The significance of determination of the fraction of creatinine-phosphokinase in patients with acute ischemic brain disease

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Heart and brain vascular diseases are the leading causes of mortality in the world. Cardiac complications can frequently occur during the development of cerebral ischemia. The aim of this study was to establish the possible changes in fractions of creatinine-phosphokinase as the sensitive laboratory index of parenchymal lesion of brain parenchyma and the presence/absence of risk factors for ischemic brain and heart disease. The study comprised 80 patients with acute ischemic brain disease (AIBD), without the history of previous coronary disease. Blood samples were taken in all patients within the first 48 hours from AIBD onset aiming to determine a total (muscular MM) and heart fraction of creatinine-phosphokinase (MB), and brain parenchyma ischemia was confirmed by CT or MR scan of the head. A detailed history of the risk factors for ischemic brain disease (IBD) and ischemic heart disease was taken from all patients with AIBD, and the profile of glycemia and lipid status were determined, and blood pressure was measured 6 times a day. Independent variables in statystical analysis were: age, degree of severity and the side of neurologic event, size of ischemic lesion and maximal values of systolic and dyastolic pressure. Dependent variables were the values of fractions of creatinine-phospokinase (CPK). Control group (n=40) comprised patients with neurologic diseases of non-vascular origin. All parameters as well as their interrelations were statistically analyzed. The results revealed significant correlation of the increased levels of CPK of MM and MB fraction with the size and place of ishemic lesion in the right cerebral hemisphere, which was highly significant for MB fraction in the total group of patients with AIBD, and for MM fraction, only for cases of more severe IBD. Highly significant increased values of those fractions were also observed compared to the control group of patients.

K e y w o r d s : **cerebral ischemia; myocardial ischemia; blood chemical analysis; creatinine; creatinine kinase.**

Introduction

Death of cardiac origin is the leading cause of mortality in patients with ischemic brain disease. Sometimes this can happen "unexpectedly" after the initial good recovery and course of the main disease. Although it is sometimes associated with the underlying heart disease, in those patients compared to the group with IBD were frequently found arrhythmia, increased levels of serum enzymes and plasma catecholamine (1–5).

Numerous experimental and clinical facts support the cerebral etiology of these changes. Thus, after the onset of IBD, blood pressure increases, as well as the serum concentrations of catecholamine and cortisol. Also, ST depres-

sion in patients with IBD was registered in 15–40% cases from previous studies. Prevalence of asymptomatic ST depression in patients with angina pectoris was 43-60% and in patients without the history of cardiac disorders was 6–8%. According to this data, ST depression was more frequently present in patients with IBD than in asymptomatic patients and less frequently in patients with the diseases of coronary arteries (6–9).

Besides, necrosis of myocardial cells was found in experimental studies after the occlusion of cerebral arteries in the absence of coronary artery disease. Also, according to our findings patients with ischemic lesion in the right cerebral hemisphere had the poorer prognosis, what was supported by the experiments and examination during the neurosurgical treatment of epilepsy, which showed that the stimulation of right insular cortex region caused the increase in blood pressure, in heart rate and in serum catheholamine, while the stimulation of the other side had the opposite effects (8–14).

Based on these facts, we formulated the following hypothesis:

– in patients with IBD, without the previous history of ischemic heart disease, existed the increase of both fractions of CPK (MM and MB) and

– the values of both fraction directly correlated with the severity and localization of IBD.

According to this hypothesis, the aim of the study was to determine the character of correlation between the clinical, neurological and laboratory findings (concentration of MM and BM fraction of CPK) in patients with IBD.

Methods

A study included 80 patients who were hospitalized in our clinic due to transient ischemic attack (TIA), reversible ischemic attack (RIA) and brain infarction (BI). Control group consisted of 40 patients without the symptoms and signs of atherosclerosis, immunologic, inflammatory, hematologic and malignant diseases.

Patients with previous history of ischemic heart disease were excluded from the study, which was the reason why standard ECG was taken for every patient.

Upon the check up in all patients were determined the degree of neurologic deficit (Canadian neurological scale) (15), functional deficiency (Bartel's daily activity scale) (16) and a side of neurologic deficiency. Blood presure was measured at 6 hours interval during the first 48 hours and afterwards three times daily (17).

Blood samples for determination of CPK concentration (MM and MB fraction) were taken from all patients with IBD and control group immediately after the admission or at least 72 hours after the clinical picture was revealed (18).

Between the third and tenth day after the onset of the disease computerized brain tomography (CT) or MRI was performed and localization and size of ischemic lesion were determined.

After the acute phase, based of neurologic examination and the degree of functional recovery as well as neuroradiologic findings we determined the degree of ischemic brain disease using Grotta scale (19).

Results were analyzed using Student's t tests, linear regression analysis and logistic regression analysis.

Results

The majority of patients were males, and mean age was 66.5 years. No significant differences of general characteristics were found between the observed groups. (Table 1).

Table1

The general characteristics of patients

	Male	Female	AGE	
IBD (n=80)	49	31	X=66.5	SD=7.67
Control group (n=40)	26	14	X=63.9	SD=8.12

Table 2

The characteristics of the observed group for mean value of blood pressure, age, size of ischemic lesion and fraction of MB and MB CPK (Student's test)

	AGE		CPK-TOT.		CPK-MB		TA SIS.		SIZE	
IBD (80)	66,5	7.67	75.1	34.2	11.2	6.2	143	14.8	2.46	1.23
TIA(14)	58.7	3.67	30.9	9.1	0.9	1.1	135	17.1	0.72	0.42
RIA(12)	61.4	4.01	43.2	11.4	1.6	1.5	144	14.9	1.32	0.57
BI-M(26)	62.2	4.04	76.3	19.5	4.9	2.8	149	12.4	2.29	1.11
BI-S(28)	68.1	4.94	110.2	31.1	19.3	3.9	156	11.5	3.73	0.87
CG (40)	63.9	8.12	37.7	10.7	0.4	0.34	136	14.5	0.0	0.0

BI-M-brain infarction-moderate; BI-S- brain infarction-severe

Comparing the observed parameters significant differences ($p<0.05$) were observed for age and degree of IBD severity. Also, significant differences ($p<0.05$) were found between the value of blood pressure and IBD severity.

Results for concentrations of CPK :

- Significant differences ($p<0.01$) were observed between patients with IBD and control group, as well as between patients with severe brain infarction (SBI) and control group ($p<0.01$) for CPK MM fraction, and a total concentration of CPK (MM + MB fraction).
- Significant differences were observed between control group and patients with TIA ($p<0.05$), between RIA patients and control group ($p<0.01$), and between SBI patients and control group for MB fraction of CPK ($p<0.001$).
- Also, comparing the observed parameters using the linear regreesion analysis we found significant correlation between the size of ischemic lesion and a total concentration of CPK ($p<0.01$), $r=0.469$ in group with IBD.
- Highly significant possitive correlation ($p<0.001$) was observed in the size of ischemic lesion and concentration of MB fraction of CPK.

Lipid oxidation is one of the theories that explains this process. Oxidation of LDL and Lp(a) occurs under the activity of free radicals and enzyme reactions. This influences the modification of apo B 100 (apoprotein specific for LDL), which leads to uncontrolled uptake of LDL from monocytes and macrophages in which degradation and deposition of cholesterol with foamy cell formation occur. This is the initial pathological process in atherogenesis and therefore is responsible for IBD (22–24).

This, we can say "cascade" system with extremly complex changes is responsible for atherosclerosis, which subsequently leads to IBD that is the most severe atherosclerotic brain complication. The main event in this system is the change in hemorrheological mechanisms, since we are dealing with the factors influencing cerebral microcirculation (25–33).

Examining these changes, we can get an insight in fine molecular changes at the level of ionic currents in ischemic tissue. Coagulation-anticoagulation system represents the dynamic balance allowing liquid blood state by cascade processes (25–33).

Decrease in oxidative phosphorylation is the initial process in IBD, followed by the production of highly ener-

Table 3

The correlation of localization of ischemic brain lesion blood pressure value age and fraction of CPK in patients with IBD

	AGE-LOC.*	CPK MM/LOC **	CPK MB/LOC***	TA SIS/LOC*
RHL-42(52.5%)	67.4	82.38	12.78	154.6
LHL-38(37.5%)	61.2	51.04	3.98	144.4

LOC-localization of ischemic brain disease; RHL-right hemisphere localization; LHL-left hemisphere localization; *$p<0.05$; **$p<0.01$; ***$p<0.001$

Discussion

Due to the rapid development of science and technology, contemporary medicine has a trend to create highly specialized experts, who tend to have a "tunnel vision". Multidisciplinary approach as well as the fact that organism is not only one organ, but a fine organization of interrelationships is frequently neglected (1–2, 20).

Vascular brain diseases are ranked the third as the cause of morbidity and mortality in most countries independently of their medical technological progress and socio-economical status. Ischemic brain disease (IBD) is represented in 80% of the cases.

Primary disorder responsible for the development of IBD is atherosclerosis of cerebral arteries. Numerous factors, such as heredity and habits influence the progression of this process. Since etiopathogenesis of atherosclerosis is still unclear, this study deals with actual processes leading to atherosclerotic cerebral changes, specially with hemorrheological and coagulation disorders. Less frequently the cause is lacunar infarction occurring due to fibrinoid degeneration of penetrating cerebral arteries caused by hypertension or brain infarction due to cardiac origin embolism (21–23).

getic phosphates necessary for maintanance of transmembrane ion potential. Consequently, there comes to an excessive neural membrane depolarization, which among other things leads to the activation of voltage-dependent calcium channels. Calcium ion influx through presynaptic membrane initiates the process of neurotransmitters release, including excitatory amino acid, which via ionotrophic and metabotropic receptors induces calcium ion accumulation in neurons. Calcium ion initiates the chain of metabolic process during which intermediary products create highly reactive oxidative matters, which can be created in other ways as well as during brain ischemia (27–33).

Besides these "local" events, during cerebral ischemia occur numerous systemic biohumoral mechanisms, mainly as compensatory, reactive and adaptive manifestations as the reaction of an organism to the strong, nonspecific stress, such as acute cerebral ischemia.

These mechanisms include the increase of circulating catecholamines, serotonin, cortisone, neuropeptides, activation of kallikrein-kinin system, and these substances become the mediators of functional disorders of other extracerebral systems, emphasizing the already existing hemorrheological disorders with the development of hypercoagulabile states.

This in turn causes consequent global hypoperfusion and increased blood viscosity with the creation of pathologic circles (2, 23, 25, 26, 35, 36).

Arteriosclerosis is the main disorder in IBD, so it is logical to assume that clinical disease manifestations of one of these organs will influence and probably initiate the pathological reactions including the manifestation of ischemic disease of other organs (25–33).

One of the studies dealing with this problem is the study of Jovicich et al. from 1989. Results of this study revealed that 30% of patients with TIA without the previous history of cardiac ischemic disease manifested symptoms of coronary disease during the exercise testing, including ECG-determined ischemia (37).

Experimental and clinical facts support the cerebral etiology of these changes. Thus, after the onset of IBD, blood pressure increases, as well as the serum concentrations of catecholamine and cortisol. Also, ST depression in patients with IBD was registered in 15-40% of cases from previous studies. Prevalence of asymptomatic ST depression in patients with angina pectoris was 43-60% and in patients without the history of cardiac disorders was 6-8%. According to this data, ST depression was more frequently present in patients with IBD than in asymptomatic patients and less frequently in patients with coronary heart disease (2–6).

Besides, necrosis of myocardial cells was found in experimental studies after the occlusion of cerebral arteries in absence of coronary heart disease. Also, according to our findings patients with ischemic lesion in the right cerebral hemisphere had the poorer prognosis (12), what was supported by experiments and examination during the neurosurgical treatment of epilepsy, which showed that stimulation of right insular cortex region caused the increase in blood pressure, heart rate and serum catheholamine, while the stimulation of the other side had the opposite effects (2–4, 12).

Results of rare recent clinical studies support these findings. So, the finding of significantly higher frequency of certain changes in continuous ECG, with frequent VA-s (ventricular arrhythmias), abnormal variations of minute heart volume, heart rate, blood pressure and cerebral flow registered by Holter ECG monitoring, power spectar analysis, transcerebral Doppler sonography etc., indicated the cerebral origin of these changes (38–43).

A certain progress was achieved recently in explaining the pathogenesis of cardiac complications including ECG changes such as ST depression and VA-s. Certain structures in CNS, which could be responsible have been identified. In some studies was confirmed that the prognosis was worse if the ischemia was in the irrigation zone of right a. cerebri media. Besides, the variations in blood pressure, increase in heart rate and level of circulating catecholamines during the stimulation of right insular cortex region supported these findings. Thus, the most important afferent input is placed in this region (13, 44–54).

There is an evidence confirming the involvement of neural structures in cardiac complications mechanisms, so that these complications are more frequently found in right cerebral hemisphere ischemia, especially with insular region involvement. The most acceptable explanation would be the rapid increase in circulating catecholamines and cortisone accompanied with age-dependent hemorrheological disorders, which were responsible for the development of cerebral and probably heart ischemia (25, 26, 46–56).

Our study supports these findings.

Conclusion

Results of our study support the idea of neural structure involvement in pathogenesis of cardiac complications and their laboratory correlates (increase in concentration of MB fraction of CPK.

Statistical analysis shows that highly positive correlation exists between the concentration of MB fraction of CPK and size and localization of ischemic brain lesion.

Further investigations of larger patients group should reveal the other, potentially important clinical, electrophysiological and laboratory predictors of the development of extracerebral (cardiac) complications in patients with IBD.

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Апстракт

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ЗНАЧАЈ ОДРЕЂИВАЊА ФРАКЦИЈА КРЕАТИНИН-ФОСФОКИНАЗЕ КОД БОЛЕСНИКА СА АКУТНОМ ИСХЕМИЈСКОМ БОЛЕШЋУ МОЗГА

Васкуларна обољења срца и мозга представљају водеће узроке смрти у цивилизованом свијету. У току развоја мождане исхемије могућ је развој кардијалних компликација. Циљ овог истраживања је био да утврди постојање промјена у фракцијама креатинин-фосфокиназе као сензитивног лабораторијског показатеља паренхимске лезије мозга и/или срца, у односу на локализацију и величину исхемијске лезије можданог паренхима и присуства/одсуства фактора ризика за исхемијску болест мозга и срца. У испитивање је укључено 80 болесника са акутном исхемијском болешћу мозга (АИБМ) без података о ранијој коронарној болести. Свим болесницима је у првих 48 сати од развоја АИБМ, узиман узорак венске крви за одређивање укупне (мишићне – ММ) и срчане фракције креатинин фосфокиназе (МВ), а потврда исхемије можданог паренхима је вршена помоћу прегледа мозга компјутеризованом томографијом или магнетном резонанцом. Такође свим

болесницима са АИБМ је детаљно узимана анамнеза о постојању фактора ризика за ИБМ и исхемијске болести срца, а у циљу потврде ових фактора одређиван је профил гликемије, липидни статус и мјерене вриједности крвног притиска 6 пута дневно. Независне варијабле код статистичке обраде биле су : животна доб, степен тежине и страна неуролошког догађаја, величина исхемијске лезије, и максималне вриједности систолног и дијастолног притиска. Зависне варијабле су биле вриједности фракција креатинин фосфокиназе. Контролну групу (n=40) су чинили болесници са неваскуларним неуролошким обољењима. Статистичком обрадом анализирани су сви параметри и њихов међусобни односи. Резултати су показали високостатистички значајну повезаност повећаних концентрација СРК- ММ и МВ фракције са величином и локализацијом исхемијске лезије у десној церебралној хемисфери, што је високостатистички значајно за МВ фракцију у укупној групи болесника са АИБМ, а за ММ фракцију за теже облике ИБМ. Такође су нађене високо статистички значајно веће вриједности ових фракција у односу на контролну групу болесника.

К љ у ч н е р е ч и : **mozak, veliki, ishemija; miokard, ishemija; krv, hemijske analize; kreatinin; kreatinin kinaza.**