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RESEARCH ARTICLE

Early Postoperative Hyperlactatemia After Extracorporeal Circulation: The Role of Standard Base Excess and Anion Gap in Differential Diagnosis

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ABSTRACT

Objectives: This study aimed to determine whether follow-up with standard base excess (SBE) and anion gap (AG) aids in the differential diagnosis of early postoperative hyperlactatemia, specifically in distinguishing between low cardiac output and lactate washout.

Methods: The study involved 1203 patients who underwent isolated coronary bypass surgery with the help of Extracorporeal Circulation (ECC). These patients were divided into two groups based on their cardiac index (CI): Group 1 consisted of 1162 patients with CI ≥ 1.8 L/min/m^2, while Group 2 had 41 patients with CI<1.8 L/min/m^2. Blood gas measurements were taken at five different time points to examine the correlation between lactate, SBE, and AG.

Results: The correlation between lactate and SBE in Group 1 was weak (r=-0.07, p<0.001). Similarly, the correlation between lactate and anion gap was weak (r=0.08, p=0.005). On the other hand, in Group 2 (CI<1.8 L/min/m^2), a much stronger correlation was observed between lactate and SBE (r=-0.49, p<0.001). However, there was no correlation between lactate and anion gap (r=-0.007, p=0.964).

Conclusion: Relying solely on SBE (standard base excess) and anion gap to distinguish hyperlactatemia is limited because they depend on various variables. Therefore, we recommend assessing hyperlactatemia by examining the patient's clinical condition, other tissue perfusion parameters, flow measurements, plasma chloride, and albumin values.

Keywords: Anion gap, extracorporeal circulation, hyperlactatemia, open heart surgery, standard base excess

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Introduction

Patients exposed to extracorporeal circulation (ECC) during open-heart surgery are at increased risk of impaired macrocirculation-microcirculation relationship.[1] Assessing the adequacy of tissue perfusion in such patients can be challenging due to underlying heart disease, comorbidities, the adverse effects of extracorporeal circulation, and the inherent limitations of tissue perfusion parameters. Monitoring multiple parameters simultaneously can help achieve intraoperative hemodynamic stability and adequate tissue perfusion.[2,3]

Lactate can be measured through blood gas analysis, which can help indicate hypoperfusion and tissue hypoxia.[4,5] Another parameter that can measure tissue perfusion is Standard Base Excess (SBE), which is immensely valuable as it can differentiate between metabolic acid-base changes and respiratory changes.[2,6] Typically, hyperlactatemia is associated with low cardiac output. However, it can also be caused by several other factors. One of the most crucial factors possible is 'washout hyperlactatemia,' which must be differentiated from hyperlactatemia caused by low cardiac output to provide appropriate treatment.

Anion Gap (AG) is another parameter calculated based on blood gas analysis and electrolyte monitoring. AG is helpful in the differential diagnosis of metabolic acidosis.[7]

In this study, we investigated whether follow-up with SBE and AG contributes to the differential diagnosis of early postoperative hyperlactatemia, whether it is due to low cardiac output or lactate washout.

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Methods

The ethics committee of Acıbadem Mehmet Ali Aydınlar University has approved the analysis of perioperative data from 1203 patients who underwent isolated coronary bypass surgery (ATADEK 2024-4/158). All principles of the Helsinki Declaration were followed when designing the study.

Patients who were under 18 years of age, required bicarbonate infusion due to arterial pH falling below 7.1, underwent preoperative resuscitation, total circulatory arrest or deep hypothermia, and had preoperative lactate elevation or moderate-to-severe liver failure were excluded from the analysis.

Our clinic follows a specific anesthesia protocol for patients who undergo open-heart surgery. Below is a detailed mention of the procedure.

Before surgery, the patient is given Alprazolam (0.5 mg PO) as a premedication. Midazolam (125 µg/kg IM) is administered thirty minutes before the surgery. A 16 G intravenous cannula is used to establish vascular access, and physiological saline infusion is initiated at 100 ml/hour. For anesthesia induction, Midazolam (50 µg/kg iv), Propofol (1–2 mg/kg iv), and Fentanyl (25–35 µg/kg iv) are used. After intubation, muscle relaxation is initiated with a Vecuronium bolus (0.15 mg/kg iv) and continued with an infusion (80 µg/kg/hour iv). For anesthesia maintenance, either Desflurane or Sevoflurane inhalation is used with a target MAC value of 0.9–1. Hemodynamic monitoring is provided through electrocardiogram (DII, V5), pulse oximetry (SpO_2), and invasive arterial pressure monitoring with right radial artery catheterization. Additionally, central venous pressure and venous oxygen saturation are monitored with right internal jugular vein catheterization.

The hematocrit (Hct) level was maintained during the ECC between 25–30%. The mean arterial pressure (MAP) was regulated at 50–80 mmHg, and the pump flow rate (CO) was set at 2–2.5 L/m^2/minute. All patients were kept under moderate hypothermia (32°C). The adequacy of tissue perfusion during ECC was determined by closely monitoring several parameters, such as venoarterial partial carbon dioxide difference (Pv-aCO_2), lactate level, arterial and venous blood gas analyses, urine output, and hemodynamic parameters.

Upon admission to the intensive care unit, patients were warmed until their rectal body temperature reached 37°C. Sedative and muscle relaxant infusions were stopped, and patients were extubated as soon as they met the extubation criteria. Morphine (0.1 mg/kg) met their analgesic needs. Routine monitoring of hemodynamics, blood gases, biochemistry, electrolytes, and body temperature was carried out regularly and as required.

The 1162 patients with preserved cardiac output (CI ≥ 1.8 L/min/m^2) were classified into Group 1, while the 41 patients with low cardiac output (CI<1.8 L/min/m^2) were classified into Group 2.

Blood gas measurements were taken at five different time points for data analysis. These time points were admission to the intensive care unit (T0), pre-extubation (T1), 30 minutes after extubation (T2), 60 minutes after extubation (T3), and 120 minutes after extubation (T4). Low cardiac output was diagnosed when the cardiac index (CI) was below 1.8 L/min/m^2 using the Fick method.

Patient characteristics were presented as mean (standard deviation), median (quartiles), and percentage. Correlations were analyzed using Pearson and Spearman tests. Statistical significance was set at $p<0.05$. The analysis was performed using SPSS version 29.

Results

Table 1 presents the patients' baseline blood gas and hemodynamic parameters. Table 2 shows demographic, blood gas, and hemodynamic data at T4 for 1162 patients with preserved cardiac output (Group 1) and 41 patients with low cardiac output (Group 2).

In Group 1 (CI ≥1.8 L/min/m^2), the correlation between lactate and SBE during T2 and T4 time points was weak ($r=-0.07$, $p<0.001$) as shown in Figure 1. At the same time points, Figure 1 also showed that the correlation between lactate and anion gap was weak ($r=0.08$, $p=0.005$).

On the other hand, Group 2 (CI<1.8 L/min/m^2) showed a much stronger correlation between lactate and SBE during T2 and T4 time points ($r=-0.49$, $p<0.001$), as depicted in Figure 2. However, Group 2 revealed no correlation between lactate and anion gap simultaneously ($r=-0.007$, $p=0.964$).

Discussion

The relationship between macrocirculation and microcirculation can be disrupted due to various situations, including comorbidities such as hypertension,[8] diabetes mellitus,[9] congestive heart failure,[10] long-lasting and bleeding surgeries,[11] the Trendelenburg position,[12] and surgeries that involve ischemia in organs.[13,14] As ECC is also one factor that disturbs this relationship, it is recommended that patients undergoing open-heart surgery with ECC be closely monitored to ensure adequate tissue perfusion.[14] In addition to basic monitoring parameters like heart rate, electrocardiography, pulse oximetry, and mean arterial pressure, advanced cardiac monitoring and parameters such as cardiac output analysis, venoarterial partial carbon dioxide difference (Pv-aCO_2), regional cerebral oxygen saturation (rSO_2), and blood gas analyses are evaluated.[1]

Lactate is a commonly used marker of tissue perfusion in clinics. It can be easily measured through blood gas analysis and can indicate if there is hypoperfusion and tissue hypoxia.[4,15] Hyperlactatemia observed in the early

Table 1. Blood gas and hemodynamic parameters for all patients

	T0 (before extubation)	T1 (Postoperative 30th min)	T2 (Postoperative 120th min)
pH	7.37 (7.33–7.41)	7.36 (7.33–7.39)	7.36 (7.33–7.40)
PaO_2, mmHg	160 (134–185)	167 (131–201)	123 (84–164)
$PaCO_2$, mmHg	38.0 (33.9–42.2)	38.7 (35.0–42.6)	39.0 (35.3–42.0)
HCO_3, mmol/L	22.0 (21.0–23.3)	22.0 (21.0–23.0)	21.8 (21.0–23.4)
SBE, mmol/L	-2.3 (-3.9 ; -0.9)	-2.8 (-4.0 ; -1.2)	-2.2 (-3.9 ; -0.9)
Hb, g/dL	10.6 (9.5–12.0)	10.4 (9.3–11.9)	10.0 (9.0–11.2)
Hct, %	33.0 (29.5–37.0)	32.7 (29.0–36.6)	31.7 (28.0–35.0)
SaO_2, %	99 (99–100)	99 (99–100)	99 (97–99)
SvO_2, %	66±10	66±9	67±9
Na, mmol/L	139 (137–141)	140 (138–142)	140 (138–142)
K, mmol/L	4.0 (3.8–4.3)	4.0 (3.8–4.3)	4.1 (3.9–4.4)
Cl, mmol/L	113 (111–116)	114 (111–116)	114 (111–116)
Ca, mmol/L	1.10 (1.05–1.13)	1.10 (1.04–1.13)	1.10 (1.03–1.12)
Lactate, mmol/L	1.6 (1.3–2.0)	1.5 (1.1–1.9)	1.3 (1.0–1.7)
AG, mmol/L	8 (5–12)	9 (5–12)	9 (5–12)
Glucose, g/dL	150 (133–173)	151 (134–174)	150 (134–171)
HR/min	88 (78–98)	87 (79–97)	87 (78–96)
MAP, mmHg	86 (77–95)	83 (75–92)	81 (73–90)

PaO_2: Partial pressure of oxygen; $PaCO_2$: Partial carbon dioxide pressure; HCO_3: Bicarbonate; SBE: Standard base-excess; Hb: Hemoglobin; Hct: Hematocrit; SaO_2: Arterial blood oxygen saturation; SvO_2: Central venous oxygen saturation; Na: Sodium; K: Potassium; CI: Confidence interval; Ca: Calcium; AG: Anion gap; HR: Heart rate; MAP: Mean arterial pressure.

Table 2. Comparison between Group 1 (CI ≥1.8 L/min/m²) and Group 2 (CI <1.8 L/min/m²) at the postoperative 120th minimum

	Group 1 (CI ≥1.8 L/min/m²) (n=1162)	Group 2 CI <1.8 L/min/m²) (n=41)	p
Age, years	59±9.4	60±8.9	NS
Male, n (%)	57	53	NS
BSA, m²	1.81±0.3	1.81±0.2	NS
At the postoperative 120th min			
HR/min	87 (77-96)	97 (82-109)	0.045
MAP, mmHg	81 (73-90)	77 (66-90)	0.043
pH	7.36 (7.34-7.40)	7.34 (7.32-7.37)	**<0.001**
PaO_2, mmHg	122 (84-164)	123 (80-152)	NS
$PaCO_2$, mmHg	39.0 (35.0-42.0)	39.0 (36.0-42.0)	NS
HCO_3, mmol/L	22.0 (21.0-23.4)	21.0 (20.0-22.3)	**<0.001**
SBE, mmol/L	-2.2 (-3.9 ; -0.8)	-3.0 (-5.0; -1.8)	0.003
SaO_2, %	99 (97-100)	99 (97-100)	NS
SvO_2, %	68±9	39±7	**<0.001**
Hb, g/dL	10.0 (9.0-11.3)	9.9 (9.0-11.1)	NS
Hct, %	31.8 (28.0-35.4)	30.2 (27.8-34.5)	NS
Lactate, mmol/L	1.3 (1.0-1.7)	5.5 (5.2-6.0)	**<0.001**
AG, mmol/L	8.4 (4.5-11.5)	11.9 (7.2-14.0)	**<0.001**
Glucose, g/dL	149 (133-170)	189 (162-210)	**<0.001**
Temperature, °C	36.4 ± 0.8	35.7 ± 0.7	**0.010**

CI: Confidence interval; BSA: Body surface area; HR: Heart rate; MAP: Mean arterial pressure; PaO_2: Partial pressure of oxygen; $PaCO_2$: Partial carbon dioxide pressure; HCO_3: Bicarbonate; SBE: Standard base-excess; SaO_2: Arterial blood oxygen saturation; SvO_2: Central venous oxygen saturation; Hb: Hemoglobin; Hct: Hematocrit; AG: Anion gap.

postoperative phase can predict poor prognosis.[16,17] Typically, hyperlactatemia is associated with low cardiac output. However, it can also be caused by several other factors, including septic shock, severe anemia, hepatic or mesenteric ischemia, the usage of Ringer's Lactate solution, renal failure, and the side effects of several drugs.[18–20]

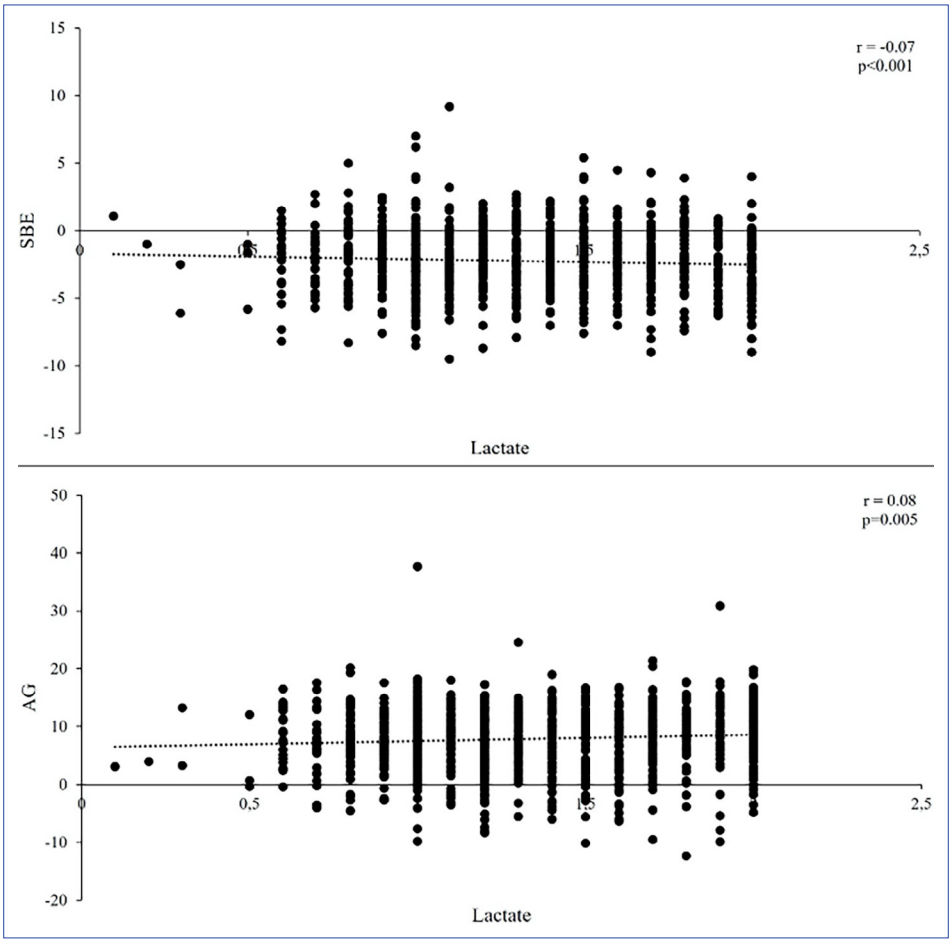

Figure 1. SBE&lactate and AG&lactate correlations in Group 1 (CI ≥1.8 L/min/m²) at the postoperative 120th minimum.

SBE: Standard base-excess; AG: Anion gap; CI: Confidence interval.

Non-pulsatile flow, dilutional anemia, hypothermia, and the inhomogeneous rewarming period during ECC can negatively affect microcirculation. This can lead to partial hypoperfusion and lactate accumulation in the tissue.[5,14] In the early postoperative period, with homogeneous warming, pulsatile flow, and increased perfusion, lactate produced during the operation passes into the bloodstream. This results in early postoperative hyperlactatemia, which occurs without cardiac output failure and is known as 'washout hyperlactatemia'.[15,19–21]

To provide the correct treatment, it is crucial to distinguish between two types of hyperlactatemia - "washout hyperlactatemia" and hyperlactatemia caused by low cardiac output. If a low cardiac output is detected, immediate action must be taken to increase it. This can be achieved through the administration of fluids or blood products, positive inotropic drugs, vasodilator drugs, vasopressor drugs, or other suitable measures. Delaying treatment for low cardiac output can lead to the patient's condition worsening due to inadequate compensation mechanisms, such as an increase in heart rate, systemic vascular resistance, and oxygen extraction rate, which can result in irreversible damage, organ failure, and even death.[16] Nevertheless, in both cases, the blood pressure, heart rate, hemoglobin level, and oxygen saturation values may remain consistent with the expected values after open-heart surgery.

Cardiac output monitoring can be achieved using invasive or semi-invasive methods, such as pulmonary artery catheterization. These techniques help assess mixed venous oxygen saturation, central venous oxygen saturation, and oxygen extraction ratio. While these parameters are believed to be similar, they each have their limitations. Therefore, it is recommended that all parameters be evaluated together whenever possible.[1]

Blood gas analysis is a crucial method of monitoring during open-heart surgery. This process helps track the gas exchange in the lungs and the levels of hemoglobin and electrolytes. SBE is a parameter obtained from blood gas analysis, and it might help differentiate the diagnosis of certain conditions. SBE can be calculated by measuring basic parameters in blood gas analysis using the formula:[22] $SBE=0.9287\times(HCO_{3^-} - 24.4+14.83\times(pH-7.4))$.

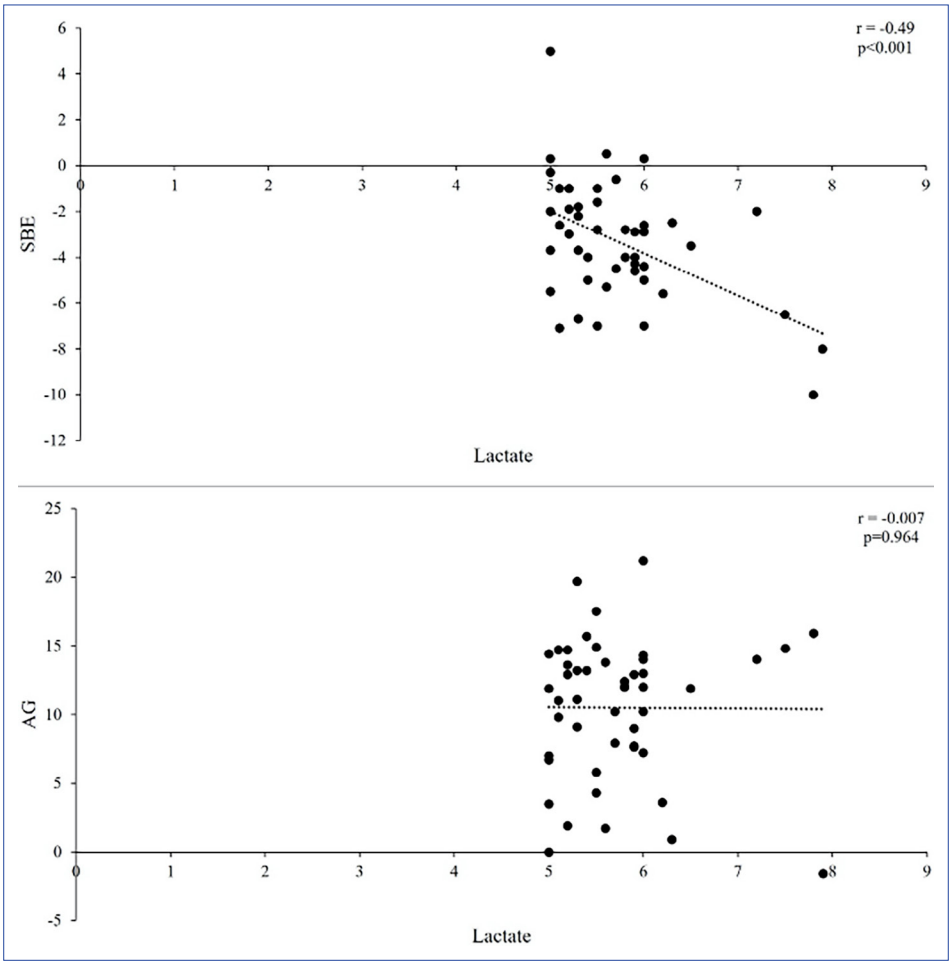

Figure 2. SBE&lactate and AG&lactate correlations in Group 2 (CI ≥1.8 L/min/m^2) at the postoperative 120th minimum.

SBE: Standard base-excess; AG: Anion gap; CI: Confidence interval.

The Anion Gap (AG) is a measure that shows the difference between the positively charged ions (cations) like Na^+ and K^+ and the negatively charged ions (anions) such as Cl- and HCO_3-. It is used to identify electrolyte imbalances, detect paraproteins like IgG, and evaluate suspected acid-base disorders. A typical range for an anion gap is 4–12 mmol/L. If the value is higher than this range, it indicates the presence of an unmeasurable anion in the environment. The formula for the anion gap is as follows:[8] Anion Gap=[UA] - [UC] = ([Na^+] + [K^+]) - ([Cl-] + [HCO_3-]). High levels of lactate in plasma are one of the causes of high anion gap metabolic acidosis.

We hypothesized that in 'washout hyperlactatemia' cases, metabolic acidosis would not worsen due to preserved cardiac output, SBE would not be negative, and there would be no significant correlation between SBE and lactate.

We conducted a study to confirm our hypothesis that there is a significant correlation between plasma lactate levels and SBE. Although the data did not thoroughly verify our hypothesis, it was very close. We observed a statistical significance between plasma lactate level and SBE in Group 1, who did not have low cardiac output. However, the correlation was weak (r=-0.07, p<0.001, Fig. 1), which might be because SBE measurement depends on other factors, such as plasma chloride and albumin levels and unmeasurable anions that can affect the base deficit.

We observed a 7-fold stronger correlation between SBE and lactate in Group 2 patients with hyperlactatemia caused by low cardiac output (r=-0.49, p<0.001, Fig. 2). Group 2 had considerably higher lactate levels (5.5 (5.2–6.0) mmol/L) than those in Group 1 (1.3 (1.0–1.7) mmol/L) (p<0.001, as indicated in Table 2). These results suggest a stronger correlation between SBE and lactate in patients with low cardiac output.

A study conducted by Noritomo et al.[23] examined the connection between SBE and lactate in critically ill patients. The study's results revealed a weak correlation between these two variables, which aligns with our analysis. The study also identified some conditions like electrolyte disorders and hypoalbuminemia that can cause changes in SBE. Moreover, the study emphasized that such variables can affect SBE independently of lactate. This finding is

not surprising since the SBE formula is structured so that lactate levels significantly influence it in patients with hyperlactatemia. The correlation between lactate and SBE is particularly noticeable in patients with high lactate levels.

After considering the limitations discussed and analyzing the data, the correlation between SBE and plasma lactate levels is expected to be higher in patient groups with low cardiac output. Plasma lactate levels dominate the SBE equation, and metabolic compensation is insufficient to counteract the mechanism causing hyperlactatemia in these patients.

While SBE can help diagnose metabolic acid-base disorders, it does not provide insight into the underlying cause. As a result, SBE may change inversely with an increase in lactate or due to accompanying electrolyte or metabolic disorders.[24,25]

We also examined the relationship between lactate and AG levels in two patient groups. In Group 1, we found a weak correlation between the two variables, with statistical significance (r=0.08, p=0.005, Fig. 1). However, in Group 2, we didn't observe any significant correlation between lactate and anion gap values (r=-0.007, p=0.964, Fig. 2).

AG is calculated by substituting the measured parameters and is a variable that can only partially help in the differential diagnosis of metabolic acidosis.[7] This weak correlation in Group 1 patients, where cardiac output is preserved, suggests that hyperlactatemia may contribute to high AG but cannot alone explain it. Moreover, concurrent disorders such as hyperchloremia or hypoalbuminemia affect AG; such electrolyte disorders and metabolic conditions need to be taken into account when interpreting AG levels, as they can significantly affect the diagnosis of metabolic acidosis.[26]

We found no connection between lactate levels and AG in Group 2 patients with low cardiac output. This lack of correlation could be due to various factors, such as metabolic disorders or electrolyte imbalances arising from acute kidney injury or fluid therapy. Moreover, the changes in AG levels may be unpredictable under such circumstances. It is also possible that the limited sample size we used to evaluate AG parameters is responsible for the absence of any significant correlation.

Conclusion

Relying solely on SBE (standard base excess) and anion gap to distinguish hyperlactatemia is limited because they depend on various variables. We suggest examining hyperlactatemia by assessing the patient's clinical condition, other tissue perfusion parameters, flow measurements, plasma chloride, and albumin values. Although the correlation between SBE and lactate can indicate hyperlactatemia caused by low cardiac output, it is not a definitive indication. Lack of correlation does not necessarily exclude the low output condition or support 'washout hyperlactatemia.'

Disclosures

Ethics Committee Approval: The study was approved by The Acıbadem Mehmet Ali Aydınlar University Ethics Committee (no: 2024-4/158, date: 14/03/2024).

Authorship Contributions: Concept – S.A.Y., M.T., B.G., F.T.; Design – S.A.Y., M.T., B.G., F.T.; Supervision – S.A.Y., M.T., B.G., F.T.; Materials – S.A.Y.; Data collection &/or processing – S.A.Y.; Analysis and/or interpretation – B.G., M.T.; Literature search – M.T., F.T.; Writing – M.T., F.T.; Critical review – S.A.Y., M.T., B.G., F.T.

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