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CLINICOBIOCHEMICAL STUDIES ON PREGNANCY TOXAEMIA IN SHEEP IN SHARKIA GOVERNORATE

(With 4 Tables)

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الدراسات الأكلينكية والبيوكيميائية لتسمم الحمل فى الأغنام بمحافظة الشرقية

إبتسام محمد جمال الدين ، فؤاد حامد السنجرى

تمت هذه الدراسة على عدد ٤٠رأس من النعاج البلدية العشارالتى تعانى من تسمم الحمل وتنتمى هذه النعاج الى مزرعة خاصة بمحافظة الشرقية. وكذلك عدد ١٠رأس من النعاج الأصحاء تماما كمجموعة ضابطة وتنتمى هذه النعاج الى مزرعة أخرى بمحافظة الشرقية.وكانت جميع الحيوانات فى الفترة الأخيرة من الحمل وكان الهدف من البحث هوالتشخيص المبكر للمرض من خلال دراسة التغيرات الأكلينيكية والبيوكيميائية للنعاج التى تعانى من تسمم الحمل. قسمت الحيوانات المصابة الى مجموعتين تبعا لشدة الاعراض الأكلينيكية: المجموعة الأولى: ظهرت عليها أعراض متوسطة. المجموعة الثانية: ظهرت عليها أعراض شديدة وشملت كل منهما عدد متساو من النعاج وهو ٢٠ رأس وقد سجلت أهم الأعراض الاكلينيكية للمرض فى المجموعتين. وتم أخذ عينتى دم من كل من الحيوان المريض والسليم: -الأولى: لدراسة التغيرات الهيماتولوجية حيث أوضحت النتائج زيادة العدد الكلى لكرات الدم الحمراء والبيضاء وهيموجلوبين الدم وزيادة حجم الخلايا المضغوطة. الثانية: لدراسة التغيرات البيوكيميائية حيث أوضحت النتائج زيادة الأجسام الكيتونية والالانين أمينو ترانسفيريز والفوسفاتيز القاعدى والأحماض الدهنية فى الحيوانات المريضة بمقارنتها بالمجموعة الضابطة بينما وجد انخفاض معنوى للجلوكوز والكلولستيرل والكالسيوم -البروتين الكلى- الألبومين والجلوبلوينات فى الحيوانات المريضة بمقارنتها بالحيوانات السليمة. وأوضحت التحاليل الوصفية الكيميائية للبول ارتفاع معدل الأجسام الكيتونية والجلوكوز والبروتين للحيوانات المصابة عند مقارنتها بالحيوانات السليمة. وعلى هذا فانه يمكن استنتاج إن نقص نسبة السكر فى الدم الناتجة عن الخلل بين كمية الجلوكوز المقدم واحتياج الحيوان خاصة اذا كان عدد الأجنة أكثر من واحد وفى الفترة الأخيرة من الحمل فيعتمد الحيوان على الدهون كمصدر للجلوكوز فتزداد نسبة ألاجسام الكيتونية ومن ثم يحدث تسمم الحمل. وعلى هذا نوصى بتقديم العليقة المتزنة وخاصة فى الشهور الأخيرة من الحمل وزيادة عدد الاجنة عن واحد كما يوصى بانه مع ظهور الاعراض الخفيفة مثل عدم القابليه للطعام وزيادة إفراز اللعاب بأن يجرى للحيوان تحليل السكر بالدم وكذلك الأجسام

الكيتونية بالدم والبول وفى حالة نقص الأولى وزيادة الثانية فإنه يجب أن تزداد كمية الكربوهيدرات فى الغذاء تفاديا لحدوث مرض تسمم الحمل.

SUMMARY

This study was carried out on 40 pregnant toxaemic native breed ewes. They were obtained from a private sheep farm in Sharkia Governorate and 10 clinically healthy pregnant ewes from another farm (served as a control group). All animals were in a late stage of pregnancy This work was done to study clinical and biochemical changes in ewes suffering from manifestations of pregnancy toxaemia as these alterations may help prediction of the disease. The diseased ewes were divided into two groups according to clinical findings: Group 1: Moderately affected. Group 2: severely affected, each group comprised 20 ewes. The clinical findings were recorded. Two blood sample were taken from each of the healthy control and the diseased animals. First blood sample was taken on sodium salt of EDTA for hematological studies. TRBCs and TWBCs counts, Hb concentration and PCV were increased significantly ($P \leq 0.01$) in the two groups compared to the healthy control. Second blood sample was collected in a clean centrifuge tube where serum was separated for biochemical examinations. Total ketone bodies –ALT-alkaline phosphatase and free fatty acids were increased significantly compared to the control group-But glucose –calcium levels-total protein –albumin-globulin-total cholesterol and triglycerides were decreased significantly compared to the control group. Urine examination indicated the presence of ketone bodies, glucose and protein in pregnant toxaemic ewes compared with the healthy control ones. It could be concluded that high concentration of ketone bodies in blood and urine in addition to hypoglycemia are markers of pregnancy toxaemia and their presence deserves early interference to safe the animal's life.

Key words: *Pregnancy toxaemia, sheep*

INTRODUCTION

Pregnancy toxaemia is a metabolic disease that causes economic losses in small ruminants, especially sheep (Cantley *et al.*, 1991). The animals were usually affected in last few weeks of pregnancy, especially if the uterus contains two or more well developed fetuses (Tontis and Zwahlen, 1987). Insufficient feeding program in these animals together with the increase in glucose consumption of rapidly growing fetuses

induce hypoglycemia and more catabolism of fat as a source of energy resulting in ketosis (Marteniuk and Herdt, 1988). Ketone bodies are normally produced by the rumen wall and liver (Radastits *et al.*, 1997). The rumen wall ketone production becomes insignificant during the clinical ketosis, however the liver is the major source of over all ketone production during ketosis (Bergman, 1971). The clinical manifestations in pregnancy toxaemia in sheep are reduced appetite, dullness, decrease ruminal movement, dyspnea, recumbency and blindness (Kronfeld, 1972 and EL.Sebaie 1995). In addition to neurological signs in the form of tremors of the muscle of the head (Scott *et al*., 1995)

The aim of this work is to study clinical and biochemical alterations in pregnancy toxaemic ewes from which we can predict the early onset of the disease.

MATERIALS and METHODS

This study was carried out on 40 pregnancy toxaemia native breed ewes of different ages (1-3 years old). They were obtained from a private sheep farm in Sharkia Governorate. Ten clinically healthy pregnant ewes (of comparable age) from another farm were used as a control group. All the study and control animals were in the last few weeks of pregnancy.

The common ration used in the affected ewes was grass hay, rice straw and small quantities of concentrates (from 100-150gm/head daily). Animals were housed in open yards without shelter in winter. The healthy ewes were fed adequate amounts of concentrate mixture and roughage. All animals in the two farms were given routinely falbazin syrup against hepatic and intestinal worm.

The diseased ewes were divided into two groups according to severity of the clinical findings: Group1: moderately affected ewes, constituted 20 animals. The findings in these ewes were loss of appetite, dullness, salivation, grinding on the teeth, sternal recumbency, moderate dehydration in addition to acetone-like odor in their breath and urine.

Group 2: severely affected ewes constituted 20 animals with more severe clinical signs: Animals showed complete loss of appetite, weakness, tremors of the muscle of the head, sternal then lateral recumbency, dehydration, blindness, severe tachycardia and severe acetone- like odor in their breath and urine.

Blood, urine and fecal samples were collected from healthy and affected ewes.

1-blood samples: Two blood samples were taken from each of the healthy and affected animals through jugular vein. The 1st sample was taken on sodium salt of EDTA for determination of TRBCS, count, TWBCS count, Hb concentration and PCV (Jain, 1986). The second sample was collected in clean centrifuge tube and serum was separated for determination of serum glucose (Trinder, 1969), Total ketone bodies (Mac Murrary *et al.,* 1984) ALT (Reitman and Frankel, 1957) Alkaline phosphatase (Belfield and Goldberg, 1971), total protein (Peters, 1968), serum albumin (Drupt, 1974) serum calcium (Gindler and king 1972), inorganic phosphorous (Goldenberg, 1966) total cholesterol (Melattini, 1978), free fatty acids (Schuster and Pilz, 1979) and triglycerides (young and postaner, 1975).

2- Urine sample were immediately analyzed using Combur-9 test strep (Boeheringer, Mannheim, Germany) for qualitative determination ketone bodies, glucose and protein.

3-Fecal samples were examined microscopically for parasitic infestation (Solusby 1982).

The obtained results were statistically analyzed using T test (Armitage and Berry, 1990).

RESULTS and DISCUSSION

Pregnancy toxaemia in sheep is a metabolic disease initiated by negative energy balance and results in excessive lipid metabolism, ketosis with subsequent hepatic lipidosis (Robert., 2000).

In the present study, The observed clinical signs of affected ewes were s similar to those previously described by Scott *et al.,* (1995); Nasser *et al.*, (1998); El Bealawy; (2000) and Abd El Rahman; (2003). The observed clinical signs may be attributed to the decline in glucose intake which leads to gluconeogenesis and more fat catabolism resulting in ketone body formation (Fouda and Gehad (1998). The neurological signs of affected animals resulted from cerebral hypoglycemia (Scott *et al.,*1995).

Fecal samples examination of both case study and control animals, revealed that all animals were free from any internal parasitic infestation.

Serum biochemical parameters in this study (Table1) revealed a significant decrease ($p \leq 0.001$) in glucose level in moderate and severe cases when compared with the control group. Similar result was obtained by Rizk and Omran (2004) in cattle and Sackran *et al.*, (2004) in goats. This may be due to excessive needs of the developing fetus (especially when multiple) to more glucose resulting in glucose deficiency and also due to disturbance in the hepatic gluconeogenesis (Linklater and Smith, 1993)

The results of the present study showed significant increase in serum ketone bodies ($p \leq 0.001$) in both moderate and severe pregnancy toxaemic ewes compared with the healthy control. Similar result was obtained by Rizk and Omran (2004) in cattle and Sackran *et al.*, (2004) in goats. This may be due to disturbance in carbohydrates and fat metabolism leading to hypoglycemia and mobilization of fat stores which leads to hepatic ketogenesis (Weirda *et al.*, 1985). The severity of hyper ketonemia would be increased if there were any limitation to utilization of ketone bodies by the peripheral tissues, either as consequence of decreased activity of ketone body utilizing enzymes in these tissues (Baird, 1982) or as a consequence of the decrease in insulin concentration (Hove, 1974).

The present study revealed significant decrease ($p \leq 0.01$) in calcium level in diseased groups. Similar results were obtained by Nasser *et al.* (1998) and El-Bealaway (2000). Robert (2000) attributed hypocalcaemia to anorexia and metabolic acidosis. Also Radostits *et al.*, (2000) concluded that at any stage of ketosis, calcium is probably lost through urine.

Inorganic phosphorous was not significantly changed in diseased groups compared with the control group. The significant increase of (ALT $p \leq 0.01$) and ALk. Phosphatase enzymes ($p \leq 0.05$) in diseased ewes compared with the healthy control in this study may be due to hepatic damage as mentioned by El-sebaie *et al.* (1992). They observed severe hepatic changes in ketotic ewes and does. Also, Reid *et al.*, (1983); Sargison *et al.* (1994) and Robert (2000) attributed the change in liver function to hepatic lipidosis which leads to disrupt hepatic cells with leakage of these enzymes into the blood.

Blood serum total protein, albumin and globulins (Table 2) were significantly decreased ($P \leq 0.01$) in studied animals. This may be due to anorexia of pregnancy toxaemic animals. These results were similar to those obtained by El-Sayed and Siam (1994) and Nasr *et al.* (1997). The decrease in blood serum total proteins may be due to inadequate diet

resulting from anorexia and mal-absorption (Benjamin, 1984). It could be attributed to the decrease in the hepatic synthesis of albumin (Kaneko, 1989).

A significant reduction of total serum cholesterol level ($p \leq 0.001$) in this study may be due to disturbance of fat metabolism in liver with fat infiltration (El-Bealawy., 2000). These results which is rather similar to those obtained by El-sayed and Siam (1994) and Nasr *et al.* (1997).

There was a significant ($p \leq 0.001$) decrease in the concentration of triglycerides in this study. Such decrease may be due to increase uptake of lipid by hepatic cells leading to hepatic lipidosis with consequent reduction in hepatic output of triglycerol which reduces the level of circulating triglycerides (Herdt *et al.*, 1997). There was a significant increase in free fatty acids ($P \leq .001$) in pregnant toxaemic ewes in this study compared with the control. The sharp increase in the concentration of free fatty acids in blood of the affected ewes could be attributed to increased mobilization of fatty acids from adipose tissues in response to an increased requirement for endogenous substrate for energy production during pregnancy (Noble *et al.*, 1971). Russel *et al.*, (1967) suggested that the plasma free fatty acids would be the most useful index of the degree of under nourishment in pregnant ewes.

There was a significant ($P \leq .001$) increase in TRBCS, TWBCS, Hb and PCV (Table3). This may be due to dehydration of pregnancy toxaemic animals. Similar results were reported by El-Bealawy *et al.* (1985) and Nasr *et al.*, (1997).

Qualitative examination of urine sample (Table 4) showed different concentration of ketone bodies in urine in moderate and severe pregnancy toxaemic ewes. The level of ketone bodies in the urine was comparable to the degree of illness, a fact that is attributed to increased fat hydrolysis by maternal tissues that convert the resulting glycerol to glucose and oxidation of fatty acids for energy (Cleon, 1988). Also glucosuria was detected in moderate and severe cases while proteins detected only in severe cases of pregnancy toxaemic that may be indicating renal affection.

It could be concluded that the alteration in biochemical parameters of pregnant toxaemic ewes are progressive from moderate to severe blood serum cases indicating that glucose deficiency causes these alteration in an ascending manner synchronous with the clinical manifestations. Thus adequate glucose in ration can prevent the occurrence of the disease. Combined estimation of ketone bodies in

serum and urine and glucose in serum can predict the disease in its early stages.

So it is recommended that pregnant ewes should receive diet containing sufficient carbohydrates especially in the last stage of pregnancy particularly when pregnancy is multiple. Periodical examination of farms is mandatory, as the appearance of minimal clinical manifestations in pregnant ewes as dullness, loss of appetite, salivation should be followed by examination of ketone bodies and glucose in blood and urine. High levels of ketone bodies in blood and urine together with low blood glucose can lead to predication of the disease and early treatment.

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Table 1: Some blood serum biochemical parameters (mean ±S.E.) in both control and pregnancy toxaemic ewes.

Parameters	Healthy control	Pregnancy toxaemic cases	
		Moderate	Severe
Glucose (mg/dl)	48.0±0.94	33.0±1.58***	24.0±2.02***
Ketone bodies (mg/dl)	5.66±0.15	7.80±0.32***	9.90±0.43***
Calcium (mg/dl)	10.0±0.35	7.08 ± 0.77**	6.2± 0.67**
Inorganic phosphorous(mg/dl)	5.5±0.25	5.2±0.13	5.7±0.24
ALT (U/L)	25.0±0.71	33.0 ±1.84**	40.00 ±2.28***
Alkaline phosphatase (U/L)	3.90±0.14	4.9 ± 0.42*	5.5 ±0.39*

*Significant (p≤.05) ** Significant(p≤0.01) *** Significant (p≤0.001)

Table 2: Proteinogram and Lipidogram in blood serum (mean ±S.E.) of both control and pregnancy toxaemic ewes

Parameters	Healthy control	Pregnancy toxaemic cases	
		Moderate	Severe
Total protein (gm/dl)	6.9±0.17	5.70 ± 0.16**	4.8±0.28***
Albumin (gm/dl)	3.4±0.13	2.9±0.11*	2.68±0.08**
Globulins (gm/dl)	3.5±0.10	2.8±0.08**	2.12±0. 31**
Total cholesterol (mg/dl)	75.0±1.41	59.0±2.52***	55.0± 2.28***
Triglycerides (mg/dl)	55.0±1.14	42.0± 2.02***	37.0 ± 2.29***
Free fatty acids (mg/dl)	26.5 ±0.79	48.5±1.36***	55.0 ± 2.47**

*Significant ($p \leq 0.05$) ** significant ($p \leq 0.01$) *** significant ($p \leq 0.001$)

Table 3: Some hematological parameters (mean ±S.E.) in both control and pregnancy toxaemic ewes.

Parameters	**Healthy control**	**Toxaemic cases Pregnancy**	
		Moderate	**Severe**
TRBCs($\times 10^6$ ul).	9.7±0.23	11.6±0.43**	13.44±0.36***
Hb (gm/dl)	10.5±0.29	12.0±0.29**	13.5±0.57**
Pcv (%)	35.0±0.70	34.0±1.70*	44.0±2.02**
TWBcs($\times 10^3$ul).	6.5±0.18	8.2±0.38**	9.5±0.43***

*Significant ($p \leq 0.05$) **significant ($p \leq 0.01$) *** significant ($p \leq 0.001$)

Table 4: Results of qualitative urine examination in both healthy control and pregnancy toxaemic ewes.

parameters	Healthy control	Pregnancy	Toxaemic ewes
		moderate	Sever
Ketone bodies	–ve	++	++++
Glucose	–ve	++	++++
Protein	ve-	-ve	++