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Retrospective Evaluation of Parenteral Nutrition in Alpacas: 22 Cases (2002–2008)

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Background: Parenteral nutrition is an important method of nutritional support in hospitalized animals, but minimal information has been published on its use in camelids.

Hypothesis/Objectives: The purpose of this study was to characterize the use of total parenteral nutrition (TPN) in alpacas, evaluate the formulations used, and determine potential complications.

Animals: Twenty-two alpacas hospitalized at the Tufts Cummings School for Veterinary Medicine (site 1: n = 8) and the Ohio State University Veterinary Teaching Hospital (site 2: n = 14).

Methods: A retrospective analysis of all alpacas that received TPN between 2002 and 2008 was performed to assess clinical indications, clinical and clinicopathologic data, and outcome.

Results: The most common underlying diseases in animals receiving TPN were gastrointestinal dysfunction (n = 16), hepatic disease (n = 2), and neoplasia (n = 2). Several metabolic abnormalities were identified in animals (n = 20/22) before TPN was initiated, including lipemia (n = 12/22), hyperglycemia (11/22), and hypokalemia (n = 11/22). Median age was significantly lower for site 1 cases (0.1 years; range, 0.01–11.0) compared with those from site 2 (4.9 years; range, 0.1–13.7; $P = .03$). Animals at site 2 also had a longer duration of hospitalization ($P = .01$) and TPN administration ($P = .004$), as well as higher survival rate ($P < .02$). Twenty-one of 22 alpacas developed at least 1 complication during TPN administration. Metabolic complications were most prevalent (n = 21/22) and included hyperglycemia (n = 8/21), lipemia (n = 7/21), hypokalemia (n = 3/21), and refeeding syndrome (n = 3/21).

Conclusions and Clinical Importance: TPN is a feasible method of nutritional support for alpacas when enteral feeding is not possible. Prospective studies are warranted to determine optimal TPN formulations for alpacas.

Key words: Camelids; Critical illness; Intravenous feeding; Nutritional support.

Nutrition is a key component of optimal treatment for hospitalized patients. However, when animals are ill or injured, they often cannot or will not eat. Enteral feeding is the first choice for nutritional support of these patients, although IV or parenteral nutrition (PN) is indicated in cases of impaired gastrointestinal function. PN can be provided as total parenteral nutrition (TPN) or partial parenteral nutrition (PPN). The authors define TPN as PN that is intended to provide total estimated energy and protein requirements, whereas PPN is intended to supply only partial energy and protein needs.[1]

Although several published studies have evaluated the utility of TPN in dogs, cats, and horses,[1–5] there is little known about its use in camelids. Three case reports or case series have been published on PN (either TPN or PPN) in alpacas.[6–8] In one, an adult alpaca with renal failure received TPN for 3 days and PPN for 10 days.[8] Furthermore, a case series described 20 crias with cryptosporidiosis, of which most received PN.[6] In the third, 31 llamas and alpacas with hypertriglyceridemia were treated with PN.[7] In addition, 2 other individual case reports of TPN in llamas have been published.[9,10] However, different formulations were used in these publications, and none of the reports specifically evaluated the formulation, clinical complications, and biochemical alterations associated with TPN.

Abbreviations:

PN	parenteral nutrition
RER	resting energy requirement
SIRS	systemic inflammatory response syndrome
TPN	total parenteral nutrition

Alpacas have specific nutrient requirements that differ from those of other ruminants because of the unique nature of their gastrointestinal tract and metabolic physiology. Llamas and alpacas consume less dry matter per unit of body weight, digest feed more completely, and have a slower gastrointestinal transit time than do other small ruminants.[11] This forestomach fermentation efficiency allows llamas and alpacas to obtain 3–5% more energy from lower quality forages than do other ruminants.[11]

Alpacas have been shown to produce less insulin and be less sensitive to the anabolic effects of insulin than are other ruminants.[12–14] Decreased insulin production and sensitivity allow hepatic gluconeogenesis to continue unabated.[a,12–14] As a result, alpacas are more prone to hyperglycemia. Alpacas also require more time to clear glucose from the circulation despite a similar number of insulin-producing pancreatic islet cells and the necessary GLUT transporters as compared with other mammals.[15,16]

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In the presence of decreased insulin concentrations and increased peripheral insulin resistance, lipolysis increases circulating concentrations of nonesterified fatty acids and alters normal lipid metabolism. This in turn predisposes camelids to lipemia and increased fat deposition, particularly in the liver and kidney.[15,17] Hypoproteinemia, by decreasing production of insulin and some enzymes necessary for glycolysis, also may contribute to developing hepatic lipidosis.[15,17,18] The feedback between decreased insulin response and lipid metabolism derangements supports the clinical impression that alpacas are predisposed to metabolic complications (eg, hepatic lipidosis) associated with TPN, which may limit the benefit of TPN formulations used for other species.[15,17,19] Therefore, the purpose of this retrospective study was to evaluate and characterize the use of TPN in alpacas. This information may be useful in developing improved TPN formulas for this species.

Materials and Methods

Case Selection

Cases were identified from a pharmacy computer database search of all alpacas that received TPN while hospitalized at the Tufts Cummings School for Veterinary Medicine Hospital for Large Animals (site 1) and the Ohio State University Veterinary Teaching Hospital (site 2) between January 2002 and December 2008. For the purposes of this study, TPN was defined as a combination of amino acids and dextrose, with or without lipids, formulated to meet resting energy requirements (RER). Additionally, the nutritional support log at site 1 was searched to ensure that no eligible cases were overlooked. Animals of all ages were included in the study.

Study Design

The medical record for each patient was reviewed retrospectively and data collected for the duration of hospitalization during which TPN was administered. The following information was assembled using a standardized data collection form: signalment; body weight and body condition score; underlying medical condition(s); duration of hospitalization before, during, and after TPN administration; estimated RER calculated by the formula 70 $\times$ (body weight in kg)$^{0.75}$; specific TPN formulation; catheter type; biochemical data; frequency and type of complications; and outcome (ie, survival to discharge, death, or euthanasia). Body condition scores were assessed on a 5-point scale, where 1 = emaciated, 3 = moderate, and 5 = obese.[20] Alpacas were divided into 2 age groups (juvenile <0.3 years, older >0.3 years) based on the expected timepoint of earliest weaning. Crias under 4 weeks of age were classified as neonatal animals. Survival was defined as discharge from the hospital.

Complications were divided into 3 categories: mechanical, metabolic, and septic**.** Mechanical complications included thrombophlebitis, catheter occlusions, or disconnected lines that interfered with the administration of TPN. Metabolic complications were defined as an abnormal serum concentration of glucose, triglyceride, bilirubin, blood urea nitrogen, sodium, chloride, calcium, or phosphorus after TPN administration in a patient with a measurement that initially was within the reference range of the institution's clinical pathology laboratory. Additionally, patients suspected of having refeeding syndrome and hepatic lipidosis were recorded. Refeeding syndrome was defined by at least two of the following characteristics: hypophosphatemia, hypokalemia, or hypomagnesemia after initiating TPN in an anorexic animal (ie, concentrations below the lower limit for the reference range after TPN administration in a patient with a measurement that initially was within the reference range of the institution's clinical pathology laboratory). Hepatic lipidosis was defined by a histopathologic confirmation of lipidosis on liver biopsy or necropsy. A diagnosis of lipemia was based on visual inspection of the serum, a triglyceride concentration >500 mg/dL, or both.

Septic complications were characterized by a systemic inflammatory response syndrome (SIRS) associated with suspected or proven infection,[21] a known focus of systemic infection (eg, septic arthritis, uveitis), or postmortem confirmation of sepsis. Confirmed sepsis was differentiated from a diagnosis of SIRS on the basis of hematologic and clinical data. More specifically, SIRS was defined by the presence of at least two of the following 5 criteria, one of which had to be an abnormal body temperature or leukocyte count[22]: tachycardia (juveniles $\geq$120 beats per minute [bpm], older animals $\geq$90 bpm); bradycardia (juveniles <70 bpm, older animals <40 bpm); tachypnea (respiratory rate $\geq$40 breaths per minute); body temperature >102.5 or $<99^{\circ}$F; abnormal leukogram ($>10\%$ immature neutrophils, leukocytosis, or leukopenia ($>18,840$, or $<4,160$ WBC/μL) based on the normal reference range reported at the Diagnostic Laboratory of site 1. The definition of SIRS has not been rigorously validated in camelids to date. It is based on established reference ranges of normal vital parameters and hematology in juvenile and adult alpacas, which vary according to source. The current definition therefore remains institution-specific.

TPN was formulated according to standard protocols at the individual hospitals. At site 1, TPN was formulated from 3 separate components: an amino acid solution (to provide between 4 and 6 g/100 kcal protein,[b,c]) plus 50% dextrose (to provide 40–60% of nonprotein calories) and 20% lipid[d] (to provide the other 40–60% of nonprotein calories). The solution was compounded aseptically by a commercially available compounder.[e] B vitamin complex[f] was added to all, and trace metals[g] to some TPN formulations, depending on clinician preference. At site 2, TPN was formulated without lipid. A commercial dextrose/amino acid solution[h] was used and administered to provide 4.25 g/100 kcal protein, plus 100% of nonprotein calories from dextrose. Calcium gluconate,[i] trace metals,[j] magnesium sulfate (as warranted by clinical signs),[k] and B complex vitamins[l] were added aseptically.

Data Analysis

Data were examined graphically and by the Kolmogorov-Smirnov test. Most data were not normally distributed and data are presented as median (range). Chi square analysis or Fisher exact test (if the expected count was <5/cell) was used to compare categorical data between study sites. Independent *t*-tests were employed to compare continuous data between study locations for normally distributed data and Mann-Whitney *U*-tests were used to compare skewed data. Associations between 2 continuous variables were determined using Spearman's correlation tests. Multivariate logistic regression analysis was performed for presence or absence of complications and survival (ie, died or euthanized versus survived to discharge) to determine whether age or study site were confounding variables. All statistical analyses were performed by commercial statistical software.[m] Results were considered statistically significant if $P \leq .05$.

Results

Patient Demographics

Twenty-two alpacas were enrolled in the study (site 1: n = 8 and site 2: n = 14). Site 1 cases were significantly younger (median age, 0.1 years; range, 0.01–11.0 years) than those at site 2 (median age, 4.9 years; range,

0.1–13.7 years; $P = .03$). More specifically, 5/8 (62.5%) alpacas at site 1 were classified as juvenile, whereas 3/14 (21.4%) animals were <0.3 years of age at site 2. Six of 8 patients at site 1 (75%) and 13 of 14 at site 2 (93%) were female ($P = .53$). Initial body weight was significantly lower in animals treated at site 1 (median, 12.2 kg; range, 5.8–75.0 kg) than at site 2 (median, 50.0 kg; range, 9.5–75.5 kg; $P = .03$). No difference was found in body condition scores between the 2 sites (site 1: median, 3; range, 1.5–3; site 2: median, 1.5; range, 1–1.5; $P = .09$) but scores were available for only 11/22 animals. Sixty-four percent (14 of 22; site 1: n = 6; site 2: n = 8) of all cases had gastrointestinal disease as either primary or secondary diagnoses. Of these, 7 were diagnosed with enterocolitis, 7 with parasitic infestation, 3 with septic peritonitis, and 1 with gastritis; some animals had multiple problems. Of the 7 parasitized animals, 4 had 1 identified parasite, and 3 had multiple parasites. Parasites identified included small coccidia, nematodirus, Eimeria macusaniensis, and sarcocystis. One animal was afflicted with nematodirus, coccidia, and sarcocystis, whereas 2 had both nematodirus and Eimeria. Other diagnoses included hepatic disease (n = 2), neoplasia (n = 2), hematologic disease (n = 1), and pharyngeal abnormalities (n = 1).

Most (n = 20) animals had at least 1 pre-existing metabolic abnormality before TPN was initiated; the most common were lipemia (n = 12/22), hypokalemia (n = 11/22), hyperglycemia (n = 11/22), and hypoglycemia (n = 4/22). The only difference between sites in pre-existing metabolic abnormalities was the incidence of lipemia (site 1: n = 1/8; site 2: n = 11/14; $P = .02$). Before starting TPN, 4 animals (site 1: n = 0/8; site 2: n = 4/14; $P = .095$) met the criteria for SIRS and 1 (site 2) had hepatic lipidosis.

TPN Administration

Animals at site 2 had significantly longer hospitalization times ($P = .01$) and longer duration of TPN administration ($P = .004$; Table 1). The number of calories administered on a kilogram basis (ie, kcal/kg/d) also differed between the 2 sites ($P < .001$; Table 1). Juvenile animals received significantly more kcal/kg/d than older animals at both institutions. Percent of the RER administered was not different between study sites. Furthermore, there was no significant difference between sites with respect to time from admittance to the hospital until initiation of TPN (site 1: median, 3.1 days; range, 0.2–8.8 days; site 2: median, 3.1 days; range, 0.4–12.2 days; $P = .86$). Alpacas at both sites had decreased appetite before starting TPN (site 1: median, 2.0 days; range, 1.0–27.0 days; site 2: median, 3.5 days; range, 0.0–12.0 days; $P = .56$). TPN was administered via 1 of 3 brands of dedicated catheter in the jugular vein in 21 of the 22 alpacas.[n,o,p] The remaining animal initially had

Table 1. Characteristics of total parenteral nutrition (TPN) administration at Tufts Cummings School of Veterinary Medicine (site 1; n = 8) and the Ohio State University College of Veterinary Medicine (site 2; n = 14).

	Site 1	Site 2	*P* Value
Duration of hospitalization (days)	5.4 (0.5–25.3)	21.4 (5.6–42.3)	.01
Juvenile	7.5 (2.4–11.6)	21.8 (15.9–30.6)	
Older	2.5 (0.5–25.3)	20.9 (5.6–42.3)	
Duration of lagtime from admit to TPN start (days)	3.1 (0.2–8.8)	3.1 (0.4–12.2)	.86
Juvenile	3.3 (2.1–6.6)	4.0 (2.2–8.8)	
Older	1.3 (0.2–8.8)	3.8 (0.4–12.2)	
Duration of decreased appetite before TPN (days)	2.0 (1.0–27.0)	3.5 (0.0–12.0)	.56
Juvenile	2.0 (1.0–2.0)	2.0 (0.0–3.0)	
Older	9.0 (6.0–27.0)[a]	4.0 (0.0–12.0)	
Duration of TPN (days)	1.5 (0.7–10.7)	8.7 (1.0–18.0)	.004
Juvenile	1.3 (0.9–5.3)	14.6 (7.7–18.0)	
Older	1.7 (0.7–10.7)	8.7 (1.0–18.0)	
Calories (kcal/kg/d)	33.7 (181–50.7)	26.4 (10.2–56.9)	<.001
Juvenile	40.4 (18.1–50.7)	39.2 (29.8–42.5)	
Older	24.8 (23.8–28.6)[a]	25.8 (10.2–56.9)[a]	
Calories (% of RER)	100 (50–100)	99 (40–207)	.47
Juvenile	100 (50–100)	100 (94–107)	
Older	100 (100–100)	99 (40–207)	
Time to reach calories goal (days)	2 (1–3)	2 (1–3)	.93
Juvenile	2 (1–2)	3 (1–3)	
Older	2 (2–3)	2 (1–3)	
Weight change during TPN (kg)	−0.5 (−1.0–0.0)	−0.5 (−10.9–6.8)	.72
Juvenile	0.0 (0.0–0.0)	0.7 (−1.1–1.5)	
Older	NA	−0.9 (−10.9–6.8)	
Insulin administered (number)	0	10	.002
Juvenile	0	3	
Older	0	7	

RER, resting energy requirements; NA, only 1 animal had sufficient data available to calculate change in body weight within this category. Juvenile animals are those <0.3 years whereas older animals are those >0.3 years. Data are presented as median (range).

[a] <0.05 between juvenile and older animals within each site.

TPN administered via a cephalic vein catheter that was later replaced with a jugular catheter.[n,o,p] At both sites, the median number of days taken to reach an animals' goal feeding rate was 2 (range, 1–3 days). Not all animals were administered the full intended goal because of logistical issues or complications (site 1: median, 100% of RER; range, 50–100% of RER; site 2: median 99% of RER; range, 40–207% of RER). There was no significant difference between sites in body weight change during TPN administration (site 1: median, −0.5 kg; range, −1.0 to 0.0 kg; site 2: median, −0.5 kg; range, −10.9 to +6.8 kg; $P = .72$). Most alpacas at both sites (site 1: n = 7/8; site 2: n = 12/14; $P = .91$) also received some form of enteral nutrition during TPN administration, although amounts could not be quantified. Insulin was administered to most (n = 10/14) alpacas at site 2, but to none of the animals at site 1 ($P = .002$). Seven of the animals that received insulin developed hyperglycemia only after initiating TPN whereas 3 had pre-existing hyperglycemia before TPN was initiated. All insulin-treated animals received significantly more dextrose and less lipid in the TPN formula than those that did not receive insulin (both $P = .005$).

Complications and Outcomes of TPN

Twenty-one of 22 animals had at least 1 complication while receiving TPN. Metabolic complications were most frequent (n = 21/22; Table 2), and included hyperglycemia (n = 8/21), lipemia (n = 7/21), hypokalemia (n = 3/21), and refeeding syndrome (n = 3/21). Development of hyperglycemia was neither related to feeding more than RER ($P = .44$) nor associated with the type of TPN formulation used ($P = .16$). Significant differences in the number of mechanical complications between sites (site 1: n = 0/8; site 2: n = 3/14; $P = .16$) were not observed. However, older animals (>0.3 years) were significantly more likely to develop metabolic ($P = .03$) complications at site 2 compared with juvenile alpacas. No factors evaluated in the multivariate analysis were independently associated with the occurrence of clinical complications.

After initiating TPN, 5 animals met the criteria for SIRS (site 1: n = 3/8 [37.5%]; site 2: n = 2/14 [14.3%]; $P = .211$). All 3 affected alpacas at site 1 were <4 weeks of age and did not meet criteria of SIRS before TPN administration. In contrast, affected animals at site 2 were 5.4 and 9.6 years of age, respectively, and were diagnosed with SIRS both before and after initiation of TPN. No cases of TPN-related sepsis were diagnosed at either site according to the a priori definition, although 1 animal was treated with antibiotics after discontinuing TPN because of clinical suspicion of infection.

Nine of 14 animals (64%) survived to discharge at site 2, whereas only 1 of 8 (12.5%) was discharged at site 1 ($P = .02$). Nonsurviving alpacas at site 1 exhibited acute enterocolitis (n = 3), septic peritonitis with adhesions caused by gastrointestinal leakage (n = 2), malignant melanoma (n = 1), and persistent dysphagia with secondary aspiration pneumonia (n = 1). Diagnoses in deceased alpacas at site 2 included gastrointestinal disease (n = 4), chronic wasting (n = 3), pneumonia (n = 2), lymphoma (n = 1), mycoplasma haemolama infection (n = 1), tooth root abscess (n = 1), hepatic dysfunction (n = 1), and renal dysfunction (n = 1). Multivariate survival analysis showed that no factors, other than site, were independently associated with survival in this small study population.

Table 2. Complications and outcomes of total parenteral nutrition (TPN) administration at Tufts Cummings School of Veterinary Medicine (site 1; n = 8) and the Ohio State University College of Veterinary Medicine (site 2; n = 14).

	Site 1	Site 2	*P* Value
Complication class			
Metabolic	7 (88%)	14 (100%)	.18
Juvenile	5	3	
Older	2	11[a]	
Mechanical	0 (0%)	3 (21%)	.16
Juvenile	0	0	
Older	0	3	
TPN-related sepsis[b]	0 (0%)	0 (0%)	NA
Metabolic complications			
Hyperglycemia	1 (13%)	7 (50%)	.16
Juvenile	1	1	
Older	0	6	
Lipemia	3 (38%)	4 (29%)	1.00
Juvenile	2	2	
Older	1	2	
Refeeding syndrome	0 (0%)	3 (21%)	.18
Juvenile	0	0	
Older	0	3	
Hypokalemia	1 (13%)	2 (14%)	.95
Juvenile	1	0	
Older	0	2	
Outcomes			
Survival to discharge	1 (13%)	9 (64%)	.02
Juvenile	1	2	
Older	0	7	
Death	4	1	
Juvenile	4	0	
Older	0[a]	1	
Euthanasia	3	4	
Juvenile	0	1	
Older	3[a]	3	
Body weight change during TPN administration (kg)	−0.5 (1.0–0.0)	−0.5(−10.9–6.8)	.72
Juvenile	0.0 (0.0–0.0)	0.7 (−1.1–1.5)	
Older	NA	−0.9 (−10.9–6.8)	

NA, only 1 animal had sufficient data available to calculate change in body weight within this category.

Juvenile animals are those <0.3 years whereas older animals are those >0.3 years. Data presented as number (%).

[a] <0.05 between juvenile and older animals within each site.

[b] One alpaca was confirmed to be septic as a consequence of intestinal rupture leading to septic peritonitis. Another individual was treated with antibiotics after discontinuing TPN because of a clinical suspicion of infection.

Discussion

Anecdotal information suggests that alpacas are more prone to metabolic complications from TPN than are

other species. This observation may be related to their underlying physiological differences in metabolism of glucose, lipid, and protein that might make the composition of TPN formulations particularly important.[15] At least 1 metabolic complication of TPN occurred in nearly all animals (21/22, 95%) in the current study compared with rates of 34–42% from published studies in dogs and cats receiving PPN[1] and cats receiving TPN, both of which used similar criteria for defining complications.[2] However, because of the comparatively long duration of TPN administration in the current study, the number of complications per TPN day was relatively low.

The current study did not detect a difference in metabolic complications based on calories provided or TPN formulation used, although the power of this small retrospective study raises the possibility of a Type II statistical error in any nonsignificant differences found in the development of complications. For example, the routine use of insulin in alpacas administered TPN at site 2 may have artificially decreased the number of hyperglycemic complications at that institution, despite the higher percentage of dextrose used in the site 2 TPN formulation. Although site 2 documented a significantly higher rate of metabolic complications in older compared with juvenile alpacas, the development of hyperglycemia after TPN administration was unrelated to age. Consistent criteria for use of insulin were not identified at either hospital, but most commonly were related to clinician preference, age of the animal, underlying illness, formulation of the PN solution, rate of TPN administration, and the intent to counteract lipemia[15] in selected patients.

Most observed complications were mild and resolved by administering insulin or adjusting the rate of the TPN infusion. In a few cases, the complications resolved even before a change in treatment was instituted. This observation suggests that there may be a period of biochemical adjustment to administration of TPN. More research on the clinical significance of changes in metabolic parameters during the time period immediately after beginning TPN administration is warranted. The high incidence of metabolic complications also underscores the importance of careful and consistent metabolic monitoring of patients receiving TPN.

In addition to metabolic complications that developed after administration of TPN, most animals (n = 20/22, 91%) had some pre-existing metabolic abnormality before TPN was initiated. The number of pre-existing metabolic abnormalities has not been published in studies of TPN in dogs and cats, and cannot be compared.[2–5] Metabolic derangements in alpacas of the current study could have been caused by the underlying disease processes, endogenous catecholamine release following illness and stress, or related to prolonged anorexia, characteristic of most patients in this series. Furthermore, the observed derangements may be related to the unique nature of metabolism in alpacas. For example, both juvenile and adult alpacas may develop lipemia under conditions of negative energy balance. Furthermore, camelids may be partially insulin resistant.[13] The associated impairment of glucose assimilation thus may predispose camelids to hyperglycemia.[14] These underlying problems may substantially affect the choice of TPN formulation in this species. Therefore, further research is warranted to evaluate the effects of pre-existing metabolic abnormalities on TPN formulation as well as how they influence monitoring of camelids receiving TPN.

Various similarities and differences need to be considered when comparing findings of the current report to those of similar retrospective TPN studies in dogs, cats, and horses.[2–5] For example, the duration of TPN administration in alpacas was longer than in studies of cats, dogs, and horses, which had a median duration of 3.5–5.0 days,[2–5] compared with the current study's longer duration of 7.6 days for the 2 sites combined. Mechanical complications in the current study were relatively low (3/22; 13.6%) compared with complication rates of 22–46% for TPN studies in dogs and cats,[2–5] although they were similar to those in horses.[23–25] The amount of body weight change seen during TPN administration was lower than in studies of cats and dogs, with a median weight loss of 0.5 kg in the current study compared with either no change or an increase of up to 3–4% in other studies.[2–5] In 1 study of foals, 40% of foals gained weight while receiving PN.[23] Our usual goal is maintenance of current weight during TPN, and we recommend increasing energy intake from TPN or from concurrent enteral feeding when this goal is not being achieved.

Gastrointestinal disease was the primary problem in alpacas at both sites, which is similar to that seen in studies of TPN in cats, dogs, and horses[2–5,23–25] as well as in case reports and case series of llamas and alpacas that received PN.[6,7,9,10] However, the study populations at the 2 sites were markedly different, which may have limited our ability to detect differences between the 2 TPN formulas. At site 2, the patients were older and tended to have more chronic disease than the patients at site 1, which were most commonly young alpacas with acute systemic illness. This difference also resulted in the alpacas at site 2 receiving TPN for a longer duration, and, possibly, improved survival compared with site 1. Four of the 7 nonsurviving alpacas at site 1 experienced a gastrointestinal leak (n = 2), neoplasia (n = 1), or persistent dysphagia (leading to euthanasia), which were considered to progress unrelated to parenteral nutritional support. The remaining 3/7 nonsurviving alpacas were neonatal crias that had acute, severe enterocolitis. In contrast, 4/5 nonsurviving alpacas at site 2 were adults ($\geq$4 years). Three of the 5 animals suffered from chronic wasting conditions, 1 had lymphoma, and 1 developed gastritis, aspiration pneumonia, and mycoplasma haemolama infection. The diversity and complexity of these cases precluded a clinically relevant assessment of risk factors of patient mortality in this small study.

Three of all 5 nonsurviving neonatal crias at site 1 ($<$4 weeks of age) developed SIRS after initiation of PN. These data could not be adequately compared with findings at site 2, because the latter institution only administered TPN in 1 neonatal cria. The relevance of this finding and its potential influence on morbidity and mortality thus remain speculative. A study of human ICU patients previously documented that SIRS and PN

were independently associated with patient mortality after control for admission conditions, severity of illness scores, and interventions.[26] Although PN prevents progressive malnutrition, a lack of enteral nutrition during TPN may lead to mucosal immunity impairment of the intestinal tract and associated increases in intestinal permeability.[27] In this context, neonatal patients may be at greater risk of developing secondary SIRS or sepsis. Nonetheless, PN is a fundamental part of human neonatal intensive care and future studies are necessary to investigate the impact of TPN on systemic inflammation in neonatal crias.

There are a number of other important limitations to this study. The retrospective design of the study prevented access to all data on all patients. There was wide variability in frequency and timing of blood parameter monitoring after initiating TPN, which hindered comparison of data on complications. In addition, some animals received more and some $<100\%$ of their initial calorie goal, resulting in a wide range of calories received as a percentage of RER. Although this was not statistically associated with complication rate or survival, it still may have influenced results. Prospective studies are warranted to determine optimal TPN formulations for alpacas. Nonetheless, this study suggests that TPN is a feasible method of nutritional support in alpacas when enteral nutrition is not possible.

Footnotes

[a] Huaman J, Villavicencio M, Guerra R, et al. Effects of insulin and hydrocortisone on the activity of glycolytic and gluconeogenic enzymes of the alpaca liver. Fed Proc 1975; 34: 659 (abstract)
[b] Travasol with electolytes, Baxter Healthcare Corporation, Deerfield, IL
[c] Travasol without electrolyes, Baxter Healthcare Corporation
[d] Intralipid, Baxter Healthcare Corporation
[e] Automix 3+3 compounder, Baxter Healthcare Corporation
[f] B vitamin complex, Veterinary Laboratories, Lenexa, KS
[g] 4 Trace elements, Abbott Laboratories, North Chicago, IL
[h] Clinimix E 4.25/25, Baxter Healthcare Corp, Clintec Nutrition Division, Deerfield, IL
[i] Calcium Gluconate 10% injection (10 mL), APP Pharmaceuticals LLC, Schaumburg, IL
[j] Multitrace-4 (Trace Elements Injection 4, USP), American Regent Inc, Shirley, NY
[k] Magnesium Sulfate Injection, USP (50%), American Regent Inc
[l] Vitamin B complex injection, Vedco Inc, St Joseph, MO
[m] Systat 11.0, SPSS, Chicago, IL
[n] MILACATH polyurethane extended use catheter, MILA, Erlanger, KY
[o] MILACATH polyurethane longterm catheter (guidewire style), MILA
[p] Angiocath Teflon intravenous catheter, Becton Dickinson, Franklin Lakes, NJ

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References

1. Chan DL, Freeman LM, Labato MA, et al. Retrospective evaluation of partial parenteral nutrition in dogs and cats. J Vet Intern Med 2002;16:440–445.
2. Crabb SE, Freeman LM, Chan DL, et al. Retrospective evaluation of total parenteral nutrition in cats: 40 cases (1991–2003). J Vet Emerg Crit Care 2006;16:S21–S26.
3. Pyle SC, Marks SL, Kass PH. Evaluation of complications and prognostic factors associated with administration of total parenteral nutrition in cats: 75 cases (1994–2001). J Am Vet Med Assoc 2004;225:242–250.
4. Lippert AC, Fulton RB Jr, Parr AM. A retrospective study of the use of total parenteral nutrition in dogs and cats. J Vet Intern Med 1993;7:52–64.
5. Reuter JD, Marks SL, Rogers QR, et al. Use of total parenteral nutrition in dogs: 209 cases (1998–1995). J Vet Emerg Crit Care 1998;8:201–213.
6. Waitt LH, Cebra CK, Firshman AM, et al. Cryptosporidiosis in 20 alpaca crias. J Am Vet Med Assoc 2008;233:294–298.
7. Waitt LH, Cebra CK. Characterization of hypertriglyceridemia and response to treatment with insulin in llamas and alpacas: 31 cases (1995–2005). J Am Vet Med Assoc 2008;232: 1362–1367.
8. Grosche A, Hoops M, Witek T. Acute kidney failure in a male alpaca caused by a severe dehydration. Praktische Tierarzt 2007;88:348–360.
9. Hovda LR, McGuirk SM, Lunn DP. Total parenteral nutrition in a neonatal llama. J Am Vet Med Assoc 1990;196:319–322.
10. Van Saun RJ, Callihan BR, Tornquist SJ. Nutritional support for treatment of hepatic lipidosis in a llama. J Am Vet Med Assoc 2000;217:1531–1535.
11. National Research Council. Providing required nutrients to small ruminants. In: Nutrient Requirements of Small Ruminants: Sheep, Goats, Cervids, and New World Camelids, Whitacre PT, ed. Washington, DC: The National Academies Press; 2007:238–243.
12. Elmahdi B, Sallmann HP, Fuhrmann H, et al. Comparative aspects of glucose tolerance in camels, sheep, and ponies. Comp Biochem Physiol A Physiol 1997;118:147–151.
13. Cebra CK, McKane SA, Tornquist SJ. Effects of exogenous insulin on glucose tolerance in alpacas. Am J Vet Res 2001;62: 1544–1547.
14. Cebra CK, Tornquist SJ, Van Saun RJ, et al. Glucose tolerance testing in llamas and alpacas. Am J Vet Res 2001;62:682–686.
15. Cebra CK. Disorders of carbohydrate or lipid metabolism in camelids. Vet Clin North Am Food Anim Pract 2009;25:339–352.
16. Araya AV, Atwater I, Navia MA, et al. Evaluation of insulin resistance in two kinds of South American camelids: Llamas and alpacas. Comp Med 2000;50:490–494.
17. Anderson DE, Constable PD, Yvorchuk KE, et al. Hyperlipemia and ketonuria in an alpaca and a llama. J Vet Intern Med 1994;8:207–211.
18. Cebra CK, Bildfell RJ, Fischer KA. Microanatomic features of pancreatic islets and immunolocalization of glucose transporters in tissues of llamas and alpacas. Am J Vet Res 2006;67:524–528.
19. Tornquist SJ, Van Saun RJ, Smith BB, et al. Hepatic lipidosis in llamas and alpacas: 31 cases (1991–1997). J Am Vet Med Assoc 1999;214:1368–1372.
20. Van Saun RJ. Nutritional requirements and assessing nutritional status in camelids. Vet Clin North Am Food Anim Pract 2009;25:265–279.
21. Levy MM, Fink MP, Marshall JC, et al. 2001 SCCM/ESICM/ACCP/ATS/SIS international sepsis definitions conference. Intensive Care Med 2003;29:530–538.
22. Goldstein B, Giroir B, Randolph A. International pediatric sepsis consensus conference: Definitions for sepsis and organ dysfunction in pediatrics. Pediatr Crit Care Med 2005;6:2–8.

23. Myers CJ, Magdesian KG, Kass PH, et al. Parenteral nutrition in neonatal foals: Clinical description, complications and outcome in 53 foals (1995–2005). Vet J 2009;181:137–144.

24. Krause JB, McKenzie HC. Parenteral nutrition in foals: A retrospective study of 45 cases (2000–2004). Equine Vet J 2007;39: 74–78.

25. Lopes MA, White NA. Parenteral nutrition for horses with gastrointestinal disease: A retrospective study of 79 cases. Equine Vet J 2002;34:250–257.

26. Chen YC, Lin SF, Liu CJ, et al. Risk factors for ICU mortality in critically ill patients. J Formos Med Assoc 2001;100: 656–661.

27. Ikezawa F, Fukatsu K, Moriya T, et al. Reversal of parenteral nutrition-induced gut mucosal immunity impairment with small amounts of a complex enteral diet. J Trauma 2008;65:360–365; discussion 366.