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Clinical Recommendations and Treatment Of Malnutrition among Chronic kidney Disease Patients

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Abstract

Background: Malnutrition refers to a state of over-nutrition or undernutrition of macronutrients elements or specific micronutrient needs, which are critical to tissues. Malnutrition is a common problem in patients with end stage renal disease (ESRD) undergoing hemodialysis (HD) that is associated with increased morbidity and mortality. The pathogenesis of malnutrition in patients with HD is multifactorial. Inadequate food intake due to anorexia and altered taste sensation combined with intercurrent illness, hyper-catabolism and reduced anabolism, dialysis procedure, chronic inflammatory state, and endocrine disorders of uraemia were reported as major causes of malnutrition in hemodialysis. Protein–energy malnutrition and wasting are strong predictors of death among hemodialysis patients. There is not a single measurement that provides complete and unambiguous assessment of the nutritional status of hemodialysis patients.

Keywords: Malnutrition, Chronic Kidney Disease

Background

Malnutrition is defined as "an imbalance between nutrient requirement and intake resulting in cumulative deficits of energy, protein, or micronutrients that may negatively affect growth, development, and other relevant outcomes," according to the American Society for Parenteral and Enteral Nutrition. Undernutrition, as defined by protein energy waste and micronutrient deficiency, is assumed in this definition **(1)**.

Malnutrition is a significant risk factor for morbidity and mortality in both developing and developed countries. Malnutrition in wealthy countries is usually associated with acute or chronic sickness, unlike in underdeveloped countries where it is linked to poor socioeconomic situations. While acute illness has the greatest influence on weight, chronic illness has the greatest impact on line. Children with chronic kidney disease (CKD) are frequently stunted, and malnutrition is a major factor in the development of growth failure in this patient group. In several investigations, a prevalence of 20–45 percent has been

recorded in children with CKD, depending on the clinical characteristics used to define malnutrition **(2)**.

According to the National Academy of Sciences' Food and Nutrition Board, a healthy adult in a stable non-pregnant, non-lactating, and non-recovery condition requires 0.6 g/kg/day of dietary protein.The "Recommended Dietary Allowance" (RDA) of protein intake, with a safety buffer, is 0.8 g/kg/day. Proteins in eggs, fish, chicken, meat, and dairy products, which have a high biologic value (HBV, nitrogen integrated into the body/total absorbed nitrogen >75 percent), should account for more than half of the protein consumed **(3)**.

Malnutrition treatment in CKD patients necessitates a multidisciplinary, multidimensional, and customized strategy. The role of the renal dietitian in assessing and monitoring the nutritional status of a kid with CKD is crucial. The nephrologist, nurses, social workers, caretakers, and therapists are among the other members of the team.

Various suggestions underline the importance of combining these variables because no single metric can accurately determine nutritional health. Following a comprehensive assessment, a tailored dietary prescription should provide enough calorie, micronutrient, and protein consumption to support the child's growth and development

Table 1 shows a comparison between protein energy wasting and protein energy deficiency. Protein energy wasting is common in CKD patients, and it's linked to stunted growth and development in kids, as well as an increased risk of cardiovascular disease, infection, and death. Anthropometric measurements in children with protein energy loss are 2 standard deviations below normal weight for age (underweight), height for age (stunting), and weight for height (wasting) **(2).**

Table 1. Comparison between protein energy malnutrition and protein energy wasting.

Clinical parameter	Protein-energy malnutrition	Protein-energy wasting
Pathogenesis	Explained by reduced nutrient and energy intake relative to metabolic demands of the body	Not completely explained by reduced nutrient and energy intake
Somatic mass		
Protein	Reduced	Reduced
Fat	May not be reduced	Reduced
Body Mass Index	May not be reduced	Reduced
Serum Albumin	May be reduced	Markedly reduced
Hypermetabolism	May be present	Present
Hypercatabolism	May be present	Present
Response to therapy	Condition improved by nutrient repletion	Condition not improved solely by nutrient repletion

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To ensure appropriate intake, supplemental feeding via nasogastric and gastric tubes may be required. Comorbidities such metabolic acidosis, gastric reflux disease, and constipation must be treated in order to effectively manage malnutrition. In patients on chronic dialysis, the importance of providing enough dialysis cannot be overstated. Finally, for growth and development to continue, thorough and ongoing monitoring of nutritional status is required, and the appropriate intervals may be found in the KDOQI clinical practice guidelines **(4)**.

Energy

Enough energy should be consumed to ensure that dietary protein is utilized effectively and that the body's nutritional stores are protected. Because of altered cellular energy metabolism, hemodialysis patients' energy metabolism is hindered and they have a negative energy balance. **(5)**. Consuming enough energy to meet ESRD patients' daily energy needs thereby maintains a positive nitrogen balance and prevents tissue damage and protein catabolism.

Patients with anorexia nervosa were frequently seen in the months following the commencement of dialysis. This is due to the fact that, despite significant changes in their lives, psychological conditions are unable to adapt to a new and restricted diet. It has been found that if protein and calorie intake are not increased in these patients, lost energy is retained in their muscle mass, and their body fat percentage decreases (**6).** According to studies, dietary energy failure occurs more frequently on dialysis treatment days than on non-dialysis treatment days **(7).** Dietary energy intake was 1.02 kcal/kg/day lower on dialysis treatment days than on nondialysis treatment days in a prospective multicenter clinical trial involving 1901 patients from the Hemodialysis Study **(8)**.

Hemodialysis patients' energy intake was shown to be low in some studies. In hemodialysis patients, poor appetite and hypermetabolism significantly restrict food intake **(9)**. When the prescribed energy requirements are compared to the quantity of energy consumed, 90 percent of patients' energy intake is found to be inadequate **(10).**

When hemodialysis patients' energy intake was 32-38 kcal/kg/day, no changes in nitrogen balance, anthropometric characteristics, or the development of a negative or positive energy balance were recorded **(11).**

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Protein

Dialysis patients' protein requirements rise as a result of dialysate losses and catabolism. In studies, it has been shown that a low protein consumption increases mortality **(12).**

Protein synthesis and breakdown are both increased by hemodialysis. Hemodialysis causes skeletal muscle nitrogen loss as a result of the procedure. Protein synthesis and breakdown both increase by 50-100 percent compared to normal levels. Interleukin-1 (IL-1), interleukin-6 (IL-6) and tumour necrosis factor alpha (TNF-) are catabolic indicators that are increased by hemodialysis. Protein breakdown is caused by an increase in cytokine production. Amino acid losses into the dialysate, higher protein catabolism, metabolic and hormonal shifts are all reasons for an increased protein need **(13).**

During hemodialysis, the dialysis fluid loses 0.2-0.3 g/kg or 6-8 g/day of protein, amino acids (aa), and peptides. With these losses due to metabolic diseases, protein catabolism rises. To avoid a nitrogen imbalance, the amino acids that have been lost must be replenished. To compensate for residual renal losses, dietary protein should be adjusted at least 1.2 g/kg/day in hemodialysis patients, according to the "National Kidney Foundation Dialysis Outcome Quality Initiative (NKF-DOQI)" and research by other investigators **(14).**

According to ESPEN, in hemodialysis patients, adjusted diet protein should be ingested at a rate of 1.1-1.2 g/kg/day and should have a biological value (of animal origin) of 50 percent. In addition, the amount of protein in the patient's diet is decided by taking into account the patient's hydration status, body weight, glomerular filtration rate, and the severity of the sickness. **(15).**

A good evaluation parameter for determining the adequacy of protein intake in dialysis patients is a BUN value < 120 mg. Protein catabolic rate of 1.2 g/kg/day was associated with low morbidity, provided adequate control of blood urea concentration, improved nutritional parameters (anthropometric measurements) and biochemical findings (blood albumin, total protein, blood, blood cholesterol, etc.) in dialysis patients, and provided a positive nitrogen balance. **(16).**

With gluconeogenesis, however, enough caloric intake is essential to prevent the utilization of protein as an energy source. Without a positive nitrogen balance, even with a high protein diet, a positive nitrogen balance is impossible to achieve. To achieve appropriate nutrition and prevent malnutrition, patients should be provided adequate calorie intakes and adequate phosphorus intakes when on a low protein diet **(17).**

Protein catabolism, branched chain amino acid breakdown, and muscle glutamine release all increase in hemodialysis patients with metabolic acidosis. The generation of ammonium and bicarbonate excretion is enabled by amino acids and glutamine metabolism. Hemodialysis patients have changes in muscle and plasma levels of branched-chain amino acids. Plasma valine, muscle valine, plasma leucine, muscle leucine, plasma isoleucine, and muscle isoleucine are all reduced as a result of hemodialysis treatment **(18).**

Branched-chain amino acids help to prevent persistent acidosis by acting as a regulator. Branched-chain amino acids levels in plasma and intracellular are increased after acidosis subsides and is enriched in branched-chain amino acids and valine during hemodialysis**.** Branched-chain amino acids help hemodialysis patients eat more. Hemodialysis patients' nutritional indicators were improved by consuming 6.6-15.7 g of essential amino acids daily **(19).**

Patients who took 12 g of oral branched-chain amino acids per day for a month had an improvement in protein and energy purchases, as well as anthropometric measurements six months later. Albumin concentrations increased from 3:31 g/dL to 3.93 g/dL **(18).**

In short-term clinical trials, animal protein (egg, dairy, etc.) has a dynamic effect on renal function. However, the long-term impacts on normal renal function remain unknown. Differences in hormones, protein metabolism, and interactions with micronutrients have been proven to highlight the varied effects of animal and vegetable proteins on renal function. Long-term use of a high-protein diet (whether animal protein or vegetable protein) in healthy persons with normal renal function may promote kidney damage and hasten chronic renal failure.

Long-term studies are needed, however, to ascertain whether the consumption of animal or vegetable protein diets has a different effect on renal function **(20).**

Carbohydrate

Carbohydrate consumption necessitates sufficient energy and the maintenance of a reserve protein pool that can be used for tissue synthesis protein.

In hemodialysis, 28 g glucose is lost when non-glucose dialysis fluid is utilized for 4 hours. The patient gained roughly 23 g of glucose when 11 mmol/L glucose was introduced to the dialysis fluid. When glucose is withdrawn from the extracellular fluid by dialysis, the glucose is lost by ingested carbohydrates, hepatic glycogen breakdown, and gluconeogenesis to avoid clinical hypoglycemia.

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Then there's an increase in protein breakdown and urea synthesis. Pyruvate is lowered in glucose-free dialysis. Glucose dialysis has no effect on pyruvate levels. Gluconeogenesis can be boosted by dialysis without glucose. Hyperglycemia, hyperinsulinemia, hyperlipidemia, obesity, and other unfavorable effects of glucose consumption exist **(21).**

In chronic renal failure, glucose metabolism deteriorates and insulin resistance develops. When combined with increased hepatic gluconeogenesis, this condition leads in rising glucose and urea levels. Uremia causes serious problems in insulin metabolism. Insulin production is diminished during rest and responds slowly to glucose infusion **(11).**

In one study, it was discovered that in the presence of chronic uremia, insulin resistance, muscle glucose uptake, and nonoxidative glucose metabolism were impaired, but that they improved following dialysis **(22).**

During fructose metabolism, uric acid is produced. Uric acid levels in the blood have been linked to fructose consumption. High levels of serum uric acid have been linked to hypertension, inflammation, chronic kidney disease, and fructose and added sugar consumption **(23).** Fruits containing fructose, on the other hand, include useful compounds such as antioxidants. As a result, fructose intake from natural fruits may be compatible with a healthy diet **(23).**

Dietary carbohydrates should be higher in order to give enough energy, safeguard the backup protein utilized for tissue protein synthesis, and cover the energy imbalance. Carbohydrates should offer 60-65 percent of daily energy. **(11).** Low protein diets make it difficult for most patients to achieve their energy requirements. As a result, glucose polymers (starch), sugar, simple sugars, and pure carbohydrate sources can fill the energy shortfall. Patients with diabetes should avoid sweets that are high in sugar **(14).**

Lipids

According to some studies, protein calorie malnutrition generally begins incipiently when the glomerular filtration rate (GFR) is around 28 to 35 mL/min/1.73 m2 or even higher, and progresses as the GFR falls below these levels **(24).**

lasma lipid levels rise significantly when the amount of GFH is reduced **(25).** Hyperlipidemia occurs when a patient's creatinine clearance is less than 50 ml/min.The accumulation of triglycerides-rich lipoproteins was linked to increased lipogenetic gene expression of enzymes and high triglyceride synthesis by renal insufficiency in Rutkowski's study. **(26).**

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Hemodialysis patients frequently have hypertriglyceridemia and hyperlipidemia. High density lipoprotein (HDL) cholesterol concentration is low, but low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) cholesterol concentrations are high. The absence of triglyceride elimination from the circulation is the primary cause of hypertriglyceridemia, there has been a decline in lipoprotein lipase and hepatic lipase enzyme activity in these patients **(27).**

Carnitine storages are reported to diminish in hemodialysis patients who are malnourished. Furthermore, during dialysis, carnitine leaves the extracellular fluid, resulting in a sharp decline in carnitine levels in the blood. Carnitine deficit is produced by a decrease in long-chain fatty acid oxidation, resulting in energy deficiency **(28).** It was discovered that adding 750 mg of carnitine to hemodialysis patients' diets reduced plasma TG and LDL cholesterol levels while increasing HDL cholesterol levels **(29).**

Because hyperlipidemia affects a substantial percentage of dialysis patients, the amount of fat in the diet should be kept to a minimum. The amount of saturated fat in the diet should be lowered, while the amount of unsaturated fat in the diet should be increased **(30).**

Because hyperlipidemia progresses in the majority of individuals with CKD, a high fat diet should be avoided. Fat should not account for more than 25% to 30% of total energy. It is recommended that the saturated fat level of the diet be lowered and the unsaturated fat content be increased.It is suggested that saturated fat intake (total energy 7%) and cholesterol intake (200 mg / day) be reduced. Monounsaturated fatty acids should account for 25-35 percent of total calories in the diet. Polyunsaturated fatty acids account about 15-20% of total energy. ten percent of the diet's total energy **(31).**

Eggs, which are recommended foods for people with a high biological value, contain a lot of cholesterol. As a result, each patient's serum cholesterol levels should be assessed individually. If a patient has hypertriglyceridemia and high cholesterol, dietary fat restriction, weight loss, increased physical activity, reduced usage of hypertonic solutions, and dietary sugar limitation are all indicated **(31).**

Dialysis patients show signs and symptoms of essential fatty acid insufficiency, such as dry and itchy skin, hair loss, and aberrant prostaglandin synthesis. Linolenic acid in fish oil contains EPA and DHA, which replace arachidonic acid in cell membranes and prevent the development of pro-inflammatory chemicals (n-3 fatty acids). The FDA recommends that n-3 fatty acid intake from dietary supplements not exceed 3 g per day **(32).**

Omega-3 dietary supplementation was found to lower triglyceride levels in trials **(33),** LDL cholesterol and CRP, as well as the Omega-6 / omega-3 polyunsaturated fatty acids ratio, were found to be essential for inflammation and mortality in hemodialysis patients **(34).**

Water and electrolytes

Fluid adjustments should be done based on the patient's edema and dehydration. Fluid consumption should be limited in hemodialysis patients who have swelling of the eyes, hands, or feet, fluid weight gain, shortness of breath, elevated blood pressure, or tachycardia**.** Patients on hemodialysis should limit their fluid intake and avoid foods including tea, coffee, soda, water, fruit juices, ice cream, sherbet, gelatin, soups, and heavy sauces. **(35)**

Dietitians, particularly renal dietitians, are frequently acknowledged as a reliable source of information on fluid management and dietary guidance **(36).** According to fluid balance dietician research, teaching patients how to deal with thirst without consuming liquids is critical. Suggestions include sucking on ice chips, cold sliced fruit, or sour sweets, as well as artificial saliva **(14).**

The HD diet emphasizes limiting sodium and fluid intake. The key pathophysiologic factor of hypertension in HD patients is extracellular volume expansion. Hemodialysis patients' water and sodium intake are regulated based on the amount of urine produced, fluid balance, and blood pressure. Potassium restriction is frequently required during hemodialysis, but the degree of restriction is determined on remaining renal function **(8).**

It is suggested that body weight increase during hemodialysis not exceed 1.5-2 kg. Hemodialysis patients should drink 500 mL plus their daily urinary output, or roughly 1000-1500 mL, on a daily basis. The amount of sodium restricted should be determined by the amount of urine produced. In oliguric patients who have more than 1 liter of urine per day, a minor salt restriction of 3-4 g per day is sufficient. Anuric hemodialysis patients can drink up to 1 liter of liquid per day and take up to 1.5-2 g of salt. Salt and water restriction should be carefully managed if hypertension or heart failure are present. Excess salt intake leads to an increase in thirst and liquid consumption. Olives, pickles, cured meats, garlic sauce, soy sauce, canned foods, sausages, processed meats, ham, chips, pretzels, and instant soups should all be avoided in hemodialysis patients' diets to limit sodium intake. Spices like vinegar and lemon can be used as a salt substitute or to add flavor to unsalted foods **(36).**

Hemodialysis therapy, as well as the degree of residual renal function, net tissue breakdown (e.g. owing to infections) and acid-base balance, have an impact on potassium levels. Serum potassium concentrations in HD patients may fluctuate due to net intestinal potassium

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absorption or excretion. Diarrhea is an example of this alteration or excretion. Dietary potassium consumption has an impact on serum potassium. This link is expected to be stronger when potassium consumption in HD patients' diets is extremely low or extremely high **(37).**

Hemodialysis patients are frequently anuric, necessitating potassium restriction. Potassium consumption for anuric HD patients should be limited to 1600-2000mg per day. Hypokalemia, which is caused by a decrease in potassium, can cause symptoms such as severe vomiting, diarrhea, and the usage of diuretics. The potassium level of the diet should be increased in this scenario **(6).**

When dialysis patients' blood potassium levels are high, the patient's diet should be evaluated as a top priority. Milk, animal products, fruits, legumes, cereals, dried fruits and vegetables should all be consumed in moderation in order to lower potassium intake.

Vitamin and minerals

Long-term hemodialysis patients may benefit from vitamin and mineral supplements, according to certain research. Patients on hemodialysis are at risk of deficiency and excess trace elements **(38)**.

Given the importance of vital trace elements in a variety of biological systems, including immune defense against oxidation and infection, it’s been suggested that the higher morbidity and mortality reported in hemodialysis patients could be attributable in part to an undiscovered trace element imbalance **(39)**.

There are numerous issues related with a lack of food intake in HD patients. Poor nutrition, restrictions on foods high in water-soluble vitamins and potassium, metabolic abnormalities caused by uremia, infection, and diseases such as gastrointestinal ailments or complications connected with restricted food intake are only a few of the scenarios that could occur. Vitamin shortages can occur as a result of a lack of vitamin-rich diets, which can lead to severe difficulties in dialysis patients **(14)**.

Vitamin B6, folic acid, and vitamin C deficits have been seen in dialysis patients **(40)**. Vitamin B6 deficiency must be continuously monitored because it is involved in amino acid consumption and lipid metabolism, as well as playing a key function as a coenzyme. Deficiencies in folic acid, vitamin B6, or vitamin B12 can have a significant impact on the ability of the other vitamins to work effectively **(41)**.

This link necessitates that everyone operates in unison in order for the metabolic pathway to function well. pyridoxine and folic acid may often diminish red cells and plasma if

vitamin B6 and folic acid supplements are not utilized in dialysis treatment **(42)**. Additional vitamin B6 intake lowers plasma cholesterol and triglyceride levels in dialysis patients, and additional folic acid intake lowers excessive homocysteine levels, which have been identified as a risk factor for cardiovascular disease **(43)**. In most studies, the recommended dose of vitamin B6 and folic acid in HD patients was between 1 mg and 10 mg per day, and in most studies, the recommended consumption was between 1 mg and 10 mg per day **(42)**.

Furthermore, vitamin C deficiency has been seen in HD patients. Increasing vitamin C intake to the recommended daily dose of 100-200 mg was formerly the go-to solution for resolving this issue. Higher doses of ascorbic acid, on the other hand, have been linked to an increase in the formation of oxalate, a vitamin C metabolite. Symptoms of oxalate accumulation include the production of calcium oxalate stones in the kidneys, calcium oxalate accumulation in internal organs and blood vessels, hypercalcemia, and hyperoxaluria **(44)**.

Vitamin C requirements for hemodialysis patients have been suggested to be 60-90 mg per day **(11)**. Additionally, iron excess is caused by ascorbic acid supplementation. It is suggested in uremic patients to prevent erythropoietin resistance. In these patients, vitamin C administration enhanced intestinal iron absorption, potentially lowering the risk of iron deficiency anemia **(45)**.

It's more difficult to tell if a patient has enough trace elements in their body if they have kidney illness. Dialysis patients have deficits in iron (Fe), calcium, and zinc. Dialysis patients frequently develop anemia as a result of an iron shortage. **(45)**. Severe blood loss might be a sign because the amount of iron absorbed in the intestine is reduced. Furthermore, because urea suppresses bone marrow, the production of erythropoietin is reduced **(14)**. After determining the patient's serum ferritin and iron levels, adding iron is indicated **(46)**.

Each intravenous iron product available obtained approval for clinical use because it increased hemoglobin levels better than oral iron supplements or controls. The response of hemoglobin to intravenous iron is related to the amount of iron administered, with 1 g of iron generally considered an adequate test of iron responsiveness. Patients with adequate levels of iron who received periodic intravenous iron supplementation maintained better serum ferritin levels and had lower requirements for ESAs than did patients who received no intravenous iron or just oral iron **(47).**

In traditional intermittent (loading) dose regimens, intravenous iron is given only when transferrin saturation is 20% and serum levels of ferritin >450 pmol/l) versus a traditional ‘load and hold’ regimen of intermittent repletion of 1g of iron (administered only when

transferrin saturation was Patients who received the maintenance regimen required 50% less ESAs overall than did those in the other group to maintain the same levels of hemoglobin. Surprisingly, the cumulative amount of iron needed over a 72-week period was identical for the two regimens. When two different threshold values for transferrin were used (20–30% versus 30–50%), a further 40% reduction in the ESA dose was achieved, although 2–3 times more iron was transiently needed to achieve the higher of the two transferrin values **(48)**

Novel methods of iron delivery new preparations that permit rapid, high-dose administration of iron could change the outpatient management of iron deficiency anemia. In a randomized study, a single 15 min injection of ferric carboxymaltose, repeated up to twice if required, resulted in 53.2% of patients with NDD-CKD achieving a ≥10 g/l increase in hemoglobin level by day 56 without ESAs, compared with only 29.9% of patients who were given oral iron supplements. Administration of two 510 mg ferumoxytol injections about 1 week apart increased the mean hemoglobin level by 10g/l **(49).**

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