



## Applied nutritional investigation

## Outcome of kidney function in adults on long-term home parenteral nutrition for chronic intestinal failure



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## ABSTRACT

**Objective:** The aim of this study was to evaluate kidney function outcome in adults on home parenteral nutrition (HPN) for chronic intestinal failure using the newly recommended equations for estimated glomerular filtration rate (eGFR) assessment in clinical practice.

**Methods:** This was an observational study with 72 patients. Clinical and biochemical parameters were collected at initiation of HPN (retrospective baseline [BL]), at inclusion in the study (cross-sectional [CS]), and at the end of a 30-mo prospective follow-up (Fup). The eGFR (mL/min/1.73 m<sup>2</sup> body surface) was calculated by the Chronic Kidney Disease Epidemiology Collaboration creatinine and categorized as normal, mildly decreased (MDKF), and chronic kidney disease (CKD) when  $\geq 90$ , 60 to 89, and  $< 60$ , respectively.

**Results:** An eGFR  $< 90$  was observed in 41.7% of patients at BL, 53.4% at CS, and 56.6% at Fup. A CKD was present in all of the patients at BL, 20.1% at CS, and 35.9% at Fup. The probability of maintaining an eGFR  $\geq 60$  was 98%, 82%, and 79% at 1, 5, and 10 y after BL, respectively (Kaplan-Meier analysis). The probability was lower in patients with MDKF at BL ( $P = 0.039$ ). The development of a CKD was significantly associated with aging and urologic diseases and numerically associated with the episodes of venous-catheter sepsis, short bowel syndrome, and a low volume of HPN.

**Conclusions:** In patients on HPN for chronic intestinal failure, decreased kidney function is a frequent finding, even at HPN commencement, demanding accurate monitoring during the treatment. Prevention of CKD primarily relies on the maintenance of fluid balance and the prevention of catheter-sepsis and urologic diseases.

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## Introduction

Chronic intestinal failure (CIF) is the long-lasting reduction of gut function below the minimum necessary for the absorption of macronutrients or water and electrolytes, such that intravenous supplementation is required to maintain health, growth, or both [1]. Home parenteral nutrition (HPN) is the primary treatment for CIF [1]. Metabolic complications can develop in patients on HPN for CIF. The pathogenesis is multifactorial owing to factors related to CIF, HPN, systemic conditions (i.e., episodes of sepsis), or

lifestyle characteristics (i.e., reduced physical activity, smoking, alcohol abuse) [1].

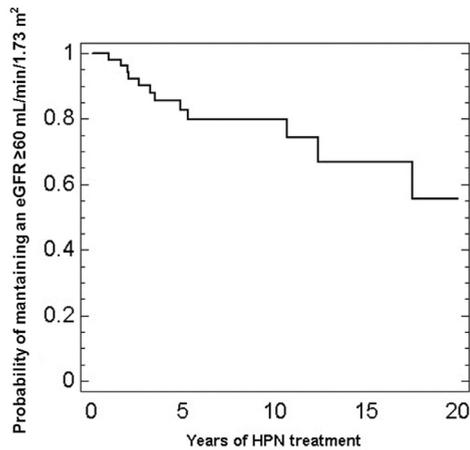
Reduced kidney function and renal stones can develop in patients on long-term HPN for CIF [1]. To date, one prospective retrospective study [2] and two cross-sectional studies [3,4] have evaluated the evolution of the kidney function and the potential risk factors for chronic kidney disease (CKD) in adults with CIF owing to non-malignant (benign) disease. An annual 3.5% decline of creatinine clearance (CrCl) [2] and a reduced kidney function in ~50% of patients [3,4] were observed. Infection rate, dehydration and hypovolemic state, duration of HPN, and patient age have been described as risk factors, but the results were not consistent among the studies [2–4]. In those studies, kidney function was assessed by the measured glomerular filtration rate (GFR).

The guidelines for the evaluation of kidney disease [5] recommend using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine equation to evaluate the estimated GFR (eGFR) in clinical practice in adults:

F.A., G.L.M., and L.P. conceived of and designed the study. F.A., A.S.S., and G.M. were responsible for the generation, collection, assembly, analysis, and interpretation of the data. F.A., M.G., and L.P. drafted the manuscript. C.G. and L.M.G. revised the manuscript. All authors approved the final version of the manuscript. This manuscript was part of F.A.'s PhD thesis for the School of Medicine of the University of Bologna, Italy.

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Years of HPN treatment	0	1	3	5	10	15	20
Patients at risk (n)	54	50	41	29	15	7	5
Probability of maintaining eGFR ≥60 mL/min/1.73 m <sup>2</sup> (%)		98	90	83	80	67	56

**Fig. 1.** Probability (Kaplan–Meier analysis) of maintaining an estimated glomerular filtration rate (eGFR)  $\geq 60$  mL/min/1.73 m<sup>2</sup> on home parenteral nutrition treatment (HPN).

$$\text{eGFR} = 141 \times \min(\text{sCr}/\kappa, 1)^\alpha \times \max(\text{sCr}/\kappa, 1)^{-1.209} \times 0.993^{\text{Age}} \\ \times 1.018 \text{ (if female)} \times 1.159 \text{ (if black)}$$

where: sCr = serum creatinine (mg/dL);  $\kappa = 0.7$  (women) or 0.9 (men);  $\alpha = -0.329$  (women) or  $-0.411$  (men); age in years; min = indicate the minimum value of sCr/ $\kappa$  or 1; max = indicate the maximum of sCr/ $\kappa$  or 1 [6].

The risk for end-stage renal failure owing to frequent and severe episodes of dehydration associated with high intestinal loss of fluids was included in the early devised criteria for intestinal transplantation [7]. Routine monitoring of kidney function is therefore recommended in patients with CIF [1].

The aim of this study was to evaluate the outcome of kidney function in adults on long-term HPN for CIF owing to benign disease using the newly recommended equation for the eGFR assessment in clinical practice.

## Materials and methods

### Study design

This was a cross-sectional, retrospective prospective study on adult patients on HPN for benign CIF cared for at the Center for Chronic Intestinal Failure of the S. Orsola-Malpighi Hospital of the University of Bologna, in Italy. All patients who had a scheduled visit between August and December 2013 were enrolled in the study if they were  $\geq 18$  y of age, had no cancer diagnosis, or were not taking an experimental drug.

Renal function and the clinical characteristics were collected at three times: enrollment in the study (cross-sectional analysis [CS]), initiation of HPN (baseline retrospective analysis [BL]), and the end of a 30-mo follow-up (prospective follow-up analysis [Fup]).

The study was approved by the Local Ethics Committee. Voluntary informed written consent was obtained from all the enrolled patients.

### Collected parameters

At BL, patient age and sex, serum creatinine (sCr) levels, and characteristics of the CIF (pathophysiological mechanism, primary disease) were retrospectively collected from patient records. The sCr concentration was considered a BL value when measured within 1 mo from the date of HPN initiation.

At CS, patient age and body mass index (BMI, kg/m<sup>2</sup>), characteristics of the HPN regimen (duration, weekly frequency of infusion, mean daily volume, amino acid and trace element content of the nutritional admixture), and presence of urologic disease were recorded.

At Fup, patient BMI, characteristics of the HPN regimen, new urologic diseases and number of catheter-related bloodstream infections (CRBSI), chronic (>3 mo) administration of potentially nephrotoxic drugs, and number of computed tomographic (CT) procedures with intravenous administration of iodinated radiocontrast that occurred between CS and Fup were recorded.

Kidney function was assessed by eGFR measurement. The eGFR was calculated by the CKD-EPI creatinine equation [6] or the creatinine-based “Bedside Schwartz” equation for patients <18 y of age at BL [see [https://www.kidney.org/professionals/KDOQI/gfr\\_calculatorPed](https://www.kidney.org/professionals/KDOQI/gfr_calculatorPed)].

Kidney function was categorized as normal (NKF) when eGFR was  $\geq 90$  mL/min/1.73 m<sup>2</sup> body surface, as mildly decreased (MDKF) when eGFR was between 60 and 89 mL/min/1.73 m<sup>2</sup>, and as CKD when eGFR was <60 mL/min/1.73 m<sup>2</sup> for  $\geq 3$  mo [5].

Urologic diseases were categorized as kidney stones, urinary tract obstruction, and recurrent urinary tract infections.

CRBSI was diagnosed by positive paired blood cultures from a peripheral vein and from the central venous catheter (CVC), with differential time to positivity. In the absence of positive cultures, returning to normal body temperature after removal of CVC from a patient with a bloodstream infection was considered indirect evidence of CRBSI [8].

The sCr was analyzed by Jaffe's assay, fully automated and run on Modular E 170 analyzer (Roche Diagnostic International Ltd, Rotkreuz, Switzerland S.P.A.).

### Statistical analysis

Variables are reported as median, range, and percentages. Differences among groups were analyzed by the Mann–Whitney U test and Kruskal–Wallis test. Differences between frequencies were analyzed by the Fisher exact test and the  $\chi^2$  test. The probability of maintaining an eGFR  $\geq 60$  mL/min/1.73 m<sup>2</sup> after starting HPN was calculated using the Kaplan–Meier method, including those patients with an eGFR  $\geq 60$  mL/min/1.73 m<sup>2</sup> at BL. The endpoint was the time of the occurrence of CKD after initiation of HPN. Group comparison was made with the log-rank test.  $P < 0.05$  was considered statistically significant. The Statgraphics centurion XV statistical package 2008 (StatPoint, Inc, Warrenton, VA, USA) was used for the analyses.

## Results

### Patient population

At CS, 31 men and 41 women, age 55.8 y (18.6–82.6 y) were included in the study. Duration of HPN was 76.3 mo (0.1–339.8 mo). The underlying diseases were mesenteric ischemia (16), Crohn's disease (14), chronic intestinal pseudo-obstruction (CIPO; 16), and others (26). The pathophysiological mechanisms of CIF were short bowel syndrome (SBS) with an end jejunostomy (19) and with a colon in continuity (32), intestinal dysmotility (17), small bowel mucosal disease (2), and fistulas (1).

BL analysis was performed in 60 of the 72 CS patients who met the criteria for the eGFR calculation at initiation of HPN.

Fup analysis was performed in 53 of the 72 CS patients who were still on HPN at the end of the 30-mo prospective follow-up. Nineteen patients did not complete follow-up because 4 were weaned from HPN, 4 were lost during follow-up (4), and 11 died. No death was due to kidney failure.

### Kidney function at different phases of HPN treatment

Reduced kidney function, either MDKF or CKD, was observed in 25 patients (41.7%) at BL, 39 (53.4%) at CS, and 30 (56.6%) at Fup. MDKF was present in 19 (31.7%), 24 (33.3%), and 11 patients

(20.8%) at BL, CS, and Fup, respectively. CKD was present in 6 patients (10%) at BL, 15 (20.1%) at CS, and 19 (35.9%) at Fup.

#### Probability of maintaining an eGFR $\geq 60$ after starting HPN

The Kaplan–Meier analysis showed that in the whole group of patients with NKF or MDKF at BL, the probability of maintaining an eGFR  $\geq 60$  mL/min/1.73 m<sup>2</sup> during the first 20 y of HPN treatment was 98%, 90%, 82%, 79%, 66%, and 55% at 1, 3, 5, 10, 15, and 20 y, respectively (Fig. 1). Therefore, the probability decreased by 18% during the first 5 y, 3% between 5 and 10 y, 13% between 10 and 15 y, and 11% in the last 5 y of treatment.

Figure 2 shows the probability of maintaining an eGFR  $\geq 60$  mL/min/1.73 m<sup>2</sup> in the subgroups of patients with either NKF or MDKF at BL. The probability was significantly lower in those with MDKF at initiation of HPN ( $P=0.039$ ). In the MDKF subgroup, the probability was 62% at both 5 and 10 y, whereas in the NKF subgroup it was 93% at 5 y and 89% at 10 y.

#### Factors associated with the kidney function categories at CS

Table 1 shows the clinical and HPN characteristics of the patient cohorts, categorized according to the kidney function at CS. A statistically significant negative association was observed between kidney function and patient age, at both BL and CS, and the presence of urologic diseases. A numerical association was observed between the presence of CKD and SBS as pathophysiological mechanism of CIF. The amino acid content of the HPN admixture significantly decreased from the NKF to the CKD subgroup.

#### Factors associated with the development of CKD during the 30-mo Fup

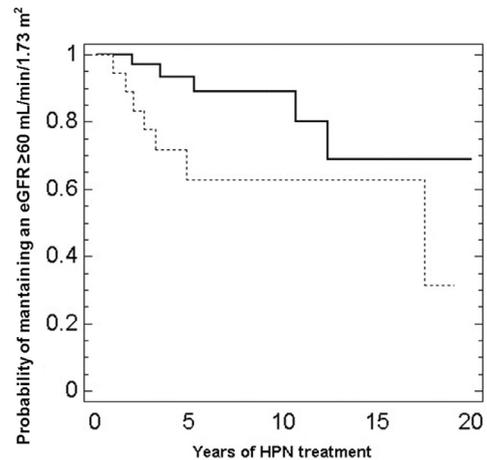
The 42 patients with NKF or MDKF at CS and who completed the Fup on HPN were categorized into two cohorts according to the kidney function evolution between CS and Fup: NKF or MDKF at CS and presence of CKD at Fup ( $n=8$ ), or NKF or MDKF at both CS and Fup ( $n=34$ ).

Table 2 shows the comparison of the clinical and HPN characteristics between the two patient cohorts. The eight patients who developed CKD during Fup showed a statistically significant higher percentage of women and higher frequency of MDKF at CS. A numerically older age at both BL and CS, lower HPN volume at CS, higher frequency of new urologic disease, and higher frequency of CRBSI episodes between CS and Fup also were observed.

## Discussion

The results of the present study gave an updated figure of the outcome of kidney function in patients on long-term HPN for CIF, assessed by the newly recommended equation for eGFR calculation in clinical practice. The frequency of reduced kidney function, either MRKF or CKD, factors associated with its development, and the probability to avoid a CKD during the treatment are reported. Although the patient population was by far larger than those of previous studies in adult patients (the largest one being 40 patients), the main limitation of the present study was the dimension of the patient cohorts, which did not allow for statistically significant differences among most of the assessed variables.

At CS, the prevalence of decreased kidney function, either MRKF or CKD was 53.4%, a result similar to those reported by previous studies, where kidney function was evaluated by measured GFR. Boncompain-Gerard et al. [3] found decreased GFR, assessed by inulin clearance, in 56% of 16 patients with an SBS who were receiving HPN. Lauerjat et al. [4] investigated 40 adult patients, 26



Patients with NKF (eGFR $\geq 90$ mL/min/1.73 m <sup>2</sup> ) at initiation of HPN							
Years of HPN treatment	0	1	3	5	10	15	20
Patients at risk (n)	35	33	28	22	10	5	3
Probability of maintaining eGFR $\geq 60$ mL/min/1.73 m <sup>2</sup> (%)		100	97	94	89	69	69

Patients with MDKF (eGFR between 89 and 60 mL/min/1.73 m <sup>2</sup> ) at initiation of HPN							
Years of HPN treatment	0	1	3	5	10	15	20
Patients at risk (n)	19	17	13	7	5	2	0
Probability of maintaining eGFR $\geq 60$ mL/min/1.73 m <sup>2</sup> (%)		94	78	63	63	63	31

Fig. 2. Probability (Kaplan–Meier analysis) of maintaining an estimated glomerular filtration rate (eGFR)  $\geq 60$  mL/min/1.73 m<sup>2</sup> on home parenteral nutrition (HPN) treatment, in patients with normal kidney function (NKF) and in those with mildly decreased kidney function (MDKF) at starting HPN.

with SBS and 14 with intestinal dysmotility. Also in this study, GFR was measured by the inulin clearance method. A decrease of GFR  $>20\%$  was observed in 52.5% of patients. A retrospective study by Buchman et al. [2] with 33 patients showed that GFR, measured as creatinine clearance, decreased by  $3.5\% \pm 6.3\%$  per year, a rate comparable with the median decreases observed in the present study ( $-2.4$  to  $-7.3\%$ ). Overall, the similarity between the present data and those from previous studies indicate that eGFR can be considered appropriate to evaluate renal function in clinical practice in patients on HPN for CIF.

The observed frequency of CKD at CS was 20.1%. A comparison with previous studies was not possible because they did not report the frequency of CKD. A recent investigation in children with a

**Table 1**

Characteristics of patient cohorts categorized according to the kidney function at inclusion in the study (CS)

	NKF <sup>a</sup> (n = 33)	MDKF <sup>b</sup> n = 24)	CKD <sup>c</sup> (n = 15)	P-value
Women, n (%)	19 (57.6)	14 (58.3)	8 (56.9)	0.949
Age at BL, y	40.1 (0.9–62.2)	44.9 (13.4–72.7)	58.5 (31.3–75)	0.007
Age at CS, y	48.5 (20.1–66.5)	54.4 (18.6–75.1)	66.4 (44.7–82.6)	<0.001
BMI, kg/m <sup>2</sup>	22.1 (17.9–24)	19.9 (10.8–26.4)	20.4 (12.6–31.5)	0.677
Primary disease, n (%)				0.179
– Mesenteric ischemia	7 (21–2)	5 (20.8)	4 (26.7)	
– Crohn's disease	4 (12.2)	5 (20.8)	5 (33.3)	
– CIPO	7 (21.2)	9 (37.5)	1 (6.7)	
– Others	15 (45.4)	5 (20.8)	5 (33.3)	
Cause of intestinal failure, n (%)				0.197
– SBS with end jejunostomy	9 (27.3)	5 (20.8)	5 (33.3)	
– SBS with colon in continuity	14 (42.4)	9 (37.5)	9 (60.0)	
– Motility disorder	7 (21.2)	9 (37.5)	1 (6.7)	
– Mucosal disease	3 (9)	0	0	
– Fistulas	0	1 (4.2)	0	
– SBS	23 (69.7)	14 (58.3)	14 (93.3)	
– Others	10 (30.3)	10 (41.7)	1 (6.7)	0.063
Urologic disease, n (%)– Stones	3 (9.1)	2 (8.3)	5 (33.3)	
– Urinary obstruction	1 (3)	4 (17.7)	4 (26.7)	0.018
– Recurrent urinary infection	3 (6.1)	0	0	
HPN duration, mo	57.8 (0.1–330.4)	42.9 (0.6–339.8)	109.2 (1.5–327.8)	0.223
HPN volume, mL/d	1917 (535–5500)	1285 (428–3500)	1400 (571–3500)	0.696
HPN amino acids, g•kg BW•d <sup>-1</sup>	1.1 (0–2.1)	0.8 (0–1.7)	0.7 (0–1.6)	0.063
Trace metal infusions <sup>d</sup> , n/wk	6 (0–7)	4.5 (1.5–7)	5 (0–7)	0.616

BL, baseline; BMI, body mass index; BW, body weight; CIPO, chronic intestinal pseudo-obstruction; CKD, chronic kidney disease; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; CS, cross-sectional; eGFR, estimated glomerular filtration rate; HPN, home parenteral nutrition; MDKF, mildly decreased kidney function; NKF, normal kidney function; SBS, short bowel syndrome.

Continuous variables data are reported as median (range).

<sup>a</sup>NKF: eGFR  $\geq$ 90; eGFR (mL/min/1.73 m<sup>2</sup> body surface) calculated by the CKD-EPI creatinine equation.

<sup>b</sup>MDKF: eGFR 89 to 60; eGFR (mL/min/1.73 m<sup>2</sup> body surface) calculated by the CKD-EPI creatinine equation.

<sup>c</sup>CKD: eGFR <60; eGFR (mL/min/1.73 m<sup>2</sup> body surface) calculated by the CKD-EPI creatinine equation.

<sup>d</sup>Trace metal infusions: One vial per infusion of any of the following multitrace element solution, Addamel N, Fresenius Kabi; Decaven 40 mL, Baxter.

history of long-term HPN, 50 due to SBS and 11 due to intestinal dysmotility, showed an eGFR <60 mL/min/1.73 m<sup>2</sup> in 4 of 70 (5.7%) [9]. Also in this case, no comparison was possible because of the different patient cohort age category.

The presence of CKD at CS was associated with older patient age at both HPN initiation and at CS, the presence of urologic diseases and, numerically, with SBS as the cause of CIF. The development of CKD during the Fup was associated with female sex and with an older age at CS. A numerically association with the frequency of episodes of CRBSI, of new urologic complication, as well as with a lower HPN volume was observed. Also these data are in keeping with those reported by previous studies. Lauerjat et al. [4] observed that the presence of urologic or nephrologic diseases and biological signs of dehydration explained most of the decrease of kidney function in their HPN patients. In the early study by Buchman et al., the episodes of bacteriemia/fungemia per year and patient age were associated with the decline of GFR and accounted for 53% of the CrCl variability [2]. A decrease of kidney function with aging is a known natural evolution [5]. Urinary stones and chronic obstruction are also known risk factors for the development of CKD [10,11]. A SBS with high-output jejunostomy may contribute to the development of reduced kidney function through recurrent episodes of acute kidney failure owing to dehydration [12]. A SBS with a colon in continuity is at risk for calcium oxalate kidney stones and of nephrocalcinosis, both risk factors for a decrease of kidney function [13]. CRBSI can be a risk factor for kidney damage in cases of organ failure related to sepsis [14]. The lower amino acid content of the HPN admixture of the CS subgroups with MDKF and CKD probably was due to the intentional prescription of a lower nitrogen load in these patients. A numeric lower HPN volume was observed in patients who developed MDKF

or CKD. The retrospective characteristic of this survey did not allow for a robust explanation of why this occurred. Nevertheless, these data are in keeping with the recommendation to maintain a normal hydration status to prevent a decrease of kidney function. The present data did not show any association between the amount of multitrace element infusions and kidney function, a previously suggested hypothesis [15]. The higher frequency of women who developed a CKD during Fup is in contrast with the knowledge of a lower rate of GFR decline in women [5]. We have not been able to find an explanation for what is probably a casual observation.

The present data showed that in patients with CIF, the probability of developing CKD during treatment is higher in those with MDKF than in those with NKF at time of treatment initiation (Fig. 2). Furthermore, the Kaplan–Meier analysis of the probability of avoiding CKD indicates that the risk for developing CKD is higher during the first 5 y of treatment (Figs. 1 and 2). Overall, the analysis shows that the risk for developing CKD during long-term HPN is low if kidney function is normal at the time of treatment initiation and that kidney function decline depends primarily on kidney function at the beginning of HPN treatment.

## Conclusion

The results of the present study demonstrated that in patients on HPN for CIF owing to benign disease, reduced kidney function is a frequent finding, requiring accurate monitoring during treatment. Maintaining normal kidney function should be a primary target of CIF management. Prevention of the development of CKD primarily relies on the maintenance of fluid balance and on the prevention of CRBS and urologic diseases, through appropriate

**Table 2**  
Characteristics of patient cohorts categorized according to the evolution of kidney function during the 30-mo prospective Fup

	NKF or MDKF* <sup>†</sup> at CS and CKD <sup>‡</sup> at Fup(n = 8)	NKF or MDKF* <sup>†</sup> at both CS and at Fup(n = 34)	P-value
Women, n (%)	7 (87.5)	15 (44.1)	0.047
Age at BL, y	45.3 (36.5–72.7)	39.0 (0.9–68.7)	0.080
Age at CS, y	54.2 (37.1–73.4)	49.1 (18.6–70.3)	0.105
BMI at CS, kg/m <sup>2</sup>	19.9 (10.8–26.4)	20.4 (12.6–31.5)	0.961
BMI at Fup, kg/m <sup>2</sup>	20.2 (10.4–24.6)	21.1 (14.1–29.5)	0.665
Primary disease, n (%)			0.548
– Mesenteric ischemia	1 (12.5)	9 (26.5)	
– Crohn's disease	2 (25)	3 (8.9)	
– CIPO	2 (25)	11 (32.3)	
– Others	3 (37.5)	11 (32.3)	
Cause of intestinal failure, n (%)			0.278
– SBS with end jejunostomy	2 (25)	8 (23.5)	
– SBS with colon in continuity	3 (37.5)	12 (35.3)	
– Motility disorder	2 (25)	11 (32.4)	
– Mucosal disease	0	3 (8.8)	
– Fistulas	1 (12.5)	0	
Urologic disease at CS, n (%)			0.151
– Stones	1 (12.5)	7 (13.2)	
– Urinary obstruction	2 (25)	5 (9.4)	
– Recurrent urinary infection	0	2 (5.9)	
New urologic disease between CS and Fup, n (%)– Kidney stones– Urinary tract obstruction			0.400
HPN duration at CS, mo	34.3 (0.6–256.6)	70.2 (0.8–339.8)	0.290
HPN volume at CS, mL/d	1114 (428–3000)	1917 (535–5500)	0.172
HPN volume at Fup, mL/d	827 (428–3000)	1855 (357–5550)	0.607
Trace metal infusions <sup>§</sup> at CS, n/wk	4.5 (1.5–7)	5 (0–7)	0.758
Trace metal infusions <sup>§</sup> at Fup, n/wk	3.5 (2.5–7)	5 (0–7)	0.660
CRBSI episodes between CS and Fup, n/y per patient	0.40 (0–0.78)	0.00 (0–1.29)	0.377
Patients receiving potentially nephrotoxic drugs <sup>  </sup> during Fup, n (%)	2 (25)	9 (26.5)	1
CT procedures with IV iodinated radiocontrast, n/patient	0.5 (0–3)	1 (0–8)	0.446
eGFR category at CS, n (%)			
– NKF	1 (12.5)	25 (73.5)	0.001
– MDRF	7 (87.5)	9 (26.5)	
– CKD	0	0	
eGFR variation between CS and Fup			
– mL/min/1.73 m <sup>2</sup>	–24 (–74 to –3)	0.5 (–38 to 41)	0.001
– %/y	–13.4 (–22 to –2)	–0.2 (–12.3 to 23.2)	<0.001

BL, baseline; BMI, body mass index; BW, body weight; CIPO, chronic intestinal pseudo-obstruction; CKD, chronic kidney disease; CRBSI, catheter-related bloodstream infections; CT, computed tomography; eGFR, estimated glomerular filtration rate; Fup, follow-up; HPN, home parenteral nutrition; IV, intravenous; MDKF, mildly decreased kidney function; NKF, normal kidney function; SBS, short bowel syndrome.

Continuous variables data are reported as median (range).

\*NKF: eGFR  $\geq$ 90; eGFR (mL/min/1.73 m<sup>2</sup> body surface) calculated by the CKD-EPI creatinine equation.

<sup>†</sup>MDKF: eGFR 89 to 60; eGFR (mL/min/1.73 m<sup>2</sup> body surface) calculated by the CKD-EPI creatinine equation.

<sup>‡</sup>CKD: eGFR <60; eGFR (mL/min/1.73 m<sup>2</sup> body surface) calculated by the CKD-EPI creatinine equation.

<sup>||</sup>Potentially nephrotoxic drugs: aspirin, mesalazyn, statin.

<sup>§</sup>Trace metal infusions: One vial per infusion of any of the following multitrace element solution, Addamel N, Fresenius Kabi; Decaven 40 mL, Baxter.

HPN regimens and medical and surgical treatments having as their objective improved intestinal function and intestinal rehabilitation [1].

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