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PECULIARITIES OF THE FORMATION OF POLYORGANIC DYSFUNCTION SYNDROME IN EXPERIMENTAL ABDOMINAL SEPSIS: KIDNEY DYSFUNCTION

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Conflict of interest

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Abstract

Introduction: A steady increase in the incidence of the abdominal sepsis puts it into the top priority in terms of both medical and socio-economic significance. Multiple organ failure/dysfunction syndrome is one of the most important components of the pathogenesis of abdominal sepsis, determining the severity of the course, features of the clinical picture, mortality and outcome.

The aim of the study is to determine the dynamics of changes in the functional state of the excretory system under the conditions of modeling of the AS in the acute experiment.

Material and methods: The study included acute experiment on 38 white rats with abdominal sepsis modeled by intraabdominal injection of a patented mixture of both aerobic and anaerobic conditionally pathogenic microorganisms' suspension and adjuvant substances influencing local host resistance. This study conforms to international and national standards of bioethics. Kidneys' functional state was evaluated by determining a set of 46 functional markers.

Results: All renal function parameters were significantly influenced by abdominal sepsis formation. At the beginning of the formation of the abdominal sepsis, the disruption of the transport of sodium ions is concentrated mainly in the area of the distal tubules. Subsequently, the zone of functional disorders also covers the proximal nephron.

Conclusions: Formation of abdominal sepsis is characterized by a complex of physiological changes in the functional state of the kidneys, both adaptive- compensatory and pathological. Renal function undergoes phase changes under abdominal sepsis with predominantly adaptive and compensatory mechanisms dominating at the beginning with further involvement of kidneys' components and formation of renal dysfunction.

Keywords: **kidney, physiology, pathology, abdominal sepsis.**

Introduction

One of the most important types of sepsis is [1, 2] abdominal sepsis (AS). Its development is characterized by the formation of multiple organ failure/dysfunction

syndrome (MODS), which often leads to adverse effects for the patient. MODS is one of the most important components of the pathogenesis of abdominal sepsis, determining the severity of the course, features of the clinical picture and mortality in AS [4, 7]. An important role in the pathogenesis of AS is occupied by the acute renal dysfunction, which is caused by common etiological factors, and the main pathogenetic mechanisms [3, 5]. Moreover, the dynamics of an acute renal dysfunction in AS remain unclear [6, 8].

The aim of the study is to determine the dynamics of changes in the functional state of the excretory system under the conditions of modeling of the AS in the acute experiment.

Material and methods

Taking into account the methodological features and tasks of the study, the work was performed in the experimental conditions (38 adolescent white rats of both genders) with AS modeled according to our own technique by intraabdominal injection of a patented mixture of both aerobic and anaerobic conditionally pathogenic microorganisms' suspension and adjuvant substances influencing local host resistance. This study was guided by generally accepted international and national standards of bioethics and approved by the Bioethics Commission of the Bukovinian State Medical University. The functional state of the kidneys in abdominal sepsis was evaluated by determining a set of markers.

Results

The results of the study are presented in the table divided into control (intact animals), and periods of 6, 24, 48, and 72 hours of AS modeling, accordingly. At the beginning of the formation of the AS, the disruption of the transport of sodium ions is concentrated mainly in the area of the distal tubules. Subsequently, the zone of functional disorders also covers the proximal nephron.

Conclusions. Formation of abdominal sepsis is characterized by a complex of physiological changes in the functional state of the kidneys, both adaptive- compensatory and pathological. Renal function undergoes phase changes under abdominal sepsis with predominantly adaptive and compensatory mechanisms dominating at the beginning with further involvement of kidneys' components and formation of renal dysfunction.

Table 1. Dynamics of changes parameters of the renal functional status under abdominal sepsis (n=38, M±m)

Parameter	**Duration of the disease**				
	Control	6 hours	48 hours	48 hours	72 hours
1	2	3	4	5	6
Diuresis, ml/2g·100g	4,43±0,116	1,02±0,119 P<0,05	3,61±0,208 P_1<0,05	3,38±0,37 P<0,05 P_1<0,05	3,27±0,19 P<0,05 P_1<0,05 P_2<0,05
Conditional diuresis, %	88,78±2,33	20,30±2,38 P<0,05	72,22±4,17 P_1<0,05	67,66±7,44 P<0,05 P_1<0,05	65,44±3,86 P<0,05 P_1<0,05 P_2<0,05
Urine Na+ mmol/l	1,41±0,39	13,36±0,51 P<0,05	0,84±0,12 P<0,05 P_1<0,05	0,82±0,055 P<0,05 P_1<0,05	1,58±0,20 P_1<0,05 P_2<0,05 P_3<0,05
Na+ excretion, mkmol/2g	6,14±1,69	13,35±1,36 P<0,05	2,92±0,34 P<0,05 P_1<0,05	2,71±0,33 P<0,05 P_1<0,05	4,99±0,49 P_1<0,05 P_2<0,05
Urine K+ mmol/l	2,45±1,41	14,57±0,65 P<0,05	7,61±0,92 P<0,05 P_1<0,05	9,07±0,43 P<0,05 P_1<0,05	15,45±1,34 P<0,05 P_2<0,05
K+ excretion, mkmol/2g	11,14±6,42	14,84±1,97	26,65±2,55 P<0,05 P_1<0,05	32,84±3,96 P<0,05 P_1<0,05	51,23±6,54 P<0,05 P_1<0,05 P_2<0,05
Urea creatinine. mmol/l	2,16±0,30	2,13±0,21	0,90±0,057 P<0,05 P_1<0,05	0,96±0,045 P<0,05 P_1<0,05	1,03±0,084 P<0,05 P_1<0,05
Plasma creatinine, mmol/l	55,67±6,40	132,29±4,36 P<0,05	105,33±1,93 P<0,05 P_1<0,05	104,50±1,78 P<0,05 P_1<0,05	105,0±1,93 P<0,05 P_1<0,05
Glomerular filtration mkl/min·100g	1436,70± 73,38	141,84± 26,96 P<0,05	254,57± 17,18 P<0,05 P_1<0,05	258,92± 29,24 P<0,05 P_1<0,05	260,964± 18,92 P<0,05 P_1<0,05
H_2O conditional reabsorption,%	97,38±0,14	93,42±0,69	87,80±0,96 P<0,05 P_1<0,05	88,86±0,62 P<0,05	89,15±0,87 P<0,05
Plasma Na+ mmol/l	135,00±2,94	151,79±1,19	146,67±1,95	150,50±1,52	154,50±1,78 P<0,05
Na+ filtration fraction mkmol/min	194,14±11,58	21,65±4,22 P<0,05	37,24±2,48 P<0,05 P1<0,05	39,22±4,45 P<0,05 P1<0,05	40,13±2,66 P<0,05 P1<0,05
Na+ excretory fraction mkmol/min	0,051±0,001	0,11±0,011 P<0,05	0,024±0,003 P<0,05 P1<0,05	0,023±0,003 P<0,05 P1<0,05	0,042±0,004 P1<0,05 P2<0,05
Na+ reabsorption fraction mkmol/min	194,09± 11,59	21,54±4,21 P<0,05	37,22±2,48 P<0,05 P_1<0,05	39,20±4,45 P<0,05 P_1<0,05	40,09±2,66 P<0,05 P_1<0,05

1	2	3	4	5	6
Na+ reabsorption,%	99,97±0,003	99,41±0,08	99,93±0,006	99,94±0,009	99,89±0,01
Urine creatinine/ Plasma creatinine	38,93±2,01	16,25±1,70 P<0,05	8,60±0,68 P<0,05 P_1<0,05	9,24±0,52 P<0,05 P_1<0,05	9,77±0,786 P<0,05 P_1<0,05
Urine Na+/Plasma Na+	0,01± 0,0029	0,088±0,0036 P<0,05	0,006± 0,0009 P_1<0,05	0,005±0,0004 P<0,05 P_1<0,05	0,01±0,001 P_1<0,05 P_2<0,05
Urine Na+/ Urine K+	3,26±1,424	0,93±0,053 P<0,05	0,11±0,015 P<0,05 P_1<0,05	0,085±0,004 P<0,05 P_1<0,05	0,11±0,014 P<0,05 P_1<0,05
Na+ clearance, ml/2g	0,046±0,013	0,088±0,008 P<0,05	0,02±0,003 P<0,05 P_1<0,05	0,028±0,002 P<0,05 P_1<0,05	0,032±0,004 P<0,05 P_1<0,05 P_2<0,05
Na+ distal transport mkmol/2g	592,13± 15,45	141,31±17,78 P<0,05	527,61± 33,15 P_1<0,05	509,56±56,17 P<0,05 P_1<0,05	501,31±31,74 P<0,05 P_1<0,05
Na+ proximal reabsorption mkmol/2g	22,70±1,38	2,44±0,49 P<0,05	3,94±0,29 P<0,05 P_1<0,05	4,19±0,48 P<0,05 P_1<0,05	4,31±0,309 P<0,05 P_1<0,05
H_2O Na+ clearance ml/2g	4,39±0,12	0,93±0,11 P<0,05	3,59±0,21 P<0,05 P_1<0,05	3,37±0,37 P<0,05 P_1<0,05	3,24±0,193 P<0,05 P_1<0,05
Urine protein, %	0,051± 0,0004	0,27±0,008 P<0,05	0,13±0,014 P<0,05 P_1<0,05	0,075±0,006 P<0,05 P_1<0,05 P_2<0,05	0,06±0,006 P_1<0,05 P_2<0,05
Protein excretion mg/2g	0,23±0,019	0,27±0,03	0,44±0,039 P<0,05 P1<0,05	0,26±0,037 P2<0,05	0,21±0,02 P2<0,05
Titred acids excretion, mkmol/2g	29,80±7,91	85,92±5,34 P<0,05	25,52±1,37	33,35±4,01	24,59±1,74
NH3 excretion, mkmol/2g	80,25±10,93	104,68±7,16 P<0,05	67,42±3,27 P<0,05	77,54±8,58	54,27±2,86 P<0,05
NH3 excretion/ Titred acids excretion	3,32±0,61	1,22±0,009 P<0,05	2,67±0,12	2,34±0,01 P<0,05	2,25±0,11 P<0,05
Na+ excretion /100 mkl glomerular filtration	0,46±0,15	10,69±1,38 P<0,05	1,21±0,18 P<0,05 P1<0,05	1,10±0,10 P<0,05 P1<0,05	1,97±0,20 P<0,05 P1<0,05 P2<0,05
Protein excretion /100 mkl glomerular filtration	0,016±0,001	0,21±0,027 P<0,05	0,18±0,013 P<0,05	0,10±0,008 P<0,05 P_1<0,05 P_2<0,05	0,08±0,005 P<0,05 P_1<0,05 P_2<0,05

1	2	3	4	5	6
Urine pH	6,21±0,02	5,46±0,09 P<0,05	7,47±0,15 P<0,05 P_1<0,05	6,91±0,05 P_1<0,05	6,78±0,09 P_1<0,05
CH+ mkmol/l	0,62±0,025	3,94±0,66 P<0,05	0,053±0,018 P<0,05 P_1<0,05	0,13±0,016 P<0,05 P_1<0,05	0,20±0,04 P<0,05 P_1<0,05 P_2<0,05
H+ excretion nmol/2 hrs	2,51±0,09	3,72±0,58 P<0,05	0,18±0,05 P<0,05 P_1<0,05	0,41±0,068 P<0,05 P_1<0,05	0,60±0,095 P<0,05 P_1<0,05 P_2<0,05
H + excretion /100 mkl glomerular filtration	0,15±0,02	3,19±0,75 P<0,05	0,079±0,023 P<0,05 P_1<0,05	0,17±0,023 P_1<0,05	0,24±0,038 P<0,05 P_1<0,05 P_2<0,05
Titred acids excretion /100 mkl glomerular filtration	5,72±0,71	69,85±8,97 P<0,05	10,37±0,76 P<0,05 P_1<0,05	13,61±1,10 P<0,05 P_1<0,05	9,72±0,82 P<0,05 P_1<0,05
NH_3 excretion /100 mkl glomerular filtration	25,58±1,71	84,58±10,45 P<0,05	27,76±2,56 P_1<0,05	31,19±1,82 P<0,05 P_1<0,05	21,49±1,46 P_1<0,05 P_3<0,05
Na+ proximal reabsorption /100 mkl glomerular filtration	10,41±0,32	14,18±0,19 P<0,05	12,87±0,14	13,38±0,18 P<0,05	13,76±0,1 P<0,05
Na+ distal transport /100 mkl glomerular filtration	1,28±0,06	0,91±0,10 P<0,05	1,79±0,16 P<0,05 P_1<0,05	1,67±0,09 P<0,05 P_1<0,05	1,67±0,15 P1<0,05

P – probability of differences compared to the data of the 6-hour period (control);

P_1–probability of differences compared to the data of the 6-hour observation period at the same group;

P_2 – probability of differences compared to the data of the 24-hour observation period.

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