Clinicopathological Studies On The Effect Of Some Bacterial Infection In Rabbits

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ABSTRACT

The present investigation was carried out on experimentally infected rabbits which obtained from the Animal House, Faculty of Veterinary Medicine, Zagazig University. A total of 45 white New Zealand rabbits apparently healthy, 0.5-0.75 kg body weight and one month age were used. The rabbits were divided into 4 groups. Group 1 (15) were kept as control. Group 2 (10) was dosed orally with 1.37ml of cooked meat medium($1X10^9$ microorganism/Kg body weight) of C.perfringens type A in 5ml sodium bicarbonate10%. Group 3 (10) was dosed orally with 5ml bacterial suspension (normal saline) containing ($5 X10^7$CFU) of E.coli . Group 4 (10) was dosed orally with 1.37ml of cooked meat medium in 5ml sodium bicarbonate 10% ($1X10^9$ microorganism/Kg body weight) of C.perfringens type A and 5ml bacterial suspension (normal saline) containing ($5 X10^7$CFU) of E.coli. Whole blood and serum samples were collected after 6hours, 1and 2weeks post infection.

Clinical examination of the experimentally infected rabbits revealed depression, dullness, browen watery diarrhea and bloated abdomen and ascitis. Haematological examination revealed a significant changes in the erythrocytic and leukocytic parameters 6hours,1and 2weeks post infection. Biochemical analysis of the same groups revealed a significant changes in the AST,ALT, ALP, total protein, albumin ,globulin, total, direct and indirect bilirubin, creatinine,Ca,P and Na. It could be concluded that the rabbit infection with *Clostridium perfringens type A and E.coli* adversely affected the hepatic and renal functions.

INTRODUCTION

Rabbit industry, in Egypt, is recently progressing rapidly as they have an economic importance. They provide a high protein meat which is poor in fat, easily digestable and nutritious. Moreover, they contribute to the production of fur, antibodies and antisera. They are used in different research aspects as ophthalmology and teratology *(1).* Diseases associated with diarrhea, cause a major financial loss in the rabbit industry. Enterotoxemia occurs in rabbits, kept in hutches, and occasionally in the individual pet rabbits. The disease is caused by *Clostridial* spp. These are anaerobic gram-postive bacilli capable of producing a powerful enterotoxin *(2).*

Escherichia coli is among the most important etiologic agents of enteritis and losses in rabbits. *E.coli* infection, among the growing rabbits, is a serious problem among the enteric diseases. The bacteriological examination revealed that *E.coli* was the predominant micro-organism isolated from ligated colon and cecum *(3).*

Colibacillosis, in White New Zealand rabbits, occurs in acute form with high mortality and destructive lesions . It affects the health condition producing high economic losses, reduces the productive activity and inducing pathological damage in different organs (intestine, liver, kidneys and heart) *(4).* The present work was designed to investigate the hematological and biochemical changes in single and mixed experimentally infected by *Clostridium perfringens type A and E.coli* in the *White New* Zealand rabbits.

MATERIAL AND METHODS

Experimental Design

A total of 45 apparently healthy white New Zealand rabbits, 0.5-0.75 kg body weight and one month age were obtained from the Animal House, Faculty of Veterinary

Medicine, Zagazig University. The rabbits were divided into 4 groups. Group 1 (15) were kept as control. Group 2 (10) was dosed orally with 1.37ml culture of cooked meat medium in 5ml 10% sodium bicarbonate ($1X10^9$ microorganism/Kg body weight of *C.perfringens type A*) ***(5)***. Group 3 (10) was dosed orally with 5ml bacterial suspension (normal saline containing 5 $X10^7$CFU) of *E.coll* ***(6)***. Group 4 (10) was dosed orally with 1.37ml culture of cooked meat medium in 5ml 10% sodium bicarbonate ($1X10^9$ microorganism/Kg body weight of C.perfringens type A) and 5ml bacterial suspension (normal saline containing 5 $X10^7$CFU) of E.coli.

Sample collection: Blood samples (5 samples from each group) were collected from the jugular vein after 6hrs and 1week post infection in gps.2 and 4, while gp.(3) samples were collected after 1 &2 weeks post infection and gp.(1) samples were collected after 6hours,1 and 2 weeks. A small amount containing EDTA was used as whole blood for hematological picture. The remaining amount was centrifuged for serum separation to be used for biochemical parameters estimation.

Bacteriological studies: The bacterial strains and bacterial risolation was obtained and carried in the aerobic and anaerobic Bacterial Section' Animal Health Research Institute' Abasia , Egypt.

Hematological studies: The erythrogram (*7*) and the leukogram (*8*) was carried out.

Biochemical studies: The serum activities of alanine aminotransferase (ALT), aspartate amino transferase (AST) and the alkaline phosphatase (ALP) were colorimetrically estimated. Serum total protein, albumin, globulin, bilirubin (total, direct and the indirect),the creatinine,Ca, P, and Na levels were determined ***(7)***.

Statistical analysis: The obtained data were statistically analyzed by F-test (one way ANOVA) in groups. 2 & 4 and "t" test in group.3 ***(9)***.

RESULT AND DISCUSSION

Enterotoxemia causes a major loss in rabbits. The disease is caused by Clostridium spp. These are anaerobic gram-postive bacilli capable of producing a powerful enterotoxin. *Clostridium perfringens type A* causes enterotoxemia in rabbits ***(10)***. Escherichia coli organisms are considered the most important etiologic agent of enteritis and losses in rabbits ***(2)***. The symptoms of experimental infected rabbits was brown to bloody fecal spoiling of the perineum and the hind legs, besides enlarged abdomen. Palpation of gases and intestinal fluid was found in addition to dehydration, anorexia. Our results are similar to those previously reported ***(11)***.

The hematological results as demonstrated in Table 1 showed a highly significant increase in the erthrocytic count in group. 2 6hours post infection due to infection which lead to hemconcentration ***(12)*** while gp.(4) showed a highly significant decrease in all erythrocytic parameters due to hemorrhagic enteritis 6hours post infection. While after 1week post infection, gps (2 & 4) showed macrocytic hypochromic anemia this was attributed to the mixed infection by the E.coli and *Clostridium* microorganism that induced lesions in the intestine. Such lesions were represented by patchy or diffuse reddening, and hemorrhage in the form of petechiae and paint brush hemorrhage in the intestinal mucosa which led to blood loss ***(12)***. The bone marrow responded to compensate the blood loss by increased the production of the erythrocytes and early released the reticlucytes (immature cell). Group. (3) showed a highly significant decrease in the erythrogram after one and two weeks from the infection. This could be attributed to the strains of E.coli which attached themselves into the enterocyte cell membranes and produce severe enteritis and hemorrhagic colitis which led to a loss of erythrocytes, epithelial necrosis, hemorrhage and emigration of granulocytes ***(12)***. These animals showed macrocytic hypochromic anemia. Our results are inconsistant with previous studies ***(4&6)*** which reported non-significant changes in all the erythrocytic parameters.

Table 1.The erythrogram (mean ± S E) in rabbits of all groups. And the leukogram (Total ($X10^3$/ µl and absolute differential leukocytic count cell/ µl)

Group	RBCs 10^6/ µL	Hb gm%	PCV %	MCV fl	MCHC %	Retic %	TLC	Lymph	Neut	Eosin	Baso	Mono
1^{6hrs} Control	5.39 b ±0.02	12.08 b ±0.1	31.4 b ±0.51	58.22 b ±1.1	38.48 b ±0.3	2.20a ±0.12	5.75a ±0.06	3620 a ±0.06	1930 c ±0.02	91.8 c ±13.84	23.00 a ±0.25	80.70b ±14.43
2^{6hrs} Clostridium	5.68 a ±0.09	13.28 a ±0.16	33.4 a ±0.6	58.76 b ±0.22	39.76 a ±0.31	2.31a ± 0.09	5.92a ±0.09	2526.4b ±0.1	3054.4a ±0.05	236.6a ±27.58	11.8 b ±0.19	95 a ±15.36
4^{6hrs} Mixed	4.81 c ±0.002	11.88 b ±0.04	30.4 b ±0.24	69.36 a ±0.47	35.54 c ±0.09	2.34 a ± 0.08	4.62b ±0.09	2054.8c ±0.1	2372.8b ±0.07	137.7 b ±12.25	0.000 c ±0.000	64.10c ±10.47
1^{1wk} Control	5.22 a ±0.05	12.16 a ±0.07	32.8 a ±0.37	62.79 c ±0.21	37.10 a ±0.19	2.00 b ± 0.04	6.03 c ±0.02	3410 b ±0.06	2380 c ±0.06	120.88 ±0.49b	36.28a ±0.15	84.72b ±14.98
2^{1wk} Clostridium	2.84 b ±0.02	9.76 c ±0.07	30.40b ±0.24	107.10 a ±0.3	32.12 c ±0.18	2.62 a ± 0.12	7.69 b ±0.18	3590 b ±0.07	3670 b ±0.15	184.78a ±18.63	30.82b ±0.73	216.86a ±18.47
3^{1wk} E.coli	4.43 ±0.08	11.52 ±0.1	33.60 ±0.51	75.82 ±0.52	34.24 ±0.23	2.26 ±0.20	6.78 ±0.24	3246.2 ±0.13	3193.7 ±0.13	179.94 ±17.92	27.64 ±0.94	138.44 ±4.76
4^{1wk} Mixed	5.15 a ±0.07	11.48b ±0.1	33.80a ±0.37	65.58 b ±0.43	33.92 b ±0.17	2.36 a ± 0.11	8.97 a ±0.27	3990 a ±0.17	4530 a ±0.11	196.2a ±14.66	35.88a ±1.1	215.6a ±23.81
1^{2wk} Control	5.23 ±0.09	12.36 ±0.13	32.60 ±0.51	62.20 ±0.37	37.89 ±0.22	2.10 ±0.10	6.28 ±0.05	3580 ±0.08	2490 ±0.07	100.62 ±15.57	12.56 ±0.09	100.82 ±15.84
3^{2wk} E.coli	4.44 ±0.01	11.60 ±0.00	33.40 ±0.25	75.20 ±0.43	34.60 ±0.25	2.54 ±0.12	9.57 ±0.22	4630 ±0.16	4340 ±0.31	307.8 ±26.71	38.28 ±0.87	249 ±24.76

Means in the same column not followed by the same letter differ significantly($p<0.05$)

Concerning the leukogram, group. (2) showed non-significant increase in the TLC, associated with neutrophilia, eosinophilia, lymphopenia and monocytosis. This may be attributed to the effect of the *Clostridium* and its toxin which caused lymphopenia (*7*). Similar findings were reported by other reports (*13*). The encountered neutrophilia and eosinophilia are assumed to the body defence by phagocytosis. They were markedly increased after one week from the infection. Our results partially agree with similar findings which reported leukocytosis, neutrophilia and lymphopenia (*14-16*). Group. (4) showed a highly significant decrease in the TLC associated with neutrophilia, eosinophilia, lymphopenia and monocytopenia. The neutrophilia and eosinophilia may be attributed to the activation of the bone marrow by particular chemical mediators. Similar results were reported previously (*7,14-16*). Group. (3) showed a highly significant increase in the TLC with a non-significant decrease in the lymphocytes, a highly significant increase in the neutrophils and monocytes with a significant eosinophilia, one week post infection. Marked leukocytosis, lymphocytosis, neutrophilia, eosinophilia and monocytosis were seen, 2 week post infection. Such result may be due to antigenic stimulation of the infecting *E.coli* (*7*). The leukocytosis was generally characterized the bacterial infection (17). Our results are in agreement with other findings (*4,6&18*).

Regarding the biochemical studies, as demonstrated in Table 2 the ALT in the present work showed a highly significant increase in groups. 2&4. Such elevation may be attributed to the congestion, besides the inflammatory and degenerative changes which occur in the liver due to the toxins produced by the bacterial agents and not regained its normal level after one week from the infection. The level of the AST showed an increase because it is a monitor to the

liver damage. Our results are in agreement with earlier result *(19)* which reported a significant increase in the activity of AST and ALT in guinea pigs S/C injected with *Clostridium tetani* toxin. The hepatocellular damage resulted in a release of the cellular enzymes *(17)*. The elevated cellular enzymes in the blood was due to degenerative changes in the liver, induced by the toxins produced by the *E.coli (18)*. The alkaline phosphatase (ALP) in the present work showed a highly significant increase in all treated groups denoting hepatocellular jaundice. Such elevation could be attributed to the damaged hepatic, renal and intestinal cells, associated with inflammation and congestion due to the bacterial toxin. Previous reports *(6,16&18)* attributed the increase in the AP to the destructive effect of the toxins, produced by the *E.coli* and *Clostridium,* on the cell membrane permeability of the hepatocytes.The degree of the elevation of the alkaline phosphatase depended upon the severity of the lesions.

Table 2. The hepatic function tests (mean ± S E) in rabbits of all groups.

Group	ALT UI	AST UI	ALP UI	Proteins (gm/dl)			Bilirubin(mg/dl)		
				Total	Albumin	Globulins	Total	Direct	Indirect
1^{6hrs} Control	20.80 b ±0.49	27.40 c ±0.74	34.10 b ±0.91	5.59 b ±0.06	3.4 a ± 0.08	2.19 c ±0.12	1.02 b ±0.01	0.45 c ±0.009	0.57 c ±0.01
2^{6hrs} Clostridium	62.40 a ±1.86	70.2 b ±3.10	36.98 c ±0.92	6.92 a ±0.45	3.5 ab ±0.19	3.42 a ±0.21	2.08 a ±0.12	1.03 b ±0.04	1.05 b ±0.09
4^{6hrs} Mixed	65.60 a ±0.6	61.00 c ±3.36	56.00 a ±1.29	6.77 a ±0.21	3.73 a ±0.12	3.04 b ±0.14	2.21 a ±0.10	0.66 c ±0.02	1.55 a ±0.11
1^{1wk} Control	27.80 b ±0.66	30.80 b ±1.74	35.60 b ±0.63	6.26 a ± 0.14	3.44 ab ±0.16	2.82 a ±0.18	1.38 b ±0.02	0.48 b ±0.02	0.9 a ±0.02
2^{1wk} Clostridium	41.60 a ±0.93	37.20 a ±1.88	39.00 a ±0.52	6.27 a ±0 .14	3.71 a ±0 .2	2.56 a ± 0.04	1.65 a ±0.12	0.73 a ±0.09	0.92 a ±0.06
3^{1wk} E.coli	35.80 ±0.49	36.20 ±1.46	43.88 ±0.19	5.99 ±0.26	3.16 ±0.14	2.83 ±0.26	1.84 ±0.02	0.56 ±0.01	1.28 ±0.01
4^{1wk} Mixed	40.20 a ±0.97	39.40 a ±1.54	40.70 a ±0.86	5.61 b ±0 .14	3.05 b ± 0.14	2.56 a ±0 .18	1.34 b ±0.05	0.51 b ±0.007	0.83 a ±0.05
1^{2wk} Control	21.80 ±0.86	29.80 ±0.58	32.72 ±0.84	6.68 ±0.07	3.61 ±0.07	3.07 ±0.09	1.03 ±0.009	0.39 ±0.007	0.64 ±0.01
3^{2wk} E.coli	30.20 ±0.58	35.80 ±0.8	40.69 ±0.64	4.62 ±0.15	2.73 ±0.04	1.89 ±0.18	1.24 ±0.01	0.45 ±0.06	0.79 ±0.06

Means in the same column not followed by the same letter differ significantly($p<0.05$)

Regarding the proteinogram, our results showed a highly significant increase in the total proteins and globulin in groups 2&4, the increase is attributed to the presence of the diarrhea which induced hemoconcentration, 6 hours post- infection. Group 4, showed a significant decrease in the total proteins one week post infection, meanwhile the albumin and globulins showed a non- significant decrease. Such findings could be attributed to the remaining of anorexia induced by the infection. The hypoproteinemia in enteric buffalo calves infected with E.coli was attributed to the damaged intestinal villi

which lead to malabsorption *(20&21)*. The proteinogram in group 3 showed a non-significant change in the total proteins, albumin and globulin one week post infection. A non-significant decrease in the total proteins, albumin and globulin could be attributed to the short period (acute state) before the sample collection after the onset of the clinical signs *(18)*. While two weeks post infection, group 3 showed a highly significant decrease in the total proteins, albumin and globulin it may due to decrease the food intake and maldigestion and malabsorbtion resulted from the enteritis. A decrease in the total protein and the albumin was recorded in experimentally infected rabbits with *E.coli (4&6)*. The serum bilirubin (total, direct and the indirect) showed a highly significant increase in the experimentally infected gps.(2&4) after 6 hours post infection. This could be attributed to the infective agent and its toxins which caused disturbance in the hepatocellular activity and destructive effect leading to hepatocellular jaundice. This elevation became non-significant after one week, but did not regain the normal level due to the mild persistent effect in group 4 but group. 2 showed a significant increase in the total and direct bilirubin. This is due to the slight disappearance of the effect. Group 3 showed a highly significant increase in the total, direct and indirect bilirubin after one week due to the ability of the *E.coli* to produce a toxic hepatic damage one week post- infection, while after two weeks the toxin of the *E.coli* produced a moderate hepatic damage which elevated the total, direct and the indirect bilirubin. An increased total, direct and indirect bilirubin has been reported in the experimentally infected rabbits with *E.coli* ($6X10^8$ and $3X10^8$ CFU/ml) due to hepatocellular damage *(4)*. Similar results were reported which indicated an increased direct and indirect bilirubin with intrahepatic disease *(22)*. A decrease in the total bilirubin, direct and indirect has been cited in the toxic hepatic disease and other liver disease *(23)*.

The creatinine level, in the present work, as demonstrated in Table 3. showed a highly significant increase in infected groups 2-4 from 6 hours to one week PI but after two weeks the creatinine level was returned to normal. This could be due to the renal lesions which became more milder after two weeks PI. *(4,6&16)*. An increase serum creatinine level in rabbits, infected with E.coli and /or Clostridium spp which attributed to the effect of the microorganisms and its toxin on the kidneys.

The serum calcium level, in the present work, showed a non-significant decrease in groups 2,3&4 6 hrs, one and two weeks post infection due to lesser effect of the experimental infection on the absorption and rabsorpion of calcium. A significant decrease in the calcium also has been reported *(19)*. A decrease in the serum calcium in rabbits experimentally infected with *E.coli* may be due to intestinal lesions which led to decreased absorption *(18)*. The serum phosphorus level showed a highly significant increase in groups 2&3. This abnormality could be attributed to the hemoconcentration (gp.2) and renal damage with phosphorus retention (gp.3). Our results contradict with that which showed non significant changes in the phosphorus-level, in rabbits experimentally infected with *E.coli (18)*. Group 4 showed a highly significant decrease after 6 hours post infection. This could be due to the partial loss in the diarrheal fluid and the extravasated plasma in the abdomen due to the increased vascular permeability by the toxin. This effect disappeared, one week post infection in gp.4 due to disappearance of the renal damage one week post infection. The serum sodium level showed a highly significant decrease in gps 2-4. This decrease could be attributed to the diarrhea which led to a partial loss of the body fluids. This decrease was alleviated in gp.3, two weeks post infection. This could be due to the ability of the defensive mechanism to overcome the infection and failed to do so in gps.2&4 one week post infection. The sodium depletion is a result of excessive loss of sodium containing fluid, through vomition and diarrhea, besides sequestration of a portion of the extra- cellular fluid *(24)*.

Table 3. The creatinine, calcium, phosphorus, sodium and potassium (mg/dl) in rabbits groups (mean ± S E).

Group	Creatinine	Ca	P	Na
1^{6hrs} Control	0.68 c ±0.02	7.58 a ±0.21	5.42 b ±0.28	142.65 a ±0.21
2^{6hrs} Clostridium	2.19 a ±0.11	7.01 a ±0.09	7.46 a ±0.16	138.88b ±1.4
4^{6hrs} Mixed	1.46 b ±0.14	7.22 a ±0.24	4.63 c ±0.12	136.02 c ±0.29
1^{1wk} Control	0.930 b ±0.01	9.43 a ±0.25	5.44 a ±0.29	144.88 a ±0.25
2^{1wk} Clostridium	1.19 a ±0.07	9.31 a ±0.56	5.12 a ±0.29	136.79 b ±3.35
3^{1wk} E.coli	1.29 ±0.02	9.15 ±0.13	7.47 ±.12	131.74 ±1.57
4^{1wk} Mixed	0.97 b ±0.02	8.57 a ±0.18	5.54 a ±0.14	137.1 b ±0.74
1^{2wk} Control	1.10 ±0.02	8.52 ±0.17	5.87 ±0.11	144.48 ±0.13
3^{2wks} E.coli	1.12 ±0.03	7.56 ±0.22	5.53 ±0.14	143.14 ±0.43

It could be concluded that the rabbit infection with *Clostridium perfringens type A and E.coli* adversely affected the hepatic and renal functions. The infection with either *Clostridium perfringens or E.coli* alone was less severe than that caused by both microorganisms, as they appear to be synergistics.

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الملخص العربي

دراسات باثولوجية إكلينيكية علي تأثير العدوى ببعض البكتريا في الأرانب

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أجريت هذه الدراسة لتقييم تأثير العدوى التجريبية بالكلوستريديوم و الميكروب القولوني كل علي حدة أو مجتمعتين معا لمعرفة تأثيرهم علي خلايا وكيمياء الدم ووظائف الكبد و الكلوه.

تم هذا العمل بإجراء فحص بكتريولوجي و تم عزل الاشيريشيا كولاى والكلوستريديم برفيرنجينس نوع أ. واستخدامت العترات المعزولة في 45 أرنبا نيوزيلانديا وقسمت كالتالي. المجموعة الأولي:وهي مكونة من 15 أرنبا تم استعمالها كمجموعة ضابطة. المجموعة الثانية: وهي مكونة من 10 أرنبا تم عدواها با لكلوستريديم برفيرنجينس نوع أ عن تركيز $X1\ 10^9$ ميكروب حي والسم الناتج منة. المجموعة الثالثة: وهي مكونة من 10 أرنبا تم عدواها بالاشيريشيا كولاى عن تركيز $X5\ 10^7$ ميكروب حي. المجموعة الرابعة: وهي مكونة من 10 أرنبا تم عدواها با لكلوستريديم برفيرنجينس نوع أ و الاشيريشيا كولاى. أوضحت نتائج الباثولوجيا الإكلينيكية والباثولوجية تأثيرا ضار علي الأعضاء الحيوية(الكبد والكلي والأمعاء) كما لوحظ زيادة في معدلات إنزيمات الكبد وصبغة البيليروبين الكلي والمباشر والغير مباشر والكرياتينين وتغير في نسبة الكالسيوم والفسفور والصوديوم كما أوضحت النتائج وجود أنيميا وزيادة في عدد الخلايا البيضاء.