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Blood Lactate/Pyruvate As a Predictor of Shock Development in Acute Myocardial Infarction

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Abstract: This study, designed to investigate the blood lactate and pyruvate levels in AMI, was carried out on 41 patients. Based upon the localization of AMI, the patients were studied under anterior AMI (n=17), anteroseptal AMI (n=9) and inferior AMI (n=15) groups. Left ventricular failure developed in five patients with anterior AMI and in one patient with anteroseptal AMI. Cardiogenic shock developed in two patients with anterior AMI. Lactate and pyruvate were analysed in the blood samples drawn 24-48 hours after AMI. CPK, CPK-MB, AST and LDH activities were determined in the sera of blood samples drawn within 6-24 hours of AMI. Enzymatic methods were performed for lactate and pyruvate analyses, kinetic methods of CPK and LDH activities, immunoassay for CPK-MB and colorimetric method for AST.

A significant difference was found between the pyruvate values of anterior and anteroseptal AMI groups and also between anterior and inferior AMI groups ($0.05>p>0.02$, $0.02>p0.01$ respectively) Lactate/pyruvate ratio was found to be significantly higher in the anterior AMI group in comparison to the inferior AMI group ($0.05>p>0.02$). The cardiac enzyme activities were found to be significantly higher in the anterior AMI group. Increased blood lactate/pyruvate ratio might be considered as a predictor of left ventricular failure and shock occurence in AMI and also as an indicator of insufficient tissue oxygenation, might serve as a determinant in choosing the correct intervention therapy.

Key Words: Acute myocardial infarction, pyruvate lactate.

Introduction

Cardiogenic shock is a leading cause of death in patients with acute myocardial infarction (AMI). In a patient with AMI and left ventricular failure, development of lactic acidosis prognosticates a poor outcome. Elevation of lactate level occurs when oxygen delivery to the nutrient capillaries falls below a critical value to maintain cellular aerobic metabolism. Increased metabolic demands, when not met by an increase in cardiac output and oxygen extractions, are associated with the development of lactic acidosis (1,2). Patients with AMI and left ventricular failure are not able to increase cardiac output, especially when exposed to metabolic stress leading to an additional increase in lactic acidosis.

Blood lactate has been suggested to predict shock development in AMI and also serves as a prognostic marker for risk of recurrent cardiac arrest and cardiovascular mortality (3, 4).

In this study, we aimed to evaluate the predictory importance of both lactate and, its only known precursor, pyruvate in AMI with different localizations.

Materials and Methods

The study was carried out on 41 patient diagnosed as AMI. The diagnosis of AMI was based on the presence of 2 or more of the following criteria: Characteristic history of prolonged chest pain, elevation in serial cardiac enzymes and characteristic electrocardiographic ST-T changes with or without development of new q waves. In the presence of only ST-T changes on electrocardiogram, elevation of cardiac enzymes was required for confirmation of AMI.

Based upon the electrocardiographic localization of AMI, the patients were grouped as:

Anterior AMI (n=17) (age: 56.12±14.87)

Anteroseptal AMI (n=9) (age: 61.22±11.24)

Inferior AMI (n=15) (age: 57.80±11.98)

Clinical symptoms of left ventricular failure (dyspnea, nocturia, tachycardia, third heart sound, rales in the lungs) were observed in 5 patients with anterior AMI and in 1 patient with anteroseptal AMI. Cardiogenic shock (defined as systolic blood pressure <90 mmHg accompanied by the clinical signs of peripheral hypoperfusion and a decrease in urinary output <20 ml/hr) developed in 2 patients with anterior AMI. Neither left ventricular failure nor cardiogenic shock was observed in patients with inferior AMI.

To determine lactate and pyruvate concentrations, fasting venous blood samples were drawn from a brachial vein, without the use of a tourniquet, 24-28 hours after AMI, the time period accepted critical for the development of left ventricular failure.

Creatine phosphokinase (CPK), creatine phosphokinase MB (CPK-MB), lactate dehydrogenase (LDH) and aspartate amino transferase (AST) activities were determined in the sera of blood samples drawn within the first 6-24 hours of AMI.

Enzymatic methods were performed for lactate and pyruvate analyses, kinetic methods for CPK and LDH activities, immunoassay for CPK-MB and colorimetric method for AST.

Statistics:

Results are expressed as mean ± SD. Statistical significance between the groups is determined by Student's "t" test.

Results

Values and statistical comparison of the analysed parameters in patients with AMI are shown in Table 1.

Table 1. Values and statistical comparison of the analysed parameters in AMI

	Anterior AMI (n=17)	Anteroseptal AMI (n=9)	Inferior AMI (n=15)
Lactate (mg/dl)	15.76±8.09	12.39±3.65	14.92±5.00
Pyruvate (mg/dl)	0.58±0.24	1.04±0.88a*	1.31±0.92b**
L/P	36.55±24.20	22.63±22.112	21.37±18.42b*
CPK-MB (U/L)	106.24±27.14	85.44±47.13	68.07±40.31b**
CPK (U/L)	1620.06±1582.68	1058.11±792.35c**	441.33±377.44b**
LDH (U/L)	1160.53±771.58	1184.54±427.42c*	379±112.33b***
AST (U/L)	242.65±169.32	162.67±144.02	88.40±41.92b**

a: Anterior AMI vs Anteroseptal AMI
b: Anterior AMI vs Inferior AMI
c: Anteroseptal AMI vs inferior AMI
* p <0.05, ** p<0.01, *** p<0.001

A significant difference was found between the pyruvate values of anterior and anteroseptal AMI groups and also between anterior and inferior AMI groups and also between anterior and inferior AMI groups (0.05>p>0.02, 0.02>p>0.01 respectively) Lactate/pyruvate ratio was found to be significantly higher in the anterior AMI group in comparison to the inferior AMI group (0.05>p>0.02). The cardiac enzyme activities were found to be significantly higher in the anterior AMI group.

Discussion

In states of tissue hypoperfusion and resultant tissue hypoxia the mitochondrial pathways cease to generate energy. In an attempt to satisfy the energy needs of the organism pyruvate-lactate shunt is activated as an emergency pathway of anaerobic metabolism. Thus, blood lactate and pyruvate values gain importance in the detection of impaired oxygenation in critical care patients. These values reflect the cumulative oxygen deficit and serve as a close correlate of survival (5, 6, 7).

In the presented study we measured blood pyruvate and lactate concentrations in patients with acute myocardial infarction of different localizations.

The blood pyruvate concentration was found to be significantly lower in the anterior AMI group when compared to both anteroseptal (0.5±0.24 vs 1.0±0.88, 0.00>p>0.02) and inferior (0.58±0.24 vs 1.31±0.92, 0.02>p>0.01) AMI groups. Higher blood lactate concentration of the anterior AMI group, however did not show significance in comparison.

Lactate/Pyruvate ratio was found to be significantly higher in the anterior AMI group in comparison to the inferior AMI group (0.05>p>0.02). This finding suggests a greater oxygen deficit and a higher possibility for the development of left ventricular failure and cardiogenic shock in anterior AMI patients than in the anteroseptal and inferior AMI patients. Supporting the above suggestion, clinical symptoms of left ventricular failure were observed in 5 patients with anterior AMI (5/17: 30%) whereas only in 1 patient with anteroseptal AMI (1/9: 17%) and in none of the patients with inferior AMI. Moreover, cardiac specific enzymes were found to be higher specifically in the anterior AMI patients.

Hands et al (8) focused specifically on the need of a an index as a prediction of shock development in patients with AMI. Such an index would be of great utility

if it is based on simple clinical and laboratory findings that are readily available for every patient. The value of peripheral blood lactate concentration as prognostic marker for risk of recurrent cardiac arrest has been demonstrated by Sheps (9) and Kessler et al (2).

According to our data however, there appears to be a higher possibility for the development of cardiogenic shock when anterior AMI presents itself not only with an increase in lactate and lactate/pyruvate ratio but also with a decrease in pyruvate level. Especially in clinical setting, the analyses of lactate and pyruvate, performed with technical ease and rapidity would offer the clinician an objective measure for both the presence and the severity of the shock state and would aid in choosing the right intervention therapy. The effectiveness of the applied therapy may best be gauged by repetitive measurements of L/P ratio (10, 11, 12, 13).

We conclude that, the concentrations of lactate and pyruvate in blood, and more specifically their ratio are good indicators of oxygen deficit and tissue oxygenation. Lactate/Pyruvate ratio is found to be increased to a higher extent in patients with acute anterior myocardial infarction, with a higher risk for tissue hypooxygenation, left ventricular failure and cardiogenic shock than in patients with inferior myocardial infarction. The measurement of both pyruvate and lactate may thus provide practical cilinical usefulness and early assessment of critically ill AMI patients.

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