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CONTEMPORARY VIEWS ON THE ROLE OF NUTRITION IN CHRONIC RENAL FAILURE – A REVIEW

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The pathological process in the kidneys in the course of chronic renal failure (CRF) leads to multiple metabolic disturbances in the whole body. Disturbances of protein, carbohydrate, fat, electrolyte and calcium-phosphate balance are the main reasons for protein-energy malnutrition in patients. The use of a proper, individually adapted diet may slow down the progression of the disease and reduce the intensity of the symptoms.

INTRODUCTION

Chronic renal failure (CRF) is a complex disease which develops as a result of chronic, progressive and irreversible glazing of the renal glomeruli and fibrosis of the renal interstitium. The disease process results in a gradual decrease in the number of functioning nephrons, which leads to increasing homeostatic, endocrine and excretory disturbances of kidney function.

The CRF clinical representation is a result of the accumulation of the end-products of protein and purine metabolism (creatinine, urea, uric acid, guanidine-acetic acid, hipuric acid and its metabolites), parathormone (PTH), inorganic phosphates and the so-called "medium molecules" and other toxic substances, the serum levels of which increase [Zdrojewski & Rutkowski, 2004].

A significant role in CRF pathogenesis is played by disturbances of kidney endocrine activity. These are manifested as decreased synthesis of erythropoietin, dihydroxycholecalciferol ($1,25(OH)_2D_3$), kinines and natriuretic factor (urodilatine) in the kidneys, while elimination and biodegradation of endocrine secretion products, such as the growth hormone, insulin and cortisol, is diminished.

Also the influence of metabolic acidosis, overhydration, electrolyte disturbances and malnutrition should not be omitted [Zdrojewski & Rutkowski, 2004].

It has recently been posited that in the clinical representation of CRF a significant role may be played by accumulation of advanced glycation end-products (AGEs) and glycated β_2-microglobulin, a basic component of the amyloid deposits in patients being haemodialysed for a long time.

Four stages of CRF development are most often distinguished depending on the degree of the clinical and biochemical symptoms and disturbances [Myśliwiec *et al.*, 2001]: (1) latent stage: characterised by a decrease in the glomerular filtration rate (GFR) to about 50% and the impairment of urine condensation (isostenuria), with minimal or no clinical symptoms; (2) compensated stage – with minor clinical symptoms, GFR drops to 25% of adequate values, evident disorders of water-electrolyte balance (polyuria, nycturia, intensive thirst), disorders of calcium-phosphate metabolism (diminished excretion of phosphates and synthesis of active vitamin D_3) and in some patients, vascularhypertension and anaemia; (3) decompensated stage with the presence of prominent clinical and biochemical symptoms and signs such as severe anaemia, neuropathy, uremic cardiomiopathy, osteodystrophy and vascular hypertension, with GFR decrease below 25%; and (4) fully symptomatic uremic stage – end stage renal disease (ESRD) – the patient requires renal replacement therapy or renal transplantation to survive.

In 2002 the National Kidney Foundation (NKF) implemented a new division of CRF (called chronic kidney disease) into 5 stages: (1) with normal or increased GFR (>90 mL/min/1.73 m^2); (2) with minor GFR decrease (60–89 mL/min/1.73 m^2); (3) with moderate decrease of GFR (30–59 mL/min/1.73 m^2); (4) with severe decrease in GFR (15–29 mL/min/1.73 m^2); and (5) kidney failure with GFR decrease below 15 mL/min/1.73 m^2 or if there is the necessity of dialysis.

Chronic kidney disease is a significant epidemiological issue, especially if we consider that it is most often diagnosed in the population of patients suffering from diabetes and arterial hypertension, which is constantly increasing.

In the year 2003 in Poland chronic renal replacement therapy was applied to 11,440 patients with CRF, 90.4% of whom (10,343 patients) were treated by maintenance haemodialysis (mHD) [Puka *et al.*, 2004].

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It is well known that in the course of mHD, the symptoms and signs of malnutrition can be detected in 20–76% of patients, although use of the term "malnutrition" for these patients is controversial [Myśliwiec *et al.*, 2001].

PATHOGENESIS OF MALNUTRITION IN CRF

Many causes of malnutrition in CRF can be distinguished. Diminished intake of nutrients and loss of nutrients into dialysate, increased catabolism of body proteins, hormonal disturbances, metabolic acidosis, and increased protein and energy demand, especially in the presence of concurrent diseases, are among the main causes of this type of malnutrition.

Individual features of a patient, such as age, physical activity, concomitant diseases and cytokine coding genes expression, subsequently modify these abnormalities.

Anorexia in CRF is caused mainly by accumulation of uremic toxins. It is also presumed that a significant role in pathogenesis of uremic anorexia is played by serotonin overproduction, caused by an increase in tryptophane concentration in the serum and brain. Increased concentrations of cholecystokinine, tumour necrosis factor alpha (TNF-α), interleukin-1 (IL-1), leptin and deficiency of neuropeptide Y also contribute to the development of malnutrition [Aparicio *et al.*, 2002]. A hypothesis that the increase of serum leptin concentration is caused by the inflammatory state was developed but subsequent studies did not confirm that [Don *et al.*, 2001].

Changes of leptin concentration among patients treated with HD are associated mainly with lipid turnover disturbances. The concentration of serum leptin is closely associated with concentration of serum insulin, total cholesterol, LDL cholesterol and triglycerides, but no relationship between concentration of leptin and content of insulin-like growth factor-1 (IGF-1) has been observed. It should be emphasized that metabolic acidosis affects leptin concentration in serum among patients treated with mHD [Stevinkel *et al.*, 2000a].

It was also found that patients who lose fat free body mass during HD have a higher concentration of C-reactive protein (CRP) and leptin in serum in comparison to those whose fat free body mass increases [Stevinkel *et al.*, 2000a].

Higher leptin concentration in serum was observed also among patients treated with peritoneal dialysis [Stevinkel *et al.*, 2000a].

Helicobacter pylori infection may be another reason for loss of appetite among patients treated with peritoneal dialysis. Applied eradication significantly improved nutritional state among these patients [Aguilera *et al.*, 2001].

Anorexia is often accompanied by nausea, vomiting and taste disturbances, and may be associated with improper dietary restrictions (*i.e.* too large reduction of daily protein intake).

Increased demand for protein is caused by intensification of catabolic processes triggered by acid-base balance disturbances (metabolic acidosis) and the inflammatory process [Lim & Kopple, 2000].

HD is known to increase proteolytic processes. During the treatment, a decrease in serum concentration of amino acids and increased secretion of free amino acids from muscles is observed. During 4–6 hours of dialysis the patient loses 2–3 g of protein, 9–11 g of amino acids, 20–30 g of glucose, some water-soluble vitamins, mineral salts and trace elements [Zdrojewski & Rutkowski, 2004].

Absorption disturbances caused by uremic toxins and coexisting heart-vessel diseases and inflammation states may also cause malnutrition development.

Malnutrition is the factor that increases mortality in patients treated with mHD. Mortality in this group of patients is connected with the existence of chronic inflammation, which constitutes the background for development of the so-called "dialysis syndrome", characterised by: malnutrition of a mixed type, *i.e.* Kwashiorkor-Marasmus (protein-energy malnutrition), sometimes cachexia, and vascular system affection leading to early and progressive atherosclerosis [Amore & Coppo, 2002; Pupim *et al.*, 2002; Quereshi *et al.*, 2002; Naicker, 2002]. The importance of this relationship (between nutrition and mortality) is especially clear if we consider the fact that cardiovascular complications are the first cause of death among dialysis patients (total mortality about 15%, cardiovascular mortality about 9%). It is estimated that amongst patients treated with HD about 40% will develop malnutrition to varying extents, and in 25% of them serious malnutrition will occur [Puka *et al.*, 2004].

TYPES OF MALNUTRITION IN CRF

In the course of CRF three types of malnutrition may be distinguished.

The first type of malnutrition includes patients nourishing correctly, taking in proper amounts of proteins and calories, but presenting reduced serum albumin content (<3.5 g/dL) [O'Keefe, 2002]. This type of malnutrition is mainly connected with increased protein catabolism associated with the inflammatory state. A very characteristic feature of these patients is a triad of disorders: malnutrition, inflammation and atherosclerotic changes (MIA syndrome) [Stevinkel *et al.*, 2000b; Sezer *et al.*, 2002].

There is common agreement that a key role in the development of malnutrition in advanced kidney failure is played by inflammatory response, mediated by cytokines and acute phase proteins: CRP, α_1-acid glycoprotein (AGP), α_1-antichymotrypsin (ACT) and others. Recently a hypothesis was put forward that the main role in malnutrition arising on the basis of the inflammatory process is played by interleukin-6 (IL-6). The reasons for the increase in IL-6 content in serum have not been fully explained, although participation of genetic factors is suggested [Panichi *et al.*, 2002; Mehrotra *et al.*, 2001] and the accumulation of IL-6 caused by diminished renal clearance of the particle is also taken into consideration.

The inflammatory response leads to increased production of free radicals (ROS), accompanied by a deficiency of antioxidant substances (vitamins C, E and plasmalogen type phospholipids are lost into dialysate). This creates oxidant and carbonyl "stress" leading to accumulation of AGEs and advanced lipooxygenation end products (ALEs). Soluble adhesion molecules, the expression of which is altered, promoting monocyte adhesion to the vessel wall, play an important role, especially in endothelial damage.

Among the second type of malnutrition there are patients

with protein deficiency caused by low protein diet and malabsorption syndrome. This type of malnutrition is frequently seen in elderly patients and is, at least theoretically, reversible while the primary disease is treated.

In clinical everyday practice we often meet the third type of malnutrition – the mixed one.

METHODS OF MALNUTRITION INVESTIGATIONS IN CRF

Malnutrition is usually manifested as deviations of anthropometric parameters, such as a decrease in skin fold thickness (fat tissue reserve indicator, fat is normally about 25% body mass) and a decrease in mid-arm muscle circumference (somatic protein reserve indicator in skeletal muscles) and deviations of biochemical parameters. The subjective global assessment (SGA) scale modified by Kalantar-Zadech is a very useful clinical tool for evaluation and monitoring of the patient's nutritional state. The test consists of nutritional anamnesis (changes of the patient's body weight, gastrointestinal signs and symptoms, difficulties with food intake, patient's invalidation, concurrent diseases) and physical examination of the patient (subjective assessment of subcutaneous tissue stores and musculature). The patient may have 1–5 points in each of the 7 assessed areas. The conventional result of a properly nourished individual is <13 points, for medium grade malnutrition 13–22 pts, and for severe malnutrition >23 pts [Kalantar-Zadech *et al.*, 1999].

The nutritional state in CRF can also be evaluated by selected serum biochemical parameters. Concentration of serum albumin is one of the most important elements of mHD patients' nutrition assessment. The decreased concentration of serum albumin is a rather late symptom of protein-caloric malnutrition because of albumin's long half-life (about 20 days). Concentration of serum albumin over 3.5 g/dL is considered to be proper, concentration between 3.4–2.8 g/dL is characteristic of light malnutrition, between 2.7 and 2.1 g/dL of moderate, and below 2.1 g/dL of severe protein-caloric malnutrition [Szczygieł, 1995]. The predictive value of albumin serum concentration markedly increases when accompanied by peripheral blood lymphocyte count evaluation [Gellert, 2002].

Among other biochemical indicators of malnutrition there are reduced indispensable amino acids and histidine concentration in serum (histidine in renal diseases becomes indispensable amino acid) and an increase in phenylalanine, methionine, cysteine concentration (exogenic to endogenic amino acids concentration ratio is lowered), reduced protein reserves in the system and hypoglycaemia, caused by considerable malnutrition on one hand, and diminished gluconeogenesis and insulin biodegradation on the other hand [Myśliwiec *et al.*, 2001].

In nutritional assessment, impaired leukocyte function and reduced lymphocyte count should also be searched for. These phenomena lead to cellular immunodeficiency. The total amount of lymphocytes in 1 mm^3 of blood for a healthy person is >1500; a reduced amount of lymphocytes within the limits 1490–1200 and 1190–800 and below 800 are considered respectively as: minor, moderate and serious malnutrition [Szczygieł, 1995].

Among anthropomeric measures, especially useful for clinical application are skin fold thickness and mid-arm muscle circumference assessments.

Mid-arm circumference (MAC) measurement is made at least twice at the mid-point of the arm (between olecranon and acromion (lat.)), and enables the calculation of mid-arm muscle circumference (MAMC). The received average is compared to standard values, which are 23.2–20.8 cm for women and 25.2–22.7 cm for men. Values of 90–75% of the normal range indicate minor malnutrition, 75–60% moderate, and below 60% severe malnutrition [Gellert, 2002]. Skin fold thickness over the triceps muscle (TSF) is calculated as the average of three measurements and compared with standard values (16.5–15.0 mm for women and 12.6–11.4 mm for men). A decrease of skin fold thickness to 89–75% of normal values suggests minor energetic reserves deficiency; 74–60% moderate, and below 60% serious energetic reserves deficiency, accompanying serious protein-caloric malnutrition [Szczygieł, 1995].

In the case of anthropometrical investigations, loss of weight during six or three months before treatment is the most significant. A loss of weight of >10% or >5%, accordingly, indicates severe malnutrition.

Because 50% of body fat is located in subcutaneous tissue, and changes of subcutaneous skin fat amount correlate with changes of total amount of fat, measurements of skin fold thickness are generally accepted as indicators of total fat amount, that is energetic reserves.

NUTRITIONAL STATE ASSESSMENT IN mHD PATIENTS

In our own research we estimated the nutritional state of 50 mHD patients (males/females 19/31, aged 55.7±14 years, treated with mHD during 35±8 months). Reference values were obtained from 50 healthy volunteers (males/females 19/31, aged 45.6±11 years).

Etiologic factors of chronic kidney failure were: glomerulonephritis – 14, tubulointerstitial nephritis – 14, diabetic nephropathy – 11, hypertensive nephropathy – 4, polycystic kidney disease – 3, other – 4 patients.

In all subjects we performed anthropometric measures (skin fold thickness, mid arm circumference, calculation of fat free body mass (FFM), SGA, BMI). We found the features of malnutrition (SGA scale >13 pts) in 13 (26%) patients treated by HD in our haemodialysis unit. In 3 (6%) patients the grade of malnutrition was severe (SGA >23 pts).

During 4 years of our follow-up observation 69.2%, *i.e.* 9 out of 13, malnourished patients (MN) died *versus* a 29.7% mortality rate, *i.e.* 11 out of 37 patients, in the well-nourished group (WN).

Medium albumin concentration in the MN group of patients was 26.9±4.5 g/dL and it was significantly lower ($p<0.001$) than in the WN group – 32.9±7.7 g/dL. This was accompanied by elevated levels of selected acute phase proteins in MN *vs.* WN group: CRP 7.2±2.1 mg/L *vs.* 2.5±1.7 mg/L, AGP 1495.7±410.2 mg/L *vs.* 1054.8±320.8 mg/L, ACT 429.54±65 *vs.* 389.06±72 mg/L, accordingly. Serum concentration of proinflammatory cytokines was elevated in MN *vs.* WN group: IL-6 40.9±12 *vs.* 29.7±7 pg/mL, IL-1α 4.02±2.3 *vs.* 2.8±1.4 pg/mL, accordingly.

Serum concentration of albumin and prealbumin signifi-

cantly correlated ($p<0.05$) with concentration of acute phase proteins CRP, AGP, ceruloplasmin (Cp) and transferrin (Tf), and might be an essential mortality indicator among patients with CRF.

We disclosed the features of minor muscle mass deficit (medium MAMC 20.85±2.1 cm) in 6 (12%) haemodialysed patients and moderate deficit (medium MAMC 17.68±2.5 cm) in 8 (16%) patients.

We also found minor deficiency in 4 (9%) haemodialysed patients (medium triceps skin fold – TSF 10.5±1.8 mm), moderate deficiency in 10 (20%) patients (medium TSF 8.2±2.7 mm) and severe deficiency in 5 (10%) patients (medium TSF 4.6±2.9 mm).

No correlation between malnutrition level and duration of dialysis therapy was observed. In our research we did not find such a correlation either in SGA or in other anthropometric parameters. On the other hand we observed a dramatically high mortality rate in the malnourished patient group (see above).

PREVENTION OF MALNUTRITION IN CRF

In 2001 reports were issued showing that during the first year of HD treatment a fall in dry body mass among patients with diabetic nephropathy was observed, in contrast to other patients with CRF, especially those well nourished at the beginning of the therapy, among whom dry body mass rose during this period [Ishimura *et al.*, 2001].

The treatment of renal failure patients depends, among other factors, on the stage of its advancement.

During latent, compensated, and early periods of decompensated renal failure (stages 1–3 by NKF) conservative treatment is applied. Since primary kidney pathology is usually irreversible in CRF patients, the most important objective of medical care is to slow down the progression of CRF, and one of the most important issues in this context is recommendation of a proper diet on one hand, and the patient's adherence to it on the other.

The rule is constant medical assistance and precise estimation of the patient's hydration level. The basis for such estimation is regular measurement of the patient's body weight.

In the latent stage of renal failure (stages 1–2 by NKF) usually no specific dietary treatment is recommended. In particular no dietary restrictions, such as a salt-free diet, low protein diet or reduction of water supply, should be introduced (unless specially recommended due to primary kidney disease).

In compensated renal failure (stage 3 by NKF), special attention should be paid to potential appearance of polyuria, the presence of which requires an increase of fluid supply, at least 700–800 mL per day more than daily diuresis.

In that stage dietary treatment is recommended. First of all phosphate content in daily diet should be reduced. Food rich in phosphorus that should be avoided includes: evaporated milk, powdered milk, hard cheese, processed cheese, whole meal bread, crisp bread, muesli, oatmeal, barley and rye flour, dough with large baking powder content, offal, fish, beans, peas, spinach, mushrooms, dried fruit, nuts, cocoa, chocolate, biscuits. Application of a low-phosphate diet (5–7 mg/kg body wt./day) for a long time is practically impossible because is not tolerated by patients. In practice, medicines are used, administered orally, which bind phosphate ions in the alimentary tract, preventing their absorption. One of them is calcium carbonate ($CaCO_3$), administered at doses from 500 to 6000 mg per day so that the concentration of phosphates in serum can be kept under the upper correct concentration value (<1.8 mmol/L) and Ca × P product below 4.44 mmol/L. Patients showing high serum phosphate levels accompanied by excessive calcium level may benefit from short-term therapy (for max. 4–6 weeks) with aluminium hydroxide. This is controversial, however, because of the proven toxicity of aluminium in chronic therapy. An effective, non-calcium phosphate-binder, restraining absorption of phosphates from the alimentary tract, is sevelamer. It is recommended to use it at large doses of 6–12 g per day [Kopple, 2001]. New perspectives of treatment of calcium-phosphate disturbances are associated with introduction of new drugs into clinical practice, *i.e.* the so-called "calcimimetics" (cinacalcet), synthetic derivatives of vitamin D (pericalcitol) and lantan compounds.

Contrary to previous stages, decompensated renal failure (stage 4 by NKF) requires constant multidirectional treatment, which is based on dietary treatment. Reduction of protein supply is one of its main rules. The basis of the daily diet should be pasta, rice, bread, sponge cake and rusks. Low protein diets (Giordano and Giovanetti "pasta-egg" diet, Połeć "potato-egg" diet) were introduced for patients with advanced renal failure treatment already in their sixties [Zdrojewski & Rutkowski, 2004].

Nowadays no very restrictive diet (reduction of protein concentration to 0.3–0.4 g/kg body wt./day) is recommended, because it can lead to development of malnutrition. A commonly accepted protein supply restriction in the diet is the amount of 0.6–0.8 g/kg body wt./day. The effectiveness of this leads to prolongation of the predialytic period by up to 40%.

In this stage it is recommended to administer fully valuable protein, which includes all exogenous amino acids, *i.e.* the white of hens' eggs. The least valuable is vegetable originated protein, because it does not contain a full range of amino acids [Zdrojewski & Rutkowski, 2004].

If indications exist, special solutions of amino acids, the so-called "renal", are administered, which contain eight indispensable amino acids: isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan and valine with the addition of histidine, and supplemented with amino acids which are not indispensable, such as: alanine, proline, serine and cysteine.

In the dietary treatment of CRF a major function is served by ketoanalogues. They are a specific substitute allowing replenishment of exogenous amino acid demand during application of a diet with restricted protein intake.

In several studies comparing the effects of a low protein diet supplemented with ketoanalogues or amino acids it was observed that their influence on delay in progression of the disease was equal, but the concentration of triglycerides in plasma was essentially lower in the ketoanalogue-supplemented diet [Zdrojewski & Rutkowski, 2004].

It was also observed that ketoanalogues administered to supplement a low protein diet and erythropoietin therapy had a positive influence not only on protein turnover, but also on

lipid management, thus contributing to inhibition of disease progression and reduction of protein content in urine [Bergesio *et al.*, 2001].

Because of increased concentration of VLDL in serum (especially pre β-VLDL fraction), LDL and IDL fraction and reduction of HDL-cholesterol concentration (hyperlipidaemia of type IV according to Fredrickson), a diet with low content of saturated fatty acids is recommended for these patients, except for butter, which contains natural vitamins (A, D and E) and should in moderate amounts be a part of the diet. Saturated fat should be replaced with unsaturated fat (oils and fats from fish) [Zdrojewski & Rutkowski, 2004].

The diet of patients with CRF should contain increased amounts of calcium. Calcium concentration level in serum should range between 2.1 and 2.4 mmol/L, and bicarbonate (HCO_3^-) concentration in serum should exceed 22 mmol/L. Ca × P product should not exceed 4.44 $mmol^2/L^2$ and concentration of iPTH should be kept in the range of 100–200 pg/mL, because exceeding these values may lead to metastatic calcifications of organs and vessels [Zdrojewski & Rutkowski, 2004].

Dietary treatment and reduction in absorption of phosphates from the alimentary tract allow calcium-phosphate management disturbances to be corrected, and in effect contributes to the reduction of secondary hyperthyroidism development, lower risk of cardiovascular complications and renal osteodystrophy. In addition these patients commonly receive active vitamin D metabolites (1000 IU/day).

Management of sodium and potassium balance is the next important issue of conservative treatment. Establishing proper sodium supply is based on assessment of current loss of this electrolyte in urine. When estimating the demand for sodium, one should take into consideration the clinical situation of a given patient, first of all arterial pressure values.

In the fully symptomatic stage of uremia patients require renal-substitutive treatment as well as strict adherence to specific dietary recommendations.

According to NKF (K/DOQI), patients below sixty years of age, with GFR<25 mL/min, and patients requiring renal replacement therapy should receive 35 kcal/kg body wt./day, and patients aged over 60 should receive 30 kcal/kg body wt./day in their diet.

The diet of patients in maintenance HD programme should contain 1.2–1.3 g of protein/kg body wt./day [Zdrojewski & Rutkowski, 2004].

Patients treated with peritoneal dialysis, while complicated by peritonitis (which causes large losses of proteins into dialysate) demand protein increased protein supply, equal to 1.4–1.6 g/kg body wt./day.

Sodium intake among haemodialysed patients should be kept within the range of 80–100 mmol/day. Intake of the following articles should be avoided: smoked meat, preserves, smoked and salted fish, hard cheese, processed cheese, blue cheese, camembert, sauerkraut, cucumber and other salted vegetable preserves, crisps, popcorn, tinned food, ready-made tinned food, ready-made packed soups and powdered or slab consommé, ready-made spice mixtures (mustard, ketchup, garlic salt, *etc*).

If daily diuresis exceeds 1.5 L, potassium intake in the diet generally should not be limited, because on one hand effective adaptive mechanisms exist, and on the other hand hypokalaemia may cause further decrease in nephrons' function. When the end stage renal disease patient becomes oliguric, an increase of potassium concentration in the serum may become life threatening because of the possibility of cardiac arrhythmias. In this situation potassium supply should be decreased to 50–65 mmol/day (2000–2500 mg). Products that should be avoided are: evaporated milk, powdered milk, groats, muesli, meat preserves with fried onion, mushrooms, potatoes, dried vegetables, pizza, beans, peas, spinach, sauces, tomato juices and puree, crisps, chips, apricots, bananas, nuts, coffee, beer, wine, fruit juices, cocoa, chocolate, fruit candies [Zdrojewski & Rutkowski, 2004].

Application of B6 vitamin and folic acid may improve lipid turnover management and contribute to reduction of homocysteine concentration, considered to be an independent risk factor of atherosclerosis.

Among patients treated with the renal-substitutive method of peritoneal dialysis, energetic demand is in about 70% covered by glucose contained in dialysis fluid. Taking that into consideration, these patients should in their diet avoid sweets, highly caloric liquids, jams, honey and products with high cholesterol content.

SUMMARY

Properly applied conservative treatment, regularly adjusted to the changing clinical situation, including arterial pressure control, prescribing proper diet, correction of anaemia, calcium-phosphate and water-electrolyte disturbances management, significantly retards progression of renal failure and is a very important part of renal-substitutive treatment.

REFERENCES

1. Aguilera A., Codoceo R., Bajo M.O., Diez J.J., del Peso G., Pavone M., Ortiz J., Valdez J., Cirugeda A., Fernanez-Perpen A., Sanchez-Tomero J.A., Selgas R., *Helicobacter pylori* infection: A new cause of anorexia in peritoneal dialysis patients. Perit. Dial. Int., 2001, 21, suppl. 3, S152–S156.
2. Amore A., Coppo R., Immmunological basis of inflammation in dialysis. Nephrol. Dial. Transplant., 2002, 17, 16–24.
3. Aparicio M., Chauveau P., Level C., de Precigout V., Lasseur C., Combe C., Nutritional status and outcome in older patients. Nephrologie, 2002, 23, 77–83.
4. Bergesio F., Monzani G., Ciuti R., Cirami C., Martinelli F., Salvadori M., Tosi P.L., Autoantibodies against oxidized LDL in chronic renal failure: Role of renal function, diet, and lipids. Nephron., 2001, 87, 127–133.
5. Don B.R., Rosales L.M., Levine N.W., Mitch W., Kaysen G.A., Leptin is a negative acute phase protein in chronic hemodialysis patients. Kidney Int., 2001, 59, suppl. 3, 1114–20.
6. Gellert R., Treatment of progressive renal failure with ketodiet. 2002, Fresenius Kabi, Warszawa, pp. 34–37 (in Polish).
7. Ishimura E., Okuno S., Kim M., Tamamoto T., Izumotani T., Otoshi T., Shoji T., Inaba M., Nishizawa Y., Increasing body fat mass in the first year of hemodialysis. J. Am. Soc. Nephrol., 2001, 12, 1921–1926.

8. Kalantar-Zadeh K., Kleiner M., Dunne E., Lee G.H., Luft C.F., A modified quantitative subjective global assessment of nutrition for dialysis patients. Nephrol. Dial. Transplant., 1999, 14, 1732–1738.
9. Lim V.S., Kopple J.D., Protein metabolism in patients with chronic renal failure: role of uremia and dialysis. Kidney Int., 2000, 58, 1–10.
10. O'Keefe A., Daigle N.W., A new approach to classifying malnutrition in the hemodialysis patient. J. Ren. Nutr., 2002, 12, 248–255.
11. Kopple J.D., National-Kidney Foundation K/DOQI clinical practice guidelines for nutrition in chronic renal failure. Am. J. Kidney Dis., 2001, 37, 66–70.
12. Mehrotra R., Kopple J.D., Nutritional menagement of maintenance dialysis patients: Why aren't we doing better? Annu. Rev. Nutr., 2001, 21, 343–379.
13. Myśliwiec M., Kokot F., Chrzanowski W., Diagnosis and treatment of renal failure latent and compensated stage. 2001, *in*: Standards of Diagnosis and Treatment in Renal Diseases (eds. B. Rutkowski., S. Czekalski). Wydawnictwo Medyczne MAKmed, Gdańsk, pp. 186–193 (in Polish).
14. Naicker S., Nutritional problems associated with end-stage renal disease in the developing world. Artificial Organs, 2002, 26, 757–759.
15. Panichi V., Migliori M., De Pietro S., Taccola D., Bianchi A.M., Giovannini L., Norpoth M., Metelli M.R, Cristofani R., Bertelli A.A., Sbragia G., Tetta C., Palla R., C-reactive protein and interleukin-6 levels are related to renal function in predialytic chronic renal failure. Nephron, 2002, 91, 594–600.
16. Puka J., Rutkowski B., Lichodziejewska-Niemierko M., Grenda R., Czekalski S., Lao M., Rowiński W., Bautembach S., Report about condition of renal replacement therapy in Poland – 2003 year. 2004, Wydawnictwo Medyczne MakMedia, Gdańsk, pp. 1–40 (in Polish).
17. Pupim L.B., Kent P., Caglar K., Shyr Y., Hakim R.M., Ikizler T.A., Improvement in nutritional parameters after initiation of chronic hemodialysis. Am. J. Kidney Dis., 2002, 40, 143–151.
18. Quereshi A.R., Alvestrand A., Divino J.C., Gutierrez A., Heimburger O., Lindholm B., Bergstrom J., Inflammation, malnutrition, and cardiac diseases predictors of mortality in hemodialysis patients. J. Am. Soc. Nephrol., 2002, 13, 1, 28–36.
19. Sezer S., Ozdemir F.N., Arat Z., Turan M., Haberal M., Triad of malnutrition, inflammation, and atherosclerosis in hemodialysis patients. Nephron, 2002, 91, 456–462.
20. Stevinkel P., Lindholm B., Lonnqvist F., Katzarski K., Heimburger O., Increases in serum leptin levels during peritoneal dialysis are associated with inflammation and a decrease in lean body mass. J. Am. Soc. Nephrol., 2000a, 11, 1303–1309.
21. Stevinkel P., Heimburger O., Lindholm B., Kaysen G.A., Bergstrom J., Are there two types of malnutrition in chronic renal failure? Evidence for relationship between malnutrition, inflammation and atherosclerosis (MIA syndrome). Nephrol. Dial. Transplant., 2000b, 15, 953–960.
22. Szczygieł B., The principles of parenteral nutrition. 1995, *in*: Internal Diseases, vol. 3 (ed. A. Wojtczak). PZWL, Warszawa, pp. 684–700 (in Polish).
23. Zdrojewski Z., Rutkowski B., Protein, carbohydrates and lipid metabolism disturbances. 2004, *in*: Dialisotherapy in Medical Practice (ed. B. Rutkowski). MAKMedia, Gdańsk, pp. 440–451 (in Polish).

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WSPÓŁCZESNE POGLĄDY NA TEMAT ZNACZENIA TERAPII ŻYWIENIOWEJ W PRZEWLEKŁEJ NIEWYDOLNOŚCI NEREK – ARTYKUŁ PRZEGLĄDOWY

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Proces chorobowy toczący się w nerkach w przebiegu przewlekłej niewydolności nerek (p.n.n.) prowadzi do wielu zaburzeń metabolicznych w całym organizmie. Upośledzenie gospodarki białkowej, węglowodanowej, tłuszczowej, elektrolitowej i wapniowo-fosforanowej stanowią główne przyczyny niedożywienia białkowo-energetycznego u chorych. Stosowanie właściwej, indywidualnie dostosowanej diety może zahamować postęp choroby oraz zmniejszyć nasilenie dolegliwości.