

Uterine parasites: A clinician's guide to pregnancy toxemia

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Abstract

Pregnancy toxemia in late gestation small ruminants is a common condition presented to large animal practitioners. The goal of this talk will be to review the current pathophysiology and therapy plans in varying circumstances in an ambulatory setting. Identifying risk factors and negative prognostic considerations needed to implement valuable therapeutic and preventative plans is critical. Following this presentation, the practitioner will be able to diagnosis and implement supportive therapy for pregnancy toxemia, identify risk factors and negative prognostic indicators, and design preventive plans for a small ruminant herd.

Key words: pregnancy toxemia, ketosis, small ruminant, pregnancy

Introduction

Periparturient metabolic disease in small ruminants is due to a failure to meet nutritional requirements during late gestation resulting in a negative energy balance. These conditions include pregnancy toxemia, or late gestation ketosis, which can result in significant morbidity and mortality. Although, large animal practitioners are presented with pregnancy toxemia as individual animal emergencies, this is ultimately a herd condition. Thus, special focus should be taken to identify and remedy risk factors within the population to limit future cases.

Risk factors for pregnancy toxemia

When identifying risk factors associated with pregnancy toxemia, overall herd management needs to be assessed to determine how nutritional needs are not being met. What is the stocking density and resultant feeder space? What is the forage/hay quality being fed during late gestation? How much grain, if any, is being provided? External factors out of our control including recent history of inclement weather limiting access to appropriate nutrition should be considered. When evaluating a small ruminant herd's risk for pregnancy toxemia, many animal characteristics should be taken into consideration. Body condition is highly associated with pregnancy toxemia risk, where lean animals (BCS \leq 2/5) or obese (BCS \geq 4/5) are more likely to develop ketosis. Dams carrying 2 or more fetuses are more likely to develop a negative energy balance in late-stage gestation as fetal growth greatly increases just prior to parturition. Other co-morbidities also put dams at increased risk such as poor dentition, lameness, or heavy parasite burden thus limited their ability to either access feed or decreased utilization of energy from feed source provided.

Clinical presentation and diagnosis

When presented with an ill, pregnant small ruminant, both pregnancy toxemia and hypocalcemia should be considered and ruled out using diagnostics. For pregnancy toxemia, clinical signs on examination are resultant of hypoglycemia and associated hypoglycemic encephalopathy. Most signs will be non-specific, including anorexia, depression and recumbency and can progress to neurologic signs including blindness and coma. Diagnostics include blood glucose and ketone body concentration which can be done in the field using hand held devices. Hypoglycemia of < 48 mg/dL and hyperketonemia, specifically beta hydroxybutyrate, of > 0.8 mmol/L is consistent with pregnancy toxemia diagnosis. Hyperketonuria can also found.

Treatment and prognosis

Two goals of treatment include: administering energy and removing factors that are depleting the dam of energy. If an animal is recumbent, IV dextrose should be administered initially then energy supplied in the form of oral propylene glycol thereafter. If the producer is capable additional doses of IV dextrose can be provided at 160 mL, 3-4x daily for 6 days. This can be paired with insulin administration at 20-40 IU daily until recovery. This inclusion of insulin is costly and requires monitoring blood glucose concentration. Research has found that animals suffering from pregnancy toxemia have impaired pancreatic response and peripheral insulin resistance associated with late gestation. Yet, this additional treatment can be costly.

Oral propylene glycol at 150-200 mL for 2 treatments, then continued daily at 60 mL per day for 6 days. Providing additional energy in the form of an energy dense ration, including corn-based grain is also critical. Other supportive care includes vitamin B, NSAID, broad-spectrum antimicrobials, and anthelmintic administration may be warranted depending on co-morbidities observed on physical examination.

Induction of parturition may be considered to eliminate the dams need of additional energy requirements. If the breeding date is known, induction of parturition can be performed in ewes > 140 days and does > 143 days of gestation. Fetometry can be performed to estimate gestational age if breeding and expected birthing date is unknown. However, due to the severity of clinical signs, the fetuses may already be compromised resulting in a high risk of abortion and still births. Furthermore, these patients are at high risk of both dystocia and retained placenta due to the dam's systemic compromise and weakness.

Several prognostic indicators have been described in published literature. Although practitioners rely on blood BHB concentrations for diagnosis of pregnancy toxemia, there has been no correlation found between BHB concentrations and outcome of disease. Acidemia, low blood pH and hyperkalemia are associated with negative clinical outcomes at the time of diagnosis.

Furthermore, clinical disease severity in the form of anorexia, ruminal hypomotility, neurologic signs and recumbency are also associated with decreased survival rate.

Prevention

Due to the large metabolic demand of carrying multiple fetuses, exponential growth, and mammary gland development late in gestation all pregnant small ruminant dams should be provided with additional energy sources with adequate bunk space to access feed. General guidelines can be applied allowing gestating small ruminant dams good quality forage at 2 lb/head/day and grain supplementation at 0.5 lb/head/day. While on farm, the practitioner should evaluate the other pregnant animals to get an idea of BCS, stage of gestation, and age groups specifically first-time pregnancies and aged dams to gauge the risk of pregnancy toxemia development in other members of the herd. Separating at risk dams to allow additional feeding and monitoring may be needed. Herd monitoring consistent with blood BHB concentration or ketonuria can also be implemented to drive further interventions including pre-emptive treatment with oral propylene glycol administration.

Conclusions

Early and accurate diagnosis of disease is critical for improved chances of maternal and fetal survival. Pregnant small ruminants should be provided an energy rich diet starting by mid gestation. If a dam is recumbent with neurologic signs, the prognosis is guarded for both dam and fetuses. Providing recommendation to improve herd management is critical to decrease risk in the entirety of the herd.

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