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BIOCHEMICAL ALTERATIONS IN PREGNANCY TOXEMIC EWES AND TRIALS OF TREATMENT

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ABSTRACT

The purpose of the current study was to assess the impact of pregnancy toxaemia on hematobiochemical and hormonal changes together with treatment trial of diseased ewes. This study was done on 15, 3-4 years old (5 healthy-10 ewes suffered from pregnancy toxaemia) were divided into 3 groups (5 /group). Gp (1) clinically healthy ewes (control group), Gp (2) diseased ewes received 150 ml of dextrose (25%) (I/V), 100 ml propylene glycol daily for3 sucessive days (orally), 1ml Cal-De-Mag kgm/ bwt and 1 ampole of vitamin B complex IM, Gp (3) diseased ewes treated with same drug and dose used in 2nd group in addition 0.4 unite insulin/kgm bwt (S/C) daily for 3 sucessive days. Urine samples were collected pre-treatment and post treatment for detection of ketone bodies, glucose and protein. Blood was drawn from each subject ewes pre-treatment and at 5 & 10 days post treatment for determination hematobiochemical and hormonal parameters. Pregnancy toxaemic ewes exhibited many signs such as anorexia, depression, disinclined to move - blindness, excessive salivation, teeth grinding, a listening attitude and sternal recumbency. the number of feti in control group was 1.40 but in ewes suffered from pregnancy toxemia were 2.20 in Gp (2) and 2.60 in Gp (3). Urine of diseased ewes revealed presence of ketone bodies, glucoserea and protein. The obtained results demonstrated that pregnancy toxemia induced significant decreases in total erythrocytic count (RBCs), hemoglobin (Hb), paked cell volume % (PCV%), insulin, triiodothyronin (T3), thyroxin (T4), immunoglobulins (IgA, IgM, IgG), super oxide dismutase (SOD), catalase (CAT) glucose, and calcium beside significant increases in cortisol, aspartate aminotransferase (AST), alanin aminotransferase (ALT), urea, creatinine and β-hydroxybutyric acid (β-HBA). Additionally marginal increases in total leukocytic count (WBCs), eosinophils, and lymphocytes coupled with insignifcant decreases in neutrophils, monocytes and basophil when compared with the control ewes were also observed. Teatment of the diseased ewes lead to reterned hemato-biochemical parameters to nearlly nomal level at 10th day post teatment when compared with control ewes. Ewes received insulin in treatment the hematobiochemical parameters was improvement than ewes not received insulin. It was determined that pregnantcy toxaemia caused numerous negative effects on hematobiochemical parameters but treatment with dextrose, propylene glycol, Cal-De-Mag, vitamin B complex and insulin could improve such negative effects

Keyword: Ewes; Pregnancy toxemia; triiodothyronin; thyroxin; immunoglobulins; blood picture; glycose; liver enzymes

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INTRODUCTION

One of the common metabolic disorders in ruminants is pregnancy toxaemia (Rook, 2000). Pregnancy toxaemia occures in straight forward pregnancies with large foetuses. Pregnancy toxaemia affects sheep and goats during the last 4 to 6 weeks of pregnancy when they are carrying twins or triplets (Radostits *et al.*, 2007). Pregnancy toxemia is a disease caused by a dysfunctional metabolism of volatile fatty acids and carbohydrates (Blood *et al.*, 1981) and fatty infiltration of the liver and kidney (Henderson *et al.*, 1982). Pregnancy toxemia occurrence is related to a negative energy balance brought by inadequate dietary intake and increased energy requirements of late gestational fast growth (Smith, 2002). It causes economical losses due to substantial mortality among the affected animals (Tawfik *et al.*, 2005). In addition, Pregnancy toxemia occurred due to negative energy demand for the fetus's rapid growth during. pregnancy toxaemia late gestation and insufficient energy intake (Abdul, 2003). Pregnancy toxemia is a metabolic disorder characterized by ketonaemia, ketonurea, hypoglycaemia, and low levels of hepatic glycogen beside showed neurological signs and weakness (Prieto,1994 and Martin and Aitken, 2000). Hypoglycaemia in ewes suffering from pregnancy toxemia was revise due to increase glucose requirement for feti in uterus (more than 40% of the total liver glucose output) beside sever changes in endocrinological status in late pregnancy (Lindsay and Oddy, 1985).

Biochemically pregnancy toxaemia characterized by marked hypoglycemia and ketosis (Nasser *et al.* (1998) beside sever changes in lipid profile represented by decrease of serum lipid values meanwhile serum macro elements (calcium, phosphorus, magnesium, sodium and potassium) significant decrease (Judith and Thomas, 1988). Pregnancy toxaemia induce impared hepatorenal function represented by significant increase in serum alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase, serum urea and creatinine (Nagamani *et al.* (1996).

Treatment of pregnancy toxaemia is done either through a caesarean delivery or artificially inducing parturition with corticosteroids, and increasing blood glucose are the two other methods (Wierda *et al.*, 1985).

This study's objective was to examine the clinical profile of pregnancy toxaemia in sheep as well as detection of the alterations in the blood picture, hormonal and some serum biochemical parameters together with treatment of the affected ewes.

MATERIAL AND METHODS

Drugs:-

1) **Dextrose (25%)**: produced by El Nasr for chemical pharmaceuticals company

2) Cal-De-Mag: produced by Pfizer for chemical pharmaceuticals company

3**) Propylene glycol:** produced by Nile for chemical pharmaceuticals company

4) Tri-B: produced by El Nasr for pharmaceuticals and chemicals industries

5) Insulin (Mixtard, 30)^{R} injectable sterile solution from Nova Nordisk AIS (Denemark) available as 10 ml vial each one milliliter contain 100 I U/ml Insulin.

Animals

The study was done on 15 ewes, 2-4 years old during last 2-4 weeks of pregnancy (5 ewes clinically healthy, and 10 ewes suffering from pregnancy toxemia) These ewes were belonged to a private farm at Abo Hamad City-Sharkia Provence. (Egypt) and showed clinical signs represented by weakness, lack of interest, and teeth-griding while alone in a corner, slight nasal discharge, standing difficulties, sternal recum-bency, lack of coordination, and, occasionally, spasms of the muscles and partial blindnes.

Experimental desgin

Ewes were divided into three equal groups (Gp) (5 ewes /group). Each group was housed separately in open yard, Gp (1) was clinically healthy ewes servd as control group, Gp (2) ewes were suffering from pregnancy toxemia treated with 150 ml of dextrose (25%) (I/V), 1 ml Cal-De-Mag/ kgm/ bwt (I/V) 100 ml propylene glycol daily for 3 sucessive days (orally) and 1 ampole tri B (IM) but Gp (3) ewes suffering from pregnancy toxemia were treated with same

drugs and doses used in the 2nd group in addition to 0.4 unit insulin/kgm bwt (S/C) daily for 3 sucessive days.

Urine samples were collected pre and post treatment for urine analysis using Comber-9test strep (Boeheringer, Mannheim, Germany) protein and ketone substances for qualitative analysis.

Four blood samples from each ewes pre treatment, 5 and 10 day post treatment were collected.

1st sample was taken in a tube contains sodium florid for estimation of glucose (**Trinder 1969)**.

2nd sample was taken in a tube contains heparin for measuring phagocytic activity and index **(Wilkinson, 1977; Lucy and Larry, 1982).**

3rd sample was taken in a tube contains EDTA for determination of haemogram and leukogram according to **Feldman *et al.* (2000)** by using cellcounter (SysmexXT-2000iv)

4th **sample** was taken and centrifuged to obtain clear for determination of

- cortisol (**Abraham, *et al.*, 1972)**, insulin **(Burtis *et al.*, 1994)**, triiodothyronine and thyroxin (**Abraham, 1981).**

- The test kits were used for colorimeteric estimation by using spectrophotometer in measuring AST-ALT (Reitman and Frankel, 1957), ALP (John, 1982), Beta-hydroxy buteric acid (Mercer *et al.,* 1986), calcium (Glinder and King, 1972), urea (Coalombe and Faurean, 1963) creatinine (Husdan and Roporpot, 1968). Super oxide Dismutase (Nishikimi *et al.* 1972), catalase (Sinha, 1972), Malondialdehyde (Nielsen *et al.,* 1997), Serum immunoglobulins (IgA, IgG, and IgM) were performed by ELISA (Erhard *et al.*, 1992).

NB Kits used in measurement of biochemical parameters were obtained from (Bio Diagnostic Egypt and Diamond. Egypt)

Statistical analysis**:-** The data was analyzed by using computerized SPSS program version 16. results are present edasmean + SE. The data were analyzed by one-way ANOVA following by Duncan's test $p \leq 0.05$ were considered significant according to Tamhane and Dunlop (2000).

RESULTS

Clinical signs:- The average number of feti in the control group was 1.40± 0.36, but in ewes suffered from pregnancy toxemia were 2.20± 0.52 in Gp (2) and 2.20± 0.41 in Gp (3) beside presence many clinical signs as weakness, inappetence, dullness, grinding on teeth, un-concerned with the examination, secluded in a corner, inability to stand, sternal recumbency, incoordination, muscular tremors and partial blindness).

Results of urie analysis:- Urine analysis revealed presence of Ketone bodies and protein in urine pregnant, ewes (table 1).

Results of blood picture analysis:- Our results demonstrated that ewes suffered from pregnancy toxemia revealed significant decreases in RBCs, Hb, PCV%, phagocytic% and index beside non significant increases in WBCs, eosinophils and lymphocytes coupled with insignifcant decreases in neutrophils, monocytes and basophil which reterned to nearlly nomal levels at 10 days post teatment when compared with the control ewes (table 3).

Results of biochemical parameters analysis

a) hormonal results:- it has been noticed that pregnancy toxemia induced significant increases in cortisol beside significant decreases in insulin, T3 and T4 (table 2).

b)biochemical results:- Pregnancy toxemia increased serum AST, ALT, urea, creatinine and BHBA coupled with significant decreases in glucose and calcium immunoglobulins (IgA, IgM, and IgG) as compared with control ewes (table 4).

c**) oxidant and antioxidant results:-** Ewes suffered from pregnancy toxemia showed significant decreases in SOD, CAT and increased MDA (table 5).

Table 1: Examination of urine samples in ewes suffering from pregnancy toxemia.

Parameters	Healthy ewes Gp (1) (n=5)	Diseased ewes				
		Pre Treatment (n=10)	Post treatment			
			Gp (2) (n=5)		Gp (3) (n=5)	
			5 day	10day	5 day	10 day
Ketone bodies	-ve	+ve	-ve	-ve	-ve	-ve
protein	-ve	ve+	-ve	-ve	-ve	-ve

Table 2: Values ± standered errors of measured hormone in healthy and pregnancy toxemic (PT) ewes (mean± SE).

Parameter	Healthy ewes Gp (1) (n=5)	Diseased ewes				
		Pre Treatment (n=10)	Post treatment			
			Gp (2) (n=5)		Gp (3) (n=5)	
			5 day	10 day	5 day	10 day
Cortisol (ng/dl)	12.06± 0.89 c	17.16± 0.42 a	15.43± 0.19b	13.05± 0.32ab	14.21± 0.51ab	12.18± 0.43c
T3 (ng/dl)	128.06± 0.98a	105.12± 0.89c	119.42± 0.78ab	126.04± 0.93a	122.22± 0.91b	128.2± 0.88a
T4 (ng/dl)	4.50± 0.32a	2.94± 0.21c	3.44± 0.33b	4.43± 0.41a	3.98± 0.55b	4.49± 0.32a
Insulin (ng/dl)	9.52± 0.87a	5.07± 0.45c	7.96± 0.21b	8.21± 0.32a	8.89± 0.55a	9.21± 0.32a

Means with different superscripts of the same row indicate significant difference at $P < 0.05$
The results show significant difference must be demonstrated by symbol a, b and c

Table 3: means values ± standered errors of blood picture and phagocytic activity in healthy and pregnancy toxemic (PT) ewes (mean± SE).

Parameters			Healthy ewes Gp (1) (n=5)	Diseased ewes				
				Pre Treatment (n=10)	Post treatment			
					Gp (2) (n=5)		Gp (2) (n=5)	
					5 day	10 day	5 day	10 day
erythrogram		RBCs (x106/µL)	9.75± 0.19a	6.32± 0.33b	7.51± 0.31b	8.98± 0.21a	8.43± 0.53b	9.58± 0.42a
		Hb (g/dL)	10.54± 0.64a	5.69± 0.44c	7.88± 0.23b	9.12± 0.52a	8.87± 0.32b	9.98± 0.45a
		PCV%	31.21± 0.92a	22.34± 0.98c	26.43± 0.8b	29.9± 0.98a	26.9± 0.97b	29.98± 0.65a
Leukocytic count		WBCs (103/mm3)	8.11± 0.75a	9.38± 0.8a	8.72± 0.9a	8.13± 0.43a	8.16± 0.54a	8.15± 0.75a
	Differential	Neut (103/mm3	2.72± 0.31a	2.67± 0.12a	2.70± 0.43a	2.71± 0.21a	2.65± 0.34a	2.71± 0.26a
		Lym (103/mm3	3.03± 0.23a	4.48± 0.30a	3.66± 0.45a	3.07± 0.44a	3.22± 0.16a	3.10± 0.41a
		Mon (103/mm3)	0.99± 0.06a	0.88± 0.13a	0.97± 0.14a	0.98± 0.20a	0.87± 0.21a	0.98± 0.21a
		Eosin (103/mm3)	0.58± 0.09a	0.60± 0.05a	0.60± 0.07a	0.59± 0.10a	0.67± 0.08a	0.57± 0.06a
		Baso (103/mm3)	0.79± 0.08a	0.75± 0.05a	0.77± 0.06a	0.78± 0.06a	0.75± 0.05a	0.79± 0.10a
Phagocytic %			69.90± 0.98a	61.81± 0.84c	65.45± 0.33b	68.23± 0.93a	64.81± 0.69b	69.09± 0.33a
Phagocytic index			4.21± 0.21a	2.15± 0.11c	3.16± 0.17b	3.99± 0.21a	3.79± 0.33b	4.09± 0.54a

Means with different superscripts of the same row indicate significant difference at $P < 0.05$.
The results show significant difference must be demonstrated by symbol a, b and c

Table 4: means values ± standered errors of measured biochemical parameters in healthy and pregnancy toxemic (PT) ewes (mean± SE)

Parameters		Healthy ewes Gp (1) (n=5)	Diseased goates				
			Pre Treatment (n=10)	Post treatment			
				Gp (2) (n=5)		Gp (3) (n=5)	
				5 day	10 day	5 day	10 day
Liver enzymes	AST (U/L)	38.69± 0.91c	52.08± 0.85a	48.12± 0.95b	40.12± 0.66c	47.09± 0.88b	39.76± 0.98c
	ALT (U/L)	29.21± 0.78c	48.77± 0.89a	38.55± 0.83b	32.43± 0.77c	35.63± 0.96ab	30.76± 0.98c
	ALP (U/L)	42.31± 0.45a	56.13± 0.32c	47.23± 0.55b	41.54± 0.44a	46.55± 0.78b	42.13± 0.55a
Kidney function	Urea (mg/dl)	16.51± 1.53c	27.09± 1.12a	20.12± 1.54b	17.13± 1.05c	18.32± 1.20b	16.21± 1.23c
	Creatinine (mg/dl)	1.88± 0.09c	3.96± 0.21a	3.05± 0.33a	1.96± 0.43c	2.21± 0.12b	1.94± 0.24c
β-HBA (mg/dl)		15.17± 0.65c	22.62± 0.71a	20.43± 0.95b	16.68± 0.86c	18.64± 0.69b	15.12± 0.92c
Calcium (mg/dl)		8.87± 0.21a	4.56± 0.10c	6.75± 0.32b	8.32± 0.21a	6.78± 0.33b	8.81± 0.66a
Glucouse (mg/dl)		78.43± 0.87a	59.32± 0.97c	68.55± 0.89b	77.85± 0.87a	70.95± 0.8b	78.85± 0.82a
immunoglobulin	IgA (g/l)	1.98 ± 0.31a	0.87 ± 0.15c	1.30 ± 0.21b	1.84 ± 0.23a	1.36 ± 0.22b	1.94 ± 0.26a
	IgG (g/l)	10.07± 0.21a	7.12± 0.69c	8.73± 0.99b	9.62± 0.76a	8.94± 0.87b	10.02± 0.98a
	IgM (g/l)	4.56± 0.21	2.66± 0.32c	3.09± 0.21b	4.98± 0.67a	3.57± 0.51b	4.41± 0.32a

Means with different superscripts of the same row indicate significant difference at $P < 0.05$
The results show significant difference must be demonstrated by symbol a, b and c

Table 5: Means Valius ± standered errors of measured oxidand - antioxidant in healthy and pregnancy toxemic (PT) ewes (mean± SE)

Parameter	Healthy ewes Gp (1) (n=5)	Diseased ewes				
		Pre Treatment (n=10)	Post treatment			
			Gp (2) (n=5)		Gp (3) (n=5)	
			5 day	10 day	5 day	10 day
SOD (u/ml)	248.43± 3.84a	231.94± 3.32c	238.61± 4.54b	245.09± 5.09a	230.98± 4.21b	247.38± 4.93a
CAT (u/l)	143.43± 1. 34a	123.54± 1. 65c	137.5± 1. 26b	141.54± 1. 34a	139.13± 1.33b	142.13± 1. 33a
MDA (nmol/ml)	17.35± 0.91bc	25.13± 0.72a	21.15± 0.67b	16.23± 0.79c	22.16± 0.48b	17.06± 0.77bc

Means with different superscripts of the same row indicate significant difference at $P < 0.05$
The results show significant difference must be demonstrated by symbol a, b and c

DISCUSSION

The obtained data revealed that ewes suffered from pregnancy toxemia showed clinical signs represented by weakness, Inability to stand, sternal recumbency, incoordination, dullness, teeth grinding, not paying attention to the examiner, sitting alone in a corner, small nasal discharge, muscle tremors, and in some cases, partial blindness) and these clincal signs were

completly disappeared at 5 days post treatment. These clinical signs may be related to hypoglycemia and ketonaemia (Abd El-Raof and Ghanem 2006). These syptoms were in accordance with those recorded by Emam and Galhoom (2008) who noticed anorexia, depression, a lack of will to move, tiredness, increased salivation, and teeth grinding in ewes with pregnant toxemia. Similar signs was observed by Ali *et al.* (2013) in ewes suffering from pregnancy toxaemia. Same signs were observed also by Prasannkumar *et al.* (2016) observed many clinical signs as Anorexia, lethargy, opisthotonos, drooping head, intermittent convulsions, a sweet-fruity breath odour, apparent blindness, bloating, teeth-grinding, and foamy salivation in goats are some of the symptoms suffering from pregnancy toxemia. Besides, Rodolfo (2019) stated that ewes suffering from pregnancy toxemia showed inability to stand, sternal recumbency, incoordination. Gaadee and Gehan (2021) and Affana *et al.* (2022) stated that pregnancy toxemia induced many clinical indicators such recumbency, decrease of physical condition, weakness, incoordination, and muscular tremors

Our esults revealed that the number of feti affects the onset of the pregnancy toxemia. These results agreed with Prieto (1994) who stated that pregnancy toxemia occured mainly in the last third of pregnancy in animals presenting two or more feti. Same results were seen by Rook (2000) who stated that pregnancy toxemia affect ewes with multiple feti.

Analysis of urine sample collected from diseased ewes revealed presence of ketone bodies and protein that disappeared at 5 and 10 days post treatment in a comparsion with the control ewes. Ketone bodies may be present in urine due to enhanced fat breakdown by bodily tissues, which then converts the resultant glycerol to glucose and uses the oxidised fatty acids as fuel (Cleon 1988). These outcomes are somewhat comparable to those attained by Gmal El-Din and El Sangary (2005) in ewes suffering from pregnancy toxaemia.These results are inagreement with Emam and Galhoom (2008) stated that pregnancy toxaemia in ewes confermed by presence of ketone bodies and protein in urine.

The present investigation showed a significant increase in cortisol beside significant decrease in insulin, T3 and T4 in ewes suffering from pregnancy toxemia that returned to nearlly nomal at 10 day post teatment as compared with control ewes. Increase in cortisol may be due to the hepatic system's inability to process cortisol (Radostitis *et al.*, 2000). These results in accordance with that obtained by Adel and Sahar (2005) who stated that diseased gaot induced significant decrease in T3 and T4. These results were in accordance with that obtained by Kulcsár *et al.* (2006) who stated that pregnancy toxemia in ewes induced significant decreases in insulin, T3 and T4 beside significant increase in cortisol. Our result was supported by result of Ismail *et al.* (2008) who found that pregnancy toxemia induced significant decreases in insulin and thyroid hormone. In addition, Abd-Elghany *et al.* (2010) found that ewes suffering from pregnancy toxemia showed significant increase in cortisol and significant decreases in insulin and thyroid hormone. Our reults were simialr to the result of Khaled, (2011) who stated that pregnancy toxemia induced significant decrease in insulin, T3 and T4. These results were accoordinecess with Mahy et al (2017) who reported that ewes suffering from pregnancy toxemia revealed significant increase of insulin. Same observation was recorded by Affana *et al.* (2022) who reported that goat insulin levels dropped and cortisol levels rose in those with pregnant toxaemia.

The obtained results showed that ewes suffered from pregnancy toxemia revealed significant decreases in RBCs, Hb, PCV%, phagocytic%, and index beside insignificant increases in WBCs, eosinophils and lymphocytes coupled with insignifcant decrease in neutrophils, monocytes and basophil which returned to nearlly nomal at

10 days post teatment when compared with the control ewes. These results agreed with Benjamin (1984) who stated that reduction in Hb in diseased ewes mighty be caused by a lack of iron, protein, and energy, all of which are necessary for the synthesis of haemoglobin and erythropoietin. Sartorelli *et al.* (1999) recorded that pregnancy toxemia depressed phagocytic process. Our results were reinforced with that of Lacetera et al (2001) who found that diseased ewes revealed decrease in RBCs, Hb, PCV %, phagocytic% and index. Likely, Abd El-Raof and Ghanem (2006) observed reduction of Hb in ewes suffering from pregnancy toxemia. Besides, Gupta *et al.* (2008) recorded that goat suffering from pregnancy toxemia revealed RBCs, Hb, and PCV% significantly droped, whereas PCV% increases WBCs, and eosinophils. Sheep suffering from pregnancy toxemia showed leukocytosis, eosinophils and lymphocytosis due to presence of inflammations (Gavan *et al.*, 2010). In addition, Abd-Elghany *et al.* (2010) reported that pregnancy toxemia induced decreases in RBCs, Hb, and PCV%. These results were in agreement with those of Tharwat and Al-Sobayil (2014) who reported that pregnancy toxemia induced non significant increases in WBCs, eosinophils and lymphocytes beside non signifcant decrease in neutrophils, monocytes, and basophil. The obtained data are in accordance with those previously obtained by Abba *et al.* (2015) who mentioned that pregnancy toxemia showed non significant leukocytosis and lymphocytosis. Phagocytic %, and index, leukocytosis, neutrophilia, monocytosis, lymphocytosis, and eosinophilia were increased in goats suffering from pregnancy toxaemia (Khan *et al.* 2021). Our results were supported by result of Gaadee and Gehan (2021) who stated that pregnancy toxemia in sheep revealed significant decrease in RBCs and Hb coupled with non significant increases in WBCs, eosinophils and lymphocyte beside insignifcant decrease in neutrophils, monocytes and basophil.

In the present investigation, it has been shown that ewes suffering from pregnancy toxemia revealed increase in serum AST and ALT, urea, creatinine and BHBA coupled with significant decreases in glucose, calcium, and immunoglobulins (IgA, IgM & IgG), concentrations and reterned to nearlly nomal at 10 days post teatment as compared with control ewes. Same changes were reported by Nonnecke *et al.* (1992) who stated that ketotic cows inhibited immuonoglobulin secretion. Elevation in serum urea might be due to decreased glomerular filtration due to a significant fatty diet infiltration of kidney (Henze, *et al.* 1998). This conclusion was agreed with those expected by Nicola et al (2001) who concluded that pregnancy toxemia induced significant decreases in immunoglobulins (IgA, IgM and IgG). The obtined data matched those reported by Abd-Elghany *et al.* (2010) who observed that pregnancy toxemia induced significant increase in β-Hydroxybutyrate and significant decreases in immunoglobulins. These results which is rather similar to those obtained by Ismail *et al.* (2010) who stated that pregnancy toxemia in goats caused a considerable drop in glucose beside increase in urea and creatinine. Another support of these results was recorded by Menzies, (2011) who stated that pregnancy toxemia caused hypoglycemia. Similarly, Khaled, (2011) stated that pregnancy toxemia induced increase in serum β-hydroxybutyrate (BHB) beside decreases in cortisol and glucose. Same results were recorded by Ali *et al.* (2013) who mentioned that pregnancy toxemia induced hypoglycemia and hypo-calcemia. These results agreed with Aly and Elshahawy (2016) who reported that ewes suffering from pregnancy toxemia showed increases in serum AST and ALT. Pregnancy toxeamic in goat induced significant increases in serum AST, ALT, β-hydroxybutyric acid, urea, and creatinine associated with decreases in glucose and calcium (Prasannkumar *et al.*, 2016). These results agreed with Mahy *et al.* (2017) who stated that ewes suffering from pregnancy toxemia revealed significant increases in

AST and ALT beside insignificant deceases in serum calcium. Pregnancy toxemia induced a notable rise in the enzymes AST, ALT, urea, and creatinine (Samir and Eman, 2019). On similar grounds Gaadee and Gehan (2021) reported that elevation of AST and ALT in ewes suffering from pregnancy toxemia might be due to hepatic injury or hepatic lipidosis, a negative energy balance, and fat mobilisation. On similar grounds, Affana *et al.* (2022) reported that pregnant women who had toxemia showed a significant drop in glucose and calcium levels beside increase in concentrations of β-Hydroxybutyric acid.

In the current work, ewes suffered from pregnancy toxemia showed significant decrease in SOD, CAT and an incease in MDA that reterned to nearlly nomal levels at 10 days post teatment when compared with the control ewes. Reduction of antioxidants during pregnancy can result in oxidative stress (Sahoo *et al.*, 2009). In addition, Khaled, (2011) stated that ewes affected by pregnancy toxemia showed an increased MDA whereas SOD and CAT were declining. Pregnancy toxemia induced significantly higher MDA and lower SOD and CAT levels (Samir and Eman 2019). Same observation was recorded by Mayra *et al.* (2021) in ewes suffering from pregnancy toxemia. Same observation was recorded by Gaadee and Gehan (2021) who recorded that serum MDA increased with decreases in SOD and CAT.

In our study, data obtained post treatment of ewes suffering from pregnancy toxemia in both groups (Gp 1 and 2) using (IV) of dextrose (25 %), (Cal-De-Mag), propylene gly-col daily for four days beside vitamin B complex in addition to insulin for Gp (3) only leading to better response and improved healthy status in the two treated group and a disappear of the clinical signs and improved hematobiochemical and hormonal parameters. Similar results were reported after treatment of the diseased goat with dextrose solution and propylene glycol (Faris *et al.,* 2005). Similar results were also reported by Cal-Pereyra *et al.* (2015) who mentioned that dextrose solution and propylene glycol is very important in treatment of ovine pregnancy toxaemia. Ketogenesis was significantly reduced by glucose and its precursor (Araujo *et al.,* 2018). The previous results was supported by finding of Yasir et al (2021) who stated that supplementation of insulin in treatment of pregnancy toxemia is important to enhance glucose utilization and help in the treatment.

It could be concluded that pregnancy toxaemia caused numerous negative effects in hematobiochemical parameters but treatment with dextrose, propylene glycol, Cal-De-Mag, vitamin B complex, and insulin) improved these parameters.

Conflict of interest: The authors declare that they have no conflict of interest.

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الكشف عن تسمم الحمل فى النعاج مع محاولات العلاج

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أجريت هذه الدراسه لتقييم تأثير تسمم الحمل على صورة الدم ,الهرمونات بعض الوظائف البيوكيميائيه مع محاولـه العلاج.
تم إستخدام عدد ١٥ نعجه فى الشهر الاخير من الحمل بإحدى المزارع الخاصة بمحافظة الشرقية تتراوح اعمارها ٣- ٤ سنه.(٥ نعجه بصحه جيده -١٠ نعجه تظهر عليها اعراض مختلفه لتسمم الحمل تم تقسيم النعاج إلى ٣ مجموعات متساويه (٥ نعجه/منها) الأولى بصحه جيده (ضابطة). المجموعه الثانية نعاج تظهرعليها أعراض مرضية لتسمم الحمل ويتم علاج النعاج المصابة باستخدام ١٠٠سم محلول الجلوكوز(٢٥%) حقنا بالوريد- ١٠٠ملى بروبيلين جليكول عن طريق الفم مرة واحدة يوميا لمدة ٣ يوم- ١ملى كال دى ماج/ كجم من وزن الجسم حقنا بالوريد- ١ امبول فيتامين ب مركب حقنا بالعضل .
المجموعه الثالثه نعاج تظهر عليها أعراض مرضية لتسمم الحمل ويتم علاجها بنفس الادويه المستخدمه بالمجموعه الثانيه بالاضافه الى ٠,٤ وحده انسولين تحت الجلد. يتم أخذ عينة بول وعينات دم من كل نعجه قبل العلاج وبعدة ب٥ و١٠ يوم وذلك للكشف عن الاجسام الكتينونبه –الجليكوز والبروتين فى البول ولدراسة تأثير تسمم الحمل على صورة الدم التغيرات البيوكيميائية و الهرمونية لهذه النعاج.
أظهرت النتائج أن عينات البول المأخوذه قبل العلاج عن وجود الاجسام الكتينونبه –الجليكوز والبروتين ولكنها اختفت بعد العلاج
اصابه النعاج بالتسمم الدموى أدى إلى وجود نقص معنوى فى العدد الكلي لكرات الدم الحمراء، تركيز الهيموجلوبين، حجم خلايا الدم المرصوصة هرمون الأنسولين وهرموني الثيروكسين والترای ايودوثيرونين الجلوكوز والكالسيوم IgA, IgM , SOD, CAT , IgG, زيادة معنويه فى العدد الكلي لكرات الدم البيضاء هرمون الكورتيزول إنزيمات الترانس امينيزس(AST-ALT) الفوسفاتيز القاعدى حامض البيتاهيدروكسى بيوتريك ,اليوريا والكرياتينين الدهون الكليه والدهون الثلاثيه
علاج الاغنام المصابة بتسمم الحمل بالمستخدمه ادى الى استجابه النعاج للعلاج وتم شفائها تمام.
نستخلص من هذة الدراسة أن الاصابه بتسمم الحمل في النعاج يمكن تشخيصة مبكرا وقبل ظهور الأعراض و ذلك بقياس مستوى الجلوكوز والأجسام الكيتونية بالبول وكذلك الجلوكوز