Clinicopathological Studies On Greenish Diarrhea In Chickens

Fakhry S. Salem, Amany A.M. Abd Allah
Sahar S. Abd El-Hamied* and Doaa I. A. Mostafa*
Clinical Pathology Department , Faculty of Vet. Medicine, Zagazig University
and *Animal Health Research Institute, Zagazig

ABSTRACT

The present study was performed to investigate the clinicopathological changes associated with greenish diarrhea in chickens. One hundred, one day old Hubbard chicks were used for this experiment. They were divided into five equal groups. The first group was kept as normal control, gp. 2 was infected orally with ***Salmonella gallinarum***, gp. 3 was infected I/M with VVNDV, gp. 4 was treated orally with lead acetate while gp. 5 was kept under temporary starvation. Chicks of all treated gps. showed greenish diarrhea at 32 days old.

Hemolytic anemia was observed, persisted in gps. 2 and 3, till the day 39 then disappeared but in gps. 4 and 5 persisted till the end of the experiment leading to hyperbiliverdenemia, biliverdinuria and greenish diarrhea. The blood films showed poikilocytosis and anisocytosis in gps. 4 and 5. The osmotic fragility test showed significant increase in gps. 4 and 5, along the experimental periods. The leukogram showed leukocytosis in gp. 2 at 32 and 39 days old, in gp. 4 along the experimental periods, leukopenia in gps. 3 and 5 at 32 and 39 days old and in gp. 5 at 46 days old.

Regarding the biochemical changes, hypoglycemia and hypocholesterolemia were recorded in gps. 2 and 3 at 32 and 39 days old, in gp. 4 along the experimental periods. Gp. 5 showed hypoglycemia along the experimental periods but hypercholesterolemia at 32 days old and hypocholesterolemia at 46 days old. Fowl Typhoid increased the ALT and CK activities but decreased the AP activity at 32 and 39 days old then returned to their normal activities at 46 days old. Gp. 3 showed significant increase in ALT activity, significant decrease in AP activity and non significant change in serum CK activity at 32 and 39 days old then returned to normal levels at 46 days old. Lead poisoning increased the activities of ALT and CK by increase the duration of treatment but decrease the AP activity along the experimental periods. Starvation decreased the AP activity and didn't affect the ALT and CK activities along the experimental periods. Hyperuricemia was seen in gps. 2, 3 and 5 which was reversible in gps. 2 and 3 and returned to its normal level at 46 days old but in gp. 5 it persisted along the experimental periods.

INTRODUCTION

Chickens are an important source of animal protein for human beings. Chicken industry in Egypt has been well established, as the annual broiler production was increased to reach 160 millions birds in 1994 which give 170 million kilograms of broiler meat *(1)*, therefore many problems may be common. Diarrhea is a clinical sign of many poultry diseases but it's not pathognomonic for any one. Examining the color, texture, consistency and volume of feces, urate and urine will provide informations about gastrointestinal, renal and hepatic diseases of the birds. Green loose feces and yellow green urates indicate hemolysis, or toxic, chlamydial, bacterial or viral hepatitis *(2)*. The etiology of greenish diarrhea vary widely. It may be bacterial as Fowl Typhoid *(3)*, viral as velogenic viscerotropic Newcastle Disease *(4)*, toxic as lead poisoning *(5)*, and starvation *(2)*. This work was performed to investigate the clinicopathological changes (hematological, biochemical and pathological) associated with greenish diarrhea in chickens.

MATERIALS AND METHODS

A-Chickens

One hundred, one day old commercial Hubbard chickens were used for this

experiment. Chickens were kept under standard hygienic conditions and on a commercial well balanced ration. All chickens were vaccinated against ND by Hitchner B1 via eye drop at 7 days old and against Gumboro disease via drinking water at 15 days old *(6)*.

B-Strains

1- ***Salmonella gallinarum***: $2.4x10^8$ viable organisms/ml was obtained from Animal Health Research Institute, Dokki, Cairo, to be used for experimental infection.

2- Velogenic viscerotropic strain of NDV (10^6 EID_{50}/ml) was obtained from Vet. Serum and Vaccine Research Institute, Abbassia, Cairo, to be used for experimental infection.

C-Chemical

Lead acetate white crystals contained about 99% lead were obtained from ADWIA Company. It was evenly distributed in ration to contain 40 mg/kg.

Experimental design

One hundred one day old chicks were used and divided equally into 5 groups each of 20 chicks.

Group 1: Was kept as normal control.

Group 2: Chicks of this group were infected orally with ***Salmonella gallinarum*** 0.5 ml/bird ($2.4x10^8$ viable organism/ml) using stomach tube at 28 days old.

Group 3: Chicks of this group were infected with velogenic viscerotropic Newcastle disease virus 0.2 ml/bird (10^6EID_{50}/ml) I/M at 28 days old.

Group 4: Chicks of this group were treated with lead acetate from 7th days old orally in the diet by the dose of 40 mg/kg ration till the end of the experiment.

Group 5: Chicks of this group were subjected to temporary starvation by fasting with providing sufficient water and limited amout of food to mentain life from 29 days old till the end of the experiment.

Blood sampling

Two blood samples were collected from chickens at 32 days old in all groups, and weekly for 2 weeks (at 39 and 46 days old) from the wing vein. The first sample (about 1 ml) was received into clean tubes containing disodium salt of EDTA anticoagulant in a dose (one drop of 10% solution/ml blood) for hematological examination. The second was three ml of blood collected without anticoagulant and used for serum separation for biochemical analysis.

Hematological studies

Erythrocytic and leukocytic counts were performed using an improved Neubauer hemocytometer and Natt and Herrick solution as a special diluent for chicken's blood *(7)*. The packed cell volume was estimated by microhematocrit centrifuge *(8)*. Hemoglobin estimation was performed using the cyanomet-hemoglobin colorimetric method after centrifugation *(9)*. Mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC) were calculated. Blood films were made, fixed by methyl alcohol, stained by Giemsa stain for detection of abnormalities in RBCs morphology *(10)*.

Osmotic fragility test was performed using Weitherman tube, NaCl 1% and distilled water *(11)*.

Clinico-biochemical studies

Serum Glucose *(12)* and cholesterol *(13)* levels were determined. Serum ALT *(14)*, AP *(13)* and CK *(15)* activities were estimated. Serum uric acid was determined *(16)*.

Statistical analysis

The obtained data were statistically analyzed by F-test *(17)*.

RESULTS AND DISCUSSION

Diarrhea in birds is clinically recognized by unformed feces, often in association with an increase in the fluid portion of the dropping. Green colored urate are suggestive of liver disease or hemolysis, this discoloration is the result of increase excretion of biliverdin (biliverdinuria) which is the bile pigment in birds *(2)*. Chicks of all treated gps. showed greenish diarrhea at 32 days old and persisted 6 days in gp. 2 with anorexia, lameness, depression, 7 days in gp. 3 with nervous

manifestation (muscular tremors, torticollis, wing and leg paralysis, opisthotonous and ataxia), loss of appetite and depression and along the experimental period in gps. 4 and 5 with weakness, ruffled feather and depression in gp. 4 and consumption of the letter, ruffled feather, depression and loss of body weight in gp. 5.

The hemogram in the present study (Table 1) revealed highly significant decrease in RBCs count, Hb concentration and PCV in gp. 2 with highly significant increase in MCV and non significant change in MCHC producing macrocytic normochromic anemia at 32 days old and macrocytic hypochromic anemia at 39 days old which denote hemolytic anemia in *Salmonella gallinarum* infected gp., disappeared at 46 days old. Septicemic salmonellosis resulting in hemolysis ***(18,19)***. The hemolytic anemia can be explained by erythrophagocytosis as a result of erythrocytic sensitization by *Salmonella gallinarum* antigen which in turn stimulate the reticuloendothelial system to destruct these sensitized erythrocytes ***(20).***

Group 3 which infected with NDV showed significant decrease in RBCs count, Hb concentration and PCV with significant increase in MCV and non significant change in MCHC producing macrocytic normochromic anemia at 32 and 39 days old indicating hemolytic anemia disappeared at 46 days old. The hemolysis of RBCs caused by NDV was due to attachment of the virus at the receptor site during replication, followed by fusion of the virus membrane with the cell membrane which may result in fusion of two or more cells and the rigid membrane of the RBCs usually results in lysis from the virus membrane fusion ***(5).*** On the other hand, it has been observed an increase in erythrocytic count in 3 days old chicks and white leghorn hens infected with ND ***(21)***. The difference in the age and species may play a role.

Group 4 treated with lead acetate showed significant decrease in RBCs count, Hb concentration and PCV with significant increase in MCV and non significant change in MCHC producing macrocytic normochromic anemia at 32 and 46 days old and normocytic normochromic anemia at 39 days old. The hematological examination of one day old chicks fed diet containing lead by dose 200 and 400 ppm revealed significant decrease in hemoglobin concentration, packed cell volume and RBCs count ***(22)*** and hemolytic anemia in birds resulted from toxicities and hypochromacia could be seen in lead toxicities ***(23).*** The anemia caused by lead is essentially explained by 2 major mechanisms. One of them includes diminishing red blood cell survival caused by increased mechanical fragility of the red blood cell membrane ***(24, 25 and 26).*** Another mechanism include interfering with erythropoiesis through impairment of heme and globin biosynthesis ***(8).***

Group 5 subjected to starvation showed significant decrease in RBCs count, Hb concentration and PCV with significant increase in MCV and non significant change in MCHC producing macrocytic normochromic anemia at 32 and 39 days old, then changed to normocytic hypochromic anemia at 46 days old which indicated hemolytic anemia and later on non regenerative anemia. The macrocytic normochromic anemia occurred in birds with food restriction ***(23).*** The hemolytic anemia in this group may be contributed to increase the osmotic fragility of red blood cell membrane. In addition to this, protein is important structural component of the erythrocyte's plasma membrane, contributing to its strength, flexibility and elasticity. Also amino acid chains form hemoglobin. Decrease protein and amino acids supplementation, erythrocyte production decrease and the life span of the cells that are produced may be shorted because of defects and hemolytic anemia occurs ***(27).*** While, the non regenerative or depression anemia in birds occurred due to nutritional deficiencies and starvation and added that starvation may lead to chronic hemolytic regenerative anemia ***(19).***

Table 1. Hemogram (mean values ±S.E) of chickens in all groups at different experimental periods (n = 5).

Days and groups		RBCs $X10^6/\mu l$	Hb gm%	PCV %	MCV fl	MCHC gm/dl	Osmotic fragility test		T.L.C. $x10^3/\mu l$
							Initial lysis %	Complete lysis %	
32 days old	(1)	3.65±0.09^{a}	12.24±0.43^{a}	37.48±0.45^{a}	102.70±0.24^{c}	32.86±1.20	0.50±0.04^{c}	0.27±0.01^{ab}	21.00±0.63^{b}
	(2)	1.55±0.07^{c}	8.10±0.21^{c}	26.00±0.63^{c}	187.46±22.68^{a}	31.20±0.61	0.53±0.02^{bc}	0.24±0.01^{b}	26.40±0.81^{a}
	(3)	1.74±0.16^{c}	9.18±0.41^{c}	28.20±1.53^{c}	165.80±9.07^{ab}	32.60±2.56	0.51±0.04^{c}	0.21±0.02^{b}	18.80±1.36^{c}
	(4)	2.15±0.08^{b}	10.62±0.36^{b}	32.00±1.52^{b}	149.74±7.66^{b}	33.66±2.35	0.61±0.02^{ab}	0.32±0.03^{a}	26.00±2.17^{a}
	(5)	2.11±0.04^{b}	9.10±0.31^{c}	32.80±0.80^{b}	155.20±3.71^{ab}	27.88±0.98	0.64±0.02^{a}	0.33±0.03^{a}	18.20±1.02^{c}
39 days old	(1)	3.62±0.07^{a}	12.50±0.23^{a}	36.82±0.38^{a}	101.84±2.81^{c}	33.96±0.74^{ab}	0.45±0.06^{b}	0.20±0.04^{b}	21.00±0.63^{b}
	(2)	2.10±0.14^{b}	8.08±0.18^{d}	28.96±1.49^{bc}	140.28±11.55^{ab}	27.99±1.56^{c}	0.49±0.04^{b}	0.20±0.06^{b}	26.40±0.81^{a}
	(3)	2.25±0.32^{b}	10.56±0.31^{bc}	31.88±1.53^{b}	140.68±8.43^{ab}	32.00±1.99^{b}	0.56±0.04^{ab}	0.22±0.01^{b}	18.80±1.36^{c}
	(4)	2.08±0.15^{b}	9.56±0.34^{c}	26.20±1.63^{c}	129.44±14.40^{bc}	36.90±1.44^{a}	0.63±0.02^{a}	0.29±0.03^{a}	26.00±2.17^{a}
	(5)	1.57±0.14^{c}	8.11±0.57^{d}	25.36±1.53^{c}	168.08±20.09^{a}	31.00±2.11^{b}	0.62±0.02^{a}	0.28±0.03^{a}	18.20±1.02^{c}
46 days old	(1)	3.72±0.09^{a}	12.48±0.29^{a}	36.54±0.53^{a}	98.61±3.65^{b}	34.18±1.03^{a}	0.52±0.0.02^{b}	0.32±0.01^{ab}	21.20±0.80^{c}
	(2)	3.52±0.17^{a}	12.20±0.32^{a}	34.40±1.33^{a}	99.10±7.86^{b}	35.87±2.22^{a}	0.54±0.02^{b}	0.26±0.01^{b}	19.80±0.58^{cd}
	(3)	3.32±0.17^{a}	12.76±0.17^{a}	35.00±0.45^{a}	106.53±6.42^{b}	36.46±0.47^{a}	0.49±0.02^{b}	0.26±0.02^{b}	19.80±0.58^{cd}
	(4)	1.53±0.21^{c}	7.96±0.35^{b}	23.80±0.58^{c}	176.60±23.26^{a}	33.80±1.50^{a}	0.60±0.02^{a}	0.60±0.02^{a}	29.20±1.02^{a}
	(5)	2.13±0.05^{b}	7.22±0.07^{b}	28.90±1.27^{b}	136.04±6.09^{b}	25.38±1.04^{b}	0.62±0.02^{a}	0.62±0.02^{a}	18.40±0.51^{d}

(1) control gp., (2) Salmonellosis, (3) ND, (4) lead poisoning and (5) Starvation.
Columns at the same period with different letters are significantly different from control gp.

Blood films of lead treated gp. (Figs. 1 and 2) showed basophilic stippling hypochromacia, anochromacia, eccentric nucleus, immature forms of RBCs, anisocytosis and poikilocytosis, These results were previously mentioned by several investigators ***(5, 23 and 25).*** Starvation stressed gp. (Fig. 3 and 4) showed red cells with misshaped nuclei, anisocytosis, poikilocytosis, hypochromacia and anochromacia. Similar finding were recorded previously ***(23).*** These abnormalities may be contributed to increase fragility of the red cells membrane ***(26).***

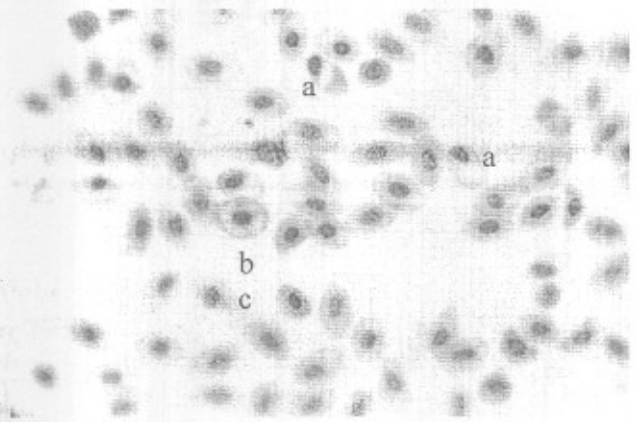

Fig. (1): Blood film of lead-treated chicken at the age of 46 days old showing eccentric nucleus(a), basophilic stippling(b) and poikilocytosis(c),. Giemsa ×1000.

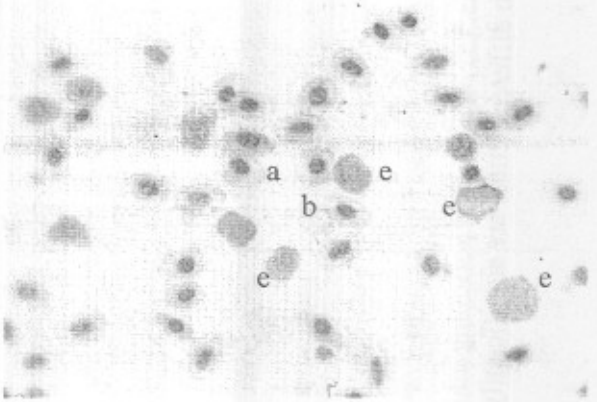

Fig. (2): Blood film of lead-treated chicken at the age of 46 days old showing anisocytosis(a), poikilocytosis(b), hypochromasia(c), anochromasia(d) and immature forms of RBCs(e), Giemsa ×1000.

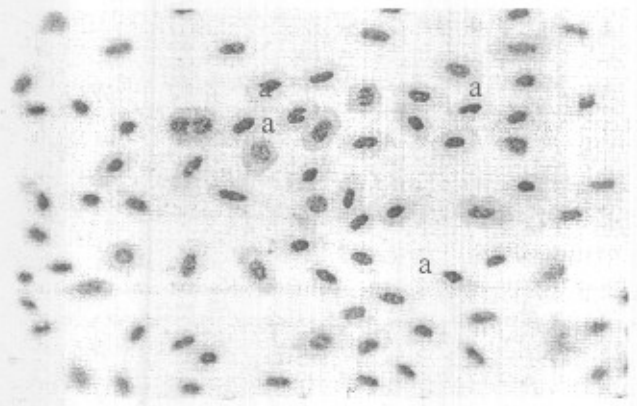

Fig. (3): Blood film of starvation stressed chicken at the age of 46 days old showing red cells with misshaped nuclei(a), Giemsa ×1000.

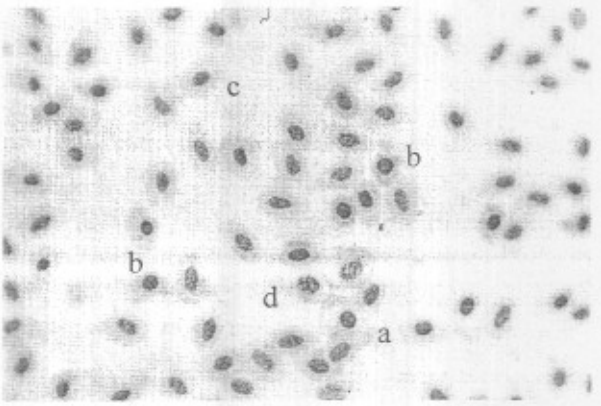

Fig. (4): Blood film of starvation stressed chicken at the age of 46 days old showing anisocytosis(a), poikilocytosis(b), hypochromasia(c) and anochromasia(d), Giemsa ×1000.

The osmotic fragility test is a measure of the resistance of erythrocytes to hemolysis by osmotic stress where the red cells exposed to decreasing strengths of hypotonic saline solutions and measuring the degree of hemolysis *(28)*. The fragilogram in the present study showed non significant changes in the osmotic fragility of red blood cell membrane in both gps. 2 and 3. Lead treated gp. showed significant increase in the osmotic fragility of RBCs membrane along the experimental periods. These results were previously reported *(24-26)* which contributed this to the inhibition of Na^+ and K^+ dependant ATPases. Gp. 5 subjected to starvation showed highly significant increase in RBCs osmotic fragility along the experimental periods. This result may be contributed to the abnormally high level of cholesterol which lead to distortions in the red blood cell shape, deformability of the cells is reduced and they destroyed in the spleen *(28)*, as approximately 50% of the red blood cell membrane is made up of cholesterol that is in equilibrium with unesterified cholesterol in the plasma, so the cholesterol content of the membrane is influenced by plasma cholesterol *(29)*. Moreover, In macrocytes and poikilocytes, there are occasional ranges of increased osmotic fragility *(30)*, and these abnormalities where proved in this group in the present study.

Leukogram in case of salmonellosis revealed leukocytosis at 32 and 39 days old. Birds respond to bacterial infection by leukocytosis *(18,19)*. The same results were previously obtained *(31)* at 2 months old chickens inoculated orally with ***Salmonella gallinarum*** 0.1 ml bacterial suspension contained $5x10^7$ viable bacteria. and *(23)* In addition to the previous explanations leukocytosis and monocytosis may occur with hemolytic anemia *(32)*. In ND infected gp. the leukogram showed leucopenia at 32 and 39 days old. These results were previously obtained *(8,33)* and indicated that leukopenia occurs with viremia (usually a mild leukopenia due to a decrease in lymphocyte). Leukopenia have been reported in pet birds that have viral infection and contributed that to the endogenous corticosterone release with temporary lymphocytes redistribution, temporary trapping of recirculating lymphocytes within lymphoid tissue to promote the antigen contact and direct destruction of lymphoid tissue *(10)*. Severe viral infection may result in leukopenia and attributed this to decrease production or increase consumption of the cells *(34)*. The same results were previously obtained *(2,19)* The leukogram of birds exposed to lead poisoning revealed leukocytosis at 32, 39 and 46 days old. The present results were consistent with previous work *(22,35)*. The changes in the total leukocytic count in this case may be contributed to the inflammatory conditions which occurred in the internal organs and intestinal tract. The starved chickens showed leukopenia along the experimental periods. The same result were previously obtained by *(36)* who ascribed this to stress reaction.

Regarding the biochemical changes, (Table 2) showed hypoglycemia and hypocholesterolemia at 32 and 39 days old in gps. (2 and 3). These results are in accordance that observed after inoculation of 6 months age chickens I/M with ***Salmonella typhimurium*** 1.0 ml contained 10^9 CFU/ml *(37)*, Infection of 29 – 40 days old chicks with VVNDV I/M 0.2 ml/bird (10^6 EID_{50} /ml) revealed significant decrease in serum cholesterol level at the 3rd day post infection *(33)*. A significant decrease in serum cholesterol level of chickens inoculated with 0.1 ml (10^6 EID_{50} / ml) NDV velogenic strain was observed 5 days post infection *(38)*. The hypoglycemia and hypocholesterolemia may be contributed to toxemia caused by bacterial and viral septicemias and liver disease, due to decrease intestinal absorption as a result of enteritis or due to decrease hepatogenic activity *(8,19)*. This may be also explained by kidney syndrome due to degeneration of renal tubules *(19,39)*.

Table 2. Some biochemical parameters(mean values ±S.E) of chickens in all groups at different experimental periods (n = 5).

Days and groups		Glucose mg/dl	Cholesterol mg/dl	ALT U/L	AP IU/L	CK U/L	Uric acid mg/dl
32 days old	(1)	272.80±7.57^{a}	133.40±3.94^{b}	7.93±0.19^{b}	157.23±3.39^{a}	256.92±17.82^{b}	7.09±0.34^{b}
	(2)	208.80±13.26^{b}	111.00±5.11^{c}	11.09±0.67^{a}	129.12±6.21^{bc}	480.44±36.15^{a}	9.07±0.57^{a}
	(3)	206.20±7.52^{b}	96.40±9.17^{c}	11.77±0.24^{a}	122.62±3.54^{c}	246.12±31.85^{b}	7.90±0.46^{ab}
	(4)	208.80±10.89^{b}	95.90±5.21^{c}	9.01±0.13^{b}	122.02±6.13^{c}	482.30±34.04^{a}	8.28±0.43^{ab}
	(5)	191.60±2.42^{b}	202.80±8.80^{a}	7.82±0.51^{b}	140.61±5.63^{b}	271.38±29.15^{b}	9.09±0.32^{a}
39 days old	(1)	271.20±7.13^{a}	136.20±3.20^{a}	8.00±0.12^{b}	153.60±6.66^{a}	293.02±31.39^{b}	7.43±0.38^{b}
	(2)	190.80±6.68^{bc}	102.60±9.50^{b}	12.35±1.13^{a}	120.84±3.81^{b}	450.46±38.10^{a}	9.19±0.44^{a}
	(3)	177.20±7.41^{bc}	100.40±6.80^{b}	12.26±1.09^{a}	87.48±7.97^{c}	310.44±22.91^{b}	8.96±0.30^{a}
	(4)	201.40±12.89^{b}	107.60±5.45^{b}	14.66±1.20^{a}	127.12±3.29^{b}	520.86±45.87^{a}	8.41±0.53^{ab}
	(5)	166.20±6.77^{c}	153.20±9.50^{a}	6.52±0.17^{b}	132.17±4.29^{b}	343.72±15.57^{b}	8.71±0.28^{a}
46 days old	(1)	263.00±5.63^{a}	148.60±4.91^{a}	7.91±0.15^{b}	154.17±3.91^{a}	270.32±32.23^{b}	7.52±0.35^{b}
	(2)	256.20±6.14^{a}	149.40±6.79^{a}	8.25±0.50^{ab}	150.66±7.06^{a}	299.44±37.35^{b}	7.41±0.45^{b}
	(3)	250.00±12.39^{a}	147.80±7.72^{a}	8.59±0.37^{ab}	144.88±10.56^{a}	272.74±32.44^{b}	7.72±0.22^{b}
	(4)	197.80±7.51^{b}	104.60±5.99^{b}	9.59±0.75^{a}	126.14±4.30^{b}	536.54±70.95^{a}	8.51±0.32^{ab}
	(5)	202.00±74.09^{b}	118.60±3.50^{b}	7.50±0.32^{b}	131.72±4.34^{b}	294.86±37.35^{b}	9.01±0.92^{a}

(1) control gp., **(2)** Salmonellosis, **(3)** ND, **(4)** lead poisoning and **(5)** Starvation.
Columns at the same period with different letters are significantly different from control gp.

Gp. 4 exposed to lead toxicity revealed hypoglycemia and hypocholesterolemia along the experimental periods. Hypoglycemia and hypocholesterolemia were recorded in birds exposed to lead poisoning may be contributed to liver and kidney damage caused by lead toxicity *(22,35)*. It may be also contributed to malabsorption due to diarrhea and enteritis. Geese with lead poisoning have slight hyperglycemia *(8)* and also Amazon parrot with lead toxicosis *(19)*. This may be related to species difference.

The starved birds showed hypoglycemia along the experimental periods. Hypoglycemia in birds may occur with starvation, malnutrition which lead to malabsorption of glucose from degenerated intestinal brush borders or malabsorption due to degenerated renal tubules *(8)*. The hypoglycemia occurred during starvation and blood glucose concentration of starved chicks started to decrease on the third day of starvation *(40)*. Birds subjected to starvation showed considerable stability even increasing on the blood glucose level for the first 72 hrs. of starvation which derived from breakdown of fat and proteins primarily from muscle tissue through gluconeogenesis *(2)*. The blood glucose level decreases when body stores were depleted and gluconeogenesis is impaired *(40)*. The hypercholesterolemia at 32 days old in starved gp. was recorded also in starvation of young broiler chicks for 48 hrs *(41)* and in fasting of 70 weeks old hens for 10 days *(2,42)*. This elevation due to the mobilization of body stores during starvation *(34)* and when the body stores were depleted the blood cholesterol level showed hypocholesterolemia at 46 days old *(43)*.

The increase in ALT activity in gps. (2,3) is related to the release of the enzyme by the degenerating liver cell *(44)*. This explanation was ensured by *(45)* who reported elevation in serum ALT activity in raptors, chickens and ducks with hepatic insult. The increase in ALT activity can be related to liver damage and other tissue damage which lead to alteration in the cell membrane permeability, this allows the escape of the enzyme into serum in abnormal high levels *(46)*. Infection I/M of one week old balady chicks with 0.25 ml broth containing $10^{3.3}$ CFU/ml *Salmonella enteritidis* also showed the same results *(31, 47)* and *(33 and 48)* due to ND. But non significant change in ALT activity in Salmonellosis and NDV respectively were recorded *(37,38)*, this may be contributed to species difference, strain of the infecting agent, age of the birds, route of administration and the degree of tissue damage.

Gp. 4 showed gradual increase in ALT activity by increasing the duration of lead poisoning which may be also related to the degree of tissue damage. Feeding 18 weeks old hens on diets containing 1000 or 1500 ppm lead chloride till 33 weeks old recorded significant increase in plasma ALT activity at the end of the experiment *(35,49)*. Gp. 5 showed non significant changes in ALT activity. The same result was previously obtained in a study conducted with hybrid large white male turkeys 6 weeks old by 16 hrs. starvation *(50)*.

The AP activity showed highly significant decrease in all gps. along the experimental periods in starved and lead treated birds, but returned to its normal level at 46 days old in case of salmonellosis and NDV infection. The main cause of serum AP reduction is the damage in the intestine *(51)* as the predominant AP isoenzyme in the plasma originates in the gut *(52)* and the fall in AP activity in the affected chicken gut is reflected by similar decrease in the serum level of this enzyme *(53)*. Moreover the reduction in the AP activity in the intestine could be due to the decrease in food consumption and the intestinal activity *(51)* or may be due to intestinal destruction and subsequent hemorrhages and release of large quantities of the enzyme in the intestine which may be not absorbed but passed out in the faeces *(54)*. In chickens the intestinal AP enzyme makes the largest contribution of plasma AP activity and is affected by intestinal disturbances, more over ducks with lead poisoning have lowered AP values because lead inhibits AP activity *(8)*.

Serum CK activity showed highly significant increase in case of salmonellosis and lead poisoning which returned to its normal level at 46 days old in gp. 2 but persisted along the experimental period in gp. 4. Gps. 3 and 5 which showed non significant changes. Elevated CK activity in avian serum may be seen with lead toxicosis and bacterial septicemia ***(8,19,37,55).*** This may be due to liver damage, myocardial infarction and other tissue damage which lead to alteration in cell membrane permeability and allows the escape of the enzyme in the serum in abnormal high amount ***(46).*** I/M injection usually does not elevate the serum CK activity unless the material is highly irritating ***(8)***. But CK activity increase in an outbreak of ND in birds over 7 weeks old ***(56)*** and decrease with *Salmonella typhimurium* infected pigeon ***(57)*** which may be related to the difference in species, age of birds, dose and strain of the microbe.

Hyperuricemia was observed in gp. 2 at 32 and 39 days old and in gp. 3 at 39 days old, and in gp. 5 at all periods. Non significant increase in uric acid in gp. 4 was noticed. Hyperuricemia in birds occur with starvation and renal disease due to decrease the rate of tubular excretion, and poor nutritional status which increase uric acid production due to catabolism of body tissues ***(8,34).*** In addition to this serum uric acid increases in case of toxemia from bacterial and viral infection ***(19).*** Hyperuricemia in salmonellosis was previously reported ***(39,47,55).*** Non significant change in serum uric acid was recorded in 6 month age chickens inoculated with *Salmonella typhimurium* ***(37)***. In case of ND increases of uric acid level was observed by several investigators ***(33,38,56).***

Significant increase in plasma uric acid in domestic fowl was recorded after repeated administration of lead acetate (200 mg/kg body weight) daily for 40 days ***(8).*** The difference from out finding may be due to difference in the dose and duration.The same result was previously obtained in case of starvation ***(50).*** No change in serum uric acid level was observed in 6 week old turkeys staved for 16 hrs ***(50).*** Also plasma uric acid did not fluctuate during 48 hrs. starvation of adult pekin ***(60).***

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الملخص العربى

دراسات باثولوجية إكلينيكية على الاسهال الاخضر فى الدجاج

فخرى سليمان سالم، أمانى احمد محمد عبد اللة، سحر سمير عبد الحميد*، دعاء إبراهيم أحمد مصطفى*

قسم الباثولوجيا الإكلينيكية-كلية الطب البيطرى-جامعة الزقازيق و معهد بحوث صحة الحيوان-فرع الزقازيق*

أجريت هذه الدراسة لمعرفة التغيرات فى خلايا الدم، والمؤشرات الكيميائية الحيوية المصاحبة للإسهال الأخضر فى الدجاج وقد استخدم لهذا الغرض عدد ١٠٠ فرخ تسمين عمر يوم واحد من نوع هبرد قسمت إلى خمس مجموعات متساوية تم معاملتها كالتالي، المجموعة الأولى: تركت بدون معاملة كمجموعة ضابطة، المجموعة الثانية: تم عمل عدوى تجريبية بميكروب (السالمونيلا جالينيرم) عند عمر ٢٨ يوم ، المجموعة الثالثة: تم عمل عدوى تجريبية بفيروس النيوكاسل (عترة ضارية) عند عمر ٢٨ يوم ، المجموعة الرابعة: تم معاملتها بأسيتات الرصاص بمعدل ٤٠ مجم/ كجم عليقة، من عمر أسبوع وحتى نهاية التجربة أما المجموعة الخامسة: تم تصويمها من عمر ٢٩ يوم.

وقد لوحظ إصابة الكتاكيت بالإسهال الأخضر فى الأربع مجموعات التى تم معاملتها. وقد أظهرت النتائج عن وجود أنيميا ناتجة عن تحلل كرات الدم فى الأربع مجموعات عند عمر ٣٢ يوم استمرت حتى عمر ٣٩ يوم فى الكتاكيت المصابة بالسالمونيلا والنيوكاسل بينما استمرت حتى نهاية التجربة فى المجموعتين المعاملة بأسيتات الرصاص و التى تم تجويعها. بالإضافة إلى ذلك لوحظ تشوهات فى حجم و شكل كرات الدم الحمراء، كما لوحظ زيادة معنوية فى قابلية غشاء خلايا الدم الحمراء للتكسير (الهشهشة) فى المجموعتين الرابعة والخامسة خلال التجربة. كما أظهر العد الكلى لخلايا الدم البيضاء زيادة معنوية فى

المجموعة الثانية عند عمر ٣٢، ٣٩ يوم و فى المجموعة الرابعة على مدى التجربة. بينما لوحظ نقص معنوى فى العدد الكلى لخلايا الدم البيضاء فى المجموعة الثالثة عند عمر ٣٢ يوم و فى المجموعة الخامسة على مدى التجربة.

وقد أثبتت النتائج نقص معنوى فى مستوى الجلوكوز والكوليستيرول فى المصل فى المجموعتين الثانية والثالثة عند عمر ٣٢ و ٣٩ يوم و فى المجموعه الرابعة على مدى التجربة. بينما أظهرت المجموعة الخامسة نقص معنوى فى مستوى الجلوكوز على مدى التجربة وزيادة معنوية فى مستوى الكوليستيرول عند عمر ٣٢ يوم. كذلك لوحظ زيادة معنوية فى نشاط انزيمي الألانين أمينوترانسفيريز والكرياتين كاينيز فى المجموعة الثانية مع نقص فى نشاط انزيم الفوسفاتيز القلوى عند عمر ٣٢، ٣٩ يوم. كما لوحظ فى المجموعة الثالثة زيادة معنوية فى نشاط الألانين أمينوترانسفيريز مع نقص معنوى فى نشاط الفوسفاتيز القلوى عند عمر ٣٢، ٣٩ يوم مع تغير غير معنوى فى نشاط الكرياتين كاينيز على مدى التجربة. بالنسبة للمجموعة الرابعة لوحظ زيادة فى نشاط انزيمى الالانين أمينوترانسفيريز والكرياتين كاينيز بزيادة مدة التجربة بينما نقص الفوسفاتيز القلوى على مدى التجربة. و قد أدى تجويع الطيور إلى قله فى نشاط الفوسفاتيز القلوى ولم يؤثر على نشاط الألانين أمينوترانسفيريز و الكرياتين كاينيز على مدى التجربة. وبقياس معدل حمض البوليك لوحظ زيادة معنوية فى المجموعات الثانيه و الثالثه و الخامسه، اختفت هذه الزيادة فى المجموعتين الثانية والثالثة عند عمر ٤٦ يوم ولكنها استمرت حتى نهاية التجربة فى المجموعة الخامسة.