Dynamic Changes of The Brain Substances Metabolism in Experimental Peritonitis

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Özet: *EKSPERİMENTAL PERİDONİTTE BEYİN METABOLİZMASINDAKİ DİNAMİK DEĞİŞİKLİKLER*

Eksperimental peridonitteki beyinde olusan metabolik değişiklikler 250-300 gram ağırlıktaki 100 erişkin erkek sıçanda araştırıldı. Kan örneklerinde arteriyovenöz farkı tespit etmek üzere her hayvanda total protem, alkanin, üre, total lipid, kolesterol, betalıpoproteinler ve glukoz tayinleri yapıldı.

Elde ettiğimiz sonuçlar experimental peridonitli sıçan beyninde total protein albumin, total lipidler, betalipoproteinler ve glikoz gibi metabolik karakterlerde değişikliklerin olduğunu göstermiştir.

Experimental peridonitin son safhasında sıçan beyninde enerji üreten maddelerin kaybolduğu bildirilmiştir. Bu duyular peridonitin rutin tedavisinin gözden geçirilerek beyin metabolizmasını düzelten ilaçların da tedaviye eklenmesi gerektiğini düşündürmektedir.

Anahtar Kelimeler: Eksperimental peridonitin, beyin metabolizması

Summary: *For studying metabolic processes of brain in animals with experimental peritonitis under dynamics an investigation was carried in 100 maturated male rats of 250-300 g. In theirblood samples we analized contents of total protein, albumin, urea, total lipids, cholesterine, beta-lipoproteins and glucose, taking in account arteriovenous difference for every substrate.*

The results obtained strongly suggested that the rat brain in experimental peritonitis was found with expressed phased changeable metabolic character of contents of total protein, albumin, total lipids, betalipoproteins and glucose.

In terminal states of experimental peritonitis the "elimination" of energetic and plastic substances from the rat brain was reported. These facts result in the necessity of critical review of routine treatment course of peritonitis and the inclusion of drugs, improving the metabolism of the brain.

Key Words: Experimental peritonitis, brain substances

Acute generalized peritonitis (AGP) gives a high mortality index till present days.

Last years investigation in peritoneal efficient treatment and reanimation successes (10) resulted in detection of a state, named as poliorganic insufficiency syndrome (2,3,7,9-11).

Physicians and scientists undertook multivariative studies off entestines, liver, kidney, lung, circulation, hemostasic functioning failures (3-6). And it seems strange that there is a poor number of works, explorating the topic of central nervous system performance, which is the regulator of all systems and organs. Some of these works deal wich functional abnormalities of central nervous system (CNS) (3,10). As substance metabolic process of brain in AGP is not clarified by clinicians and experimentators by the reasons of specific localisation of the organ, making difficult metabolism studying, the aim of this work is to investigate the metabolic processusstate in brain in experimental peritonitis under dynamics.

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MATERIAL and METHODS

We took maturated male rats of 250-300 g as experimental animals. Experimental peritonitis was performed by N. M. Baklykov method (1965) introducing 1ml/100 g of 1% soluble autofeces with our modification following preliminary reproduction of destructive focus by 10% soluble of calcium chloric.

The observation of metabolic processes in the brainunder dynamics in experimental peritonitis was performed by arterial blood samplings from left ventricle and samplings of venous return going from brain, made by punctionning of internal jugular vein. 5 minutes before blood samplings, animals were made soluble solipsol injection (0,2 g/100 g of weight).

Total protein, contents of albumin, total lipids, cholesterine, beta-lipoproteids, glucose, urea were analized in samples with respect of arteriovenous difference (AVD) of every substrate. Protein was studied by biureth device, albumin-by bromcresole one, urea-by urease method, glucose-by enzymatic glucosooxidant, cholesterine-by Ilk method with biochemical analysator FP-901, total lipids content-by "Bio-LA-Test" sets (Tchecoslovakia), beta-lipoproteins-by turbodimetric method.

4 series of experiments were carried out relatively to the 1t, 2d and 3d days of experimental peritonitis. The 4th blood sampling was taken on the 4-5th day from agonized animals. Control group was formed from 20 intact maturated male rats.

Investigation results

Among nitrous metabolism we analysed contents of total protein, albumin and urea at the moments of blood coming in the brain (arterial one) and coming from (jugular venous one).

Control trial showed the deep decreasing of total protein content from 67,4 +/-1,9 g/l to 60,2 +/-1,5 g/l ($p<0,05$) in the venous return, as well as the unreliable diminuation of floating albumin from 34,8 +/-1,9 g/l to 32,2 +/- 0,9 g/l. Uratemia in venous blood increased from 5,23 +/-0,6 mMOL/l to 6,2 +/-1,1 mMOL/1 ($P> 0,05$), i.e. experimental animals brain retained the total protein and albumin. An insignificant urealy increasing is due evidently to consumption of protein molecules for cellular structure formation.

Reproduction of the pathological process resulted in significant AVD reducing in general protein and albumin content to 2,6 g/l and 2,3 g/l relatively. Approximately proportional were AVD changes of urea (see table 1).

Experimental peritonitis progressing in animals showed an allogic metabolic imaging of the three mentioned above indices of nitrous exchange.

Simultaneously we reported a continuous reduction of total protein and albumin of arterial blood (40,9 +/-1,2 g/l and 21,8 +/0,8 g/l relatively) on the background of slightly marked reducing of total protein and albumin in venous blood (47,4 +/-1,4 g/l and 24,6 +/-0,5 g/l relatively). When passing through the brain blood was found during the second day not only loosing protein and albumin, but eliminated of these substances, hypothetically, due to cellular structure desintegration. On this background ureal content sharply increased in arterial (12,46 +/- 1,8 mMOL/l) and venous blood (11,18 +/- 0,84 mMOL/l). To the third-forth days the experimental animal state aggravated severely and the tendency of protein-albumin elimination progressed, coming to its maximum in agonized animals. At this very time we reported unexpective and incomprehensive ureal reduction both in arterial and venous blood.

Analysis of lipid metabolism showed that when passing through the brain blood was found lipid content slightly reduced from 4,2 +/- 0,16 g/l to 4,01 +/- 0,14 g/l ($P>0,05$) , on the contrary blood beta-lipoproteids reduced significantly from 25,2 +/-0,5 till 19,87 +/- 0,65 conv. units, as well as cholesterine content from 1,93 +/-0,1 mMOL/l to 1,14 +/- 0,2 mMOL/l ($P<0,05$), see table 2.

These findings testify in check animal sample that the brain retains lipid metabolic products. We suppose that this is due to lipid necessity for cellular membrane construction and for compensation of energy in normal physiological brain state. During the first day of experimental peritonitis we reported the decreasing of all the

Table I: Dynamics of nitrous metabolic variable changes in the brain in experimental peritonitis.

Indices	Check sample		1st day		2st day		3st day		Terminal state	
	A	V	A	V	A	V	A	V	A	V
Total protein	67,4 +-1	60,2 9+-1	52,8 5+-6	50,2 8+-8	40,9 2+-1	47,4 3+-1,	52 4+-1,	58 6+-1,	46,6 7+-1,	56,2 3+-1,3
Albumin	34,8 +-1	32,2 9+-0	27,1 9+-0	24,8 8+-0,	21,8 7+-0,	24,6 8+-0	31,1 5+-1,	32,3 2+-1,	25 7+-0,	32,4 6+-1,4
Urea	5,2 +-0,6	6,2 +-1,	5,4 1+-0,	6,2 7+-0,	12,5 3+-1,	11,2 8+-0,	2,7 8+-0,	3,3 2+-0,	4,1 5+-0,	4,7 3+-0,3

three parameters of lipid metabolism: total lipids, beta-lipoproteins and cholesterine, relatively from 3,8 +/- 0,2 to 3,18 +/- 0,23 g/l; from 14,8 +/- 0,45 to 12,1 +/-0,81 conv. units (P< 0,05) and from 1,3 +/- 0,2 to 0,98 +/- 0,12 mMOL/l (P>0,05). We detected also the AVD index growth in total lipids from 0,13 to 0,62 g/l with simultaneous diminuation of AVD index in beta-lipoproteins and cholesterine from 5,33 to 2,08 conv. units and from 0,79 to 0,4 mMOL/l relatively. During the second day of the pathological process development the diminuation of all the three studied components of lipid metabolism took place in arterial blood with their slight decrease in venous blood (see table 2). As a result we found out inversion of the brain to these studied substances. Blood circulation through the brain resulted in unreliable increasing of total lipids and beta-lipoproteids contents. As for cholesterine, its content in both artery and vein was unchangeable. In general, lipid metabolic process showed great similiarity with protein metabolic features in rat with experimental peritonitis during the second day of pathology development.

During days succeded until animal agony the character of lipid metabolism to total lipids and lipoproteins in the brain remained stable, associated with approximatively equal decrease of total lipids both in arterial and venous blood to the 3d day to 1,53 +/- 0,36 g/l and 1,85 +/- 0,23 g/l (P< 0,05) relatively with expressed reliable increase of this parameter in terminal state up to the normal control level.

Beta-lipoproteid content after its minimal accurance turned to slight growth so that during the agony substrate content in venous blood got initial level and in arterial blood it was significantly less-16,3 +/-0,7 conv. units (P<0,01). But at any time of observation, beginning from the 2d day, the process of eliminaton of the both substrates took place in the brain. Cholesterine was found with undifferencial changes.

Hungry rats of the check sample were found with an accurate high level of glucose content in arterial blood in comparison with venous one-6,16 +/-0,2 mMOL/l and 5,66 +/- 0,26 mMOL/l (P> 0,05) relatively, moreover in the serie of 7 animals we detected the reducing of glucose in 6 cases in blood, passed through the brain (see drawing 1), that points on the reliable absorption of this substrate by the experimental organ.

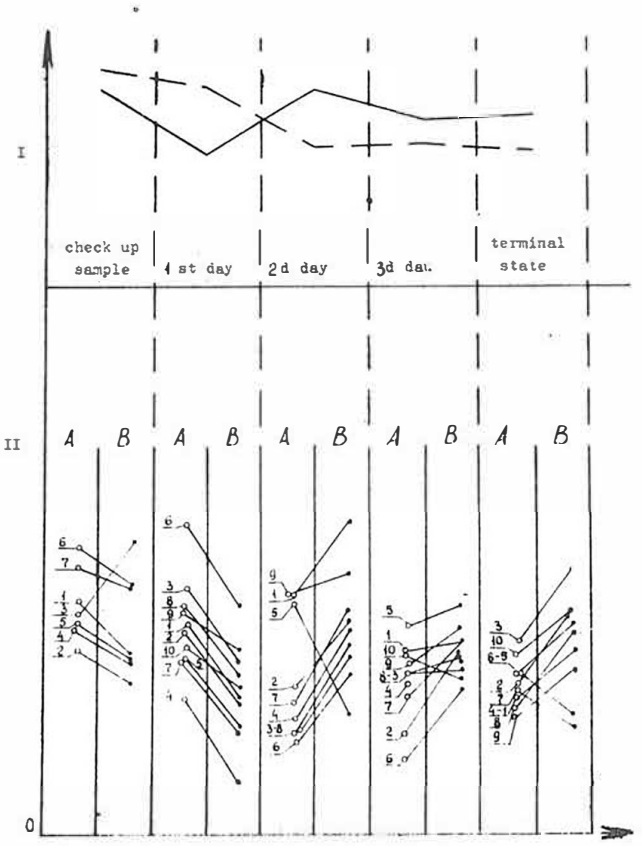
After the reproduction of the experimental peritonitis during the first day of observation the intensity of glucose absorbtion by the brain encreased expressively (see drawing 1). It was testified by the glusoce content index growthing in AVD from 0,50 to 1,95 mMOL/l in all 10 experimental animals. The aggravation of pathological process in animal sample during the second day of observation led to inverse character of glucose metabolism in the brain (see drawing 1). During the second day of pathology on the background of reliable diminuation of glucose in arterial blood from 5,68 +/- 0,36 mMOL/l to 3,96 +/- 0,48 mMOL/l (P< 0,05) we revealed an accurate increasing of glucose content in venous blood, coming from the brain, from 3,73 +/- 0,24 to 0,38 mMOL/l (P< 0,01). These features were reported in 8 cases of 9.

Close to the 3d day of experimental peritonitis the significant reduction of AVD on the account for glucose concentration reduction in venous

Table II: Dynamics of lipid metabolic variable changes in the brain in experimental peritonitis.

Biochem indices	Check sample		1st day		2st day		3st day		Day of the death	
	A	V	A	V	A	V	A	V	A	V
Total lipids	67,4 +-0,16	4,01 +-0,14	3,8 14+-0,2	3,18 2+-0,23	2,35 +-0,42	2,65 42+-0,3	1,53 +-0,36	1,86 +-0,23	3,92 +-0,46	4,46 46+-0,34
Beta-poprot	25,2 +-0,5	19,87 +-0,65	14,8 +-0,45	12,1 +-0,81	10,2 81+-0,56	11,5 +-0,3	11,45 +-0,28	13,15 +-0,2	16,3 +-0,7	18,8 +-0,92
Choles terine	1,93 +-0,1	1,14 +-0,2	1,3 +-0,2	0,98 +-0,12	1,06 +-0,22	1,1 +-0,3	1,54 +-0,31	1,25 +-0,2	1,22 +-0,21	1,52 +-0,1

Biochemical indices of AVD according to the experimental peritonitis



I- middle parameters
II - individual parameters
A- artery
V - vein

and increasing in arterial blood took place, testifying the inhibition of glucose metabolic process intensity, though in terminal stage the picture of this metabolism and its intensity were found as during the 2d day.

Summaring the findings results, characterizing metabolic processes in the brain of rats with experimental peritonitis we may report the staging of changes occured. The reproduction of pathological process resulted in diminuation of total protein, albumin, glucose, total lipids, cholesterine, beta-lipoproteins within two days of observation.

In addition, during the 1st day positive AVD characterized all studied substrates. As for glucose and total lipids in this period of time, we reported their slight increasing. This fact pointed out the expressed absorbtion of all these substrates by the brain in rats with primary stages of experimental peritonitis. During the second day of mentioned pathology on the background of progressing decrease of these substrates in arterial blood, i. e. under conditions of their deficiency in the brain, supposed on account for their deficit synthesis in the main biochemical structures as liver, lung etc., all these materials were eliminated from the brain. So, we hypothesize the brain was transferred in a producer of total protein, albumin, total lipids, beta-proteins and glucose.

We also suppose that the negative AVD in lipid and protein metabolism is due to the desintegration of cellular structures. As for carbohydrate exchange, the activity of gluconeogenesis was manifestated in severe dysfunctions of metabolism by the several scientists and physicians, f.

e. H. H. Huppiy and A.B. Gruzmann (1990). M. Y. Anestyady (1976) shared this opinion of the correlation between the desintegration of protein in acute intestinal obstruction with the gluconeogenesis growth. This imaging was the same during succeded days of observation. It is supposed that revealed metabolic disorders in the brain are the reason of the followed disfunctions of the central nervous system. It is of evidence that the treatment course in peritonitis should be reanalized and supplement by the medicines improving metabolism of the brain.

CONCLUSIONS

1. The method of the brain metabolism studies by the detection of the difference between substrate contents in forward and reverse flows of blood-arterial and venous ones-may be strongly considered as a reliable method.

2. The rat brain in experimental peritonitis is found with the expressed phased changeable metabolic character of total protein, albumin, total lipids, beta-proteins and glucose.

3. In terminal states of experimental peritonitis we reported the "elimination" of energetic and plastic substances from the rat brain, what is hypothetically the reason of functional lesions of the central nervous system.

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