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Pregnancy Toxemia (Ketosis) in Ewes and Does[1]

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Abstract

This article is meant to provide producers with a general overview of pregnancy toxemia, or ketosis, in small ruminants. The producer is always advised to consult with their veterinarian to ensure the best plan is in place for their scenario, including diagnosis, treatment, and prevention. Each individual case is unique; therefore, the approach to each situation is not necessarily the same.

Introduction

Pregnancy toxemia, also known as ketosis or twin-kid/lamb disease, is a serious condition of does and ewes. It most commonly occurs during the last 4–6 weeks of pregnancy. The final growth stage of the fetus and the start of lactation result in a profound increase in energy demands for the dam. Some does and ewes are unable to compensate for this increase and enter a negative energy balance, also called ketosis. This is especially true for pregnancies with multiple kids or lambs, where energy demands are significantly increased compared to singleton pregnancies. Other contributing factors include stressful events, such as transportation or weather changes, decreased rumen capacity due to fetal growth, and a decline in nutrition. Even brief periods of withholding feed for transportation, or decreased appetite due to another systemic disease, can lead to a decline in energy balance, which may result in the onset of pregnancy toxemia.

Two predominant forms of the disease exist. The first form is defined as obesity-related pregnancy toxemia. This form tends to occur in does and ewes with significantly elevated body condition scores (BCS ≥ 4.0, where a BCS of 1.0 is emaciated and 5.0 is obese). Refer to Small Ruminant Body Condition Scoring for more information regarding proper utilization of the BCS system. In these cases, the elevated body condition score leads to a predisposition for fat mobilization, hepatic (liver) lipidosis, hypoglycemia (low glucose), and ketosis when faced with a negative energy balance. The second form of the disease is defined as starvation pregnancy toxemia and is related to inadequate nutrition (under-conditioning), multiparity (multiple fetuses), or advanced maternal age. In these cases, primary hypoglycemia and hypocalcemia (low calcium) predominate within 1 to 3 weeks of parturition (birth) and lead to a rapid decline in health.

Signs of Disease

While the pathogenesis behind pregnancy toxemia is not fully understood, the consequences can be quite severe and result in significant economic losses on both large-scale and small production herds. Be on the lookout for early signs of pregnancy toxemia in does and ewes that are reaching late-stage gestation.

Common clinical signs of pregnancy toxemia may include:

1. Decreased appetite.
2. Grinding teeth or other signs of discomfort.
3. Sudden decrease in milk production (in animals currently lactating).
4. Decreased energy; weakness, muscle tremors, or recumbency.
5. Disorientation, changes in vision, star gazing or convulsions.
6. Poor or rough haircoat.

Any of these clinical signs may occur in combination or alone, with other abnormalities possible as well.

Diagnosis

Diagnosis is made based on a mixture of clinical signs, history, and clinical pathological findings. Early changes include the presence of ketonuria (ketones in urine), which often develops before ketonemia (ketones in blood). Common bloodwork abnormalities include ketonemia, metabolic acidosis, hypoglycemia (although hyperglycemia does not rule out pregnancy toxemia), hypocalcemia, and hypokalemia. Animals with neurologic symptoms may also have decreased cerebrospinal fluid (CSF) glucose values, thought to be related to hypoglycemic encephalopathy. In some cases, a nonspecific but marked increase in white blood cells (neutrophils) may occur.

A particularly helpful way to screen for pregnancy toxemia is through measurement of beta-hydroxybutyrate (BHB) levels in blood. When glucose stores are depleted, fat is mobilized to the liver for conversion into an alternative energy source known as ketone bodies. BHB is one of the ketone bodies produced, and it provides essential energy delivery throughout the body when the supply of glucose is exceeded by metabolic demands. Under normal conditions,

the level of BHB within the body should be less than 0.8 mmol/L. Subclinical disease with mild to moderate pregnancy toxemia is associated with serum-BHB levels of 0.8 mmol/L–1.6 mmol/L. More significant clinical signs begin to develop at serum-BHB levels above 1.6 mmol/L. Evaluation of BHB levels can be used on an individual basis for monitoring disease progression and can also be useful for herdwide screening for signs of malnutrition that may predispose animals to disease development. When serum-BHB levels rise above 0.7 mmol/L, spillover of other ketone bodies into the urine occurs, known as ketonuria. Measurement of urine ketones with commercial qualitative test strips can provide additional diagnostic information but is less sensitive as an early indicator of disease. Producers should work with their veterinarians to assess the aforementioned parameters and determine the animal's status and appropriate intervention plan.

Treatment

At the earliest onset of clinical signs, intervention under veterinary guidance is paramount because the disease can progress quickly. It is critical to identify if the animal has any comorbidities, which should be treated concurrently. The mainstays of treatment include administration of energy sources and removal of factors that cause negative energy balance. In the early stages of the disease, adjustments to nutrition or supplementation with oral propylene glycol may be beneficial. However, propylene glycol is bitter and can worsen decreased appetites; it can be ineffective if anorexia or decreased rumen function develops. In such cases, the most effective form of supporting energy intake is through intravenous dextrose supplementation. Simply administering oral sugar supplements is not a primary or effective treatment. Care must also be taken to correct for other bloodwork abnormalities such as decreased blood pH (acidosis) and decreased calcium due to elevated BHB levels, which are commonly seen in these cases.

In many cases, consideration for the importance of the dam versus the offspring can be utilized to decide the treatment plan. If the dam is of primary concern, then an emergency cesarean section (C-section) or induction of parturition should be considered. For cases where the offspring is of primary concern, it is important to ensure reliable breeding dates are available for estimation of gestation age. Induction of parturition or C-section may still be considered in these cases, as long as the offspring are within a week of their due date. Supplementation of nutrition or removal of the offspring from the dam should be performed for the offspring after parturition, because the production of milk demands a large amount of resources from the dam.

Prevention

Several methods of prevention can be implemented to reduce the occurrence of disease. Providing adequate and well-balanced nutrition, especially in the late stages of pregnancy, is an excellent way to support the increasing energy demands that ewes and does experience. On average, an increase of 50% more feed is required for single kid/lamb pregnancies, whereas a 75% increase in feed is required for twin pregnancies. Slow introduction of grains (to avoid rapid pH changes in the rumen) over time can help to manage the need for increased energy without a sudden increase in the volume of feed intake as demand increases. Providing energy-dense forages as part of the diet is also important to maintain healthy rumen function. Energy-dense forages include those with high amounts of energy, measured by Total Digestible Nutrients (TDN) per unit of dry matter. Examples of energy-dense forages include ryegrass, chicory, and certain legumes.

Another method of prevention is based on identifying which does or ewes are at an increased risk of pregnancy toxemia. Having your veterinarian perform pregnancy evaluation for fetal counts (via ultrasound at 40–90 days of gestation) helps identify which animals are at increased risk. For dams with multiple fetuses entering late-state gestation, higher-energy feeding and more intense monitoring and management practices can be implemented. Additionally, it is important to keep accurate records of which does or ewes have previously experienced pregnancy toxemia, as they may be more susceptible with future pregnancies.

Insufficient time between parturition and breeding back is another predisposing factor and is therefore an important consideration for pregnancy toxemia prevention. Both gestation and lactation require significant metabolic contributions, especially when combined. The cumulative metabolic demands often exceed the reserves and daily intake of energy and lead to higher incidence of pregnancy toxemia. This is particularly true in animals that lose body condition score throughout their pregnancy or lactation. Insufficient recuperation time prior to breeding back can lead to continued decline in condition with each cycle and further increase the risk of pregnancy toxemia.

Does and ewes should ideally be kept in medium body condition throughout the pregnancy (BCS 3.0–3.5). Over-conditioning (BCS > 4.0) can increase susceptibility to stressors in late-stage pregnancy, predispose ewes and does to metabolic dysregulation, and lead to abdominal fat stores that limit how much feed the rumen can hold. Closely monitoring does and ewes during the first half of pregnancy is important to ensure they maintain medium body condition when their metabolic demand is lower.

General Recommendations

Early intervention is key for improving outcomes and survival in cases of pregnancy toxemia. Always consult with your veterinarian if you have concerns of pregnancy toxemia or observe signs of underlying illness in your ewes

or does. Your veterinarian may initially recommend administering glucose or electrolyte solutions, but medical evaluation is always the best course of action.

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