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8 Nutrition in peritoneal dialysis

Guidelines

A. All patients should receive nutritional counselling based on an individualized plan of care.
(Evidence level C)

B. Nutritional status should be assessed every 6 months using a panel of measures.
(Evidence level C)

Serum albumin alone is not a clinically useful measure for protein/energy nutritional status in peritoneal dialysis patients.
(Evidence level B)

We recommend that subjective global assessment (SGA), protein intake (as assessed from the protein equivalent of total nitrogen appearance, nPNA, or by dietary recall) and an assessment of protein nutrition should be used.
(Evidence level C)

C. The target for dietary protein intake is generally regarded to be $\geq$1.2 g/kg/day; it should not be below 0.8 g/kg/day in any patient. On average, the achieved nPNA needs to be $\geq$1 g/kg/day, adjusted for patient's age and physical activity.
(Evidence level C)

D. In non-obese patients (BMI $<$27 kg/m^2), dietary energy intake should be 35 kcal/kg/day, adjusted for age and taking the energy derived from peritoneal glucose absorption into account. In malnourished patients, the energy and protein intake should be normalized to the desirable body weight.
(Evidence level C)

E. Metabolic acidosis (venous bicarbonate $<$25 mmol/l) should be avoided.
(Evidence level A)

F. When malnutrition develops in a PD patient, the adequacy of the dialysis prescription should be assessed, underdialysis should be excluded and other causes such as inflammation should be investigated.
(Evidence level C)

Commentary on Guideline 8: nutrition in peritoneal dialysis

Guideline A. Nutritional counselling

In general, nutritional counselling improves compliance with nutritional recommendations [1,2] and it is likely that this is also true for peritoneal dialysis (PD) patients.

Guideline B. Assessment of nutritional status

For PD patients, nutritional status should be routinely assessed using a panel of measures. Such panels have included Marckmann's nutritional index and Harty's nutritional index [1,3]. The frequency of using these measures has not been verified, but a 6 monthly review is desirable. Serum albumin, prealbumin, creatinine and creatinine index, dietary interviews and diaries, protein equivalent of nitrogen appearance (nPNA), subjective global assessment (SGA), anthropometry and dual-energy X-ray photon absorptiometry (DEXA) are all measures utilized to assess nutritional status, which will be detailed below, and their evidence for use will be substantiated. The importance of regular follow-up and acting on trends in the nutritional state cannot be overemphasized.

Subjective global assessment. SGA is a useful and reproducible instrument for assessing the nutritional status of PD patients [1,4–8]. It is inexpensive and it can be performed rapidly, and it gives a global score of protein-energy nutritional status. There are several different versions of SGA that have been applied in different studies. However, no systematic studies comparing different versions of SGA (or the different components included in SGA) have been reported. Therefore, we cannot recommend any particular version of SGA to be superior. One parameter in SGA that is likely to be important is the degree of anorexia, which has been reported to be a strong independent predictor of mortality in haemodialysis patients [9].

Serum albumin level. The serum albumin level, at the time of initiation of chronic dialysis therapy or during the course of dialysis therapy, is a predictor of future mortality risk [10–13]. However, one needs to bear in mind that serum albumin also reflects several non-nutritional factors which are frequently present in PD patients, including infection, inflammation, hydration status, peritoneal and urinary albumin losses, and acidaemia [14–16]. Hypoalbuminaemia in dialysis patient does not necessarily indicate protein-energy malnutrition. The patient's clinical status must be examined when evaluating changes in the serum albumin concentration, which is weakly and inversely correlated with serum acute phase proteins [17]. Similar arguments apply for prealbumin, a negative acute phase reactant which also may not correlate with changes in other nutritional parameters. In addition, the prealbumin level is related to residual renal function [16,18]. Therefore, there is insufficient evidence to conclude that prealbumin is a more sensitive or accurate index for malnutrition than serum albumin.

Protein equivalent of total nitrogen appearance. It is important to monitor protein intake, and the use of nPNA as an estimate of protein intake is well validated [19] and simple to use in the clinical setting. However, there are several important limitations to nPNA as an estimate of dietary protein intake (DPI). First, nPNA approximates DPI only when the patient is in nitrogen equilibrium or steady state [20]. It will change in anabolic or catabolic situations and in circumstances of marked variation in protein intake (e.g. in a diabetic patient with gastroparesis). nPNA may overestimate DPI when the protein intake is <1 g/kg/day, possibly due to protein catabolism. Normalizing PNA to body weight can be misleading in obese, malnourished and oedematous patients [21]. It is recommended that for individuals who are $<90\%$ or $>115\%$ of standardized body weight, the oedema-free adjusted body weight is used.

Anthropometry and hand-grip strength. The anthropometric parameters that are generally assessed include body weight, height, skeletal frame size, skin-fold thickness, midarm muscle circumference, percentage of the body mass that is fat, the percentage of usual body weight, the percentage of standard body weight and the body mass index (BMI) [22–24]. These various measures provide different information concerning body composition, and it is therefore advantageous to measure more than one of these parameters. Although the use of anthropometrics is an indirect and rather insensitive method with several errors (including sensitivity to hydration status), the estimation of lean body mass and fat mass agree reasonably well with results from DEXA [16]. As the use of anthropometrics is easy and cheap to apply, it can be recommended for routine assessment of nutritional status, bearing the limitations in mind. There has recently been an increased focus on functional tests [1], and hand-grip strength is a cheap and simple method that agrees reasonably well with other measures of nutritional status [16,25]. It has, furthermore, been demonstrated to predict outcome in PD patients [25], and may thus be recommended for routine follow-up of PD patients.

Dual-energy X-ray absorptiometry. This provides accurate data on body composition which are superior to anthropometry, creatinine kinetics and bioelectrical impedance [22,26,27]. Although the estimation of fat mass using DEXA is unaffected by abnormalities in hydration status that are common in PD patients [28,29], the estimation of lean body mass will be affected by the hydration status.

Creatinine kinetics. Creatinine kinetics have also been used to calculate lean body mass (LBM) in PD patients from creatinine excretion in the urine and dialysate [30]. However, the estimated LBM from creatinine kinetics is usually markedly lower than with other methods such as total body potassium [24,31]. Furthermore, LBM estimated from creatinine kinetics is dependent on the creatinine content in the diet and the metabolic degradation of creatinine, which is poorly understood in uraemia [31]. Finally, the variation in LBM with repeated measures of LBM using creatinine kinetics is unacceptably high [31]. Therefore, creatinine kinetics are not a good method for monitoring of LBM in PD patients.

Follow-up of a panel of nutritional markers. We recommend that body weight, SGA, protein intake (as assessed from nPNA or dietary recall) and some assessment of protein stores should be used for follow-up of the patients. There are several potential measures of protein nutrition that may be used for follow-up, but in particular DEXA, anthropometics or hand-grip strength seem to be of value [1,4–7,16,22–25]. As stated above, the frequency of using these measures has not been verified but a 6 monthly review is desirable. As nutritional status may differ greatly between patients due to differences in age, co-morbidity and clinical history, it is at present not possible to define targets for nutritional status. Instead the regular follow-up and acting on trends in the nutritional state cannot be overemphasized, although this approach has not been validated systematically. A prospective decline in the nutritional state should result in careful evaluation and treatment of the patient.

Guideline C. Dietary protein intake (DPI)

The recommended DPI for clinically stable PD patients is still controversial, but up to 1.3 g/kg/day has been advocated. This recommendation is based primarily on early studies [32,33], which were based on an analysis of relatively young active males on CAPD. These and others studies have shown that a DPI of 1.0–1.2 g/kg/day is associated with a neutral or positive nitrogen balance, with a relationship between protein intake and nutritional parameters [32–34].

However, none of these studies show that this DPI of >1.2 g/kg/day impacts on malnutrition or preserves nutritional status. Similar to the recommendations in healthy adults, the recommended intake of 1.2 g/kg/day is based on the assumption that this intake should be within the safe limit for 97.5% of the population (mean ± 2 SD). Still, many (but not all) patients have a stable nutritional status on a protein intake <1.2 g/kg/day. Nitrogen balance studies [32–34] indicate that a protein intake of ≥1.0 g/kg body weight/day is enough in most patients, and in a nitrogen balance study of CAPD patients receiving an individualized diet composed to resemble the patients' spontaneously chosen energy and protein intake at home, some of the patients were in a neutral or positive nitrogen balance with a protein intake as low as 0.7 g/kg body weight/day [34]. In addition, an intake of ≥1.2 g/kg body weight/day cannot be achieved by the vast majority of PD patients in spite of relatively stable nutritional status [7,35–37]. Therefore, we consider a protein intake of ≥1.0 g/kg body weight/day as acceptable if the patient has no declining trend in nutritional status. However, when DPI (or nPNA in steady state) is <0.8 g/kg/day, there is a clear need to reassess the patient.

Guideline D. Dietary energy intake

Dietary energy intake for adult PD patients should be 35 kcal/kg/day, adjusted for age. There are supporting data that the energy requirements are systematically different in PD patients compared with the general population, and it is generally considered that the energy requirements are lower in older patients, and a caloric intake of 30 kcal/kg body weight/day may be recommended in patients with an age above 60 years. There are no data that this calorie intake will reduce morbidity or mortality or improve the nutritional state in malnourished patients. The rationale is based on increased mortality rates in low body weight patients [38], and aggressive energy intake may correct this. In the PD patients, this recommended intake includes both diet and the energy intake derived from peritoneal glucose absorption. The glucose absorption from the dialysate may be calculated easily (if the glucose concentration in the drained 24 h dialysate is measured) by using the equation: glucose absorbed (mmol) = glucose concentration in infused dialysate (mmol/l) × infused dialysate volume (l) − glucose concentration in drained dialysate (mmol/l) × drained dialysate volume (l). In CAPD patients with normal peritoneal transport capacity, ~60% of the daily dialysate glucose load is absorbed, resulting in a glucose absorption of ~100–200 g glucose/24 h [39].

Guideline E. Metabolic acidosis

Metabolic acidosis is an important stimulus for net protein catabolism [40] and elicits the transcription of genes for proteolytic enzymes in muscle including the ubiquitin–proteasome pathway [41,42]. In non-dialysed chronic uraemic patients, the correction of metabolic acidosis decreases protein degradation [43] and improves nitrogen balance [44].

CAPD patients with metabolic acidosis were reported to be more malnourished compared with other patients using a composite nutritional score [45]. The buffer concentration in the dialysate (lactate 40 *vs* 35 mmol/l, bicarbonate/lactate 25/15 *vs* 25/10 mmol/l, bicarbonate 39 *vs* 34 mmol/l) needs to be individualized, and many patients will need the higher concentrations to avoid acidosis [46–49]. In PD patients using the lower buffer concentrations, acid–base status may be improved by a switch to the higher dialysate buffer concentrations, and oral bicarbonate supplementation may also be used to improve the acid–base status.

Correction of acidosis (from an HCO_3 level of 19 to 26 mmol/l) with oral sodium bicarbonate in seven CAPD patients resulted in an improvement in protein turnover with decreased body protein degradation [50]. Also, improvement in the acid–base status in CAPD patients has been shown to result in increased body weight and plasma branched chain amino acids, and reduced muscle levels of ubiquitin mRNA [51]. Correction of acidosis with oral sodium bicarbonate for 2 weeks in 11 PD patients showed that urea appearance decreased significantly whereas there was no significant change in protein intake, indicating that the bicarbonate supplementation may have induced net anabolism [52]. Two hundred consecutive new CAPD patients were randomized, in a single-blind fashion, to receive a high (lactate 40 mmol/l) or low (lactate 35 mmol/l) alkali dialysate for 1 year [53]. Calcium carbonate and sodium bicarbonate were also used to correct acidosis in the high alkali dialysate group. At 1 year, the mean venous serum bicarbonate was 27.2 mmol/l in the high alkali dialysate group, and 23.0 mmol/l in the low alkali dialysate group ($P < 0.001$). It was concluded that better correction of metabolic acidosis leads to greater increases in body weight and midarm circumference, but not in triceps skinfold thickness, in the first year of CAPD. There were also fewer hospital admissions and hospitalization days in the high alkali dialysate group. The improvement in morbidity, in terms of number of admissions and days in hospital per year, may be associated with the improvement in the nutritional state.

A randomized placebo-controlled, double-blind trial of oral sodium bicarbonate supplementation in 60 CAPD patients (30 in each group) with mild acidosis (plasma bicarbonate <25 mmol/l, on average 22.9 mmol/l) and a Kt/V <2.1 demonstrated that the patients randomized to oral bicarbonate (2.7 g/day) showed improved bicarbonate levels, a better nutritional status as regards SGA and nPNA, and had shorter hospitalization compared with the control group [54].

Thus, full correction of acidosis is an obvious goal for treatment in CAPD patients, and oral bicarbonate should be prescribed even when the blood standard bicarbonate level is only marginally decreased.

Guideline F. Dialysis dose

There is much controversy about the relationship of delivered dose and nutritional status [55]. This is based on a cross-sectional correlation between Kt/V and nPCR, which is mathematically coupled. Overall, prospective studies of adequacy and nPNA or DPI have not shown any correlation. There is little direct evidence that increasing dialysis dose improves nutritional status, except from one small prospective study which suggested that nutritional parameters (nPNA and albumin) may improve with increased dialysis dose in malnourished patients without co-morbidity [56]. Another study in Asian patients showed increased protein intake in anuric patients by increasing the dose of CAPD from three to four daily exchanges [57,58], but not when the dose was increased from four to five exchanges per day [57]. Furthermore, the large randomized ADEMEX study ($n = 965$) of increased dialysis dose did not show any clear improvements in nutritional parameters (nPNA, body weight, plasma albumin, prealbumin and transferrin) [35]. However, these patients in general did not suffer from marked malnutrition. In patients who have no residual renal function or a declining residual renal function, there is a concomitant decrease in DPI. Residual renal function may therefore be an important parameter for nutritional intake in PD patients.

Several categories of patients that might require additional nutritional support include severely malnourished patients with complications, such as diabetic patients with gastroparesis, or hospitalized dialysis patients who often ingest even lower amounts of protein and energy [56]. Nutritional support can be given by oral supplements [59] of fortified energy and protein, intraperitoneal amino acids, nasogastric feeding or parenteral nutrition. It may also be appropriate to have nutritional support during severe episodes of peritonitis, e.g. with intraperitoneal amino acids. It has also become increasingly evident that malnutrition in patients with chronic renal failure, apart from that due to inadequate food intake, is also often related to increased protein catabolism induced by chronic inflammation [60–62]. This second type of malnutrition should preferably be denoted 'wasting' [62,63], and usually responds poorly to increased food intake. In such cases, it is important also to look for, and treat the causes of inflammation, including optimal treatment of co-morbidity.

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Appendix

Calculation of PNA in peritoneal dialysis patients

The formulae for calculation of PNA (PCR) in chronic PD patients have been validated for CAPD. However, they are generally applied to all chronic PD patients. In chronic PD patients, the following formulae apply using total urea appearance and protein loss in dialysate and urine [19]:

$$\text{PNA (PCR)} = 15.1 + 0.195\ \text{urea appearance (mmol/24 h)} + \text{protein losses (g/24 h)}$$

The 24 h urea excretion by peritoneal dialysate and residual renal urea excretion is measured from a 24 h collection of dialysate and urine. Preferably, protein losses are also measured.

If protein losses are not assessed, a simplified equation can be used:

$$\text{PNA (PCR)} = 20.1 + 0.209\ \text{urea appearance (mmol/24 h)}$$

Note that the change in total body urea nitrogen in chronic PD patients is negligible and is not included in these equations. Other equations have also been used and give relatively similar results [1,19].

Normalization of PNA in peritoneal dialysis patients

The PNA should be normalized or adjusted to specific body size. The most common normalization and the one recommended by the DOQI Haemodialysis Work Group is to normalize to the distribution volume of urea (V) divided by 0.58.

$$\text{nPNA(nPCR)(g/kg/day)} = (\text{PNA})/(\text{V}/0.58)$$

However, this equation has not been validated for PD patients and will introduce further errors in the estimation of nPNA as V is usually not directly determined, but calculated from the Watson equation.

There are no data to support other normalization techniques, but normalization to aBW_{ef} (where aBW_{ef} is the actual oedema-free body weight) may be the preferred normalization technique if the patient has a relatively normal BMI ($19 < \text{BMI} < 27$). The DOQI Nutrition Work Group recommends the use of the following normalization formula [1]:

$$\text{nPNA} = (\text{PNA})\text{aBW}_{ef}$$

However, in malnourished and in obese patients, the PNA should not be normalized using aBW. We suggest that in these patients PNA should be normalized to desirable body weight.