HYPOMAGNESEMIA, HYPOCALCEMIA, AND RUMINITIS IN UNGULATES: AN UNDER-RECOGNIZED SYNDROME?

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Abstract

Macro- and microminerals are critical in maintaining normal physiologic processes. Requirements for domestic ungulates have been documented and are available through the National Research Council (NRC).[4] Normal values have not been established for most wildlife species. Due to specialized feeding habits, collection of baseline data for each species is needed to develop complete diets and detect nutritional diseases.[3] Several cases of hypomagnesemia in captive ungulates are described in this report.

Introduction

Formation of a new zoological collection presented a unique opportunity to evaluate animals obtained from many different sources and management conditions. Serum mineral panels were added to the routine bloodwork performed on ungulates to help determine species-specific reference ranges for this collection.

Calcium

The majority of the body's calcium (Ca) is stored in the skeleton. It plays a major role in muscle function, nerve conduction, and enzymatic processes.[5] In general, the concentrations of Ca in roughages are highest in legumes (alfalfa) and lowest in grains. Absorption of Ca from the diet is affected by multiple factors, including the presence of oxalates, adequate concentrations of Ca and phosphorus (at a 1:1 to 2:1 ratio), and relative balance of parathyroid hormone, calcitonin, and vitamin D. Hypocalcemia may be due to an imbalance in homeostasis, rather than a dietary deficiency.[5] Acute signs include: stiffness, tetany, weakness, decreased rumen motility, shock and death. Chronic signs of hypocalcemia include reduced feed intake, poor growth, rickets, osteomalacia, lameness, hunched stance, and pathologic fractures.

Phosphorus

Phosphorus (P) is important in skeletal structure and as a source of chemical energy (ATP). Serum concentrations are not as closely regulated as those of Ca and vary with dietary intake and renal excretion. Concentrations in feedstuff are high in oil seeds, moderate in grains, and lowest in grasses and legumes.[5] High P diets tend to suppress Ca absorption and predispose to the development of uroliths.

Magnesium

Seventy percent of the total body magnesium (Mg) is found in bone. It also plays a role in muscle contraction, energy metabolism, and Ca metabolism.[5] Serum concentrations are not under close hormonal control, but vary with dietary intake and renal excretion. The availability is lower in forages than grains. Multiple factors affect absorption including serum Ca and rumen pH. Serum Mg concentrations depend on constant dietary absorption. Magnesium is primarily absorbed in the rumen, and efficiency of absorption tends to decrease with age. Clinical signs of hypomagnesemia are seen when concentrations drop below 18 ppm in cattle.[1] There may be a considerable lag between development of hypomagnesemia and appearance of clinical signs. Signs of tetany often coincide with development of acute hypocalcemia with preexisting hypomagnesemia.[1] Acute signs are observed with a rapid decline in serum Mg, such as when a ruminant is put on lush pasture ("grass staggers"). Chronic hypomagnesemia usually develops over months in response to a marginal diet.[5] Clinical signs include: poor growth rate or body condition, nervous signs (twitching ears, kicking at abdomen, hyperexcitability, chomping jaws, frothy salivation), tetany, and seizures. Signs can be precipitated by stress, and may progress to death. Although the whole herd may be involved, usually only a few animals show clinical signs during any given period.

Role of Feed Management

Rumen acidosis can be a cause of mineral imbalances. Improper feed management can lead to subacute and chronic rumen acidosis. Decreased rumen pH is associated with intake of fermentable carbohydrates, decreased effective fiber intake, stress, and overall decreased feed intake.[2] Signs associated with rumen acidosis are: laminitis, poor body condition, variable feed intake, abnormal rumen motility, and changes in fecal consistency. Factors that may prevent chronic rumen acidosis include: avoiding limited access time to feed concentrates or competition for feeders; providing adequate roughage (stemmy hay); addition of buffers and fiber to the diet; providing a balanced diet; and minimizing stress.

Methods

Serum samples for mineral analyses were collected over 4 yr during routine exams (animals were either in restraint device or immobilized) and submitted to Michigan State Animal Health Diagnostic Laboratory (Lansing, Michigan 48909 USA). Most ungulates were fed a commercial pelleted diet (Mazuri ADF 16 or 25, St. Louis, Missouri 63166 USA), small amounts of produce, alfalfa and/or Bermuda grass hay, and fresh cut browse (average percentage DM: 1% pellets; 1.5-2% hay; 0.3% browse). Nutrient and mineral concentrations of feed are analyzed as part of the quality control program. Animals were fed in barns or holding areas. In addition, animals had access during the day to a habitat containing multiple species of grasses, shrubs, and trees. Although data regarding consumption of diet fed is available, amount of habitat vegetation consumed is unknown.

Results and Discussion

Initially, results were compiled to determine this collection's reference ranges for each species. The first indication of possible clinical signs associated with hypomagnesemia occurred in an adult female kudu (*Tragelaphus strepsiceros*) in January 1999. Initially this animal presented for weight loss, rough hair coat, and retained placenta following birth of a weak calf. She later had an acute onset of muscle fasciculations, hunched posture with stiff gait, and lethargy. Values at that time showed hypocalcemia and hypomagnesemia (Ca = 4.8 mg/dl, P = 8.2 mg/dl, Mg = 12 ppm).

A second female kudu showed tetanic muscle contractions during the first trimester of pregnancy. This animal had serum Ca = 6.0 mg/dl, P = 10.0 mg/dl, and Mg = 8.06 ppm. A review of records showed that 6 mo previously this animal had a dystocia and bloodwork at that time revealed serum Ca = 7.1 mg/dl, P = 8.7 mg/dl, and Mg = 9.34 ppm.

Three adult male kudu presented with bilaterally symmetric facial/cervical alopecia and loss of body condition. Initial diagnostics showed no significant abnormalities except an inverse Ca:P ratio and hypomagnesemia. One male kudu began exhibiting a hunched posture with shifting and stretching of its hindlimbs when standing. Follow-up examination showed a progression in loss of condition, evidence of laminitis, hypocalcemia (Ca = 6.7 mg/dl), hyperphosphatemia (P = 14.6 mg/dl), and hypomagnesemia (Mg = 10.3 ppm).

A second male kudu was immobilized for examination. Although the anesthetic procedure was uneventful, approximately 3.5 hr after reversal, this kudu was observed to suddenly stiffen and fall into lateral recumbency with muscle fasciculations. The animal's condition deteriorated rapidly despite supportive care, and it died within 20 min of initial signs. Bloodwork revealed severe hypocalcemia (Ca = 4.5 mg/dl), hyperphosphatemia (P = 13.5 mg/dl), and hypomagnesemia (Mg = 7.75 ppm). Necropsy findings included evidence of chronic/active rumenitis and laminitis.

The third, less severely affected, male kudu was immobilized to further investigate the syndrome. Laboratory results were similar to the other cases (Ca = 6.8 mg/dl, P = 12.0 mg/dl, Mg = 9 ppm). Due to evidence of a herd problem, diet changes were implemented (addition of chelated minerals (4-Plex, Zinpro, Eden Prairie, MN), buffers (sodium bicarbonate, magnesium oxide), and increased effective fiber (white oak, soy hulls, citrus pulp). Six months after the diet change, this kudu was immobilized for a follow-up exam. General body condition and hoof and hair growth had improved. However, despite an uneventful anesthetic procedure, the animal developed stiff gait, hunched posture, and muscle fasciculations the following day. Results from the initial immobilization showed hypocalcemia (Ca = 6.4 mg/dl), hyperphosphatemia (P = 13.6 mg/dl), and hypomagnesemia (Mg = 7 ppm). This animal was treated with 23% calcium gluconate (250 ml s.c., 125 ml i.v.; Phoenix Scientific Inc., St. Joseph, MO 64506), 50% magnesium sulfate (2 ml i.v., 35 ml s.c.; American Regent Laboratories Inc., Shirley, NY 11967), and i.v. fluids (25 L LRS, 375 ml 5% dextrose in LRS). The animal's condition continued to deteriorate and it was euthanized. Necropsy findings confirmed chronic rumenitis.

Although the kudu herd appeared to be most severely affected, other species also were at risk based on review of medical records. Several eland (*Taurotragus oryx*) had developed evidence of laminitis and one showed facial tremors and stiffness postimmobilization. This individual had serum Ca = 8.9 mg/dl, P = 9.2 mg/dl, and Mg = 13 ppm. Other affected eland typically had magnesium values in the range of 12-16 ppm, often associated with inverse Ca:P ratios. Nyala (*Tragelaphus angasii*) also appeared likely to develop clinical problems associated with mineral imbalances. An adult male nyala developed facial and flank alopecia. Serum minerals were Mg = 12.1 ppm, Ca = 6.9 mg/dl, and P = 8.9 mg/dl. An additional immobilization occurred 6 mo later. The procedure was uneventful, however the animal developed signs of shifting leg lameness and hypermetria the afternoon of the immobilization. These signs rapidly progressed to recumbency, seizures, and death. Lab results (Ca = 7.1 mg/dl, P = 9.8 mg/dl, Mg = 8 ppm) and clinical signs were consistent with hypomagnesemia. Chronic rumenitis was found at necropsy.

A compilation of mineral results for various ungulates was made and species grouped as grazers, browsers, or intermediate feeders. In most of the grazers, ranges and mean values for serum Mg fell in expected ranges (Table 1). Occasional low values were seen in individual gemsbok (*Oryx gazella gazella*) and scimitar-horned oryx (*Oryx dammah*). Among the browsers, kudu and nyala herd mean Mg values were lower than the other species (Table 2). Bongo (*Tragelaphus eurycerus*) may also be a potential concern with slightly lower mean Mg values. Eland appear to be at risk among the intermediate feeders (Table 3). The eland herd's mean value was also lower than other species.

It is speculated that feeding concentrates may play a role in the development of rumen acidosis/chronic rumenitis and subsequently affect Ca:P balance and Mg absorption. Addition of buffers and chelated minerals may ameliorate some of these changes, however, the primary underlying etiology is still incompletely understood. Preliminary data suggest that Ca:P balance and Mg concentrations may be improved if treated early.

Mineral imbalance may result from inappropriate diets, concentrate feeding resulting in alterations in rumen pH, and concurrent disease. Measurement of trace minerals in exotic ungulates may be an underutilized diagnostic tool in assessing herd health. Clinical signs of alopecia, loss of body condition, lameness, muscle fasciculations, and seizures have been observed with hypocalcemia concurrent with hypomagnesemia in kudu, nyala, and eland. Evaluation of diet and feed management, along with serum mineral concentrations, should be performed to allow veterinarians and nutritionists the ability to recognize, treat, and prevent mineral imbalances leading to clinical signs.

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Table 1. Selected mineral values in grazer species at Disney's Animal Kingdom.

Grazer species	Ca (ppm)	P (ppm)	Mg (ppm)		SD	*n*
			range	mean		
Goat (*Capra hircus*)	90-116	37-97	21-29			
Cattle (*Bos taurus*)	85-110	45-80	20-35			
Ankole (*Bos taurus*)	91-105	76-109	18-24	20.6	1.9	8
Banteng (*Bos javanicus*)	87-119	65-114	17-27	21.7	2.7	10
Gemsbok (*Oryx gazella gazella*)	71-119	43-124	13-26	19.2	2.9	19
Scimitar horned oryx (*Oryx dammah*)	69-113	59-124	12-26	20.6	3.5	29
Sable antelope (*Hippotragus niger*)	73-113	40-104	15-25	21.3	2.4	24
Wildebeest (*Connochaetes taurinus*)	91-109	41-124	18-25	19.9	2.4	11