FREE AMINO ACIDS IN BLOOD PLASMA OF PATIENTS WITH MYOCARDIAL INFARCTION

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S u m m a r y. Myocardial infarction (MI) is a pathological condition resulting from total occlusion of coronary blood supply to some part of the heart muscle. This causes necrosis of the heart muscle of various extent. The most frequent background of the development of myocardial infarction is coronary atherosclerosis. The majority of patients with coronary atherosclerosis experience ischemia during effort and a characteristic chest pain. Free amino acids (AAs) play an important role both in human physiology and pathology. The pool of free amino acids varies, because amino acids are supplied to this pool, as well as uptaken and used as needed by the body. MI poses a direct life threat as it considerably affects other body functions, and changes blood serum free amino acids concentrations.
The objective of the study was determination of the level of free amino acids pro□le in patients with MI, and assessment of the level of free amino acids in patients with MI under the age of 65, and 65 years old and older. MI as a life threatening state causes dynamic changes in free amino acid pro□le; however, this is more signi□cant in the patients aged over 65. On day 0 in the group of patients ≥ 65 the level of amino acids was statistically signi□cantly higher, however it decreased during the rest of the observation period.

K e y w o r d s: cardiovascular diseases, age, amino acid pro□le

INTRODUCTION

Over the centuries, people have recognized factors causing diseases and learnt to diagnose and cure them. The introduction of mandatory protective vaccinations has minimized the epidemic of dangerous infectious diseases which have threatened mankind. The symptoms of heart disease in the form of angina pectoris were already known to physicians in ancient times. Galen Claudius, a Roman doctor, referred clinical symptoms of the disease to the organ, naming angina pectoris 'cardialgia' [3, 6]. Scienti□c observations concerning the relationship between economic status and morbidity due to cardiovascular diseases conducted over the years initially indicated a higher risk of coronary disease in individuals with a high socio-economic status (SES). Cardiovascular diseases were perceived not only as a consequence of improved life standard and prosperity, but also as a model clinical problem resulting from the modernization of societies enjoying better life [30]. Poland is characterized by an especially unfavourable situation with respect to the occurrence of cardiovascular diseases and their risk factors. Therefore, the problem of prevention of cardiovascular diseases has become one of the most important tasks aimed at the reduction of morbidity due to heart diseases, the complication of which is myocardial infarction [4, 25, 27].

Myocardial infarction is a pathological condition resulting from total occlusion of coronary blood supply to some part of the heart muscle. This causes necrosis of the heart muscle affecting an area of various extent. The most frequent background of the development of myocardial infarction is coronary atherosclerosis. Myocardial infarction and its consequences are the main cause of deaths not only in Poland, but worldwide [6, 14, 17].

In the majority of cases, the direct cause of myocardial infarction is a total occlusion of the lumen of the coronary artery, most frequently caused by intravascular thrombus due to the detached atherosclerotic plaque. In 90% of cases, atherosclerosis is the primary cause of MI. Also critical cardiac ischemia which lasts too long (longer than 30 minutes) leads to myocardial infarction [9, 21, 28, 31].

Another cause of infarction is a sudden increase in the demand of the heart muscle for oxygen with the existing impairment of the coronary flow reserve, i.e. the capacity for an adaptive increase in the blood flow through the coronary arteries. The condition is escalated when the sympathetic nervous system is stimulated by physical effort, acute pain, psychological stress, shock, thyroid storm, considerable acceleration of the heart rate, elevated body temperature, and an abrupt withdrawal of drugs blocking beta adrenergic receptors. Thus, myocardial infarction is the complication of a severe form of ischemic coronary disease [13, 18, 29].

Proteins are organic compounds indispensable for the existence of a cell. They are of decisive importance for biochemical processes which are the essence of all life functions [15, 22]. Proteins are not only the essential elements of the cell structures, but also the factors controlling biological transformations in the body. Both structural proteins and proteins regulating biochemical processes in the body wear out and are subjected to degradation. Amino acids (AAs) are the structural units of proteins and peptides. They contain two functional groups, i.e. an amino group and a carboxyl group. Therefore, they may bind together by peptide bonds. Amino acids are necessary for the formation of biologically active amines, neurotransmitters, hormones, hem, or purines [8, 15, 16].

In the cells, proteins are constantly renovated in a continuous process of degradation, and subsequent re-synthesis from free amino acids. Free amino acids, which are present in the organisms of humans and animals, constitute the so-called 'pool of free amino acids'. They may originate from alimentary proteins, decomposition of the body's own proteins, synthesis of endogenous amino acids, or conversion into other amino acids [24]. Many pathological processes, such as pancreatitis or septic states of various origin impair body homeostasis [5]. Free amino acids play an important role, both in human physiology and pathology. The pool of free amino acids varies, because amino acids are supplied to this pool and used as needed by the body. Cardiovascular diseases such as myocardial infarction exert an effect on the functioning of the whole body. They may also affect protein metabolism, especially changes in the level of free amino acids in the blood plasma.

AIM OF THE STUDY

The objectives of the study were:

1. determination of the level of free amino acids in patients with MI,
2. evaluation of the level of free amino acids in the examined patients with myocardial infarction aged under 65, and 65 years and older.

MATERIALS AND METHODS

The clinical material for the conducted study was the blood collected from the patients treated for MI. The blood was taken from the cubital vein four times: on admission to hospital (day 0), and subsequently on the first, third and seventh day of hospitalization. Blood was taken from the cubital vein fasting, in the morning hours; 5 ml of blood was collected into a test tube, and left at the temperature of 4°C until thrombus formation. Subsequently, the blood was centrifuged at 3,000 x g for 15 minutes in order to separate the plasma. From the centrifuged plasma, proteins were removed using 6% sulphosalicylic acid. The levels of free amino acids in the supernatant obtained were determined by ion exchange chromatography method using an automatic amino acid analyzer AAA 400-INGOS (Prague, The Czech Republic).

Characteristics of the study groups.

The study was conducted in the group of 128 patients, 88 males (68.75%) and 40 females (31.25%), aged 48–83 (mean age of males 62.43, and females – 66.35), hospitalized for myocardial infarction in the Świętokrzyskie Cardiology Centre in Kielce (Table 1). The examined patients were divided into two groups: group 1 – patients under 65yrs, and group 2 – patients 65 years old and older (Table 3).

Table 1. Characteristics of the examined group.

Gender	No. of patients	%	No. of patients < 65yrs	%	No. of patients ≥65 yrs	%
Males	88	68.75%	50	92.32%	38	60.32%
Females	40	31.25%	15	23.08%	25	39.68%
Total	128	100%	65	100%	63	100%

Table 2. Age of the examined patients.

Gender	M	SD	Min	Max
Males	62.43	9.75	48	78
Females	66.35	12.27	50	83
Total	63.81	10.76	48	83

Analysis of amino acids concentrations

Amino acids were separated using one-column system, 3 x 200 mm, filled with joinit resin OSTION LG FA (Ingos Prague, The Czech Republic). For the separation of amino acids, five lithium citrate buffers were used at pH - 2.9; 3.1; 3.35; 4.05; 4.90. The eluted amino acids were transferred into the tephlone-coated capillary and reacted with the incoming ninhydrin forming colored compounds. Amino acids were identified by retention time compared to the standards. Acidic and alkaline amino acids were separated at 38-39°C, and neutral amino acids at 59-60°C. Amino acid serum concentrations were expressed in micromoles per 1cm^3 (μmol/cm^3).

Statistical analysis.

The results were statistically analysed and presented as tables and graphs. The investigated variables of the examined population were expressed as arithmetic means, and the minimum and maximum values and standard deviation. The differences between specified categories were calculated by statistical tests, the selection of which was determined by the level of measurement of the variables. The differences between the variables were determined using Student's t-test; $p < 0.05$ was considered statistically significant [12].

RESULTS

The maintenance of the normal concentration of plasma amino acids depends on the balance between amino acids supplied and used up by the body. Each disease causes disorders of various extent in the homeostasis of the body. The cardiac muscle becomes weakened by myocardial infarction, and simultaneously exerts a considerable effect on the functioning of the whole body. The determination and comparison of the levels of free amino acids in the blood plasma of the patients with MI was the object of the presented study.

Table 3 presents mean concentrations of the examined AAs in the groups of patients, minimum and maximum range, and standard deviation.

Table 3. Concentrations of free amino acids in the blood serum of patients with MI [μmol/cm3].

		patients aged < 65 yrs								patients aged ≥ 65 yrs							
		Day 0		Day 1		Day 3		Day 7		Day 0		Day 1		Day 3		Day 7	
		M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
alkaline	LYS	0.181	0.037	0.199	0.032	0.234	0.066	0.230	0.345	0.182	0.051	0.164	0.035	0.199	0.026	0.212	0.025
	HIS	0.080	0.014	0.089	0.017	0.088	0.021	0.086	0.019	0.076	0.015	0.072	0.014	0.079	0.023	0.080	0.015
	ARG	0.074	0.200	0.065	0.016	0.116	0.062	0.099	0.039	0.079	0.058	0.040	0.017	0.058	0.014	0.083	0.031
acidic	ASP	0.040	0.009	0.046	0.015	0.046	0.019	0.049	0.016	0.041	0.009	0.036	0.010	0.042	0.011	0.037	0.009
	GLU	0.352	0.187	0.345	0.173	0.396	0.198	0.362	0.169	0.308	0.183	0.295	0.169	0.322	0.189	0.345	0.153
sulphuric	MET	0.018	0.004	0.017	0.006	0.025	0.007	0.023	0.006	0.017	0.006	0.020	0.009	0.021	0.004	0.022	0.006
aromatic	TYR	0.086	0.023	0.088	0.016	0.093	0.027	0.090	0.023	0.080	0.024	0.071	0.014	0.082	0.020	0.080	0.027
	PHE	0.065	0.012	0.074	0.010	0.083	0.020	0.074	0.016	0.068	0.024	0.065	0.017	0.072	0.013	0.069	0.010
branched-chain	VAL	0.233	0.043	0.255	0.047	0.321	0.116	0.291	0.078	0.314	0.169	0.274	0.085	0.292	0.088	0.264	0.054
	ILE	0.062	0.015	0.065	0.026	0.105	0.035	0.098	0.030	0.084	0.031	0.077	0.028	0.093	0.019	0.087	0.016
	LEU	0.123	0.025	0.155	0.024	0.217	0.088	0.200	0.072	0.163	0.080	0.152	0.040	0.176	0.043	0.162	0.024
non-protein	TAU	0.076	0.032	0.072	0.038	0.087	0.049	0.089	0.044	0.066	0.033	0.077	0.041	0.074	0.043	0.078	0.032
	ORN	0.089	0.022	0.101	0.022	0.099	0.023	0.099	0.017	0.096	0.021	0.078	0.022	0.086	0.017	0.091	0.019
	CIT	0.036	0.018	0.024	0.017	0.019	0.016	0.020	0.012	0.028	0.018	0.017	0.008	0.020	0.009	0.022	0.005
small protein	SER	0.110	0.021	0.124	0.029	0.149	0.039	0.135	0.023	0.111	0.032	0.106	0.032	0.135	0.022	0.118	0.017
	GLY	0.214	0.060	0.225	0.059	0.241	0.086	0.275	0.093	0.207	0.063	0.188	0.034	0.232	0.054	0.258	0.076
	ALA	0.402	0.080	0.389	0.102	0.396	0.114	0.490	0.260	0.383	0.096	0.356	0.131	0.338	0.081	0.403	0.090
	THR	0.130	0.037	0.146	0.046	0.181	0.083	0.181	0.051	0.133	0.038	0.120	0.030	0.180	0.049	0.164	0.046

The values of concentrations of free AAs in the blood plasma of patients with MI in the age group < 65 varied considerably, and were low on day 0 and day 1. On subsequent days, these levels were within the norm or higher. The concentrations of free AAs in the blood plasma of patients with MI aged ≥65 varied considerably during the whole period of study. The values of these amino acids most often remained below the reference value.

The results of Student's t- test (Tables 4-7) present the concentrations of free AAs in the blood plasma of patients in the groups aged < 65 years and ≥ 65 determined during the period of study.

Table 4. Concentrations of amino acids in the blood plasma of patients with MI (μmol/cm3) on day 0.

		Patients aged < 65yrs		Patients aged ≥65yrs		T	p
		M	SD	M	SD		
alkaline	LYS	0.1805	0.037	0.1820	0.051	0.017	0.898
	HIS	0.0800	0.014	0.0761	0.015	1.054	0.309
	ARG	0.0738	0.200	0.0792	0.058	0.202	0.655
acidic	ASP	0.0397	0.009	0.0406	0.009	0.141	0.708
	GLU	0.3517	0.187	0.3077	0.183	0.813	0.371
sulphuric	MET	0.0180	0.004	0.0166	0.006	0.969	0.329
aromatic	TYR	0.0856	0.023	0.0795	0.024	1.003	0.321
	PHE	0.0648	0.012	0.0676	0.024	0.352	0.555
branch-chain	VAL	0.2325	0.043	0.3140	0.169	6.946	**0.011**
	ILE	0.0617	0.015	0.0837	0.031	12.833	**0.001**
	LEU	0.1232	0.025	0.1631	0.080	7.169	**0.010**
non-protein	TAU	0.0762	0.032	0.0659	0.033	1.465	0.231
	ORN	0.0890	0.022	0.0965	0.021	1.822	0.182
	CIT	0.0364	0.018	0.0278	0.018	1.620	0.212
small protein	SER	0.1102	0.021	0.1112	0.032	0.023	0.881
	GLY	0.2135	0.060	0.2071	0.063	0.164	0.687
	ALA	0.4020	0.080	0.3829	0.096	0.706	0.404
	THR	0.1297	0.037	0.1332	0.038	0.130	0.720

On day 0, statistically significantly higher levels of VAL, ILE, LEU were observed in the group aged ≥ 65 (Table 4).

Table 5. Concentrations of amino acids in the blood plasma of patients with MI (μmol/cm3) on day 1.

		Patients aged < 65yrs		Patients aged ≥65yrs		T	p
		M	SD	M	SD		
alkaline	LYS	0.1986	0.032	0.1638	0.035	16.473	**0.000**
	HIS	0.0892	0.017	0.0722	0.014	18.103	**0.000**
	ARG	0.0654	0.016	0.0400	0.017	24.216	**0.000**
acidic	ASP	0.0463	0.015	0.0363	0.010	8.616	**0.005**
	GLU	0.3447	0.173	0.2950	0.169	1.228	0.272
sulphur	MET	0.0173	0.006	0.0201	0.009	2.178	0.145
aromatic	TYR	0.0885	0.016	0.0706	0.014	20.599	**0.000**
	PHE	0.0743	0.010	0.0645	0.017	7.757	**0.007**
branch-chain	VAL	0.2549	0.047	0.2738	0.085	1.205	0.277
	ILE	0.0654	0.026	0.0773	0.028	2.879	0.095
	LEU	0.1546	0.024	0.1519	0.040	0.109	0.742
non-protein	TAU	0.0719	0.038	0.0770	0.041	0.258	0.613
	ORN	0.1011	0.022	0.0777	0.022	15.595	**0.000**
	CIT	0.0243	0.017	0.0167	0.008	3.957	0.053
small protein	SER	0.1239	0.029	0.1064	0.032	5.113	**0.027**
	GLY	0.2255	0.059	0.1877	0.034	9.056	**0.004**
	ALA	0.3892	0.102	0.3563	0.131	1.204	0.277
	THR	0.1462	0.046	0.1203	0.030	6.561	**0.013**

On day 1, in the age group < 65 the values of concentrations of free AAs: LYS, HIS, ARG, ASP, TYR, PHE and ORN, SER, GLY, THR were statistically significantly higher in comparison to the age group ≥ 65. Other differences were statistically insignificant (Table 5).

Table 6. Concentrations of amino acids in the blood plasma of patients with MI (μmol/cm3) on day 3.

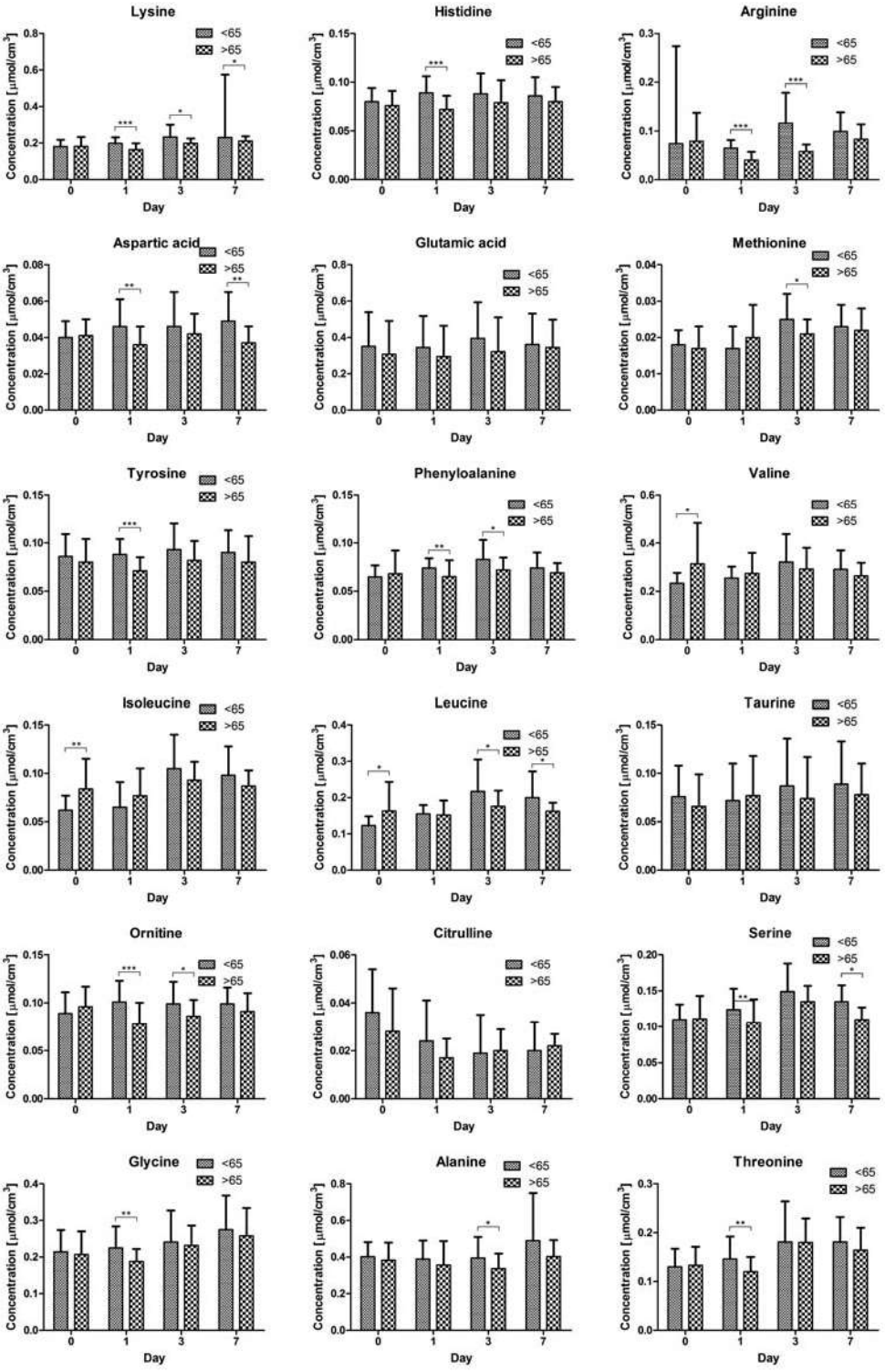
		Patients aged < 65yrs		Patients aged ≥65yrs		T	p
		M	SD	M	SD		
alkaline	LYS	0.2336	0.066	0.1987	0.026	6.939	**0.011**
	HIS	0.0884	0.021	0.0794	0.023	2.658	0.108
	ARG	0.1158	0.062	0.0581	0.014	15.147	**0.000**
acidic	ASP	0.0458	0.019	0.0416	0.011	1.069	0.305
	GLU	0.3958	0.198	0.3223	0.189	2.133	0.149
sulphur	MET	0.0248	0.007	0.0213	0.004	5.184	**0.026**
aromatic	TYR	0.0935	0.027	0.0818	0.020	3.592	0.063
	PHE	0.0830	0.020	0.0725	0.013	5.972	**0.017**
branch-chain	VAL	0.3207	0.116	0.2917	0.088	1.189	0.280
	ILE	0.1054	0.035	0.0929	0.019	2.845	0.097
	LEU	0.2171	0.088	0.1756	0.043	5.190	**0.026**
non-protein	TAU	0.0872	0.049	0.0740	0.043	1.267	0.265
	ORN	0.0995	0.023	0.0857	0.017	6.666	**0.012**
	CIT	0.0190	0.016	0.0203	0.009	0.125	0.725
small protein	SER	0.1488	0.039	0.1345	0.022	3.050	0.086
	GLY	0.2407	0.086	0.2321	0.054	0.210	0.648
	ALA	0.3964	0.114	0.3376	0.081	5.282	**0.025**
	THR	0.1810	0.083	0.1798	0.049	0.005	0.946

On day 3, the concentrations of LYS, ARG, MET, PHE and LEU, ORN, ALA were statistically significantly higher in the group < 65 compared to the group ≥65. The remaining AA levels were not statistically significantly different between the groups. (Table 6).

Table 7. Concentrations of amino acids in the blood plasma of patients with MI (μmol/cm3) on day 7.

		Patients aged < 65yrs		Patients aged ≥65yrs		T	p
		M	SD	M	SD		
alkaline	LYS	0.2298	0.345	0.2123	0.025	4.396	**0.041**
	HIS	0.0857	0.019	0.0804	0.015	1.247	0.269
	ARG	0.0993	0.039	0.0833	0.031	2.046	0.160
acidic	ASP	0.0489	0.016	0.0373	0.009	9.935	**0.003**
	GLU	0.3623	0.169	0.3453	0.153	0.142	0.708
sulphuric	MET	0.0234	0.006	0.0217	0.006	1.091	0.301
aromatic	TYR	0.0904	0.023	0.0802	0.027	2.413	0.126
	PHE	0.0743	0.016	0.0690	0.010	2.112	0.152
branch-chain	VAL	0.2907	0.078	0.2644	0.054	2.033	0.159
	ILE	0.0975	0.030	0.0867	0.016	2.639	0.110
	LEU	0.2003	0.072	0.1618	0.024	6.453	**0.014**
non-protein	TAU	0.0891	0.044	0.0780	0.032	1.104	0.298
	ORN	0.0989	0.017	0.0906	0.019	2.897	0.095
	CIT	0.0195	0.012	0.0222	0.005	0.960	0.333
small protein	SER	0.1347	0.023	0.1182	0.017	9.005	**0.004**
	GLY	0.2754	0.093	0.2580	0.076	0.576	0.451
	ALA	0.4900	0.260	0.4031	0.090	2.463	0.122
	THR	0.1813	0.051	0.1643	0.046	1.732	0.193

On Day 7, statistically significant differences between the groups were observed only in the case of LYS, ASP, and LEU and SER. In all cases, the level of the above-mentioned AAs was statistically significantly higher in the age group < 65. Other values were not statistically significantly different between the groups (Table 7).



F i g u r e 1. Analysis of the level of amino acids in blood plasma of patients with myocardial infarction. The data presented as the mean and standard deviation (n=60; * - $p<0.05$; ** - $p<0.01$; *** - $p<0.001$; t-Student test).

DISCUSSION

Cardiovascular diseases are the leading causes of mortality, and are responsible for nearly 50% of all deaths. The main cardiovascular diseases include the complication of ischemic heart disease, e.g. myocardial infarction, and cerebrovascular diseases, which are the cause of 35% of all deaths in Europe. However, considerable differences between individual countries are observed with respect to such indicators as age, gender, and distribution of the conditioning factors. [26]

Clinical symptoms of acute coronary syndrome (ACS) occur in nearly 30% of individuals aged over 65. In developed countries, it remains the most frequent cause of deaths among males aged over 45, and females aged over 65. In the population of the elderly, females are a considerably higher percentage of patients with ACS. Cardiovascular diseases are often accompanied by other disorders, such as arterial hypertension or diabetes. In the aetiology of myocardial infarction dynamic changes develop within the blood vessels. With time the changes narrow vascular lumen, and sometimes even occlude the vessel completely [11].

In the course of a coronary event acute-phase proteins increase considerably [23]. In the situation of oxygen deficiency there is an insufficient blood flow to the tissues. This causes an enhanced protein catabolism and change in the AA profile. These disorders are observed in MI. On the cellular level, that results in anaerobic metabolism, and resultant metabolic acidosis. It impairs the function of cells and may even lead to their irreversible damage. The conducted studies showed variations in the free AA profile in the blood plasma of the patients from the moment of admission to hospital until day 7 of hospitalization. The concentration of free AAs in the plasma of patients with MI varied according to the duration of treatment. On day 0. a significantly statistically higher level of VAL, ILE, LEU was observed in patients aged 65 and older.

On day 1 of hospitalization, statistically significant differences in the concentrations of LYS, HIS, ARG, ASP, TYR, PHE and ORN, SER, GLY, THR were observed between the groups of examined patients. In cases of the aforementioned AAs the values were higher in the group aged < 65. The concentration of other AAs were not statistically significantly different. However, on day 3 of hospitalization, the concentrations of LYS, ARG, MET, PHE and LEU, ORN, ALA were statistically significantly higher in the group aged < 65. No statistically significant differences were found with respect to the remaining amino acids. On day 7 of hospitalization, statistically significant differences in the values of LYS, ASP and LEU and SER were recorded between the groups of the examined patients. The level of those AAs was higher in the group < 65. Other values were statistically insignificant between the groups.

Bertolini et al. [2] observed the effect of age and gender on the levels of amino acids in human blood. Also, Armstrong [1], who conducted studies on a large population, indicated a lower level of free amino acids in the plasma of females. The importance of transformations of amino acids in the pathogenesis of many diseases has been noted in recent years. The results helped introduce new analytical methods to determine the concentrations of free amino acids in the body fluids.

Górski et al. [7] presented the most important data concerning the use of energy substrates by the healthy heart muscle in ischemic disease and diabetes. The heart muscle possesses a unique capacity to use various energy substrates and immediately convert them. It may use energy from glucose, free fatty acids, lactic acid, and ketone bodies. The heart muscle may use some amino acids as energy substrates, including glutamine, glutamic acid, as well as aspartic acid, and alanine.

Moreover, there is a need to conduct research into amino acid metabolism in obesity, considering the fact that an increased level of free amino acids in the plasma may stimulate the secretion of insulin. Studies by Zwaigzne et al. [32] confirmed the extent and effect of the reduction of body weight as a result of weight loss treatment on the concentration of free amino acids in the plasma and blood cells. The researchers found the concentration of free amino acids in the blood plasma and red blood cells in obese children changed together with weight loss. Their levels were close to the values of free amino acids in the children with normal body weight [19]. Observations by other researchers indicate that there is a further need to investigate amino acids metabolism in obesity. The results of studies confirm the presence of changes in protein metabolism in obese children.

In their study, Iłżecka et al. [10] determined the concentration of amino acids in the plasma of patients with atherosclerosis. They found that amino acids may play an important role in the etiopathogenesis of the disease. The concentrations of amino acids varied. In the whole group, considerably decreased values of isoleucine, leucine,

tyrosine, and valine were observed. The clinical status of patients concerning severe athersoclerotic burden exerted a signi□cant effect on the levels of amino acids, with the highest concentrations of alanine observed in terminal patients. Nevertheless, no statistically signi□cant relationship was found with concomitant diseases, or between the clinical pathological symptoms of these diseases and duration of the disease.

Changes in free amino acid pro□le in the plasma of patients in acute coronary syndromes result from disorders developing in the body due to pathological processes. In the course of an acute coronary event, acute-phase proteins increase considerably [20]. In the situation of acute oxygen de□ciency, the tissue blood flow is insuf□cient. It causes enhanced protein catabolism and changed amino acid pro□le. On the cellular level, anaerobic metabolism develops, leading to metabolic acidosis. It impairs the function of cells and may even lead to their irreversible damage. In physiological conditions, the concentrations of free amino acids depend on many factors, such as gender, age, nutrition, intensity of physical activity and the state of health. In order to maintain undisturbed synthesis of all body proteins, a positive nitrogen balance is necessary, which is possible only when the human body is in the state of health. In the course of myocardial infarction, free amino acids are used in a different way.

The results of our study revealed different free amino acid pro□le in the plasma of patients aged < 65 and ≥ 65 who had undergone myocardial infarction. The determination of aminogram and its interpretation may constitute a basis for the application of the supplementation of amino acids, which are the source of energy for the body cells.

CONCLUSIONS:

Myocardial infarction as a life threatening state causes dynamic changes in free amino acid pro□le; they are considerably more pronounced in patients aged under 65.

On day 0 in the group of patients aged 65 years and older the level of amino acids was statistically signi□cantly higher, however it decreased during the rest of the observation period.

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