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The Severity of Hypoalbuminemia Predicts the Response to Intradialytic Parenteral Nutrition (IDPN) in Hemodialysis Patients

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Abstract

Background—Intradialytic parenteral nutrition (IDPN) is currently used infrequently to correct hypoalbuminemia in maintenance hemodialysis (MHD) patients. We hypothesized that severity of baseline hypoalbuminemia correlates with the success rate of the IDPN therapy in MHD patients.

Methods—In a prospective and contemporary cohort of 196 hypoalbuminemic MHD patients who received IDPN through Pentec Health, predictors of IDPN response were examined using multivariate logistic regression.

Results—Out of 196 hypoalbuminemic MHD patients, 134 subjects had severe hypoalbuminemia defined as baseline serum albumin <3.0 g/dL. The average period of IDPN therapy was 5.8±2.4 months. Baseline serum albumin was lower in MHD patients who responded to the IDPN (2.68±0.47 g/dL). The multivariate logistic regression analysis adjusted the associations for age, gender, diabetes, and IDPN time. The presence of severe hypoalbuminemia (serum albumin <3.0 g/dL) at baseline was associated with 2.5 time higher chance of responding to IDPN (95% confidence interval [CI]: 1.3–4.9, p=0.006). The same severe hypoalbuminemia was associated with 3.5 times increased likelihood of serum albumin correction by at least 0.5 g/dL (95% CI: 1.8–6.8, p<0.001).

Conclusions—Improvement of hypoalbuminemia occurs in most hypoalbuminemic MHD patients who receive IDPN therapy. The likelihood and magnitude of the response to IDPN is associated with the severity of baseline hypoalbuminemia. These associations need to be verified in controlled trials.

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Potential Conflict of Interests:

Ms. D. Scholl is an employee of Pentec Health, the provider of the IDPN that is used in this study, and Dr. S. Lindenfeld is a former executive and current consultant of Pentec.

Keywords

Hypoalbuminemia; intradialytic parenteral nutrition (IDPN); chronic kidney disease (CKD); hemodialysis; protein-energy wasting (PEW)

Introduction

In the United States, approximately 400,000 individuals with chronic kidney disease (CKD) stage 5, undergoing maintenance hemodialysis (MHD) treatment, have an unacceptably high mortality rate of 20–25% per year in the USA.[1] Even though most CKD patients die of cardiovascular diseases, traditional cardiovascular risk factors such as hypercholesterolemia or hypertension do not appear to be associated with death risk.[2–4] However, a low serum albumin, [5] low protein intake [6] and/or low body mass index (BMI) or weight loss [7] are strong predictors of high mortality in MHD patients. The foregoing measures are all surrogates of protein-energy wasting (PEW), [8] a condition that is common in individuals with advanced CKD and which is also referred to as uremic malnutrition, [9] kidney disease wasting, [10] or malnutrition-inflammation-cachexia syndrome [11] due to its close link to chronic inflammation.[11–14] Since the PEW is associated with death risk in CKD patients, interventions that improve nutritional status may improve survival.[15] Hypoalbuminemia, the most commonly used surrogate of PEW in dialysis patient population, has strong association with increased mortality.[5, 16] If defined as a serum albumin <3.8 g/dL in CKD patients, [8] hypoalbuminemia is observed in approximately half of all MHD patients.[5] Epidemiologic evidence suggests that correcting hypoalbuminemia may improve survival in MHD patients.[5]

The intradialytic parenteral nutrition (IDPN), a known intervention to provide nutritional support parenterally during the hemodialysis treatment session, is currently used infrequently to correct hypoalbuminemia in MHD patients.[17] According to some, but not all, previous studies, the IDPN has been an effective nutritional intervention for malnourished CKD patients, especially those in whom oral food intake cannot be corrected. [18] The mixed data about success rate of the IDPN could be due to the possibility that certain characteristics in different patients are associated with the degree of response to IDPN. Hence, we hypothesized that IDPN responders, i.e., those whose baseline serum albumin [S-albumin] increased continuously during the IDPN therapy, have unique characteristics that can help identify better responders. We studied a contemporary cohort of 196 MHD patients who had received IDPN from 2002 to 2007.

Methods

Patient Population

Subjects participating in this study were hypoalbuminemic MHD patients who were referred to Pentec Health by diverse hemodialysis clinics for the IDPN therapy. The referrals were usually instigated by independent Registered Dietitians (RD) who worked in the dialysis facilities under supervision or direct request of the nephrologists in charge of providing medical care to the patient in question. In this study, the inclusion criteria were MHD

outpatients who received IDPN for three or more months, but no more than twelve months, and whose IDPN therapy ended between January 2002 and May 2007. Some patients continued to receive IDPN after May 31, 2007, but data collection was discontinued on that date. MHD patients with a starting serum albumin of > 3.5 g/dl and those with missing core data were excluded.

IDPN Intervention

The IDPN applied in this study was individually designed for each MHD patient and administered according to the protocol shown in **Table l**. Separate pump, equipment, supplies and filters were utilized. It was administered into venous chamber over the entire hemodialysis treatment three times weekly. The IDPN solution was not given simultaneously with blood transfusion or in the same line as intravenous iron. The infusion was initiated in smaller volume and increased incrementally up to 250–350 ml/hr infusion rates. The volume was calculated into fluid removal. Clear solutions of protein and dextrose were given initially. Lipids were added as 3-in-1 solution by the 7th treatment whereas a lipid test dose was performed initially by recommended 30 min infusion.

IDPN Composition

As shown in Table 2, the administered IDPN provided ~ 700–1200 Calories composed of 70–910 non-protein Calories and 40–112 g amino acids per treatment. The non-protein portion averaged 560 Calories, that of amino acids 66 g. IDPN contained a 15% or 20% amino acid solution composed of essential and nonessential amino acids. Based on 70% dextrose the individual amount of dextrose varied with body weight and diabetes status. Patients generally received 0.4 to 0.8 g dextrose per kg body weight per minute of infusion. The majority of subjects also received either 10% or 20% lipids providing 1.1 or 2 Calories per milliliter intralipid solution. The total amount of applied fluid per IDPN treatment was 500–1000 ml.

Laboratory Tests

In addition to the monthly hemodialysis blood work, phosphorus, magnesium and potassium were monitored pre and post IDPN treatment once a week for the first two weeks. Triglyceride levels were checked prior to the first lipid infusion. Monitoring of blood glucose was recommended pre, mid and post each treatment until stable levels were achieved, especially for diabetic patients during the first four weeks of therapy. Hyperglycemia was managed according to dialysis unit protocol or physician order. A copy of the subjects' routine monthly laboratory test results was requested from dialysis unit personnel. Only serum albumin was recorded for the purposes of this study.

Statistical Methods

Chi-square and student t tests were employed to examine the differences of proportion and values between two groups. Multivariate logistic regression models were fitted to construct odds ratio (OR) of response to IDPN before and after controlling for age, gender, diabetes, and IDPN time. OR values include 95% confidence interval (CI) levels. Restricted cubic spline graphs were utilized as exploratory data analyses to illustrate systematic relations

between baseline serum albumin and the likelihood of response to IDPN. This method also served to examine the non-linear associations of continuous serum albumin as an alternative to inappropriate linearity assumptions.[19] A *p*-value <0.05 or a 95% CI that did not span 1.0 was considered to be statistically significant. A p-value between 0.05 and 0.10 was considered to indicate a potentially significant trend in order to mitigate the chance of Type II error, i.e., accepting the null hypothesis when it should be rejected. Descriptive and multivariate statistics were carried out with the statistical software "Stata version 10.0" (Stata Corporation, College Station, Texas).

Results

From January 1, 2002, through May 31, 2007, 196 MHD patients from 56 outpatient dialysis clinics in fourteen states of the United States were referred for IDPN therapy and received IDPN therapy for 3 to 12 consecutive months. For this study, required data including baseline serum albumin and ending serum albumin as well as age, gender, dialysis vintage and history of diabetes mellitus were available in all 196 MHD patients. As shown in Table 3, the MHD average treatment period (dialysis vintage) was 5.7 years. Fifty-seven percent of the subjects were women. Patients were on average 64 years old, and 54% of the patients were diabetics. The average serum albumin level prior to the IDPN treatment was 2.68 g/dl. In this cohort the serum albumin value increased at an average of 0.40 g/dL during the course of IDPN therapy; 134 subjects (68%) had severe hypoalbuminemia, defined as a baseline serum albumin <3.0 g/dL.

Table 4 compares the IDPN responders, i.e., those whose serum albumin increased over the course of IDPN therapy (72% of the patients), to non-responders, i.e., those with no change or drop in serum albumin (28% of the patients). Among the responders, 59% of the patients showed an improvement in serum albumin of +0.5 g/dL or higher. The only statistically significant difference between the two groups was the lower baseline serum albumin in the responder group. Among the IDPN responders there were 37% more patients with severe hypoalbuminemia, i.e., serum albumin <3.0 g/dL.

Using logistic regression analysis and after controlling for age, gender, diabetes mellitus and IDPN time, the odds ratio of response to IDPN for each 0.5 g/dL lower serum albumin was 85% higher in the continuous model of serum albumin (see Table 5). Age, gender or length of therapy was not significantly associated with the response rate. Figure 1 is the cubic splines analyses indicating that the association between baseline serum albumin and response to IDPN is linear. After dichotomizing serum albumin at 3.0 g/dL, the chance of responding to IDPN was 2.5 times higher in subjects with severe hypoalbuminemia (95% confidence interval [CI]: 1.3–4.9, p=0.006), as also shown in Table 5. Finally, additional multivariate logistic regression analyses adjusted for the same covariates showed that the likelihood of serum albumin improvement by at least 0.5 g/dL during the IDPN therapy was 3.5 times higher in patients with severe hypoalbuminemia (95% CI: 1.8–6.8, p<0.001).

Discussion

We found that the efficacy of the IDPN therapy was predicated by the severity of baseline hypoalbuminemia in 196 MHD patients who participated in this prospective clinical study. Patients with the lower baseline serum albumin and higher proportion of severe hypoalbuminemia responded better to the IDPN therapy. Patients' demographics were not associated with the IDPN success rate. These findings, especially if verified in additional studies, may help better identify patients who would more likely benefit from the IDPN or patients who may appear less responsive to this treatment.

Individuals with CKD have an exceptionally high mortality rate and a high burden of cardiovascular disease.[20] At least one out of every five of the 400,000 MHD patients in the US dies every year.[21–23] Even though half of all these deaths are attributed to cardiovascular disease, [21] measures of the PEW, and not traditional cardiovascular risk factors, are the strongest predictors of mortality in MHD patients.[10, 13] The confounding effect of PEW on the associations between traditional cardiovascular risk factors and clinical outcome is so strong that these conventional associations appears paradoxically inversed.[3, 24] Such consistent observations reiterate the overwhelming role of the PEW in leading to poor outcomes including high death rate in CKD patients. Indeed, hypoalbuminemia, a known surrogate of PEW and possibly inflammation, is one of the strongest predictors of mortality in MHD patients.[5] Hence, it is biologically plausible that effective nutritional interventions that can correct PEW and hypoalbuminemia can lead to greater survival.[25] However, the causal link between correction of hypoalbuminemia and improved survival in CKD patients has not yet been tested in randomized controlled trials.

Several retrospective studies evaluated the effect of IDPN on clinical outcomes in MHD patients. Chertow et al [26] examined mortality in 1,679 MHD patients who received IDPN for 12 months vs. 22,517 non-randomized control MHD patients who did not and found that IDPN had salutary effect on serum levels of biochemical surrogates of nutritional status. Among patients who received IDPN, those whose serum albumin was less than or equal to 3.4 g/dL displayed a significant reduction in the relative risk of death.[26] Capelli et al [27] studied 81 MHD hypoalbuminemic patients, in whom 50 patients received IDPN for an average of 9 months. They found a better survival rate (64% versus 52%) in the MHD patients treated with IDPN. Serum albumin increased by 12% in the survivors of the IDPN-treated group.[27] A recent study in 22 acutely ill MHD patients who received IDPN for 1.5 to 48 months showed that weight loss in all patients ceased after approximately 2 months of IDPN. Protein nitrogen apperance, serum albumin, prealbumin (transthyretin), cholesterol and creatinine levels all increased significantly.[28] However, some other interventional studies failed to show evident improvement of nutritional status or clinical outcome with IDPN or other nutritional interventions.[17][29] A recent randomized trial in 186 malnourished MHD patients who also received oral nutritional supplements concomitantly did not show improvement in 2-year mortality or other outcome measures in the IDPN group.[29] However, this prospective study demonstrated that an improvement in serum prealbumin (transthyretin) during nutritional therapy was associated with a decrease in morbidity and mortality in malnourished MHD patients.[29] Hence, large-scale, prospective

randomized interventional studies that are not confounded by concurrent nutritional support of other types are needed to ascertain the impact of IDPN on clinical outcomes.

In our study the likelihood of response of the IDPN therapy was related to the degree of severity of the baseline hypoalbuminemia. Subjects with lowest serum albumin levels responded more effectively compared to those with higher serum albumin concentrations. Whether this observation is the true effect of IDPB, due to the oncotic shifts of albumin, or other mechanisms, it implies that a reverse pyramid (funnel) effect exist, in that in low albumin ranges the IDPN can effectively improve the level of serum albumin, where as in mild hypoalbuminemia much larger amount of or more intense nutritional support is needed as depicted in Figure 2. Nevertheless, the foregoing hypothesis may oversimplify the sophisticated nature of the factors related to the nutritional status and the pathophysiology or PEW and nutritional support in CKD.

Our study should be qualified for its limitations including significant degree of heterogeneity, esp. since the MHD patients originated from 56 dialysis centers across the country. Due to the administrative nature of the collected data, we did not have information on race or ethnicity, nor was data on comorbid condition available. Other biochemical data such as serum phosphorus or creatinine, or blood hemoglobin or protein catabolic rate were not available for all patients. However, baseline and post-IDPN data were available on all patients along with age sex, dialysis vintage time, IDPN therapy length and presence or absence of diabetes mellitus,

Conclusions

Our analyses in 196 hypoalbuminemic MHD patients shows that the response to the IDPN therapy is related to the severity of baseline hypoalbuminemia, in that patients with severe baseline serum albumin levels respond better to the IDPN therapy than those with mild to moderate hypoalbuminemia. Our findings may indicate a so-called funnel phenomenon (Figure 2). If these findings can be confirmed in additional studies, they may have important implications for future randomized controlled trials or patient care management and reimbursement policies, because more effective risk stratification of the malnourished dialysis patients can be achieved leading to improving efficiency of the IDPN and other nutritional supports in CKD patients. Given several advantages of the IDPN including its convenience (since it is administered during dialysis treatment and thus does not require additional clinic visits or prolonged dialysis time), the IDPN has the potential of noteworthy improvement of nutritional status and reducing morbidity and mortality in CKD patients. Nevertheless, well-designed controlled trials are needed to examine these and other hypotheses related to the IDPN therapy.

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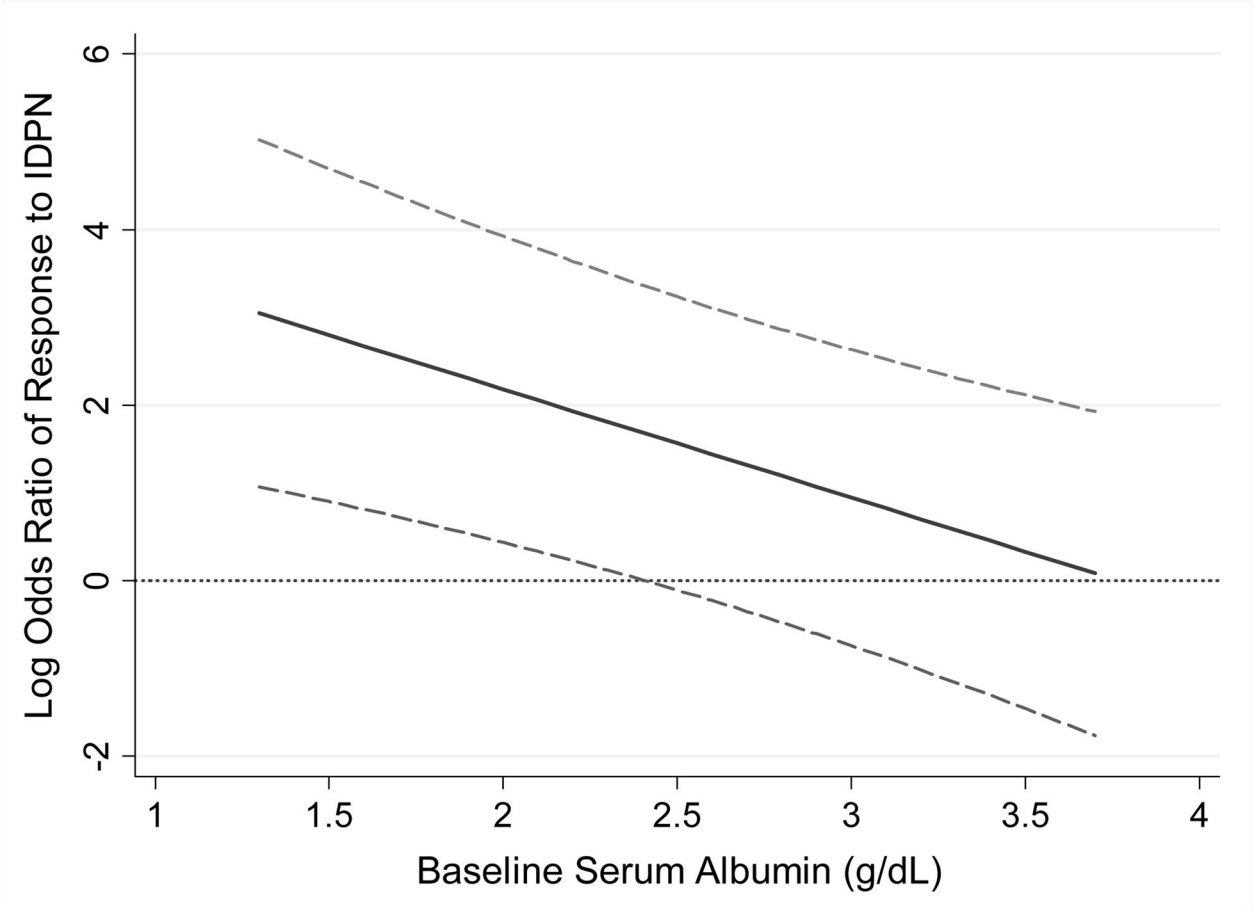

Figure 1.
Association between baseline serum albumin and odds ratio of response to IDPN treatment, adjusted for patients demographics and length of treatment, based on cubic spline analysis.

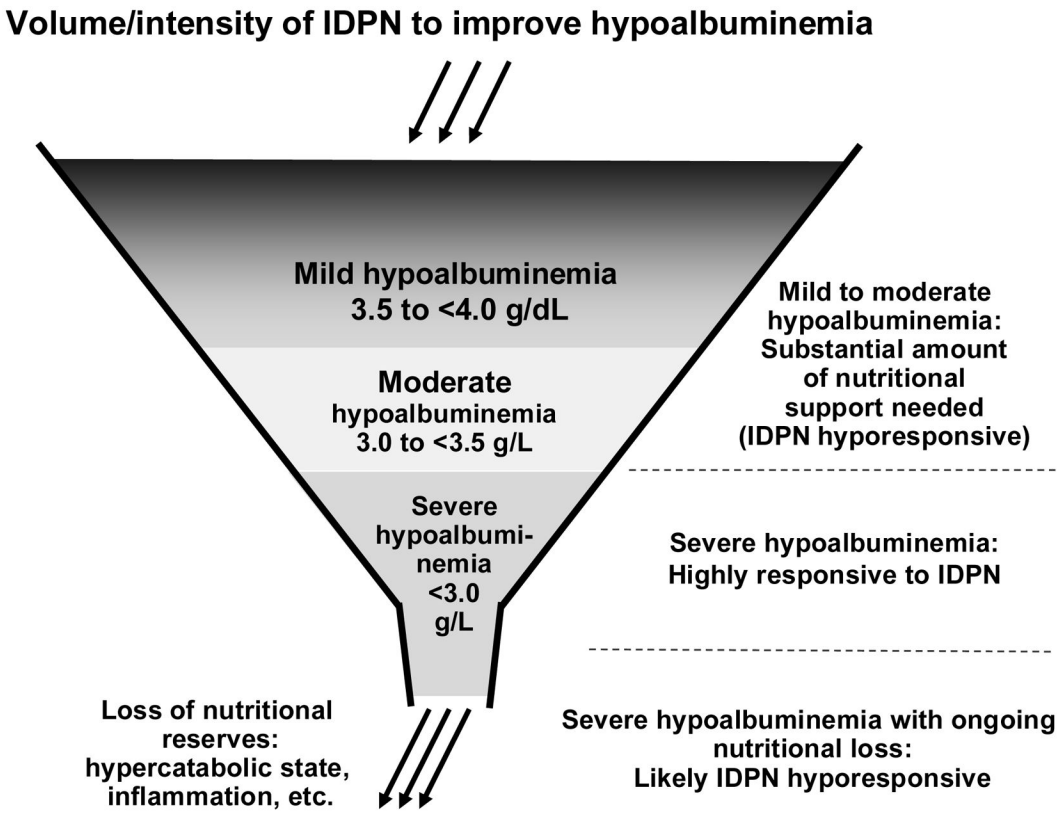

Figure 2.

Hypothetical model of reverse pyramid or funnel phenomenon to explain the association between the likelihood of response to IDPN based on the degree of severity of hypoalbuminemia

Table 1

The IDPN therapy protocol in MHD patients

- Infused over entire dialysis treatment
- Volume calculated into fluid removal
- Infused into venous chamber
- Separate pump, equipment, supplies and filters
- IDPN should not be given simultaneously with a blood transfusion or in the same line as IV iron
- Initiated in smaller volume and increased incrementally
- Clear solutions of protein and dextrose given initially
- Lipids can be added as 3-in-1 solution by 7^{th} treatment
- Test dose (30 min infusion) recommended with the lipid initiation
- Goal rate reached by 7th treatment
- infusion rates are 250–350 ml/hr

Table 2

Composition of the IDPN Solution

IDPN composition	Minimal amount	Maximal amount	Average
Amino Acids	40g	112g	66g
Dextrose	3 mg/kg/min	9 mg/kg/min	6 mg/Kg/min
Lipids	10%	20%	1.5 Cal/ml
Calories	900 Kcal	1400 Kcal	1150 Kcal
Fluid	500 ml	1000 ml	750 ml

Table 3

Baseline demographic, clinical, and laboratory values of 196 MHD patients who received IDPN therapy

Variable	Mean±SD	Minimum	Maximum
Proportion of women (%)	53%		
Diabetes mellitus (%)	54%		
Subject's Age (years)	64±15	22	92
Dialysis vintage (years)	5.7±5.7	0.3	10
IDPN Treatment period (months)	5.8±2.4	3	12
Albumin level pre study (g/dL)	2.68±0.47	1.3	3.7
Albumin level post study (g/dL)	3.08±0.60	1.3	4.6
Changes of Albumin level (g/dL)	0.40±0.61	−0.3	+2.0

Table 4

Comparing data of responders and non-responders to IDPN therapy

MHD patients' characteristics	Responders (n=142)	Nonresponders (n=54)	p-value
Age (yrs)	64±15	65±14	0.8
Diabetes mellitus	53%	57%	0.6
Gender (% women)	54%	50%	0.6
IDPN time (months)	5.7±2.2	6.1±2.8	0.3
Serum albumin (mg/dL)	2.62±0.47	2.85±0.44	0.002
Serum albumin <3.0 mg/dL (%)	74%	54%	0.006
Increased albumin ≥0.5 mg/dL (%)	59%	n/a	n/a

Table 5

Odds ratio (OR) of responding to IDPN derived from the logistic regression analysis

	Unadjusted		Multivariate adjusted	
	OR (95% CI)	**p-value**	**OR (95% CI)**	**p-value**
Baseline serum albumin (each 0.5 g/dL lower)	1.78 (1.22–2.60)	0.003	1.85 (1.26–2.73)	0.002
Baseline severe hypoalbuminemia (<3.0 vs. >=3.0 g/dL)	2.45 (1.27–4.70)	0.007	2.54 (1.31–4.93)	0.006