Plasma carnitine levels in patients receiving home parenteral nutrition[1,2]

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ABSTRACT Patients on long-term home parenteral nutrition (HPN) are known to frequently develop hepatic steatosis or steatohepatitis. The etiology of this steatosis or steatohepatitis is unknown, but carnitine deficiency has been one of the postulated mechanisms. The importance of L-carnitine in hepatic fatty acid oxidation and the steatosis observed in primary and acquired carnitine deficiencies prompted us to determine plasma carnitine levels in 37 patients receiving long-term HPN. Thirteen patients (35%) had low total and free plasma carnitine levels. Fifteen of the 37 HPN patients were matched for age and sex with 15 patients with Crohn's disease who did not require HPN. Mean total and free plasma carnitine values were significantly lower ($p < 0.001$) in these 15 HPN patients (32.2 ± 11.9 and 28.4 ± 10.8) when compared to Crohn's patients not requiring HPN (49.1 ± 10.9 and 46.4 ± 11.5). Associations were not detected between plasma carnitine and clinical or biochemical parameters that might have explained the low values. *Am J Clin Nutr* 1986;43:85–91.

KEY WORDS Carnitine, home parenteral nutrition, Crohn's disease, short bowel syndrome, steatosis, steatohepatitis

Introduction

Carnitine (B-hydroxy-Y-trimethylaminobutyric acid) is synthesized from lysine and methionine in the liver and kidney. The L-form of this compound is biologically active in transporting long-chain fatty acids from sites of activation in the cytoplasm to sites of B oxidation in the mitochondria of animal tissues (1).

In humans, primary carnitine deficiency syndromes include systemic (SCD) and myopathic (MCD) forms. SCD presents in early life with a clinical picture of acute encephalopathy resembling Reye's syndrome (2, 3). Attacks are often precipitated by intercurrent illness associated with caloric deprivation and are characterized by hypoglycemia, hypoprothrombinemia, hyperammonemia, elevated concentrations of serum liver enzymes, and hepatic steatosis. Hepatic and muscle carnitine contents are low while serum values are variable (2, 3). MCD is characterized by muscle weakness, low skeletal-muscle carnitine content, and excess lipids in skeletal muscles (3, 4).

Acquired carnitine deficiency has been described in cachectic cirrhotic patients (5), newborn infants receiving total parenteral nutrition (TPN) (6), and an adult receiving long-term TPN (7). Combinations of inadequate oral dietary intake, absence of L-carnitine in TPN solutions, and decreased hepatic synthesis probably contributed to carnitine deficiency in these situations.

We previously reported (8) persistent abnormalities in conventional liver tests that prompted one or more liver biopsies in 9 of 60 (15%) of our adult home parenteral nutrition (HPN) patients. Of the nine patients, steatohepatitis (eg fat + mononuclear cell inflammation) was present in eight, cholestasis in three, and fibrosis in three. Clinically, three patients had progressive jaundice and one died from hepatic encephalopathy. The etiology of

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the steatohepatitis and cholestasis in patients on long-term HPN is unknown. Carnitine deficiency is one of the frequently postulated mechanisms that has been implicated in HPN-associated steatohepatitis; however, this hypothesis has never been tested.

The importance of L-carnitine in hepatic fatty acid oxidation (9) and the steatosis observed in primary and acquired carnitine deficiency prompted us to determine plasma carnitine levels in our patients receiving long-term HPN, who frequently develop hepatic steatosis or steatohepatitis (or both).

Materials and methods

Seventy-one patients were treated with HPN between October, 1975 and June, 1984 for a total of 2428 patient-months. Fifty-nine percent (42/71) of these patients are currently receiving HPN. In the past 9 mo, 37 HPN patients returned for examination and had plasma collected for carnitine determination. All 37 patients had received HPN for at least 1 mo before carnitine determinations. Five of these patients had a percutaneous liver biopsy for histology and quantitative carnitine analysis.

Demographic data of the 37 patients are shown in **Table 1.** The 20 males and 17 females ranged in age from 9 to 71. Indications for HPN were short bowel syndrome (n = 26), hypomotility (n = 6), chronic adhesive obstruction (n = 4), and enterocutaneous fistulae (n = 1). Lengths of remaining small bowel were: all (n = 10) 3–6 feet (n = 14), 0–3 feet (n = 12), and none in one patient. Duration of HPN in months ranged from 1 to 87 (mean 32 mo). Eighty-seven percent (32 patients) received HPN 5 or more nights/wk, 8% (3 patients) 4 nights/wk, and 5% (2 patients) only 2 nights/wk. There was no statistically significant difference in sex distribution with respect to primary disease, indication for HPN, length of remaining small bowel, and nights of HPN infusion/wk.

Blood for L-carnitine analysis was drawn at approximately 8 AM, before oral intake of food and after the overnight HPN infusion. L-carnitine was measured by a sensitive radioisotope enzymatic assay (10). In this type of assay, free and esterified carnitine concentrations are measured by a combined enzymatic-radioisotopic method. The [^{14}C] acetyl moiety of [*acetyl*-1-^{14}C] acetyl-coenzyme A is transferred to free carnitine by the catalytic action of pigeon breast muscle carnitine acetyltransferase. Unreacted [*acetyl*-1-^{14}C] acetyl-coenzyme A is separated from the product [*acetyl*-1-^{14}C] acetylcarnitine by binding the former to an ion-exchange resin in a small column. The column effluent containing labeled acetylcarnitine is assayed in a liquid-scintillation spectrometer. Carnitine present as fatty acylcarnitine esters is determined by difference following alkaline hydrolysis. The method is applicable to serum, plasma, urine, and tissue specimens. Results are reported as free and total carnitine. The difference of the two values is a measure of short-chain and long-chain fatty acylcarnitine esters.

Total and free carnitine concentrations were determined from stored sera in 15 control patients with Crohn's disease (ie regional enteritis) who did not require HPN and who were participating in another study.

Levels of serum aspartate aminotransferase (AST), alkaline phosphatase (AP), serum bilirubin (SBR), and albumin were determined at periodic intervals during HPN. After a patient's first dismissal from the hospital after HPN, liver tests were done monthly for at least the first 3 mo. Thereafter, liver tests were done every 3 mo unless the presence of abnormalities made shorter intervals desirable.

Details of our HPN program have been previously described (11). HPN formulas are individualized, but in general, patients receive cyclic overnight infusions of approximately 2000 kcal and 85 g of amino acids for an average nonprotein kcal-to-nitrogen ratio of 150:1. Each patient receives approximately 4.9 g of L-methionine and 4.9 g of L-lysine daily. A minimum of 0.8 g of protein/kg of body weight is administered each day to all patients. Most receive between 1.0 and 1.5 g/kg body weight/day. All patients receive at least two 500-cc bottles (550 kcal/bottle) of 10% fat emulsion each week and a few patients receive 20% or more of their daily calories as intravenous fat. Electrolytes, minerals, vitamins, and trace elements are given in amounts to maintain normal blood concentrations. HPN infusion time varied from 8 to 16 h/day. Patients were encouraged to eat, but voluntary refusal of oral food was common and was related to anorexia and fear of worsening of diarrhea and pain.

Seven-day diet records were obtained on 22 HPN patients. Each diet record was reviewed by registered dietitians (JN and SR) for determination of average daily oral energy (kcal) and protein (g) intakes. Mean intakes were calculated from only those days reflecting typical eating patterns. Calorie and protein values were derived using standard composition tables (12, 13).

Comparisons between means were done with analysis of variance or *t* tests if only two groups were tested. Rank sum tests were used to compare groups if the distributions were non-Gaussian. The association between continuous variables was estimated with Pearson's correlation coefficient or with Spearman's rank correlation when the distributions were non-Gaussian.

The following parameters on HPN patients were prospectively collected and later retrieved by a Clinfo Computer System: age, sex, weight (kg), percent ideal body weight, primary disease, indication for HPN, length of remaining small bowel, months on HPN, nights of HPN infusion/wk, total kcal/day, oral kcal/day, intravenous kcal/day, total protein g/day, oral protein g/day, intravenous (iv) protein g/day, conventional liver tests (AST, AP, and SBR), and serum albumin values.

Results

Thirteen patients (35%) had low total and free plasma carnitine levels (patients 1–13, **Table 2**). Liver biopsies were performed in five patients, including 3 of these 13 patients. Hepatic carnitine was low in only one of the five patients (Table 2). Four of the five liver biopsies contained either grade 1 (n = 3) or no fat (n = 1). The steatosis was characterized by both macrovesicular and microvesicular fat.

TABLE 1
Demographic and clinical data on HPN patients

Patient	Age	Sex	Weight	% Ideal body weight (IBW)	Primary disease	Indication for HPN	Length of remaining sm bowel	Months on HPN	Nights infused per week
	yrs		*kg*				*ft*		
1	57	M	78.0	110	Crohn's	Short bowel	3–6	24	7
2	41	F	58.2	105	Crohn's	Short bowel	0–3	80	5
3	32	M	77.3	110	Crohn's	Diffuse small bowel disease	All	21	6
4	59	M	75.0	111	Scleroderma	Hypomotility	All	18	7
5	40	M	64.8	100	Crohn's	Short bowel	3–6	56	7
6	19	M	55.5	82	Idiopathic intestinal pseudo-obstruction	Hypomotility	All	53	7
7	53	F	51.8	95	Ischemic bowel	Short bowel	0–3	58	7
8	34	F	54.0	91	Gardner's syndrome	Short bowel	0–3	48	7
9	52	M	77.2	102	Chronic ulcerative colitis	Enterocutaneous fistulae	All	7	7
10	57	F	54.0	98	Multiple postoperative enteric fistulae	Short bowel	3–6	21	5
11	16	M	62.0	90	Intestinal lymphangiectasia	Malabsorption	3–6	13	7
12	62	F	59.0	93	Radiation enteritis	Chronic obstruction, Short bowel	3–6	35	6
13	57	M	71.3	102	Lymphoma	Short bowel	3–6	26	7
14	36	F	46.2	84	Ischemic bowel	Short bowel	None	42	7
15	9	M	35.2	120	Idiopathic intestinal pseudo-obstruction	Hypomotility	All	20	7
16	71	M	60.7	103	Volvulus	Short bowel	0–3	70	4
17	53	F	55.9	94	Crohn's	Short bowel	0–3	34	7
18	33	M	67.2	100	Crohn's	Short bowel	3–6	31	7
19	32	F	41.83	115	Crohn's	Short bowel	3–6	43	7
20	55	M	75.0	115	Ischemic bowel	Short bowel	3–6	14	2
21	61	F	42.5	72	Scleroderma	Hypomotility	3–6	1	7
22	67	F	55.6	91	Radiation enteritis	Short bowel	All	5	7
23	33	M	56.8	83	Crohn's	Short bowel	0–3	80	6
24	51	M	53.6	83	Ischemic bowel	Short bowel	0–3	24	7
25	43	F	50.0	84	Crohn's	Short bowel	3–6	87	7
26	52	F	49.0	89	Volvulus	Short bowel	0–3	45	6
27	52	M	60.7	90	Radiation enteritis	Obstruction	All	1	7
28	27	F	58.2	93	Gardner's syndrome	Hypomotility	All	24	7
29	35	M	79.5	116	Massive abdominal trauma	Short bowel	3–6	24	2
30	61	F	54.5	91	Crohn's	Short bowel	0–3	85	7
31	43	M	77.0	103	Cancer	Chronic adhesive obstruction	All	1	7
32	39	F	57.7	95	Ischemic bowel	Short bowel	3–6	42	4
33	55	F	60.0	96	Ischemic bowel	Short bowel	0–3	38	5
34	71	M	60.4	95	Idiopathic intestinal pseudo-obstruction	Hypomotility	All	4	4
35	68	F	56.5	103	Chronic adhesive obstruction	Short bowel	0–3	22	7
36	44	M	61.8	92	Crohn's	Short bowel	0–3	7	7
37	47	M	73.3	113	Wegener's granulomatosis	Short bowel	3–6	4	7

TABLE 2
Nutrient intake, liver enzymes, and carnitine values on HPN patients

Patients	Average oral kcal/day	Average iv kcal/day	Average oral protein (g)/day	Average iv protein (g)/day	AP (76–239 U/L, normal)	AST U/L (<31 U/L, normal)	Total SBR (≤1.1 mg/dl, normal)	Albumin (3.1–4.3 g/dl, normal)	Free plasma carnitine Male 28–69 nmol/ml Female 19–60 nmol/ml	Total plasma carnitine Male 37–89 nmol/ml Female 30–73 nmol/ml	Hepatic carnitine (free/total) nmol/ml nonprotein collagen Free 3–14 nmol/ml Total 5–20 nmol/ml
1	1800	1403	122	85	189	17	1.5	2.67	23.2	24.6	
2	1647	1725	50	70	478	28	0.3	3.26	17.0	18.4	
3	1346	862	57	85	117	21	0.2	2.40	22.4	25.1	
4		1709		85	146	42	0.6	3.27	25.8	28.0	
5		1725		55	308	49	0.4	3.14	27.3	27.9	
6		2275		85	357	141	0.8	3.52	25.8	26.9	5.48/6.64
7	2504	1018	111	85	205	32	0.2	3.60	13.6	16.0	
8	771	2039	30	85	353	97	1.2	3.36	10.5	13.5	
9		2658		127	617	45	1.3	3.11	24.1	25.9	
10	2330	690	63	85	1183	75	1.0	3.37	17.7	21.2	
11		3137	31	127	320	22	0.4	1.81	23.4	26.3	
12	395	1725	11	85	338	53	0.6	2.98	12.5	13.6	2.58/3.34
13		1754		85	622	303	15.5	2.01	20.1	25.0	7.40/8.13
14	1011	1882	11	85	391	231	4.5	2.94	22.9	27.3	3.41/6.00
15	90	2172	5	85	558	38	0.7	3.77	32.7	35.8	3.98/4.91
16	2220	1882	102	55	203	32	0.7	3.46	31.4	31.6	
17		862		85	217	25	0.5	3.17	22.4	32.4	
18		1725		85	268	40	0.8	3.61	50.5	52.2	
19	2365	596	87	43	419	45	0.3	3.17	19.9	24.0	
20	2899	1293	100	64	193	32	1.1	3.54	35.3	38.7	
21		2275		64	88	22	0.3	2.80	20.1	23.6	
22		1440		85	313	99	0.7	2.77	22.5	24.7	
23		1114		43	243	24	0.8	4.24	69.9	74.5	
24		2275		127	213	26	0.6	3.52	32.0	46.0	
25	2278	1035	83	85	485	25	0.3	3.05	27.2	28.4	
26		1207		85	291	41	0.8	3.70	23.4	29.6	
27	854	1443	15	85	166	38	0.6	2.79	44.3	47.9	
28	160	862	1	63	135	30	0.5	3.89	20.0	25.6	
29		690		85	280	20	0.5	3.32	32.0	42.3	
30		345		55	185	20	0.7	3.47	22.6	31.9	
31		2361		85	171	18	0.2	2.45	38.2	44.0	
32	1230	1725	66	100	363	37	0.4	3.64	19.5	23.8	
33	1716	862	66	85	121	24	0.5	3.88	27.1	35.0	
34	1086	2275	32	85	151	33	0.6	2.62	36.2	40.3	
35	800	690	23	85	345	21	0.3	3.24	22.2	28.2	
36	3450	1314	132	85	317	20	0.3	3.70	65.4	69.5	
37	2349	1204	164	85	122	27	0.9	3.14	43.3	50.6	

Grade 2 steatosis in one patient was associated with the lowest hepatic carnitine.

Fifteen of the 37 HPN patients were closely matched for age and sex with 15 patients with Crohn's disease who did not require HPN. Mean total and free plasma carnitine concentrations were significantly lower ($p < 0.001$) in these 15 HPN patients (32.2 ± 11.9 and 28.4 ± 10.8, respectively) when compared to Crohn's patients not requiring HPN (49.1 ± 10.9 and 46.4 ± 11.5, respectively) (**Table 3**).

Table 2 details the carnitine concentrations, conventional liver tests, and food intake data for all HPN patients. AP values ranged from 88 to 1183 U/L (normal, 75–220 U/L); mean 310 U/L. Only 6 of 37 patients (16%) had values greater than twice the upper limits of normal. AST values ranged from 17 to 303 U/L (normal < 31 U/L); mean 50 U/L. Six patients (16%) had AST values greater than twice the upper limit of normal. Total serum bilirubin values ranged from 0.2 to 15.5 mg/dl. All but two patients had total SBR values less than twice the upper limits of normal.

No significant associations were found between plasma carnitine concentrations and age, primary disease, HPN indication, length of remaining small bowel, months on HPN, nights of HPN infusion/wk, conventional liver tests (AP, AST, and SBR), and serum albumin.

We examined the relationship between plasma carnitine and nutrient intakes. Specifically, we failed to find significant correlations between plasma carnitine levels and average total kcal/day, oral kcal/day, kcal given intravenously each day, total daily protein, protein intakes by mouth each day, and intravenous protein daily. Similarly, no significant relationships were detected when plasma carnitine levels were analyzed against these nutrient intakes when the intakes were expressed per kg of body weight.

When only those patients with low total and free carnitine levels were examined in the same fashion with respect to nutrient intakes we again failed to find significant correlations between carnitine values and age, primary disease, HPN indication, length of remaining small bowel, months on HPN, nights of HPN infusion/wk, conventional liver tests, and albumin.

Discussion

Thirty-five percent of adult patients who received HPN for an average of 32 months had low free and total plasma carnitine concentrations. The carnitine values in 15 HPN patients were significantly lower than 15 age- and sex-matched patients with Crohn's disease who did not require HPN. Associations were not detected between carnitine levels and clinical or biochemical parameters that might have

TABLE 3
Free and total plasma carnitine values in patients on HPN and patients with Crohn's disease not receiving HPN

	HPN (n = 15) $\bar{x} \pm SD$	Patients with Crohn's not on HPN (n = 15) $\bar{x} \pm SD$	p value
Free plasma carnitine $\frac{nmol}{ml}$	28.4 ± 10.8	46.4 ± 11.5	<.001
Total plasma carnitine $\frac{nmol}{ml}$	32.2 ± 11.9	49.1 ± 10.9	<.001
		Mean ± SD	Range
Normal values*: Free plasma carnitine $\frac{nmol}{ml}$			
Male		46.8 ± 10	28–69
Female		40.1 ± 9.5	19–60
Total plasma carnitine $\frac{nmol}{ml}$			
Male		59.3 ± 11.9	37–89
Female		51.5 ± 11.6	30–73

* Normal values derived from 85 normal controls.

explained the low values by a lack of substrate availability or defects in synthesis or absorption. Patients received considerable amounts of the amino acid precursors of L-carnitine—lysine and methionine. It is unlikely that impaired hepatic synthesis caused the low carnitine levels since there were no correlations with the liver enzyme values. We did not measure urinary carnitine values, so we cannot exclude excessive urinary losses as a causative factor. However, 24-h urinary carnitine excretions in three similar HPN patients were low (7, 14). Protein-calorie malnourished patients who were receiving fat-free TPN (adequate supply of lysine and methionine)for 8–45 days also had low urinary excretion of carnitine except during operative stress or infection (or both) where it increased two- to sevenfold that of normal values (15).

Low total plasma carnitine levels developed within 2 wk in two adult patients receiving carnitine-free total parenteral nutrition (TPN) (16). Plasma carnitine decreased by an average of 33% between the 15th and 40th days without enteral nutrition in 10 adult surgical patients who received continuous lipid-containing, carnitine-free TPN (17).

There are a limited number of reported carnitine values in patients on long-term HPN. Kuettel and colleagues (18) detected low mean values for total plasma and red blood cell carnitine in a group of 20 clinically stable patients receiving HPN for 3 mo to 9 yr. Worthley et al (14) reported decreased levels of plasma and urinary carnitine in two patients requiring HPN for 34 and 39 mo. In another report (7), Worthley and colleagues described an adult patient receiving TPN who developed symptoms and signs that were attributed to L-carnitine deficiency. Hyperbilirubinemia, muscle weakness, and hypoglycemia developed in an adult patient who received cyclic TPN for 1 yr. His free and total carnitine levels were reduced to one-half the normal values and the urinary-free carnitine was markedly diminished. Intravenous administration of L-carnitine at 400 mg/day for 7 days followed by a maintenance dose of 60 mg/day corrected the plasma carnitine and was associated with a return to normal of bilirubin values, a subjective improvement of muscle strength, and absence of reactive hypoglycemia. We have not observed a similar constellation of symptoms in any of our HPN patients, and although some have had weakness or hyperbilirubinemia (or both), multiple factors other than carnitine deficiency were potential causes of these abnormalities.

We did not have enough liver biopsies for histology and carnitine analysis to make meaningful observations about a possible correlation between steatosis and carnitine concentrations. Sachan et al (19) recently reported that alcohol-induced hepatic steatosis in rats could be significantly decreased by supplementing the ethanol diet with 1% DL-carnitine, 0.5% L-lysine, and 0.2% L-methionine. The ability to decrease hepatic lipid content was greater for carnitine than for its amino acid precursors.

The lack of symptoms that have been reported with carnitine deficiency and the absence of an association between plasma carnitine and serum liver enzymes in our patients do not exclude carnitine deficiency as a causal factor in HPN-associated steatosis or steatohepatitis. If a causal relationship exists between low tissue-carnitine concentrations and hepatic steatosis or steatohepatitis in patients receiving HPN, carnitine supplementation should decrease the steatosis concomitant with enhanced oxidation of long-chain fatty acids.

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