Pregnancy Toxaemia in Goat: a Case Report

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ABSTRACT

A four years old Khari goat was brought to Madi Veterinary Hospital with the history of parturition 3 days ago with triplet’s kid, in-appetence and weakness. Vitals signs were within normal physiological range. Laboratory diagnosis revealed low glucose level (27 mg/dl) and was diagnosed as pregnancy toxemia on the basis of clinical and laboratory findings. Calcium-borogluconate, corticosteroid, B-complex and jiggery were given as glucose replacement, glucose precursor, metabolic supporter and source of glucose respectively. Goat became responsive with after two days of treatment.

**Keywords:** Corticosteroids, Glucose replacement, Khari

INTRODUCTION

Pregnancy toxemia (PT) is also known as twin lamb disease, a metabolic disorder of pregnant small ruminants caused by an abnormal metabolism of carbohydrates and fats, which occurs at the final stage or few days after pregnancy (Brozos, C et.al, 2011). The occurrence of this disorder may be either due to poor nutrition or excessive demand from multiple rapidly growing fetuses but sometimes both contributed together (Lima et al., 2012). Nearly 80% of fetal growth occurs during the last six weeks of gestation (Bergman E.N., 1993, Navarre and Pugh, 2002). The normal blood glucose and ketone bodies range in does is 40-60 mg/dl and 1-4 mg/dl respectively. There is significant decrease in the blood glucose level and increase in the level of ketone bodies in PT in goat. During the final month of gestation, the energy requirement of a pregnant doe carrying twins or triplets is 180% or 240% greater, respectively, than that of a doe with a single fetus and obese ewes and does are at greater risk for developing PT (Navarre and Pugh, 2002). Obese ewes or does carrying multiple fetuses are at higher risk to develop the disease because of the limited space for adequate intake of feed (Ermilio EM, Smith MC. 2011). It is characterized by hypoglycemia and hyperketonemia. Rapid fetal development at the late gestation periods causes rapid mobilization of the fat stores to assure adequate energy. The liver also increases gluconeogenesis to facilitate glucose availability to the
fetus. However, in a negative energy balance, this increased mobilization may overwhelm the capacity of liver resulting in hepatic lipidosis. At the same time, ketone bodies are being produced and accumulated, which eventually leads to excessive ketone bodies in blood circulation, thus increasing the susceptibility to pregnancy toxemia (Menzies, 2011). In severe outbreaks, morbidity can reach up to 20% with a mortality rate of 80% of affected animals (Ismail et al., 2008). Early clinical signs of PT in ewes and does include in-appetence, listlessness, aimless walking, muscle twitching, fine muscle tremor, opisthotonos, and grinding of teeth. As the condition progresses, blindness, ataxia and finally recumbence, coma and death will occur in animals that are not diagnosed and treated with the condition. This study outlines the clinical approach in management of PT in a doe. This clinical case management highlighted that institution of correct treatment at the right time was able to give better prognosis and success.

CASE HISTORY

History of patient
A four-year-old Khari cross female goat weighing 30 kg is brought in with the main complaints of weakness and lack of appetite. She had the history of parturition 3 days ago having three kids. The state of the deworming and vaccinations were out of date.

Physical examination, clinical and laboratory findings
The doe was unable to stand properly on its legs with the body condition score (BCS) 2 out of 5 according to Ghosh (2019). Physical examination shows normal vitals with decreasing ruminal motility, lethargic, weak, off feed. Animal had pale mucous membrane and increased capillary refill time. Blood glucose level was found 27 mg/dl. Based on the history, physical examination, clinical examination, and laboratory findings, pregnancy toxemia was determined in this instance (Ketosis).

TREATMENT

20 ml of calcium-borogluconate injection (THICAL, Vetoquinol containing 20% glucose along with calcium, magnesium) was given S/C once daily for two days as glucose replacement. 40mg of triamcinolone as corticosteroids (TRICORT 40, 1 ml ampule, CADILA pharmaceuticals) was given IM once daily for two days for the initiation of gluconeogenesis. 10 ml of vitamin B-complex injection (Conciplex, Concept Pharmaceuticals) was given IM once daily for two days to support metabolic process of the goat. 100gm of jiggery was provided orally in drinking water for 3 days as the chief source of glucose and energy. The main objective of treatments in pregnancy toxemia is to increase the formation of glucose for utilization at tissue level, and increase the usage of ketone bodies to prevent acidosis and electrolytes disorder. However, treatments with drugs are only effective in an early disease diagnosis.
PROGNOSIS

The doe was bright and alert and responsive with increase in appetite after 2 days of treatment. Recovery rate was higher with better performance after the treatment.

RESULTS AND DISCUSSION

Jones and Navarre (2014) state that PT is typically detected in malnourished animals, however in the current case, the affected goat was underweight and carrying multiple fetuses, which placed her at higher risk due to her limited rumen area for a sufficient intake of feed and low blood glucose level. Tinkler (2014) asserts that clinical indicators including anorexia, depression, and detachment from the herd are the first step in the diagnosis of PT in a pregnant doe, which is the primary concern in the current case. As the disease progressed the animal developed neurological signs such as depression, tremors, star-grazing, ataxia, circling, teeth grinding, and blindness as a result of hypoglycemia-induced encephalopathy. (Andrews 1997). In the final stage without proper diagnosis and treatment, affected animal will be in recumbency and coma where the prognosis at this stage is very poor with low survival rate (Tinkler 2014). Biochemically, significant reduction in the level of glucose and calcium, and an increased level of Serum Glutamic Pyruvic Transaminase (SGPT), Serum Glutamic Oxaloacetic Transaminase (SGOT), Blood Urea Nitrogen (BUN), creatinine, β-hydroxyl Butyric Acid (BHBA), and Non-Esterified Fatty Acid (NEFA) in the pregnancy toxemic goats had been observed by Vasava, P. R. et.al., 2016 in Gujarat, India. In this case blood glucose was found level were found to be decreased. Laboratory diagnosis of serum BHBA and urine ketone level can be performed to further diagnose pregnancy toxemia (Ramin et.al., 2007).

According to Ermilio and Smith (2011), based on their observations from a study conducted on sheep and goats, the primary therapeutic strategy for pregnant toxemia is to restore glucose level with re-establish metabolism and dehydration condition. In this instance, calcium borogluconate, 20 ml S/C, was administered to the doe to make up for the low glucose level. In addition to borogluconate, oral glucose replacement with 100gm of jiggery was given once daily as source of glucose for 3 days. Corticosteroids and/or ACTH have been used as treatments to enhance gluconeogenesis and raise blood glucose levels (LW, H. 1958). Dexamethasone, a glucocorticoid, has been used to successfully treat hyperglycemia in field situations. (Yadhav et al., 2018). In the present case, the doe was parturited three days prior, therefore we utilized the corticosteroid triamcinolone to boost gluconeogenesis and subsequently the blood glucose level. There are a few crucial things to keep in mind when preventing and treating pregnancy toxemia. For example, during the latter few months of gestation, high-energy supplement feed like 0.5–1 kg of cereal grain like corn, oats, or barley should be provided (Edmondson et.al., 2012). Prior to the two months of parturition, the does with BCS less than 2 must be identified.
and fed with high energy concentrates and essential minerals that contain calcium and magnesium. Animals require a high performance deworming product as well as potable water. Additionally, vitamin B12 may be administrated as an appetite stimulant. It accelerates the goat’s body metabolic rate, increases the absorbable glucose concentration in the body.

CONCLUSION

Glucose is the animal’s major energy source, and the demand for glucose in the body increases several times during pregnancy. PT results from alternating the glucose hemostatic mechanisms in response to the increased nutritional demand of the developing fetus and pregnant mother. Early diagnosis and treatment are crucial to save the life of the dam and fetus. Treatment mostly includes the administration of the rich energy glucose source to the affected animals. It is suggested to take a sample of late gestation doe to test for serum BHB or glucose concentration to determine the extent of the risk in the rest of the flock.

REFERENCES

12. Lima MS, Pascoal RA, Stilwell GT and Hjerpe CA, 2012. Clinical findings, blood chemistry values
and epidemiologic data from dairy goats with pregnancy toxemia. Bov Practitioner, 46: 102-110