BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE

BENHA VETERINARY MEDICAL JOURNAL

CLINICOPATHOLOGICAL STUDIES ON THE EFFECT OF ENTERIC DISEASES IN BROILER CHICKS

Gheith, I.M.[a], Fararh, K.M.[b], Bakry, H.H.[c], Hosney G.A.[d]

[a]Departement of Biotechnology, Animal Health Research Institute, Dokki, Egypt, [b]Department of Clinical pathology, Faculty of Veterinary Medicine, Benha University, Egypt, [c]Department of Forensic Medicine, Faculty of Veterinary Medicine, Benha University, Egypt, [d]Departement of Pathology , Animal Health Research Institute, Dokki, Egypt.

A B S T R A C T

60 Cobb chicks one day old were used in this study and were fed balanced ration. The chicks were divided into 3 groups (n= 20/group). Group (A) used as control group, group (B) and group (C) was experimentally infected with *Campylobacter jejuni* (*C.jejuni*) at the 4th day and *Clostridium perfringens* (*C. perfringens*) type (A) at the 10th day of age, respectively. Blood sample were collected at 2nd, 4th and 6th weeks of age to examine hematological, biochemical, immunological parameters and acute phase parameters. The present results showed that there were a significant decrease in lymphocytes number, albumin, total protein, cholesterol, calcium in group (B) and (C) compared to group (A) and significant increase in total leukocytes count, heterophilis number, fibrinogen, ESR, glucose, creatinine, uric acid, AST, and haemagglutination inhibition antibody titer. It is concluded that *C.jejuni* and *C. perfringens* type (A) are important enteric disease that caused lack of production efficiency of broiler chicks as a result of their negative effect on the function of liver, intestine and pancreas. Therefore it is necessary to find the best treatment to counteract the effect of enteric diseases on broiler chicks.

KEY WORDS: Acute phase parameters, Campylobacter, Chicken, Clostridium, Enteric diseases

(BVMJ 22(2): 17-26, 2011**)**

1. I N T R O D U C T I O N

Enteric diseases of broiler chicks are the most important diseases that threaten the economics of the poultry industry because of loss of productivity, increased mortality, reduced welfare of birds and illness to human as the poultry act as asymptomatic carriers and as reservoir of these enteric pathogens. *Campylobacter* and *Clostridia* are the main enteric pathogenic microorganism in broiler chicks, that colonizing the intestinal tract and are major causes of bacterial food poising in the world today [4, 29] that continue catch the attention of researchers, food processers and consumers. *Campylobacters* are small gram-negative, nonspore-forming, curved spiral or rod shaped bacteria that are microaerophilic in nature [15]. It has been noted that once *Campylobacter* is established within an individual bird, horizontal transmission [30]. The symptoms caused by *C. perfringens* type A usually are relatively mild and limited to the elderly and very young, but occasionally death may occur due to dehydration [36] often occurs rapidly through the flock [17]. *C. perfringens* is a gram-positive anaerobic spore-forming bacterium, able to produce various toxins and enzymes responsible for the associated lesions and symptoms. *C. perfringens* strains are classified into five toxinotypes (A, B, C, D and E), based on the

production of four major toxins (α, β, ε and ι). Therefore the aim of this study was to examine the effect of experimental infection of *C. jejuni* and *C. perfringens* on hematological, biochemical, immuno-logical parameters and changes in acute phase parameters in broiler chicks.

2. MATERIAL AND METHODS

2.1. Birds:

A total of 60 one day old chicks (Cobb breed) were used. They were obtained from (Alahram Company).the chicks were randomly allocated into four groups (20 chicks / group). Group (A) act as control group, while group (B) and (C) were experimentally infected with *Campylobacter jejuni* and *C. perfringens*, respectively. The chicks were housed in clean well-ventilated previously fumigate room. The room floor was bedded by fresh clean chopped wheat straw forming a deep litter of 3.5 cm depth, which was turned over weekly and changed every two weeks. Each group of bird was provided by suitable feeder and water. The broiler chicks were fed on well-balanced diet prepared from a corn-soybean meal based diet. Starter diet was given till the 20^{th} days of age followed by chicks were fed on finisher diet which was given from the 21^{st} day till the end of the experiment. The chicks were vaccinated against most common viral diseases, which infect the broiler chicks.

2.2. Experimental infection microorganism

2.2.1. Campylobacter jejuni:

At the 4^{th} day of age, each chick of group (A) and (B) was orally infected with 0.1 ml saline containing (2.5×10^{8} CFU) *C.jejuni*, which was kindly obtained from Department of Poultry diseases, National Research Center, Dokki, Giza, Egypt.

2.2.2. Clostridium perfringens:

At the 10^{th} day of age, each chick of group (C) and (D) was infected via subcutaneous route with 0.1 ml saline containing (9×10^{8} CFU) *C. perfringens* type (A), which was kindly obtained from Department of Bacteriology in Animal Health Research Institute, Dokki, Giza, Egypt.

2.3. Blood sampling

Blood samples were collected from heart and placed on tube contain EDTA as anticoagulant for hematological examination and in plane tube for separation of serum to be used in biochemical and immunological tests .The blood were collected at 2^{nd}, 4^{th} and 6^{th} weeks of age.

2.4. Clinicopathological assays

2.41. Hematological assay:

TLC, DLC and ESR were determined according to methods described previously [7].

2.4.2. Biochemical assays:

Total proteins, albumin, serum electrophoresis, total cholesterol, AST, glucose, creatinine, uric acid, calcium and fibrinogen were estimated using commercial kits (Stanbio, USA) [33].

2.4.3. Immunological assay:

Haemagglutination inhibition (HI) test was performed on serum samples according to methods described previously [21].

2.5. Statistical analysis:

The obtained data were analyzed with one way ANOVA using the statistical software package SPSS for Windows (version 11).

3. RESULTS

Group (A) showed no clinical signs throughout the experimental period, meanwhile group (B) showed depression, decrease of appetite and diarrhea, and group (C) showed in-appetence, ruffled feather, huddling, anorexia, dehydration

and sternal recumbency. Regarding to hematological parameters in group (B) and (C), significant increase in (TLC) at 4th and 6th weeks of age was recorded and significant lymphopenia was found at 6th week of age compared to group (A) results, while significant heterophilia was recorded throughout of the experimental period (table 1). Concerning to acute phase parameters, there were significant increase in fibrinogen concentration and ESR results in group (B) and (C) at 4th week of age till the end of experimental period, while the concentration of albumin revealed significant decrease at 4th and 6th weeks of age (table 2). The concentration of total proteins, Alpha 1 and Beta globulins concentration of group (B) and (C) showed significant decrease at 2nd week of age till the end of experimental period, however Alpha 2 concentration showed significant decrease at 4th and 6th week. Meanwhile gamma globulins concentration in group (B) showed no significant changes at 2nd and 6th weeks of age and significant increase was seen at 4th week of age, while the data of group (C) showed significant increase at the 2nd and 4th weeks of age, but no significant changes were observed at 6th week. Regarding to A/G ratio, significant increase was detected at 6th week of age (table 3). The cholesterol results showed significant decrease in in group (B) and (C) at 2nd week of age till the end of experimental period compared to group (A).

Table 1 Leukogram of different groups

Parameter	Age (Week)	Group (A)	Group (B)	Group (C)
Total leuk. count ($\times10^3$/µl)	2nd week	18.80±3.03^a	22.00±4.30^a	21.80±3.40^a
	4th week	19.00±2.23^a	23.00±3.87^b	26.60±1.67^c
	6th week	21.00±2.30^a	24.00±1.50^b	27.00±2.90^c
Lymphocytic count ($\times10^3$/µl)	2nd week	12.40±1.67 ^{a}	11.20±0.83 ^{a}	11.6±1.14 ^{a}
	4th week	13.40±0.70 ^{a}	11.60±1.81^a	13.00±0.70 ^{a}
	6th week	16.00±1.22 ^{a}	13.00±1.00^b	14.20±2.16^c
Heterophilic count ($\times10^3$/µl)	2nd week	5.40±0.54^a	8.40±1.14^b	7.20±1.78^c
	4th week	5.60±1.90^a	8.40±1.90^b	11.00±1.50^c
	6th week	4.00±0.71^a	10.40±1.67^b	10.20±1.10^c
Monocytic count ($\times10^3$/µl)	2nd week	1.400±0.140^a	1.800±0.283 ^{a}	1.600±0.140 ^{a}
	4th week	1.400±0.116 ^{a}	1.800±0.288 ^{a}	1.600±0.140 ^{a}
	6th week	1.600±0.220 ^{a}	1.600±0.216 ^{a}	1.200±0.303 ^{a}

Means (± SD) with different superscripts (a, b and c) within a row are significantly different at $P < 0.05$.

Table 2 Changes in acute phase proteins parameters of different groups

Parameter	Age (week)	Group (A)	Group (B)	Group (C)
Fibrinogen (g/dl)	2nd week	0.63±0.09^a	0.72±0.14^a	0.73±0.69^a
	4th week	0.50±0.05^a	0.76±0.12^b	0.78±0.10^c
	6th week	0.50±0.08^a	0.70±0.17^b	0.78±0.69^c
Albumin (g/dl)	2nd week	1.10±0.14^a	0.88±0.08^a	0.84±0.04^a
	4th week	1.20±0.20^a	0.87±0.02^b	0.84±0.04^c
	6th week	1.27±0.05^a	0.80±0.02^b	0.78±0.02^c
ESR (mm) (1st hour)	2nd week	0.78±0.17^a	0.85±0.05^a	0.84±0.04^a
	4th week	0.88±0.18^a	1.02±0.19^a	1.12±0.18^b
	6th week	0.72±0.13^a	1.21±0.22^b	1.30±0.15^c

Means (±SD) with different superscripts (a, b and c) within a row are significantly different at $P< 0.05$.

Regarding to A/G ratio, significant increase was detected at 6th week of age (table 3). The cholesterol results showed significant decrease in in group (B) and (C) at 2nd week of age till the end of experimental period compared to group (A). Regarding to the activity of AST, significant increase throughout the experiment was seen (table 4). The results of serum glucose concentration revealed significant hyperglycemia and significant increase in creatinine concentration and uric acid throughout the experimental period. Significant hypocalcaemia throughout the experimental period in group (B) and (C) was seen (table 5). The HI antibody titers of group (B) and (C) were higher than those of group (A) from 2nd week of age till the end of experiment (table 6).

Table 3 Changes in serum total protein and serum electrophoretic pattern of different groups

Parameter	Age (week)	Group (A)	Group (B)	Group (C)
Total proteins (g/dl)	2nd week	2.55±0.43[a]	1.87±0.06[b]	1.88±0.23[c]
	4th week	2.05±0.56[a]	1.85±0.32[b]	1.63±0.49[c]
	6th week	2.74±0.27[a]	1.64±0.23[b]	1.55±0.32[c]
Alpha (1) globulins (g/dl)	2nd week	0.372±0.02 [a]	0.027±0.01[b]	0.081±0.02[c]
	4th week	0.378±0.06 [a]	0.145±0.05[b]	0.111±0.01[c]
	6th week	0.386±0.02 [a]	0.016±0.00[b]	0.038±0.03[c]
Alpha (2) globulins (g/dl)	2nd week	0.451±0.07 [a]	0.355±0.04[a]	0.378±0.04[a]
	4th week	0.478±0.01 [a]	0.343±0.04[b]	0.255±0.052[c]
	6th week	0.487±0.06 [a]	0.389±0.04[b]	0.319±0.02[c]
Beta globulins (g/dl)	2nd week	0.337±0.07 [a]	0.088±0.17[b]	0.134±0.05[c]
	4th week	0.333±0.04 [a]	0.133±0.05[b]	0.163±0.02[c]
	6th week	0.301±0.04 [a]	0.089±0.00[b]	0.062±0.01[c]
Gamma globulins (g/dl)	2nd week	0.258±0.05 [a]	0.344±0.03[a]	0.376±0.02[b]
	4th week	0.233±0.04[a]	0.338±0.02[b]	0.316±0.04[c]
	6th week	0.282±0.07[a]	0.313±0.03[a]	0.371±0.61[a]
A/G ratio	2nd week	0.78±0.09[a]	1.08±0.18[a]	0.86±0.08[a]
	4th week	0. 84±0.15 [a]	0.90±0.05 [a]	0.99±0.08 [a]
	6th week	0.87±0.02 [a]	0.99±0.09 [b]	0.99±0.10 [c]

Means (± SD) with different superscripts (a, b and c) within a row are significantly different at $P < 0.05$.

Table 4 Total cholesterol and Activity of serum Aspartate aminotransferase (AST) of different groups

Parameter	Age (Week)	Group (A)	Group (B)	Group (C)
Total cholesterol (mg/dl)	2ndweek	151.70±6.40[a]	127.21±17.57[b]	126.91±11.55[c]
	4th week	152.41±12.84[a]	130.92±11.12[b]	134.79±6.80[c]
	6th week	150.23±5.13[a]	131.30±7.84[b]	132.52±7.03[c]
AST(U/L)	2nd week	142.40±6.73[a]	192.40±8.64[b]	199.20±10.32[c]
	4th week	156.90±10.11[a]	193.20±13.16[b]	201.20±8.46[c]
	6th week	151.30±7.96[a]	194.60±14.04[b]	202.20±8.25[c]

Means (± SC) with different superscripts (a, b and c) within a row are significantly different at $P < 0.05$.

4. DISCUSSION

Broiler chicks are the most important and cheapest sources of animal protein in Egypt, But with rapid progressive expansion of poultry industry to meet the increasing demand of meat, enteric bacterial pathogens such as *Salmonella*, *Campylobacter* and *C. perfringens* can cause economic losses to the poultry industry and also have public health significant as the poultry act as asymptomatic carriers and as a reservoir of these enteric bacterial pathogens which

cause illness to human [24]. In the present study group (A) (control group), showed no clinical signs throughout the experimental period, while group (B) which orally infected with *C. jejuni*, showed depression, decrease of appetite and diarrhea. Similar signs were observed by in earlier reports [31, 39]. Our result disagree with some studies [34] stated that experimental infections with *C. jejuni* did not produce any clinical abnormalities in broiler chicks aged either 2 days or 3 weeks, although intestinal colonization was achieved by inoculation via both the oral and cloacae routes. Group (C), which was experimental with *C. perfringens*, showed ruffled feather, huddling, anorexia, dehydration and sternal recumbency. Similar sign was previously observed previously [10, 22].

Table 5 Changes in serum glucose, creatinine, uric acid, and calcium concentration of different groups

Parameter	Age / week	Group (A)	Group (B)	Group (C)
Glucose (mg/dl)	2nd week	257.98±35.08^{a}	340.00±14.40^{b}	357.47±18.45^{c}
	4th week	297.14±38.60^{a}	381.51±12.16^{b}	382.35±26.90^{c}
	6th week	284.36±39.26^{a}	366.89±30.52^{b}	387.56±21.46^{c}
Creatinine (mg/dl)	2nd week	0.90±0.07^{a}	1.40±0.15^{b}	1.40±0.26^{c}
	4th week	0.86±0.08^{a}	1.44±0.15^{b}	1.60±0.10^{c}
	6th week	0.89±0.10^{a}	1.52±0.08^{b}	1.62±0.08^{c}
Uric acid (mg/dl)	2nd week	12.32±1.12^{a}	17.17±2.03^{b}	18.22±0.93^{c}
	4th week	11.63±0.61^{a}	19.99±1.75^{b}	19.32±0.73^{c}
	6th week	12.65±2.52^{a}	19.89±2.41^{b}	19.07±1.15^{c}
Calcium (mg/dl)	2nd week	8.07±0.62^{a}	5.64±0.74^{b}	5.24±0.46^{c}
	4th week	8.39±0.88^{a}	5.40±0.65^{b}	5.00±0.70^{c}
	6th week	8.34±0.50^{a}	5.24±0.28^{b}	5.10±0.88^{c}

Means (± SC) with different superscripts (a, b and c) within a row are significantly different at $P < 0.05$.

Table 6 Results of Haemagglutination inhibition (HI) test of experimentally infected chicks and control group

Parameter	Age /week	Group (A)	Group (B)	Group (C)
HI antibody titer	2nd week	3.2±1.56	3.4±1.26	3.5±1.00
	4th week	2.3±1.00	3.4±1.75	3.8±1.62
	6th week	3.3±1.14	3.6±1.01	3.6±1.94

Means (± SC) with different superscripts (a, b and c) within a row are significantly different at $P < 0.05$.

Evaluation of leukogram revealed leukocytosis at 4th and 6th weeks of age and significant lymphopinia at 6th week of age was found in group (B) and (C), when compared with group (A), moreover there were significant heterophilia throughout the experimental period. The finding of leukocytosis may be occurred as patho-physiological response during acute phase response as reaction of host to infection and inflammation [19]. The pervious data reflect a stress condition on the chicks due to bacterial infection. Heterophilia in infected group may be related to that heterophil is the 1st cell of defense in the body, which attack and engulf the microorganism. Moreover, may be related to tissue destruction [35] caused by bacterial infection also as a normal response to bacterial infection [7, 37]. Our data disagree with earlier studies [13, 38] who reported that during acute phase response, decrease in leukocytes was observed. Lymphopeinia may be also resulted from infection by *C. perfringes* due to necrosis of foillular lymphocytes in

bursa of Fabricius which was recorded in chicks [8].

Acute phase proteins are group of plasma proteins which are synthesized in the liver and released into blood stream by a variety of stimuli including inflammation and bacterial infection [18]. Acute phase response may be increased (positive acute phase proteins) or deceased (negative acute phase proteins) during inflammatory disorders [26]. Gruys et al. [14] conclude that in veterinary medicine, determination of acute phase proteins gives valuable clinical information on infection and inflammatory conditions. In our study, fibrinogen concentration showed significant increase at 4^{th} week of age till the end of experimental period. These results are in harmony with previous results [23] which showed an increase in fibrinogen concentration during bacterial infection. The results disagree with Nazifi et al. [28] who reported that no significant changes in fibrinogen concentration during infection. This increase suggestive to occurred due to bacterial infection and inflammation [5]. That fibrinogen is one of plasma proteins secreted by hepatocytes and its level increased (positive acute phase proteins) approximately 90 minute after onset of a systemic inflammatory reaction [32]. Concerning to albumin, the concentration of albumin revealed significant decrease at 4^{th} and 6^{th} weeks of age. Similar results were previously obtained by Mazur-Gonkowska et al. [23]. Our findings were confirmed by Murata [27] who mentioned that albumin behaves as a negative acute phase proteins, which decrease during bacterial infection. Hypoalbuminemia may be related to loss of albumin through intestine as results of lesions in intestine and also as a result of decrease the production of albumin by liver due to liver lesions. Moreover may be as a result of the reduction in feed intake in infected groups. Assessment of ESR result, at 6^{th} week of age significant increases were observed in group (B) and (C) .This could be attributed to bacterial infection and the inflammation. This opinion supported by Gadzinski and Julian [10] who mentioned that ESR is an indirect index of acute phase protein concentration and it is sensitive but not specific index of plasma proteins changes, which result from inflammation and infection. Further support of this concept came from Alsemgeest [1] who mentioned that bacterial infection usually leaded to strong systemic acute phase response. ESR is increased in associations with tissue damage, inflammation and an increase in fibrinogen in plasma [5] that erythrocytes normally have net negative charge and therefore repel each other but during the inflammation, fibrinogen, which have positive charge increase and bind to RBCs thereby reducing the negative charge and allowing rouleaux formation to occur, which increase ESR. The fore mentioned results of ESR were obtained in the same hand with previous fibrinogen finding. Our result indicated that infected groups with *Campylobacter* and *Clostridium* showed acute phase response. These results are in fit with the concept of Miettinen et al. [25] who reported that whole bacterial cells are shown to induce production of proinflammatory cytokines, such as tumor necrosis factor α and interleukin 6 which stimulated the acute phase response [2].

The results of total proteins in chicks of group (B) and (C) showed significant hypoprotinemia at 2^{nd} week of age till the end of experimental period, which may be due to loss of protein through intestine as a result of enteritis and decrease in liver production of protein. Also hypoprotenima may be due to decrease in feed intake. These results simulate those reported by Gheith [12]. Regarding to electrophoric patterns of group (B) and (C) , alpha 1 concentration, showed significant decrease at 2^{nd} week of age till the end of experimental period, alpha 2 concentration showed significant decrease at 4^{th} and 6^{th} weeks of age and beta globulins concentration showed significant decrease

throughout the experimental period compared with group (A). Significant hypoglobinemia (alpha and beta globulins) may be as results of liver lesions that liver is the site of production of alpha and beta globulins [5]. Our results disagree with Gheith [12] who observed increase in alpha and beta globulins during bacterial infection in broiler chicks. Concerning to gamma globulins, group (B) showed significant increase at 4th week of age, while group (C) showed significant increase at the 2nd and 4th weeks of age. Hyper gammaglobulinemia could be related to increase of immunoglobulin due to antigenic stimulation [37] caused by *C. jejuni* and *C. perfringens* type (A).

Our result showed significant decrease in cholesterol concentration in group (B) and (C) from the 2nd week of age till the end of experimental period. This decrease may be attributed to septicemia and liver lesions which caused by bacterial infection [11] as *Campylobacter* and *Clostridium*. On the same ground [5] said that in some forms of hepatic failure, decreased cholesterol synthesis can lead to decrease blood cholesterol concentration. Moreover, the enteritis and hepatic lesion in group (B) and (C) may be cause decrease in intestinal absorption and hepatic lipogenic activity [3]. These results are disagreeing with Tanaka et al. [36] who mentioned that increase in plasma cholesterol may be associated with bile duct hyperplasia. AST activity showed significant increase throughout the experiment in group (B) and (C), this increase could be related to hepatic affection, as AST is indicator of hepatocellular damage [16]. Moreover, the increase in AST indicates damage in liver. These results agree with these previously obtained by Gheith [12]. Regarding to concentration of serum glucose, group (B) and (C) showed significant hyperglycemia throughout the experiment period. These increase occurred as a results of stress on the chicks due to bacterial infection. During stress epinephrine release and mobilize the stored glycogen in liver, also stress increase glucocorticoides which they stimulate glycogenlysis and gluconegenesis [20] our results also agree with Gruysn and Snel [13] who mentioned that during acute phase response increase adrenocorticotrophic hormone (ACTH) and glucocorticoids were occurred. Concerning to the calcium concentration, the present study revealed significant hypocalcaemia throughout the experimental period in group (B) and (C), when compared with group (A).These decrease may attributed to enteritis that reduced calcium absorption, increased execration and may be related to hyoalbuminemia [5]. Our results are in agreement with Gruysn and Snel [13] who mentioned that during acute phase response decrease in calcium level of serum was reported. Regarding to uric acid and creatinine concentration, there were significant increase in group (B) and (C) throughout the experimental period compared with group (A). These results agree with Gheith [12]. This elevation may be related to renal disease [20]. Uric acid is the major end product of nitrogen metabolism and it is produced by liver and kidney, moreover the blood uric acid can used as an indicator of renal function in bird and severe renal disease may result in an increased creatinine concentration [37]. Also hyperuricumia in bird may be as a result of protein degradation due to poor nutritional statues [5] and massive tissue destruction by bacteria. With respect to the antibody titers against Newcastle virus, the present study revealed that the HI antibody titers of group (B) and (C) were higher than those of group (A) from 2nd week of age till the end of experiment. This may be related to the effect of bacterial cell which enhance proliferation of immune cells [6]. These results related to increase in immunoglobulins in these groups, which manifested and confirmed by increase in gamma globulins, moreover these results may be attributed to antigenic stimulation which accompany variety of infectious and hepatic diseases [37].

5. CONCLUSION

It is concluded that *C. jejuni* and *C. perfringens* type (A) are important enteric disease that caused lack of production efficiency of broiler chicks as a result of their negative effect on the function of liver, intestine and pancreas. Therefore it is necessary to find the best treatment to counteract the effect of enteric diseases on broiler chicks.

6. REFERENCES

1. Alsemgeest, S.P.M. 1994. Blood concentrations of acute-phase proteins in cattle as markers for disease. PhD Thesis Utrecht, the Netherlands: Utrecht University. ISBN: 90-3-0573-0.
2. Baumann, H., Gauldie, J. 1994. The acute phase response. *Immunol.* **15**: 74 -80.
3. Black, D.G. 1981. Avian Clinical Pathology. In: Refresher course on aviary and Caged Birds University of Sydney **55**: 224 -248.
4. Brynestad, S., Granum, P.E. 2002. *Clostridium perfringens* and foodborne infections. *Int. J. Food Microb.* **74**: 195-202.
5. Coles, E.H. 1986. Avian Clinical Pathology. In: Veterinary Clinical Pathology. 4th Ed., Washington Square, Philadelphia, Toronto.
6. De Simone, C., Vesely, R., Bianchi Salvadori, B., Jirillo, E. 1993. The role of probiotics in modulation of the immune system in man and in animals. *Int. J Immunotherap.* **9**:23-28.
7. Feldman, B.F., Zinkl, J.G., Jain, N.C. 2000. Schalm's Veterinary Hematology. 5th Ed., Lea and Febiger, Philadelphia, USA.
8. Frame, D.D., Bickford, A.A. 1986. An outbreak of coccidiosis and necrotic enteritis in 16-week-old cage-reared layer replacement pullets. *Avian. Dis.* **30**: 601-602.
9. Gabay, C., Kushner, I. 1999. Acute phase proteins and other systemic responses to inflammation. *Erratum in N. Engl. J. Med.* **340**: 448 - 454.
10. Gadzinski, P., Julian, R.J. 1992. Necrotic enteritis in turkeys. *Avian. Dis.* **36**: 92-98.
11. Galvin, C.E. 1980. Laboratory diagnostic aids in pet birds practice. Proc. of the American Animal Hospital. Association South Bend. IN. Pp.41.
12. Gheith, E.M. 2008. Clinicopathological studies on effect of probiotics on broiler. M.V.Sc. Thesis, Fac. Vet. Med., Benha University.
13. Gruysn, E., Snel, F.W.J. J. 1994. Animal models for reactive amyloidosis bailliere's. *Clin. Rheumatol.* **8**:599-611.
14. Gruys, E., Obwolo, M.J., Toussaint, M.J.M. 1994. Diagnostic significance of the major acute phase proteins in veterinary clinical chemistry: a review. *Vet. Bull.* **64**: 1009-1018.
15. Halablab, M.A., Mohammed, K.A.S., Miles, R.J. 2008. Growth and survival of *Campylobacter* pathogens in the presence of different metabolic inhibitors. *J. Medical. Sci.* **8**: 262-268.
16. Harr, K.E. 2002. Clinical chemistry of companion avian species: A review. *Vet. Clin. Pathol.* **31**: 143-151.
17. Horrocks, S.M., Anderson, R.C., Nisbet, D.J., Ricke, S.C. 2009. Incidence and ecology of *Campylobacter jejuni* and *coli* in animals. *Anaerobe* **15**: 18 -25.
18. Jamieson, J.C., Wayne, S., Belo, R.S., Wright, J.A., Spearman, M.A. 1992. The importance of N-linked glycoproteins and dolichyl phosphate synthesis for fusion of L6 myoblasts. *Biochem. Cell Biol.* **70**: 408 -412.
19. Johnson, R.W. 1997. Inhibition of growth by pro-inflammatory cytokines an integrated view. *J. Anim. Sci.* **75**: 1244-1255.
20. Kaneko, J.J. 1997. Clinical biochemistry of domestic animals. San Diego (CA): Academic Press.
21. King, D.J., Seal, B.S. 1998. Biological and Molecular characterization of Newcastle disease virus (NDV) field isolates with comparisons of reference NDV strains and pathogenicity chicken or embryo passage of selected isolates. *Avian Dis.* **42**: 507-516.
22. Long, J. R. 1973. Necrotic enteritis in broiler chickens. I. A review of the literature and the prevalence of the disease in Ontario. *Can . J. Comp Med.* **37**: 302-308.

23. Mazur-Gonkowska, B., Koncicki, A., Krasnodebska-Depta, A. 2004. Assessment of acute phase response in turkeys experimentally infected with *Escherichia coli* or haemorrhagic enteritis virus EM: *Bulletin-of-the-Veterinary-Institute-in-Puawy* **48**: 19-23.
24. Mead, P.S., Slutsker, L., Dietz, V., Mccaig, L.F., Bresee, J.S. 1999. Food-related illness and death in the United States. *Emerg. Infect. Dis.* **5**: 607-625.
25. Miettinen, M., Vuopio-Varkila, J., Varkila, K. 1996. Production of human tumor necrosis factor alpha, interleukin-6, and interleukin-10 is induced by lactic acid bacteria. *Infect Immun.* **64**: 5403-5405.
26. Morley, J.J., Kushner, I. 1982. Serum C-reactive protein levels in disease. *Ann N Y Acad Sci.* **389**: 406-418
27. Murata, H. (2007): Stress and acute phase protein response: An inconspicuous but essential linkage. *Vet. J.* **173**: 473-474.
28. Nazifi, S., Dadras, H., Hosseinian, S.A., Ansari-Lari, M., Masoudian, M. 2010. Measuring acute phase proteins (haptoglobin, ceruloplasmin, serum amyloid A and fibrinogen) in healthy and infectious bursal disease virus-infected chicks. *Comp. Clin. Pathol.* **19**: 283-286.
29. Newell, D.G., Fearnley, C. 2003. Sources of *Campylobacter* Colonization in Broiler Chickens. *Appl. Environ. Microbiol.* **69**: 4343–4351.
30. Petit, L. Gilbert, M., Popoff, M.R. 1999. *Clostridium perfringens*: toxinotype and genotype. *Trends in Microbiology.* **7**:104-110.
31. Ruiz-Palacios, G.M., Escamilla, E., Torres, N. 1981. Experimental *Campylobacter* diarrhea in chickens. *Infect. Immun.* **34**: 250-255.
32. Ruminy, P. 2001. Gene transcription in hepatocytes during the acute phase of systemic inflammation: from transcription factors to target genes. Inflam. Res. **50**: 383-390.
33. Salkie ML (1996). A retrospective study of the relative utility of electrophoresis, immunoelectrophoresis, immunofixation, and nephelometry in the investigation of serum proteins.Clinical Biochemistry, **29**(1): 39-42
34. Shanker, S., Lee, A. and Sorrell, T.C. 1988. Experimental colonization of broiler chicks with *Campylobacter jejuni*. *Epidemiol. Infect.* **100** :27-34.
35. Steven, L., Michael, A. 2002. Fundamentals of Veterinary Clinical Pathology. 2nd Ed., Low State Press.
36. Tanaka, D., Isobe, J., Hosorogi, S., Kimata, K., Shimizu, M., Katori, K., Gyobu, Y., Nagai, Y., Yamagishi, T., Karasawa, T., Nakamura, S. 2003. An outbreak of food-borne gastroenteritis caused by *Clostridium perfringens* carrying the cpe gene on a plasmid. *Jap. J. Inf. Dis.* **56**: 137-139.
37. Thrall, M.A. 2004. Veterinary Hematology and Clinical chemistry. Lippincott Williams and Wilins, Maryland, USA.
38. Titball, R.W., Naylor, C.E., Basak , A.K. 1999. The *Clostridium perfringens* α toxin. *Anaerobe*. **5**: 51- 64.
39. Welkos, S.L. 1984. Experimental gastroenteritis in newly hatched chicks with *Campylobacter*. *J. Med. Microbiol.* **18**: 233-248.

مجلة بنها للعلوم الطبية البيطرية
BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE

دراسات باثولوجيه اكلينيكية عن تأثير الامراض المعوية علي بداري التسمين

ابتسام محمد غيث[1]، خالد مصطفي فراراه[2]، حاتم حسين بكري[3]، جيهان حسني[4]

[1] قسم البيوتكنولوجي – معهد بحوث الصحه الحيوانيه بالدقي، [2] قسم الباثولوجيا الاكلينكيه– كلية الطب البيطري – جامعة بنها، [3] قسم الطب الشرعي – كليه الطب البيطري – جامعه بنها، [4] قسم الباثولوجيا – معهد بحوث الصحه الحيوانيه بالدقي

الملخص العربى

اجريت هذه الدراسه على 60 كتكوت عمر يوم واحد من سلاله الكب، تغذت علي عليقه متزنة، قسمت الي ثلاث مجموعات (20 كتكوت/المجموعة) وكانت المجموعه الاولي بمثابه مجموعه ضابطه. وقد تم اجراء عدوي تجريبية للمجموعه الثانيه بالكامبيلو بكتر جيجيناي عند عمر اربع ايام اما المجموعه الثالثه فقد تم اجراء عدوي تجريبه بالكولوستيريديا بريفرنجيس عند عمر 10 ايام. وقد. تم تجميع عينات الدم في الاسبوع الثاني والرابع والسادس من عمر الكتاكيت لفحص التغيرات الدمويه، البيوكيميائية، و المناعية. واظهرت نتائج المجموعه الثانيه والثالثه مقارنه بنتائج المجموعه الاولي ان هناك انخفاض ملحوظ في عدد الخلايا الليمفاويه والزلال والبروتين الكلي والكلولستيرول والكالسيوم وزياده معنويه في عدد كرات الدم البيضاء والهتيروفيل و الفيبرينوجين و سرعه ترسيب الدم والجلوكوز والكرياتينين وحمض البوليك و ونشاط انزيم الاسبرتيت امينو ترانس فريز وتركيز الاجسام المضاده ضد تحصين النيوكسل. وخلصت نتائج الدراسه علي ان الكامبيلو بكتر جيجيناي والكولوستيريديا بريفرنجيس من اهم الامراض المعويه التي تسببت في عدم كفاء الانتاج في بداري التسمين نتيجه لتاثيرها السلبي علي وظيفه الامعاء والكبد والبنكرياس ولذالك فمن الضروري ايجاد العلاج الامثل لمواجه تلك الامراض.

(مجلة بنها للعلوم الطبية البيطرية: عدد 22 (2)، ديسمبر 2011: 17-26)