrospective informative search was performed using a spatial-vector descriptive model, which was supplemented by a manual search of relevant articles. 37 modern literature sources were selected, of which 91.9 % — in the last 10 years, 78.4 % — in the last 5 years.

Evidence synthesis

Peripheral nervous system disorders account for about 90 % of the neurological consequences of EI, which, according to reports from combustiological departments, significantly exceed similar ones after thermal injuries. Most of these are high-voltage electrical burns [25].

Peripheral nerves are primarily conductive due to their low resistance compared to other tissues, making them susceptible to injury from electrical burns [1, 6]. Indeed, nerves are the most sensitive organ to electrical burns due to their low resistance to the passage of electrical current. They therefore serve as much better natural conductors of electricity than skin, muscle, and bone [1, 2, 13, 14, 16, 24]. Therefore, current can pass through the body in the absence of an accompanying skin burn. Electrical burns cause direct damage to the nerve as well as indirect damage through the formation of post-traumatic edema [1].

The most common complication of electrical neurotrauma is mononeuropathy (including carpal tunnel syndrome), followed by polyneuropathy and polyradiculopathy [23].

Neuropathy has been documented since the 1970s and is considered the most common and disabling complication of electrical burns. However, these neuropathies are often underdiagnosed or ignored. The prevalence of neuropathies after EI varies considerably, ranging from 2 to 84 %, with an average of 13.5 % [1, 8, 33].

Electricity causes myelin damage through direct effects and through damage to the vascular endothelium, leading to impaired blood supply to the nerves [13]. Compartments often compress nerves and contribute to the progression of peripheral neuropathy [18]. The mechanism of peripheral neuropathy involves axonal damage, resulting in both motor and sensory deficits. High-voltage electrical trauma can affect peripheral nerves, primarily causing axonal rather than demyelinating damage [7, 20]. Vascular and nerve damage caused by electrical trauma triggers the progressive development of necrosis [27].

Mononeuropathy usually affects individual nerves of the upper extremities, but is not limited to them, as these are common points of current entry. Median and ulnar neuropathies can be secondary to long-bone fractures [24]. The nature of the injury is important, as many patients complain of paresthesia or persistent numbness at the points of entry and exit of the electrical current, which is quite common. Neurological symptoms are thought to arise from structural lesions such as hemorrhage, edema, or chromatolysis of pyramidal cells. However, organic damage cannot explain the delayed onset of symptoms, which sometimes appear days or years after EI [12, 32, 34]. Typical symptoms include numbness and tingling sensations (paresthesia). Paresthesia may be transient and may develop suddenly or gradually after contact with an electrical source [2, 14, 16]. This rare complication of EI can be easily overlooked or misdiagnosed as neuropraxia, which can delay appropriate treatment [3]. Electrical injury results in nerve palsies caused by a variety of peripheral nerve lesions [16]. The ulnar, median, and radial nerves have the highest incidence of persistent dysfunction. Median and/or ulnar nerve palsies following EI are rare but devastating [8].

Partial or complete loss of peripheral nerve function, mostly confined to the area of local contact, is usually temporary, and full recovery can be expected unless the nerve is involved in local tissue damage [25].

Carpal tunnel syndrome is a fairly common mononeuropathy that occurs with electrical burns [1]. The wrist has an awkward anatomy where low-resistance tissues are affected by high-resistance tissues such as bone and tendon. The generation of high heat from the inside out causes the wrist area to conduct electrical current more than other parts of the body [8]. Carpal tunnel syndrome is a pathology of burn victims with serious functional consequences, as it is often ignored and treated late. Early clinical and electromyographic diagnosis is essential to ensure surgical treatment before irreversible intraneural damage occurs. Ultrasound may be useful for early detection of median nerve injury at the wrist when electromyography is not possible, and to rule out other underlying etiologies for carpal tunnel syndrome following electrical burns. If the diagnosis is confirmed, prompt surgical release of the nerve may be considered to avoid the development of irreversible intraneuronal ischemic lesions [1]. Immediate decompression with fasciotomy and carpal tunnel release appears to be the most promising treatment option. Loss of neurological function can be avoided if the median nerve is released [8]. Acute and chronic pain from electrical burns may mask symptoms of tunnel neuropathy. Median nerve injury at the wrist is the most common burn neuropathy, followed by ulnar nerve injury [1]. Electrical injuries can affect the function of the human hand in terms of reducing self-esteem of hand function, manual dexterity and tactile gnosis [30].

Polyneuropathy is associated with direct damage to peripheral nerves at the points of entry or exit of electric current, or with a polyneuritic syndrome involving nerves located distant from the points of contact. Various combinations of lesions of the median, ulnar, radial, sciatic and femoral nerves and

their more distant branches are observed [4, 25]. The localization of the damage may be completely outside the limits of that diagnosed in neure injuries or may be limited to neuromuscular synapses, which explains the occurrence of muscle weakness and, above all, rapid fatigue.

Polyradiculopathy is characterized by the involvement of multiple spinal nerve roots where there are multiple points of electrical current entry. This pathological condition manifests as numbness, tingling, pain, or muscle weakness in the areas innervated by the affected nerves. Causes may include mechanical compression (compartment syndrome), edema, or degenerative changes in the nerves [4, 25].

Peripheral nervous system involvement is quite common, both sensory and motor. Immediate symptoms are often reversible and have a better prognosis than late effects [12]. In a Canadian multicenter study of patients who had previously experienced electric shock, 9 % of respondents reported muscle weakness, 6 % reported tingling in the extremities, and 9 % reported numbness in the extremities within one year after EI [23, 25].

Sensory dysfunction may be associated with damage to sensory nerve endings [4]. The deficit may be patchy, with sensory deficits not corresponding to motor changes and being distributed unevenly [11, 17, 21, 26]. Sensory impairments are often accompanied by pain, numbness, and/or paresthesia along the path of the current [4]. They may develop in the affected limb for a long time after the initial injury. These symptoms are the result of incomplete nerve regeneration or permanent nerve damage, requiring long-term pain management. In addition, muscle weakness and atrophy may persist due to irreversible damage to peripheral nerves or muscle tissue. In deep burns involving nerve trunks, the prognosis for recovery is poor, and complete loss of nerve function is quite common. Electrical injuries can result in long-term motor deficits and muscle weakness, requiring significant rehabilitation efforts. Rehabilitation and physiotherapy are crucial for the treatment of these complications, although some patients never fully recover [31].

Causalgia is a complex regional pain syndrome type I (Sudek's syndrome), characterized by intense burning pain in the area of the affected nerve, which may be accompanied by autonomic and trophic (painful ischemia) disorders, edema, changes in skin temperature and color, limited mobility and stiffness in the affected limb (usually the upper limb), which is caused by damage to peripheral nerves due to electrical current or repeated surgical interventions. It is diagnosed on the basis of clinical criteria and determination of nerve damage using electroneuromyography.

Histopathological findings include focal petechial hemorrhages, perivascular space dilation, peripheral neuronal fragmentation, vascular damage, and ballooning myelin sheath dilation [23]. Degenerative changes are seen in peripheral nerves at both the input and output zones and in distant areas [22].

Whenever a neurodeficit is identified, appropriate investigation is warranted to exclude contributing factors (compartment syndrome).

Fasciotomy and nerve decompression are associated with improved peripheral nerve function in the long term. Patients who underwent fasciotomy had significantly lower rates of peripheral neuropathy than those who did not (odds 45 *vs* 92 %, $p < 0.0001$) [6].

Autonomic dysfunction after electrical trauma occurs in approximately 60 % of victims of high- and low-voltage electrical trauma [30].

EI victims are prone to autonomic neuropathy and sympathetic skin response disorders. Delayed autonomic dysfunction can occur weeks or months after the initial injury [31]. Symptoms can be diverse, including tachycardia, significant blood pressure fluctuations, headache, anxiety, hyperhidrosis, gastrointestinal problems, dyssomnia (sleep disturbances), and shortness of breath.

Arrhythmias are a direct consequence of electrical trauma. The combination of persistent sinus tachycardia and episodes of increased sweating or chills, acute persistent hypertension, and psychomotor agitation suggests a central hyperadrenergic state with the addition of autonomic surge. Abnormal results of autonomic function tests, significantly elevated plasma and urine noradrenaline levels (in the absence of pheochromocytoma), and a striking response to clonidine (0.4 mg/day) with plasma noradrenaline returning to normal within one week of treatment support this assumption [4, 22, 28].

Claude Bernard—Horner syndrome (oculosympathetic syndrome) is a pathological condition caused by dysfunction of the sympathetic nervous system after EI [4]. It is accompanied by blepharoptosis, sometimes with slight elevation of the lower eyelid («inverted ptosis»), miosis, enophthalmos, dyshidrosis, decreased ciliospinal reflex (reaction to skin irritation in the neck area, manifested by pupil dilation), injection of conjunctival vessels, and hyperemia of the facial skin on the affected side.

Keraunoparalysis (Charcot's palsy) is a temporary syndrome that lasts several hours after a high-voltage, electric arc, or lightning strike. It occurs in the limb along the path of the current and is associated with vascular spasm in this area due to a burst of catecholamine release. The limb becomes cold, pale, numb, and the pulse is not determined, which is sometimes confused with compartment syndrome. High-voltage EI produces various forms of paralysis: flaccid or spastic; temporary or prolonged; immediate or delayed; paraplegia or quadriplegia; unilateral or bilateral [4, 24]. As a rule, acute paralysis is mostly reversible, but delayed paralysis can be debilitating and lead to only partial recovery [4, 18].

Bladder dysfunction is a neurourological consequence of EI to the spinal cord or brain, or is under-

stood as an independent consequence of electric shock [36].

Sexual dysfunction is manifested by reduced libido and impotence [9, 34].

Delayed lesions of the autonomic nervous system, as well as complex regional pain syndromes, may also include changes in skin temperature and color, burning and skin sensitivity, edema, motor and autonomic disorders [4, 5].

Movement disorders have been reported, and the differentiation between them as central and peripheral syndromes is emphasized. In particular, parkinsonism, choreoathetosis (combining chorea with rapid chaotic movements and athetosis with slow «twisting» movements), dystonia, myoclonus, and cerebral tremor are defined. Electrical origin of myoclonus has been reported, as well as demyelination [4].

Chronic neuropathic pain (allodynia) occurs in a proportion of electrocuted individuals, although accurate statistics are not available. Some patients may complain of intermittent discomfort without any apparent physical damage, whereas other complications may present with severe pain with obvious tissue damage [37].

Pain is a common and complex complaint following EI. It is often multifactorial and appears disproportionate to any measurable neuropathy [34]. Post-traumatic pain results from both direct EI and secondary injuries from fractures, dislocations, and falls. Thus, pain management is of paramount importance in EI and its sequelae [15].

With significant destruction, all extero- and interoreceptors close to the lesion area instantly die (burn out) and as a result the reflex component drops out. Therefore, the pain is more intense in the low voltage group than in the high voltage group [10, 30].

People with a history of nonspecific EI are at increased risk of being diagnosed with unspecified pain or undiagnosed soft tissue disorders. The risk of nonspecific pain is related to the severity of the injury in both the short and long term. Patients who were hospitalized for more than 1 day had a significantly higher risk of developing nonspecific pain compared with controls. The risk of developing pain as a long-term sequelae is related to the severity of neurogenic pain. Pain as a long-term sequelae of EI was reported by 8 % of the study population during the 1-year follow-up. Approximately 10 % of professional electricians with a history of unspecified injury reported persistent pain and muscle disorders 1 to 45 years after the injury [29].

Pain as a long-term or delayed physiological consequence of EI can sometimes be overlooked, especially after neuronal damage with less pronounced immediate physiological symptoms. This group of patients presents with various forms of pain that are very difficult for the physician to associate with a specific clinical diagnosis. Patients who have sustained persistent low-voltage nerve damage are often referred for specialized consultations and examinations that are usually ineffective in diagnosing long-term conditions. This may be because the effects of EI are similar to many other conditions, so specialists perform examinations that are unable to detect organic changes. Thus, these patients are often examined by specialists from different medical specialties and as a result, they are given unspecified diagnoses due to the lack of objective data and positive clinical tests. More than 95 % of diagnoses, such as «chronic pain of unknown etiology» or «pain in the arm (leg)», could not be confirmed by repeated examination. This is due to the fact that many clinical symptoms are not sufficiently recognized by the medical community as consequences of electric shock [29].

The literature notes that many patients do not experience satisfactory pain relief after electric shock, regardless of treatment, but a combination of somatic and psychosocial methods leads to quite favorable results [34].

Conclusions

1. Peripheral nervous system complications of electric shock can debut immediately after the incident or after a long time.
2. Peripheral nervous system damage in EI is quite difficult to diagnose.
3. Peripheral nervous system complications of EI significantly impair the quality of life of patients.
4. Timely performed nerve decompression techniques improve treatment outcomes.

There is no conflict of interest.

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References

1. Albert T, Perrot P, Lecoq FA, Lancien U. Post-burn carpal tunnel syndrome: A systematic review. Hand Surg Rehabil. 2025 Jun;44(3):102134. doi: 10.1016/j.hansur.2025.102134.
2. Almusawi YK, Al-Sammarraie MRH, Shikara MD. The Effect of An Electric Current on Human Body (A Review). Al-Esraa University College Journal for Medical Sciences. 2023;4(5):2. doi: 10.70080/2790-7937.1016.
3. Anand S, Findlay AR, Anand G. Carpal tunnel syndrome following an electrical injury in a child. Case Reports 2015;2015:bcr2014208092. doi: 10.1136/bcr-2014-208092.
4. Andrews CJ, Reisner AD. Neurological and neuropsychological consequences of electrical and lightning shock: review and theories of causation. Neural Regen Res. 2017 May;12(5):677-686. doi: 10.4103/1673-5374.206636.
5. Beukes F, Smith Z, Wilscott-Davids C, Mentoor I. Electrifying evidence: Ten years of fatal encounters with electricity at Tygerberg mortuary. J Forensic Leg Med. 2025 May;112:102875. doi: 10.1016/j.jflm.2025.102875.
6. Blears EE, Ballou J, Weitzner A, Caffrey J, Dellon AL. The Roles of Fasciotomy and Peripheral Nerve Decompression in Electric Burn Patients: A Systematic Review and Meta-Analysis. Microsurgery. 2025 Feb;45(2):e70036. doi: 10.1002/micr.70036.
7. Blyth E, Vujcich E, Dunn D. High-Voltage Electrical Burn Requiring Urgent Scalp Reconstruction after Developing a Brain Abscess. Eur Burn J. 2024 Sep 3;5(3):288-295. doi: 10.3390/ebj5030027.
8. Cartı EB, Uçar AD, Yıldırım M. Is carpal tunnel release safe in electrical burn decompressive therapy: Six-years experience. Turk J Surg. 2021 Jun 30;37(2):87-95. doi: 10.47717/turkjsurg.2021.4379.
9. Dhaniwala NS, Date S, Dhaniwala MN. Effects of electrical injury on musculoskeletal system: a case report. MOJ Orthop Rheumatol. 2019;11(4):160162. doi: 10.15406/mojor.2019.11.00493.
10. Dhital BM, Regmi S, Regmi SR, et al. Cardiac Arrhythmias in Patients with Electrical Injury: Hospital Based Study. Nepalese Heart Journal. 2020;17 (2):29-32. doi: https://doi.org/10.3126/njh.v17i2.32675.
11. Electrocution and Electrical Injury — Emergency Management in Children. Guidelines. CHQ-GDL-00735. 26/03/2025. 11p. Available from: chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://www.childrens.health.qld.gov.au/__data/assets/pdf_file/0011/180200/gdl-00735.pdf.
12. Gentges J, Schieche C. Electrical injuries in the emergency department: an evidence-based review. Emerg Med Pract. 2018 Nov;20(11):1-20. Available from: https://pubmed.ncbi.nlm.nih.gov/30358379/.
13. Guimarães F, Camões J, Mesquita A, Gomes E, Araujo R. A Case Report: Low Voltage Electric Injuries Culminating in Cardiac Arrest and Direct Lung Injury. Cureus. 2020 Oct 30;12(10):e11261. doi: 10.7759/cureus.11261.
14. Jiang H, Zhang Y, Zhang J, et al. Acupuncture for the treatment of thalamencephalic and mesencephalic injury secondary to electrical trauma: A case report. Front Neurosci. 2023 Mar 6;17:1139537. doi: 10.3389/fnins.2023.1139537.
15. Joshi MV, Bhakne P, Kulkarni CA, Palekar TJ, Phansopkar P. The Power of Movement: A Comprehensive Case Study of Physiotherapeutic Approaches in Electrical Injury Rehabilitation. Cureus. 2024 Jul 15;16(7):e64615. doi: 10.7759/cureus.64615.
16. Karamoy OO, Edwar PPM. Electrical burn injury in a 12-year-old male: a case report. Bali Medical Journal. 2024;13(3):1224-1228. doi: 10.15562/bmj.v13i3.5330.
17. Khartade HK, Shrivastava S, Vashisht J, et al. Delayed Death Due to Lightning: An Autopsy Case Report Highlighting Diagnostic and Medicolegal Issues. J Burn Care Res. 2023 Jul 5;44(4):996-999. doi: 10.1093/jbcr/irad070.
18. Khor D, AlQasas T, Galet C, et al. Electrical injuries and outcomes: A retrospective review. Burns. 2023 Nov;49(7):1739-1744. doi: 10.1016/j.burns.2023.03.015.
19. Kravets OV, Yekhalov VV, Gorbuntsov VV. Central neurological injuries in electrical trauma (review). International Neurological Journal (Ukraine). 2025;21(8):594-600. doi: 10.22141/2224-0713.21.8.2025.1227.
20. Kwon KH, Kim SH, Minn YK. Electrodiagnostic Study of Peripheral Nerves in High-Voltage Electrical Injury, Journal of Burn Care & Research. July-August 2014;35(4):e230-e233. doi: 10.1097/BCR.0b013e31829b39b9.
21. Mansueto G, Di Napoli M, Mascolo P, et al. Electrocution Stigmas in Organ Damage: The Pathological Marks. Diagnostics (Basel). 2021 Apr 10;11(4):682. doi: 10.3390/diagnostics11040682.
22. Mishalov VD, Khokholeva TV, Bachynskyi VT, et al. authors; Mishalov VD, editor. Forensic medicine: Textbook. Kyiv; 2018, 575 p. Ukrainian.
23. Morosanu V, Balasa R, Morosanu S, Baróti B, Roman-Filip I. Delayed Demyelinating Disease of the Central Nervous System Following Low-Voltage Alternating Current Electrical Injury: A Case Report and Review of the Literature. Cureus. 2023 Aug 22;15(8):e43951. doi: 10.7759/cureus.43951.
24. Narang S, Manoharan GK, Dil JS, Raja A. Electrical Injuries and Neurosurgery: A Case Report and Review of Literature. Indian J Neurotrauma 2023;20:65-70. doi: 10.1055/s-0041-1739481.
25. Nielsen KJ, Carstensen O, Kærgaard A, Vestergaard JM, Biering K Neurological symptoms and disorders following electrical injury: A register-based matched cohort study. PLoS ONE.2022;17(3):e0264857. doi: 10.1371/journal.pone.0264857.
26. O'Keefe KP, Danzl DF, Moreira ME, Ganetsky M. Electrical injuries and lightning strikes: Evaluation and management/ UpToDate. 22 Jul 2025. https://www.uptodate.com/contents/electrical-injuries-and-lightning-strikes-evaluation-and-management.
27. Ozdemir M, Akkoc MF, Selçuk CT, Erbatur S. Effectiveness of reverse first dorsal metatarsal artery flap for reconstruction of distal foot in electrical injuries. Burns. 2025 Mar;51(2):107335. doi: 10.1016/j.burns.2024.107335.
28. Parsaik AK, Ahlskog JE, Singer W, et al. Central hyperadrenergic state after lightning strike. Clin Auton Res. 2013 Aug;23(4):169-73. doi: 10.1007/s10286-013-0197-7.
29. Poulsen PH, Carstensen O, Kærgaard A, et al. Unspecified pain and other soft tissue disorders following electrical injuries: a register-based matched cohort study. Int Arch Occup Environ Health. 2022 May;95(4):799-809. doi: 10.1007/s00420-021-01802-y.
30. Rådman L, Wold A, Norman K, Olausson H, Thordstein M. Hand Function After an Electrical Accident-A Case-Control Study. J Occup Environ Med. 2023 Mar 1;65(3):242-248. doi: 10.1097/JOM.0000000000002720. Epub 2022 Oct 4.
31. Ragasivamalini B, Balambighai V, Sureshkumar SA. Super-refractory Status Epilepticus and Cerebellar Involvement Following High-Voltage Electrical Injury. Cureus. 2025 Jan 15;17(1):e77472. doi: 10.7759/cureus.77472.
32. Roshanzamir S, Keshavarzi E. Sympathetic skin response impairment: A good predictor of bone loss in electrical burn victims. Burns. 2020 Mar;46(2):394-399. doi: 10.1016/j.burns.2019.07.034.
33. Thomas J, Sreekumar NC, Shankar C, James A. Complications and Outcome of Electrical Burns in Manipal, India: 6-Year Institutional Report. World J Plast Surg. 2020 Jan;9(1):14-21. PMID: 32190586; PMCID: PMC7068176.
34. Wesner ML, Hickie J. Long-term sequelae of electrical injury. Can Fam Physician. 2013 Sep;59(9):935-9. Availble from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3771718/.
35. Wold A, Rådman L, Norman K, Olausson H, Thordstein M. Clinical Evaluation of Nerve Function in Electrical Accident Survivors with Persisting Neurosensory Symptoms. Brain Sci. 2022 Sep 27;12(10):1301. doi: 10.3390/brainsci12101301.
36. Yiannopoulou KG, Papagiannis GI, Triantafyllou AI, et al. Neurological and neurourological complications of electrical injuries. Neurol Neurochir Pol. 2021;55(1):12-23. PMID: 33026644.
37. Zemaitis MR, Lopez RA, Huecker MR. Electrical Injuries. [Updated 2025 Jan 20]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK448087/.

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Ушкодження периферичної нервової системи при електротравмі (огляд літератури)

Ураження електричним струмом можуть мати неврологічні наслідки для постраждалих. Когортні ретроспективні дослідження виявили, що в близько 40 % випадків ураження електричним струмом високої напруги показники периферичної нервової провідності були аномальними.

Збір доказів. Проведено ретроспективний пошук інформації з використанням просторово-векторної описової моделі, доповненої ручним пошуком відповідних статей. Відібрано 37 сучасних літературних джерел, з них 91,9 % — за останніх 10 років, 78,4 % — за останніх 5 років.

Синтез доказів. На порушення з боку периферичної нервової системи припадає близько 90 % неврологічних наслідків електротравми, що, за звітами комбустіологічних відділень, значно перевищують такі після термічних травм. Найпоширенішим ускладненням електричної нейротравми є мононейропатія (зокрема синдром зап'ясткового (карпального) каналу), рідше трапляються полінейропатія та поліорадикулопатія. Електричні опіки спричиняють прямі пошкодження нерва, а також непрямі пошкодження через утворення посттравматичного набряку. Наслідком електричної травми є параліч нервів, зумовлений ураженнями периферичних нервів. Часткова або повна втрата функцій периферичного нерва, яка переважно обмежується ділянкою локального контакту, зазвичай є тимчасовою, і можна очікувати повного відновлення, якщо нерв не залучений до пошкодження місцевих тканин. Ураження периферичної нервової системи поширене явище з боку як сенсорної, так і моторної системи. Негайні симптоми часто минають і мають кращий прогноз, ніж віддалені наслідки. Сенсорна дисфункція може бути пов'язана з пошкодженням чутливих нервових закінчень. Вегетативні розлади після електротравми трапляються в близько 60 % постраждалих від високовольтної та низьковольтної електротравми. Симптоми можуть бути різноманітними (тахікардія, значні коливання артеріального тиску, головний біль, тривожність, гіпергідроз, проблеми зі шлунково-кишковим трактом, дисомнія (порушення сну) та відчуття нестачі повітря). Хронічний нейропатичний біль (алодинія) виникає в частини уражених електричним струмом. Точних статистичних даних щодо цієї патології не існує. Деякі пацієнти можуть скаржитися на періодичні неприємні відчуття без будь-яких явних фізичних пошкоджень, інші ускладнення можуть виявлятися сильним болем із явним пошкодженням тканин.

Висновки. Ускладнення електротравми з боку периферичної нервової системи можуть дебютувати одразу після інциденту або через тривалий час, їх складно діагностувати, вони значно знижують якість життя пацієнтів. Своєчасно виконані прийоми декомпресії нервів поліпшують результати лікування.

Ключові слова: електротравма, периферична нервова система, вегетативна нервова система, алодинія.

ДЛЯ ЦИТУВАННЯ

- Kravets OV, Yekhalov VV, Gorbuntsov VV. Peripheral nervous system damage following electrical injury (review). Український неврологічний журнал. 2026;1:5-10. doi: 10.30978/UNJ2026-1-5. English.
- Kravets OV, Yekhalov VV, Gorbuntsov VV. Peripheral nervous system damage following electrical injury (review). Ukrainian Neurological Journal. 2026;1:5-10. http://doi.org/10.30978/UNJ2026-1-5.