Treatment and Control of Peri-Parturient Metabolic Diseases: Pregnancy Toxemia, Hypocalcemia, Hypomagnesemia

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KEYWORDS

• Goat • Hypocalcemia • Hypomagnesemia
• Metabolic diseases • Pregnancy toxemia • Sheep • Treatment

In general, peri-parturient metabolic diseases in ewes and does—pregnancy toxemia, hypocalcemia, and hypomagnesemia—are caused by the failure of animals to have their nutritional requirements met during late pregnancy and/or early lactation. These disorders can be significant causes of peri-parturient mortality of ewes and does.[1–3] Decreased intake of the respective nutrients, usually in association with increased requirements of the animals, contributes to development of the pathologic conditions. Various factors can predispose the animals to these diseases. The early stages of the pathologic conditions are characterized by reduced appetite, which leads to further reduction of the intake of nutrients, in turn precipitating development of the disease and increasing morbidity. This article provides guidelines on the treatment and control of 3 peri-parturient diseases in ewes and does, namely pregnancy toxemia, hypocalcemia, and hypomagnesemia.

PREGNANCY TOXEMIA

Pregnancy toxemia ("twin-lamb disease") is a metabolic disorder of pregnant small ruminants, caused by an abnormal metabolism of carbohydrates and fats, which

The authors have nothing to disclose.
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Vet Clin Food Anim 27 (2011) 105–113
doi:10.1016/j.cvfa.2010.10.004 **vetfood.theclinics.com**
0749-0720/11/$ – see front matter

occurs at the final stage of pregnancy. The disease occurs more frequently in ewes than in does; lean (body condition score <2 in the 5-point scale) or obese (body condition score ≥4) animals, as well as animals carrying 2 or more fetuses, are at higher risk of developing the disease. The disease is characterized by development of hypoglycemic encephalopathy. Animals show anorexia, depression, neurologic signs, and blindness, followed by recumbency and coma.[4] The salient paraclinical findings are hypoglycemia and hyperketonemia/hyperketonuria of affected animals.

Treatment of Clinically Ill Female Animals

Treatment of the disorder should be based on 2 general principles: (a) administration of energy sources and (b) removal of factors that increase energy requirements of affected animals. The efficacy of the treatment depends on early instigation which, in turn, relies on timely and correct diagnosis of the disease. However, even in cases of early instigation of treatment, this may still fail. In animals with signs of the terminal stage of the disease (neurologic signs, blindness, recumbency), treatment often leads to transient improvement of the general condition of the animal, which could subsequently deteriorate, with eventual death of the animal. In such cases, for welfare reasons euthanasia of affected animals would be recommended, even before instigation of treatment. Substandard welfare of sick animals adds to the financial constraints of treatment, which can be expensive but frequently fruitless. In fact, Sargison[5] has reported that, despite a full course of treatment of toxemic ewes, only one-third of the animals would likely survive.

In hospitalized animals, intensive care involves indwelling intravenous catheterization, followed by administration of glucose (5–7 g) every 3 to 4 hours until full recovery.[1]

In veterinary practice situations, emergency pharmaceutical treatment consists of oral administration of propylene glycol (600 mg/mL). Rook[1] recommended administration of 100 to 200 mL twice daily, whereas other investigators recommend 60 mL twice daily,[6,7] which is considered less likely to cause side effects. A better approach would be to start treatment with 2 doses, each 150 to 200 mL, on the first day, thereafter reducing it to 60 mL per dose. The regime should be followed for up to 6 days, depending on the improvement of the animal's condition. Alternatively, glycerol (60 mL/animal, twice daily for 3–6 days) may be administered. Sodium propionate, liquid molasses, sodium lactate, or ammonium lactate may also be used as glucose sources, but they are not metabolized as quickly as propylene glycol. High doses of all these substances can disrupt normal function of the animal's ruminal flora, thus predisposing to ruminal acidosis. Oral administration of a concentrated dextrose plus electrolytes solution at a dose of 160 mL/animal, 3 to 4 times daily for 3 to 6 days, has also been found to be effective.[8,9]

Administration of recombinant bovine somatotropin at 0.15 mg/kg body weight (BW)[7] or single administration of a slow-release formulation at 160 mg/kg BW[10] has been shown to be of some benefit. Administration of insulin (intramuscular administration of protamine zinc insulin, 20–40 IU/animal daily, every 2 days until recovery) restores glucose uptake and has been suggested as an adjunct to the energy treatments described here, to increase recovery rates of seriously ill ewes[1,11]; in financial terms, however, its use would be justified only in animals of high reproductive value. Flunixin meglumine (intramuscular administration at 2.5 mg/kg BW for up to 3 days) can also be of help as an adjunct therapy to the aforementioned protocols.[12]

Administration of broad-spectrum anthelmintic(s) for effective treatment of gastrointestinal nematodes and liver trematode parasites should be considered. In such a case administration of levamisole is not recommended, as it has been associated with causing abortion in animals during late pregnancy.[13]