Editor’s Column

Chair’s Column

Intradialytic Parenteral Nutrition

Nutrition Support Practices During the Peri-Extubation Period

Pathophysiology of Aging and Targeting Malnutrition in this Population

Use of a Pureed by Gastrostomy Tube (PBGT) Diet to Promote Oral Intake: Review and Case Study

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Support Line Index 2014
Intradialytic Parenteral Nutrition
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Abstract
Malnourished patients on hemodialysis may not respond to oral nutrition interventions due to gastrointestinal (GI) symptoms, medications, frequent hospitalizations, comorbidities, deteriorating functional status, and socioeconomic challenges. Intradialytic parenteral nutrition (IDPN) is a nutritional supplement that can fill the gap between what patients are able to consume via oral diet and/or enteral feeding and what they need to consume to meet their estimated energy and nutrient needs. Clinicians face challenges in identifying who can benefit from the therapy, determining optimal IDPN formulas, and obtaining reimbursement for the therapy.

Introduction
IDPN is an intravenous nutrition support therapy given to patients during thrice-weekly hemodialysis. It is infused via the venous drip chamber of the dialysis machine, thus entering the body with the dialysis-cleansed blood. IDPN alone is insufficient to meet all energy, protein, and nutrient requirements, but it does provide a consistent amount of energy and protein with each dialysis session to supplement the patient’s oral and/or enteral intake.

The following guidelines for using IDPN are from the 2000 National Kidney Foundation Kidney Disease Outcomes Quality Initiative (1):

- IDPN may be beneficial for malnourished patients or patients unable to consume sufficient energy and protein to meet nutrient requirements.
- A trial of intensive nutrition counseling with oral supplements and/or tube feeding should be attempted and/or considered before initiating IDPN.
- The combination of IDPN and oral diet or tube feeding should meet the patient’s energy and protein needs.

A variety of clinical criteria have been used to justify the use of IDPN in hemodialysis patients, including chronic nausea, vomiting, diarrhea, and anorexia; weight loss; reduction in lean body mass; hypoalbuminemia; and a decreased protein catabolic rate (2–5). To meet total nutrition needs, the patient must be able to consume sufficient intake to complement the nutrition provided by IDPN. The patient must understand that IDPN is a supplement to the oral and/or enteral diet. A decision tree (Figure) can assist the clinician in identifying candidates for IDPN and monitoring potential outcomes of the therapy (2). The Figure also addresses intraperitoneal nutrition (IPN), which is a nutrition supplement of amino acids given in conjunction with peritoneal dialysis. Information on IPN is provided elsewhere (2,3).

Composition of IDPN
Historically, IDPN was primarily administered as a source of energy with a moderate amount of protein, averaging 1,000 kcal from dextrose and lipids and 50 to 60 g amino acids (AAs) in quantities of 1 liter or more (2,3). The current trend is to reduce the dextrose and lipid content and increase the protein in overall quantities of less than 1 liter. The changes have been made in an attempt to reduce the complications previously experienced with IDPN infusions (3,6,7). An example of an IDPN formulation is shown in Table 1.

Amino Acids
The basis of IDPN is the AA solution containing essential and nonessential AAs. The amount of protein in IDPN during each dialysis session should be approximately 1.2 to 1.4 g/kg dry body weight (3,6,8). A total of 1 g/kg may be provided to patients with hepatic encephalopathy who are sensitive to protein. A more concentrated AA formulation allows the clinician to provide more protein with less volume. For example, 100 g of protein can be given in 1,000 mL (10% AA), 667 mL (15% AA), or 500 mL (20% AA).

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Adequacy of dialysis is measured by Kt/v, a unitless measure that examines solute clearance over time (6). The AAs in IDPN may increase urea generation and decrease Kt/v, but the overall removal of uremic toxins may not be reduced, according to conversations with renal nutrition experts. Therefore, Kt/v may be an invalid indicator of dialysis adequacy during the administration of IDPN. The equation for Kt/v has not been studied or validated during IDPN infusion. Some facilities hold IDPN when measuring Kt/v.

Dextrose

The dextrose in IDPN infused during a condensed timeframe can contribute to hyperglycemia, particularly in patients with diabetes mellitus and insulin resistance. Studies have reported temporary hyperglycemia in patients without diabetes receiving IDPN (9,10). In one study, a dose of 125 g dextrose in IDPN resulted in an average increase in blood glucose levels of 63 mg/dL (9). Insulin can be added to the IDPN bag and/or administered subcutaneously, depending on the amount of dextrose provided and the resulting blood glucose levels. Clinicians should measure arterial or peripheral glucose before starting IDPN, halfway through the infusion, and 30 to 60 minutes after discontinuation of the IDPN infusion (2). The goal is to maintain serum glucose levels of less than 200 mg/dL during infusion (2) and less than 180 mg/dL postinfusion (11) while avoiding life-threatening hypoglycemia. If the patient can eat, a snack should be provided 30 minutes before stopping the IDPN infusion to help prevent rebound hypoglycemia when IDPN is stopped. Dextrose content is generally between 40 and 80 g per therapy. The reduced amount of dextrose in IDPN from more than 125 g/bag to less than 50 g/bag for patients with glucose intolerance has improved glycemic control and reduced the need to treat with insulin (2,3).

Lipid

Hyperlipidemia is a potential complication for all dialysis patients, and IDPN can contribute to an elevation in serum lipids. Serum triglycerides should be measured before administering the first intravenous fat emulsion (IVFE)-containing IDPN infusion and again before the second infusion of IVFE. Serum triglycerides greater than 400 mg/dL suggest poor lipid clearance (12). Prior to infusing IVFES, patients should receive a test dose to determine if they have an allergy to the IVFE components. A prudent approach is to defer the addition of IVFES to IDPN for 1 to 4 weeks until the patient has demonstrated tolerance to the AA and dextrose mixture. If an adverse effect occurs with the addition of an IVFE, it can be stopped and the AA/dextrose solution resumed. The focus on higher levels of AA and lower amounts of total energy in IDPN has decreased the need to use IVFES. The patient’s requirement for fat can be met through the oral diet and/or enteral feeding.

Micronutrients

Vitamins and trace elements are not routinely added to IDPN because the micronutrients could be lost in the dialysate as the blood circulates in the body and returns to the dialyzer. A general rule of thumb for limiting losses through dialysate is to add injectable vitamins during the final 30 minutes of IDPN for patients who do not tolerate oral multivitamins. However, because of periodic injectable multivitamin shortages, these micronutrients should be reserved for patients unable to tolerate any oral vitamin supplementation.

Electrolytes are not generally added to IDPN because dialysis patients often have electrolyte imbalances. However, electrolytes can be added, keeping in mind that electrolytes are added as salts. For example, sodium and potassium are combined with phosphorus, acetate, or chloride.

Infusion and Monitoring of IDPN

There are no evidence-based guidelines for initiation of IDPN formulations. A generally safe approach for initiating administration is to infuse approximately half the goal volume and 0.8 to 1 g/kg protein (2). The volume and concentration of IDPN can be progressed to goal, depending on glycemic control and patient tolerance. Predialysis electrolyte, glucose, and triglyceride levels should be monitored until stable while initiating the IDPN. Other laboratory data that are collected with the patient’s monthly dialysis laboratory analysis are generally sufficient for monitoring. Information from dietary records/dietary recall/nutrition history and weight changes can allow the clinician to monitor the sufficiency and effectiveness of IDPN and determine when the patient is ready to be transitioned off of the therapy. Due to the supplemental nature of IDPN and its lower content of dextrose than in PN, there is no need to wean the patient gradually off the therapy; it is simply not provided during the next dialysis session. The patient’s comorbidities and metabolic response to IDPN therapy determine the frequency of necessary monitoring and the parameters to follow. In addition, guides on monitoring and administration of IDPN are available from companies providing IDPN. Because IDPN is a supplement used in conjunction with an oral diet, it should not produce the long-term complications seen with PN. However, long-term studies have not been reported.

Identifying Malnutrition in Hemodialysis Patients

IDPN has been reported to improve weight, appetite, and survival in malnourished patients on hemodialysis (10,13–22). Defining and identifying malnutrition continues to be elusive, and the effect of nutrition intervention is also difficult to measure. The criteria on which the malnutrition diagnosis is based vary from physical signs and symptoms to laboratory
malnutrition:
• Inadequate food intake to meet needs
• Acute or chronic unintentional weight loss
• Loss of subcutaneous fat
• Loss of muscle mass
• Fluid accumulation
• Functional assessment

The degree of malnutrition (severe or non-severe) in each etiology-based category is determined by the amount of change over a specific period of time. As an example, the characteristics for CKD are shown in Table 3. Note that the presence of hepatic proteins is not included as a characteristic of malnutrition due to the (Continued on next page)

values or a combination of both. An estimated 25% of dialysis patients are severely malnourished (19). Interpreting the full impact of these percentages requires confidence in and understanding of the criteria used to determine the presence and severity of malnutrition.

The difficulty in defining and diagnosing malnutrition in patients with chronic kidney disease (CKD) contributes to the inability to compare data on the prevalence of malnutrition and the effectiveness of nutrition interventions for its prevention and treatment. Members of the International Society of Renal Nutrition Metabolism (ISRN) developed a definition of malnutrition in the CKD population that considers causes, manifestations, and diagnostic characteristics (23). The term developed by the ISRN is protein-energy wasting (PEW), which takes into account the intertwined relationship of poor nutrient intake, inflammation, and uremia. The factors that promote the development of PEW include protein losses during dialysis, endocrine disorders, and comorbid conditions. Many of these factors work synergistically to affect nutritional status negatively.

PEW manifests as anthropometric and laboratory changes as well as increased mortality and morbidity risks. The diagnostic criteria set by the ISRN for PEW are listed in Table 2, and the patient must meet at least one criterion in three of the four categories. Findings in the laboratory category may be difficult to interpret due to the presence of inflammation as well as the impact of cholesterol-lowering medications or large urinary or GI losses. Anthropometric measurements, such as mid-arm muscle circumference or triceps skinfold, are not routine in most dialysis units. Clinicians primarily rely on weight changes and evaluation of nutrient intake to determine the presence of PEW.

The Academy of Nutrition and Dietetics (Academy) and the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) have collaborated to develop definitions and characteristics of malnutrition (24,25).

The three categories of etiology-based malnutrition are:
- Starvation-related (social or environmental circumstances) malnutrition, which is chronic starvation in the absence of inflammation.
- Chronic disease-related malnutrition, which is associated with mild-to-moderate inflammation seen with chronic diseases such as kidney disease.
- Acute disease- or injury-related malnutrition, which is associated with acute and severe inflammation and is the type of malnutrition seen in intensive care units.

At least two of the following characteristics are used to determine the presence of malnutrition:
- Inadequate food intake to meet needs
- Acute or chronic unintentional weight loss
- Loss of subcutaneous fat
- Loss of muscle mass
- Fluid accumulation
- Functional assessment

Table 2. Diagnostic Criteria for Protein Energy Wasting (PEW) (23)

<table>
<thead>
<tr>
<th>Diagnostic Category</th>
<th>Diagnostic Criteria</th>
</tr>
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<tbody>
<tr>
<td>Laboratory</td>
<td>Serum albumin &lt;3.8 g/dL</td>
</tr>
<tr>
<td></td>
<td>Transthyretin &lt;30 mg/dL</td>
</tr>
<tr>
<td></td>
<td>Cholesterol &lt;100 mg/dL</td>
</tr>
<tr>
<td>Body fat mass and weight</td>
<td>Body mass index &lt;23</td>
</tr>
<tr>
<td></td>
<td>Total body fat mass &lt;10%</td>
</tr>
<tr>
<td></td>
<td>Weight loss: 5% over 3 months or 10% over 6 months</td>
</tr>
<tr>
<td>Dietary intake</td>
<td>&lt;0.8 g/kg/day protein</td>
</tr>
<tr>
<td></td>
<td>&lt;25 kcal/kg/day</td>
</tr>
<tr>
<td>Muscle mass</td>
<td>Muscle mass loss: 5% over 3 months or 10% over 6 months</td>
</tr>
<tr>
<td></td>
<td>Mid-arm muscle circumference: decreased &gt;10% compared to 50th percentile of reference population</td>
</tr>
</tbody>
</table>

Table 3. Characteristics Associated with Chronic Kidney Disease and Mild-to-Moderate Inflammation

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Severe Malnutrition</th>
<th>Non-Severe Malnutrition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy Needs</td>
<td>≥1 month of consuming ≥75% energy needs</td>
<td>≥1 month of consuming ≤75% energy needs</td>
</tr>
<tr>
<td>Weight</td>
<td>&gt;5%, 7.5%, 10%, or 20% of weight loss over 1, 3, 6, and 12 months, respectively</td>
<td>5%, 7.5%, 10%, or 20% of weight loss over 1, 3, 6, and 12 months, respectively</td>
</tr>
<tr>
<td>Muscle/Fat Mass</td>
<td>Moderate-to-severe loss of fat or lean body mass or localized or generalized fluid accumulation</td>
<td>Mild loss of fat or lean body mass or localized or generalized fluid accumulation</td>
</tr>
<tr>
<td>Functional Capacity</td>
<td>Measurably reduced functional measures</td>
<td>Minimally or not reduced functional measures</td>
</tr>
</tbody>
</table>

absence of evidence that hepatic proteins respond to changes in nutrient intake (26). Hepatic proteins (albumin and transthyretin) are primarily reflective of the presence and severity of inflammation (27). Serum hepatic proteins and body weight are affected by factors other than nutrition that could skew interpretation of the patient’s nutritional status and the effect of IDPN (27–29).

Consistency in the use of either of these diagnostic tools from the ISRNM and A.S.P.E.N. is important when identifying the presence of malnutrition and determining the impact of a nutrition intervention. When evaluating the effectiveness of IDPN, consider total energy and protein intake (IDPN plus oral diet/enteral feeding), factors that affect inflammation (e.g., hospitalizations, infections, comorbidities, or surgical procedures), and fluid status (3).

Effectiveness of IDPN
An evidence-based analysis of the effectiveness of IDPN published in 1999 indicated that all but one of the studies supporting the efficacy of IDPN produced level “C” data, meaning the research studies were not well designed, randomized, or controlled (30). A 2010 systematic review of the scientific literature found only three acceptable randomized, controlled trials and determined that the evidence was insufficient for either net benefit or net harm with IDPN (31). The authors recommended the creation of a clinical trial or registry for all patients treated with IDPN to accumulate clinical outcome data for evaluation of IDPN effectiveness.

In the absence of such a clinical trial or registry, current support for use of IDPN is primarily based on expert opinion, anecdotal research findings, and editorial consensus rather than strong scientific evidence. Unfortunately, the sample size and treatment time required to produce scientific evidence of IDPN efficacy contribute to a prohibitive price tag (19,30).

Reimbursement
Individuals with permanent kidney failure, regardless of age, are eligible for Medicare benefits. Eligibility usually starts in the fourth month of dialysis. For those who have an employer- or union-sponsored plan, Medicare may become the secondary payor for up to 30 months (32). IDPN is not part of the prospective payment system that combines or bundles the Medicare payment to approved centers for dialysis services. Services such as equipment and supplies for dialysis, end-stage renal disease laboratory tests, or administrative services are included in the payment as a bundle of services provided. Therefore, an IDPN supplier may be able to bill Medicare or another insurance plan separately for IDPN (32). It is important to evaluate all of a patient’s insurance plans to determine whether IDPN therapy, including supplies, such as the pump and pole, can be covered.

Reimbursement for IDPN may be provided by Medicare Part D, Medicaid, or commercial insurance; IDPN is a non-covered benefit under Medicare Part B (33). Medicare Part D is the prescription drug plan offered to anyone with Medicare that went into effect on January 1, 2006 (34). All Medicare/Medicaid patients are automatically enrolled in Medicare Part D. Medicare Part D covers most prescription drugs not already covered under Medicare and is managed by regional Prescription Drug Plans. Because IDPN is considered to be a Part D compound or drug, AAs, dextrose, and IVFES may be covered if these drugs are on the plan’s formulary. Preauthorization is often required (34).

Commercial insurers, health maintenance organizations, preferred provider organizations, and Medicaid programs may cover IDPN based on guidelines or the medical policy established by each payor. Before starting IDPN, it is important to verify the patient’s specific plan benefits and determine coverage for not only the IDPN but for any supplies or pump needed to administer IDPN. Often, plans offer information on their websites outlining general coverage policies for therapies such as IDPN.

Each patient should be informed of his or her out-of-pocket expenses for annual deductibles, co-pays, or co-insurance before initiating IDPN therapy. The amount owed by the patient depends on the services provided and the coverage of the health plan(s) for these services.

Conclusion
IDPN should be reserved for hemodialysis patients who have not been successful in increasing oral intake after receiving intensive nutrition counseling and oral supplements. IDPN is not a “stand-alone” feeding modality; adjunctive oral diet and/or enteral feeding is required to meet the patient’s total nutrient needs. It is important to evaluate the desired outcomes critically against those that can be achieved by IDPN modalities. IDPN should be reserved for the patient with kidney disease who is unable to receive sufficient nutrients, either orally or enterally, to maintain weight and functional status.

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References