NUTRITION FOR CHRONIC KIDNEY DISEASE

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2010
Definition of Chronic Kidney Disease

Chronic kidney disease is defined as kidney damage for three or more months, as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifested by pathologic abnormalities or markers of kidney damage, including abnormalities in the composition of the blood or urine or abnormalities in imaging tests. Or GFR < 60 mL/minute/1.73 m² for three months or more, with or without kidney damage.\(^1,2\)

The normal level of GFR varies according to age, sex, and body size. Normal GFR in young adults is approximately 120 to 130 mL/minute/1.73 m² and declines with age by approximately 1 mL/minute/1.73 m² per year after the third decade.

Kidney failure is defined as either (1) GFR less than 15 mL/minute/1.73 m² (which in most cases will be accompanied by signs and symptoms of uremia) or (2) a need to start kidney replacement therapy (dialysis or transplantation).\(^3\)

Stages for Chronic Kidney Disease

According to the National Kidney Foundation (NKF) Kidney Disease Outcome Quality Initiative (K/DOQI) Clinical Practice Guidelines, CKD is classified in five stages, based on estimated GFR and irrespective of diagnosis.\(^1,2,4\)

The stages of CKD reflect gradual adaptation to nephron loss. In the early phase, stages 1 and 2, the patient is asymptomatic, blood urea nitrogen (BUN) and serum creatinine (SCr) are normal or nearly normal, and acid-base, fluid, and electrolyte balance are maintained through an adaptive increase of function in the remaining nephrons. A reduction of GFR to 30 to 59 mL/minute/1.73 m² defines stage 3, moderate impairment of GFR. The patient usually has no symptoms; SCr and BUN are increased, and the levels of hormones such as erythropoietin, calcitriol, and parathyroid hormone (PTH) are usually abnormal. Stage 4, severe impairment of GFR, involves a further loss of kidney function. Symptoms, if present, are mild; patients may have anemia, acidosis, hypocalcemia, hyperphosphatemia, and hyperkalemia. The final stage of kidney disease, stage 5, defined by a GFR of less than 15 mL/minute/1.73 m², is usually characterized by worsening of all the aforementioned symptoms. At this stage, the institution of renal replacement therapy is required, usually when the true GFR falls below 10 mL/minute or the creatinine clearance (CrCl) is below 15 mL/minute.\(^4\)

Nutrition and Kidney Disease

A high prevalence of malnutrition exists in patients with renal failure. Several surveys have reported protein-calorie malnutrition in up to 40% of this patient population.\(^5\)
TABLE 3
Stages of Chronic Kidney Disease

<table>
<thead>
<tr>
<th>Stage Description</th>
<th>GFR (mL per minute per 1.73 m²)</th>
<th>Metabolic consequences</th>
</tr>
</thead>
<tbody>
<tr>
<td>At increased risk</td>
<td>Higher than 60 (with risk factors for chronic kidney disease)</td>
<td>-</td>
</tr>
<tr>
<td>1 Kidney damage (early) with normal or elevated GFR</td>
<td>90 or higher</td>
<td>-</td>
</tr>
<tr>
<td>2 Kidney damage with mildly decreased GFR (early renal insufficiency)</td>
<td>60 to 89*</td>
<td>Parathyroid hormone level begins to rise (GFR of 60 to 80).</td>
</tr>
<tr>
<td>3 Moderately decreased GFR (moderate kidney failure)</td>
<td>30 to 59</td>
<td>Calcium absorption decreases (GFR below 50). Lipoprotein activity declines. Malnutrition develops. There is onset of left ventricular hypertrophy and/or anemia (erythropoietin deficiency).</td>
</tr>
<tr>
<td>4 Severely decreased GFR (pre-end-stage kidney disease)</td>
<td>15 to 29</td>
<td>Triglyceride concentration begins to rise. Hyperphosphatemia or metabolic acidosis develops. There is a tendency toward hyperkalemia.</td>
</tr>
<tr>
<td>5 Kidney failure (end-stage kidney disease [uremia])</td>
<td>&lt; 15 (or dialysis)</td>
<td>Azotemia develops.</td>
</tr>
</tbody>
</table>

GFR = glomerular filtration rate.

*May be normal for age.


Herselman et al. demonstrated an association between a composite score for protein-energy malnutrition and infection-related morbidity in a group of haemodialysis patients. Data from the large Dialysis Outcomes and Practice Patterns Study (DOPPS) cohort confirm that malnourished dialysis patients have an increased risk of mortality.

During progression of CKD, the requirements and utilization of different nutrients change significantly. These changes ultimately place kidney disease patients at higher risk for protein-calorie malnutrition. In addition, the presence of protein-calorie malnutrition is an important predictor of poor outcome in these patients. Understanding the applicable nutritional principles and the available methods for improving nutritional status of these patients is essential.

There are a number of factors that contribute to malnutrition in patients with renal failure (Table 1).

**Diagnosis and Monitoring Malnutrition**

There is not a single measurement that provides complete and unambiguous assessment of the nutritional status of haemodialysis patients. Ideally, a nutritional marker should not only predict outcome, but it should also be an inexpensive, reproducible and easily performed test that is not affected by such factors as inflammation, gender, age and systemic diseases. No such ideal nutritional marker is available at present.11

Malnutrition should be diagnosed by a number of assessment tools including:

(1) **Clinical assessment**

The clinical assessment of nutritional status should begin with a thorough history focusing on recent changes of dietary intake, weight and gastrointestinal symptoms.9 Current and usual body weight, changes in body weight, and weight for height (body mass index) are all used to assess overall nutritional status.12

(2) **Subjective global assessment (SGA)**12

Uses eight clinical measures including dietary intake, comorbid disease or gastrointestinal symptoms, physical examination, changes in body weight, and functional capabilities to produce a semiquantitative nutritional assessment. An overall assessment of nutritional status is generated from scores in each category:

- 1 - 2 : severely malnourished
- 3 - 5 : mildly to moderately malnourished
- 6 - 7 : increased risk of malnourishment to well nourished

(3) **Anthropometry**12

Anthropometry measures body composition (fat stores and fat free mass) using calipers and a tape measure, commonly mid upper arm circumference (MUAC) and triceps skinfold thickness (TSF); mid arm muscle circumference (MAMC) can then be calculated. Comparison with standards must use renal anthropometric tables. Serial measurements are of more significance to highlight changes in muscle mass or fat.
Normalized PNA provides an independent and less time consuming assessment of dietary protein intake. Nitrogen balance, the difference between intake and losses, is zero in the steady state or slightly positive. Both net protein breakdown under fasting conditions and dietary protein requirements are strongly influenced by body mass. In order to normalize PNA it should be related to body weight of the patient. When determining nPNA, patients should be stable and neither anabolic nor catabolic. The protein equivalent of total PNA can be estimated from interdialytic changes in urea nitrogen concentrations in serum and urine.

Serum albumin and serum prealbumin

Although the serum albumin concentration is a reasonable marker of body protein stores and a reliable predictor of mortality in dialysis patients, the conclusion that a low serum albumin concentration is simply due to a low-protein diet can be very misleading. The serum albumin concentration responds relatively slowly to changes in protein stores because the half-life of albumin is approximately 20 days. The serum concentration of prealbumin has a half-life of about two days and therefore changes more rapidly with variations in nutritional status. However, the problem of other factors causing changes in serum albumin levels (e.g., inflammation) undoubtedly applies to serum prealbumin. Compared with serum albumin, prealbumin concentrations in hemodialysis patients appear to have a special advantage as markers because they are more highly correlated with complications.

Serum transferrin

Not a good indicator of nutritional status as serum levels increased in iron deficiency, and reduced levels occur in uraemia per se, during an acute phase response, iron loading, and in patients with proteinuria.

Technical investigations (bioimpedancemetry, dual X-ray absorptiometry, near-infrared reactance)

Protein Metabolism

Several treatment-related conditions predispose chronic dialysis patients to negative protein balance. There are inevitable losses of amino acids during both hemodialysis (HD) and peritoneal dialysis (PD), ranging from 5 to 8 g of amino acids per HD session and 5 to 12 g/day of amino acids during PD. Losses may be higher with high efficiency HD or when peritonitis is present.

Chronic kidney disease patients have well-defined abnormalities in their plasma and to a lesser extent in their muscle amino acid profiles. Commonly, essential amino acids concentrations are low and nonessential amino acid high. An important influence is the progressive loss of kidney tissue, where metabolism of several amino acids takes place. Specifically, glycine and phenylalanine concentrations are elevated whereas serine, tyrosine, and histidine concentrations are decreased. Plasma and muscle concentrations of branched-chain amino acids (valine, leucine, and isoleucine) are reduced in chronic dialysis patients. Among these, valine displays the greatest reduction. In contrast, plasma citrulline, cystine, aspartate, methionine, and both 1- and 3-methylhistidine levels are increased.
Energy Metabolism

Patients with kidney failure have a less well-defined minimum energy requirement, which is dependent on the resting energy expenditure, the activity level of the patient, and other ongoing illnesses. Resting energy expenditure is elevated in chronic dialysis patients compared with normal controls matched for age, sex, and body mass index, and is further increased during the HD procedure when catabolism is at maximum due to amino acids losses.8

Recent studies suggest that REE in patients receiving HD therapy is greater than in control subjects, greater during dialysis than before and after dialysis, and greater during dialysis days than nondialysis days. Most recent report also concluded that energy expenditure is greater in patients receiving HD therapy than in patients with CKD stage 1 to 4.13

Lipid Metabolism8

Dyslipidemia is quite common in CKD patients, and abnormalities in lipid profiles can be detected in patients once kidney function begins to deteriorate, suggesting that uremia is associated with lipid disorders.

In HD patients, the most common abnormalities are elevated serum triglycerides and very-low-density lipoproteins, and decreased low-density and high density lipoproteins (LDLs, HDLs). The increased triglyceride component is thought to be related to increased levels of apolipoprotein CIII, an inhibitor of lipoprotein lipase. A substantial number of chronic HD patients also have elevated lipoprotein (a) (lp[a]) levels. Patients on PD have higher concentrations of serum cholesterol, triglycerides, LDL cholesterol, and apolipoprotein B, even though the mechanisms that alter the lipid metabolism are similar to those seen in chronic HD patients. Patients on PD also have higher concentrations of lp(a).

Large cross-sectional studies have identified that low rather than high cholesterol concentrations are associated with an increased risk of mortality in chronic dialysis patients. In contrast, a large multicenter study showed that, in a cohort of diabetic patients on HD, those who died from a cardiovascular event had higher median cholesterol, LDL cholesterol, LDL/HDL ratio, and apolipoprotein B concentrations at the time of initiation of dialysis.

Sodium Metabolism9

As CKD progresses, the ability to excrete the daily dietary sodium intake diminishes. Sodium retention occurs in most forms of CKD and contributes to the development of hypertension and hence progressive end-organ hypertensive injury. In dialysis patients excess sodium intake is associated with higher interdialytic weight gains and intradialytic hypotension as a result of the need for ultrafiltration of large fluid volumes over a relatively short time interval.

Potassium Metabolism9
Hyperkalemia is usually uncommon until the GFR falls to less than 20 mL/minute in the absence of distal tubular dysfunction (hyperkalemic distal renal tubular acidosis) or agents that block distal potassium secretion (e.g., angiotensin-converting enzyme inhibitors or nonsteroidal anti-inflammatory drugs).

**Calcium and Phosphorus Metabolism**

In early renal failure, serum calcitriol levels fall, and secondary hyperparathyroidism is nearly universal. As renal failure progresses, a reduction in parathyroid expression of vitamin D receptor as well as calcium receptor renders these glands resistant to both calcitriol and calcium. Dietary phosphorus further increases parathyroid hyperplasia in addition to parathyroid hormone synthesis and secretion.

**Vitamin, and Trace Element Metabolism**

Vitamin deficiencies may occur in renal failure. Once the patient is receiving dialysis, water-soluble vitamin losses may accelerate, particularly those of vitamin B₁ (thiamine). The requirements for fat-soluble vitamins are even more difficult to establish than the requirements for water-soluble vitamins. It has also been suggested that fat-soluble vitamins may participate in some of the complications of kidney failure.

Vitamin A concentrations are usually elevated in chronic dialysis patients, and intake of even small amounts leads to excessive accumulation. Vitamin E levels in chronic dialysis patients are not well defined, and there have been reports of increased, decreased, or unchanged concentrations. Vitamin K supplementation is usually not recommended in chronic dialysis patients unless they are at high risk for developing vitamin K deficiency. It is important to recognize that the daily requirements of vitamin B₆, folic acid, and ascorbic acid are often increased in chronic dialysis patients.

Serum aluminum is the most important trace element in these patients, because elevated levels have been shown to be associated with dialysis dementia as well as bone disease.

**NUTRITION THERAPY FOR CHRONIC KIDNEY DISEASE**

The goals of nutritional management for patients with CKD are the following: (1) prevention of malnutrition, (2) limitation of nitrogenous waste accumulation, (3) normalization of metabolic disturbance, (4) prevention of progressive renal disease, and (5) minimization of cardiovascular risk.

**Protein**

Dietary protein restriction has been recommended as a therapeutic approach for retarding the progression of CKD. The results of several recent studies on this subject are conflicting. The results of the largest clinical trial, the Modification of Diet in Renal Disease (MDRD) study, did not demonstrate a benefit of dietary protein restriction on progression of kidney disease. On the other hand, three meta-analyses indicate that such diets may be beneficial in slowing the progression of disease, albeit only to small extent. If such diets are to be used, it is important to assure that patients are not at risk for malnutrition.
For individuals with a GFR less than 25 mL/minute who are not undergoing maintenance dialysis, a low protein diet that provides 0.60 g protein/kg body weight/day should be considered. However, if the individual cannot tolerate such a diet or is unable to maintain an adequate intake of calories, then the individual should eat up to 0.75 g protein/kg body weight/day. Application of dietary protein restriction varies widely among practitioners. For a stable, well-nourished patient who has an established progressive kidney disease (CKD stages 1 to 4), a daily protein intake equivalent to the Recommended Dietary Allowance (RDA) - 0.8 g protein/kg body weight/day - is reasonable. For a patient who is obese or underweight, the amount of protein should be based on an appropriately defined body weight.

Amino acid and protein losses occur with dialysis, requiring a higher intake than that needed for healthy adults. Current protein recommendations for patients receiving HD or PD therapy are 1.2 g protein/kg body weight/day.

**Calories**

Calorie needs depend upon equations derived from direct and indirect calorimetry. The Body Mass Index (BMI) could be used to identify the appropriateness of weight for height. Total calorie needs can be then adjusted for weight goals (i.e., gain, loss, or maintenance), metabolic status (anabolic or catabolic), and activity level.

For individuals with a GFR less than 25 mL/minute who are not undergoing maintenance dialysis, the recommended intake is 35 kcal/kg body weight/day for those younger than 60 years and 30 to 35 kcal per kg for individuals who are 60 years and older.8,13

The recommended daily energy intake for maintenance haemodialysis or chronic peritoneal dialysis patients is 35 kcal/kg body weight/day for those who are less than 60 years of age and 30-35 kcal/kg body weight/day for individuals 60 years or older.

**Carbohydrate**

Several organizations have recently published new or updated dietary guidelines referred to as Therapeutic Lifestyle Changes (TLC) in response to growing evidence that suggests that nutrition has a positive role to play in preventing disease. Some common guidelines include increased consumption of fresh fruits and vegetables and whole grains and a reduced intake of total fat, saturated fatty acids, and cholesterol. Applying these guidelines translates into recommendations that carbohydrates provide 50% to 60% of calories and fats provide 25% to 30% of total calories. These recommendations should be considered for patients with CKD, because of the high incidence of CVD in this patient group. The Lifestyle or TLC guidelines are compatible with CKD diets, because the recommendations include reduced intake of red meat.

**Fat**

The fatty acid composition of the fat also plays a role in CVD prevention. Saturated and transfatty acids are known to modify serum lipoprotein patterns toward a pattern that is associated with CVD risk. Fatty acid
recommendations have been to keep saturated fatty acid intake at less than 10% of total calories and linoleic acid (n-6 polyunsaturated fatty acids) as 5% to 10% of total calories. Perunicic-Pekovic GB et al. showed a beneficial effect of ω-3 fatty acids on inflammatory markers, nutritional parameters and lipid profiles of patients on dialysis.  

Although no specific guidelines are currently available for patients with CKD, applying these fatty acid recommendations to stable, well-nourished patients in order to potentially lower CVD risk is reasonable. The US Food and Drug Administration (FDA) has identified 3 g/day of ω-3 fatty acids as the amount Generally Recognized as Safe (GRAS). This guideline can be applied to the CKD diet by adding cold-water fish, a main dietary source of ω-3 fatty acids, twice a week in lieu of red meat or poultry.

**Sodium and Fluid**

Because of the complex interactions between dietary sodium, hypertension, CVD, and CKD, the NKF-K/DOQI Clinical Practice Guidelines on Hypertension and Antihypertensive Agents (2004) recommends a dietary sodium intake of less than 2.4 g/day for most patients with CKD. Individualized modification is needed if the patient has a sodium-wasting disease or is prescribed medications that cause sodium loss.

The recommended sodium intake for patients receiving HD therapy is usually 2 g per day. This intake complements the fluid restriction required for patients with anuria (500 mL or more of urine per day) of 1000 mL per 24 hours. Because of the continual nature of PD therapy and the osmotic nature of the glucose-containing dialysate solutes, sodium and fluid losses occur. Therefore, restricting sodium (less than 3 g/day) and fluid is typically unnecessary.

Fresh foods such as cereals, vegetables, fruit, meat and dairy products contain plenty of sodium. Here are some tips to reduce the amount of sodium:

1. Choose low salt foods
2. Do not add salt at the table or in the cooking
3. Clearing up myths about salt
4. Count the salt

Here are some tips to control the fluid intake:

1. Use jugs and measuring cups to accurately measure the fluid intake
2. Spread the fluid allowance over the day - don’t drink it all at once
3. Drink from small rather than large cups
4. Limit salt and salty foods as these make thirsty.
5. Choose foods low in salt
6. Try mints, peppermints or chewing gum
7. Brush teeth to freshen mouth
8. Suck on lollies or a slice of lemon instead of drinking
9. Freeze some of fluid allowance.

**Potassium**
Dietary intake of potassium is not restricted in patients with CKD unless potassium retention, the need to prescribe potassium-retaining medications, or both are evident. The NKF-K/DOQI suggest a potassium intake greater than 4 g/day for stages 1 to 2 and ranging between 2 and 4 g of potassium per day for CKD stage 3 to 4.

Whereas dialysis therapy removes potassium from the serum, dietary potassium intake determines how much potassium accumulates between treatments. The usual dietary potassium prescription for patients receiving HD therapy is 2 g/day. Patients receiving PD therapy are treated with greater frequency and are therefore allowed a more liberal prescription, typically in the range of 4 g daily.

**Phosphorus and Calcium**

The recommended dietary phosphorus intake is 800 to 1000 mg per day. Restricting dietary phosphate, prescribing phosphate binders, and ensuring adequate dialysis are all necessary to maintain serum phosphorus levels between 2.5 and 5.5 mg/dL, when the plasma levels of parathyroid hormone (PTH) are elevated, or when both levels are elevated. Serum levels of phosphorus are controlled by diet modification and medication. Total dietary intake of calcium has become a concern because of the evidence that an excessive calcium intake can exacerbate vascular and other extraskeletal calcifications. The amount of dietary calcium that can be labeled as excessive in the population of patients with CKD has not to date been defined by any research. The RDA for calcium is 1200 mg (for both men and women aged 19 to 75 years).

The K/DOQI recommendations specify that the total dose of elemental calcium resulting from the use of calcium-based phosphate binders should not exceed 1500 mg per day and that the total calcium intake, from dietary sources and phosphate binders combined, should not exceed 2000 mg per day. Calcium-containing phosphate binders are not recommended for patients undergoing dialysis who are hypercalcemic, defined as corrected serum calcium greater than 10.2 mg/dL.13

**Vitamin and Trace**

High doses of vitamin C may result in elevated plasma levels of oxalate. Supplementation should not exceed 75 to 90 mg/day. Supplementation with fat-soluble vitamin A is not recommended. Requirements for fat-soluble vitamins E and K are thought to be similar to those of general population.

Vitamin formulations containing high-dose folic acid have become available for the patient undergoing dialysis in response to the literature suggesting that hyperhomocysteinemia is linked to CVD and poor outcome in this patient population, supplements in the range of 1 to 5 mg folate per day may be prudent. In addition, vitamin supplements containing vitamin E, selenium, and zinc have also become available.13

Recommendations for providing trace element supplements for uremic patients are controversial for several reasons; it is very difficult to determine whether body stores are sufficient, insufficient, or excessive, and it is difficult to prove that symptoms are reversed solely by the administration of trace elements.9

### Table 2
Selected nutritional parameters for varying levels of kidney failure

<table>
<thead>
<tr>
<th>Nutritional parameter</th>
<th>Normal kidney function</th>
<th>Stage 1–4 Chronic kidney disease</th>
<th>Stage 5 Hemodialysis</th>
<th>Stage 5 Peritoneal dialysis</th>
<th>Transplant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calories (kcal/kg/d)</td>
<td>30-37</td>
<td>35 &lt; 60 yrs</td>
<td>35 &lt; 60 yrs</td>
<td>35 &lt; 60 yrs</td>
<td>30-35 initial</td>
</tr>
<tr>
<td></td>
<td>30-36 ± 60 yrs</td>
<td>30-36 ± 60 yrs</td>
<td>30-36 ± 60 yrs</td>
<td>30-36 ± 60 yrs</td>
<td>25-30 for maintenance</td>
</tr>
<tr>
<td>Protein (g/kg/d)</td>
<td>0.8</td>
<td>0.6-0.75</td>
<td>1.2</td>
<td>1.2-1.3</td>
<td>1.3-1.6 initial</td>
</tr>
<tr>
<td></td>
<td>50% HBV</td>
<td>50% HBV</td>
<td>50% HBV</td>
<td>50% HBV</td>
<td>1.0 for maintenance</td>
</tr>
<tr>
<td>Fat (% total kcal)</td>
<td>30%–35%</td>
<td>Patients considered at highest risk for cardiovascular disease; emphasis on PUFA, MUFA; 250–300 mg cholesterol/day</td>
<td>&lt;10% saturated fat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium (mg/d)</td>
<td>Unrestricted</td>
<td>2,000</td>
<td>2,000</td>
<td>2,000</td>
<td>Unrestricted; monitor medication effect</td>
</tr>
<tr>
<td>Potassium (mg/d)</td>
<td>Unrestricted</td>
<td>Correlated to laboratory values</td>
<td>2,000–3,000</td>
<td>3,000–4,000</td>
<td>Unrestricted; monitor medication effect</td>
</tr>
<tr>
<td>Calcium (mg/d)</td>
<td>Unrestricted</td>
<td>≤ 2,000 from diet and medications</td>
<td>≤ 2,000 from diet and medications</td>
<td>1,200</td>
<td></td>
</tr>
<tr>
<td>Phosphorus (mg/d)</td>
<td>Unrestricted</td>
<td>Correlated to lab values</td>
<td>800–1,000</td>
<td>800–1,000</td>
<td>Unrestricted unless indicted</td>
</tr>
<tr>
<td>Fluid (ml/d)</td>
<td>Unrestricted</td>
<td>Unrestricted with normal urine output</td>
<td>1,000 + urine output</td>
<td>1,000 + urine output Monitored; 1,500–2,000</td>
<td>Unrestricted unless indicted</td>
</tr>
</tbody>
</table>

*Unrestricted* indicates any intake is possible within normal limits; *unlimited* indicates no dietary recommendation is made.

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*a* Mean as guidelines only for initial assessment; individualization to patient’s own metabolic status and co-existing metabolic conditions is essential for optimal care.

*b* HBV-high biological value.

*c* PUFA-polyunsaturated fatty acids.

*d* MUFA-monounsaturated fatty acids.
STRATEGIES FOR TREATMENT OF MALNUTRITION IN KIDNEY FAILURE PATIENTS

Repetitive comprehensive dietary counseling by an experienced dietitian is an important step to improve dietary intake; another is detection of early signs of malnutrition. For cases in which dietary counseling to improve nutritional status is unsuccessful, other forms of supplementation such as enteral (including oral protein, amino acid, and energy supplementation; nasogastric feeding tubes; percutaneous endoscopic gastrostomy or jejunostomy tubes) and intradialytic parenteral nutrition (IDPN) may be considered.\(^8\)

Several reports have emphasized the effective use of IDPN as a potential therapeutic intervention in malnourished chronic dialysis patients. Pupim et al. suggest that IDPN acutely improves net protein synthesis and increases albumin fractional synthetic rate.\(^6\) Chertow et al. in a retrospective analysis of more than 1500 chronic HD patients treated with IDPN, decreasing risk of death with the long-term use of IDPN was reported, particularly in patients with serum albumin concentrations below 3.5 g/dL and serum creatinine concentrations below 8 mg/dL.\(^17\)

Enteral nutrition (EN) by means of oral nutritional supplements (ONS) and tube feeding (TF) offers the possibility of increasing or ensuring nutrient intake in cases where normal food intake is inadequate. These guidelines are intended to give evidence-based recommendations for the use of ONS and TF in nephrology patients.

Patients with acute renal failure (ARF) and critical illness are characterized by a highly catabolic state and need depurative techniques inducing massive nutrient loss. EN by TF is the preferred route for nutritional support in these patients. EN by means of ONS is the preferred way of refeeding for depleted conservatively treated chronic renal failure patients and dialysis patients.\(^18\)
BIBLIOGRAPHY
