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INFLUENCE OF INFECTED EXPERIMENTAL BILE PERITONITIS ON OPTIC DENSITY OF THE BLOOD PLASMA AND FUNCTIONAL STATE OF KIDNEYS

Keywords: *optical density of the blood plasma, infected bile peritonitis,kidneys, "loss" syndrome of sodium ions, excretion of titrated acids.*

Abstract. *The clinical course of infected experimental bile peritonitis in investigations on 86 white non-linear male-rats 24 hours following discrete flow of autobile from the common bile duct of rat through the formed defect of its wall by means of thermocoagulation and introduction of the contents of the small intestine was shown to be accompanied by growth of optical density of the blood plasma at wave length 250, 310,320,340 nm with the development of the "loss" syndrome of sodium ions against a background of reduction of titrated acids excretion and ammonia with urine.*

Introduction

Experimental modeling of the infected bile peritonitis was known to be carried out by means of introduction of sterile medical bile [2, 13, 14] and the contents of the small intestine into the abdominal cavity. At the same time the given method is not rather efficient, since it requires for its initiation damage factors,that occur mostly in clinic, does not take into account the guiding mechanisms of the infected bile peritonitis onset, spreading and progression of intraabdominal inflammatory process, does not reproduce local changes of the damaged organ and, as a whole, is not a representative model adequate to clinical form of the infected bile peritonitis. A model of the infected bile peritonitis at the expense of discrete introduction of autobile from the common bile duct of rat through the formed defect of its wall by means of thermocoagulation and additional introduction of the contents of the small intestine, was elaborated by us to solve the problem. Under such conditions the development of the infected bile peritonitis may cause changes of optical density of the blood plasma and results in renal damage, that probably, will assist complication of the clinical course of the infected bile peritonitis [6, 7, 16]. Simultaneously, optic density of the blood plasma and functional state of kidneys when simulating the infected bile peritonitis due to discrete introduction of autobile from common bile duct of rat through the formed defect of its wall by means of thermocoagulation and additional introduction of the contents of the small intestine are not sufficiently studied.

Purpose

To elucidate changes of optical density of the blood plasma and functional state of kidneys at experimental infected bile peritonitis after discrete escape of autobile from the common bile duct of rat through the formed defect of its wall by means of thermocoagulation and additional introduction of the contents of the small intestine.

Material and methods

Studies were conducted on 76 white non-linear male-rats weighting 0.16-0.18 kg under conditions of hyposodium diet. Experimental modeling of infected bile peritonitis was carried out by means of discrete flow of autobile from the common bile duct through the formed defect of its wall by means of thermocoagulation and additional introduction of 0.5 ml of the contents of the small intestine [9].

Renal function was studied introducing water from water-pipe into rat stomach in quantity 5% of body weight with the help of metallic probe with further collection of urine during 2 hours. Euthanasia of the animals was carried out immediately after urine collection by means of decapitation under ether narcosis. The blood was drawn into vials with heparin. Creatinine concentration was determined in blood plasma and urine according to the reaction with picric acid, sodium ions, potassium - by the method of flame photometry on PhF-1.

Protein concentration in urine was determined by sulfosalicylate method. Glomerular filtration according to endogenic creatinine clearance, relative, proximal, distal reabsorption of sodium ions, its filter fraction and clearance, protein excretion, creatinine, sodium and potassium ions, water clearance free of sodium ions, concentrated index of endogenic creatinine, indices of acid regulatory renal function

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according to formulae, adduced in the article, were calculated [10, 11]. Optic density of blood plasma in the range of wave lengths from 250 to 340 nm was determined [6].

Statistical processing of data was carried out by means of computer program "Statgrafics" and "Excel 7.0".

Discussion of the results

The results of the research have shown that the clinical course of experimental infected bile peritonitis in 24 hours following discrete flow of autobile from the common bile duct of rat through the formed defect of its wall by means of thermocoagulation and additional introduction of 0,5 ml contents of the small intestine is accompanied with optic density growth of the blood plasma at wavelength 250, 310, 320, 340 nm (table 1). Diuresis, relative diuresis, concentration and excretion of ions of potassium with urine, blood plasma creatinine remained without changes (table 2). Simultaneously, concentration and excretion of creatinin, its concentrating index, glomerular filtration increased. Concentration and excretion of the urine protein didn't experience changes, with the exception of standardized protein excretion on 100 mcl of glomerular filtrate which underwent inhibition (table 3). Investigations of acid regulatory renal function have detected reduction of standardized indices of excretion of titrated acids and ammonia (table 4).

The elaborated method of the infected bile peritonitis modeling at the expense of the wall coagulation of the common bile duct of rat and additional introduction of the small intestine contents reproduces destructive and inflammatory processes in it, which occur in case of infected peritonitis onset in patients. Perforation of the organ creates the conditions for escape of endogenic bile, contained in its lumen, into the abdominal cavity and gradual peritonitis development, which corresponds mostly to the character of pathological process in people. The presence of necrotized edges of the defective organ creates all the necessary conditions for polonged bile coming

Table 1

Indices of optic density of the blood plasma (D-um od) in ranges of wave lengths (250-340 nm) under conditions of modeling of infected bile peritonitis (x±Sx)

Indices	Control (n=9)	Infected bile peritonitis (n=10)
D_{250}, um od	0,176±0,0099	0,223±0,0150 p< 0,05
D_{260}, um od.	0,237±0,0154	0,282±0,0238
D_{270}, um od.	0,337±0,217	0,386±0,0327
D_{275}, um od.	0,372±0,0252	0,422±0,0338
D_{280}, um od.	0,380±0,0257	0,423±0,0337
D_{285}, um od.	0,336±0,0226	0,369±0,0316
D_{290}, um od.	0,235±0,0157	0,264±0,0218
D_{300}, um od.	0,059±0,0036	0,058±0,0043
D_{310}, um od.	0,009±0,0007	0,014±0,0013 p< 0,01
D_{320}, um od.	0,005±0,0001	0,010±0,0015 p< 0,02
D_{340},um od.	0,002±0,0005	0,004±0,0003 p< 0,05

P –probability of differences in comparison with the control;
n –number of observations

Table 2

Indices of the renal function under conditions of the infected bile peritonitis modeling (x±Sx)

Indices	Control (n=9)	Bile peritonitis (n=10)
Diuresis, mg/2 h · 100 g	4,33±0,300	4,62±0,125
Relative diuresis, %	86,77±5,999	92,52±2,516
Concentration of potassium ions in urine, mm/l	4,38±0,138	4,15±0,279
Excretion of potassium ions, mcm/2 h ·100 g	18,99±1,388	18,92±0,844
Creatinine concentration in urine, mm/l	0,439±0,0266	0,685±0,0613 p< 0,01
Creatinine excretion, mcm/2 h · 100 g	1,89±0,170	3,18±0,322 p< 0,01
Creatinine concentration in blood plasma, mm/l	51,2±2,82	45,9±4,91
Concentrating index of endogenic creatinine, od.	8,63±0,449	17,13±2,800 p< 0,02
Glomerular filtration, mcl/min · 100 g	306,4±19,32	676,32±120,00 p< 0,02
Protein concentration in urine, g/l	0,024±0,0042	0,017±0,0012
Protein excretion, mg/ 2 h · 100 gr	0,112±0,0245	0,078±0,0055
Protein excretion, mg/100 mcl C_{cr}	0,037±0,0086	0,015±0,0024 p< 0,02

p –probability of differences in comparison with control; n –number of observations

Table 3

Indices of sodium ions transport under conditions of infected bile peritonitis modeling (x±Sx)

Indices	Control (n=9)	Infected bile peritonitis (n=10)
Concentration of sodium ions in urine, mm/l	0,617±0,0264	3,175±0,4131 p< 0,001
Concentration of sodium ions in blood plasma, mm/l	138,6±4,71	138,25±1,057
Filtration fraction of sodium ions, mcm/min · 100 g	42,50±2,875	93,59±16,656 p< 0,02
Excretion of sodium ions, mcm/2 h · 100 g	2,69±0,250	14,81±2,093 p< 0,001
Excretion of sodium ions, nm/100 mcl C_{cr}	0,892±0,0855	2,88±0,563 p< 0,01
Absolute reabsorption of sodium, mcm/min · 100 g	42,47±2,874	93,46±16,656 p< 0,02
Relative reabsorption of sodium, %	99,947±0,0112	99,82±0,036 p< 0,01
Concentrating index of sodium ions, um. od	0,004±0,0001	0,023±0,0030 p< 0,001
Clearance of sodium ions, ml/2 h ·100 g	0,019±0,0012	0,107±0,0154 p< 0,001
Clearance of water free of sodium ions, мл/2 h · 100 g	4,32±0,298	4,51±0,120
Distal reabsorption of sodium ions, mcm/2 h · 100 g	607,2±56,91	623,9±14,39
Proximal reabsorption of sodium ions, mml/2 h · 100 g	4,49±0,300	10,59±1,987 p< 0,02
Distal reabsorption of sodium ions, mcm/100 mcl C_{cr}	1,659±0,1464	1,019±0,1598 p< 0,01
Proximal reabsorption of sodium ions, mm/100 mcl C_{cr}	12,19±0,331	12,78±0,203

p- probability of differences in comparison with control; n –number of observations

Table 4

Indices of acid adjusting renal function under conditions of infected bile peritonitis modeling (x±Sx)

Indices	Control (n=9)	Bile peritonitis (n=10)
Excretion of titrating acids, mcm/2 h ·100 g	68,55±5,953	66,55±7,460
Ammonia excretion, mcm/2 h ·100 g	363,0±36,08	392,74±68,433
Ammonia coefficient, un.	5,24±0,095	5,51±0,398
Concentration of hydrogen ions in urine, mcm/l	0,018±0,0044	0,018±0,0031
Excretion of hydrogen ions, nm/2 h · 100 g	0,083±0,0222	0,083±0,0149
Excretion of hydrogen ions, nm/100 mcl C_{cr}	0,026±0,0064	0,014±0,0021
Excretion of titrating acids, nm/100 mcl C_{cr}	22,40±1,611	11,36±1,134 p< 0,001
Hydrogen excretion, nm/100 mcl C_{cr}	118,4±9,97	60,82±5,690 p< 0,001

p –probability of differences in comparison with control; n –number of observations

from the lumen of organ into the abdominal cavity, development of pre-focal inflammation of the wall of the organ , that is, the area of the largest pathological lesion - the source of the lingering injury of the abdominal cavity is modeled. The development of the infected bile peritonitis is confirmed by growth of a number of microorganisms in bile: E.coli - 7,58±0,39 lg Kyo/ml, S.faecalis - 7,49±0,39 lg Kyo/ml, B.fragilis - 5,47±0,39 lg Kyo/ml, P.niger - 5,07±0,39 lg Kyo/ml. Escape of the infected bile into the abdominal cavity resulted in lesion of intestinal wall at the expense of the influence of hydrophobic bile acids [12] and microorganisms cited above. It promoted the development of essential dysbacteriosis in the lumen of small, large intestine [1, 3] and excessive escape of bile acids, endotoxin and pathogenic microflora into portal vein. Under the influence of the damaging action of hydrophobic bile acids and endotoxin on hepatocytes translocation of bile acids, endotoxin and pathogenic microflora into the blood, which activated the process of unlimited proteolysis and fibrinolysis was observed. Optic density of the blood plasma increased and protein breakdown in the urine with reduction of its concentration and excretion in this biological fluid occurred against a background of proteolytic activity of the blood plasma growth. At the same time not essential growth of optic density of the blood plasma only at wave lengths 250, 310, 320 and 340 nm, is explained by the additional influence of pathogenic microflora coming into the blood at infected bile peritonitis. In the mechanism of decompensation of kidneys the most significant pathogenic links were damages of the proximal portion of nephron at the expense of endotoxin action of gram-negative microflora to which the receptors in this portion of renal tubules were revealed [8. 15]. Besides, injuries of the proximal portion of nephron could be caused by the products of lipid peroxidation, thromboxane A2, factor of tumor-a necrosis, which are also produced under endotoxin influence [4].

Injuries of this part of nephron, containing a large quantity of lysosomes could be stipulated by excessive activation of proteolysis and detergent action of hydrophobic bile acids [11]. Derangements of energy metabolism of proximal and distal tubules were caused by the products with average molecular weight, which might damage mitochondria. The products of lipid peroxidation stimulated thromboxane A2 accumulation in renal cortical substance, which was the cause of the secondary damage of the distal part of nephron. Indices changes of sodium ion transport with the presence of growth of its concentration and excretion with urine, its clearance and concentrating index according to lowering of its distal transport are explained by the development of the "loss" syndrome of the given cation with urine and "concealed" damage of the proximal part of nephron [5], since reabsorption in the latter increased at the expense of processes of the passive transport under conditions of high filtrating nephron load against a background of derangement of active energy dependent mechanisms of reabsorption in this part of renal tubules. Reduction of standardized indices of excretion of titrating acids and ammonium are stipulated by additional damage of the renal tubules in case of infected bile peritonitis.

Conclusion

The clinical course of the infected experimental bile peritonitis after discrete autobile escape from the

common bile duct through the formed defect of its wall by means of thermocoagulation against a background of introduction of the small intestine contents is accompanied by an increase of optic density of the blood plasma at wave length 250, 310, 320, 340 nm and development of "loss" syndrome of sodium ions against a background of excretion decrease of titrating acids and ammonium with urine.

Perspectives of further investigations

Perspectives of further investigations as to elucidation of the role of new mechanisms of the damage of inner organs under conditions of the development of polyorganic insufficiency syndrome in case of the suggested model of the infected experimental bile peritonitis is substantiated.

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ВПЛИВ ІНФІКОВАНОГО ЕКСПЕРИМЕНТАЛЬНОГО ЖОВЧНОГО ПЕРИТОНІТУ НА ОПТИЧНУ ГУСТИНУ ПЛАЗМИ КРОВІ ТА ФУНКЦІОНАЛЬНИЙ СТАН НИРОК

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Резюме. У дослідах на 76 білих нелінійних щурах - самцях показано, що перебіг інфікованого експериментального жовчного перитоніту через 24 год після дискретного надходження автожовчі зі спільної жовчної протоки щура через сформований дефект її стінки шляхом термокоагуляції та введення вмісту тонкого кишечнику супроводжується зростанням оптичної густини плазми крові за довжин хвиль 250, 310, 320, 340 нм із розвитком синдрому "втрати" іонів натрію на тлі зниження екскреції кислот, що титруються та аміаку з сечею.

Ключові слова: оптична густина плазми крові, інфікований жовчний перитоніт, нирки, синдром "втрати" іонів натрію, екскреція аміаку та кислот, що титруються.

ВЛИЯНИЕ ИНФИЦИРОВАННОГО ЭКСПЕРИМЕНТАЛЬНОГО ЖЕЛЧНОГО ПЕРИТОНИТА НА ОПТИЧЕСКУЮ ПЛОТНОСТЬ ПЛАЗМЫ КРОВИ И ФУНКЦИОНАЛЬНОЕ СОСТОЯНИЕ ПОЧЕК

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Резюме. В опытах на 76 белых нелинейных крысах - самцах показано, что течение инфицированного экспериментального желчного перитонита через 24 часа после дискретного поступления автожёлчи с общей желчной протоки крысы через сформированный дефект ее стенки путем термокоагуляции и введения содержимого тонкого кишечника сопровождается увеличением оптической плотности плазмы крови при длинне волны 250, 310, 320, 340 нм с развитием синдрома "потери" ионов натрия на фоне снижения экскреции титруемых кислот и аммиака с мочой.

Ключевые слова: оптическая плотность плазмы крови, инфицированный желчный перитонит, почки, синдром "потери" ионов натрия, экскреция титруемых кислот и аммиака.

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