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Diet in Renal Diseases: An Art of Science

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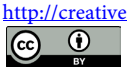

Abstract

Purpose of Review: Chronic kidney disease (CKD) is associated with a limited ability to excrete fluids, electrolytes, uremic toxins and other end-products of catabolism. Studies on adverse renal outcomes with dietary patterns are limited. **Methods**: Comprehensive search in PubMed of papers published until June 2024 describing prospective cohort studies on renal nutritional therapy (RNT) with at least 3 years of follow up. **Results:** RNT should include adequate yet limited amounts of calories, fluids, protein, lipids, sodium, potassium, and phosphorus. RNT is an adjuvant to specific drug-therapy in 1) certain complications viz. fluid overload, anemia and renal osteodystrophy, and 2) specific kidney diseases viz. glomerulopathies, tubulopathies, polycystic kidney disease, calcium oxalates urolithiasis and cystinuria, as well as 3) types of renal failure viz acute and chronic and its treatment viz. hemodialysis, peritoneal and transplantation. **Conclusion**: RNT is patient-specific and should be systematically planned to delay the progression of CKD as well as to prevent and treat its complications.

Keywords

Calories, Diet, Disease, Electrolytes, Fluid, Kidney, Lipids, Nutrition, Protein, Vitamins

1. Introduction

Dietary restrictions are essential for patients with (a) specific malnutrition disorders viz. genetic and acquired disorders in obesity as well as absorption defects in carbohydrates, amino acids, fat metals, protein and metals, (b) obesity, cardiovascular diseases, hypertension, stroke, type 2 diabetes, metabolic syndrome, and (c) multi- or specific organ disease [1]. Patients with chronic kidney disease (CKD) have limited excretory function and hence require specific dietary restrictions [2]. It is a vital organ for (a) glomerular excretion of metabolic (ure-

mic) toxins, (b) intravascular volume homeostasis via tubular reabsorption of electrolytes and water as well as the renin angiotensin aldosterone system, (c) maintenance of stable blood osmolality via antidiuretic hormone action, (d) metabolic adjustment of acid-base imbalance, (e) stability of serum electrolytes via tubular reabsorption/secretion of sodium, potassium, magnesium, calcium, and (f) manufacturing of vital hormones viz. erythropoietin and active form of vitamin D. Therefore, we conducted this review evaluate renal nutritional therapy (RNT) in those patients and its associations with CRD complications in an attempt to delay CRD-progression and improve patient's survival and quality of life.

2. Methods

We performed a comprehensive search in PubMed of papers published until June 2024 describing prospective cohort studies on RNT with at least 3 years of follow up without date or language restriction. The 2 investigators manually searched the reference lists of eligible studies, clinical practice guidelines, reviews, and other relevant studies. Methodological queries regarding study inclusion were discussed among two investigators to achieve consensus.

3. Results

Electronic searching retrieved multiple studies yet 45 cohort studies fulfilled the criteria of inclusion. Specific dietary adjustments in patients with CRD are summarized in Table 1.

3.1. Fluid Balance

In most patients with acute renal failure, CKD and end-stage ones (on dialysis program), fluid retention is a major complication resulting in intravascular volume expansion that can culminate into severe refractory hypertension, pulmonary oedema and death. Hence, fluid restriction is essential, with allowance calculated as "urine + 500 ml". The 500 ml covers the loss of fluids through the skin and lungs. This life-saving rule applies to most patients with CKD viz. acute and chronic glomerulopathy, advanced tubulointerstitial/vascular diseases, and obstruction. Exceptions entail patients with dehydration, non-oliguric acute tubular necrosis (ATN), recovering polyuric phase of ATN, and chronic reflux nephropathy. Moreover, certain kidney diseases such as calcium oxalate urolithiasis, cystinuria, autosomal dominant polycystic kidneys, and recurrent urinary tract infections indicate high fluid intake with 2 liters/day to cleanse their urinary tract from stones and bacteria [3] [4]. Finally, the myth of "kidney protection with excessive water intake" should be discouraged since (a) intact thirst center dictates water urges after sensing increased serum osmolality, and (b) extravagant polydipsia leads to undue day/night polyuria and may generate disease-phobia. Such a "false myth" was confirmed in a randomized controlled study [5]. Exceptions of the latter are those with impaired thirst center *i.e.* the

Table 1. Dietary restrictions in healthy subjects and patients with chronic renal disease (CKD).

Nutritional items	Normal subjects requirements	CKD (stage 4)		
		Requirements	Food items	
			Permissible	Exclude
Fluids	At thirst	At thirst	Urine + 500 ml	
Energy (kcal/kg/day)	35	23		
Protein (g/kg/day)	0.8	0.6	Fish + limited poultry and diary products	Red meat
Saturated fat	<30% of calories	<30% of calories	Corn, sunflower, olive oils	Red meat, butter, margarine, baked and fried food
Cholesterol (mg/day)	200	200		
Sodium (mg/day)	<2300	<2000	Unprocessed food	Table salt, canned and frozen food especially if if oedema, fluid overload and hypertension
Potassium (mg/day)	4000	<2500	Grapes, vegetables, rice, eggs and tuna	Fruits, juice, dates, concentrated tomato (especially if oliguric)
Calcium (mg/day)	1000	2000	May need Ca & activated Vit D supplement	If calcium oxalate urolithiasis
Phosphorus (mg/kg/day)	800	800	limited dairy products	Red meat
Magnesium (mg/day)	400	<200		Mag-antacids & laxatives/enemas

elderly and those with mental disabilities [6].

3.2. Protein

Dietary proteins are normally assembled to build and maintain muscle, bone, skin, connective tissue, internal organs, and blood. Moreover, their ingredients are essential to fight disease and heal wounds. In contrast to carbohydrates and lipids, excess proteins are not stored. They are degraded to form (a) urea and other nitrogenous waste, (b) inorganic ions (Phosphorus (P), Hydrogen, potassium (K), sodium (Na), and sulphates), which are primarily excreted by the kidney. Hence, in those with reduced kidney-reserve; excessive protein-intake leads to glomerular hyperfiltration followed by secondary glomerulosclerosis and progressive kidney loss. Such a phenomenon is evident in complementary bodybuilders [7]. Hence, the aims of RNT should be to (a) ensure sufficient dietary protein intake to avoid body protein degradation (principally skeletal muscles), (b) limit the accumulation of nitrogenous waste products and inorganic ions characteristic of uremia that leads to anorexia and subsequent malnutrition (morbidity & mortality), and (c) slow the rate of progression of CKD. In healthy adults, the safe level of protein intake is 0.8 g/kg daily. If reduced, adaptive response: more effective utilization of dietary essential amino acids as seen in

MDRD study without changes in anthropometric measurements. Hence, in patients with nephrotic syndrome 0.8 g of protein daily and in those with CRD 0.6 is adequate [8]. Higher intake is not indicated, even in proteinuric patients, since the liver compensates for that and liver production will be lowered if intake is high. Exceptions for that are those with excessive loss viz. nephrotic patients with proteinuria > 5 g/day which indicates an increase of protein intake by 1 g/day protein for each 1 g urinary protein excretion [9]. If less (starved), wasting develops. Such essential nutrients are available in chicken breast, fish and plant proteins [10]. Nearly 10% of dialysis patients are severely malnourished and 33% are at moderate stage. Factors that increase protein requirements are (a) anorexia due to inadequate intake due to uremia, co-morbidities, medications, depression, (b) superimposed illnesses (e.g. sepsis), (c) increased requirements due to protein loss in dialysate or catabolism induced by hemodialysis, especially with bioincompatible membranes. Hence, dialysis patients should consume 1.2 g/kg daily [11].

3.3. Lipids

Fats are an essential energy source and for the maintenance of healthy microvasculature. However, patients with CKD are at higher risk of atherosclerosis viz. cerebrovascular disease, ischemic heart disease and peripheral arterial disease [12] [13]. The latter was attributed to enhanced vascular atherosclerosis, uremic toxins, abnormal lipid modifications, vascular calcifications, oxidative stress and endothelial dysfunction [14]. Moreover, secondary hyperlipidemia is common in CKD, with prevalence at 20% - 70% [2]. The non-nephrotic patients have (a) high VLDL, decreased HDL-cholesterol and (b) high triglycerides due to defective catabolism. In nephrotic ones, TG and LDL-cholesterol are increased [15]. Moreover, elevated levels of TG in the blood might also result from enhanced hepatic lipogenesis and VLDL secretion in response to high glucose concentrations, especially in patients on peritoneal dialysis since standard PD solutions contain high levels of glucose [16]. To limit atherosclerosis, <30% of calories should be from fat, mainly from saturated fat and low cholesterol [17]. HMG-CoA reductase inhibitors should be used and fibrate should be avoided for increased risk of myopathy. Hence, in patients with CKD, the amount should be limited as well as restricted only to monounsaturated and polyunsaturated fats viz. canola oil, nuts, oatmeal, olive oil, salmon, and sesame oil. The saturated and trans fats can raise serum cholesterol levels and clog blood vessels. Saturated fats, are solid at room temperature and are high in animal products such as red meat, poultry, and butter. On the other hand; the trans ones are often found in baked goods and fried foods, as well as in hydrogenated vegetable oils such as margarine and vegetable shortening.

3.4. Calories (Energy)

In both predialysis and dialysis patients, energy expenditure (and hence energy

requirements) are similar to healthy individuals *i.e.* 35 kcal/kg daily to avoid body protein wasting. In the elderly and obese, lower energy intake can be cautiously prescribed 30 kcal/kg daily [17]. If less, you can add glucose polymers (polycose) to beverages, high-density oral supplements (Nepro, Suplena) and candy bars (Regain). In general, diets rich in calories (due to increased fat and/or carbohydrates) are deleterious for the kidneys. Diets with high fat content have been reported to cause renal damage in non-obese animals that did not develop type 2 diabetes, suggesting a direct effect of high fat/high calorie intake on the kidney [18]. Such phenomenon has been shown to be related to (a) podocyte injury secondary to inflammasome activation [19], (b) down-regulation of the sirtuin type 1 (Sirt 1)–adiponectin axis [20], and (c) altered expression of cytoskeleton genes [21]. In addition to promoting direct kidney damage, high caloric intake also affects renal disease indirectly (through the development of obesity and type 2 diabetes).

3.5. Sodium

Na is an essential element for blood pressure homeostasis yet excessive amounts are associated with; (a) hypertension, (b) fluid overload and pulmonary oedema, as well as (c) excessive oedema in nephrotic states (d) progressive secondary glomerulosclerosis, and increased cardiovascular risk [22] [23]. The U.S. Food and Drug Administration recommends that adults limit their Na intake to no more than 2300 mg per day [24]. Table salt, some seasonings, and certain sauces such as soy sauce and teriyaki are obvious sources of high Na. However, occult sources include processed foods viz. canned, frozen and snack ones (chips and crackers). The amount of Na in foods and beverages can be found on the product's Nutrition Facts label. Hence, low-Na diet entails (a) eating fresh, frozen (without sauce or seasoning), or canned (low Na or no salt added) fruits and vegetables, (b) unprocessed meats instead of processed meats, (c) cooking from scratch, (d) using spices, herbs, and salt-free seasonings instead of salt, and (e) using alternative seasonings, such as lemon juice and hot pepper sauce. Moreover, salt substitutes should be avoided since Na is replaced with something that is really excreted and has cardiotoxic accumulation [25].

3.6. Potassium

K is an intracellular anion. Its normal level is controlled by glomerular filtration and subsequent tubular reabsorption in the distal tubule. The 2015 American Dietary Guidelines Advisory Committee report recommends a daily K-intake of 121 mmol per day and in the event of poor K-intake, the kidney can lower K-excretion to about 5 to 25 milliequivalents, thus maintaining a baseline serum K-level of 3.5 mmol/L [26]. Unfortunately, K-imbalance is rarely symptomatic and lethal tachyarrhythmias and myocardial paralysis may be the only manifestations of severe hypo and hyperstates [27]. HypoK is caused by; (a) gastrointestinal losses via vomiting or diarrhea, and (b) renal losses via uncontrolled diabe-

tes or excessive thiazides/loop diuretic-use. On the other hand; hyperK is associated with (a) physiological catabolism and (b) excessive cell damage viz. sepsis, hemolysis and rhabdomyolysis and tumor necrosis with/without chemotherapy. Glomerular filtration is the main excretory route. Its level may be normal or even low in chronic tubulointerstitial disease (chronic reflux nephropathy), type 1 - 3 renal tubular acidosis, Bartter's syndrome and Gitelman's. Hence, the risk of hyperK is high in glomerulopathy and oliguric renal diseases viz. dehydration, advanced tubulointerstitial diseases and obstructive uropathy the risk of hyperkalemia. Moreover, even with GFR > 20%, life-threatening hyperK can be encountered in (a) with K-retaining drugs viz. Spironolactone, Beta-blockers and ACEI/ARB, (b) type IV RTA including diabetics, and (c) primary and secondary adrenal diseases. Hence, low-K diet is essential for such patients with potential for hyperK. High-K foods include fruits, juice, vegetables, dates, tomatoes, potatoes, brown rice, bran cereals, whole-wheat bread, pasta, beans and nuts. Low-K foods include apples, peaches, grape, carrots, white rice. In addition to diet and avoidance of hyperkalemic drugs, ancillary medications for hyperK include (a) increasing fecal K^+ excretion by the cation-exchanging resin sodium polystyrene sulfonate for levels 5 - 6 mmol/L and with the healthy gut as well as IV furosemide if fluid overload, (b) glucose-short acting insulin infusions and beta2 agonists for levels at 6 - 7 mmol/L, and IV calcium gluconate and/or dialysis if cardiac arrythmias or wide QRS-complexes [28].

3.7. Phosphorus

Failing kidneys are unable to filter even the normal P product of catabolism. The resultant hyperP can induce (a) muscle weakness, (b) secondary hyperparathyroidism leading to brittle bones, and (c) progressive vascular calcification leading and ischemic vasculopathy [29]. A diet limited to $\leq$1000 mg of P per day is indicated and includes avoidance of meat, poultry, fish, cereals, dairy and processed products, flavored drinks, beans and nuts [30]. However, such a diet limits essential food elements and is rarely sufficient to control hyperP. Hence, P-binders are the corner stone in its management, with a preference for the polyallylamine Sevelamer hydrochloride (Renagel) or carbonate (Renvela) over Calcium or Aluminum P-binders to avoid worsening of visceral/vascular calcifications due to increasing P-Ca product in the former and Aluminum accumulation in brain/bone marrow in the later. However, severe nausea and decreased appetite are associated with their use, and with azotemia, patients were declared uremic and were placed prematurely on renal replacement therapy.

3.8. Calcium

Calcium (Ca) is an essential electrolyte for the functioning of muscles as well as the circulatory and digestive system, and is essential for bone formation and blood cell synthesis [31]. Ca-balance is mainly regulated by (a) Ca absorption in the intestine aided by vitamin D3 and (b) exchange in the bones. Nevertheless,

falsely high values may be obtained in (a) patients with liver or renal failure, (b) in hyperlipidemia and hemolysis, (c) upright posture, (d) Venous occlusion of the arm during venipuncture, (e) recent meal. Hence, it is better to be done at an early morning fast, in a sitting position and without the application of a venous tourniquet. Moreover, since nearly 90% of the protein-bound Ca is bound to albumin; assessment of physiologically active serum Ca indicates measurement of ionic one or its corrected form to serum albumin. CRD is associated with poor absorption of gut-Ca due to a lack of activation of 1-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol (vitD3). Poor mineralization of bones leads to renal rickets. Moreover, retention of P due to as well as the low serum Ca leads to activation of parathyroid glands (secondary hyperparathyroidism) to "push" P through tubules yet its side effect is parathyroids-leaching of bone Ca with resultant hyperparathyroid bone disease (Osteitis fibrosa cystica) [32]. The resulting high Ca-P product leads to extraosseous and vascular calcifications, adding to the increase in cardiovascular morbidity and mortality associated with CRD [33]. Hence, maintaining a balanced serum Ca level with Ca supplementation, active forms of vitamin D, calcimimetics and P-binders is essential in the management of CRD. The recommended daily intake of dietary Ca is 800 to 1000 mg/day with an increased dose of active vitamin D analogs to suppress secondary hyperparathyroidism. However, suppression of parathyroid glands, with correction of hyperP, hypoCa, calcimimetics and active forms of vitamin, should not exceed 9 times to avoid adynamic bone disease. Moreover, Ca-supplementation should not be used solely as P-binders to avoid high Ca-P products and vascular calcifications [34]. In CRD-patients with uncontrolled hyperP, hypoCa and low 1,25 vit D, progressive hyperplasia of the parathyroid glands culminates into adenomatous changes with severe hyperCa (tertiary hyperparathyroidism) that indicates excision of the adenomatous gland [35]. In patients with urolithiasis; Ca and different forms of vitamin D should be restricted to avoid further stone formation [3].

3.9. Magnesium

Magnesium (Mg) is a major intracellular cation. It is a co-factor of many enzyme systems, including adenosine triphosphate-dependent ones. Approximately 70% of Mg ions are stored in bone. The serum Mg level is kept constant within very narrow limits. Regulation takes place in the kidney's ascending loop of Henle. HypoMg can be seen in (a) hereditary tubular defect of Getileman syndrome, (b) secondary to acute and chronic gastrointestinal loss with vomiting and diarrhea, (c) inappropriate or overzealous use of diuretics, laxatives, (d) kidney loss in recovery polyuric phase of ATN, and (e) chronic use of Tacrolimus in transplant patients. Moreover, in malnourished and diabetic patients especially after hydration and refeeding (refeeding syndrome). In the latter, multiple essential electrolytes are needed viz. K, P and Mg as well as all vitamin B especially B1 [36]. On the other hand, hyperMg is rarely seen except after inappropriate Mg therapy.

3.10. Uric Acid

It is the end-product of purine catabolism. In the kidney, it is freely filtered, with a net 90% reabsorbed in the proximal tubule by different transporters [37]. Hyperuricemia is common in certain kidney diseases viz. analgesic nephropathy and following treatment with diuretics, Cyclosporin A, antineoplastics, and tumor lysis [38]. Xanthine oxidase inhibitors (Allopurinol and Febuxostat) are indicated in the treatment of significant hyperuricemia (>600 umol/L) with/without gout and urate urolithiasis. On the other hand, animal studies and clinical trials, failed to confirm the benefit of such drugs for mild/moderate asymptomatic hyperuricemia to avoid crystal-dependent or -independent chronic renal injury [39]. Moreover, severe hypersensitivity syndromes were reported following those drugs as well as increased cardiovascular events following their discontinuation that was attributed to rebound activation of renin angiotensin aldosterone system [40].

3.11. Fast-Foods

Quick-service restaurants provide quick, easy, and inexpensive meals. To balance fast food items, (a) broiled, steamed and grilled items are preferred over deep fat-fried ones, (b) removal of skin and avoidance of extra crispy is essential to lower fats and also Na from seasonings of coating, (c) avoid salty fries with high calories and Na, (d) eating chicken breast without the salty dressing, and (e) avoid tomatoes, hot sauce, juices for their high content of Na and K.

3.12. Trace Elements

Trace elements are essential micronutrients required in amounts ranging from 50 micrograms to 18 milligrams per day. They include iron, magnesium, zinc, selenium, copper, iodine, and manganese. The kidney is the principal route of their excretion; supplementation is not recommended and excessive exposure is associated with toxicity.

1) To avoid hyperMg, Mg-containing laxatives and antacids should be avoided in patients with CRD since the kidney is responsible for Mg-excretion.

2) Postmortem studies in dialysis patients indicated (a) high-levels of nickel and chromium due to contaminated dialysate, (b) low red-cells bromide in chronic ambulatory peritoneal dialysis patients due to dialysate loss, and (c) low plasma yet high tissue zinc due to dialysis [41]. Unfortunately, and in CKD patients, zinc-supplementation are still being promoted as a measure to increase B lymphocytes counts and granulocytes motility as well as to improve taste and sexual dysfunction.

3.13. Vitamins

Most people get enough vitamins and minerals to stay healthy by eating various foods each day, Vitamins are co-factors in multiple enzymatic activities. In patients with CKD, water-soluble vitamins are essential due to (a) decreased intes-

tinal absorption, (b) impaired enzymatic activity and the presence of circulating inhibitors, and (c) dialysate losses. Folic acid and vitamin C are removed by conventional hemodialysis and should be supplemented in those patients. Vitamin B12 is not removed by conventional hemodialysis yet removed by high-flux membranes. Hence, it should be supplemented in high-flux dialysis and hemofiltration/hemodiafiltration. Patients with CKD are unable to activate 25 vit D to 1,25 vit D, which is 100 times more potent than 25 vit D and hence has limited absorption of calcium and subsequent (a) symptomatic hypoCa, (b) further activation of parathyroid glands in addition to hyperP, and (b) decrease mineralization of bone leading to renal rickets. However, iatrogenic hyperCa should be avoided for its (a) detrimental effect on kidney function in predialytic patients, (b) exacerbation of urolithiasis, (c) tissue and vascular calcifications, and (d) gastrointestinal and neurological side-effects. However, supplementations with other fat-soluble vitamins (A, E and K) should be avoided since they are poorly removed by failing kidneys and hemodialysis with resultant toxicity especially with vitamin A. Moreover, vitamin E supplementation is not indicated in CRD patients since its level is usually normal and has not been related to erythrocyte survival. Hence, routine multivitamin supplementation is not necessary in CKD and should individualized [42].

3.14. Acute Renal Failure

If ARF is self-limited and catabolism is modest, 0.6 g of protein/kg daily is adequate. However, due to dialysate losses, patients supported with peritoneal and hemodialysis dialysis may require 1.2 g/kg daily while continuous hemofiltration/hemodiafiltration needs up to 2 g/kg daily. In the absence of hypermetabolic states viz. sepsis, trauma, and burns, the energy requirements are similar to normal subjects (35 kcal/kg daily). However, adjustment for hypermetabolic states has been suggested with an increase of 1.2 to 1.6 X basal energy expenditure. In an oliguric state, fluids should be limited to 500 ml+ losses with restrictions of Na and K to 2 g/kg daily. Oral and tube-feeding are the preferred routes with products designed for those with CRD to promote mucosal integrity. Total parenteral nutrition, through a central vein and using conventional amino acid solutions, is typically used yet with potential risk of complications (electrolytes imbalance, acid-base disturbances and catheter sepsis.

3.15. Anemia

Anemia is prevalent in 2/3 of patients with CKD and is associated with cognitive impairment, sleep disturbances, CKD progression, cardiovascular comorbidities, and higher mortality [43]. It is inevitable due to decreased erythropoietin production. An exception is in those with autosomal dominant polycystic kidney disease since erythropoietin production is unrestricted from the increased peritubular cells encircling the enlarging cysts. Prior to correction of anemia with erythropoietin stimulating agent (ESA), (a) 2 hematological elements

should be adequate viz. iron and vitamin B12 and (b) optimal level of correction. In adults, iron deficiency is rarely nutritional. It is due to gastrointestinal leaks and/or menorrhagia in females as well as filter/tubes losses during initiation and closure of hemodialysis. On the other side vitamin B12 absorption requires (a) separation from proteins by gastric hydrochloric acid, (b) combination with an intrinsic factor from gastric parietal cells to form an absorbable complex in the terminal ileum and (c) intact ileum. Hence, it is associated with (a) autoimmune deficiency of intrinsic factor which is prevalent in 20% of patients, (b) achlorhydria due to pernicious anemia, (c) drugs that lower hydrochloric acid e.g. proton pump inhibitors or those that lower release of intrinsic factor e.g. metformin. Hence, correction of anemia entails (a) confirmation of adequate stores of iron and VB12. Testing for iron stores should be done by measurement of transferrin saturation% and not ferritin which is increase with inflammatory conditions (infections and autoimmune diseases). (b) Correcting leaking defects to avoid future losses. In patients with CKD, significant anemia that persists despite adequate VB12 and iron stores, indicates parenteral erythropoietin-use. Treatment of significant anemia, to improve oxygen carrying capacity, is to hemoglobin at 110 - 120 g/L. Overzealous use of erythropoietin that raised hemoglobin above those levels was associated with multiple cardiovascular thrombotic events in this patient population with premature and progressive atherosclerosis [44] [45]. The last word is on folic acid supplementation which is rarely encountered in patients with CKD unless (a) strict vegetarians, and (b) on maintenance hemodialysis with loss during treatment sessions.

4. Discussion

The global prevalence of CKD is increasing rapidly and is currently estimated to be 9.1% [46]. In USA, they are a leading cause of death. Nearly 37 million US adults have it, and 40% have severe disease and are unaware of it and hence undiagnosed. Due to hyperfiltration in the remaining glomeruli, CKD tends to get worse over time with 360 patients starting maintenance dialysis every 24 hours. Diabetes and high blood pressure are the leading causes of CKD, accounting for 3 out of 4 new cases. Moreover, in 2019, the cost of Medicare treatment of CKD was $87.2 billion and that for dialysis patients was an additional $37.3 billion [47]. A good diet should provide limited yet adequate amounts of (a) calories (energy) for daily tasks, (b) proteins to avoid loss of muscle-mass and prevent infections yet without inducing hyperfiltration/hypertrophy that increases glomerulosclerosis and subsequently kidney loss, (c) fluid and certain salts that are normally filtered by the glomerulus to limit fluid overload and electrolyte/s intoxication, and (e) fats in a disease with multiple risk factors for atherosclerosis.

5. Conclusion

RNT is patient-specific and its systematic implementation, with adjuvant drug-therapy, can prolong patients and kidney survival as well as improve their

quality of life.

Author's Contributions

Prof/Kamel El-Reshaid conceived of the scientific work, participated in its design and drafted the manuscript. Dr. Shaikha Al-Bader participated in the study design and data collection.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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