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**PREGNANCY TOXAEMIA IN SHEEP AND GOATS  
(CLINICAL AND BIOCHEMICAL ASPECTS)**  
(With 4 Tables)

By

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تسمم الحمل في الأغنام والماعز  
الأعراض الإكلينيكية والصورة البيوكيميائية

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إشتمل البحث على فحص عدد ٦٠ من الأغنام والماعز والتابعة لمزرعة برج العرب بكفر الشيخ للأغنام والماعز وكان الهدف من البحث دراسة الأعراض الإكلينيكية والبيوكيميائية للحالات التي تعاني من تسمم الحمل. وقد سجلت أهم الأعراض الإكلينيكية متمثلة في فقدان الشهية والخمول الذي أتبعه الأعراض العصبية متمثلة في العمى والرقاد والإغماء والموت في بعض الحالات. وأوضحت التحاليل البيوكيميائية لمصل الدم على إنخفاض معنوي في معدلات الجلوكوز والدهون الثلاثية والكولسترول والماغنسيوم والألبومين في الحيوانات المريضة بمقارنتها بالسليمة - بينما حدثت زيادة معنوية في معدلات الكورتيزون والأحماض الدهنية واليوريا وأنزيمات الكبد وكذلك الألبا والجاماجلوبولين في الحيوانات المريضة بمقارنتها بالسليمة.

## SUMMARY

This work was carried out to study clinical and biochemical aspects of field cases of ewes and does suffering from pregnancy toxemia. The first signs observed were anorexia and depression which may be followed by locomotor disturbances (incoordination and star gazing), recumbency, blindness, coma and death. Laboratory findings of present data showed ketonuria, lower serum values of glucose, triglycerides, cholesterol, magnesium and albumin. Higher serum cortisol, free fatty acids, urea, liver enzymes, alpha and gamma globulins were observed. The obtained data were tabulated and discussed.

*Key words: Sheep & Goats - Pregnancy Toxaemia*

## INTRODUCTION

Pregnancy toxemia is a potentially total metabolic condition of sheep and goats which can cause substantial losses. It occurs in ewes and does during the last 2 to 4 weeks of gestation caused by a negative energy balance resulting from increased energy demands for rapid fetal growth in late gestation and insufficient intake (Smith, 1990). Adult ruminant obtains very little glucose from its diet and its metabolic requirements for glucose are supplied by gluconeogenesis in the liver and kidney (Bergman, 1973). During pregnancy and lactation, the requirements for glucose increase where considerably, in the pregnant animal the fetus and uterus utilize glucose as a major energy source (Lindsay, 1973) and during lactation large quantities of glucose are removed by the mammary glands for lactose synthesis (Annison and Linzell, 1964). As a result of the energy deficit, some mobilization of lipid reserves appear to occur which results in a doubling of the plasma free fatty acids, giving rise to a fatty liver and increased ketone bodies in blood and urine (Chaiyabuter, et al., 1982 and Kimberling, 1988)

Main clinical manifestations in early pregnancy toxemia disease in ewes and does are reduced appetite, dullness, hypoglycaemia, ketonaemia and ketonuria. In a more advanced stage a severe ketoacidosis, haemoconcentration, hyperglycaemia and uraemia often accompanied by dyspnea, recumbency and blindness (Kronfeld, 1972) and El-Sebaie (1995). Excessive salivation and fine muscle fasciculation were occasionally observed in the head region causing movements of the overlying skin and twitching of the ears (Sargison, et al., 1994 and Scott, et al., 1995). The condition can be

diagnosed provisionally by a clinical examination of animals and confirmed biochemically by a marked increase in the concentration of 3 - hydroxybutyrate and a corresponding decrease in the plasma glucose concentration (Scott and Woodman, 1993). The aim of this work was to investigate serum biochemical alterations in field cases of pregnancy toxæmia in ewes and does which may provide a diagnostic aid in such conditions.

## **MATERIAL and METHODS**

Both pregnant ewes and does were included in this study. Fifty-two native breed ewes ranging from 2 to 4 years -old at Meseer, Kafr El- sheikh, production farm were transferred from concentrate to drawa feeding during the last month of pregnancy. Three mortality and 20 abortion cases beside severe to moderate clinical signs characteristics of pregnancy toxæmia had occurred a few days later. These signs include inappetance, dullness, incoordination, grinding on the teeth, sternal recumbency, drowsiness, salivation and blindness.

The diseased does were allotted into two groups (A and B) according to the locality and nutrition regimen. In a flock of 60 pregnant Barky and Demashky does (2.5-5 years old) at Borg El-Arab sheep and goat production farm diseased cases had recorded (group A). They exhibited one or more of the following symptoms, anorexia, depression, disinclination to move, recumbency and drowsiness. Three cases died and necropsy findings showed paleness of the liver and kidney. The history of the flock indicated that these animals were inadequately fed (about 100 gm concentrate/ head/ day plus rice straw add lib.).

Several cases of suspected naturally occurring pregnancy toxæmia in pregnant native breed does were observed in small private farms at El- wahat Al- Daqlia (group B). These were seen especially during bad weather days. Diseased cases showed loss of appetite, weakness, muscle tremors of the head and sternal recumbency. The post-mortem examination of the emergency slaughtered case revealed greasy friable pale liver and abdominal fat necrosis. In this locality goats fed mainly grasses beside dates, concentrate or barley and housed in open yards without shelter in winter.

Blood and urine samples were collected from 7 and 11 clinically affected does from groups A and B, respectively as well as 8 affected ewes. Blood and urine Samples were also collected from apparently healthy pregnant does and ewes, 10 samples from each samples for detection of

different causes of abortion (vaginal swabs) and serum were also taken from aborted cases and examined at the Animal Health Research Institute. Urine samples were immediately used for detection of ketone bodies using coumbour-9 test-strips (Boehringer Monnheim, Germany). Blood serum samples were subjected to glucose determination on the day of sampling according to Siest et al (1981), cortisol after Schlaghedee et al. (1992) and free fatty acids as described by Schuster, and Pilz, (1979). Serum alkaline phosphatase (ALP) and inorganic phosphorus after Kilchling and Freiburg (1951). Serum total lipids, phospholipids, triglyceride, cholesterol, total protein, urea, alanine amino-transferase (ALT), aspartate amino-transferase (AST), calcium and magnesium were estimated using reagent kits (Biomerieux, Marcy-L Etoile, France). Serum protein electrophoretic pattern was assayed according to Davis (1964) and Ornstein (1964). Statistical analysis was carried out after Snedecor and Cochran (1974).

## RESULTS

Blood serum concentrations of glucose, phospholipids, triglyceride, calcium and magnesium showed a marked decrease in pregnancy toxæmic ewes and does compared to non-toxæmic ones (Tables, 1 and 3). Serum cholesterol levels was only decreased in toxæmic ewes.

Serum cortisol, free fatty acids, urea, ALT, AST, and ALP were generally increased in toxæmic ewes and does than in non-toxæmic ones. In significant difference were seen between toxæmic and non toxæmic animals for: total lipids and total protein. While serum albumin concentrations were dropped in all toxæmic animals, alpha and beta globulins as well as total globulins were elevated especially in toxæmic ewes compared to apparently healthy ones (Tables, 2 and 4).

## DISCUSSION

Anorexia and depression were the first signs observed in both pregnancy toxæmic ewes and does. Sternal recumbency and locomotor disturbances including incoordination in ewes and muscle tremors in goats were previously recorded by El-Sherif *et al.* (1978), Smith (1990) and El-Sebaie *et al.* (1992) and El-Sebaie (1995). Forbes and Singleton (1964) described that the nervous symptoms may be due to an inability of the nerve cells to utilize sugar, perhaps as a result of high cortisol levels. Moreover,

excess salivation and blindness showed in severe toxemic cases of ewes were in accordance with the previous reports of Buswell *et al.* (1986) and Sargison *et al.* (1994).

Laboratory findings revealed the presence of ketone bodies in the urine samples of clinically affected animals. The results obtained for serum glucose indicate that the ketotic ewes and does (group A) were hypoglycemic. These results could be confirmed with those obtained by Storry and Rook (1962) and Jonsson and Pehrson (1972) who found that the glucose levels are related to the animals energy status, values falling with a negative energy balance. Affected does in group B, in spite of showing clinical signs of pregnancy toxemia, had serum glucose concentration near the normal range. These results indicated that exposure to cold resulted in an increase in mobilization of glucose as well as free fatty acids to be used in the thermogenic process (Terashima *et al.*, 1982)

Serum cortisol concentrations showed a marked increase in diseased animals when compared to healthy ones. Similar result were recorded by Lindler (1959) who found that in ketotic sheep, the circulating cortisol levels were almost 3 times those of healthy sheep. Kimberling (1988) postulated that the stress and low caloric intake in pregnancy toxemic animals have a profound effect on the kidney and adrenal gland with a significant reduction in renal blood flow and glomerular filtration rate which raise plasma renin activities and elevate plasma cortisol levels.

Concerning serum lipid pattern, the observed decrease in phospholipids and triglyceride in ketotic animals was in agreement with Payne (1977) who mentioned that in ketosis, serum lipid values especially phospholipids and triglyceride were reduced. Henricson *et al.* (1977) concluded that the lower triglyceride content is the result of reduced appetite in animals with hyperketonaemia. The observed decrease in serum cholesterol in the ketotic ewes could be confirmed with the result obtained by kamel (1992). The sharp increase in the concentrations of free fatty acids in the sera of both affected ewes and does could be attributed to the increased mobilization of fatty acids from the adipose tissues in response to an increased requirement for endogenous substrate for energy production during pregnancy (Noble *et al.*, 1971). Haughey (1973) found that fat catabolism is a major change in cold-stressed neonatal lambs. Russel *et al.* (1967) suggested that plasma free fatty acids would be the most useful index of the degree of under nourishment in pregnant ewes.

The elevated values of serum urea in the diseased animals can be fully explained by the observation of Parry and Tylor (1956) who found fatty

infiltration in the tubular epithelium of the kidneys of ketotic ewes. The increase in the activities of serum enzymes (ALT, AST & ALP) in diseased animals is a strong evidence for the degree of their liver damage. EL-Sebaie *et al.* (1992) observed severe hepatic changes in ketotic ewes and does.

The present decrease in serum calcium concentrations in diseased does (group A) tends to support the conclusion of Yoshida (1979) and Blood *et al.* (1983) where at any stage of ketosis calcium is probably lost through urine with consequent uremia of compensating acidosis. While the present decrease in serum magnesium concentrations in both ewes and does could be confirmed with the results obtained by Terashima *et al.* (1982).

The obtained results for serum protein electrophoretic pattern (Tables, 2 and 4) in ketotic animals showed a significant drop in the albumin concentration and a significant elevation in the concentrations of alpha and beta globulins and consequently total globulins. Vihan and Rai (1984) reported hypoalbuminaemia in pregnancy toxemic goats, while Ceron *et al.* (1994) observed non significant decrease in serum albumin, slight increase in alpha and beta globulins and a consistent increase in gamma globulins in ketotic goats. They postulated that the increase in alpha fraction in ketotic animals may be due to the combining of alpha globulin fractions with lipid compounds to facilitate their transport as lipoproteins (in ketosis there is a mobilization of reserve triglycerides to compensate the lack of blood glucose). Similarly, beta globulins are involved in lipid transport

Kimberling (1988) described that the total feed requirements for the single bearing ewe during the last 6 weeks is 1.5 x maintenance and 2 x maintenance for a twin bearing ewe. The additional energy for this period is best supplied by concentrate feeds as the rumen capacity is limited by fetal expansion. Accordingly the animals included in this study receive low feed requirements (less concentrates or only drawa) which led to occurrence of the disease. Finally it could be concluded that early diagnosis of ovine pregnancy toxemia is vital for satisfactory treatment and pregnant ewes should receive its full requirements during the last stage of pregnancy to avoid and prevent its occurrence.

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Table (1) some Laboratory findings in toxæmic pregnant and apparently healthy ewes.

Parameter	Diseased ewes	Healthy ewes
Glucose (mg/dl)	26.96* ± 2.80	36.24 ± 3.14
Cortisol (ng/ml)	48.87*** ± 3.11	30.32 ± 1.84
Phospholipids(mg/dl)	64.13 ± 4.00	80.06 ± 5.20
Triglyceride (mg/dl)	41.50* ± 3.06	51.43 ± 2.84
Cholesterol (mg/dl)	65.40* ± 2.63	76.11 ± 3.56
Free fatty acids(mg/dl)	49.83*** ± 2.88	23.33 ± 1.96
Total protein (gm/dl)	6.45 ± 0.33	7.03 ± 0.24
Urea (mg/dl)	34.75* ± 2.17	28.36 ± 2.08
ALT (U/L)	30.77** ± 1.50	24.24 ± 0.63
AST (U/L)	41.85*** ± 0.98	27.13 ± 0.75
ALP (mmol U/L)	5.61* ± 0.60	3.58 ± 0.35
Phosphorus (mg/dl)	5.26 ± 0.48	6.39 ± 0.41
Calcium (mg/dl)	9.61 ± 0.43	10.52 ± 0.53
Magnesium (mg/dl)	2.03* ± 0.16	2.69 ± 0.21

\* Significant at:

\* P<0.05

\* P<0.01

\* P<0.001

Table (2) Serum protein electrophoretic pattern in toxæmic and healthy ewes.

Fraction percentage	Diseased ewes	Healthy ewes
<b>Albumin</b>	29.75* ± 1.12	33.15 ± 1.03
<b>Alpha-globulins</b>	20.73 ± 0.90	21.12 ± 0.84
Alpha-1	5.77 ± 0.23	5.92 ± 0.29
Alpha-2	4.49** ± 0.21	3.35 ± 0.26
Alpha-3	10.47 ± 0.60	11.85 ± 0.38
<b>Beta-globulins:</b>	11.13* ± 0.66	9.12 ± 0.55
Beta-1	6.16* ± 0.30	5.34 ± 0.24
Beta-2	4.97* ± 0.41	3.78 ± 0.28
<b>Gamma-globulins:</b>	38.39 ± 1.70	36.61 ± 0.89
Gamma-1	25.67 ± 1.24	24.26 ± 1.08
Gamma-2	12.72 ± 0.54	12.35 ± 0.41
<b>Total globulins</b>	70.25* ± 1.12	66.85 ± 1.03
<b>A/G Ratio</b>	0.42* ± 0.25	0.50 ± 0.22

\* Significant at:

P < 0.05

P < 0.01

Table (3) Some laboratory findings in Toxaemic and apparently healthy does.

Parameter	Diseased does		Healthy does
	Group A	Group B	
Glucose (mg/dl)	28.75* ± 2.18	31.55 ± 2.12	37.12 ± 2.06
Cortisol ng/ml	46.22*** ± 2.42	n.d.	18.76 ± 1.05
Total lipids (mg/dl)	339.87 ± 19.40	n.d.	315.30 ± 20.86
Phospholipids (mg/dl)	50.38*** ± 3.22	63.18* ± 4.36	77.16 ± 4.23
Triglyceride (mg/dl)	37.56** ± 3.58	61.43 ± 2.84	54.20 ± 4.00
Cholesterol (mg/dl)	69.14 ± 4.10	n.d.	62.05 ± 5.46
Free fatty acids (mg/dl)	38.56*** ± 2.66	53.17*** ± 3.80	20.70 ± 1.56
Total protein (gm/dl)	6.34 ± 0.22	6.65 ± 0.30	6.58 ± 0.27
Urea (mg/dl)	35.17* ± 2.11	38.30** ± 3.24	29.28 ± 1.18
ALT (U/L)	27.12*** ± 0.48	33.41*** ± 1.36	22.92 ± 0.37
AST (U/L)	36.70*** ± 0.84	42.70*** ± 1.75	26.11 ± 0.57
ALP (mmol U/L)	5.06*** ± 0.30	6.53*** ± 0.48	3.16 ± 0.19
Phosphorus (mg/dl)	7.89* ± 0.48	6.82 ± 0.56	6.17 ± 0.38
Calcium (mg/dl)	8.53 ± 0.22	9.18 ± 0.36	9.87 ± 0.34
Magnesium (mg/dl)	1.94* ± 0.48	1.81* ± 0.11	2.36 ± 0.14

n.d. = Not determined.

\* Significant at:

P < 0.05

P < 0.01

P < 0.001

Table (4) serum protein electrophoretic pattern in toxæmic and healthy does.

Fraction	Diseased does		Healthy does
	Group A	Group B	
Albumin	28.44* ± 1.08	29.25* ± 1.16	32.47 ± 0.85
Alpha-globulins	15.29* ± 0.87	14.78 ± 0.76	13.09 ± 0.39
Alpha-1	7.18 ± 0.52	6.88 ± 0.44	6.05 ± 0.56
Alpha-2	8.11 ± 0.64	7.90 ± 0.36	7.04 ± 0.47
Beta-globulins:	22.78** ± 0.48	21.14 ± 0.83	20.16 ± 0.68
Beta-1	11.64 ± 0.51	10.32 ± 0.49	11.08 ± 0.47
Beta-2	11.14* ± 0.48	10.82 ± 0.56	9.07 ± 0.60
Gamma-globulins:	33.49 ± 1.60	34.83 ± 1.08	34.28 ± 1.39
Gamma-1	24.00 ± 1.21	23.90 ± 0.95	24.29 ± 1.15
Gamma-2	9.49 ± 0.54	10.93 ± 0.78	9.99 ± 0.45
Total globulins	71.56* ± 1.08	70.75* ± 1.16	67.53 ± 0.85
A/G Ratio	0.40* ± 0.022	0.41* ± 0.026	0.48 ± 0.015

\* Significant at:

\*P < 0.05

\*\*P < 0.01

