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AFLATOXICOSIS AS A FIELD PROBLEM IN SOME COW FARMS IN KAFR EL SHEIKH PROVINCE

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Abstract

Analysis of diet samples used in feeding private dairy cows in Desouk in Kafr El Sheikh Province showed that rations were contaminated with aflatoxin (75-150 p.p.b.) which was reflected on physiological blood parameters showing low level of red blood corpuscles (R.B.Cs.), Mean corpuscular haemoglobin concentration (MCHC) and high level of packed cell volume (P.C.V.), mean corpuscular volume (M.C.V.) and mean corpuscular haemoglobin (M.C.H.). In contaminated group, serum showed low level of total protein, iron, copper, magnesium, sodium and potassium, as well as, high level of asparate amino transferase (AST) and alkaline phosphatase live enzymes.

INTRODUCTION

Lactating cows occasionally are subjected to eat hazardous aflatoxin which leads to loss of weight and decrease in milk yield. Lynch *et al.* (1970) reported that levels of 0.02 mg/kg of aflatoxin or more decreased weekly body weight, while, serum alkaline phosphatase value increased. Polan *et al.* (1974) fed concentrate containing either 10, 50, 250 or 1250 p.p.b of aflatoxin B1 (AFB_1) for 14 days and they found that AFM1 in milk increased to day 4 with little change to day 14 in cows fed 250 and 1250 p.p.b of AFB_1. Traces of AFM_1 were found at the 50 p.p.b dietary level and none at 10 p.p.b and the concentration AFB1 must exceed 46 p.p.b for AFM_1 to be detectable in milk. Moorthy *et al.* (1984) reporteted that aflatoxin in a powdered rice culture (NRRL 2999) was added to the feed of calves (1 year old

35-40 kg body weight) at 10 µg / kg body weight for up to 514 days. The cattle calves remained apparently healthy and were killed between 512 and 514 days. Post mortem revealed fatty changes in the liver with mild hyperplasia.

Galhotra *et al.* (1986) administrated 0.25 mg aflatoxin/kg body weight in 3 divided doses on alternate days to six healthy male buffalo-calves. Four of the dosed calves died between 4th and 8th days, 2 were killed on the 10th day. They showed an increase of alkaline phosphatase and acid phosphatase in non-Kupffer cells. Hepatorenal injury caused loss of liver serum enzymes. Adam (1988) mentioned that cows fed mainly on maize, silage supplemented with minerals and ground peanut cake containing very high concentration of aflatoxin B1 ranging from 130 to 295 mg/kg brought liver malfunction.

Rajendran *et al.* (1992) recorded outbreak of aflatoxicosis in Jersy cattle. The clinical signs included rejection of food, lethargy, decreased milk yield, ascites and abortion, and of 117 animals affected, 55 (47%) died after acute or subacute or chronic illness. The liver was affected in all cases. The outbreak coincided with a new consignment of feed in which up to 5 p.p.m of aflatoxin was found. Teglia *et al.* (1995) produced experimental aflatoxicosis in 9 rabbits with a mean weight of 4 kg using oral doses of 400 µg of aflatoxin B1. The histopathology of the lesions were found in the liver, kidneys and heart. Sahoo *et al.* (1996) fed graded levels of crude aflatoxins to young healthy Newzeland White rabbits. The aflatoxin treated rabbits showed lymphopenia with compensatory neutrophilia and reduction in total serum protein concentration.

The aim of this work is to investigate changes in physiological some parameters in blood of dairy cows as influenced by aflatoxin contaminated rations, as well as, to determine aflatoxin concentration in the rations.

MATERIALS AND METHODS

Private milk farms claimed of the drop of milk yield and debility of dairy cows. The anmials were not protozoal-infested, and were without symptoms of diseases. These animals, early lactating were kept on dry rations; they were of nearly the same age.

The animals were divided into groups in two regions in Desouk, the first group ate contaminated dry rations with aflatoxin (Gl, 40 animals), and the second group

(G2, 40 animals), healthy group ate dry ration free from aflatoxin and kept as control.

Analysis of aflatoxin in dry rations was chemically estimated according to Robberts and Patterson (1975).

Blood samples were taken from all animals for haematological and biochemical analysis according to the following methods:

Red blood corpusles (RBCs), haemoglobin, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC) by Schalm (1975); packed cell volume (PCV) by Moxine and Benjamine (1970); iron by Ramsay (1958); copper by Khalifa *et al.* (1972); calcium as described by Bett and Fraser (1959); inorganic phosphorus by Klichling and Freiburg (1952); magnesium by Neil and Neely (1956); sodium and potassium by Flame photometery using 410C flame photometer by Oser (1979); total proteins by Hoffmann and Richterrich (1970); liver enzymes, alkaline amino transferase (ALT) 4, asprate transferase (AST) according to Reitiman and Frankel (1957) and alkaline phosphatase by Kilichling and Freiburg (1952) were adopted. Statistical analysis of data for mean, standard error and "t" test was carried out according to Snedecor and Cochran (1976).

RESULTS

Table 1 shows blood parameters with low level of RBCs, MCHC, high level of PCV, MCV, MCHC, Low level of serum iron and copper in first group (G1).

Table 2 shows serum parameters with low level of total protein, magnesium, sodium and potassium in first group (G1).

In table 3, liver enzymes show increase in level of AST and alkaline phosphatase in serum of blood of cows fed on aflatoxin contaminated dry ration.

On analysing the ration of affected cows, it was found to contain from 75 to 15 ppb aflatoxin.

DISCUSSION

Our investigation revealed that, in dairy cows fed on aflatoxin contaminated ration, there were low number of RBCs, MCHC, high level of PCV, MCV, MCHC which are indicative to nutritional anaemia as reflected in low iron and copper values in serum of affected animals as described by Coles (1989).

Table 1. Effect of aflatoxin contaminated rations on some haematological parameters, iron and copper of dairy cows.

Values	Group 1 N = 40	Group 2 (control) N = 40	T.value
RBCs 10^6 µl	4.15±0.52***	10.56±1.40	P<0.001
Haemoglobin Hb g/d	10.55±0.57	10.43±0.36	N.S.
PCV %	45.51±1.00***	31.21±2.63	P<0.001
MCV Fl	126.09±7.59***	3.03±1.71	P<0.001
MCH Pg.	30.41±1.27	10.59±0.42	P<0.001
MCHC g/dl	25.19±1.5**	34.79±2.00	P<0.01
iron mcg/dl	107.74±7.17**	244.27±0.33	P0.001
Copper mcg /dl	62.16±0.33**	89.08±1.37	P<0.01

N.S. = Not significant

Moreover, there were reduction in magnesium, total protein sodium and potassium due to aflatoxin which reduced food intake and interfered with food nutrients. Reduced total protein may be due to insufficient protein synthesis in the liver. Lynch et al. (1970) reported that, weekly body weight loss happened at level of 0.02 mg/kg or more of aflatoxins. Blood *et al.* (1989) recorded that aflatoxin reduces food intake, weight gains and milk production. Rajendran *et al.* (1992) studied outbreak of aflatoxicosis in a herd of 162 Jersy cattle, and reported that clinical signs included rejection of food, decreased milk yield. Saho and Chattopodhgay (1996) found that graded levels of crude aflatoxin produced on chemically by *Aspergillus parasiticus* NRRL 2999 when fed to young healthy white rabbits, reduced level of total serum protein concentration; depletion of lymphocytes might be a contributory factor for immunosuppression.

As regards the biochemical change, high level of AST and alkaline phosphatase noticed is due to the liberation of the enzyme from the damaged liver cells in the circulatory system. Lynch *et al.* (1970) reported that serum alkaline phosphatase increases at level 0.02 mg/kg or more of aflatoxin. Ray *et al.* (1986) found that the biochemical analysis of serum of cows fed on peanut containing 77 µg/g of aflatoxin B1 revealed hepatic dysfunction indicated by high values of serum lactate dehydrogenase, aspartate transaminase and total bilirubin. Blood *et al.* (1989) recorded that the serum AST levels increased with liver disease in all species. Rajendran *et al.* (1992) reported that the liver was affected in acute and chronic aflatoxicosis in which up to 5 p.p.m of aflatoxins was consumed. Tfeglia et al. (1995) administrated oral doses of 400 µg of aflatoxins to rabbits, and they noticed that histopathological lesions were detected in the liver, kidney and heart.

Table 2. Effect of aflatoxin contaminated rations on some haematological parameters, iron and copper of dairy cows.

Values	Group 1 N = 40	Group 2 (control) N = 40	T.value
Total protein (g/dl)	5.77±0.33***	8.12±0.33	P<0.001
Calcium (mg/dl)	11.27±0.5	11.92±0.42	N.S.
Inorganic phosphorus (mg/dl)	6.87±0.24	6.87±0.47	N.S.
Calcium : phosphorus ratio	74.17±3.35	79.71±3.97	N.S.
Magnesium (mg/dl)	1.89±0.11**	2.49±0.14	P<0.01
Sodium mg Eq/L	81.67±3.20***	141.68±1.17	P<0.001
Potassium mg Eq/L	3.00±3.20***	4.50±0.1	P<0.001
Na / K ratio	28.16±0.61**	31.32±0.75	P<0.01

N.S. = Not significant

It could be concluded that, low amount of aflatoxins (75-150 ppb) in the ration of lactating cows reported in the present work affects animal health causing nutritional anaemia and liver hepatocellular degenerations as shown by Coles (1986).

Table 3. Effect of contaminated dry rations with aflatoxin on liver enzymes.

Values	Group 1 N = 40	Group 2 (control) N = 40	T.value
ALT µ/L	19.97 ± 4.27	17.22±2.04	N.S.
AST µ/L	45.96±2.14***	28.96±2.18	P<0.001
Akaline phosphatase µ/L	3.18±0.21***	1.93±0.16	P<0.001

N.S. = Not significant ± S.E.

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الأفلاتوكسين كمشكلة حقلية فى بعض مزارع الأبقار فى كفر الشيخ

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لوحظ فى بعض المزارع الاهلية فى دسوق بمحافظة كفر الشيخ مشكلة حقلية حيث شملت مزارع الابقار الحلابة نقصا كبيراً فى كمية اللبن مع وجود ضعف عام فى الحيوانات وفقدان الشهية. وبعد تحليل عينات العلائق الجافة وجد بها سموم افلاتوكسين ب١ ، من ٧٥ الى ١٥٠ جزء فى البليون. وعند تحليل ٤٠ عينة من الدم والسيرم لوحظ نقص كبير فى عدد كريات الدم الحمراء وارتفاع فى حجم كريات الدم ومؤشرات الدم ونقص كبير فى البروتين الكلى والماغنسيوم والصوديوم والبوتاسيوم والحديد والنحاس وانزيمات الكبد. كما وجدت زيادة ملحوظة فى خمائر الترانس أمينيز والفوسفيتيز القلوى مما يشير الى تأثر تسممى ضار بوظيفة الكبد.