BIOCHEMICAL AND CYTOLOGICAL ALTERATIONS OF PERITONEAL FLUID AFTER INTRAPERIOTENEAL INJECTION OF THIOGLYCOLATTE AND *ESHERICHIA COLI* IN RATS

Rania M.E.Bakr.[1], Farid A.S.[1], Abd-Allah O.A.[2] and Fararh K.M.[1*]

[1]Clinical Pathology Department, Faculty of Veterinary Medicine, Benha University, Egypt. [2]Clinical Pathology Department, Faculty of Veterinary Medicine, Suez Canal University, Egypt.

*correspondence: KHALED.FRARAH01@fvtm.bu.edu.eg

ABSTRACT

The differential diagnosis of peritoneal fluid alterations due to pathological conditions is a common clinical problem. However, the capability to distinguish Infectious from non-infectious causes of peritoneal fluid alterations using available biochemical techniques would obviate many expensive and time-consuming diagnostic studies on patients presenting with peritoneal fluid changes of unknown etiology. For demonstrating the effects of chemical and bacterial peritonitis on peritoneal fluid analysis, thioglycolatte medium (TG) and *Escherichia coli* (*E. coli*) were used for induction of chemical and bacterial peritonitis, respectively. 90 male rats were divided into 5 groups, each group contains 18 rats. One group used as control, two groups were injected with 3% TG (1or 2 ml/200 gm b.wt.) intraperitonealy and other two groups were injected with *E. coli* (0.5 or 1×10^8 CFU/mL) intraperitonealy. Collection of peritoneal fluid samples were done at 1 day, 1 week and 2 weeks after induction of peritonitis. Results of both models showed significant changes in peritoneal fluid analysis. Both modelsshowed significant increases in total protein concentration, specific gravity and cholesterol levels all over the experimental period. Chemical peritonitis model showed significant elevation in triglycerides level at 1 day after induction of peritonitis. While bacterial peritonitis model showed non-significant changes in triglycerides level at all times after induction of peritonitis. Regarding to cytological examination of peritoneal fluid, both modelsshowed significant increases intotal nucleated cell count (TNCC), red blood cells (RBCs), monocytes, neutrophils and mesothelial cell counts at all times after induction of peritonitis. Chemical peritonitis model showed significant increases mainly in mesothelial cells compared to bacterial peritonitis model. On the other hand, bacterial peritonitis model showed significant increases mainly in neutrophils compared to chemical peritonitis. Both models showed significant increases in lymphocytes count at 1 day after induction of peritonitis compared to the control group. Therefore, we concluded that peritoneal fluid could be used for differential diagnosis of peritonitis.

Keywords: peritoneal fluid, experimental peritonitis, thioglycolatte, *Esherichia coli*, peritoneal cytology, peritoneal chemistry.

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1. INTRODUCTION

Analysis of peritoneal fluid obtained by abdomenocentesis is a useful diagnostic and prognostic aid for assessing abdominal diseases [1-3]. Normal peritoneal fluid is an ultrafiltrate of plasma. It is formed by similar process that

involves the forces of Starling's law and anatomic structures. Generally, plasma filtrate leaves the capillaries and enters interstitial space then diffuses into the abdominal cavity, from which it is removed by lymphatic system and returned to plasma [4].It is clear to slightly turbid, pale yellow to straw colored and odorless.The peritoneal fluid plays an important role in allowing the abdominal organs to move freely; this movement is required for normal function of gastrointestinal and urinary tracts [5]. The most important causes of peritoneal fluid alterations includes septic and aseptic causes such as hepatic cirrhosis, tuberculosis peritonitis, spontaneous bacterial peritonitis, congestive heart failure and chronic indigestion.The differential diagnosis of peritoneal fluid is a common clinical problem [6]. Therefore, peritoneal fluid is important in the diagnosis of several disorders like liver cirrhosis, malignancy, pancreatitis, heart failure, hepatic venous obstruction, nephrotic syndrome, myxedema and peritonitis due to infection or other causes [7]. Also,peritoneal fluid analysis is helpful in diagnosing the cause of abdominal discomfort in animals with gastro-intestinal complications since peritoneal fluid composition reflects the pathophysiologic state of the visceral and parietal mesothelial surfaces [8]. A great deal of hope has been invested in ascitic cytology to provide the diagnosis at the expense of other investigations [9]. Evaluation of peritoneal fluid involves a visual assessment, in which parameters such as color and turbidity are grossly assessed, and a laboratory study including biochemical and cytological examinations. The results of peritoneal fluid analysis can be used to form predictive values, sensitivity and specificity when determining the need for intervention in peritonitis [10, 11]. Therefore, the present work was designed to compare between different severities of experimental chemical and bacterial peritonitis using gross assessment, biochemical analysis and cytological examination of the peritoneal fluid.

2. MATERIALS AND METHODS

2.1. Animals

The present study was carried out on a total number of 90 male white Albino rats (180-200 gm b.wt.). They were obtained from the United Company (Cairo, Egypt). They were housed for two weeks in the same environmental and nutritional conditions similar to those under which the experiment was performed for accommodation. Rats were randomly allocated into five groups (18 rats/group) and housed in separate cages. Each group of rats was provided by suitable feeder and water.

2.2. Induction of peritonitis

a) Thioglycollate medium (TG) was obtained from Difco Laboratories (Detroit, Michigan, USA). It was rehydrated by suspending 30 gm of the medium in 1000 ml of cold distilled water and then heated to boiling degree to dissolve it. To sterilize the medium, it was placed in the autoclave for 15 minutes at 121ºC. The medium was kept for 2 weeks after it was cold in dark place before used according to the previous method [12]. Then it was injected intraperitoneally (IP.) at zero day and collection of samples was performed after 1 day, 1 week and 2 weeks.

b) Saline broth of *Esherichia coli* (*E. coli*) was kindly obtained from Department of Microbiology and Immunology, Faculty of Veterinary Medicine, Benha University, Egypt. It was prepared by making a suspension of *E. coli* culture in broth at a concentration of 0.5 or 1×10^8 CFU/mL using turbidimetric method according to the previous method [13]. Then *E. coli* was injected IP to induce peritonitis at zero day; after that collection of peritoneal fluid

samples was performed after 1 day, 1 week and 2 weeks.

2.3. Experimental design

Rats were divided into 5 groups (each one contains 18 rats) as following: Group (C): were used as control. Group (TG_L): were injected with TG (low dose 1 ml/200 gm body weight) IP. Group (TG_H): were injected with TG (high dose 2ml/ 200gm body weight) IP. Group (EC_L): were injected with *E. coli* (low dose 0.5×10^8 CFU) IP. Group (EC_H): were injected with *E. coli* (high dose 1×10^8 CFU) IP.

2.4. Peritoneal fluid collection

Peritoneal fluid was collected according to the previous researcher [14]. First we injected 10 ml of phosphate buffer saline IP, and then making massage for abdomen to dislodge any attached cells into phosphate buffer saline, after that we collected as much fluid as possible and deposit the collected fluid in tubes kept on ice then spin the collected fluid at 1500 rpm for 8 minutes, discard the supernatant which used for biochemical analysis and resuspend the cells in PBS for counting.

2.5. Peritoneal fluid examination

Biochemical examinations include measurement of total protein, cholesterol and triglycerides according to the methods adopted previously [15-17]. Diagnostic kits used for estimation of different biochemical parameters as total protein, cholesterol and triglycerides were obtained from Qumica Clinica Alpicada (QCA) (Spain) and specific gravity was obtained from Macherey-Nagel (MN) (Germany). Cytological examination includes RBC_S count, total nucleated cells count (TNCC) anddifferential cell count by using Geimsa's stained smears were done according to standard techniques [18].

2.6. Statistical Analysis

Statistical analysis was performed using the statistical software package SPSS for Windows (Version 16.0; SPSS Inc., Chicago, Ill.). Student's *t*-test was used to determine significant differences between two experimental groups. The significance of differences between more than two groups was evaluated by one-way analysis of variance (ANOVA). If one-way ANOVA indicated a significant difference, then differences between individual groups were estimated using Fisher's least significant difference (LSD) test.Results are expressed as the mean ± standard error of mean (SEM). A *P*-value of less than 0.05 was considered significant.

3. RESULTS

3.1.Peritoneal fluid analysis in thioglycolatte-induced peritonitis

The recorded data demonstrated in (Tables 1 and 2) showed significant increases in total protein, SPG, cholesterol, TNCC and RBC_S at 1 day, 1 and 2 weeks after induction of peritonitis compared to the control group. Also TG_L and TG_H groups showed significant increases in triglycerides level at different times after induction of peritonitis. Regarding to differential nucleated cell count, TG_L and TG_H groups showed significant increases in monocytes, neutrophils and mesothelial cells counts at 1 day, 1 and 2 weeks after induction of peritonitis compared to the control group. Also, TG_L and TG_H groups showed significant increases in lymphocytes and mast cells counts at different times after induction of peritonitis compared to the control group. Normal lymphocyte and mitotic lymphocyte were recorded (photo 1). There were non-significant changes in eosinophilic and basophilic counts at all times after induction of peritonitis compared to the control group.

3.2. Peritoneal fluid analysis in E.coli-induced peritonitis

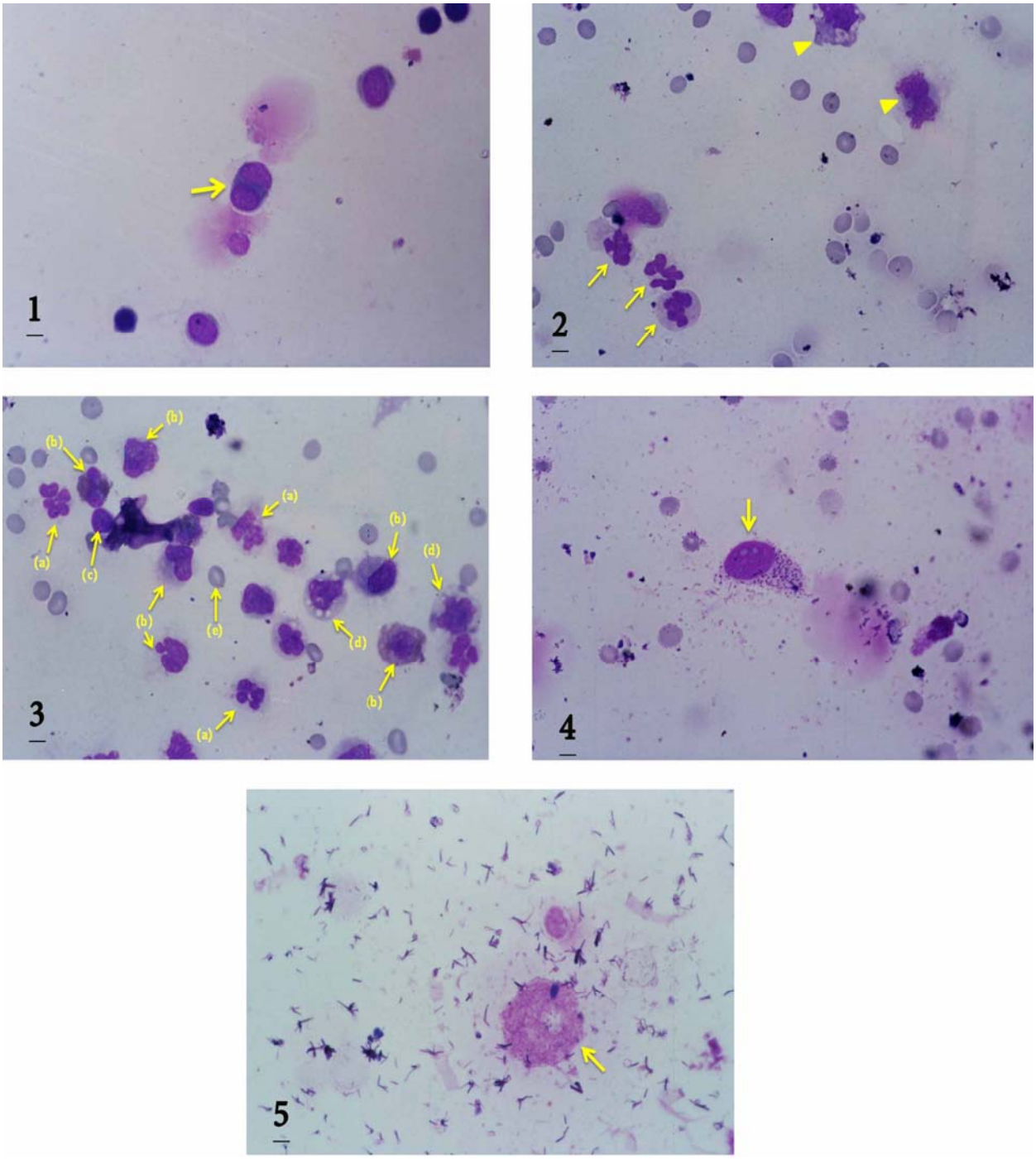

Photo 1: Cyto-centrifugation of rat abdominal fluid 1 day after thioglycolatte- treatment (2ml/gm body weight IP) shows mitotic lymphocyte (arrow), normal lymphocytes and mesothelial cells. Photo 2: Cyto-centrifugation of rat abdominal fluid 1 day after *E. coli* infection (1×10^8 CFU/ml, IP) shows neutrophils (arrows), macrophage (arrowhead) and RBCs. Photo 3:Cytocentrifugation of rat abdominal fluid 1 day after *E. coli*-infection (1×10^8 CFU/ml, IP) shows several degenerated neutrophil (with swollen nuclear chromatin) (a), reactive lymphocytes (b), small lymphocyte (c), macrophage (d), and RBCs (e). Photo 4: Cyto-centrifugation of rat abdominal fluid 1 day after *E. coli*-infection (1×10^8 CFU/ml, IP) shows macrophage cell engulfing bacteria and residues. Photo 5: Cyto-centrifugation of rat abdominal fluid 1 day after *E. coli*-infection (1×10^8 CFU/ml, IP) shows mast cell (arrow) surrounded with fibrin filaments.

Table (1): Changes of peritoneal fluid in thioglycolatte-treated rats after 1 day, 1 week and 2 weeks of treatment compared with the control group.

Parameter& Groups	Total protein (gm/dl)	SPG	Cholesterol (mg/dl)	Triglycerides (mg/dl)	TNCC ($\times 10^3/\mu L$)	RBCs ($\times 10^3/\mu L$)
C	0.19 ± 0.02^{a}	1.025 ± 0.00^{a}	0.57 ± 0.12^{a}	0.00 ± 0.00	0.59 ± 0.05^{a}	0.04 ± 0.01^{a}
TG_{L1}	0.34 ± 0.04^{b}	1.031 ± 0.00^{b}	2.24 ± 0.13^{b}	$0.13\pm0.09^{*}$	15.28 ± 0.81^{b}	1.70 ± 0.09^{b}
TG_{L2}	0.33 ± 0.02^{b}	1.031 ± 0.00^{b}	3.12 ± 0.38^{ce}	$0.07\pm0.07^{*}$	6.18 ± 0.73^{c}	0.75 ± 0.04^{c}
TG_{L3}	0.34 ± 0.02^{b}	1.031 ± 0.00^{b}	2.91 ± 0.33^{cb}	$0.10\pm0.10^{*}$	2.90 ± 0.40^{dh}	0.52 ± 0.03^{d}
TG_{HI}	0.34 ± 0.02^{b}	1.031 ± 0.00^{b}	3.17 ± 0.36^{ce}	0.00 ± 0.00	22.26 ± 1.52^{e}	2.85 ± 0.55^{e}
TG_{H2}	0.34 ± 0.01^{b}	1.031 ± 0.00^{b}	2.69 ± 0.31^{cb}	0.00 ± 0.00	2.73 ± 0.23^{fh}	0.63 ± 0.03^{fc}
TG_{H3}	0.30 ± 0.01^{b}	1.031 ± 0.00^{b}	2.22 ± 0.15^{cbd}	$0.96\pm0.14^{*}$	1.71 ± 0.12^{g}	0.40 ± 0.04^{gd}

Data represent mean values ± SEM (N=6), TG_{L1} (1 day), TG_{L2} (1 week), TG_{L3} (2 weeks), TG_{H1} (1 day), TG_{H1} (1 week), TG_{H3} (2 weeks). *($P \leq 0.05$) with the corresponding control group using student t-test. Data with different superscripts letter in the same column are significantly different at ($P \leq 0.05$) (ANOVA test).

Table (2): Changes of differential nucleated cells count of peritoneal fluid in thioglycollate-treated groups after 1 day, 1 week and 2 weeks of treatment compared with the control group.

Parameter & Groups	Lymphocyte ($\times10^3/\mu L$)	Monocyte /Macrophage ($\times10^3/\mu L$)	Neutrophil ($\times10^3/\mu L$)	Mesothelial ($\times10^3/\mu L$)	Mast ($\times10^3/\mu L$)	Eosinophil ($\times10^3/\mu L$)	Basophil ($\times10^3/\mu L$)
C	0.11±0.01[a]	0.02±0.00[a]	0.02±0.00[a]	0.41±0.03[a]	0.00±0.00	0.00±0.00	0.00±0.00
TG_{L1}	0.76±0.02[b]	0.77±0.03[b]	0.76±0.02[b]	12.98±0.50[b]	0.00±0.00	0.00±0.00	0.00±0.00
TG_{L2}	0.74±0.10[b]	0.43±0.02[c]	0.37±0.01[c]	4.57±0.44[c]	0.06±0.01*	0.00±0.00	0.00±0.00
TG_{L3}	0.43±0.02[c]	0.14±0.03[d]	0.20±0.02[dg]	2.05±0.24[d]	0.05±0.00*	0.00±0.00	0.00±0.00
TG_{HI}	0.89±0.03[db]	1.33±0.05[e]	1.11±0.04[e]	18.92±0.94[e]	0.00±0.00	0.00±0.00	0.00±0.00
TG_{H2}	0.13±0.04[a]	0.10±0.01[fd]	0.16±0.03[fg]	2.18±0.75[fd]	0.13±0.04*	0.00±0.00	0.00±0.00
TG_{H3}	0.23±0.04[a]	0.15±0.03[fd]	0.10±0.01[fg]	1.17±0.08[g]	0.03±0.00*	0.00±0.00	0.00±0.00

Data represent mean values ± SEM (N=6), TG_{L1} (1 day), TG_{L2} (1 week), TG_{L3} (2 weeks), TG_{H1} (1 day), TG_{H1} (1 week), TG_{H3} (2 weeks). *($P \leq 0.05$) with the corresponding control group using student *t*-test. Data with different superscripts letter in the same column are significantly different at ($P \leq 0.05$) (ANOVA test).

Table (3): Changes of peritoneal fluid in *E. coli*-infected groups after 1 day, 1 week and 2 weeks of infection compared with the control group.

Parameter& Groups	Total protein (gm/dl)	SPG	Cholesterol (mg/dl)	Triglycerides (mg/dl)	TNCC ($\times 10^3/\mu L$)	RBCs ($\times 10^3/\mu L$)
C	0.20 ± 0.01^{a}	1.025 ± 0.00^{a}	0.66±0.12a	0.00±0.00	0.47 ± 0.02^{a}	0.03 ± 0.01^{a}
EC_{L1}	0.32 ± 0.02^{b}	1.031 ± 0.00^{b}	$4.92\pm.0.66^{b}$	0.00±0.00	1.88 ± 0.08^{be}	4.50 ± 0.87^{b}
EC_{L2}	0.38 ± 0.03^{bd}	1.031 ± 0.00^{b}	2.73 ± 0.38^{c}	0.00±0.00	1.59 ± 0.14^{bef}	1.14 ± 0.06^{c}
EC_{L3}	0.34 ± 0.00^{b}	1.031 ± 0.00^{b}	2.59 ± 0.51^{ce}	0.00±0.00	1.28 ± 0.04^{bef}	0.81 ± 0.03^{d}
EC_{H1}	0.55 ± 0.05^{c}	1.031 ± 0.00^{b}	3.88 ± 0.59^{cb}	0.00±0.00	3.30 ± 0.30^{c}	4.11 ± 0.24^{eb}
EC_{H2}	0.43 ± 0.03^{d}	1.031 ± 0.00^{b}	4.25 ± 0.62^{cbd}	0.00±0.00	2.04 ± 0.22^{d}	1.18 ± 0.07^{fc}
EC_{H3}	0.45 ± 0.02^{d}	1.031 ± 0.00^{b}	3.51 ± 0.64^{cb}	0.00±0.00	1.75 ± 0.13^{e}	0.82 ± 0.03^{gd}

Data represent mean values ± SEM (N=6), EC_{L1} (1 day), EC_{L2} (1 week), EC_{L3} (2 weeks), EC_{H1} (1 day), EC_{H1} (1 week), EC_{H3} (2 weeks). Data with different superscripts letter in the same column are significantly different at ($P \leq 0.05$) (ANOVA test).

Table (4): Changes of differential nucleated cells count of peritoneal fluid in *E. coli*-infected groups after 1 day, 1 week and 2 weeks of infection compared with the control group.

Parameter& Groups	Lymphocyte ($\times10^3/\mu L$)	Monocyte /Macrophage ($\times10^3/\mu L$)	Neutrophil ($\times10^3/\mu L$)	Mesothelial ($\times10^3/\mu L$)	Mast ($\times10^3/\mu L$)	Eosinophil ($\times10^3/\mu L$)	Basophil ($\times10^3/\mu L$)
C	0.09±0.01[a]	0.02±0.00[a]	0.02±0.00[a]	0.32±0.02[a]	0.00±0.00	0.00±0.00	0.00±0.00
EC_{L1}	0.18±0.03[bc]	0.09±0.02[b]	0.75±0.04[b]	0.80±0.05[b]	0.00±0.00	0.01±0.00*	0.01±0.00*
EC_{L2}	0.15±0.02[ac]	0.09±0.01[b]	0.47±0.05[c]	0.85±0.04[b]	0.00±0.00	0.00±0.00	0.00±0.00
EC_{L3}	0.15±0.02[ac]	0.10±0.02[b]	0.25±0.03[d]	0.76±0.04[b]	0.00±0.00	0.00±0.00	0.00±0.00
EC_{H1}	0.26±0.03[bc]	0.16±0.02[b]	1.35±0.07[e]	1.41±0.04[c]	0.00±0.00	0.06±0.00*	0.03±0.00*
EC_{H2}	0.20±0.02[bc]	0.12±0.03[b]	0.61±0.01[fb]	1.14±0.04[c]	0.00±0.00	0.00±0.00	0.00±0.00
EC_{H3}	0.21±0.01[bc]	0.14±0.02[b]	0.35±0.04[gcd]	1.08±0.9[c]	0.00±0.00	0.00±0.00	0.00±0.00

Data represent mean values ± SEM (N=6), EC_{L1} (1 day), EC_{L2} (1 week), EC_{L3} (2 weeks), EC_{H1} (1 day), EC_{H1} (1 week), EC_{H3} (2 weeks). *($P \leq 0.05$) with the corresponding control group using student *t*-test. Data with different superscripts letter in the same column are significantly different at ($P \leq 0.05$) (ANOVA test).

The recorded data demonstrated in (Tables 3 and 4) showed significant increases in total protein, SPG, cholesterol, TNCC and RBC_S at 1 day, 1 and 2 weeks after induction of peritonitis. While EC_L and EC_H groups showed non-significant changes in triglycerides level at all times after induction of peritonitis compared to the control group. Regarding to differential nucleated cell count, EC_L and EC_H groups showed significant increases in monocytes, neutrophils and mesothelial cell counts at 1 day, 1 and 2 weeks after induction of peritonitis compared to the control group. Many neutrophil were recorded (photo 2), also several degenerated neutrophils with swollen nuclear chromatin and reactive lymphocyte were recorded (photo 3). In addition, macrophage engulfing bacteria and residues was found (photo 4). EC_L and EC_H groups showed significant increases in lymphocytes, eosinophilic and basophilic counts at 1 day after induction of peritonitis. One mast cell was recorded (photo 5) while, there were non-significant changes in mast cells count at all the experimental periods.

4. Discussion

Peritoneal fluid analysis has a considerable potential aid to hematological and clinical examination in the diagnosis of abdominal disorders since it can directly assess the volume of peritoneal fluid, cellularity and protein characteristics, thereby giving an indication of inflammatory changes in the peritoneal cavity. It may be also valuable in the diagnosis of chronic peritonitis and other different diseases in different animals [19].

The present work shed more light on the effects of chemical and bacterial peritonitis on peritoneal fluid analysis and investigated the influence of peritonitis on hematological, biochemical parameters and histopathological examinations.

Concerning the results of peritoneal fluid analysis, TG_L and TG_H groups showed significant increases in total protein and SPG at 1 day, 1 and 2 weeks after induction of peritonitis compared to the control group. High protein level in peritoneal fluid occurs most commonly due to presence of chemotactants in the peritoneal cavity resulted from the inflammatory process. Inflammation in the peritoneal cavity increased vascular permeability that allows plasma (and its protein,) to ooze out of the blood into exudate in peritoneal fluid [20]. These results agreed with previous data[21]. TG_L and TG_H groups showed significant increases in cholesterol at 1 day, 1 and 2 weeks after induction of peritonitis compared to the control group. In addition, TG_L and TG_H groups showed significant increases in peritoneal triglycerides at 2 weeks while, TGL group showed significant increases in triglycerides level at 1 day and 1 week after induction of peritonitis. These results similar with those obtained by previous researcher [22] who owed increased levels of cholesterol and triglycerides as a result of lymphatic obstruction which leads to rupture of lymphatic channel causing increased exudation of chyle with a relatively high lipid content. In addition, cholesterol concentration was significantly higher because of degeneration of cellular components, such as WBCs and RBCs, in the fluid or because cholesterol can exit the vasculature due to increased capillary permeability in vasculitis [23].

Concerning peritoneal total nucleated cells count (TNCC) and RBC_S count results, TG_L and TG_H groups showed significant increases at 1 day, 1 and 2 weeks after induction of peritonitis compared to the control group. Differential nucleated cells count was revealed significant increases in neutrophils, monocytes and mesothelial cells in TG_L and TG_H groups at 1 day, 1 and 2 weeks after induction of peritonitis compared

to the control group. In addition, TG_L and TG_H groups showed significant increases in lymphocytes at 1day after induction of peritonitis compared to the control group. Meanwhile, mast cells were increased significantly in TG_L and TG_H groups 1 and 2 weeks after induction of peritonitis compared to the control group. These results similar with that obtained by previous studies [24] which reported that leukocytosis with significant increases in neutrophils, macrophages and lymphocytes after induction of chemical peritonitis in peritoneal fluid with no significance in basophils and eosinophils. The exudation of protein rich fluid due to peritonitis is usually accompanied by the migration of leukocytes (mostly neutrophil) into the effusion because of chemotactic substance in the fluid. The vascular permeability is typically increased by the effects of inflammatory mediators (e.g., histamine, bradykinin, leukotrienes) that were released from inflamed tissue. The inflammatory mediators may also cause selective vasodilation, so that more blood enters the inflamed tissues and thus increases the hydraulic pressure in the capillaries [25]. Peritonitis cause damage to mesothelium, which increase mesothelial cells in peritoneal fluid. Beside their structural functions, mesothelial cells are involved in inflammatory responses of peritoneal cavity, including antigen presentation, cytokine production, release of oxidants and proteases, and promotion of the migration of neutrophils [26].

The results of peritoneal fluid analysis in bacterial peritonitis showed significant increases in protein levels and SPG in EC_L and EC_H groups at 1 day, 1 and 2 weeks after induction of peritonitis. These findings agree previous researcher [27]. The inflammatory mediators released due to peritonitis and associated with bacteria ensue vascular damage, which increases the vascular permeability to plasma proteins [28]. Our results disagree with [29] who recorded non-significant changes in SPG and significant decreases in total protein in peritonitis. Regarding to cholesterol results, EC_L and EC_H groups showed significant increase at 1 day, 1 and 2 weeks after induction of peritonitis compared to the control group. Interestingly, triglyceride concentrations showed no significant changes in EC_L and EC_H groups. Formation of "pseudochyle" which typically contains cholesterol but not triglycerides can occur in long-standing effusions [30].

EC_L and EC_H groups showed significant increase in RBC_S at 1 day, 1 and 2 weeks after induction of peritonitis compared to its control. This results agree with previous data [31]. Several studies have quantified cytokine activity in theperitoneal fluid of human patients with various diseases ofthe abdomen. TNF and IL-6 are pro-inflammatory endogenous mediatorthat can contribute to the pathologic events of peritonitis by inducing the release of other mediators, such as interleukins, eicosanoids, platelet-activating factor, tissue factor, and plasminogen-activator inhibitor. When produced locally in the peritoneal cavity, TNF may be an important contributor to the development of intra-abdominal inflammation associated with presence of red cells in the exudate. In addition, it may gain access to the circulation and contribute to systemic deterioration [32].

EC_L and EC_H groups showed significant increases in TNCC at all times after induction of peritonitis compared to the control group. Regarding to differential nucleated cells, EC_L and EC_H groups showed significant increases in monocytes, neutrophils and mesothelial cells at all the experimental period. Also, EC_H group showed significant increases in lymphocytes at 1 day, 1 and 2 weeks after induction of peritonitis. EC_L and EC_H groups showed significant increases in basophils and eosinophils at 1 day only after induction of

peritonitis. These findings were similar partially withprevious studies [33] who recorded that the number of TNCC, neutrophils, monocytes and band neutrophils significantly increased in peritonitis. When foreign pathogens are introduced into the normally sterile peritoneal cavity, invading bacteria are rapidly absorbed into the lymphatic circulation via host mesothelial cells [34-36]. Following detection by innate immune cells and triggering of several defense pathways, bacteria are recognized and degraded by various phagocytes, and fragments are displayed for recognition by other cells of the immune system [37, 38]. Simultaneously, a local acute inflammation ensues with consequent release of histamine from mast cells, followed by an acute vascular dilatation and leak of activated inflammatory components including complement, cytokines, immunoglobulins, clotting factors, fibrin and polymorphonuclear cells [39].

5. CONCLUSION:

From the previous results, we can conclude that peritoneal fluid analysis is an important tool in diagnosis of different types of peritonitis. Chemical and bacterial peritonitis lead to several changes in peritoneal fluid analysis. Chemical peritonitis model showed significant increases in TNCC specially mesothelial cell. However, bacterial peritonitis model showed significant increases in TNCC specially neutrophils.

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مجلة بنها للعلوم الطبية البيطرية
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التغيرات البيوكميائية والخلوية فى السائل البريتونى بعد حقن مادة الثيوجليكولات وبكتيريا الإشيرشيا كولاى داخل التجويف البريتونى فى الفئران

رانيا محمد السيد بكر[1]، أيمن سمير فريد[1]، أسامة على عبد الله[2] و خالد محمد فرارة[1]
[1]قسم الباثولوجيا الإكلينيكية-كلية الطب البيطري-جامعة بنها، [2] قسم الباثولوجيا الإكلينيكية-كلية الطب البيطري-جامعة قناة السويس

الملخص العربي

إن تحليل السائل البريتونى يعد هاماً فى تشخيص اضطرابات البطن المختلفة بما فى ذلك التهاب التجويف البريتونى. التهاب التجويف البريتونى الكيميائي والبكتيري يؤدى إلى العديد من التغيرات فى السائل البريتونى. لكي نوضح تأثير التهاب التجويف البريتونى الكيميائي والبكتيري على تحليل السائل البريتونى، فإن مادة ثيوجليكولات (TG) وبكتيريا الإشيريشيا كولاى (E.coli) تم استخدامهم لإحداث الالتهاب البريتونى.100 فأر تم تقسيمهم إلى خمس مجموعات، كل مجموعة تحتوي على 20 فأر. تم استخدام مجموعة واحدة ضابطة، ومجموعتين تم حقنهم بمادة الثيوجليكولات 3% (1 أو 2 مليلتر لكل 200 مليجرام من وزن الجسم) داخل التجويف البريتونى والمجموعتان الأخيرتان تم حقنهم ببكتيريا الإشيريشيا كولاى (1/2 أو 1 × 10^8 CFU.) داخل التجويف البريتونى. تم تجميع عينات السائل البريتونى بعد يوم، أسبوع وأسبوعان من إحداث التهاب التجويف البريتونى. نتائج كلا النموذجين تبين تغيرات عديدة فى تحليل السائل البريتونى. كلا النموذجين أحدثا زيادة في تركيز البروتين الكلى، الكوليسترول والكثافة النوعية (SPG) بعد كل الأوقات من إحداث التهاب التجويف البريتونى بالمقارنة مع المجموعة الضابطة. التهاب التجويف البريتونى الكيميائي أحدث زيادة فى مستويات الدهون الثلاثية بعد يوم واحد من إحداث التهاب التجويف البريتونى بالمقارنة مع المجموعة الضابطة، بينما التهاب التجويف البريتونى البكتيري لم يحدث أي تغيرات في مستويات الدهون الثلاثية بعد كل الأوقات من إحداث التهاب التجويف البريتونى بالمقارنة مع المجموعة الضابطة. الفحص الخلوي للسائل البريتونى فى كلا النموذجين أوضح زيادة فى العدد الكلى للخلايا ذات النواة (TNCC) كرات الدم الحمراء، خلايا منوسيت (monocytes), خلايا النيتروفيل (neutrophils) و خلايا الميزوثيليل (mesothelial cells) بعد كل الأوقات من إحداث التهاب التجويف البريتونى بالمقارنة مع المجموعة الضابطة. التهاب التجويف البريتونى الكيميائي أحدث تغيرا ملحوظا في خلايا الميزوثيليل (mesothelial cells) بالمقارنة بالتهاب التجويف البريتونى البكتيري. على العكس فان التهاب التجويف البريتونى البكتيري أحدث تغيرا ملحوظا فى خلايا النيتروفيل (neutrophils) بالمقارنة مع التهاب التجويف البريتونى الكيميائي. كلا النموذجين أحدث زيادة في الخلايا الليمفاوية (lymphocytes) بعد يوم واحد من إحداث التهاب التجويف البريتونى بالمقارنة مع المجموعة الضابطة.

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