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ACUTE KIDNEY INJURY IN ELECTRICAL TRAUMA

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Abstract. Introduction. Electrical burns are a major cause of high mortality and morbidity rates worldwide. The leading cause of death in such cases is usually refractory myoglobinuric acute kidney injury (AKI).
Evidence collection. A retrospective informative search was conducted using a spatial–vector descriptive model supplemented by a manual search for relevant articles.
Matherials and methods. The scientific literature was found using the search engines Scopus, CrossRef, Google Scholar and PubMed.
Evidence synthesis. Victims of electrical trauma frequently develop significant hypovolemia as a result of massive tissue damage caused by extravascular fluid leakage (sequestration), which leads to a further reduction of intravascular volume. Hypovolemia due to extravascular fluid loss may result in prerenal azotemia and acute tubular necrosis, typically aggravated by untimely or insufficient fluid resuscitation. Progressive ischemia in electrical injury, together with edema and tissue necrosis, induces metabolic acidosis, perpetuating a vicious cycle. The renal form of AKI is caused by acute tubular necrosis resulting from renal hypoperfusion, hemolysis, rhabdomyolysis, and inter-fascial compartment syndrome. Three major mechanisms are involved in the pathogenesis of AKI: intrarenal vasoconstriction, formation of hemoglobin or myoglobin casts in distal renal tubules, and the direct cytotoxic effect of myoglobin on nephron endothelium. Rhabdomyolysis occurs in 14–42 % of electrical burn cases. Because the definitions of rhabdomyolysis vary widely, the exact incidence of AKI in rhabdomyolysis is difficult to establish but is estimated to range from 13 % to 48 %. Free myoglobin enters the systemic circulation because of massive skeletal muscle necrosis (rhabdomyolysis) and may lead to pigment-induced acute kidney injury. A blood pH below 7.2 is an indication for bicarbonate infusion until normalization (but not beyond). The only effective treatment for AKI patients with elevated creatinine and potassium levels is renal replacement therapy.
Conclusions. The main etiological factor of AKI in electrical injury is the presence of free hemoglobin or myoglobin in the bloodstream. Timely and adequate fluid resuscitation prevents and limits AKI in electrical trauma. The combined use of intermittent and prolonged veno-venous hemodiafiltration represents the optimal methods of extracorporeal detoxification in myoglobinemia.

Keywords: electrical injury, rhabdomyolysis, acute kidney injury, treatment.

INTRODUCTION

Electrical burns are responsible for high mortality and morbidity rates worldwide. Mortality varies from country to country depending on socioeconomic status. In developed countries, the mortality rate ranges from 3–15 %, while in developing countries it reaches 21–27 %. The leading cause of death in such cases is refractory myoglobinuric acute kidney injury [1]. Electrical injuries are associated with a higher incidence of acute kidney injury (AKI) compared with purely thermal burns. The incidence of AKI following electrical burns varies across countries from 1.5 % to 12.7 %. In most study groups, men predominated, accounting for up to 90.47 % of all victims [2-4]. Risk factors for AKI include older age, a larger total body surface area (TBSA) of burn injury, and the presence of contact electrical burns. In developing countries, rural residents are at greater risk of developing AKI after electrical trauma (ET), likely due to delays in receiving initial medical care [2].

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EVIDENCE COLLECTION

The selected literature sources were included in the study if they: (1) were published in Ukrainian, English, Spanish, or Chinese; (2) reported acute functional and morphological renal injuries caused by electrical trauma; (3) provided data on the prevalence of renal dysfunction in electrical injury; (4) employed an observational design (cohort or cross-sectional). A retrospective informative search was conducted using a spatial-vector descriptive model supplemented with a manual search for relevant articles.

MATHERIALS AND METHODS

The scientific literature was found using the search engines Scopus, CrossRef, Google Scholar and PubMed, and supplemented by a manual search for relevant articles using the keywords: electrical trauma, hemolysis, rhabdomyolysis, acute kidney injury. A total of 36 relevant scientific sources were analyzed, of which 97.2 % were published within the last 10 years and 69.4 % within the last 5 years.

EVIDENCE SYNTHESIS

The kidney is the organ responsible for eliminating metabolites excessively produced by damaged skin and other tissues in electrical trauma (ET). This overloads renal function, particularly in the removal of lipid derivatives [5]. Acute renal injury from electrical trauma may result from direct visceral damage [6]. Case reports describe incidents such as victims «hanging» on live wires, electric trauma during «train-surfing,» or injuries from illegal electrofishing devices.

Prerenal AKI in ET is most often caused by dehydration, hypovolemia, and cardiovascular failure [7-9]. Victims with massive tissue injury often develop marked hypovolemia due to extravascular fluid loss (sequestration), leading to further reduction in intravascular volume.

High-voltage current can cause violent muscle contractions, massive necrosis of deep structures, vessels, and nerves, contributing to impaired tissue neurotrophy, hypotension, and electrolyte imbalance [5]. In such cases, AKI arises from pathophysiological changes caused by electrical burns and is mainly due to decreased cardiac output against a background of severe intravascular fluid deficit.

Hypovolemia resulting from extravascular fluid loss can lead to prerenal azotemia and acute tubular necrosis [8-11]. This is usually aggravated by delayed or insufficient fluid resuscitation. Renal perfusion may not be adequately maintained by infusion therapy based on TBSA calculations alone [12]. Progressive ischemia, tissue edema, and necrosis lead to metabolic acidosis and perpetuate a vicious cycle [13-15].

The renal form of AKI is caused by acute tubular necrosis resulting from renal hypoperfusion, hemolysis, rhabdomyolysis, and inter-fascial compartment syndrome [7, 14, 16-18].

A decrease in urine output, even under conditions of adequate fluid administration, is often the first sign of AKI. Oliguria develops as a complication of rhabdomyolysis with myoglobinuria, causing pathological redistribution of fluid throughout the body in the form of anasarca and bilateral pleural effusion. Both anasarca and pleural effusion result from impaired renal excretory function [13].

Three major mechanisms are involved in the pathogenesis of AKI:

- Intrarenal vasoconstriction;
- Formation of hemoglobin or myoglobin casts in distal renal tubules;
- Direct cytotoxic effect of myoglobin on nephron endothelium [7–9, 12, 19].

Free hemoglobin. Intravascular fluid is an electrolyte solution, thus having high electrical conductivity and low resistance. For this reason, electrical trauma directly damages erythrocytes by electrical current as well as by high temperature generated by current passage through tissues.

When electrical trauma causes massive hemolysis, free hemoglobin released from red blood cells decomposes under heat, conjugates with haptoglobin, and is transported to the liver. Unconjugated hemoglobin is freely filtered through glomeruli and excreted in urine, leading to pigmenturia. Free hemoglobin exerts direct toxic effects on the renal tubular endothelium, causing degenerative changes and obstruction of renal tubules by the formation of hemoglobin casts (acidic hematins), which contribute to the development of acute hemoglobinuric nephrosis. This process is further aggravated by dehydration, acidosis, electric shock, and endotoxemia. Urine typically becomes bright red (lacquer-colored) without visible formed elements of blood [1, 13, 19].

Rhabdomyolysis develops in 14–42 % of electrical burn cases [2]. Because definitions of rhabdomyolysis vary widely, the precise incidence of AKI in rhabdomyolysis is difficult to determine, but it is estimated to range from 13 % to 48 % [6].

Free myoglobin enters the systemic circulation as a result of massive skeletal muscle necrosis (rhabdomyolysis) and may lead to pigment-induced acute renal injury [11, 18, 20].

Myoglobinuria is a common finding in patients with electrical burns: 36.4 % of them have pigmenturia lasting 48–72 hours, and in high-voltage injuries, the proportion of such patients can reach 75–100 % [21].

At plasma myoglobin concentrations below 92 μg/L, the protein is freely filtered through the glomeruli and reabsorbed in renal tubules. When its level rises under conditions of acidosis, the iron-containing fragment of myoglobin reacts with urinary glycoproteins (Tamm–

Horsfall protein), forming insoluble complexes that obstruct renal tubules. The renal threshold for free myoglobin is 150–170 μg/L. Normally, its urinary concentration does not exceed 5 μg/L, but an increase to 20 μg/L indicates rhabdomyolysis.

Creatine phosphokinase (CPK) activity rises within 2–12 hours after ET and gradually decreases within 7–10 days. Persistent elevation of CPK indicates a high risk of AKI. Levels of rhabdomyolysis markers correlate directly with renal function parameters and the need for renal replacement therapy (RRT). CPK and myocardial CPK-MB correlate within 6 hours after ET, while free myoglobin concentration correlates on the third day. If no positive dynamics are observed, persistent elevation of free myoglobin is a valid marker of renal injury risk and mortality [22].

Serum creatine-creatinine phosphotransferase may exceed the upper normal limit by more than five times and reach values over 10,000 U/L, peaking on day 3 and halving every 24–48 hours [6].

Myoglobinuric nephrosis and pathophysiology AKI is the most significant complication of rapid myoglobin release and occurs due to myoglobin-induced renal vasoconstriction with subsequent ischemia, formation of myoglobin casts in distal convoluted tubules (causing obstruction), and the nephrotoxic effect of myoglobin on epithelial cells of proximal tubules [1, 6, 15, 23, 24].

Rhabdomyolysis is characterized by myoglobinemia and myoglobinuria, which increase the risk of AKI and water–electrolyte imbalances that may lead to structural renal damage [13, 25, 26]. The condition is exacerbated by cortical ischemia and decreased glomerular filtration rate due to generalized hypovolemia [1, 23, 24].

Free myoglobin reaches renal tubules approximately four times faster than unconjugated hemoglobin. Consequently, urine takes on a dark brown, almost black, color. Concurrently, serum creatinine and urea levels rise against a background of metabolic acidosis and hyperkalemia – a condition defined as myoglobinuric nephrosis [8, 10, 13, 19, 27].

Early identification of rhabdomyolysis is challenging, since the classic triad of symptoms – muscle pain, weakness, and dark urine – may be masked when patients are sedated or receiving inadequate fluid therapy [6].

A 2.6-fold increase in lactate concentration indicates tissue hypoperfusion and metabolic acidosis (pH ≤ 7.29) and is often accompanied by stress hyperglycemia (> 50%) [7–9]. Elevated levels of lactate dehydrogenase (LDH) and myoglobin are associated with a higher risk of hospitalization [28].

Blood urea is not a reliable independent indicator, as it can also rise in non-renal conditions such as dehydration or a high-protein diet [13].

An increase in hematocrit or hemoglobin indicates hemoconcentration due to plasma volume deficit, although hematocrit levels may later fall due to erythrocyte damage during resuscitation [12].

Diagnostics. Early identification of signs of AKI is essential to prevent its progression. AKI may be detected clinically or through laboratory findings. Clinically, it can present with or without oliguria; therefore, laboratory confirmation is necessary. Diagnostic indicators of AKI include:

- urine osmolality < 400 mOsm/kg,
- urinary sodium concentration > 40 mEq/L,
- and the presence of brown granular casts, as well as granular or cuboidal tubular epithelial cells in urine sediment [13].

Serum creatinine does not accurately reflect the degree of renal injury in patients with electrical burns and AKI. It may overestimate glomerular filtration rate (GFR) by 10–20 %. Changes in GFR are more informative than fluctuations in serum creatinine, since muscle injury actively affects creatinine production and clearance even during hospitalization [12].

Elevated serum levels of glutamate-oxaloacetate transaminase (GOAT) and glutamate-pyruvate transaminase (GPT) are nonspecific and are often increased only in patients with cutaneous burns [29].

Symptoms of AKI range from mild elevation of muscle enzyme levels to life-threatening conditions associated with extreme enzyme activity, electrolyte imbalance, and AKI itself [13].

Renal dysfunction due to rhabdomyolysis is one of the most concerning complications of high-voltage electrical injury and may be associated with hyperkalemia [30]. A common pathogenic pathway in rhabdomyolysis is increased intracellular cytoplasmic and mitochondrial calcium levels. This may result from decreased adenosine triphosphate (ATP) reserves or direct membrane disruption. ATP depletion leads to dysfunction of Na^+/K^+-ATPase and Ca^{2+}-ATPase pumps, which are essential for maintaining muscle cell integrity. This results in the release of muscle enzymes into the bloodstream. Renal vasoconstriction, direct and indirect tubular injury (ischemic), and intratubular obstruction by acidic myoglobin derivatives also contribute to renal damage [13].

AKI in electrical trauma is diagnosed according to KDIGO criteria:

- increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours, or
- increase in serum creatinine ≥ 1.5 times the baseline within the previous 7 days, or
- urine output < 0.5 mL/kg/h for more than 6 hours [2].

Treatment. The primary goal of infusion therapy in AKI is to achieve **euvolemia** and ensure adequate renal perfusion. In patients with AKI accompanied

by oliguria or anuria, excessive fluid overload should be avoided. Timely correction of hypotension helps prevent AKI and accelerate recovery. The target mean arterial pressure (MAP) is 65 mm Hg [7–10, 15, 20, 31].

Balanced crystalloid solutions are preferred over 0.9 % sodium chloride, as they reduce mortality. Synthetic colloids offer no advantage over crystalloids in AKI, except in cases of profound hypovolemia. Moreover, synthetic colloids increase the risk of requiring renal replacement therapy (RRT). In cases of vasoplegia and hypovolemia, vasopressor and inotropic agents are used alongside fluids to restore cardiac output. The first-line vasopressor for AKI is norepinephrine [7–9].

Serum electrolyte levels – particularly potassium – should be monitored every 2–4 hours after initiation of treatment, depending on prior values, renal function, and clinical signs [10].

Management of hyperkalemia includes:

- stabilization of cardiomyocyte membranes,
- shifting potassium into the intracellular compartment,
- and removal of excess potassium from the body.

Therefore, treatment should include calcium gluconate and β_2-agonists [32–34].

The effectiveness of cation-exchange resins for preventing hyperkalemia in AKI patients in not supported by strong evidence [7–9].

In patients with hyponatremia and signs of extracellular volume depletion (hypotension, reduced skin turgor, elevated hematocrit), cerebral salt wasting may develop; sodium correction should therefore be performed cautiously [10].

Metabolic acidosis is common in AKI but rarely requires specific treatment (if urine pH > 6.5), except in severe cases. Previous recommendations to alkalinize urine in rhabdomyolysis have now been disproven. Intravenous sodium bicarbonate may be used based on measured blood and urine pH values. A blood pH below 7.2 is an indication for bicarbonate infusion until normalization – but not beyond physiological range – and correction of arterial blood gases [7–9, 19].

In some cases, myoglobinuria may be managed by administering an initial bolus of 25 g mannitol in adults (0.5 g/kg in children), followed by continuous infusion up to 0.5 g/kg/h. Normalization of free hemoglobin levels can usually be achieved soon after adequate fluid resuscitation and maintaining a urine output of 50–70 mL/h. However, if adequate diuresis is not achieved within 30 minutes, mannitol infusion should be discontinued to prevent rebound edema or the «ricochet syndrome» [1, 10, 19, 21, 33].

Saluretics (loop diuretics) in rhabdomyolysis should be used only for managing hypervolemia and have no other indications in AKI treatment [7–9].

The only effective method for patients with AKI accompanied by elevated creatinine and potassium levels is renal replacement therapy (RRT). One of the most common extracorporeal detoxification methods is hemodialysis, although it is known that standard hemodialysis alone does not effectively remove free hemoglobin from plasma [1, 12, 22, 33].

Some studies have shown benefits of early use of advanced Prismaflex membranes (AN69 ST150) for myoglobin clearance. Recently, novel cytokine adsorbers have been proposed for this purpose; a prospective randomized controlled trial on this topic is currently underway [31, 35].

Continuous veno-venous hemofiltration (CVVH) is a widely accepted adjunctive measure for myoglobin removal [12, 13, 36]. Combining intermittent veno-venous hemodiafiltration and prolonged veno-venous hemodiafiltration effectively reduces azotemia, hyperkalemia, rhabdomyolysis products, and markers of cytolytic syndrome [7–9].

CONCLUSIONS

1. The primary etiological factor of AKI in electrical trauma is the presence of free hemoglobin or myoglobin in the bloodstream.
2. Early and adequate fluid resuscitation prevents and limits AKI in electrical trauma.
3. The combined use of intermittent and prolonged veno-venous hemodiafiltration represents the optimal extracorporeal detoxification methods in patients with myoglobinemia.

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КРАВЕЦЬ О.В., ЄХАЛОВ В.В., МИНКА Н.В., СТАНІН Д.М., МАРТИНЕНКО Д.А.

ГОСТРЕ ПОШКОДЖЕННЯ НИРОК ПРИ ЕЛЕКТРИЧНІЙ ТРАВМІ

АНОТАЦІЯ

Вступ. Електричні опіки є причиною високих показників смертності та захворюваності в усьому світі. Основною причиною смерті в таких випадках буває рефрактерне міоглобінурійне гостре пошкодження нирок.

Збір доказів. Було зроблено ретроспективний інформативний пошук із використанням просторово-векторної описової моделі, що була доповнена ручним пошуком відповідних статей.
Матеріали та методи. Наукову літературу було знайдено за допомогою пошукових систем Scopus, CrossRef, Google Scholar та PubMed.
Синтез доказів. У постраждалих від електротравми внаслідок масивного пошкодження тканин часто розвивається досить виражена гіповолемія, яка викликана екстравазальним витоком (секвестрацією) рідини, що призводить до подальшого зменшення внутрішньосудинного об'єму. Гіповолемія внаслідок екстраваскулярного витоку рідини може призвести до преренальної азотемії та гострого канальцевого некрозу. Зазвичай цьому сприяє несвоєчасна або недостатня рідинна ресусцитація. Прогресуюча ішемія при електричній травмі, набряк та тканинний некроз обумовлюють метаболічний ацидоз та замикають порочне коло. Ренальну форму гострого пошкодження нирок спричиняє гострий тубулярний некроз внаслідок гіпоперфузії нирок, гемолізу, рабдоміолізу та міжфасциального компартмент-синдрому. У патогенезі гострого пошкодження нирок приймають участь три провідні механізми: внутрішньониркова вазоконстрикція, утворення гемоглобінових або міоглобінових циліндрів у дистальних ниркових канальцях та пряма цитотоксична дія міоглобіну на ендотелій нефронів. Рабдоміоліз розвивається у 14 – 42 % випадків електричних опіків. Оскільки визначення рабдоміолізу дуже різняться, точну частоту ГПН при рабдоміолізі важко встановити, але вважається, що вона коливається від 13 % до 48 %. Вільний міоглобін надходить до загального кровотоку внаслідок масивного некрозу м'язової тканини (рабдоміолізу) і може ускладнюватися пігмент-індукованим гострим ураженням нирок, pH крові нижче 7,2 є показанням до введення препарату до його нормалізації (але не більше). Єдиним ефективним методом лікування пацієнтів з ГПН із підвищенням рівнів креатиніну та калію є замісна ниркова терапія.
Висновки: Основним етіологічним фактором ГПН при електротравмі є наявність у кровотоці вільного гемоглобіну або міоглобіну. Своєчасно розпочата адекватна рідинна ресусцитація дозволяє запобігати та обмежувати ГПН при електротравмі. Дискретна та подовжена вено-венозна гемодіафільтрація в їх поєднанні є оптимальними методами екстракорпоральної детоксикації при міоглобінемії.

Ключові слова: електрична травма, рабдоміоліз, гостре пошкодження нирок, лікування.

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