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Changes of cortisol levels and index of lipid peroxidation in cerebrospinal fluid of patients in the acute phase of completed stroke

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Background. *Brain ischemia initiates series of biochemical reactions that could, directly or indirectly, induce and extend processes that damage numerous cellular and subcellular structures. One of the reactions of organism to ischemia is the increased release of glucocorticoid hormones, included in regulation of effects of numerous mediators/modulators that could be released in the acute phase of brain ischemia. Considering that brain infarction induced systemic response of organism to stress, we presumed that it reflected the contents of cortisol in the cerebrospinal fluid (CSF) during the acute phase of the disease, and that cortisol influenced damaging processes of lipid peroxidation in CNS initiated by ischemia. The aim of our investigation was to define temporal dynamics and manner correlation of cortisol concentration and index of lipid peroxidation (ILP) in the CSF patients in the acute phase of completed stroke.* ***Methods.*** *In this investigation we followed changes of cortisol concentration and ILP in the CSF of 53 patients in the acute phase of completed stroke. Control group included 14 age and sex matched patients, subjected to diagnostic lumbar radiculography because of the sudden motor deficiency onset, without disturbances in the CSF passage and without pain and the consequences of anti-pain and anti-inflammatory therapy. From the perspective of the duration of period after an ischemic episode patients were divided into four groups: group A: 0–6 hours (n=12), group B: 7–12 hours (n=14), group C: 13–24 hours (n=14), and group D: 24–48 hours of postischemic period (n=13). Concentration of cortisol in the CSF was measured by quantitative RIA method (Cortisol Bridge kit, Biodata). The ILP was determined according to the spectrophotometric method.* ***Results****. Concentration of cortisol in the CSF of patients with completed stroke was of amount 69 ± 6.7 pmol/ml CSF and was significantly increased ($p<0.001$) compared to the values of the control group (2.49 ± 0.29 pmol/ml CSF). From 0 to 6 hours after the ischemic insults concentration of cortisol in the CSF the amount was 15.9 ± 2.4 pmol/ml CSF ($p<0.001$), then was progressively increasing and was maximal from 13 to 24 hours after insults (123.2 ± 7.1 pmol/ml CSF), ($p<0.001$). In patients with completed stroke ILP in the CSF was twice increased in the course of 48 hours compared to controls (0.54 ± 0.08 nmolMDA/ml CSF), ($p<0.05$). By comparing the observed parameters we found significant negative correlation between this two indicators in period from 7 to 24 hours ($r=-0.77$), ($p<0.001$).* ***Conclusion.*** *The main potentially protective effect of the increased CSF cortisol concentration in patients with completed stroke in the acute phase could be the decrease of deleterious effects of lipid peroxidation reactions induced by ischemia. This mechanism could be the attempt of organism to compensate ischemia-disturbed homeostasis.*

K e y w o r d s : **brain ischemia; brain infarction; cerebrospinal fluid; hydrocortisone; lipid peroxidation; homeostasis.**

Introduction

Brain ischemia, as a powerful stressogenic factor, induces series of organism defensive mechanisms which have the common aim to protect the disturbed homeostasis (1, 2). As well as the local brain ischemic damage, brain infarction also includes systemic changes which represent general response of organism to stress (1, 3).

The mechanisms that include the reaction of organism to stress, are the powerful and almost immediate activation of regulatory hypothalamus-hypophysis-adrenal cortex-axis (HHA-axis), with consequently increased release of glucocorticoid hormones in blood. The increased necessity of organism for glucocorticoids was especially expressed in the acute period of stress (4–6). Cortisol was included in regulation of numerous mediators/modulators effects that could be released in stress, and thus influenced the quality of organism responses (7).

Glucocorticoid hormones showed various effects on CNS. It was shown that they made changes in synthesis or degradation of numerous cell proteins induced by glucocorticoids (5, 8, 9). Glucocorticoids also participated in regulation of maturation and differentiation of the cells in CNS, as well as in the modulation of electrophysiological activity, neuronal transmission, metabolism of biogenic amines and neuropeptides (8, 10, 11).

More than 95% of total glucocorticoid effects was attributed to cortisol, which was mostly present in blood as a bound form (12, 13). The importance of glucocorticoid hormones and their presence in CSF in different pathological circumstances in CNS have still not been sufficiently explained.

Some investigations clearly showed the increased activity of HHA-axis in the early phase of ischemic insult, as well as the existence of significantly negative correlation between the activity of HHA-axis and relative distance of injury (infarction) from the brain frontal lobus (14, 15). It was assumed that frontal injuries led to the disturbed balance between biogenic amines and cortisol systems, considering that biogenic amines (noradrenaline at the first place) regulated the deliver of releasing factors for corticosteroids (4, 16).

In the course of the acute ischemic insult patients were frequently exposed to the repeated stress (various cardiovascular complications, infections, emotional reactions, etc.). Repeated stress enhanced the magnitude of adrenal cortex sensibility for ACTH, and in this way prolonged the circumstances for hypercorticism (15, 17, 18). This prolonged extreme hypercorticism potentiated series of negative consequences on metabolism, cardiovascular and immune systems. High lethality rate was registered in patients with extreme values of cortisol in plasma and catecholamines in urine (19–22).

Numerous studies demonstrated that glucocorticoids in brain inhibited rising, releasing or activity of many mediators which induced the damage of blood vessels endothelium and rising edema (2, 7, 12, 23–25). It was also shown that glucocorticoids inhibited formation of prostaglandins and leukotriens by inhibiting ciklooxygenase and lipooxygenase pathways, and inducing the inhibition of phospholipase A_2 in cells (2, 12, 22–27). Glucocorticoids showed this effect by the synthesis induction of protein inhibitors of phospholipase A_2 in cells (12, 27). Certain studies showed that glucocorticoids inhibited lavage of proteins through endothelium, not only by suppressing vasodilatative mediators (prostaglandins, bradiquinins, histamins, etc.), but also by decreasing micro-vascular permeability through induction of synthesis of corresponding protein inhibitors in endothelial cells (2, 25–28). Glucocorticoids probably had the role of free radicals scavengers and thus had improved cells survival and decreased brain swelling (5, 23).

Considering that brain infarction induced systemic response of organism to stress, we assumed that it reflected contents of cortisol in the CSF during the acute phase of disease, as well as the cortisol-influenced damaging processes of lipid peroxidation in CNS, initiated by ischemia. The aim of our investigation was to define temporal dynamics and correlation of cortisol concentration and index of lipid peroxidation in the CSF patients with the acute phase of completed stroke.

Methods

Investigation included 53 patients of both sexes with completed stoke, between the ages of 55 and 70. These patients were under no therapy at the moment of samples collection. Completed stroke was confirmed by CT scan.

From the perspective of the period of duration after an ischemic episode patients were divided into four groups: 1) 0–6 hours (n=12); 2) 7–12 hours (n=14); 3) 13–24 hours (n=14); and 4) 24–48 hours of postischemic period (n=13).

Control group included 14 patients age and sex matched, subjected to diagnostic lumbar radiculography with sudden onset of motor deficiency, without disturbances in the CSF passage, without therapy and with no symptoms and signs of the acute inflammatory, neurodegenerative or psychiatric diseases.

Samples of CSF (1 ml) were collected by lumbar punction in the period from 8 to 10 hours in the morning in plastic tubes and were frozen until the appropriate biochemical analysis. Concentration of cortisol in the CSF was measured by quantitative RIA method (Cortisol Bridge kit, Biodata).

Index of lipid peroxidation (ILP) was determined according to the method of Andreeva, et al. (29) after stimulation of lipid peroxidation *in vitro* with Fe-salts. Thiobarbituric acid reacted with malondyaldehyde (MDA), which originated from polyunsaturated fatty acids in the process of lipid peroxidation, and formed a colored complex. Concentration of generated MDA was measured spectrophotometrically on 533 nm.

All the values were presented as the mean value ± standard error (SE). Student's *t*-test for the independent samples was used for the evaluation of statistical differences

between the examined parameters. Correlation between single measured parameters was determined by measurement of regression correlation coefficient.

Results

Concentration of cortisol in the CSF of patients with completed stroke was 69 ± 6.7 pmol/ml CSF and was significantly increased ($p<0.001$) compared to the values of the control group (2.49 ± 0.29 pmol/ml CSF) (Fig. 1). From 0 to 6 hours after the ischemic insults concentration of cortisol in the CSF was 15.9 ± 2.4 pmol/ml CSF ($p<0.001$), then was progressively increasing and reached maximal values from 13 to 24 hours after the insults (123.2 ± 7.1 pmol/ml CSF), ($p<0.001$) (Fig. 1).

Index of lipid peroxidation in the CSF was increased twofold in patients with completed stroke in the course of 48 hours, compared to controls (0.54 ± 0.08 nmol MDA/ml CSF), ($p<0.05$) (Fig. 2). Follow up of temporal dynamics of ILP in the CSF of stroke patients was the least increased in the group from 0 to 6 hours after ischemia (0.74 ± 0.07 nmol MDA/ml CSF), ($p<0.05$), but was gradually increasing in the other groups and in the group from 25 to 48 hours after ischemia was 1.89 ± 0.26 nmol MDA/ml CSF (Fig. 2).

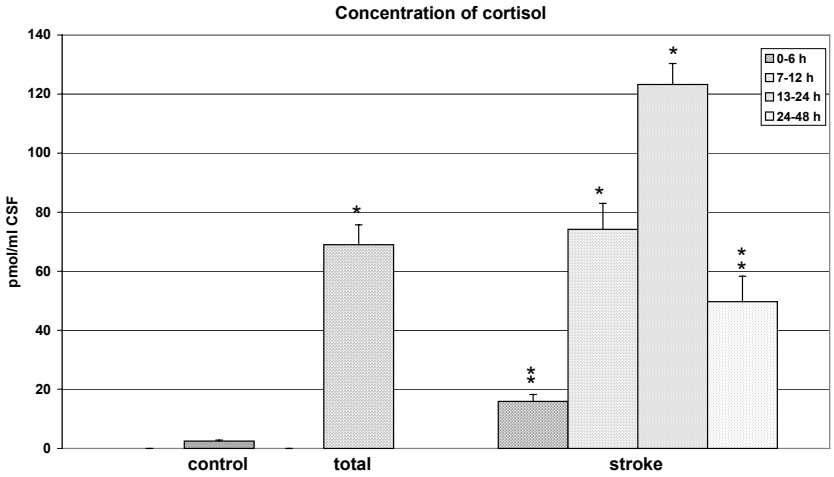

Fig. 1 – Concentration of cortisol in the CSF of patients with completed stroke. Values are given as pmol/ml CSF. Mean ± SE

* - Student's t-test, $p< 0.001$.
Total - average value of all patients with completed stroke;
Control - concentration in the control group.

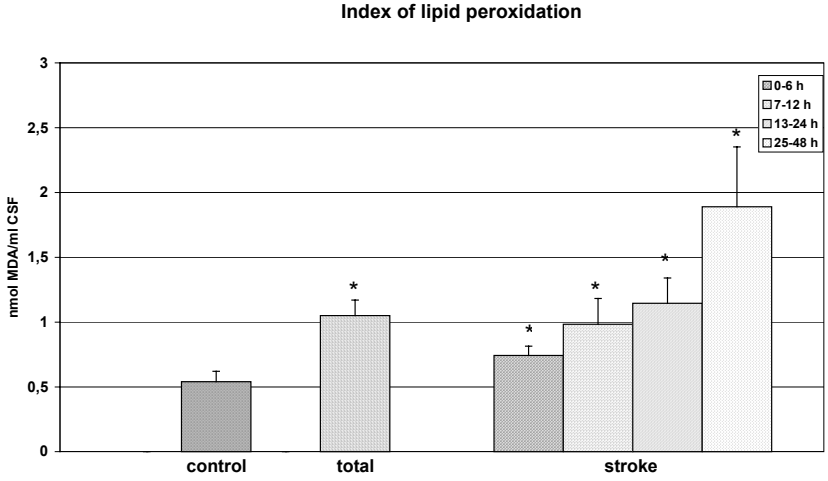

Fig. 2 – Index of lipid peroxidation in the CSF of patients with completed stroke. Values are given as nmol MDA/ml CSF. Mean ± SE

* - Student's t-test, $p< 0.05$.
Total - average value of all patients with completed stroke;
Control - value in the control group.

Comparing the observed results significant negative correlation was found between the concentration of cortisol and values of ILP in the CSF in period from 7 to 24 hours after insults (r=-0.77, p<0.001) (Fig. 3).

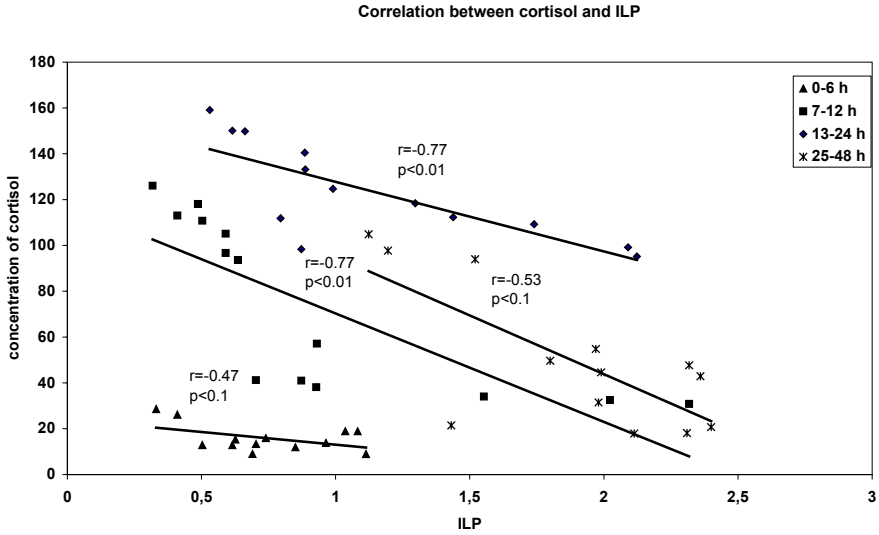

Fig. 3 – Correlation between concentration of cortisol and index of lipid peroxidation in the CSF of patients with completed stroke.
r - correlation coefficient;

Discussion

Ischemic lesions in CNS are pathological basis for neuronal cell damage and comprise activation of diverse mediator systems and homeostatic responses. Brain ischemia triggers a cascade of free radical reactions, causing structural and functional damage of biomolecules, disturbances in intracellular metabolism, as well as the damage of the adaptive response capabilities, all resulting in neuronal cell death (30, 31). It is known that the increased production of active oxygen species in the forebrain cortex and striatum is present in the early stage of brain ischemia. This process leads to chain reaction of free radical propagation, increase in superoxide anion/superoxide dismutasa ratio, and the decreased activity of glutathione reductase. Thus it is suggesting the insufficient function of antioxidative defense system for superoxide anion and hydrogen peroxide removal, as well as for removal of other peroxides formed in reactions of reactive oxygen species and biomolecules (32, 33).

Lipid peroxidation, as a consequence of free radical reaction, causes damage to cell membranes and their function. Our results indicated that the index of lipid peroxidation in the CSF of patients with completed stroke increased progressively during first 48 hours. Amplification and propagation of lipid peroxidation during the acute period of completed stroke were also observed. Consequences of these toxic reactions were alterations in cell membrane permeability and neurotransmitter receptor presentation, disturbance of cell membrane function and integrity, accompanied with malfunction of membrane enzymes and extracellular/intracellular communications (34).

Degradation of cell membrane phospholipids increased the level of free fatty acids, above all the arachidonic acid (35, 36). Clinical studies, including patients with the acute brain infarction, demonstrated significant rise of $PGF_{2\alpha}$ and PGE_2 content in CSF, and the increase of peroxidation products, originated mostly during arachidonic acids cascade (37). Application of selective enzyme blockaders in arachidonic acids cascade decreased brain edema and protected neurons in certain regions of brain from ischemic damage (36, 38). To a certain extent, these effects were caused by the decreased production of free radicals in arachidonic acids cascade (36, 38).

Integrity of cellular and subcellular membranes was the fundamental assumption for normal activity and prevention of irreversible functional and structural damage of neurons. The increased release of glucocorticoids was one of the initiated protective mechanisms during the acute brain infarction. The level of cortisol in CSF of patients with the acute brain infarction was about 28 times higher compared to the control group, with the peak values measured 13–24 hours after ischemia.

Some experimental studies indicated that glucocorticoid hormones inhibited the process of lipid peroxidation in all brain structures (2, 27, 39, 40). It was also shown that the application of glucocorticoid hormones immediately before unilateral carotid ligation prevented ischemic alteration of EEG activity and decreased brain edema (21, 39). Furthermore, the application of glucocorticoid hormones after the experimental cardiac arrest led to recovery of normal ECG activity, the increase of Na^+, K^+, ATPase activity and clinical recovery (21, 39). The results of other studies indicated glucocorticoid-induced reduction of free radical and

lipid peroxide formation in all brain structures, as well as the increased activity of neuronal Na^+, K^+, ATPase during the early phase of brain concussion and subarachnoid hemorrhage (21, 41, 42). Our results indicated the presence of inverse linear correlation between the concentration of cortisol and the index of lipid peroxidation in CSF of patients with the acute brain infarction 7–24 hours after the insult ($r=-0.77$, $p<0.001$). The increased level of cortisol in CSF of patients with brain infarction during the acute period probably represented one of protective responses, aiming to restrain destructive effects of lipid peroxidation reaction triggered by ischemia.

Although the mechanisms of glucocorticoid-induced suppression of lipid peroxidation process have not still been well understood, it was assumed that it included the inhibition of dien formation (intermediary product during the process of lipid peroxidation) and physical interaction with cell membrane, preventing spreading of lipid peroxidation chain reaction (39, 40). Glucocorticoids probably reduced the process of lipid peroxidation by stabilizing cell membrane, thus protecting Na^+, K^+, ATPase activity, reducing the damage of mitochondrial membranes and preserving electrochemical ionic gradient, as well as cell energy metabolism (21, 23, 40, 42). It was also shown that glucocorticoids induce synthesis of phospholipase A_2 protein inhibitor, reducing arachidonic acid release and production of prostaglandins, leucotriens and toxic hydro-peroxides and endoperoxides, released in arachidonic acid cascade (25, 26, 42).

Conclusion

Results obtained in this study indicated that the acute brain infarction induced numerous pathophysiological processes and reciprocal defensive mechanisms, reflected as biochemical changes in the CSF.

Index of lipid peroxidation in the CSF as the indicator and the consequence of neuronal cell membranes damage was increasing progressively during the first 48 hours after the brain infarction.

Concentration of cortisol in the CSF was increased in the acute phase of brain infarction with maximal values in period from 13 to 24 hours after the ischemic episode and the was the indicator of the systemic response degree expression of organism.

Considering that in the period from 7 to 24 hours after the brain infarction there was the inverse linear correlation between the concentration of cortisol and the index of lipid peroxidation in the CSF, it was assumed that cortisol acted neuroprotectively in the acute period of brain infarction, primarly by reducing the disruptive effects of lipid peroxidation reaction.

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A p s t r a k t

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PROMENE KONCENTRACIJE HIDROKORTIZONA I INDEKSA LIPIDNE PEROKSIDACIJE U CEREBROSPINALNOJ TEČNOSTI BOLESNIKA U AKUTNOJ FAZI INFARKTA MOZGA

Uvod. Ishemija mozga pokreće niz biohemijskih reakcija koje direktno ili indirektno dovode do inicijacije i širenja procesa koji oštećuju mnoge celularne i subcelularne strukture. U okviru reakcije organizma na ishemiju dolazi do povećanog oslobađanja glukokortikoidnih hormona, koji su uključeni u regulaciju dejstva mnogih medijatora/modulatora koji se oslobađaju u akutnoj fazi ishemije mozga. S obzirom na to da infarkt mozga dovodi do pokretanja sistemskog odgovora organizma na stres, pretpostavili smo da se to odražava na sadržaj hidrokortizona u cerebrospinalnoj tečnosti (CST) tokom akutne fazi bolesti, kao i da hidrokortizon utiče na ishemijom pokrenute oštećujuće procese lipidne peroksidacije u CNS-u. Cilj ovog istraživanja je bio da tokom akutne faze infarkta mozga definišemo vremensku dinamiku promene i karakter povezanosti koncentracije hidrokortizona i indeksa lipidne peroksidacije (ILP) u CST. **Metode.** U ovom istraživanju praćena je promena koncentracije i ILP u CST kod 53 bolesnika sa infarktom mozga u akutnoj fazi. Kontrolnu grupu predstavljalo je 14 ispitanika podvrgnutih dijagnostičkoj lumbalnoj radikulografiji zbog naglog razvoja motornog deficita, bez smetnji u pasaži likvora, koji nisu koristili antidoloroznu i antiinflamatornu terapiju. Dinamika promene koncentracije hidrokortizona i indeksa lipidne peroksidacije praćena je u CST bolesnika sa akutnim infarktim mozga, koji su podeljeni u sledeće grupe: A. grupa: 0–6 časova nakon infarkta mozga (n=12), B. grupa: 7–12 časova nakon infarkta mozga (n=14), C. grupa: 13–24 časa nakon infarkta mozga (n=14) i D. grupa: 25–48 časova nakon infarkta mozga (n=13). Koncentracija hidrokortizona u CST određivana je kvantitativno, RIA metodom (Cortisol Bridge kit, Biodata). Indeks lipidne peroksidacije u CST određivan je spektrofotometrijskom metodom. **Rezultati.** Koncentracija hidrokortizona u CST kod ispitivanih bolesnika sa akutnim infarktom mozga iznosi 69,0 $\pm$ 6,7 pmol/ml CST i statistički značajno je povećana ($p<0,001$) u odnosu na kontrolu (2,49 $\pm$ 0,29 pmol/ml CST). Do šestog časa nakon insulta koncentracija hidrokortizona u CST iznosi 15,9 $\pm$ 2,4 pmol/ml CST ($p<0,001$), zatim dalje raste i maksimum dostiže 13–24 časa nakon insulta (123,2 $\pm$ 7,1 pmol/ml) ($p<0,001$). Kod bolesnika sa infarktom mozga tokom prvih 48 časova izmeren ILP u CST u ovoj studiji iznosi 1,07 $\pm$ 0,12 nmol MDA/ml CST, što je statistički značajno povećanje ($p<0,05$) u odnosu na vrednost izmerenu u kontrolnoj grupi ispitanika (0,54 $\pm$ 0,08 nmol MDA/ml CST). Dobijeni rezultati pokazuju i statistički značajnu negativnu povezanost između ova dva pokazatelja u periodu od 7 do 24 časa nakon infarkta mozga ($r=-0,77$) ($p<0,001$). **Zaključak.** S obzirom da se hidrokortizon oslobađa u okviru akutne reakcije organizma na stres, to porast njegove koncentracije kod CST bolesnika u akutnoj fazi infarkta mozga može imati za cilj smanjenje oštećujućih efekata ishemijom pokrenutih reakcija lipidne peroksidacije i može predstavljati pokušaj organizma da na taj način kompenzuje narušenu homeostazu.

K lj u č n e r e č i : **mozak, ishemija; mozak, infarkt; cerebrospinalna tečnost; hidrokortizon; lipidi, peroksidacija; homeostaza.**