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A Review On Lactic Acidosis In Cardiac Surgery

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Abstract

The normal range of lactate is 0-2mmol/L and value above it is considered as hyperlactatemia. It may arise due to adequate and inadequate oxygen supply mechanisms. The major reason is likely a stress-induced aerobic mechanism which shows raised levels of lactate. Increased lactate levels can cause acidifying effects on the blood. There are many etiological reasons for lactic acidosis like disease conditions, deficiency and alcohol. Early and late onset are two categorizations for patients with hyperlactatemia after cardiac surgery. One of the major trigger factors for lactic acidosis in cardiopulmonary surgery patients is tissue hypoperfusion. So preventive measures before cardiac surgery should be taken.

Introduction

In patients undergoing heart surgery, lactate level is a crucial biomarker and an excellent predictor of treatment results. In a number of studies, hyperlactatemia (HL) is a reliable indicator of mortality and serious morbidity following cardiac surgery [1]. In accordance with the underlying cause, Cohen and Woods separated lactic acidosis into two groups: lactic acidosis accompanied by or not by symptoms of tissue hypoxia [2]. Oxygen deprivation may be the result of insufficient blood flow brought on by a decline in cardiac output, a reduction in haemoglobin levels, or a problem with target cells' ability to absorb oxygen. In order to diagnose and evaluate hypoxia and lactic acidosis in those who are in cardiac failure, the level of blood lactate is measured. Hyperlactatemia is a typical metabolic syndrome following heart surgery with cardiopulmonary bypass. Its mechanism is intricate and debatable. The most frequent factor causing hyperlactatemia is tissue hypoxia (type A). According to research, type B (absence of tissue hypoxia) could be an additional factor in the development of hyperlactatemia after heart surgery. It has been demonstrated in several investigations that hyperlactatemia can be adrenaline induced. This mechanism is known as aerobic glycolysis. This type of glycolysis is a stress response rather than due to oxygen shortage [3].

In one of the studies after cardiac surgery, HL was detected in 1 in 10-20 percent of the overall of adult patients, and it was linked to severe postoperative morbidity and death. According to a research by Broder and Weil, there was a high probability of circulatory shock-related death in more than 88% of patients with blood lactate levels more than 4.0 mmol/L. The chances of surviving shock decrease from 90% to 10%, as stated by the same authors, when lactate levels in the blood rose from

2.0 to 8.0 mmol/L. In hospitals and intensive care units, serum lactate levels are often utilized as a guidance for doctors to initiate and enhance early therapy in shock patients and so aid to reduce morbidity and mortality. Despite having normal arterial blood gases (ABG), individuals after heart surgery under cardiopulmonary bypass (CPB) may still have lactic acidosis as a result of tissue hypoperfusion. Consequently, monitoring lactate levels in individuals undergoing heart surgery may be more sensitive than ABG measurement alone and may subsequently help in early diagnosis of an imbalance between demand and supply of oxygen [4].

Although lactic acidosis can have a number of different causes, having elevated serum lactate levels after heart surgery has been linked to an elevated chance of postoperative infection, cardiopulmonary dysfunction, renal impairment, longer hospital stay, and higher mortality. It has been established that aiming for normal lactate levels in the postoperative setting can shorten hospital stays after heart surgery [5].

However, as per recent research, not all types of HL are detrimental following cardiac surgery. In contrast to patients who developed hyperlactatemia later after surgery, Maillet and colleagues observed that the timing of HL plays a significant effect and that individuals with an immediate postoperative elevated lactate level have much worse results. As a result, the clinical results following heart surgery may be affected differently depending on the onset of hyperlactatemia (early versus late) [1]. This article aims to summarise the information on the etiology of hyperlactataemia during cardiac surgery, provide an explanation of lactate physiology as it relates to the field of cardiac surgery, and demonstrate the intricate connection between the development of HL and patient outcomes.

Pathophysiology

Category of Hyperlactatemia

There are two major types of lactic acidosis: Type A, caused by a lack of oxygen, and Type B, irrespective of that, seems to be sufficient oxygen supply [6]. Type A lactic acidosis is defined by poor tissue oxygenation, whereas Type B lactic acidosis is defined by a low level of tissue hypoxia. A high lactate to pyruvate (L/P) ratio is frequently linked to type A lactic acidosis, which indicate either a global or local deficiency of tissue oxygen transport. Hepatic, mesenteric, and limb ischemia are three causes of limited oxygen transport. An expected L/P ratio is linked to Type B lactic acidosis, which is characterized by rapid aerobic metabolism. Drug overdose and poisonings (cyanide, methanol, salicylates), ketoacidosis due to diabetes, hepatic malfunction (reduced lactate clearance), and thiamine insufficiency (impaired pyruvate dehydrogenase function) are additional reasons of Type B acidosis. Depending on a specific aetiology, the L/P ratio might be normal or high [7].

Important Causes of Hyperlactatemia

Seizure

Depending on the category of seizure, lactate levels may be dramatically raised. It is very important for the physician to understand that higher levels of lactate in this situation are sporadic. Once the episode has passed, lactate is no longer produced and is quickly eliminated. Extended periods of time with persistently high lactate levels after having a seizure may indicate a distinct or concurrent base etiology and require for additional examination [8, 9].

Diabetic Ketoacidosis

Elevated lactate levels may develop in diabetic ketoacidosis (DKA), a condition that is not commonly noticed, but unlike other disease states, they do not appear to be related to more serious

outcomes [5]. In a historical analysis of 68 individuals with DKA, Cox et al. discovered that 40% had lactate levels above 4 mmol/L. There was no association between lactate and mortality or duration of stay in the intensive care unit in this group. It is possible that elevated lactate levels in DKA may be caused by both a changed metabolic profile and hypoperfusion, which is indicated by the fact that lactate levels have a positive relationship with glucose and a poor relationship with thiamine levels. However, additional study is needed [10,11].

Malignancy

Individuals with rapidly progressing leukaemia or lymphoma, usually affecting the liver, are a majority of cancer patients who appear with high levels of lactate that are linked to their disease. Although the aetiology is unclear, it is probably linked to the excessive production of glycolytic enzymes by tumors, mitochondrial malfunction, decreased hepatic clearance, and possibly malnutrition, which results in thiamine deficiency [12, 13].

Burns

Lactate had been identified to be a powerful indicator of prognosis in cases of serious burns. According to Jeng et al., the very first lactate point was a helpful variable to distinguish between survivors and nonsurvivors [14]. Similar outcomes were observed in one more prospective trial by Kamolz et al applying an early lactate limit of 2 mmol/L. Additionally, they demonstrated a link between immediate lactate elimination and reduced mortality [15].

In addition, sepsis with multi-organ dysfunction is a significant cause of morbidity and mortality in burns patients. When working with burns patients, lactate levels should be acquired and taken into account, though its utility in terms of therapeutic outcome is uncertain [16].

Thiamine Deficiency

Thiamine acts as a coenzyme for a number of cellular enzymes, such as pyruvate dehydrogenase and alpha ketoglutarate dehydrogenase, which are necessary for the tricarboxylic acid cycle and aerobic glucose metabolism, respectively. Thiamine deficiency supports anaerobic metabolism, which results in higher lactate generation. Thiamine deficiency has been linked to the formation of increased lactate levels in blood and cerebrospinal fluid. States of nutritional deficit, such as alcoholism, persistent atrophy disorders, hyperemesis gravidarum, anorexia nervosa, and gastric bypass surgery, are risk factors for thiamine deficiency. When there is no other explanation for high lactate levels, it is important to take into account the frequently neglected but treatable condition of thiamine deficiency [17].

Lactulose

Lactate and acetate are produced in the intestines from synthetic, nondigestible disaccharide known as lactulose. The colonic epithelium may receive lactate if excessive amounts of lactulose are given, or if lactulose is retained in the gut, leading to systemic L- and D-hyperlactatemia. Lactulose administration to healthy individuals only causes slight elevations in plasma L- and D-lactate [18].

Carbon Monoxide

Carbon monoxide attaches to haemoglobin with a higher affinity than oxygen, and forms carboxyhemoglobin preventing efficient oxygen transport [19]. In addition to lowering oxygen supply by shifting the oxyhemoglobin curve to the left, carbon monoxide also causes oxidative stress and direct cellular injury [19]. The primary cause of hyperlactatemia from carbon monoxide

poisoning is assumed to be tissue hypoxia [20].

Alcohol

The relationship between increased lactic levels and alcohol is still uncertain, and studies have produced conflicting findings. Despite the fact that ethanol may raise lactate levels in an experimental environment, clinically significant elevated lactate levels are uncommon in healthy individuals. Lactate level increase should not be ascribed only to the possible effects of ethanol; rather, other reasons of highly raised lactate levels in these patients should be ruled out and treated if present. Patients who consume alcohol may be more susceptible to other conditions that raise lactate levels, such as a lack of thiamine, convulsions, sepsis, and other harmful substances. In addition to ethylene glycol toxicity, increased lactate levels have also been linked to other alcohols like propylene glycol and methanol [17].

Hyperthyroidism

Through amplification of glycolysis and the hexose monophosphate pathway, hyperthyroidism increases basal metabolic rate, which in turn increases glucose metabolism and hyperlactatemia. In addition, due to the loss of thiamine reserves, hyperthyroidism and thyroid storm can be considered as risk factors for thiamine deficiency [18].

Propofol

Acute metabolic acidosis, heart failure, rhabdomyolysis, lipemia, and hepatomegaly or hepaticlipidosis are all symptoms of propofol infusion syndrome, also known as PRIS [21].

Renal failure, lactic acidosis, and hyperkalemia are additional frequent features of this condition [22]. People who receive propofol for extended periods of time are more likely to develop PRIS. The two main pathophysiologic processes for PRIS are thought to be impaired fatty acid oxidation and mitochondrial malfunction [23,24].

Lactic Acidosis After Cardiac Surgery

There are numerous studies which mention the link between cardiac surgery and lactic acidosis. The etiological factors may include both hypoxic and non-hypoxic causes. Usual normal range of serum lactate is 0-2 mmol/L, but the reference range is 0.75-2mmol/L. Higher levels have been associated with in hospital mortality [25]. Increased lactate levels are observed during or immediately following the start of cardiopulmonary bypass, and in the following 4-24 hours of the postoperative intensive care unit (ICU) stay. In 10 to 21% of patients having on-pump cardiac surgery, HL develops during or shortly after CPB and commonly lasts until ICU arrival which is called as "Immediate hyperlactaemia" (IHL) or Early Onset Hyperlactaemia. For cardiac anaesthesia and intensive care clinicians, it is an important finding that frequently prompts worries about a challenging intra or postoperative course. Late-onset HL (LHL) is a condition that has been linked to cardiac surgery. Observational data shows elevated lactate of 4 to 14 hours after operation, which returns to normal within 12 to 24 hours [18, 26-28]. Following both emergency and elective cardiac surgery, this happens in 14-20% of adult patients. Perfusion deficits are produced while using cardiopulmonary bypass in the group of people who have had cardiac surgery specifically, as perfusion of the organs is insufficient for demand if core temperature rises under CPB. Lactate levels >10 mmol/l at early time was found to have a 100% positive predictive value in a retrospective small study, that indicated postoperative mortality. Liver failure contributed in some patients' elevated systemic lactate levels. Longer CPB duration and decreased oxygen supply (DO₂) during CPB were independent risk factors for development of hyperlactatemia. Circulatory dysfunction is evident even in critically ill surgical patients, according to Meregalli et al., who also claimed that there is no sign of shock in these patients. It is assumed that the presence of hyperlactatemia suggests "occult

hypoperfusion" in some patients, which would account for their higher mortality [29].

Studies and Reports

A strong predictor and a reliable biomarker in open-heart surgery and critically ill patients is Lactate [30-33]. Modern technological developments have helped to increase the number of cardiac surgeries. While treating the patients who have already undergone cardiac surgery, identifying risk factors for mortality and disability are crucial. In individuals with systemic hypoperfusion, hypoxia, metabolic acidosis and hyperlactatemia are frequent events [34]. In 2014, Marjan Joudi et.al conducted a descriptive study among 228 patients where the findings demonstrated that among the variables assessed, the number of blood transfusions, ejection fraction (EF) prior to surgery, diabetes mellitus, length of time using a pump, and blood storage time, respectively, significantly increased postoperative serum lactate compared to pre-operative levels [35]. A retrospective study conducted by Maket al. in 195 cardiac surgery patients undergoing bypass and/or valve surgery. They have concluded that patients who have undergone cardiac surgery who had late peaking blood lactate >30 h post-operatively are more likely to have a poor outcome in the intensive care unit and die. In post-CPB patients, lactate persistence is a better indicator of prognosis than peak lactate. In contrast to a simple post-CPB lactate washout, patients in a condition of prolonged hypoperfusion are more likely to experience serious surgical complications and die [36].

Renew et al., in 2016 reported that 121 (1.26%) of the 9580 cardiac surgery patients who satisfied the inclusion criteria developed HL and cardiogenic shock was the most frequent reason (53.8%). In that study, a total of 49 (40.5%) patients passed away while receiving care and 40 (33.1%) were sent home. The remaining 32 (26.4%) patients were transferred to a skilled-care institution [34].

More research is necessary to fully understand these findings, especially how persistent hyperlactatemia affects decision-making in this complicated patient population.

Conclusion

The majority of the patients who undergo heart surgery are likely to develop both hypoxia and non-hypoxia conditions which can cause hyperlactatemia. Most likely, stress-induced increased oxygenated metabolism, where the higher lactate emerges from a cumulative effect on the lactate or pyruvate equilibrium, is the main non-hypoxic cause. It is crucial to keep in mind, nevertheless not all postoperatively developed de novo hyperlactatemia is benign. Hyperlactatemia and acidosis are common features of complications like cardiac arrest, septic shock and ischemia brought on by myonecrosis or mesenteric infarction. Routine monitoring should be done in the intensive care unit to ensure that the patient is not at risk of developing hyperlactatemia and also we should be attentive to maintain normotensive and normovolemic hemodynamic status.

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