Nutritional support for treatment of hepatic lipidosis in a llama

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- ➤ Hepatic lipidosis is a disease characterized by abnormal accumulation of lipid in the liver and is associated with high mortality in camelids.
- ➤ Partial parenteral nutrition with enteral supplementation can be used to maintain adequate energy intake and minimize further lipid mobilization.
- ➤ Because of the distinctive metabolism of camelids, supplementation with higher amounts of amino acids (relative to nonprotein calories) in parenteral solutions rather than those traditionally provided to other species may be required.
- ➤ Management of hepatic lipidosis in llamas should focus on early recognition and treatment of the primary disease process combined with aggressive nutritional support.

Following a 3-week period of sustained high environmental temperatures (> 35 C [> 95 F]), 2 lactating female llamas from a single farm in central Oregon died within 1 week of each other; hepatic lipidosis was diagnosed on the basis of results of histologic evaluation. A third llama from the same farm, a 3-year-old female that was 3 months into her first lactation and 10 weeks pregnant, was anorectic for approximately 24 hours and was found recumbent one morning. The referring veterinarian, who was also the owner of the llama, performed a physical examination, collected a blood sample, and initiated symptomatic treatment prior to referring the llama to Oregon State University. The referring veterinarian reported that a mass of firm dry feces could be palpated per rectum and appeared to be in the spiral colon. Results of biochemical analysis revealed slightly decreased BUN concentration, hypokalemia, hyperglycemia, and increased total bilirubin, **γ-glutamyltransferase (GGT)**, **alkaline phosphatase (ALP)**, and **aspartate aminotransferase (AST)** activities (**Table 1**, day –1). A CBC revealed leukocytosis and neutrophilia with a left shift (1,230 cells/µl; reference range, 0 to 147 cells/µl). All other hematologic variables and fibrinogen concentration were within reference ranges. The llama was treated with lactated Ringer's solution, supplemented with 10 mEq/L of potassium chloride (4 L, IV), ceftiofur sodium[a] (2.2 mg/kg [1.0 mg/lb] of body weight, IV), and flunixin meglumine[b] (1.1 mg/kg [0.5 mg/lb], IV). A solution consisting of water (2.5 L), 220 g magnesium hydroxide, 25 ml dioctyl sodium succinate, and 30 g potassium chloride was administered via orogastric tube. A commercially available solution consisting of calcium, phosphorus, and magnesium salts[c] was also administered IV.

Following initial treatment, the llama and her nursing cria were transported to the Veterinary Teaching Hospital at Oregon State University. On admission, the llama was in lateral recumbency and was bradycardic (heart rate, 40 beats/min), tachypneic (respiratory rate, 56 breaths/min), and slightly hyperthermic (rectal temperature, 39 C [102.2 F]). She was somewhat alert to her surroundings. The llama weighed 224 lb (102 kg) and had a body condition score of 3 on a scale of 1 to 10.[1] Peristaltic sounds could be not be detected, and she was assessed to be mildly dehydrated on the basis of clinical findings. Palpation per rectum confirmed the presence of a possible dry fecal mass in the spiral colon, further suggesting a decreased hydration status. Hepatopathy was a concern, given the previous diagnosis of hepatic lipidosis in the other clinically affected llamas from this farm.

Initial diagnostic tests consisted of a CBC and serum biochemical analysis, including serum bile acid concentrations and **sorbitol dehydrogenase (SDH)** activity to further aid in assessment of hepatic function. Although the serum was not grossly lipemic, serum triglyceride concentrations were measured to assess the ability of the liver to export lipid. Amylase (603 U/L; reference range, 420 to 1,200 U/L) and lipase (59 U/L) activities were measured to evaluate whether there was pancreatic involvement. Although a reference range for lipase activity in llamas could not be found, the determined value was within reference range for other species.[2] Hematologic abnormalities included leukocytosis, lymphocytosis (5,346; reference range, 689 to 4,848 cells/µl), and neutrophilia with a left shift (1,450 cells/µl; reference range, 0 to 147 cells/µl) with many toxic neutrophils (Table 1, day 0). Biochemical abnormalities included hyperglycemia, hyperbilirubinemia, decreased BUN, hypokalemia, hypoproteinemia, slight hyponatremia, and increased ALP and creatine kinase activities. Hepatocellular damage and cholestasis were evidenced by increased GGT, AST, bile acids, and SDH analytes. Retrospective analysis of a serum sample obtained at the time of initial evaluation in the field revealed an increased concentration of **nonesterified fatty acids (NEFA;** 1.14 mEq/L; reference range, < 0.6 mEq/L), which is consistent with negative energy balance and excessive fat mobilization.[3] A urine sample collected midstream revealed a

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Table 1—Selected hematologic and serum biochemical values for a 3-year-old lactating female llama with hepatic lipidosis

Variable	Day of treatment relative to day of admission (day 0)							Reference range*
	−1	0	2	5	7	8	12	
WBC (cells/μl)	24,600	29,700	20,500	17,300	17,100	ND	10,600	8,000–21,400
Neutrophils (cells/μl)	19,434	22,869	17,220	12,975	13,680	ND	6,996	4,711–14,686
BUN (mg/dl)	21.0	17.0	18.0	13.0	11.0	11.0	9.0	24.0–44.0
Creatinine (mg/dl)	2.5	2.4	1.9	1.4	1.3	1.2	1.5	1.5–2.7
Glucose (mg/dl)	171	331	482	207	127	162	109	89–132
Total protein (g/dl)	5.5	4.8	4.5	4.6	4.6	4.5	5.4	5.3–7.3
Albumin (g/dl)	3.7	3.1	2.9	2.9	2.8	2.7	3.2	3.0–4.2
ALP (U/L)	112	107	124	1426	ND	703	303	12–97
CK (U/L)	63	987	131	106	ND	172	31	0–552
AST (U/L)	2,558	2,128	1,938	2,298	1,721	1,367	844	69–241
GGT (U/L)	155	161	296	616	ND	413	268	16–46
Sodium (mEq/L)	152	147	141	143	150	148	154	149–158
Potassium (mEq/L)	2.8	2.0	1.9	2.4	3.2	3.7	6.3	3.8–7.3
Calcium (mg/dl)	8.8	9.1	9.6	9.3	10.6	10.3	8.7	8.4–10.8
Phosphorus (g/dl)	3.2	1.5	1.3	1.2	1.4	4.8	5.6	1.3–10.1
Magnesium (g/dl)	ND	2.4	0.7	0.6	1.2	1.7	3.0	1.6–4.9
SDH (U/L)	ND	40.9	40.0	61.2	14.1	ND	9.4	0–15
Bile acid (μmol/L)	ND	1,112	997	371	14	ND	16.2	15–30
Total bilirubin (mg/dl)	1.0	1.2	1.0	0.3	ND	0.1	0.1	0–0.3

ALP = Alkaline phosphatase. CK = Creatine kinase. AST = Aspartate aminotransferase. GGT = γ-glutamyltransferase. SDH = Sorbitol dehydrogenase.
*Adult llama reference ranges obtained from the Clinical Pathology Laboratory, College of Veterinary Medicine, Oregon State University.

specific gravity of 1.033, pH 9, 2+ glucose, 1+ protein, ketone negative, and 1+ bacteria. Alkalinity of the urine and hyperglycemia may have caused the increased proteinuria and glucosuria, respectively. Ultrasonographic evaluation of the abdomen revealed an abnormal appearance of the liver, with decreased parenchymal echogenicity and many small diffuse hyperechoic foci throughout the hepatic parenchyma. These hyperechoic foci had acoustic shadowing; these foci were believed to be the result of mineralization rather than fibrosis because of the strong echos that were produced as well as the acoustic shadowing. Results of a Baerman fecal examination were negative. Results of coagulation assays revealed no abnormalities. Histologic examination of a hepatic tissue biopsy specimen revealed moderate diffuse lipid accumulation within the hepatocytes; results of bacteriologic culture of hepatic tissue were negative.

On the basis of the hematologic findings of neutrophilia with a left shift and toxic neutrophils without a known cause, the treatment plan continued to be supportive. Administration of ceftiofur sodium (2.2 mg/kg [1.0 mg/lb], IV, q 12 h) and flunixin meglumine (0.25 mg/kg [0.11 mg/lb], IV, q 8 h) was continued. A 14-gauge 13-cm (5 1/4 in) catheter[d] was placed into a jugular vein, using sterile technique. After receiving lactated Ringer's solution (5 L, IV) for rehydration, **partial parenteral nutrition (PPN)** was initiated, which consisted of a solution of 880 ml of 50% dextrose, 2,000 ml of 8.5% amino acids,[e] 100 ml of 23% calcium gluconate,[f] 5 ml of vitamin B complex, and 30 ml of potassium chloride (4 mEq/ml) in a base of lactated Ringer's solution. Total solution volume was 4 L, with a final concentration of 0.54 kcal/ml, 11% dextrose, 4.6% amino acids, 0.6% calcium, and 40 mEq/L potassium. This solution was administered at a rate of 260 ml/h during the first 24 hours and maintained at that rate continuously with an infusion pump. Acid-base status was monitored throughout the duration of fluid therapy. On the day of admission, the llama was in a state of metabolic acidosis with respiratory compensation, as evidenced by a P_{CO_2} of 14 mm Hg (reference range, 43 to 49 mm Hg), base excess of –14.3 mEq/L (reference range, +3 to –3 mEq/L), and pH of 7.41 (reference range, 7.34 to 7.43). Acid-base status was rapidly corrected and maintained within reference ranges with the administration of fluids. Insulin (30 units NPH,[g] SQ, q 12 h) was administered to improve glucose uptake and inhibit continued lipolysis.[3,4] Blood glucose concentration was monitored every 6 hours to ensure hyperglycemia would not be further induced by PPN supplementation and to monitor the efficacy of insulin treatment. The llama and her cria were housed in a double-bedded stall to prevent pressure necrosis of the sternum; both were monitored for attitude and behavior every 2 hours.

The nursing cria that was admitted with the llama was alert and nibbling at hay offered in the stall. After weighing the cria (95.5 lb [43.4 kg]) on admission, it was decided to leave her with the dam and monitor for signs of stress that would be expected because of the inability to nurse. The cria was observed nursing often for brief periods throughout the day. Presence of milk in the udder was confirmed by manual expression, but the amount did not appear adequate to meet the cria's nutritional needs. However, because the cria was eating grass hay and alfalfa pellets, she was allowed to stay with the dam because of the stress that may be caused by sudden weaning.

The day following admission the llama appeared stable, remaining quiet in sternal recumbency, but was still anorectic. She was transfaunated with 1 L of filtered bovine rumen fluid mixed with 1 L of warm water and 250 g of alfalfa meal. Partial parenteral nutrition was continued at 260 ml/h, along with insulin, ceftiofur, and flunixin meglumine, as described. Various foods were offered, including grass and alfalfa hays, fresh grass clippings, pelleted llama feed, willow branches, blackberry leaves, and a molasses corn-oats-barley mixture in an attempt to stimulate eating. Within 48 hours of admission, the llama was standing, alert to her surroundings, and eating small amounts of

grass hay. She urinated normally and passed some dry feces. A second transfaunation was performed, and auscultation of the abdomen revealed a continued absence of pregastric compartmental motility sounds. The WBC count was primarily within reference range at this time, but neutrophilia with a left shift (205 cells/µl; reference range, 0 to 147 cells/µl) was still evident. Results of serum biochemical analysis revealed the same abnormalities as those detected on initial examination, with additional abnormalities of hypoalbuminemia and hypomagnesemia (Table 1, day 2). Most of these biochemical abnormalities may be attributed to continuing anorexia and hepatic dysfunction. Correction of the metabolic acidosis detected at admission may account for the continued decrease in serum potassium concentration. Hypomagnesemia may be attributed to fluid therapy and inadequate oral intake of nutrients. The treatment regimen and PPN were continued unchanged; insulin was administered only when blood glucose concentration exceeded 400 mg/dl.

During the next week of hospitalization, the llama continued to eat small amounts of grass hay and remained alert, spending increasing amounts of time standing. Partial parenteral nutrition was continued, but potassium chloride in the mixture was increased from 40 to 45 mEq/L (day 5). Two additional transfaunations were performed during the first week of hospitalization. In addition to the filtered rumen fluid and alfalfa meal, an oral electrolyte product[h] containing dextrose was added to the enteral solution. After the fourth transfaunation, some peristaltic sounds could be heard over the left paralumbar fossa. A sample of fluid obtained from the first compartment of the stomach revealed small- and medium-sized protozoa.

The llama's clinical improvement during the first week of treatment was reflected in the fact that abnormal hepatic enzyme values peaked and started to decline (Table 1, days 5 to 9). A second ultrasound-guided biopsy was performed on the liver 1 week after admission. Histologic findings were unchanged. The llama was weaned off PPN over a 3-day-period by decreasing the flow rate by 50% per day beginning the ninth day of hospitalization. The llama and her cria were taken outside for walks and grazing. Treatment with antibiotics and flunixin meglumine were discontinued on day 11, and the llama was discharged from the hospital 12 days following admission. At the time of discharge (day 12), most of the previously abnormal serum biochemical values were within reference ranges, excluding AST, GGT, ALP, and BUN. The consistently low BUN concentrations throughout treatment of this llama were most likely attributable to inadequate enteral intake of protein. The continued increase of AST, GGT, and ALP activities may be a result of longer half-lives, continued leakage, or induction of these enzymes. At the time of discharge from the hospital, the llama and her cria weighed 220 lb [100 kg] and 104.5 lb [47.5 kg], respectively. One month following discharge, the llama was reported to be doing well and eventually delivered a clinically normal healthy cria at the end of her current gestation. The previous nursing cria had gained weight at a normal rate and was also reported to be thriving.

Hepatic lipidosis is an abnormal accumulation of lipid in the liver that usually develops in response to negative energy balance-induced mobilization of adipose tissue. Clinical syndromes of hepatic lipidosis are recognized in most species but are most commonly seen in anorectic cats[5,6] and periparturient ruminants.[7-9] Reports of hepatic lipidosis in camelids are limited[3,4,10]; however, a prevalence rate approaching 4% of all sick camelids has been suggested.[3] Contrary to other reports of 1 or 2 llamas with hepatic lipidosis,[4,10] the llama in our report was not ketotic, possibly because of the hyperglycemia, which is consistent with findings of another study.[3] It is likely that the pathogenesis of hepatic lipidosis in llamas is multifactorial, but it appears from the literature and our clinical experience that it is more of a secondary disease process rather than a simple overdemand for energy.[3] Illness, poor nutritional state, environmental stresses, and parasitism are but a few of the potential factors that induce periods of anorexia and either increase adipose mobilization or suppress tissue uptake of lipoproteins. In the llama of our report, it was hypothesized that the anorexia was a result of sustained high environmental temperatures (> 3 weeks' duration), which was most likely the stressor that induced the disease process. The llama of this report, possibly because of her pregnancy and lactation status, had a syndrome more similar to that seen in most ruminants, which is characterized by high serum NEFA concentration without an increase in triglyceride concentration. Hepatic lipidosis in camelids may also be evidenced by only moderate increases in serum NEFA concentration with hyperlipidemia.[3,4]

Mortality rate resultant from hepatic lipidosis is quite high for all species, even with parenteral nutritional support.[3-10] Parenteral nutritional support was selected in this instance as the best method to rapidly decrease ongoing nutrient deficits and decrease fat mobilization. Enteral nutritional support was not chosen as a primary modality because of the stress induced by daily intubation, which could potentially increase cortisol-induced lipolysis, and the impracticality of leaving a nasogastric tube in place in a llama, which is an obligatory nasal breather. In addition, the ability of the gastrointestinal tract to digest and absorb sufficient quantities of nutrients to support daily nutritional requirements was in question. Enteral supplementation with bovine rumen fluid, alfalfa meal, and electrolytes with dextrose was used to inoculate and stimulate microbial activity within the pregastric fermentation vat. It was anticipated that this enteral support, in addition to offering various forages, browse, and grains, would help stimulate the llama's appetite. Any end-product outflow from the fermentation system in the form of volatile fatty acids or microbial protein would be advantageous to maintaining integrity of the digestive tract as well as provide nutrients in addition to those provided parenterally.

Supportive treatment for hepatic lipidosis focuses on energy supplementation to minimize negative energy balance resultant from anorexia. Improving energy

balance decreases the rate of adipose-tissue mobilization, thus decreasing fatty acid uptake by the liver. Maintenance and resting metabolizable energy requirement (kcal per day) for llamas has been determined to be 84.5 × **metabolic body weight** ($W_{kg}^{0.75}$) and 59.3 × $W_{kg}^{0.75}$, respectively.[11] The lactational energy requirement for llamas is unknown but may be estimated by increasing basal energy by 50 to 100%. The mammary gland requires large amounts of glucose for lactose synthesis and amino acids for milk protein production. We estimated the total daily metabolizable energy requirement to be between 2,700 and 3,800 kcal/d. Parenteral nutrition provided approximately 2,200 kcal/d, in addition to any enteral component. On the basis of the minimal change in body weight of the llama during the period of hospitalization and the gain in body weight of the cria, our delivery of energy from parenteral and enteral sources was a reasonable approximation of actual requirements.

Typically, an emulsified lipid source is used as a source of energy in most parenteral solutions to minimize use of protein for energy and decrease osmolality of the solution. It has been demonstrated that addition of fat in the diet is beneficial in treating hepatic lipidosis in cats[5,6] but not in dairy cattle.[12] The importance of limiting or including lipid intake in camelids undergoing hepatic lipidosis is not known. Because the clinical findings in the llama of this report appeared more similar to those of hepatic lipidosis in ruminants, we elected not to use lipid in our parenteral solution. However, individual camelids with hepatic lipidosis and hyperlipidemia may be different and may respond to lipid supplementation, similar to cats.

Glucose was the sole source of nonprotein energy in the parenteral solution. Given our use of a predominate glucose solution, it was important to monitor blood and urine glucose concentrations repeatedly to avoid or correct abnormalities as they developed. Camelids do not assimilate exogenous glucose well as a result of their naturally low serum insulin concentrations.[13] Dairy cows with hepatic lipidosis also have decreased circulating insulin concentrations.[7,14] Insulin treatment is not well-documented in camelids but was used here based on the assumption of low circulating concentrations in our llama and its use in a previous report.[4] On the basis of clinical response reflected in blood glucose concentrations, insulin appeared effective in facilitating glucose and triglyceride uptake and inhibiting hormone-sensitive lipase activity, thereby slowing mobilization of adipose tissue and reducing the delivery of NEFA to the liver.

Formulation recommendations for parenteral nutrition solutions suggest a ratio of 150 to 200 nonprotein calories for every 1 g of nitrogen. The formula we used had a nonprotein calorie to nitrogen ratio of only 55:1. This low ratio would presumably lead to amino acids being catabolized for energy and not being used for protein synthesis. Protein requirements for llamas are not defined and are complicated by the presence of microbial and host animal needs. One study suggested a maintenance digestible protein requirement of 2.38 $g/W_{kg}^{0.75}$.[15] Using a true absorption coefficient of 75%,[16] this would result in a maintenance metabolizable protein requirement (gram per day) of 1.79 × $W_{kg}^{0.75}$. This suggests a maintenance requirement of 57.4 g/d or approximately 0.6 g/kg for llamas. This value is in agreement with the suggested maintenance protein supplementation rate for adult healthy horses[17] and humans.[18] Recommendations for protein supplementation in parenteral solutions for humans ranges from 0.8 to 2.2 g/kg for adult to newborn, respectively.[18] One report of **total parenteral nutrition** (**TPN**) in a neonatal llama delivered 2.1 g of protein/kg.[19] Other reports of TPN use in calves suggest a protein requirement of 3.5 g/kg.[20,21] The parenteral solution we used delivered approximately 170 g of amino acids/d, equivalent to 1.7 g/kg. The higher rate of protein delivery to our adult llama was extrapolated to account for protein needs of lactation as well as address issues of amino acid use for gluconeogenesis. No adverse effects of this amount of protein supplementation, as evidenced by alterations in biochemical profiles, were observed throughout the duration of parenteral support.

Other complications potentially associated with parenteral nutrition were not observed in the llama of this report, suggesting adult llamas can tolerate the use of this procedure well. The potential value of individual llamas and their body size allow for the use of parenteral nutrition, contrary to most situations with adult cattle. Management of hepatic lipidosis in llamas should focus on early recognition of the primary disease process in conjunction with aggressive nutritional support. Although the supportive treatment used in our llama could be debated, we feel that our approach to nutritional supplementation provides a reasonable protocol for clinicians to address nutritional support for debilitated camelids.

[a]Naxcel, Upjohn Company, Kalamazoo, Mich.
[b]Banamine, Schering-Plough Animal Health, Kenilworth, NJ.
[c]Norcalciphos, Smith-Kline Beecham, Exton, Pa.
[d]Angiocath, Becton-Dickinson Co, Sandy, Utah.
[e]8.5% FreAmine III (Amino Acid Injection), McGaw Inc, Irvine, Calif.
[f]Calcium gluconate 23% solution, Agri Laboratories Ltd, St Joseph, Mo.
[g]NPH Iletin I (100 U/ml), Eli Lilly and Co, Indianapolis, Ind.
[h]Biolyte, Upjohn Co, Kalamazoo, Mich.

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