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CONSENSUS REPORT

Expert working group report on nutrition in adult patients with renal insufficiency (Part 2 of 2)

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Introduction

The second part of the report deals with the nutritional therapy of 1) patients with chronic renal failure on haemodialytic treatment or peritoneal dialysis, 2) patients with nephrotic syndrome, 3) patients with acute renal failure and 4) kidney transplanted patients. Each of these situations is unique as to the aetiology of malnutrition and nutritional treatment. Haemodialytic treatment or peritoneal dialysis are in themselves additional catabolic stimuli. Preventing protein-energy malnutrition in hemodialysis patients is important because of its prognostic relevance. Peritoneal dialysis patients pose a special problem because the energy intake can be in excess, while the nitrogen balance is often negative, and they are at risk of developing a kwashiorkor-like syndrome. The nutritional risk of patients with acute renal failure and the difficulty in nourishing them adequately is outlined. Specific recommendations are given for the pre-transplant, early post-transplant and late post-transplant phases.

Nutritional therapy in patients with chronic renal failure: patients on hemodialysis

Additional causes of malnutrition during hemodialysis treatment

The prevalence and effects of protein-energy malnutrition (PEM) in dialysis patients have been outlined in the first part of this report. Specific aspects regarding the causes of PEM in maintenance hemodialysis (MHD) patients will be described.

Anorexia is often responsible for low nutrient intake in hemodialysis (1, 2). This may be related to uremic toxicity, or to psychosocial and economic factors (loneliness, depression, ignorance, poverty, alcohol and drugs), to inadequate unpalatable diets, or to gastro-intestinal motility disturbances.

Intercurrent or underlying illnesses are frequently present which interfere with protein and energy metabolism and are responsible for hypercatabolism.

Dialysis promotes a net protein catabolism and induces a reduction of protein synthesis (3). Four to nine grams of free amino acids and 2–3 g of peptide-bound amino acids are removed during each session. The frequent re-use of filters exacerbates the amino acid and albumin loss (4, 5).

Under certain conditions, blood-membrane interaction can be a catabolic stimulus, resulting in an increased release of amino acid from muscle (6). When more biocompatible synthetic membranes were used, this increased release of amino acids was not observed (7). Bioincompatible membranes may exert negative effects on nutrition, by increasing the release of proteases and cytokines (IL-2, TNF-a), via prostaglandin liberation and/or IGF-2 suppression (8, 9). However the effect of biocompatibility on nutritional status is still a matter of debate (10, 11).

Underdialysis is often an important cause of PEM. An adequate dialysis dose improves quality of life and survival (12–14). Protein catabolic rate, protein intake, dialysis adequacy, anorexia, endogenous catabolism and nutritional status are influenced by each other (15). Current recommendations for dialysis dose based on conventional dialysers suggest a Kt/V for urea[1] of

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[1] Kt/V_{urea}: K: urea clearance(ml/min); t: length of dialysis (min); V_{urea}: distribution volume of urea (ml). This index gives an appropriate estimation of dialysis dose.

1.2–1.4 (8), but it may be lower in dialysers, with improved clearance of larger solutes. The underdialysis may also be related to increased morbidity as an independent factor, not necessarily related to nutrition.

Other catabolic factors related to dialysis might be the composition of the dialysis fluid (use of acetate), its contamination with endotoxins as well as losses of blood, glucose and other nutrients (water soluble vitamins, carnitine).

A good nutritional state at the entry in dialysis is a strong prerequisite for an uncomplicated long term dialysis (16, 17).

Nutritional therapy

Besides nutritional treatment, the nutritional management of MHD patients should focus on:

- adequate dialysis;
- counteracting catabolic stimuli (acidosis, infections, anaemia, cardiovascular instability, etc.);
- avoiding drugs that reduce appetite and intestinal motility.

Protein intake

MHD patients should eat at least 1.2 g/kg/day (1.2–1.4 g/kg/day), calculated for ideal body weight (14, 16, 18, 19). The percentage of high biological value proteins should be higher than 50%. The dialysis dose must be adequate to control nitrogen waste products and acidosis in higher nitrogen intake (20). If the daily protein intake falls below 1.2 g/kg, the patients are at risk of developing PEM. A lower intake (1.0–1.1 g/kg/day) requires a close monitoring of nutritional status and is allowed only in patients with normal protein status (21).

Energy intake

The energy intake should preferably be higher than 35 kcal/kg/day, and it must be increased according to physical activity (14, 16, 18, 19). Energy requirements are also related to the changes of lean body mass (22). Additional energy intake is needed if hypermetabolic situations supervene, such as infections or surgery.

Fluids and electrolytes (16, 18, 23)

Water and fluid intake must be strictly controlled: an amount of 500–800 ml/day (taking into account the fluid content of foods) plus residual diuresis is generally allowed. Low to moderate sodium intake (60–100 mEq/day, 3.56 g of NaCl) is prescribed. If the patient still has some residual kidney function only a moderate reduction of dietary potassium (K) intake will be necessary. However, in the presence of metabolic acidosis, hypermetabolism, and administration of angiotensin converting enzyme inhibitors high predialysis K plasma values (>6 mEq/L) are frequent; in these conditions total K intake should be ≤ 1 mEq/kg. In order to decrease potassium intake, it can be useful to throw away cooking water after preparing vegetables, particularly potatoes and to avoid steamed and microwaved vegetables. The calcium intake should be higher than 1000–1500 mg/day, and phosphorus should be limited. However, the phosphorus restriction makes the diet unpalatable, especially if the protein intake is high: the American and European Dietetic Associations recommend 17 mg/kg ideal weight. A diet rich in protein and calcium and poor in phosphorus is difficult to obtain: a careful exclusion of some foods (24) and meticulously monitoring phosphate plasma level, will allow the use of lowest possible amount of phosphate-binding products. Calcium salts are effective phosphate binders provided that they are taken during meals. Calcium carbonate or acetate supplements are largely used for intestinal binding of phosphate, to correct metabolic acidosis and to provide the required calcium supplements.

Vitamins

Losses through the dialysis membranes and dietary manipulations used to lower K and phosphorus intake can be responsible for some disorders in vitamin status. Low plasma levels of ascorbic acid and pyridoxine have been described in MHD patients (16, 25, 26). Daily supplements of vitamin C (30–60 mg), B6 (10–20 mg) and folic acid (1mg) are suggested. Larger supplements of folic acid (2.55 mg/day) may be useful to lower high homocysteine levels (25, 26). However, no long-term studies are available on whether high dosages of folic acid are toxic in chronic renal failure. B_1 and B_2 supplements are also used generally; vitamin A is always increased in uremia and its supplementation is contraindicated.

In the early phase of dialytic treatment, the nutritional habits of uremic patients change dramatically. After a long period of low protein diet, with nearly ad libitum or even enhanced fluid intake, the patients are asked to eat large amounts of protein and to limit their water intake. This period has been called 'phase of adaptation' (16, 23, 27), and it is psychologically very difficult, partly because of the dependence on dialysis and because of the side effects of the treatment. The patients should therefore be encouraged to follow the new nutritional guidelines, and frequent dietary counselling is advisable. Special formula products for haemo-dialysis treatment can be useful especially in malnourished patients, who are not able to increase their nutrient intake (28). Sometimes it may be necessary to reduce the dietary restrictions relating to potassium and phosphorus intake. If malnutrition develops artificial nutrition can be considered.

Intradialytic parenteral nutrition (IDPN)

An easy access to the blood flow is offered during dialysis sessions, and energy and nitrogen can be infused

to patients. Initially, rather disappointing results have been found after short-term (29) and even long term treatment (30); little objective benefit was observed before 4 months of treatment. However, other studies (31) showed beneficial effects and even a reduced mortality has been demonstrated in malnourished haemodialysis patients treated with IDPN in retrospective analysis (32, 33). The amino acid formula infused to uremic patients should be adapted to the metabolic changes of uremia (34), and both nonessential and essential amino acids should be included. Good results were also noticed on nutritional and immunological status (33–35). An increase in spontaneous eating was also reported (34, 35).

During a single hemodialysis seance 50 g of amino acids and 800–1000 kcal or more can be easily infused (36). Energy supply can be given as glucose or glucose-fat mixture. A ratio of 30% lipid and 55% glucose is usually well tolerated. Hence in most malnourished dialysis patients, eating less than recommended intakes, IDPN makes it possible to reach an adequate energy and protein intake.

Conclusions

Correcting PEM can be more difficult that preventing its occurrence during MHD because of peculiar aspects such as the anorexia and catabolism promoted by dialysis. It is therefore very important for the patient to have optimum nutritional status before the commencement of dialysis. The intake of water, sodium and potassium should also be carefully monitored. Intradialytic artificial nutrition can be used to correct transient disturbances in nutrient intake but should not be recommended as a long-term alternative to oral nutrition in regular dialysis patients.

Nutritional therapy in patients with chronic renal failure: patients on continuous ambulatory peritoneal dialysis (CAPD)

Additional causes of malnutrition in CAPD patients

1. The constant absorption of glucose from the dialysate in peritoneal cavity and the abdominal distension can suppress appetite (37).
2. Loss of free amino acids is lower than in hemodialysis (1.2–3.4 g/24 h) but large amounts of proteins are also lost (from 5–15 g/day). The loss of protein can increase by 50–100% if mild peritonitis supervenes, but it can be much higher if the infection is severe (38). As a consequence of the above, an excess of energy over protein net intake can ensue, and obesity can develop that can conceal a dangerous kwashiorkor-like protein malnutrition.
3. Chronic or acute peritoneal inflammation is per se a catabolic stimulus, at the level of body protein mass.
4. Physical inactivity is frequent, mainly because of the time-consuming dialysis procedures.

Nutritional treatment

Some differences from haemodialysis patients should be noted as far as nutritional allowances are concerned (38, 39).

1. Protein intake should be 1.2–1.5 g/kg/day at least 50% of high biological value (HBV), according to nutritional status and to protein loss in the dialysate. Additional 0.1–0.2 g/kg/day should be prescribed if peritoneal inflammation occurs.
2. Energy intake: the glucose absorption from the dialysate must be taken into account. The total energy requirement, as in MHD patients, is $\geq$ 35 kcal/kg/day, but the glucose absorption can account for 25–30% of energy requirements. This is one of the causes of the severe hypertriglyceridaemia that is often present in these patients; the oral energy supply should favour lipids (30–40% of total energy intake, diet+dialysate) and complex carbohydrates (25–40% of total energy intake, according to glucose absorption). Simple sugars should be restricted.
3. Vitamins: pyridoxine (10 mg) and vitamin C (100 mg) supplements are recommended (40).

Intraperitoneal nutrition

The intraperitoneal infusion of amino acids was tested in the early 1980s, but it raised many problems due to selection of patients (38), unspecific amino acid formula, insufficient buffering of solutions, with overall discouraging results.

When new solutions were available (1.1% new amino acid formula, 40 mmol/L lactate), promising results were obtained (41, 42): improved nitrogen balance, higher protein plasma levels and a good correction of plasma amino acid concentration were noticed. The treatment with intraperitoneal amino acid solutions seems to be a very promising nutritional treatment (43, 44). In the future new studies are needed to better identify the patients that especially need this treatment (45).

Growth factors

While recombinant human growth hormone (rhGH) is a well established therapy with limited side-effects for chronically uremic children with growth retardation (46), the possible positive effects of GH in adult uremic patients treated with haemodialysis or peritoneal dialysis are still debated and under investigation. The administration of rhGH both to maintenance haemodialysis (47) and to CAPD (48) patients was associated with an anabolic response as demonstrated by improved nitrogen retention and decreased nitrogen appearance,

and also suggested by changes in amino acid plasma concentrations (49). The administration of rhIGF-1 might also improve nitrogen metabolism in CAPD patients (50). Haemodialysis patients treated with rhGH showed an increased muscle protein synthesis without effect on the rate of muscle protein degradation (51).

Conclusions

Peritoneal dialysis presents two peculiar nutritional aspects: the first is the large amount of calories that the patient receives with the peritoneal infusion fluid (dialysate); the second is the chronic and sometimes massive loss of proteins through the peritoneal membrane. Nutritional indications/recommendations to these patients must take into account both of these factors.

Recommendations for nutritional treatment of dialysed patients

Haemodialysis
Proteins: 1.2–1.4 g/kg ideal body weight/day, 50% HBV. Energy: 35 kcal/kg/day or more. High calcium-low phosphorus intake is recommended. Daily Vit C, B6 and folic acid supplements.

Peritoneal dialysis
Proteins: 1.2–1.5 g/kg/day (or more if peritonitis occurs). Energy: 30–35 kcal/kg/day as dietary intake, plus dialysate energy. Minimal simple sugars are allowed. Daily Vit C and B6 supplements.

If malnutrition supervenes, intradialytic or intraperitoneal artificial nutrition can be useful.

Suggestions for future research

1. Controlled studies of effects on nutritional status of different protein-energy intakes in patients.
2. Controlled studies on the efficacy of IDPN (vs oral supplements).
3. Modulation of nutrient requirements in relation to age.
4. Modulation of protein catabolism in dialysis by mean of drugs: Indomethacin? Pentoxifilline? GH, IGF-1?

Nutrition in the nephrotic syndrome

Despite the fact that the kwashiorkor-like syndrome of long-standing severe nephrotic syndrome (NS) is not completely due to low albumin levels, and several mechanisms are involved, malnutrition is a common finding in this syndrome. Besides the well-known disturbance of visceral protein and lipid metabolism (increased albumin synthesis and decreased catabolism, increased VLDL, LDL and IDL [52]), a severe involvement of muscle metabolism has been described (53).

In the past, nutritional treatment of NS was traditionally characterized by a high protein intake, in order to compensate for the protein urinary loss and low albumin/protein plasma levels. Higher protein intake experimentally induces increased protein synthesis and increased proteinuria with a negative nitrogen balance and lower plasma protein levels (54). In man, high protein intakes (1.6–2.0 g/kg/day) are able to increase albumin synthesis, at the site of albumin in RNA transcription, the fractional catabolic rate of albumin, the renal excretion of albumin and protein, the plasma phosphate and renin activity (55, 56). Increased expression of TGF-β, IGF-1 and PDGF are described with higher protein intake. Neither plasma proteins nor skeletal muscle protein synthesis are improved. Therefore, a high protein intake seems to be harmful for the kidney and for nutritional status. With a lower protein intake (55, 56) protein excretion is definitely decreased.

Changes in glomerular capillary pressure and haemodynamics are responsible for changes in proteinuria. The quality of dietary proteins/aminoacids might be involved (57, 58). Branched chain amino acid infusion does not influence renal haemodynamics (59). A direct beneficial relationship between the reduction of urinary protein loss obtained by low protein diet in patients with nephrotic syndrome and the subsequent course of their renal function has been described (60–62).

An interesting evolution in the nutritional treatment of nephrotic syndrome was more recently proposed (63, 64) by means of diets with 0.6–0.7 g/kg/day of proteins, all of vegetable origin, supplemented with essential amino acids (EAA) or keto-analogues. This treatment was able to decrease proteinuria and hyperfiltration, while improving the lipid pattern. The albumin plasma level was not negatively affected. A similar effect on plasma lipid was described using a soy-based vegetal protein diet (0.7 g/kg/day), and no evident signs of malnutrition were noticed (65).

Conclusions

A relatively low protein diet (0.8–1.0 g/kg/day independently from proteinuria) seems to be the most acceptable and safe solution for patients with NS, without renal insufficiency. The span between the two figures can account for the differences in the amount of proteinuria. No definitive data are available on the effects on nutritional status and on long term effects on renal functions of dietary quantitative replacement of large amount of proteinuria (66). The commonly suggested regimen of 0.6 g/kg/day supplemented with 1 g HBV proteins for each gram of proteinuria exceeding 3 g/day provides, as a mean, lower protein intake and, while decreasing urinary protein losses, it has no significant positive effect on the progression of renal failure (23). Lower protein intakes, supplemented with keto and/or EAA, with

larger use of vegetable proteins are also probably useful and safe.

Energy intake should be 35 kcal/kg/day, or higher, according to physical activity: 30% should derive from fat (low in saturated and higher in oleic and linoleic acid content) (67).

Recommendations for nutritional treatment of patients with nephrotic syndrome

Proteins: relatively low-protein diet (0.8–1.0 g/kg/day), without replacement of urinary protein loss.
Energy: 35 kcal/kg/day or more, according to physical activity.

Nutritional therapy in patients with acute renal failure (ARF)

Protein-energy malnutrition in ARF

A negative nitrogen balance frequently characterizes ARF and sometimes severe hypercatabolism[2] is present. Many factors are involved, such as endocrine abnormalities (hypersecretion of glucagon, catecholamines, cortisol, parathormone, insulin resistance), metabolic acidosis, imbalance between protease and antiprotease, excessive cytokine activity (TNF, Il-1, Il-6), immobilisation, 'uremic toxins', bleeding, nutrient losses (haemodialysis, peritoneal dialysis, haemofiltration), associated events (trauma, burns, sepsis, shock, multiple organ failure), reduced food intake, etc. (71, 72). This metabolic situation can greatly affect the clinical course of the disease, as it entails further accumulation of urea and other uremic toxins. It promotes the development of mineral and electrolyte problems (hyperkalaemia, hyperphosphataemia, metabolic acidosis), it produces lean body mass depletion, and may have adverse effects on the recovery of renal function (71). For these reasons, the acute renal failure syndrome, especially if complicated by shock or sepsis, has an extremely high mortality rate (73). Although the hypothesis that complete nutrition is able to improve prognosis in ARF patients (74) has not been definitively confirmed (75, 76), the markedly negative nitrogen balance strongly suggests that such patients should be supplied with an optimal amount of nutrients.

Therapeutical regimens and efficacy of support in ARF

Nutrition support of ARF patients particularly demands an integrated and overall view on energy, protein, and fluid and electrolyte metabolism, and a careful and accurate assessment of nitrogen and electrolyte balances.

Energy

ARF per se is not generally associated with increased energy expenditure (77–79), which is, if anything, lower than in patients affected by other acute events. However, coexisting acute traumatic events do increase resting energy expenditure (e.g. in sepsis the increase is approximately 30%) (77, 79). In hypercatabolic patients with acute renal failure, relatively low levels of energy supply (26 kcal/kg/day) have been associated with a better nitrogen balance, compared to higher supplies (35 kcal/kg/day) (80). As a general rule, a higher energy intake is suggested in patients with higher urea nitrogen appearance and worse nitrogen balance, but more than 40 kcal/kg/day are seldom used and are potentially dangerous (73). Energy expenditure should therefore be measured, or calculated, and then corrected depending on the concomitant condition: in most cases it does not exceed 1.3 basal energy expenditure (77), though it may reach 1.5–1.7 in some cases (81). The calculation of energy expenditure can be based on Harris-Benedict equations or on WHO equations, that seem to give better estimates of resting metabolic rate (73, 82). To avoid the risk of overfeeding, the calories to be administered should be calculated on the patients' estimated dry weight as ARF patients are often hyperhydrated or have overt oedemas (81).

In some replacement therapy situations, heat loss occurring during dialysis should also be taken into consideration (77). The presence of reduced glucose tolerance and insulin resistance caused by acute uremia itself or acidosis, or increased gluconeogenesis (83) suggest the need for careful monitoring of blood glucose levels and the possible use of insulin in glucose solutions to attain euglycaemic levels.

In both acute and chronic kidney failure, there is a decreased ability to utilize exogenous lipids (84, 85), and even the use of medium-chain triglycerides has not been shown to offer any advantages over long-chain triglycerides (84). This experimental finding, together with the frequent occurrence of hypertriglyceridaemia, makes it advisable to limit the percentage of lipids to 20–25% of the total energy and to monitor frequently triglyceridaemia during treatment (86). Lipid intake is in any case considered of great importance since lipids, besides being a concentrated, low osmolarity source of energy which produces lower quantities of CO_2, are carriers of essential fatty acids (77, 87).

Nitrogen

The total quantity of nitrogen to be administered to ARF patients depends on a number of factors mainly related to clinical conditions (degree of catabolism), extent of impairment of renal function, route of delivery of the nutrients and whether replacement therapy has

[2]Definition of hypercatabolism in kidney failure: daily increase of serum urea nitrogen over ~11 mmol/L (68), or urea nitrogen appearance (UNA) >15 g/day, or a ratio of srum urea nitrogen to serum creatinine >10 (69), or UNA 5–10 g/day (moderate catabolism) and >10 /day (high catabolism) (70).

been instituted. In the most severe cases replacement therapy, or the possibility of resorting to it, makes it possible to provide ARF patients with quantities of nitrogen, fluid and electrolytes similar to those administered to critical patients with normal kidney function.

Nutritional support in ARF should not be started very early after injury. The utilization of amino acid for protein synthesis might be impossible until uremia is controlled, and large quantities of glucose and amino acids during the 'ebb phase' (first 48 hours after trauma) might increase renal oxygen consumption and even aggravate tubular damage and the loss of renal function (88, 89).

Patients with non oliguric ARF, from primary renal involvement (such as drug induced ARF), with expected short duration of renal shut-down (1–2 weeks) are less catabolic: if they receive conservative therapy and are able to continue oral feeding, nitrogen provision should not exceed 0.55–0.6 g/kg/day of proteins of high biological value. Patients with anorexia and nausea who have a functional digestive tract may be given total enteral nutrition, with a 0.55–0.6g/kg/day protein intake. To obtain an adequate energy intake, specific products for patients with renal failure, with low protein to energy ratio can be used (90). Protein intake might be gradually increased up to 0.8 g/kg/day if BUN levels are below ~36 mmol/L; 0.6–1.0 range of protein or essential amino acids (EAA) and non essential amino acids (NEAA) has been also suggested in these patients (91). In ARF, besides EAA, also NEAA histidine, arginine, tyrosine, serine, and cysteine become indispensable, while others, such as phenylalanine and methionine, may accumulate (91, 92). If more than 0.4–0.5 g/kg/day of amino acids are infused, non essential amino acids, and in particular those from the urea cycle (arginine, ornitine or citrulline) must also be given (91). The use of EAA alone must be avoided, as the occurrence of important amino acid imbalances and severe clinical consequences have been described (73, 81, 92).

Severely catabolic patients usually receive replacement therapy (haemodialysis, peritoneal dialysis, haemofiltration). This allows administration of a higher quantity of nitrogen, as well as a freer supply of fluid and electrolytes. Patients with such conditions are hardly ever able to feed themselves adequately per os and may therefore receive enteral nutrition or, more often, because of the impaired gastrointestinal motility, vomiting or diarrhoea, total parenteral nutrition (TPN). Protein and/or amino acid requirements in these patients range from 1.0–1.5 g/kg/day depending on the severity of catabolism. There is no firm evidence that increasing the protein intake further results in better nitrogen balance but it may promote the appearance of nitrogen waste products (76). However, prescribed protein/amino acid intake is higher (up to 1.5–2.5 g/kg/day) in more severe ARF patients treated with CVVH, CVVHD, CVVHDF (73, 93), which have greater weekly urea clearances. In acutely ill hypercatabolic patients, with superimposed ARF, positive nitrogen balance was achieved only if the protein intake was higher than 1 g/kg/day, but interestingly the nitrogen balance was improved by a relatively low energy intake (26 kcal/kg/day) (80).

Replacement therapy also produces a considerable loss of amino acids and/or protein with the dialysate (94), especially with high flux dialysers (95). This loss should be integrated by artificial nutrition, so an additional amount of protein or aminoacids (0.2 g/kg/day) is recommended (77). The use of haemodiafiltration makes it possible to achieve a positive nitrogen balance, even over a long-term period, by administering larger quantities of amino acids (96, 97). CAVH allow the provision of adequate nutrition support to critically ill patients with ARF (98). Higher amino acid intake (2.5 g/kg/day) improved nitrogen balance in comparison with lower intake (1.2 g/kg/day), while requiring more aggressive haemofiltration (99). Peritoneal dialysis, haemofiltration and haemodiafiltration also allow delivery of non-negligible amounts of glucose (100). Despite the fact that no optimal amino acid composition for artificial nutrition has been defined, NEAA should be administered together with EAA. The optimal ratio between the two has not yet been definitively established, but it should be higher than in currently available formulations (1:1): a value of 3 or 4:1 has been suggested (91). Some of the amino acids considered non-essential in normal subjects (arginine, tyrosine, cysteine, serine) may be considered 'conditionally essential' in renal failure and their provision may become indispensable (77). Pharmacokinetic studies on plasma and tissue concentration of the single amino acids in uremic subjects (101–105) suggest that nutrient formulae should contain each amino acid in different proportions from those used in non-uremic patients. The advantages of branched chain enriched amino acid mixtures have not been demonstrated (77, 81).

Infectious and metabolic complications of TPN are frequent and must be strictly monitored (76). An increased risk for arrhythmias related to central vein catheter was also described (106).

Fluid, electrolytes, vitamins

Because trace elements are excreted mainly by the kidney, their parenteral administration to ARF patients requires great care. It must, however, be remembered that zinc, manganese, copper, selenium and chromium can also be effectively eliminated in the gastroenteric tract. Among vitamins, vitamin A should probably be avoided because of the possibility of accumulation, as reported in chronic renal failure, and signs of toxicity should be carefully monitored. Vitamin C should not exceed 30–50 mg/day, because inappropriate supplementation may result in secondary oxalosis. Even though vitamin D from body storage may provide lasting protection against deficiency, its active renal metabolite

(1,25 OH-cholecalciferol) can be rapidly depleted, making repletion necessary. Vitamin K, E, B_6 and folate requirements are also increased in acute renal failure (40).

Recommendations for nutritional treatment of patients with acute renal failure

1. Artificial nutrition is indicated in patients with ARF whose spontaneous oral ingestion of nutrients is not adequate. The maximum duration of fasting or hypoalimentation tolerated by these patients can vary between 2–7 days, depending on the degree of catabolism, the previous nutritional status, the type of treatment (conservative or dialytic) and the severity of the prognosis.
2. The amino acid solutions to be delivered parenterally to ARF patients should contain both EAA and NEAA; the optimal ratio has not yet been established and can range from 2:1–4:1. If more than 0.4–0.5 g/kg/day are supplied, the addition of NEAA is mandatory. Composition of the amino acid mixture should be tailored to meet the specific metabolic requirements of uremia (histidine, taurine, tyrosine). Nitrogen amounts depend on the degree of catabolism, intercurrent illness, type of treatment (conservative or replacement) and residual renal function.
3. Calorie requirements in ARF depend more on the intercurrent events than on renal failure itself which does not increase energy expenditure. In most cases requirements are met by 1.3 times the basal energy expenditure. Lipid provision is useful, but the percentage should not exceed 20–25% of non-protein calories. Special problems may arise from the need to limit fluid and electrolyte intake. Care should be taken in administering vitamins A and C.

Suggestions for future research

1. Test if TPN regimens with EAA and NEAA, and with higher EAA proportions, especially BCAA, can be used more efficiently;
2. Test if adequate artificial nutrition can accelerate the recovery of renal function;
3. Demonstrate a true effect of (artificial) nutrition on: (a) survival; (b) clinical status; (c) rate of complications; (d) hypermetabolism; (e) nutritional status; and (f) recovery of renal function;
4. Test the effects of IGF-1 on recovery of renal function.

Nutrition in renal transplantation

Although renal transplantation, by restoring renal function, also generally restores the freedom to ingest a variety of foods and beverages, careful attention should still be paid to nutritional problems in this clinical condition. Distinct recommendations should be issued for the pre-transplant, early post-transplant and late post-transplant phases.

Pre-transplantation period

Nutritional monitoring and dietetic counselling are particularly required in transplant recipient candidates. Elderly, diabetic, obese, and young people must be particularly followed. If severe malnutrition is recognized, a period of artificial nutrition might be suggested. If uncontrolled diabetes, obesity, severe dyslipidaemia and/or hypertension are present, the patients are at risk of developing or rapidly worsening atherosclerosis. Careful correction of these risk factors by means of diet and drugs must be initiated before the patients are transplanted. Smoking adds a severe risk and should be stopped. A prudent program of weight control should be begun in obese patients.

Because of immunosuppressive therapy, bone disease can rapidly worsen in the post-transplant period: calcium, phosphate, parathyroid hormone, bone status and need for calcium salts and Vitamin D must be regularly assessed. Regular exercise is useful to ameliorate body composition, to lower cardiovascular risk and to improve bone status.

Early post-transplantation period

The surgical trauma during transplantation is generally mild and bowel function is rapidly restored. Enteral or parenteral artificial nutrition are not generally needed, as spontaneous oral nutrient intake is resumed. However, surgical trauma, short-term starvation, high-dose steroid therapy, pre-existing protein-energy malnutrition, and a variable delay in restoring kidney function make transplanted uremic patients prone to develop acute PEM in the early post-transplant period. Cushing syndrome appearance is minimized by high protein intake (107). Recovery from uremic PEM is slow, some authors found that 3 months after transplantation the muscle abnormalities (low protein, changed water content and amino acid concentration) were not corrected, possibly because of immunosuppressive therapy (108). Other authors found an altered water, electrolyte and protein pattern, typical of uremia, 13 months after transplantation, while after 9 years, muscle composition was normal despite chronic steroid therapy (109). Cyclosporine has a nitrogen sparing effect in comparison with high-dose steroid therapy (110).

In the early post-transplant period 30–35 kcal/kg/day is an adequate energy intake: it can be adjusted to corrected nitrogen balance to minimize the nitrogen losses. The protein intake should be in the high range to obtain a neutral nitrogen balance: 1.3–1.5 g/kg/day (up to 2.0 g in most catabolic patients [111]) must be

Table 1 Recommendations for nutritional treatment of adult patients with renal insufficiency

Clinical condition	Non-protein energy, kcal/kg/day*	Protein, g/kg/day
Hemodialysis	≥35	1.2–1.4
Peritoneal dialysis	≥35 (glucose absorptiom from dialysate can account for 25–30% of energy needs)	1.5–1.5 (>50% HBV)
Nephrotic syndrome	≥35	0.8–1.0
Acute renal failure		
Non oliguric, non hypercatabolic patients	In most patients: = 1.3 BEE	0.55–1.0
Hypercatabolic, dialysed Patients	≥ 1.3 BEE	1.0 to 1.5 (or more, see text). NEAA+EAA (ratio>1:1)
Renal transplantation		
Pre–op period	Correction of malnutrition	Correction of malnutrition
Early post–op period	30–35	1.3–1.5
Last post–op period	Adapted to maintain an ideal body weight	1.0

HBV: high biological value; EAA: essential amino acids; NEAA: non essential amino acids, BEE: Basal energy expenditure. *Adapted to individual needs in case of underweight or obesity

prescribed in patients, especially if treated with high doses of steroids. The lower range (1.3 g/kg/day) is suggested in patients with less catabolic combined therapy (cyclosporine + steroid).

Carbohydrates: diabetes frequently appears after renal transplantation as a result of corticosteroid therapy, and it can require dietary and pharmacological treatment (112). More often (about 70%) the diabetes disappears within one year after transplantation (112); 50% of energy as carbohydrates might be a prudent dietary approach to treat post-transplant hyperglycaemia (113).

Lipids: if increased glucose and/or triglyceride plasma concentrations suggest strictly limiting the carbohydrate intake, it might be difficult to limit the fat intake to 30% of total energy. However, a high (>1) polyunsaturated/saturated fatty acids ratio, with high oleic acid and limited alcohol consumption must be indicated since the very early post-transplant period.

The frequent hypophosphataemia that immediately follows renal transplantation suggests avoiding the oral phosphate binders and treating patients with adequate Vitamin D. The phosphorus content of the diet does not need to be limited.

Late post-transplantation period

The late phase of the post-transplant period can still be characterized by increased protein catabolism, and the muscle protein status can be impaired in this phase (109, 114). However, it has been reported that muscle metabolism and nutrition are normal 9 years after transplantation (109). The decline in renal function in these patients is probably prevented by a reduction in daily protein intake, chronic renal rejection being also related to non-immunological factors (115, 116). An amount of one gram of protein/kg/day is prescribed, the energy intake being adapted to the maintenance of desirable body weight.

One of the most severe metabolic risks in transplanted patients is hyperdyslipidaemia. The most common lipid pattern is characterized by high total cholesterol and high LDL cholesterol levels (with low HDL3 cholesterol and normal HDL cholesterol). Prednisone, renal dysfunction, proteinuria, cyclosporine, increased body weight and inappropriate food intake (117) are the most important factors responsible for dyslipidaemia. The guidelines of the National Cholesterol Education Program (118) can be a useful dietary approach to post-transplant dietary treatment. Besides dietary counselling, which is the first step in the treatment of dyslipidaemia, and drug therapy, regular physical exercise had been found to be a useful tool (119).

Recommendations for nutritional treatment of renal transplant recipients

1. Before transplantation, correction of obesity, diabetes, hypertension and dyslipidaemia must be initiated. If severe malnutrition is present, artificial nutrition might be suggested.
2. In the early post-transplant phase, the patients are at risk of acute PEM. The protein intake must be in the high range especially in the patients treated with high doses of steroids.
3. In the late post-transplant phase a lower amount of protein is suggested, the decline of renal function being possibly prevented by prudent daily protein intake.

Suggestions for future research

To determine the optimal protein intake to prevent decline of renal function and negative nitrogen balance.

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