



## Successful Medical Management of Pregnancy Toxemia in Goats

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### ABSTRACT

Pregnancy toxemia is a metabolic disorder that occurs in does and ewes during the late stage of pregnancy. Pregnant does that have low energy levels and having multiple numbers of fetuses are more susceptible to toxemia. The present study was carried out in twenty five goats in advanced stage of pregnancy with the history of anorexia, torticollis, grinding of teeth, salivation and rigors. On clinical examination of animals, they were dull, depressed with tachycardia, tachypnea, opisthotonus and pale conjunctival mucous membrane. The biochemical parameters revealed hypoglycemia and hypocalcemia. Urine samples were collected and urine analysis revealed positive for ketone bodies. The goats were successfully treated with 25% dextrose i/v as a bolus, multiple electrolytes solution containing 5% dextrose i/v, glycerin orally and Vitamin B-complex injection intramuscularly and all the twenty five animals survived. Out of 25 animals medical termination of pregnancy was done in 21 cases and four animals delivered a live kid.

**Keywords:** Goats, Pregnancy Toxemia, BHBA (Beta hydroxy butyric acid), Ultrasonography, Treatment

Pregnancy toxemia is a metabolic disease also called pregnancy ketosis, twin lamb disease, lambing or kidding sickness and ovine ketosis (Sharma *et al.*, 2014). Ketosis in goat is called as Pregnancy toxemia and in cow is called as acetonemia (Radostits *et al.*, 2009). Ketosis is a condition characterized by abnormally elevated concentration of ketone bodies in the body tissues and fluids when fatty acid are incompletely metabolized, occurs in cows and ewes during times of increased bodily mobilization of fat stores, usually just after they have given birth or in late gestation (Smith, 2009). Pregnancy toxemia usually occurring in ewes and does during the last 2-4 weeks of gestation. It is seen in females carrying multiple fetuses and may result from their inability to consume adequate energy to match metabolic demands. It is characterized by anorexia, weakness and depression (Smith, 2009). Predisposing factors such as poor management and hygiene, teat injuries and faulty milking machines are known to hasten the entry

of infectious agents and the course of the disease (Majic *et al.*, 1993).

### MATERIALS AND METHODS

Twenty five non-descriptive breed pregnant does of 2-4 years old, 30-35 kg body weight and 130-140 days of gestation were presented to Madras Veterinary College Teaching Hospital, Chennai-7, showing clinical signs of dullness, drowsiness, anorexia, acetone odour from the mouth, grinding of teeth, dyspnea and nervous signs such as star gazing posture, opisthotonus, torticollis and incoordination.

Blood glucose level was estimated by one touch select glucometer and blood BHBA (Beta hydroxy butyric acid)

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level was estimated by ketone meter. Urinalysis reagent strips were used to detect the presence of ketone bodies, glucose and pH level in urine. Hold the strip in a horizontal position, dip it into each urine sample individually, remove it after one minute and compare it to chart colors to record the results.

Radiography examination of the abdominal lateral view was done. B-mode ultrasound was used for examination of the liver by using a 3.5 MHz curvilinear transducer applied on the 7-12 intercostal space on right side of the animal body.

Blood sample was collected in EDTA vial for complete blood count and without anticoagulant vial for estimation of biochemical parameters. Complete blood count (CBC) was assessed with an automatic cell counter (Mindray BC Vet 2800). Parameters assessed were: red blood cell count (RBC), hemoglobin (Hb), PCV, PLT count, white blood cell count (WBC), WBC differential count including neutrophils, lymphocytes, monocytes. The serum concentration of alanine amino transferase (ALT), alkaline phosphatase (ALP), total protein, calcium, albumin, cholesterol, GGT, blood urea nitrogen (BUN) and creatinine, were determined by automated serum biochemistry analyser (A-15 Biosystem) by using standard kits.

Common treatment for ewes affected with pregnancy toxemia include the oral administration of glycerol or propylene glycol solution (2 oz. twice a day), intravenous glucose and more than 135 days of gestation, injection of dexamethasone or beta methasone to induce parturition (Radostits *et al.*, 2009), intravenous 100-300 ml dextrose (25%) bolus IV, B vitamins, remove fetuses (induce labor or c-section) with the aim of eliminating the metabolic demand for energy, of the gravid uterus.

## RESULTS AND DISCUSSION

All affected does had showed the clinical signs (Fig. 1) of dull, depressed, anorexia, acetone odour from the mouth, opisthotonos, torticollis, grinding of teeth, frothy salivation, tremors, incoordination, star gazing posture (Fig. 2). The vital signs like rectal temperature and respiration rate were normal range but the heart rate was increased than normal healthy goats (Table 1).

The clinical signs of dull, depression and anorexia in the present study might be due to nervous abnormalities in pregnancy toxemia does (Abdelaal *et al.*, 2013, Reddy *et al.*, 2014), acetone odour from the mouth might be due to increase in ketone bodies (Abdelaal *et al.*, 2013). Opisthotonus due to the hypoglycemia, ketonemia and neurodegeneration, torticollis (Reddy *et al.*, 2014).

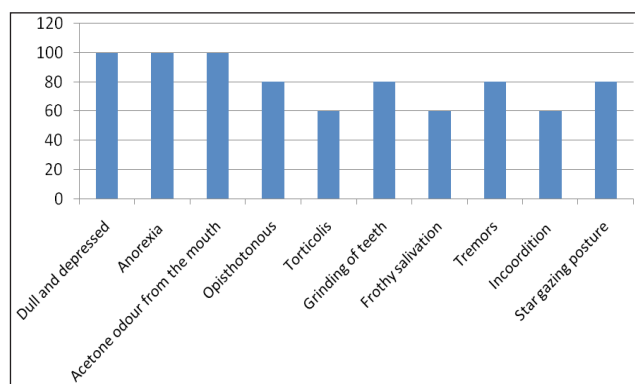


Fig. 1: Clinical signs in pregnancy toxemic goats

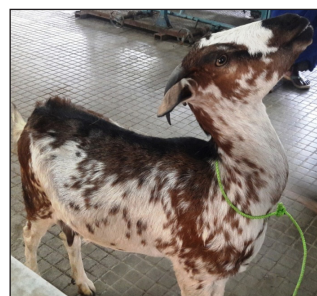


Fig. 2: Pregnancy toxemia - Star gazing posture in a doe



Fig. 3: One touch glucometer showing hypoglycemia

Table 1: Mean ± standard error values of vital signs of pregnancy toxemia (PT) affected goats

Parameters (Unit)	PT Affected goats (Mean±SE)
Rectal temperature (°F)	101.52 ± 0.511
Respiration rate (per minute)	26.8 ± 0.841
Heart rate (per minute)	121 ± 10.001

Grinding of teeth may be due to cerebral hypoglycemia (Balikci *et al.*, 2009 Abdelaal *et al.*, 2013). Frothy salivation, tremors, incoordination, star gazing posture might be due to the nervous degeneration (Barakat *et al.*, 2007). The vital parameters temperature and respiration rate are

normal in range but increased heart rate was similar with the study of Prasannkumar *et al.* (2016).

The mean values of haematological parameters showed increased level of total leucocyte count, neutrophil and decreased level of lymphocyte but Hb, PCV, RBC and platelets values are within the normal range (Table 2). The pregnancy toxemia does had hypoglycemia (Fig. 3), hypocalcemia, hypoproteinemia, hypoalbuminaemia and decreased level of cholesterol level and increased level of Beta hydroxy butyric acid (BHBA) (Fig. 4). There was increase in the mean value of AST, ALP, GGT, BUN, creatinine and triglyceride level (Table 2). Urine analysis, showed positive for ketone bodies (Fig. 5) in the urine (++++).

**Table 2:** Haematological and biochemical parameters of pregnancy toxemia affected goats (Mean±SE).

Parameters	Mean ± SE
Hb(g/dl)	8.7±0.044
RBC(m/cmm)	12.95±0.089
PCV (%)	24.02±0.123
Total leucocyte count (WBC)/cmm	23391.3±205.408
Platelets (/cmm)	306160±1351.791
Lymphocyte (%)	48.6±0.464
Neutrophil (%)	49.12±0.281
Monocyte (%)	4.304 ± 0.073
Glucose (mg/dl)	34.78±0.228
Calcium (mg/dl)	8.71±0.0254
BHBA (mmol/dl)	5.91±0.05
AST (IU/L)	232.4±1.603
ALP(IU/L)	296±32.577
GGT(IU/L)	54.8±4.893
Total protein (g/dl)	4.83±0.056
Albumin (g/dl)	2.24±0.026
Cholesterol (mg/dl)	56.18±2.418
BUN (mg/dl)	26.71±0.976
Creatinine (mg/dl)	2.02±0.141
Triglyceride (mg/dl)	115.48±2.65

Increased TLC in the present study might be due metabolic acidosis (ketoacidosis), infections, localized inflammatory process and tissue necrosis of liver (Gupta *et al.*, 2007; Tharwat and Fahd Al Sobayil, 2014 and Abba *et al.*, 2015). Neutrophilia could be due to hepatic lipidosis in which exposure of hepatocytes to fatty acids elicits

inflammation, increase of oxidative stress, apoptosis and production of fibrogenic cytokines (Smith and Sherman, 2009). Lymphopenia in the present study may be due to toxic increase of BHBA and acetoacetate which leads to inhibition of caprine lymphocytic proliferation and reduced caprine T-lymphocyte blastogenesis (Smith and Sheramn, 2009 and Hefnawy *et al.*, 2011 and Abba *et al.*, 2015).



**Fig. 4:** A Ketometer showing hyper -ketonemia



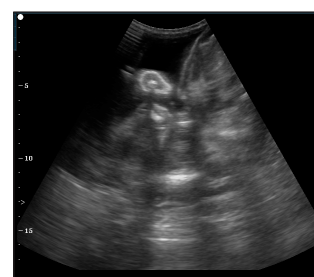
**Fig. 5:** Urine samples showed positive for ketone bodies



**Fig. 6:** Radiography examination showed multiple fetuses



**Fig. 7:** Ultrasonography - Hyper-echogenicity of liver parenchyma



**Fig. 8:** Ultrasonography - fetal skeleton



**Fig. 9:** After treatment affected does delivered a two live kids

Increased blood BHBA level in pregnancy toxemia does which could be attributed to the disturbance in the carbohydrate and fat metabolism leading to increase the lipolysis of tissues fat and release the long chain fatty acids which were converted by the hepatocytes to ketone bodies hepatic ketogenesis (Rook, 2000; Hefnawy *et al.*,



2010). Increased triglyceride level may be due to lipolysis of tissue and release of long chain fatty acids that can be stored as triglyceride in the liver it might be converted to ketone bodies (Barakat *et al.*, 2007; Abba *et al.*, 2015).

Hypoglycemia in the present study might be due to increased BHBA there was net energy balance (NEB), which causes hypoglycemia (Reddy *et al.*, 2014; Rani *et al.*, 2015). Hypocalcaemia might be due to high need of Ca for fetal skeleton development and when doe carry twin or triplet in last trimester, there was a greater risk of the development of pregnancy toxemia (Anoushepour *et al.*, 2014; Rodolfo *et al.*, 2014).

Decreased serum total protein in the present study might be due to the impairment of liver function (Ulvund, 1990). Low serum cholesterol level could be caused by a fat infiltration in the liver and a low output of lipoprotein (Sevinc *et al.*, 2003).

Increased ALT which could be attributed to the damage in hepatic cells, increasing its membrane permeability, leading to release the cellular enzymes to circulation, this damage resulted from increase the metabolism of the body fats due to energy deficiency which in turn increases the circulating free fatty acids that reach the liver and subsequently induce fatty infiltration (Kaneko *et al.*, 1997).

Increased ALP values in the present study may be due to inflammation of the hepatic epithelial cells surrounding the bile ducts or may be from renal origin. Increased serum GGT which was attributed to damage in liver parenchyma (Sargison *et al.*, 1994).

Increased BUN and creatinine attributed to severe kidney dysfunction accompanied with acidosis which is the result of increased ketone body in general circulation, fatty infiltration in tubular epithelium of kidney leads to elevation of both parameters (Lima *et al.*, 2012; Rodolfo *et al.*, 2014).

Urine analysis showed aciduria and ketonuria in the present study might be due to increase the glucose demand than the dietary supply in pregnancy leading to increase the mobilization of long chain fatty acids from adipose tissues a marked rise in circulating non esterified fatty acid and ketone bodies, which in return, descend in urine (Gonzalez *et al.*, 2011; Albay *et al.*, 2014).

The radiography examination showed multiple fetuses in examination of lateral view of abdomen (Fig. 6). The ultrasonography examination showed more echogenicity of liver parenchyma than the normal healthy animal liver parenchyma (Fig. 7), visible of fetal skeleton (Fig. 8).

Ultrasonography of liver in pregnancy toxemia cases appeared more echogenicity than the normal liver parenchyma. The hyperechoic features may be attributed to changes in the nature of the liver tissues that increases the attenuation of the ultrasound beam (Cebra *et al.*, 1997; Szebeni *et al.*, 2006).

The goats were successfully treated with 25% dextrose i/v as a bolus, multiple electrolytes solution containing 5% dextrose i/v, glycerin @ 60 g orally and Vitamin B-complex injection @ 3 ml intramuscularly and all the twenty five animals survived. Out of 25 animals medical termination of pregnancy was done in 21 cases and four animal delivered a live kid (Fig. 9).

Olfati *et al.* (2013) reported the use of oral administration of glycerol or propylene glycol solutions (2 oz. twice a day), intravenous glucose, intravenous 5% dextrose drip, dextrose 50% bolus intravenously and Vitamins B complex injection in pregnancy toxemia ewes. Ermilio and Smith (2011) reported that the main therapeutic plan in pregnancy toxemia is to restore the glucose metabolism and dehydration status, which were the main focus. In the present case, the doe was promptly administered dextrose and fluid therapy to replace the glucose and electrolyte loss, as well as to rehydrate and at the same time to flush out the ketone bodies.

## CONCLUSION

The present study was carried out in twenty five goats in advanced stage of pregnancy with the history of anorexia, torticollis, grinding of teeth, salivation, rigors and star gazing posture. The biochemical parameters revealed hypoglycemia and hypocalcemia. Blood glucose estimation by one touch glucometer and Blood BHBA estimation by ketone meter were found to be useful in the diagnosis of pregnancy toxemia in goats. The goats were successfully treated with 25% dextrose i/v as a bolus, multiple electrolytes solution, glycerin orally and Vitamin B-complex injection.

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