

Chronic kidney disease (CKD) is defined as abnormalities of kidney function or structure that persist for >3 months and have health implications.¹ The worldwide (C) 2021 American Society for Parenteral and Enteral Nutrition prevalence of CKD is estimated to be 9.1%.² A diagnosis of CKD is correlated with higher risks of cardiovascular and all-cause mortality, acute kidney injury, and CKD progression, among other comorbidities.¹ Patients with CKD, especially those approaching end-stage kidney failure and undergoing maintenance dialysis, frequently experience gradual decline in their nutrition status; the latter is typified by such deranged metabolic and nutrition status changes³ displayed as concurrent depletion of body protein and energy reserves, eventually resulting in fat and muscle wasting, in addition to shrinkage of visceral protein pool.^{4–6} This state of disorder, coined as protein–energy wasting (PEW), has a worldwide prevalence of 11%–54% and 28%–54% in patients who have stages 3–5 non-dialysis-dependent CKD or require maintenance dialysis,⁷ respectively. In addition, insufficient dietary intake of energy and protein can stem from dietary restrictions, comorbidities that affect the gastrointestinal function, low socioeconomic status, depression, physical disability that hampers food provision and preparation, and factors pertaining to peritoneal dialysis, such as abdominal discomfort, peritoneal glucose absorption, and early satiety with peritoneal dialysate infusion.^{3, 13, 14} Furthermore, the pathophysiology of PEW can be caused by other highly common factors, including a low level of physical activity, endocrine derangements, metabolic abnormalities, and dialysis-specific catabolism and nutrient losses.^{3, 13–16} Although the obligatory losses of water-soluble vitamins, proteins, and amino acids are known to occur during both peritoneal dialysis and hemodialysis processes,^{15, 16} the losses of energy and glucose are highly applicable during the hemodialysis process, as glucose is replenished via the peritoneal route.^{15, 16} Notably, increased protein catabolism and inflammation induced by dialysis were evident in both peritoneal dialysis and hemodialysis patients,^{15–17} though the effect of catabolism was found to be higher in the latter, with the exception of those with peritonitis.¹⁵ Eventually, PEW may lead to cardiovascular diseases, infections, frailty, and depression, with these complications exacerbating PEW.^{3, 13} In spite of the multifactorial attributions of PEW, it is realized that many of the causative factors are related to decreased nutrient intake,⁵ as indicated in Figure 1. The International Society of Renal Nutrition and Metabolism (ISRNM) proposed an etiological model for PEW (Figure 1).^{3, 12, 13} A crucial factor in the etiology of PEW is insufficient oral intake of energy and protein owing to anorexia, as a consequence of the dialysis process, retained uremic toxins, inflammation, metabolic acidosis, and intercurrent illnesses.^{3, 13} Concurrently, these conditions are independently related to the progression of PEW. This review article aims to focus on different nutrition support approaches in the treatment of PEW in CKD, including nutrition counseling, oral nutrition supplementation, enteral tube feeding, partial parenteral nutrition (PN) therapies such as intraperitoneal PN (IPPEN) and intradialytic PN (IDPN), and total PN, in addition to providing the recommended goals for nutrition support. Of importance, the prevalence of PEW increases with dialysis vintage,^{8–10} contributing substantially to increased morbidity and mortality and negatively impacting quality of life.^{3, 11} The potential causes of PEW in CKD are complicated.