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Management of Sepsis Patient with Multiple Trauma: A Case Report

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ABSTRACT

Background: In critically ill patient, source control surgery is one of the significant determinants of patient treatment and makes it successful. This case report was aimed to describe the management of sepsis patient with multiple trauma. **Case presentation:** A 20-year-old woman presented with blunt trauma in the chest, abdomen, and right superior and inferior extremities. After the surgical procedure, the patient was treated in the ward with skin traction and simple wound care; a week later, the wound became worse, abscess inside, necrotic tissue and smelled terrible, and the woman had fever and jaundice somnolence, and other clinical symptoms manifested and became sepsis. She underwent twice a debridement surgery for a nasty wound. After that, she was treated in intensive care. A multidisciplinary team was required to treat the patient. **Conclusion:** Surgical intervention, wound cleaning, and appropriate and adequate antibiotics are also given to prevent worsening sepsis in the future.

1. Introduction

Multiple trauma is a high-risk incidence of sepsis because of necrotic wounds, bone exposure, hidden hematoma. Sepsis is a well-documented risk factor for poor outcomes following severe traumatic injury. In addition to a high injury severity score (ISS), many operations, and an extended stay in an intensive care unit or hospital, other risk factors for post-traumatic sepsis include massive transfusions of packed red blood cells and a high level of ISS. Because prolonged hospital stay, and repeated operation procedures, increase multiple drug-resistant microorganisms. The multidisciplinary interplay of specialists and intensivists is needed. This case report was aimed to describe the management of sepsis patients with multiple trauma.

2. Case Presentation

A 20-year-old woman was involved in a traffic accident (she rode a motorbike and was hit and run over by a car). She was hospitalized in the ward. She underwent debridement surgery on the Upper and Lower right extremity and right leg skin traction. A week later, she underwent twice debridement on different days. She had broken right humerus, radius, ulna, and femur in three different places, right tibia, fibula fracture, open wound, and bone exposed in the right thigh. The wound was terrible; it was a lot of pus, abscess, necrotic tissue, and an intense scene. We

found a small hematoma on the chest, jaundice. Nevertheless, there was no damage to the abdomen. After surgery, she was hospitalized in ICU because we needed to know the patient intensively and multidisciplinary specialists.

General examination show the patient was alert, had a fever (38ºC), and hemodynamic is stable. Respiratory rate was 23 times per minute. Patient was given oxygenation with NRM 8 L/minutes. The abdomen was no distention. Laboratory examination show total bilirubin level was 20.3; direct bilirubin level was 13.6; indirect bilirubin level was 6.7; procalcitonin level was 7.9. The patient was treated with antibiotics meropenem (3x1 gram) and levofloxacin (1x 750) via drip intravenous.

The patient was consulted to thoracic surgeon, digestive surgeon, plastic surgeon, cardiologist, and orthopedics. The consultation result from thoracic surgeon stated there was not something emergency. Digestive surgeon stated there was biller obstruction in MRI results and they planned ERCP (endoscopic retrograde cholangiopancreatography). The digestive surgeon postponed the ERCP procedure on the patient because the direct bilirubin decreased from 18 to 4 (different days in several examinations). The echocardiography examination from cardiologist show mild mitral valve regurgitation, mild tricuspid valve regurgitation, mild pulmonary valve regurgitation, minimal pericardial effusion and ejection fraction 61.

The orthopaedic doctor performed OREF (open reductional external fixation) from the femur to the tibia, then the plastic surgeon performed wound management by using a tool in the form of NPWT (negative pressure wound therapy). After the operation was completed, the patient was transferred to the intensive care unit to receive more extensive medical attention.

When the patient entered the ICU, the patient's condition was alert, blood pressure (140 /64 mmHg, MAP 98, HR: 125), NRM 10 l / min. Postoperatively, the patient was still bleeding about 1000 cc, and then the patient was given two units of blood resuscitation. After that, hemodynamics was still not stable; blood pressure (121/80 mmHg, MAP 94, HR 123) performed four units of crystalloid resuscitation and two units of colloid, the hemodynamics began to decrease. Blood pressure (110/58 mmHg, MAP 77, HR 133) and on physical examination, pale conjunctiva from routine blood examination found HB 8.5; the patient was given 1 unit of blood resuscitation, NPWT was stopped, and pressure bandages were applied.

After the action was carried out, the hemodynamic condition was still unstable and decreased; blood pressure (92/55 mmHg, MAP 88, HR 141) during a physical examination, there was bleeding in the proximal femur area, which was then applied with a pressure bandage to stop the bleeding and the patient was given 300 ccs of 0.9% NaCl resuscitation. After resuscitation, hemodynamics began to stabilize, but breathing problems and the patient began to appear restless and short of breath. Monitor vital signs, blood pressure (102/61mmHg), MAP 76, HR 144, RR: 30, PaO2: 80 mmHg, temperature: 37,5ºC. On physical examination, crackles were found in both lung fields and when a chest X-ray was performed. The intensivists gave directions to the doctor, who was tasked with intubating immediately so that the patient's O_2 needs could be met. The patient received paracetamol 500 mg, gelofusin 250 cc/infusion pump and fentanyl 100 mg, roculax 30 mg.

The patient is on a ventilator (Pins 21, Pasb 11, PEEP 8, FiO2 80%, RR: 14 I: E = 1:1,6). She got two ampoules of furosemide, fluid was not given, then urine output got 3.150 cc fluid balance -1.550cc, then the fluid was given back gradually due to CVP -2. The next day, 1 unit of blood was transfused when the blood came in, the fluid was temporarily stopped after the transfusion was complete, and the fluid was given back restrictively; the CVP was maintained and monitored until it reached 7. The next day, the ventilator was weaned, and a chest X-ray was taken to review its progress; after being checked, there were no signs of pulmonary oedema in the two lung fields. Then the patient was extubated and given a rebreathing mask of 8 L/minutes. The oxygen was lowered gradually then checked on physical examination there is no sound ronchi. Antibiotics were still given until the fourteenth day according to culture and after re-checking for

procalcitonin the results improved. The patient two days later was reviewed for the condition of the wound, the action was carried out in the operating room. Before being transferred to a private room, a patient is taken back to the intensive care unit (ICU) for further wound care.

3. Discussion

Our patient with multiple trauma, risk of trauma in long term period and need frequent surgery. For the lower right extremity high risk of infection is life-threatening for this patient. In ICU patients has comorbid (tricuspid valve regurgitation, mitral valve regurgitation, pulmonary valve regurgitation) who gave massive transfusion and fluid at the same time. It became a hazard for this patient with heart congenital comorbid.

This patient also has severe sepsis, when the patient was ongoing bleeding. Then, the patient was consulted plastic surgeon. They decides to perform NPWT and wound compression, which temporarily stops the bleeding. However, the patient rebleeds, and as a result of the bleeding proximal femur and wound compression, the bleeding is stopped. After the bleeding ceased, the hemodynamics stabilized, and we took care of the hemodynamics. When hemodynamics deteriorated to 80/40 mmHg, HR 150 x/minutes, and we were unable to administer vasopressors, we administered inotropics and resuscitated the patient with PRC and FFP.

The patient developed shortness of breath, increased RR, but saturation remained 90-100, and we took a thorax photo. The outcome was pulmonary edema, which we treated with two ampoules of furosemide to restore negative balance. According to an analysis of cause-specific mortality, this was mostly owing to a decrease in MODS and ARDS-related death.[1] On the other hand, the death toll from brain injuries has grown. It's important to keep in mind that overall mortality is on the decline, so these observed changes should be seen in that light.[1] Before the turn of the twentieth century, organ failure was a more common cause of mortality than brain injury.[1] Many factors, both before and after a person went to the hospital, are likely to have played a role in the decline in mortality over the last few decades. Advances in diagnostic instruments, resuscitation techniques, as well as peri-operative and surgical procedures, have all contributed to the decline in the number of people who die as a result of road traffic accidents.[2]

In addition, several advancements in trauma care, such as the prevention and treatment of MODS/ARDS/sepsis, may account for the observed shift toward a higher proportion of brain injury-related deaths. According to Nast-Kolb et al, MODS-related mortality decreased as a result of improved trauma management and intensive care unit care.[3] They emphasized the necessity of numerous specific, significant modifications, including volume resuscitation, mechanical ventilation with airway pressure limiting, damage control treatment, and early enteral feeding are some of the options available.[4]

Interestingly, the fatality rate in the intensive care unit due to hemorrhage has risen over time. According to a major review, when the entire trajectory from pre-hospital to intensive care unit was followed, the risk of exsanguination-related death decreased over time (about 20 percent in 20 years).[5] The authors hypothesized that improved bleeding management and implementation of ATLS resulted in a reduction in mortality within 60 minutes of admission. It was discovered that by providing 24-hour access to an onsite CT scanner and implementing the damage-control strategy, it was possible to significantly reduce the risk of exsanguination shortly after hospital admission.[5]

Prior to the advent of damage control resuscitation approximately two decades ago, surgeons operated and performed definitive procedures. This usually resulted in metabolic abnormalities and/or mortality, as critically damaged patients frequently lack the physiological reserves necessary for definitive surgery. In contrast, patients who survive the initial stages of trauma treatment in the emergency room and operating room but die in the intensive care unit as a result of new-onset or uncontrolled postoperative bleeding. Our findings imply that there is a proportionate increase in exsanguination while admission to the ICU (in

comparison to a decrease in other causes of death). There were no significant differences between studies that looked at changes in ICU mortality due to exsanguination over a 15-year period, according to one that was included in this review.[6] It may be wise to concentrate on exsanguination prevention and treatment in the critical care unit setting, given these findings.[6]

The initial step in resuscitating a trauma patient is to correct overt indications of hypovolemia (hypotension, tachycardia, altered mental status, and oliguria). However, attaining these objectives cannot be deemed sufficient since, as previously stated, they do not detect latent hypoperfusion during compensated shock. As a result, resuscitation aimed at detecting and correcting perfusion deficits early has been advocated. [7]

As previously stated, early detection and intervention using a pulmonary artery catheter are recommended, as is resuscitation at supraphysiologic values of the heart's index and oxygen supply, as well as the completion of many controlled clinical trials. Three meta-analyses were conducted to consolidate their findings. The first identified methodological problems in primary investigations, making it difficult to draw firm findings.[7] Researchers found a non-significant trend toward a lower mortality rate and a shorter stay in the ICU when the analysis was restricted to studies that began hemodynamic optimization prior to surgery.[7]

In their meta-analysis, Ivanov and co-workers reported a significant reduction in morbidity when hemodynamic resuscitation of critically ill patients was guided with a pulmonary artery catheter.[8] Ivanov and co-workers Kern and Shoemaker examined the influence of supranormal hemodynamic optimization on people at high risk of developing cardiovascular disease. They determined that mortality was lowered in studies when treatment was initiated prior to the onset of organ failure and where the control group's mortality surpassed 20%.[9]

Hemodynamic optimization appears to be useful only if initiated promptly after trauma and is oriented toward correcting the underlying perfusion deficit. Thus, anaerobic metabolic indicators such as lactate or base excess must be analyzed and monitored in order to detect shock recovery at the cellular level.[10]

For the last 50 years, lactic acidosis has been associated with changes in oxygen supply. Its prognostic significance in hemorrhagic shock has been proven in terms of predicting infectious complications and multiorgan failure as well as indicating the risk of death.[10]. Numerous writers have demonstrated that early lactic acidosis clearance is associated with a greater chance of survival.[10] All patients who achieved acidosis correction during the first 24 hours survived. Mortality rose as the time interval between normalization and renormalization increased.[10]

Other indicators of metabolic acidosis have been associated with death. The most thoroughly researched is based deficiency. Hemorrhagic shock has been shown to be associated with an increased risk of death and a variety of organ dysfunction syndromes when the severity of metabolic acidosis is measured using this method.[11] The rate at which the base deficit normalizes has not been associated with prognosis. We compared lactate and base deficits. They had a strong connection in the initial evaluation, although lactate performed better during IV fluid resuscitation and in identifying compensated shock. [11]

Regional perfusion analysis may provide the most sensitive approximation.[12] Preliminary results on the detection of regional CO2 in sensible microvascular beds as a sign of hypoperfusion are encouraging.[12] In animal studies and a few published case reports, occult hypoperfusion has been diagnosed by measuring oxygen tension or saturation in specific tissues.[12] Regrettably, none of these strategies have been thoroughly examined, and none have gained widespread use.[12] Red blood cell transfusion is a common procedure in trauma patients. A multicenter investigation discovered that 55% of trauma patients admitted to the intensive care unit got transfusions throughout their stay.[13]

Blood transfusions are often based on the belief that the higher the hemoglobin level, the better when it comes to increasing blood's ability to carry oxygen. This view has been challenged by an understanding of the

critically ill patient's tolerance for low hemoglobin levels and by knowledge of the immunosuppressive effects of transfusion. Increased risk of infection, blood-borne illness, multiorgan failure and mortality as compared to comparable critically ill individuals.[14]

In a randomized clinical trial, the threshold for RBC transfusion was established in which 838 euvolemic critically ill patients with hemoglobin levels less than 9.0 g/dL were randomly assigned to receive RBC transfusions to maintain Hb concentrations between 10.0 and 12.0 g/dL (Liberal group) or to get RBC transfusions only when Hb concentrations fell below 7.0 in order to keep Hb concentrations between 7.0 and 9.0 g/dL (S group) (Restrictive group).Overall, neither group had a statistically significant difference in 30-day mortality, length of stay, or morbidity. Hill and colleagues validated similar findings in a meta-analysis comparing liberal and limited transfusion techniques. They discovered an extra 40% reduction in transfusion exposure in individuals assigned to restriction groups.[15] The current recommendation is to administer transfusions to keep hemoglobin concentrations between 7.0 and 9.0 g/dL in patients in critical care who are under the age of 55 and do not have significant heart disease.[16]

It has a lot of different causes, but the main ones are hypothermia, fibrinolysis caused by tissue trauma and endothelial damage, and the dilution of coagulation factors and platelets.[16] Occasionally, a person who has been hurt will already have a preexisting condition, like liver or hematologic disease, or will be taking anticoagulants. All of these things might make the person more likely to bleed, and they should be taken into account during the anamnesis.[16]

The entity is identified by bleeding from a variety of locations, including intravenous sites, the nasogastric tube, the vesical catheter, and surgical drains, as well as nonmechanical bleeding in the surgical field. As with so many other things, it is preferable to avoid complications than to have to address them later. Hypothermia prevention and treatment, as well as shock reduction, are accomplished through prompt interruption of the hemorrhage and aggressive hemodynamic resuscitation. Generally, prophylactic platelet or plasma administration does not prevent coagulopathy.[17]

Platelet count, activated partial thromboplastin time, prothrombin time-international normalized ratio, and fibrinogen concentration are simple laboratory tests used to identify coagulopathy and guide the administration of fresh frozen plasma, platelets, and cryoprecipitate.[18] Damage control surgery is an extremely effective adjunct procedure in the coagulopathic patient undergoing surgery. It enables rapid management of nonmechanical bleeding, restricts surgical extension, and breaks the vicious circle of bleeding–hypothermia–coagulopathy.[18]

In patients with known or suspected sepsis, the qSOFA predicts death and prolonged ICU stay using three variables: a Glasgow Coma Score of 15, a respiratory rate of 22 breaths per minute, and systolic blood pressure of 100 mmHg. When at least two of these characteristics are present concurrently, the patient is classified as qSOFA positive. In support of the 3rd International Consensus Conference on Sepsis Definitions, data analysis revealed that the qSOFA score was a predictor of poor outcome in patients with known or suspected infection; however, there was no data to support its use as a screening tool.[19]

In patients with suspected sepsis or septic shock, we advocate obtaining suitable routine microbiologic cultures (including blood) before beginning antimicrobial medication if this does not cause a considerable delay in starting antimicrobial therapy (i.e. < 45 minutes). Although this recommendation has not been updated in this version, it remains valid. It is difficult to distinguish the signs and symptoms of sepsis from those of many other disorders.[20,21,22] With no "gold standard" test to diagnose sepsis, the bedside provider cannot make an independent differential diagnosis of sepsis in a patient with organ dysfunction on the basis of symptoms alone. Indeed, up to a third or more of patients initially diagnosed with sepsis are later diagnosed with non-infectious conditions.[23,24]

Procalcitonin is undetectable in healthy individuals, but it rapidly rises in response to pro-inflammatory stimuli, most notably bacterial infections, which cause the body to respond. Procalcitonin levels, when

combined with clinical examination, may aid in the diagnosis of dangerous bacterial infections and prompt early antibiotic treatment. According to a meta-analysis of 30 investigations, procalcitonin has a sensitivity of 77% and a specificity of 79% for sepsis in critically ill patients (3244 patients).[25]

In the setting of sepsis and septic shock, beta-lactam drugs may exhibit changes in critical pharmacokinetic characteristics, resulting in subtherapeutic concentrations.[26,27] Administering an antibiotic over a longer period of time, such as an extended IV infusion (infusing the antibiotic for at least half of the dosage interval) or a continuous infusion, produces higher sustained beta-lactam concentrations than normal intermittent IV infusion (≤ 30 minutes). Antibiotic loading doses must be administered prior to continuous infusion to avoid delays in attaining effective beta-lactam concentrations.[28] Extensive and continuous infusions will take up more space in the vein/lumen than intermittent infusions over the course of treatment. Drug stabilization and inter-drug compatibility are important considerations in ensuring the efficacy of antibiotic and other IV drug regimens.[29]

This technique is feasible and has modest economic costs, and there is no indication that extended beta-lactam infusion results in worse outcomes than shorter infusions. As a result, if the requisite equipment is available, we recommend sustained beta-lactam infusion over typical bolus infusion in patients with sepsis and septic shock. More research is needed to assess the long-term effects of beta-lactam treatment, the impact on antimicrobial resistance development, and the expense of protracted beta-lactam administration versus bolus beta-lactam administration.[30]

Patients who are at high risk for MDR organisms should be monitored closely, we recommend empiric treatment with two gram-negative drugs to ensure appropriate coverage, whereas in patients at low risk for MDR organisms, we recommend empiric treatment with a single agentas the use of two agents appears to have little benefit and may increase the risk of side effects associated with antimicrobials, such as direct toxicity, infection with *Clostridium difficile,* and development.[31] Patients with severe disease, such as septic shock, who are at high risk of resistant infections, require gram-negative bacilli to be covered twice on an empirical basis.[31]

Table 1. Planned duration of empirical antimicrobial therapy in RCTs of shorter versus longer duration of therapy according to clinical syndrome

Population/syndrome	**RCT/systematic review (Data extracted from)**		**Shorter duration**	**Longer duration**	**Outcomes**
Pneumonia	[301]	Capellier (2012)	8 days	15 days	No difference
	[301, 302]	Chastre (2003)	8 days	15 days	No difference
	[302]	El Moussaoui (2006)	3 days	8 days	No difference
	[301-303]	Fekih Hassen (2009)	7 days	10 days	No difference
	[302-303]	File (2007)	5 days	7 days	No difference
	[302-303]	Kollef (2012)	7 days	10 days	No difference
	[302-303]	Leophonte (2002)	5 days	10 days	No difference
	[301]	Medina (2007)	8 days	12 days	No difference
	[302-303]	Siegel (1999)	7 days	10 days	No difference
	[302-303]	Tellier (2004)	5 days	7 days	No difference
Bacteremia	[302]	Chaudhry (2000)	5 days	10 days	No difference
	[302]	Runyon (1991)	5 days	10 days	No difference
	[304]	Yahav (2018)	7 days	14 days	No difference
Intra_abdominal infection	[305]	Montravers (2018)	8 days	15 days	No difference
	[293]	Sawyer (2015)	Max. 5 days	Max. 10 days	No difference
Urinary tract infection	[302]	Peterson (2008)	5 days	10 days	No difference

4. Conclusion

Patients with multiple trauma require effective, comprehensive, and fast treatment. When in the ICU requires resuscitation with specific goals, ventilation needs to be maintained so that oxygenation supply is met, hemodynamic and urine output need to be monitored so that tissue perfusion can be maintained properly; these things need to be considered to prevent worsening in the future, Surgical intervention, wound cleaning and appropriate and adequate antibiotics are also given to prevent worsening of sepsis in the future.

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