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ORIGINAL RESEARCH

The Relationship between Total Carbondioxide and Lactate Levels to Hypovolemic Shock Post-CABG in ICU Dr Kariadi General Hospital

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ABSTRACT

Background: A total of 2.4% of patients after Coronary artery bypass grafting (CABG) required reoperation due to postoperative bleeding leading to vasodilatory shock syndrome. The condition of metabolic acidosis was common in shock patients where a low serum TCO2 value could be used as an indicator. Lactic acidosis was the most common cause of metabolic acidosis in hospitalized patients. TCO2 measurement was more useful to determine anion gap, but still rarely utilized. The correlation of TCO2 and lactate levels were not known well in hypovolemic shock. This research aims to analyzing the correlation of serum TCO2 values and lactate levels to the condition of hypovolemic shock post CABG.

Method: This study was an observational prospective cohort study. 23 subjects were post-CABG patients undergoing treatment at Dr. Kariadi as of January-February 2023. Data were obtained from hemodynamics status, measuring serum TCO2 and lactate levels in blood samples from patients taken 1 hour after surgery. The data were then processed using fischer's exact test.

Results: 26.1% subjects were experiencing hypovolemic shock post-CABG. Correlation analysis between lactate, TCO2 artery, and TCO2 vein to the condition hypovolemic shock post-CABG showed strong correlation ($p=0.009$; $p=0.003$; $p=0.003$). The correlation of lactate levels and TCO2 values both artery and vein resulted strong correlation ($p=0.026$). Cut-off lactate level delta was 5.8; cut-off TCO2 Artery delta was 20.5; cut-off TCO2 vein delta was 21.55 which were all measured using the ROC curve.

Conclusion: TCO2 serum and lactate levels have strong correlation to the condition hypovolemic shock post-CABG patients.

Keyword: Coronary Artery Bypass Graft; Hypovolemic shock; Lactate level ; TCO2 serum.

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INTRODUCTION

Shock after cardiac surgery is a serious condition with high morbidity and mortality. As many as 18.5% of patients undergoing cardiac procedures and 15.1% of patients undergoing coronary artery bypass grafting (CABG) experienced complications and were readmitted to hospital 30 days after initial hospitalization.[1] In a study of a total of 12,652 CABG patients, 2.4% of patients required reoperation due to postoperative bleeding. The four forms of shock—cardiogenic, hypovolemic, obstructive, and distributive—can occur separately or in combination.[2]

Elderly patients are more likely to experience hypovolemic shock due to fluid loss because they have less physiologic reserves. Cardiopulmonary bypass (CPB) can also be complicated by a systemic inflammatory response characterized by profound vasodilatation. This vasodilatory shock syndrome, which is especially noted after prolonged CPB, is associated with endothelial injury and release of cytokines and other inflammatory mediators.[2]

Lactic acidosis is the most common cause of metabolic acidosis in hospitalized patients. Lactic acidosis occurs when lactic acid production exceeds lactic acid clearance. Increased lactate production is usually caused by impaired tissue oxygenation, either from decreased oxygen delivery or defects in mitochondrial oxygen utilization. If the stimulus for lactic acid production is treated by treatment of the underlying disease (eg, restoration of perfusion in a patient in shock), oxidative processes will metabolize the accumulated lactate and regenerate bicarbonate.[3,4]

The patient's volume status has a major influence on absorption, because sodium is reabsorbed along with this bicarbonate. Thus, volume contraction stimulates sodium and bicarbonate reabsorption. This causes an increase in the total CO2 content. Low total CO2 levels result from metabolic acidosis or compensate for respiratory alkalosis.[4]

Serum total carbon dioxide (TCO2) is usually included in routine serum analysis, so it can be more readily available than other indicators of metabolic acidosis. Several previous studies have used serum TCO2 to estimate the amount of metabolic acidosis and to predict patient prognosis. Serum bicarbonate (HCO3-) and base deficit can be markers of shock. However, previous studies have shown that HCO3- levels from calculations are often not in line with HCO3- measurement results and are influenced mainly by the anion gap caused by lactate and ketones.[3] Measurement of serum TCO2 values is more useful clinically in determining the anion-gap, but has not been applied as a routine examination in patients with shock and other hemodynamic disorders postoperatively. The relationship between serum TCO2 and lactate levels in hypovolemic shock is not clearly known. Based on this background, the aim of this study was to determine the relationship between total serum CO2 and lactate levels on hypovolemic shock in postoperative Coronary Artery Bypass (CABG) patients.

METHODS

This study was an analytical observational with a prospective cohort approach which was carried out at Dr. Kariadi General Hospital Semarang from January to February 2023. Participants were ICU patients who were about to undergo CABG surgery, aged 18-70 years, with normal albumin and electrolyte levels. Patients with a history of chronic kidney disease, uncontrolled diabetes mellitus for the last 3 months, cardiac tamponade, chronic lung disease or lung infection, and hormonal disorders (Cushing's syndrome, aldosterone hormone disorders) were excluded. A total of 23 participants obtained by consecutive sampling were required for an α level of .05, a power of 80%, and a dropout rate of 10%. The primary outcomes were assessed for the relationship between serum total CO2 (TCO2) and lactate levels in hypovolemic shock. Written informed consent was obtained from the participants. Furthermore, participants will undergo CABG surgery with standard procedures at Dr. Kariadi Hospital, Semarang. Then the patient's blood was taken 1 hour after surgery to measure serum TCO2 levels and lactate levels. Hemodynamic parameters and the incidence of hypovolemic shock were monitored and recorded at 1 hour postoperatively.

Univariate analysis was used for descriptive analysis which was presented in tabular form and frequency distribution for categorical variables, as well as the average value (Mean) and standard deviation for numerical variables. Bivariate analysis used the Pearson Chi Square correlation test if the data was normally distributed and the Fischer's Exact test if the data was not normally distributed to determine the relationship between variables. Significant value if $p < 0.05$. The diagnostic accuracy of serum TCO2 levels and lactate levels for hypovolemic shock was assessed by calculating the area of the Area Under the Curve (AUC) on the Receiver Operating Characteristic (ROC) curve.

RESULTS

Twenty three patients with mean (SD) age of 57.5 years were recruited

(Table 1). 87% of the patients were male while 13% were female. BMI was 25.78 kg/m2, lactate was 5.31. On arteryTCO2 examination, it was 21.42 and venous was 22.23.

Table 1. Participants Characteristics

Variables	N (%)	Mean ± SD
Gender		
Male, No. (%)	20 (87.0)	
Female, No. (%)	3 (13.0)	
Age, y		57.52 ± 6.75
BMI, Kg/m^2		25.78 ± 3.98
Lactate		5.31 ± 2.22
Artery TCO_2		21.42 ± 3.22
Vena TCO_2		22.23 ± 4.52
Hypovolemic Shock		
Yes	6 (26.1)	
No	17 (73.9)	

There was a significant relationship between lactate levels (p = 0.009), arterial TCO2 (p = 0.003) and venous TCO2 (p = 0.003) to the condition of hypovolemic shock (Table 2). There was also significant relationship between Lactate to Arterial TCO_2 (p = 0.026) and Venous TCO_2 (p = 0.026). (Table 3)

Table 2. Lactate and TCO_2 correlation based on hypovolemic shock

Variables	Hypovolemic Shock		p Value	OR (95% CI)
	Yes (n=6)	No (n=17)		
Lactate				
≥ 5.80	5 (62.5%)	3 (37.5%)	0.009$^{£}$*	23.33 (1.95 – 279.43)
< 5.80	1 (6.7%)	14 (93.3%)		
Artery TCO_2				
≤ 20.50	5 (71.4%)	2 (28.6%)	0.003$^{£}$*	37.50 (2.77 – 507.48)
> 20.50	1 (6.3%)	15 (93.8%)		
Vena TCO_2				
≤ 21.55	5 (71.4%)	2 (28.6%)	0.003$^{£}$*	37.50 (2.77 – 507.48)
> 21.55	1 (6.3%)	15 (93.8%)		

*Significant ; $^{£}$Fisher's exact

Table 3. Lactate relationship to arterial and venous TCO_2

Variables	Lactate		P Value	OR (95% CI)
	≥ 5.80	< 5.80		
Artery TCO_2				
≤ 20.5	5 (62,5%)	2 (13,3%)	0.026£*	10.83 (1.37 – 85.44)
> 20.5	3 (37,5%)	13 (86,7%)		
Vena TCO_2				
≤ 21.55	5 (62,5%)	3 (37,5%)	0,026£*	10.83 (1.37 – 85.44)
> 21.55	2 (13,3%)	13 (86,7%)		

*Significant ; £Fisher's exact

Sensitivity, specificity, positive predictive value, negative predictive value for lactate, artery and venous TCO_2 for hypovolemic shock can be seen in table 4. It was known that the sensitivity for lactate, arterial and venous TCO_2 were the same. Meanwhile, the specificity, positive predictive value, negative predictive value, and accuracy of TCO_2 were superior to Lactate.

Table 4. Lactate and TCO2 diagnostic table based on hypovolemic shock

Variables	Hypovolemic Shock		Sens. (%)	Spe. (%)	PPV (%)	NPV (%)	Acc. (%)
	Yes	No					
Lactate							
≥ 5.80	5	3	83.33	82.35	62.50	93.33	82.61
< 5,80	1	14					
Artery TCO_2							
≤ 20,50	5	2	83.33	88.24	71.43	93.75	86.96
> 20,50	1	15					
Vena TCO_2							
≤ 21,55	5	2	83.33	88.24	71.43	93.75	86.96
> 21,55	1	15					

Abbreviations : Sens = Sensitivity; Spec = Specificity; PPV = Positive Predictive Value; NPV = Negative Predictive Value; Acc = Accuracy.

The cut-off calculation of lactate levels for the diagnosis of hypovolemic shock used the receiver operating characteristic (ROC) curve. The cut-off delta value of lactate levels was 5.8 (AUC = 0.868; sensitivity 83.3%; and specificity 82.35%). The results of the lactate level delta examination are considered positive if they are more or equal to the cut-off value, and are considered negative if they are less than the cut-off value. Calculation of cut-off arterial TCO2 levels for the diagnosis of hypovolemic shock used the receiver operating characteristic (ROC) curve. The cut-off delta value of arterial TCO2 levels was 20.5 (AUC = 0.946; sensitivity 83.3%; and specificity 88.24%). The results of the delta examination of arterial TCO2 levels are considered positive if they are more or equal to the cut-off value, and are considered negative if they are less than the cut-off value.

DISCUSSION

The serum TCO_2 level is a measure of serum bicarbonate replacement. The total CO_2 concentration includes serum bicarbonate and available forms of carbon dioxide (ie, carbonate anion and carbonic acid). In general, serum bicarbonate accounts for about 95% of the total CO_2 concentration. The concentration of carbonic acid is proportional to the partial pressure of carbon dioxide (p CO_2) in the blood. Multiplying p CO_2 by the constant (0.03) estimates the concentration of carbonic acid. A change in hydrogen ion concentration (pH) results from a change in either bicarbonate or carbon dioxide. Measurement of the total CO_2 content can help us explain acid-base disturbances (when the pH and p CO_2 are known). Furthermore, because we often measure the total CO_2 content as part of automated chemical determinations, this measurement can provide the first clue to acid-base disturbances. The body produces about 1 mEq/kg of hydrogen ions daily (mainly from protein metabolism). The kidneys normally excrete this daily acid load. Failure of excretion forces the reactions of H+ and H CO_3−, resulting in a decrease in the bicarbonate concentration.[3,4]

Total carbon dioxide (TCO2) is one of the accordion scoring tests usually included in routine serum chemistry analysis, and therefore it may be more accessible than other indicators of metabolic acidosis. This test measures the total amount of carbon dioxide in the blood, and about 95% of the carbon dioxide in the blood is in the form of bicarbonate. Carbon dioxide is used to diagnose, monitor, and treat a variety of potentially serious conditions related to changes in the body's acid-base balance. Because the serum T CO_2 concentration almost directly reflects the blood bicarbonate concentration and the T CO_2 value is directly proportional to the HCO3. There have been several previous studies using serum T CO_2 to estimate the amount of metabolic acidosis and to predict patient prognosis.[3,4] Serum T CO_2 concentrations of 20 mmol/l or less are usually associated with 28-day mortality in patients with sepsis.[5]

In this study, there was a correlation between arterial and venous T CO_2 and lactate levels on hypovolemic shock. This is in line with the research conducted by Cheah K et al[3], namely the use of ET CO_2 in the emergency room has great potential to be used as a non-invasive monitoring method for shock patients. Besides that research performed by Jin Hee Kim et al in patients with sepsis or septic shock. The relationship between serum T CO_2 and 28-day mortality, bicarbonate, pH, lactate, and anion gap determined by the cubic spline curve found that a serum T CO_2 concentration of 20 mmol/l or less was associated with 28-day mortality in patients with sepsis. This study demonstrated the role of serum T CO_2 in diagnosing shock.[4,7]

Symptoms of hypovolemic shock may be related to volume depletion, electrolyte imbalance, or acid-base disturbances that accompany hypovolemic shock. Patients with volume depletion may complain of thirst, muscle cramps, and/or orthostatic hypotension. Shock in cardiac surgery intensive care unit (ICU) patients is a serious condition with high morbidity and mortality. Prompt identification of the underlying condition and correct management of life-threatening physiological damage is essential to save the patient. Shock in patients undergoing cardiac surgery frequently occurs with several potential causes playing a role. Postoperative complications of cardiac surgery that may cause hypotension include cardiac complications such as regurgitation or prosthetic paravalvular thrombosis, cardiac tamponade, myocardial infarction, or the development of atrial fibrillation or ventricular dysrhythmias. So the importance of examination to determine the state of shock in patients appropriately.[1,2]

One of the tests that can be used as a marker of shock is lactate. This can assist in the initial assessment of shock and its predictive value, and the subsequent response to therapy. Elevated lactate suggests a relatively slow response to hypoperfusion and may lead to treatment decisions being made in response to conditions that may not reflect the patient's current clinical status. Lactate levels may remain elevated for some time after the conditions that caused the increase, especially in liver failure where lactate metabolism is impaired. In addition, current understanding of lactate metabolism indicates that lactate formation in shock occurs by a complex process that cannot be explained by tissue hypoperfusion alone. If lactate is not cleared within 24 hours, the prognosis appears to be poor, and persistently elevated lactate levels appear to be associated with almost complete death. Failure to clear lactate within 24 hours seems to connote a poor prognosis, and persistently elevated lactate levels appear to be associated with near-total death. However, because of its prognostic value, low cost, and ease of monitoring, serum lactate remains a useful part of the overall assessment and monitoring and may identify a subset of patients who may benefit from early goal-directed resuscitation.[3,8]

In addition, there are other tests that can be used as a marker of shock, namely the total serum CO_2 level. Low total CO_2 levels result from metabolic acidosis or compensate for respiratory alkalosis. Bicarbonate levels below 10 mEq/L actually identify metabolic acidosis as the cause, because compensation for respiratory alkalosis will not push bicarbonate that low. If a metabolic acidosis is present, a distinction must be made between increased anion gap and nonanion gap acidosis. In summary, total serum CO_2 can provide clues to acid-base abnormalities. When used together with pH and pCO2, these measurements help us determine the possible causes of metabolic imbalances, especially in acutely ill patients.[3] In this study it has been found that both arterial and venous T CO_2 have an accuracy (Area Under the Curve/AUC) of using serum Total CO_2 (T CO_2) as a marker of hypovolemic shock after Coronary Artery Bypass Graft.[4,8]

Lactate is the result of anaerobic metabolism that occurs in body cells and is an indicator of work overload or hypoxia. Under conditions of hypovolemic shock, lactate has a strong influence on the level of total CO_2 (T CO_2) in the body. According to a study conducted by Chen et al. (2017), increased lactate production in conditions of hypovolemic shock will cause an increase in T CO_2. This is due to changes in the lactic acid and carbon dioxide cycles which result in high T CO_2. In hypovolemic shock, the body will experience disturbances in blood flow and oxygen flow to body tissues. This will cause hypoxia and decrease the efficiency of oxygen burning by body cells. As a result, cells will switch to using anaerobic metabolism for energy and produce lactate as a by-product.[4]

The lactate produced will affect the formation of CO_2. This process is called the lactate-carbon acetate cycle which converts lactic acid to carbon dioxide and water. The addition of T CO_2 in the body will increase arterial CO_2 and cause an increase in the concentration of HCO3- in the blood, which leads to respiratory acidosis. However, the study conducted by Al-Sarraj et al. (2016) showed that an increase in T CO_2 does not always lead to respiratory acidosis. In hypovolemic shock, high T CO_2 can result in increased formation of bicarbonate and reduced respiratory acidosis. Overall, the effect of lactate on T CO_2 in hypovolemic shock is complex and depends on several factors such as the rate of lactate production, blood and oxygen flow, and the efficiency of bicarbonate formation.[6-8]

Both T CO_2 and lactate levels can be used as markers of hypovolemic shock, this cannot be separated because the mechanism of both is closely related to acid-base conditions in the human body such as metabolic acidosis that occurs in patients with shock. Metabolic acidosis can be measured by pH, p CO_2, bicarbonate concentration, and base deficit obtained from arterial blood gas analysis (Arterial BGA). Additionally, lactate concentration or anion gap is measured to assess the causes and associated factors of metabolic acidosis.[4]

So the above theory supports the findings in this study which show that both lactate and TCO2 levels are associated with hypovolemic shock after CABG surgery.

CONCLUSION

The use of serum Total CO2 (TCO2) and lactate levels has a significant relationship to the condition of postoperative hypovolemic shock Coronary Artery Bypass Graft. There is a significant relationship between serum Total CO2 (TCO2) and lactate levels on hemodynamic conditions after Coronary Artery Bypass Graft. The results of the diagnostic test with the ROC curve showed that the cut-off value of the lactate level delta was 5.8 (AUC=0.868; sensitivity 83.3%; and specificity 82.35%); The cut-off delta value of Arterial TCO2 levels was 20.5 (AUC=0.946; sensitivity 83.3%; and specificity 88.24%); The delta cut-off value of venous TCO2 levels was 21.55 (AUC=0.936; sensitivity 83.3%; and specificity 88.24%)

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