Biochemical And Pathological Studies In Sheep Suffering From Neuromuscular Disturbance

Gehan H El-Sakkar*, Arafa MM and Youssef SF****
* Pathology Dept., Zagazig Veterinary Lab.
**Biochemistry Dept., Animal Health Research Institute, Dokki

ABSTRACT

Blood and serum samples were collected from 25 adult sheep (9-24 month old) in Kalubia governorate. The animals were suffered from stiffness, weakness, trembling in limbs, incoordination and inability to stand. Five apparently healthy adult sheep were used as control. Examination of two ration samples (offered to affected and non affected animals) proved to be balanced. Ration was free from rancidity and aflatoxin, while ochratoxin was detected in the ration which had been eaten by sheep showed clinical sings. Such ration was deficient in phosphorus, calcium, magnesium and copper, while the molybdenum level was high when compared with the ration given to healthy animals. The values of total RBCS, PCV, Hb, MCH and MCHC were significantly low in the affected sheep compared to that of the control. Analysis of serum microelements revealed significant decrease in copper, iron and zinc levels. Estimation of serum minerals revealed decreased calcium, phosphorus, magnesium, sodium and potassium levels. Serum triglycerides, glucose cholesterol, total protein and albumin were significantly decreased while serum globulin was not changed. A significant increase was found in the activities of AST, ALT. ALP, LDH and cortisol in diseased sheep when compared to control. Macroscopic examination revealed enlarged and congested liver. Some cases showed whitish foci and petechial hemorrhages on the liver surface. The lungs showed congestion and kidneys were swollen and pale. Atrophy of the spleen was seen in some cases, while others showed hemorrhage. The intestine was congested and revealed ulcerated mucosa. Microscopically the liver showed focal hepatic hemorrhage, cloudy swelling, and hypertrophic kupffer cells, associated with sinusoidal leucocytosis besides with fibrous tissue proliferation in portal tract surrounding dilated bile duct containing necrotic debris. The kidneys showed necrotic renal tubular epithelium. The lungs showed focal acquired atelectasis and compensatory focal alveolar emphysema, in addition to intralobular bronchiole containing necrotic debris in addition to showing hyperplasia of bronchial epithelium. The spleen showed focal splenic hemorrhage and lymphocytic necrosis and depletion. The intestine showed necrosis of intestinal villi associated with massive leukocytic infiltration and mucosal edema. Focal cerebral hemorrhage and focal gliosis were seen in the brain sections.

Finally all the investigated parameters, in the ochratoxicosis affected animals, revealed decreased dietary mineral level, specially calcium, phosphorus and copper. Such deficiencies were suggested to be the real cause of the problem. Based on the results of the diseased sheep are partially induced by ochratoxicosis and mediated by copper, calcium, phosphorus and magnesium, deficiencies and elevated molybdenum and ferric.

INTRODUCTION

Ochratoxin is a common feed contaminant, which may impair animal health and lead to accumulation of residues in the edible tissue of slaughtered animals *(1)*. Mycotoxins depress T and B lymphocytic activity, suppress antibody production, and reduce complement and interferon activity, besides the impairment of macrophage function *(2)*. The signs of ochratoxicosis ranged from death (acute toxicity), immunosuppression, hepatotoxicosis, nephrotoxicosis and neurotoxicosis *(3)*.

Mineral deficiency, in adult sheep, appears as a cause of failure of some animals to make satisfactory weight-gain, in spite of the presence of balanced feed and abundant water with absence of signs of any primary disease, meanwhile the other sheep appear in satisfactory

health body condition *(4)*.Nutritional surveys in different areas of Egypt have revealed deficiency in soil-trace elements and consequently in the grazing fodder . Thus, deficiency signs due to subnormal intake of phosphorus, calcium, copper, zinc, cobalt and selenium have been observed in certain areas *(5)*. Ochratoxin is among the most abundant food contaminating mycotoxin in the world *(6)*.Approximately 25% of world annual crops are affected by mycotoxins *(7)*. Economic losses from mycotoxicosis in agriculture are due to the effects on livestock productivity (morbidity), losses in crops as well as costs, besides of regulatory programs directed toward mycotoxins which have been estimated by billions of dollars *(8)*.

The present study was planned to diagnose the probable cause of neuromuscular disturbance among adult sheep via histopathological studies of some organs and detection of the mean levels of some biochemical parameters, and hematological profile.

MATERIAL AND METHODS

Animals

Determination of some biochemical analysis were conducted on 25 sheep (9-24 month old) belonging to Kalubia Governorate. They were suffering from enteritis, depression, unsatisfactory body-weight-gain and difficult movement (neuromuscular disturbance). Other five apparently healthy sheep represented the control group.

Sampling

1. Blood

Blood samples collected from the jugular vein of each animal were divided to two portions. The first blood sample was mixed with EDTA for hematological examination *(9)*. The second blood sample were left to coagulate, and clear serum was separated for estimation of total protein *(10)*, albumin *(11)*, ALT and AST *(12)*, LDH *(13)* and ALP *(14)*, besides glucose *(15)* cortisol *(16)*, calcium *(17)*, inorganic phosphorus *(18)*, magnesium *(19)*, sodium and potassium *(20)*, cholesterol *(21)*, triglycerides *(22)* and copper, iron, zinc and molybdenum *(23)*.

2. Ration and water

Ration samples were collected from the manger and ration-store, for detection of aflatoxin and ochratoxin *(24)*. Peroxide number, oxidative rancidity and acid number were evaluated *(24)*. They were analyzed chemically for humidity %, ash%, crude protein % and ether extract % *(24)*.

3. Pathological examination

Tissue specimens were collected from the liver, kidneys, brain, intestine, spleen and lungs, immediately after slaughtering and directly fixed in 10% neutral buffered formalin. Five micron thick serial paraffin sections were prepared, stained with hematoxylin and eosin *(25)* and examined microscopically.

4. Statistical analysis

The obtained data were analyzed using t-student test *(26)*.

RESULTS and DISCUSSION

Signs

The main clinical signs, observed on the affected sheep from a Kalubia Governorate Farm, were stiffness, weakness, incoordination trembling of the limbs, inability to stand, diarrhea, anorexia and poor growth. Some cases showed diarrhea and pneumonia. Several previous studied showed similar clinical signs *(27-29)*.

Ration analysis:

Analysis of both collected ration samples revealed balanced crude protein and total fat content. Both rations were free from rancidity, aflatoxin B_1- B_2- G_1 - G2, but ochratoxin was detected (50 ppb). On the other hand Ca, P, Mg and Cu- contents were low while Fe and Mo-levels were higher in the ration offered to the affected sheep than that of the healthy ones (Table 1).

Table 1. Ration composition offered to diseased and apparently healthy animals.

Diet ingredients	Diseased animals	Healthy animals
Crude protein %	14.7	16.2
Total fat %	3.1	3.2
Total Ca %	0.34	0.84
Total P %	0.25	0.71
Total Mag %	0.27	0.35
Total Cu %	0.03	0.08 – 0.18
Total Fe %	0.20	0.4 – 0.11
Total Zn %	5.2	5.1
Total molybdenum %	0.9	0.49
Acid No	3.1	5.2
Aflatoxin G1	- ve	- ve
G2	- ve	- ve
B1	- ve	- ve
B2	- ve	- ve
Ochratoxin	50 ppb	- ve

ppb = Part Per Billion.

Hematological analysis

Table 2 shows the hemogram of the apparently healthy and affected sheep. The mean values of the RBCs count, Hb, PCV, MCH and MCHC were significantly decreased in the affected sheep when compared with the healthy ones. Those results pointed out coexistence of clinical marginal anemia with clinical laboratory mineral deficiency. The mean values of the total and differential leukocytic counts in the apparently healthy and diseased sheep revealed a significant leukopenia in the latter. The total leukocytic count was significantly decreased when compared with the healthy ones. Such a decrease in the total leukocytic count may be attributed to anorexia and immunodepression *(30)*.

Table 2. Hemogram in both healthy and diseased sheep.

Parameters		Healthy animals (n=5)	Diseased animals (n=25)
RBC ($\times 10^6$ mm)		6.3 ± 0.23	4.1 ± 0.12**
Hb (g/dl)		8.4 ± 0.13	7.9 ± 0.55*
PCV (%)		31.6 ± 0.72	30.17 ± 1.5
MCV (fl)		52.5 ± 0.41	53 ± 0.9
MCH (pg)		14 ± 0.17	11.58 ± 0.3*
MCHC (g/dl)		27.36 ± 0.12	22 ± 0.6*
WBCs ($\times 10^3$ mm)		7.3 ± 0.4	4.7 ± 0.3*
Differential leukocytic count $\times 10^3$	Neutrophil %	31 ± 1.2	34 ± 1.8
	Eosinophil %	5.13 ± 0.4	5.0 ± 0.2
	Lymphocyte %	5.8 ± 3.4	52.0 ± 3.6
	Monocyte %	6.0 ± 0.2	8.4 ± 0.3
	Basophil	0	0

* Significant when compared with the control at $P < 0.01$ using student test.
** Significant when compared with the control at $P < 0.001$ using student test.

Biochemical exanimation

The mean values of activities of serum AST, ALT, ALP and LDH in the healthy and affected sheep are presented in Table 3. They were significantly increased in the diseased sheep. The mean values of serum total protein, glucose, cholesterol and triacylglycerol were decreased in the diseased sheep Table 3. The significant decrease in the serum total protein and albumin levels in the diseased is probably due to the stress of diarrhea and its impact on the hepatic parenchyma resulting in a failure of protein synthesis *(9)*. Hepatic damage was monitored by the significant increase in the AST, ALT, ALP and LDH activities in the diseased sheep. Moreover, the decrease in the total serum protein and albumin may be attributed to increased levels of cortisol in the diseased animals which causes catabolism of protein leading to negative nitrogen balance and increased urinary elimination of nitrogen. Elevated liver enzymes in the blood of sheep fed copper deficient diet, indicating hepatic dysfunction *(31)*. The decreased serum glucose level in the diseased animals may be due to copper deficiency *(32)*. The significant decrease in the total serum cholesterol and triacylglycerol levels in the diseased sheep, than the control ones, may reflect either reduced food intake or early hepatic damage. Similar results reduction in serum cholesterol level was recorded in copper deficient animals *(33)*. The high values of serum cortisol may be attributed to hypocuprosis via influencing the adrenal cortex.

Table 3. Serum biochemical profile of healthy and diseased sheep.

Parameters	Healthy animals (n = 5)	Diseased animals (n = 25)
AST IU/ L	35 ± 0.8	46 ± 0. 2**
ALT IU/ L	15 ± 0.9	30 ± 0.9**
ALP IU/ L	89 ± 0.6	115 ± 1.33**
LDH IU/ L	150 ± 9.2	670 ± 7.6**
Cortisol mg/dl	5.7 ± 0.14	8.7 ± 0.1*
Total protein g/l	7.6 ± 0. 2	6.4 ± 0.2*
Albumin g/dl	3.9 ± 0.17	2.11 ± 0.13*
Globulin g/dl	3.7 ± 0.16	4.3 ± 0.2
Glucose mg/dl	69 ± 1.2	45.5 ± 01.04*
Cholesterol mg/dl	124 ± 1.02	90 ± 2.1*
Triglyceride mg/dl	94 ± 0.9	84 ± 0.44*

* Significant when compared with the control at $P < 0.01$ using student test.
** Significant when compared with the control at $P < 0.001$ using student test.

The significant decrease in the serum calcium, phosphorus, magnesium, sodium and potassium levels (Table 4) in the diseased animals could be attributed to their low concentration in the offered ration together with presence of diarrhea which decreased the intestinal absorption of most nutrient *(4)*. The serum copper, zinc and iron were highly significantly decreased ($P<0.001$) in the diseased sheep while the serum-molybdenum-level was significantly increased when compared with the healthy ones. The significantly decreased serum copper level in the diseased animals may be attributed to the high concentration of molybdenum in the ration which reduces the availability of dietary copper due to formation of insoluble cupric-thiomolybdate complex in the rumen *(34)*. This antagonistic relationship between copper and molybdenum was previously recorded *(35)*. The encountered decrease in the serum zinc and iron levels may be attributed to interference with their absorption from the gastrointestinal tract, besides anorexia *(4)*.

Table 4. Some macro- and microelements in both the healthy and diseased sheep.

Parameters	Healthy animals (n = 5)	Diseased animals (n = 25)
Copper µg/dl	60.3 ± 2.6	49.8 ± 3.1**
Zinc µg/dl	80.08 ± 6.1	59 ± 4.1**
Iron µg/dl	87.3 ± 4.1	63 ± 1.3*
Molybdenum µg/dl	68 ± 3.1	90.6 ± 4.8**
Calcium mg/dl	10.2 ± 0.3	8.4 ± 1.3*
Phosphorus mg/dl	5.4 ± 0. 9	3.2 ± 0.1**
Magnesium mg/dl	2.8 ± 0.2	2.0 ± 0.3*
Sodium mg/L	143.5 ± 6.1	136 ± 5.7*
Potassium mg/L	5.0 ± 0.19	3.9 ± 0.8*

* Significant when compared with the control at $P < 0.01$ using the student test.
** Significant when compared with the control at $P < 0.001$ using the student test.

Pathological examination

a. Macroscopically

The liver of some diseased sheep was enlarged, soft, pale and its capsule showed petechial hemorrhages. These changes were previously cited *(28,29)*. Meanwhile the examined kidneys were enlarged and petechial hemorrhages were prominent which agreed with findings of *(28,36)*. The lungs of some diseased sheep were congested and enlarged. The lungs, in other cases, were soft in consistency and pale. Its visceral pleura was studded with petechial hemorrhages. The spleen was atrophied and sometimes hemorrhagic. The small intestine was congested and frequently showed ulcerated mucosa. The brain was soft in consistency, congested and presented multiple petechial hemorrhages in the cerebrum and cerebellum . The previous PM findings were recorded by several authers *(28, 29, 36)*.

b.Microscopically

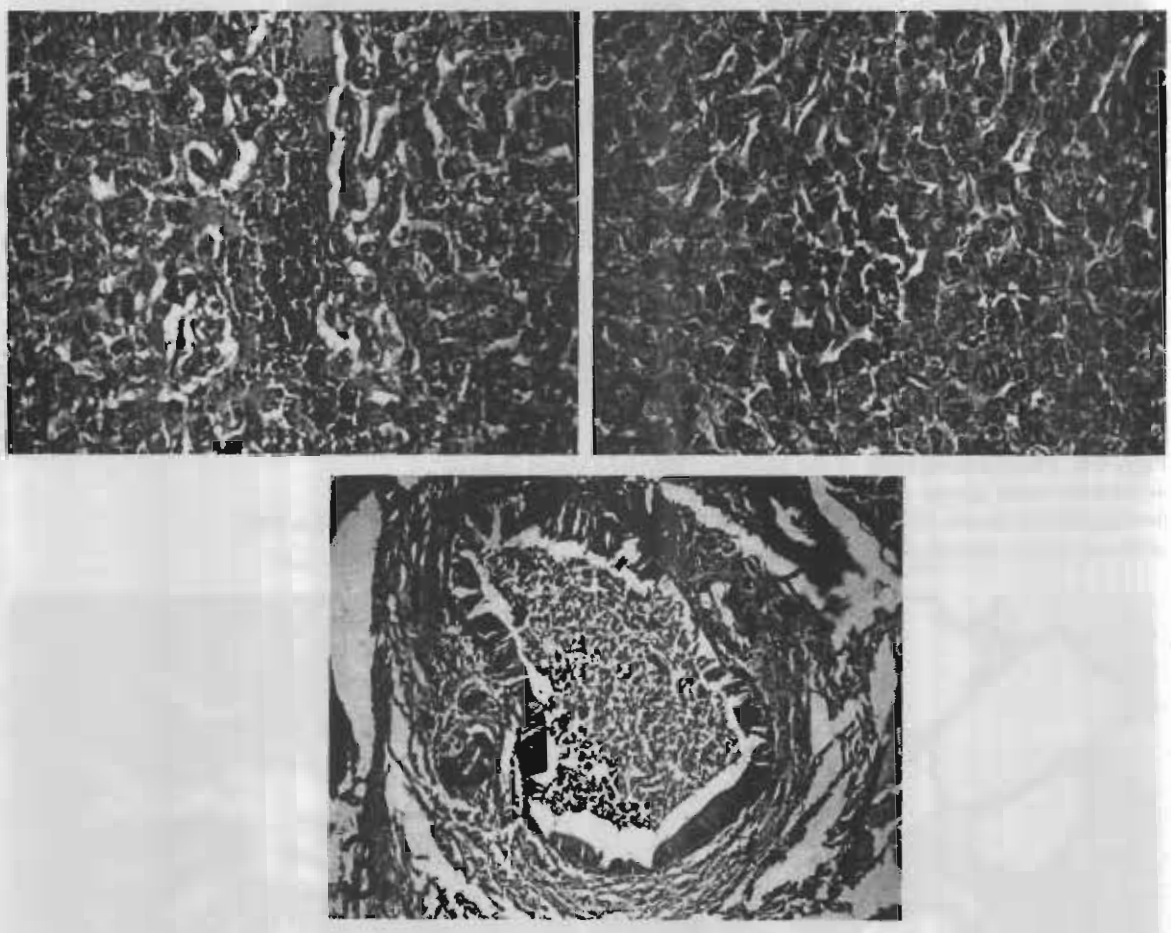
Mycotoxins encompass a wide spectrum of different components which affect target organs and systems, notably, the liver and kidneys, besides the nervous and immune systems *(37)*.The liver of the obligatory slaughtered diseased sheep showed focal hemorrhages (Fig.1). The lesions of mycotoxicosis depend on the toxin level, and ranged from poor performance to general hemorrhagic syndrome *(29,38)*. Other cases showed hypertrophic kupffer cells associated with sinusoidal leucocytosus (Fig. 2). The portal tracts showed fibrous tissue proliferation surrounding dilated bile duct containing necrotic debris (Fig. 3). These changes are reported in previous studies *(1,28,29,36)*. The proliferated fibrous connective tissue in the portal triads is diagnostic and may be due to the direct effect of ochratoxin. Moreover the examined kidneys revealed necrotic renal tubular epithelium and dilated bowman's space (Fig. 4). Similar findings were previously described by *(28,29,36,39,40)*. Coagulative nerosis of the renal tubular epithelium with pyknotic neuclei, were observed in the diseased animals (Fig.5). Such lesions is characteristic for ochratoxin related nephrotoxicosis *(41,42)*. Other examined case showed focal tubular necrosis with mononuclear leukocytic infiltration (Fig.6). Nephritis is evidenced by necrosis of renal tubule *(1,28,29,39)*. The lungs showed focally acquired atelectasis with compensatory focal alveolar emphysema (Fig.7) *(29,43,44)*. In other cases the Intralobular bronchiole containing necrotic debris and showing hyperplasia of bronchial epithelium (Fig.8). Similar lesions were previously reported *(40)*. Meanwhile the examined spleen showed focal splenic hemorrhage (Fig. 9). Another examined case revealed lymphocytic necrosis and depletion from the white pulp (Figs. 10 & 11). Lymphocytic depletion was probably due to immunosuppressive effect of mycotoxins *(1,28,29)*. Mycotoxin reduced the efficacy of the acquired immunity and the diminished effect of

mycotoxin-cell-mediated-immunity may be a reflection of the amphipathic effect of the toxin *(45)*.

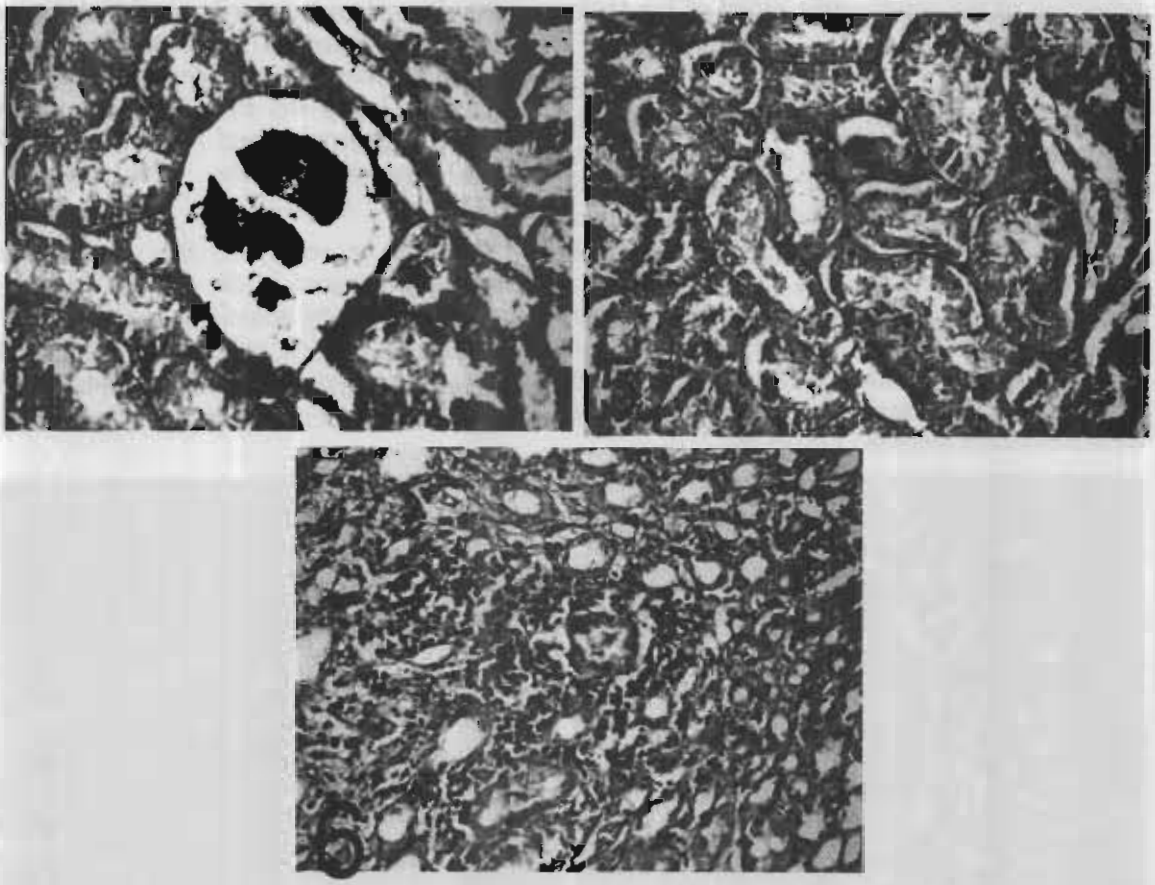
Intestine: Some examined cases showed necrotic villi and edematous lamina propria with leukocytic infiltration (Fig. 12). Other cases showed marked necrosis of the intestinal villi associated with massive leukocytic infiltration (Fig. 13). Meanwhile necrosis of the crypts of Lieberkuhn was associated with leukocytic infiltration (Fig. 14). Finally, mucosal edema and necrotic villi were detected (Fig. 15). Similar necrosis of the intestinal villi, and crypts of Lieberkuhn with massive leukocytic infiltration were previously recorded *(28,36,40)*.

Brain: The examined brain of the diseased sheep showed congested cerebral blood vessels and small focal cerebral hemorrhage (Figs. 16 & 17). Other examined cases showed focal gliosis (Fig. 18). Finally the examined brain showed neuronal degeneration and neuronophagia (Fig. 19). The previous lesions were recorded by *(29,44)*.

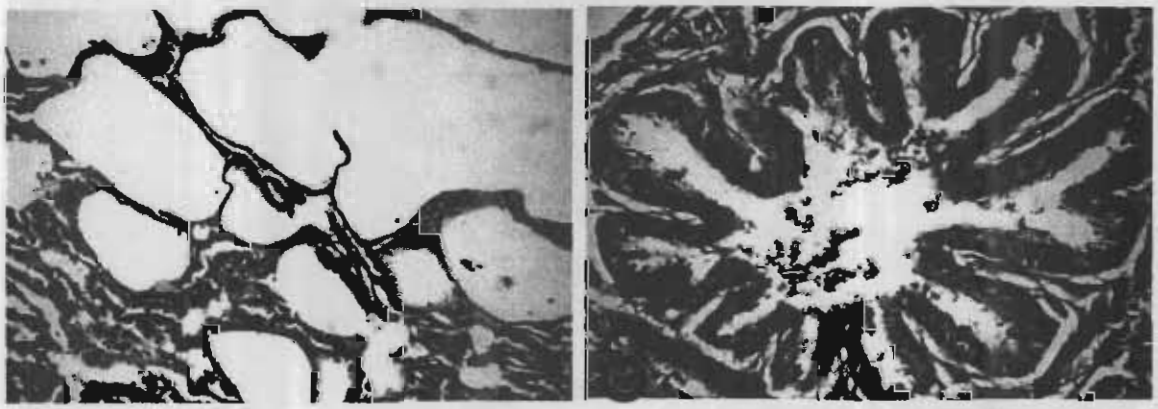
It could be concluded that the signs and lesions, encountered in the diseased sheep, are partially induced by ochratoxin and mediated by deficiency of copper, calcium, phosphorus and magnesium with elevated molybdenum and iron.



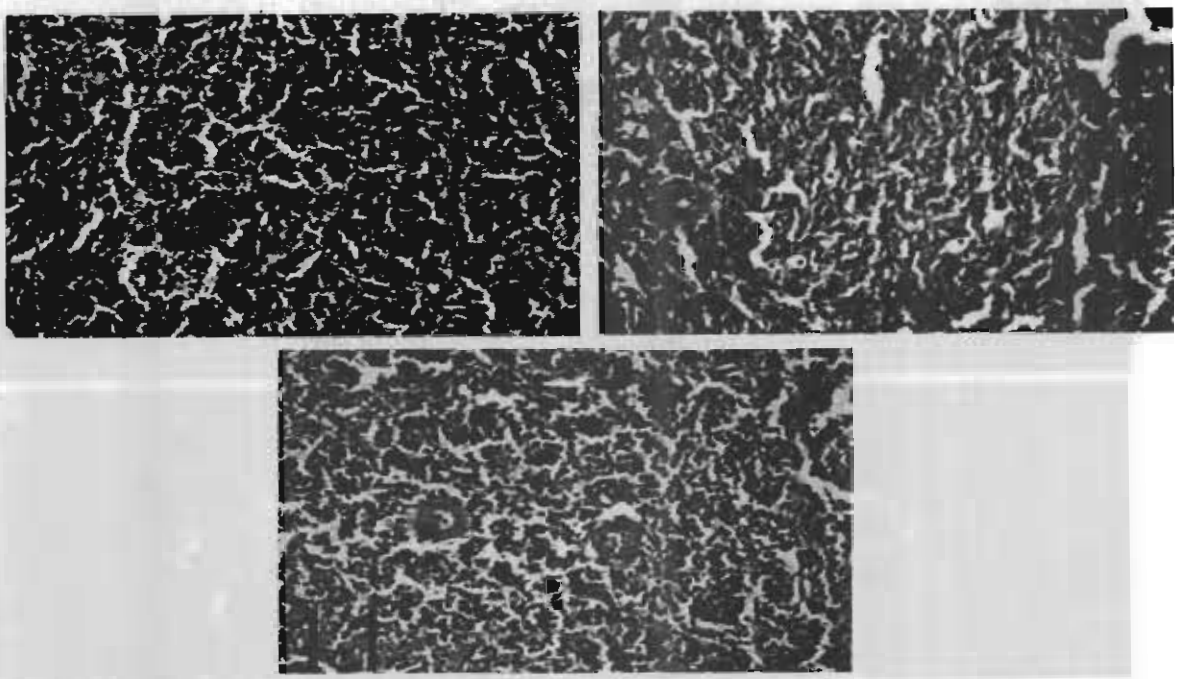
Figs. 1-3. Photomicrograph of the liver of diseased sheep showed
1- Focal hemorrhage. (H & E x 200).
2- Hypertrophic kupffer cells associated with sinusoidal leucocytosis (H & E x 200).
3- portal tract with fibrous tissue proliferation surrounding dilated bile duct containing necrotic debris. (H & E x 100).



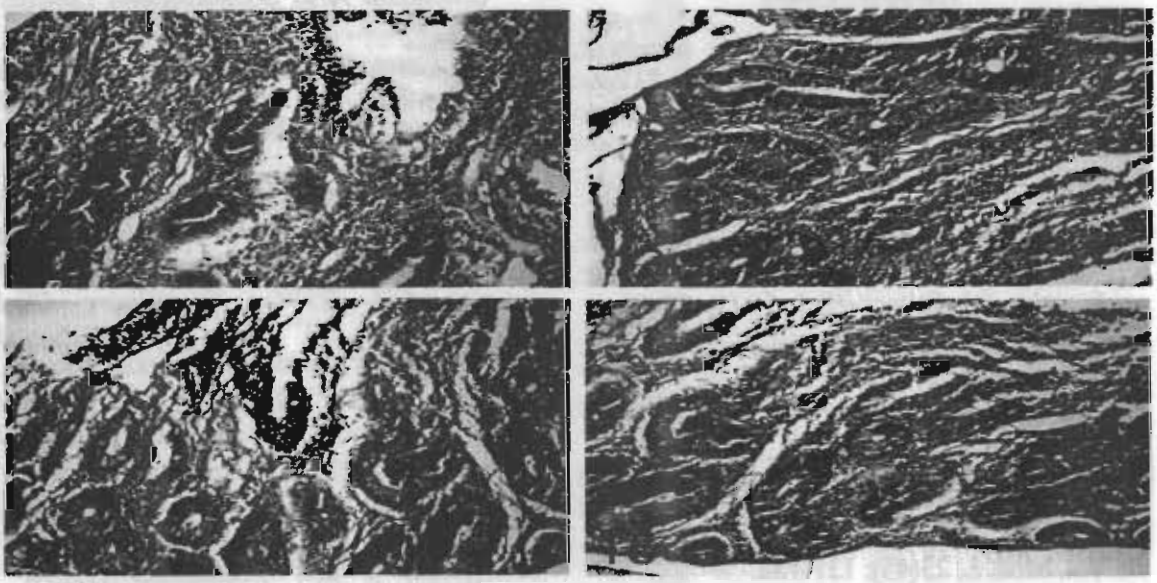
Figs. 4-6. Photomicrograph of the kidney of diseased sheep showed
4- Necrotic renal tubular epithelium and dilated bowman's space ,(H & E x 200).
5- Coagulative nerosis of the renal tubular epithelium with pyknotic neuclei, (H & E x 200).
6- Focal tubular necrosis with mononuclear leukocytic infiltration (H & E x 200).



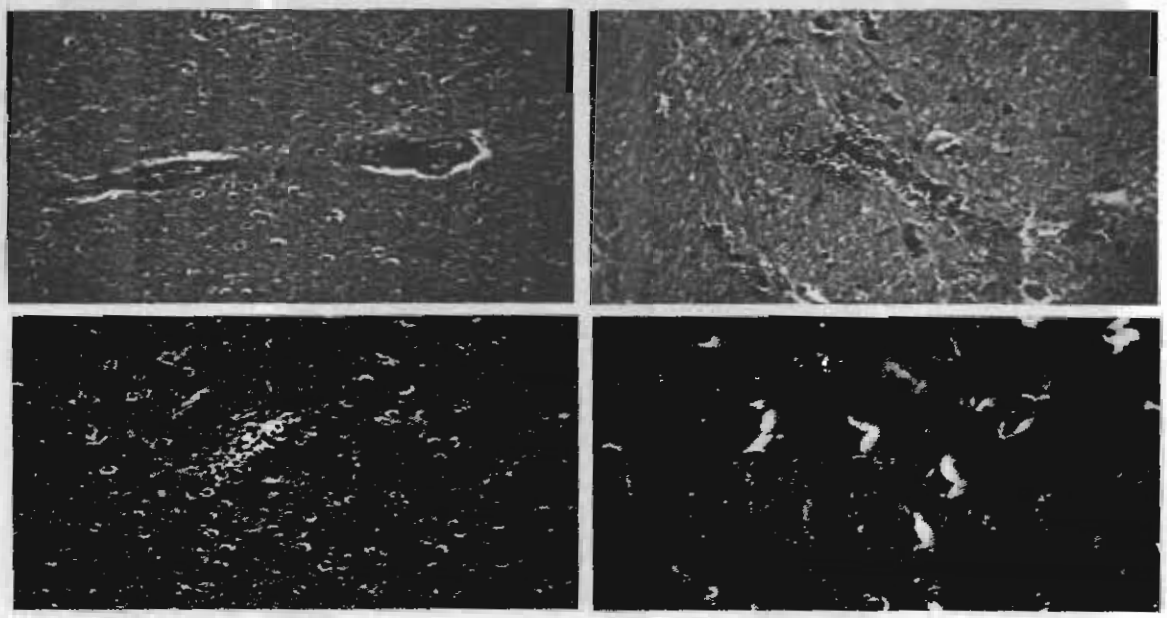
Figs. 7, 8. Photomicrograph of the lung of diseased sheep showed
7-Focal acquired atelectasis and compensatory focal alveolar emphysema ,(H & E x 100).
8-Intralobular bronchiole containing necrotic debris and showing hyperplasia of bronchial epithelium. (H & E x 100).



Figs. 9-11. Photomicrograph of the spleen of diseased sheep showed
9-Focal hemorrhage (H & E x 200).
10- Lymphocytic necrosis and depletion (H & Ex 200).
11- Lymphocytic depletion from the white pulp. (H & E x 200).



Figs. 12-15. Photomicrograph of the intestine of diseased sheep showed
12-Necrotic villi and edematous lamina propria with leukocytic infiltration (H & E x 100).
13-Marked necrosis of intestinal villi associated with massive leukocytic infiltration (H & E x 100).
14)-Necrotic crypts of Lieberkuhn associated with leukocytic infiltration (H & E x 200).
15): Mucosal edema and necrotic villi (H& E x 100).



Figs. 16-19. Photomicrograph of the brain of diseased sheep showed
16-Congested cerebral blood vessels. (H & Ex 200).
17-Small focal cerebral hemorrhage. (H & E x200).
18- Focal gliosis. (H & E x 200).
19-Neuronal degeneration and neuronophagia,. (H & E x 200).

REFERENCES

1. ***Biro K, Solti L, Vetro I, Bago G, Glavits R, Szabo E and Gremmels J (2002):*** "Tissue distribution of ochratoxin A as determined by HPLC and Elisa and histopathological effects in chickens." Avian Pathology, 31: 141-148.
2. ***Corrier DE (1991):*** "Mycotoxicosis: Mechanisms of immuno-suppression." Vet. Immuncel. Immunopathol., 30 (1): 73-87.
3. ***Hallinger K and Ekperigin HE (1999):*** "Mycotoxicosis in food producing animals." Vet Clin. North. Amm Food Anim. Pract, 15 (1): 133-165.
4. ***Abdel-All S T I, Ismail M N and Amer A A (1990):*** "Investigation on rickets problem of fattening calves at Qena province." Assiut. Vet. Med. J., 23(45): 131-138.
5. ***Nasser MH (1995):*** "Diagnosis of copper deficiency in cattle." 3rd Ed. Congress Egyptian Society for cattle Diseases, 3-5 Dec., Assiut, Egypt.
6. ***Morshdy AM, Hussein MA, El-Desoky K and El-Sebaey SS (2009):*** "Mould and ochratoxin A in Soyabean and Luncheon." Zag. Vet. J., 37 (3): 57-62.
7. ***Cost Council for agricultural science and technology (1989):*** "Mycotoxins: Economics and health Risks." Report No. 116. Ames, IA.
8. ***Trail F, Mahanti N and Linz J (1995):*** "Molecular biology of aflatoxin biosynthesis." Microbiology, 141: 755-765.
9. ***Coles EH (1986):*** "Veterinary Clinical Pathology." 4th Ed., W.B. Saunders Company, Philadelphia, London, Toronto.
10. ***Weichselbaum RE (1946):*** "Determination of total protein." Am. J. Clin. Path., 16: 40.
11. ***Drupt F (1974):*** "Colorimetric method for determination of albumin." Pharm. Biol., 9: 777-779.
12. ***Reitman S and Frankel S (1957):*** "A colorimetric determination of serum glutamic oxaloacetic and glutamic pyruvic transaminase." Am. J. Clin. Path., 28:56-58.
13. ***Cabaud PG and Worblewskj F (1958):*** "Colorimietric measurement of lactic dehydrogenase activity of body fluids." Am. J. Clin. Path., 30: 231-236.

14.-Eastman JR and Bixter D (1977): "Kinetic, colorimetric method for measurement of alkaline phosphatase activity in serum." Clin. Chem., 23: 1769.

15.Siest G, Henry J and Schiële F (1981): "Interpretation des examens de laboxatoire." Karger Ed., P. 206.

16.Calstorn K, Balton N, Kallner A and Kini O R (1988): "Cortisol in assay of reproductive hormones when, why and how." IFCC and Farons diagnostica, 23.

17.Glinger EM and King JD (1972): "Rapid colorimetric determination of calcium in biological fluids." Am. J. Clin. Path., 58: 376.

18.Klichling H and Freiburg Br (1951): "Inorganic phosphorus in serum in clinical photometric." 3rd Ed., Vet. Ges.mbH, Stuttgart.

19.Niel DW and Neely RA (1956): "Determination of magnesium in serum." J. Clin. Path., 9: 162.

20.Burriel M and Ramirez OM (1951): "Manual of Flame Photometrically Flame Photometry Theory and Application." American Elsevier publishing Co. Inc. New York.

21.Richmond W (1973): "Enzyme preparation for cholesterol investigation." Clin. Chem., 19: 1350-1356.

22.Young D and Pestaner L (1975): "Determination of serum triglycerides." Clin. Chem. J., 21(5).

23.Allain, P and Maurous, V (1979): "Microdetermination of lead, cadmium, copper, zinc and iron in blood and urine by graphite furunce atomic absorption spectrophotometer." Clin. Chem. Acta., 91:41.

24.AOAC Association of Official Analytical Chemists (1990): "Natural poisons." Chap. 26, Washington DC, USA.

25. Bancroft JD and Gamble M (2002): "Theory and practice of Histological Technique." 5th edition, Churchill Livingston, London, Edinburgh, New York, Philadelphia, St. Louis, Sydney.

26.Petrie A and Watson P (1999): "Statistics for Veterinary and Animal Science." 1st Ed., pp. 90-99, The Blackwell Science Ltd, United Kingdom.

27.Blood DC and Radostits, OM (1989): "Veterinary Medicine." 7th Ed. Ballire, Trindal.

28.Refaat M, Abou Elmagd M and Fahmy S (2006): "Mycotoxicosis and opportunistic organisms in Arabian foals. Egypt J. Comp. Path. and Clinic. Path. V. (19) No. (3).

29.Abou Salem M, Hamoda F and Tantawy A (1999): "A field study on mycotoxicoses in horses." Zagazig Vet. J. V. (27), No. (4).

30.Mogongo FO , Gombe KS and Ogaa JS (1985): The influence of cobalt vitamin B deficiency as stress or affect- Veterinary Laboratory Medicine and Clinical Pathology . 3rd Ed. Iowa State University press, Ames, Iowa USA.

31.Fell BF (1981): Pathological consequences of copper deficiency and cobalt deficiency . Rec. Vet. Sci. 10 : 303-305.

32.Duncan JR, Keith WP and Eduaried AM (1994): Veterinary laboratory Medicine and Clinical Pathology 3rd, Iowa State University Press, Ames , Iowa State , USA.

33.Hamam RJ , Judson GJ , Renter DJ, Mclaren LD and Mcfarlane JD (1980): Effect of Vit. B12 injections on the growth of young merino sheep . Aust. J. Agnc. Res. 31 : 347-355.

34.McDonald P, Edwards RA and Greennajg JFD (1979): "Animal Nutrition" Third Ed., E.L.BS impression 1979, Longman Group Ltd., London

35.Suttle NF (1994): "Meeting the copper requirements of ruminants." in Gamsworthy, P. C. and Cold, D. J. A. (eds.) Recent Advances in Animal Nutrition. Nottingham University Press, Nottingham, P. 173- 188.

36.Ali A, El-Deeb K and Abdin M (2004): "Pathological and biochemical changes in broilers feed ration contaminated with mycotoxins." 7th vet. Med. Zag. Conference.

37.Kuiper G (1995): "Mycotoxin: risk assessment and legislation." Toxicol. Lett. Vol. Dec. 82/ 853-859.

38.LeBars J and LeBars P (1996): "Recent acute and subacute mycotoxicosis

recognized in France." Vet. Res, 27 (4-5): 383-394.

39.Mehdi NA, Cartton QWW and Tuite J (1981): "Citrinin mycotoxicosis in broiler chickens." Food and cosmetics Toxicology 19 (6): 723-733.

40.Lebdah MA (1998): "Studies on some mycotic diseases affecting chicken at Sharkia Governorate." 4th Vet. Med. Zagazig. Congress.

41.Huff J E (1991): "Carcinogenicity of ochratoxin A in experimental animals." TARC scientific publications, 155: 229-244.

42.Boorman G, McDonald M, Imostom S and Persing R (1992): "Renal lesions induced by ochratoxin A exposure in the F344 rat. Toxicologic Pathology, 20, 236-245.

43.Kadri MM, Mohamed MH and Soroor FE (2000) "Pathologic studies on some lung affections in sheep in Sharkia Governorate: I-Mycotic and parasitic affections." Egypt. J. Comp. Pathol. & Clin. Pathol.,13 (1):17-26.

44.Thomson RG (Reginald G) (2001): "Thomson`s Special Veterinary Pathology." [edited by] McDonald McGavin, Willian W. Corltan, James F. Zachary 3rd ed.

45.*Deloach NR, Drewsk A and Nagi A (1987):* "Interaction of T-2 toxin with bovine carrier. Toxicol. Pharma (1987): 8: 123-131.

الملخص العربى

دراسات بيوكيميائية وهستوباثولوجية علي اغنام التسمين المصابة باضطرابات عصبية عضلية

جيهان هلال الصقار*، محمود محمد عرفه، صفوت فؤاد يوسف****

* قسم الباثولوجى - معمل بيطرى الزقازيق

** قسم الكيمياء – معهد بحوث صحة الحيوان

تم إجراء هذه الدراسة على 25 رأس من الأغنام تتراوح أعمارها من 9- 24 شهر فى محافظة القليوبية كانت تعانى من عدم القدرة على الحركة- وضعف عام ونقص فى الوزن واسهال تتغذى على عليقة ملوثة بالأوكراتوكسين بالإضافة إلى عدد (5) أغنام سليمة تتغذى على علائق متكاملة كمجموعة ضابطة. وقد تم دراسة الأعراض الإكلينيكية واخذ عينات من دم الأغنام المصابة كما تم اخذ عينات من الأحشاء الداخلية للحيوانات المذبوحة اضطراريا للفحص الباثولوجى وقد أسفرت النتائج عن ما يأتى:

بالنسبة لصورة الدم: لوحظ انخفاض معنوى لمستوى العدد الكلى لكرات الدم الحمراء الهيموجلوبين وحجم الخلايا المضغوطة فى الحيوانات المريضة. مما يوضح أن الحيوانات تعانى من انيميا. كما لوحظ انخفاض ملحوظ فى العد الكلى لخلايا الدم البيضاء ونسبة الخلايا القاعدية عند مقارنتها بالضابط بالنسبة للتحليل البيوكيمائى لمصل الدم وجد الامر:

أ- انخفاض معنوى فى مستويات الكالسيوم- الفوسفور- الماغنسيوم- الصوديوم- البوتاسيوم.

ب- انخفاض معنوى فى مستويات النحاس- الحديد- الزنك مع ارتفاع معنوى فى مستوى المولبيدنيوم فى الاغنام المصابة عند مقارنتها بالمجموعة الضابطة.

جـ- انخفاظ ملحوظ فى مستوى البروتين الكلى والالبيومين والجلوكوز والكولسترول والدهون الثلاثية.

د- ارتفاع ملحوظ فى انزيمات الكبد والفوسفات القلوى والكورتيزون مقارنة بالمجموعة الضابطة.

كما أجريت دراسة باثولوجية على الأعضاء الداخلية وثبت فيها وجود نزف بؤرى بالكبد وحبيبات السيتوبلازم بالكبد ونخر ببعض خلايا الكبد مع اتساع بالقناة المرارية. كما لوحظ نخر فى الطلائية المبطنة لأنابيب الكلى أو نخر بؤرى بالانابيب مع تغلغل خلوى كما لوحظ زيادة سمك الطلائية المبطنة للشعب الهوائية بالرئة مصاحب بتراكم مخاط داخل الشعب. بالإضافة إلى اتساع الحويصلات الهوائية وبالطحال شوهد نزف بؤرى أو نخر بالإضافة إلى نخر لبطانة الأمعاء مصحوب بتغلغل خلوى أو نخر بالأهداب المعوية. أما بالمخ فقد شوهد احتقان بالأوعية الدموية ونزف بؤرى

ومن الدراسة يتضح أن العلائق الملوثة بسموم الاوكراتوكسين المقدمة للأغنام والتى بها أيضا نقص شديد فى الأملاح الأساسية والنادرة حيث توجد نسبة لا تكفى لاحتياجاتها مما أدى الى ظهور العرج وعدم القدرة على الحركة الذى يستلزم معه ضرورة التصدى لتلك الظاهرة واتخاذ الاحتياطات اللازمة لمقاومة تلك الأعراض من خلال إعطاء مثل هذه الحيوانات مركبات وفيتامينات واملاح تعويضية لهذا النقص حتى تتلافى الخسائر الاقتصادية الناتجة بحيث ان تكون العلائق خالية تماما من وجود السموم الفطرية المختلفة وبخاصة سموم الاوكراتوكسين.