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lumen through the multiple layers of platelet membranes. The current causes the membrane protein losses its three dimensional property, distorts, and forms intramembrane pores. The process culminates in irreversible cell death due to mixed intracellular and extracellular components[2].

Statistically, platelet count difference were observed between intervention groups day-7, day-10, and day-14 with the intervention group day-3 only but not to control group. This finding is consistent to return of platelet count within normal limit after low platelet count on day-3. Previous studies showed that platelet count return within normal limit in 10-14 days with subsequent variable-length of transient thrombocytosis[6].

In this study, normal platelet count was reached on day-7. Theoritically, on sixth day post electric injury, normal circulating platelet is followed by mild thrombocytosis. Platelet "refill" depends to megakariocyte sitoplasm fragmentation. Low platelet count stimulates megakariocyte forming from small acetylcholinesterase positive (SAChE +) cells. On sixth day post electric injury, production rate from megakariocyte is capable to maintain normal platelet count in circulation[11].

Increase in platelet count in this study was not subsequently followed by a period of thrombocytosis, seen from normal platelet count in day-7, day-10, and day-14. This finding is consistent in several previous study in severe burn. Those studies conclude that in patients with severe thrombocytopenia on third to four days post trauma experienced no thrombocytosis[8]. In this study, due to specimen collecting was not continuous, we can't accurately follow platelet count day by day, especially on day-3 and day-4. One study mentioned a period of thrombocytosis rarely occurs in patients with severe thrombocytopenia and related to incidence of sepsis in burn patients[7,8]. A further study is encouraged to investigate any relation between platelet count alteration in acute and subacute phase of burn and incidence of sepsis in electrically injured rats.

First limitation of this study was no platelet count performed in the end of acute phase. This makes the length of thrombocytopenia period impossible to be concluded. Another limitation is direct counting of platelet through blood smear test. This method requires skillful personel in smearing blood sample. Subjectivity is relative high and precision is hard to achieve. Common pitfalls are inspecting difference between platelet and stain particle, inequality in platelet distribution, and method of reading the fields[12]. We suggest further study to use automatic animal hematology analyzer to minimize error.

Conclusion

We observed platelet count difference in acute phase of electric burn injury and no difference in platelet count difference in subacute phase of electric burn injury.

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Case Report: Risk of Electric Injury on Delayed Initial Treatment

Ulfa Elfiah
Dissa Yulianita Suryani

Abstract: 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. 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.

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, 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 are 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 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

Disclosure: *The authors herely declare they have no financial interest in the information discussed in this article*

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 PRESENTATION

A 26 years old male came to Dr. Soebandi General Hospital 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. 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 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 dim sound in the basal right and left lung, auscultation showed a decreased vesicular sound and there was crackles in the basal right and 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.

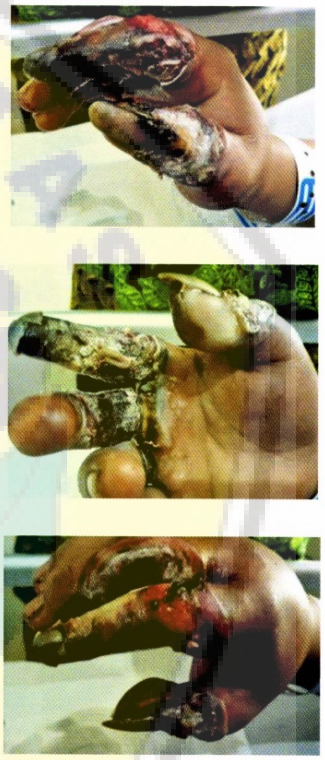

Figure 1. Clinical photograph of burns entered due to electric injury.

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.

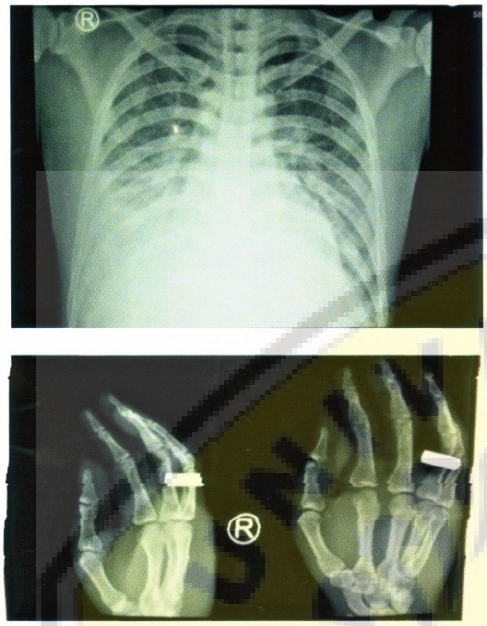

Figure 2. Thorax x-ray (2A) and manus dextra (2B)

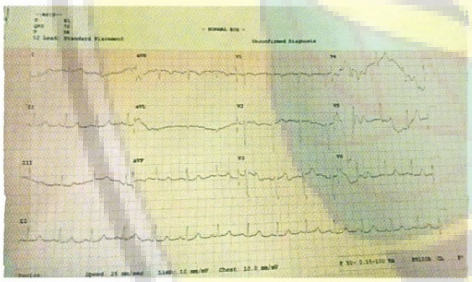

Figure 3. Normal ECG result.

Investigation was carried out by laboratory test, ECG, 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).

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).

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, 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), 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 (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