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The Role of Passive legs Raising Position in Hypovolemic Shock: A Case Report

Ira Rahmawati[1]*, Ade Dilaruri[2], Sulastyawati[1], Supono[1]
[1]Department of Nursing, Poltekkes Kemenkes Malang, Indonesia
[2]Arifin Achmad General Hospital, Riau Province, Indonesia
Corresponding author: irarahmawati.polkesma@gmail.com

ABSTRACT

Background: Hypovolemic shock can be catastrophic rapidly unless recognized and treated promptly. Although gastrointestinal loses might be the cause of hypovolemic shock in the elderly, it rarely causes a change in the blood gas analysis results.

Purpose: This study aimed to report a case of a 75-year-old male with signs of hypovolemic shock caused by gastrointestinal loses and discuss the effect of passive leg raising procedure as an early nursing intervention in patients presenting with shock.

Methods: The method used in this paper is a case study. The subject in this study is a 75-year-old male presenting to our Emergency Room with signs of shock which was caused by gastrointestinal problems. Passive leg raising or also known as modified Trendelenburg Position was performed as the patient presenting with low blood pressure. The results of physical dan laboratory investigation, as well as the nursing and medical therapies were presented, analyzed and discussed based on the recent literature.

Results: There was an increase in the Mean Arterial Pressure (MAP) after the intervention was given for five minutes followed by fluid resuscitation with 1000 ml of warm Normal Saline. The patient responded well to the intervention given in the ED and was sent to the Intermediate Ward for further treatments.

Conclusion: Passive leg raising might be beneficial to be performed in patients with hypovolemic shock as it increases the venous blood return the heart.

Keywords: Shock, Hypovolemic Shock, Case Report, Passive Leg Raising, Modified Trendelenburg Position.

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BACKGROUND

Shock is defined as a cardiovascular collapse leading to significant decrease in tissues perfusion (Wacker & Winters, 2014). It is characterized by systolic blood pressure below 90 mmHg and mean arterial pressure (MAP) lower than 60 mmHg or reduced of the MAP by more than 30% for more than 30 minutes, oliguria and decreased peripheral perfusion (Kalla & Herring, 2013; Worthley, 2000). Hypovolemic shock can be catastrophic rapidly unless recognised and treated promptly. Although gastrointestinal loses might be the cause of hypovolemic shock in the elderly, it rarely causes a change in the blood gas analysis results. Passive leg raising or also known as modified Trendelenburg position is not only a simple method to predict patients' hemodynamic improvement after fluid resuscitation, but may also improve patient's blood pressure by increasing blood return to the heart from the lower limbs. Having said that, the evidence on this topic is still lacking and need to be studied further.

OBJECTIVE

The aimed of this paper is to report a case of hypovolemic shock with metabolic acidosis following gastroenteritis and discuss the role of passive leg raising procedure in critically ill patients.

Case Presentation

A 75-year-old male was brought to the Emergency Department (ED) by an ambulance with signs of shock. The patient has diarrhoea for five days with frequency of more than seven times a day. It was described as watery diarrhoea with no blood in the stool. The patient had been seen by a general practitioner (GP) and given hydralyte and antiemetic drug. However, it became progressively severe and the patient was found lying on the bed by his relative after having diarrhoea for more than 10 times since early morning that day. His past medical history includes hypertension controlled with atenolol, Iron deficiency controlled with iron tablet and stroke.

On arrival in the ED, the patient was conscious and verbally responded but clearly in a hypotensive state with pale, cold skin, capillary refill time of 4 seconds and decreased urine output. The patient looked extremely lethargic. His blood pressure (BP) on arrival was 70/50 mmHg, heart rate (HR) 65 beats per minute, respiratory rates (RR) 14 breaths per minute with clear breath sounds and equal chest compliance, temperature (T) 34. 9º Celsius and Oxygen saturation 97% on oxygen 4 L/minute delivered via nasal cannula. Physical examination on the abdomen revealed generalised abdominal tenderness.

Complete blood count was performed with unremarkable results except for serum lactate (4.2 mml/L) and packed cell volume (0. 53 L/L). The Arterial Blood Gasses Analysis (ABG) results are shown in the table below.

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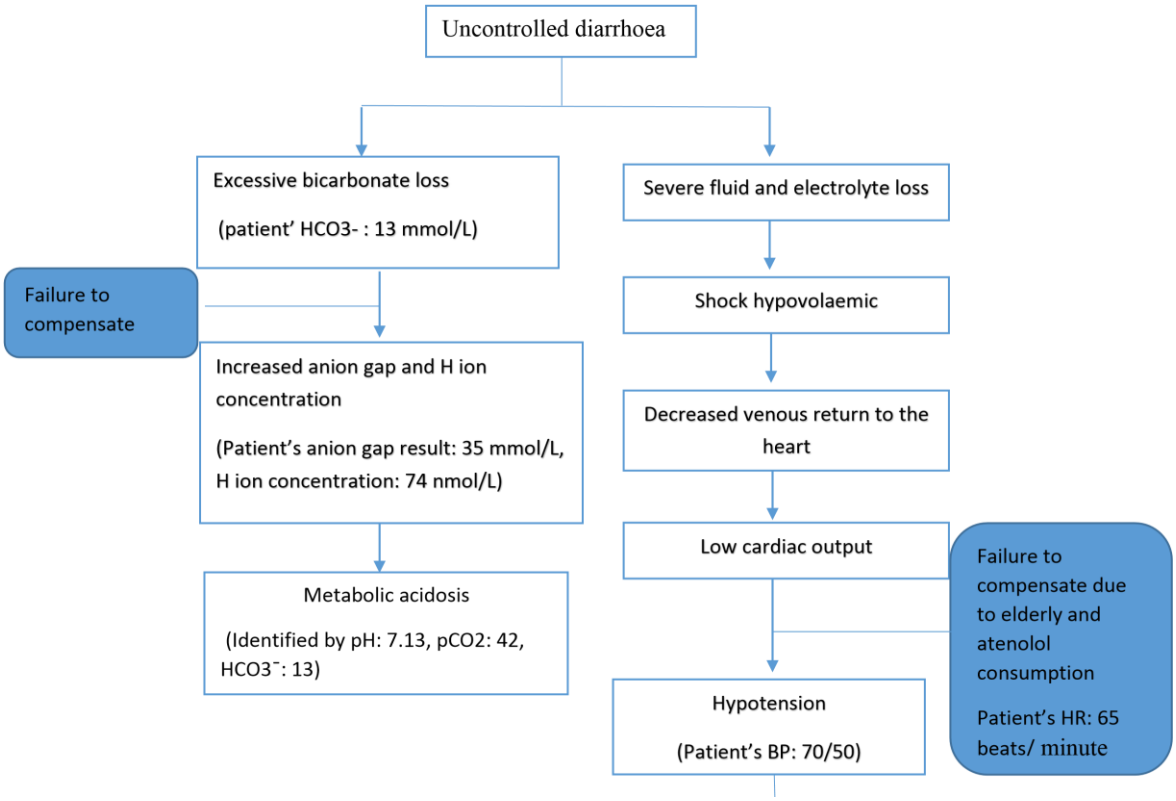
Table. 1 Blood Gasses Analysis Results

Measure	Result
pH	7.13
pO2 (mmHg)	82
pCO2 (mmHg)	42
HCO3- (mmol/L)	13
Potassium (mmol/L)	5.3
Sodium (mmol/L)	125
Chloride (mmol/L)	82
Anion Gap (mmol/L)	35
H ion Concentration (nmol/L)	74
Glucose (mmol/L)	9.3
Lactate (mmol/L)	4.2
Ionized Calcium (mmol/L)	1.00

A Chest x-ray and a 12 lead electrocardiogram (ECG) were also performed at the ED. The chest x-ray revealed normal results, and the ECG revealed sinus rhythm with peaked T wave.

Analysis of the Clinical Findings

The flowchart below has been formed to analyse the changes in patient's clinical features resulting from prolonged diarrhoea.



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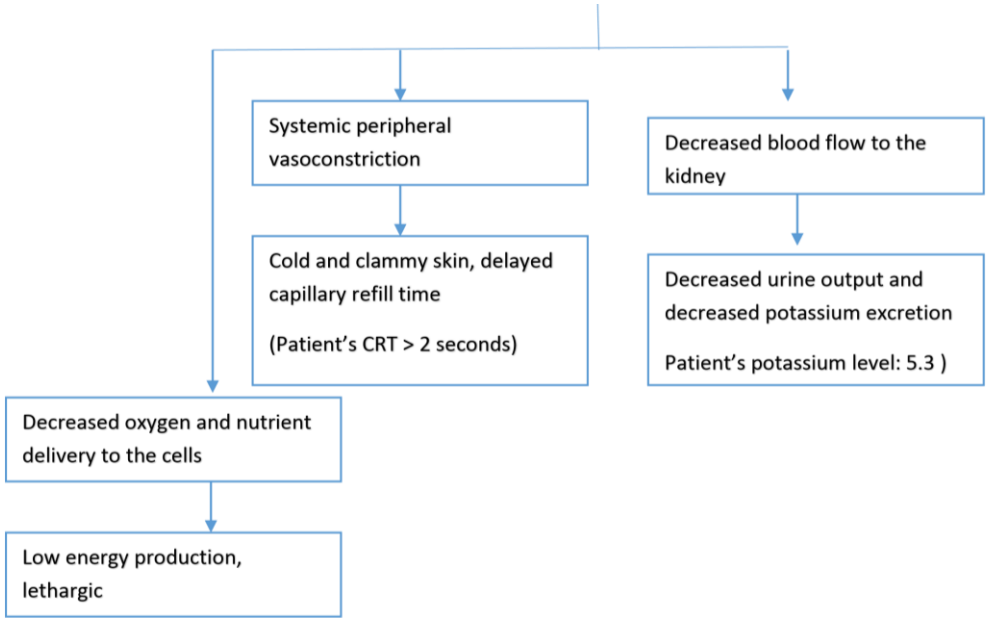

Figure 1. Analysis of the Clinical Findings

On arrival in the ED, the patient was in a shock state resulting from uncontrolled and prolonged watery diarrhoea. This patient presented with severe hypotension, cold and clammy skin, lethargic and no urine output despite indwelling urinary catheter (IDC) insertion. The HR of 65 beats/minutes indicates inadequate compensatory mechanisms to increase cardiac output, which can be correlated with atenolol (a beta-blocker agent) that he consumes every day. The patient had high level of serum lactate (4.2 mml/L) and increased packed cell volume (0.53 L/L) which indicate severe circulatory compromise and dehydration.

Our patient was also suffered from uncompensated metabolic acidosis indicated by low pH (7. 13), low $HCO3^-$ (13 mmol/L), increase in anion gap (35 mmol/L) and normal PCO2 (42 mmHg). Metabolic acidosis may be caused by excessive loss of bicarbonate in urine or stool (Goel & Calvert, 2012). The massive loss of bicarbonate in this patient results in increased anion gap to 35 mmol/L (the normal range is 7-17) which is the hallmark of uncompensated metabolic acidosis (Goel & Calvert, 2012).

Upon arrival at the ED, Passive legs raise (PLR) was performed and the BP was checked after 5 minutes. The patient's mean arterial pressure (MAP) increased by 5 mmHg. Fluid resuscitation was then commenced in the ED with warm normal saline 1000 ml over ten minutes. After that, another 1000 ml of warm normal saline was given over 30 minutes. The patient was positioned supine with the legs raised at about 30 degree. The patient was responded well to the fluid resuscitation identified by the improvement in his haemodynamic features. The BP increased to 90/60 mmHg after fluid administration. Then, the patient was monitored closely including the urine output aiming for urine output 0.5 – 1 ml/kg/hour.

DISCUSSION

Shock is a severe circulatory failure caused by an imbalance between demand and supply of cellular oxygen. Hypovolemic shock is identified by loss of intravascular volume caused by either haemorrhagic or non-haemorrhagic causes, which then resulted in preload reduction (Lier, Bernhard, & Hossfeld, 2018). On arrival in the ED, the patient was in a shock state resulting from uncontrolled and prolonged watery diarrhoea. Massive loss of

body fluid will eventually lead to decreased ventricular filling causing low cardiac output (Stuart & Bament, 2013). Further, insufficient cardiac output may cause hypotension and inadequate perfusion to the body organs including kidney, liver and heart, which may eventually cause hepatic failure, cardiac failure or even multiple organ failure (Curtis, Ramsden, & Friendship, 2007). This patient presented with severe hypotension, cold and clammy skin, lethargic and no urine output despite indwelling urinary catheter (IDC) insertion. The HR of 65 beats/minutes indicates inadequate compensatory mechanisms to increase cardiac output, which can be correlated with atenolol (a beta-blocker agent) that he consumes every day. Poor compensatory mechanism to the shock is also typical in elderly patients (Curtis et al., 2007; Kalla & Herring, 2013).The patient had high level of serum lactate (4.2 mml/L) and increased packed cell volume (0. 53 L/L) which indicate severe circulatory compromise and dehydration (Stuart & Bament, 2013).

At the ED, our patient was conscious and verbally responded but clearly in a hypotensive state with pale, cold skin, capillary refill time of 4 seconds and decreased urine output. These symptoms are typical in patients presenting with shock hypovolaemic (Stuart & Bament 2013). The patient was in the stage four of shock hypovolaemic, which is identified by severe hypotension, oliguria, lethargic, and drop in adenosine triphosphate (ATP) production (Kalla & Herring 2013). Upon patient's arrival at the ED, we performed passive leg raising to evaluate the patient's response to fluid loading as well as a technique to give rapid fluid loading. Improvement of the patient's systolic blood pressure after five minute of the procedure indicated positive responses. Fluid resuscitation was then commenced for this patient with warm normal saline 1000 ml over ten minutes. After that, another 1000 ml of warm normal saline was given over 30 minutes. Normal saline is the first choice of crystalloid fluid that strongly recommended for shock hypovolaemic with hyponatraemia as happened in this patient (Stuart and Bament 2013). This patient was responded well to the fluid resuscitation identified by the improvement in his haemodynamic features. The BP increased to 90/60 mmHg after fluid administration. Then, the patient was monitored closely including the urine output aiming for urine output 0.5 – 1 ml/kg/hour.

Passive leg raising or also known as modified Trendelenburg position is a simple method to predict patients' hemodynamic improvement after fluid resuscitation (Elwan et al., 2018; X. Monnet & J. L. Teboul, 2015). In spontaneously breathing patients, this maneuver can be performed in patients presenting with severely low blood pressure, shock or clinical signs of dehydration. The purpose of this procedure is to transfer venous blood from the lower body toward the intra-thoracic compartment and assess its effect on cardiac output, MAP or stroke volume (Maizel et al., 2007; Pottecher et al., 2010). Passive leg raising is similar to a fluid challenge with no fluid given. Therefore, preventing the risks of excessive fluid therapy which dangerous in critically ill patients.

The manoeuvre is carried out by first, putting the patient supine or preferably semi-recumbent. Semi –recumbent position will increase the effect of leg raising on cardiac preload as it mobilizes venous blood from the splanchnic area (X. Monnet & J.-L. Teboul, 2015). Then, positioning the patient supine with legs elevated to 30 degrees. The effects of this manoeuvre are then assessed by several methods. The effects of passive leg raising can be performed by measuring the cardiac output (CO) and stroke volume (SV) non-invasively using echocardiography or evaluating changes in stroke volume and carotid flow time following PLR (Jalil et al., 2018). A study conducted in 2015 measured cardiac output (CO), stroke volume (SV), heart rate and blood pressure at the baseline, during passive leg raising and after fluid loading. This study found that CO and SV increased by more than 12% during the PLR manoeuvre, proofing that PLR a reliable bedside method to predict positive improvement after fluid resuscitation (Maizel et al., 2007).

In addition to predict whether a patient will response positively to fluid resuscitation, a PLR manoeuvre may also serve as a rapid fluid loading by mobilizing about 300 ml of blood from the lower limbs toward the heart (Caille et al., 2008). Indeed, PLR manoeuvre has been widely utilised as one of the initial treatment for patients with hypovolemic shock (Gaffney, Bastian, Thal, Atkins, & Blomqvist, 1982). However, The American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (2010) do not recommend neither against the PLR position as an optimal position in shock (Markenson et al., 2010).

PubMed and google scholar were further searched to identify clinical studies determining the role of PLR manoeuvre in critically ill patients. We focused on studies that investigated weather PLR manoeuvre improves the patients' outcomes. We found only few studies that documented the duration and extent of changes in haemodynamic parameters produced by this manoeuvre. A study conducted in 2014 found that this manoeuvre lessened the severity of hypotension and tachycardia following a lower limb's tourniquet deflation in patients with a total knee arthroplasty (Huang et al., 2014). However, a study involving 30 healthy individuals in 2018 found contrary results. A PLR position in these individuals induced decreased systolic blood pressure immediately during PLR and after PLR maneuver (Pickett, Bridges, Kritek, & Whitney, 2018).

In cases of cardiac arrest, a recent experimental study was conducted to evaluate the PLR effects on survival rate to 30 days in out-of-hospital cardiac arrests. The study included 3554 cardiac arrest cases with 44% of the cases were given PLR maneuver during CPR and 56% of the cases did not received PLR maneuver. This study found that there is no evidence that PLR maneuver increases the rate of survival post cardiac arrest to 30 days (Holmén, Herlitz, Jimenez-Herrera, Karlsson, & Axelsson, 2019). Having said that, this manoeuvre provides approximately 300 ml extra blood to the heart, thus increasing stroke volume. Therefore, the short-term effects of PLR manoeuvre in patients with hypovolemic shock or cardiac arrests might still be beneficial.

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