

Research Article

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Clinical and Therapeutic management of Pregnancy toxemia in a Ewe

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Keywords

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Abstract

In the present study, Nellore Jodipi ewe was presented with the history of recent lambing, anorexia, weakness of hind limbs, unable to stand and recumbency. Clinical examination of serum samples revealed reduction in glucose (36 mg/dL), reduction in calcium levels (7.5 mg/dL), presence of the ketone bodies in the urine and elevated blood β -hydroxybutyrate (>1.4 mmol/l). Based on the clinical signs, serum sample and urine sample examination the case was diagnosed as pregnancy toxemia and therapy was initiated with 50% dextrose and Ringers lactate intravenously along with symptomatic therapy.

Introduction

Metabolic diseases occupy a substantial part of ruminant pathology. Pregnancy toxemia in ruminants is a nutritional stress syndrome affecting mainly adult and multiparous animals in good body condition during the last 3 to 6 weeks of gestation (Schlumbohm and Harmeyer, 2008). The disease is also known as ovine ketosis, twin-lamb disease, ewe sleepy sickness, lambing sickness or pregnancy paralysis and it is characterized by hypoglycemia and hyperketonemia resulting from incapacity of the animal to maintain adequate energy balance. Negative energy balance during late pregnancy in ewes is an important cause of pregnancy toxemia. Symptoms include depression, anorexia, weakness, staggering gait, apparent blindness, recumbency, coma, and death (Al-Qudah, 2011). Risk factors include multiple fetuses, ingestion of poor quality diet, decreased dietary energy level, genetic factors, obesity, lack of good body condition or high parasite load and lack of exercise (Rook, 2000). Olfati and Moghaddam

(2013) reported that crossbred ewes carrying multiple pregnancies are much more likely to experience ketosis. Further, it can also be observed in poorly nourished sheep with only a large single fetus (Bani Isail et al., 2008). The present study describes about the pregnancy toxemia and its successful therapeutic management in a Nellore Jodipi Ewe.

Materials and Methods

Blood samples were collected through puncture of the jugular vein using sterile 21G needles into anticoagulated vacutainers and sample was obtained in the morning before feeding. Blood β -hydroxybutyric acid (BHBA) concentrations were determined. Samples for biochemical analysis were transported and stored at 40° . Analysis was conducted within 2 hours after sampling. The following indices were determined: glucose and calcium. The biochemical tests were

performed using colorimetric method with an semi - automated biochemical analyser.

Case History and Observations

A Nellore Jodpi ewe of third parity was presented to the Veterinary dispensary, Chakrayapeta, Kadapa with the history of recent lambing, anorexia, weakness of hind limbs and recumbency. Clinical examination revealed heart rate (94/min), respiratory rate (30/min),

gasping (Fig. 1) and unable to stand (Fig. 2). Haematology did not reveal any significant abnormalities except elevated blood - hydroxybutyrate (>1.4 mmol/l). Serum biochemical examination revealed hypoglycemia (36 mg/dL), reduction in calcium levels (8.8 mg/dL). Urine examination revealed presence of ketone bodies. Based on the history, clinical and biochemical findings, the present case was diagnosed as pregnancy toxemia.



Fig. 1 Affected animal showing gasping



Fig. 2 affected animal was unable to stand.

Treatment and Discussion

The ewe was treated with 250 ml of 50% dextrose intravenously and Ringers lactate at the rate of 10 ml/kg b. wt intravenously. Calcium injection (20 ml of calcium Sandoz), Inj. Neurovet (3 ml) were also given. The owner was advised to repeat the treatment for two more days along with oral supplementation. By the next day the ewe was able to stand and started taking feed.

The clinical signs observed in the present study were in accordance with the Al- Qudah, (2011) who stated that symptoms of ketonemia in ewes includes depression, anorexia, weakness, staggering gait, apparent blindness, recumbency. Hypocalcemia and ketone bodies in urine was observed in the present study and a similar finding was also reported by Brozos et al., (2011). Therapy was initiated with intravenous administration of 50% dextrose and it was in accordance with the Radostits et al., (2007).

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