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CLINICOPATHOLOGICAL STUDIES ON CAPRINE AFLATOXICOSIS

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SUMMARY

Out of 80 diseased goats, aflatoxin was detected in serum of 50% of them. Examination of 600 samples of feed utilized by these animals yielded aflatoxin and toxigenic A. flavus in 52.2% of the samples. Experimental induction of aflatoxicosis in goats gave similar symptoms which were observed in field animals. Biochemical and haematological examinations of blood of experimentally intoxicated goats revealed significant alteration in most of the examined values including liver and kidney function tests, total protein, albumin and globulin. Histopathological examination of internal organs of these animals showed obvious changes in all tissues particularly in the liver and kidney.

INTRODUCTION

Aflatoxicosis represents one of the most causes of serious mycotoxicosis in man as well as in poultry, livestock, and other animals (Madhaven et al., 1965). It produced by strains of Aspergillus flavus and Aspergillus parasiticus, which are frequent contaminants of harvested feeds and food stored under conditions of high humidity and temperature (Hassan et al., 2002 and Hassan and Mogda, 2003).

The susceptibility of animals to aflatoxin is affected by species-breed variations and nutritional factors (Raisbeck et al., 1991 and Pier, 1992). The susceptibility of different animal species to aflatoxin appears to be variable. Hepatic damage was the principal injury induced in all animals; however, acute poisoning was manifested as icterus of the mucous membranes and widespread haemorrhages (Newberne et al., 1973).

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The aim of the present work, therefore, was detection of aflatoxicosis in kids as well as studying the effect of aflatoxin on the clinicopathological parameters and tissue organs of kids.

MATERIALS AND METHODS

Serum samples:

A total of 80 samples of serum were collected from diseased conditions suspected of having mycotoxicosis, at Borg El Arab and El Wadi El Gedid goat farms during the last 3 years. Symptoms included high mortality and morbidity rates, diarrhoea, salivation, cough, sneezing with mucopurulent discharges and dyspnea. The collected serum samples were analysed for measurement of aflatoxins.

Grains and feed stuffs samples:

A total of 600 samples of different grains and feedstuffs which were used by infected animals were collected for measurement of aflatoxins and isolation of mould. These included 50 samples from each of the following (Yellow corn, barley, wheat, beans, soybean, hay, straw, barseem, tibn, cotton seed cake, wheat bran and mixed feed).

Experimental animals:-

Ten male, healthy, cross breed kids weighing 15-18 kg, housed under hygienic conditions, and fed on balanced ration and water ad libitum were used.

Detection of aflatoxins in serum:

Aflatoxins in serum of suspected animals was extracted according to the method described by Hansen (1993) using the immunoaffinity column.

Isolation of mould from contaminated grains and feed stuffs:

Mould and yeast colonies particularly Aspergillus flavus and Aspergillus parasiticus were isolated and identified using methods recommended by Conant et al. (1954).

Detection of aflatoxin in feed:

Extraction and detection of aflatoxin in feed was performed as recommended by Gabal et al. (1994).

Experimental induction of caprine aflatoxicosis (Clark et al., 1984):

The total number of male cross breed domestic kids (10) was randomly classified into two groups. The first group consisted of 2 animals and kept under healthy conditions as control individuals whereas the other 8 animals were given aflatoxins orally in 3 doses (mg/kg of body weight/day), 0.1 for 34 days; 0.2 for 18 days and 0.4 for 10 days as gradual increasing doses of aflatoxins. During the experimental work, changes in behaviour, feed intake, clinical signs and body weight gain were reported. At the end of the experiment, the goats were sacrificed and post-mortem as well as histopathological examinations were performed.

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Haematological and serum biochemical studies:

Blood was collected by jugular vein puncture (with and without anticoagulant) 5 times during the experimental period (0 time, 15 and 34 days after the 1st dose, 15 days after the 2nd dose and 7 days after the 3rd dose). Haemogram was estimated after Jain (2000). Serum was separated for determination of the following parameters; total protein according to the Biuret method after Henry (1957), serum albumin according to Webster (1974), serum globulins were calculated by subtracting the obtained value of albumin from the values of total protein. Albumin /Globulin ratio (A/G ratio) was calculated according to the results of albumin and globulin. Serum enzyme activities (AST, ALT & ALP) were determined according to Reitman and Frankel (1957) and Belfield and Goldbery (1971), serum cholesterol was assayed after Fasce (1982). Serum triglycerides according to Young and Paster (1975), serum bilirubin after Jendrassik et al. (1983), serum creatinine following the method of Henry (1974). Serum urea was determined after Tabacco et al. (1979), serum calcium after Stern et al. (1957) and serum inorganic phosphorus according to Yee (1968).

Histopathological examination:

Formalin fixed tissue specimens were embedded in paraffin and sectioned at 4-6 microns thickness. Sections were stained by Haematoxylin and Eosin stain for routine histopathological examination (Drury et al., 1976).

Statistical analysis:

Data of haematological and serum biochemical parameters of control and treated groups were statistically analyzed according to Petrie and Watson (1999).

RESULTS AND DISCUSSION

The results of the present work are shown in Tables (1-7) and Figures (1-4).

Table (1): Detection of aflatoxin in serum of infected animals by immuno-affinity column.

Total No. of examined animals	+ve animals		Amount of aflatoxin in serum of diseased animals (ppb)		
	Number	%	Max	Min	Mean
80	40	50	9.3	3.6	5.38

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Table (2): Toxigenicity of *A. flavus* species recovered from feed stuffs.

Samples tested*	Total No. of tested samples = 50				Mean amount of each type of aflatoxin (ppb)				
	+ve for *A. flavus*		-ve for aflatoxin						
	No.	%	No.	%	B1	G1	B2	G2	Total
Yellow corn	25	50	25	50	525	-	110	90	725
Barley	10	20	9	18	800	77.50	-	-	877.50
Wheat	25	50	20	40	925	-	345	155	1425
Beans	5	10	5	10	130	-	-	25	155
Soybean	35	70	-	-	-	-	-	-	-
Hay	10	20	5	10	176.7	-	-	-	176.70
Straw	15	30	-	-	-	-	-	-	-
Barseem	15	30	-	-	-	-	-	-	-
Tibn	15	30	11	22	750.75	-	-	-	750.75
Cake	20	40	-	-	-	-	-	-	-
Wheat bran	45	90	36	72	430	932	780	288	2430
Mixed feed	29	58	19	38	681.5	-	-	50	731.50

*50 samples of each were examined.

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Table (3) Changes in body weight gain of treated goats with aflatoxin (mean ± S.E.).

Time (day)		Mean body weight (kg)
0 Time	Control	18.08 ± 0.67
	Treated	17.32 ± 0.58
15 days after 1st dose	Control	20.19 ± 0.68
	Treated	18.14 ± 0.61*
34 days after 1st dose	Control	25.42 ± 0.43
	Treated	20.06 ± 0.28*
15 days after 2nd dose	Control	29.14 ± 0.59
	Treated	22.58 ± 0.76*
7 days after 3rd dose	Control	32.14 ± 0.52
	Treated	26.15 ± 0.64*

* Significantly different from the control $P < 0.05$

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Table (4) Changes in haemogram of goats treated with aflatoxin (mean ± S.E.).

Parameter / Time		Hb (g/dl)	PCV (%)	RBCs ($X10^6$/µl)	MCV (fl)	MCH (pg)	MCHC (g%)
0 Time	Control	8.4 ± 0.08	36.24 ± 1.04	14.3 ± 0.66	25.68 ± 1.60	5.92 ± 0.34	23.2 ± 0.67
	Treated	8.2 ± 0.11	34.86 ± 1.20	13.7 ± 0.69	25.70 ± 1.63	6.04 ± 0.30	23.6 ± 0.66
15 days after 1st dose	Control	8.8 ± 0.38	38.04 ± 1.11	14.02 ± 0.61	27.28 ± 1.23	6.46 ± 0.43	23.2 ± 0.89
	Treated	7.7 ± 0.23*	39.10 ± 0.98	14.40 ± 0.55	27.29 ± 1.12	5.40 ± 0.30*	19.9 ± 0.98*
34 days after 1st dose	Control	9.9 ± 0.12	37.68 ± 1.08	15.94 ± 0.41	23.68 ± 0.81	6.20 ± 0.12	26.32 ± 0.60
	Treated	8.16 ± 0.42*	34.60 ± 0.88*	13.90 ± 0.14*	24.90 ± 0.79	5.90 ± 0.26	23.70 ± 1.47
15 days after 2nd dose	Control	10.12 ± 0.17	38.64 ± 0.86	15.56 ± 0.50	24.94 ± 1.05	6.50 ± 0.19	26.20 ± 0.82
	Treated	8.03 ± 0.43*	36.40 ± 0.73*	13.78 ± 0.43*	26.50 ± 0.84	5.85 ± 0.36*	22.02 ± 0.92*
7 days after 3rd dose	Control	10.68 ± 0.30	37.46 ± 0.85	16.32 ± 0.47	23.08 ± 1.20	6.56 ± 0.19	28.56 ± 0.92
	Treated	9.94 ± 0.09	37.28 ± 0.83	15.32 ± 0.40	24.40 ± 0.90	6.50 ± 0.15	26.69 ± 0.46*

* Significantly different at $P < 0.05$

Table (5) Changes in total and differential leucocytic count in goats treated with aflatoxin (Mean ± S.E.).

Parameter / Time		WBCs (X10³/µl)	Differential leucocytic count (X10³/µl)			
			Neutrophil	Lymphocyte	Monocyte	Eosinophil
0 Time	Control	9.62 ± 0.52	3.60 ± 0.09	5.1 ± 0.71	0.85 ± 0.05	0.15 ± 0.05
	Treated	10.12 ± 0.66	3.40 ± 0.63	5.8 ± 1.21	0.79 ± 0.15	0.09 ± 0.02
15 days after 1st dose	Control	10.24 ± 0.33	3.20 ± 0.28	5.7 ± 1.06	1.16 ± 0.26	0.08 ± 0.02
	Treated	10.92 ± 0.58	4.60 ± 1.02	4.8 ± 1.42	1.33 ± 0.39	0.00
34 days after 1st dose	Control	9.86 ± 0.50	3.30 ± 0.50	3.0 ± 0.98	1.60 ± 0.63	0.00
	Treated	12.24 ± 0.57*	5.90 ± 0.43*	4.6 ± 1.10	1.64 ± 0.84	0.00
15 days after 2nd dose	Control	12.12 ± 0.35	4.20 ± 0.42	5.6 ± 0.63	2.29 ± 0.28	0.00
	Treated	14.78 ± 0.46*	6.40 ± 0.56*	6.1 ± 1.02	2.15 ± 0.53	0.00
7 days after 3rd dose	Control	11.94 ± 0.63	4.00 ± 0.86	6.0 ± 0.62	1.95 ± 0.63	0.00
	Treated	10.46 ± 0.37*	4.60 ± 1.16	4.3 ± 0.42*	1.58 ± 0.47	0.00

* Significantly different at P < 0.05

Table (6) Changes in serun proteins and enzyme activities of goats treated with aflatoxin (mean ± S.E.).

Parameter / Time		T. protein (g/dl)	Albumin (g/dl)	Globulin (g/dl)	A/G ratio	ALT (u/l)	AST (u/l)	ALP (u/l)
0 Time	Control	5.9 ± 0.27	3.20 ± 0.13	2.74 ± 0.21	1.20 ± 0.11	66.1 ± 1.37	30.66 ± 0.91	67.3 ± 2.65
	Treated	6.32 ± 0.25	3.18 ± 0.16	3.14 ± 0.26	1.05 ± 0.12	66.3 ± 0.87	31.42 ± 1.15	68.3 ± 3.44
15 days after 1st dose	Control	6.9 ± 0.37	3.59 ± 0.06	3.29 ± 0.40	1.17 ± 0.17	67.8 ± 0.48	31.1 ± 0.48	66.22 ± 3.67
	Treated	5.9 ± 0.23*	3.13 ± 0.05	2.81 ± 0.19	1.13 ± 0.05	68.96 ± 0.33*	34.6 ± 0.66*	75.14 ± 3.03*
34 days after 1st dose	Control	6.82 ± 0.17	4.22 ± 0.12	2.62 ± 0.50	1.61 ± 0.05	67.5 ± 0.83	30.8 ± 0.34	68.6 ± 3.63
	Treated	5.76 ± 0.21*	3.03 ± 0.21*	2.73 ± 0.14	1.11 ± 0.07*	77.6 ± 0.87*	36.98 ± 0.87*	87.58 ± 3.59*
15 days after 2nd dose	Control	6.9 ± 0.13	4.45 ± 0.18	2.45 ± 0.04	1.82 ± 0.04	68.5 ± 0.92	31.26 ± 0.27	80.92 ± 3.55
	Treated	5.6 ± 0.18*	3.02 ± 0.23*	2.62 ± 0.02	1.15 ± 0.08*	79.8 ± 0.74*	38.14 ± 0.57*	102.94 ± 3.48*
7 days after 3rd dose	Control	6.94 ± 0.12	4.38 ± 0.09	2.56 ± 0.03	1.71 ± 0.03	68.3 ± 1.02	33.12 ± 0.43	79.96 ± 0.51
	Treated	5.01 ± 0.17*	2.95 ± 0.05*	2.06 ± 0.03*	1.43 ± 0.04*	77.96 ± 1.18*	40.36 ± 0.71*	100.18 ± 4.46*

* Significantly different at $P < 0.05$

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Table (7) Changes in some serum biochemical parameters of goats treated with aflatoxin (mean ± S.E.).

Parameter / Time		Cholesterol (mg/dl)	Triglycerides (mg/dl)	Bilirubin (mg/dl)	Phosphorus (mg/dl)	Calcium (mg/dl)	Urea (mg/dl)	Creatinine (mg/dl)
0 Time	Control	212.0 ± 3.23	101.93 ± 1.58	0.12 ± 0.02	3.80 ± 0.17	6.77 ± 0.79	19.77 ± 0.15	0.96 ± 0.03
	Treated	209.6 ± 5.89	102.78 ± 1.87	0.11 ± 0.01	3.77 ± 0.21	6.98 ± 0.66	19.83 ± 0.20	0.99 ± 0.04
5 days after 1st dose	Control	210.2 ± 5.34	101.70 ± 1.38	0.16 ± 0.03	3.84 ± 0.23	8.45 ± 0.59	20.27 ± 0.61	1.12 ± 0.03
	Treated	220.2 ± 6.50	104.20 ± 1.19	0.18 ± 0.02	3.79 ± 0.22	7.62 ± 0.57	21.13 ± 0.75	1.16 ± 0.05
14 days after 1st dose	Control	215.4 ± 4.38	106.08 ± 1.83	0.19 ± 0.04	4.08 ± 0.31	8.95 ± 0.40	20.93 ± 0.14	1.21 ± 0.02
	Treated	228.2 ± 4.40*	114.34 ± 1.46*	0.26 ± 0.03	2.66 ± 0.25*	7.97 ± 0.24*	25.27 ± 0.21*	1.30 ± 0.01*
15 days after 2nd dose	Control	219.8 ± 3.79	110.28 ± 1.26	0.22 ± 0.02	4.03 ± 0.10	9.02 ± 0.35	21.41 ± 0.14	1.31 ± 0.02
	Treated	232.6 ± 3.40*	121.94 ± 1.91*	0.29 ± 0.02*	3.20 ± 0.15*	8.12 ± 0.23*	29.1 ± 0.11*	1.43 ± 0.04*
7 days after 3rd dose	Control	223.0 ± 2.29	118.20 ± 1.20	0.39 ± 0.01	4.30 ± 0.19	8.93 ± 0.19	20.88 ± 0.25	1.34 ± 0.01
	Treated	238.8 ± 3.91*	127.50 ± 1.05*	0.46 ± 0.02*	3.63 ± 0.21*	8.01 ± 0.27*	22.44 ± 0.33*	1.50 ± 0.02*

* Significantly different at P < 0.05

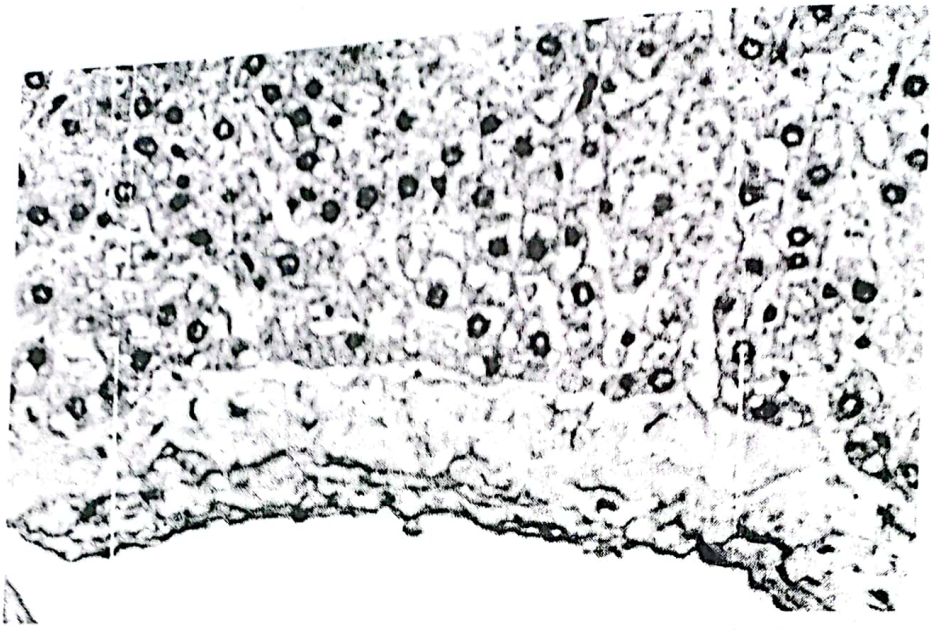

Fig. (1): Liver of goat administered aflatoxin showing proliferated fibrous tissue around the bile duct with partial replacement of the hepatic parenchyma (H & E, X 40).

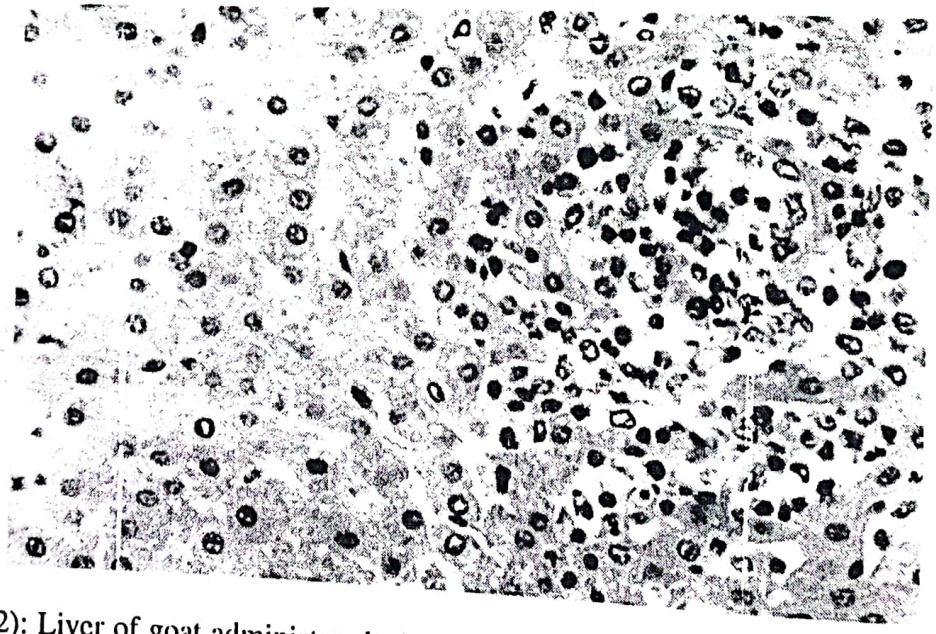

Fig. (2): Liver of goat administered aflatoxin showing oedema, inflammatory cells infiltration and hyperplastic epithelial lining of the bile ducts (H & E, X 40).

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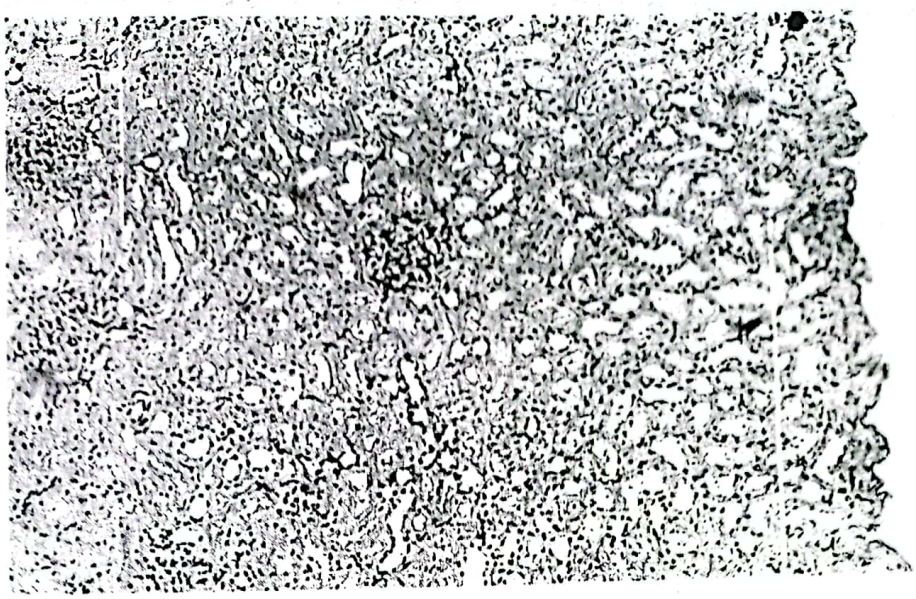

Fig. (3): Kidney of goat administered aflatoxin showing thickened and fibrotic renal capsule at the end of the experimental period (H & E, X 40).

Fig. (4): Kidney of goat administered aflatoxin showing fibrosis among the tubules (H & E X 40).

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In the present work, serum examination of 80 cases suspected to be under outbreak of mycotoxicosis, revealed that 40 cases (50%) were affected with aflatoxins at a mean level of 5.38 ppb (max. 9.3 ppb and min. 3.6 ppb). The detection of aflatoxins in blood and milk of farm animals was previously reported by Sabino et al. (1995) and Fernandez et al. (1997) who detected aflatoxin B1 and M1 in urine and faeces at levels 61.82 ppb and 27 ppb, respectively.

Mycological examination of 600 samples of different feed stuffs (collected from these farms) revealed the isolation of 1110 fungal isolates representing 8 genera and 6 species. The genus Aspergillus was the most predominant isolate recovered from the examined samples (62.3%) followed by Mucor spp (54.7%) and Penicillium spp (35.3%). Similar findings have been reported by other authors (Hassan, 1990; Aja and Emejuaiwe, 1994; Lisker et al., 1994; Hassan et al., 2002 and Hassan and Mogda, 2003).

It is interesting to report here that species of A. flavus group were the most common isolates (41.5%). The isolated Aspergillus flavus strains produced different levels of aflatoxins particularly those isolated from wheat bran gave the highest yield of aflatoxins (2430 ppb), followed by Aspergillus flavus isolated from wheat, barley, tibn, mixed feed and yellow corn (1425, 877.50, 750.75, 731.50 and 725 ppb, respectively). In contrast, A. flavus isolated from hay and bean gave the lowest yield of aflatoxin (176.50 and 155 ppb, respectively), (Table, 2). These results are in accord with results of several authors, Hassan (1990); Abramson et al. (1992) and Hassan (1998).

Goats exposed to aflatoxin showed clinical symptoms including salivation, dry cough, sneezing, dullness, weakness with slowing of approach avoidance behaviour and loss of appetite. Diseased goats started to lose their normal vitality after receiving the first dose of the toxin, with reduction in the mean body weight and feed consumption than that of control.

Post mortem examination revealed that, the liver was enlarged and congested, the kidneys were pale but the cortex was congested, the gall bladder was distended and engorged with bile while the heart was flabby with hydropericardium and the lung was oedematous, congested with presence of severe, petechial haemorrhages. Such observations coincide with those of Abdel Salam et al. (1989).

Results of blood picture in the present study revealed that red blood cells (RBCs), haemoglobin (Hb) and packed cell volume (PCV) were decreased during the experimental period which

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come in parallel with the results of Roger et al. (1991) in lambs and Brucato et al. (1986) in calves and may have been occurred due to either blood loss or kidney and liver affection.

Values of serum total protein, albumin and globulins were decreased significantly with continuous administration of aflatoxins which correlated well with the inhibitory effect of aflatoxin on protein synthesis, and liver damage (Neathery et al. 1980). Reduction of albumin values resulted in decrease in A/G ratio. It has been reported that aflatoxin interfer with carbohydrate oxidation, a process which is essential for providing energy needed for protein synthesis (Fernandez et al. 1997).

Activities of ALT, AST and AP were increased and it has been reported to occur due to altered permeability of hepatocytes (Roger et al. 1991; Edrington et al. 1994) and myocytes (Harvey et al. 1995). Elevation of AP also may be due to hepatic necrosis caused by aflatoxin administration leading to thickening of bile ducts and intrahepatic colestasis. Elevation of bilirubin, cholesterol, triglycerides probably occurred due to hepatic disease. Similar findings have been reported in sheep (Suliman et al. 1987; Roger et al. 1991), goats (Clark et al. 1984) and cattle (Helferich et al. 1986).

The significant elevation in serum cholesterol and triglycerides due to aflatoxicosis may result in different degrees of arteriosclerosis, phlebitis and venous occlusion which leads to insufficient blood supply specially to the lung tissues, therefore the exchange of gases in lung alveoli becomes inadequate (Roger et al., 1991 and Edrington et al., 1994).

The obtained results concerned with kidney function tests demonstrated significant increase in serum urea and creatinine values which indicate circulatory insufficiency or nephropathy (Suliman et al. 1987; Soliman, 1998). The kidney affection was supported by histopathological examination of the same samples.

Aflatoxins are believed to decrease the availability or quantity of bile salts in the gastrointestinal tract, resulting in decreased absorption of fat soluble vitamins (Cheeke and Shull, 1985). If vitamin D assimilation is impaired, Ca and P absorption may likewise decrease, which may explain the lowered blood. Ca and P concentrations seen in treated goats in this study.

The liver and kidney affection was supported by histopathological examination which revealed thickening and sclerosis in the wall of the blood vessels and hyperplastic bile duct. The hepatocytes showed circumscribed round vacuoles in the

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cytoplasm (fatty change) and focal inflammatory cells infiltration inbetween the atrophied hepatocytes (Soliman, 1998) (Figs, 1-4).

In conclusion, from the forgoing results it appears that aflatoxicosis in goat affects the production, biochemical parameters and tissues of internal organs. Therefore, the breeding of goat, as other animals, must be given more care to avoid aflatoxicosis by using healthy feeds and clean environment.

REFERENCES

Abdel-Salam; E.B.; El-Tayeb, A.E.; Nor Eldin, A.A. and Abdel-Magide, A.M. (1989): "Aflatoxicosis in fattening sheep." Vet. Rec., 124 (18): 487-488.

Abramson, I.; Richter, W.; Rintelen, J.; Sinha, R.N. and Schuster, M. (1992): "Ochratoxin A production in Bavarian Cereal grains." Arch. Environ. Contamin. Toxicol., 23 (2): 259-263.

Aja, N.W. and Emejuaiwe, S.O. (1994): "Aflatoxin producing fungi associated with Nigerian maize." Environ. Toxicol. Water Quality, 9 (1): 17-21.

Belfield, A. and Goldberg, D.M. (1971): "Method for determination of alkaline phosphatase." Enzyme, 12: 561-564.

Brucato, M.; Sundlof, S.F.; Bell, J.V. and Edds, G.T. (1986): ìAflatoxin B1 toxicosis in dairy calves pretreated with selenium-vitamin E.î Amer. J. Vet. Res., 47 (1): 179-183.

Cheeke, P.R. and Shull, L.R. (1985): "Natural toxicants in feeds and poisonous plants." AVI Publishing Westport, CT, pp. 393-477.

Clark, J.D.; Hatch, R.C.; Miller, D.M. and Jain, A.V. (1984): "Caprine aflatoxicosis: experimental disease and clinical pathologic changes." Amer. J. Vet. Res., 45 (6): 1132-1135.

Conant, N.F.; Martin, D.S.; Smith, D.T.; Baker, R.D. and Callaway, J.L. (1954): "Manual of Clinical Mycology." Philadelphia, W.B. Saunders Company, USA.

Drury, R.A.; Wallington, E.A. and Cancersor, R. (1976): "Carleton's Histological Techniques." 4th Ed., Oxford University Press, London, New York, Toronto.

Edrington, T.S.; Harvey, R.B. and Kubena, L.F. (1994): "Effect of aflatoxin in growing lambs fed ruminally degradable or escape protein sources." J. Anim. Sci., 72 (5): 1274-1281.

Fasce, C.F. (1982): "Colorimetric method for determination of serum cholesterol." Clin. Chem., 18: 901-906.

Fernandez, A.; Belio, R.; Ramos, J.J.; Saez, M.C. and Sanz, T. (1997): "Aflatoxins and their metabolites in the tissues, faeces and urine from lambs feeding on an aflatoxin contaminated diet." J. Sci. Food Agric., 74 (2): 161-168.

Gabal, M.A.; Hegazy, S.M. and Nagwa, Y. Hassanien (1994): "Aflatoxin production by Aspergillus flavus field isolates." Vet. Human Toxicol., 36: 519-521.

Hansen, T.J. (1993): "Quantitative testing for mycotoxins." VICAM, American Association of Cereal Chemists, Inc., 38 (5): 40-44.

Harvey, R.B.; Edrington, T.S.; Kubena, L.F.; Elissalde,

CamScanner

M.H.; Corrier, D.E. and Rottinghaus, G.E. (1995): "Effect of aflatoxin and diacetoxyscirpenol in ewe lambs." Bull. Environ. Contam. Toxicol., 54 (3): 325-330.

Hassan, A.A. (1990): "Fungal contamination of food." M.V.Sc. Thesis, Mic. Fac. Vet. Med., Cairo Univ.

Hassan, A.A. (1998):"Mycosis in turkeys." Vet. Med. J. Giza, 46 (4B): 857- 865.

Hassan, A.A. and Mogda, K.M. (2003): "New trials of use of molasses and garlic extracts for compating mycotoxicosis" Kafr El- Shikh. Vet. Med. J., 1(1): April 2003.

Hassan, A.A.; Karatum, K.M. and El- Khawaga, A. (2002):"Effect of selenium in broiler chicken fed diet containing F. monilfarne culture material supplied known level of Fumonisin B1" Egypt. J. Comp. and Clinic. Path., 15 (1): 98- 110.

Helferich, W.G.; Baldwin, R.L. and Hesieh, D.P.H. (1986): "Aflatoxin B1 metabolism in lactating goats and rats." J. Anim. Sci., 62 (3): 697-705.

Henry, R.J. (1957): "Colorimetric method for determination of serum total protein." Anal. Chem., 62: 1491.

Henry, R.J. (1974): "Clinical Chemistry, Principles and Techniques." 2nd Edition, Harper and Row, p. 525.

Jain, N.C. (2000): "Schalm's Veterinary Haematology." 5th Ed., Lea and Fenbiger, Philadelphia, PA.

Jendrassik, I. et al. (1983): "Determination of serum bilirubin." Biocehm. 72: 97-81.

Lisker, N.; Michaeli, R. and Frank, Z.R. (1994): "Aspergillus flavus and other mycoflora of groundnut kernels in Israel." Mycotoxin Res., 10 (1): 47-52.

Madhaven, T.V.; Garner, R.C. and Holscher, M.A. (1965): "Effect of prednisolone on liver damage in rats induced by aflatoxin." J. Pathol. Bacteriol., 93 (8): 443-447.

Neathery, M.W.; Moss, W.H.; Wyatt, R.W.; Miller, Gentry, R.P.; Smith, O. and Heinmiller, S. (1980): "Aflatoxicosis and zinc metabolism in dairy calves." J. Dairy Sci., 63 (1): 145-146.

Newberne, P.M.; Wogan, G.N. and Hall, A.H. (1973): "Chronic aflatoxicosis in sheep." J. Am. Vet. Med. Assoc., 163 (11): 1262-1270.

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

Pier, A.C. (1992): "Major biological consequences of aflatoxicosis in animal production." J. Anim. Sci., 70 (5): 3964-3770.

Raisbeck, M.F.; Rottinghaus, G.E. and Kendall, J.D. (1991): "Effect of naturally occurring mycotoxins on ruminants." In: J.E. Smith and R.S. Henderson (Ed): Mycotoxins and Animal Foods. Pp. 647-677. CRC Press, Boca Raton, FL.

Reitman, S. and Frankel, S. (1957): "A colorimetric determination of serum glutamic oxaloacetic and glutamic pyruvic transaminase." Am. J. Clin. Path., 28: 56-58.

Roger, C.H.; Leon, F.K.; Timothy, D.P. and William, E.H. (1991): "Diminution of aflatoxin toxicity to growing lambs by dietary supplementation with hydrated sodium calcium aluminosilicate." Am. J. Vet. Res., 52 (1): 152-156.

Sabino, M.; Purchio, A. and Milanez, T.V. (1995): "Aflatoxins B1, M1 and aflatoxicol in tissues and urine of calves receiving aflatoxin." Food Add. and Contam., 12 (3): 467-472.

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Soliman, A.A. (1998): "Effect of aflatoxins in contaminated rations in performance of sheep." Ph.D. Thesis, Animal Nutrition, Fac. Agric., Cairo University.

Stern, J.; Dawra, P.K. and Dollear, F.C. (1957): "Determination of serum calcium." Clin. Chim. Acta, 2: 576.

Suliman, H.B.; Mohamed, A.F.; Awadelsied, N.A. and Shommein, A.M. (1987): "Acute mycotoxicosis in sheep: Field cases." Veterinary and Human Toxicology, 29 (3): 241-243.

Tabacco, A.; Frank, Z.R. and Zuber, M.S. (1979): "Determination of serum urea." Clin. Chem., 25 (2): 336.

Webster, D. (1974): "Determination of serum albumin." Clin. Chim. Acta, 53: 109.

Yee, H.Y. (1968): "Determination of serum inorganic phosphorus." Clin. Chem., 14: 898.

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

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