15

Diabetes and Renal Replacement Therapy

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INTRODUCTION

The renal dietitian is an important member of the clinical team looking after patients with diabetes and end stage renal failure (ESRF). An understanding of the metabolic and nutritional changes that occur prior to and during ESRF is essential in order to provide nutritional advice to these patients. This is applicable irrespective of whether they are treated with haemodialysis (HD), peritoneal dialysis (PD) or renal transplantation.

PREVALENCE

Diabetes has become the commonest cause of ESRF in Western countries. In the UK around 16% of patients starting renal replacement therapy (RRT) have ESRF due to diabetic nephropathy (1). This figure is considerably higher in areas of the country where there are ethnic populations with an increased susceptibility to diabetes (2).

DIALYSIS INITIATION

Ideally, all diabetic patients approaching ESRF should be involved in the development of their own personalised care plan. This will enable a negotiation

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of the necessary dietary changes leading up to and extending to the initiation of elective dialysis (3).

Dialysis guidelines from the National Kidney Foundation promote the early initiation of RRT for diabetic patients, due to an increased susceptibility to uraemic symptoms at lower serum creatinine levels than non-diabetic subjects (4). Early RRT for diabetic renal failure not only relieves the symptoms of nausea, anorexia and vomiting but also helps reduce overall mortality. However, despite these recommendations, dialysis is frequently delayed due to either personal resistance or inadequate dialysis resources.

Some diabetic patients with already compromised renal function, will require the emergency initiation of dialysis during an intercurrent illness. However, this does not mean that all will require long-term RRT.

GLYCAEMIC CONTROL

Achieving good glycaemic control is important for all patients with ESRF as this can retard the progression of the microvascular and macrovascular complications (5). Good glycaemic control at the start of dialysis has also been shown to improve mortality risk. For patients on continuous ambulatory peritoneal dialysis (CAPD) hyperglycaemia increases circulating advanced glycation end products (AGE), which have been implicated in causing endothelium and peritoneal membrane damage with loss of ultrafiltration capacity. Good glycaemic control reduces thirst, which in turn helps to reduce fluid associated weight gain.

RENAL REPLACEMENT THERAPY

A brief outline on the principles of RRT is given below. Most patients with ESRF initially require dialysis, either haemodialysis or peritoneal dialysis. Only a minority of individuals, usually those with a suitable relative willing to donate a kidney, will have immediate access to a renal transplant.

DIALYSIS

While survival on dialysis continues to improve, diabetic patients still do less well than non-diabetic patients (6). Results from the Italian Cooperative Peritoneal Study Group Registry show the 10-year patient survival for the 301 diabetic patients to be less than half that of the 1689 non-diabetic subjects (20.6% vs 55.6%) (7). Higher mortality rates among diabetic patients receiving HD also occur (8), but with good glycaemic control these rates can be improved (9).

Peritoneal dialysis is the preferred mode of treatment for diabetic patients with microvascular and macrovascular co-morbidities. Continuous ambulatory peritoneal dialysis allows for a slow ultrafiltration process that provides greater cardiovascular stability than HD. Blood pressure control is easier and residual renal function is preserved for longer. It also provides incidentally for an alternative route of insulin administration. Initial concerns that diabetic CAPD patients may have higher dialysis-associated infection rates have not been confirmed (10,11).

DIALYSIS PROCEDURES

Continuous Ambulatory Peritoneal Dialysis

CAPD is a method of long-term dialysis that requires a permanent intra-peritoneal catheter. The peritoneum acts as a semi-permeable membrane that allows diffusion of solutes and facilitates the removal of water by ultrafiltration. The daily CAPD regimen comprises a drain-in period, followed by a dwell time of approximately 4 h and a drain-out period. This cycle is usually repeated four times each day (12,13).

Haemodialysis

Haemodialysis requires the surgical construction of an arterio-venous fistula that is usually sited in the non-dominant forearm using the cephalic vein and either the radial or brachial artery. After six to eight weeks the fistula has usually thickened sufficiently to allow it to be cannulated with two large-bore needles that take blood to and from the dialysis machine. Blood is pumped through a semi-permeable membrane filter in the dialysis machine allowing removal of excess solutes and fluid. Haemodialysis is repeated every two to three days with the time on dialysis dependent on the patient's body size and residual renal function. If a patient has no permanent access and requires emergency dialysis this can be done through a temporary neckline or a semi-permanent catheter in a central vein (14).

FACTORS INFLUENCING NUTRITIONAL STATUS IN DIALYSIS PATIENTS

Approximately 40% of dialysis patients exhibit some degree of protein and energy malnutrition and this is associated with an increased risk of morbidity and mortality. In the Modification in Renal Disease Feasibility Study (MDRD) in which 840 patients were prospectively studied, 42% of CAPD patients and 30% of HD patients were considered to be malnourished (15). Contributing factors to protein energy malnutrition occurring in dialysed patients are shown in Table 15.1.

Table 15.1 Factors implicated in protein–energy malnutrition in dialysis patients

Factors	Comments
Reduced nutritional intake	Energy and protein intakes consistently lower than requirements Uraemic symptoms can continue for upto three months after starting dialysis
Underdialysis	Nutritional intake deteriorates with inadequate dialysis
Gastroparesis	Abdominal distension Vomiting Early satiety
Dialysis-related effects	Abdominal discomfort with infusion of PD dialysate Protein losses, 6–12 g amino acids during one HD session Daily protein losses on CAPD of 5–15 g/day Peritonitis protein losses up to 20 g/day
Metabolic/endocrine factors	Hyperparathyroidism Hyperglucagonaemia Insulin resistance Vitamin D deficiency
Co-morbidity and infections	IHD and episodes of hypotension limiting dialysis time PVD can limit vascular access for HD Infections of vascular access in HD patients and peritonitis in PD

NUTRITIONAL ASSESSMENT/SCREENING

Ideally the nutritional status of patients approaching ESRF and starting dialysis should be monitored. As a single marker of nutritional status is unreliable, a number of nutritional parameters, as outlined in Chapter 14, can help identify those who are malnourished. Dual-energy X-ray absorptometry remains a useful method for assessment of lean body mass (15).

Nutrition scores such as subjective global nutrition assessment (SGA), based on clinical, physical and subjective measures are useful tools. The SGA is considered better suited to assessing study populations.

Serial biochemical flow charts are useful in assessing nutritional status (see Table 15.3). A decline in pre-dialysis serum urea, creatinine, potassium and phosphate may be indicative of a loss of lean body mass, rather than an improvement in nutritional status (17). Additional points to consider in the nutritional assessment of dialysis patients are given in Table 15.2.

Table 15.2 Additional factors in the nutritional assessment of dialysis patients

Factors	Comment
Anthropometry	Ensure measurements are taken at patient's dry weight: Post-dialysis for the HD patient After drained-out period for CAPD Adjust for oedema
Serum biochemistry	Serial biochemical measurements are useful in monitoring nutritional status
Protein catabolic rate (nPCR)	An indirect marker of protein intake in stable dialysis patients The nPCR for stable HD patients >1.2 g/kg/day The nPCR for stable PD patients 1.3 g/kg/day Patients with nPCR <0.8 g/kg/day require a dietary intake assessment

NUTRITIONAL REQUIREMENTS

Prior to dialysis most diabetic patients will have followed a diet that attempted to balance their carbohydrate intake with other aspects of their diabetes management. Many will already be on a diet that is low in phosphate and potassium and some will also be on a low-protein diet. Prior to dialysis, nutritional intake is usually inadequate with most patients having a negative energy balance (18). Intensive dietetic counselling is required for all patients starting dialysis to help them improve their dietary intake within the constraints of the combined diabetic/renal diet.

Energy Requirements for Dialysis Patients

Energy requirements to achieve neutral nitrogen balance in stable diabetic dialysis patients are similar to those of healthy non-diabetic adults (35 kcal/kg body weight), with lower requirements for subjects over 65 years of age (30–35 kcal/kg body weight) (19). Patients on CAPD receive part of their energy requirements from dialysate glucose (see below) (20).

If CAPD patients have difficulty in meeting their recommended dietary energy intakes due to early satiety, they should be encouraged to eat after 'drain-out' and to wait 20–30 min before commencing the next dialysate bag. Avoiding fluids at mealtimes can also improve appetite by minimising stomach distension. If energy requirements are not achieved despite dietetic input, nutritional supplements should be considered (21).

Table 15.3 A checklist for interpreting blood results of dialysis patients

Biochemistry (normal range)	Low	Good	Acceptable	High
Urea (mmol/l) (2.5–5.5)	<20 vegetarian status poor appetite residual function	20–28 check dialysis adequacy or urea reduction ratio to ensure patient not underdialysed	28–32	>32 high protein intake inadequate dialysate drugs, i.e. steroids catabolism acidosis
Creatinine (μmol/l) (55–125)	<700 small muscle mass (check BMI) residual function	700–1200 normal levels in dialysis patients		>1200 depends on size of patient. May be underdialysed
Potassium (mmol/l) (3.5–5.5)	<3.5 residual function diarrhoea vomiting malnourished re-feeding	3.5–5.5 (PD) 3.5–6.00 (HD)	6.0 (PD), 6.5 (HD) check diet, drugs, blood transfusion underdialysed hyperglycaemic acidotic	
Phosphate (mmol/l) (0.8–1.4)	<0.8 too many binders malnourished re-feeding	0.8–1.8	1.8–2.0	>2.0 check diet and binders check Ca and PTH levels
Calcium (mmol/l) adjusted (2.15–2.6)	<2.15 check adjusted for albumin	2.15–2.6	2.6–2.8	>2.8 check $CaCO_3$ tablets bone disease active vitamin D high PTH
Albumin (g/l) (35–55)	<30 infection (CRP) nephritic malnourished	33–45 NB: 30–34 may indicate malnutrition	45	>45 underdialysed constipated acidotic

NB: Use pre-dialysis HD bloods. This information is not applicable to other groups of renal patients.

Energy Gains and Losses from Glucose Fluxes During Dialysis

Energy requirements for CAPD patients are partially met from absorbed dialysate glucose. Requirements from dietary intake are therefore lower at 30 kcal/kg body weight (25 kcal/kg if obese) and 25–30 kcal/kg body weight if older than 65 years (21).

Patients absorb approximately 70% of dialysate glucose, amounting to 300–600 kcal or 2.5–17 g of glucose per hour during dwell times (22) (See table 15.4). Bag volumes range from 1 to 3 litres, with higher dextrose concentrations and larger volumes used to maximize solute clearance and ultrafiltration. The exact amount of glucose absorbed will depend on the dialysate glucose concentration and volume, the number of exchanges, the dwell time between each exchange and the permeability of the patient's peritoneal membrane. Membrane permeability is likely to be increased in people with diabetes. Increasing glucose loads can predispose to hyperglycaemia, hyperinsulinaemia, hyperlipidaemia, and obesity.

On the basis of a 70% absorption rate of glucose from dialysate fluid, between 2.5–17 g of glucose can be absorbed per hour during dwell times. This glucose load can predispose to hyperglycaemia, leading in turn to hyperinsulinaemia, hyperlipidaemia and obesity.

During HD blood values frequently fall below 4.0 mmol/l when patients are dialysed against a glucose-free dialysate (23).

Such patients should be advised to either eat a snack before or during dialysis. The choice of a slowly absorbed, low-glycaemia snack is ideal for this purpose.

Due to the metabolic problems associated with glucose as the main osmotic agent for inducing ultrafiltration in PD, alternative osmotic agents are now becoming available. High molecular weight glucose polymer solutions such as Icodextrin appear to be safe and effective. Icodextrin has the advantage of a reduced glucose and calorific load but requires dwell times of between 8–12 h and is therefore often left *in situ* overnight. No studies of use in diabetic patients have so far been published (24).

Table 15.4 Approximate energy provided by glucose-containing dialysate

Dialysate (1 l)	Grams of absorbed glucose	Energy provided (kcal)
1.36% dextrose	10	40
2.27% dextrose	16	60
3.86% dextrose	27	100

NUTRITIONAL REQUIREMENTS IN DIALYSIS PATIENTS

The general principles surrounding the nutritional requirements of patients with ESRF receiving dialysis are generally applicable to both diabetic and non-diabetic subjects and are briefly outlined below.

DIETARY PROTEIN

Protein requirements are increased in dialysis patients due to protein losses during dialysis (20) (see Table 15.5) and can be as high as 20 g albumin daily in CAPD with peritonitis. These losses are further increased in the diabetic patient (25) due to greater peritoneal membrane permeability. For non-diabetic stable dialysis patients daily protein intake recommendations based on NKF-KDOQI data (21) and supported by nitrogen balance studies are 1.2 g/kg/day for HD patients and 1.2–1.3 g/kg/day for CAPD patients. Adequate total energy intake is required to maximise the effectiveness of dietary protein utilisation (20).

MINERAL AND VITAMINS

Potassium

Hyperkalaemia in dialysis patients is a potential cause of sudden death. Recommendations to keep serum potassium levels between 3.5–6.5 mmol/l pre-dialysis for HD patients and 3.5–5.5 mmol/l for CAPD patients were published in 2002 (26). Potassium restrictions are usually unnecessary in CAPD patients due to the continuous nature of this form of dialysis. By way of contrast, HD patients accumulate potassium between dialysis sessions.

Phosphate

Hyperphosphataemia is very prevalent among patients with ESRD and is a cause of hyperparathyroidism, metastatic calcification (when the serum calcium–phosphate product exceeds 5.5 mmol/l) (27) and has been associated with excess cardiovascular mortality in HD patients (28). The recommended

Table 15.5 Amino acid and protein losses during dialysis

Dialysis Modality	Amino acids (g)	Proteins/peptides (g)
CAPD/day	2–3.5	5–15
HD/session	6–12	2–3

dietary intake of phosphorus for HD and PD dialysis patients is approximately 17 mg/kg/day. The removal of phosphate during dialysis is limited due to the high distribution volume for phosphate and the rapid rebound of serum phosphate following dialysis. Hyperphosphataemia is common and dietary restriction of dairy products, bony fish and offal meats combined with the use of phosphate buffers remains the best means of minimising hyperphosphataemia. Medical and dietary treatment of hyperphosphataemia is aimed at keeping the parathyroid hormone level within two to three times the upper limit of the normal range and the alkaline phosphatase and calcium concentrations within the normal range.

Sodium

On starting dialysis sodium intake should be limited to below 100 mmol, equivalent to 6 g salt/day. When dialysis patients become anuric their fluid and sodium intake needs to be further restricted, to 1 litre fluid/day and 80–100 mmol sodium/day. In patients able to maintain a urinary output above 1l/day, fluid restriction of 1.5–2 l/day and more flexible sodium intake may be appropriate. Residual urinary excretion is maintained for longer in PD than HD patients and hence fluid and sodium intake can initially be more liberal in PD patients.

Vitamins

The role of vitamin supplementation in dialysis patients is controversial. Vitamin status is compromised in dialysis patients due to poor nutritional intake and the cooking methods required for a low-potassium and low-phosphate diet. Fat-soluble vitamins other than vitamin D are not routinely prescribed due to the risk of vitamin A toxicity. Vitamin D is prescribed for bone protection and the prevention of hyperparathyroidism. Among the water-soluble vitamins 10 mg/day of pyridoxine and 60 mg/day of ascorbic acid are recommended as low concentrations can occur in dialysis patients. In addition folic acid may also be low and there may be a need for supplementation.

L-carnitine is an essential co-factor in fatty acid and energy metabolism and recent work suggests that it might be effective in reducing the erythropoetin requirements for controlling anaemia. The US Food and Drug Administration department have recently approved its use in the prevention and treatment of carnitine deficiency in HD patients. Currently, however, there is insufficient evidence to support its routine use in such patients (29).

NUTRITIONAL MANAGEMENT IN DIALYSIS PATIENTS

There are general principles regarding the nutritional management of patients on dialysis with ESRF. These are generally applicable to both diabetic and non-diabetic subjects (30,31) and are briefly covered below.

PREVENTION OF HYPERKALAEMIA

Hyperkalaemia in the HD patient may be due to dietary indiscretions, either as a result of unfamiliar foods of high potassium content or increased portion sizes of known potassium-containing foods. Patients on haemodialysis are usually advised to limit their potassium intake to less than 1 mmol/kg/day. Patients therefore need to avoid high-potassium foods, limit the intake of fruit and vegetables and cook using techniques to lower potassium levels.

Patients with residual urinary function can still excrete some urinary potassium and therefore may safely consume more fruit and vegetables provided there is close monitoring. Hyperkalaemia is less common in CAPD due to continuous potassium removal on dialysis.

In diabetic patients with poor glycaemic control, undergoing dialysis, hyperkalaemia can result from insulin insufficiency. Improving glycaemic control can reduce serum potassium levels. Insulin requirements can drop following the commencement of haemodialysis. In addition to poor glycaemic control, hyperkalaemia is also associated with a number of other non-dietary causes of hyperkalaemia.

PREVENTION OF HYPERPHOSPHATAEMIA

Patient awareness of phosphate-containing foods is essential in order to limit phosphate intake. Dairy products have two to three times more phosphate than equal quantities of protein derived from meat and therefore need to be limited. Milk intake should not exceed 1–3 pt/day and other dairy-containing foods such as chocolate, cheese and yoghurt should be restricted. Other high-phosphate foods such as offal and offal-containing products, veal and fish with edible bones, such as sardines, pilchards and shell fish also need to be limited.

Phosphate binders reduce the absorption of dietary phosphate but when they are comprised solely of calcium salts they can result in hypercalcaemia leading to metastatic calcification. For this reason phosphate binders made from non-calcium salts, such as Sevelamer, may be preferred for the prevention of hyperphosphataemia. To be effective, phosphate binders containing calcium carbonate should be taken before meals as the ability of this salt to bind with phosphate is pH-dependent. The calcium in the phosphate binders may have

the added advantage of helping patients achieve the necessary daily calcium requirements.

SODIUM INTAKE

Restricting sodium intake helps minimise thirst and hypertension. In certain individuals this can be difficult as so many convenience foods have a high salt content. A diet history can usually help in eliminating the very high sodium sources.

Interdialytic weight gains should not exceed 3% of the patient's dry weight. This is particularly important in diabetic HD patients, as excessive weight gains have been linked with increased mortality, possibly due to increasing hypertensive and cardiovascular stress.

HYPOALBUMINAEMIA AND PROTEIN MALNUTRITION

Hypoalbuminaemia can cause fluid and sodium retention, and is also linked to increased mortality. Medical management includes further fluid restriction and increased dialysis ultrafiltration. The use of hypertonic CAPD solutions to stimulate ultrafiltration can result in hyperglycaemia and an increase in protein loss. Dietary management is aimed at increasing protein intake. This is particularly a problem in vegetarian patients who rely on pulses and dairy products. While these products are good for diabetic control, they do deliver a greater phosphate load and increase the need for phosphate binders. For malnourished dialysis patients with poor appetites, high-protein energy dense comfort foods can provide a valuable source of energy and protein. When patients are unable to meet their protein requirements due to factors such as a reduced appetite, an inability to prepare foods or a lack of sufficient funds, high protein sip feeds should be considered.

Amino acid-containing dialysates for CAPD patients provide a potential means to improve nitrogen balance in severely hypoalbuminaemic patients. Further benefits to the use of such solutions require further evaluation (32,33).

ADDITIONAL NUTRITIONAL SUPPORT

Haemodialysis patients who continue to report weight loss and poor nutritional intake and who are unable to take nutritional supplements should be considered for either enteral support or intradialytic parenteral nutrition (IDPN) (34). Nasogastric feeding is the preferred short-term option for hospitalised inpatients but this is not usually practical in the outpatient setting. The relatively recent introduction of Percutaneous Endoscopic Gastrostomy (PEG) feeding in HD patients has been shown to be successful in many cases. Balancing the patient's fluid allowance with their nutritional requirements is a

major factor when prescribing the feed. The use of high energy dense (2 kcal/ml), low electrolyte feeds has made it possible to feed intermittently dialysed HD patients without causing fluid overload. However, not all patients will require this type of 'renal' feed. Patients with a low serum potassium and phosphate, i.e. CAPD patients, may require a high electrolyte feed of similar energy density. Continual review of serum biochemistry and fluid requirements is essential when prescribing this type of feed.

Interdialytic Parenteral Nutrition (IDPN) can be considered when nasogastric or PEG feeding in HD patients is not established. This consists of giving patients 1 l of a parenteral feed containing between 800–1000 kcals and 50 g amino acids via the venous return line during each dialysis session. Although the overall efficacy of IDPN is not well established, improvements in appetite, immune and nutritional status have been reported (35). A drawback to IDPN is that it can not be given during the dialysis-free days of the week.

SPECIFIC NUTRITIONAL MANAGEMENT IN DIABETIC DIALYSIS PATIENTS

The dietary management of diabetic patients receiving dialysis has to be adapted to achieve the best attainable glycaemic control. Dietary management also has to address any other aspects of the metabolic syndrome which may be present, such as obesity, dyslipidaemia and insulin resistance.

GLYCAEMIC CONTROL

Although it is a popular patient myth that the principles of a good diabetic diet cannot be achieved within the confines of dialysis dietary restrictions, in reality there is sufficient scope for dietary adjustments using lower glycaemic index food choices to prove this wrong. However, not all low glycaemic foods are suitable for dialysis patients; potatoes, yam, cassava, sweet potato and green banana all contain significant amounts of potassium. Suitable low glycaemic foods include porridge, pasta, basmati rice, couscous and granary breads. Boiling vegetables in large volumes of water can reduce the potassium content considerably. Hence, when an individual's staple diet includes carbohydrate foods with a high potassium content, suitable cooking methods can make many more of these foods available for inclusion in the diet.

Since there is a high prevalence of diabetic ESRF in ethnic minority populations, a number of cultural and religious factors that can influence glycaemic control need to be considered. The most important of these is a wish to fast for religious observance, even though exemptions can be obtained. For example, during Ramadan, fasting can last up to 18 h in the summer months with no foods or drinks being taken between dawn and sunset. Of necessity

large meals are usually taken before dawn and after sunset and are likely to include fried foods and carbohydrate-rich meals including specially prepared sweet foods. During such periods, without expert adjustment of treatment regimens, large fluctuations in blood glucose can occur. Hypoglycaemia may also be a problem among haemodialysis patients who are not being dialysed against a glucose-containing dialysate. Dietary advice should be directed towards limiting sweet foods taken after sunset and promoting low glycaemic foods such as basmati rice, chapati or naan breads made with wholewheat flour and not besan flour (chick pea) which is high in potassium.

OBESITY

Weight reduction using dietary intervention is generally considered suitable within the general population when the BMI is above 30 kg/m^2. A considerable number of CAPD patients are either obese or have a tendency to become so. The weight management of CAPD patients is difficult as many are receiving additional calories from the glucose absorbed during dialysis. Increasing physical activity is often unrealistic in a population confined to their house for much of the day.

DYSLIPIDAEMIA AND OTHER CARDIOVASCULAR RISK FACTORS

The dyslipidaemia of ESRF is characterised by raised plasma triglycerides with a normal cholesterol level. This form of dyslipidaemia becomes worse after starting CAPD. The lipid profile on CAPD is generally more atherogenic than with other dialysis modalities. A possible contributory factor for this atherogenic lipid profile is the glucose absorption from the dialysates giving rise to enhanced triglyceride synthesis. Chronic hyperinsulinaemia is a major component of the metabolic syndrome and a recognised risk factor for atherosclerosis (36). There is a preferential sieving of the smaller cardio-protective lipoprotein: HDL-cholesterol among the proteins lost into the dialysate rather than that of other lipoproteins (37).

The dyslipidaemia of ESRF is an independent risk factor for atherosclerosis in ESRD (38). To date there are no convincing trials to show that the 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statin) are equally as effective as they are for non-renal-compromised patients, and although widely used, their benefits are not unequivocally established (39,40). Dietary management of glycaemic control for diabetic patients on PD improves hypertriglyceridaemia and overall dyslipidaemia. The effectiveness of dietary manipulation of plasma lipids in PD patients is limited and it could be argued that if adhered to, properly constructed dialysis diets are close to optimal lipid-lowering recommendations (41,42). General healthy eating advice given to patients with normal renal function may not be appropriate, as many PD

patients are undernourished and advice to reduce saturated fat should not be at the expense of reducing total protein or energy intake. Current recommendations are that 35% of energy should be derived from fat (41), with emphasis placed on reducing the saturated to polyunsaturated fat ratios.

HYPOGLYCAEMIC AGENTS AND DIALYSIS

The diet for the diabetic patient on dialysis needs to reflect their diabetic treatment. An understanding of how ESRF and dialysis interacts with the action of hypoglycaemic agents and insulin is important for the renal dietitian if the prescribed diet is to minimise periods of hypo- and hyperglycaemia.

ORAL HYPOGLYCAEMIC AGENTS

Oral agents are rarely used in Type 2 diabetic patients undergoing dialysis. By the time diabetic patients have developed ESRF extensive β-cell failure will have occurred and most patients will have already been switched to insulin. During the pre-dialysis period metformin is contraindicated as are the longer acting sulphonylureas such as glibenclamide. An insulin secretagogue like repaglinide, whose metabolism is not dependent on renal function for clearance, is safe. The glitazone class of insulin sensitisers can also be used in subjects with moderate renal failure. However, in reality, by the time patients are requiring CAPD or HD, insulin provides not only the safest but also the most flexible and easiest way to optimise glycaemic control.

INSULIN

Insulin requirements in ESRD patients are difficult to predict as renal and extra-renal breakdown of insulin is diminished on the one hand and its half-life is prolonged on the other. With advancing renal failure this effect is antagonised by insulin resistance, which usually improves with regular dialysis. On initiation of CAPD, the timing of insulin doses should be adjusted to complement the pattern of glucose absorption. Very strict glycaemic control becomes increasingly difficult in dialysis patients because of the risk of intradialytic and nocturnal hypoglycaemia. A balance around optimising glycaemic control needs to be made as good glycaemic control reduces catabolism and improves nutritional status. One expert committee has suggested an HbA_{1c} target of below 8%.

Administrating insulin via the intraperitoneal route can improve glycaemic control and lessen hyper- or hypoglycaemic episodes. Intraperitoneal insulin absorbed into the portal venous circulation results in a more physiological portal to peripheral insulin ratio of approximately 3:1 and lower systemic

insulin levels (43). However this route of administration promotes a more atherogenic lipid profile and may cause focal hepatic fat accumulation, and there also remains a theoretical risk of peritonitis.

SUMMARY

As the prevalence of diabetes grows so will the need for renal replacement therapies. The dietary management of patients as they approach ESRF should focus on minimising uraemic symptoms while ensuring a sufficient protein and energy intake to prevent malnutrition. Both the nutritional status and the glycaemic control of an individual as they start dialysis are independent important predictors of their future morbidity and mortality. Achieving glycaemic control in patients undergoing regular dialysis requires a careful balance between the dialysis prescription and meal planning. While the nutritional requirements, constraints and demands are by and large similar for the diabetic and non-diabetic patient, the need to address atherosclerotic cardiovascular risk factors is still greater in the diabetic patient. Today many of the diabetic patients undergoing dialysis are malnourished with poor glycaemic, lipid and metabolic profiles. There is a real need for well-constructed nutritional studies in the diabetic dialysed population to formulate evidence-based dietary interventions that will help to extend the patient's life expectancy to that of a non-diabetic dialysed subject.

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