Chapter

Medical Nutrition Therapy in Renal Replacement Therapy

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Abstract

This chapter discusses Medical Nutrition Therapy in three modalities of renal replacement therapy (RRT), outlining the nutrient requirements for patients on maintenance hemodialysis, peritoneal dialysis, and kidney transplant in acute and chronic phases. The chapter takes note of the drastic impact of RRT on the patient's nutrition status and overall well-being, which puts them at high risk of morbidity and mortality, and thus emphasizes timely and regular comprehensive nutrition assessment to enable appropriate intervention. Recognizing that there are different modalities of RRT and that patients have different physiological characteristics as well as different laboratory test values, which may also vary for individual patients each time tests are run, nutrition therapy is individualized each time. The chapter takes a closer look at protein-energy wasting, a condition common among patients undergoing RRT, which is a predictor of mortality, discussing its prevention and treatment measures. Finally, the chapter takes a closer look at electrolytes, specifically potassium, sodium, calcium, and phosphorous, in relation to mineral bone disease.

Keywords: nutrition therapy, nutrient recommendations, nutrition assessment, protein energy wasting, mineral bone disease

1. Introduction

1.1 Medical nutrition therapy

Medical nutrition therapy (MNT) is an individualized nutrition evidence-based process that aims at treating and managing medical conditions. MNT comprises comprehensive nutrition assessment, diagnosis, specialized therapies, diet modifications, and nutrition counseling [1, 2].

A Renal Dietitian/Nutritionist, Registered Dietitian Nutritionist (RDN), a nutrition professional, or an international equivalent does the comprehensive nutrition assessment, including anthropometric measurements, nutrition-focused physical findings, monitoring and evaluating appetite, dietary intake, body weight changes, and biochemical data. This also assesses the effectiveness of MNT [3]. Renal replacement therapy significantly affects nutrition status, which in turn affects the well-being of the patients. Malnutrition is common among patients undergoing RRT, with more undernutrition leading towards those undergoing hemodialysis, while

overnutrition may also be found among patients on peritoneal dialysis (PD) and post-transplant patient in the chronic phase. Malnutrition is common among patients with end-stage renal disease, with a prevalence ranging from 18 to 75% for patients on hemodialysis [4]. Patients often do not take adequate calories due to dietary restrictions, which is most common with hemodialysis, and reduced appetite caused by uremia. Other factors that cause malnutrition are loss of nutrients during RRT through dialysis membrane, metabolic acidosis, inflammation, as well as the catabolic effects of RRT [5].

1.2 Nutrition assessment

Comprehensive nutrition assessment, including but not limited to a history of dietary intake, appetite, body weight and body mass index (BMI), other anthropometric measurements, biochemical data, and nutrition-focused physical findings, is required. This should be done at least within the first 90 days of starting dialysis and monitored regularly. Nutrition assessment is the first step of the nutrition care process; therefore, doing it correctly will ensure appropriate intervention. A combination of screening tools and laboratory parameters is recommended [6].

For patients on maintenance hemodialysis (MHD) and PD, it is reasonable to measure body weight and composition at least monthly and at least quarterly for transplant patients to monitor for any changes. The routine anthropometric measurements include waist circumference, skin fold measurements, and creatinine kinetics. Although BMI should be used routinely for its usefulness in predicting mortality, it should not be used in isolation because it is not sufficient to diagnose Protein-energy wasting (PEW) unless it is less than 18 kg/m^2. Rather, percent change in the usual body may be more reliable for determining the risk of PEW [3].

The comprehensive nutrition assessment will inform the nutrition intervention prescribed. Based on the treatment plan, the renal nutritionist or international equivalent should therefore monitor key nutrition care outcomes, such as dietary nutrient intake, body composition, and serum biomarker levels, after which the plan will be re-assessed and adjusted accordingly to achieve the goals established. The Renal Dietitian will work with the multidisciplinary renal team throughout the nutrition care process. The patient and/or caregiver will be educated on dietary recommendations based on individual needs. Studies show that nutrition education effectively increases patients' compliance with dietary prescriptions [7].

The use of a combination of tools is recommended, including the global subjective tool (SGA) and malnutrition inflammation score (MIS).

When determining the energy requirements, the RDN should consider some factors, including but not limited to RRT modalities, level of physical activity, age, sex, weight status, disease-specific determinants, metabolic stressors, treatment goals, and the patient's overall health status. Studies suggest that diet therapy may aid in lowering dialysis doses to be used safely and effectively even as the glomerular filtration rate continues to decline [8].

2. Medical nutrition therapy in different renal replacement therapy modalities

This section discusses the different nutrient requirements for the different RRT modalities.

2.1 Nutrition therapy in maintenance hemodialysis

The goal of nutrition intervention in MHD patients is to optimize their nutritional status, control blood glucose and blood pressure and fluid overload, keep renal biochemistry within safe limits, and make dietary advice as practical as possible to assist in compliance. For this reason, all renal patients must have adequate renal-specific dietetic or nutritional support [9].

2.1.1 Energy requirements

Chronic Kidney Disease (CKD) impairs energy metabolism; therefore, it is prudent to maintain adequate energy intake, which is necessary to prevent PEW. To maintain a neutral nitrogen balance and nutrition status, studies suggest that energy intake should range between 30 and 35 kcal/kg/day [3].

2.1.2 Protein requirements

For metabolically stable patients, guidelines recommend a dietary protein intake of 1.0–1.2 g/kg body weight per day to maintain a stable nutritional status. However, higher dietary protein levels may need to be considered for patients at risk of hyperglycemia and/or hypoglycemia to maintain glycemic control. Dietary fat should not be restricted because they may be important sources of calories [10].

2.1.3 Potassium

Renal dieticians should focus on individualization, consistent checks on serum potassium levels, and clinical judgment, which would provoke the utilization of other interventions other than dietary restrictions to attain normal serum levels of potassium when appropriate. Studies suggest that dietary potassium restriction may limit heart-healthy diets and lead to the intake of more atherogenic diets [10].

It is important to note that there have not been any clinical trials done on how modifying diet can influence serum potassium levels in patients with CKD [3]. Several factors could influence the shift in serum potassium levels, including [11]: medications such as angiotensin-converting enzyme (ACE) inhibitors, thiazides, and loop diuretics; gastrointestinal problems (vomiting, diarrhea, constipation); acid–base balance; glycemic control; and catabolic state.

Individualized potassium recommendations can improve patient outcomes and quality of life. Moreover, pinpointing the root cause of hyperkalemia would be ideal to help with appropriate interventions. Lindsey suggests the following reflections to help in finding the root cause of hyperkalemia [12]: If the potassium level is consistent with the current trend; if it could be a laboratory error; if there are medications that would affect potassium levels/recent dose change; if there is constipation; the patient's carbon dioxide and blood sugar trend; recent muscle mass loss, reduced appetite, and recent food intake. The author further clarifies that restricting fruits and vegetable may not have a positive impact since most potassium in diets come from coffee, tea, savory foods, beer, animal protein, and dairy.

2.1.4 Phosphorous

Recent studies point out that restrictions on dietary phosphorous may lead to worse survival and poorer nutrition status [10]. The KDQOI guidelines

recommend that to reach the decision of restricting dietary phosphorus, there needs to be the presence of progressively or persistently high serum phosphate levels, taking into consideration the trends rather than a single laboratory value and after paying attention to concomitant calcium and parathyroid hormone (PTH) levels [3].

In MHD patients, if the nutrition requirements cannot be met through the oral and enteral intake, intradialytic parenteral nutrition is recommended to improve and maintain nutritional status [3].

2.2 Nutrition therapy in peritoneal Dialysis

Guidelines recommend comprehensive and regular nutrition assessment for patients with PD, including body measurements, patient appetite, nutrition-related laboratory markers, clinical status, and dietary intake. However, different factors influence dietary recommendations, and as such, dietary recommendations are not yet universal [13]. Energy requirements range from 30 to 35 kcal/kg/day, with patients below 60 years of age proposed to get 35 kcal/kg/day and those older than 60 years to get 30 kcal/kg/day incorporating the calories from the dialysate into the calculations, which are usually mostly the dextrose because absorption occurs into the patient's body [3].

Patients undergoing PD do quite a number of exchanges in a day; thus, they experience losses of essential elements and nutrients, including amino acids, peptides, vitamins, and trace elements. Dietary restrictions are, therefore, minimal compared to MHD patients. Guidelines suggest that dietary protein should range from 1.0 to 1.3 g/kg/day and even be higher up to 1.5 g/kg/day during peritonitis [3, 13]. Dietary potassium is generally not restricted, while sodium recommendation is <4 g based on serum levels. Phosphorous allowable is between 800 and 1000 mg/day, and phosphate binders with meals are recommended if serum levels are high. The fluid is adjusted based on the dextrose concentration of the dialysate.

2.3 Nutrition therapy in continuous renal replacement therapy

Continuous Renal Replacement Therapy (CRRT) is the modality of choice for critically ill patients. While it permits better control of fluids and is hemodynamically tolerated better than intermittent hemodialysis in critically ill patients, it has greater effects on nutrition [14–16]. The clearance of CCRT is not only specific to uremic toxins; it also clears low molecular substances, which are essential. Macronutrients and micronutrients are also cleared from the patient's blood into the waste [17–20]. Studies are limited on nutrition requirements, and as such, it is impossible to generalize given the different CCRT performance modalities, types of fluids, and different prescriptions [21]. However, some studies suggest that energy requirements range from 20 to 35 kcal/kg/day with a proportion of 60–70% being carbohydrates and 30–40 being lipids, respectively, considering the anabolic and catabolic phases while considering non-nutritional calories and being cautious about overfeeding [6, 19, 22]. Protein requirements range from 1.5 to 2.5 g/kg/IBW/day [23, 24]. There is no standard recommendation for the electrolytes, vitamins, and trace elements, but the medical team should continue monitoring the critically ill patients, checking the serum levels, and correcting/or adjusting the fluids/feeds as appropriate. The medical team should monitor serum levels of phosphorous, potassium, and calcium and adjust as appropriate.

2.4 Nutrition therapy in kidney transplant

Nutrition therapy is very crucial in the acute phase of the post-transplant period (up to eight weeks) to provide adequate nutrition. This would enable wound healing and prevent catabolism, prevent infections, correct clinically significant electrolyte and metabolic abnormalities caused by the immunosuppressive medications, and aid in restoring kidney function. In the chronic phase, nutrition helps to stabilize and prevent deterioration of kidney function and prevents the development of new-onset diabetes after transplant, hypertension, hyperglycemia, anemia, dyslipidemia, and bone disease [25, 26]. Adequate calories are recommended. Therefore, energy requirements should be between 30 and 35 kcal/kg/day and protein 1.2–2.0 g/kg/day in the early period post-transplant [26, 27]. After the first months, protein intake should be reduced to about 0.8 g/kg/day in patients with adequate graft function while adjusting both energy and protein intake for physical activity levels, gender, and age [28]. Some studies suggest that the protein recommendation for the chronic phase post-transplant for the recipients without diabetes should be 0.6–0.8 g/kg/day, while for those with diabetes, it should be 0.8–0.9 g/kg/day [29]. Dietary potassium in the acute phase ranges between 2 and 4 g if the patient has hyperkalemia and unrestricted in the chronic phase unless hyperkalemic. Fluids are generally unrestricted in both phases, and phosphorous should be given as the daily required intake and supplemented if the patient has hypophosphatemia in the acute phase. In the acute phase, sodium should be restricted if blood pressure and fluids dictate in the acute phase while in thechronic phase, sodium should range between 2 and 4 g if the patient has hypertension and/or edema.

Hypophosphatemia is often common in post-transplantation, especially in the first months, and often to lead osteodystrophy and osteomalacia; therefore, it is prudent to prescribe high-phosphorous intake through diet or supplements [3].

The nutrient requirements for each RRT modality are summarized in **Table 1**.

Nutrient requirements	**Modality**				
	Hemodialysis	**PD**	**CRRT**	**Transplant**	
				Acute phase	**Chronic phase**
Energy (kcal/kg/day)	30–35 [3] <age60 35 kcal >age60 30 kcal	30–35 [3] <age60 35 kcal >age60 30 kcal	Anabolic phase: 20–25 Catabolic phase: 25–35 [22]	25–35 [3] 30–35 [27] 30–35 [26]	Adjust to maintain body weight
Protein (g/kg/day)	1.0–1.2 [3]	1.0–1.2 [3] 1.2–1.3 [13] 1.3–1.5	1.5–1.7 [23] 1.7–2.0 [22] 2.0–2.5 [24]	1.2–2.0 [26–28]	0.8–1.0 limit with chronic graft dysfunction
Potassium (mg/day)	2300	Not restricted	Monitor serum levels and adjust/ correct as appropriate	2000–4000 if hyperkalemic	Unrestricted unless hyperkalemic

Nutrient requirements	Modality				
	Hemodialysis	PD	CRRT	Transplant	
				Acute phase	Chronic phase
Sodium (mg/day)	<2400	3000–4000 Based on labs	Monitor serum levels and adjust/ correct as appropriate	Restrict if BP/ fluids dictate	2000–4000 with HTN and/ or edema
Phosphorous (mg/day)	800–1000	800–1000 Use phosphate binders	Monitor serum levels and adjust/ correct as appropriate	DRI Supplemented if serum levels are low	DRI
Calcium (mg/day)	<2000	<2000	Monitor serum levels and adjust/ correct as appropriate	1200–1500	1200–1500 [26]
Fluids (ml)	500–700 + urine output 1000 if anuric	Adjusted based on dextrose concentrations of dialysate	Individualized	Generally unrestricted	Generally unrestricted

DRI; Daily Recommended Intake.

Table 1.
Nutrient requirements in different RRT modalities.

3. Protein energy wasting

The International Society of Renal Nutrition and Metabolism (ISRNM) proposed the term protein-energy wasting (PEW) to characterize multiple metabolic alterations related to uremia, hypercatabolism, cachexia, and malnutrition associated with morbidity and mortality in kidney disease [2, 3, 30].

The major and most common cause of PEW is inadequate protein and energy intake, compounded by anorexia due to uremia, inflammation, dialysis procedure, and acidemia [31]. Insufficient nutrient intake may also result from glucose absorption from peritoneal dialysate and early satiety feeling, poor economic status, depression, and illness that affects gastrointestinal functions [32]. Nutrient loss during RRT, such as peptides, amino acids, vitamins, trace elements, and glucose, further put the patients at risk of PEW [33].

Studies suggest that adequate energy and protein intake, as recommended in **Table 1**, would help prevent PEW. Other strategies include dialysis adequacy, correcting metabolic acidosis, and treating inflammation and co-morbidities such as diabetes [34–36]. When standard preventive measures cannot reduce the loss of energy and protein stores, nutrition supplementation would suffice. A renal dietitian or equivalent can assess the patient for oral and enteral nutrition supplementation and further intradialytic parenteral nutrition [31].

4. Mineral bone disease

Many CKD patients are at an increased risk of developing CKD- Renal mineral bone disease (MBD). They develop bone lesions symptomatically showing up as pain, including back pain, tendon ruptures, pruritus, and an increased incidence of pathological fractures. Studies show that patients with renal mineral bone disorders are predisposed to calcification of the cardiovascular system and, consequently, increased morbidity and mortality.

This has led to a shift in the treatment of renal mineral bone diseases from just looking at a single biomarker, such as serum calcium levels, to further considering the disease's physiology, thus a look into serum phosphate and parathyroid hormone levels (PTH) [37].

Before the knowledge of fibroblast growth factor 23 (FGF23) and its influence on secondary hyperparathyroidism, phosphate retention was considered the main factor in the disorder occurring [38].

A series of physiological events are triggered by retained phosphate, including hyperphosphatemia, low vitamin D3, and reduced calcium levels, which stimulate parathyroid hormone secretion enhancing phosphate excretion and secondary hyperparathyroidism in end-stage renal disease [39].

There have been observations by authors that hyperphosphatemia and hypocalcemia were evidence of calcitriol deficiency, suggesting that it would be the main culprit to secondary hyperthyroidism noting the complexity of the disease because of the several elements in the pathophysiology [37].

Several studies have documented a strong relationship between serum Fibroblast growth factor 23 levels and creatinine clearance, noting that a decline in renal function had an increase in the FGF23 levels. Further, patients with End Stage renal disease would have up to a 1000-fold above the normal growth factor levels attributed to the reticence of phosphate and decline in renal clearance [40, 41].

The dire consequences associated with secondary hyperparathyroidism place an emphasis on the need to promptly manage CKD renal mineral bone diseases with a keen look and follow-up checks on markers such as serum calcium, phosphate, and parathyroid hormone and calcitriol levels. In light of this, the KDIGO guidelines recommend the onset of management dependent on serial trends of the markers [42].

Through proper nutrition education and counseling, there is a need to limit daily phosphate intake to less than 800 mg, and this is possible by educating the patients on how to read food labels to look out for high phosphate-containing foods and carbonated drinks as well as additives.

This should be done with close nutritional assessment and monitoring as most food sources that are protein in nature are rich in phosphorus. This, in turn, would help prevent protein-energy malnutrition in chronic kidney disease.

Individualized nutrition plans, as far as dietary sources of phosphates are concerned, should have the priority with consideration to intestinal absorption. Plant-based phosphate sources have lower intestinal absorption than those inorganic sources.

The use of phosphate binders has come a long way to help reduce intestinal absorption by allowing the formation of a non-absorbable complex with phosphorus in the food. Three classes of these binders are in use currently, that is, calcium-based binders, aluminum-based binders, and non-calcium-based binders. Caution should

be taken with aluminum-based binders as their long-term use has been related to osteomalacia and encephalopathy.

The choice between calcium-based and non-calcium-based binder should, on the other hand, be guided by serum calcium, calcitriol levels, and parathyroid hormones Lest hypercalcemia occurs.

Low cholesterol levels, low uric acid levels, and anti-inflammatory effects have been attributed to using Savelamer, a non-calcium phosphate binder making it have some prominence, particularly where the serum calcium levels are normal. Dialysis is another efficient way of eliminating phosphorus from the bloodstream, yet this is possible with consideration of the type of dialysis, length of dialysis, and type of dialysate.

The most common length of hemodialysis is 4 hours with thrice-a-week sessions that eliminate up to 2600 mg of phosphate levels. However, this may be slightly lower for low resources settings where dialysis is done twice a week. Peritoneal dialysis will succeed at up to 220mmg of phosphate elimination when done four times a day with two-liter exchanges.

4.1 Emerging treatment options for CKD MBD

A new class of phosphate binders, sucroferric oxyhydroxide (PA21) and ferric citrate (JTT-751), are iron-based, calcium-free phosphate binders recently advanced into clinical practice. These have also been used to fix anemia in CKD and attenuate vascular calcification.

Klotho supplementation has also been suggested as a prophylactic or therapeutic therapy for averting secondary hyperparathyroidism. Equally, the usage of anti-FGF23 monoclonal antibodies (FGF23-Ab) to counteract the negative effects of high levels of FGF23 in animal models has been assessed. While counteraction of FGF23 has been characterized by improvement in secondary hyperparathyroidism, increased levels of serum phosphate, aortic calcification, and higher risk of mortality have been reported. Therefore, the therapeutic applicability of FGF23-Ab in humans is yet to be proven.

5. Conclusion

Renal replacement therapy has a great impact on the nutrition status and the overall well-being of renal patients. This chapter discussed medical nutrition therapy focusing on different modalities of renal replacement therapy. It delved into nutrition assessment. Its benefits, and nutrient requirements for each renal replacement therapy. It, finally, discussed the nutrition management of protein energy wasting and mineral bone disease, conditions which are very common among people with end-stage renal diseases.

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Conflict of interest

The authors declare no conflict of interest.

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References

[1] Sikand G, Handu D, Rozga M, de Waal D, Wong ND. Medical nutrition therapy provided by dietitians is effective and saves healthcare costs in the Management of Adults with Dyslipidemia. Current Atherosclerosis Reports. 2023;**25**(6):1-12. DOI: 10.1007/s11883-023-01096-0

[2] Lu M, Pilla SJ, Oh SH. Diabetes mellitus: Dietary management. In: Caballero B, editor. Encyclopedia of Human Nutrition. 4th ed. Oxford: Academic Press; 2023. pp. 234-251. DOI: 10.1016/B978-0-12-821848-8.00021-4

[3] Ikizler TA et al. KDOQI clinical practice guideline for nutrition in CKD: 2020 update. American Journal of Kidney Diseases. 2020;**76**(3):S1-S107. DOI: 10.1053/j.ajkd.2020.05.006

[4] As'habi A, Najafi I, Tabibi H, Hedayati M. Prevalence of protein energy wasting and its association with cardiovascular disease risk factors in Iranian peritoneal Dialysis patients. Iranian Journal of Kidney Diseases 2019;**13**(1):8

[5] Visiedo L et al. The impact of nutritional status on health-related quality of life in hemodialysis patients. Scientific Reports. 2022;**12**(1):3029. DOI: 10.1038/s41598-022-07055-0

[6] Ostermann M, Lumlertgul N, Mehta R. Nutritional assessment and support during continuous renal replacement therapy. Seminars in Dialysis. 2021;**34**(6):449-456. DOI: 10.1111/sdi.12973

[7] Paes-Barreto JG et al. Can renal nutrition education improve adherence to a low-protein diet in patients with stages 3 to 5 chronic kidney disease? Journal of Renal Nutrition. 2013;**23**(3):164-171. DOI: 10.1053/j.jrn.2012.10.004

[8] Hanafusa N, Lodebo BT, Kopple JD. Current uses of dietary therapy for patients with far-advanced CKD. Clinical Journal of the American Society of Nephrology. 2017;**12**(7):1190-1195. DOI: 10.2215/CJN.09340916

[9] Rahman T et al. Provision of renal-specific nutrition knowledge for changing dietary practice in Bangladeshi hemodialysis patients. PEC Innovation. 2022;**1**:100028. DOI: 10.1016/j.pecinn.2022.100028

[10] Kalantar-Zadeh K et al. Dietary restrictions in Dialysis patients: Is there anything left to eat? Seminars in Dialysis. 2015;**28**(2):159-168. DOI: 10.1111/sdi.12348

[11] Udensi UK, Tchounwou PB. Potassium homeostasis, oxidative stress, and human disease. International Journal of Clinical and Experimental Physiology. 2017;**4**(3):111-122. DOI: 10.4103/ijcep.ijcep_43_17

[12] Zirker L. Updated Guidelines Challenge Current Standard for Potassium Intake in CKD. Florida, USA: Renal and Urology News; 2023. Available from: https://www. renalandurologynews.com/home/ departments/renal-nutrition-update/ diet-advice-for-blood-pressure-controlgoes-beyond-sodium-restriction/ [Accessed May 12, 2023]

[13] Kimball T, Holotka J, Habura I, Pawlaczyk K. Nutritional status in peritoneal Dialysis: Nutritional guidelines, adequacy and the Management of Malnutrition. Nutrients. 2020;**12**(6):1715. DOI: 10.3390/nu12061715

[14] Bonnassieux M et al. Renal replacement therapy modality in the ICU and renal recovery at hospital discharge. Critical Care Medicine. 2018;**46**(2):e102-e110

[15] Rabindranath KS, Adams J, MacLeod AM, Muirhead N. Intermittent versus continuous renal replacement therapy for acute renal failure in adults. Cochrane Database of Systematic Reviews. 2007;**18**(3):CD003773. DOI:10.1002/14651858.CD003773.pub3

[16] Symons JM et al. Demographic characteristics of pediatric continuous renal replacement therapy: A report of the prospective pediatric continuous renal replacement therapy registry. Clinical Journal of the American Society of Nephrology. 2007;**2**(4):732-738

[17] Fiaccadori E, Regolisti G, Maggiore U. Specialized nutritional support interventions in critically ill patients on renal replacement therapy. Current Opinion in Clinical Nutrition & Metabolic Care. 2013;**16**(2):217-224

[18] Frankenfield DC, Badellino MM, Reynolds HN, Wiles CE III, Siegel JH, Goodarzi S. Amino acid loss and plasma concentration during continuous hemodiafiltration. Journal of Parenteral and Enteral Nutrition. 1993;**17**(6):551-561

[19] Honoré PM et al. Nutritional and metabolic alterations during continuous renal replacement therapy. Blood Purification. 2013;**35**(4):279-284

[20] Bellomo R et al. High protein intake during continuous hemodiafiltration: Impact on amino acids and nitrogen balance. The International Journal of Artificial Organs. 2002;**25**(4):261-268

[21] Fishman G, Singer P. Metabolic and nutritional aspects in continuous renal replacement therapy. Journal of Intensive Medicine. 2023. DOI: 10.1016/j.jointm.2022.11.001

[22] Onichimowski D, Goraj R, Jalali R, Grabala J, Mayzner-Zawadzka E, Czuczwar M. Practical issues of nutrition during continuous renal replacement therapy. Anaesthesiology Intensive Therapy. 2017;**49**(4):309-316. DOI: 10.5603/AIT.a2017.0052

[23] Singer P et al. ESPEN guideline on clinical nutrition in the intensive care unit. Clinical Nutrition. 2019;**38**(1):48-79

[24] McClave SA et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (ASPEN). JPEN. Journal of Parenteral and Enteral Nutrition. 2016;**40**(2):159-211

[25] Veroux M et al. Nutrition in kidney transplantation. The International Journal of Artificial Organs. 2013;**36**(10):677-686

[26] Hong SH, Kim EM, Rha MY. Nutritional intervention process for a patient with kidney transplantation: A case report. Clinical Nutrition Research. 2019;**8**(1):74-78. DOI: 10.7762/cnr.2019.8.1.74

[27] Akbulut G, Gencer-Bingol F. Medical, nutritional therapy for renal transplantation in the COVID-19 pandemic. World Journal of Transplantation. 2021;**11**(6):212-219. DOI: 10.5500/wjt.v11.i6.212

[28] Losappio V et al. Nutrition-based Management of Inflammaging in CKD and renal replacement therapies. Nutrients. 2021;**13**(1):267. DOI: 10.3390/nu13010267

[29] Tiger NB. Owner's manual: Nutrition Care for Your Kidney Transplant. Journal of Renal Nutrition. 2019;**29**(3):249-255

[30] Carrero JJ et al. Etiology of the protein-energy wasting syndrome in chronic kidney disease: A consensus statement from the International Society of Renal Nutrition and Metabolism (ISRNM). Journal of Renal Nutrition. 2013;**23**(2):77-90. DOI: 10.1053/j.jrn.2013.01.001

[31] Alp Ikizler T et al. Prevention and treatment of protein energy wasting in chronic kidney disease patients: A consensus statement by the International Society of Renal Nutrition and Metabolism. Kidney International. 2013;**84**(6):1096-1107. DOI: 10.1038/ki.2013.147

[32] Wright M et al. Disturbed appetite patterns and nutrient intake in peritoneal dialysis patients. Peritoneal Dialysis International. 2003;**23**(6):550-556

[33] Ikizler TA, Flakoll PJ, Parker RA, Hakim RM. Amino acid and albumin losses during hemodialysis. Kidney International. 1994;**46**(3):830-837

[34] Schoenfeld P, Henry R, Laird N, Roxe D. Assessment of the nutritional status of the National Cooperative Dialysis Study population. Kidney International. Supplement. 1983;**13**:S80-S88

[35] de Brito-Ashurst I, Varagunam M, Raftery MJ, Yaqoob MM. Bicarbonate supplementation slows progression of CKD and improves nutritional status. Journal of the American Society of Nephrology. 2009;**20**(9):2075-2084

[36] Goldstein SL, Ikizler TA, Zappitelli M, Silverstein DM, Ayus JC. Non-infected hemodialysis catheters are associated with increased inflammation compared to arteriovenous fistulas. Kidney International. 2009;**76**(10):1063-1069

[37] Moe SM, Drüeke T, Lameire N, Eknoyan G. Chronic kidney disease-mineral-bone disorder: A new paradigm. Advances in Chronic Kidney Disease. 2007;**14**(1):3-12. DOI: 10.1053/j.ackd.2006.10.005

[38] Isakova T et al. Fibroblast growth factor 23 is elevated before parathyroid hormone and phosphate in chronic kidney disease. Kidney International. 2011;**79**(12):1370-1378. DOI: 10.1038/ki.2011.47

[39] Habas ES, Eledrisi M, Khan F, Elzouki A-NY. Secondary hyperparathyroidism in chronic kidney disease: Pathophysiology and management. Cureus. 2021;**13**(7):e16388. DOI: 10.7759/cureus.16388

[40] Gutierrez O et al. Fibroblast growth factor-23 mitigates hyperphosphatemia but accentuates calcitriol deficiency in chronic kidney disease. Journal of the American Society of Nephrology. 2005;**16**(7):2205-2215. DOI: 10.1681/ASN.2005010052

[41] Damasiewicz MJ, Toussaint ND, Polkinghorne KR. Fibroblast growth factor 23 in chronic kidney disease: New insights and clinical implications. Nephrology (Carlton). 2011;**16**(3):261-268. DOI: 10.1111/j.1440-1797.2011.01443.x

[42] Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD Update Work Group. KDIGO 2017 clinical practice guideline update for the diagnosis, evaluation, prevention, and treatment of chronic kidney disease-mineral and bone disorder (CKD-MBD). Kidney International Supplements (2011). 2017;7(1):1-59. DOI: 10.1016/j.kisu.2017.04.001