Serum Lactate as a Prognostic Factor in Coronary Arteries Bypass Graft Operations

Thesis

Submitted for partial fulfillment of the master degree in Intensive Care

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Ain Shams University
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2019

Acknowledgement

First and foremost, I feel always indebted to Allah the most kind and most merciful and we owe to him for his care and guidance in every step in our life.

I wish to express my great attitude and thanks to Prof .Dr Amr Esam_eldeen Abd_elhameed, Professor of Anaesthesia and Intensive Care, Faculty of medicine, Ain Shams University for accepting to supervise this work and for his valuable supervision and guiding comments, he generously devoted much of his precious time and provided unlimited in depth guidance, I sincerely appreciate all the encouragement and support given by him.

I am profound grateful to Dr. Mohamed Mourad Mohsen Mohamed Ali, Lecturer of Anaesthesia and Intensive Care, Faculty of medicine, Ain Shams University, for his kind and close supervion, constant fatherly advice , support , scientific guidance and his trust in my performance and my work.

Thanks to all the patients who were included in this study and cooperated in this research

Finally thanks to my great family and my wife for their support and encouragement.

Ahmed Mohamed Abd Elkader

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List of Abbreviations

Abbr.	Full term
χ^2	:Chi square test
2VD	: Two vessel disease
1VD	: Single vessel disease
3VD	: Three vessel disease
ACC	**:** American College of Cardiology
ACT	**:** Activated clotting time
AHA	: American Heart Association
AKI	**:**Acute Kidney Injury
ATP	: adenosine tricyclic phosphate
AVR	: aortic valve replacement
BIMA	: bilateral internal mammary artery
BMI	: Body mass index
CABG	: Coronary Artery Bypass Grafting
CHD	**:** Coronary heart disease
CHF	**:** Congestive heart failure
CKD	**:** chronic kidney disease
COPD	**:** Chronic Obstructive Pulmonary Disease
CPS	: Cardiopulmonary Bypass
CVD	: Cardiovascular disease
CVP	**:** Central venous pressure
DM	**:** Diabetes Mellitus
EACTS	**:**European Association for Cardio-Thoracic Surgery

ECG	: Electrocardiogram
EF	: Ejection Fraction
ESC	: European Society of Cardiology
ESRD	: End Stage Renal Disease
GDMT	: Guideline-directed medical therapy
HDL-C	: High density lipoprotein cholesterol
HTN	: Hypertension
ICU	: Intensive care unit
IVC	: Inferior Vena Cava
LAD	: Left anterior descending artery
LDH	**:** Lactate dehydrogenase
LDL-C	**:** Low density lipoprotein cholesterol
LIMA	**:** Left internal mammary artery
LV	: Left Ventricle
MI	**:** Myocardial infarction
mL	: Mille Litre
MR	**:** Mitral regurge
MV	**:** Mechanical Ventilation
NCEP ATP III	: National cholesterol education program Adult Treatment Panel III
O2	: Oxygen
OxPhos	**:** oxidative phosphorylation
PCI	**:** Percutaneous Coronary Intervention
ScvO2	**:** Mixed Venous Oxygen Saturation
SIHD	**:** Stable ischemic heart disease

STEMI	**:**ST segment elevation myocardial infarction
STS	: Society of Thoracic Surgeons
SVC	: Superior Vena Cava
SYNTAX	:Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery
TOE	: Trans esophageal echocardiography
VT	: Ventricular Tachycardia
WHO	**:** Word Health Organization
XCL	: Cross clamp

INTRODUCTION

Cardiovascular diseases (CVDs) are the number one cause of human mortality and morbidity worldwide. Every year, more and more people die from these diseases than from any other illnesses. In 2016, 17.9 million people died from CVDs, constituting 31% of all global deaths. Heart attack and stroke make up 85% of these deaths and the number of deaths from CVDs in the world is predicted to reach 23.6 million by 2030 ***(Monika, et al., 2019)***

Furthermore, Coronary Artery Disease (CAD) is one of the important diseases which affects the patients' survival, prognosis and quality of life, The most preferred treatment approach especially in multi_vessel CAD is Conventional Coronary Artery Bypass Grafting (CABG) surgery with Cardiopulmonary Bypass (CPB) (***Hillis et al., 2012***).

On the other hand, CPB usage and cardiologic arrest might cause various adverse effects. Nowadays, cardiac surgery is routinely performed with lower mortality rates in many centers worldwide. Nevertheless, postoperative morbidity, in relation to various risk factors, still seems

to be common and complications such as arrhythmias, ventricular dysfunction, infection, gastrointestinal dysfunction, acute lung injury and renal disorders may develop (***Nishimura et al., 2014***) .

Many variables measured in critically ill patients have been used to estimate the severity of disease, prognosticate morbidity and mortality, evaluate costs of treatment, finally indicate specific treatment and monitor the adequacy of treatment and its timing. It is unlikely that one measurement can replace all of these, but in the remainder of this manuscript we will show that lactate levels may come close. Although in our mind lactate levels are strongly linked to tissue hypoxia, they may follow many more metabolic processes not related to tissue hypoxia and, therefore, subject to many disturbances found in various clinical situations.***(Bakker, et al.,2013)***

Clinical findings and lab test results on admission to the intensive care unit (ICU) reflect the most recent pathophysiological findings. The events in the hours that follow admission are often a development of those events . Based on this information, the changes in these parameters on admission as well as in the outcome of ICU patients have

been used to establish the risk of death and blood lactate levels are one of the most commonly used methods ***(Sanz ,et al.,2002).***

Blood lactate levels are used in several situations, such as marker for tissue hypoperfusion in shocked patients, indicator of adequate post-shock resuscitation, prognostic index after resuscitation, prognostic factor in case of severe diseases and as etiologic diagnosis ***(Kliegel et al .,2004)***.

Most cases of hyperlactatemia in critically ill patients are due to inappropriate tissue oxygenation. This condition may results from respiratory disorders with poor blood oxygenation or from circulatory disorders that cause tissue hypoperfusion. As patients with tissue hypoperfusion do not always show clinical signs, hyperlactatemia may be the only marker for this disorder ***(Meregalli et al ., 2004).***

Lactic acidosis is defined as a metabolic acidosis in which arterial blood lactate levels are equal to or greater than 45 mg/dl (5 mmol/l) and the arterial pH is less than 7.35 ***(Stacpoole et al .,1993)***.

Critically ill patients may have normal lactate levels up to 18 mg/dl,although arterial blood reference values can reach as much as 10 mg/dl.Values between 18 and 45 mg/dl are described as being in the gray zone, whose Importance has not been established yet. In critically ill patients whose arterial blood lactate levels amount to 18 and 45 mg/dl, the main goal is to determine whether there is hypoperfusion, because if it is not detected, there may be potentially deleterious consequences ***(Koliski, et al.,2005).***

AIM OF THE WORK

This study aims to verify the use of hyperlactatemia as a marker for tissue hypo perfusion (anaerobic metabolism) and as a prognostic index in patients undergoing CABG.

Chapter (1)

Lactate (History, Metabolism and Uses)

i. History of lactate:

The first description of lactate originates from 1780 when Karl Scheele found lactate in sour milk. It took almost 70 years before the German physician-chemist **Joseph Scherer** demonstrated the presence of lactate in human blood. Where Scherer analyzed blood drawn from a young woman who had just died from what we now call septic shock, it was **Carl Folwarczny** in 1858 who demonstrated the presence of lactate in the blood of a living patient ***(Kompanje ,et al, 2007).***

(Araki , et al, 1891) made an important observation that has shaped our association of increased lactate levels and tissue hypoxia. These authors observed that when they interrupted oxygen supply to muscles in mammals and birds, lactic acid was formed and increased .