Nutritional management of encapsulating peritoneal sclerosis with intradialytic parenteral nutrition

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Introduction

The learning objectives of the case study were:

- To discuss the management of a patient with encapsulating peritoneal sclerosis.
- To discuss the nutritional recommendations of adults with end-stage kidney disease (ESKD) on haemodialysis.
- To discuss intradialytic parenteral nutrition as a nutrition therapeutic intervention.
- To discuss refeeding syndrome as a complication of nutrition intervention.
- To discuss ethical considerations regarding parenteral nutrition in South Africa.

Case study

A 52-year-old male with ESKD secondary to hypertension was treated on continuous ambulatory peritoneal dialysis for 10 years. The peritoneal dialysis was stopped because of recurrent peritonitis. From July 2012 to May 2013, he was placed on haemodialysis three times a week. He presented with severe vomiting, abdominal distension and constipation indicative of total bowel obstruction in September 2012. Since he could not tolerate oral feeds and clinically presented with severe malnutrition, his nutritional management included total parenteral nutrition (TPN) to improve his nutritional status in preparation for surgical exploration and intervention. After two weeks of TPN, the patient was fit for surgery. The laparotomy was remarkable in finding adhesive lesions, for which an enterolysis was successfully completed. Postoperatively, the patient recovered uneventfully and was discharged in September 2012. He then received counselling and oral nutritional supplements.

Six months later, the patient was readmitted with recurring symptoms of vomiting and weight loss. After appropriate abdominal investigations, a partial bowel obstruction was again confirmed. He was admitted for two weeks to optimise his nutritional management because he was at risk of refeeding syndrome. Thereafter, he was managed as an outpatient with intensive nutritional monitoring at each dialysis session.

Diagnosis

The diagnosis was ESKD treated with haemodialysis, and complicated with recurring partial bowel obstruction secondary to encapsulating peritoneal sclerosis (EPS) and severe protein energy malnutrition (PEM).

This case study presents the patient's nutritional management from February 2013 to May 2013. During this time, he received a semi-diet, oral nutritional supplements and a trial of intradialytic parenteral nutrition (IDPN).

Anthropometry

At baseline, the patients' pre- and post-dialysis weight was recorded, including his measured weight loss over six months. The anthropometry measurements are summarised in Table I.

Table I: Summary of anthropometric measures

Measurement	Value in February 2013
Usual weight (kg)	55
Weight loss over the preceding six months	21.8% (12 kg)
Pre-dialysis weight (kg)	43.5
Post-dialysis weight (kg)	43
Height (cm)	169
Body mass index (kg/m^2)	15
Ideal body weight (kg)	53-57

The patient presented with grade III PEM. He had consistently undergone severe unintentional weight loss of 21.8% in the six months preceding February 2013. His measured pre- and post-dialysis weight indicated a nominal intradialytic weight gain of 0.5 kg, indicative of poor oral fluid intake. Haemodialysis patients are usually allowed to gain 2-2.5 kg of their dry weight between dialysis sessions.[1]

Table II: Biochemistry profile

	Reference ranges	Day 1 pre-dialysis (baseline)	Day 1 post-dialysis	Day 3 pre-dialysis	Day 3 post-dialysis	Three months post-IDPN or pre-dialysis
Albumin (g/l)	35-52	25	26	25	26	35
Calcium (mmol/l)	2-2.5	2.1	2.1	2.2	2.1	2.3
Magnesium (mmol/l)	0.6-1.1	0.7	0.6	0.8	0.7	0.9
Phosphate (mmol/l)	0.8-1.4	0.9	0.8	1	0.8	1.5
Sodium (mmol/l)	135-147	131	135	133	136	135
Potassium (mmol/l)	3.3-5.3	3.6	3.5	3.7	3.6	4
Urea (mmol/l)	2.1-7.1	18	15	22	20	25
Creatinine (umol/l)	64-104	383	255	386	370	500
eGFR (ml/minute/1.73m^2)	-	17	23	16	15	10
C-reactive protein (mg/l)	0-10	80	-	-	-	45

eGFR: estimated glomerular filtration rate, IDPN: intradialytic parenteral nutrition

Biochemistry

Routine blood tests during parenteral nutrition were monitored according to the protocol used in Tygerberg Academic Hospital. The patient's full blood count indicated normochromic normocytic anaemia due to chronic disease, caused by erythropoietin deficiency in chronic kidney disease. Glucose, triglycerides and liver function tests remained normal throughout the intervention. All other post-dialysis values were influenced by fluid shifts and the dialysis effect.[2] Table II indicates the biochemistry profile of the patient on initiation of the IDPN intervention (baseline), as well as at day 3, and three months post-intervention, and the effect of dialysis on the results.

At baseline, the low serum albumin concentration was probably a reflection of fluid retention and the underlying inflammatory process, and therefore limited the specificity of serum albumin as a nutritional marker.[2] Low to normal urea and creatinine levels in a patient with ESKD are reflective of PEM, while an improvement in nutritional status would be characterised by these levels increasing to a higher level.[2] The lower range of normal electrolyte levels pre-dialysis is also reflective of PEM, and is considered to be a "red flag" sign of refeeding syndrome.

The results in the next three-month intervention reflected a slight increase in electrolytes concentrations, including urea and creatinine. This was attributed to the aggressive nutritional intervention with IDPN, during which the patient's intake (50% of which was voluntary) was supplemented with oral nutritional supplements and IDPN.

Clinical investigation

The patient appeared weak and frail, with severe generalised muscle wasting and mild abdominal distension due to the partial bowel obstruction. Expected clinical signs of mild to moderate fluid overload were not observed since the patient's measured intradialytic weight gain was only 0.5 kg because of his poor oral intake. Other expected clinical signs included Cushing's syndrome, e.g. fat redistribution and weight gain. However, clinical signs of the steroid treatment were masked owing to the patient's emaciation (severe PEM).

Diet history

In September 2012, the patient was counselled postoperatively and given a renal diet. The RenalSmart® web-based programme was used to formulate a nutritional prescription which included the South African renal exchange lists,[3] as well as oral nutritional supplements 2-3 times per day, depending on his oral intake and fluid status.

On readmission in February 2013, the patient could only tolerate approximately 50% of his prescribed diet because of recurring symptoms of partial bowel obstruction and persistent vomiting. Macronutrient intake consisted of 427 kcal, 22 g protein, 50 g carbohydrates and 15 g fat.

Medical management

Cessation of continuous ambulatory peritoneal dialysis treatment was recommended on diagnosis of EPS to prevent further peritoneal damage, and haemodialysis was initiated. The patient received tamoxifen 10 mg per day (a selective oestrogen receptor modulator used in fibrosclerotic disorders) and prednisone 20 mg per day (a corticosteroid) to suppress the inflammatory process associated with EPS.[4,5] Surgical treatment in this case included enterolysis, a procedure performed to remove adhesive lesions obstructing the bowel.[6,7]

Surgery can reverse the state of bowel obstruction, but it does not improve peritoneal deterioration, leading to the reformation of the capsules and recurrence of EPS, usually in the ensuing 6-12 months postoperatively.[4]

On readmission, the patient presented with partial bowel obstruction. Surgery was not indicated in view of the patient's emaciation.

Nutritional management

After readmission in February 2013, the patient was referred to the dietitian for re-evaluation of the nutritional prescription to optimise his nutritional status. After exhaustive nutritional interventions with oral and enteral support to establish acceptability and

Table III: Initial prescription (actual body weight of 43 kg)

Nutrient	Recommendation	Calculated requirements	Oral intake (semi-diet)	Half of the IDPN
Energy (kcal/kg)	20	860	427	490
Protein (g/kg)	1.2	52	22	14
Nitrogen (g/kg)	0.2	8.3		2.3
Carbohydrates (g/kg)	2-3	86-129	50	63
Fat (g/kg)	0.7-1.5	30-65	15	25
Fluid (ml/day)	1 000-2 000	1 000- 2 000	400	380

IDPN: intradialytic parenteral nutrition

Table IV: Maintenance prescription (lower range of ideal body weight of 53 kg)

Nutrient	Recommendation	Calculated requirements	Oral intake (semi-diet and sip feeds)	IDPN
Energy (kcal/kg)	30-35	1 590-1 855	900	980
Protein (g/kg)	1.2	64	35	28
Nitrogen (g/kg)	0.2	10.2		4.5
Carbohydrates (g/kg)	3-5	159-265	120	125
Fat (g/kg)	0.7-1.5	37-80	30	50
Fluid (ml/day)	1 000-2 000	1 000-2 000	600-800	760

IDPN: intradialytic parenteral nutrition

Table V: Follow-up prescription (higher range of ideal body weight of 57 kg)

Nutrient	Recommendation	Calculated requirements	Oral intake (semi-diet and sip feeds)	IDPN
Energy (kcal/kg)	30-35	1 710-1 995	800	980
Protein (g/kg)	1.2	68	20	56
Nitrogen (g/kg)	0.2	10.9		9
Carbohydrates (g/kg)	3-5	171-285	110	125
Fat (g/kg)	0.7-1.5	40-86	20	50
Fluid (ml/day)	1 000-2 000	1 000-2 000	600	1 000

IDPN: intradialytic parenteral nutrition

tolerance, supplemental parenteral nutrition was recommended, and supplemental IDPN initiated. The patient was managed for two weeks in hospital to monitor the risk of refeeding syndrome. Thereafter, he was stable enough to be managed as an outpatient. It was determined that 3-6 months using IDPN would improve the patient's nutritional status. The nutritional goals focused on preventing refeeding syndrome, correcting electrolytes and fluid balance, and maintaining weight (and preferably facilitating weight gain) to improve the nutritional status of the patient. A combination of guidelines was used, including the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI™), and the European Society of Parenteral and Enteral Nutrition (ESPEN) and refeeding syndrome guidelines to calculate the requirements.[2,8,9]

Initially, in February 2013, the nutritional calculations were based on actual dry weight of 43 kg since the patient's BMI was classified as grade III PEM, and he was at risk of refeeding syndrome. The nutritional prescription included a semi-diet as it was better tolerated. The oral nutritional supplements were stopped to avoid overfeeding when IDPN was initiated. To prevent refeeding syndrome, only half of the premixed IDPN bag was given, which combined with oral intake, met the calculated requirements (Table III).

After two weeks, an ideal weight of 53 kg (BMI of 18.5 kg/m^2) was used during the maintenance prescription to optimise the nutritional status (Table IV). The nutritional prescription included a semi-diet with full-volume IDPN. Oral nutritional supplements were also reintroduced as the risk for refeeding syndrome decreased at this stage of the patient's management. On the days without IDPN, he was encouraged to increase his oral intake as much as possible depending on tolerance. The patient was carefully monitored to avoid overfeeding occurring since an ideal body weight was used, combined with a higher range of requirements.

After two months of intervention, the patient's oral intake decreased even further, and the IDPN prescription was increased to meet requirements using an ideal body weight of 57 kg (a BMI of

20 kg/m^2). Because of the patient's severe PEM and poor intake, the follow-up prescription of IDPN (Table V) provided approximately 50% of his requirements. The plan was to initiate TPN immediately if the patient's intake deteriorated even further.

Micronutrient imbalances were corrected intravenously when IDPN was initiated to prevent electrolyte shifts associated with refeeding syndrome. Prophylactic electrolytes were also given since the IDPN is electrolyte-free and the patient was experiencing ongoing losses, i.e. vomiting and losses during dialysis. The IDPN contained vitamins and trace elements, but additional daily supplementation with water-soluble vitamins was given to address dialysis losses.[1] All other micronutrients and minerals were given according to the NKF KDOQI™ redommendations.[2]

A motivation was written, using evidence-based literature, for the patient to receive IDPN. The hospital pharmacy placed the order with the company who supplies the IDPN. IDPN is prepared under strict sterile conditions as a single disposable bag. The stock was delivered and stored (< 7°C) in the fridge in the renal unit. The IDPN was initiated at the start of haemodialysis and infused through an intravenous pump over four hours into the venous return of the dialysis machine. A constant flow was maintained over the entire dialysis session. Hanging of the IDPN occurred according to strict sterile technique and hospital protocol. Since the patient had a negative fluid balance, additional fluid did not have to be removed. Hyperglycaemia was treated at the discretion of the attending nephrologist. The patient did not receive a snack during dialysis, but only at the end of the session, and determined by blood glucose levels. The patient was discharged approximately one hour after dialysis after glucose monitoring and stabilisation.

In addition to routine blood tests during parenteral nutrition, pre- and post- dialysis electrolytes were also measured and corrected accordingly. Continuous monitoring of blood glucose levels and blood pressure pre- dialysis, during dialysis and post-dialysis were performed according to protocol. The patient's weight was also measured daily, including pre- and post-dialysis to maintain a euvolaemic state post-dialysis.

Even though there was improvement in the patient's nutritional parameters during the three-month trial of IDPN, the partial bowel obstruction caused by EPS worsened, and he required TPN prior to the scheduled surgery. The patient passed away due to surgical complications.

Discussion

EPS is a rare but severe complication of long-term peritoneal dialysis, and is defined by the International Society for Peritoneal Dialysis as a clinical syndrome that involves recurrent gastrointestinal obstruction, including sclerosis, calcification, peritoneal thickening and cocooning or encapsulation of the intestines.[4] Clinical symptoms of partial or total bowel obstruction are present, caused by a fibrous cocoon that covers the intestines, leading to weight loss, malnutrition, infection and eventually death.[5] The prevalence of EPS is 0.5-2.5%. An increasing incidence relates to the duration of peritoneal dialysis. The mortality rate is 25-55% in the first year of diagnosis.[6] The diagnosis of EPS lacks specificity and relies on clinical, radiographic or macroscopical evaluation.[4] The first signs of EPS, i.e. nausea and changes in the stool pattern, are often not recognised, and may be accompanied by increased levels of inflammatory markers or blood-stained ascites. Patients present with signs of bowel obstruction at diagnosis.[4,5] Theories on the pathogenesis of EPS include inflammation, fibrosis and angiogenesis (growth of new blood vessels in the body).

The stages of EPS include:

- *Asymptomatic:* Ultrafiltration failure and ascites.
- *Inflammatory:* Weight loss, diarrhoea or changes in the stools, and blood-stained ascites.
- *Progressive or encapsulating:* Abdominal complaints, e.g. nausea and vomiting, and gastrointestinal obstruction.
- *Obstructive:* Complete ileus.

PEM is very common in patients with ESKD undergoing maintenance haemodialysis. Prevalence varies between 18% and 70%, and it is one of the strongest predictors of mortality and morbidity.[2] PEM increases with duration on dialysis, and is more severe in the elderly.[8] Maintenance haemodialysis is associated with a high mortality rate. There is a five-year survival rate of 35%.[2] PEM can be attributed to a multitude of interrelated causes, such as chronic inflammation, co-morbidities, metabolic acidosis and decreased appetite, with inadequate protein and energy intake, as well as inflammatory disorders.[10,11] ESKD patients should undergo routine and continuous monitoring of nutritional parameters, including pre-dialysis serum albumin, percentage of usual body weight, percentage of standard body weight, subjective global assessment, dietary assessment and measurement of a normalised protein equivalent of total nitrogen appearance (a measure of the net protein degradation and protein intake in dialysis patients).[2]

The average requirement of dietary protein intake (DPI) in healthy individuals is estimated to be 0.65 g protein/kg/day. The recommended dietary allowance is 0.83 g protein/kg/day.[12] A DPI of 1.2 g/kg/day is necessary to maintain a neutral or positive nitrogen balance in stable haemodialysis patients.[13] The DPI is reported to be low in dialysis patients. Mean levels of DPI vary from 0.94-1 g protein/kg/day. This means that half of ESRD patients ingest less than this amount of protein.[13] The NKF KDOQI™ guidelines recommend a DPI of 1.2 g/kg/day for clinically stable haemodialysis patients. The ESPEN guidelines even recommend intake up to 1.4 g/kg/day.[2,8] Since it is difficult for many ESRD patients to meet this prescription, this goal may require oral nutritional supplements, and enteral or parenteral nutrition, singly or in combination, depending on the clinical setting.

Patients should also receive intensive nutritional counselling with an individualised plan that is reviewed at least quarterly.[2] If these conservative measures are unsuccessful, oral nutritional supplements must be considered.[2] Enteral nutrition must be considered as first-line therapy in patients who are unable to eat adequately with a functional intestinal tract.[8] Studies suggest that energy and protein intake can be increased by 20-50% with oral nutritional supplements or enteral nutrition.[13] Limitations to the use of enteral nutrition are severe malnutrition when oral nutritional supplements do not meet nutritional requirements; a non-functional

gastrointestinal tract (clinically significant bowel resection, short bowel syndrome, complete mechanical obstruction when surgery is not an option, or enteritis that requires bowel rest); or even a disability that restricts oral intake and enteral nutrition.[13] In this case, total enteral nutrition was not indicated because of partial bowel obstruction. Through regular monitoring of the patient's nutritional status and patient counselling, inadequate nutrient intake, changes in nutritional status and the failure of these interventions strategies were identified, and the need for IDPN established.

Intervention strategies which focus on oral nutritional supplements and IDPN alone, and ignore the inflammatory component, may not have a significant impact on nutritional status. Besides poor nutrient intake, serum albumin concentration is also influenced by age, fluid overload, capillary leakage and inflammation.[14] Serum albumin negatively correlates with markers of inflammation, including C-reactive protein, fibrinogen and interleukin-6.[13] Therefore, inflammation mediated by proinflammatory cytokines causes hypoalbuminaemia and loss of lean muscle mass. Caution must be used with the interpretation of serum albumin, and inflammatory factors should be taken into consideration.

Since the patient was severely malnourished and had presented with insufficient oral intake over more than six weeks, he was at high risk of developing refeeding syndrome. This can be fatal, and while it is caused by rapid initiation of feeding after a period of undernutrition (more than five days), it is characterised by fluid and electrolyte shifts, including hypophosphataemia, hypomagnesaemia and hypokalaemia, as well as metabolic and clinical complications.[15] To prevent refeeding syndrome, treatment should be started slowly and increased over 4-7 days, while monitoring electrolytes daily for the first week, and replacing them accordingly.[15] During the nutritional management of this case, initial requirements were calculated using refeeding syndrome guidelines based on actual weight to prevent overfeeding with subsequent fluid and electrolyte shifts.

IDPN formulations contain essential and non-essential amino acids, as well as dextrose and lipids.[16] They can overcome limitations in oral intake, and are given during dialysis to ensure compliance and maintenance of fluid balance. They provide a reasonably large amount of supplementation in a short time.[17] Since IDPN is only given when the patient is on dialysis (three times a week), it is not formulated to supply all protein and energy requirements. It is not a long-term support modality, but a therapy that is used to increase protein and energy intake in patients requiring "resuscitation".[13] IDPN usually provides 800-1 200 kcal/day thrice weekly from carbohydrate and lipid emulsions, and 30-60 g protein.[8] The aim of providing IDPN is to improve quality of life and reduce PEM-associated complications, as well as hospitalisation and mortality.[13]

The use of IDPN is currently recommended in patients who cannot meet their nutrient needs orally, after other reversible causes have been sought, and/or in those who cannot tolerate enteral nutrition because of gastrointestinal intolerance, or in those who do not require TPN.

Based on existing data, as well as expert opinion, the following criteria may be indications for IDPN:[10,11,13,16,17]

- Evidence of PEM, and inadequate dietary protein and/or energy intake (protein < 0.8 g/kg and/or calories < 25 kcal/kg, and/or a subjective global assessment rating of severe malnutrition).
- Weight loss more than 10% of ideal body weight, or 20% of usual body weight (no time constraints).
- Serum albumin below 34 g/l (a three-month rolling average).
- Evidence that comprehensive nutritional assessment and dietary counselling have failed to achieve the expected outcome.
- The inability to administer or tolerate adequate oral nutrition, including oral nutritional supplements and enteral nutrition.
- Evidence that a patient is intolerant of enteral nutrition, enteral nutrition cannot meet the individual's nutritional needs, or is not feasible (a three-month trial).
- Evidence that the following were either eliminated or addressed in the patient: anorexia caused by a uraemic state, an altered taste sensation, intercurrent illness, emotional distress, an impaired ability to ingest food, an unpalatable prescribed diet, a catabolic response to a superimposed illness, inadequate dialysis, gastroparesis or constipation.

Multiple studies have documented that IDPN improves nutritional parameters. However, a recent randomised controlled trial in haemodialysis patients with PEM showed that the addition of IDPN to oral nutritional supplements did not further improve nutritional status.[8] Therefore, IDPN should only be initiated in haemodialysis outpatients with PEM who cannot tolerate oral nutritional supplements. Generally, the combination of oral nutritional supplements and IDPN can only provide 7-8 kcal/kg/day and 0.3-0.4 g protein/kg/day.[8] According to ESPEN guidelines, oral nutritional supplements and IDPN are generally unable to provide satisfactory nutritional requirements in patients with severe PEM with a spontaneous intake of less than 20 kcal/kg/day and 0.8 g protein/kg/day.

ESPEN recommends that enteral nutrition should be considered when oral nutritional supplements or IDPN fail to improve nutritional status, and that enteral nutrition should always be preferred to parenteral nutrition. Parenteral nutrition is only indicated when enteral nutrition is impossible or insufficient.[8] Enteral nutrition would not have produced better results than oral intake in this patient because of the partial bowel obstruction preventing nutrient absorption. After a multidisciplinary discussion, IDPN was considered to be the next best option for this patient who was being treated on an outpatient basis. It was determined that TPN would be the last option if IDPN failed as the former would require hospitalisation since home-based parenteral nutrition has not yet been established in South Africa. The patient still met 50% of his prescribed intake. Therefore, a trial of IDPN was initiated to improve his quality of life, and to avoid hospitalisation.

The use of specific parenteral solutions is not yet supported by data. When considering which macronutrients to administer during IDPN, the various metabolic abnormalities of patients on haemodialysis should be taken into account. Haemodialysis patients have altered protein metabolism, with impairments in branched-chain amino acids, essential amino acids, tyrosine and sulphur amino acids

metabolism. Intradialytic infusion of amino acids prevents a decrease in plasma amino acids, and a subsequent decrease in protein synthesis.[8] Glucose and lipid metabolism are also altered in this population, and the use of hypertonic glucose is limited by insulin resistance, glucose intolerance and the risk of post-dialysis hypoglycaemia.[8] Lipid emulsions should be provided to prevent essential fatty acid deficiencies, and to decrease the osmolarity of the solution in order to increase the tolerance of providing the parenteral nutrition peripherally.[8]

According to the ESPEN, the following guidelines must be taken into consideration to ensure optimal tolerance of the IDPN: [8]

- IDPN should be infused at a constant rate, thrice weekly, during a typical four-hour haemodialysis session.
- IDPN delivery should be progressively increased from 8 ml/kg/IDPN in the first week, to a maximum of 16 ml/kg/IDPN, and not exceeding 1 000 ml per session.
- IDPN should be associated with ultrafiltration (volume for volume).
- Seventy-five mmol of sodium should be added per litre of IDPN solution in order to compensate for sodium losses owing to ultrafiltration.

One of the advantages of using IDPN in acutely ill patients with no need of enteral nutrition (which carries risks of fluid overload, aspiration and gastrointestinal intolerance), is no need for central venous catheter insertion (as for TPN with its own risks of sepsis and costs).[13] In South Africa, home-based TPN is not available, making IDPN the best alternative to parenteral nutrition support for the outpatient care of renal patients on haemodialysis. Strictly defined criteria must be followed before IDPN is initiated to prevent indiscriminate use in chronic haemodialysis patients who are overtly malnourished.[16] IDPN should be started as early as possible, and maintained for approximately 4-6 months. It can be prolonged indefinitely if required in a clinical setting.[13,16] The disadvantages of IDPN include the cost since it is not available on state tender or on medical aid, and if not prescribed with caution, can result in overfeeding. Metabolic complications from overfeeding are serious and can be fatal. Overfeeding is associated with hyperglycaemia, azotaemia, fat overload syndrome, hypertriglyceridaemia, hepatic steatosis, hypercapnia, and increased risk of bloodstream infection.[18] Therefore, it is of utmost importance that the nutritional prescription must be constantly monitored by an experienced dietitian so that metabolic stability is maintained and recovery promoted.

Conclusion

The incidence of malnutrition in chronic kidney disease remains unchanged over time, whereas patient management and dialysis techniques continue to progress. IDPN is a convenient, non-invasive, safe therapy which provides nutrition support during the haemodialysis procedure. It is a method of nutritional supplementation, and not total nutritional support. Strict definitive criteria must be followed. A trial of IDPN may be warranted, but it is more than likely that TPN will be needed. Further large studies need to be undertaken to provide data on IDPN's effectiveness with regard to quality of life, hospitalisation and survival, and to validate it as a therapeutic intervention in the appropriate clinical setting. Optimal nutrition support must be provided for the patient's sake. Unquestionably, IDPN is cost-effective over the long term.

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