Nutrition Management in Hemodialysis

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Katrina Campbell, Megan Rossi and Juan Jesus Carrero

5.1 Introduction

Dietary intervention is a cornerstone strategy in the management of end-stage kidney disease (ESKD). In fact, during the 1960s, before dialysis was accepted as a regular form of renal replacement therapy, many patients were treated with diet alone. The role of the kidneys includes the elimination of metabolic waste products as well as maintenance of fluid, electrolyte, and hormone homeostasis. Thereby, ESKD requiring renal replacement therapy is associated with a range of metabolic and nutritional issues. Undergoing hemodialysis treatment, where only partial replacement of renal function is possible, the resulting metabolic and nutritional consequences require a range of management approaches.

Potentially significant dietary changes are necessary for patients undergoing hemodialysis treatment. The overarching goals for the nutritional management in hemodialysis include:

1. Optimizing nutritional status, including prevention and treatment of protein-energy wasting (PEW) and correction of nutrient deficiency
2. Management of electrolyte and fluid balance

This chapter will briefly address the range of factors that affect nutritional status in ESKD, including the prevalence, methods of assessment, and management of the following issues:

- PEW
- Electrolyte disturbance
- Fluid balance
- Vitamin and mineral deficiencies

5.2 Protein-Energy Wasting in Hemodialysis

Protein Energy Wasting (PEW) refers to nutritional problems related to altered protein and energy metabolism. This is influenced by two major factors. The first factor is an imbalance between protein and energy intake and requirements, attributed to inadequate intake. The second factor is the catabolic processes associated with dialysis and metabolic consequences of end-stage disease (including inflammation and oxidative stress) resulting in accelerated breakdown of protein stores. In clinical practice, it may be difficult to separate these two processes, which work synergistically while exacerbating one another (Fig. 5.1).

The following section will address the prevalence, etiology, and methods of assessment and management of PEW in hemodialysis.

5.2.1 Prevalence of PEW and Effect of PEW on Outcome in Hemodialysis

PEW remains a common issue even in the modern day dialysis patient. As illustrated in Fig. 5.1, approximately 20–60 % of patients undergoing hemodialysis around the world may have PEW [1–9]. Importantly, we should bear in mind that the actual prevalence may be higher, as these data are from observational studies that include only those patients who are clinically stable.

Nutrients are the substrates for energy, tissue synthesis, and metabolism, and are necessary for life. Undernutrition and/or micro/macronutrient deficiencies specifically are contributors to the metabolic complications and poor outcomes of hemodialysis patients. Most markers of PEW have been

M. Rossi (✉)
School of Medicine, University of Queensland, Brisbane, QLD, Australia
e-mail: megan.rossi@uq.net.au

K. Campbell
Department of Nutrition & Dietetics, Princess Alexandra Hospital, Woollongabba, QLD, Australia

J. J. Carrero
Department of Renal Medicine, Karolinska Institute, Huddinge, Sweden

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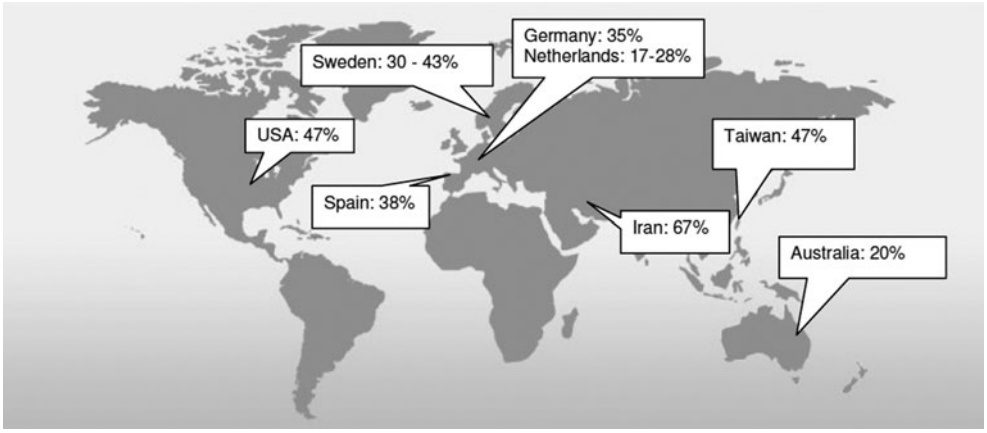

Fig. 5.1 Prevalence of protein-energy wasting in hemodialysis populations throughout the world

associated with poor quality of life, infections, atherosclerosis, cardiovascular events, graft rejection, and mortality [1, 2, 10–12]. Simple markers of nutritional status, such as serum albumin, serum prealbumin, and poor appetite, are strongly associated with the incidence of hospitalizations [13, 14], which impacts health-care costs. Health-care costs for PEW hemodialysis patients have been suggested up to threefold as compared with non-PEW individuals [13]. Although we currently lack of randomized controlled trials targeting PEW to reduce hard outcomes in hemodialysis patients, three large epidemiological analyses have explored this issue based on the potential of providing oral nutritional support to hypoalbuminemic patients. In one study, hypoalbuminemic individuals receiving nutritional support had a 34% reduction in 1-year mortality risk as compared to those who did not receive it [15]. Nutritional support in persistently hypoalbuminemic hemodialysis patients reduced hospitalizations rates during the subsequent year by approximately 20% [16] versus those who did not receive it. Implementation of a protocol to provide nutritional support during hemodialysis upon diagnosis of hypoalbuminemia and to maintain albumin within normal range associated with 20–30% reduced mortality as compared to similar patients not receiving nutritional support. Despite these reports being observational in nature, they provide solid background regarding the importance of ensuring good nutritional status in hemodialysis patients.

PEW has short-term impact on mortality, and its consequences are so rapid and devastating that in epidemiological studies things that are normally risk factors appear as protective. A clear example is the association between cholesterol and mortality. In hemodialysis patients, a high- rather than a low-level of cholesterol associates with improved survival [17], which is opposite to the effect observed in the general population. When chronic kidney disease (CKD) patients are stratified according to the presence/absence of PEW, it is observed that this mortality paradox is seen only in people with signs of PEW [17]. The explanation is likely that patients undergoing hemodialysis die of the short-term consequences of PEW and do not live long enough to die of cardiovascular disease associated with high cholesterol. In this case, high cholesterol may actually be a sign of higher fat stores that allows the patient to survive the wasting process longer. A similar paradox has been reported repeatedly for obesity [18]; dialysis patients are at such high risk of PEW that obesity may provide a measure of protection by excess energy store to stand the PEW catabolic process. Hyperhomocysteinemia, an important cardiovascular risk factor in the general population, has also been associated with improved survival in hemodialysis patients [19]. Again, homocysteine levels in this setting may be a reflection of overall better amino acid stores.

5.2.2 Etiology of PEW Is Multifactorial in ESKD

There are a wide range of drivers that affect the nutritional and metabolic state in CKD (Fig. 5.2). Understanding the features that contribute to the etiology of PEW is critical to inform appropriate assessment and treatment strategies. Not all of these alterations are directly or fully tackled by adequate nutritional support and will not be discussed in this chapter. These include, for instance, inflammation-induced hypercatabolism, increased energy expenditure, hormonal disorders (such as insulin resistance or growth hormone alterations), and poor physical activity and/or frailty. A multifaceted therapeutic approach for this complex syndrome is therefore necessary.

5.2.2.1 Appetite

Reduced appetite in ESKD is an independent predictor of poor outcome [10, 20] and important contributor of PEW, as a result of driving an inadequate dietary intake. Appetite disturbance present in ESKD is generally reported between 35 and 50% of hemodialysis patients from samples in Europe and the USA [10, 12, 20–22]. Appetite is typically driven by the endocrine system; however, in hemodialysis patients, factors related to the dialysis procedure, alterations in the gastrointestinal system, as well as hedonic and social implications are also important to consider.

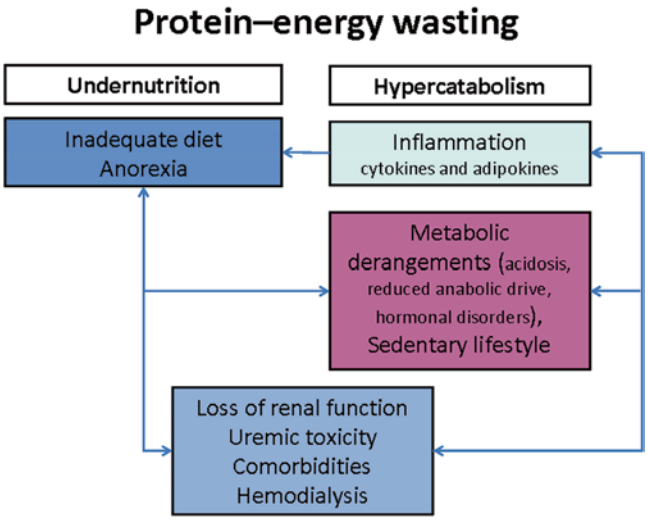

Fig. 5.2 A simple overview of etiology of protein-energy wasting in dialysis

Appetite hormones and neuropeptides serve to regulate hunger to respond with adequate energy intake; however, their actions are altered in ESKD. Studies indicate that patients undergoing dialysis treatment who exhibit appetite disturbance show signs of slower eating and report higher ratings of fullness prior to meals compared with controls [23]. This response has been associated with high circulating levels of anorectic hormones (cholecystokinin (CCK), leptin and peptide-YY (PYY)) [24, 25] and stimulation of serotonin [26]. To add to this picture of dysregulation, ghrelin, an appetite-stimulating hormone, appears to have reduced function in ESKD [27]. These appetite hormones, which are typically cleared by dialysis, peak prior to a dialysis session, resulting in reduced appetite leading up to dialysis [28]. Therefore, we see a typical cycling of appetite along dialysis days with reduced appetite being common before dialysis session [29].

5.2.2.2 Effect of Hedonic Drivers of Food Intake

The food and drink "experience" or hedonic factors driving appetite and food intake may be negatively influenced by a range of disturbances that manifest in ESKD, both physiological and psychological. CKD changes both smell and taste functions, thereby reducing the ability to detect basic tastes for salt and bitter, as well as reducing taste sensitivity compared with healthy controls [30–32]. Taste is thought to be affected in CKD patients by a range of factors, including reduced saliva volume and altered composition, as well as reduced neural function resulting in impaired activity of taste receptors [33]. Other oral manifestations including a high prevalence of oral disease, increased uremic by-products, buffering, and reduced salivary flow rate increase erosion and malocclusion [34]. Such dental problems may create chewing or biting problems, interfering with the ability to consume a variety of nutrient-dense foods [35]. Hedonic experience can also be influenced in the setting of hemodialysis by a range of psychological factors, including anxiety due to past (or present) food restrictions or coping with the disease, and presence of depression, which has been demonstrated to be a strong driver of appetite in hemodialysis patients [21]. These together with a range of social issues, including food security [36] and social isolation [37], dialysis patients experience a range of factors that influence their food experience and therefore it is important to consider them in the context of nutritional management.

5.2.2.3 Gastrointestinal Disturbance

Gastrointestinal symptoms are also potential contributors to PEW observed in ESKD. Prevalent conditions in dialysis patients include constipation, impaired gastric emptying, and motility disorders [38–41]. The pathogenesis of these disorders is largely unknown; however, it may be related to bacterial overgrowth in the small [42] and large [43] intestines. This state of "dysbiosis" has been hypothesized in ESRD as a driver of increased inflammation and anorexia [43]. In relation to this, comorbid diabetes may also increase the risk of diabetic gastroparesis, resulting in delayed gastric emptying, nausea, and prolonged satiety [44]. Nonetheless, the prevalence of gastrointestinal symptoms in ESKD patients with diabetes does not appear to be any different to the remaining ESKD population, although the studies are few [39, 40].

5.2.2.4 Inflammation

Inflammation is a major contributor to PEW and cardiovascular disease in dialysis [45, 46]. ESKD is characterized by persistent low-grade, inflammatory state [47]. Increased concentration of inflammatory cytokines and adipokines are due to both reduced renal clearance and stimulation of increased production [48]. Furthermore, factors that have been hypothesized to promote a state of chronic inflammation in dialysis patients include membrane bio-incompatibility, comorbid conditions, persistent infection, diet, and genetic factors [48].

Inflammation contributes to PEW as a driver for appetite dysregulation and protein catabolism. Key inflammatory cytokines trigger both central and peripheral mechanisms to drive appetite regulation [49]. High concentrations of each of these cytokines have been reported in the dialysis population and are associated with uremic anorexia [10, 50, 51]. Furthermore, muscle wasting is a significant consequence of chronic inflammation [52, 53]. The action of IL-6 as a result of muscle proteolysis appears to stimulate further protein catabolism [54]. Therefore, the synergistic action of poor appetite and increased muscle wasting resulting from the inflammatory cascade represents a key mechanistic driver of PEW. Treatment targeting the source of inflammation (i.e., optimizing dialysis therapy, including access and prescription,

appropriate fluid management, etc.) is critical with nutrition interventions having limited success in isolation.

5.2.2.5 Dialysis Procedure

The hemodialysis procedure in itself is a catabolic stimulus: Interaction between the blood flowing through the dialysis membrane gives rise to an inflammatory cascade, which appears to be dependent on the dialysis membrane used [55]. Furthermore, inflammatory stimuli include limited clearance of uremic toxins, particularly protein-bound uremic toxins, along with increased gut ischemia leading to increased endotoxemia. Amino acid and protein losses during the dialysis session, together with low nutrient intake, promote low nutrient availability for muscle synthesis and acute-phase reactant synthesis [56–58]. The consequence is breakdown of muscle protein to compensate for these losses [59, 60]. Concurrent amino acid supplementation during the dialysis session can prevent or reverse these adverse effects [61, 62]. Furthermore, optimizing dialysis provision and/or increasing the frequency of the dialysis procedure has been associated with improvements in nutritional markers [63, 64]; however, this has not been confirmed in a subsequent randomized trial [65]. Finally, hemodialysis results in a more rapid loss of residual renal function, which has been shown to relate to rates of malnutrition [66]. Proposed mechanisms for this include reduced regulation of amino acid metabolism, particularly conversion of essential amino acids (phenylalanine to tyrosine and glycine to serine), thereby limiting the amino acid profile available for protein synthesis [67] .

5.2.2.6 Metabolic Acidosis

Metabolic acidosis is a common consequence of the reduced buffering capacity of the kidney in ESKD and an important contributor to net protein catabolism and uremic anorexia. Correction of acidosis has shown to improve nutritional status [68], likely through decreased protein turnover, improved appetite, and total protein intake. The mechanism of action through decreased protein degradation has been demonstrated in both hemodialysis [69] and peritoneal dialysis [70].

5.2.3 Assessment of Protein-Energy Wasting in Hemodialysis

Systematic screening and assessment of nutritional status is essential in the management of hemodialysis patients. The key goal of this process is to identify potential nutrition risk early (screening) and undertake thorough assessment in order to form a diagnosis of PEW and indicate targets for intervention, evaluation, and monitoring [71]. An ideal nutrition assessment tool should not only predict outcome, but also respond to nutritional therapy, without being affected by nonnutritional factors. In addition, nutrition assessment in hemodialysis must be easily applied in practice, preferably achievable during or soon after the dialysis treatment.

However, there is not a single measure that can provide a valid assessment of nutritional status; therefore, nutrition assessment is based on a combination of measures. Nutritional laboratory biomarkers are and can be influenced by uremic retention (and conversely residual renal function), fluid status, inflammation (as many nutritional markers also function as acute-phase reactants), and renal replacement therapy (losses into dialysate). Anthropometry and body composition tools are affected by fluid status. Careful consideration of all these confounding factors must be given before making a diagnosis. Given that drivers of PEW are complex and multifactorial, parameters for assessment therefore need to capture a range of measures, including body composition, biochemical parameters, and dietary intake [72]. An overview of nutrition assessment parameters is provided in Table 5.1.

5.2.3.1 Anthropometry and Body Composition for PEW Assessment

Monitoring of weight and body composition is useful to identify depleted fat and/or muscle stores; however, the precision is dependent on the tool used [73]. In general, the most clinically applicable tools are the least precise. For example, assessment of weight and weight change is a standard routine practice in the dialysis setting. Weight gain or loss is influenced by fluctuations in body water related to breaks in dialysis therapy; however, long-term trends of adjustments to "dry" or target weight may provide insights into actual weight change. Even when the weight change is established, it is not known the degree of weight loss from muscle wasting, compared with fat mass. Anthropometric measures including skinfold thickness (in particular, triceps and biceps) and circumferences (typically mid-arm) are also applicable to routine care and may be used together with weight to identify where weight changes may be coming from. Handgrip strength is another clinically applicable tool that can be used to assess change in muscle function over time and has been shown to be a good predictor of outcome [74, 75].

More advanced methods, including body composition instruments, are more likely to be applied in a research situation rather than in daily practice in the hemodialysis setting. Dual X-ray absorptiometry, total body potassium, and total body nitrogen are generally isolated from the research setting due to their high cost and limited application to the clinical practice setting. Bioimpedance analysis (BIA) tools are becoming more common in the routine assessment of nutrition status in hemodialysis. BIA relies on several assumptions; it is important to use equipment validated for dialysis patients and to also account for consistent hydration status. As this is constantly variable in the hemodialysis patients, it is important to perform this measurement at a consistent timeframe, for example, 30 min after dialysis. Longitudinal

Table 5.1 Overview of parameters used in hemodialysis for assessing protein-energy wasting

Assessment tool	Ease of measurement	Clinical applicability	Considerations
Anthropometry and body composition			
Weight and weight change, including BMI	High	Moderate	Does not distinguish body compartments. Dry weight change of 5 % or more clinically applicable
Lean muscle mass (and/or fat mass) using body composition instruments	Low	High	Tools to assess directly are expensive and not clinically applicable (e.g., total body potassium, total body nitrogen); or open to error due to indirect measure and body water fluctuations (bioimpedance, DEXA)
Anthropometrics including skinfold thickness and mid-arm muscle circumference	Moderate	Moderate	Require training to optimize validity and reproducibility, low-cost and to be undertaken after dialysis session
Handgrip strength	High	Moderate	Measure of muscle function, non-invasive. Evaluation of longitudinal change required
Biochemistry			
Serum proteins	High	Low	Inverse relationship with inflammation and hydration status
Inflammation markers	Moderate	Moderate	Indicator of stress response, may decrease protein synthesis and raise energy expenditure
Nutrition assessment tools			
Subjective global assessment	High	High	Draw on a range of data from medical histories and physical examination to evaluate overall nutritional status
Malnutrition inflammation score			
Dietary intake			
Adequacy of protein and energy intake (diet history)	Moderate	High	For reliable data from detailed diet histories require skills and training, however, important to evaluate, given the high protein and energy requirements in hemodialysis
Adequacy of protein intake (PNA)	High	Moderate	PNA can be calculated by estimating the generation of urea nitrogen in blood. Assumes patient is metabolically stable

BMI body mass index, *DEXA* dual-energy X-ray absorptiometry, *PNA* protein of nitrogen appearance

changes from BIA have been used to predict body cell mass in dialysis patients [76] and were associated with morbidity and mortality [77].

5.2.3.2 Biochemistry for PEW Assessment

Biochemical parameters are commonly used to estimate dietary needs and to monitor nutritional status [78]. However, this assessment method requires caution in interpretation. In clinically stable hemodialysis patients, protein of nitrogen appearance (PNA) can be used to estimate protein intake. The total nitrogen appearance of the body should be equal to or slightly smaller than the nitrogen intake. Because urea nitrogen appearance is highly correlated with total nitrogen appearance and measurement of total nitrogen losses in urine, dialysate, and stool is inconvenient and laborious, regression equations to estimate PNA have been developed. In hemodialysis patients, PNA can be calculated by estimating the generation of urea nitrogen in blood [79], usually followed by normalization (nPNA) by body weight or body weight derived from the urea distribution space. nPNA assessment is recommended with a monthly frequency [79]. nPNA would not be a valid indicator of protein intake in cases of catabolism, growth/anabolism (children, pregnant women, recovering from an intercurrent illness), or day-to-day changes in dietary protein intake. PNA should not be used to evaluate nutritional status in isolation, but rather as one of several independent measures when evaluating nutritional status.

Synthesis of serum proteins commonly used to assess nutritional status (albumin, prealbumin, etc.) is directly impacted by inflammation. Therefore, there is a direct inverse correlation between serum proteins and serum inflammatory markers in dialysis patients [80], rendering the assessment of nutrition status using serum proteins problematic. A low serum albumin concentration is highly prognostic; however, it may not only reflect an acute-phase response, but also be the result of fluid overload and dialysate loss. This is also reflective of other serum proteins, including pre-albumin, transferrin, and retinol-binding protein. Inadequate dietary protein intake can affect serum protein in the short term, as it decreases the rate of serum protein synthesis [81]. However, longer term, compensatory shifts in serum protein from extravascular to intravascular space occur, thereby limiting the value of serum proteins for evaluating nutritional status. To overcome some of these limitations, it can be useful to evaluate inflammatory markers, such as C-reactive protein (CRP), and interdialytic fluid gains to assess the validity of these markers for predicting PEW.

Pre-dialysis serum bicarbonate can provide an indication of the etiology for PEW. Metabolic acidosis may lead to stimulation of protein breakdown and subsequent muscle wasting, indicated by low serum bicarbonate. However, in the event of both low and high pre-dialysis, bicarbonate may be indicative of PEW risk. When low, this may indicate severe malnutrition due to the lack of endogenous protein [82, 83].

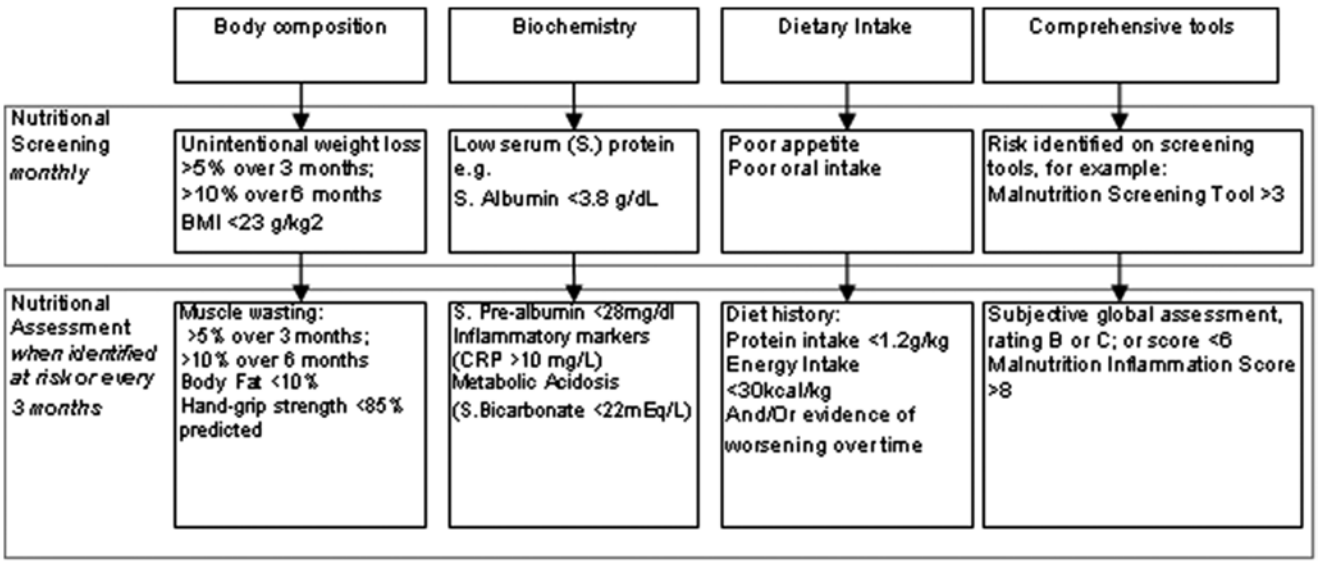

Fig. 5.3 Suggested nutrition screening and assessment parameters for use in hemodialysis

5.2.3.3 Nutrition Assessment Tools for PEW Assessment

The most comprehensive nutrition assessment tools to evaluate PEW in the hemodialysis setting include the subjective global assessment (SGA) and the malnutrition inflammation score (MIS) [84]. These tools combine features of a medical history (e.g., weight change, gastrointestinal symptoms, dietary intake change, functional capacity, and in the case of MIS, biochemistry) as well as a physical examination (accounting for fat and muscle wasting). SGA differs from MIS, by not requiring biochemistry, and is also based on a global rating rather than a summative score. Both tools have been shown to be prognostic indicators of clinical outcome, although may not be sensitive to detect small changes over time [85] .

5.2.4 Treatment of Protein-Energy Wasting

Once a nutrition screening and assessment process is in place (as detailed in Fig. 5.3), it is critical to be followed up by an appropriate management plan to treat PEW, or indeed prevent the exacerbated nutrition risk [71]. The recommended energy and protein requirements in hemodialysis are 35 kcal/kg/day (over 30 kcal/kg/day for > 60 years old) and over 1 g protein/kg/day [72, 86]. Most studies demonstrate that these targets are rarely met, particularly for protein. In the event of PEW, nutrition support is required. There are a number of different forms of nutrition support as outlined in Fig. 5.4.

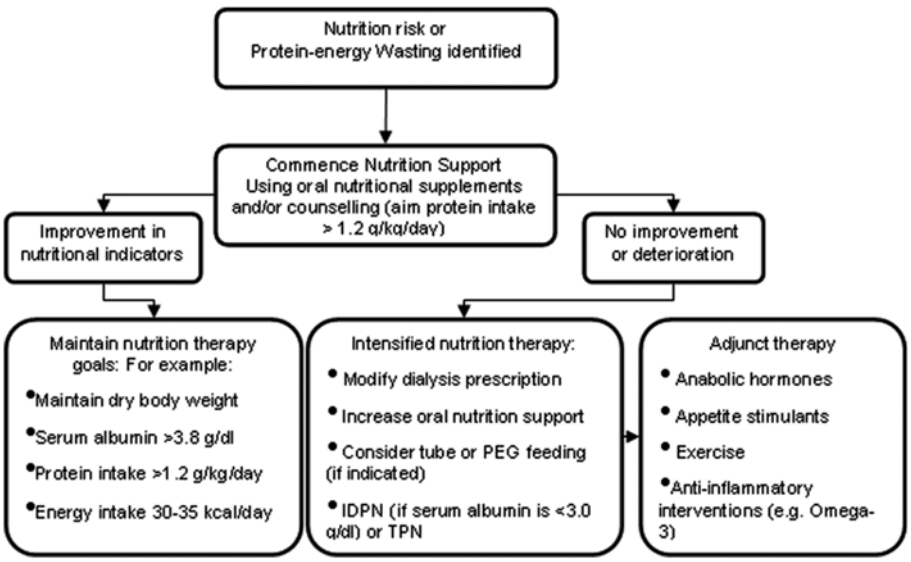

Fig. 5.4 Treatment of protein-energy wasting algorithm. (Adapted from [71])

5.2.4.1 Oral and Enteral Nutritional Supplements

Oral nutritional supplements (ONS) are considered a first-line treatment for PEW in hemodialysis. In addition to dietary counseling to optimize nutritional intake from food, ONS can provide an added 7–10 kcal/day and 0.3–0.4 g/kg protein/day [87]. Provision of ONS to dialysis patients has shown improvements in serum albumin, in the order of 0.23 g/dL [88]. Additional benefits observed have included increased body weight [89], lean body mass [90], global nutrition status, and quality of life (QOL) [91]. Recent large observational studies have demonstrated reduced hospitalizations [15] and improved survival [16] in hemodialysis patients in those who received ONS, compared with patients who did not.

ONS are best incorporated into routine intake away from main meals and/or provided during a dialysis session. Meals and ONS provided on dialysis have several benefits including improved protein turnover [61] and compliance, and should therefore be considered in all patients at risk of PEW [92].

Enteral nutrition, in the form of tube feeding, is an option for patients who are unable to tolerate sufficient oral intake. This involves nasogastric tubes (through nose to stomach), percutaneous endoscopic gastroscopy (PEG, direct to stomach), or jejunostomy tubes (through to the jejunum) [93]. Generally, tube feeding would be utilized in the situation of comorbid conditions impacting the nutritional status and/or functional oral intake, including dysphagia or severe anorexia.

5.2.4.2 Intradialytic Parenteral Nutrition

Intradialytic parenteral nutrition (IDPN) provides nutrition support during the hemodialysis procedure directly via the venous access. IDPN is considered when ONS have been tried and intake remains considerably inadequate (e.g., <20 kcal/kg/day) [89]. Formations of IDPN come in the form of multi- or single macronutrients (dextrose, amino acids, and/or lipids) and, therefore, may be somewhat individualized for the patients needs [71]. The effectiveness of this treatment over any other nutrition support option has yet to be demonstrated; however, it is a safe and convenient option for patients who cannot meet their needs orally.

5.2.4.3 Other Treatments

There are a range of other treatments that warrant consideration, including optimization of dialysis, use of appetite stimulants, and growth hormone. Appetite stimulants such as megestrol acetate have been evaluated in pilot randomized-controlled trials in maintenance hemodialysis patients [94, 95]. Although this agent has been shown to improve appetite and food intake, it has been associated with increase in body fat, not muscle mass, notwithstanding considerable side effects [96]. However, a pilot study in malnourished dialysis patients demonstrated improved energy balance with subcutaneous ghrelin administration [97]. Finally, small, short-term metabolic studies investigating the use of growth hormone in maintenance hemodialysis have demonstrated an indication for achieving positive nitrogen balance (reviewed in [71]). However, important consideration into side effects of growth hormone, including hyperglycemia and acromegaly, has prevented its approval for treatment of PEW in the maintenance hemodialysis population. This is an area which is likely to receive increasing attention, in addition to agents targeting inflammation and gut microbiota in the prevention and treatment of PEW in hemodialysis.

5.3 Electrolyte Disturbance

5.3.1 Hyperkalemia

Disturbance in potassium balance is a management challenge in kidney disease, in particular, for anuric patients receiving hemodialysis treatment. While a small percentage is chronically hypokalemic, hyperkalemia is by far the more common disturbance of potassium homeostasis. Hyperkalemia is potentially life threatening with muscular cells highly sensitive to changes in intracellular concentrations of potassium, precipitating muscle weakening, paralysis, and potentially fatal arrhythmias[98]. Hyperkalemia is a risk factor for sudden cardiac death, the leading cause of mortality in hemodialysis patients [99], and is associated with a twofold risk of all-cause and cardiovascular mortality [100].

There are a number of different causes of elevated serum potassium, many of which are not diet related. Common causes in the dialysis population include acute infection, medications such as angiotensin-converting-enzyme inhibitors and non-steroidal anti-inflammatory agents, and factors that may indirectly be related to suboptimal nutrition, including metabolic acidosis, increased catabolism, poor glycemic control, and constipation.

5.3.1.1 Assessment of Hyperkalemia

Hyperkalemia, categorized as mild or moderate (serum potassium 5.5–6.5 mEq/L) to severe (>6.5 mEq/L), is often asymptomatic and detection generally relies on biochemical tests, or electrocardiography, in the acute setting. An understanding of the underlying cause of hyperkalemia is needed when considering treatment options to avoid any unnecessary dietary restrictions in this population already at high risk of malnutrition. For instance, hyperkalemia can also occur in situations of underdialysis or alterations in the gastrointestinal (GI) tract (site of potassium elimination). Steroids, ACEIs, and potassium-sparing diuretics may raise potassium levels. Acidosis and hyperglycemia promote loss of intracellular potassium and raise potassium levels.

Table 5.2 Example of a simple potassium food guide

High (>5 mmol/serve[a])	Medium (3–5 mmol/serve[a])	Low (≤2 mmol/serve[a])
Fruit		
Banana	Pear	Canned fruit (drained)
Fruit mixes (fresh juice/dried)	Melon	Berries
Peach	Plum	Rhubarb
Vegetables		
Starchy vegetables	Broccoli	Asparagus
Tomato	Carrot	Peas
Avocado	Silver beet	Lettuce
Dairy		
Cow, butter and soy milk	Ice cream	Cheese
Yogurt	Creamed rice	Rice milk
Extra foods		
Iced coffee	Liquorice	Oatmeal/plain biscuits
Worcestershire sauce	Chocolate	Plain muesli bars

Unit conversion: 1 mmol potassium = 39 mg potassium
[a] Based on standard portion size

A diet history targeting sources of potassium is a method for determining whether diet may be the primary or a contributory cause of hyperkalemia. Identification of total potassium intake as well as the sources of high potassium foods is needed for targeted intervention. Food frequency questionnaires using a checklist of high potassium foods, as exemplified in Table 5.2, may also add to the dietary assessment. This technique may assist patient recall of high potassium foods consumed less frequently although potentially in high quantities, contributing to the unexplained occasional hyperkalemia.

Twenty-four-hour urine tests are another method of assessing potassium intake, although logistics including timeliness and patient burden limits its clinical applicability.

5.3.1.2 Management of Hyperkalemia

In the case of hyperkalemia where diet has been identified as a contributing factor, limiting intake of high potassium foods is recommended as the first-line intervention. This generally precedes medical treatments such as potassium exchange resins and changes to the concentration of the dialysis bath. As a guide, limiting potassium intake to 1 mmol/kg of ideal body weight through education on potassium sources and individualizing meal plans may help in the treatment or prevention of hyperkalemia.

Depending on the resources available, however, intervention can be as basic as providing patients with lists of high, medium, and low foods from each food group with the recommendation of avoiding foods from the "high" category. Only reputable food lists obtained from government agencies should be used, many of which are freely accessible and reviewed by qualified dieticians [101]. It is important that dialysis patients do not exclude any food groups from their diet (including fruit and vegetables), instead select the lower potassium options within each food group. Following this method limits the risk of malnutrition, nutrient deficiencies, and enhances patient satisfaction.

Individualized counseling with a qualified dietitian is the gold standard diet intervention for hyperkalemia. This management strategy allows recommendations to be tailored to patients' normal diet intakes, enhancing patient knowledge and compliance. In specific dialysis populations, generally younger patients, up-skilling using a potassium point system may be an effective strategy. Patients are given a daily potassium allowance (calculated based on 1 mmol/kg) and are educated on individual foods' potassium contents. This technique promotes patient autonomy, allowing patients to select how they use their daily allowance of potassium. Nonetheless, the lack of mandatory labeling for potassium on nutrition information panels is a major barrier for many patients.

Food preparation techniques including soaking and boiling have been shown to decrease the potassium content by up to 70% in some foods [102]. However, it is important to consider the loss of other water-soluble nutrients when recommending this technique.

5.3.1.3 Key Management Strategies

1. Dietary counseling
   a. Limiting foods from the high potassium category (see Table 5.2)
   b. Potassium point system (higher level knowledge)
2. Food preparation techniques
3. Potassium exchange resins
4. Adjusting concentration of dialysis bath

5.3.2 Hyperphosphatemia

The kidneys play a vital role in mineral metabolism, maintaining homeostasis between serum and tissue stores of essential minerals including phosphorus. The kidney's ability to excrete phosphorous is progressively compromised with deterioration in kidney function leading to hypophosphatemia and hormonal disturbances. This presents as CKD-mineral and bone disorder (CKD-MBD), which encompasses mineral, bone, and extra skeletal (vascular) abnormalities. Despite a lack of intervention studies linking phosphorous manipulation to clinical outcomes, the strength of observational and experimental data has warranted the development of guidelines for phosphorous control [103].

Table 5.3 Guideline recommendations for dietary intake on hemodialysis

Nutrient	Guideline recommendation
Energy [86]	35 kcal/kg 30–35 kcal/kg >60 years
Protein [86]	For clinically and weight stable patients aim for at least 1.2 g/kg of ideal body weight/day protein
Sodium [86]	Less than 2.3 g/day (or <100 mmol/day)
Fluid [86]	Target range: 500 mL plus previous day urine output
Phosphate [103]	Target range: < 1.6 mEq/L Phosphorus intake of 800–1000 mg/day and aiming for 10–12 mg/g
Potassium [86]	Target range: Potassium 3.5–5.5 mEq/L Low potassium diet: individualized, approximately 40 mg/kg IBW or adjusted weight [141]

5.3.2.1 Assessment of Hyperphosphatemia

Routine blood tests are used to measure phosphorous, with KDIGO guidelines recommending a target below 1.6 mmol/L (see Table 5.3). Test results should be based on trends rather than single laboratory values when determining the need for intervention. In the short term, significant elevation of phosphorous may present as severe itchiness, while long-term elevation can manifest in visible calcification deposits in bones and joints of extremities.

There are two main forms of dietary phosphorous, organic and inorganic phosphorous, which need to be targeted in diet history assessments (whether diet history records or food frequency questionnaires are employed). Sources of organic phosphorus include animal products such as dairy, meat, fish, and eggs, as well as plant foods such as whole grains, legumes, and nuts. Inorganic phosphorus is found primarily in processed foods in the form of food additives for a range of properties including anticaking, leavening, emulsification, flavor enhancement, and color and moisture retention. The phosphorus content of foods is determined not only by the total amount but also by the bioavailability of the phosphorous. Organic phosphorus from plant and animal sources is absorbed at a rate of 20–40 and 40–60%, respectively, while inorganic forms of phosphorus are thought to be absorbed between 90 and 100% [104] (see Table 5.4 for a list of common phosphorous-based food additives).

5.3.2.2 Management of Hyperphosphatemia

The National Kidney Foundation-Kidney Disease Outcomes Quality Initiative (NKF-KDOQI) guidelines and the European Best Practice Guidelines recommend daily phosphorus intake of 800–1000 mg/day for patients on maintenance hemodialysis therapy. However, intakes adjusted to dietary protein requirements (10–12 mg/g protein) may be more appropriate for patients with higher protein needs [105].

Dietary restrictions must be carefully recommended and followed up because limiting naturally rich phosphorus foods can increase the risk of undernutrition and low protein intake [106]. Restriction should be directed toward processed foods with phosphorous-based additives. This should be the first-line intervention because of the high bioavailability of the phosphorous additives in addition to the low nutrient density of most processed foods. Educating patients to identify phosphorous-based additives on the food ingredient lists is an effective strategy shown to lower serum phosphorous levels [107]. This strategy is becoming more important with the increasing use of phosphorus-based additives in the food supply [107]. A barrier to this strategy, however, is that phosphorus listing on the nutrition panel is not mandated. In addition, the ingredient list commonly reports additives as E-numbers instead of names in much of Europe and other non-US countries. This makes it difficult to determine which foods contain phosphorus additives. The name and E-number for each of the 18 commonly used additives are provided in Table 5.4 [108].

Often a simplified message of promoting home-cooked meals from fresh ingredients and limiting processed and takeaway foods is a more practical approach to achieve restriction of phosphorous additives. Food preparation techniques including boiling have been shown to decrease the phosphorus content considerably [109]. However, again, it is important to consider the loss of other water-soluble nutrients when recommending this technique.

The next line strategy is to ensure that a low phosphorus to protein ratio is adopted and/or dietary protein is not excessive (e.g., <1.5 g/kg, see dietary protein guidelines in Table 5.5). One strategy to balance the phosphorous intake without compromising on protein is to limit high phosphorus to protein ratio foods. Ideally, foods with ratios of 12–16 mg

Table 5.4 Common phosphorous-based food additives

E-number	Additive name	E-number	Additive name
101	Riboflavin	452	Polyphosphates
322	Lecithins	541	Sodium aluminum phosphate acidic
338	Phosphoric acid	627	Disodium guanylate
339	Sodium phosphates	631	Disodium inosinate
340	Potassium phosphates	635	Disodium 5′-ribonucleotides
341	Calcium phosphates	1410	Monostarch phosphate
343	Magnesium phosphates	1412	Distarch phosphate
442	Ammonium phosphatides	1413	Phosphated distarch phosphate
450	Diphosphates	1414	Acetylated distarch phosphate
451	Triphosphates	1442	Hydroxy propyl distarch phosphate

Table 5.5 Phosphorous-to-protein ratio of selected food items [2]

Food	Phosphorous-to-protein ratio
Seafood	
Orange roughy fish	4.5
Tuna, canned in water	6.4
Lobster	9.0
Salmon, sockeye	10.0
Crab, blue	10.2
Rainbow trout	11.0
Chicken egg	
Egg white	1.4
Egg substitute	10.1
Whole egg	13.3
Egg yolk	24.7
Meat	
Lamb	6.3
Beef (excludes organ meats)	7.0
Chicken breast	7.5
Pork (excludes organ meats)	9.3
Frankfurter, beef	14.1
Chicken liver	16.5
Dairy	
Cream cheese	16.7
Soymilk	17.4
Cheddar cheese	20.4
Milk, low fat (2%)	28.3

of phosphorous to 1 g of protein are recommended [110]. It is important to note, however, that more restrictive prescription of dietary phosphate is associated with poorer indices of nutritional status and, therefore, it is paramount that patients are given clear messages not to overrestrict protein intakes to achieve phosphate targets [111]. Phosphorous from plant sources, such as whole grains, is not essential to restrict due to the importance of their dietary fiber, vitamin and mineral content, and the low bioavailability of plant-based phosphorus. Suggestions of typically ingested foods according to phosphorus/protein content are listed in Table 5.5.

Despite optimal dietary management, phosphate binders remain a common adjunct therapy. There are different types of phosphate binders on the market, which vary in cost, although the data to date do not support superiority of the more expensive novel non-calcium binding agents [112]. To enhance the effectiveness of this medication, educating patients on matching their binder medication to the phosphorous load of their meals can improve serum levels [113]. Although this self-adjusting binder technique promotes autonomy, limits dietary restrictions, and enhances patient satisfaction, it is time intensive to implement and is restricted by patients' cognitive capacity. Another important, but often overlooked, point is to ensure that patients are taken binders appropriately, such as timing at the start of meals.

5.3.2.3 Key Management Strategies

1. Restrict phosphorous-based additives
   a. Promotion of fresh food is best
   b. Check ingredient lists for phosphorous-based additives (higher level knowledge)
2. Ensure dietary protein is not excessive (see protein guidelines)
3. Limit foods with high phosphorus: protein ratios
4. Phosphate binder prescription
   a. Ensure appropriate use and compliance to binders
   b. Self-adjusting binder education (higher level knowledge)

5.3.3 Fluid Balance

Fluid overload is highly prevalent in dialysis patients. In fact, acute fluid overload is a common cause for not only emergency dialysis but also hospital admissions manifesting as heart failure and pulmonary edema. This contributes a significant cost burden on the health-care system [114]. Chronic hypervolemia is thought to be the cause of at least 80% of all hypertension in dialysis patients [115]. Furthermore, fluid overload is closely linked to markers of cardiovascular disease and stroke, the leading cause of morbidity and mortality in this population. In addition, removal of excessive fluid during dialysis requires high ultrafiltration rates, leading to an increased risk of hypotensive episodes and cramps.

5.3.3.1 Assessment

There is a lack of consensus on the definition for excessive fluid gains, termed interdialytic weight gain (IDWG). Excessive IDWG may be defined using an absolute amount (i.e., 2–5 kg) or a percentage of the individual's body weight (usually 4%). Due to the lack of consensus surrounding the recommended cutoffs, it is important to develop and communicate local policies and standards based on the dialysis unit, or individual patient, accounting to comorbidities. Furthermore, despite the existence of many assessment tools, no single method has emerged as a gold standard.

The average IDWG from six consecutive sessions (over a 2-week period) is generally used to determine compliance to fluid restrictions. Peripheral edema, hypertension, and visible distension of jugular veins are commonly used in the clinical setting to determine fluid overload.

Biochemical assessment of sodium is a poor indicator of hydration due to the body's tight control of this parameter. There are a series of serum natriuretic peptides that hold promise as prognostic biomarkers of fluid status, although to date their lack of specificity limits utilization in practice [115].

Bioimpedance analysis is another method that has shown to be useful for determining fluid status, although most of the validation studies have been undertaken in the nonuremic population. Nonetheless, recent studies have demonstrated that clinical decision-making based on hydration management from bioimpedance resulted in improved management and reduced cardiovascular markers such as arterial stiffness and all-cause mortality [116].

More invasive measures of chronic fluid overload that offer good prognosis for cardiovascular risk include left ventricular dysfunction and hypertrophy from echocardiogram or cardiac magnetic resonance imaging.

5.3.3.2 Management

Most patients' fluid intakes are limited to 0.5 L fluid/day (plus a quantity equal to any residual urine output). However, prescribing a fluid allowance without a sodium restriction is futile, with thirst strongly linked to sodium intake. In fact, for every 8 g of salt, 1 L of fluid is required to meet the associated thirst [117]. Therefore, compliance to sodium guidelines of less than 6 g of salt/day (equivalent of 2 g sodium) is fundamental for achieving fluid control.

Patients often fear salt restricted diets due to their association with bland, un-pleasurable food. Identified barriers to adherence to a salt restrictive diet are (a) perceived taste/palatability of low-sodium foods, (b) convenience/difficulty (e.g., time, availability of low-sodium foods, interference with socialization, and cost) or, (c) lack of knowledge or understanding (e.g., lack of perceived benefit and inability to identify low-sodium foods). For this reason, it is important to begin any sodium dietary education with reassuring patients of sodium's acquired taste and, thus, slow decreases over time lead to increased salt sensitivity. With this in mind, it is important that realistic goals are set and sodium reduction occurs gradually over several months.

Bread, baked products, pre-cooked foods, and sausages are the most common sources of sodium in a Western diet besides the salt added to meals. Most of the sodium (75%) comes from processed foods and, therefore, advocating for fresh, unprocessed food should underpin all sodium education. Other principles such as not adding salt to cooking, but instead utilizing other salt-free flavors and spices such as garlic, freshly ground pepper, and dry mustard powder, can enhance compliance without compromising flavor. Caution should also be given against using salt substitutes due to their high potassium content. Fortunately, there is mandatory labeling of sodium on nutrition information panels, enhancing the transparency for patients. As a general rule, foods with more than 120 mg of sodium per 100 g should be limited, and the importance of checking-specific brands is also apparent, with some brands containing several fold more sodium for equivalent food products [118]. Individualized counseling with a qualified dietitian remains the gold standard. This allows for patient-specific recommendations of food alternatives based on the patient's reported diet history. This method maybe perceived as less overwhelming for patients who struggle with adjusting their dietary habits.

Clearing up myths is another important strategy to increase patient awareness. Common myths include the need for extra salt in hotter months as well as for preventing dialysis-associated cramps.

Once patients have a grasp on sodium restrictions, education on what constitutes a fluid becomes more relevant. Anything that forms a liquid at mouth temperature or even foods with high fluid contents, such as rice and melon fruits, should be considered in fluid allowances. There are a number of government approved resources available which offer practical tips including the use of peppermints or slices of lemon to stimulate saliva flow, as well as freezing some of the fluid allowance to extend its thirst-quenching capacity [101].

5.3.3.3 Key Management Strategies

1. Limiting processed foods
2. Replacing salt in cooking with other flavors and spices
3. Reading food labels (higher level knowledge)
4. Choosing lower salt food options within each food group
5. Dispelling sodium myths
6. Educating on what constitutes a fluid
7. Practical tips for fluid management
   a. Stimulating saliva
   b. Extending fluid allowance

5.3.4 Vitamins and Trace Elements

There are a range of factors that contribute to vitamin and mineral disturbances common in the hemodialysis population, which manifest as both primary and secondary deficiencies. Primary causes, defined by low nutrient intakes, may result from symptoms of anorexia, taste changes, as well as the burden of potassium and oxalate dietary restrictions. Secondary causes include medication interactions, particularly with phosphate binders; enhanced gastrointestinal malabsorption, possibly relating to gut edema; altered kidney and cellular synthesis and metabolism, specifically with vitamin D; and the significant loss of water-soluble vitamins in dialysate. Toxicity from vitamin and trace elementals is also a concern in this population due to their limited clearance, particularly in anuric patients.

Studies have reported that more than 90% of maintenance hemodialysis patients exhibit some level of vitamin abnormality [119] and similar prevalences have been observed with trace elements, particularly in anemic patients [120].

The literature linking vitamin and elemental supplementation with clinically relevant outcomes is sparse. One prom-

inent observation study, which has led to a significant uptake in routine supplementation, demonstrated that patients who consumed water-soluble vitamins had better nutrition status, in addition to a 16% decrease in mortality, compared to those who did not [121]. Importantly, the benefit of vitamin supplementation persisted even after adjusting for traditional risk factors such as age, gender, race, body mass index, and other potential confounders.

Nonetheless, the importance of undertaking prospective intervention studies to confirm this association is clear. This has been highlighted by the disappointing results of a number of intervention studies demonstrating a lack of efficacy for homocysteine lowering therapy (through vitamin B supplementation) on clinical outcomes, despite initial promise suggested in observation studies [122].

5.3.4.1 Assessment

The hemodialysis population's complex biochemistry and nutrient metabolism limit the application of the recommended dietary intake (RDI) reference values which are targeted at the general population [123]. This shortcoming makes assessment of nutritional adequacy an ongoing challenge for dialysis patients. In addition, the lack of consensus on optimal methods to assess nutritional status for many vitamins and minerals further compounds the issue.

Nonetheless, the European Renal Association in conjunction with the European Dietitian and Transplant Nurses Association (ERA-EDTNA) has published recommendations for nutrient adequacy in the dialysis population [124]. The ERA-EDTNA make clear the distinction, however, between their recommendations based on expert opinion and clinical guidelines, which have been hampered by the lack of research in this area.

There are large differences in the distribution and size of body stores between nutrients and, therefore, assessment of adequacy requires a range of techniques. Common methods include (1) dietary intake, (2) serum or plasma concentration, (3) urine concentration, and (4) enzymatic activity. In addition, clinical manifestations of deficiency or toxicity, particularly where early signs are well defined, may offer better insight into overall body adequacy. In fact, the ERA-EDTNA have suggested that zinc supplementation should be given in the case of chronic inadequate protein/energy intakes with physical symptoms evoking signs of zinc deficiency (such as impaired taste or smell, skin fragility, and peripheral neuropathy), rather than relying solely on serum measures.

There are a number of robust, non-invasive techniques for measuring vitamins including erythrocyte transketolase activity coefficients (ETK-AC) (thiamine adequacy) and erythrocyte glutamic pyruvic transaminase (EGPT) activity (pyridoxine adequacy) [125]. Unfortunately, the complexity and cost associated with these biochemical measures limits the translation into routine clinical care in many dialysis units.

5.3.4.2 Management

Following a balanced diet is the preferred method to achieve recommended nutrient intakes as it limits not only the risk of toxicity that presents with taking commercial supplements but also the interaction between nutrients. For example, iron supplements have been shown to promote zinc deficiencies through inhibiting absorption [124].

The significant impact of dietary intake on nutrient adequacy in hemodialysis patients was demonstrated in a study that compared the vitamin intake of patients reliant on processed foods with those relied on traditional meals, and found the former group were significantly lower, particularly in B6 and folic acid [126].

Nonetheless, dietary intakes are often insufficient to meet the increased needs of many vitamins and trace elements in this population, as outlined in Table 5.6.

5.3.4.3 Vitamin Supplementation

The ERA-EDTNA working group is the only body to provide recommendations on a compressive list of vitamin and mineral supplementations, with many other groups opting against due to the lack of evidence in this area [127]. Since the inception of these recommendations in 2007, there has only been one significant change. The ERA-EDTNA's recommendation for vitamin E supplementation (400–800 IU/day) was based on the findings of a high-impact study which demonstrated that α-tocopherol supplementation in maintenance hemodialysis prevented vascular events [128]. Unfortunately, subsequent studies, including Heart Outcomes Prevention Evaluation (HOPE) [129] and HOPE-The Ongoing Outcomes (HOPE-TOO), have not only showed no benefit but also a possible risk for heart failure with vitamin E supplementation [130]. For this reason, prudence dictates that recommendations for supplementation of vitamin E should be withdrawn until further research is undertaken.

Vitamin D is unique to the other fat-soluble vitamins in that its metabolism, bioactivity, and supplementation requirements are dependent on phosphocalcic metabolism and bone status. For this reason, clinical guidelines recommend vitamin D supplementation should be individualized [131].

Due to the limited clearance of fat-soluble vitamins, toxicity from this group poses a significant risk. Irrespective of that, caution in supplementing water-soluble vitamins, such as vitamin C, can also be detrimental, with levels well below what is considered toxic in the general population, proving to be harmful [132].

5.3.4.4 Trace—Element Supplementation

Like vitamin D, the need for iron supplementation is variable and depends on a number factors including hemoglobin levels and the use of erythropoiesis-stimulating agents. Therefore, guidelines recommend routine evaluation and individualized management of iron stores should be followed, with supplementation in the form of intravenous iron if needed.

Table 5.6 Vitamin and trace element requirements [138–140]

	RDA/AI[a] 19–50 years	Recommended supplementation on hemodialysis	RDI/AI recommended (%)	Food sources	Toxicity[b]
Water-soluble vitamins					
Thiamine (B1)	1.2 mg (male); 1.1 mg (female)	1.1–1.2 mg	100	Enriched, fortified, or whole-grain products, including ready-to-eat cereals	No
Riboflavin (B2)	1.3 mg (male); 1.1 mg (female)	1.1–1.3 mg	100	Organ meats, milk, bread products and fortified cereals	No
Pyridoxine (B6)	1.3 mg ≥50 years: 1.7 mg (male) 1.5 mg (female)	10 mg	>700	Fortified cereals, organ meats, fortified soy-based meat substitutes	Yes
Ascorbic acid (C)	90 mg (male); 75 mg (female)	75–90 mg	100	Citrus fruits, tomatoes, potatoes, Brussel sprouts, cauliflower, broccoli, strawberries	Yes
Folic acid (B9)	400 µg	1 mg	250	Enriched cereal grains and breads, dark leafy vegetables, fortified ready-to-eat cereals	Yes
Cobalamin (B12)	2.4 µg	2.4 µg	100	Fortified cereals, organ meats, fortified soy-based meat substitutes	No
Niacin (B3, nicotinamide, nicotinic acid)	16 mg (males); 14 mg (females)	14–16 mg	100	Meat, fish, poultry, enriched and wholegrain breads and bread products, fortified ready-to-eat cereals	Yes
Biotin (B8)	30 µg[a]	30 mg	100	Liver and smaller amounts in fruits and meats	No
Pantothenic acid (B5)	5 mg[a]	5 mg	100	Chicken, beef, potatoes, oats, cereals, tomato products, liver, kidney, egg yolk, broccoli, whole grains	No
Fat-soluble vitamins					
Retinol (A)	900 µg (males); 700 µg (females)	Nil	n/a	Liver, dairy products, fish, darkly colored fruits, leafy vegetables	Yes
Alpha-tocopherol (E)	15 mg	Up to RDA if deficiency exists	n/a	Vegetable oils, unprocessed cereal grains, nuts, fruits, vegetables, meats	Yes
Vitamin K	120 µg[a] (male); 90 µg (female)	Unknown	n/a	Green vegetables (collards, spinach, salad greens, broccoli), brussel sprouts, cabbage, plant oils and margarine	No
Calciferol (D)	15 µg 20 µg (>70 years)	Individualized approach	n/a	Fish liver oils, flesh of fatty fish, egg yolk, fortified dairy products and fortified cereals	Yes
Trace elements					
Iron	8 mg (men; women post-menopause); 18 mg (women pre-menopause)	IV iron dose case specific[c]	n/a	Fruits, vegetables and fortified bread and grain products such as cereal (nonheme iron sources), meat and poultry (heme iron sources)	Yes
Zinc	11 mg (men); 8 mg (women)	Nil	n/a	Fortified cereals, red meats, certain seafood	Yes
Selenium	55 µg	Nil	n/a	Organ meats, seafood, plants (depending on soil selenium content)	Yes

[a] *RDAs* recommended dietary allowances, *AIs* adequate intakes
[b] For normal individuals defined by the presence of an upper limit
[c] [131]

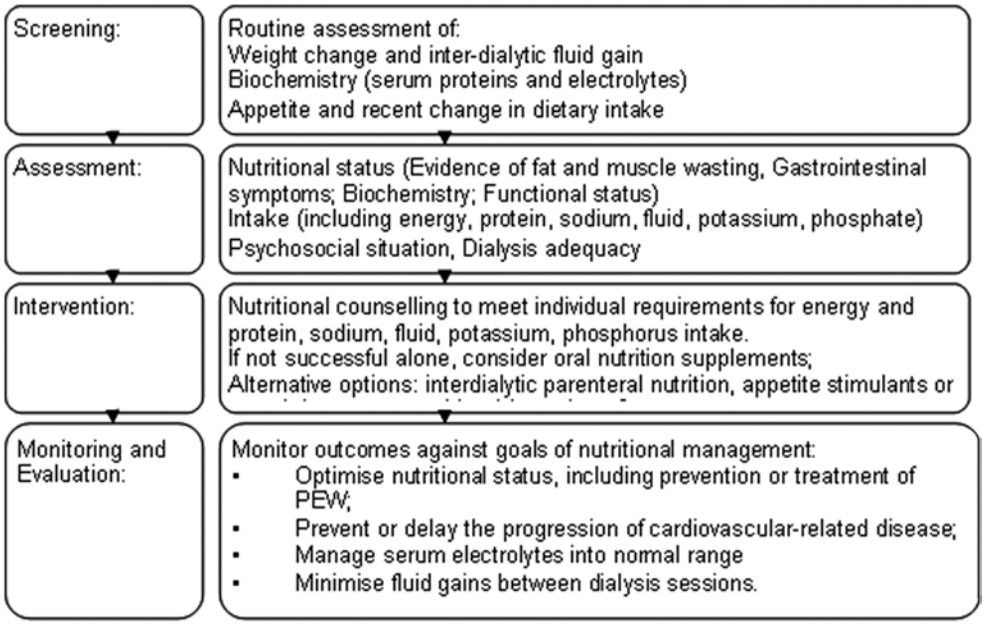

Fig. 5.5 Overview of the process for providing medical nutrition therapy for hemodialysis patients

Despite the lack of routine recommendation for both zinc and selenium, studies have shown symptom improvement with supplementation [133, 134]. Therefore, supplementation for 3–6 months may be considered where symptoms evoking signs of deficiency are suspected.

The high prevalence of commercial dietary supplements in the general population, which was reported to be 50% in a large cohort of older Americans [135], highlights the importance of reviewing patients' supplement use. Purchase of regular vitamin and mineral supplements should be strongly discouraged, where supplements such as B-100 or multivitamins can contain dangerously high amounts of B vitamins as well as containing hazardous minerals (phosphorous and potassium) and vitamins (A and K). There are a number of renal-specific formulations available, which comply with the recommended dose defined in Table 5.6.

5.4 Summary of Nutritional Management in Hemodialysis

The goal of nutritional management in hemodialysis is to (1) optimize the nutritional status, including prevention or treatment of PEW, (2) prevent or delay the progression of cardiovascular-related disease, (3) manage bone mineral metabolism through optimizing phosphate management, and (4) manage serum electrolytes and fluid. Dietary requirements for dialysis patients span both macronutrients (protein and energy) and micronutrients (vitamins and trace minerals) and essential nutrients in the form of amino acids and fatty acids. Optimizing nutritional status requires adherence to minimum requirements. Guideline recommendations for intake in maintenance dialysis patients are summarized in Table 5.3 [71, 79, 86, 105, 136].

Figure 5.5 outlines the process that should be undertaken for the nutritional management of hemodialysis patients. Providing routine review of dialysis patients results in improved outcomes, including reduced rates of malnutrition, improvements in control of serum phosphate and potassium [137].

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