

Clinical and Serobiochemical Studies on Naturally-Occurring Pregnancy Toxaemia in Shamia Goats

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Abstract: Detailed study of mild and advanced cases of pregnancy toxaemia was carried out on 39 affected goats. Total fifteen of these goats were diagnosed as mild cases of the disease, whereas 24 of them were considered as advanced cases. The estimation of blood glucose, plasma protein, lactate, pyruvate, Creatine Kinase (CK), Lactate Dehydrogenase (LDH), Aspartate Aminotransferase (AST) and urea nitrogen was carried out. The results were compared with those obtained from healthy pregnant goats. The blood glucose level was low in affected goats. However, plasma lactate, pyruvate, urea nitrogen concentration and creatine kinase and aspartate aminotransferase activities in the same goats were highly increased. These results suggest that the main cause of death among pregnancy toxaemic goats may be due to hepatorenal dysfunction.

Key words: Pregnancy, toxaemia, Shamia goats, serobiochemical, AST, LDH

INTRODUCTION

Pregnancy toxaemia is a potentially total metabolic condition of sheep and goats which can cause substantial losses. It occurs in does during the last 2-4 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). The requirements for glucose increase considerably in the pregnant animal and the fetus and uterus utilize glucose as a major energy source (Lindsay, 1973). 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, mobilization of lipid reserves results in a doubling of the plasma free fatty acids giving rise to fatty liver and increased ketone bodies in blood and urine (Chaiyabuter *et al.*, 1982).

In early pregnancy toxemia disease in does, the main clinical manifestations are reduced appetite, dullness, hypoglycaemia, ketonaemia and ketonuria. In a more advanced stage, severe ketoacidosis, haemoconcentration, hyperglycemia and uraemia are often accompanied by dyspnoea, recumbency and blindness (El-Sebaie, 1995). Excessive salivation and muscle fasciculation were occasionally observed in the head

region causing movements of the overlying skin and twitching of the ears (Sargison *et al.*, 1994; Scott *et al.*, 1995).

Ketosis in ruminants is a disease caused by impaired metabolism of carbohydrates and volatile fatty acids. Biochemically, it is characterized by ketonaemia, ketonuria, hypoglycaemia and low levels of hepatic glycogen. Clinically, the disease in cattle (acetonaemia) and in ewes and does (pregnancy toxaemia) are rather different entities and occur in different parts of the pregnancy-lactation cycle but the biochemical disturbance is essentially the same and they occur under similar conditions of management; all of which lead to a state of negative nutritional balance. The disease in cattle responds readily to treatment and is self-limiting; but the disease in sheep and goats is highly fatal (Blood *et al.*, 1983).

The present study was carried out in an attempt to obtain further information on the clinical, haematological and biochemical aspects and methods of treatment of field cases of pregnancy toxaemia in Shamia goats.

MATERIALS AND METHODS

Animals: Thirty nine pregnant Shamia goats were admitted to the Veterinary Clinic, College of Veterinary Medicine during the period between October 2004 to April 2006 with suspected pregnancy toxaemia symptoms including inappetence, star-gazing (Fig. 1) and recumbency.

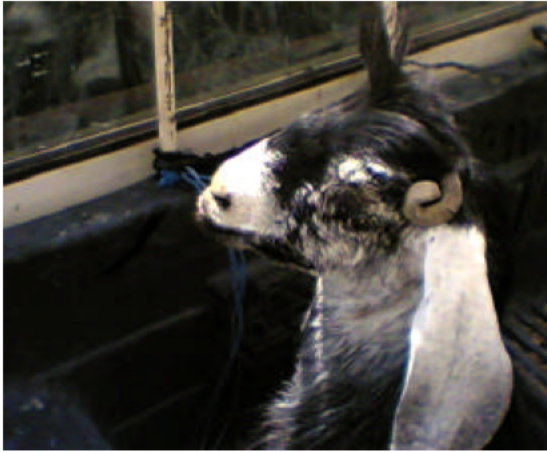


Fig. 1: Star grazing goat with mild stage of pregnancy toxemia

Clinical examination: All pregnant goats admitted to the veterinary clinic had in uterus 2-3 feti. Rectal temperature, heart beat, pulse and respiratory rates were determined. Visible mucous membranes and ruminal motility were examined.

Collection of blood samples: Blood samples were collected from the jugular vein of animals in 2 mL Ethylene Diamine Tetra Acetic Acid (EDTA) vacutainer tubes for hematological analysis and into 5 mL plain tubes for serum biochemical analysis. Serum samples were stored at -20°C until analyzed.

Haematological parameters: The analysis of haematological parameters was performed according to the methods of Jain (1993). Packed Cell Volume (PCV) was measured in a microhaematocrit centrifuge (Hawksley and Sons Ltd, England). Haemoglobin concentration (Hb) was estimated by cyanmethaemoglobin method, Red Blood Cell (RBC) and White Blood Cell (WBC) counts were made by Coulter counter (ZF6, Shimadzu, Kyoto, Japan).

Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH) and Mean Corpuscular Haemoglobin Concentration (MCHC) were calculated from PCV, RBC and Hb values.

Glucose tolerance test: This was performed on 24 h fasting goats which were given intravenous injection of 50% dextrose at a dose of 1 g kg⁻¹ body weight. Collected blood samples at 0, 1, 2, 3, 4, 5 and 6 h post-dosing were kept in chilled tubes from healthy goats and at 0, 2, 4 and 6 h from pregnancy toxemic Shamia goats.

Determination of biochemical parameters: The VETTEST 8008 biochemical and Analyzer (Sanofi Animal Health Ltd, England) was used to determine the activities of serum Aspartate Aminotransferase (AST), Lactic Dehydrogenase, (LDH) and Creatine Kinase (CK) and concentrations of total protein, albumin, globulin, bilirubin and urea by commercial kits (Roche products, Hertz, UK). Serum glucose was assayed one day after the experiment using Trinder assay kit (Sigma Chemical Corporation, Italy).

Treatment: The treatment for admitted goats with pregnancy toxemia started by re-hydration of the animal using 25% dextrose mixed by electrolyte and vitamin B complex (Fortemil Ourfino Animal Health, Brazil) at dose of 500 mL given by intravenous route.

Termination of pregnancy was completed by giving combination of dexamethasone as sodium phosphate 2mg by the dose of 3 mL intramuscular (Dexafar Farvet Laboratories B.V., Holland).

Hormone therapy by using prostaglandin F2 (Synchromati® BREMER PHARMA, Germany) 1 mL intra muscular.

An oral dose of propylene glycol (GLYCOL-M-SAVET, Saudi Arabia) at 100-200 mL for 5-10 days was given to support the liver and the kidney.

Sources of calcium and phosphorus were also given to all cases by using (Fosvitan, Diana, Spain) at 20 mL intramuscularly for 4 days.

Caesarian section also was done in 15 goats. The operation was done routinely using xylazine (Rombun 2%, Bayer, Germany) and local infiltration anaesthesia of the left flank using lidocain 2% (Vetoquinol, France).

Statistical analysis: The data were compared by student "t" test (Schlaggedee *et al.*, 1992) and $p < 0.05$ was considered statistically significant.

RESULTS AND DISCUSSION

Anorexia and depression were the first signs observed in both mild and advanced pregnancy toxemic goats. Sternal recumbency and locomotor disturbances including incoordination in gait and muscle tremors of goats were previously recorded by El-Sherif *et al.* (1978), Smith (1990) and El-Sebaie (1992, 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. In this study, salivation and blindness observed in goats suffering from toxemia were in accord with the findings of Buswell *et al.* (1986) and Sargison *et al.* (1994).

Table 1: Percentage fall from mean±SD of glucose (mg dL⁻¹)

Time (h)	Healthy goats		Toxaemic goats	
	Value	% fall from 0-6 h	Value	% fall from 0-6 h
0	55.16±2.11		(0)33.18±1.09	
2	206±7.1	55.0	(2)161.67±5.88	62.80
4	122.5±6.1	80.05	(4)106.8±4.9	78.3
6	57.16	99.40	(6)55	98

*p<0.05

Table 2: Haematological parameters (mean±SD) of pregnancy toxaemic goats

Parameters	Goats	
	Healthy	Diseased
RBC (10 ⁶ mm ⁻³)	7.30±0.33	5.79±0.22
Hb (g dL ⁻¹)	12.86±0.26	11.40±0.24
PCV (%)	44.80±0.70	42.00±4.06
MCV (m ³)	61.21±3.12	70.96±7.36*
MCH (pg)	17.45±0.92	19.18±0.91
MCHC (%)	28.55±0.37	28.84±3.22
WBC (×10 ³ mm ³)	5.56±0.27	5.61±0.43

*p<0.05

Table 3: Blood biochemical parameters (mean±SD) in goats with pregnancy toxaemia

Parameters	Goats	
	Health (7)	Diseased(6)
Glucose (mg dL ⁻¹)	55.16±2.3	33.10±1.6
Pyruvate (mol L ⁻¹)	172.10±12.1	240.00±22*
Oxalacetate (mmol L ⁻¹)	100.20±9.1	85.00±8.1
Lactate (mg dL ⁻¹)	5.10±0.65	10.00±1.1*
Plasma total volatile Fatty acids (mEq L ⁻¹)	45.10±1.4	42.00±1.6
Non-esterified fatty acids (mg dL ⁻¹)	4.86±0.57	10.20±1.1*

*p<0.05

Table 4: Serobiochemical values of goats with pregnancy toxaemia

Parameters	Healthy goats	Toxaemic goats
AST (i.u)	6.7±0.3	68±0.3*
LDH (i.u)	128±12	126±13
CK (i.u)	66.3±4	100.2±6*
BUN (mg dL ⁻¹)	6.2±0.5	90±0.6*

*p<0.05

Laboratory findings revealed the presence of ketone bodies in the urine of clinically affected goats. The results obtained for serum glucose indicated ketotic goats (Table 1). Story and Rook (1962) and Jonsson and Pehrson (1972) found that the glucose levels were related to the animals' energy status, values falling with a negative energy balance. Affected does 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).

As presented in Table 2 there were no significant differences in haematological parameters except MCV that showed increased level (p<0.05).

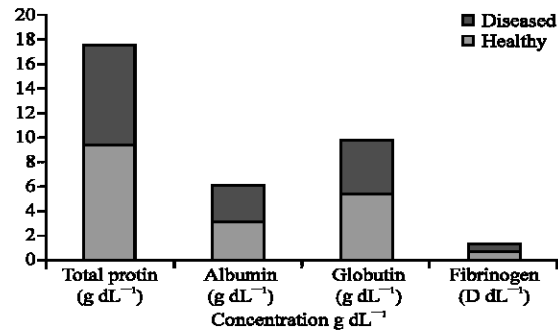


Fig. 2: Serum total protein and fractions of goats with pregnancy toxaemia

Concerning serum lipid pattern (Table 3), 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 triglycerides were reduced. Henricson *et al.* (1977) concluded that the lower triglyceride contents are the result of reduced appetite in animals with hyperketonaemia. The sharp increase in the concentrations of free fatty acids in the sera of both affected goats could be attributed to the increased mobilization of fatty acids from the adipose tissue 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.* (1976) suggested that plasma free fatty acids would be the most useful index of the degree of under nourishment in pregnant goats.

The elevated values of serum urea (Table 4) 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 goats. The increase in the activities of serum enzymes (LDH, AST and ALP) in diseased animals are a strong evidence for the degree of their liver damage. EL-Sebaie *et al.* (1992) observed severe hepatic changes in ketotic goats.

The obtained results for serum protein electrophoretic pattern in ketotic animals (Fig. 2) 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.

Despite the fact that all goats in the study were ketotic, there was a wide range of plasma glucose

concentrations. In this study, the response to concentrated oral rehydration solution therapy was not related to the severity of hypoglycaemia at initial presentation. Several additional factors, including the individual goat's capacity for gluconeogenesis (Marteniuk and Herdt, 1988) may influence the prognosis of the disease.

In non-pregnant goats, plasma glucose concentrations have been shown to be significantly higher 2 h after dosing with 160 mL of an oral rehydration solution than when the recommended dose of glycerol or propylene glycol was given (Buswell *et al.*, 1986).

McClymont and Setchell (1956b) noted that after development of clinical signs of ovine pregnancy toxemia, when insulin-induced hypoglycaemia was less than a few hours duration, returned to normality was rapid, but when hypoglycaemia was prolonged, goats did not recover. In this study, goats with naturally-occurring pregnancy toxemia whose plasma glucose concentrations rose significantly within 48 h of treatment, made a complete recovery. Conversely, goats whose plasma glucose concentrations were unaltered, or fell following treatment, died or eventually required human destruction. The finding of persistent hypoglycaemia in unresponsive cases of caprine pregnancy toxemia in this study provides indirect support for the postulate that irreversible cerebral pathologic changes develop in unresponsive goats with pregnancy toxemia (Jeffrey and Higgins, 1992). There is a suggestion that the hepatic gluconeogenic response may be different between susceptible and non-susceptible ewes (Marteniuk and Herdt, 1988).

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