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NUTRITIONAL REGIMEN OF NEW THERAPEUTIC STRATEGIES IN CHRONIC RENAL FAILURE

D. Paskalev, V. Ikonomov[1], H. Sapundziev[1]

Clinic of Hemodialysis and [1]Clinic of Nephrology, Prof. Paraskev Stoyanov Medical University of Varna

We are what we are because of the kind of the kidneys we have.
Homer Smith

ABSTRACT

Nowadays the number of the patients with chronic renal failure and the syndrome of end-stage renal disease permanently increases. New dietary regimens are necessary to improve the status of the patients with severe kidney diseases. Nutrition plays an essential role in the contemporary approach to the management of the patients with renal failure. Low-protein diet delays the progression of the renal failure. The meta-analysis of the effect of dietary protein restriction on the course of diabetic and non-diabetic renal diseases outlines the importance of the adequate correction of the uremic syndrome and the absence of side effects of this nutritional regimen. The development of appropriate diet therapy to be administered at the predialytic stage of the disease could prevent or at least delay the progression of chronic renal failure and, in this way, it represents a challenge for the nephrologists and hope for the patients.

Key words: dietary therapy, protein restriction, chronic renal failure, end-stage renal disease, glomerular filtration rate

Contemporary approach to the management and the new dietary therapy of kidney diseases with chronic renal failure (CRF) defined a standpoint of modern nephrology.

Circumstances will inevitably dictate the strategy of dietary regimen in a patient with CRF who could be critically ill, or sitting in an outpatient consulting room. Obviously it is essential to confirm the stage of renal failure, with particular emphasis on reversible factors, which require immediate by specific therapeutic complication by the best form of replacement therapy.

Spontaneous slowing of progression is also seen in only 10 to 12% of cases of patients with CRF whose progression has apparently stopped are unlikely, to consent to a change of supplements in low protein diet. The trial the Modification of Diet in Renal Disease (MDRD) study (1994) evaluated the beneficial effect of dietary protein restriction in patients with renal diseases of diverse etiology.

The objective of these studies was to determine whether a low-protein diet retards the progression of renal failure. MDRD study indicated that patients with moderate renal failure (GFR at 25-55ml/min; study A) faster decline in mean GFR during the first 4 months after randomization but a slower mean decline thereafter in patients who were prescribed a low-protein diet (0,58g/kg/day) compared to a usual protein diet (1,3g/kg/day), but no significant difference in the level of GFR at 3 years of follow-up. In patients with more advanced renal insufficiency (GFR levels at 13-24ml/min; study B) there was a slower mean decline in GFR (3,6 ml/min/year). MDRD study revealed a longer time to renal failure in patients with a GFR less than 25ml/min/1,73m^2, it is indicated to prescribe dietary protein intakes of 0,6g/kg/day (Table 1).

Many of human controlled studies have not shown an unequivocal benefit by strong evidence, including the lack of randomization, the use of retrospective analysis, that criticized the concentrations of serum creataine or creatinine clearance to assess the changes of decline of renal function. In the results of the intention-to-tread analysis did not prove a beneficial effect of the very-low-protein diet (0,28g protein/kg/day) compared to the low-protein diet (0,58 protein g/kg/day) in the progression of renal disease (no significant effect on the time to renal failure or death). Secondary analyses of the MDRD study (2,3) revealed a longer time to renal failure in group B in patients with lower total protein intake but did not use the keto-amino acid supplement with GFR less than 25ml/min.

In cases of advanced GFR values less than 25ml/min a diet providing 0,60g protein/kg body weight/day may reduce the rate of loss of renal function or delay the early onset of the uremic syndrome of end-stage renal disease (ESRD). An adequate intake of calories must also be insured at rise of catabolizing body protein if the caloric intake is inade-

Address for correspondence:
H. Sapundziev, Clin. of Nephrology, Prof. P. Stoyanov Medical University of Varna, 55 Marin Drinov St, BG-9002 Varna, BULGARIA
E-mail: officeub@mail.bg

Table 1. Scheme for protein restriction in dependence on renal function

Stage of CRF	Daily permitted protein supply	Restriction of phosphate	Daily permitted calcium supply	Hyperlipidemia diet + statine
Initial CRF (CrCl 50-70ml/min)	0,2-1,2 g/kg bw/d	<900 mg/d	500-1200 mg/d	aimed LDL <100 mg/dl
Mild CRF (Cr Cl 25-50 ml/min)	0,6-0,8 g/kg bw/d	700-900 mg/d	1000-1200 mg/d	total chol. <160 mg/dl
Advanced CRF	Protein balanced diet	<700 mg/d	1200-1500 mg/d	
(Cr Cl 5-25 ml/min)	<0,6 g/kg bw/d ca 45g	Ca carbonate	1200-1800 mg/d	total chol. <210 mg/dl
Hemodialysis (CrCl <5 ml/min)	1,2-1,4 g/kg bw/d normal diet under HD	700-900 mg/dl		

quate on periodic assessment of dietary adequacy and compliance (by measuring the 24h urinary excretion of urea).

The meta-analysis of effect of dietary protein restriction on the progression of diabetic and non-diabetic renal diseases were considered also its importance in adequate correction of uremic syndrome and an absence of side effects in the long or short term of this nutritional regimen. Thus can virtually arrest the downward course of CRF in predialytic patients for at least one year (9).

The use of low-protein diets and lipid-lowering regimen for individual patients to a greater or lesser extent when some factors determine a Kt/v in dialysis patients result a lower than that prescribed. There is a direct relationship between food intake during hemodialysis and measurement of optional value of standard kt/v (has to be 1,2 for non-diabetic patients and 1,4 for diabetics, and Urea Reduction Percentage/PRU) of 65-70% (NKF – DOQI Guideline limits) in the determination of dialysis adequacy.

Adequate hemodialysis achieves a high survival rate, a low morbidity and a better standard of living. Intradialysis food intake influences the result of dialysis that the effective circulating volume diseases. A week after, it to be calculate again in low-protein regimen of food intake (6).

Dietary phosphorus restriction should begin when the glomerular filtration rate is a 60-70 ml/min and can be achieved by restricted dietary phosphorus intake. Phosphorus is contained in almost all foods and it is difficult to limit the intake of dietary phosphorus to less than 900-1000mg per day. That 60-70% of consumed phosphate was absorbed from low-phosphate diet in inadequate dialysis, when safe phosphate-binding therapy.

Involved mechanisms of renal tabular reabsorption of organic phosphate are under the control of dietary. Endocrine and metabolic factors, but not fully clarified in terms of homeostasis and pathophysiology. Vitamin D therapy used to manage secondary hyperparathyroidism may increase intestinal absorption of both calcium and phosphate. Excess total body calcium may not be reflected in serum calcium levels for patients with renal failure and most patients are in positive calcium balance.

The excessive calcium intake that oral or i.v. calcitriol can be used safely only when calcium and phosphorus balances are controlled. Approximately 50% prevalence of hyperphosphatemia (>1,6mmol/L) and cutoff point for "poor control" was defined >2,1 mmol/L which was present in 39% of dialysis population. Elevated Ca x P product was associated with a higher risk of mortality statistically significant above 5,8 $mmol^2/L^2$. Poor phosphorus control significantly increased the risk for cardiovascular death (7). Some authors recommended target levels of less than 1,8mmol/L for phosphorus and less than 4,4$mmol^2/l.^2$ for Ca x P product in permanent nutritional status. Controlling the Ca x P product through dietary phosphorus restriction and phosphorus-binding agents is the most common means of preventing metastatic calcifications into tissues.

The dietary therapy in CRF patients is usually recommended on a conventional low-protein (0,6g/kg b. w. per day) diet.

A very important step in the clinical management in the predialytic stage of chronic uremic patients with (creatinine clearance less than 25ml/min/1,73m^2 serum creatinine level below 350mmol/L) has been the assessment of nutritional status a very-low-protein diet (0,3g/kg b. w. per day) supplemented with essential amino acids and keto acids as a factor correlating with a progression of CRF. Nowadays there are numerous studies about the effect of using mixtures of essential amino acids (Propac, Promod, Magnacal). Progressive protein-energy malnutrition is a heavy complication in patients on periodical hemodialysis treatment in 30% of them and in all with marked anaemia. On periodical hemodialysis treatment oral intake of nutritional supplements shows the possibility to treat protein-energy malnutrition. The protein intake to be increased to the preferential norm of 1,2-1,4g/kg b. w. /day by Nutridial (Nutrim Co), a Bulgarian nutritional supplement with protein concentration of 62%. This is significantly cheaper compared to same treatment with other foreign analogue (9).

Keto acid supplementation (ketoanalogue given in the form of Ketosteril®, Fresenius, Germany, or Clintec®, France) slows progression of renal failure more than amino acids with a diet containing 0,3g per kg ideal weight of protein and 7-9mg per kg ideal weight of phosphorus Also added with vitamins, calcium carbonate, or 10g per day of essential amino acids. There has been no change in blood pressure, phosphaturia and proteinuria, but the nutrition has been maintained. Keto acid supplemented regimen halted

the progression of moderately-severe CRF for at least an year (herein after serum creatinine levels at changeover of 6,6 – 7,4mg/dl) (10).

A low Na^+ of 20-40mmol/day diet may reduce salt retention mainly in nephrotic syndrome) and the danger of diuretic therapy. In patients with heavy nephritic syndrome (proteinuria >5g/day), hypoproteinuria until the serum albumin below 30g/l, with azothemia (>150mmol/L creatinine) and an increase at over 1,5 times in total cholesterol with increased production of very low density lipoprotein (VLDL) and low density lipoprotein (LDL) In diabetic patients combined pancreatic and kidney transplantation before heavy accelerated macro- and microvasculopathic complications and before muscle wasting precludes rehabilitation affords to retain functional status, thereby avoiding a risk of posttransplantation mortality.

In protein-energy malnutrition of patients on hemodialysis there was no correlation between decreased TNF-α, transferrin or albumin levels and reduced anthropometric parameters in the high prevalence of malnutrition in CRF children, which becomes more pronounced when treatment by hemodialysis is initiated (1).

For haemodalysis patients with diabetic nephropathy the diet does appear to affect the response to epoietin. Differences in mean survival time were significant between those with cardiovascular disease and without cardiovascular disease (5). Treatment of renal anaemia with epoetin reduces oxidative stress as the concomitant administration of vitamin C and vitamin E (15mg/kg/daily) may improve this therapeutic effect (10).

The low-protein diet decreases the risk of highly prevalent comorbid risk factors through progressive CRF that plague the long-duration restricting life quality optimal food intake, especially in atherosclerotic heart disease and cerebrovascular catastrophes that induce a high rate of withdrawal from food (as a form of suicide). Compliance with prescribed amino acids and keto analogues was assessed by questioning the patients by a physician and dietician.

The number of patients with ESRD has increased over the past 10 years and continues to grow at a rate of 7-9% per year in the United States and with prevalence rate of a 400 persons per million in Europe that the financial cost of caring for them is enormous, particularly in face of economic health state aided resources. Therefore, the development of dietary therapy at predialytic stage to prevent, or at least to slow into years the progression of CRF, greatly contisuted to the development a new medical strategy: challenge to nephrologists and hopes for patients with CRF.

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