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Medical Nutrition Therapy for Hemodialysis Patients

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1. Introduction

Nutrition in hemodialysis is very important in decreasing complications and improving quality life of patients. Nutrition program on patients with chronic renal failure on dialysis plays an important role in the process of treatment.

The purposes of medical nutrition therapy in dialysis patients are to promote the nutrition to correct patients' appetite, to correct systemic complications composed by the loss of nephrons in progress, to reduce of protein catabolism to the lowest level, to relieve or prevent the cardio-vascular, cerebrovascular, peripheral vascular diseases formation, to prevent increasing fluid and electrolyte disorders, to reduce uremic symptoms such as itching, nausea, vomiting, loss of appetite and to ensure optimum nutrition. In addition, medical nutrition helps to avoid high-potassium and sodium from the diet, to prevent pulmonary edema, hypertension and heart failure, to prevent renal osteodystrophy keeping the consumption of calcium and phosphorus under control, to prevent protein energy malnutrition with saving patients' food consumption and detecting nutritional status with methods such anthropometric measurements, laboratory findings, subjective global assessment (SGA) (Cianciaruso 1995, Kopple 2004, Mahan 2012). Negative changes (hyperkalemia, hiperfosfotemi, peripheral and pulmonary edema) in fluid-electrolyte balance occur in patients who do not comply to the diet.

In this chapter, assessment of nutritional status in hemodialysis patients and preparation of individual dietary training programs for patients will be discussed.

2. Assesment of the nutritional status

Regular assessment of nutritional status in hemodialysis patients is important and early detection of malnutrition can be helpful in improving this condition (Fouque 2003).



The results of studies indicate that hemodialysis patients are at risk of malnutrition. The evaluation methods used in the nutritional status showed that 18-75% prevalence of malnutrition in hemodialysis patients, malnutrition could cause a worse outcome and subsequent mortality(Dwyer 2005). Chazot's study was assessed the nutritional status of twenty hemodialysis patients receiving hemodialysis treatment more than 20 years and was showed that hemodialysis treatment caused to malnutrition the long period of time(Chazot 2001).

Malnutrition occurs depending on several factors in hemodialysis patients. Especially, there is reduction of protein-energy intake because of inappropriate dietary restrictions, anorexia, and taste alterations, promoting malnutrition in most patients entering dialysis (Lavılle 2000). Studies illustrate that there are two types of malnutrition in dialysis patients: The first type is specified by uraemic syndrome and reduction in serum albumin levels due to decreasing energy and protein intake. It should be provided improvement with adequate energy and protein intake. The second type is associated with inflammation and atherosclerosis, high cardiovascular mortalite(MIA Syndrome). Prominent features of this type, proinflammatory cytokines, increased oxidative stress, increased protein catabolism, increased resting energy expenditure, hypoalbuminemia (Stenvinkel 2000, Baltzan 1998). İn addition, malnutrition due to poor nutrition, chronic volume overload congestive heart failure and systemic hypertension, uraemic bone disease and extraskeletal metastatic calcification due to hyperphosfotemia development are other adverse conditions encountered as a result of the diet incompatibility.

In general, there are catabolic and inflammatory situation in patients with end-stage. Patients receiving dialysis treatment are seen in tissue loss in the course of time. At the start of dialysis treatment, having a high level adipocyte tissue can be advantageous for individuals. Dialysis patients who have excess body fat mass are being protected against this situation because of more energy storage. Recent data shows that patients who are overweight or obese had higher rates of survival than normal or in hemodialysis patients. Low serum albumin level (hypoalbuminemia) revealed that the obese are less in HD patients. Reduction in mortality in overweight patients was reported as well as indicators of nutritional status of overweight HD patients was significantly higher than underweight HD patients and to be shorter than the duration of hospital stay. (Glanton 2003, Guida 2004, Kalantar-Zadeh 2005)

Different methods are used in the evaluation of nutritional status in hemodialysis patients. Biochemical, anthropometric measurements, nitrogen and energy balance techniques, record of food intake, subjective global assessment, bioimpedance analysis (BIA), Dual-Energy X-ray Absorptiometry (DEXA), creatinine kinetics, neutron activation analysis and nuclear magnetic resonance spectrometry and serum markers: albumin, pre-albumin, insulin-like growth factor-1 (IGF-1) and transferrin; main proteins of the acute phase (C-reactive protein (CRP), serum amyloid A), secondary proteins of the acute phase (fibrinogen, ferritin, complement), cytokines (interleukin-6 (IL-6), tumour necrosis factor) are used to assess the nutritional status of patients with chronic renal failure (Basile 2003).

Some studies (Beddhu 2002, Panichi 2006) describe hypoalbuminemia in HD patients as a strong indicator for mortality and morbidity. As a result of malnutrition, albumin synthesis

decreases and develops hypoalbuminemia. In fact, the serum albumin level is a powerful way directly correlated with dietary protein, but recent literature emphasizes that the effect of serum albumin concentration on the inflammatory response. Albumin is a negative acute phase protein, except nutritional status, and its synthesis is supressed during inflammation. For this reason, there are limitations in the use of serum albumin level in order to assess the nutritional status of patients due to be affected by malnutrition and inflammatory reactions (Santos 2003). Indeed, because of longer half life, it cannot be a sensitive indicator for nutritional therapy. In studies, significant negative correlation was found between prealbumin and CRP (Kaysen 1995, Owen 1998, Sathishbabu 2012). Prealbumin is a negative marker of inflammation level that correlates positively and significantly with other nutritional markers in ESRD patients on hemodialysis (Sathishbabu 2012). Because of the shorter half life of prealbumin, many authors consider prealbumin to be a better marker of nutrition than serum albumin (Mittman 2001, Kalantar-Zadeh 2003). That is considered one of the indicators of uremic malnutrition less than 29mg/dl of serum prealbumin levels in patients on dialysis, serial measurements are recommended in the evaluation of nutritional status (Pupim 2004). Serum creatinine concentration (less than 10 mg/dl) should be evaluated for PEM and skeletal muscle wasting, because it indicates reduced dietary protein intake and skeletal muscle mass(Janardhan 2011).

Subjective Global Assessment (SGA) is often preferred by experts to assess the nutritional status in chronic dialysis patients as relatively quick, easy, and cheaper than other methods (Mutsert 2009). İt is important that SGA was proposed by the National Kidney Foundation (NKF) Kidney Disease/Dialysis Outcomes and Quality Initiative (K/DOQI) for nutritional assessment in the adult dialysis patients(K/DOQI 2000).

Subjective Global Assessment (SGA) reveals that there are seven components to assess nutritional status; two components related to physical examination (indicator of fat and muscle loss and nutritional status-associated with changes in fluid balance) and five components of medical history (weight change, diet, gastrointestinal symptoms, functional capacity, disease and nutrition relationship needs) (Steiber 2004). While SGA scoring points are given in each section of 1-7 and are categorized as 1-2 points (bad), 3-5 points (moderate), 6-7 points (normal). If it is received from this SGA most 6 or 7 points refers mild malnutrition. Most of 3, 4 or 5 points show moderate malnutrition. Most of the findings of sections 1 or 2 points received are recognized as marked malnutrition and severe malnutrition (Janardhan 2011). European Best Practice Guidelines (EBPG) on diagnosis and monitoring of malnutrition proposed that the SGA can be used to determine malnutrition in hemodialysis patients (Fouque 2007).

Nutritional history and dietary record provide information about nutrition of patients and determine for malnutrition development at risk whether or not. Because of record of food intake is taken long-term, bored patients may cause to give false information. Therefore, record of food intake 3-day to get more accurate for patients (Kalantar-Zadeh 2003).

3. Energy

Enough energy should be taken for the effective use of dietary protein and the protection of the nutrients stores of body. Energy metabolism is impaired and is composed of negative energy balance because of disrupted cellular energy metabolism in hemodialysis patients (Mak 2011). Therefore, to consume enough energy identified by the daily energy requirements of ESRD patients provides a positive nitrogen balance and preventing tissue destruction and protein catabolism.

The anorexia nervosa was often encountered in patients in the next few months from the start of dialysis therapy. This is because, even though dramatic changes in their lives, psychological conditions, can not be adapted to a new and restricted diet. It has been reported if protein and energy intake are not increased in these patients, lost energy is stored with muscle mass of patients, and the amount of body fat is decreased (Fouque 2003). The studies have suggested that the dietary energy failure is more on dialysis treatment days than non dialysis treatment days (Burrowes 2003, Rao 2000). In a prospective multicenter clinical trial that included 1901 participants of the Hemodialysis Study, dietary energy intake was 1.02 kcal/kg/day less on dialysis treatment days than on nondialysis treatment days. (Burrowes 2003, Stark 2011).

Some studies indicated that energy intake was low in hemodialysis patients. Poor appetite and hypermetabolism fairly reduce food intake in hemodialysis patients (O'Keefe 2002, Nakao 2003, Morais 2005, İkizler 2002, Pumpkin 2002). When the recommended energy requirements compared with consumed amounts, it is concluded that energy intake is inadequate in 90% of patients (Rocco 2002)

When energy intake of hemodialysis patients was 32-38 kcal/kg/day, have not been reported any increasing or decreasing in nitrogen balance and anthropometric parameters, and developing a negative or a positive energy balance. (Kopple 2004).

Studies demonstrated that low-energy and with low protein diet cause weight loss and malnutrition in patients. For these reasons, sedentary, non-obese dialysis patients's requirements of energy coming from all sources should be determined, according to NKF-DOQI, ESPEN and EDTNA-ERCA 2002; respectively, 35 cal/kg/day (under the age of 60), 30-35 cal/kg/day(over the age of 60); 35 cal/kg/day and 30-35 cal/kg (ideal body weight)/day. (Kopple 2001, Kopple 2004, Cano 2006, Fouque 2003). In some studies, it was shown that hemodialysis patients should receive daily energy as 30-40 kcal/kg (Kalantar-Zadeh 2003, Stenvinkel 2000).

4. Protein

Protein requirement increases due to the dialysate losses and catabolism in hemodialysis patients. In research, it is emphasized that the inadequate protein intake increases mortality (Ohkawa 2004).

Raj et al's study showed that hemodialysis increases both protein synthesis and degradation. The net effect of hemodialysis is loss of nitrogen in skeletal muscle. Protein synthesis and degradation increases by 50-100% of normal values. Hemodialysis causes to increase in catabolic indicators such as interleukin-1 (IL-1), interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF- α). This increasing in the production of cytokines causes in protein degradation. Reasons for increased protein requirement; amino acid losses into the dialysate, increased protein catabolism, metabolic and hormonal changes(Raj 2007).

There are 0.2-0.3 g/kg or 6-8 g/day of protein, amino acids (aa) and peptide losses with the dialysis fluid during hemodialysis. Protein catabolism increases with these losses due to metabolic disorders. The lost in amino acids needs to be replaced to avoid negative nitrogen balance. According to "National Kidney Foundation Dialysis Outcome Quality Initative (NKF-DOQI)" and studies by other investigators to compensate for residual renal losses, dietary protein should be adjusted at least 1.2 g/kg/day in hemodialysis patients as indicated (Kopple 2001, Mahan 2012, Kalantar-Zadeh 2003, Locatelli 2005).

According to ESPEN, adjusted diet protein should be consumed as 1.1-1.2 g / kg / day and should be high in the biological value (of animal origin) of 50 % protein in hemodialysis patients (Mehrotra 2001, Karalis 2002, Cano 2006). Furthermore, the amount of protein of the patient's diet is determined by considering the state of hydration adjusted body weight, glomerular filtration rate and with the course of illness (Nissenson 2008). To determine the adequacy of protein intake in dialysis patients, a good evaluation parameter is BUN value under 120 mg. When 1.2 g / kg / day protein intake, it was indicated protein catabolic rate is associated with low morbidity, provided adequate control of blood urea concentration, improved the nutritional parameters (anthropometric measurements) and biochemical findings (blood albumin, total protein, blood, blood cholesterol, etc.), provided a positive nitrogen balance in dialysis patients (Bergstrom 1993, Amanda 2010).

However, it is required that adequate caloric intake prevent the use of protein as an energy source with gluconeogenessis. Otherwise, a positive nitrogen balance can not be provided in spite of high protein intake. When patients were given a low protein diet, should be followed adequate energy intakes and adequate phosphorus intakes of patients to ensure optimal nutrition, and to prevent malnutrition (Locatelli 2005, Gribotto 2012).

Metabolic acidosis in hemodialysis patients increases protein catabolism, the branchedchain amino acid degradation and muscle glutamine release. Amino acids and glutamine metabolism allow the formation of ammonium and bicarbonate excretion. Changes at branched-chain amino acids levels of muscle and plasma occur in hemodialysis patients. As a result of hemodialysis treatment, plasma valine, muscle valine, plasma leucine are low, muscle leucine, plasma isoleucine, muscle isoleucine are normally observed (Cano, Fouque 2006). Branched-chain amino acids play a regulatory role against chronic acidosis. After acidosis subside is given a support and enriched with branched-chain amino acids and valine during hemodialysis, branched-chain amino acids level of plasma and intracellular are enhanced. (Raj 2000).

Branched-chain amino acids improve appetite in hemodialysis patients. 6.6-15.7 g daily intake of essential amino acids in hemodialysis patients corrected the their nutritional parameters. In patients who underwent 12 g oral branched-chain amino acid a day showed improvement in protein and energy purchases in one month, in the anthropometric measurements six months later. Consantrations of albumin increased 3:31 g / dL to 3.93 g / dL. (Cano, Fouque 2006) According to Raj, although amino acid repletion increased in muscle protein synthesis, no decrease in muscle protein breakdown during HD treatment was observed (Raj 2007)

There is a dynamic effect of animal protein (such as egg, dairy etc.) on renal function in short-term clinical trials. But long-term effects on the normal kidney functions are still unknown. There are mechanisms shown to reveal the different effects of animal and vegetable proteins on renal function including differences in hormones, protein metabolism and interaction with micronutrients. Healthy individuals with normal renal function, long-term consumed high-protein diet (whether of animal protein or vegetable protein) may cause kidney damage and accelerate chronic renal failure. However, long term studies are necessary to determine the different effect of the consumption of animal or vegetable protein diet on renal functions (Bernstein 2007).

5. Carbohydrate

Carbohydrate intake requires enough energy and to maintain reserve protein that can be used for the synthesis protein of tissue.

When dialysis fluid not containing glucose is used for 4 hours, 28 g glucose is lost in hemodialysis. However, when 11 mmol / L glucose was added to the dialysis fluid, the patient gained approximately 23 g of glucose. When glucose is removed by dialysis in the extracellular fluid, loss of the glucose is completed with absorbed carbohydrates, destruction of liver glycogen, and glyconeogenesis in order to avoid symptomatic hypoglycemia. Then, increased protein breakdown and urea synthesis begin. Glucose-free dialysis is reduced pyruvate. Pyruvate does not change with glucose dialysis. Glyconeogenesis may be stimulated with glucose-free dialysis. However, there are negative effects of glucose intake such as hyperglycemia, hyperinsulinemia, hyperlipidemia, obesity etc(Lindholm 1998).

Deterioration of glucose metabolism and insulin resistance develops in chronic renal failure. This situation results in rising levels of glucose and urea when coupled with increased hepatic gluconeogenesis. Insulin metabolism in uremia shows severe abnormalities. Basal insulin secretion is reduced and receives limited response to glucose infusion (Kopple 2004).

In one study, it was observed occurrence of the insulin resistance impaired, muscle glucose uptake and nonoxidative glucose metabolism, in the presence of chronic uremia, but recovered after dialysis (Foss 1996).

Uric acid is generated during fructose metabolism. Serum uric acid levels have been found to correlate with fructose intake. High serum uric acid was associated with hypertension, in-

flammation, chronic kidney disease and the intake of fructose and added sugars (Feig 2008, Brymora 2012). But fruits containing fructose have some beneficial substances such as antioxidants. Therefore, it is possible that fructose intake from natural fruits with regular diet. (Jalal 2010, Brymora 2012).

Carbohydrate from the diet should be higher to provide enough energy, to protect the back-up protein to be used for tissue protein synthesis, to cover the energy deficit. It should provided 60-65% of daily energy from carbohydrates (Kopple 2004). Most patients have difficulty in meeting energy needs with low protein diets. For this reason, the energy gap can be covered by glucose polymers (starch), sugar, simple sugars, pure carbohydrate sources. Patients with diabetes should avoid concentrated sweets (Mahan 2012).

6. Lipids

Recent evidence suggests that protein calorie malnutrition often begins incipiently when the glomerular filtration rate (GFR) is about 28 to 35 mL/min/1.73 m2 or even higher (Kopple 1994) and continues to fall gradually as the GFR decreases below these values (Laville 2000). Reduced quantity of GFH causes a significant increase in plasma lipid levels(Liu 2004). Especially, hyperlipidemia consists when creatinine clearance is below 50 ml/min in patients. In Rutkowski's study, accumulation of triglycerides-rich lipoproteins was associated with increased lipogenetik gene expression of enzymes and the high quantity triglycerides production by renal deficiency (Rutkowski 2003, Liu 2004).

Usually, there are hypertriglyceridemia and hyperlipidemia in hemodialysis patients. Low-density lipoprotein (LDL) and very low density lipoprotein (VLDL) are high concentration, high density lipoprotein (HDL) cholesterol concentration is low. The main reason of hypertriglyceridemia is the lack of removal of triglycerides from the circulation (Kwan 2007, Lacquaniti 2010). In these patients, it has been reported decreased lipoprotein lipase, hepatic lipase enzyme activity.

Generally it is known to decrease in carnitine storages in hemodialysis patients with malnutrition. In addition, carnitine leaves from the extracellular fluid during dialysis therapy and this situation causes a sudden drop in serum level of carnitine. Carnitine deficiency is caused by deterioration of long-chain fatty acid oxidation and thus deficiency of energy(Matera 2003, Flanagan 2010). It was determined to put on 750 mg/day carnitine supplementation in diet of hemodialysis patients, reduced the level of plasma TG and LDL cholesterol and increased HDL cholesterol levels(Naini 2012).

Hyperlipidemia develops in a large part of dialysis patients, the amount of fat in the diet should not be higher. Saturated fat content of the diet should be reduced and unsaturated fat content should be increased (Vaziri 2006).

Hyperlipidemia progresses in the majority of patients with CKD; therefore, content of fat in the diet should not be high. Total energy from fat should not exceed 25% to 30. İt should be reduced saturated fat content of the diet and increased unsaturated fat content.

It is recommended reducing saturated fat intake (total energy <7%) and cholesterol intake (<200 mg / day). Total fat content of the diet should be between 25-35% of energy and monounsaturated fatty acids 15-20% of total energy, polyunsaturated fatty acids 10% of total energy of the diet (Nissesson 2008). Recommended foods for patients with a high biological value such as meat, eggs contain high cholesterol. Therefore assessment of serum cholesterol levels should be specific for each patient. If patients have hypertriglyceridemia and high cholesterol, regulation dietary fat content, weight control, increased physical activity, reducing the use of hypertonic solution, restriction of simple sugars of dietary intake are recommended.

Signs and symptoms of deficiency of essential fatty acids such as dry and itchy skin, hair loss, abnormal prostaglandin synthesis are observed in dialysis patients. EPA and DHA which replace arachidonic acid in cell membrane and prevents the formation of pro-inflammatory compounds are part of linolenic acid in fish oil (n-3 fatty acids). According to FDA, intake of n-3 fatty acid with food supplements should not exceed 3 g / day (Vergili-Nelsen JM 2003).

The studies were reported that omega-3 food supplementation reduced levels of triglyceride (Bouzidi 2010, Skulas-Ray 2008), LDL cholesterol and CRP (Saifullah 2007), as well as Omega-6 / omega-3 polyunsaturated fatty acids ratio was important for inflammation and mortality rate in hemodialysis patients(Noori 2011, Daud 2012).

7. Water and electrolytes

The fluid adjustment should be made according to edema and dehydration in the patient. In hemodialysis patients, if conditions such as swelling of the eyes, hands or feet, fluid weight gain, shortness of breath, increased blood pressure or tachycardia are observed, fluid consumption should be restricted (Hegel 1992, Saran 2003). Hemodialysis patients should reduce fluid intake and should limit food consumption such as tea, coffee, soda, water, fruit juices, ice cream, sherbet, gelatin, soups and heavy sauces.

Dietitians, especially renal dietitians, are most often cited as the trusted source on providing information on fluid management and delivering dietary advice (Smith 2010). Research about fluid balance dietician indicates that it is important to teach patients how to deal with thirst without drinking liquids. Proposals such as sucking on ice chips, cold sliced fruit, or sour candies and using artificial saliva are recommended (Mahan 2012).

Controlling sodium and fluid intake are important components of the HD diet. Extracellular volume expansion is the main pathophysiologic determinant of hypertension in HD patients. Water and sodium intake in hemodialysis patients are adjusted according to the amount of urine, fluid balance and blood pressure. With hemodialysis, potassium restriction is often necessary, but the measure of restriction depends on residual renal function (Stark 2011).

Body weight gain during hemodialysis is recommended and should not exceed 1.5-2 kg. A recommended daily amount of fluid of hemodialysis patients should be 500ml + the urinary output in a day or around 1000-1500 ml. Sodium restriction should be based on the amount of urine. A mild salt restriction as 3-4 g / day is sufficient in oliguric patients that have an amount of urine totaling more than 1 liter per day. Anuric hemodialysis patients may consume up to 1 liter of liquid 1-1.5-2 g / daily of salt. If hypertension or heart failure is present, salt and water restriction should be more monitored more delicately. Excess salt intake causes an increase for the feeling of thirst and liquid intake (Fouque 2003, Lindley 2009).

To reduce sodium intake in hemodialysis patients, olives, pickles, cured meats, garlic sauce, soy sauce, canned foods, sausages, processed meats, ham, chips, pretzels and instant soups should be removed from the diet. Different spices, such as vinegar and lemon, can be used for consumption of unsalted foods or as a salt substitute.

Potassium levels are affected by hemodialysis therapy with the degree of residual renal function and net tissue breakdown (e.g. due to infections) and acid-base status. In HD patients, serum potassium concentrations may change to net intestinal potassium absorption or excretion. An example of this change or excretion is diarrhea. Serum potassium is impressed by dietary potassium intake. It is thought this relationship is stronger when the potassium intake is very low or very high in diets of HD patients (Kaveh 2001, Noori 2010).

Potassium restriction is often required because hemodialysis patients are usually anuric. Anuric HD patients are recommended to restrict their potassium intake to 1600-2000mg daily. Hypokalemia may occur with symptoms such as severe vomiting, diarrhea, diuretic use, due to the reduction of potassium. In this case, the potassium content of the diet should be increased (Fouquo 2003).

When blood potassium levels in dialysis patients are high, treatment of the patient's diet should be reviewed as a priority. The food consumption should be limited to reduce the intake of potassium levels, such as milk, meat products, fruits, legumes, cereals, dried fruits and vegetables, etc.

8. Vitamin and minerals

Some studies demonstrate vitamin and mineral supplements for the long-term hemodialysis patients. Hemodialysis patients are potentially at risk of deficiency and excess of trace elements (Inamoto 2003). Given that essential trace elements play key roles in multiple biological systems, including immunological defense against oxidation and infection. It has been hypothesized that the increased morbidity and mortality seen in hemodialysis patients may in part be due to the imbalance of trace elements that has not yet been recognized (D'Haese 1996, Coombes 2012).

In HD patients, there are many problems associated with the lack of food intake. Poor nutrition, restriction of foods that are rich in water-soluble vitamins, foods that are rich in potassium, metabolic disorders caused by uremia, infection and diseases such as gastrointestinal

diseases or complications associated with reduced intake of foods are some of the possible scenarios. The lack of foods containing vitamins leads to vitamin and deficiencies that could cause of further possible complications in dialysis patients. (Mahan 2012).

In dialysis patients, B6, folic acid and vitamin C deficiencies have been observed (Coveney 2011). Vitamin B6 deficiencies, especially as it plays in amino acid utilization and lipid metabolism and maintains a critical role as a coenzyme, are very important to monitor closely. Deficiencies in either folic acid, vitamin B6 or vitamin B12 can greatly affect to capacity of the others to function properly (Wierzbicki 2007). This bond requires all to work in synchrony for optimum performance of the metabolic pathway. If Vitamin B6 and folic acid supplements are not used in dialysis treatment, pyridoxine and folic acid may often reduce red cells and plasma (Steiber 2011). In dialysis patients, an additional intake of vitamin B6 reduces plasma cholesterol and triglyceride levels and additional intake of folic acid can reduce the high levels of homocysteine, which has been determined to be a risk factor for cardiovascular disease (Dumm 2003). Vitamin B6 and folic acid intake in HD patients are higher than normal healthy subjects, and respectively, the recommended intake varied between 1 mg and 10mg per day in most studies (Steiber 2011).

In addition, the loss of vitamin C has been observed in HD patients. Increasing vitamin C in the diet to a recommended amount of 100-200 mg/daily was at once the standard suggestion of mending this problem. However, the intake of higher doses of ascorbic acid was found to possibly lead to the accumulation of oxalate, which is the metabolite of vitamin C. With oxalate accumulation, formation of calcium oxalate stones in the kidneys, the accumulation of calcium oxalate in internal organs and blood vessels, hypercalcemia and hiperoxalemia are all symptoms (Moyad 2009). Recently, the daily requirement of vitamin C in patients undergoing hemodialysis is suggested at 60-90 mg/daily (Kopple 2004). In addition, ascorbic acid supplementations, are composed of iron overload. In uremic patients, it is recommended to prevent resistance to erythropoietin. Vitamin C supplementation increased intestinal iron absorption in these patients, which may reduce the incidences of iron deficiency anemia (Handelman 2011).

Thiamine sources are whole grain and enriched bread and cereals, peas, beans, nuts, brown rice, and meats. It is absent in rice and some cereal products. Thiamine nutritional value is lost with cooking, polishing and purifying. Thiamine is not stored in the body and is excreted in the urine, because of a water-soluble vitamins (Steiber 2011). The addition of thiamin is controversial among some experts. However, 30 mg thiamine has been shown to support the improving of the activity of translocases red blood cells. Thiamine requirements should be 1.5 mg / daily, when dialysis patients have operations, infections have a high risk of developing, convulsions of the neurological symptoms can occur and large quantities of glucose adds to the diet(Fattal-Valevski 2011, Fouque 2003).

25 (OH) D3 levels of dialysis patients are known to be lower than the normal population. Treating vitamin D deficiencies shows the important contributions and progressions towards enhancing the quality of life with dialysis patients (Cheng 2007). The studies showed 25(OH)D3 levels significantly lower than 15 ng / mL (37 nmol / L) in patients. The lowest value of vitamin D is accompanied by high levels of secondary hyperparathyroidism (Gha-

zali 1999). There are several reasons for this a) The patient should have a specific catered diet, but this diet may incude the reduction of the intake of vitamin D foods (milk, fish, cream, butter, etc.). b) The endogenous synthesis of vitamin D3 decreases in individuals over 60 years, due to increased melanin and reduced contact with sunlight in the skin (Godar 2012, Holick 1987), c) Urinary path 25 (OH) D3 and vitamin D binding protein loss is high (Saha 1994). d) The decrease of glomerular filtration rate (GFR)(Kawashima 1995, Thadhani 2012).

Hemodialysis treatment does not provide a change for vitamin A levels. B-carotene, ubiquinol, and laykopen levels were lower in patients that didn't have renal failure. The intake of dietary vitamin A should not exceed the RDA in HD patients of 800-1000 mg/ day(Koople 2004).

Increased oxidative stress and cardiovascular risks are associated with hemodialysis patients. The antioxidant properties of vitamin E may be useful in preventing or reducing these risks. HD patients are recommended 400 IU/Daily intake of vitamin E (Galli 2004, Mann 2004, Kopple 2004).

Protein and phosphorus restriction, loss of appetite, and vitamin D deficiencies increase the need of calcium in HD patients. Support of calcium and control of serum phosphorus levels, by using calcium-containing phosphate-binding agents, are balanced simultaneously (Miller 2010). Calcium acetate or calcium carbonate are effective with reducing concentration of serum phosphorus, simultaneously, correcting hypocalcemia and negative calcium balance (Isakova 2009, Miller 2010). However, the use of vitamin D and calcium in hemodialysis patients concluded the risk for severe hypercalcemia and renal osteodystrophy (Tilman 2009). Increasing calcium during treatment should be done carefully. As a result, to ensure the positive balance of calcium levels in dialysis patients, 1000-1500 mg of calcium should be taken daily.

Lack of phosphorus excretion in the human body can be closely related to the glomerular filtration rate. Even if a single nephron loses its function, it may result in the accumulation of phosphorus in the plasma while showing an inability to the discharging of phosphorus (Kopple 2004). When GFR decreased 120 mL / min to 25 mL / min, the accumulation of phosphorus was observed very clearly in the plasma. In hemodialysis patients, the level of serum phosphorus 2.5-4.5 mg / dL, and patients who have a glomerular filtration rate (GFR) between 25 mL/min/1.73 m2 and 70 mL/min/1.73 m2, 8 mg/kg/d to 10 mg/kg/d of phosphorus may be given with the 0.55 g/kg/d to 0.60 g/kg/d of protein. High biological value protein sources including essential amino acids are rich foods from phosphorus, therefore there are difficulties on the limitation of phosphorus. For this reason, the absorption of phosphorus is prevented with the phosphorus binding agents from the outside. Egg white is a rich source of high biological value protein have one of the lowest phosphorus-protein ratios and is also deprived from cholesterol; therefore, it is a particularly healthy food source of protein for patients on dialysis (Noori 2010). Whole eggs instead of egg whites, whole bread instead of white bread, dried beans instead of peas and preferably fish (cod, tuna) that have a low phosphorus / protein ratio, should be consumed to reduce dietary phosphorus intakes in dialysis patients (Cupisti 2003). The active form of vitamin D is added in the treatment, this is an important step in the control of serum parathyroid hormone activity (Steiber 20109). About 80% absorption of dietary phosphorus from the gastrointestinal tract requires the use of phosphorus-binding agents (Locatelli 2002, Guarneri 2003, Noori 2010, Noori 2010). Niacin working with a different mechanism than phosphate binders, is helpful to lower phosphate levels while causing a decrease transport of phosphate without interfering with the sodium-phosphate pump in the GI lumen (Mahan 2012, Cheng 2006).

Patients with kidney disease are more difficult to assess whether there is sufficient amount of trace elements in the body. Iron (Fe), calcium and zinc deficiencies are demonstrated in dialysis patients. Frequently anemia is shown in dialysis patients due to an iron deficiency (Tarng 1999, Vinay 2009). Because the amount of iron absorbed in the intestine is decreased, severe blood loss can be a symptom. In addition, the formation of erythropoietin decreases due to bone marrow suppression by urea (Mahan 2012). Adding iron is recommended after assessing the patient's serum ferritin and iron levels (Rambod 2008). Intravenous iron therapy can be applied to patients for the treatment of anemia. With this treatment, hemoglobin was shown to be removable at 5-7 g / dL to 10 g / dL. Due to the fact that erythropoietin therapy increases usage of iron, it is recommended for patients to take iron supplementation.

Uremic symptoms, such as anorexia, impaired taste sensation, reduced oxidative stress improved immune function and sexual dysfunction are associated with Zn deficiency in HD patients. CRP is a sensitive marker of inflammatory activity; an association between decreased plasma Zn concentrations with higher CRP levels in hemodialysis patients has been noted (Guo 2010). Concentrations of serum Zn may affected from medications used by hemodialysis patients such as calcium carbonate, calcitriol (Dashti-Khavidaki 2010), aluminum phosphate-binders. For these reasons, Zn, Fe, magnesium (Mg) are needed, respectively, 15 mg / day, 10-18 mg / day, 200-300 mg / daily in dialysis patients (Fouque 2003). In addition, good sources of zinc are meat, poultry, nuts, and lentils and fortified breakfast cereals (Rucker 2010).

Mild selenium deficiency also appears to increase susceptibility to oxidant stress (Klotz 2003, Rayman 2002), which may be especially relevant to HD patients in whom oxidative stress is markedly increased (Stenvinkel 2003) and may contribute to accelerated atherosclerosis. Selenium deficiency may contribute to the risk of infection (Field 2002) and perhaps to uremic cardiomyopathy, thus contributing to the increased risk of CVD in the HD population. The selenium content of grains and seeds is variable, and depends on the selenium content of the soil and the form in which selenium is present (Rucker 2010). Selenium is also present in some meats, seafood, and nuts (particularly brazil nuts); levels in these foods may again be influenced by ambient soil levels (Rayman 2000). Some studies demonstrated oxidative stress and atherosclerosis is associated with selenium deficiency, because of its link to infection and uremic cardiomyopathy. Selenium deficiency increases risk of cardiovascular disease in HD patients(Fujishima 2011).

Recommended dietary nutrient intake for hemodialysis patients are shown below in Table 1 (Nissesson 2008, Rucker 2010, Fouque 2007).

Macronutrients and Fiber	
Dietary protein intake (DPI)	• 1.2 g/kg/d for clinically stable patients
	(at least 50% should be of high biological value)
Daily energy intake (DEI)	• 35 kcal/kg/d if <60 years
	• 30–35 kcal/kg/d if 60 years or older
Total fat	25–35% of total energy intake
Saturated fat	<7% of total energy intake
Polyunsaturated fatty acids	Up to 10% of total calories
Monounsaturated fatty acids	Up to 20% of total calories
Carbohydrate	Rest of calories (complex carbohydrates preferred)
Total fiber	"/>20–25 g/d
Minerals and Water (Range of Intake)	
Sodium	750–2000 mg/d
Potassium	2000-2750 mg/d
Phosphorus	800-1000 mg/d
Calcium	<1000 mg/d
Magnesium	200–300 mg/d
Iron	10-18 mg/d
Zinc	15 mg/d
Selenyum	55 μq/d
Water	Usually 750–1500 mL/d
Vitamins (Including Dietary Suppleme	nts)
Vitamin B1 (thiamin)	1.1–1.2 mg/d
Vitamin B2 (riboflavin)	1.1–1.3 mg/d
Pantothenic acid	5 mg/d
Biotin	30 µg/d
Niacin	14–16 mg/d
Vitamin B6 (pyridoxine)	10 mg/d
Vitamin B12	2.4 μg/d
Vitamin C	75–90 mg/d
Folic Acid	1–5 mg/d
Vitamin A	800-1000 μg/d
Vitamin D	1000-1500 IU
Vitamin E	400-800 IU

 Table 1. Recommended Dietary Nutrient Intake for Hemodialysis Patients

9. Conclusion

To evaluate the amount of food intake and food preference, the patient's diet history should be taken. The patient's age, gender, social environment, economic, psychological, and educational status and history of the disease should be considered due to nutrition effect. Also, including weekends, during the 3-7 days whole foods is recorded by the patient along with the amount. Daily intake of calories and nutrients of the patients are calculated with information from those records. In addition, laboratory values and SGA as a scoring tool are very important for preparing a appropriate diet for HD patients.

The hemodialysis therapy should be dealt with by a multidisciplinary team, as recommended for other high risk populations (Morais 2005). A part of medical nutrition therapy is to provide nutrition education and periodic counseling by dietitians. For effective intervention, dietitians should present a guide for educating HD patients about individual nutritional needs. This guide should provide information about food sources, nutrients and usage exchange food lists. Adapting to patients requirements of intakes should be based on their laboratory values. Patients may be predisposed to receiving lower than recommended amounts of energy and macro-nutrients to the diet and patients who received information or counseling about their diet must be followed up closely by renal dietitians (Mahan 2012).

If a patient has diabetes, the control of blood sugar is required with a specialized diet therapy. Due to high serum glucose levels, osmolality increases, water and potassium are pulled out of cells. There are the relationship between glycemic control and survival of hemodialysis patients (Mahan 2012). Poor glycemic control causes to macrovascular complications and generation of advanced glycation end products (AGEs)(Ricks 2012). The diet for diabetes management can be modified for a patient on dialysis.

Recently, dialysis treatment is increasing in elderly patients with end-stage renal disease (ESRD) (Tamura 2009). Elderly hemodialysis patients have some diseases such as ischemic heart disease, diabetes mellitus, infectious diseases, bone fracture, cerebrovascular disease in common with ESRD. Specific prescriptions should prepare for elderly dialysis patients such as longer treatment time, nutritional support, and a personalized treatment schedule(Burns 2003). In addition, tube feeding and parenteral interventions may reinforce protein and energy intake among patients with malnutrition and anorexia.

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References

- [1] Mahan K, Escott-Stump S, Raymond JL (2012) Krause's Food and the Nutrition Care Process, 13. Edition, Elsevier.
- [2] Stark S, Snetselaar L, Hall B, Stone RA, Kim S, Piraino B, Sevick M A (2011) Nutritional Intake in Adult Hemodialysis Patients, Top Clin Nutr, Vol. 26, No. 1, 45-56.
- [3] D'Haese PC, De Broe ME (1996) Adequacy of dialysis: trace elements in dialysis fluids. Nephrol Dial Transplant 11(Suppl. 2):92–97.
- [4] Fouque D, Guebre-Egziabher F (2007) An update on nutrition in chronic kidney disease, Int Urol Nephrol, 39:239-246.
- [5] Fouque D, Guebre-Egziabher F, Laville M (2003) Advances in anabolic interventions for malnourished dialysis patients. J Renal Nutr 13:161–165.
- [6] Ohkawa S, Kaizu Y, Odamaki M, Ikegaya N, Hibi I, Miyaji K, Kumagai H: (2004) Optimum dietary protein requirement in nondiabetic maintenance hemodialysis patients. Am J Kidney Dis, 43:454-463.
- [7] Coombes JS, Fasset RG (2012) Antioxidant therapy in hemodialysis patients: a systematic review, Kidney International (2012) 81, 233–246.
- [8] Vaziri ND, Moradi H (2006) Mechanisms of dyslipidemia of chronic renal failure. Hemodial Int,; 10: 1–7.
- [9] Inamoto H, Kata M, Suzuki K (2003) Deficiency of Vitamins and Minerals in the Dialysis Diet: The State of 33 Essential Nutrients. Nephrology Dialysis Transplantation. 18:448.
- [10] Mutsert R, Grootendorst DC, Boeschoten EW, Brandts H, Manen JG, Krediet RT, Dekker FW (2009) Subjective global assessment of nutritional status is strongly associated with mortality in chronic dialysis patients, Am J Clin Nutr 2009;89:787–93.
- [11] Laville M, FouqueD (2000) Nutritional aspects in hemodialysis, Kidney International, Vol. 58, Suppl. 76, pp. S-133-S-139.
- [12] Steiber A, Kalantar-Zadeh K, Secker D, McCarthy M, Sehgal A (2004) Subjective Global Assessment in Chronic Kidney Disease: A Review, Journal of Renal Nutrition, Vol 14, No 4 (October): pp 191-200.
- [13] K/DOQI (2000) National Kidney Foundation: Clinical practice guidelines for nutrition in chronic renal failure. Am J Kidney Dis(suppl 2)35:S1-140,
- [14] Dwyer JT, Larive B, Leung J, Rocco MV, TOM Greene T, Burrowes J, Chertow GM, Cockram DB, Chumlea WC, Daugirdas J, Frydrych A, Kusek JW forthe hemo study group (2005) Are nutritional status indicators associated with mortality in the hemodialysis (HEMO) study?. Kidney nt Vol68: 1766-1776.

- [15] Stenvinkel P, Heimbürger O, Lindholm B, Kaysen, GA, Bergström J (2000) Are There Two Types of Malnutrition in Chronic Renal Failure Evidence for Relationships Between Malnutrition, Inflammation and Atherosclerosis (MIA Syndrome). Nephrol Dial Transplant. 15:953-960.
- [16] Baltzan MA, Shoker AS (1998) Malnutrition and Dialysis, Kidney Int.:53:999.
- [17] Chazot C, Laurent G, Charra B, Blanc C, Vovan C, Jean G, Vanel T, Terrat J C, Ruffet M (2001) Malnutrition in Long Term Hemodialysis Survivors. Nephrol Dial Transplant. 16:61-69.
- [18] Basile C (2003) The effect of convection on the nutritional status of haemodialysis patients, Nephrol Dial Transplant,18 [Suppl 7]: vii46–vii49.
- [19] Burrowes JD, Larive B, Cockram DB, Dwyer J, Kusek JW, McLeroy S, Poole D, Rocco MV, (2003) For the HEMO Study Group. Effects of dietary intake, appetite, and eating habits on dialysis and nondialysis treatment days in hemodialysis patients: cross-sectional results from the HEMO Study. J Ren Nutr.;13(3):191-198.
- [20] Rao M, Sharma M, Juneja R, Jacob S, Jacob CK (2000)Calculated nitrogen balance in hemodialysis patients: Influence of protein intake, Kidney International, Vol. 58, pp. 336–345.
- [21] O'Keefe A, Daigle NW(2002) A new approach to classifying malnutrition in the hemodialysis patient, J Ren Nutr.;12:248-55.
- [22] Morais AC, Silva MA, Faintuch J, Vidigal E J, Costa RA, Lyrio DC, Trindade CR, Pitanga KK (2005) Correlation of nutritional status and food intake in hemodialysis patients, Clinics, 60(3):185-92
- [23] Nakao T, Matsumoto H, Okada T, Kanazawa Y, Yoshino M, Nagaoka Y, Takeguchi F (2003) Nutritional management of dialysis patients: balancing among nutrient intake, dialysis dose, and nutritional status. Am JKidney Dis.: 41(3 Suppl 1):S133-6.
- [24] Beddhu S, Kaysen GA, Yan G, Sarnak M, Agodoa L, Ornt D, Cheung AK (2002) Association of serum albümin and atherosclerosis in chronic hemodialysis patients. Am J Kidney Dis: 40(4):721-7.
- [25] Panichi V., Rizza, M.G., Taccola, D., Paoletti, S., Mantuano, E., Migliori, M., Frangioni, S., Filippi, C., Carpi, A. (2006) C reactive protein in patients on chronic hemodialysis with different techniques and different membranes. Biomed Pharmacother; 60(1):14-7.
- [26] Santos NCJ, Draibe SA, Kaimmura MA, Canziani MEF, Cendoroglo M, Júnior AG, Cuppari L (2003)Is serum albumin a marker of nutritional status in hemodialysis patients without evidence of inflammation. Artificial Organs 27(8):681-686.
- [27] Mittman N, Morell MA, Kyin KO, ChattapadhyayJ (2001) Serum prealbumin predicts survival in hemodialysis and peritoneal dialysis: 10 years of prospective observation. Am J Kidney Dis;38(6): 1358-64.

- [28] Sathishbabu M, Suresh S (2012) A study on correlation of serum prealbumin with other biochemical parameters of malnutrition in hemodialysis patient, Int J Biol Med Res; 3(1): 1410-1412.
- [29] Kaysen GA, Rathore V, Shearer GC, Depner TA (1995) Mechanisms of hypoalbuminemia in hemodialysis patients. Kidney Int 1995; 48: 510-16.
- [30] Owen WF, Lowrie EG(1998)C-reactive protein as an outcome predictor for maintanence hemodialysis patients. Kidney Int; 54:627-636.
- [31] Pupim LB, İkizler A.(2004) Assessment and monitoring of uremic malnutrition. Journal Of Renal Nutrition, 14,6-19.
- [32] Janardhan V, Soundararajan P, Vanitha Rani N, Kannan G, Thennarasu P, Ann Chacko R, Maheswara Reddy CU (2011) Prediction of Malnutrition Using Modified Subjective Global Assessment-dialysis Malnutrition Score in Patients on Hemodialysis, Indian J Pharm Sci., Jan-Feb; 73(1): 38–45.
- [33] Kalantar-Zadeh K, Ikizler TA, Block G, Morrel M, Kopple JD (2003) Malnutrition-inflammation complex syndrome in dialysis patients:causes and consequences. American Journal Of Kidney Disease, 42(5), 864-881.
- [34] Fouque D, Vennegoor M, Wee PT, Wanner C, Basci A, Canaud B, Haage P, Konner K, Kooman J, Martin-Malo A, Pedrini L, Pizzarelli F, Tattersall J, Tordoir J, Raymond Vanholder R (2007) EBPG guideline on nutrition. Nephrol Dial Transplant;22(2):ii45–
- [35] Kalantar-Zadeh K, Abbott KC, Salahudeen AK, Kilpatrick RD, Harwich TB (2005)Survival advantages of obesity in dialysis patients. Am J Clin Nutr:81:543-54.
- [36] Glanton CW, Hypolite O, Hshien PB, Agodoa LY, Yuan CM, Abott K(2003) Factors associated with improved short term survival in obeses end stage renal disease patients. Ann Epidemiol. 2003; 13: 136-143.
- [37] Guida B, Trio R, Nastasi A, Lacetti R, Pesola D, Torneca S, Memoli B, Cianciaruso B (2004) Body composition and cardiovascular risk factors in pretransplant hemodialysis patients. Clin Nutr 23:363-72.
- [38] Rocco MV, Paranandı L, Burrowes JD (2002) Nutritional Status in the HEMO Study Cohort at Baseline Hemodialysis. Am JKidney Dis. 39:245-256.
- [39] Ikizler TA, Pupim LB, John R. Brouillette JR, Levenhagen DK, Farmer K, Hakim RM, Flakoll PJ (2002) Hemodialysis stimulates muscle and whole body protein loss and alters substrate oxidation. AmJ Physiol Endocrinol Metab; 282: E107-116.
- [40] Pupim LB, Flakoll PJ, Brouillette JR, Levenhagen DK, Hakim RM, Ikizler TA(2002) Intradialytic parenteral nutrition improves protein and energy homeostasis in chronic hemodialysis patients. J Clin Invest;110: 483-492.
- [41] Kopple JD, Massery CG(2004) Nutritional management of renal disease. Second edition, wiliams&wilkins, Lippincott.

- [42] Raj DS, Adeniyi O, Dominic EA, Boivin MA, McClelland S, Tzamaloukas AH, Morgan N, Gonzales L, Wolfe R, Ferrando A (2007) Amino acid repletion does not decrease muscle protein catabolism during hemodialysis, Am J Physiol Endocrinol Metab 292: E1534–1542.
- [43] Kopple JD(2001) The national kidney foundation k/doqı clinical practice guidelines for dietary protein ıntake for chronic dialysis pateints. Am. J. of Kidney Disease ,38 (4) Supplement 1, S68-S73.
- [44] Canoa N, Fiaccadorib E, Tesinskyc P, Toigod G, Drumle W, DGEM: Kuhlmann M, Mann H, Horl WH(2006). ESPEN guidelines on enteral nutrition: adult renal failure. Clinical Nutrition,25,295-310.
- [45] Fouque D (2003) Nutritional requirements in maintenance hemodialysis. Advances İn Renal Replacement Therapy, 10(3),183-193.
- [46] Stenvinkel P, Lindholm B, Heimbürger M (2000) Elevated serum levels of soluble adhesion molecules predict death in predialysis patients:association with malnutrition, inflamation and cardiovascular disease, Nephr. Dialysis Transpl.,15,1624-1630.
- [47] Mehrotra R, Kopple JD (2001) Nutritional management of maintance dialysis patients. why aren't we going better?, Annual Review of Nutrition,21, 343-379.
- [48] Karalis M (2002) Ways to increase protein intake, J.Renal Nutrition, 13(3), 199-204.
- [49] Cano N, Fouque D, Leverve X (2006) Application of Branched- Chain Amino Acids in Human Patological States: Renal Failure. J. Nutr, 136, 299-307.
- [50] Raj D, Ouwendyk M, Francoeur R and Pierratos A. (2000) Plasma amino acid profile on nocturnal hemodialysis. Blood Purif, 18, 97–102.
- [51] Bernstein A, Treyzon L, Li Z (2007) Are high protein, vegetable-based diets safe for kid ney function? A review of the literature. Journal American Dietetic Association, 107,644-650.
- [52] Locatelli F, Vecchio LD, Pozzoni P (2005) Clinical benefits of slowing the progression of renal failure, Kidney International , 68(99), S152- S156.
- [53] Lim VS, Kopple JD(2000) Protein metabolism in patients with chronic renal failure.role of uremia and dialysis. Kıdney International, 58, 1-10.
- [54] Gribotto G, Bonanni A, Verzola D (2012) Effect of kidney failure and hemodialysis on protein and amino acid metabolism, Curr Opin Clin Nutr Metab Car, 15:78–84.
- [55] Foss MC, Gouveia LM, Neto MM, Paccola GM, Piccinato CE (1996) Effect of Hemodialysis on Peripheral Glucose Metabolism of Patients with Chronic Renal Failure. Nephron. 73:48-53.
- [56] Rigalleau V, Combe C, Blanchetier V, Aubertin J, Aparicio M, Gin H (1997) Low protein diet in uremia: Effects on glucose metabolism and energy production rate, Kidney Int., 51:1222-27.

- [57] Feig DI, Kang DH, Johnson RJ (2008) Uric acid and cardiovascular risk, N Engl J Med; 359: 1811–1821.
- [58] Brymora A, Flisiniski M, Johnson RJ, Goszka G, Stefaniska A, Manitius J (2012) Lowfructose diet lowers blood pressure and inflammation in patients with chronic kidney disease, Nephrol Dial Transplant, 27: 608–612.
- [59] Jalal DI, Smits G, Johnson RJ, Chonchol M (2010) Increased fructose associates with elevated blood pressure. J Am Soc Nephrol; 21: 1543-1549.
- [60] Liu Y, Coresh J, Eustace JA, Longenecker J, Jaar B, Fink N, Tracy R, Powe NR, Klag MJ (2004) Association Between Cholesterol Level and Mortality in Dialysis Patients. JAMA. 291:451-459.
- [61] Rutkowski B, Szolkiewicz M, Korczynska J, Sucajtys E, Stelmanska E, Niewoglowski T, Swierczynski J (2003) The Role of Lipogenesis in the Development of Uremic Hyperlipidemia. Am J Kidney Dis. 41:84-88.
- [62] Lindley EJ (2009) Reducing sodium intake in hemodialysis patients. Semin Dialysis, May-Jun;22(3):260-3.
- [63] Cianciaruso B, Brunori G, Kopple JD, Traverso G, Panarello G, Enia G, Strippoli P, Vecchi A, Querques M, Viglino G, Vonesh E, Maiorca R (1995) Crosssectional comparisons of malnutrition in continuous ambulatory peritoneal dialysis and hemodialysis patients. Am Journal of Kidney Disease, 26, 475-86.
- [64] Lindholm B, Wang T, Heimburger O and Bergstrom J (1998) Influence of different treatments and schedules on the factors conditioning the nutritional status in dialysis patients. Nephrol Dial Transplant, 13 [Suppl 6], 66–73.
- [65] Vergili-Nelsen JM (2003) Benefits of fish oil supplementation for hemodialysis patients. J Am Diet Assoc;103: 1174-1177.
- [66] Noori N, Dukkipati R, Kovesdy CP, Sim JJ, Feroze U, Murali SB, Bross R, Benner D, Kopple JD, Kalantar-Zadeh K (2011) Dietary omega-3 fatty acid, ratio of omega-6 to omega-3 Intake, inflammation, and survival in longterm hemodialysis patients. Am J Kidney Dis.;58(2):248-256.
- [67] Bouzidi N, Mekki K, Boukaddoum A, Dida N, Kaddous A, Bouchenak M(2010) Effects of omega-3 polyunsaturated fatty-acid supplementation on redox status in chronic renal failure patients with dyslipidemia. J Ren Nutr.;20(5):321–328.
- [68] Skulas-Ray AC, West SG, Davidson MH, Kris-Etherton PM (2008)Omega-3 fatty acid concentrates in the treatment of moderate hypertriglyceridemia. Expert Opin Pharmacother.;9(7):1237-1248.
- [69] Saifullah A, Watkins BA, Saha C, Li Y, Moe SM, Friedman AN(2007)Oral fish oil supplementation raises blood omega-3 levels and lowers C-reactive protein in haemodialysis patients – a pilot study. Nephrol Dial Transplant.;22(12):3561–3567.

- [70] Daud ZA, Tubie B, Adams J, Quainton T, Osia R, Tubie S, Kaur D, Khosla P, Sheyman M(2012) Effects of protein and omega-3 supplementation, provided during regular dialysis sessions, on nutritional and inflammatory indices in hemodialysis patients, Vascular Health and Risk Management,8: 187–195.
- [71] Kaveh K, Kimmel PL(2001)Compliance in hemodialysis patients: multidimensional measures in search of a gold standard. Am J Kidney Dis;37:244–66.
- [72] Noori N, Kalantar-Zadeh K, Kovesdy CP, Murali SB, Bross R, Nissenson AR, Kopple JD, (2010) Dietary Potassium Intake and Mortality in Long-Term Hemodialysis Patients, Am J Kidney Dis., 56(2): 338–347.
- [73] Aviva Fattal-Valevski (2011)Thiamine (Vitamin B1), Journal of Evidence-Based Complementary & Alternative Medicine, 16: 12.
- [74] Ghazali A,Fardellone P, Pruna A, Atık A, Achard JM, Oprisiu R, Brazier M, Remond A, Moriniere P, Garabedian M, Eastwood J, Fournier A (1999) Is low plasma 25-(OH) vitamin D a major risk factor for hyperparathyroidism and Looser's zones independent of calsitirol? Kidney Int, 55:2169-2177.
- [75] Cheng S, Coyne D(2007)Vitamin D and outcomes in chronic kidney disease. Curr Opin Nephrol Hypertens;16:77–82.
- [76] Holick MF(1987)Photosynthesis of vitamin D in the skin: effect of environmental and life-style variables. Fed Proc., 46(5):1876–1882.
- [77] Godar DE, Pope SJ, Grant WB, Holick MF (2012) Solar UV doses of young americans and vitamin D₃ production, Environ Health Perspect 120:139–143.
- [78] Saha H(1994)Calcium and vitamin D homeostasis in patients with heavy proteinuria, Clin Nephrol, 41:290–96
- [79] Kawashima H, Kraut JA, Kurokawa K(1995) Metabolic acidosis suppresses 25-hydroxyvitamin D3-1a-hydroxylase in the rat kidney. J Clin Invest, 70:135–140.
- [80] Thadhani R, Appelbaum E, Pritchett Y, Chang Y, Wenger J, Tamez H, Bhan I, Agarwal R, Zoccali C, Wanner C, Lloyd-Jones D, Cannata J,Thompson T, Andress D, Zhang W, Packham D, Singh B, Zehnder D, Shah A, Pachika A, Manning WJ, Solomon SD (2012) Vitamin D therapy and cardiac structure and function in patients with chronic kidney disease, JAMA, February 15,Vol 307, No.7.
- [81] Rucker D, Thadhani R, Tonelli M (2010)Trace element status in hemodialysis patients, Seminars in Dialysis, 23,4:389–395,
- [82] Cheng S, Coyne DW(2006) Niacin and niacinamide for hyperphosphatemia in patients undergoing dialysis, Int.Urol.Nephrol.,38,171.
- [83] Mak RH, Ikizler AT, Kovesdy CP, Raj DS, Stenvinkel P, Kalantar-Zadeh K (2011)Wasting in chronic kidney disease, J Cachexia Sarcopenia Muscle, 2:9–25.

- [84] Bergstrom J(1993). Nutritional requirements of hemodialysis patients. Mitch W, Klahr S (Ed.). Nutrition and Kidney, U.S.A: Little, Brown and Company, 2nd ed.: 263-293.
- [85] Kwan BC, Kronenberg F, Beddhu S, Cheung AK(2007) Lipoprotein metabolism and lipid management in chronic kidney disease. J Am Soc Nephrol; 18: 1246–1261.
- [86] Lacquaniti A, Bolignano D, Donato V, Bono C, Fazio RM, Buemi M(2010) Alterations of Lipid Metabolism in Chronic Nephropathies: Mechanisms, Diagnosis and Treatment, Kidney Blood Press Res., 33:100-110.
- [87] Matera M, Bellinghieri G, Costantino G, Santoro D, Calvani M, Savica V(2003)History of L-carnitine: implications for renal disease. J Ren Nutr, 13:2-14.
- [88] Flanagan JL, Simmons PA, Vehige J, Willco MDP, Garrett Q (2010) Rol of carnitine in disease, Nutrition & Metabolism, 7:30.
- [89] Naini AE, Sadeghi M, Mortazavi M, Moghadası M, Harandi AA (2012) Oral Carnitine supplementation for Dyslipidemia in Chronic Hemodialysis Patients, Saudi J Kidney Transp., 23(3):484-498.
- [90] Smith K, Coston M, Glock K, BS1, Elasy TA, Wallston KA, PhD4, Ikizler TA, Cavanaugh KL, (2010) Patient Perspectives on Fluid Management in Chronic Hemodialysis, Ren Nutr.; 20(5): 334-341.
- [91] Dumm GN, Giammona A (2003) Variations in the lipid profile of patients with chronic renal failure treated with pyridoxine. Lipids in Health and Disease, 2,1-7.
- [92] Coveney N, Polkinghorne KR, Linehan L, Corradini A, Kerr PG (2011)Water-soluble vitamin levels in extended hours hemodialysis, Hemodialysis International, 15(1): 30-38.
- [93] Wierzbicki AS(2007)Homocysteine and cardiovascular disease: a review of the evidence. Diab Vasc Dis Res.;4:143-50.
- [94] Steiber AL, Kopple J(2011) Vitamin Status and Needs for People with Stages 3-5 Chronic Kidney Disease, J Renal Nutr, 21(5):355-368.
- [95] Galli F, Buoncristiani U, Conte C, et al: (2004)Vitamin E in uremia and dialysis patients. Ann N YAcad Sci 1031:348-351,
- [96] Mann JFE, Lonn EM, Y1 Q, Gerstein HC, Hoogwerf B J, Pogue J, Bosch J, Dagenais GR, Yusuf S (2004)Effects of vitamin E on cardiovascular outcomes in people with mild-to-moderate renal insufficiency: results of the HOPE study. Kidney Int 65: 1375-1380.
- [97] Miller JE, Kovesdy CP, Norris KC, Mehrotra R, Nissenson AR, Kopple JD, Kalantar-Zadeh K (2010) Association of Cumulatively Low or High Serum Calcium Levels with Mortality in Long-Term Hemodialysis Patients, Am J Nephrol; 32:403–413.

- [98] Isakova T, Gutie' rrez OM Chang Y, Shah A, Tamez H, Smith K, Thadhani R, Wolf M (2009) Phosphorus Binders and Survival on Hemodialysis, J Am Soc Nephrol 20: 388–396.
- [99] Drueke TB, Touam M (2009) Calcium balance in haemodialysis—do not lower the dialysate calcium concentration too much(con part), Nephrol Dial Transplant, 24:2990—93
- [100] Locatelli F, Fouque D, Heimburger O, Drüeke TB, Canata- Andia JB, Hörl W, Ritz W (2002) Nutritional status in dialysis patients: a european concensus. Nephrology Dialysis Trasplantation, 17, 563-572.
- [101] Guarneri R, Antonione G (2003) Mechanisms of malnutrition in uremia. Journal of Renal Nutrition, 13(2), 153-157.
- [102] Noori N, Kalantar-Zadeh K, Kovesdy CP, Bross R, Benner D, Kopple JD (2010) Association of Dietary Phosphorus Intake and Phosphorus to Protein Ratio with Mortality in Hemodialysis Patients, Clin J Am Soc Nephrol 5: 683–692.
- [103] Noori N, Sims JJ, Kopple JD, Shah A, Colman S, Shinaberger CS, Bross R, Mehrotra R, Kovesdy CP, Kalantar-Zadeh K (2010) Organic and inorganic dietary phosphorus and its management in chronic kidney disease, Iranian Journal of Kidney Diseases, 4:2
- [104] Cupisti A, Morelli E, Alessandro C, Lupetti S and Barsotti G. (2003) Phosphate control in chronic üremia: Don't forget diet. J Nephrol, 16, 29-33.
- [105] Moyad MA, Combs MA, Crowley DC, Baisley JE, Sharma P, Vrablic AS, Evans M(2009) Vitamin C with metabolites reduce oxalate levels compared to ascorbic acid: a preliminary and novel clinical urologic finding. Urol Nurs 29:95-102,
- [106] Handelman GJ (2011) New insight on vitaminc in patients with chronic kidney disease, JRenal Nutr,21(1):110-112.
- [107] Locatelli F, Andrulli S, Memoli B, Maffei C, Vecchio CD, Aterini S, Simone WD, Mandalari A, Brunori G, Amato M, Cianciaruso B, Zoccali C (2006) Nutritional-inflammation status and resistance to erythropotein therapy in haemodialysis patients, Nephrol Dial Transplant, 21:991.
- [108] Tarng DC, Huang TP, ChenTW, Yang WC (1999)Erythropoietin hyporesponsiveness: from iron deficiency to iron overload. Kidney Int.; 55(suppl 69):S107-118.
- [109] Deved V, Poyah P, James MT, Tonelli M, Mann BJ, Walsh M, Hemmelgarn BR (2009) Ascorbic acid for anemia management in hemodialysis patients: a systematic review and meta-analysis, Am J Kidney Dis 54:1089-1097.
- [110] Rambod M, Kovesdy CP, Kamyar Kalantar-Zadeh K (2008) Combined high serum ferritin and low iron saturation in hemodialysis patients: the role of inflammation, Clin J Am Soc Nephrol 3: 1691–1701.

- [111] Fujishima Y, Ohsawa M, Itai K, Kato K, Tanno K, Turin TC, Onoda T, Endo S, Okayama A, Fujioka T (2011) Serum selenium levels are inversely associated with death risk among hemodialysis patients, Nephrol Dial.Transp.,26:10,3331-38.
- [112] Stennett AK, Ofsthun NJ, Kotanko P, Gotch FA (2010) Kinetic modeling as a route to rational dialysis prescriptions—urea, phosphorus, calcium, and more, US Nephrology, 5(2):18–20.
- [113] Klotz LO, Kroncke KD, Buchczyk DP, Sies H(2003)Role of copper, zinc, selenium and tellurium in the cellular defense against oxidative and nitrosative stress. J Nutr 133:1448S-1451S.
- [114] Rayman MP(2002)The argument for increasing selenium intake, Proc Nutr. Soc, 61:203–215.
- [115] Stenvinkel P(2003) Interactions between inflammation, oxidative stress, and endothelial dysfunction in end-stage renal disease. J Ren Nutr, 13:144–148.
- [116] Field CJ, Johnson IR, Schley PD(2002) Nutrients and their role in host resistance to infection. J Leukoc Biol 71:16-32.
- [117] Saran R, Bragg-Gresham JL, Rayner HC, Goodkin DA, Keen M, Van Dijk PC, Kurokawa K, Piera L, Saito A, Fukuhara S, Young EW, Held PJ, Port FK (2003) Nonadherence in hemodialysis: associations with mortality, hospitalization, and practice patterns in the DOPPS, KidneyInternational, vol. 64, no. 1, pp. 254–262,.
- [118] Hegel MT, Ayllon T, Thiel G, Oulton B (1992) Improving adherence to fluid restrictions in male hemodialysis patients: a comparison of cognitive and behavioral approaches, Health Psychology, vol. 11, no. 5, pp. 324–330.
- [119] Joni Ricks, Miklos Z. Molnar, Csaba P. Kovesdy, Anuja Shah, Allen R. Nissenson, Mark Williams, Kamyar Kalantar-Zadeh (2012) Glycemic Control and Cardiovascular Mortality in Hemodialysis Patients With Diabetes, A 6-Year Cohort Study, Diabetes March, 61:3, 708-715.
- [120] Burns A(2003) Conservative management of end-stage renal failure: Masterly inactivity or benign neglect? Nephron Clin Pract 95: c37–c39.
- [121] Tamura MK, Covinsky KE, Chertow GM, Yaffe K, Landefeld CS, McCulloch CE (2009) Functional status of elderly adults before and after initiation of dialysis, N Eng J Med, 361;16.

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