Disorders of Protein and Lipid Metabolism Associated with Chronic Renal Failure and Chronic Dialysis

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ABSTRACT

Patients with chronic renal failure treated with maintenance dialysis demonstrate many abnormalities of protein, carbohydrate, and lipid metabolism. Losses into dialysate of amino acids and glucose during the course of hemodialysis and protein losses during peritoneal dialysis, associated with inadequate dietary intake and/or increased endogenous catabolism, may result in chronically negative nitrogen balance and a state resembling clinical malnutrition. Hypertriglyceridemia and low levels of high density lipoprotein cholesterol are also characteristic of the chronic dialysis patient. Patients receiving peritoneal and chronic ambulatory peritoneal dialysis may be at particular risk for hypertriglyceridemia because of high obligate glucose loads. Literature relating to these areas is reviewed, and specific recommendations outlined for dietary protein, amino acid, carbohydrate, and lipid content and composition in the chronic renal failure and chronic dialysis patient.

Introduction

There are numerous disorders of carbohydrate, protein, and lipid metabolism seen in patients with chronic renal failure. Similar metabolic abnormalities may be seen in patients suffering from malnutrition of any source, including low levels of complement, albumin, transferrin, and many amino acids. Serum transferrin level in particular appears to correlate well with nutritional status. Malnutrition in renal failure patients is usually multifactorial in etiology, with poor dietary intake secondary to dietary restriction, anorexia, or depression, intercurrent catabolic illnesses, and dialysis-related losses all playing a role.

It is the purpose of this report to summarize, place into perspective, and suggest therapeutic approaches to some of these disorders. Because of the scope of
the topic, our review is limited to discussions of protein, amino acid, and nitrogen balance, and disorders of lipid metabolism.

**Protein and Amino Acid Metabolism and Nitrogen Balance**

Serum concentrations of essential amino acids are decreased in renal failure, whereas those of nonessential amino acids are frequently increased. Tyrosine concentrations tend to be reduced, as are the ratios of tyrosine:phenylalanine and total essential to nonessential amino acids. The conversion of phenylalanine to tyrosine is impaired. Elevated concentrations of products of amino acid and protein metabolism may accumulate because of decreased urinary clearance, increased production, or decreased degradation. The kidney itself degrades many peptides and small proteins; however, in renal failure, serum concentrations of such compounds as Bence-Jones protein, gamma globulin light chains, alpha-1 microglobulin, beta-2 microglobulin, lysozymes, fibrinogen degradation products, and ribonuclease may all increase.

Protein and amino acid metabolism and nitrogen balance will be reviewed first in the chronic dialysis patient and then in the patient with chronic renal failure who is under medical management but does not yet require dialysis.

The loss of amino acids during hemodialysis varies from three to 22 g per dialysis, with the rate of amino acid loss averaging between one to two g per hr (table I), 20 to 30 percent of the loss consisting of essential amino acids. When a dialysis bath without glucose is used, amino acid loss will be approximately 50 percent higher than when a glucose-containing bath is used. This may relate to an increased transfer of amino acids from tissues to liver for gluconeogenesis. Wathen et al studied 10 chronic dialysis patients after a three hour fast, comparing glucose-free and glucose-containing (200 mg per dl) dialysate. Acetate concentration of dialysate was 35 mEq per L in all cases. Decreased blood levels were found of glucose, insulin, lactate, and pyruvate with glucose-free dialysate. In addition, a close parallel between plasma glucose and insulin levels was seen in the absence of any statistically significant changes in plasma glucagon levels, which remained considerably above normal. The lack of any reciprocal changes in glucagon relative to changes in plasma insulin and glucose levels suggested an impaired feedback system, perhaps related to chronically elevated glucagon levels. A total glucose loss of about 28 g into the dialysate was seen over the six hours of glucose-free dialysis. The decrease in plasma glucose levels seen, however, reflected only a small fraction of this loss (approximately 21 percent), suggesting that the remainder (22 g) must have come from hepatic glucose production during the duration of dialysis. If the marked decrease in blood pyruvate is accepted as an indication of gluconeogenesis, then these data suggest that gluconeogenesis becomes active during dialysis with glucose-free, but not with glucose containing, dialysate.

Acetate ions have energy content, since they can be metabolized to CO₂ and water and can be considered fragments of amino acids. Owing to its small molecular weight, 59 Daltons, acetate has a dialysance similar to that of urea (60 Daltons).
Gonzalez et al\textsuperscript{16} have demonstrated a transfer of 17.7 g per hr using a one square meter surface area dialyzer; others\textsuperscript{29} have reported a transfer of 14.5 g per hr. A positive acetate transfer of 17.5 g per hr could theoretically provide the patient with a positive caloric balance of some 70 calories per hr.\textsuperscript{9} Even assuming a caloric loss of 18 calories per hr utilizing a glucose-free bath (net loss of glucose 4.5 g per hr), and eight calories per hr from amino acid loss (assuming net loss of 2 g per hr), the patient would still, assuming full caloric utilization of acetate, receive a positive caloric balance of 43.5 calories per hr.\textsuperscript{9}

Addition of a solution of five percent synthetic amino acids to the dialysis bath, resulting in a bath composition similar to normal plasma, prevented the anticipated one to two g per hr loss of amino acids.\textsuperscript{9} A bath containing twice the previous concentrations of amino acids supplied amino acids to the patient at a rate of 1.5 to 2 g per hr. Infusion of five percent amino acid solution intravenously over the same time period (four hrs) at the end of dialysis resulted in considerably higher serum levels than those obtained by adding the amino acids to the dialysate. Thus, although administering amino acids via dialysate was satisfactory, only about half of the amino acids were transferred to the patient; hence, it was more economical to administer the amino acids intravenously at the conclusion of dialysis.

**Nitrogen Balance Measurements**

Borah et al\textsuperscript{5} performed daily measurements of nitrogen balance at two levels of protein intake in five chronic hemodialysis patients. During high (1.4 g per kg body weight) protein intake, nitrogen balance was positive on non-dialysis days and negative on dialysis days, resulting in a cumulative nitrogen balance for the week of study of essentially zero. During low (0.5 g per kg) protein intake, nitrogen balance was approximately zero on non-dialysis days but was again negative on dialysis days, resulting in a negative cumulative balance for the study period. The negative nitrogen balance observed on dialysis days was associated with a higher rate of urea generation (determined by kinetic modeling) that was most evident in the hours immediately following dialysis. The average overall negative nitrogen balance on dialysis days in these studies was a negative 2.4 g nitrogen per patient per day, an amount consistent with previously reported losses into dialysate averaging one to three g of amino acid nitrogen per dialysis. Catabolism of intracellular proteins must occur in response to dialysis to maintain intracellular amino acid pools despite the loss of amino acids in the dialysate. It can be speculated that the accelerated rise in blood urea nitrogen during the post-dialysis period is a reflection of the stimulation of urea generation resulting from the increased protein catabolic rate on dialysis days.

**Proteins in Hemodialysis**

Although amino acid and peptide loss during hemodialysis is about one to two g per hr of dialysis, there is little or no plasma protein loss into hemodialysis fluid; however, in chronic peritoneal dialysis, losses of 0.5 to 2.0 g of protein per liter of dialysis fluid have been observed (table II), and under optimal conditions, a loss of between 0.2 to 1.0 g per L will occur.\textsuperscript{22} Kluthe et al\textsuperscript{22} recommend a dietary intake for chronic hemodialysis patients of 1.2 g per kg of protein, one g per kg of protein plus 0.2 g per kg of essential amino acid supplementation, or one g per kg protein plus 0.2 g per kg of high biologic value protein (table III). This recommendation, given in conjunction with 35 kilocalories per kg total energy intake, is adequate for a well dialyzed hemodialysis patient who is in good condition, but it does not cover additional
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needs when stress situations, bleeding, or infection occur. Because of protein losses into dialysate, peritoneal dialysis patients will also require a higher protein intake, possibly of an additional amount approaching losses in dialysate. Dialysate protein losses in patients receiving continuous ambulatory peritoneal dialysis, using four exchanges per day, average 12 ± 2 g per day32 (table II).

In summary, patients undergoing hemodialysis three times weekly should probably receive 1.0 to 1.2 g protein per kg per day, possibly including or supplemented by 0.2 g per kg per day as essential amino acids.22,23 Patients receiving maintenance peritoneal dialysis will require a higher protein intake, approximately 1.2 to 1.5 g per kg per day, because of losses of both protein and amino acids during dialysis (table III). Giordano et al14 have presented data suggesting that administration of 1.2 g protein per kg per day will result in positive nitrogen balance in the majority of patients on chronic ambulatory peritoneal dialysis using 10 L of dialysate per day.

At least half of the dietary protein intake for dialysis patients should be of high biologic value.23 Caloric intake, as discussed previously, should be at least 35 kilocalories per kg per day. During peritoneal dialysis in normoglycemic patients, approximately 5 to 18 g of glucose per hr is absorbed when the dialysate contains 1.5 percent glucose, and 25 to 60 g of glucose per hr is absorbed with 4.25 percent glucose. With continuous ambulatory peritoneal dialysis, approximately 22 g of glucose will be absorbed from each six hr exchange with 1.5 percent dialysate, and 52 g from each 4.25 percent exchange32 (table II). Hence, patients undergoing peritoneal dialysis may benefit from a lower dietary carbohydrate and caloric intake (table III).

Supplementation with intravenous or oral essential amino acids is probably not necessary if stable chronic hemodialysis patients ingest diets providing the previously stated requirements. In patients with dietary intake less than this, or in those who are septic or otherwise hypercatabolic, oral or intravenous supplementation between or during dialyses may be indicated. Caloric and amino acid supplements may be administered orally in quantities sufficient to meet the goals if this is required and can be accomplished. If this is not possible, amino acids can be infused into the venous line of the dialyzer over the entire course of each dialysis, during the last one to two hrs of dialysis,14 or at the conclusion of dialysis.9 Glucose-containing dialysate, 200 mg per dl, should be used and additional oral carbohydrate may be required at the end of

| TABLE II: Peritoneal Dialysis Net Transfer of Nutrients |
|-----------------|-----------------|-----------------|
|                  | Intermittent PD | Chronic Ambulatory PD |
|                  | g per hr        | g per 6 hrs     |
| Glucose:         |                 |                 |
| 1.5 percent      | +5              | +22             |
| dialysate        | -18             | -25             |
| 4.25 percent     | +25             | +50             |
| dialysate        | -60             | -60             |
| Total protein    | -0.2            | -2.5            |
| Albumin          | -2.5            | -3.5            |

**TABLE III: Recommended Dietary Intake for Chronic Dialysis Patients**

<table>
<thead>
<tr>
<th></th>
<th>Hemodialysis</th>
<th>Intermittent PD</th>
<th>Chronic Ambulatory PD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein† (at least half of high biologic value)</td>
<td>1.2</td>
<td>1.2 - 1.5</td>
<td>1.3 - 1.5</td>
</tr>
<tr>
<td>Calories§</td>
<td>35</td>
<td>35</td>
<td>25 - 30</td>
</tr>
<tr>
<td>Carbohydrates§</td>
<td>30 - 35</td>
<td>30 - 35</td>
<td>25 - 30</td>
</tr>
<tr>
<td>Polyunsaturated: 1:1</td>
<td>1:1</td>
<td>1:1</td>
<td>1:1</td>
</tr>
<tr>
<td>Saturated fat ratio</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

*Peritoneal dialysis †Kcal per kg per day §g per kg per day ¶Percent of total calories
Dialysis to prevent reactive hypoglycemia. These types of supplementation should be considered in any chronic dialysis patient developing evidence of protein-caloric malnutrition who cannot be controlled by usual dietary measures.

Dietary Management in Patients With Chronic Renal Failure

These patients are those with chronic renal failure who do not yet require dialysis. Patients with moderate renal failure (serum creatinine 4.5 to 12.0 mg per dl) were treated with a high caloric diet containing 0.5 to 0.7 g per kg per day protein supplemented with eight essential amino acids plus histidine. The patients demonstrated decreased uremic symptoms, blood urea nitrogen, and urinary excretion of methylguanidine and guanidinosuccinic acid, associated with improved nitrogen balance.1 Patients with creatinine clearances of 3 to 20 ml per min have been managed with a 30 g protein diet, supplemented by 10 g of a mixture of essential amino acids and histidine, or with a mixture of the ketoanalogues of essential amino acids, essential amino acids, and histidine. Total nitrogen content of the diet was five to six g, and caloric content was 2000 to 3000 kilocalories.44

Nonessential amino acids may have superior nutritional value compared to essential amino acids as a source of nonessential nitrogen.27-33 Diets containing mixtures of nonessential and essential amino acids may be preferable for chronic renal failure patients.

A direct relationship between the serum urea nitrogen to serum creatinine ratio and protein intake in nondialyzed patients with chronic renal failure has been demonstrated.25 This relationship may be useful for selecting the optimal quantity of protein prescribed in these patients. For example, to maintain the serum urea nitrogen at 60 mg per dl in a man with serum creatinine of 10 mg per dl, the ratio of serum urea nitrogen to serum creatinine would be 6.0, and about 40 grams protein per day will maintain this ratio at 6.0 in men.25 Protein restriction is rarely necessary until glomerular filtration rate is below 25 ml per min.23 Below this level, Kopple25 suggests restriction of daily protein intake to up to 90 g for creatinine clearance of 20 to 25 ml per min, up to 70 g for 15 to 20 ml per min, and up to 50 g for 10 to 15 ml per min (table IV).

When the glomerular filtration rate is 4 to 10 ml per min, diets providing 40 grams of protein per day (0.55 to 0.60 g per kg per day), containing 28 g of protein of high biological value, have been recommended.23,24 These diets maintain neutral or positive nitrogen balance while reducing uremic symptoms. Protein intake may be increased gram for gram in the presence of urinary protein losses. When the glomerular filtration rate is below 4 to 5 ml per min, dietary protein restriction becomes less satisfactory, and lower protein diets (18 to 25 g per day of primarily high biologic value protein) may induce muscle wasting.23 In general, institution of dialysis should not be postponed beyond this point. However, if dialysis cannot be instituted, amino acid or ketoacid diets, as will be discussed later, may be considered. Modifications of the low protein diet, which provide 16 to 22 g of protein per day of mixed biological value with the addition of supplemental amino acids or keto acids, may maintain nutrition and reduce uremic toxicity in patients with creatinine clearance below 4 to 5 ml per min (table IV). For a short period of time, a diet providing 0.47 g per kg per day of the nine essential amino acids can be used with a very low protein diet (3 g per day).23

Keto Acid Therapy in Chronic Renal Failure

Walser42 has reviewed the principles of ketoacid therapy in uremia. He points out that ketoanalogues might suppress urea
formation by an amount of nitrogen equal to the stoichiometric quantity required to aminate them, which represents approximately nine percent by weight. Thus, a ketoanalogue supplement of 11 g might reduce urea nitrogen production by one g. Reduction of nitrogen intake by this amount, without inducing negative nitrogen balance, might produce a substantial fall in blood urea nitrogen concentration and result in symptomatic improvement in chronically uremic individuals. The rationale for ketoacid therapy is that these compounds reduce urea production by diverting nitrogen of urea precursors toward protein synthesis. Walser has been able to maintain positive nitrogen balance in uremic patients ingesting a virtually protein free diet supplemented with the ketoanalogues of branched chain amino acids, the hydroxy-analogues of methionine and phenylalanine, and the four remaining essential amino acids as such, plus glycine.

Beneficial effects of ketoacids in patients with chronic renal failure are likely to occur only in those taking a diet of less than 30 g of protein daily, and supplementation with essential amino acids or their ketoanalogues may not be indicated or necessary in well nourished dialysis patients. Although the alpha keto analogues of all essential amino acids except lysine and threonine can be converted by human subjects into the corresponding amino acid, there are too few quantitative estimates of the efficiency of conversion to permit calculation of the amounts of alpha keto analogues that are required to meet amino acid requirements. Efficiency of utilization of different alpha keto analogues may vary greatly. Efficiency of conversion to the amino acid will depend upon route of administration, organ distribution and relative activities of the enzymes in the pathway for alpha keto acid metabolism, as well as the relative concentrations of the alpha keto acid analogues and the corresponding amino acids in blood and body fluids. Thus, the role of ketoacid therapy in patients with chronic renal failure remains imprecisely defined but is probably a considerably more limited one than had initially been proposed.

Lipid Metabolism

Abnormalities of plasma lipids are seen in at least 50 percent of chronic dialysis patients. Levels of serum triglycerides are significantly increased in both undialyzed uremic patients and in dialyzed patients. The hypertriglyceridemia associated with dialysis may be partially related to increased synthesis of fatty acids from acetate. Levels of high density lipoproteins in chronic dialysis patients are approximately half those in normal individuals without renal failure. Cholesterol levels are generally within normal limits. In one series of 131 patients on hemodialysis, 65 percent had abnormalities in plasma lipoproteins: Type IV in 39 percent, Type IIB in 21 percent, and Type IIA in 5 percent. Triglyceride levels were higher in dialysis patients than in control subjects, but there was no statistical difference in cholesterol values between the two groups.

In transplant recipients, both plasma cholesterol and triglycerides are significantly higher than in normal controls. Plasma cholesterol levels are significantly higher in transplant patients than in
dialysis patients, but no difference is usually seen in triglyceride levels. In one series of 126 dialysis patients studied, 28 percent showed a normal serum lipid pattern, 6 percent type IIa, 11 percent type IIb, and 56 percent type IV. Among 101 transplant recipients, 29 percent had a normal lipid profile, 18 percent type IIa, 16 percent type IIb, and 35 percent type IV. Serum triglyceride concentrations in children on peritoneal dialysis are not significantly different from those on hemodialysis, but both are significantly higher than in patients on medical management and after transplantation.

The hypertriglyceridemia of chronic renal failure may be related to excessive production or to impaired degradation. The hyperinsulinemia characteristic of uremia could play a role in causing overproduction of free fatty acids and triglycerides. This may be the case but probably is of minor importance. The increased glucagon levels seen in uremia probably do not play a major role in regulating lipid metabolism. The observation that serum free fatty acid levels are not elevated in chronic uremia despite elevated glucagon levels suggests that glucagon does not stimulate lipolysis in man. There is a poor correlation between plasma insulin levels and serum triglyceride concentrations in uremia, casting doubt on a role for either insulin resistance or hyperinsulinemia in the etiology of uremic hypertriglyceridemia.

There is considerably more evidence to support the concept of impaired lipoprotein catabolism in uremia. Lipoprotein lipase in adipose tissue has been found to be reduced in dialysis patients with hypertriglyceridemia and normal in dialysis patients with normotriglyceridemia. Decreased adipose tissue lipase has been found in uremic subjects. Post-heparin lipase activity, an indirect measure of lipoprotein lipase, is also reduced in uremia. This may be related to decreased formation or impaired release of the enzyme. Ibel's et al demonstrated reduced post-heparin lipase activity and triglyceride clearance in patients with renal failure and hyperlipoproteinemia. Finally, a nondialyzable factor which inhibits lipoprotein lipase activity has been demonstrated in uremic plasma. Thus, the bulk of evidence supports the conclusion that the major cause of uremic hypertriglyceridemia is decreased lipolytic activity and not increased triglyceride production.

Frank et al reported that significant hypertriglyceridemia was first observed when creatinine clearance fell to 50 ml per min. The incidence continued to rise as creatinine clearance fell further with the highest rate developing at a creatinine clearance of less than 10 ml per min. Hypertriglyceridemia was correlated with plasma glucagon levels but not with growth hormone or insulin levels. Plasma cholesterol levels remained normal with deteriorating renal function and showed no correlation with plasma glucagon, growth hormone, or insulin levels. There was a decrease in the prevalence of hyperlipidemia after five years of maintenance hemodialysis therapy. It was speculated that plasma growth hormone and glucagon through an effect on plasma triglyceride, and plasma insulin by an effect on plasma cholesterol, might play a role in the decline of hyperlipidemia with increased duration of dialysis.

In a prospective controlled study of hemodialysis vs. peritoneal dialysis, previous findings of hypertriglyceridemia among peritoneal dialysis and hemodialysis patients have been confirmed by us. A significant difference was not demonstrated between peritoneal dialysis and hemodialysis. Lower values were noted by us for HDL cholesterol in dialysis patients, as has been reported by others. HDL levels in hemodialysis patients were significantly lower than those observed in controls; values for peritoneal dialysis patients were lower, although not
significantly so, than controls. The difference between controls and peritoneal dialysis patients might, of course, have reached significance if a larger sample size had been available. The issue is of considerable importance, since HDL levels have an inverse relationship with the risk of atherogenesis, and relative preservation of HDL values may represent an advantage to peritoneal dialysis patients.

The subject of accelerated atherosclerosis in chronic dialysis patients remains controversial. Lindner et al reported a marked increase in deaths from atherosclerotic causes in patients on long-term hemodialysis. Subsequent studies have suggested that atherosclerosis observed in chronic dialysis patients may be related more to pre-existing risk factors such as hypertension, antedating the onset of dialysis, than to accelerated atherosclerosis directly related to the chronic dialysis procedure. Epidemiologic data do not suggest that triglyceride excess per se plays a major role in accelerated atherosclerosis.

Low levels of HDL cholesterol are seen in undialyzed patients with chronic renal failure. Levels of HDL cholesterol in patients on dialysis are also significantly lower than values from control and non-uremic subjects with similar levels of triglyceride excess. Epidemiologic studies have shown consistent inverse associations between HDL cholesterol and coronary heart disease mortality in diverse population groups. Moreover, HDL cholesterol predicts risk of coronary heart disease independent of other lipid levels. Among older subjects, HDL cholesterol is the best single lipid indicator of coronary heart disease risk. Since levels of lipoprotein lipase correlate directly with levels of HDL cholesterol, the consistent finding of impaired removal of triglyceride in undialyzed and dialyzed uremic patients is of great interest. The low levels of HDL cholesterol are most likely secondary to a combination of decreased adipose tissue LPL, abnormal apoprotein composition of HDL, and decreased lecithin-cholesterol acyltransferase activity.

Treatment of the hyperlipidemia associated with chronic renal failure and chronic dialysis is a difficult problem. Clofibrate will effectively reduce triglyceride levels, but its route of excretion is renal, and dosage must be markedly reduced in renal failure, with potential risk of muscle toxicity. Intensive hemodialysis has been shown to reverse the insulin resistance and decrease insulin levels as well as triglyceride synthesis in some studies, but this seems to be a relatively ineffective and impractical approach.

Dietary modification has demonstrated considerable promise in controlling hypertriglyceridemia in the chronic dialysis patient. Twenty dialysis outpatients with hyperlipoproteinemia were given diets for one month planned to reduce their intake of fat and cholesterol and to raise the ratio of polyunsaturated to saturated fats to 1:1. Carbohydrate intake was increased to maintain the same caloric values. Plasma levels of triglyceride fell significantly in patients with Type IV and Type IIB hyperlipoproteinemia. The authors postulate that simple dietary modification may be an effective treatment for dialysis related hyperlipidemia. Serum triglycerides may also be lowered with a diet in which the carbohydrate content is reduced from 50 percent of total calories to 35 percent, the fat content is increased from 40 percent to 55 percent of calories, and the polyunsaturated to saturated fatty acid ratio is raised from 0.2 to 2.0.

Cattran et al reported a 33 percent reduction in plasma triglyceride levels by decreasing dietary carbohydrate from 45 percent to 20 percent of total calories ingested for three weeks in eight stable chronic dialysis patients. There was a 33 percent increase above control values.
after three weeks of high carbohydrate intake (60 percent of total calories ingested). Dietary fat intake was increased from 35 percent to 60 percent of total calories during the low carbohydrate period and decreased to provide 22 percent of total calories during the high carbohydrate period, maintaining a constant caloric intake in all study periods. Polyunsaturated to saturated fat ratio was also kept constant at 0.10 to 0.15:1. Cholesterol levels remained unchanged and within normal limits. No significant correlation between either insulin or glucagon levels and triglyceride concentration were observed. These data suggest that reduction in dietary carbohydrate may be an effective long-term therapeutic approach to the hypertriglyceridemia seen in chronic dialysis patients.

Chronic ambulatory peritoneal dialysis, with its continuous obligate glucose load, may have significant implications regarding lipid profile and risk of accelerated atherosclerosis in patients so treated. As noted previously, approximately 22 g of glucose will be absorbed in the course of a six hr exchange with 1.5 percent dialysate, and 52 g with 4.25 percent dialysate. Many patients receiving chronic ambulatory peritoneal dialysis demonstrate high triglyceride levels and a tendency towards obesity, perhaps related to this increased carbohydrate load. Long-term implications of this hypertriglyceridemia are unknown but are of potential concern. A highly significant decrease in serum triglycerides and cholesterol in chemically diabetic rats was seen when activated charcoal was administered as one percent of their diet. Thirty-five g per day of oxystarch plus 35 g per day of activated charcoal resulted in decrease of mean serum cholesterol concentration from 200 mg per dl to 140 mg per dl after a four-week trial. Hypertriglyceridemia was corrected in three of four patients. It is speculated that activated charcoal, which is inert as an intestinal nitrogen binding sorbent, may lower serum lipids by direct intragut binding of lipids and bile acids.

Renal transplantation can be expected to increase cholesterol levels as a result of the steroid therapy associated with transplantation. Hypertriglyceridemia will persist in 44 percent of patients three years after transplant, primarily owing to increased triglyceride production. Glucocorticoids may also impair peripheral removal of very low density lipoprotein. Triglyceride serum levels and production rate will be reduced partially toward normal by shifting to alternate day steroids.

In summary, optimal management of the hyperlipidemia associated with chronic renal failure and chronic dialysis would appear to consist of dietary modification, whereby carbohydrate intake is limited to perhaps 30 to 35 percent of total calories, and fat intake increased as necessary to maintain desired caloric intake (table III). Attempts to increase the polyunsaturated: saturated fat ratio to perhaps 1:1 may provide additional benefit. The patient receiving chronic ambulatory peritoneal dialysis may benefit from a greater reduction in dietary carbohydrate intake, as well as minimization of use of excessively hypertonic exchanges. Dietary protein would be administered as outlined earlier in this article. The role and indications for additional measures to control lipids in the chronic renal failure patient remain imprecisely defined.

References


