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Amino Acid Profile of Blood Serum in Normal Conditions and In Myocardial Infarction

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Abstract

This study focuses on changes in the amino acid composition of blood serum in patients with myocardial infarction. The amino acid profile is considered to reflect the metabolic changes that occur during a heart attack. Analysis of changes in free amino acids in the blood in this category of patients can contribute to a deeper understanding of metabolic disorders in myocardial infarction and expand our understanding of potential targets for metabolic correction.

Keywords: Amino acids, blood serum, nonessential amino acids, essential amino acids, troponin I, glycine, chromatogram, myocardial infarction.

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1. Introduction

According to estimates by the World Health Organisation and relevant cardiology societies, the incidence of myocardial infarction will remain high by 2030, with the total number of myocardial infarction cases increasing by 10–15%, especially among people over 65 years of age. In addition, up to 80% of deaths from cardiovascular disease will be associated with ischaemic events, including myocardial infarction [1].

Recently, particular attention has been paid to changes in the amino acid composition of blood serum as a reflection of the metabolic changes that occur during a heart attack. Amino acids play an important role not only as structural components and substrates for energy metabolism, but also as active participants in inflammatory, immune and

regenerative processes. Under conditions of hypoxia and necrosis, proteolysis is activated, the consumption of amino acids for gluconeogenesis and adaptive metabolism increases, and intracellular compounds are released into the systemic bloodstream [1,2,3].

Changes in amino acid composition can be twofold: on the one hand, they are the result of pathological tissue destruction and the body's stress response, and on the other hand, they represent a potential diagnostic and prognostic resource. Metabolomic studies in recent years have shown that certain amino acid patterns correlate with the severity of myocardial damage, the level of inflammation, the degree of cardiac tissue remodelling, and disease outcomes [1,3].

Thus, studying the amino acid composition of blood serum in myocardial infarction allows not only to deepen the understanding of pathophysiological processes occurring in the acute and subacute periods of the disease, but also to identify new biomarkers that are significant for diagnosis, risk stratification, and assessment of the regenerative potential of cardiac tissue.

The aim of this study was to evaluate alterations in the serum amino acid profile in patients with myocardial infarction in comparison with normal amino acid levels observed in healthy individuals.

2. Methods

Samples taken from patients with a confirmed diagnosis of acute myocardial infarction using troponin I (Finecare FIA Meter analyser manufactured by Guangzhou Wondfo Biotech Co., Ltd., PRC) upon admission to the Republican Specialised Scientific and Practical Medical Centre of Cardiology (RSCC). Blood from male donors of appropriate ages was also used as samples for the control group. To isolate free amino acids from the blood plasma of patients, 1 ml of 20% TCA was added to 1 ml of the test sample. After 10 minutes, the precipitate was separated by centrifugation at 8000 rpm for 15 minutes. After separating 0.1 ml of supernatant, it was freeze-dried. The hydrolysate was evaporated, the dry residue was dissolved in a mixture of triethylamine-acetonitrile-water (1:7:1) and dried. This operation was repeated twice to neutralise the acid. Phenylthiocarbamyl derivatives (PTC) of amino acids were obtained by reaction with phenylthiocyanate according to the method of Steven A., Cohen Daviel. The amino acid derivatives were identified by high-performance liquid chromatography (HPLC). HPLC conditions: Agilent Technologies 1200 chromatograph with DAD detector, 75x4.6 mm Discovery HS C18 column. Solution A: 0.14M CH3COONa + 0.05% TEA pH 6.4, B:CH3CN. Flow rate 1.2 ml/min, absorption 269 nm. Gradient %B/min: 1-6%/0-2.5 min; 6-30%/2.51-40 min; 30-60%/40.1-45 min; 60-60%/45.1-50 min; 60-0%/50.1-55 min.

3. Results

We conducted research to study the amino acid spectrum in the blood of patients with myocardial infarction. For this purpose, blood samples were obtained from eight patients admitted to the cardiac intensive care unit of the Republican Specialised Scientific and Practical Medical Centre of Cardiology with a diagnosis of acute myocardial infarction. The latter was diagnosed based on the determination of troponin I levels in the blood. The average troponin I level in the blood of the patients we studied was 27.55 ± 5.90 ng/ml, ranging from 8.09 to 50.00 ng/ml. The reference values are less than 0.29 ng/ml. The blood of 8 male volunteers was used as a control.

The study showed that in myocardial infarction, there were no statistically significant changes in the content of the amino acids serine, threonine, proline, tyrosine, valine, histidine, isoleucine, leucine, tryptophan, and phenylalanine compared to normal values.

Of the 18 amino acids we studied, 8 were found to be statistically significant. Of these, 5 amino acids are non-essential amino acids.

The glycine content was reduced by 39.7% from the normal value. While the normal range of values was 69.80÷125.48 mmol/L, in myocardial infarction it was 28.37÷92.18 mmol/L.

The asparagine content was found to be 48.9% below normal. The fluctuations in values were 79.40÷142.14 mmol/L in normal cases and 29.52÷102.48 mmol/L in cases of myocardial infarction.

The glutamine content in the blood during myocardial infarction was 80.6% lower than normal. Normal values ranged from 112.01 to 355.73 mmol/L, while during myocardial infarction they ranged from 30.11 to 135.48 mmol/L.

Cysteine levels were also lower than normal (by 60.6%). While normal values ranged from 103.75 to 497.06 mmol/L, during myocardial infarction they ranged from 44.24 to 129.42 mmol/L.

Unlike all other amino acids, the alanine content in the blood during myocardial infarction was, on the contrary, 49.1% higher. The normal range was 18.18÷55.34 mmol/l,

while during myocardial infarction it was 40.63÷69.14 mmol/l.

A statistically significant decrease was also observed in the content of some essential amino acids.

For example, the arginine content in the blood during myocardial infarction was also found to be 29% below normal. At the same time, the normal range was 63.89÷195.58 mmol/L, while during myocardial infarction it was 55.74÷164.52 mmol/L.

The methionine content in the blood during myocardial infarction was also 57.4% lower than normal, with normal values ranging from 38.07 to 411.10 mmol/l and myocardial infarction values ranging from 33.24 to 124.79 mmol/l.

In myocardial infarction, a significant decrease compared to normal values was also observed in the lysine content (by 49.6%). The range of values was 30.30÷201.18 mmol/l for normal values and 30.99÷70.93 mmol/l for myocardial infarction.

4. Discussion

The results of the present study demonstrated significant disturbances in the amino acid composition of blood serum in patients with myocardial infarction, indicating pronounced metabolic remodeling associated with myocardial ischemia and necrotic injury. Among the analyzed amino acids, the most marked statistically significant decrease was observed in glutamine levels, while methionine concentration was also reduced. In contrast, alanine levels were significantly increased compared with the control group.

The observed reduction in glutamine concentration is consistent with contemporary metabolomic studies showing enhanced glutamine utilization during acute cardiovascular stress. Glutamine serves as an important substrate for energy production, nitrogen transport, immune-cell activation, and antioxidant defense. During myocardial infarction, inflammatory responses and oxidative stress markedly increase cellular glutamine consumption. Recent metabolomic analyses of patients with acute coronary syndrome demonstrated decreased circulating glutamine levels associated with myocardial hypoxia and increased catabolic metabolism [4,5]. Furthermore, reduced plasma glutamine has been linked with impaired mitochondrial function and enhanced inflammatory activation in cardiovascular diseases [6].

A significant decrease in methionine identified in this study may reflect disruption of sulfur-containing amino acid metabolism. Methionine plays a crucial role in methylation reactions through S-adenosylmethionine formation and participates in antioxidant defense via glutathione synthesis pathways. Previous investigations have shown that ischemic myocardial damage is accompanied by alterations in methionine metabolism and increased oxidative stress, contributing to endothelial dysfunction and progression of ischemic heart disease [7,8]. Clinical metabolomics studies published in recent years also report decreased methionine availability in patients with acute myocardial infarction, suggesting increased utilization in stress-related metabolic pathways [9].

In contrast to the decreased amino acids, alanine concentration in blood serum was significantly elevated in patients with myocardial infarction. This finding agrees with reports demonstrating activation of anaerobic glycolysis during myocardial ischemia. Alanine formation is closely linked to pyruvate metabolism via transamination reactions catalyzed by alanine aminotransferase. Under hypoxic conditions, enhanced glycolytic flux promotes conversion of pyruvate into alanine, reflecting adaptive metabolic responses aimed at maintaining energy homeostasis [10]. Similar increases in alanine levels have been described in metabolomic profiling studies of acute myocardial ischemia and systemic hypoxia [11].

Taken together, the detected imbalance between essential and nonessential amino acids confirms that myocardial infarction is associated with profound metabolic alterations involving energy metabolism, oxidative stress regulation, and amino acid interconversion. Modern metabolomic evidence suggests that serum amino acid profiling may serve as a sensitive biochemical indicator of myocardial injury severity and may help identify potential targets for metabolic therapy in cardiovascular diseases [5,9,11].

5. Conclusion

Thus, among the amino acids in the blood serum of patients with myocardial infarction that we studied, the most pronounced statistically significant decrease was observed in the content of the replaceable amino acid glutamine, by 80.6% compared to the control group. Among the essential amino acids, the most statistically significant decrease was observed in the content of methionine, by 57.4% compared to the control group. Unlike all other amino acids, the content of alanine in blood serum in myocardial infarction was, on the contrary, 49.1% higher.

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