Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.285188.3353 Volume 30, Issue 4, July 2024
Manuscript ID ZUMJ-2404-3353
DOI 10.21608/ZUMJ.2024.285188.3353
REVIEW ARTICLE

Hyperlactatemia and Coronary Artery Bypass Grafting

Gehad Saeed Bayoumy Fath Al-Bab Ibrahim [1*], Mamdouh ElSharawy [1], Karim ElFakharany [1], Dina Ashraf Abdelhady [2], Dina Said Shemais [1]
[1] Cardiothoracic Surgery Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt
[2] Clinical Pathology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt

***Corresponding author:**
Gehad Saeed Bayoumy Fath Al-Bab Ibrahim

Email:
dr.gehad1996@gmail.com

Submit Date 25-04-2024
Accept Date 10-05-2024

ABSTRACT

Background: Surgical revascularization is becoming more and more necessary for high-risk and elderly individuals with complicated coronary artery disease. After coronary artery bypass grafting (CABG), there is a substantial risk of both morbidity and death from cerebrovascular damage. The surgical approach used for CABG, and in particular the degree of aortic manipulation, has frequently been associated with the etiology of postoperative neurological problems, despite the possibility of several contributing factors. When revascularization (CABG) is necessary for coronary artery disease, the aortic clamping technique (SCT\DCT) is used. This approach affects the blood lactate level, which can lead to many postoperative problems. Elevated blood lactate level after cardiac surgery is an indicator of systemic hypoperfusion and tissue hypoxia. **Aim of the review**: to evaluate the relationship between postoperative blood lactate levels and outcome in patients undergoing open heart surgery and to verify the clinical impact of hyperlactatemia (HL) and low lactemia (LL) after coronary artery bypass grafting (CABG**)** in terms of postoperative morbidity and mortality rate.

Keywords: Hyperlactatemia; CABG; Cardiac Surgery

INTRODUCTION

Monitoring lactate levels during CABG surgery can provide important information about tissue oxygenation and can help guide the management of the patient. Research has demonstrated that higher lactate levels following CABG surgery are linked to a higher likelihood of postoperative complications and mortality. Therefore, lactate levels are often monitored during CABG surgery to help ensure that tissue oxygenation is maintained at a sufficient level. This can be done by periodically measuring lactate levels in arterial blood samples. If lactate levels are found to be elevated, steps can be taken to improve tissue oxygenation, such as increasing oxygen delivery or improving cardiac output **[1],[2].** Lactate is produced by the body as a byproduct of anaerobic metabolism, which occurs when there is insufficient oxygen available to meet the energy demands of the cells. The pathway of lactate production is called anaerobic glycolysis, and it takes place in the cytoplasm of the cells **[3].** The process of anaerobic glycolysis can be summarized in the following steps: Glucose is transported into the cell and converted to hexokinase, an enzyme that produces glucose-6-phosphate. Phosphohexose isomerase is the enzyme that changes glucose-6-phosphate into fructose-6-phosphate. The conversion of fructose-6-phosphate is to 2 molecules of pyruvate through a series of enzymatic reactions that involve enzymes such as aldolase and enolase. Pyruvate is then converted to lactate by the enzyme lactate dehydrogenase (LDH). This reaction also involves the transfer of hydrogen ions from NADH to pyruvate, forming NAD+. In the first stage of the Cori cycle, lactate dehydrogenase converts pyruvate to lactate as a substitute mechanism for regenerating NAD+, which is needed for further glycolysis. After glycolysis, lactate is created from pyruvate in a 10:1 ratio, which is subsequently transformed in the liver (the second half of the Cori cycle) to regenerate glucose under baseline physiological conditions with a normal oxygen tension **[4].** Lactate production will rise under conditions that promote glycolysis, but the lactate–pyruvate ratio will not change. When oxygen

supply cannot keep up with tissue demands, pyruvate builds up and oxidative phosphorylation, or the electronic transport chain, which supports aerobic metabolism, fails, greatly increasing the rate at which lactate is produced. Skeletal muscles will produce lactate to support glycolysis during times of high metabolic demand, such as exercise. The muscles will then oxidize the lactate during the recovery phase. Pyruvate, the byproduct of glycolysis, is the only source of lactate. Lactate can be oxidized into glucose by gluconeogenesis, which can then turn it back into pyruvate **[4].** Gluconeogenesis (**part of the Cori cycle**) mostly takes place in hepatocytes, whereas oxidation happens in the skeletal muscles and renal cortex. Thirty percent of lactate is metabolized by the kidney, compared to sixty percent by the liver. The balance of production, conversion, and clearance is reflected in the serum lactate level. Lactate can build up through aerobic metabolism with increased glycolysis, anaerobic metabolism during tissue hypoxia, and impaired lactate conversion due to pyruvate dehydrogenase inhibition or relative deficiency **[2].** The production of lactate during anaerobic glycolysis is an important mechanism for the body to generate energy when the oxygen supply is limited. It allows the cells to continue to produce ATP, which is the energy currency of the body, even when there is not enough oxygen available to support aerobic metabolism. However, if lactate production continues for an extended period, it can lead to tissue acidosis and cellular damage **[2].** Cardiomyocytes can use lactate when it is abundant, but they mostly employ glucose and fatty acids for energy production. The heart uses lactate as fuel, but lactate consumption falls during and after surgery, even as lactate production rises. For this reason, hyperlactatemia might be an adaptive rather than detrimental response. Research indicates that during shock conditions, the heart uses lactate as its main energy source. In an animal model, inhibiting the heart's ability to use lactate resulted in cardiac decompensation and death **[4].**

TYPES OF HYPERLACTATEMIA

- ***Type A hyperlactatemia*:** Results from insufficient perfusion, while type B hyperlactatemia is caused by adequate perfusion.
- ***Type A Lactic Acidosis:*** Because of its link to metabolic acidosis, elevated lactate in the presence of insufficient perfusion has historically been referred to as type A lactic acidosis. The typical environment for type A lactic acidosis is anaerobic metabolism. Insufficient perfusion may manifest as regional, global, or microcirculatory. Low cardiac output syndrome and uncompensated shock state are two instances of systemic insufficient perfusion. Limb ischemia is one instance of regional or local malperfusion. Because pyruvate dehydrogenase is inhibited and pyruvate is diverted to lactate generation in the situation of hypoxia, type A lactic acidosis will increase the lactate–pyruvate ratio. type A lactic acidosis appears to originate from the bypass itself **[5].** In the myocardium and peripheral tissues, cardiopulmonary bypass itself raises lactate and lactate-pyruvate levels. This is linked to varying inflammatory, microcirculatory, and mitochondrial responses during hypothermia and cardiopulmonary bypass, and it is secondary to insufficient delivery despite calculated perfusion needs. It is unknown which tissue beds—the heart, lungs, intestines, and skeletal muscles—contribute considerably to serum lactate production during bypass. Furthermore, cardiopulmonary bypass induces a strong inflammatory response throughout the body, resulting in the production of free radicals and reactive oxidative species **[5].** These alterations lead to mitochondrial dysfunction by reducing their ability for oxidative phosphorylation, which shunts pyruvate to lactate and causes type B hyperlactatemia when combined with any ischemia/reperfusion event. Moreover, endogenous catecholamine release triggered by the stress response of cardiac surgery raises glycolysis and gluconeogenesis, which in turn increases the generation of glucose and lactate **[2], [5].**
- ***Type B hyperlactatemia* (Figure 1):**
Elevated lactate in the setting of adequate perfusion **[6]**, no metabolic acidosis coexists so type b hyperlactatemia is better than type B lactic acidosis as an expression. Stress conditions that promote glycolysis will produce more lactate but keep the lactate–pyruvate ratio at 10:1 because pyruvate will be balanced but converted into lactate by the Krebs cycle. When type B hyperlactatemia is present, the patient is usually less acidemic than anticipated (a normal pH may even be present, depending on the severity of hyperchloremia or bicarbonate deficiency), warm and well perfused upon examination, end-organ function (urine output, mentation) is adequate, and the arteriovenous oxygen (A-VO2) difference is normal or narrow **[5], [7].** Hyperglycemia is related to hyperlactatemia. In addition to exogenous hormones, the stress response triggers gluconeogenesis and catabolism. Serum glucose functions as a stand-alone biomarker of the stressed state. Despite enhanced tissue perfusion and greater cardiac output, exogenous adrenaline raised lactate

levels and was linked to hyperglycemia in sepsis patients. Since insulin stimulates pyruvate dehydrogenase, hyperlactatemia is a contributing factor to insulin resistance. It is hypothesized that insulin resistance following surgery raises serum lactate levels even more **[8].** Diabetes mellitus significantly reduces the glucose-lactate connection. Given that individuals with diabetes are more likely to experience hyperglycemia and that diabetes should thus apparently have a favorable influence on lactate levels, this may appear counterintuitive. However, the lactate level was suppressed by DM. Greco and colleagues examined the relationship between hyperlactatemia, glucose, and diabetes in 4098 patients following heart surgery and discovered that patients with DM had a strong attenuation of the stress-induced increase in lactate levels, which is why postoperative lactate levels are significantly attenuated by diabetes mellitus. Furthermore, in multiple studies where the correlation between hyperglycemia and unfavorable outcomes is significantly repressed in DM patients, the suppressive impact of DM on stress hyperlactatemia has also been found with stress hyperglycemia **[9], [10].**

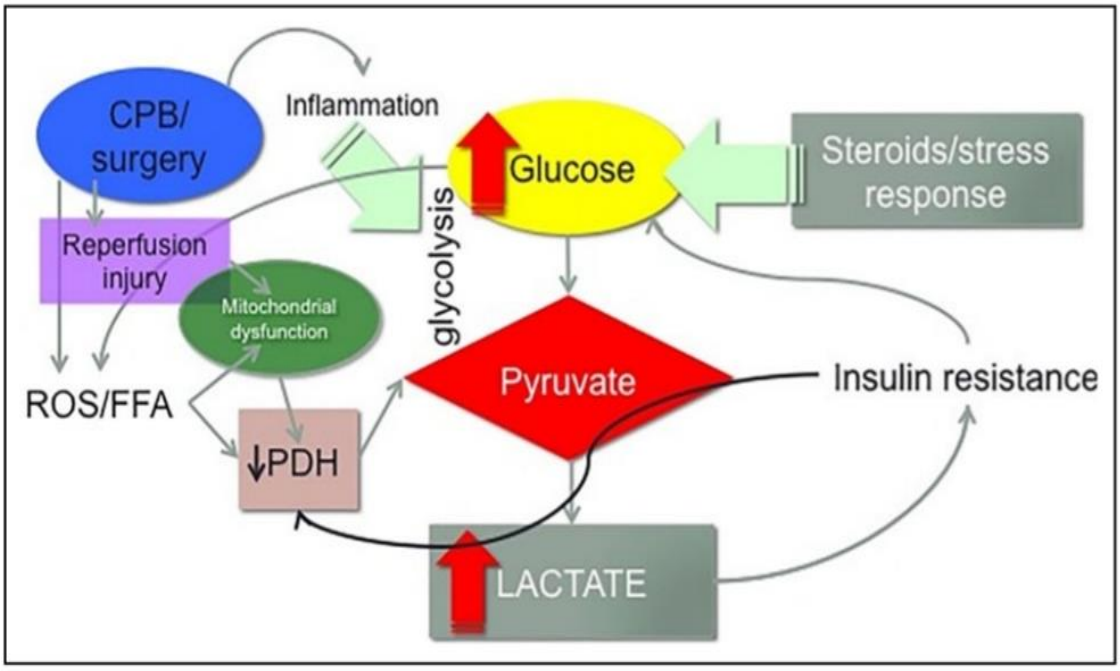

Figure (1): Contributing factors to hyperlactatemia and hyperglycemia in type B lactic acidosis. CPB indicates cardiopulmonary bypass; PDH, pyruvate dehydrogenase; ROS/FFA, reactive oxygen species/free fatty acids **[9], [10].**

➢ ***Another classification of hyperlactatemia:***

Elevated postoperative lactate has been described based on the timing of onset (early or late). Early onset refers to the period from bypass to admission to the intensive care unit (ICU), and late-onset is the period from 6 to 12 hours following ICU admission. Elevations in lactate that occur early in life have been linked to higher death rates. **Maillet et. al (2003)** found that in two studies involving adult cardiac surgery patients, patients with a lactate level >3 mmol/L at the time of ICU admission had a 14.9% mortality rate, compared to 1.5% in those with a lactate level of 3 mmol/L during cardiopulmonary bypass. This finding was linked to higher intra-aortic balloon pump usage, longer postoperative mechanical ventilation, and longer ICU stays **[2].**

Blood lactate levels between 0.4 and 2.0 mmol/L are considered normal. Blood lactate levels exceeding 4.0 mmol/L are categorized as severe HL, while levels between 2 and 4.0 mmol/L are regarded as mild to moderate HL. Hyperlactatemia that manifested early, but not later, was linked to higher rates of morbidity and death. Research has also shown that the intricacy of the treatment, the duration of cardiopulmonary bypass, and the blood glucose level are significant indicators of elevated risk of endocarditis. Crucially, following heart surgery, late hyperlactatemia (LHL) was quite prevalent, benign, and self-limiting **[9], [10], [11].**

FACTORS CONTRIBUTING TO HYPERLACTATEMIA

Rather than being merely perceived as a measure of tissue hypoxia, hyperlactatemia is best viewed as a

biomarker of the multifactorial stress response. Even in settings with adequate cardiac output and tissue perfusion, a number of additional factors, including systemic inflammation, fever, and tissue damage that drive cortisol and adrenergic signaling, contribute to the stress response in addition to tissue hypoxia **[12].** The cause of post-cardiac surgery hyperlactatemia is complex. Hyperlactatemia may manifest even in the absence of hypoperfusion of the tissues. The cellular metabolism may be negatively impacted by the anesthetics and low temperatures during CPB, which can result in a decreased liver clearance of lactate **[12].** Furthermore, because catecholamines affect oxidative glucose metabolism, their use during heart surgery may raise lactate levels **[13].** Stress conditions change both glycolysis and the mitochondrial flow of pyruvate independent of tissue hypoxia by raising endogenous catecholamines and cortisol. Pretreatment with α- and β adrenergic blockers reduced lactate buildup in an animal model of hemorrhagic shock. Further research bolsters the idea that β2 adrenergic receptor stimulation could be the cause of hyperlactatemia **[14].** Using epinephrine infusion to stimulate adrenergic pathways and adrenergic blockade, then to stop them, a role for the adrenergic system has been confirmed in shock states. Even in situations with enough oxygen, stress causes the release of endogenous catecholamines (such as adrenaline), which stimulate the action of Na+/K-ATPase and raise lactate generation. In adults suffering from septic shock, Levy et al.'s study showed that suppression of Na+/K- ATPase prevented increases in lactate and pyruvate. These findings provide evidence that adrenergic signaling, which is activated under stressful situations (heart surgery, for example), promotes glycolysis and indirectly lactate formation **[4], [15].** Hyperglycemia and postoperative epinephrine injection were separate risk factors for LHL **[16].** Raper et al. found that following heart surgery, hyperglycemia and adrenaline were usually linked to a lactate concentration of 5 mmol/L. It is commonly recognized that β2-agonists can cause severe lactic acidosis in individuals suffering from acute asthma, and adrenaline possesses a potent β2 mimetic effect that leads to heightened gluconeogenesis, lipolysis, and glycogenolysis **[16], [17].** Elevated levels of free fatty acids inside cells prevent pyruvate from being converted to acetyl-coenzyme A, which increases the generation of lactic acid. Additionally, activation of β-adrenergic receptors increases the concentration of plasma glucose, which in turn increases the substrate available for glycolysis **[16].** Postoperative hyperglycemia highlights the possible function of adrenaline in initiating the onset of hyperlactatemia and was a potent independent risk factor linked to LHL. Hyperglycemia can cause hyperlactatemia and is often linked to normothermic CPB. Patients undergoing heart surgery who exhibit normal hemodynamics have been found to have elevated endogenous glucose levels, which have been linked to the endogenous release of stress hormones and cytokines that cause insulin resistance. Chioléro et al **[9], [18].** In individuals experiencing cardiogenic shock following heart surgery, hyperglycemia, and elevated nonoxidative glucose availability may play a major role in hyperlactatemia. Even yet, for ICU patients, the lactate concentration is a reliable indicator of the severity of the illness. Its predictive significance following myocardial infarction has not been satisfactorily established. Generally speaking, mild hyperlactatemia is regarded as benign **[9].** Extended periods of CPB were also linked to IHL (immediate hyperlactatemia upon ICU admission). Additionally linked to postoperative hyperlactatemia, non pulsatile CPB has been implicated in regional hypoperfusion. An intraoperative oxygen debt may have resulted from the patient's inability to wean off of CPB, as intraoperative vasopressor use was also found to be an independent risk factor for IHL and to represent intraoperative hemodynamic instability **[19].**

Preoperative Risk Factors: **Demers et al. (2000)** postulated that the hyperlactatemia observed during CPB is caused by a variety of preoperative variables and co-morbidities. They found that risk variables for hyperlactatemia were age, congestive heart failure, poor left ventricular ejection fraction, hypertension, diabetes mellitus, reexamination, and emergency procedures **[11].** Cardiopulmonary bypass (CPB) tissue hypoxia may result from hemodialysis, inefficient peripheral oxygen delivery, and hemodynamic instability, all of which are associated with increased postoperative morbidity and death. After CPB, hyperlactatemia usually happens more frequently in operations that call for longer CPB durations. A peak postoperative lactate of ≥3.0 mmol/L was predicted by two parameters, which were identified. Preoperative IABP implantation and severe renal damage following surgery **[14].**

Complications of Hyperlactatemia: Following cardiac surgery, several anesthetic, surgical, and postoperative parameters, as well as the patient's preoperative status, are directly linked to morbidity and death **[20].** Patients undergoing cardiac surgery

with CPB may experience hyperlactatemia (HL) for a variety of reasons, including tissue hypoxia, CPB itself, nonhypoxic factors such as medication therapy, cardioplegia solution, and hypothermia. According to a recent study by Demers et al., HL is identified in 10–20% of adult patients undergoing cardiac surgery and is linked to a considerable increase in postoperative morbidity and death. More than 88% of patients in research by Broder and Weil who had blood lactate levels more than 4.0 mmol/L were at high risk of dying from vascular shock. The same scientists found that as blood lactate levels rise from 2.0 to 8.0 mmol/L, the likelihood of surviving shock drops from 90% to 10% **[10], [11].** There is established evidence linking HL and lactic acidosis to higher rates of morbidity and death. A negative prognostic factor is a blood lactate concentration greater than 3.5 mmol/l at the time of transfer to the intensive care unit. Elevated blood lactate levels in the intensive care unit following heart surgery are often viewed as a metabolic illness. Serial lactate measurements may help identify patients who are at high risk of developing mortality and morbidity and enable appropriate preventive interventions to be implemented. It's still unclear if a high intraoperative blood lactate concentration during perfusion is a sign of a bad result. Prior research has not linked HL during perfusion to long-term mortality, whereas peak blood lactate levels during CPB≲2.0mmol/L are linked to worse surgical outcomes. Elevated blood lactate levels during the first hour of surgery (early HL) and six hours after surgery (late HL) were found to be highly indicative of significant cardiac complications, including atrial fibrillation and the need for extended inotropic support. -Compared to individuals with serum lactate levels <4 mmol/L, those with levels >4 mmol/L had a 5.66 times higher risk of requiring extended inotropic support and a 3.5 times higher risk of developing AF. A number of factors, including intraoperative factors such as surgical procedures, myocardial protection, and metabolic disruption under continuous positive pressure heart rate, influence the outcome of heart surgery. Keeping an eye on these variables during surgery could result in timely therapeutic intervention that could enhance surgical outcomes **[6].** IABP may be required more frequently during surgery, particularly in patients with EF<40%. Complex surgeries, including aortic root replacement, intracardiac repairs, and revision procedures, have been linked to elevated serum lactate levels. length of hospital and intensive care unit stays for HL patients. When compared to patients with lactate <4 mmol/L, patients with peak serum lactate ≥4 mmol/L had a significantly longer stay in the intensive care unit and hospital. Raised lactate levels have the potential to indicate tissue hypoxia and are linked to a higher risk of organ failure, extended mechanical breathing, and even death following surgery. As a result, keeping an eye on lactate levels during CPB can assist inform treatment decisions and reveal important details regarding tissue oxygenation **[21].**

Observed Complications as Recorded by Some Studies: A greater EuroSCORE, a lower left ventricular ejection fraction, a lower preoperative hemoglobin level, a higher incidence of renal illness, and a history of blood transfusions are among the predisposing factors associated with patients who experience problems **[22].** To have an additional intervention for bleeding or re-exploration, pericardial effusion, perforated intestine, intestinal ischemia/obstruction, peripheral ischemia, and left ventricular assist device, return to the operating room (OR) **[23].** The individuals with hyperlactatemia were more likely to utilize IABP. There was also a noticeably increased risk of sequelae such as acute renal damage, heart bleeding, and cardiogenic shock. When serum creatinine levels were compared, it was discovered that patients presenting with lactate >3.0 mM had higher peak postoperative creatinine values, further characterizing renal impairment **[23].** Measured in terms of length of ICU stay, duration of mechanical ventilation, and hospital mortality, patients with hyperlactatemic conditions fared worse, spending more time in the ICU, more time on mechanical ventilation, and experiencing higher hospital mortality **[24].** Higher rates of postoperative surgical procedures, IABP, and sequelae such as cardiogenic shock, cardiac bleeding, and renal failure were linked to mortality **[25].** Patients with pre-existing heart failure, hypertension, chronic obstructive pulmonary disease (COPD), and renal failure were more likely to not make it out alive. Non-survivors also showed a marked rise in the need for postoperative IABP, extended cardiopulmonary bypass (CPB) times, and emergent surgery. Higher peak lactate levels and later times to peak lactate levels were linked to death. Thirty hours was the 90th percentile for the time to peak lactate. The 90th percentile was exceeded by a noticeably higher percentage of non-survivors: lactate peak was achieved at >30 h in 64.7% of mortalities. According to certain research, survivors reached their maximal lactate in 7.5 hours, while non-survivors took an average of 37.6 hours. It was observed that these variations affected

renal function. In non-survivors, acute renal injury was more common. Additionally higher were the peak creatinine and the % increase in postoperative creatinine. Mortality was linked to prior renal failure, severe peripheral vascular disease, age, congestive heart failure, and reoperation **(26).**

Preventive Measures: Several tactics can be used to reduce the possibility of tissue hypoxia and lactate generation during CPB. For instance, keeping the patient's temperature within a specific range can aid in lowering the tissues' oxygen requirement and metabolic rate. Tissue hypoxia and lactate generation can also be lessened by making sure there is sufficient tissue perfusion and limiting the length of CPB. Increased morbidity and mortality in the adult cardiac surgery population have been linked to HL during CPB. According to earlier research, a strategy of hemodynamic optimization that targets a serum lactate level under 3 mmol/L in the first 6 hours and under 2 mmol/L in the first 12 hours of ICU admission in patients with early hyperlactatemia may lower the incidence of major complications. This strategy also takes into account other parameters like cardiac index, calculated oxygen delivery, and central venous oxygen saturation. Adjusting the way heart surgery is managed. For non-elective surgery in particular, hemodynamic stabilization before CPB may reduce or eliminate IHL. For high-risk patients, this may include using an intra-aortic balloon pump support more frequently **[27].** Restricting pump hypotension could help prevent possible visceral oxygen debt during surgery. Enhancing myocardial protection might help avoid or lessen postoperative poor cardiac output and make it easier to wean off of CPB. Even for non-diabetic patients, a rigorous surveillance strategy for postoperative hyperglycemia and treatment with continuous IV insulin therapy should enhance prognosis **[28].** Furthermore, norepinephrine and dobutamine may be given to hypotensive patients who have retained left ventricular function but are not responding to fluid management, and first-line epinephrine medication should be reevaluated **[29].**

REFERENCES

1. Duval B, Besnard T, Mion S, Leuillet S, Jecker O, Labrousse L, Rémy A, Zaouter C, Ouattara A. Intraoperative changes in blood lactate levels are associated with worse short-term outcomes after cardiac surgery with cardiopulmonary bypass. Perfusion. 2019 Nov;34(8):640-50.
2. Stephens EH, Epting CL, Backer CL, Wald EL. Hyperlactatemia: an update on postoperative lactate. World J. Pediatr. Congenit. Heart Surg... 2020 May; 11(3):316-24.
3. Sarkar M, Prabhu V. Basics of cardiopulmonary bypass. Indian J Anaesth. 2017 Sep 1;61(9):760-7.
4. Yılmaz Ş, Yurtseven N, Tuygun AK, Yavuz Y, Canik S. Hyperlactatemia after coronary artery bypass surgery: risk factors and effect on mortality. Turkish J Thorac Cardiovasc Surg. 2011 Jan 1;19(1):30-5.
5. Redant S, Hussein H, Mugisha A, Attou R, Bels DD, Honore PM, Laet CC. Differentiating hyperlactatemia type A from type B: how does the lactate/pyruvate ratio help?. J transl med. 2019 Jul 11;7(2):43-5.
6. Hawkes, C. Uneven dietary development: linking the policies and processes of globalization with the nutrition transition, obesity, and diet-related chronic diseases. Global health. 2006; 2: 1-18.
7. Al Maqrashi Z, Sedarous M, Pandey A, Ross C, Wyne A. Refractory hyperlactatemia and hypoglycemia in an adult with non-Hodgkin's lymphoma: a case report and review of the Warburg effect. Case Reports in Oncology. 2021 Jul 30;14(2):1159-67.
8. Demir ZA, Balcı E, Özay HY, Bahçecitapar M. Intraoperative Hyperglycemic Stress Response and Oxygen Extraction Ratio in Cardiac Surgery. Journal of the Society of Thoracic Carido-Vascular Anaesthesia & Intensive Care. 2022 Sep 1;28(3).
9. Algarni KD. The effect of hyperlactatemia timing on the outcomes after cardiac surgery. The Cardiothoracic Surgeon. 2020 Dec;28:1-8.
10. Faizal A, Raman AP, Ravikumar TD. Proportion of Hyperlactatemia during Cardiopulmonary Bypass and its Impact on Post-operative Outcomes in Coronary Artery Bypass in a Tertiary Care Centre.
11. Demers P, Elkouri S, Martineau R, Couturier A, Cartier R. Outcome with high blood lactate levels during cardiopulmonary bypass in adult cardiac operation. Ann Thorac Surg. 2000 Dec 1;70(6):2082-6.
12. Matteucci M, Ferrarese S, Cantore C, Cappabianca G, Massimi G, Mantovani V, Rossi MB, Beghi C. Hyperlactatemia during cardiopulmonary bypass: risk factors and impact on surgical results with a focus on the long-term outcome. Perfusion. 2020 Nov;35(8):756-62.
13. Bellomo R. Bench-to-bedside review: lactate and the kidney. Crit care. 2002 Aug;6:1-5.
14. Daniel III WT, Kilgo P, Puskas JD, Thourani VH, Lattouf OM, Guyton RA, Halkos ME. Trends in aortic clamp use during coronary artery bypass surgery: effect of aortic clamping strategies on neurologic outcomes. J Thorac Cardiovasc Surg. 2014 Feb 1;147(2):652-7.

15. Naik R, George G, Karuppiah S, Philip MA. Hyperlactatemia in patients undergoing adult cardiac surgery under cardiopulmonary bypass: Causative factors and its effect on surgical outcome. Ann Card Anaesth. 2016 Oct 1;19(4):668-75.

16. Farogh A, Younas A, Hassan A, Ahmed N, Bashir T, Iftikhar S, Afshan S. Base Excess or Hyperlactatemia: Identifying the Superior Risk Factor for Increased Morbidity and Mortality in Cardiac Surgery. Pak J Med Health Sci. 2022 May 13; 16(04):263-.

17. Manthous CA. Lactic acidosis in status asthmaticus: three cases and review of the literature. Chest. 2001 May 1;119(5):1599-602.

18. Chioléro RL, Revelly JP, Leverve X, Gersbach P, Cayeux MC, Berger MM, Tappy L. Effects of cardiogenic shock on lactate and glucose metabolism after heart surgery. Crit Care Med. 2000 Dec 1;28(12):3784-91.

19. Aktar N, Asif Rahim AM, Shahidullah AY, Iftekher Hossain AH, Ahmed F. Blood lactate level as predictor of early outcome after off-pump coronary artery bypass grafting. J Cardiovasc Med. 2020 Jan 1;12:120-5.

20. Maillet JM, Le Besnerais P, Cantoni M, Nataf P, Ruffenach A, Lessana A, Brodaty D. Frequency, risk factors, and outcome of hyperlactatemia after cardiac surgery. Chest. 2003 May 1;123(5):1361-6.

21. Chambers KA, Park AY, Banuelos RC, Darger BF, Akkanti BH, Macaluso A, Thangam M, Doshi PB. Outcomes of severe sepsis and septic shock patients after stratification by initial lactate value. World J Emerg Med. 2018;9(2):113.

22. Hajjar LA, Almeida JP, Fukushima JT, Rhodes A, Vincent JL, Osawa EA, Galas FR. High lactate levels are predictors of major complications after cardiac surgery. The J Thorac Cardiovasc Surg. 2013 Aug 1;146(2):455-60.

23. de Varennes B, Lachapelle K, Cecere R, Ergina P, Shum-Tim D, Tchervenkov C, Sampalis J. Application of the Parsonnet scoring system for a Canadian cardiac surgery program. Canad J Cardiol. 2007 Nov 1;23(13):1061-5.

24. McNelis J, Marini CP, Jurkiewicz A, Szomstein S, Simms HH, Ritter G, Nathan IM. Prolonged lactate clearance is associated with increased mortality in the surgical intensive care unit. Am J Surg.. 2001 Nov 1;182(5):481-5.

25. Ranucci M, Isgrò G, Romitti F, Mele S, Biagioli B, Giomarelli P. Anaerobic metabolism during cardiopulmonary bypass: predictive value of carbon dioxide derived parameters. Ann Thorac Surg. 2006 Jun 1;81(6):2189-95.

26. Okorie ON, Dellinger P. Lactate: biomarker and potential therapeutic target. Crit Care Clin.. 2011 Apr 1;27(2):299-326.

27. Aubourg C, Collard A, Léger M, Gros A, Fouquet O, Sargentini C, Rineau E. Risk Factors and Consequences of Late-Onset Hyperlactatemia After Cardiac Surgery With Cardiopulmonary Bypass: A Single-Center Retrospective Study. J Cardio Thorac Vasc Anesth.. 2022 Nov 1;36(11):4077-84.

28. Van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in critically ill patients. New Eng J Med. 2001 Nov 8;345(19):1359-67.

29. Zhou Y, Yang C, Jin Z, Zhang B. Intraoperative use of cell saver devices decreases the rate of hyperlactatemia in patients undergoing cardiac surgery. Heliyon. 2023 May 1;9(5).

To Cite:

Fath Al-Bab Ibrahim, G., ElSharawy, M., ElFakharany, K., Abdelhady, D., Shemais, D. Hyperlactatemia and coronary artery bypass grafting: Review article. *Zagazig University Medical Journal*, 2024; (1339-1345): -. doi: 10.21608/zumj.2024.285188.3353