

Some Biochemical and Pathological Changes in Turkey Induced By Gentamicin and Their Modulation with Vitamin E

Abo El-Fetouh, E H, Halla M Khalil* Gehan, Nabil and Soad Mekawy**

(Histopathology, Biochemistry* and Clinical Pathology** Department)

(Animal Health Research Institute (Zagazig branches))

ABSTRACT

The present study was designed to investigate the protective role of vitamin E against gentamicin toxicity. A total of 80, 4 weeks -old turkey poult were divided into 4 groups. 1st group healthy non-treated turkeys (control group), 2nd group gentamicin (5mg/kgm B.wt/day) treated, 3rd group vitamin E (10mg/kg B.wt/day) treated, 4th group treated with gentamicin plus vitamin E. Five turkeys poults from each group were sacrificed at 1st day post treatment, blood samples and Specimen from internal organ were taken for hematobiochemical and histopathological study. Five turkey poults from each group were weighted at start and 1st day post treatment for detection effect of the durgs in body weight.

The present work revealed that, gentamicin induced a significant reduction in RBCs count, Hb, PCV %, total protein, albumin, globulin and rise in AST, ALT, ALP, uric acid, and creatinine beside insignificant rise in WBCs count and body weight in turkey at 1st day post treatment. Vitamin E induce significant rise in RBCs count, Hb, PCV % total WBCs count, total protein, albumin globulin and insignificant increase in body weight, AST, ALT, ALP, uric acid, creatinine and body weight in turkey at 1st day post treatment.

Necropsy revealed swollen kidneys and liver with hemorrhagic streaks on its surface. Petechial hemorrhages present on skeletal muscles. Microscopically, kidneys showed degenerative changes and necrosis of the renal tubules. Liver showed severe vacuolar and hydropic degeneration of the hepatic parenchyma.

Oral administration of vitamin E to turkey poult gentamicin treated caused improvement in hematobiochemical parameters and pathological lesions.

From the results, it can be concluded that gentamicin is a highly toxic to turkey birds but vitamin E has potential protective effects against gentamicin toxicity.

INTRODUCTION

Aminoglycoside is a group of bactericidal antibiotics. Gentamicin is an amino-glycoside antibiotic isolated from micromonospora purpura (1). It is widely used for treatment of infections with G-ve bacteria (2). Gentamicin is an effective drug used to control many diseases in poultry, however, its clinical use is partially limited due to its toxicity (3). The mechanism of action of gentamicin involves irreversible inhibition of bacterial ribosomes and impairs protein synthesis, (4). Gentamicin toxicity results from accumulation and retention of

aminoglycoside in proximal convoluted tubular cells (5).

Vitamin E includes 8 isomers in 2 classes designatd (tocopherol & tocotrienols) (6). Vitamin E has antioxidants effect in the body (7). Antioxidat activity of vitamin E contributes to stabilization of the oxidation sensitive fatty acids in the cellular metabolism. Free radicals catalyze lipid peroxidation is a continuous process, causing damage to cellular and intracellular structures, vitamin E inhibit these processes (8). Vitamin E stimulates immune system of animals (9).

The objective of this study was to illustrate the harmful effect of gentamicin and to evaluate the possible protective effect of Vitamin E on hematological and some serum biochemical parameters in turkey poult.

MATERIAL AND METHODS

Drugs

- 1- Gentamicin (Garamycin[®]) Memphis Co. intramuscular injection. Each ml of solution contains 40-mg Gentamicin sulfate.
- 2- Vitamin E was obtained as a viscous oil of vitamin E (Alpha-Tocopherol): It is available as soft gelatin capsules (400 I.U.).

Birds and Experimental design

A total of 80, 4 weeks old mixed breed turkey poult were used in this investigation. Turkeys were floor reared in separate units along, fed on a balanced ration free from any medication and water was provided ad-libitum. Turkeys were divided into four equal groups (20 birds each). 1st group was healthy turkeys non-treated (control group). 2nd group healthy turkeys injected I. M with gentamicin (5 I.U/kg b wt/day) for 5 successive days (10), 3rd group healthy turkeys orally treated with vitamin E (10 I.U/kg b.wt /day) for 5 successive days and 4th group healthy turkeys treated with gentamicin and vitamin E for the same previous dose and period.

Blood samples

At 1st day post treatment 5 birds from each group were slaughtered and 2 blood samples were taken from each bird, 1st sample was taken in tube contain EDTA for hematological study (11), 2nd sample was taken for obtaining serum for detection of total protein (12), albumin (13), Globulin was calculated as difference between total protein and albumin, AST and ALT (14), ALP (15),

uric acid (16) and creatinine (17) were determined.

Body weight

Turkeys in all groups were weighted individually at the start of the experiment and at 1st day post treatment for calculation weight gain and feed conversion rate.

Histopathological studies

Specimens from liver and kidneys were taken from slaughtered bird then fixed in 10% neutral formalin and embedded in paraffin. Sections of 5 microns thickness stained by H&E and microscopically examined (18).

Statistical analysis

Obtained data were statistically analyzed (19).

RESULTS

The present work revealed that, gentamicin induced a significant decrease in erythrocytic count, hemoglobin, packed cell volume %, total protein, albumin, globulin and a significant increase in AST, ALT, ALP, uric acid, and creatinine beside insignificant rise in total leukocytic count and body weight in turkey at 1st day post treatment. Vitamin E induce significant increase in erythrocytic count, hemoglobin, packed cell volume%, leukocytic count, total protein, albumin, globulin and insignificant increase in body weight, AST, ALT, ALP, uric acid and creatinine in turkey at 1st day post treatment. (Tables, 1 -3)

Turkey poult treated with gentamicin only (2nd group) grossly, showed the liver and kidneys were congested, enlarged, hemorrhagic and the kidneys pale in its colour. The heart of most cases undergoes pericarditis. Microscopically, liver exhibited various degenerative changes with severe congestion of the portal blood vessels and hepatic sinusoids and portal leukocytic infiltration (Fig, 1). The hepatic cells showed clear large necrotic area

beside heterophils and lymphocytes (Fig, 2). Other cases the hepatic cells suffered from telangiectiasis (Fig, 3). The renal tubules showed intense necrotic changes in the tubular epithelium with congested capillaries (Fig, 4), other cases undergo hemorrhages, necrosis and leukocytic infiltration were seen (Fig, 5). Vacuolar and hydropic degeneration were seen in some cases (Fig, 6). The heart exhibited severe pericarditis that represented by heterophilic infiltration, congestion of some blood vessels and edema (Fig, 7). Other cases

of the cardiac tissue suffered from edema and lymphocytic infiltration (Fig, 8). Turkey poult treated with gentamicin and vitamin E (4th group) microscopically, the hepatic cells exhibited mild coagulative necrosis (Fig 9) and apparently normal hepatic parenchyma (Fig 10) Kidneys of turkey poult treated with gentamicin and vitamin E (4th group) with mild degenerative changes (Fig 11). Heart of turkey poult treated with gentamicin and vitamin E (4th group) exhibited mild pericarditis (Fig 12) and apparently normal myocardial muscles (Fig 13)

Table 1. Effect of gentamicin and vitamin E on erythrogram of broiler chickens (n=5)

Group	Parameters	Erythrogram			WBCs (106/c.mm)
		RBCs (106/c.mm)	HB (g m %)	PCV%	
Control		5.37±0.40	10.54±0.78	30.27±0.80	17,48 ±0,69
Gentamicin		4.30±0.21*	7.69±0.64*	26.11±0.48*	18,04±0,87
Vitamin E.		7.02±0.57*	13.09±0.50*	33.13±0.68*	19,89 ±0,48*
Genta & Vit.E.		4.18±0.84	11.05±0.83	30.02±0.79	18,13 ±0,80

*Significant at $P \leq 0.05$

** Significant at $P \leq 0.01$

Table 2. Effect of gentamicin and vitamin E on protein picture of broiler chickens (n=5)

Group	Parameters	T.P. gm/dl	Albumin gm/dl	Globulin (gm/dl)	A/G Ratio
Gentamicin		2.78± 0.44*	1.51± 0.19*	1.27± 0.18*	1.14± 0.14
Vitamin E.		6.29± 0.31**	3.43± 0.35*	2.86± 0.26*	1.17± 0.19
Genta & Vit.E.		4.59± 0.47	2.54± 0.38	2.09± 0.30	1.19± 0.24

Table 3. Effect of gentamicin and vitamin E on biochemical parameters of broiler chickens (n=5)

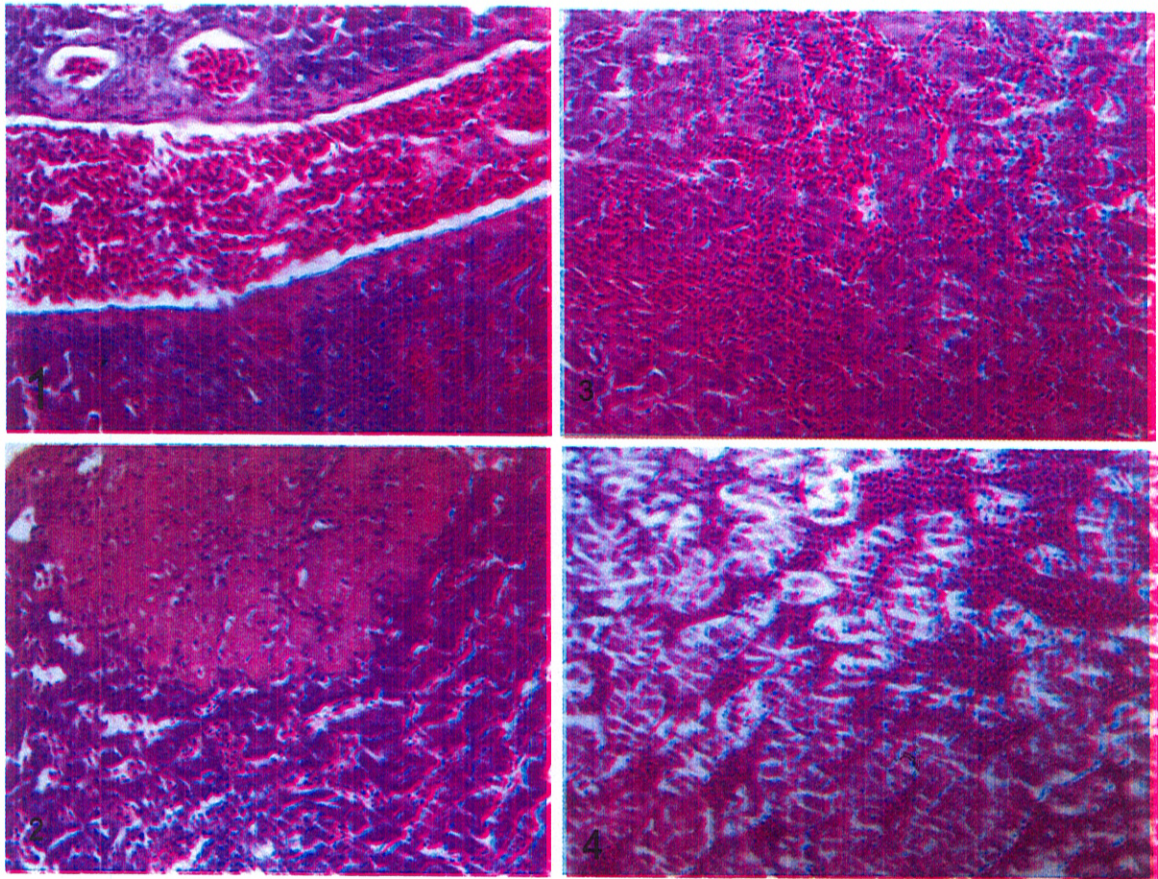
Group	Parameters	Liver enzymes			Kidney function(mg/dl)	
		AST(U/L)	ALT(U/L)	ALP(U/L)	Uric acid	Creatinine
Control		83.17 ± 1.63	26.68 ± 0.82	119.30±1.30	4.98±0.60	1.79±0.30
Gentamicin		89.64 ± 1.41	30.16 ± 0.96*	122.43±1.42*	6.59±0.36*	2.61±0.15*
Vitamin E.		84.05 ± 1.35	27.02 ± 0.79	120.18±1.80	5.05±0.28	1.85±0.18
Genta & Vit.E.		84.37 ± 1.55	27.62 ± 0.98	120.38±1.95	5.02±0.48	1.82±0.19

*Significant at $P \leq 0.05$

** Significant at $P \leq 0.01$

Table 4. Effect of gentamicin and vitamin E on body weight pre (n=5).

Body weight		Control group	Gentamicin	Vitamin E.	Genta & Vit.E.
Initial	gm at 28 day of age	869.49±7.39	866.04±3.59	870.16±6.40	865.03±9.60
Final	gm at 1 st day post treatment	910.46±8.92	912.18±7.50	915.39±5.58	916.17±7.22
Gain	gm	40.97◇0.35	46.14◇0.49	45.23◇0.63	51.14◇0.53



- Fig. 1. Photomicrograph of the liver of turkey poult treated with gentamicin only (2nd group) showing severe congestion and leukocytic infiltration (H&EX300)
- Fig.2. Photomicrograph of the liver of turkey poult treated with gentamicin only (2nd group) showing large area of coagulative necrotic and leukocytic infiltration (H&EX300)
- Fig. 3. Photomicrograph of the liver of turkey poult treated with gentamicin only (2nd group) showing telangiectasis (H&EX300)
- Fig. 4. Photomicrograph of the kidneys of turkey poult treated with gentamicin only (2nd group) showing severe necrosis of the tubular epithelium (H&EX300)

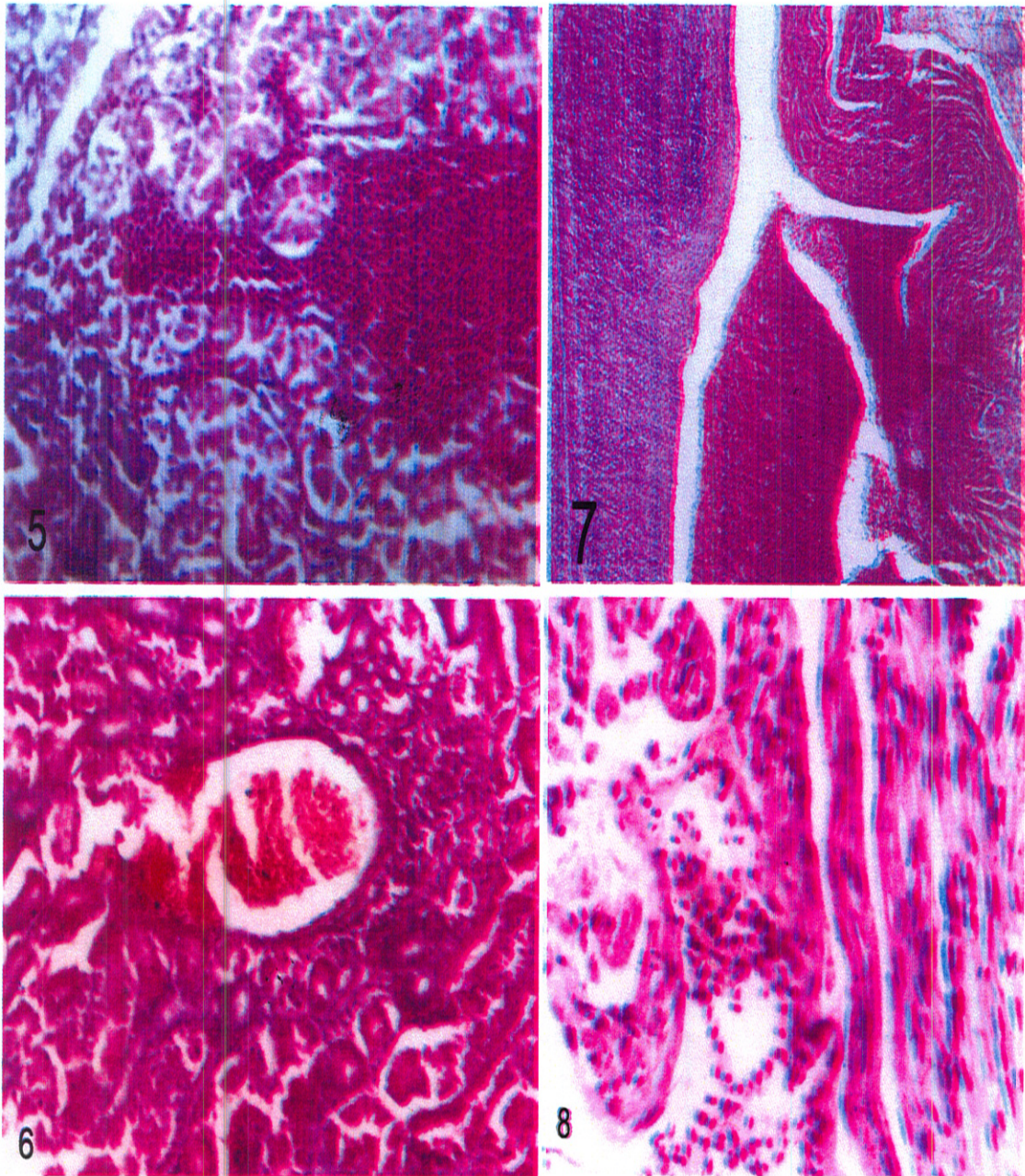
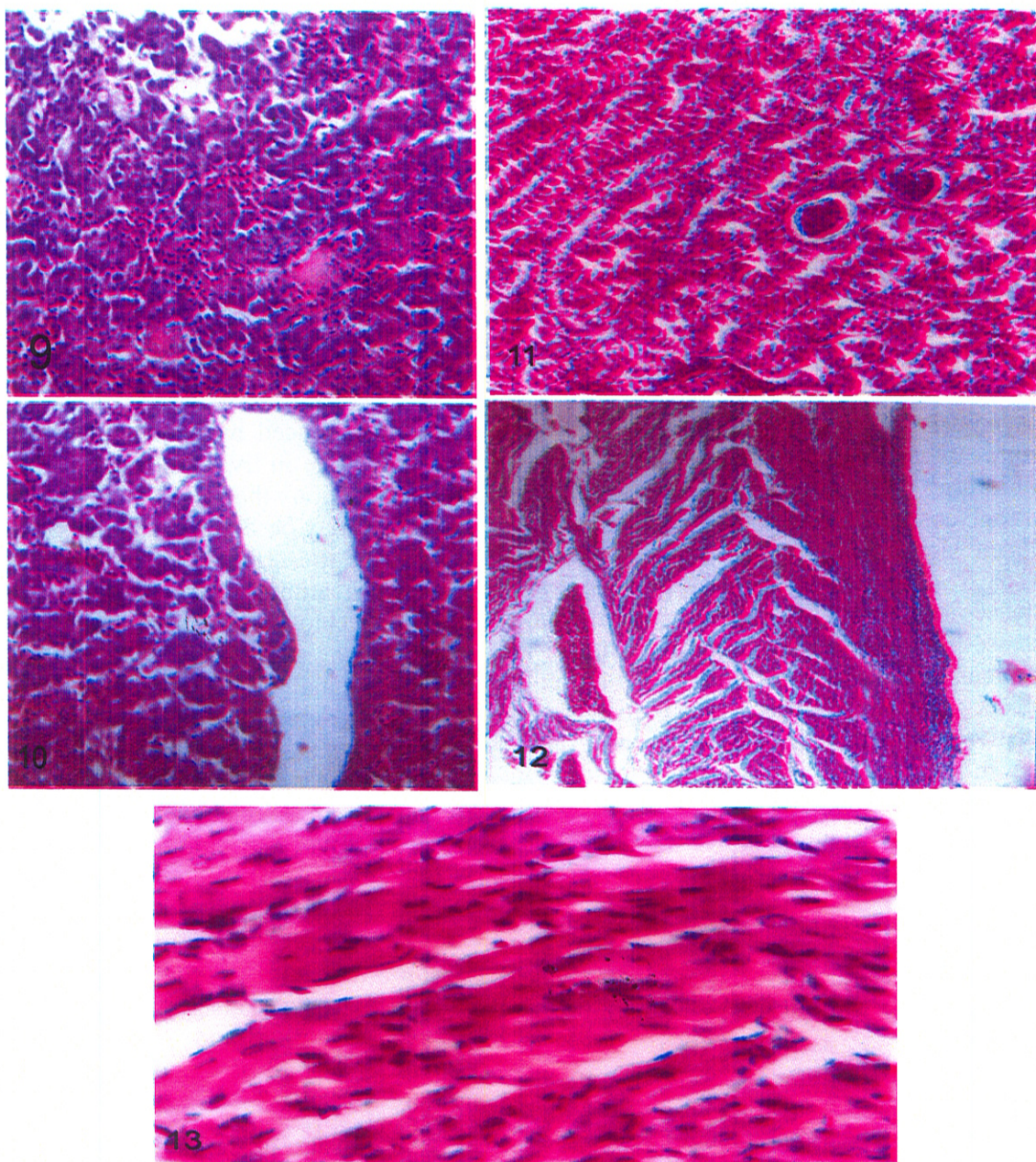


Fig.5. Photomicrograph of the kidneys of turkey poult treated with gentamicin only (2nd group) showing necrosis, hemorrhages and focal leukocytic aggregation among the renal tubules (H&EX 300)

Fig.6. Photomicrograph of the kidneys of turkey poult treated with gentamicin only (2nd group) showing hydropic degeneration in the epithelium lining the renal tubules (H&EX300)

Fig.7. Photomicrograph of the heart of turkey poult treated with gentamicin only (2nd group) showing severe pericarditis (H&EX300)

Fig.8. Photomicrograph of the heart of turkey poult treated with gentamicin only (2nd group) showing edema and lymphocytic infiltration (H&EX1200)



- Fig.9. Photomicrograph of the liver of turkey poults treated with gentamicin and vitamin E (4th group) showing mild coagulative necrosis (H&EX300)
- Fig.10. Photomicrograph of the liver of turkey poults treated with gentamicin and vitamin E (4th group) showing apparently normal hepatic parenchyma (H&EX300)
- Fig.11. Photomicrograph of the kidneys of turkey poults treated with gentamicin and vitamin E (4th group) showing mild degenerative changes (H&EX300)
- Fig.12. Photomicrograph of the heart of turkey poults treated with gentamicin and vitamin E (4th group) showing mild pericarditis (H&EX1200)
- Fig.13. Photomicrograph of the heart of turkey poults treated with gentamicin and vitamin E (4th group) showing apparently normal myocardial muscles (H&EX1200)

DISCUSSION

Gentamicin induced significant decrease in total RBCs count, Hb, PCV, beside in significant increase in WBCs count but vitamin E induce a significant increase in RBCs, Hb, PCV and WBCs. Anemia induced by gentamicin may be due to erythropoietin deficiency following injury to the kidney by gentamicin, site of erythropoietin production (20). Also gentamicin can chelate iron and decreased iron level may interfere with Hb biosynthesis (21). Same changes in blood picture were induced by aminoglycoside antibiotics in chickens (22,23). Another aminoglycoside antibiotic (netilmicine): induced significant decrease in RBCs count, Hb, PCV (24). Vitamin E induced significant increase in RBCs, Hb, PCV in rabbits (25). Increase in hemogram post vitamin E treatment may be due to that vitamin E protects the biological membrane from oxidative damage with consequent beneficial effect in preservation and keeping erythrocytes from any hazard or damage effect with consequent increase in erythrocyte (26).

In the present study, gentamicin induces a significant reduction in total protein, albumin and globulin in turkey. Meanwhile, vitamin E induces significant elevation in serum total protein, albumin and globulin. This results in agreement with the results observed (27-28) in chicken. Another aminoglycoside (netilmicine): induces significant decrease in total protein in rats (29). Reduction in albumin post gentamicin treatment may be due to destructive effect of gentamicin on liver cells producing albumin (31). Similar trend was reported that vitamin E induced significant rise in serum total protein, albumin and globulin in chicken. Serum protein picture post vitamin E treatment may be due to increased absorption of globulin and essential protein digested products and improve protein synthesis in liver (32).

In the present work, gentamicin induced significant increase in AST, ALT, ALP, uric acid and creatinine in turkey poults but vitamin E induced insignificant increase in

AST, ALT, ALP, uric acid and creatinine. These results come in harmony with the results turkeys (33) and in broilers (34). Gentamicin is rapidly excreted by glomerular filtration and the small % is reabsorbed by the epithelial cells of the proximal tubuls, lead to accumulation within the renal cortex. Accumulation of the reabsorbed drug leads to nephrotoxicity (35). Therefore the nephrotoxic effect of aminoglycosides due to their marked accumulation and retention in proximal tubular cells (2). Rise of liver enzymes, uric acid and creatinine induced by gentamicin was mainly due to damage in liver and kidney (30). In the same direction the rise in uric acid and creatinine may be due to nephrotoxic effect of gentamicin (36). Rise in liver enzymes, uric acid and creatinine post gentamicin treatment may be due to degenerative changes in liver and tubular necrosis in the kidneys (37). Gentamicin induces desquamation and atrophy of tubular epithelial cells in rats (38). Moreover, (39) stated gentamicin induce severe tubular necrosis in rats associated with rise serum urea, creatinine. Similar structural changes in liver and kidney were observed by (28). Gentamicin induces acute tubular necrosis in kidney and liver fatty change, vacuolar degeneration, necrotic areas and cellular infiltration around portal triads. Vitamin E induce insignificant rise in liver enzymes, creatinine and uric acid in chicken (40). Also, vitamin E induce insignificant rise in liver enzymes (41).

The insignificant increase in the body weight and weight gain in gentamicin and vitamin E. in turkeys run parallel with recorded that antimicrobials produced an increase in the growth rate in growing chicks, increase body weight gain with improved feed conversion through inhibiting pathogenic organisms which damage gut epithelium impairing food absorption, inhibiting pathogenic organisms which compete for growth factors in the gut and inhibiting organisms producing toxic substances affecting growth (42). Also vitamin E induce improving the efficiency of digestion and increase in body weight in rabbits (43).

Gross alterations observed in our study were in close resemblance to those reported in chickens (27,34). Lesions arising post gentamicin treatment may be due to drug accumulation in the proximal convoluted tubules, leading to loss of brush border integrity and hepatic degenerative changes (45). Pathological changes in the liver post gentamicin treatment are parallel to previous observation (46).

The present data revealed that vitamin E ameliorate toxic effect of gentamicin and improved in hematobiochemical parameters and pathological lesion. Same results were reported in rats (47). In addition, it has been reported that found vitamin E induce improvement in both hepatic and renal lesion which induced by gentamicin in dogs (48). This may due to the ability of vitamin E to improve the endogenous antioxidants (49) or due to ability of vitamin E to scavenge the free radicals (50).

From the results, it can be concluded that gentamicin is a highly toxic to turkey birds but vitamin E has potential protective effects against its toxicity.

REFERENCES

1. **Karlowsky J, Zelenitsk Y and Zhanel, G (1995):** Aminoglycoside adaptive resistance Pharmacotherapy 17: 549 – 55.
2. **Mary J, Richard A, Pamel, C and Michael C (2000):** Lippincotts illustrated reviews: pharmacology". Philadelphia. New. York. Press. 314– 317.
3. **Lietman P and smith C (1983):** Aminoglycoside nephrotoxicity in human. J. Infect Dis 5, 2:284–292.
4. **Abo-Nora M, El-Katan Y and Ali M (2007):** Influence of propolis on nephrotoxicity induced by gentamicin in goats. 5thInt.,Sci. Conf. Mansoura.37-44
5. **Ali B (2002):** Effect of treatment with the medicinal plant *Rhazya stricta* decne on gentamicin nephrotoxicity in rats. Phyto medicine, 9: 385-389.
6. **Mensink R Houeligen A and Hornstra G (1999):** A vitamin E concentrate rich in tocotrienols had no effect on serum lipids, lipoproteins or platelet function in men with mildly elevated serum lipid. Am. J. Clin. Nutr. 69:213-219.
7. **Meydani M (1995):** Vitamin E. Lancet, 345: 170-175.
8. **Droke E and Loerch S (1989):** Effect of selenium and vitamin E on performance and humoral response of steers to feed lot environment. J. Ani Sci, 67:35-39
9. **Bonnette, E, Kornegay, E and Hammeberg, C (1990):** Humoral immune response of weaned pigs treated with vitamin E. J. Animal. Sci. 68:337-345
10. **Sarkozy G, Iaczay P and Horrath, E (2002):** Treatment of experimentally induced *P. multocida* infection in broiler and turkeys. J. Vet. Med. 49(3) 30-34.
11. **Jain N (1986):** Schalm's Vet Hematology 4thEd, Lea and Fibiger, Philadelphia
12. **Doumas B, Certor R, Peers T and Schaflr R (1981):** A candidat reference -method for determination of total protein in serum. Clin. Chem. (27):1642-1647
13. **Drupt, F (1974):** Colorimetric method for determination of albumin .Phar .Bio (9) 777
14. **Reitman S and Frankel S (1957):** Colorimetric determination of S.AST, S. ALT enzymatic activity. An.J.Clin.Path, 28:56
15. **John D (1982):** laboratory mothed for det. of alkaline phosphatas 9thEd. 580.
16. **Fawcet J and Scott J (1960) :** Determination of urea .J. Clin. Path. (13) 156
17. **Husdan H and Roporpot A (1968):** Estimation of cratinin. Clin. Chem 14, 22

18. **Bancroft J, Steven A and Turner D (1990)**: Theory and practice of histological techniques 3rd Ed. Churchill Livingstone, London and New York.
19. **Petri A and Watson A (1999)**: Statistical for Vet. Animal Sci Ltd United Kingdom
20. **Naeshiro Y, Ishimura F and Sato S (1997)**: Possible mechanism for the anaemia induced by gentamicin in rats. *Comp. Haemat. Inter.* 7(4) 220-225.
21. **Priuska E and Schacht J (1995)**: "Formation of free radical by gentamicin and iron and evidence for an iron-gentamicin complex". *Bioc Phar.* 50: 49-52.
22. **Helmy O (2000)**: Effects of some aminoglycosides in chickens. Master degree of Pharmacology, Fac. of Vet. Med., Zag. Univ.
23. **Malhat Seham M (2006)**: Some Pharmacological Studies on Aminosidine in Chickens. 8th Sci. Vet. Med. Zag., 38-46
24. **Gammaz H and Abd-Alla O (1991)**: Effects of netilmicin on some hematological and blood serum biochemical parameter in albino rats. *Zag Vet. J.* 19 (3) 96.
25. **Enver Y, Sibel K and Ahmet L (2004)**: Effect of vitamin E on hematological biochemical parameters in endotoxaemic in rabbits. *Bull vet inst* 48, 105-108.
26. **Abbas S (2002)**: Effect of vitamin E and selenium on performance and some blood serum constituents in Saidi lambs. *Assiut Vet. Med. J.* 47 (94) 29-38
27. **Khan I, Khan M, Salemi Z and Javed I (2008)**: Pathological and biochemical effects of gentamicin in Chicken Turkish J. of Vet Ani. Sci. 32 (5) 45-51
28. **Muhammad K, Muhammad Z, Ijaz J and Ahrar K (2009)**: Pathological effects of gentamicin in broiler chicks. *Exp. & Toxic Path.*, 61 (5) 25-32
29. **Shalaby M and Amer H (1990)**: Nephrotoxicity of netilmicin in rats. *Benha Vet. Med. J.* (1) 47-58.
30. **Kaneko J (1989)**: Biochemistry of domestic animal Acad. Press Inc New York
31. **El-Zaiat A, Attia S and Shoeib M (2006)**: Effects of clay and vitamin E addition to the dietary mandara strain polluted with the lead on blood components, hatchability and histological changes of the testis. *Zag. Vet. J.* 34 (3) 161-175
32. **Abd El-Latif S (1999)**: Nutritional interrelationships of vitamin E and selenium on laying Japanese quail. *Egypt. J. Nutr. Feeds.* 2. 711 - 718.
33. **Donaldson W and Christensen V (1995)**: possible toxic effects of Gentamicin in newly hatched turkey poults. *Poultry Science Assoc.* 4:271-275
34. **Saleemi K, Zargham M, Javed I and Khan A (2009)**: Pathological effects of gentamicin I/M administered to broiler chicks. *Exp. Toxicol. Pathol.* 61 (5) 25-32
35. **Edson R and Terrell C (1999)**: The aminoglycoside. *Mayo. Clin. Proc.* 74: 19
36. **Schumacher J, Wilson R, Spano J and Hughes F (1991)**: Effect of diet on gentamicin-induced nephrotoxicosis in horses. *Am. J. Vet. Res.* 52 (8) 274-278.
37. **Ijaz K, Muhammad K and Ahrar K (2008)**: Pathological and biochemical effects of gentamicin in chickens. *Turk. J. Vet. Anim. Sci.* 32 (5) 45-51.
38. **Karadeniz I A, Yildirim N and Celebil, F (2008)**: Effect of ginseng on gentamicin sulphate-induced kidney toxicity in rats. *Rev Méd. Vét.* 159 (4) 15-20
39. **Kumar K, Shfw A and Ratnk K (2000)**: Carvdilol, beta blocker with antioxi-dant property protect against gentamicin nephrotoxicity in rats. *Life Sci*, 66, 61-69
40. **Attia A, Ayyat M and El-Zaiat A (2005)**: Role of clay or vitamin E in layer hens fed diets contaminated by lead. 2nd paper: Blood components and lead residues in the

- tissues and eggs. Allatt. Takar, 54 (2) 179-190
41. *Tsereteli B, Andronikali T and Lremashvih N (1994):* Vitamin E supplements in diets for birds. Zoot, 5. 17 - 18.
42. *Abd El-Aziz M (2002):* Handbook of Veterinary Pharmacology, 5th Ed.
43. *Abd El-Hasseb A, Khalid E and Mohey M (2004):* Testicular physiology responses to selenium and vitamin E in rabbits. Alex. J. Vet. Med. 21(1): 162-167
44. *Williams P, Hottendrf G and Bennet D (1986):* Inhibition of renal membrane binding and nephrotoxicity of aminoglycoside. J. Pharm. Exp. Ther., 237:19- 25.
45. *Baliga S, Ueda N, Walker P and Shah R (1999):* Oxidant mechanisms in toxic acute renal failure. Drug. Metab. Rev., 31, 971-997.
46. *Begg E and Barclay M (1995):* Aminoglycoside. Br. J. Clin. Pharma. 39: 77-83
47. *Abdel-Naim A, Abdel-Wahab H and Attia F (1999):* Protective effects of vitamin E against gentamicin-induced nephrotoxicity in rats. Pharm Res. 40 (2) 83 - 87
48. *Varzi R, Avizeh A and Shahriari E (2007):* Effect of vitamin E on gentamicin-induced nephrotoxicity in dogs. J Vet Pharmacol Ther.30 (5) 77- 81
49. *Beytut E, Mine E and Akskal M (2003):* Effect of vitamin E on antioxidative defense in rats kidney treated with of glucocorticoid. Cell Bioch Funct 16 (40)10 -18
50. *Mohammad A (2000):* Biochemical studies on some antioxidants in blood of diabetic rats. Ph. D. Sc Thesis of biochemistry, Fac. Vet. Med. Zagazig Uni.

الملخص العربي بعض التغيرات البيوكيميائية والباثولوجية في الرومي المحدث بالجنتاميسين ومعالجتها باستخدام فيتامين هـ

عزت ابو الفتوح حمودة، هاله محمد محمد خليل*، جيهان نبيل عبد الوهاب، سعاد مكاوي**
أقسام (الباثولوجيا الكيمياء* الباثولوجيا الاكلينيكية**): معهد بحوث صحة الحيوان (فرع الزقازيق):

تم تقسيم ٨٠ كتكوت رومي عمر ٤ اسبوع إلى ٤ مجموعات متساوية وكانت المجموعة الأولى كتاكيت رومي بصحة جيدة غير معالجة (مجموعه ضابطة):. المجموعة الثانية كتاكيت رومي بصحة جيدة وتم إعطائها الجنتاميسين بالحقن في العضل بجرعه ٥ مجم/كجم من وزن الجسم لمدة خمس ايام متتالية. المجموعة الثالثة كتاكيت رومي بصحة جيدة وتم إعطائها فيتامين هـ في مياه الشرب بجرعه ١٠ مجم/كجم من وزن الجسم لمدة خمس ايام. المجموعة الرابعة كتاكيت رومي بصحة جيدة وتم إعطائها الجنتاميسين وفيتامين هـ بنفس الجرعه والمده السابقه. تم ذبح ٥ كتكوت رومي عند اليوم الاول من نهايه العلاج وتم أخذ عينتين دم عند اليوم الاول من نهايه العلاج في جميع المجاميع السابق ذكرها. أخذت العينة الأولى على هيبارين وذلك لدراسة التأثيرات على صورة الدم والأخرى لفصل المصل وذلك لقياس بعض المؤشرات البيوكيميائية. يتم اخذ عينات من الكبد والكلى لعمل شرائح باثولوجية لدراسة تأثير الجنتاميسين وفيتامين هـ على تلك الاعضاء باثولوجيا.

تشير النتائج أن الجنتاميسين أدى إلى حدوث نقص معنوي في عدد كرات الدم الحمراء، تركيز الهيموجلوبين، حجم خلايا الدم المرصوصة، البروتين الكلي، الالبومين و الجلوبيولين وزيادة معنوية في، انزيمات الكبد (ALT-AST): حمض اليوريك والكرياتينين كما أدى إلى نقص معنوي في البروتين الكلي، الالبومين و الجلوبيولين وزيادة غير معنويه في عدد كرات الدم البيضاء.

وأظهرت النتائج أن فيتامين هـ أدى إلى حدوث زياده فى عدد كرات الدم الحمراء, تركيز الهيموجلوبين ، حجم خلايا الدم المرصوصة ، كرات الدم البيضاء البروتين الكلى ، الاليومين و الجلوبيولين وزياده غير معنويه فى , , ALP, ALT, AST الكرياتينين وحمض اليوريك.

بالفحص الظاهري لكتاكت الرومى المعطاه الجنتاميسين فقط(المجموعه الثالثه): وجد ان الكبد والكلى بهما احتقان وتضخم وانزفه كما ان القلب به التهابات فى غشاء التامور.وبالفحص المجهرى للكبد (المجموعه الثانيه): وجد تنكسات مختلفه واحتقان وارتشاح للخلايا الالتهابيه بالاضافه الى اماكن بها تنخر. بالنسبه للكلى فيوجد بها نخر بالنبيبات الكلويه بالاضافه الى احتقان وارتشاح بالخلايا الالتهابيه مع تنكسات مختلفه فى بعض الحالات الاخرى.اما بالنسبه للقلب فيه وزمه بالاضافه الى التهاب غشاء التامور.اما بالنسبه للمجموعه الرابعه فوجد بها نخر تخثرى خفيف واتساع خفيف للحبيبات الكديه مع خلايا كديه طبيعيه والكلى بها ارتشاح خالى بسيط للخلايا الالتهابيه والقلب يوجد به التهاب خفيف بغشاء التامور وعضلات القلب وجدت بحاله سليمه. وتلاحظ أن استخدام الجنتاميسين وفيتامين هـ أديا إلى عودة هذه الوظائف إلى المستوى الطبيعي في مصل كتاكت الرومى .

مما سبق واستنادا إلى التغيرات فى صورة الدم والتغيرات البيوكيميائية في مصل كتاكت الرومى والتاثيرات الباثولوجيه التى احدثها الجنتاميسين يمكن القول أن ذلك العقار له تاثيرات عكسيه على الجسم ولكن استخدام فيتامين هـ امكنه التغلب على الاثار العكسيه للجنتاميسين لذلك ينصح باستخدام فيتامين هـ للتغلب على تلك الاثار العكسيه اثناء العلاج بالجنتاميسين