Electrical Injuries: Clinical Presentation, Pathophysiology, and Multidisciplinary Management in Emergency Medicine, Paramedics, Pharmacy, Laboratory Sciences, Public Health, Radiologic Technology, and Environmental Health Practice

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Abstract

Background: Electrical injuries are a distinct form of trauma caused by the passage of electrical energy through or around the human body. They differ from thermal and mechanical injuries due to their ability to affect multiple organ systems simultaneously. The severity ranges from minor cutaneous burns to fatal cardiac arrest and irreversible neurological damage. These injuries remain a significant cause of preventable morbidity and mortality in both domestic and occupational settings.
Aim: This study aims to provide a comprehensive overview of electrical injuries, including classification, epidemiology, pathophysiology, clinical presentation, evaluation, management, complications, prognosis, and preventive strategies within a multidisciplinary healthcare context.
Methods: A structured narrative review approach was used to synthesize current evidence from clinical literature, emergency medicine guidelines, burn care protocols, and multidisciplinary healthcare practice sources. Data were analyzed across emergency medicine, paramedicine, pharmacy, laboratory diagnostics, radiology, public health, and rehabilitation sciences to construct an integrated clinical framework.
Results: Electrical injuries are classified into flash, flame, lightning, and true electrical injuries. Pathophysiological effects are governed by voltage, current type, tissue resistance, and exposure duration. Clinical manifestations include arrhythmias, respiratory arrest, burns, rhabdomyolysis, neurological deficits, and psychological sequelae. Evaluation requires ECG monitoring, laboratory testing including CK and renal function, and targeted imaging. Management involves airway protection, resuscitation, fluid therapy, burn care, and early surgical consultation. Complications include compartment syndrome, acute kidney injury, and long-term neurological and psychological disorders. Multidisciplinary care improves survival and functional outcomes.
Conclusion: Electrical injuries require rapid systemic assessment and coordinated multidisciplinary management due to their hidden internal severity and unpredictable clinical course.

Keywords: Electrical injury, burns, trauma, arrhythmia, rhabdomyolysis, multidisciplinary care, emergency medicine, burn management

Introduction

Electrical injury represents a distinct category of trauma that differs fundamentally from mechanical forms of injury, including both blunt force and penetrating mechanisms. These injuries arise when electrical energy, either from low-voltage or high-voltage sources, is transmitted through the human body. Such exposure is frequently associated with a wide spectrum of clinical consequences, ranging from localized tissue damage to severe systemic complications, contributing significantly to both morbidity and mortality. In most clinical scenarios, electrical injuries occur as accidental events and are largely preventable through appropriate safety measures and environmental control. When survival beyond the initial insult is achieved, the physiological impact may extend across multiple organ systems, leading to dysfunction involving neurological, cardiovascular, musculoskeletal, and integumentary structures [1][2][3][4]. From a pathophysiological and clinical classification perspective, electrical injuries are commonly divided into four principal categories based on the mechanism and interaction of electrical energy with the human body. The first category, flash injuries, results from an electrical arc phenomenon in which the current does not traverse the body. Instead, the injury is primarily external, manifesting as cutaneous burns due to intense thermal energy generated by the arc discharge. The second category, flame injuries, occurs when an electrical arc ignites surrounding materials, particularly clothing, leading to secondary thermal burns that may be more extensive than the primary electrical exposure itself. The third category, lightning injuries, is characterized by exposure to extremely high-voltage electrical energy over a very short duration. In such cases, current frequently travels over the surface of the body in a phenomenon known as flashover, while internal current passage may be variable and inconsistent depending on exposure conditions. The fourth and most direct form, true electrical injury, occurs when the individual becomes an integral part of an active electrical circuit. In these cases, electrical current enters and exits the body at identifiable contact points, producing a defined pathway of injury that can result in deep tissue destruction and multi-organ involvement.

Etiology

Electrical injuries are commonly categorized according to the voltage level involved, primarily into low-voltage injuries defined as exposure to less than 1000 volts, and high-voltage injuries defined as exposure to 1000 volts or more. This classification is clinically significant because voltage magnitude is strongly associated with the severity of tissue damage, systemic involvement, and risk of mortality. In North American settings, domestic electrical systems typically operate at 110 to 120 volts, whereas many regions globally utilize standard household currents ranging from 220 to 240 volts. Although these voltages are considered low in industrial terms, they are still capable of producing clinically important injuries in domestic environments, especially when exposure is prolonged or when safety mechanisms fail. Common household sources include electrical outlets, extension cords, and portable appliances, all of which may become hazardous under conditions of faulty insulation, wet environments, or direct contact with conductive materials. In pediatric populations, low-voltage injuries frequently occur through oral exploration behaviors such as biting or chewing electrical cords. These incidents may not always result in immediate loss of consciousness or cardiac arrest, which can lead to underestimation of injury severity. However, even low-voltage exposure in children can produce substantial physiological harm depending on several modifying factors. These include the duration of contact, which may lead to sustained muscle contraction or tetany, the total body size of the child, and the surface area and anatomical location involved in the electrical contact. In certain cases, the pathophysiological consequences of low-voltage exposure may resemble those seen in high-voltage injuries, particularly when current traverses vital structures or persists long enough to cause deep tissue injury [5][6][7][8]. High-voltage injuries are more commonly associated with occupational and environmental exposures. Electrical transmission infrastructure, such as overhead power lines, typically carries voltages ranging from 69 to 765 kilovolts, while distribution systems operate between 4 and 34.5 kilovolts. These elevated voltage levels are frequently implicated in severe workplace injuries, particularly among individuals working in construction, maintenance, and electrical utilities. Epidemiological data indicate that at least half of all fatal electrical injuries in occupational settings are linked to direct or indirect contact with power lines, while approximately one-quarter are attributed to machinery or powered industrial tools. Lightning strikes represent an extreme form of high-voltage exposure, with estimated voltages ranging from 100 to 150 million volts. Despite their brief duration, lightning events can produce profound multisystem injury due to the massive energy transfer involved and the unpredictable pathways of current dispersion through the human body.

Epidemiology

Electrical injuries represent a significant public health burden, with substantial mortality and morbidity reported annually. In the United States alone, these injuries are responsible for approximately 1000 deaths each year, reflecting the severity and potential lethality of electrical exposure. Among these fatalities, nearly 400 cases are attributed to high-voltage electrical injuries, which are commonly associated with industrial and occupational environments where exposure to powerful electrical sources is more likely. Lightning-related incidents also contribute significantly to mortality statistics, accounting for an estimated 50 to 300 deaths annually, depending on environmental conditions and geographic variation. In addition to fatal outcomes, nonfatal electrical shock events are considerably more frequent, with at least 30,000 reported cases each year. These nonfatal injuries still carry important clinical consequences, often requiring medical evaluation, burn care, and long-term follow-up due to potential multisystem involvement. From a healthcare utilization perspective, electrical injuries constitute approximately 5% of all admissions to specialized burn units in the United States, highlighting their relevance within acute trauma and burn management services. The epidemiological distribution of these injuries also demonstrates clear age-related patterns. Pediatric populations account for around 20% of all electrical injury cases, with the highest incidence observed among toddlers and adolescents. In younger children, injuries frequently occur in domestic environments, often related to accidental contact with electrical outlets, cords, or household appliances. This reflects the vulnerability of children within the home setting, where supervision and environmental safety measures may be insufficient. In contrast, electrical injuries among adults are predominantly associated with occupational exposure. These injuries are especially common in industries involving construction, electrical maintenance, and utility work, where direct contact with energized systems poses a persistent risk. In fact, electrical trauma is recognized as the fourth leading cause of work-related traumatic deaths, underscoring its occupational significance and the need for strict adherence to safety protocols and regulatory standards. Overall, the epidemiological profile of electrical injuries demonstrates a dual burden affecting both domestic and workplace environments, with distinct risk patterns across age groups and exposure settings [9].

Pathophysiology

The physiological basis of electrical injury is rooted in the movement of electrons through conductive media under the influence of a potential gradient, progressing from regions of higher electron concentration toward areas of lower concentration. This fundamental process governs the generation and transmission of electrical energy and is mathematically described by Ohm's law, which establishes that electrical current is directly proportional to voltage and inversely proportional to resistance. This relationship provides the foundational framework for understanding tissue injury in electrical exposure, as variations in voltage and resistance directly influence the magnitude of current passing through biological structures. The potential gradient, defined as the difference in electron concentration between two points, determines voltage levels and varies significantly depending on the source of electrical energy. In clinical contexts, electrical injuries are broadly categorized into low-voltage exposures, defined as less than 1000 volts, and high-voltage exposures, defined as 1000 volts or greater. Household electrical systems in the United States typically operate at 110 to 120 volts, although certain appliances may utilize currents up to 240 volts, whereas industrial systems and high-tension transmission lines may exceed 100,000 volts, representing a substantially greater hazard due to increased energy transfer capacity [10]. Electrical current, denoted as I, represents the flow of electric charge and is measured in amperes. It reflects the quantity of electrons traversing a conductive pathway and, in the context of human injury, corresponds to the amount of electrical energy delivered through bodily tissues during exposure. The physiological tolerance to electrical current varies between individuals and is significantly influenced by neuromuscular excitability, as higher currents induce involuntary muscular contraction, potentially culminating in sustained tetanic contraction that prevents voluntary disengagement from the source. This phenomenon is clinically significant, as prolonged contact duration directly increases the extent of tissue damage.

Resistance, represented as R and measured in ohms, describes the opposition offered by biological tissues to the flow of electrical current. Within the human body, resistance is not uniform and varies according to the composition and physicochemical properties of different tissues. Highly vascularized and water-rich tissues, including nerves, skeletal muscles, and blood vessels, exhibit low resistance and therefore serve as efficient conductors of electricity. Conversely, tissues such as bone, adipose tissue, and keratinized skin demonstrate significantly higher resistance, thereby limiting current flow. Skin resistance is further influenced by structural and environmental factors, including thickness, hydration status, and degree of keratinization, with dry and thickened skin exhibiting greater resistance compared to moist mucosal surfaces or disrupted skin barriers such as abrasions, lacerations, or puncture wounds. Reduced resistance in these compromised areas facilitates deeper penetration of electrical current, increasing the likelihood of internal organ involvement. High-resistance skin typically results in substantial energy dissipation at the point of contact, producing prominent cutaneous burns while simultaneously limiting the amount of current transmitted to deeper structures. In contrast, low-resistance skin may exhibit minimal external injury despite significant internal tissue damage, as a larger proportion of electrical energy is conducted into deeper anatomical compartments. This dissociation between external burn severity and internal injury underscores the clinical principle that visible cutaneous findings are not reliable indicators of the extent of systemic involvement. Current density, defined as the amount of current per unit cross-sectional area of tissue, further influences injury patterns. As electrical current traverses the upper limb, it initially distributes relatively evenly through low-resistance tissues such as muscle and neurovascular structures. However, at anatomical constrictions such as joints, where cross-sectional areas are reduced and high-resistance structures such as tendons and bone predominate, current becomes concentrated within remaining conductive tissues, thereby increasing localized energy deposition and susceptibility to injury.

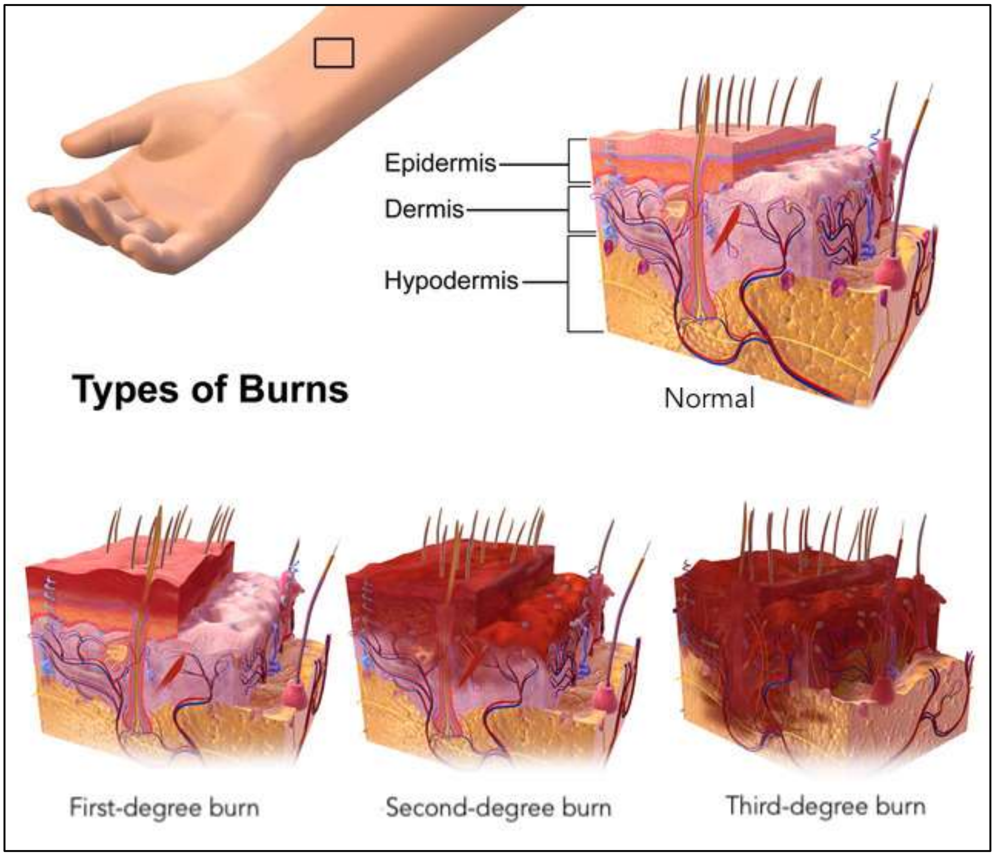

Fig. 1: Degree of Burns.

The trajectory of electrical current through the body is critically determined by contact points, traditionally described as entry and exit sites. In clinical practice, the hand most commonly serves as the point of entry, whereas the feet frequently function as the exit pathway. However, precise identification of entry and exit sites is often unreliable due to complex current dispersion patterns and the possibility of multiple contact points or "skip lesions." For this reason, the term contact points is preferred in clinical documentation. The anatomical pathway of current plays a decisive role in determining organ involvement. Current traversing the cranial region may result in central nervous system injury, while thoracic pathways, particularly those extending from upper to lower limbs, may involve the myocardium and predispose to life-threatening arrhythmias. The severity of injury is influenced by multiple interrelated variables collectively described by the Kouwenhoven factors, which include current type, magnitude, exposure duration, tissue resistance, current pathway, and electrical field strength [10][11]. The type of current, classified as alternating current or direct current, exerts a significant influence on physiological outcomes. Alternating current, commonly used in domestic electrical systems at frequencies of approximately 50 to 60 Hz, periodically reverses direction and is particularly associated with sustained muscle contraction and tetany. Direct current, in contrast, flows unidirectionally and is typically encountered in battery systems, defibrillators, and cardioverting devices. While both current types are capable of producing injury, alternating current is generally considered more hazardous at equivalent voltage levels due to its propensity to induce prolonged muscular contraction, thereby increasing exposure duration. This neuromuscular effect is most pronounced at frequencies between 40 and 110 Hz, which coincide with standard household electrical systems. In such circumstances, contraction of flexor musculature in the hand may result in involuntary gripping of the electrical source, further prolonging contact time and exacerbating tissue injury [11].

The perception of electrical current begins at very low thresholds, typically around one milliampere, where individuals may experience a tingling sensation. The let-go current represents the maximum level of current at which an individual retains voluntary control to release the electrical source despite muscular contraction. This threshold varies depending on physiological and anthropometric factors, including muscle mass and body weight. In an average adult male weighing approximately 70 kilograms, the let-go current is estimated to be approximately 15 milliamperes for alternating current and 75 milliamperes for direct current, whereas in pediatric populations, this threshold is significantly lower, typically between 3 and 5 milliamperes. This reduced tolerance contributes to the increased vulnerability of children in electrical exposure scenarios. Protective electrical devices such as circuit breakers and fuses are engineered to prevent electrical fires and equipment damage rather than direct human protection. These devices typically activate at current levels between 15 and 20 amperes, which are substantially higher than human physiological thresholds. In contrast, ground fault circuit interrupters are specifically designed for human safety, detecting current imbalances as low as 4 to 6 milliamperes and rapidly interrupting the circuit to prevent injury, making them particularly important in high-risk environments such as bathrooms and kitchens. The physiological effects of electrical injury are further modulated by current frequency. Low-frequency alternating current is particularly associated with sustained tetanic contraction, increasing the likelihood of prolonged exposure and subsequent tissue damage, whereas higher-frequency currents are less likely to produce such effects. In general, alternating current is considered three to five times more injurious than direct current when matched for voltage and amperage. Direct current typically produces a single, forceful muscular contraction that may propel the individual away from the source, thereby limiting exposure duration.

Current density remains a critical determinant of tissue injury severity, as it is influenced by the surface area through which electrical energy is transmitted. A smaller contact area results in higher current density and consequently greater localized tissue destruction, whereas a larger contact area distributes energy more widely and reduces focal injury intensity. This principle explains why low-voltage exposures confined to small anatomical regions may produce injuries comparable in severity to high-voltage exposures distributed over broader areas. Clinical analogies such as monopolar electrocautery illustrate this concept, where high current density at the instrument tip results in targeted tissue coagulation while a larger dispersive pad minimizes injury at the return site. Electrical field strength ultimately governs the extent of biological damage at the cellular level. Low field strength exposures typically produce transient sensory disturbances without structural injury, whereas high field strength exposures induce profound electrochemical and thermal tissue damage, including protein denaturation, coagulative necrosis, hemolysis, vascular thrombosis, and disruption of musculoskeletal integrity. These effects may extend beyond localized injury to produce systemic complications such as massive edema, vascular congestion, and compartment syndrome. Secondary physiological consequences include hypovolemia, hypotension, and widespread tissue ischemia. Severe muscle destruction may precipitate rhabdomyolysis, leading to myoglobin release, pigment-induced nephropathy, and significant electrolyte disturbances. Collectively, these pathophysiological cascades substantially increase the risk of acute kidney injury and multi-organ dysfunction in affected individuals [12].

History and Physical

Electrical injuries present a broad clinical spectrum that varies according to the mechanism of exposure, voltage level, and pathway of current through the body. These injuries are generally classified into flash, flame, lightning, and true electrical injuries, each demonstrating distinct clinical characteristics and symptom profiles. Despite this classification, initial clinical evaluation must prioritize stabilization through a structured, algorithm-based approach, with immediate identification and management of life-threatening conditions such as airway compromise, respiratory failure, cardiac arrest, or major trauma preceding focused injury assessment. True electrical injury represents the most clinically significant form, in which the patient becomes part of an active electrical circuit. Clinical presentation in such cases is highly variable, ranging from minimal symptoms to catastrophic multisystem failure. Patients may present with cardiac arrhythmias, including life-threatening dysrhythmias, respiratory arrest, altered consciousness or coma, associated blunt trauma from falls or blast effects, and varying degrees of thermal or electrical burns. Some individuals may report transient sensory disturbances or unpleasant paresthesia without visible external injury, while others demonstrate extensive tissue destruction with profound pain and functional impairment. A comprehensive history is essential and should include detailed characterization of the electrical source, including voltage classification, alternating or direct current exposure, duration of contact, environmental context, and any associated traumatic events. This information is critical in determining injury severity and guiding subsequent diagnostic and therapeutic decisions [12].

Low-voltage alternating current injuries may present with subtle or superficial cutaneous burns; however, clinical severity should not be underestimated, as prolonged exposure or sustained muscle tetany can result in severe internal injury. Even low-voltage exposures are capable of inducing cardiac arrest, respiratory arrest, ventricular fibrillation, or seizure activity, particularly when current traverses vital anatomical regions. Consequently, electrical exposure must be considered in the differential diagnosis of unexplained cardiac or respiratory arrest, especially when the event is unwitnessed or occurs in high-risk environments. Collateral information obtained from witnesses or emergency responders is often essential in reconstructing the exposure event and estimating electrical parameters. High-voltage alternating current injuries are typically associated with more severe clinical manifestations, including deep thermal burns, prolonged loss of consciousness, and higher incidence of cardiac arrest at the scene. In such cases, the exact voltage is often unknown, and clinicians must rely on contextual clues such as occupational setting, industrial exposure, or proximity to power distribution systems to estimate the likely energy level involved. Regardless of voltage classification, all patients require thorough and systematic physical examination to assess the full extent of injury, as external findings frequently underestimate internal damage. High-voltage injuries are associated with significantly increased morbidity, including compartment syndrome, rhabdomyolysis, acute renal failure, and limb loss, often necessitating surgical intervention. Mortality rates in high-voltage exposure range from 5% to 30%, whereas low-voltage injuries demonstrate substantially lower mortality rates, typically below 1% to 3% [6][13]. Cardiac vulnerability is particularly important, as ventricular fibrillation may occur with current exposure as low as 50 to 120 milliamperes, which is within the range of household electrical systems. Electrical injury may also cause direct myocardial damage, leading to early or delayed arrhythmias such as sinus tachycardia or premature ventricular contractions, although long-term cardiac sequelae are relatively uncommon. When current passes through the thoracic region, respiratory compromise may occur due to paralysis of respiratory musculature rather than direct pulmonary injury, as lung tissue has low electrical conductivity and is rarely directly affected.

Cutaneous findings in true electrical injury are often misleading, as severe internal injury may exist despite minimal external burns. Burn severity is typically greatest at contact points, with intensity determined by both current density and duration of exposure. Smaller contact areas concentrate current density, resulting in more severe localized tissue destruction, whereas larger contact areas distribute energy more diffusely. The clinical decision regarding surgical intervention, including fasciotomy or amputation, is guided not solely by burn appearance but by integrated assessment of physiological, neurological, and vascular status. Extremity involvement warrants a high index of suspicion for compartment syndrome, which results from extensive muscle necrosis and subsequent edema secondary to electroporation and cellular disruption. Progressive swelling within closed fascial compartments elevates intracompartmental pressure, leading to ischemia and potential irreversible tissue damage. This condition must be distinguished from eschar syndrome, which arises from circumferential full-thickness burns and results in external constriction rather than internal compartmental pressure. Clinical evaluation of both conditions relies on recognition of the classical "six Ps," including disproportionate pain, paresthesia, paralysis, pallor, poikilothermia, and pulselessness, although these signs may not all be present in early stages. Electrical injuries may also be associated with musculoskeletal complications such as posterior shoulder dislocation, a rare but recognized consequence of high-voltage exposure or seizure-like tetanic contraction. Unlike anterior dislocations, posterior dislocations occur when forceful internal rotator muscle contraction overwhelms the external rotator muscles, displacing the humeral head posteriorly from the glenoid cavity. Patients typically present with the affected limb adducted and internally rotated, and diagnosis may be delayed due to subtle clinical presentation. High-risk individuals include those exposed to high-voltage injury or those experiencing prolonged muscle tetany following low-voltage alternating current exposure [15].

Flash Electrical Injury

Flash electrical injury results from exposure to the intense thermal energy produced by an electrical arc discharge rather than direct passage of electrical current through the body. This form of injury is characterized by the generation of extremely high temperatures, reaching approximately 19,000 °C, sufficient to cause rapid thermal destruction of surrounding tissues. The underlying mechanism involves the movement of electrical current between two electrodes through a medium that is normally nonconductive. During this process, surrounding gases become ionized, leading to the formation of plasma, which represents the fourth state of matter. The ionization process produces a localized region of extreme energy concentration, accompanied by visible light and explosive thermal release. This phenomenon may occur in both natural and artificial environments, with lightning representing the natural form of large-scale electrical arc discharge, while industrial applications include welding, plasma cutting, and other high-energy electrical processes. In clinical contexts, flash burns differ fundamentally from true electrical injuries because the electrical current typically does not traverse the human body. Instead, injury results primarily from radiant heat and thermal energy exposure at or near the body surface. As a result, flash burns resemble conventional thermal burns more closely than classical electrocution injuries. These injuries are not associated with defined entry or exit points, as current conduction through the body is minimal or absent. The severity of tissue damage is variable and depends on proximity to the arc, duration of exposure, and environmental conditions. Cutaneous manifestations range from superficial erythema and hyperemia in mild cases to deep partial-thickness or full-thickness burns characterized by dry, leathery eschar formation and extensive tissue necrosis in more severe exposures. Pediatric populations represent a particularly vulnerable group for flash electrical injuries due to exploratory behaviors involving electrical cords and household devices. A common mechanism involves oral exposure, where children bite or suck on electrical wires or appliances, resulting in arc formation between the external and internal surfaces of the oral cavity. This can produce significant injury to perioral structures, including potential involvement of the orbicularis oris muscle. When the burn extends across the oral commissure, it may lead to significant aesthetic and functional impairment, including lip deformities and compromised oral competence. These injuries often progress over time, with significant edema and eschar formation developing within 48 to 72 hours following the initial insult. A critical complication in such cases involves vascular involvement, particularly when the labial artery is affected by necrotic tissue. In these situations, delayed hemorrhage may occur when the eschar separates, typically within two to three weeks after injury. This delayed bleeding can be severe and requires anticipatory management strategies. Continuous monitoring and multidisciplinary follow-up involving burn specialists, oral surgeons, or plastic surgeons are essential to optimize functional and cosmetic outcomes. Caregiver education is a crucial component of management, particularly regarding early recognition of bleeding complications. Parents or guardians should be instructed to apply firm local pressure, such as compressing the lips manually, while urgently transporting the child to an appropriate medical facility for definitive care [13].

Flame Electrical Injury

Flame electrical injury arises when electrical energy produces sufficient heat to ignite surrounding combustible materials, most commonly clothing, resulting in secondary thermal injury rather than direct electrical tissue damage. In this mechanism, the primary insult is mediated through radiative and conductive heat transfer following ignition initiated by an electrical source. Electrical current itself may not necessarily traverse the body in a significant manner; instead, the injury is predominantly thermal in nature once ignition occurs. This distinction is clinically important because flame burns in electrical contexts often combine features of both electrical exposure and conventional thermal trauma, thereby complicating clinical assessment and management. A key mechanism in flame electrical injury involves ignition of clothing or nearby combustible materials due to electrical discharge or arc formation. Once ignition occurs, prolonged exposure to flames results in progressive thermal damage to the skin and underlying tissues. In many cases, patients sustain additional contact burns when burning garments remain in direct contact with the skin surface, intensifying the depth and extent of injury. The severity of tissue destruction is further exacerbated when synthetic fabrics such as polyester or nylon are involved, as these materials have low melting points and can liquefy under heat exposure. Once melted, they may adhere firmly to the skin, producing deeper burns and increasing the risk of full-thickness tissue necrosis. This adherence complicates wound debridement and is associated with more extensive long-term morbidity, including scarring and contracture formation.

Lightning Electrical Injury

Lightning injury represents a unique and extreme form of electrical trauma characterized by exposure to massive electrical energy generated during atmospheric discharge. In most cases, affected individuals are outdoors and become part of the lowest resistance pathway between the cloud and the earth, effectively serving as a conduit for electrical current. Despite the extraordinary voltage involved, the clinical spectrum of lightning injury is highly variable, ranging from mild or superficial cutaneous burns to catastrophic multisystem failure resembling high-voltage industrial electrical exposure. The variability in presentation is largely influenced by factors such as current dispersion, duration of exposure, and environmental conditions at the time of strike. One of the most distinctive dermatological findings associated with lightning injury is the presence of Lichtenberg figures, which are transient, fern-like erythematous patterns on the skin surface. Although not universally present, their occurrence is considered highly suggestive of lightning exposure and serves as an important diagnostic clue in appropriate clinical contexts. In addition to cutaneous manifestations, lightning injuries frequently involve multiple organ systems. Otorhinolaryngological complications may include ruptured tympanic membranes due to pressure wave transmission. Ophthalmic injuries can also occur, ranging from transient visual disturbances to cataract formation and retinal damage. Neurological involvement is particularly prominent in lightning injury and may manifest as seizures, transient or permanent paralysis, cognitive impairment, and sensory disturbances such as paresthesias. A notable and distinctive neurological phenomenon associated with lightning exposure is keraunoparalysis, also referred to as Charcot paralysis. This condition is characterized by transient flaccid paralysis, often involving all four limbs, accompanied by loss of proprioception and vascular instability. Keraunoparalysis typically resolves spontaneously, with recovery occurring over a period ranging from several hours to a few weeks. The transient nature of this condition differentiates it from structural neurological injury, although it may initially mimic more severe spinal cord pathology, necessitating careful clinical evaluation and monitoring [16].

Evaluation

Individuals affected by electrical injuries require simultaneous assessment as both trauma patients and cardiac patients due to the multisystem nature of electrical exposure. Initial evaluation must prioritize life-threatening conditions using structured resuscitation principles, followed by focused cardiovascular and systemic assessment. All adult patients who sustain electrical injury should undergo a baseline electrocardiogram and continuous cardiac monitoring to detect immediate and delayed rhythm disturbances. The need for prolonged observation is determined by clinical risk factors, including the presence of chest pain, electrocardiographic abnormalities, a transthoracic current pathway, episodes of cardiac arrest, loss of consciousness at the scene, or pre-existing cardiac disease. Patients who do not demonstrate significant clinical findings or ECG abnormalities at presentation are generally at low risk for delayed cardiac complications, as evidence indicates that most clinically relevant arrhythmias manifest within the first 24 to 48 hours of observation [17][18][19][20]. In patients presenting with low-voltage electrical exposure, normal ECG findings, and absence of cardiac symptoms or underlying cardiovascular disease, discharge may be considered safe following comprehensive physical examination and appropriate clinical assessment. Similarly, pediatric patients exposed to household electrical currents, particularly low-voltage sources, may also be discharged when no evidence of significant injury is identified and when cardiac history and examination findings are unremarkable. These decisions must be supported by careful documentation and exclusion of occult injury, as subtle presentations may mask deeper tissue involvement.

Laboratory evaluation plays an essential role in identifying systemic complications associated with electrical injury. Recommended baseline investigations include complete blood count, comprehensive metabolic profile with particular attention to electrolyte imbalance and renal function, and urinalysis to assess for myoglobinuria. The presence of myoglobin in urine necessitates further assessment of serum myoglobin levels due to the risk of muscle breakdown and renal toxicity. In cases where rhabdomyolysis is suspected or respiratory compromise is present, arterial blood gas analysis may be required to evaluate acid-base status and oxygenation. Creatine kinase measurement is a key diagnostic marker for muscle injury and should be obtained in all patients with suspected deep tissue involvement. When clinical suspicion exists for thoracic current passage, or when chest pain, arrhythmias, or ECG abnormalities are present, cardiac biomarkers such as CK-MB and troponin should be evaluated to assess myocardial injury. Radiological assessment should be guided by clinical presentation and suspected injury mechanisms. Chest radiography is indicated in patients with cardiopulmonary instability, respiratory symptoms, suspected thoracic trauma, or those requiring resuscitation following electrical exposure. It is also useful in evaluating complications following falls or blunt trauma associated with electrical incidents. Neuroimaging with computed tomography of the head is warranted in patients with altered mental status, seizures, focal neurological deficits, loss of consciousness, or suspected cranial trauma. In such cases, cervical spine immobilization is recommended until spinal injury is excluded, and further cervical imaging may be required depending on clinical findings. However, routine cervical imaging is not necessary in patients without neurological deficits or significant trauma indicators. A critical principle in the evaluation of electrical injury is the poor correlation between external burn severity and internal tissue damage. The absence of visible cutaneous injury does not exclude significant deep tissue or organ involvement. Consequently, additional imaging modalities such as computed tomography or ultrasound may be required based on the suspected anatomical trajectory of the electrical current and associated clinical findings. The selection of imaging should be individualized and directed by suspected injury distribution rather than external appearance alone. Severe electrical injuries, particularly those involving high-voltage exposure or prolonged contact with low-voltage current, may result in extensive tissue necrosis, necessitating surgical intervention. Fasciotomy is often required in cases complicated by compartment syndrome or progressive tissue edema. Early surgical consultation is essential in such scenarios, as delayed intervention significantly increases the risk of irreversible ischemia, infection, and eventual limb loss. Prompt recognition and multidisciplinary management are therefore critical in optimizing functional outcomes and reducing long-term morbidity associated with electrical trauma [20].

Treatment / Management

The management of electrical injuries requires immediate, structured, and multidisciplinary intervention, beginning at the point of emergency department arrival. Patients must be rapidly assessed and stabilized using established Advanced Cardiovascular Life Support and Advanced Trauma Life Support principles, with simultaneous attention to airway, breathing, and circulation. Given the unpredictable multisystem involvement of electrical trauma, continuous cardiac monitoring is indicated in all patients who have sustained more than minor low-voltage burns, as electrical current can precipitate immediate or delayed arrhythmogenic events even in the absence of overt external injury. Airway management is a critical priority in patients presenting with facial or oral burns, respiratory distress, hypoxia, altered consciousness, or any clinical feature suggestive of compromised airway patency. Early oxygen supplementation should be initiated, followed by escalation to advanced airway interventions when necessary, including endotracheal intubation or emergency surgical airway procedures such as cricothyrotomy in cases of airway obstruction or severe thermal injury. Electrical burns involving the head and neck region may progress rapidly due to tissue edema, making early airway protection essential to prevent catastrophic respiratory compromise. In addition to airway stabilization, spinal protection must be considered in patients with suspected high-energy trauma, falls, or loss of consciousness. Cervical spine immobilization, and in selected cases full spinal precautions, should be implemented until structural injury is excluded. A systematic primary trauma survey is required to identify concurrent injuries such as pneumothorax, internal hemorrhage, or fractures, which may result from either the electrical event itself or secondary traumatic mechanisms such as falls or explosive force [20][21].

Following initial stabilization, attention must be directed toward the prevention and treatment of systemic complications, particularly rhabdomyolysis and burn-related shock. Patients with significant burns or suspected muscle breakdown, as indicated by myoglobinuria, require aggressive intravenous fluid resuscitation. The therapeutic goal is to maintain adequate renal perfusion, with urine output targeted between 1 and 2 mL/kg/hour. This strategy is essential to minimize the risk of acute kidney injury secondary to myoglobin precipitation within renal tubules. Adjunctive therapies such as osmotic diuretics like mannitol, loop diuretics such as furosemide, and urine alkalinization using sodium bicarbonate may be considered in selected cases. However, these interventions should be applied cautiously, as they may exacerbate hypovolemia in patients with extensive burns exceeding 20% of total body surface area or in those with concomitant traumatic injuries, where hemodynamic instability is a significant concern. Vascular access is a fundamental component of acute management. All adult patients require prompt establishment of intravenous access, with intraosseous access serving as an alternative when peripheral cannulation is unsuccessful. In patients with severe trauma, cardiac arrest, or hemodynamic instability, central venous access may be required for rapid administration of fluids and medications. Wherever possible, vascular access should avoid burned tissue due to increased infection risk; however, in life-threatening situations, access through injured tissue may be temporarily necessary when alternative sites are unavailable, as resuscitation remains the immediate priority over infection prevention [21].

Burn-specific management must be initiated early, including wound care, analgesia, and tetanus prophylaxis according to immunization status. Careful neurovascular assessment of affected limbs is essential prior to splinting or bandaging, as electrical injuries frequently involve deep tissue damage not immediately apparent on external examination. Immobilization of injured extremities may be required to prevent further tissue damage and reduce pain. Disposition decisions are based on clinical severity, systemic involvement, and risk of delayed complications. Patients with cardiac arrest, respiratory compromise, syncope, chest pain, abnormal electrocardiographic findings, significant burns, or major trauma require inpatient admission for close monitoring. Many of these patients benefit from transfer to specialized burn units or intensive care settings where multidisciplinary expertise is available. Early involvement of trauma surgeons, critical care physicians, orthopedic specialists, and burn care teams is essential to optimize outcomes and prevent complications such as infection, compartment syndrome, and organ failure. Rehabilitation planning and discharge education are integral components of long-term care. Patients should receive comprehensive counseling regarding potential sources of electrical exposure in both domestic and occupational environments, with emphasis on preventive safety measures. Education should also address possible long-term sequelae, including neurological deficits, chronic pain syndromes, psychological trauma, and functional impairment. Structured follow-up appointments should be arranged based on injury severity to ensure ongoing monitoring, rehabilitation, and early identification of complications [22].

Emerging Therapies

Recent advances in translational medicine have introduced novel therapeutic strategies aimed at improving outcomes in electrical injury. Poloxamer-188, a nonionic surfactant composed of hydrophilic and hydrophobic segments, has demonstrated promising potential in repairing disrupted cellular membranes. Electrical trauma is characterized at the cellular level by electroporation and membrane destabilization, and Poloxamer-188 has been shown to restore membrane integrity, thereby limiting progressive muscle and nerve damage. Experimental studies suggest that it may also attenuate inflammatory responses and reduce secondary tissue degeneration. Although initially studied in conditions such as sickle cell disease and ischemia-reperfusion injury, its application in electrical burn management is currently under investigation. Ongoing clinical trials are evaluating its safety profile, pharmacokinetics, and therapeutic efficacy, with the aim of determining its potential incorporation into future treatment protocols [21][22]. Regenerative medicine approaches, particularly stem cell-based therapies, represent another promising frontier in the management of electrical injuries. Mesenchymal stem cells derived from bone marrow, adipose tissue, and umbilical cord sources have demonstrated significant immunomodulatory and regenerative properties. These cells contribute to tissue repair by reducing inflammatory cascades, inhibiting fibrotic processes, and enhancing angiogenesis, which is essential for wound healing and tissue regeneration. Given the extensive neuromuscular and vascular damage associated with high-voltage injuries, stem cell therapy offers a biologically plausible strategy to enhance cellular survival and improve functional recovery. Preclinical studies have also indicated potential benefits in reducing oxidative stress and limiting delayed neuropathy, a common complication following electrical trauma. Although still experimental, ongoing research aims to define optimal delivery methods, dosing strategies, and long-term outcomes, with the potential to transform future standards of care [23][24]. Hyperbaric oxygen therapy represents an additional adjunctive treatment modality for severe electrical injuries, particularly in cases involving extensive soft tissue damage. This therapy involves administration of 100% oxygen under increased atmospheric pressure, thereby enhancing oxygen diffusion into hypoxic tissues. Improved tissue oxygenation promotes angiogenesis, reduces edema, and supports fibroblast activity, which collectively accelerates wound healing. Hyperbaric oxygen therapy may also reduce infection risk and improve the viability of skin grafts in patients with extensive burns. In the context of electrical injury, where microvascular damage and tissue hypoxia are common, this modality offers supportive benefits in tissue preservation and functional recovery, particularly when integrated into a comprehensive multidisciplinary treatment plan [25].

Differential Diagnosis

The differential diagnosis of electrical burns requires careful clinical consideration due to the wide range of conditions that can mimic or coexist with electrical injury. Chemical burns represent an important alternative diagnosis, particularly in occupational settings where corrosive substances may produce tissue damage similar in appearance to electrical thermal injury. Thermal burns from direct flame exposure also closely resemble electrical burn patterns, especially when clothing ignition is involved, making accurate history taking essential. Intracranial hemorrhage should be considered in patients presenting with altered consciousness, seizures, or neurological deficits following an electrical event, particularly when associated with falls or high-energy trauma. Ocular burns and chemical injuries may present with pain, visual disturbance, or corneal damage that can overlap with electrical arc-related eye injury. Respiratory arrest is another critical differential, especially in cases of thoracic current passage or associated trauma, requiring immediate exclusion in emergency settings. Rhabdomyolysis may present with muscle pain, weakness, and dark urine, and may be either a primary consequence of electrical injury or an independent metabolic or traumatic condition. Seizures and status epilepticus must be differentiated from post-electrical neurological dysfunction or hypoxic brain injury, particularly when events are unwitnessed. Syncope is frequently encountered and may be cardiac, neurogenic, or situational in origin, requiring careful distinction from transient electrical shock effects. Ventricular fibrillation remains a critical consideration, as it may represent both a primary electrical complication and an alternative diagnosis in cases of sudden collapse [23].

Prognosis

The prognosis of electrical injuries is highly variable and depends on multiple interrelated factors, including the voltage level, anatomical distribution of injury, duration of exposure, and the presence of systemic complications. High-voltage injuries are generally associated with significantly worse outcomes compared to low-voltage exposures due to deeper tissue penetration and greater likelihood of multi-organ involvement. The development of complications such as cardiac arrhythmias, renal failure, compartment syndrome, or extensive burns further worsens prognostic outcomes. Neurological involvement and delayed tissue necrosis also contribute to long-term morbidity and functional impairment. Despite these risks, advances in modern medical care have markedly improved survival rates and functional recovery. Improvements in intensive care management, early aggressive resuscitation protocols, refined surgical techniques, and the use of advanced wound care technologies, including bioengineered skin substitutes, have collectively enhanced patient outcomes. Early recognition and multidisciplinary intervention remain central determinants of prognosis, particularly in preventing irreversible tissue loss and systemic complications [23].

Complications

Electrical injuries involving high electrical field strength are associated with extensive cellular and tissue damage due to combined electrochemical and thermal effects. This results in protein denaturation, coagulative necrosis, hemolysis, vascular thrombosis, and structural damage to muscles and tendons, often leading to avulsion injuries. These primary effects are frequently compounded by secondary complications, including severe tissue edema resulting from vascular injury, thrombosis, and muscle swelling. Progressive edema may culminate in compartment syndrome, a limb-threatening condition requiring urgent intervention. Fluid shifts associated with tissue injury can lead to dehydration, hypovolemia, and hypotension, further compromising organ perfusion. Severe muscle destruction significantly increases the risk of rhabdomyolysis, characterized by myoglobin release, myoglobinuria, electrolyte disturbances, and metabolic acidosis. These systemic derangements place patients at high risk of acute kidney injury due to tubular obstruction and nephrotoxic effects of myoglobin. Overall, complications of electrical injury frequently extend beyond localized tissue damage and involve multisystem physiological disruption [24].

Long-Term Sequelae of Electrical Injuries

Electrical injuries may result in persistent long-term sequelae affecting multiple organ systems, with neurological complications being among the most prevalent. Patients may experience chronic neuropathy, recurrent seizures, syncope, tinnitus, paresthesias, persistent weakness, and impaired balance or gait ataxia, reflecting both central and peripheral nervous system involvement. Cognitive and psychological sequelae are also significant and may include impaired memory and attention, irritability, depressive disorders, post-traumatic stress, and reduced overall psychological functioning. These effects may persist long after initial physical recovery and significantly impair quality of life. Ocular complications such as cataract formation may develop following electrical exposure due to lens protein disruption. Physical sequelae are equally diverse and include chronic pain syndromes, fatigue, joint contractures, muscle spasms, pruritus, headaches, febrile episodes, and reduced range of motion due to soft tissue fibrosis and musculoskeletal injury. These long-term outcomes highlight the multisystem nature of electrical trauma and the need for prolonged follow-up care [23][24][25].

Consultations

Management of electrical injuries requires a coordinated multidisciplinary approach involving several medical specialties to address the complex and multisystem nature of the condition. General and burn surgeons play a central role in wound assessment, debridement, and surgical management of necrotic tissue. Orthopedic surgeons are frequently required for the management of fractures, musculoskeletal injuries, and compartment syndrome. Critical care and trauma specialists provide essential support in hemodynamic stabilization and management of systemic complications such as shock and respiratory failure. Neurologists and neurosurgeons are involved in evaluating and treating central and peripheral nervous system injuries, including seizures and spinal involvement. Cardiologists are essential in managing arrhythmias and myocardial injury associated with electrical conduction through the thorax. Psychiatric consultation is often required for psychological sequelae such as post-traumatic stress and depression. Physiotherapists play a key role in long-term rehabilitation, focusing on mobility restoration, functional recovery, and prevention of contractures [25].

Patient Education

Prevention of electrical injuries relies heavily on adherence to safety protocols and environmental risk reduction strategies. At the household level, preventive measures include the use of child-proof electrical outlet covers, proper storage and positioning of electrical cords out of reach of children, and strict compliance with manufacturer instructions for all electrical appliances. Damaged electrical equipment, particularly those with exposed or frayed wiring, should be promptly replaced to minimize risk of exposure. The use of electrical devices in wet environments such as showers or bathtubs must be strictly avoided due to increased conductivity and risk of severe injury. In occupational settings, adherence to electrical safety procedures is critical, including deactivation of power sources at the circuit breaker and confirmation of de-energization using non-contact voltage testers prior to maintenance work. Patient education prior to discharge is essential and should include counseling on potential environmental sources of electrical exposure, recognition of early symptoms of complications, and awareness of long-term neurological, psychological, and physical consequences. Structured follow-up appointments should be scheduled to monitor recovery, manage complications, and support rehabilitation efforts [26][27][28].

Conclusion

Electrical injury represents a multisystem traumatic event with complex mechanisms that extend beyond visible skin damage. Clinical outcomes depend on voltage, current pathway, exposure duration, and tissue resistance. High-voltage exposure produces deeper tissue destruction, higher complication rates, and increased mortality compared with low-voltage injury. However, even low-voltage exposure can cause life-threatening arrhythmias, neurological impairment, and internal organ injury without significant external burns. Early recognition and structured emergency management reduce preventable deaths. Airway protection, cardiac monitoring, fluid resuscitation, and early surgical evaluation remain central to care. Delayed complications such as compartment syndrome, rhabdomyolysis, renal failure, and chronic neurological deficits require ongoing surveillance. Recovery is not limited to acute survival. Long-term functional, psychological, and occupational rehabilitation determine final outcomes. Multidisciplinary involvement improves prognosis. Emergency medicine, burn surgery, cardiology, neurology, critical care, rehabilitation, and mental health services must work in coordination. Prevention strategies remain essential because most injuries are avoidable through electrical safety practices in home and workplace environments. Electrical injury should be treated as a systemic disease process rather than a localized burn injury. Outcomes improve when clinicians maintain high suspicion for hidden internal damage, even in patients with minimal external findings. Early intervention and structured follow-up define survival and quality of recovery.

References

1. Burnham T, Hilgenhurst G, McCormick ZL. Second-degree Skin Burn from a Radiofrequency Grounding Pad: A Case Report and Review of Risk-Mitigation Strategies. PM R. 2019 Oct;11(10):1139-1142.
2. Kim MS, Lee SG, Kim JY, Kang MY. Maculopathy from an accidental exposure to welding arc. BMJ Case Rep. 2019 Feb 03;12(2)
3. Carrano FM, Iezzi L, Melis M, Quaresima S, Gaspari AL, Di Lorenzo N. A Surgical Instrument Cover for the Prevention of Thermal Injuries During Laparoscopic Operations. J Laparoendosc Adv Surg Tech A. 2019 Jan 30
4. Lovaglio AC, Socolovsky M, Di Masi G, Bonilla G. Treatment of neuropathic pain after peripheral nerve and brachial plexus traumatic injury. Neurol India. 2019 Jan-Feb;67(Supplement):S32-S37.
5. Trivedi TK, Liu C, Antonio ALM, Wheaton N, Kreger V, Yap A, Schriger D, Elmore JG. Injuries Associated With Standing Electric Scooter Use. JAMA Netw Open. 2019 Jan 04;2(1):e187381.
6. Daskal Y, Beicker A, Dudkiewicz M, Kessel B. [HIGH VOLTAGE ELECTRIC INJURY: MECHANISM OF INJURY, CLINICAL FEATURES AND INITIAL EVALUATION.]. Harefuah. 2019 Jan;158(1):65-69.
7. Bailey ME, Sagiraju HKR, Mashreky SR, Alamgir H. Epidemiology and outcomes of burn injuries at a tertiary burn care center in Bangladesh. Burns. 2019 Jun;45(4):957-963.
8. Von Caues S, Herbst CI, Wadee SA. A retrospective review of fatal electrocution cases at Tygerberg Forensic Pathology Services, Cape Town, South Africa, over the 5-year period 1 January 2008 - 31 December 2012. S Afr Med J. 2018 Nov 26;108(12):1042-1045
9. Karray R, Chakroun-Walha O, Mechri F, Salem I, Drira H, Nasri A, Damak A, Rekik N. Outcomes of electrical injuries in the emergency department: epidemiology, severity predictors, and chronic sequelae. Eur J Trauma Emerg Surg. 2025 Jan 27;51(1):85.
10. Culnan DM, Farner K, Bitz GH, Capek KD, Tu Y, Jimenez C, Lineaweaver WC. Volume Resuscitation in Patients With High-Voltage Electrical Injuries. Ann Plast Surg. 2018 Mar;80(3 Suppl 2):S113-S118.
11. Esses SI, Peters WJ. Electrical burns; pathophysiology and complications. Can J Surg. 1981 Jan;24(1):11-4.
12. Rouse RG, Dimick AR. The treatment of electrical injury compared to burn injury: a review of pathophysiology and comparison of patient management protocols. J Trauma. 1978 Jan;18(1):43-7.
13. Shih JG, Shahrokhi S, Jeschke MG. Review of Adult Electrical Burn Injury Outcomes Worldwide: An Analysis of Low-Voltage vs High-Voltage Electrical Injury. J Burn Care Res. 2017 Jan/Feb;38(1):e293-e298.
14. Schweizer R, Pedrazzi N, Klein HJ, Gentzsch T, Kim BS, Giovanoli P, Plock JA. Risk Factors for Mortality and Prolonged Hospitalization in Electric Burn Injuries. J Burn Care Res. 2021 May 07;42(3):505-512.
15. Ketenci IE, Duymus TM, Ulusoy A, Yanik HS, Mutlu S, Durakbasa MO. Bilateral posterior shoulder dislocation after electrical shock: A case report. Ann Med Surg (Lond). 2015 Dec;4(4):417-21.
16. Jacob NE, M R. Keraunoparalysis: Fleeting Paralysis Following a Lightning Flash. Cureus. 2024 Oct;16(10):e70773.
17. Pawlik AM, Lampart A, Stephan FP, Bingisser R, Ummenhofer W, Nickel CH. Outcomes of electrical injuries in the emergency department: a 10-year retrospective study. Eur J Emerg Med. 2016 Dec;23(6):448-454.
18. Davis C, Engeln A, Johnson EL, McIntosh SE, Zafren K, Islas AA, McStay C, Smith WR, Cushing T., Wilderness Medical Society. Wilderness Medical Society practice guidelines for the prevention and treatment of lightning injuries: 2014 update. Wilderness Environ Med. 2014 Dec;25(4 Suppl):S86-95.
19. Gentges J, Schieche C. Electrical injuries in the emergency department: an evidence-based review. Emerg Med Pract. 2018 Nov;20(11):1-20.
20. Lee DH, Desai MJ, Gauger EM. Electrical Injuries of the Hand and Upper Extremity. J Am Acad Orthop Surg. 2019 Jan 01;27(1):e1-e8.
21. Wong SW, Yao Y, Hong Y, Ma Z, Kok SH, Sun S, Cho M, Lee KK, Mak AF. Preventive Effects of Poloxamer 188 on Muscle Cell Damage Mechanics Under Oxidative Stress. Ann Biomed Eng. 2017 Apr;45(4):1083-1092.
22. Moloughney JG, Weisleder N. Poloxamer 188 (p188) as a membrane resealing reagent in biomedical applications. Recent Pat Biotechnol. 2012 Dec;6(3):200-11.
23. Surowiecka A, Chrapusta A, Klimeczek-Chrapusta M, Korzeniowski T, Drukała J, Strużyna J. Mesenchymal Stem Cells in Burn Wound Management. Int J Mol Sci. 2022 Dec 05;23(23)

24. Wang M, Xu X, Lei X, Tan J, Xie H. Mesenchymal stem cell-based therapy for burn wound healing. Burns Trauma. 2021;9:tkab002.
25. Murias R, Court Y, Merbilhaá O, Fariña G, Pace EL, Biglia A, Igoillo MC, Echezuri J, Garone A, Crocenzi A, Portas M. Therapeutic approach to electric burn with platelet rich plasma, grafts and hyperbaric oxygenation. Medicina (B Aires). 2021;81(2):297-300.
26. Wesner ML, Hickie J. Long-term sequelae of electrical injury. Can Fam Physician. 2013 Sep;59(9):935-9.
27. Nisar S, Keyloun JW, Kolachana S, McLawhorn MM, Moffatt LT, Travis TE, Shupp JW, Johnson LS. Institutional Experience Using a Treatment Algorithm for Electrical Injury. J Burn Care Res. 2021 May 07;42(3):351-356.
28. Gille J, Schmidt T, Dragu A, Emich D, Hilbert-Carius P, Kremer T, Raff T, Reichelt B, Siafliakis A, Siemers F, Steen M, Struck MF. Electrical injury - a dual center analysis of patient characteristics, therapeutic specifics and outcome predictors. Scand J Trauma Resusc Emerg Med. 2018 May 31;26(1):43.