

Module 15.4.

Nutrition support in peritoneal dialysis patients

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Learning Objectives:

- To know the causes of protein energy wasting in patients on peritoneal dialysis (PD);
- To learn how to evaluate nutritional status in PD patients;
- To learn the nutritional requirements in PD patients;
- To know the different techniques of nutritional support in PD patients;
- To learn the best approach to nutritional support in malnourished PD patients.

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Key Messages:

- Protein energy wasting (PEW) is found in approximately 30-40% of patients on peritoneal dialysis (PD);
- PD patients with PEW have a compromised prognosis;
- Many causes of PEW in PD patients are similar to those in haemodialysis (HD) patients. These include insufficient food intake, abnormal nutrient metabolism, inflammation, and hormone abnormalities;
- Specific factors due to PD additionally affect the nutritional status of PD patients. These are a lower eating drive, important protein losses through the peritoneal effluents, an increased energy expenditure in PD patients with no residual renal function, and finally the extra glucose load from PD solutions;
- Compared to HD patients, the body composition in PD patients is characterized by fluid overload and gain of fat mass. Loss of lean body does not appear different from that observed in HD patients;
- PEW can be detected by a decrease in body mass index, a body weight or muscle mass loss, and low serum albumin and prealbumin;
- Nutritional support, preferably oral nutritional supplements (ONS) or, in the absence of compliance, amino acid-based intra-peritoneal parenteral nutrition (AA-IPP), is able to improve nutritional status;
- In PD patients at high risk of PEW, as defined by insufficient spontaneous intake, no residual renal function and inflammation, intensified dietary counselling, and, if necessary, ONS or AA-IPP are worth trying initially;
- In PD patients with established PEW with spontaneous intakes more than 20 kcal/day, dietary counselling and ONS/AA-IPP should be first prescribed; but enteral nutrition (EN) may be necessary if the above measures do not improve the nutritional status;
- In patients with established PEW, with spontaneous intakes less than 20 kcal/day, or in stress conditions, daily nutritional support is necessary. EN is preferred to intravenous parenteral nutrition, provided that the gut is functional.

1. Introduction

Protein-energy wasting (PEW), formerly called “malnutrition”, is a precise nutritional status, defined by a loss of protein and body mass, often accompanied by decreased functional capacity, which is prevalent in dialysis patients (1). Peritoneal dialysis (PD) is a well established treatment modality in patients with chronic kidney disease stage 5 (CKD 5), as an alternative to haemodialysis (HD). The incidence and prevalence of PEW among patients undergoing PD are high (2). In a cross-sectional study, its prevalence was 42%, versus 32% in patients treated by haemodialysis (HD) (3). In particular, serum total protein and albumin tended to be lower, midarm muscle circumferences were similar and relative body weight, skinfold thickness, and the estimated percentage of body fat tended to be greater in PD patients (3). Differences in nutritional status between PD and HD patients may be explained by specific factors associated with PD treatment. These include fluid overload, protein losses in PD effluents and the absorption of glucose from PD solutions across the peritoneal membrane. In addition, loss of residual renal function is associated with an increased risk of PEW in PD patients (4). The addition of peritoneal glucose energy does not prevent some PD patients from remaining in negative energy balance because of poor nutritional intake (4), thus emphasizing the need to monitor the nutrition status of these patients closely. On the other hand, many PD patients develop an increase in body weight, body fat and truncal fat mass, especially during the first year after starting a PD programme (5). Polymorphisms in the uncoupling protein 2 gene appear to explain differences between PD patients who are gaining and those who are not gaining body fat (5). In spite of important protein losses in the dialyzates, muscle wasting does not appear to be more pronounced in PD than in HD patients (6). Several nutritional parameters in PD patients correlate with increased morbidity and mortality, which is consistent with observations in patients treated by HD. Low serum albumin (2), reduced urea nitrogen appearance (7), decreased lean body mass (8), low serum creatinine (9), low handgrip strength (10), and low nutrition scores as measured by the subjective global assessment (SGA) (8), are all associated with high morbidity and mortality in this setting. Unfortunately, whether any improvement in the above parameters, as a result of nutritional support, has a positive impact on the outcome of PD patients is unknown. To address this question would need large multicentre trials, which are unlikely to be performed in this population. Since no such data are available, data obtained in other conditions and clinical experience are relied upon to suggest that nutritional status in PD patients should be assessed regularly and nutritional support begun if PEW is detected. The purpose of this paper is to discuss the main issues related to nutritional status and nutrition support in PD patients.

2. Specific factors inducing protein energy wasting in PD

Insufficient food intake, abnormal nutrient metabolism, inflammation, and hormone abnormalities are the main causes of PEW in patients undergoing either HD or PD (11). However, the following factors specific to PD also affect nutritional status in PD patients.

2.1. Eating behaviour and appetite

Clinical experience suggests that PD patients are more likely to report early satiety or feelings of fullness. A study which investigated eating behaviour in dialysis patients indicates that those undergoing PD have a lower food intake and a poorer appetite compared to HD patients (12). Impaired gastric emptying may explain this abnormality since the instillation of dialysate into the peritoneal cavity may contribute to abnormal gastric electrical activity (13) as well as to prolongation of gastric emptying time (14). A number of explanations have been forwarded to explain the suppression of appetite in PD patients. The abdominal distension, produced either by intraperitoneal instillation of large volumes of dialyzate or the large ultrafiltration induced by hyperosmolar PD solutions, (as shown in rabbits (15)), contributes. The nutrient-specific role of PD fluids does

likewise, as demonstrated in a rat model of PD: the instillation of glucose-based dialysate resulted in a dose-dependent suppression of carbohydrate intake only, whereas the instillation of an amino acid-based dialysate produced a dose-dependent suppression of both carbohydrate and protein intake (16). This latter observation may be relevant because a variety of PD fluids including glucose-based, amino acid-based and glucose polymer-based solutions are available commercially. However, whether these experimental findings in animals are reproducible in PD patients is unknown. The inadequate total urea and creatinine clearance secondary to loss of residual kidney function is an important contributing factor for anorexia in PD patients. This is evident from the independent association between residual kidney function, but not PD clearance, with dietary energy and protein intake as well as the intake of micronutrients in PD patients (4, 17). These data clearly indicate the qualitative difference between residual kidney function and PD clearance. It therefore follows that anuric PD patients are clinically found to be more wasted by different nutrition parameters including handgrip strength, serum albumin and subjective global assessment (SGA), as compared to patients with preserved residual kidney function (18). Inflammation is associated with reduced dietary energy and protein intake in PD patients (19), thus representing another contributing factor to anorexia in PD patients. Markedly elevated serum leptin, which is common in PD patients, may represent an additional specific factor which may suppress appetite (20). These observations in PD patients are in line with experimental evidence showing that the control of energy homeostasis is centrally regulated in the hypothalamus by peripheral circulating hormones and inflammatory cytokines, of which leptin appears to play a key role in appetite regulation (21). Finally, cardiovascular disease including circulatory congestion may also contribute to anorexia and reduced dietary intake in PD patients (22).

2.2. Gastrointestinal symptoms

Gastrointestinal symptoms such as abdominal pain, constipation, diarrhoea, and stool urgency are more frequent in dialysis patients than in the general population (23). However, PD patients appear to have a similar prevalence of gastrointestinal symptoms to those of HD patients in one cross-sectional comparative study (23).

2.3. Protein losses

Therapy-associated losses of protein during PD are much higher than in HD, as are losses of protein-bound substances, such as trace elements. They approximate to 10 g/day, with losses of free amino acid losses amounting to 3-4 g/day, 30% being essential amino acids (24). Protein losses may increase dramatically to between 15 and 100 g/day in severe peritonitis (25). Protein losses from PD effluents mainly include albumin, and immunoglobulins, whereas transferrin and β 2-microglobulin are lost in small amounts. In patients undergoing automated PD (APD), comparable or slightly higher 24-hour dialysate protein losses are observed compared with those reported in continuous ambulatory peritoneal dialysis (CAPD) patients (26).

2.4. Glucose absorption through the peritoneal membrane

Because peritoneal solutions with a high glucose content are standard, PD treatment is associated with a glucose uptake of 100 to 200 g/day (24), which is further increased during peritonitis. Glucose absorption can be estimated in terms of calories absorbed. However, this amount of energy can vary considerably from individual to individual, depending on peritoneal membrane transport characteristics and dwell times of PD solutions. The average peritoneal glucose energy intake is about 8 kcal/kg/day, leading to extra energy provision of 300 to 750 kcal/day (27, 28). The glucose absorbed accounts for 12 to 34% (mean 20%) of total energy intake (25). Consequently, PD patients, although having a low spontaneous energy intake of about 23-24 kcal/kg/day (29), receive a relatively high total energy intake of 29 to 33 kcal/kg/day (30, 31). This

compares favourably with the total energy intake observed in HD patients which varies between 22 and 29 kcal/kg/day (32).

The high glucose load may contribute to hyperinsulinaemia, insulin resistance, induction or aggravation of diabetes, hypertriglyceridaemia which is observed in 60% of PD patients (24), and increased LDL and VLDL cholesterol.

3. Energy expenditure in PD patients

Some studies suggested that the resting energy expenditure (REE) in end-stage renal disease (ESRD) patients was no different from, or even lower, than in healthy controls (33, 34). In contrast, one study showed that HD patients had increased REE, which further increased during HD (35). In PD patients, REE may be normal or elevated and has been associated with an increased mortality and specifically with cardiovascular deaths (36). Several factors may contribute to an elevated REE in PD patients, one of which is loss of residual renal function. Even though the diseased kidneys may lose their excretory function, they remain metabolically very active and make a significant contribution to the REE of PD patients. Resting hypermetabolism together with reduced dietary energy and protein intake may contribute jointly to the more severe PEW seen in anuric PD patients than in those with preserved residual renal function (37). Inflammation and the presence of cardiovascular disease are other important factors associated with an elevated REE in PD patients. PD patients with previous circulatory congestion are indeed more inflamed, as evident by their higher C-reactive protein and lower serum albumin, than PD patients with no circulatory congestion. These patients also have significantly lower dietary energy and protein intake and higher REE, thus resulting in worse negative energy balance and more PEW among those with circulatory congestion (22). Finally, there is also some suggestion that severe secondary hyperparathyroidism may contribute to an increased REE in HD patients, and this is likely to be true also for PD patients (38).

4. Body composition analysis in PD patients

Clinical observations suggest that overhydration is a common finding in PD patients. In addition, the enhanced loss of proteins in PD increases the risk of protein wasting. However, with the extra glucose load, body weight may increase over time, due to increased body fat mass. The body weight increases between 1 and 5 kg during the first year of PD treatment, and tends to stabilize (or slightly increases) thereafter (39-41).

Precise body composition studies in PD patients are rare, but they appear to corroborate clinical findings. Total body water as assessed by deuterium oxide dilution is in fact no higher than in controls (40). However, the ratio extracellular/intracellular water, using bromide dilution to measure the extracellular water compartment, is increased (40). Body cell mass, assessed by total body potassium count, decreases over time during PD, whereas body fat may increase in parallel (41). Hence, body composition in PD patients is characterised by a relative fluid overload, high fat mass, low fat-free mass and low serum albumin and prealbumin.

5. Nutritional assessment in PD patients

Serum albumin, prealbumin, creatinine and creatinine index, dietary protein intake (DPI) dietary energy intake (DEI), obtained by interviews and diaries, protein equivalent of nitrogen appearance (nPNA), subjective global assessment (SGA), anthropometry, handgrip strength and dual-energy X-ray photon absorptiometry (DEXA) are all measures used to assess nutritional status in dialysis patients including those on PD treatment.

5.1. Serum albumin and prealbumin

There is substantial evidence that serum albumin is a powerful biochemical predictor of clinical outcomes in dialysis patients, including PD patients (2). Low serum albumin often indicates PEW in PD patients (11). However, hypoalbuminaemia in these patients may also reflect confounding factors which are frequently present in PD patients, including infection, inflammation and overhydration (42). Similar limitations also apply for serum prealbumin, which is a negative acute phase reactant like albumin. In addition, the prealbumin level is related to residual renal function (43). In spite of these limitations, serum albumin and prealbumin still remain useful makers in the assessment of nutritional status in PD patients.

5.2. Subjective global assessment

SGA is a useful and reproducible instrument for assessing the nutritional status of PD patients (2, 8, 44). It is inexpensive, can be performed rapidly, and reflects 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 the SGA) have been reported. Therefore, no particular version of SGA can be said to be superior to another.

5.3. Dietary protein intake and protein equivalent of nitrogen appearance

nPNA which can be calculated from urea concentrations in blood and dialysates, is an estimate of DPI. It is well validated and simple to use in the clinical setting (45). However, there are several important limitations of nPNA as an estimate of DPI. First, nPNA approximates DPI only when the patient is in nitrogen equilibrium or steady state. 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). Second, nPNA may overestimate DPI when the protein intake is <1 g/kg/day, possibly due to protein catabolism. Finally, normalizing PNA to body weight can be misleading in obese, malnourished and oedematous patients (46). It is thus recommended that, for individuals who are <90% or >115% of standardized body weight, the oedema-free adjusted body weight is used to normalize PNA.

5.4. Anthropometric measurements

The anthropometric parameters that are generally assessed include body weight, height, skin-fold thickness, midarm muscle circumference, percentages of body mass components, the percentage of usual body weight, the percentage of standard body weight, and the body mass index (BMI). These various measures provide different pieces of information concerning body composition and it is advantageous to measure more than one of them. The use of anthropometrics is easy and cheap to apply. However, this is an indirect and rather insensitive method with several errors, including sensitivity to hydration status and operator-dependency. These measures may be suggested for routine bedside assessment of nutritional status, bearing these limitations in mind (47).

5.5. Hand grip strength

There has recently been an increased focus on functional tests. Hand-grip strength is a cheap and simple method that agrees reasonably well with other measures of nutritional status and especially with body lean muscle mass. It has been demonstrated to predict outcomes in PD patients and may thus be recommended for their routine follow-up (10).

5.6. Dual-energy X-ray absorptiometry

DEXA provides accurate data on body composition which are superior to anthropometry, creatinine kinetics and bioelectrical impedance (48), and is considered the gold standard test in assessing body composition. Although the estimation of fat mass using DEXA is unaffected by the abnormalities in hydration status that are common in PD patients, the estimation of lean body mass may nonetheless be affected by the hydration status.

5.7. Recommendations for nutrition status monitoring

It is recommended that nutritional status should be assessed at least every 6 months using a panel of measures. Regular follow-up is needed in order to identify significant changes or trends in nutritional parameters, which may require nutritional interventions and investigation of causes of PEW (47). **Table 1** summarizes the panel of nutritional parameters in PD patients and the proposed intervals of measurements for each of them.

Table 1. Proposed monitoring schedule for nutritional parameters in PD patients

Nutritional parameter	Interval (months)
Dietary interview	6 - 12
BMI	1
nPNA	1
Midweek predialysis creatinine	1
Serum albumin	1 - 3
Serum prealbumin	1 - 3

6. Nutritional requirements in PD patients

6.1. Energy requirements

Energy intake in PD patients represents the sum of dietary intake and the absorption of glucose from the dialysate. Metabolic balance studies of patients on chronic ambulatory peritoneal dialysis (CAPD), eating their usual diets, showed a strong correlation between total energy intake and nitrogen balance, irrespective of the duration of dialysis (28). A few studies have addressed the energy requirements of PD patients (28, 46). Based upon these studies, international panels of experts recommend a total (oral + peritoneal) energy intake of 30 to 35 kcal/kg/day in PD patients, as shown in **Table 2** (47, 49-51). Some guidelines take into account the patient's age, since physical activity, and thus active energy expenditure, is known to decrease with age. It is important to adapt energy requirements to physical activity, which is low in dialysed patients (52). A recent study indeed showed that the vast majority of PD patients are sedentary or have little physical activity, as assessed with a pedometer (53).

Table 2. Recommendations for protein and energy supply in adult PD patients

	ESPEN (49)	NKF-KDOQI (50)	EBPG (47)	ISRNM (51)
Protein intake, g/kg/day	1.2-1.5 (> 50 % HBV*)	1.2-1.3 (> 50 % HBV*)	1.3	> 1.2 Peritonitis: > 1.5
Energy intake, kcal/kg/day	35**	< 60 y. 35** > 60 y. 30-35**	< 60 y. 35** > 60 y. 30**	30-35**

* HBV: High biological value

**Energy intake including energy supply (glucose) from PD fluids

ESPEN: European Society for Clinical Nutrition and Metabolism

NKF: National Kidney Foundation

EBPG: European Best Practice Guidelines

ISRNM: International Society of Renal Nutrition and Metabolism

6.2. Protein requirements

Three publications report nitrogen balance studies conducted in PD patients (28, 54, 55). They demonstrated that a positive or neutral nitrogen balance was obtained in relatively young patients taking 1.2 g/kg/day of protein. Based upon these studies, international panels of experts recommend protein intakes between 1.2 to 1.5 g/kg/day (**Table 2**). ESPEN suggests the use of at least 50% as high biological value protein (50). The ISRNM suggests increasing protein intakes to more than 1.5 g/kg/day in PD patients with peritonitis (47, 49-51).

It is noteworthy that in both HD and PD, metabolic balance studies have investigated a very small number of stable dialysis patients, over a short period. Whether lower energy and protein requirements are sufficient to maintain a positive or neutral nitrogen balance in some patients, for instance elderly patients or patients with little physical activity, requires further determination. In the only study which investigated this question, Uribarri *et al* demonstrated, in 49 PD patients, that body weight, oedema-free/fat-free mass and anthropometric parameters could be maintained over a 6-month period with a total energy intake of 29 kcal/kg/day and a protein intake of 1 g/kg/day (56). However, these PD patients were relatively obese for their height, and when adjusted for the patients' overweight condition, energy intake rose to recommended levels.

6.3. Mineral / Vitamins requirements

Studies investigating the mineral and vitamin requirements in PD patients are not available. Recommendations are similar to those for patients treated by HD. Losses of water-soluble vitamins through PD effluents suggests the replacement of pyridoxine (10 mg) and vitamin C (100 mg) (**Table 3**).

Table 3. Mineral/Vitamin requirements of patients on PD

Phosphate, mg/d*	800 – 1000*
Potassium, mg/g	2000 – 2500*
Sodium, g/d	1.8 – 2.5*
Fluid, ml	1000 + urine volume
Pyridoxine, mg	10
Vitamin C, mg	100

* individual requirements may differ in acute conditions

7. Methods for nutritional support

Nutritional support in PD patients includes nutritional counselling, oral nutritional supplements (ONS), amino acid-based intraperitoneal parenteral nutrition (AA-IPPN), intravenous parenteral nutrition (IVPN) and enteral nutrition (EN) with tube feeding.

7.1. Nutritional counselling

In patients treated by HD, nutritional counselling improves compliance with nutritional recommendations. It is likely that this is also true for PD patients.

7.2 Oral nutritional supplements

Data regarding the use of ONS in PD patients are limited. However, at least 10 studies, of which admittedly only 4 were randomized (**Table 4**) have examined the effect of nutritional support on nutritional status in PD patients with PEW (57-60). Although these studies showed mixed results, they report that ONS may improve nutritional status considerably if patients are compliant. On the other hand, intolerance or non-adherence to ONS was observed in over 50% of PD patients in those studies, leading to a high drop-out rate. This is probably why no effect could be demonstrated in other studies, in particular in the already underpowered trials. Finally, studies which have used high biological value supplements, such as calcium caseinate or egg albumin-based supplements, appeared to show greater benefits than studies using standard ONS. Taken together, available data suggest that PEW could be improved by ONS in adherent PD patients.

Table 4. Randomized trials of nutritional support in PD patients

Authors	N	Type of patients	Design	Months	Nutritional significant effects
Eustace et al. (2000) (57)	47	HD/PD	RCT: Essential aminoacids (3.6 g with meals 3 times daily) vs placebo	3	↑ albumin only in HD, not in PD
Aguirre Galindo et al. (2003) (58)	100	PD	RCT: 100% natural protein vs 50% calcium caseinate + 50% natural protein. All patients receiving 35 kcal/kg/d and 1.4 g prot/kg/d		↑ albumin in both groups, more pronounced with calcium caseinate
Gonzalez-Espinoza et al. (2005) (59)	28	PD	RCT: egg albumin-based ONS vs control group	6	↑ albumin in interventional group, ↑ DPI, DEI and nPNA in interventional group
Moretti et al. (2000) (60)	49	HD/PD	Crossover controlled trial: standard ONS vs control group	12	↑ albumin and nPNA in interventional group ↓ albumin and nPNA in control group

7.3. Amino acid-based intraperitoneal parenteral nutrition

AA-IPPN consists mainly in the intraperitoneal administration of a 1.1% amino acid-based solution (61). Metabolic studies during intravenous infusion of ($^2\text{H}_3$) and intraperitoneal leucine (^{13}C) showed that intraperitoneal amino acids were incorporated in protein synthesis (62). The analysis of 11 studies of intraperitoneal AA infusions, including 4 randomized trials, showed an improvement in nitrogen balance (19), and the fasting plasma amino acid pattern (63). However, nutritional parameters were improved in only some and not all cohort series (19). In a 3-year, randomized, prospective, controlled study, 60 CAPD patients were assigned randomly either to replace one exchange daily with AA dialysate or to continue with dextrose dialysates. Dietary protein intake increased in the AA group. Biochemical nutritional parameters including nPNA, albumin and cholesterol decreased in the dextrose group but remained stable or increased in the AA group. The nutritional benefit appeared more prominent in women, whose lean body mass and body mass index were maintained in the AA group but not in the dextrose group (64). It should be noted that the degree of malnutrition in patients recruited to this study was generally mild. Whether an AA dialysate has similar benefit in moderate to severe PEW is not known.

In APD patients, data regarding this issue are very limited. A random-order short-term cross-over study in 8 APD patients showed that the use of AA plus dextrose in dialysates improved rates of protein synthesis (L-[1- ^{13}C] leucine) and 24-h net protein balance, when compared to the use of dextrose alone (65).

AA-IPPN may be associated with hypokalaemia and hypophosphataemia (61). Some patients may also develop mild acidosis (66). These possible side effects underline the need for close monitoring during this treatment.

Based upon this evidence, we suggest that AA-IPPN is considered in PD patients with evidence of PEW, when nutritional requirements cannot be ensured by the oral or enteral routes.

7.4. Intravenous parenteral nutrition

IVPN has been poorly investigated in PD patients. ESPEN recommends a combination of carbohydrate and fat to address the energy supply (67). The use of specific formulae for parenteral nutrition are not yet supported by controlled data in this setting. Present data suggest that intravenous IVPN should be limited to undernourished and stressed PD patients, or to patients with severe encapsulating peritonitis (67, 68), when nutritional requirements cannot be ensured by oral or enteral routes.

7.5. Tube feeding

In paediatric nephrology, small infants on PD are routinely tube fed. However, information regarding enteral nutrition (EN) by tube feeding in adult PD patients is not available. Clinical experience and common sense suggest that tube feeding may be considered in two situations: a) if AA-based IPPN or ONS are insufficient to cover nutritional needs and b) in catabolic acute conditions in which neither oral intake nor AA-based IPPN are possible. Although used in children treated by PD, percutaneous endoscopic gastrostomy or jejunostomy (PEG/PEJ) are not recommended in adult PD patients due to the risk of peritonitis (67).

8. Impact of nutrition support on outcome in PD patients

Studies to determine whether morbidity and mortality of PD patients may improve with these interventions are still needed and would require large multi-centre trials. ONS and AA-IPPN, may improve nutritional parameters in malnourished PD patients, but their potential benefit in terms of improved patient survival is not known since no studies were adequately powered to address mortality in these patients. In HD patients, recent data have shown the effectiveness of nutritional support in reducing morbidity and mortality in malnourished patients in whom serum prealbumin improved (69). Although the same results could be expected in PD patients, no prospective data have demonstrated the effect of nutrition support on outcome in this population. Until these data become available, nutritional status must be assessed regularly in PD patients so as to detect PEW promptly and to secure early nutritional intervention.

9. Decision tree for nutritional follow-up and support in PD patients

9.1. Non-acutely ill PD patients with PEW

A proposed decision tree is pictured in **Figure 1**. In PD patients with mild PEW or considered at risk of PEW, as defined by insufficient spontaneous food intake, inflammation, anuria and/or cardiac congestion, intensified dietary counselling must be given. At the same time all possible causes of PEW must be addressed and corrected if possible. This includes for instance the correction of metabolic acidosis and the eradication of sources of infection/inflammation. If this is not sufficient to cover nutritional requirements, ONS should be prescribed and can usually be relied upon, provided that the patient is compliant. If adherence to ONS is not obtained, the use of AA-based IPPN may be an alternative to cover protein needs and to improve nutritional parameters.

In patients with established PEW but spontaneous intakes of more than 20 kcal/day, both dietary counselling and ONS should be readily advocated; EN may be necessary if the above measures are unable to improve nutritional status.

In patients with established PEW and spontaneous intakes less than 20 kcal/day, or in stress conditions, daily nutritional support is necessary. EN is preferred to IVPN. However, IVPN is indicated when EN is impossible or insufficient.

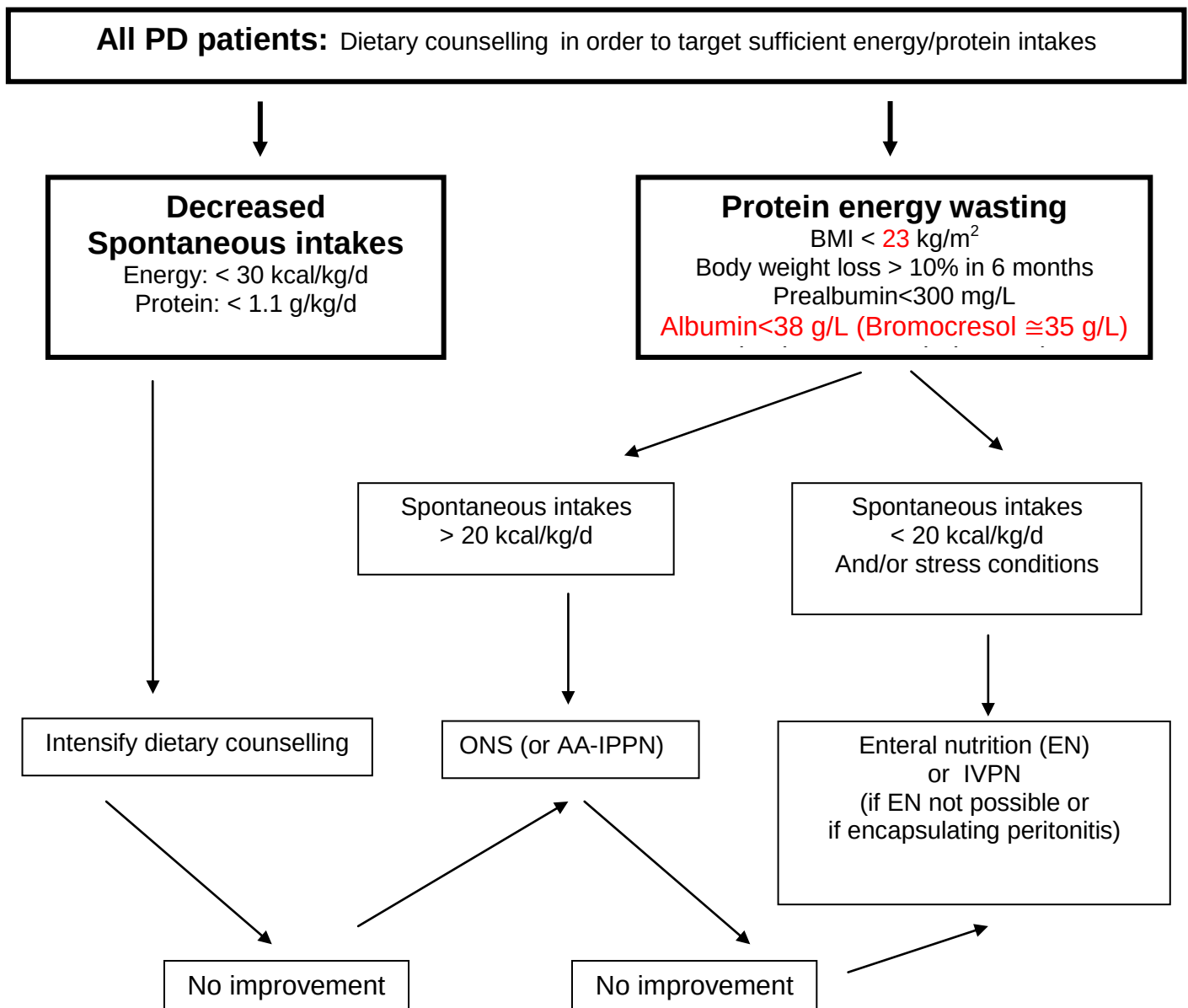


Fig. 1. Proposed algorithm to prevent and/or to treat PEW in PD patients

9.2. Acutely ill PD patients with PEW

The decision to use IVPN in acutely ill and/or stressed PD patients depends on gut function. If EN is not feasible, IVPN should be administrated to cover nutritional requirements and should be adapted to stress conditions, as in acute kidney injury (67).

10. Perspectives to improve nutritional management in PD patients

It is likely that a multi-modal approach to PEW in PD patients, combining nutritional support with exercise and anabolic hormones may improve nutritional status to a greater extent than nutrition support alone, but this approach should be tested in randomised controlled trials.

The glucose load in PD patients is likely to be an important contributing factor to many metabolic disorders seen in these patients such as hyperglycaemia, hyperinsulinaemia (70), hyperleptinaemia (71) and insulin resistance (72). In addition, it is thought that glucose absorption may be responsible, at least in part, for fat accumulation in these

patients. The development of strategies aimed at reducing glucose load using icodextrin, a glucose polymer PD fluid, has been shown to improve the lipid profile and insulin resistance of these patients (73). Glucose exposure in the PD regimen can be further reduced by replacing glucose-based PD fluids with both icodextrin and amino-acid based PD fluids during the same day. This combination has been recently shown to improve glycated haemoglobin in diabetic PD patients in a randomized controlled trial (74). Whether this improves body composition, as well as nutritional parameters should be addressed in the future.

11. Summary

All PD patients should be regularly monitored in order to detect early signs of PEW. In some groups of PD patients at higher risk of PEW, i.e. anuric PD patients, those with cardiac congestion, inflammation and low nutritional intakes, intensified nutrition counselling and nutritional support, preferably in the form of ONS, may help to cover nutritional requirements. Once PEW is established, ONS or AA-IPPN may improve nutritional status. Whether morbidity and mortality can be reduced by nutritional support in these patients is unknown, although it is likely that it will, considering the positive data obtained in HD patients. The reduction in glucose load obtained by using glucose-sparing solutions improves the metabolic profile of PD patients, although the impact of these solutions on body composition and patient outcomes still needs to be addressed.

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