HORMONAL HAEMATOLOGICAL BLOOD BIOCHEMICAL
CHANGES IN PREGNANCY TOXAEMIA IN BALADY GOATS
(CAPRINES CAPRINA) WITH TRAILS OF TREATMENT AS A
FIELD STUDY

By
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SUMMARY

This study aimed to investigate the effect of different degree of pregnancy
toxaemia on hormonal, blood picture and some biochemical changes together
with treatment of affected goats. This study was carried out on 29 Balady goats
aged 2-3 years during last 2-4 weeks of pregnancy (5 healthy and 24 goats
suffered from different degree of pregnancy toxaemia) in Sharkia Province.
Goats were divided into two main groups, First group contained 5 healthy goat
free from internal and external parasites served as control group, Second group
contained twenty four diseased goats were subdivided into three equal
subgroups(8 in each) according to the severity of the clinical signs of pregnancy
toxaemia. First subgroup goats suffered from slightly symptoms, second
subgroup of goats suffered from moderate symptoms, third subgroup goats
suffered from severe symptoms).

Treatment carried out in diseased group were received 100 ml dextrose
(25%) together with 100 ml of Ringer lactate solution in addition 50 ml calcium
preparation (Cal-De-Mag) injected intravenously followed by oral dosing of
propylene glycol daily for four days. Two blood samples were collected from
each animal by Jugular vein puncture before and after10 and 20 days of
treatment. The first sample was taken in heparinized tube for haematological
examinations and the second one was taken in centrifuge tube to obtain clear
serum for hormonal and biochemical analysis.

Urine analysis of pregnancy toxaemic goats revealed presence of different
concentrations of ketone bodies according to degree of pregnancy toxemic goats,
glucosuria detected only in moderate and sever cases but protein percentage only
in urine of sever cases of pregnancy toxemic goats.

The present investigation revealed that, goats suffering from pregnancy
toxaemia showed a significant decrease in total erythrocytic count, haemoglobin
concentration, packed cell volume%, insulin, triiodothyronin (T3) thyroxin
(T4),total protein, albumin, globulin, A/G ratio, glucose and cholesterol
associated with significant increase in leukocytic count, cortisol, transaminases
(AST-ALT) total lipid, triglycerides β-hydroxybutyric acid(β-HBA),Urea and
creatinine showed highly significant progressive elevation as pregnancy toxemia advanced beside non significant changes in serum alkaline phosphatase activity.

All previous parameters showed a significant improvement towards the values of control ones at 20 post treatment. Trail of treatment based on high level of injection dextrose, lactates, calcium, magnesium and vitamin B.

It could be concluded that the different degree of pregnancy toxemia induced several adverse effects on haemogram, hormonal, liver and kidney functions in goat which need about 20 days post treatment to return to the normal levels.

**INTRODUCTION**

Pregnancy toxemia is one of the major metabolic diseases affecting sheep and goat causing economical losses due to substantial mortality among the affected animals (Tawfik et al., 2005). The disease occurs mainly in the last third of pregnancy, with greater incidence in animals presenting two or more fetuses (Prieto, 1994). Pregnancy toxemia appears in the last 2-4 weeks of gestation periods.

Pregnancy toxemia caused by negative energy demand for the rapid fetal growth during the late gestation and insufficient energy intake (Abdul, 2003). Ovine and caprine pregnancy toxemia are diseases caused by impaired metabolism of carbohydrates and volatile fatty acids. Biochemically, it is characterized by ketonaemia, ketonurea, hypoglycaemia, low levels of hepatic glycogen (Blood et al., 1981) and fatty infiltration of the liver and kidneys (Henderson et al., 1982).

There are two factors involved in the development of hypoglycaemia. Firstly, the glucose requirement of the uterus may increase to more than 40% of the total liver glucose output, Secondly the endocrinological status changes in late pregnancy (Lindsay and Oddy, 1985). Adequate plane of nutrition in the second half of pregnancy and improving glycogenic fatty acid production in the rumen may help in suppression of the problem (Radosits et al., 2000).

Treatment pregnancy toxemia by two ways, the first one is to reduce the glucose requirements, either by caesarean section or by artificially inducing parturition using corticosteroids and the second one is to increase blood glucose (Wierda et al., 1985).

The present study focused on evaluation the effect of different degree of pergnancy toxemia on blood picture, hormonal, liver as well
as kidney functions in goat and with trial of treatment.

MATERIAL AND METHODS

1) Animals

The present investigation was carried out on Twenty nine goats during the last 2-4 weeks of pregnancy goats (24 clinically suffering from different degree of pregnancy toxemia and others 5 were clinically healthy) their age ranged from 2-3 years old. These goats were belonged to a private farm at Sharkia Province.

2) Experimental design

In this study, goats were divided into two groups. Each group were housed separately in open yard.

1) First group contain of five healthy goats free from internal and external parasites which served as control group.

2) Second group contained twenty four diseased goats were divided into three equal subgroups (8 in each) according to degree of the appeared symptoms.

A-First subgroup: goats which in-appetence, dullness and grinding of the teeth, without paying attention to the examiner. Goats isolated in a corner, slight nasal discharge (slightly affected).

B-Second subgroup: goats were appeared weakness, inability to stand, sternal recumbency, incoordination, anorexia and in some cases, muscular tremors and partial blindness (Moderately affected).

C-Third subgroup: Goats were clinically characterized by depression, labour breathing, lateral recumbency, head tilt, frothy salivation, and the odor of ketone which was detected in the breath. Some does showed complete blindness and comma (Severely affected).

3) Urine samples

Urine samples were analyzed using Comber-test strep (Boeheringer, Mannheim, Germany) for qualitative determination of ketone bodies, glucose and protein.

4) Blood samples

Two blood samples were collected from control goats as well as from diseased one before and after treatment by 10 and 20 days post treatment.

4.1) Haematological values

First sample was collected in heparenized tube for estimation erythrocytic count, haemoglobin content, packed cell volume and total leucocytic count according to (Jain, 1993).

4.2) Hormonal assay and biochemical studies

Second blood sample was collected in centrifuge tube to obtain clear serum for determination of
cortisol by radioimmunoassay method described by Abraham et al. (1972), insulin Burtis et al. (1994), triiodothyronine (T3) and thyroxin (T4) hormone Abraham (1981). Also, serum transaminases (AST-ALT) were determined according to method described by Reitman and Frankel (1957), alkaline phosphatase (John, 1982), total protein (Doumas, 1974), albumin (Drupt, 1974) globulin was calculated as difference between total protein and albumin and A/G ratio was determined, total lipid (Knight et al., 1972) triglycerides (Royer, 1969) cholesterol (Richmon, 1973), Beta-hydroxy buteric acid (Mercer et al., 1986), glucose (Trinder, 1969), urea (Coalome and Fauorean, 1963), creatinine (Husdan and Roporpot 1968).

5) Drugs:
1) Dextrose (25%): produced by El-Nasr for chemical pharmaceuticals company Egypt.
2) Ringer lactate solution: produced by El Nasr for chemical pharmaceuticals company Egypt.
3) Cal-De-Mag: produced by Pfizer for chemical pharmaceuticals company Egypt.
4) Propylene glycol: produced by Nile for chemical pharmaceuticals company Egypt.
5) Tri-B: produced by El Nasr for pharmaceuticals and chemicals industries Egypt.

6) Treatment trial:-
Goats in subgroups(1 and 2) which suffering from light and moderate pregnancy toxaemia were received 100 ml dextrose (25%) together with 100 ml of Ringer lactate solution in addition 50 ml calcium preparation (Cal.De.Mag.) injected intravenously followed by oral dosing of propylene glycol daily for four days but goats in subgroup (3) which suffering from severe pregnancy toxaemia were received 150 ml dextrose (25%) together with 150 ml of Ringer lactate solution in addition 100 ml calcium preparation (Cal.De.Mag.) injected (I/V) and 2 ml of vitamin B-complex injected (I/M) followed by oral dosing of propylene glycol twice in first day then one time daily for four days.

7) Statistical analysis:-
The obtained data were statistically analysed according to Petrie and Watson (1999).

RESULTS AND DISCUSSION
Gestational toxemia is a metabolic disorder characterized by hypoglycaemia and hyperketonaemia as a result of the incapacity of the animal to maintain an adequate energy balance (Hay and Baird, 1991). The clinical picture comprise neurological manifestations and weakness (Prieto, 1994).
Clinical signs of pregnancy toxemia in examined goats were graded into three stages. The first stage was a light affection, the goats characterized by inappetence, dullness and grinding of the teeth, without paying attention to the examiner. Goats isolated in a corner. The second stage was a moderate affection characterized by weakness, inability to stand, sternal recumbency, incoordination, anorexia and in some cases, muscular tremors and partial blindness. The third stage was severe affection characterized by depression, labour breathing, lateral recumbency, head tilt, frothy salivation, and the odour of ketone was detected in the breath. Some does showed complete blindness and coma. These symptoms were recorded previously by Scott and Woodman (1993) in the same species. The severity of the clinical signs was proportionally correlated with the decreased glucose level and inequally with the increased β-HBA in the serum. This may indicate that the signs were initially produced by hypoglycaemia rather than ketonaemia. This conclusion is in agreement with those expected by Scott et al. (1995) who concluded that the impairment of glucose utilization is the real cause for the nervous signs.

Table (1) show the qualitative analysis of urine of pregnancy toxemic goats in this study revealed presence of different concentrations of ketone bodies in urine according to degree of disease, glucosuria detected only in moderate and severe cases but proteinuria was detected only in urine of severe pregnancy toxemic goats. The level of ketone bodies in the urine was comparable to the degree of illness. These results were rather similar to those obtained by El-Din, Ibtisam and El Sangary (2005) in ewes. The presence of ketone bodies in the urine may be attributed to increased fat hydrolysis by the maternal tissues that convert the resulting glycerol to glucose and oxidation of fatty acids for energy (Cleon, 1988).

Table (2) results in this study revealed significant decrease in total erythrocytic count, haemoglobin content and packed cell volume percent in goats suffering from light, moderate and severe cases of pregnancy toxemia associated with significant increase in leukocytic count (table, 2). These results are in agreement with those of Mohamed et al. (2004) and could be attributed to deficiency of energy, protein and iron that are required for erythropoietin production and haemoglobin synthesis (Benjamin, 1984). The leukocytosis were also observed during all stages of the disease. The above mentioned results were supported by the studies of El-Sebaie, (1995) and Abd El-Raof.
and Ghanem (2006). These results could be attributed to the release of glucocorticoids under the effect of pregnancy toxemia, which causes an increase in the movement of granulocytes from the bone marrow to the peripheral blood (Maianti et al., 1990).

Results in table (3) revealed that the significant elevation in cortisol level in goats suffered from pregnancy toxemia. The obtained results coincided with Adel and Sahar (2005) who stated that, the stress of under nutrition, especially at the late stage of pregnancy stimulates the secretion of corticotrophin releasing factor (CRF) from the hypothalamus which stimulates the pituitary to release adrenocorticotropic hormone (ACTH), The end product of the action of adrenocorticotropic hormone namely cortisol. Another explanation comes from Radostitis et al. (2000) who recorded that the increase in cortisol level due to hepatic failure to metabolize cortisol. The significant decrease in insulin hormone in the present study was comparable with the results obtained previously by El-Sayed and Siam (1994) in goat. Decrease in the insulin hormone in the present study may be as a reflex of decreased glucose level for facilitating the hepatic synthesis of glucose (Hart et al., 1978).

Statistical analysis of the obtained results in Table (3) revealed a significant reduction in triiodothyronine and thyroxine. Close similarity was seen between the obtained results and those finding of Abdoou and Shaheed Iman (2004) in goats.

In table (4), it has been found that the transaminases (AST-ALT) levels in the serum of goats suffering from pregnancy toxemia showed a gradual increase as the pregnancy toxemia advanced. Our results were reinforced with that of Radostitis et al. (2000) in sheep and goat. These results may be due to fatty infiltration of the liver as suggested by Reid et al. (1983). Recently, another explanation comes from Nassif et al. (2005) who recorded that the change in liver enzymes in pregnancy toxemic goat may be due to damage of some hepatic cells which resulted from hyperketonaemia and lipolysis.

Table (4) revealed a significant increase of serum urea and creatinine in all cases of pregnancy toxemia in goat. This finding was supported by the observation by Pugh et al. (2002) who noticed elevation in serum urea and creatinine of pregnancy toxamic sheep and goat. These results might be attributed to the reduced glomerular filtration as a result of extensive fatty infiltration of the kidneys (El-Sayed,
et al., 2002) or might be attributed to renal dysfunction as a result of extensive degenerative changes of the kidneys (Abd El-Rahman, 2003).

Table (5) show the proteinogram of the diseased goat revealed hypoproteinemia associated with hypoalbuminemia, hypoglobulinemia and insignificant decrease in A/G ratio especially in the severely affected goat. This may be due to anorexia of pregnancy toxemic goats. The obtained data are in accordance with those previously obtained by Kaneko et al. (1997) and Abd El-Raof and Ghanem (2006) in diseased goats and ewes, respectively, who concluded that malnutrition especially in the late stage of pregnancy led to inadequate provision of amino acid substrate for general protein production and/or due to the metabolic action of glucocorticoids on body protein Katzung (1984). Nasr, et al. (1997) stated that the decreased in proteinogram in does suffered from pregnancy toxemia attributed to the reduction of albumin synthesis in hepatic insufficiency and albuminuria (Kaneko, et al. 1997).

Table (6) demonstrated a significant increase in total lipid, triglycerides, β-hydroxybutyric acid and significant fall in cholesterol and glucose in diseased goats if compared with healthy ones. The obtained results was supported by the finding reported by Nassif, et al. (2005) in does. Significant elevation in total lipid, triglycerides and β-hydroxybutyric acid in affected goat could be attributed to the starvation and lipolysis of the adipose tissue and the release of long chain fatty acids which were converted by the liver into ketones (Abd El-Rahman and Hassan, 2002). Decreased cholesterol in the present study might be attributed to the liver insufficiency and disturbance of fat metabolism with fat infiltration in liver (El-Bealawy 2000). The recorded hypoglycemia in the present study may be due to the effect of the long period of starvation and the developing fetuses (Anderwes, 1997) also due to disturbance of hepatic gluconeogenesis (Smith and Sherman, 1994). Schlumbohm and Harmeyer (2004) recorded that the high ketone body concentrations suppress the endogenous production of glucose by approximately 30% and facilitate the development of pregnancy toxemia in pregnant ewes. Also, Rook (2000) attributed the hypoglycaemia in pregnancy toxaemia to imbalance between the demand for glucose and the rate at which it is supplied. Another explanation for hypoglycaemia recorded by Vernon et al. (1981) was attributed to the lower circulating levels of insulin, together with higher levels of growth hormone, progesterone and prolactin.
in late pregnancy that tend to encourage hypoglycaemia.

In this investigation, data obtained after treatment indicated that, slight and moderate diseased cases were treated successfully with intravenous injection of dextrose (25%) together with Ringer lactate solution in addition to (Cal-De-Mag) followed by oral dosing of propylene glycol daily for four days. Similar results were also reported after treatment of the diseased goat with dextrose solution and propylene glycol (Faris et al., 2005). Glucose and its precursor will quickly reduce ketogenesis (Berman and Roberts, 1967), but three goats from the third subgroup (sever pregnancy toxemic goat) were found dead at second and third day of treatment. The mortalities may be attributed to the development of severe acidosis and irreversible brain damage due to hyperketonemia and hypoglycaemia. This result is in harmony with Mohamed et al. (2004).

From the previously mentioned results, we could conclude that the pregnancy toxaemia is affected with adverse effects on erythrogram, hormonal and some urine and biochemical parameters which help in early diagnosis of pregnancy toxaemia in goat. Also to avoid the occurrence of pregnancy toxaemia in pregnant goats, animals must be given enough food to supply the digestible energy needed throughout the gestation period to avoid the occurrence of pregnancy toxaemia.
Table (1) Qualitative examination of the urine in late pregnant goats, clinically normal and those suffering from different degree of pregnancy toxaemia.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Healthy goats (n=5)</th>
<th>Diseased goats</th>
<th>Light pregnancy toxemia (n=8)</th>
<th>Moderate pregnancy toxemia (n=8)</th>
<th>Sever pregnancy toxemia (n=5)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
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<td>Pre treatment</td>
<td>Post treatment</td>
<td>Pre treatment</td>
</tr>
<tr>
<td>Ketone bodies</td>
<td>-ve</td>
<td>+</td>
<td>-ve</td>
<td>-ve</td>
<td>++</td>
</tr>
<tr>
<td>Glucose</td>
<td>-ve</td>
<td>+</td>
<td>-ve</td>
<td>-ve</td>
<td>++</td>
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<tr>
<td>protein</td>
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<td>-ve</td>
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</tbody>
</table>
Table (2): Haemogram and leukocyte in late pregnant goats, clinically normal and those suffering from different degree of pregnancy Toxaemia.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Healthy goats (n=5)</th>
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<td>Light pregnancy toxemia (n=8)</td>
<td>Moderate pregnancy toxemia (n=8)</td>
<td>Sever pregnancy toxemia (n=5)</td>
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<td></td>
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<td>Pre treatment</td>
<td>Post treatment</td>
<td>Pre treatment</td>
<td>Post treatment</td>
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<tr>
<td>RBCs (10^6/cm^3)</td>
<td>8.74 ± 0.69</td>
<td></td>
<td>6.15± 0.34**</td>
<td>6.53± 0.28*</td>
<td>7.39± 0.31</td>
<td>5.54± 0.31**</td>
</tr>
<tr>
<td>HB (gm/dl)</td>
<td>13.0± 0.64</td>
<td></td>
<td>10.32± 0.21**</td>
<td>11.2± 0.16*</td>
<td>12.7± 0.39</td>
<td>9.42± 0.52**</td>
</tr>
<tr>
<td>PVC (%)</td>
<td>37.8± 1.19</td>
<td></td>
<td>33.62± 1.32*</td>
<td>34.8± 0.94</td>
<td>35.7± 0.86</td>
<td>32.86± 1.32*</td>
</tr>
<tr>
<td>WBCs (10^3/cm^3)</td>
<td>10.05± 0.81</td>
<td></td>
<td>13.64± 0.62**</td>
<td>12.43± 0.52*</td>
<td>11.5± 0.61</td>
<td>13.98± 0.75**</td>
</tr>
</tbody>
</table>

* Significant at P< 0.05  ** Significant at P< 0.01  *** Significant at P< 0.001
### Table (3) Cortisol, insulin, T3 and T4 levels in late pregnant goat clinically normal and those suffering from different degree of pregnancy toxaemia.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healthy goats (n=5)</th>
<th>Light pregnancy toxemia (n=8)</th>
<th>Moderate pregnancy toxemia (n=8)</th>
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<td></td>
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<td>Pre treatment</td>
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<td></td>
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<td>10</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Cortisol (ng/dl)</td>
<td>13.62± 1.94</td>
<td>18.23± 0.61**</td>
<td>17.02± 0.48*</td>
<td>15.8± 1.03</td>
</tr>
<tr>
<td>Insulin (ng/dl)</td>
<td>8.21± 0.59</td>
<td>5.11± 0.46**</td>
<td>6.11± 0.64*</td>
<td>7.92± 0.92</td>
</tr>
<tr>
<td>T3 (ng/dl)</td>
<td>147.02± 8.53</td>
<td>114.85± 9.45*</td>
<td>119.43± 7.94*</td>
<td>129.83± 8.63</td>
</tr>
<tr>
<td>T4 (µg dl)</td>
<td>3.92± 0.12</td>
<td>2.74± 0.45*</td>
<td>2.92± 0.32*</td>
<td>3.1 8± 0.62</td>
</tr>
</tbody>
</table>

* Significant at P< 0.05  ** Significant at P< 0.01  *** Significant at P< 0.001
Table (4): Liver and kidney function in late pregnant goats, clinically normal and those suffering from different degree of pregnancy toxaemia.

<table>
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<th>parameter</th>
<th>Healthy goats (n=5)</th>
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<tr>
<td></td>
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<td>Light pregnancy toxemia (n=8)</td>
<td>Moderate pregnancy toxemia (n=8)</td>
<td>Sever pregnancy toxemia (n=5)</td>
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<td>Pre treatment</td>
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<td>20</td>
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<tr>
<td>AST (μ/l)</td>
<td>74.18± 2.63</td>
<td>85.10± 1.92**</td>
<td>80.23± 1.06*</td>
<td>76.23± 1.34</td>
<td>87.10± 1.46*</td>
<td>84.31± 1.75</td>
</tr>
<tr>
<td>ALT (μ/l)</td>
<td>41.39± 2.19</td>
<td>50.12± 1.05*</td>
<td>47.36± 1.03*</td>
<td>43.25± 1.63</td>
<td>52.31± 1.63*</td>
<td>49.32± 1.51*</td>
</tr>
<tr>
<td>Alkaline Phosphatase (μ/l)</td>
<td>65.12± 1.42</td>
<td>63.24± 1.32</td>
<td>64.15± 1.76</td>
<td>64.93± 1.76</td>
<td>62.32± 1.39</td>
<td>63.41± 2.45</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>24.74± 0.48</td>
<td>29.75± 1.64**</td>
<td>27.92± 1.06</td>
<td>25.98± 0.75</td>
<td>30.95± 1.47**</td>
<td>28.96± 1.59*</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.90± 0.07</td>
<td>2.41± 0.20*</td>
<td>2.12± 0.12</td>
<td>1.96± 0.08</td>
<td>2.54± 0.19**</td>
<td>2.37± 0.24</td>
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</tbody>
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* Significant at P< 0.05  ** Significant at P< 0.01  *** Significant at P< 0.001
Table (5): Serum proteinogram in late pregnant goats, clinically normal and those suffering from different degree of pregnancy toxaemia.

<table>
<thead>
<tr>
<th>parameter</th>
<th>Healthy goats (n=5)</th>
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<tbody>
<tr>
<td></td>
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<td>Light pregnancy toxemia (n=8)</td>
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<tr>
<td></td>
<td>Pre treatment</td>
<td>Post treatment</td>
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<tr>
<td>T. protein (gm%)</td>
<td>9.16±</td>
<td>5.24±</td>
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<td></td>
<td>0.92</td>
<td>0.32**</td>
</tr>
<tr>
<td>Albumin (gm%)</td>
<td>4.05±</td>
<td>2.12±</td>
</tr>
<tr>
<td></td>
<td>0.47</td>
<td>0.28**</td>
</tr>
<tr>
<td>Globulin (gm%)</td>
<td>5.11±</td>
<td>3.12±</td>
</tr>
<tr>
<td></td>
<td>0.82</td>
<td>0.15*</td>
</tr>
<tr>
<td>A/G ratio</td>
<td>0.79±</td>
<td>0.68±</td>
</tr>
<tr>
<td></td>
<td>0.17</td>
<td>0.09</td>
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</table>

* Significant at P< 0.05  ** Significant at P< 0.01
Table (6) Lipogram and glucose in late pregnant goats, clinically normal and those suffering from different degree of pregnancy toxemia.

<table>
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<tr>
<th>Parameter</th>
<th>Healthy goats (n=5)</th>
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</tr>
<tr>
<td>T lipids (mg/dl)</td>
<td>390.62±6.37</td>
<td>412.72±4.63*</td>
<td>405.4±3.74</td>
<td>384.72±6.83</td>
<td>426.41±7.62**</td>
<td>412.8±4.82*</td>
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<td></td>
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<td>473.30±4.36</td>
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<td></td>
<td>500.79±4.87*</td>
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</tr>
<tr>
<td>Triglycerides  (mg/dl)</td>
<td>476.87±6.49</td>
<td>496.76±4.73*</td>
<td>483.7±7.63</td>
<td>479.89±5.09</td>
<td>499.91±6.93*</td>
<td>488.79±7.84</td>
</tr>
<tr>
<td></td>
<td></td>
<td>511.80±7.25</td>
<td></td>
<td></td>
<td>527.80±7.25</td>
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</tr>
<tr>
<td>β-HBA (mg/dl)</td>
<td>10.62±0.71</td>
<td>13.204±0.84*</td>
<td>12.31±0.93</td>
<td>11.83±0.73</td>
<td>13.29±0.85*</td>
<td>13.15±1.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15.42±0.95**</td>
<td></td>
<td></td>
<td>13.82±0.89*</td>
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</tr>
<tr>
<td>Cholesterol    (mg/dl)</td>
<td>60.75±1.95</td>
<td>49.06±1.82**</td>
<td>55.82±0.95*</td>
<td>57.93±1.04</td>
<td>46.72±2.75**</td>
<td>52.73±1.93*</td>
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<tr>
<td></td>
<td></td>
<td>52.98±2.65**</td>
<td></td>
<td></td>
<td>56.84±1.04**</td>
<td></td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>65.83±1.92</td>
<td>53.62±2.42**</td>
<td>58.93±0.97*</td>
<td>63.83±0.89</td>
<td>52.98±2.65**</td>
<td>56.84±1.04**</td>
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<tr>
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<td>50.93±1.97***</td>
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<td></td>
<td>59.07±1.30*</td>
<td></td>
</tr>
</tbody>
</table>

* Significant at P< 0.05  ** Significant at P< 0.01  *** Significant at P< 0.001
REFERENCES


