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FATTY LIVER SYNDROME IN LAYING HENS IN UPPER EGYPT

(With 12 Tables)

By

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ظاهرة التشحم الكبدي في الدجاج البياض بصعيد مصر

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تمت دراسة ظاهرة ترسيب الدهون فى كبد الدجاج البياض فى قطعان مزارع البياض فى محافظات بنى سويف والمنيا وأسيوط وذلك من خلال الملاحظات الحقلية لعدد ٢٣ قطيع بياض لمدة عام كامل. لوحظت هذه الظاهرة أثناء أشهر الصيف فى قطيعين من الدجاج البياض بمحافظة المنيا وقطيع بياض بمحافظة أسيوط والتى تربى فى بطاريات ولم تلاحظ هذه الظاهرة فى القطعان البياض التى تربى تربية أرضية. وقد وجد انخفاض فى انتاج البيض بين (١٠-١٥%) وارتفاع فى نسبة النفوق (٢,٥% شهريا) فى القطعان المصابة. وكذلك وجد تضخم فى حجم الكبد مع تدهن ملمسه واصفرار لونه فى بعض الحالات. كما وجد بالكبد أنزفة مع تجمع الدهن فى التجويف البطنى وحول الفونصة والمعدة الغدية فى الحالات المصابة. وقد وجد ارتباط زيادة نسبة عسر المياه بحدوث هذه الظاهرة. تمت دراسة استحداث ظاهرة ترسيب الدهون فى كبد الدجاج البياض تجريبيا من خلال تأثير عوامل مختلفة مثل عسر المياه- ارتفاع درجة حرارة الطقس- نقص الكالسيوم و اضافة سموم الأفلاتوكسين. وقد اظهرت التجارب أن جميع هذه العوامل أحدثت ظاهرة ترسيب الدهون وكانت النتائج ملموسة فى الصفة التشريحية وزيادة وزن الجسم, نسبة وزن الكبد الى وزن الجسم, نسبة الرطوبة فى الكبد, نسبة الدهن فى الكبد- ومكونات مصل الدم (اختبار وظائف الكبد - الكوليستيرول- تراى جليسريد والدهون الكلية).

SUMMARY

The prevalence of Fatty liver syndrome (FLS) in laying flocks in Beni-Suef, El Menia and Assiut Governorates was recorded through field observations of 23 laying flocks for one year. FLS was observed in 2 flocks of caged layers in El Menia and one flock in Assiut Governorates while no cases were observed in floor pen raised flocks. Cases of FLS were observed in summer months and the affected flocks showed drop in egg production (10-15%), increased monthly mortality (2.5%), enlarged, greesy, yellow livers with lipids, some cases had liver haemorrhages, more abdominal fat and more fat around the gizzard and proventriculus. Water hardeness was associated with appearance of FLS. The induction of FLS in laying hyline hens was experimentally studied using hard water, high atmospheric temperature, calcium deficient ration and aflatoxin. The results of experimental induction of FLS by these factors indicated that all of them can induce FLS as observed by clinical symptoms, pathological lesions, increase in body weight, liver weight, liver % of body weight, liver fat %, decrease in liver moisture and increase in certain serum constituents (GOT, cholesterol, triglycerides and total lipids).

Key words: *Fatty liver, Laying hens.*

INTRODUCTION

Fatty liver syndrome is a major commercial problem which faces breeder and layer flocks in Egypt. The results in increased yellow friable liver condition with high lipid content, excess abdominal fat, rupture of liver may occur leading to haemorrhage and death.

Fatty liver syndrome (FLS) was firstly described by (Couch, 1956). Different synonyms were given as liver haemorrhagic syndrome (Nesheim and Norvel, 1970); fatty liver (Roberson and Trujullo, 1975) and fatty liver haemorrhagic syndrome (Wolford and Polin, 1972 a,b).

Lombardi et al. (1968); O'Hea and Leveille (1969); Sallmann (1973); Butler (1975, 1976) and Leveille et al. (1975) reported that the cause of FLS was due to one or more of the following factors.

1- An increased flow of lipids from the fat depots to the liver.
2- An intensified denovo synthesis.
3- A defect in the transport of fat from the liver cell plasma to the blood stream. Many factors predispose to FLS as genetic factors, housing

system, nutrition, water hardness and hormones (Couch 1956; Griffith et al., 1969; Haghighi-Rad and Polin, 1982; Roland 1985; Squires and Leeson, 1988, and Hansen and Walzem, 1993).

Couch (1968), Deacon (1968), Greuel and Hartfiel (1968) and Reedy (1968) observed an increase in body weight of the affected hens from 25-30%, drop in egg production, palness of the comb with cyanosis of the tips and enlargement of liver.

Couch (1968), Deacon (1968); Greuel and Hartfiel (1968); Reedy (1968); Wolford et al. (1971); Rothenbacher and Schwartze (1972) and Sallmann (1973) found on post-mortem examination (P.M.) accumulation of fat on the abdominal cavity and visceral organs, liver enlarged and rounded, very friable and pale brown to yellow in colour. The liver tissue bulges at the cutting surface and shows a pasty consistency. There are simple or mutiple haemorrhages on liver. Rupture of the liver capsule causing intrenal bleeding and death.

The aim of this work is to report the prevalence of FLS in Upper Egypt through field observations and to induce the syndrome by different dietetic, managemental and nutritional factors.

MATERIAL and METHODS

Experimental birds:

75 hens of Hyline breed were provided by Shosha farm for table egg production (El-Menia Governorate). Birds were 75 weeks of age and were weighted individually and selected according to weight for uniformity. The hens were maintained in wire cages under constant lighting and environmental temperature. The hens were randomly distributed into 5 groups for induction of FLS by different factors.

Reagent Kits:

1-Cholesterol kits:

Kits used for determination of cholesterol were provided by Bio Merieux vitek, Inc. Kits ref. 61224.

2- Triglycerides Kits:

Kits used for determination of serum triglycerides were provided by bio Merieux vitek, Inc. Kits ref. 61226.

3- Lipid kits:

Kits used for determination of serum total lipids were provided by bioMerieux vitek, Inc. Kits ref. 61402

4- Transaminase kits:

Used for determination of GOT activity were provided by Bio Merieux vitek, Inc. Kits ref. 61691.

Chemicals:

1- Diethyl ether LR (ether solvent) $(C_2H_5)_2O$ used for extraction of liver fat.

2- Concentrated sulfuric acid (Adwic company) used for serum determination of serum lipid.

3- Sodium hydroxide (NaOH) 0.4 N (Alamia Company for chemicals) used for determination of GOT.

Instruments:

- Soxhelt apparatous was used for liver fat extraction.
- Spectrophotometer was used for serum analysis.
- Atomic Absorption Spectroscopy was used for water quality analysis.

Field observations on FLS:

A total of 23 flocks of laying hens in Beni-suef, El-Menia and Assiut Governorates were put under observation for any changes refering to FLS by clinical examination, recording total mortalities and daily egg production and P.M examination of mortalities.

Livers were removed, weighted and kept in freezer for determination of liver moisture and liver lipid content.

Blood samples:

Blood samples were collected from chickens from the previous farms and serum was separated in sterile glass vials and kept in a frozen state -20°C until used for biochemical analysis (determination of serum total lipids, cholesterol, triglycerides, and GOT activity).

Moisture content of the liver:

The moisture content of the liver was determined by drying the liver samples for 24 h at 70°C using hot air oven and the difference in weight was estimated as moisture content (Pearson and Butler, 1978 a):

Liver fat content:

The liver fat content was determined according to the method of Folch et al.(1957) using Soxhelt apparatus.

Biochemical analysis:

1- Determination of serum total lipids, enzymatic cholestrol and triglycerides:

They were according to the method of Coles (1980).

2- Determination of GOT activity:

GOT activity was determined according to the method of Reitman and Frankel (1957).

Criteria for estimation of fatty liver syndrome:

Degree of affection with FLS was determined by the following criteria:

- Body weight changes. liver weight, liver weight% to body weight, liver lipid content.
- Serum content of cholesterol, triglycerides, total lipids and activity of GOT.
- Postmortem changes (liver changes and abdominal fat)

Experimental design

Experimental induction of FLS in laying hens:

I- Induction by hard water:

The quality of water was changed by addition of 100 p.p.m. calcium chloride ($CaCl_2$) and 50 p.p.m. magnesium chloride ($MaCl_2$.) to the normal water **(Jensen et al.,1976)** The water was available adlibitum but the feed was restricted according to the breed requirements. Regular water and restricted feed was available for control group. The birds of experimental and control groups (15 birds each) were observed for 8 weeks.

II- Induction by high atmospheric temperature:

A group of 15 birds of Hyline breed was maintained at 30-32°C in wire cages for 8 weeks (Wolford, 1971). The temperature source was electrical heater. The control group was kept at 20-22°C atmospheric temperature. The normal regular water was available adlibitum and the feed was restricted as normal basal diet.

III- Induction by calcium deficient ration:

To study the effect of calcium deficient ration on induction of FLS. A group of 15 birds of Hyline breed was allowed to take calcium dificent diet (2%) Bolden and Jensen (1985). The feed and normal water were available adlibitum. The control group was fed on restricted normal basal diets and the normal regular water were available adlibitum. The birds were observed for 8 weeks.

IV- Induction by Aflatoxin:

To study the effect of aflatoxin on induction of FLS, a group of 15 birds of Hyline breed was maintained in wire cages. 10 micrograms aflatoxin (1 ml of aflatoxin extract) were dissolved in 1 ml chloroform, then completed to 100 ml chloroform in a volumetric flask. Every one kilogram of normal basal diet was thoroughly mixed with 10 micrograms

aflatoxin dissolved in chloroform. Then the chloroform was allowed to evaporate overnight in an oven at 50°C. Samples from the normal basal diet were taken for analysis for aflatoxin. This group was fed at a dosage level of 10mg/kg diet for 4 weeks. The feed was restricted and the normal water was available adlibitum (Hamilton and Garlich, 1970).

RESULTS

Results of field observations:

Cases of FLS were observed in two flocks of caged layers in ElMenia and one flock of caged layers in Assiut Governorate. No cases were observed in floor pen raised hens. Positive cases of FLS were observed in June-September (summer months).

Affected flocks had a history of ideal curve of egg production which showed declination at 45-50 weeks of age (10-15% drop). Mortalities of these flocks increased and reached 2.5% per month in comparison with 0.7-0.9% mortalities in previous months and in non affected flocks.

The affected flocks seemed to be healthy, in a good condition and sometimes were over weight and difficult to be distinguished from non affected flocks except some birds had pale enlarged combs and wattles with scaly like dandruf on the comb and wattles.

P.M. examination of affected cases revealed extreme accumulation of fat in the abdominal cavity and around visceral organs (gizzard and proventriculus). Liver was enlarged in size, greesy in texture, friable and yellowish in colour. In some cases, liver was covered with a layer of thick clotted blood throughout the parietal surface and the clots were attached to the liver with fibrin originating from rupture of the organ. Kidneys were somewhat enlarged and pale. Sometimes, the oviduct had a fully formed egg in the distal part.

Analysis of water samples showed increased hardness of water due to high concentration of calcium and magnesium (130 p.p.m. $CaCl_2$ and 40 p.m.m. $MaCl_2$). Analysis of ration samples proved that ration was balanced and free from aflatoxin.

Result of experiment I:

Results of the effect of hard water is shown in tables (1-3). There is a significant decrease in body weight (B.W.) of birds raised on hard water when compared to control group raised on normal water (Table 1). There is a significant ($P<0.01$) increase in liver weight (L.W.), liver% of

body weight (L% of B.W.) and liver fat (L.F) while there is a significant ($P<0.01$) decrease in liver moisture% (L.M.) of experimental group raised on hard water compared to control group. (Table 2). There is a significant ($P<0.01$) increase in serum constituents (GOT, cholesterol, Triglycerides and lipids) in experimental birds raised on hard water in comparison to control group raised on normal water. Table (3).

Results of experiment II:

There is a significant ($P<0.01$) decrease in B.W of experimental birds raised under high atmospheric temperature (30-32°C) when compared to control birds raised under optimal temperature (20-22°C) (Table 4). There is significant ($P<0.01$) increase in L.W.%, L.% of B.W. and L.F. while there is significant ($P<0.01$) decrease in L.M of experimental group raised under high temperature in comparison to control birds raised under optimal temperature (Table 5). There is a significant ($P<0.01$) increase in serum constituents in experimental birds raised under high temp. in comparison to control group raised under optimal temp. (Table 6).

Results of experiment III:

There is a significant ($P<0.05$) increase in B.W. in experimental birds fed on calcium dificient diet when compared to control birds fed normal basal diet (Table 7). There is a significant ($P<0.01$) increase in L.W.,L.% of B.W and L.F. while there is a significant ($P<0.01$) decrease in L.M.% in birds fed calcium deficient diet in comparison to birds fed normal basal diet (Table 8). There is a significant ($P<0.01$) increase in serum constituents of birds fed calcium deficient diet when compared to control birds fed normal basal diet (Table 9).

Results of experiment IV:

There is a significant ($P<0.01$) decrease in B.W. of birds treated with aflatoxin with restricted feeding when compared to control group (Table 10).

There is a significant ($P<0.01$) increase in L.W., L.% of B.W and L.F% and significant ($P<0.01$) decrease in L.M.% in birds treated with aflatoxin with restricted feeding when compared to control group (Table 11). There is a significant ($P<0.01$) increase in serum constituents of birds treated aflatoxin with restricted feeding when compared to control birds (Table 12).

DISCUSSION

In the present wrok we tried to throw some light on the prevalence of FLS in laying flocks in Upper Egypt through field observation of 23 laying flocks and through experimental induction of the disease in Hyline Layers.

Results concering the prevalence of FLS showed that the FLS occurred during hot summer months. Similar observations were reported by Couch, (1956); Ringer and Sheppard (1963); Griffith et al. (1969) and Nesheim et al. (1970). The syndrome was observed in caged layers of 45-50 weeks of age with drop in egg production (10-15%) below nomal. The clinical and pathological changes were consistent with previous reports of Couch (1956, 1968); Deacon (1968); Greuel and Hartfiel (1968); Reedy (1968), Wolford (1971); Rothenbacher and Schwartze (1972) and Sallmann (1973).

The induction of the disease by hard water showed that there were a decrease in body weight gain (109 gm) and an increase in liver weight (L.W.), liver body weight ratio (L.% of B. W.) and liver fat % (L. F.%), while there was a decrease in liver moisture % (L. M. %) and increase in GOT activity, serum cholesterol, serum triglycerides and serum total lipids. The same results were reported by Durfor and Becker (1964), Hurwitze and Bornstein (1966) and Jensen et al. (1976 , 1977). They can explain excess calcium reduce feed consumption.

The induction of the disease by high atmospheric temperature showed that there were increase in body weight gain (163 gm), L. W., L% of B.W. and L.F. %, while there was a decrease in L.M.% and an increase in GOT, cholesterol, triglycerides and total lipids of serum. Our results are in agreement with those of Barton (1966), Sunde (1966), Griffith and Schexnaider (1970), Wolford (1971), Pearson and Butler (1978 b) and Akiba et al. (1983). They reported that the glycogen content of liver was increased at 29°C and was positively correlated with the lipid content, this was due to an excessive intake of metabolisable energy in the form of carbohydrate. On the other hand, the results disagree with those of Lee et al. (1975).

The induction of the disease by calcium deficient ration showed that there was an increase in body weight gain (165 gm), increase in L.W., L.% of B.W., L.F.% while there was decrease in L.M.% and

increase in serum GOT, cholesterol, triglycerides and total lipids. The same results were reported by Roland et al. (1974, 1985), Harms et al. (1977), Mongin and Sauveur (1979), Haghighi-Rad and Polin (1981), Bolden and Jensen (1985) and Roland (1985). The low levels of dietary calcium may result in increased feed consumption. On the contrary, our results are in disagreement with the results of Bolden and Jensen (1983) and Maurice et al. (1983).

The induction by aflatoxin showed a decrease in body weight (93 gm), and increase in L.W., L.% of B.W. and L.F. %, while there was a decrease in L.M. % and an increase in serum GOT, cholesterol, triglycerides and total lipids. Our results are in agreement with Hamilton and Garlich (1970), Osborne et al. (1975), Doerr et al. (1976) and Dalvi and Ademoyero (1983). They used 0.5-1.0 mg/g diet at 21 weeks of age for 8 weeks. They suggested that aflatoxins inhibit fat digestion and lipid transport

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Table 1: Effect of hard water on body weight

Treatment	I. B. W (mean) gm	F. B. W. (mean) gm	C. B. W. (mean) gm
Hard water	1403 ± 50	1294 ± 80	-109
Normal water	1394 ± 60	1335 ± 70	-59

I. B. W = intial body weight
F. B. W.= Final body weight
C. B. W.= change of body weight
+ = body weight gain
- = body weight loss

Table 2: Effect of hard water on liver

Treatment	L.W (mean) gm	L.% of B. W (mean) %	L. M.% (mean) %	L. F.% (mean) %
Hard water	38.80	2.99	61.95	19.74
Normal water	23.70	1.77	69.40	10.62

L.W = Liver weight
L.% of B. W = Liver % of body weight
L. M.% = liver moisture%
L. F.% = liver fat%

Table 3: Effect of hard water on serum constituents

Treatment	GOT (mean) µ/L	Cholesterol (mean) G/L	Triglycerides (mean) G/L	Lipids (mean) G/L
Hard water	242 ± 5	1.36 ± 0.11	3.25 ± 0.66	20.69 ± 6
Normal water	121 ± 8	0.65 ± 0.08	1.60 ± 0.3	8.29 ± 2

Table 4: Effect of high atmospheric temperature on body weight

Treatment	I. B. W (mean) gm	F. B. W. (mean) gm	C. B. W. (mean) gm
High temp. 30-32°C	1425 ± 40	1262 ± 90	-163
Optimal temp. 20-22°C	1394 ± 60	1335 ± 70	-59

Table 5: Effect of high atmospheric temperature on liver

Treatment	L.W (mean) gm	L.% of B. W (mean) %	L. M.% (mean) %	L. F.% (mean) %
High temp. 30-32°C	39.3	3.11	58.00	25.43
Optimal temp. 20-22°C	23.7	1.77	69.40	10.62

Table 6: Effect of high atmospheric temperature on serum constituents

Treatment	GOT (mean) µ/L	Cholesterol (mean) G/L	Triglycerides (mean) G/L	Lipids (mean) G/L
High temp. 30-32°C	307 ± 9	1.93 ± 0.6	4.16 ± 0.66	25.98 ± 4.2
Optimal temp. 20-22°C	121± 8	0.65 ± 0.23	1.60 ± 0.3	8.29 ± 2.1

Table 7: Effect of calcium deficient ration on body weight

Treatment	I. B. W (mean) gm	F. B. W. (mean) gm	C. B. W. (mean) gm
calcium deficient ration	1370 ± 50	1535 ± 30	+165
normal ration	1394 ± 60	1335 ± 70	- 59

Table 8: Effect of calcium deficient ration on liver

Treatment	L.W (mean) gm	L.% of B. W (mean) %	L. M.% (mean) %	L. F.% (mean) %
Calcium deficient ration	42.40	2.76	57.78	24.30
Normal ration	2.70	1.77	69.40	10.62

Table 9: Effect of calcium deficient ration on serum constituents

Treatment	GOT (mean) µ/L	Cholesterol (mean) G/L	Triglycerides (mean) G/L	Lipids (mean) G/L
Calcium deficient ration	331 ± 5	2.35 ± 0.3	3.81 ± 0.2	9.99 ± 1
Normal ration	121 ± 8	0.65 ± 0.8	1.60 ± 0.3	8.29 ± 2

Table 10: Effect of Aflatoxin on body weight of birds raised on restricted feeding

Treatment	I. B. W (mean) gm	F. B. W. (mean) gm	C. D. W. (mean) gm
Aflatoxin	1300 ± 80	1207 ± 70	-93
-	1394 ± 60	1335 ± 70	- 59

Table 11: Effect of Aflatoxin on liver of birds raised on restricted feeding.

Treatment	L.W (mean) gm	L.% of B. W (mean) %	L. M.% (mean) %	L. F.% (mean) %
Aflatoxin	38.50	3.18	58.09	23.28
-	23.70	1.77	69.40	10.62

Table 12: Effect of Aflatoxin on serum constituents of birds raised on restricted feeding.

Treatment	GOT (mean) μ/L	Cholesterol (mean) G/L	Triglycerides (mean) G/L	Lipids (mean) G/L
Aflatoxin	378 ± 4	2.49 ± 0.3	4.98 ± 0.2	28.04 ± 4.0
-	121 ± 8	0.65 ± 0.8	1.60 ± 0.3	8.29 ± 2.0