DIET IN CHRONIC RENAL FAILURE

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Dietary management in chronic renal failure (CRF) has received special attention with its possible role in the stabilization and /or further progression of deterioration, of renal function(l,2). Chronic renal insufficiency is usually defined as a reduction in the glomerular filtration rate (GFR) to 25-50% of the normal and CRF as a rate below 25% of the normal(3). Dietary factors that require modification in CRF include the intakes of protein, energy, minerals, electrolytes and vitamins. Hypertension, proteinuria, intraglomerular hydrostatic pressure and renal parenchymal calcification may also be influenced by the diet(l).

Protein and Energy Intakes

The goals of management of children with CRF is not only symptomatic and preservation of renal functions but also promotion of growth and maturation(4). Experimental studies in the animals have shown that high protein diets promote further glomerular damage(l). Proteins lead to the release of glucagon from pancreas and this is considered to release a hypothetical substance 'glomerulopfessin' from the liver. Increased renal blood flow, glomerular filtration and sclerosis follow leading to decline in renal function(l-5). Animals fed low protein diet develop less severe renal lesions, less proteinuria and live longer(6). A meta-analysis of studies supported the effectiveness of low protein diets in delaying the onset of end stage renal disease(7). However, two recent studies in children given low protein diets (0.8-1.lg/kg/day) observed the similar rate of progression of renal disease after three years when compared with a control group of children fed normally(8,9). Yet another large study has found little evidence that rigorous protein restriction slows the progression of renal disease in humans(10).

With increasing azotemia, the dietary protein should be progressively decreased to maintain blood urea nitrogen levels lower than l00 mg/dl(1,3). Although nutritionally safe low protein diets have yet to be established in CRF. It is usual to recommend the protein intakes of 1.8 g/kg/d to infants 1.0 to 1.5 g/kg/d between 1 to 2 years and 1 g/ kg/d between 2 to 16 years(2).

Protein intake should not fall below RDA levels as growth retardation and failure to thrive become a major concern in CRF(4). Proteins should constitute 6-10% of the calorie intake and 65-70% of the proteins should be of high biological value such as milk, egg, meat, fish, *etc.* However, high phosphate content precludes use of milk as a major source of protein in CRF(1,3,5,11). Although children can effectively utilize the essential amino acids and keto acid analogues,

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there is no evidence that use of low protein formulas result in sustained growth or alter the course of renal insufficiency (4). Besides these diets are costly and have poor compliance(3-5).

Children with CRF should be given 100% of the recommended dietary allowance of the calories and must have ongoing assessment of nutritional status and growth(4). Reduced energy supply results in increased protein, catabolism and growth failure in CRF(1,4,5). Overnight continuous and slow administration of feeds through an intragastric tube is an effective method for the maintenance of energy intakes in infants and young children(2). Such feeds help to encourage the oral intakes during the daytime.

As hyperlipedemia may contribute to continuing glomerular injury, dietary control of lipid intake may be important. Lipids act as a substrate for the production of vasoactive substances, attract inflammatory cells and increase proteinuria leading to glomerular damage. However, hyperlipedemia is very uncommon in CRF in Indian patients(5,12).

Sodium and Water Intake

The capacity of the diseased kidney to excrete sodium and water is impaired with the progressive fall in GFR. Water is lost by solute excretion through the kidney and when combined with a reduced ability to concentrate, urine excretion does not diminish as expected in response to dehydration(l,2). Most children with CRF are able to maintain salt and water homeostasis until end stage renal disease and hence do not need rigorous restriction of salt and water(l,11).

The recommended daily intake of about 2 g of sodium per day is optimally achieved if foods with high salt content are avoided and no salt is added to the diet. Sodium intake should be restricted to 0.5 g in the presence of hypertension, edema or cardiac failure(l,11). Some progressive renal disease such as renal dysplasia and interstatial nephropathy are associated with high urinary sodium losses and may require salt supplements(2). Clinical estimate of excess or poor salt intake in CRF is useful in its management. Excess weight gain and/or edema may be the predominant manifestation of the former; poor weight gain, a high blood urea in relation to creatinine or clinical signs of salt depletion may be indicative of the latter. Optimal attention to the sodium balance is important to achieve steady growth rates(2).

Potassium Intake:

The serum potassium level is usually maintained within the normal until the GFR is reduced to 10-25% of the normal and in the face of moderate variations in the intakes(l,4). However, patients with diabetic nephropathy, interstitial nephropathy and post-obstructive renal damage are at risk to develop hyperkalaemia due to syndrome of hyporeninemichypoaldosteronism(l,4).

Potassium intake is restricted when the GFR declines to 10 ml/min/1.73m^2 and/or in the presence of hemolysis, acidosis, dehydration and gastrointestinal bleeding(2,11). Persistent hypokalemia (<3 mEq/L) requires potassium replacement(l,11).

Acidosis

Metabolic acidosis in CRF increase muscle protein breakdown and de-

creases nitrogren utilization. Further chronic acidosis is mitigated by the buffering of hydrogen ions by the bone salts at the cost of skeletal system(l,2,5).

Alkali therapy should begin with the starting dose of oral bicarbonate 2-3 mEg/kg/d and should be adjusted to maintain serum bicarbonate level above 18 mEq/L(11). As amino acids carry acid radicals, the restriction of protein intake helps to prevent severe acidosis(5).

Calcium, Phosphorus and Vitamin D

As the kidneys are the principal site of activation of vitamin D, they play an important role in the regulation of calcium and phosphorus metabolism. In CRF, osteodystrophy can occur early with a 50% reduction of GFR, while serum creatinine concentration may show little change(3,13).

Restriction of dietary phosphates and proteins in CRF has been shown to prevent renal osteodystrophy. This dietary restriction of phosphates is beneficial even in early renal insufficiency when the serum phosphate level is normal, because it helps in reducing hyperparathyroidism and raise serum calcitriol concentration(4,14). Serum phosphate level should be maintained as low normal by the use of phosphate binders such as calcium carbonate (40% elemental calcium), which is also useful in providing recommended calcium intake. Aluminium hydroxide should not be used in children due to increased risk of aluminium toxicity(2-4,11).

Vitamin D administration is necessary for the treatment of renal osteodystrophy and to promote normal growth. Standard vitamin D is effective in large doses (10,000 to 50,000 IU/day) but more potent forms such as dihydrotachysterol (15 to 45 ug/kg/day) or calcitriol 20 to 60 ng/kg/day are the agents of choice(l,4,ll). Another vitamin D preparation, 1-alpha hydroxyvitamin D_3 may also be in used the maintenance dose of 40-80 ng/kg/day. Careful monitoring of serum and urinary calcium levels is essential to prevent risks associated with these preparations such as hypercalcemia, metastatic calcifications and possibly accelerated course of renal deterioration(3,11).

Vitamins and Minerals

Anemia is a major manifestation of CRF and is characterized by normal indices and a low reticulocyte count principally caused by decreased erythropoietin production. Treatment with erythropoietin may not be effective in the presence of concomitant iron and folate deficiency. Iron and folate deficiencies should, therefore, be corrected if present. Folic acid and pyridoxine requirements may be increased(l,4).

Dietary Counselling

Dietary treatment in chronic renal failure forms an integral part of therapy. However, dietary restrictions in CRF pose practical problems as children do not readily accept dietary changes. Besides anorexia, electrolyte disturbances and multi-drug therapies add to the problems in dietetic modifications.

High energy foods including sugar, jelly, jam, honey, oils and butter are useful adjuncts to supply the recommended daily allowance of the calories(l,5). The intake of high potassium sources like coconut water, dried fruits, nuts and soft drinks need to be avoided. Fresh fruits may be taken in restricted quantities

such as one per day (orange, guava, apple) or one slice of big sized fruit such as pineapple or papaya(5). Cooking of meat and vegetables in large amounts of water and chopping them into smaller pieces leaches out potassium and water soluble vitamins in the cooking liquid. Salty snacks, chips and chocolates should also be avoided. No restriction is required on cooked vegetables, rice, wheat, oils, sugar.

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