Braz. J. vet. Res. anim. Sci.,
São Paulo, v. 37, n. 2, p. 146-152, 2000.

Experimental peritonitis in horses. Hematological and biochemistry aspects *

Peritonite experimental em eqüinos. Aspectos hematológicos e bioquímicos

Luiz Cláudio Nogueira MENDES[1]; Luiz Carlos MARQUES[2]; Ruben Pablo SCHOCKEN-ITURRINO[3]; Fernando Antônio de ÁVILA[3]; Euclides Braga MALHEIROS[4]

CORRESPONDENCE TO:
Luiz Carlos Marques
Departamento de Clínica e Cirurgia Veterinária
Faculdade de Ciências Agrárias e Veterinárias da UNESP, Campus de Jaboticabal
Via de Acesso Prof. Paulo Donato Castellane, s/n
14884-900 – Jaboticabal – SP
e-mail: lmarques@fcav.unesp.br

1-Departamento de Clínica, Cirurgia e Reprodução Animal do Curso de Medicina Veterinária da UNESP, Araçatuba – SP
2-Departamento de Clínica e Cirurgia Veterinária da Faculdade de Ciências Agrárias e Veterinárias da UNESP, Jaboticabal – SP
3-Departamento de Microbiologia da Faculdade de Ciências Agrárias e Veterinárias da UNESP, Jaboticabal – SP
4-Departamento de Ciências Exatas da Faculdade de Ciências Agrárias e Veterinárias da UNESP, Jaboticabal – SP

SUMMARY

Sixteen adult horses were randomly divided into 4 equal groups (GI, GII, GIII and GIV) of 4 animals and each group was injected intraperitoneally with one of the following suspension: Group I, 100 x 10^7 colony-forming units (CFU) of *E. coli* diluted in 500 ml of 0.9% saline; Group II, 100 x 10^7 CFU of *Bacteroides fragilis* in 500 ml of 0.9% saline; Group III, 100 x 10^7 CFU of *E. coli* in combination with 100 x 10^7 CFU of *B. fragilis* in 500 ml of 0.9% saline; Group IV, 500 ml of 0.9% saline. Leukopenia appeared in all animals inoculated with bacteria within the first six hours of the experiment. After this period, leukocytosis was observed in some inoculated horses. Horses inoculated with pure cultures of either *E. coli* or *B. fragilis* demonstrated mild and self-limiting peritonitis, whereas those inoculated with a combination of both bacteria demonstrated laboratory findings of higher intensity and duration.

UNITERMS: Peritonitis; Horses; *Escherichia coli*; *Bacteroides fragilis*.

INTRODUCTION

The peritoneum is composed of a single layer of mesothelial squamous cells overlying loose areolar connective tissue and adipose tissue[10]. Inflammation of the peritoneum of the horse may occur as a primary condition or, more commonly, as a secondary complication and may be associated with either infectious or non-infectious disease[17] and is characterised by exudation of serum fibrin, and protein into the peritoneal cavity[20].

Peritoneal injury triggers many physiological responses that are intended to control or eliminate the peritoneal contaminant. Although these responses are beneficial initially to the patient, they may progress to the point where they have deleterious effects[11].

Clinical signs of peritonitis depend on the primary disease process, infectious agents involved and extend of disease, most often they are nonspecific but suggestive of gastrointestinal dysfunction like colic, ileus, pyrexia, anorexia, weight loss, and diarrhea[20]. Bacteria most commonly isolated from the peritoneal cavity after large bowel contamination include *Escherichia coli* and other enterobacteria, *Streptococcus spp, Proteus spp, Bacteroides spp* (particularly *B. fragilis*), and *Clostridium spp*[1,3,11]. The association between *E. coli* and *B. fragilis* has synergistic effects and this synergism with involvement of obligate anaerobes and facultative organisms enhances the pathogenicity of some bacteria that are relatively non-pathogenic in normal circunstances[16].

Leukocytosis with neutrophilia, leukopenia with neutropenia, increased plasma fibrinogen and alkaline phosphatase activity were observed in horses with naturally acquired peritonitis[5,6,14]. Protein sequestration and fluid exudation into the peritoneal cavity lead to hypoproteinemia and dehydration[20]. Other laboratory findings included lymphocytosis, monocytosis and a high gamma glutamyl transferase activities[4].

There have been few detailed studies of peritonitis in adult horses[14] and the experimental models used in

* Supported by FAPESP.

peritonitis studies in horses are based on surgical procedures with the objective of investigating the adhesions formation, treatments[1,2,13], and changes in the peritoneal fluid[22].

A clear understanding of the host responses to peritoneal injury is essential in determining the evolution, prognosis and treatment of peritonitis. The objective of the present investigation was to study the haematological and biochemistry aspects of horses to a bacterial offense to the peritoneum.

MATERIAL AND METHOD

Sixteen healthy horses (twelve male and four female) of various breeds, ranging from 3 to 10 years were used. The horses were randomized in four groups (GI, GII, GIII and GIV) of four animals each. During the study the horses were housed in individual stalls, fed commercial ration (3 kg/animal/day), coast-cross (*Cynodon dactilon L*) hay and water *ad libitum*.

Bacteroides fragilis was isolated from a human patient with peritonitis in the Department of Microbiology, University Hospital, Faculty of Medicine of Ribeirão Preto – São Paulo University, and was cultivated at the Anaerobic Laboratory of the Microbiology Department, Faculty of Agronomic and Veterinary Sciences, Campus of Jaboticabal, São Paulo State University, using Jang; Hirsh method[12]. *Escherichia coli* was isolated from a sample of feces from a healthy horse at the Microbiology Department, at the same university, using Edwards; Ewing method[7]. The inoculum was standardized as 10×10^7 colony-forming units (CFU) per millilitre.

For the inoculations, paracentesis was performed according to the technique described by White II[24], and the animals were inoculated intraperitoneally as described in Tab.1.

Blood samples were collected from the jugular vein of all animals at intervals of 0, 2, 4, 6, 8, 10, 12, 24, 36, 48, 60, 72, 120, 168 and 216 hours after inoculations (HAI). Samples for hemogram were collected in tubes containing ethylenediaminetetraacetic acid (EDTA) as anticoagulant. Blood for serum used in other biochemical analyses was collected into tubes containing no anticoagulant. Blood counts, plasma fibrinogen, total plasma protein, creatinine, blood urea nitrogen, gamma glutamyl transferase, alkaline phosphatase, bilirubin and aspartate aminotransferase were determined.

Red and white cell counts and hemoglobin levels were determined using a cell counter[a] annexed to a haemoglobinemeter[b]. Total plasma protein concentrations in the plasma were determined using the biuret method[c], and plasma fibrinogen was measured by refratrometry[19]. Analysis of blood urea nitrogen, creatinine, bilirubin, gamma glutamyl transferase, alkaline phosphatase and aspartate aminotransferase was performed by modified diacetyl, Lustosa-Basques, Sims-Horn, modified Szasz, modified Roy and Reitman-Frankel methods, respectively, using colorimetric kits[d].

Data were analyzed using a randomized design. Tukey test was used to compare data obtained from the uninfected control group and infected groups at each observation time. Results were considered to be significant at the $p < 0.05$ level.

RESULTS

A significant difference ($p < 0.05$; $p < 0.01$) in the red cell counts between infected and uninfected control horses occurred at times 4, 6, 8 and 10 HAI in GI, 6, 24 and 60 HAI in GII and 8 and 10 HAI in GIII (Tab. 2). A significant difference ($p < 0.05$; $p < 0.01$) in the packed cell volume between infected and uninfected control horses occurred at times 12 and 168 HAI in GI, 12 HAI in GII and 6, 8, 10, 12, 24 and 72 HAI in GIII (Tab. 3). A significant difference ($p < 0.05$; $p < 0.01$) in the hemoglobin values between infected and uninfected control horses occurred at times 6 and 12 HAI in GII and 6, 8, 10, 12 and 72 HAI in GIII (Tab. 4). A significant difference ($p < 0.05$; $p < 0.01$) in the total leukocytes counts between infected and uninfected control horses occurred at times 2, 12, 24, 36 and 48 HAI in GI, 4, 24, 36 and 48 HAI in GII and 2, 4 and 6 HAI in GIII (Tab. 5). A significant difference ($p < 0.05$; $p < 0.01$) in the neutrophils counts between infected and uninfected control horses occurred at times 2, 24 and 72 HAI in GI and 24 HAI in GIII (Tab. 6). A significant difference ($p < 0.05$; $p < 0.01$) in the lymphocytes counts between infected and uninfected control horses occurred at times 2 and 72 HAI in GI and 4 HAI in GIII (Tab. 7). A significant difference ($p <$

Table 1
Treatment of horses by intraperitoneal injection. Jaboticabal-SP, 1998.

Group	Inoculum/dose
I	*E. coli* (100×10^7 CFU) +500 ml of 0.9% saline
II	*B. fragilis* (100×10^7 CFU) + 500 ml of 0.9% saline
III	*E. coli* (100×10^7 CFU) + *B. fragilis* (100×10^7 CFU) + 500 ml of 0.9% saline
IV	500 ml of 0.9% saline

CFU = Colony-forming units

[a] CC-510-Celm, Barueri, SP, Brazil.
[b] HB-520-Celm, Barueri, SP, Brazil.
[c] Protein Kit - Lab test, SP, Brazil.
[d] Lab test, SP, Brazil.

Table 2

Mean erythrocytes count and T values, four groups of horses studied. Jaboticabal – SP, 1998.

	Erythrocytes (N x 10^3)				T values		
HAI	Mean GI	Mean GII	Mean GIII	Mean GIV	GI X GIV	GII X GIV	GIII X GIV
0	5752.5	6897.5	5050.0	5387.5	0.4318^{NS}	0.0591^{NS}	0.4011^{NS}
2	5542.5	5826.2	6215.0	5067.0	0.3303^{NS}	0.1236^{NS}	0.2493^{NS}
4	6155.0	6422.5	5962.5	4692.5	0.0414^{*}	0.0924^{NS}	0.1545^{NS}
6	5827.5	6815.0	7590.0	4562.5	0.0358^{*}	0.0052^{**}	0.0523^{NS}
8	6162.5	7142.5	6235.0	4992.5	0.0420^{*}	0.1172^{NS}	0.0264^{*}
10	6035.0	5990.0	6805.0	4780.0	0.0104^{*}	0.1487^{NS}	0.0021^{**}
12	5900.0	5930.0	6525.0	5037.5	0.2588^{NS}	0.2693^{NS}	0.0868^{NS}
24	5247.5	5962.5	5665.0	4602.5	0.2616^{NS}	0.0241^{*}	0.0946^{NS}
36	5372.5	5687.5	5055.0	4800.0	0.1965^{NS}	0.0771^{NS}	0.6972^{NS}
48	4935.0	5637.5	5125.0	4625.0	0.5990^{NS}	0.0889^{NS}	0.4110^{NS}
60	4450.0	6335.0	5505.0	4520.0	0.9023^{NS}	0.0260^{*}	0.1000^{NS}
72	5987.5	5087.5	5356.6	4732.5	0.4797^{NS}	0.8138^{NS}	0.4913^{NS}
120	4775.0	5230.0	4936.6	4492.5	0.4061^{NS}	0.2382^{NS}	0.1868^{NS}
168	4252.5	4900.0	4603.3	4570.0	0.2142^{NS}	0.4494^{NS}	0.9528^{NS}
216	4570.0	4545.0	4826.6	4730.0	0.8402^{NS}	0.7861^{NS}	0.5821^{NS}

NS = Non-significant; * $p < 0.05$; ** $p < 0.01$.

Table 3

Mean packed cell volume and T values, four groups of horses studied. Jaboticabal – SP, 1998.

	Packed cell volume (%)				T values		
HAI	Mean GI	Mean GII	Mean GIII	Mean GIV	GI X GIV	GII X GIV	GIII X GIV
0	29.50	40.25	30.25	29.00	0.8230^{ns}	0.1557^{ns}	0.5986^{ns}
2	27.25	30.50	31.50	27.75	0.7215^{ns}	0.3514^{ns}	0.1548^{ns}
4	29.75	31.25	31.75	27.50	0.3522^{ns}	0.3492^{ns}	0.0629^{ns}
6	28.00	36.25	34.50	27.00	0.6121^{ns}	0.1063^{ns}	0.0090^{**}
8	30.50	33.00	36.50	27.25	0.2795^{ns}	0.0918^{ns}	0.0019^{**}
10	29.25	31.00	38.00	26.25	0.1977^{ns}	0.2960^{ns}	0.0028^{**}
12	29.00	32.75	38.00	24.75	0.0293^{*}	0.0168^{*}	0.0002^{**}
24	26.75	32.00	33.00	25.25	0.4400^{ns}	0.0699^{ns}	0.0044^{**}
36	25.00	29.50	29.25	26.75	0.3559^{ns}	0.2709^{ns}	0.1963^{ns}
48	23.75	28.75	29.00	25.00	0.6297^{ns}	0.2431^{ns}	0.1630^{ns}
60	23.75	31.50	28.75	23.75	1.0000^{ns}	0.0878^{ns}	0.1331^{ns}
72	24.00	26.75	29.33	23.50	0.8005^{ns}	0.3026^{ns}	0.0421^{*}
120	25.50	30.00	27.33	25.25	0.8648^{ns}	0.1383^{ns}	0.2446^{ns}
168	23.00	29.75	26.66	26.25	0.0483^{*}	0.3171^{ns}	0.8804^{ns}
216	24.75	31.00	27.66	25.75	0.6182^{ns}	0.1655^{ns}	0.2588^{ns}

NS = Non-significant; *$p < 0.05$; **$p < 0.01$.

Table 4

Mean hemoglobin and T values, four groups of horses studied. Jaboticabal – SP, 1998.

Parâmetro	Subprodutos								FVF
	FS1	SIT	FA	FCO1	FCO2	FS2	FP	TB	
a	37,80	33,11	23,56	22,53	21,20	42,42	35,60	14,79	40,74
b	62,15	71,16	52,09	24,59	27,64	59,53	50,59	84,29	74,67
c	0,077	0,055	0,062	0,412	0,709	0,053	0,017	0,019	0,017
P	99,95	104,27	75,65	47,12	48,84	101,95	86,19	99,08	115,41
p2	87,13	85,29	62,94	45,98	48,08	85,64	58,84	55,85	75,04
p5	75,48	70,38	52,39	44,45	47,01	73,05	48,43	38,00	59,68
p8	68,28	62,10	46,30	43,12	46,03	66,14	44,46	30,96	53,82

NS = Non-significant; * $p < 0.05$; ** $p < 0.01$.

0.05; $p < 0.01$) in the plasma fibrinogen between infected and uninfected controls horses occurred at time 72 HAI in GI, 12, 48 and 216 HAI in GII and 48 and 72 HAI in GIII (Tab. 8). A significant difference ($p < 0.05$; $p < 0.01$) in blood urea nitrogen between infected and uninfected control horses occurred at times 6, 8 and 10 HAI in GI, 6, 10, 12, 24, 48 and 60 HAI in GIII (Tab. 9).

Analysis of aspartate aminotransferase, gamma glutamyl transferase, alkaline phosphatase, bilirubin, creatinine and total serum protein did not show significant differences.

Table 5

Mean leukocytes count and T values, four groups of horses studied. Jaboticabal – SP, 1998.

	Hemoglobin (g%)				T values		
HAI	Mean GI	Mean GII	Mean GIII	Mean GIV	GI X GIV	GII X GIV	GIII X GIV
0	10.37	13.77	11.02	9.87	0.6680^{ns}	0.0664^{ns}	0.2945^{ns}
2	10.32	11.82	12.27	9.72	0.6363^{ns}	0.1985^{ns}	0.1310^{ns}
4	10.37	12.72	11.47	9.40	0.5019^{ns}	0.1768^{ns}	0.1327^{ns}
6	9.85	12.50	12.67	9.27	0.6322^{ns}	0.0232*	0.0203*
8	10.67	12.70	13.22	9.62	0.4267^{ns}	0.0571^{ns}	0.0204*
10	11.07	11.97	14.10	9.10	0.2231^{ns}	0.1359^{ns}	0.0132*
12	10.67	12.37	13.35	8.80	0.1579^{ns}	0.0461*	0.0082**
24	9.55	11.85	11.80	9.15	0.7665^{ns}	0.1325^{ns}	0.0763^{ns}
36	8.95	11.32	11.05	9.25	0.8011^{ns}	0.1929^{ns}	0.1652^{ns}
48	8.57	10.50	11.02	8.92	0.7998^{ns}	0.3761^{ns}	0.1693^{ns}
60	8.65	11.47	10.97	8.40	0.8211^{ns}	0.0870^{ns}	0.0671^{ns}
72	9.40	10.52	11.06	8.50	0.3941^{ns}	0.0692^{ns}	0.0420*
120	9.50	11.50	9.93	8.67	0.4168^{ns}	0.0892^{ns}	0.3457^{ns}
168	9.32	10.70	10.26	9.35	0.9816^{ns}	0.2707^{ns}	0.4080^{ns}
216	8.35	11.27	10.56	8.90	0.6766^{ns}	0.0557^{ns}	0.0863^{ns}

NS = Non-significant; * $p < 0.05$; ** $p < 0.01$.

Table 6

Mean neutrophils count and T values, four groups of horses studied. Jaboticabal – SP, 1998.

	Leukocytes/mm^3				T values		
HAI	Mean GI	Mean GII	Mean GIII	Mean GIV	GI X GIV	GII X GIV	GIII X GIV
0	14775	9000	11425	9925	0.0545^{ns}	0.3844^{ns}	0.4609^{ns}
2	5600	7550	5425	9875	0.0043**	0.1290^{ns}	0.0162*
4	6925	5525	5475	10725	0.5466^{ns}	0.0080**	0.0328*
6	10700	8725	5925	11100	0.8477^{ns}	0.1232^{ns}	0.0269*
8	12850	10600	7900	11575	0.6013^{ns}	0.5655^{ns}	0.1637^{ns}
10	16550	13175	8775	12950	0.2020^{ns}	0.7931^{ns}	0.1044^{ns}
12	18550	12450	10150	11075	0.0496*	0.3219^{ns}	0.6469^{ns}
24	16600	13050	11575	10900	0.0223*	0.0223*	0.7568^{ns}
36	16950	11550	12775	11000	0.0194*	0.0194*	0.5200^{ns}
48	16025	12775	8200	8975	0.0129*	0.0129*	0.6986^{ns}
60	15725	13050	9275	10775	0.0938^{ns}	0.0938^{ns}	0.6569^{ns}
72	11825	11100	11300	11475	0.8863^{ns}	0.8863^{ns}	0.9420^{ns}
120	17175	8750	14000	10975	0.0747^{ns}	0.0747^{ns}	0.2044^{ns}
168	18850	11425	17600	12375	0.0663^{ns}	0.0663^{ns}	0.2727^{ns}
216	17700	10625	20200	11700	0.0573^{ns}	0.6712^{ns}	0.1448^{ns}

NS = Non-significant; * $p < 0.05$; ** $p < 0.01$

Table 7

Mean lymphocytes count and T values, four groups of horses studied. Jaboticabal – SP, 1998.

	Lymphocytes (%)				T values		
HAI	Mean GI	Mean GII	Mean GIII	Mean GIV	GI X GIV	GII X GIV	GIII X GIV
0	39.75	55.25	43.25	41.25	0.8771^{ns}	0.1057^{ns}	0.8015^{ns}
2	66.00	45.00	69.00	37.25	0.0026**	0.3832^{ns}	0.0564^{ns}
4	43.25	42.50	65.50	35.50	0.3562^{ns}	0.4064^{ns}	0.0275*
6	21.75	28.75	34.75	33.00	0.1068^{ns}	0.5831^{ns}	0.8544^{ns}
8	23.00	25.25	36.50	26.75	0.6196^{ns}	0.8605^{ns}	0.2702^{ns}
10	27.75	30.75	36.00	33.25	0.5318^{ns}	0.7630^{ns}	0.4782^{ns}
12	20.25	30.75	35.75	27.75	0.4619^{ns}	0.7121^{ns}	0.2490^{ns}
24	23.75	36.50	21.25	40.25	0.0527^{ns}	0.5859^{ns}	0.0711^{ns}
36	34.25	31.25	31.00	37.50	0.6619^{ns}	0.4480^{ns}	0.5033^{ns}
48	35.50	39.50	49.25	45.25	0.3415^{ns}	0.5770^{ns}	0.7081^{ns}
60	40.00	33.75	40.75	34.25	0.5358^{ns}	0.9440^{ns}	0.5993^{ns}
72	60.00	43.00	31.67	31.25	0.0049**	0.1211^{ns}	0.9609^{ns}
120	46.25	53.00	23.67	39.75	0.5833^{ns}	0.2091^{ns}	0.1666^{ns}
168	34.75	38.00	25.00	33.00	0.8851^{ns}	0.6912^{ns}	0.5614^{ns}
216	35.75	48.25	26.00	43.25	0.4887^{ns}	0.5281^{ns}	0.1491^{ns}

NS = Non-significant; * $p < 0.05$; ** $p < 0.01$.

Table 8

Mean fibrinogen and T values, four groups of horses studied. Jaboticabal – SP, 1998.

	Fibrinogen (g/dl)				T values		
HAI	Mean GI	Mean GII	Mean GIII	Mean GIV	GI X GIV	GII X GIV	GIII X GIV
0	0.58	0.51	0.54	0.55	0.5945^{ns}	0.6333^{ns}	0.8790^{ns}
2	0.54	0.54	0.54	0.58	0.5097^{ns}	0.6506^{ns}	0.5097^{ns}
4	0.50	0.60	0.54	0.52	0.2070^{ns}	0.4961^{ns}	0.6704^{ns}
6	0.51	0.55	0.61	0.60	0.2040^{ns}	0.5097^{ns}	0.6704^{ns}
8	0.57	0.42	0.61	0.57	1.0000^{ns}	0.0667^{ns}	0.5891^{ns}
10	0.54	0.42	0.60	0.61	0.3436^{ns}	0.1129^{ns}	0.7908^{ns}
12	0.60	0.44	0.63	0.65	0.4880^{ns}	0.0127*	0.6202^{ns}
24	0.57	0.57	0.68	0.65	0.1682^{ns}	0.0591^{ns}	0.6479^{ns}
36	0.65	0.58	0.68	0.68	0.3559^{ns}	0.2110^{ns}	1.0000^{ns}
48	0.65	0.52	0.80	0.64	0.8439^{ns}	0.0254*	0.0047**
60	0.80	0.55	0.77	0.67	0.2782^{ns}	0.0656^{ns}	0.1002^{ns}
72	0.78	0.60	0.85	0.65	0.0311*	0.4197^{ns}	0.0059**
120	0.74	0.63	0.82	0.63	0.1135^{ns}	1.0000^{ns}	0.0734^{ns}
168	0.68	0.61	0.89	0.73	0.5891^{ns}	0.2764^{ns}	0.2515^{ns}
216	0.78	0.55	1.01	0.70	0.5986^{ns}	0.0195*	0.0905^{ns}

NS = Non-significant; * $p < 0.05$.

Table 9

Mean blood urea nitrogen and T values, four groups of horses studied. Jaboticabal – SP, 1998.

	Blood urea nitrogen (mg/dl)				T values		
HAI	Mean GI	Mean GII	Mean GIII	Mean GIV	GI X GIV	GII X GIV	GIII X GIV
0	27.84	28.10	28.75	19.52	0.0821^{ns}	0.0799^{ns}	0.0563^{ns}
2	25.91	27.11	28.57	20.42	0.2845^{ns}	0.0594^{ns}	0.1024^{ns}
4	25.38	25.84	30.30	20.55	0.2128^{ns}	0.4541^{ns}	0.0851^{ns}
6	30.02	25.21	34.15	20.40	0.0296*	0.1533^{ns}	0.0171*
8	31.02	24.14	33.25	20.27	0.0391*	0.9298^{ns}	0.0527^{ns}
10	30.28	27.62	34.70	17.75	0.0394*	0.1292^{ns}	0.0192*
12	27.07	27.75	37.37	19.12	0.0759^{ns}	0.5216^{ns}	0.0046**
24	26.10	32.21	40.15	17.82	0.0633^{ns}	0.1203^{ns}	0.0062**
36	26.72	44.87	41.17	17.57	0.1650^{ns}	0.1992^{ns}	0.1657^{ns}
48	24.50	20.22	34.10	16.92	0.0816^{ns}	0.3002^{ns}	0.0356*
60	23.97	29.28	35.60	16.30	0.1108^{ns}	0.3726^{ns}	0.0164*
72	24.74	21.53	36.86	21.02	0.3883^{ns}	0.6009^{ns}	0.1438^{ns}
120	24.52	27.62	23.76	18.32	0.0736^{ns}	0.1274^{ns}	0.1048^{ns}
168	22.39	17.52	21.30	19.55	0.4568^{ns}	0.2943^{ns}	0.7036^{ns}
216	20.98	24.15	25.10	20.42	0.9215^{ns}	0.3401^{ns}	0.3499^{ns}

NS = Non-significant; * $p < 0.05$; ** $p < 0.01$.

DISCUSSION

Increases in red cell counts, packed cell volume and hemoglobin occurred at different times in all groups of inoculated horses. An elevated packed cell volume and polycythemia may be seen early in the disease process, reflecting the degree of dehydration present and splenic contraction[21,23]. In the horses of this experiment, diarrhea and fluid loss into the abdominal cavity were responsible for alterations of the water-electrolytes status.

Leukopenia appeared in all animals inoculated with bacteria within the first 6 hours of the experiment. After this period, leukocytosis was observed in some inoculated horses. In peracute or acute peritonitis, severe leukopenia with an absolute neutropenia and degenerative left shift are present because of the margination of neutrophils and migration of neutrophils into the peritoneal cavity[18]. The first host defense against infection was provided by neutrophils activated by chemotactic factors in the bloodstream and moved to the infected region, causing transient neutropenia. Neutrophilia then occurs as a consequence of cell production by bone marrow[18]. In addition, losses greater than production occur in acute inflammations of large surfaces such as the peritoneum when neutrophils migrate to limit injury[23]. Neutropenia occurs also during toxin absorption by the peritoneum[14].

Leukocytosis with neutrophilia has been reported to occur in natural peritonitis in horses[6,14,15]. Probably leukopenia is not detected in natural equine peritonitis because laboratory evaluation is performed several hours after the beginning of the inflammatory process in the peritoneum, but, experimentally, leukopenia was also observed, which was statistically significant between 12 and 72 hours after surgery, a longer period than the

observed in this experiment probably due to the surgical model used intending to study adhesions formation[1].

Fibrinogen is a plasma protein that, when transported to the extravascular space, plays an important role in organism defense and aids in finding the pathologic process. Plasma levels of fibrinogen are considered to be important for the evaluation of the inflammatory response. Variations in plasma fibrinogen levels occurred in all inoculated groups but different from those reported in literature[6,14], but increased plasma fibrinogen levels in equine peritonitis have also been demonstrated[1,4]. The present data stated that the evaluation of plasma fibrinogen was of little value for the prognosis of equine peritonitis, similar result was reported else where[9].

Significantly increased in blood urea nitrogen occurred in animals of group III. Any catabolic process results in alteration of these parameters, including the inflammatory process and fever being important causes of azotemia in horses[8]. Clinical and pathological alterations were more intense probably because the energetic and protein metabolism was more intense in animals of group III than in the other groups explaining the increased blood urea nitrogen.

Analysis of aspartate aminotransferase, gamma glutamyl transferase, alkaline phosphatase, and bilirubin did not showed significant alterations in all inoculated animals, but the pattern observed was not useful for the diagnosis or prognosis of horse peritonitis. However, an increase in alkaline phosphatase and gamma glutamyl transferase activities has been reported in horse peritonitis[4].

CONCLUSION

We conclude that the peritonitis in horses is a complex process, and the stage of the disease and the etiologic agent determined the alterations present in peripheral blood and in the blood chemistry parameters, which can be useful for diagnosis and/or prognosis of clinical cases of peritonitis.

RESUMO

Dezesseis eqüinos adultos foram distribuídos aleatoriamente em 4 grupos (GI, GII, GIII e GIV) constituídos por quatro animais, recebendo cada grupo o seguinte inóculo por via intraperitoneal: GI (100 X 10^7 unidades formadoras de colônia (UFC) de *Escherichia coli* diluídos em 500 ml de solução salina 0,9% estéril); GII (100 X 10^7 UFC de *Bacteroides fragilis* diluídos em 500 ml de solução salina 0,9% estéril); GIII (100 X 10^7 UFC de *Escherichia coli* associados a 100 X 10^7 UFC de *Bacteroides fragilis* diluídos em 500 ml de solução salina 0,9% estéril); GIV (testemunho - 500 ml de solução salina 0,9% estéril). Leucopenia ocorreu em todos os animais inoculados com bactérias, nas primeiras seis horas após as inoculações. Posteriormente a este período, verificou-se em alguns eqüinos inoculados leucocitose. Os eqüinos inoculados com culturas puras de *E. coli* ou *B. fragilis* apresentaram peritonites brandas e autolimitantes, enquanto os inoculados com a associação destas bactérias, apresentaram alterações laboratoriais de maior intensidade e duração.

UNITERMOS: Peritonite; Eqüinos; *Escherichia coli*; *Bacteroides fragilis*.

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Received: 23/07/1998
Accepted: 03/09/1999