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Nutritional Screening and Assessment in Chronic Kidney Disease

By Guillermina Barril and Mar Ruperto

Nutritional risk and malnutrition related to chronic kidney disease (CKD) are common disorders that usually appear from CKD stages 3–5 and are more frequent among patients undergoing renal replacement therapy, mainly among those receiving hemodialysis therapy.

The prevalence of malnutrition has been reported in up to 54% of patients living with CKD, leading to a significant increase in morbidity and mortality (1–3). Nutritional screening is a preassessment method of nutritional status to identify patients at risk of malnutrition and, in turn, to indicate nutritional assessment for those with increased nutritional risk and/or probable malnutrition. Since the 1980s, several nutritional screening tools have been implemented in CKD (Figure 1).

The subjective global assessment (SGA), originally developed by Detsky and colleagues in the 1980s (4), was adapted and validated in 1996 as a seven-point scale (7-point SGA) (5, 6). Recommended by clinical practice guidelines for regular nutritional assessment in patients with CKD and undergoing dialysis (7), this 7-point SGA is based on clinical history data (body weight, dietary intake, gastrointestinal symptoms, and functional capacity, as well as comorbidities related to nutritional needs) and includes a physical examination of body mass (subcutaneous fat and muscle) and the detection of edema. Studies (8, 9) have shown that low 7-point SGA scores are associated with a high risk of mortality in patients living with CKD and undergoing dialysis. In 1999, the dialysis malnutrition score (DMS) was developed (10), which used the original 7-point SGA scale and included a score from 1 to 5 for each item. Subsequently, the Malnutrition-Inflammation Score (MIS) questionnaire, a semiquantitative tool that is based on the subjective 7-point SGA and also includes objective parameters (body mass index, serum albumin, and total iron binding capacity) (11), has been extensively correlated in previous studies (11, 12) with hospital admission and mortality. MIS is a validated nutritional screening tool for patients with CKD and undergoing dialysis (11, 12) and has been recommended for routine use for the nutritional assessment of patients with kidney failure (7). The Dialysis Outcomes and Practice Patterns Study (9) used the quantitative modified SGA (m-SGA), developed in 2002, based on caregiver ratings of weight loss, appetite loss, gastrointestinal symptoms, and disease burden. Patients with a severe m-SGA score had significantly higher mortality risk compared with those with moderate or normal m-SGA scores.

An expert panel in 2008 (13) suggested using specific markers from four different categories—biochemistry, body mass, muscle mass, and dietary intake—for the clinical diagnosis of the so-called protein-energy wasting (PEW) syndrome. Three of these four categories should be included, with at least one being a biochemical marker. PEW is a complex syndrome that, combined with the inflammation, uremic toxicity, and endocrine-metabolic disorders of CKD, has been shown to significantly increase the mortality rate at a 5-year follow-up (13) (Figure 2).

Most recently in 2019, unified diagnostic criteria for disease-related malnutrition were proposed within the framework of the Global Leadership Initiative on Malnutrition (GLIM) (14). The GLIM approach includes one phenotypic criterion (low body mass index, unintentional body weight loss, or low muscle mass) and at least one etiologic criterion (reduced food intake, disease burden, or inflammation state) for diagnosing disease-related malnutrition. At present, the applicability of GLIM criteria in CKD and dialysis is still being developed. Further studies with large samples are warranted to validate GLIM criteria for the diagnosis of PEW.

In summary, the first step in detecting nutritional risk can be performed using well-established and validated nutritional screening tools, whereas nutritional assessment requires the combination of several parameters to diagnose PEW in populations with CKD and undergoing dialysis. A single marker by itself is not able to identify or diagnose nutritional disorders. ■

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Figure 1. Timeline of nutritional screening tools and diagnostic criteria used in populations with CKD and undergoing dialysis

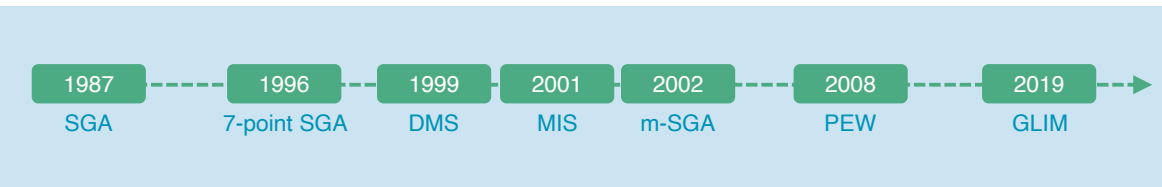
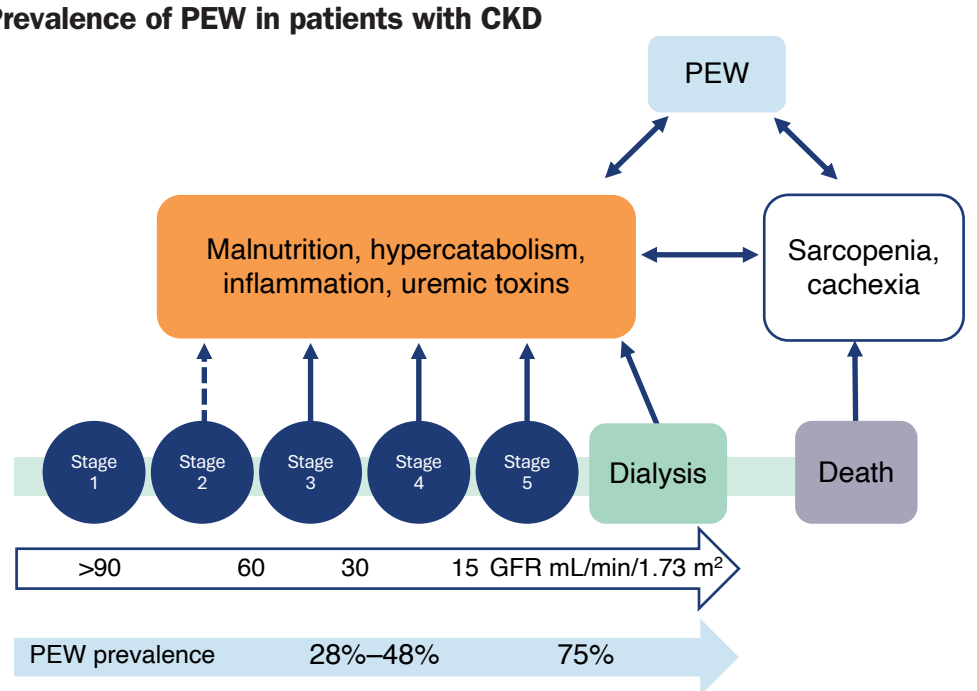


Figure 2. Prevalence of PEW in patients with CKD



Conceptual scheme modified from Hanna et al. (15). GFR, glomerular filtration rate.

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A Potential Paradigm Shift: Potassium Binders in K+-Restricted Diets in Patients With CKD

By Deborah J. Clegg and Biff F. Palmer

Individuals with chronic kidney disease (CKD) and those receiving kidney replacement therapies are often prescribed diets that are extremely challenging to adhere to. In addition to being low in phosphorus, these diets typically are restrictive in potassium (K^+)-containing foods. The rationale for K^+ restriction among this patient population was derived years ago and was based on K^+ balance studies in which individuals with CKD receiving dialysis were given K^+ supplements. When given as K^+ salts, development of hyperkalemia was common and provided the rationale for K^+-restricted diets. More recent data suggest that consumption of diets rich in fruits and vegetables (food items rich in K^+) in individuals with CKD and on dialysis do not significantly increase plasma K^+ concentration (1, 2). Reflexively encouraging patients with CKD not to consume fresh fruits and vegetables and/or a Mediterranean diet has the potential for harm since these diets have proven health benefits. As a result, there is a growing trend focusing on liberalization of K^+ in the diet among people with advanced CKD and/or on dialysis. One strategy to allow patients to ingest diets higher in dietary K^+ is with simultaneous use of new K^+-binding drugs. A shift toward a more lenient, plant-based diet may be plausible and may enhance compliance while fostering better overall health (3).

Patiromer and sodium zirconium cyclosilicate are new oral drugs that function as K^+-binding agents in the gastrointestinal tract and have demonstrated efficacy in treating hyperkalemia. Both drugs have demonstrated sustained efficacy and tolerability when used on a chronic basis. Patiromer is a nonabsorbed polymer that binds K^+ in exchange for calcium and acts primarily in the colon. Sodium zirconium cyclosilicate has a nonabsorbed microporous structure allowing for binding of K^+ in the gastrointestinal tract in exchange for sodium. Both drugs reduce plasma K^+ in patients with CKD, enabling the chronic use of renin-angiotensin-aldosterone system inhibitors, and in patients with heart failure, CKD, and established cardiovascular disease. Diet was not controlled in studies of these drugs, but patients were instructed to avoid high K^+ intake. What is not known is whether novel binding agents could enable patients who are vulnerable to hyperkalemia to increase their consumption of K^+-enriched fruits and vegetables without inducing hyperkalemia. Such trials would be of great utility. If such trials demonstrated that these drugs were effective in liberalizing the diet, patients with high risk of CKD would be granted the health benefits of K^+-enriched diets and likely would enjoy a better quality of life (4, 5).

The current management of individuals with hyperkalemia is to reflexively impose dietary restrictions on fresh fruits and vegetables, depriving them of the cardiovascular benefits of these foods. This strategy has the potential to contribute to ongoing development of atherosclerosis in patients with CKD. Dietary surveys conducted by the National Health and Nutrition Examination Survey indicate that the average consumer takes in approximately 2000 mg of K^+ per day, and therefore, K^+ has been listed as a nutrient of concern because of inadequate intake (6). This is important because patients with advanced CKD and undergoing dialysis are prescribed low K^+ diets, which provide 2000 mg per day—exactly what consumers are typically (inadequately) eating. We contend that there is insufficient evidence to justify the extent to which K^+ restriction is commonly enforced in many patients with CKD. In cases of hyperkalemia, it is important to note that there are nondietary factors such as metabolic acidosis, poorly controlled diabetes mellitus leading to hypertonic states, increased catabolism, tissue breakdown, constipation, and medications, all of which contribute to hyperkalemia and should be considered first prior to dietary restriction. In addition, there are several characteristics of diets enriched in fruits and vegetables that serve to limit development of hyperkalemia (Table). Nevertheless, dietary counseling remains essential, especially for individuals consuming large quantities of foods rich in K^+ additives or high in sodium content. ■

Table. Characteristics of a diet enriched in fruits and vegetables that minimize hyperkalemia

Carbohydrate load causing stimulation of insulin release and shift of K^+ into cells
Increased alkali content • Shift of K^+ into cells • Increased K^+ secretion brought about by the pH effect on the renal outer medullary channel in the collecting duct
High fiber content • Increased stool bulk and less K^+ absorption • Decreased constipation • Increased kidney K^+ secretion via gastric-kidney crosstalk
Lack of exogenous administration of K^+ for flavoring as commonly present in processed foods

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Dr. Clegg reports having provided consultation to AstraZeneca Pharmaceuticals regarding dietary potassium management in patients with CKD. Dr. Palmer reports having participated in advisory boards for AstraZeneca Pharmaceuticals and Bayer HealthCare Pharmaceuticals.

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