ELSEVIER

Nutrition 24 (2008) 103–107

NUTRITION

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International ward rounds

Better living through chemistry, constant monitoring, and prompt interventions: 26 years on home parenteral nutrition without major complications

Maria Luisa Forchielli, M.D.[a,*], Denise Richardson, R.N.[b], Judah Folkman, M.D.[d], Kathleen Gura, Pharm.D.[c], and Clifford W. Lo, M.D.[b]

[a] *Department of Pediatrics, S. Orsola-Malpighi, Bologna University, Bologna, Italy*
[b] *Clinical Nutrition Service, Department of Gastroenterology and Nutrition, Children's Hospital, Harvard Medical School, Boston, Massachusetts, USA*
[c] *Department of Pharmacy, Children's Hospital, Harvard Medical School, Boston, Massachusetts, USA*
[d] *Laboratory of Surgical Research, Department of Surgery, Children's Hospital, and Departments of Cell Biology and Surgery, Harvard Medical School, Boston, Massachusetts, USA*

Manuscript received February 25, 2007; accepted October 6, 2007.

Abstract

Objective: We discuss 26 y of home parenteral nutrition (HPN) in an otherwise healthy patient with severe short bowel syndrome demonstrating a decrease in life-threatening complications after various management changes.

Methods: The patient is a 41-y-old male with a midgut volvulus from malrotation who developed short bowel syndrome and has been HPN dependent since the age of 15 y. His surgical and nutritional data were collected retrospectively and prospectively and included nutritional history, anthropometric parameters, laboratory results, activity levels and types, and treatments for complications. His entire HPN course has been prospectively followed.

Results: Since becoming HPN dependent, the patient's energy intake range has been 20–45 kcal · kg^{-1} · d^{-1}, with 0.8–1.6 g of protein · $kg^{-1} \cdot d^{-1}$. He receives HPN with electrolytes, multivitamins, and trace elements nightly and his intravenous fat emulsion ranges from one to seven times per week. Adjustments to magnesium, iron, zinc, selenium, vitamin E, and carnitine are often required. During his first years of HPN, he had six episodes of catheter-related sepsis and two central venous catheter occlusions. The current central venous catheter has been in place for >13 y without infection or replacement. He developed mild osteopenia but has maintained an active lifestyle without fractures. In the second and third decades of HPN, episodes of hepatic dysfunction occurred, with improvement or resolution using various interventions including oral fish oil.

Conclusion: This case illustrates the successful management of a life-long HPN-dependent patient in whom PN complications have been minimized, including a very recent occurrence of parenteral nutrition–associated cholestasis.

Keywords: Nutritional support; Short bowel syndrome; Cholestasis

Introduction

Many technologic advances have occurred since home parenteral nutritional (HPN) support was pioneered in the early 1970s [1]. Thanks to these improvements and a better understanding of short bowel syndrome (SBS) [2–5], patients can now leave the hospital, lead a near-normal lifestyle, and become productive members of society. Experience with long-term HPN in infants began at the Children's Hospital in 1968 [6]. This report illustrates how a patient has been successfully maintained on HPN without interruption for >26 y with few complications, including a late occurrence of parenteral nutrition–associated cholestasis (PNAC) that promptly resolved. This case description follows the hospital's ethical requirements. The patient has given his permission to report his case.

* Corresponding author. Tel.: +39-051-6363870; fax: +39-051-6237876.
E-mail address: luisa.forchielli@unibo.it (M. L. Forchielli).

0899-9007/08/$ – see front matter
doi:10.1016/j.nut.2007.10.001

Case report

The patient is a 41-year-old male with SBS who has been on HPN for 26 y after sustaining a midgut volvulus secondary to intestinal malrotation at age 15 y. The patient was in excellent health until he presented to his local hospital with abdominal pain and vomiting. He was sent home on a liquid diet but returned to the hospital in shock with a rigid abdomen and was operated on emergently. His infarcted bowel was resected from the ligament of Treitz to the midtransverse colon due to superior mesenteric artery compromise with primary anastomosis. Postoperatively, he developed high-grade obstruction at the anastomosis with food refusal. He was then transferred to the Children's Hospital in Boston for postoperative management. After a relatively brief admission, he was sent home on cyclic HPN and oral feedings, which have continued without interruption since then. Initially he was placed on a hypoallergenic formula (Flexical, Mead Johnson, Indianapolis, IN, USA).

Despite being HPN dependent, the patient entered the workforce after graduating from college, is married with two children, is an outdoor surveyor and mountain guide, and continues to be active in sports with only mild fatigue with heavy exertion, although somehow is angry for being unable to completely resume his prior lifestyle.

His energy intake is 20–45 kcal $\cdot$ kg^{-1} $\cdot$ d^{-1} with 0.8–1.6 g of protein $\cdot$ kg^{-1} $\cdot$ d^{-1}. Intravenous fat emulsions, up to 37% of total calories, have been infused 1–7 d/wk and as infrequently as twice per month. Initially, Liposyn II 10% (Hospira Inc., Lake Forest, IL, USA) was piggybacked once to twice weekly into his central venous catheter (CVC), but he developed dysgeusia and, unbeknown to us, skipped lipids. After noting an abnormal triene/tetraene ratio, he received Liposyn II 20% (Hospira). Due to persistent dysgeusia, he started receiving his current fat emulsion, Intralipid 20% (Baxter/Fresenius-Kabi, Clayton, NC, USA), which, unlike Liposyn II (a soybean/safflower combination), consists solely of soybean oils. Afterward, his dysgeusia resolved.

Multivitamins and minerals were added to HPN according to his requirements. During different stages of HPN, he required adjustments such as two to three times the recommended doses of magnesium, iron, selenium, zinc, vitamin E, and carnitine. Due to recurrent low serum bicarbonate levels, oral baking soda was supplemented to offset bicarbonate loss by diarrhea. Oral carbohydrate intake was also monitored to avoid excesses.

During his early years on treatment, total nutrient admixtures were stopped due to catheter occlusions from lipid deposits. Total nutrient admixtures were resumed when the introduction of dual-chamber containers allowed optimal admixture stability of the parenteral formulation.

Throughout his HPN course, his weight has been relatively stable, remaining around his ideal body weight (IBW). His current weight is 3% above his IBW (lean body mass = arm muscle area 85th–95th percentile versus fat mass = triceps skinfold 25th percentile). His adult height is 174 cm.

As our patient's oral feedings improved with time, HPN and rehydration fluids were adjusted accordingly. However, his caloric needs have never been orally met due to poor absorption and chronic diarrhea. His bowel transit time was initially 20 min but has since increased to 90 min. His most recent stool output was 500–1500 mL/d. His duodenum–jejunum length is only 10 cm and extremely dilated. Intestinal-lengthening procedures and intestinal transplantation were discussed but refused by the patient. Meanwhile, supplemental pancreatic enzymes and diphenoxylate (Lomotil) have partially improved stool frequency and volume. His current HPN supplies 3200 mL 7 nights per week, with 1 g of protein $\cdot$ kg^{-1} $\cdot$ d^{-1}, 2000 kcal/d plus 100 mL of lipids twice per week, and daily amounts of selenium, zinc, and carnitine. He receives iron dextran (5 mg) and vitamin K (10 mg) weekly. To counter the effects of hypergastrinemia, cimetidine was initially added but later switched to an oral proton pump inhibitor.

In his early years on HPN, our patient developed osteopenia (vertebral bone density measurement 0.713 = Z score -2.21 g/cm^2). Aluminum toxicity was not the cause. The calcium and phosphorus in his HPN have prompted supplementation of oral calcium. He also experienced six episodes of CVC sepsis (3 of *Staphylococcus epidermidis*, 1 of *Candida lusitaniae*, 1 of an unidentified yeast, and 1 of *Enterobacter agglomerans*), mostly due to his active lifestyle. As an adolescent, he often swam in lakes despite advice to the contrary. *Staphylococcus epidermidis* occurred after the patient began self-mixing his HPN. At one point, he received his HPN solution compounded with amino acid powders contaminated with pyrogens. The catheter had to be removed twice. Two episodes of CVC occlusion due to lipid deposits resolved with ethanol administration. His current CVC, a Hickman 9.6-Fr single-lumen catheter, has been in place for >13 y.

To increase calories on a transient peripheral HPN, our patient drank his HPN solution. This unusual practice has been used in patients with glycogen storage disease type I [7]. Diluting the HPN with a lemon-flavored carbonated beverage, although unpalatable, enabled him to maintain adequate caloric intake and weight for a short time. However, a second attempt, as an alternative to sports drinks while working outdoors, was very irritating to his mouth and esophagus.

In his second and third decades on HPN, he experienced two major episodes of abnormalities in liver function tests (LFTs) unrelated to CVC sepsis. The liver showed fatty infiltration without evidence of biliary abnormalities or cirrhosis by ultrasound or recent magnetic resonance imaging. Hepatitis and other causes of hepatic disease were excluded. The patient has always refused a liver biopsy. Various interventions have been performed or maintained to prevent PNAC including 1) cyclic HPN (over 10–12 h, preferably overnight) with ad libitum per os food assumption; 2) lim-

itation of intravenous carbohydrate (60% of total calories with adjustments according to clinical situations) and fat (1 g/kg infused twice a week); 3) HPN removal of copper and manganese; 4) a trial of intravenous growth hormone (0.1 $mg \cdot kg^{-1} \cdot d^{-1}$), oral glutamine (0.45 $g \cdot kg^{-1} \cdot d^{-1}$), and a diet of complex carbohydrates (60% of total calories) and low fat (20%) [8]; 5) cycles of bacterial overgrowth treatment with oral metronidazole (Flagyl 20 $mg \cdot kg^{-1} \cdot d^{-1}$); 6) alcohol avoidance; 7) carnitine supplementation on HPN (100 mg/d); 8) use of oral ursodeoxycholic acid (Actigall 300 mg three times a day); and recently 9) use of ω-3 fish oil (500 mg as 12 capsules/d). Interventions 5 to 8 were started together; interventions 5 and 8 were discontinued 6 mo later with LFT normalization. Oral metronidazole and ursodeoxycholic acid have been restarted on at least two other occasions of LFT elevations. Recently he developed a direct bilirubin (DB) level of 7.4, a total bilirubin level of 9.1 mg/dL, thrombocytopenia, coagulation impairment, and splenomegaly (normal blood flow direction in the spleen and liver). After decreasing intravenous lipids in favor of ω-3 fish oil, his LFT values declined to a total bilirubin level of 0.8 mg/dL (normal level <1.2 mg/dL), a DB level of 0.6 mg/dL (normal level <0.4 mg/dL), alanine aminotransferase level from 327 to 132 U/L (normal level <30 U/L), and alkaline phosphatase level from 584 to 274 U/L (normal level <120 U/L). The patient has been on ursodeoxycholic acid and ω-3 fish oil for the past 8 mo. As for the other interventions, item 2 has been maintained since first liver impairment, whereas items 3 and 4 were discontinued thereafter. Standard doses (1 mg of copper and 0.5 mg of manganese daily) were added back later. Cyclic HPN has been maintained since the early days after the operation. Consumption of all foods ad libitum has always been encouraged.

Interestingly, his dependence on HPN has left him with a very favorable lipid profile: total cholesterol level 81 mg/dL (normal level <200 mg/dL), high-density lipoprotein level 36 mg/dL, triacylglycerol level 27 mg/dL (normal level <250 mg/dL), and low-density lipoprotein level 28 mg/dL (normal level <160 mg/dL). His recent essential fatty acid profile showed a normal triene:tetraene ratio, an α-linolenic acid level of 33 mmol/L (normal level 50–130 mmol/L), a γ-linolenic acid level of 97 mmol/L (normal level 16–150 mmol/L), a linoleic acid level of 1624 mmol/L (normal level 2270–3850 mmol/L), an arachidonic acid level of 203 mmol/L (normal level 30–250 mmol/L), and an eicosapentaenoic acid level of 119 mmol/L (normal level 14–100 mmol/L).

Expenses were a source of anxiety for our patient and his family. With medical insurance, the costs of HPN were initially high ($220–$680/d). To save money, he aseptically mixed his own solutions ($50/d) after purchasing a laminar airflow hood until lower costs and extended insurance coverage allowed him to rely on home-care companies. He comes regularly to the HPN clinic (two to three times per year) for follow-up on his nutritional intake, anthropometric measurements, laboratory tests (chemistry and complete blood cell count with trace elements, vitamins, and lipid profile occasionally) and maintains telephone contact as necessary or extra visits if complications occur. This has avoided hospital admissions for the past 13 y and limited costs. A summary of the patient's complications is presented in Table 1.

Discussion

The relevance of this case lies in a relatively low occurrence of HPN complications over an exceptionally long HPN course and the development of PNAC after 26 y of HPN with resolution after multiple therapies. This patient required, and became dependent on, HPN due to his severe SBS status with 10 cm of duodenum–jejunum anastomosed to the midtransverse colon after midgut volvulus secondary to malrotation. HPN supplied him with fluid and nutrients that were continuously adjusted especially during the first years after resection. Many studies have reported on patients with long-term HPN, but their time spans of observation were all much shorter. In a European survey by Van Gossum et al. [9] of adult patients on long-term HPN, the

Table 1
Summary of complications of a very long-term HPN course in a patient with short bowel syndrome

Type of complication	First decade on HPN	Second decade on HPN	Third decade on HPN
Nutrition related			
Electrolytes	Major corrections	Minor corrections	Minor corrections
Vitamins	Vitamin E supplementation	Vitamin E supplementation	None
Trace elements	Selenium, iron addition	Selenium, zinc, iron addition	Selenium, zinc, iron addition
Other	None	Carnitine addition	Carnitine addition
CVC related			
Sepsis	6 episodes	None	None
Occlusion	1 episode	1 episode	None
Metabolic			
Osteopenia	Present	Corrected	Corrected
PNAC	Not present	Increase in liver function tests but with normal bilirubin	Present with abnormal direct bilirubin in resolution
No. of hospital admissions	8	1	None

CVC, central venous catheter; HPN, home parenteral nutrition; PNAC, parenteral nutrition–associated cholestasis

average was 7 y. There was a patient on HPN for 24 y, but no details were provided on his medical history [9]. In addition, the cohort was followed for only 1 y compared with the 26 y of HPN of our case. A more recent case report of a patient on HPN for 27 y has described an apparently miserable life, with the patient going through a continuous series of septic episodes, a failed intestinal transplantation, and eventually PNAC during a septic episode followed by death [10]. Similar considerations apply to studies in children. Studies by Colomb et al. [11] and Tung et al. [12], in particular, are retrospective in children with intestinal failure on long-term HPN. In these studies, the patients who received the longest HPN course were on them for approximately 15 and 10 y, respectively, and the patients whose intestinal failure was due to SBS for 15 and 9 y. Neither study mentioned outcomes specifically for these children.

Short bowel syndrome is an intestinal failure secondary to congenital or acquired loss of portions of the small bowel with impaired nutrient digestion and absorption, water and electrolyte losses, and eventual failure to thrive [2–5]. Length and functioning of the remaining gut (including the colon), presence of the ileocecal valve, and patient age at time of the event are the main predictors of recovery and appropriate nutritional interventions. Common causes of SBS include gastrointestinal atresia, abdominal wall defects, necrotizing enterocolitis, midgut volvulus, gastrointestinal ischemia, radiation enteritis, pseudo-obstruction, inflammatory bowel disease, and neoplasia. In patients with SBS secondary to chronic diseases, enteral nutrition is less effective due to residual intestinal functional deficits. Reduced absorptive capacity and decreased transit time initiate and reinforce hypergastrinemia and pancreatic enzyme inactivation and increase motility with osmotic and secretory diarrhea. Macro- and micronutrient malabsorptions depend on the extent and location of gastrointestinal resection, functional status of the remaining intestine, and its adaptive ability. Patients with stable SBS without disease absorb approximately 62% of calorie intake, with higher rates for proteins (81%) than carbohydrates (61%) and fat (54%) [13]. In patients with severe SBS, dietary nitrogen absorption is 21–61% [14]. Bacterial overgrowth, especially without the ileocecal valve, increases local and systemic inflammation and nutrient loss. Approximately 800 and 1100 kcal/d are lost in adults with SBS with and without a colon, respectively, and bile acids, fat, and fat-soluble vitamins in a terminal ileum loss >100 cm [15]. A large load of nutrients reaching the colon may cause 1) metabolic acidosis for carbohydrate conversion by lactobacilli to levorotatory lactic acid (D-lactic acid) with a risk of neurologic impairment [2]; 2) calcium oxalate, with insoluble salts leading to hyperoxaluria and renal stone formation if calcium and fats combine [16]; and 3) an increase in stool-reducing substances from malabsorbed carbohydrates. Fortunately, these carbohydrates can be converted by bacteria into short-chain acids, an important energy source and trophic factor for colonic mucosa in patients with SBS [17].

Our patient's dependency on HPN is consistent with the literature, where HPN tapering is deemed possible in residual bowel lengths of 50–70 cm (110–150 cm if the colon is absent) in adults [18] or 10–20 cm (with or without the ileocecal valve) in infants and children in whom adaptation is more likely. Although an adolescent at the time of resection, intestinal adaptation has since occurred, as demonstrated by the transit time improvement and resumption of minimal enteral absorption, thus limiting electrolyte and trace element corrections in our patient's HPN. This adaptation allows the provision of 20–30 kcal $\cdot$ kg^{-1} $\cdot$ d^{-1} and 0.8–1 g of protein $\cdot$ kg^{-1} $\cdot$ d^{-1}, which is lower than recommended (35–40 kcal $\cdot$ kg^{-1} $\cdot$ d^{-1} based on IBW and 80–100 g of protein/d) [13] but considered appropriate because he maintains his IBW and active lifestyle without energy excesses for his liver. More calories (40–45 kcal $\cdot$ kg^{-1} $\cdot$ d^{-1}, 1.5–1.6 g of protein $\cdot$ kg^{-1} $\cdot$ d^{-1}) were given in the postresection period when losses and requirements (adolescent) were higher. His oral diet has always been an ad libitum diet with suggestions to choose complex carbohydrates over fat especially during liver impairment. This is considered optimal for patients with SBS (especially with jejunostomy), with higher absorption of fluid, calcium, magnesium, zinc, copper, and fat [19]. Lower dietary fat content reduces fat stool excretion and liver impairment. On that diet he has not had sepsis, whereas he had CVC sepsis episodes when on the initial elemental diet, likely due to bacterial translocation through cytokine activation, mucosal damage, and/or bacterial overgrowth [20].

Patients with extreme SBS develop complications from the baseline disease and the need for long-term HPN (e.g., CVC complications, hepatic dysfunction, bone demineralization, and other metabolic alterations) [3,5]. Vigilant monitoring is crucial. Our patient has had some infectious and metabolic-related complications without lasting consequences once promptly faced. CVC sepsis episodes have been reduced to being exceptionally rare since his first years on HPN, when he had problems managing his line. That time corresponds to his adolescence.

His nutritional deficiencies were more consistent with his poor enteral absorption and diarrhea than with his prolonged use of HPN. Since the addition of vitamins and trace elements to HPN, deficiencies have become uncommon even in patients with long-term HPN. Apart from the recent intravenous multivitamin shortage in the United States and scanty reports on vitamin B12 deficiency in addition to choline in patients with long-term HPN [21,22], it is the baseline disease with poor oral intake and diarrhea that exposes patients on long-term HPN to nutrient deficiencies. Trace elements (selenium and zinc in particular) more than vitamins usually require extra supplementations. Among vitamins, fat-dependent ones need intravenous adjustments likely due to fat malabsorption in patients with SBS (e.g., vitamin E in our case). Extra supplementations of trace elements may prove toxic. Similarly, standard intravenous compound mixtures of trace elements usually contain man-

ganese in large quantities with potential accumulation in the brain and liver dysfunction [23,24]. Effects can be reversible with dose adjustment. Copper also can interfere with liver function and needs monitoring.

Hepatic dysfunction first appeared during the second decade of HPN but developed into overt PNAC just a few months ago (DB level >2 mg/dL), without a concomitant triggering event or any forewarning. PNAC, a negative outcome of patients with SBS on HPN [3,5,25], has a multifactorial origin: lack of enteral feeds, infections, toxic substances (e.g., manganese), components of total parenteral nutrition (e.g., carbohydrate or fat excess, methionine content), or its contaminants. PNAC development is inversely related to patient age and positively related to the duration of total parenteral nutrition (typically during the initial phase of HPN). Only two patients developed PNAC after a prolonged HPN course: one after 13 y, probably associated with a parenteral fat intake $>1\ g \cdot kg^{-1} \cdot d^{-1}$ [26], and the other after 27 y [10]. In a study by Cavicchi et al. [26], the median PNAC occurrence was after 17 mo of HPN and the 13-y occurrence was an extreme case. However, the PNAC definition in that study (DB level >3.5 mg/dL) versus the definition in our study (DB level >2 mg/dL) might not make them fully comparable. In a case report by Fairman et al. [10], PNAC seemed a spurious event because it developed in a very ill and septic patient after a failed intestinal transplantation. In our patient, the multiple preventive interventions aimed at liver protection and bowel adaptation delayed a PNAC occurrence to after 26 y on HPN. His recent alcohol misuse may have contributed to PNAC, but the addition of fish oil and ursodeoxycholic acid, associated with a reduction of parenteral fat intake, has normalized his bilirubin. Ursodeoxycholic acid (Actigall) is a well-established therapy for PNAC [27]. A few PNAC cases have recently been resolved with intravenous fish oil [28], but experience using oral supplements is limited.

In conclusion, long-term survival with good quality of life is possible with SBS and intestinal failure. However, constant monitoring is required to prevent HPN complications, among which liver impairment always appears to be around the corner, but also solvable.

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