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A CASE REPORT: RISK OF ELECTRIC INJURY ON DELAYED INITIAL TREATMENT

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ABSTRACT

Introduction: Electric Injury is a very aggressive burn injury with severe functional and aesthetic consequences caused by progressive and prolonged tissue necrosis. Necrosis that attacks the skeletal muscle can lead to rhabdomyolysis which results in complications if not treated properly.
Case Illustration: A complicated case of electric injury in Dr. Soebandi Jember General Hospital, a 26 years old man came to the emergency room with complaints of severe shortness of breath and urinary disorders. The patient had a history of having an electric shock in his right hand when turning on the fan a week prior of admission. The examination showed that the patient had bilateral pulmonary effusion, generalized edema and acute tubular necrosis (ATN) which was characterized by oliguria and even anuria accompanied by hematuria. Other symptoms experienced by patients are anterior uveitis, subconjunctival hemorrhage, and hematemesis.
Discussion: Electric injuries can have a wide range of effects on the body, and their management requires a multidisciplinary approach to address the various complications that may arise. Treatment of electric injuries often involves various surgical procedures, including skin grafts, flaps, or amputation, depending on the severity of the injury.
Conclusions: Early identification of potential complications in electric injury cases and effective patient follow-up can reduce the risk of Acute Kidney Injury (AKI) occurrence in patients with electric injuries.

Highlights:

1. Electric injuries are aggressive burns that can lead to severe tissue necrosis, rhabdomyolysis, and a range of complications, including acute kidney injury (AKI).
2. A multidisciplinary approach and early identification of potential complications in electric injury cases are crucial for reducing the risk of Acute Kidney Injury (AKI) and improving patient outcomes.

INTRODUCTION

Electric injury, a relatively common form of mechanical trauma, can occure as a result of lightning strikes, low voltage or high voltage electric shock, and is often associated with high morbidity and mortality. Almost all electric injuries occur accidentally and cannot be prevented. If the patient is not treated immediately, fatal damage to electric injury can result in multiple organ or tissue dysfunction.[1]

This phenomenon of electric injury is relatively rare. Although this case is rare, the morbidity and mortality from this incident is very high. In the United States, the American Burn Association estimates that 4,400 people have been injured in electric accidents and 400 have died from electric injury each year, most are related to work (miners, electricians, dan construction workers). The average victim is a young adult or teenager, who is often

injured as a result of outdoor adventure activities (such as climbing electric poles, or exploration of dangerous places) and children involved in household accidents[2]. In a study done by Liu, it was found that 0.5% of deaths were related to electrical injuries, and among these deaths, 60%-70% were caused by low voltage electricity and sometimes caused by short circuits from car batteries in America and China.[3]

In Indonesia, there is not many collective data on electric injury, one of epidemiological research on electric injury patients in the Burn Unit, Cipto Mangunkusumo General Hospital in 2009 – 2010 shows that as many as 11.8% patients treated at the Burn Unit were electric injury in origin[4]. A study by Martina and Wardhana in the Burn Unit, Cipto Mangunkusumo General Hospital from January 2011– Desember 2012 explained that as many as 76 adult patients died from burns, and 14% of the dead patients were caused by electricity[5]. Other data, based on the results of a preliminary study obtained from the Dr. Soebandi Jember General Hospital in March 2016–September 2017, there were 11 patients admitted due to elecric injury[6].

In the case of electric injury, survived patients often experience body function disorder and some require reconstructive surgeries[7]. Fatal complications caused by electric injury, especially in cardiac such as arrhythmias and respiratory arrest may cause death. The final result of electric injury depends on the intensity or voltage of exposure, the direction of electric current entering the body, the state of the body, and immediate and adequate therapy. Immediate and precise diagnosis is influenced by selective laboratory results. Proper handling and treatment reduce and even eliminate the losses especially caused by complications on electric injury.

CASE ILLUSTRATION

A 26 year old male came to Dr. Soebandi General Hospital and complained of severe shortness of breath. He also complained that his left eye was rather blurred and very little urine. The patient was exposed to an electric shock in his right hand when holding a damaged wall fan cable one week prior to admission. The electric shock lasted for more or less 5 minutes and the patient was unconscious. According to the patient's family, the voltage of his house is 220 volt. When he arrived at the emergency room at the private hospital, the patient regained consciousness and complained of dizziness, nausea, and blood vomiting with the remaining food twice. The patient was only treated for 2 days. Furthermore, the patient at his own request asked to be treated at home.

During home care, patient get 4.5 litres of fluid therapy in 4 days under the supervision of a nurse. Since being treated at home, patient complained of little amount of urine. On the 7th days after the incident, the patient felt difficulty in breathing, so the patient came to Dr. Soebandi Jember General Hospital.

When arrived at Dr. Soebandi Jember General Hospital, patient complained of shortness of breath and continuous hiccups. Hemodynamic was stable with blood pressure 144/90 mmHg, pulse 95 bpm regular and strong, respiratory rate 32 bpm with SpO2 96% and axillary temperature 36 °C. There was no prior history of hypertension.

The sign of anemia was found at physical examination of the head and neck. In addition, palpebral edema and redness of the sclera of the both eyes, and turbidity in the cornea of the patient's left eye.

Physical examination of the thoracic region starting from the inspection found retraction and lagging of the right lung motion,

palpation obtained from the right pulmonary fremitus decreased, percussion obtained a dimsound in the basal right and left lung, auscultation showed a decreased vesicular sound and there was crackles in the basal rightand left lung.

Physical examination of the abdominal region and genitalia, inspection slightly distended abdomen and scrotal edema with positive translumination, the percussion was shifting dullness positive. The initial urine can be obtained 150 mL/24 hours with cloudy yellow color.

Figure 1. Clinical Photograph of Burns Enteredue to Electric Injury.

Physical examination of the extremities was found at burns entry of the former electric shock only at the right hand as necrotic in digiti II as high as the middle and distal phalang, bone exposed at digiti I with tendon, tendon exposed at digiti III with a granulation at the edge of the wound. Exit burns in this patient were not found (Figure 1). Pitting edema was found in the examination of both lower extremities.

The investigation was carried out by laboratory test, ECG, and x-ray photos of the chest, and manus dextra. ECG results in the form of a sinus rhythm and no abnormalities were found (Figure 3). On thorax x-ray, extra pleural effusion is obtained, whereas manus dextra x- ray is not found abnormalities (Figure 2). Laboratory results showed anemia, increased leukocytes, increase in serum creatinine and hyponatremia and hypoglycemia. Whereas in the urinalysis proteinuria and erythrocytes are found (Table 1).

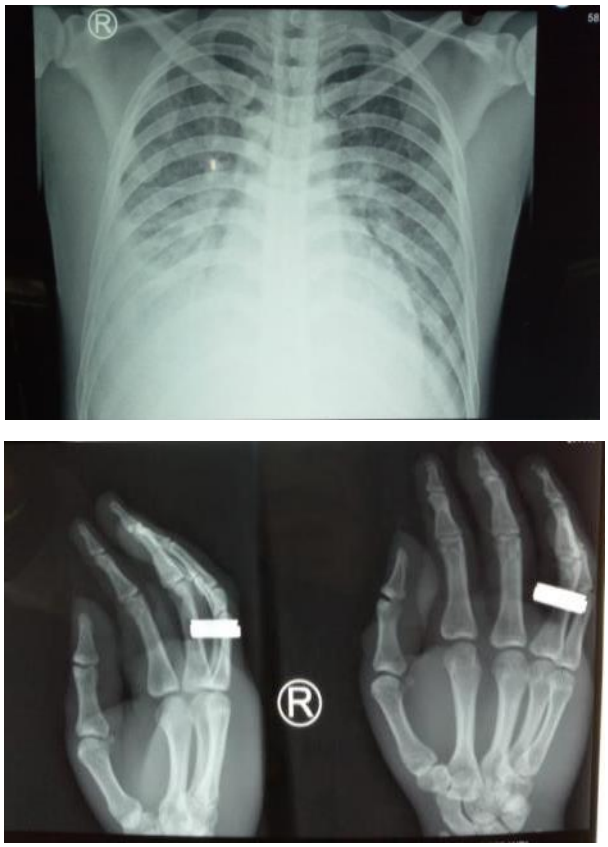


Figure 2. Thorax X-ray (2A) and Manus Dextra (2B)

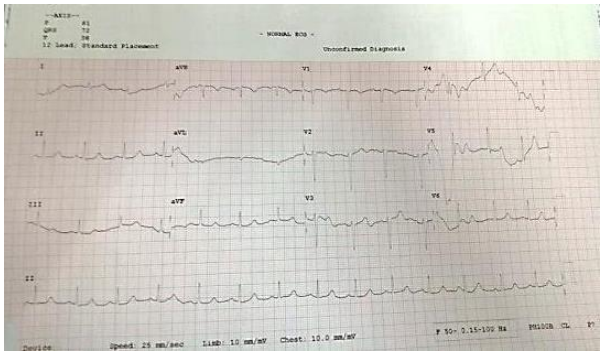
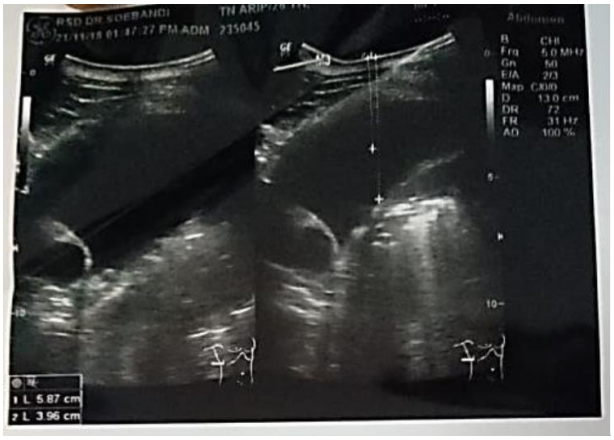


Figure 3. Normal ECG Result.

Table 1. Results of Laboratory Test.

HB	10.3 gr/dl
WBC	15.4 10^9/L
HCT	29.6 %
Thrombocyte	316 10^9/L
Albumin	3.1 gr/dl
Glucose	64 mg/dl
Natrium	110.6 mmol/L
Kalium	3.82 mmol/L
Chlorida	78.0 mmol/L
Calcium	1.87 mmol/L
Creatinin	16.0 mg/dl
BUN	135 mg/dl
Urea	288 mg/dl
Urid Acid	12.9 mg/dl
Urinalysis	
Color	Cloudy yellow
pH	5.0
Density	1.015
Protein	+1 (25 mg/dL)
Glucose	Normal
Leucosyte macro	Negative
Blood macro	Negative
Eritrocyte	2-5
Leucocyte	0-2
Epitel Squamous	0-2
Yeast	Positive

Result of thoracic marker USG showed that the intensity of echo fluid in both pleural cavity, (Figure 4). Abdominal USG results showed right kidney: normal size, echo cortex intensity increased, sinus cortex line unclear. Left kidney: normal size, echo cortex intensity increased, sinus cortex line unclear, visible stone in the middle pole size 0.87 cm. Emerging intensity of echo fluid in both abdominal cavity and bilateral thoracic cavity (Figure 5).



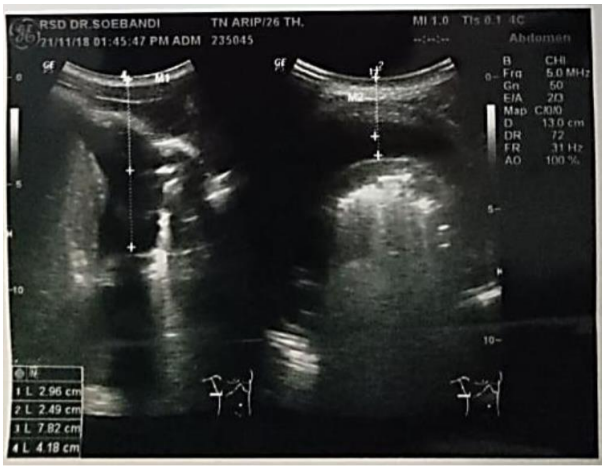
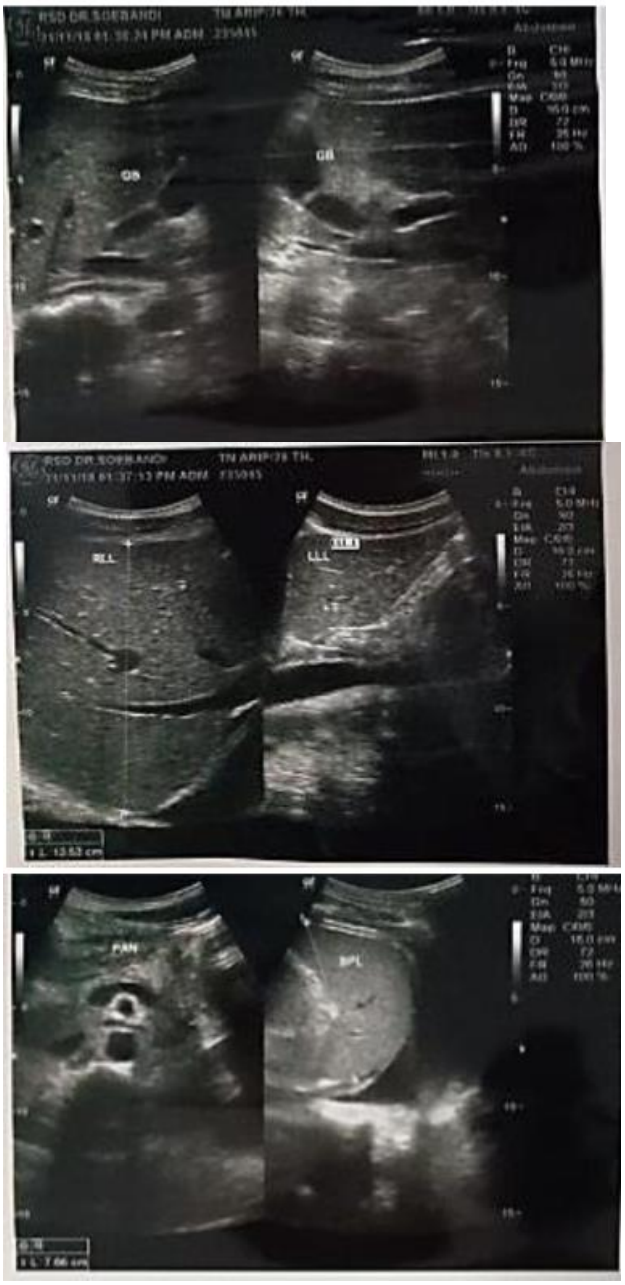


Figure 4. Results of Thoracic Marker USG



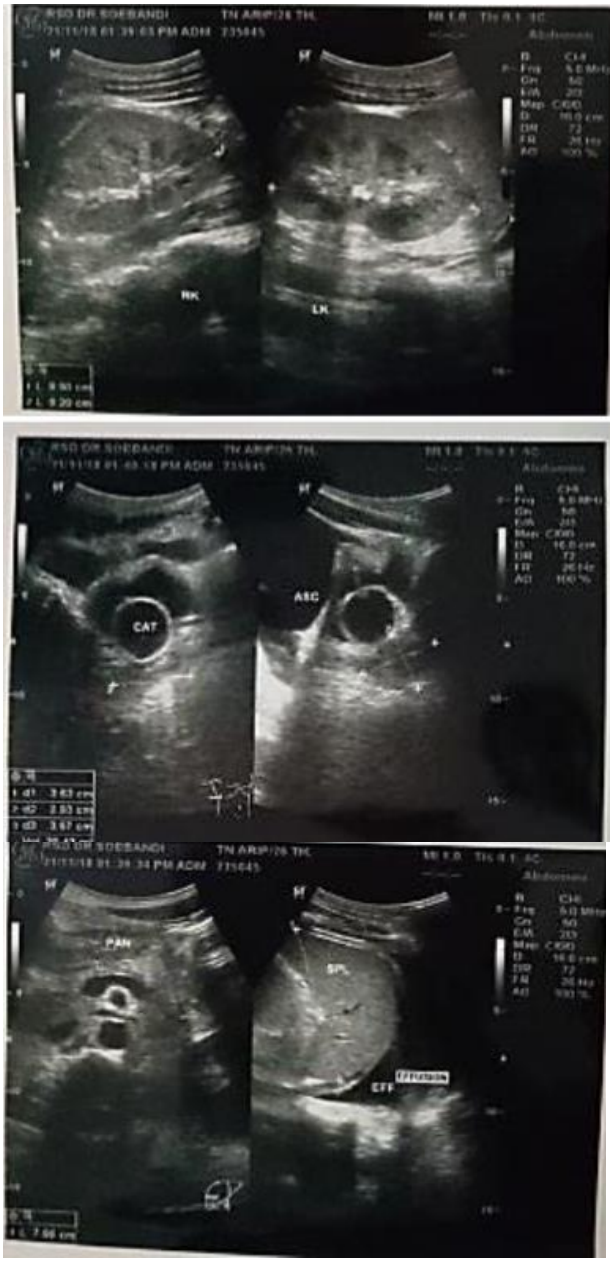


Figure 5. Results of Abdominal USG

Based on physical examination and workup results, patients diagnosed by kidney failure with pulmonary edema as a result of acute tubular necrosis accompanied by anterior uveitis, subconjunctival hemorrhage, and hematemesis as a result of electric injury with inappropriate initial treatment.

Patient Management

Based on patient's history and physical examination, management is given to improve general conditions with correction of hypoglycemia, hyponatremia, administration of antibiotics and ranitidine.

Severe shortness of breath due to uremia and bilateral pleural effusion are treated through hemodialysis. After 24 hours post hemodialysis, the results of renal function examination improved (Table 2), and pleural puncture was immediately in patients the results were obtained 150 mL of clear yellow color at right pleural puncture and no fluid was found at left pleural puncture.

Eye complaints are defined as uveitis anterior ocular sinistra (OS) and subconjunctival bleeding ocular dextra dan sinistra (ODS) due to electrical mechanical trauma. Management by ophthalmologist was given statrol ed 5x1 drops of OS, often ed 5x1 drops of OS, and methylprednisolone oral 3x4 mg for 7 days.

Wound management with debridement and reconstruction of the right hand with abdominal flap 3 days after pleural puncture.

Table 2. Post-hemodialysis Laboratory Results

Natrium	133.8 mmol/L
Kalium	3.60 mmol/L
Chloride	99.6 mmol/L
Calcium	2.65 mmol/L
Creatinin	6.2 mg/dl
BUN	67 mg/dl
Urea	144 mg/dl
Urid Acid	5.5 mg/dl

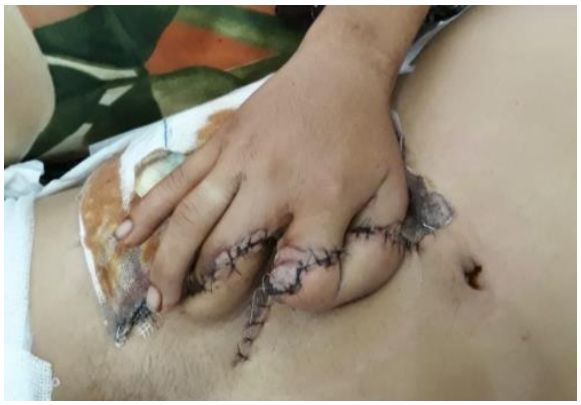


Figure 6. Post-abdominal Flap Surgery

Surgical actions on the entry wound include debridement, digit II phalang distal-media amputation, and defect closure with abdominalflap (Figure 6).

DISCUSSION

Electricity is the flow of electrons (outer particles negatively charged from an atom) through a conductor. The object that collects electrons become negative, and when electrons flow away from this object through a conductor, an electric current is created, measured in amperes. The power that causes electrons to flow is called voltage, and is measured in volts. Anything that blocks the flow of electrons through a conductor creates resistance, which is measured in ohms. Electric injury will occur when a person comes into contact with the current produced by a source. These sources can be man-made (for example, power lines in utility companies) or natural, such as lightning strikes.8 In this case, the patient is exposed to an electric shock from household appliances in the form of a fan, but an electric shock comes from a fan cable connected to the home electric current so that the current voltage is 220 V.

The severity and complications of electric injury depend on the amount of stress, resistance, type of current, current path and duration of contact. There are three main mechanisms for the occurrence of electric injury, they are: (1) electrical energy causes direct tissue damage, alters the potential of resting membrane cells, and generates muscle tetany; (2) conversion of electrical energy into heat energy, causing large tissue damage and coagulative necrosis; (3) mechanical injury with direct trauma resulting from falls or severe muscle contractions.[9]

This case is classified as electric injury low voltage (<1000) because the voltage is 220 V. Although low voltage, this electric injury can cause the patient to become unconscious, the appearance of tissue necrosis, myoglobinuria and hemoglobinuria.[10] In this case, the patient was unconscious and progressive tissue necrosis occurred in his right hand. The case examined by Buja et. Al stated that they also treated patients with amputation not only from the fingers but also on the forearm, which was caused by low voltage.[10]

Blood vessels, muscles, and nerves contain high levels of electrolytes and water so that resistance is low, and electricity conductors are the best compared to bone, fat, and skin. Hard skin areas are very good resistors, while moderate amounts of water or sweat on the skin surface can significantly reduce resistance.[9]

Electric current can flow through two types of currents, namely direct current (DC / direct current) or alternating current (AC / alternating current), where the flow of electrons changes direction in rhythmic mode. Alternating current is the most common type of electricity in homes and offices, standardized to a frequency of 60 cycles/second (Hz). The case of this patient's electric injury was a type of air conditioner because it happened at home. Alternating current voltage with the same voltage as DC is considered to be about 3 times more dangerous, because the cyclic flow of electrons causes the tetani muscle to extend the victim's exposure to the source. Tetani muscle occurs when fibers are stimulated at 40-110 Hz; standard electric current 60 Hz is in the range. If the source of contact is the hand, when there is a tetanic muscle contraction, contraction of the extremity flexor will cause the victim produce continuous prolonged contact with the source. Conversely, high DC voltage often causes large muscle contractions and causes the victim to bounce due to the short duration of contact with the source stream.[9]

In its flow through the body, the electric current always looks for the

shortest and fastest route to the exit point. There are several main lines of electric shock cases that are most common in the community, namely: (1) the first line is the electric current through the hand holding an electric conductor then the current flows to find the shortest route to the neutral conductor in the form of soil through the foot; (2) The second path is the obliq path, most likely the electric current passes through the heart organ which has its own electrical system, the electric current that passes through this organ then interferes with the heart's electrical process; (3) The third path occurs when the contralateral hand holds an electrical conductor, then the electric current from the hand will flow to the contralateral hand holding the conductor.[11]

In this case, a burn wound was found only at the source contact point (entry wound), but no ground contact points were found (exit wounds). Entrance and exit wounds do not always appear together, sometimes only entry wounds, sometimes also just wounds come out or both appear together.[12] Descriptive research conducted by Guntheti et. al stated from 62 patients, there were 13 (20.96%) cases where no entry or exit wounds were found, only 25 (40.32%) of the wounds were included, only 2 (3.23%) out cases and 22 (38.48%) cases.[13]

The pathophysiology of electrically induced ocular injuries is complex. Electrically induced ocular injuries have been associated with many pathological changes such as cataracts, macular edema, eyelid edema, epithelial corneal keratitis, uveitis, and pupillary abnormalities. Among other things, cataracts are the most common complication in which the lens is the most sensitive tissue to electricity and heat generated in the eye followed by low-resistance parts of the eye such as the retina and optic nerve. Whereas in the case of electric injury, the unilateral mechanism of uveitis (inflammation of the iris and ciliary body) after an electrical injury is unknown. Electric current may have spread only to the left eye as well, the heat generated by the flow of current through the eyes can cause a variety of cellular or interullular changes that may result in uveitis.[14]

Management of anterior uveitis in these patients is categorized as severe anterior uveitis based on symptoms and physical examination. The principle of handling is to maintain visual acuity, relieve ocular pain, eliminate ocular inflammation, and prevent synechiae formation.[15]

Most subconjunctival bleeding occurs spontaneously. However, this bleeding can also occur spontaneously due to trauma, postoperative bleeding, systemic diseases such as hypertension, diabetes and clotting factor disorders. Subconjunctival bleeding is bright red or dark red sclera. The inflammatory process can occur, but is usually not severe. Bleeding can also expand in the first 24 hours, but afterwards it starts to decrease due to the absorption process. The absorption process takes place within 1-2 weeks.

Gastrointestinal complication can also occur in electric injury. This is associated with a sequel's "stress response" which increases the secretion of stomach acid and peptic acid. The pathogenesis of "curling's ulcers" and stress ulcer is a change in the quality and quantity of mucosubstantion stomach, loss of integrity of mucous barrier, bile acid reflux and digestive enzymes, acid hypersecretion, hypoproteinemia or negative nitrogen balance, mucosal ischemia during opening of submucosal arteriovenous shunts, local vasoconstriction or the development of microvascular thrombus after intravascular coagulopathy. Gastrointestinal lesion can occur within 5 hours after the incident. Within 72 hours, many cases develop gastroduodenal

ulcerations which result in large bleeding, or perforation.[16]

Initial administration of oral or enteral food that starts within 6 hours after the incident is an effective supplement in preventing stress-induced ulceration. Early feeding reduces the incidence and severity of bacterial translocation by maintaining the integrity of the mucosal barrier. The use of antacids, and H2 receptor blockers has become an established practice in an effort to prevent bleeding. H2 receptor blockers, for example Ranitidine, administered at a dose of 150 mg twice a day for about 3-6 weeks, is a normal practice today, with the idea of reducing acid secretion by blocking histamine H2 receptors on parietal cells of the gastric mucosa.[16]

Electric shock can also cause disruption of cell membrane permeability due to electroconformational denaturation of proteins (macromolecules). When electrified, it means that there is direct contact between the current source and the conductor (body surface tissue), and there is an electron displacement as well as the process of transferring ions in the solution. At that time, the process of electrolysis and exothermal reaction, electrochemistry, followed by changes in pH, oxygen concentration, and release of toxic substances into the surrounding tissue. Electric current through the cell layers shows a phenomenon called the electroporation process. The process takes place in the cell membrane; Negatively charged membrane proteins denaturate. Protein denaturation especially occurs around the location of cell lines and gates (the location of sodium pumps, calcium pumps, and potassium channels). These cell membrane proteins lose their three-dimensional structure, experience distortion or form intramembrane pores so that the intracellular components are easily mixed with extracellular components; the process takes place so fast.[17] The amount of intracellular fluid to extracellular will form edema.

Complaints of shortness of breath in patients due to uremia and the presence of pleural effusion in both lungs of patient where the right lung has more fluid. Right pleural puncture results obtained clear yellow liquid and included the type of transudate based on the light's criteria. Pleural effusion is influenced by a balance between fluid production, fluid absorbance, and several defense forces such as plasma osmolality, hydrostatic pressure, venous pressure, and permeability of capillary walls.18 Pleural effusion in this case can be due to increased permeability of the capillary wall due to the electroporation process and due to the process of rhabdomyolysis which causes Acute Tubular Necrosis (ATN).

Patients in this case experience acute renal failure or commonly known today as Acute Kidney Injury (AKI) which occurs after burns are mostly caused by reduced cardiac output, which is mainly caused by fluid loss. This is usually caused by delayed or inadequate fluid resuscitation but can also be caused by muscle damage or substantial hemolysis. Reducing urine output even though adequate fluid administration is usually the first sign of AKI. This will be followed by an increase in serum creatinine and urea concentrations.[19] AKI after electric injury which increases creatinine and potassium levels is an indication for hemodialysis. Blood urea is not a useful independent indicator because it increases in non-kidney conditions such as dehydration and high-protein diets.[20]

Oliguria in this case is caused by the presence of ATN which occurs due to the process of rhabdomyolysis into myoglobinuria on electric injury causing fluid accumulation throughout the body in the form of anarchic edema and bilateral pleural effusion. This edema and bilateral pleural effusion occurs because the

kidneys are unable to function as excretory organs. While rhabdomyolysis is a syndrome in which pain occurs and necrosis of skeletal muscle by releasing muscle enzymes into the circulation.

Rhabdomyolysis can result in elevated levels of myoglobulin, creatinine phosphokinase (CK), lactatdehydrogenase (LDH). The symptoms range from moderate increases in muscle enzymes to life- threatening diseases associated with extreme increases in enzymes, electrolyte imbalances (especially hyperkalemia), and continuing into AKI. The last common pathway for injury to rhabdomyolysis is an increase in free intracellular ionized cytoplasm and mitochondrial calcium. This may be caused by reduced adenosine triphosphate (ATP), cellular energy sources, and/or by direct injury and rupture of the plasma membrane. ATP depletion causes pump dysfunction Na/K-ATPase and Ca 2+ ATPase which are important to maintain the integrity of muscle cells, resulting in the release of muscle enzymes in the circulation. The mechanism of myoglobin which causes damaged glomerular filtration is unknown. Previous studies have shown that renal vasoconstriction, direct tubular injury, indirect tubular injury (ischemic), and intraluminal blockade by myoglobin play a role.[21]

The importance of detecting ATN signs early, is to prevent the continuation of AKI. ATN signs can be found through clinical or laboratory. Clinical ATN can be accompanied by oliguria or without oliguria. So laboratory results are needed to establish the diagnosis of ATN in the form of urinary osmolality <400 mOsm / Kg, amount of urine sodium> 40 mEq / L, creatinine ratio <20, percentage of sodium> 2, and in urine sediments found renal, granular and muddy tubular epithelium brown granular casts. Management of ATN includes a hemodynamic improvement in the form of preventing hypotension which can cause disruption of the glomerular filtration rate due to the mechanism of renal arterial vasoconstriction. If the weight of the treatment is hemodialysis.[22]

Hematuria in the case of electric injury is the result of denatured protein (pigmenturia) in the form of free hemoglobin released from red blood cells degraded by heat conjugated by haptoglobin and transferred to the liver. When large amounts of free hemoglobin are produced in electric injury, non-conjugated free hemoglobin passes through the glomerulus and is excreted in the urine causing hematuria. This can cause degenerative changes in tubular cells, occlusion of renal tubules by formation of hemoglobin casts, and finally AKI, especially if combined with dehydration, acidosis, shock, or endotoxemia. Hemoglobin is freely absorbed by degraded tubular epithelium to globin and hem.[20]

Progressive ischemia in electric injury is also a serious complication. Thrombus formation in arterioles, narrowing of vessels decreases blood flow, and tissue necrosis can occur.[23] This is a factor that contributes to amputation decisions. Therefore, in this case, amputation is done in the part that has necrosis. The main goal in handling electrical injuries on the hand is to restore the function of the hand to the maximum. Plastic surgery procedures, such as skin grafts or artificial dermis, are useful for first-time surgery, and regional or free flaps are useful if the injury is severe. In case of electric injury with high tension, groin, and abdominal flaps are the most commonly used flaps. In this case, abdominal flaps are performed on low-voltage electrical injuries.[23]

CONCLUSION

Electric injury has complex

complications, so that appropriate treatment to prevent complications from occurring has a very important role. This case explains the effect of acute tubular necrosis (ATN) which continues to be the acute kidney injury (AKI). The progressive ischemic effect of electric injury accelerates rhabdomyolysis. Detecting possible complications on electric injury and good follow-up of patients will minimize the occurrence of AKI in patients with electric injury.

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CONFLICT OF INTEREST

There is no conflict of interest in this study.

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The authors declare they have no financial for any institution.

AUTHORS CONTRIBUTION

All authors contributed to the study design, data curation, methodology, manuscript wrote and revision, administration.

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