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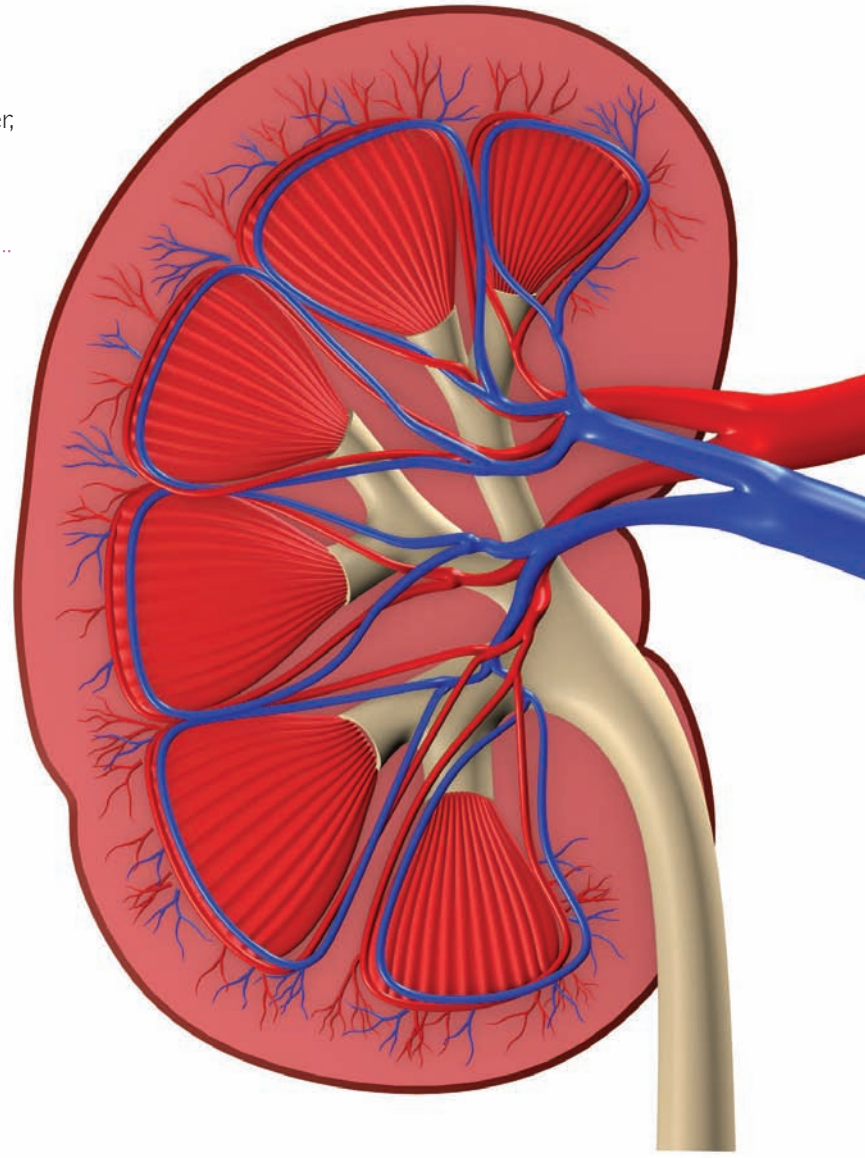
Malnutrition and Chronic Kidney Disease

Diane Green, Dietetic Manager, Salford Community Health, NHS Salford, UK



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Introduction

Malnutrition occurs in a high percentage of patients with renal failure and is associated with increased mortality and morbidity in this population. The cause of malnutrition is often multi-factorial but frequently contributed to decreased food intake. Providing calories and protein that are appropriate for a patients stage of kidney disease allows adequate nutrition and avoids unnecessary dietary restrictions.

A high prevalence of malnutrition exists in patients with renal disease. Various studies show signs of malnutrition in 23-76% of haemodialysis (HD) patients and 18-50% of peritoneal dialysis (PD) patients.[1-3] Variations in the prevalence in malnutrition can be attributed to age, case mix, co-morbid conditions and quality of dialysis. The renal population often have a reduced body weight, depleted energy (fat tissue) stores, loss of somatic protein (low muscle mass) and low levels of serum albumin, transferring pre-albumin and other visceral proteins. The aetiology of malnutrition in CRF is complex and may include many factors e.g. poor food intake due to anorexia, nausea and vomiting due to uraemic symptoms, hormonal derangements and acidosis. Research consistently reports inadequate oral intake as a major factor.[1]

Assessment of malnutrition in CKD

Many methods have been used to assess the presence of malnutrition in chronic kidney disease (CKD). A history of weight loss and symptoms such as nausea and anorexia may indicate impending or established malnutrition. Anthropometry measurements, such as BMI, mid-arm muscle circumference, skin fold thickness and hand grip strength[4] may all be useful tools. Biochemical markers (serum albumin, pre-albumin and transferrin) have been used to evaluate nutritional status. Of these biochemical markers, serum albumin has been most commonly used to assess malnutrition.[5] In nephrology it is well-established that a low serum albumin level is a strong predictor of total and cardiovascular mortality in HD and PD patients. On the other hand, in non-renal patients, no association has been found between serum albumin and cardiovascular disease, suggesting that low albumin *per se* does not necessary contribute to cardiovascular mortality.

Decreased nutrient intake

There are a number of factors that contribute to malnutrition in patients with renal failure (see **Table One**). Decreased intake of protein and calories are the most evident factors. Studies have demonstrated that even patients with a mild decline in glomerular filtration rate (GRF) i.e. GFR <50mls/min, have a decreased calorie and protein intake.[6] Studies have also documented that dietary protein progressively declines with decreasing GFR.[6]

Co-morbid conditions frequently contribute to deceased intake and malnutrition. One example is gastroparesis, which is likely to contribute to a poor intake in those patients with renal failure who have diabetes. Several studies have documented a high incidence of impaired gastric motility in maintenance dialysis patients.[7]

Overzealous diet restrictions can also contribute to decreased intake. The provision of a renal diet that limits protein, salt, potassium, phosphate and fluid may further limit dietary intake in a patient with existing malnutrition and poor oral intake. Dietary intervention should not be initiated until nutritional status and dietary habits have been assessed and clear needs for dietary restrictions are established. Underlying causes for electrolyte abnormalities such as poor glucose control, use of potassium sparing diuretics causing hyperkalaemia, need to be investigated before imposing dietary restrictions. Acchiardo *et al* [8] have proposed that the primary cause of hypoalbuminaemia in CKD is malnutrition. However, poor food intake does not often result in hypoalbuminaemia if CKD is not present and although food intake is markedly lower in patients with anorexia nervosa, serum albumin levels and catabolism of protein have been shown to be similar to those of control subjects.

Serum albumin levels may be low even in apparently well nourished HD patients, and they decrease in relation to the degree of malnutrition.[4] Inflammation can cause hypoalbuminaemia by suppressing albumin synthesis and by causing transfer of albumin from the vascular to the extravascular space. The combination of inflammation and reduced protein intake will lead to a significant reduction in serum albumin concentration.

Table One: Factors Contributing to Malnutrition in Renal Failure

- Decreased intake
  - Anorexia
  - Gastroparesis
  - Interperitoneal instillation of diaylsate in CAPD
  - Uraemia
- Diet Restrictions
- Loss of nutrients in dialysate
- Concurrent illness in hospitalisation
- Increased inflammatory markers
- Chronic blood loss
- Acidosis
- Endocrine disorders
  - Insulin resistance
  - Hyperglucogonemia

Increased nutrient losses

Patients who receive maintenance dialysis experience a loss of nutrients as a direct result of dialysis. HD results in a loss of 6-12g amino acids, 2-3g peptides and negligible amounts of protein per dialysis sessions.[9] During PD, patients lose only 2-4g amino acids, but experience a loss of 8-9g protein per day (including 5-6g albumin).[9] Patients on PD can lose over 15g protein each day during periods of peritonitis. This increase protein losses can last for several days after the peritonitis is treated.[9]

Patients receiving maintenance dialysis also have protein losses due to frequent blood sampling for labs. A patient with normal haemoglobin will lose approximately 16g protein with each 100mls of blood removed.[9]

Increased catabolism

Patients with renal failure are frequently 'anabolism challenged'. The increased acute phase reactants observed with renal failure and dialysis inhibits hepatic production of albumin and increased catabolism of skeletal muscle tissue.[9] Acidosis is an additional factor that precipitates catabolism in this population.[10] Provision of bicarbonate to maintenance dialysis patients decreases the protein catabolic rate and improves nutritional status.

Protein requirements

Protein requirements for patients with chronic kidney failure are dependent on the acute or chronic nature of the renal failure and the presence and type of dialysis. The nutritional status and adequacy of current intake of the patient should also be considered. A reduced protein intake may decrease uraemic symptoms and delay the need for dialysis in a stable patient with chronic renal insufficiency. However, a reduced protein intake is not advisable in the setting of significant malnutrition or inadequate calorie intake.

Protein requirements for patients receiving dialysis are increased above requirements for healthy adults. HD and PD increase nitrogen losses. In addition, there is information that HD is an inflammatory and catabolic process.[11] The European guideline is 1.0-1.2g protein/kg/day for stable haemodialysis patients and 1.2-1.5g protein/kg/day for stable PD patients.[12]

Energy intake

Energy intake is also critical in dialysis patient. Several studies have demonstrated that the energy requirements in these patients are not different from that of healthy populations. The recommended daily energy intake is 35kcals/kg/IBW/day. Patients on PD absorb calories from the glucose in the dialysis fluid and this should be included in the calculation of dietary energy intake.

Nutritional intervention

The frequent occurrence of malnutrition in patients with renal failure and the consistent association between markers of malnutrition and poor outcomes in this population emphasise the need for appropriate and timely intervention. Patients with CKD provide the dietitian with additional challenges of providing nutritional support with confines of fluid and any phosphate or potassium restrictions. Dietary supplements have the advantage of being fairly inexpensive and simple to administer. Several of the available supplements have been formulated specifically for renal patients. It is recommended that supplements are energy and protein dense and low in phosphate within a low volume. A large retrospective analysis of dietary supplements in dialysis patients demonstrated that their use was associated with an increased serum albumin level in those patients, as well as increases in body weight and anthropometry measures.[13] However, despite these results, many patients are not compliant with the use of supplements. This may be because of the fluid volume, monotony, or they may be unpalatable to the patient. It is important that the supplements prescribed are selected appropriately and that patients be carefully monitored for compliance.

If oral supplements cannot to tolerated, tube feeding should be considered. In practice, tube feeding can be very effective in improving nutritional status in malnourished patients. Patients who require extended nutrition support may be appropriate for long-term feeding access, such as a percutaneous endoscopic gastrostomy (PEG). Peritoneal dialysis is generally contraindication to PEG placement due to the risk of peritonitis. There is limited randomised data specific to maintenance HD patients receiving PEG feeding. Cohort data suggests that PEG feeds are safe and effective in patients receiving long-term HD.[14]

Conclusion

Although a large body of evidence exists to demonstrate that increasing intake will improve measures of nutritional status, no studies exist to determine whether provision of additional protein and calories to reach the target recommendations will change the outcomes of mortality and morbidity. No randomised, perspective or controlled trials have been carried out to examine this question. However, the benefits of an adequate diet would seem to outweigh the potential risk of over feeding. The main risk of increasing protein and energy intake is that every effort should be made to ensure that patients consume an adequate diet which may require a relaxation of some of the usual dietary restrictions. It also requires an effort by the dietitian to help patients understand their requirements and remain sensitive to patient's ethnic food habits. These efforts should result in well nourished patients, as well as improved patient outcomes.

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