METABOLIC DISEASES

Hypocalcemia, Pregnancy Toxemia, Polioencephalomalacia

These diseases have similar causes, similar symptoms, and can occur together but their treatments are very different.

HYPOCALCEMIA (Parturient Paresis, Milk Fever)

Occurs from 6 weeks before to 10 weeks after kidding. Seen most commonly in does with multiple fetuses, diets high in mineral fed prepartum, and can be triggered by a period of reduced feed intake (i.e.- bad weather, transportation etc.) 24 hours prior to symptoms.

Symptoms include initial excitability and stiffness progressing to involuntary tremors, rapid heart rate and respiration, hypothermia, bloat, unsteadiness, stargazing, paralysis, coma and death within hours.

Main differential diagnosis if occurring before kidding is pregnancy toxemia, and these can occur together.

Caused by low blood calcium due to increased demand for fetal skeletal development before kidding, and for milk production after kidding. The metabolic mechanism mobilizing calcium from the bone requires 24 to 72 hours to activate. Activation can be suppressed in the face of high mineral supplementation. Symptoms develop with sudden increase in demand, delay in response and reduced intake occurring together.

Treatment requires calcium supplementation, preferably IV for fastest results. Calcium borogluconate 23%, 50-150 ml given IV *slowly*, monitoring heart rate and rhythm. Oral administration of calcium gel or subcutaneous administration of calcium solution prevents relapse, but often fails to achieve high enough blood levels to treat acute cases.

Prevention includes late pregnancy diets lower in calcium (but adequate in energy and protein), avoiding supplements high in calcium and phosphorus, and eliminating stressors including movement, fasting, parasitism, and shipping in the last 8 weeks of pregnancy.

PREGNANCY TOXEMIA (Twin disease, Ketosis)

Occurs in the last 4 weeks of gestation with rapid rise in fetal development and metabolic need. Nearly twice maintenance levels of dietary protein and energy are needed by animals carrying twins. Thin or over fat, older does with multiple fetuses are most at risk. Reduced feed availability or intake in late gestation will trigger symptoms (bad weather, shipping, poor feed year, rumen capacity decreased by pregnancy and obesity, illness or lameness).

Symptoms begin with decreased appetite, rapid weight loss and listlessness progressing to aimless wandering, involuntary twitching or tremors, teeth grinding, blindness, weakness, recumbency, coma and death over 2-4 days. Toxemia from fetal death and decomposition can complicate.

Caused by increased glucose demands by the fetus(es). Glucose is supplied by 1) dietary intake and 2) conversion of body fat to glucose by the liver. If intake is inadequate and fat metabolism is accelerated the liver becomes infiltrated with fat causing failure, and byproducts of fat metabolism accumulate in the bloodstream causing ketoacidosis. Ketosis in turn suppresses appetite which reduces intake further and accelerates self-poisoning.

Treatment is aimed at supplying glucose, correcting fluid and pH balance and eliminating glucose demand. Early cases can be treated with propylene glycol 60 ml twice daily for 3 days and oral electrolytes with glucose 2-4 quarts 2-3 times daily until stabilized. Severe cases will need IV treatment with sodium bicarbonate to correct the acidosis, 60 to 100 ml 50% dextrose to correct the hypoglycemia and fluids to correct hydration and electrolytes. In both situations supplementation with calcium and electrolytes as well is usually helpful. The decision to terminate the pregnancy to eliminate demand depends on the value of the doe and/or kids, her condition, and whether the kids are still alive determined by ultrasound. The pregnancy can be terminated by caesarian

section if the doe is valuable enough, well enough and far enough along, otherwise induce with dexamethasone 5-10 cc IV or IM.

Prevention is aimed at adequate, regular intake of protein and energy in late gestation and avoiding management circumstances that interfere with that. Animals should be entering late gestation in moderate body condition, have plenty of feeder space, have procedures done *before* the last month of pregnancy.

POLIOENCEPHALOMALACIA (PEM, cerebrocortical necrosis)

Can occur as primary disease or secondary to other illness.

Caused by thiamine (Vitamin B1) deficiency; possibly caused by high sulfur intake. Thiamine is usually produced by the rumen microbes. Deficiency caused by upset in microflora (i.e.-moldy feed, prolonged antibiotic therapy), ingestion of certain plants (bracken fern), or prolonged diarrhea (i.e. - coccidiosis).

Symptoms progress from ataxia to recumbency, stargazing, blindness, seizures, coma and death.

Treatment consists of *early* administration of Thiamine 10 mg per lb IV, IM, SQ every 8 hours to effect. B Complex injectable is commonly used – much larger volumes are needed to provide adequate thiamine levels. Response usually occurs within 24 hours, but can take 3 or more days. Dexamethasone 10-20 cc given IV, IM or SQ once may help reduce cerebral swelling. Rumen transfaunation or probiotics will help re-establish normal rumen microbe populations.

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