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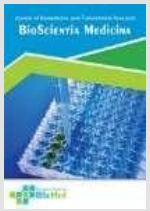

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Burn Wound and Traumatic Inhalation due to Marapi Volcano Eruption

Deddy Saputra[1], Arief Gusman[1*], Melati Purnama Sari[1]

[1]Department of Plastic Surgery, Faculty of Medicine, Universitas Andalas, Padang, Indonesia

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***Corresponding author:**
Arief Gusman

E-mail address:
arief.gusman@outlook.com

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A B S T R A C T

Background. Burns can be caused by various etiologies, including heat and chemicals from volcanic eruptions. Mount Marapi was a commonly visited hiking site prior to its eruption on December 3th 2023. **Case Presentation.** This case reports a 19 year old woman with severe full thickness burns, covering 40.5% of total body surface area (TBSA) with inhalation trauma caused by inhaled volcanic ash from the Mount Marapi's eruption. Airway assessment was done through bronchoscopy examination.While being treated in the burn unit, the patient's overall condition deteriorated, so the patient was moved to the intensive care unit (ICU). Severe burns usually can cause circulatory disruption and trigger systemic inflammatory responses. Meanwhile, inhalation injury and sepsis also contributes to a higher risk of death in burn patients. Burns with inhalation trauma require specific treatment procedures. **Conclusion.** This study summarizes the experience of treating victim of the Mount Marapi eruption and multidisciplinary approach to achieve optimal patient care.

1. Introduction

Burn injuries can be caused by various etiologies, but the majorities are associated with hot liquid, fire or electrical sources. Each causes have its different physiological dan pathophysiological responses.[1] The World Health Orgaization (WHO) reported that 11 million burn injuries occur annually worlwide, accounting for an estimated 180.000 deaths every year, with around 90% happens in low- and middle-income countries.[2] One of the casualties of these burn injury are from volcanic eruptions, which often results in a large mass victims.[3]

The Mount Marapi, located in the West Sumatra, has been a site of several explosive eruptions over the years. It was also a common tourist attraction for hiking prior to its eruption in December 3th 2023. The fatalities in this eruption involved 23 hikers who was found dead, 11 person was injured, with 2 of them was sent to M. Djamil General Hospital, requiring advanced medical care.[4] This case report presented one of the victim, a 19 years old female with severe burns and traumatic inhalation. Thermal and chemical injuries from volcano eruption lead not only to skin necrosis, but also traumatic inhalation, deep tissue damage, metabolic changes and multiple organ failures.[5] Spesific cause of burn injury determines different treatment approach. This paper summarises the experience of treating volcano eruption victims, and the multidiciplinary approach to optimalized patient care.[6,7]

2. Case Presentation

A 19-year-old woman came to the Emergency Room at M. Djamil Hospital with severe full-thickness burns, with an area of 40.5% on the face, both upper legs, and both lower legs, airway obstruction, and compartment syndrome 24 hours after the injury. Due to limited burn units at the previous hospital, the patient was referred to our hospital. Before the patient was referred, the patient had received resuscitation and stabilization.

When admitted to our emergency room, the patient was conscious and cooperative, with blood pressure 116/65 mmHg, mean arterial pressure 82, heart rate 143x/minute and oxygen saturation 100%. When in the emergency room, the general surgeon immediately consulted a plastic surgeon and planned for emergency debridement after stable hemodynamics. The patient was admitted and treated in the Burns Unit for further care.

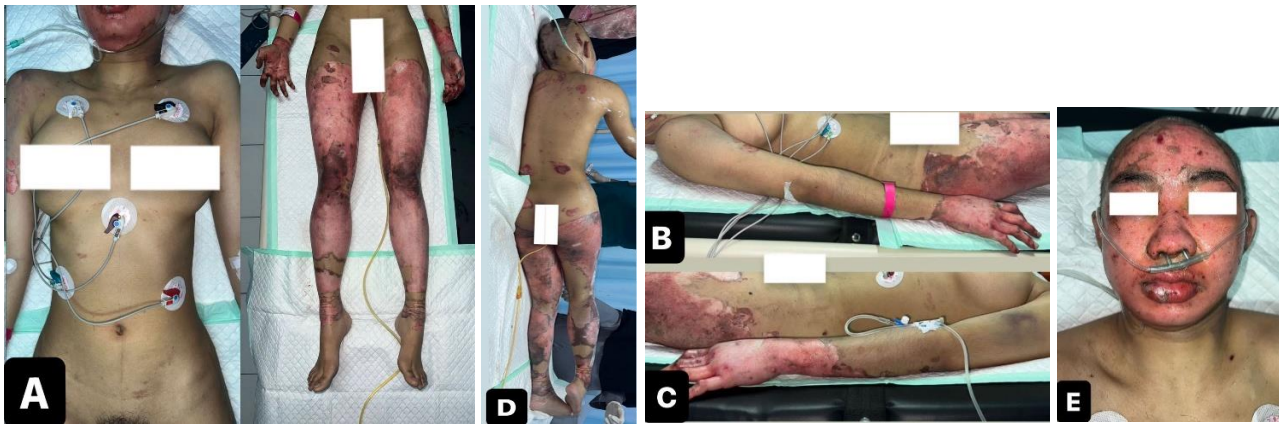

Figure 1. (A) Patient initial condition at Dr. M. Djamil General Hospital on the first day, front view, (B) right side view, (C) left side view, (D) back view, and (E) the face.

From the initial laboratory examination results in the burn unit, the leukocyte result was 13,820/mm^3, hypoalbuminemia and followed by respiratory alkalosis. The patient was given antibiotic therapy, hydration, and education on extremity elevation and coughing. In our unit, patients are bathed from head to toe, including the burned area. On the second day of treatment in the burn unit, the patient complained of chest palpitations after giving the second antibiotic injection with hemodynamic blood pressure 92/78 mmHg, heart rate 240x/minute and an electrocardiography examination and blood laboratory examination were carried out. The patient was immediately consulted to the heart and blood vessels department, diagnosed with stable supraventricular tachycardia and the patient received diltiazem injections.

The patient was transferred to the operating room on the fourth day of treatment. Debridement and escharotomy were performed after the bronchoscopy was performed. The results of bronchoscopy were grade 1 inhalation trauma with redness and edema found in the right bronchus. No soot was found on bronchoscopy examination, and the lavage fluid was examined at the anatomical pathology department. After that, the plastic surgeon performed debridement and escharotomy. Necrotic tissue was found on the fingertips, but not all of the necrotic tissue could be cleaned thoroughly because the operation time was quite long, so it was carried out for further debridement. After the surgical process, the patient is extubated and sent to the burn unit.

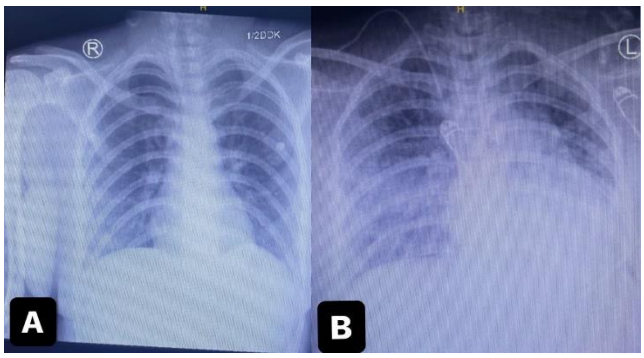

Figure 2. CXR on the (A) first day and (B) fourteenth day of hospitalization.

On the fifth day, the patient complained of chest palpitations for the second time, with unstable supraventricular tachycardia and cardioversion was performed, with the result that the unstable SVT turned into sinus tachycardia and the use of diltiazem was stopped. On the seventh day, the patient complained of shortness of breath that had never been felt before, the patient required additional effort to exhale. Complaints of increasing shortness of breath were felt to increase, so a blood gas analysis was carried out. Then, the patient was consulted to an intensive care doctor for airway and respiratory management and monitoring in the Intensive Care Unit (ICU). In the ICU, intubation was performed with hemodynamic blood pressure 86/56 mmHg, mean arterial pressure 63, heart rate 135x/minute and SpO_2 64%.

The initial laboratory results in the ICU after the intubation showed that the Hb value was 8.8, leukocytes 7,880/mm3 platelets 1150,000 and albumin 1.6. The patient was given a blood transfusion of 1 unit of PRC and 25% albumin. In addition to fluid and blood resuscitation, patients are also given inotropes to achieve target mean arterial pressure. The patient also had impaired renal function, urea 73 and creatinine 2.3, and severe metabolic acidosis with pH 7.24, PCO_2 32, PaO_2 241, BE -12.6, HCO_{3-} 13.7, and SaO_2 100%. The patient is given fluid hydration and meylon correction. There was an electrolyte imbalance with sodium 129 and potassium 5.3. The patient is given sodium and potassium correction. Coagulopathy was also found, showing INR 1.57 APTT 48, D-dimer >10,000. Procalcitonin above 100. Sputum culture received with pseudomonas aeruginosa. Blood pressure in the ICU after resuscitation reached the target. Hemodynamic blood pressure 104/62 mmHg, with mean arterial pressure 77, heart rate 126x/minute, and SpO_2 100% with total urine output 0.42 cc/kg/hour.

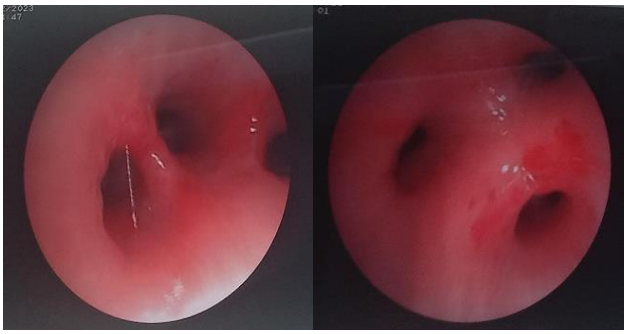
Figure 3. Bronchoscopy image showed inflammation with hyperemic and edema of the bronchial mucosa.

On the third day of ICU treatment, there was a significant change in the results of the leukocyte examination, 19,020 and procalcitonin was still above 100, so antibiotic therapy was changed. The patient showed compensated metabolic acidosis. Ureum value 109, creatinine 1.7. Then, the patient was consulted to the hypertension kidney department and diagnosed with improvement in kidney disorders. The patient was also diverted to have a tracheostomy placed due to indications of long-term care. When the tracheostomy was performed, stenosis was found at the end of the trachea. On the fourth day of ICU, there was no improvement in the platelet value even though continuous platelet transfusion and platelet apheresis had been carried out; the thrombocyte value was 45,000, then the patient was consulted to the medical oncology hematology department, diagnosed with overt DIC. Additionally, the nasogastric tube showed a blackish residue, so steroid use was discontinued. While being treated in the ICU, the patient received antibiotic therapy of cefoperazone sulbactam 3 x 2 grams and levofloxacin 1 x 750 mg, which was then replaced with ceftazidime 3 x 2 grams, amikacin 1 x 1100 mg, omeprazole drip 120 mg/24 hours continuously, fartisone 2 x 100 mg, resfar 1 x 5000 IU. On the seventh day of ICU treatment (14 days after injury), the patient's condition worsened. The patient's condition was very unstable even though he had been given norepinephrine, dobutamine and vasopressin. The patient died from the probable cause of death being septic shock and multiple organ failure.

3. Discussion

Thermal injury is the main cause of 90% burns incidents, where the depth of injury depends on the heat temperature and contact duration. Volcanic eruptions can cause thermal injuries due to direct contact with fire, radiant heat or chemicals from volcanic ash, resulting in deep burns, partial or full thickness burns.[8] Burns can have both local and systemic impacts, which occur if the burn involves more than 30% of the total body surface area (TBSA). In this patient, burns of 40.5% TBSA were found, so this patient is at high risk of undergoing circulatory disorders and plasma extravasation which causes edema, resulting in increased systemic vascular resistance and decreased peripheral blood flow.[9]

Severe burns are known to cause a hypermetabolic state, which involve metabolic, hormonal, and inflammatory dysregulation. This hypermetabolic response has detrimental effects on the structure and function of major organs, such as heart, liver, skeletal muscle and skin. This condition is triggered by a persistent increase in catecholamines, cortisol, glucagon and dopamine. The hypermetabolic state and lack of oxygen simultaneously have the potential to prolong the anaerobic glycolysis process, resulting in the patient falling into a condition of lactic acidosis.[8,10] In addition, increases in catecholamines and other catabolic compounds, such as cortisol and glucagon, stimulate hyperdynamic cardiovascular responses. This is caused by the damage in mitochondrial morphology and structure which reduces cardiac mitochondrial replication in heart cells, disturbing transport chain and causing ATP deficiency in cardiac tissue as responses to burns. This condition causes a continuous increase in the need for oxygen for cardiac cells which cannot be compensated for by burn patients, resulting in further damage to myocytes which causes heart dysfunction, in the short and even long term.[27] Decreased heart function, lack of fluids due to increased evaporation of water, also major tissue damage from the burn itself, all together cause an increase in heart rate and decreased contractility. Williams et al. reported that the heart rate of burn patients increased by around 170% of the normal value at initial admission.[11] Arrhythmias in burn patients can be influenced by several important factors other than catecholamines, including significant changes in fluid status and electrolyte disturbances related to resuscitation.[12]

Cardiovascular disorders and endocrine regulation can also impact urinary dysfunction. In burn patients, there is a decrease in renal blood flow and glomerular filtration rate (GFR) due to hypovolemia and decreased cardiac output. Continuous kidney disorders can cause acute tubular necrosis (ATN), kidney failure or

death.[8] Folkestad et al. reported that acute kidney injury (AKI) occurred in approximately 38% of burn patients treated in the ICU. AKI in burn patients is a marker of ongoing multiple organ damage and associated with increased mortality rates and length of stay in burn patients.[13]

Burn wounds are divided into three zones, the coagulation zone, stasis zone and hyperemic zone. The coagulation zone is surrounded by a stasis zone that experiences vascular transudate, inflammation and low perfusion. The stasis zone itself can have the chance to improve or even become necrotic in the first few days if the vascular perfusion is poor, resulting in more necrotic tissue, hence a wider and deeper burn area than before.[14] These patients are going through microvascular disorders, significant inflammatory responses and cardiovascular disorders which caused perfusion to become increasingly impaired. This not only causes worsening of the burn area which makes larger necrotic tissues, but also worsen the patient's overall condition systemically.[14]

Inflammation from extensive tissue damage can also be a challenge considering the risk of systemic inflammatory response syndrome (SIRS) and multiple organ failure.[15-20] Meanwhile, the inflammatory response also have important role in the healing process, triggering a cascade of cytokines and growth factors that protect against the risk of infection.[21] Inflammatory factors involved cytokines, including IL-6, IL-1β, tumor necrosis factor α (TNF-α), interferon-γ , transforming growth factor-β, platelet-activating factor (PAF) and IL-8. These compounds are produced by various types of cells, but the ones that play the most roles are macrophages and monocytes that is found in areas of inflammation.[22-24]

Sepsis plays a major role on morbidity and mortality of burn patients. Every study regarding the management of burn wounds always emphasizes that maximum efforts must be made to control the rate of sepsis in burn patients, because around 73-85% of all deaths that occur in acute conditions are caused by sepsis.[15] Any burn wound infection should be treated proactively with systemic antibiotic therapy, but treatment is becoming increasingly difficult and challenging due to drug resistance of many bacterial strains.[8]

Inhalation trauma is also a significant predictor of prognosis in burn patients. Volcanic ash contains heterogeneous minerals consisting of a mixture of amorphous and crystalline minerals, namely silica (SiO_2).[25] Pathophysiological changes in inhalation trauma are mediated by microvascular changes that caused by heat and chemical injury. Heat causes protein denaturation in bronchial mucosa which stimulates complement activation, thereby releasing histamine which leads to edema.[19] Damage to the lower airways, from the trachea to the lung parenchyma, is more likely to be caused by inhaled chemicals, causing chemical irritation that stimulates the release of neuropeptides from the tracheobronchial tissue. This neuropeptide induces an inflammatory cascade consisting of bronchoconstriction, increased vascular permeability, formation of reactive oxygen species (ROS) and vasodilation. [26]

Bronchoscopy is the gold standard procedure for diagnosing upper airway injuries, with the most frequent findings being hyperemia, edema, presence of soot, and mucosal erosion.[19] Pulmonary complications, particularly pneumonia, have been found to occur more frequently in patients with inhalation injury than in burn patients without airway injury, especially when combined with large burn areas.[15] Monteiro et al. reported that the agent most frequently isolated in burn patients with inhalation injury was *Pseudomonas aeruginosa*, both from sputum and blood sampling.[17] A systematic review by Galeiras et al. reported that in-hospital mortality due to inhalation injury in burn patients had an odds ratio of 3.2.[18] To this day, there is no consensus among leading burn centers regarding optimal mechanical ventilation settings for these patients. Supportive care is still an alternative therapy for inhalation trauma patients.[19,27] During treatment in the ICU, the patient's condition continued to decline. Indicated by a decrease in hemodynamic condition even though it has been on inotropes, vasopressin and epinephrine

support. This condition is also accompanied by the high procalcitonin which indicates that there was ongoing high incidence of inflammation and infection in patients.[28]

4. Conclusion

Management of severe burn patients with inhalation trauma, particularly which caused by volcanic eruptions, is complex and has its own challenges. Infection and systemic inflammation controls during hospitalization should always be a top priority to reduce the risk of mortality in these patients. Optimal patient care must be attempted through approach from multidisciplinary perspectives.

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