Diet In Renal Diseases

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There are only a few renal diseases in which it is possible to influence the changes in the kidney by dietary means. These include many of the inborn tubular disorders which are rare. The main aspects of renal diseases that are being considered here are the dietary management of (a) renal failure, and (b) renal stones.

DIET IN RENAL FAILURE

Carbohydrates and fats can be given freely to the patient with renal failure, but proteins should be prohibited, and minerals and water greatly restricted. This was the basis of Bull’s famous regime, a nauseating concoction of olive oil and glucose, which was administered orally or via an intragastric route, and, if it induced vomiting, the vomitus was strained and given again. More palatable versions of this were devised in our country, with cream and sugar mixed and frozen to make a reasonably palatable ice cream. Fortunately, the advent of dialysis has made this diet obsolete.

In the 1960s, Giordano and Giovannetti showed that restriction of dietary protein improved the symptoms of the patient with chronic renal failure, lowered blood urea and made the patient feel better. This therefore became standard practice, both for chronic and for acute renal failure. Incidentally, protein-rich foods are also abundant in minerals, and restriction of their intake helps the patient in more ways than one.

We continue to practise restriction of protein in the diet, allowing a limited amount of protein of high biological value to provide essential amino acids. The amount of protein should not be reduced below 0.6 gm/kg/day, and this should be of high biological value, as otherwise the patient will be unable to maintain a satisfactory nitrogen balance. This came to be known as the Giordano-Giovanetti diet, and many modifications of this are in use all over the world, and in India. The habitual diets of many healthy Indians do not provide even this amount of protein, and subsisting on this level of protein intake should pose no problem.

A modification of this diet was to reduce the protein intake even below this level and add essential amino acids or their keto analogues. The high cost of these supplements precludes their use in our country. It is essential that adequate energy be provided so that endogenous protein is protected, and 35 kcal/kg/day is the accepted figure.

Progression of chronic renal failure: While our original intention in reducing dietary protein was to produce symptomatic improvement in our patients, it soon became obvious that the rate of loss of residual renal function was faster in patients who did not restrict their protein intake than in those who did. The restriction of protein intake could protect the kidneys and keep them going for a much longer time. This has been amply proved in animal studies, and over a short term in humans, but is still doubtful in the long run in humans, which is what matters to us. It appears from the work of Rosman et al. that protection was only noted in patients who had far advanced disease, and was not very effective in the earlier stages. Further, women were not as much benefitted as men, and only patients with chronic glomerulonephritis had significant change in the rate of decline in renal function.

The mechanism of such protection seems clear on animal studies. Amino acids release glucagon from the kidneys, and this releases a still hypothetical substance known as angiotensin from the liver. This increases renal blood flow and glomerular filtration in remnant nephrons, and this has been shown to promote focal glomerular sclerosis in the glomeruli, thus accelerating the decline in renal function. All amino acids, and therefore all proteins, do not have this effect. It seems safe to allow vegetable proteins in greater measure.

There is also evidence to suggest that high phosphorus intake is detrimental to the kidneys, and meat and milk contain an abundance of phosphorus in highly absorbable form. Further, meat is a very rich source of potassium, and the restriction on its intake can be virtually life saving to patients with chronic renal failure.

Keto analogues of essential amino acids, given with a very low protein diet, have been used to slow progression of renal failure, and are effective in patients who are compliant. However, compliance is a serious problem and the cost of these keto-acids is high. These measures are successful only in moderate renal failure, and not when the disease is far advanced.

I remain sceptical about the ability of protein restriction to reduce the rate of decline of renal function in chronic renal failure, as so many of our patients consume far less amounts of protein than the figures recommended, and yet the decline continues at more or less the same rate as that described in more affluent societies. There is, however, the need to maintain some degree of restriction, especially on animal protein, because symptoms are less in patients on protein restricted diets, and also because these diets have less phosphorus and acid radicals.

Lipids contribute to continuing glomerular injury. Macrophages ingest lipoproteins and are stimulated to produce growth factors which promote collagen synthesis and proliferation of mesangial cells. Lipids serve as a substrate for the production of eicosanoids, which are vasoactive and increase proteinuria, with its harmful effects on the glomerulus, and also attract inflammatory cells. Lowering of lipid levels slows the deterioration in renal function in glomerular diseases associated with the nephrotic syndrome. The use of fish oil shifts eicosanoid synthesis to less vasoactive and chemotactic metabolites, decreases proteinuria, and reduces glomerular scarring. The need to lower cholesterol levels in nephrotic patients, with or without renal failure, is thus established. However, hyperlipidaemia is very uncommon in chronic renal failure in Indian patients.

Potassium: Hyperkalaemia can lead to sudden death in chronic renal failure, and should be guarded against. The capacity of the kidney and of the colon to excrete potassium increases in chronic renal failure as there is increased activity of the renin-angiotensin-aldosterone system. When renin production is defective as in dia-
betics, the risk of developing hyperkalaemia is far greater.

The Indian diet and mode of cooking offer advantages for the patient with chronic renal failure. The major source of potassium is meat, and even non-vegetarians in India do not consume meat in large quantities as in the West. Further, our common mode of cooking meat and vegetables, chopping them into pieces and boiling them in water, leaches out the potassium. My experience has been that only a small minority of Indian patients have dangerous levels of serum potassium. These patients should be watched and appropriate precautions taken. For the rest, it should suffice to caution against taking too much potassium.

Acidosis: Severe acidosis can cause tachypnoea by stimulating the respiratory centre. It also causes venous contraction which increases venous return to the heart, and reduces cardiac contractility, and the two together produce pulmonary oedema. It is thus a life-threatening situation. Furthermore, some of the acid is buffered within cells in exchange for potassium and therefore acidosis causes hyperkalaemia with its attendant dangers. Chronic acidosis is buffered in bone, with the loss of calcium, and osteodystrophy results. It is therefore wise to keep serum bicarbonate levels above 18 mEq/l. As amino acids carry acid radicals, the restriction of protein intake helps to prevent severe acidosis. Severe acidosis in Indian patients is a rarity.

Salt and water: The capacity of the diseased kidney to excrete salt and water varies from patient to patient, and therefore the intake of these two items must be individualised. Whereas in other oedematous states it is enough to restrict salt intakes, in renal failure the capacity of the kidney to excrete free water is also impaired, and therefore a limit must be placed on water intake too.

Recommendations for diet in chronic renal failure: The average Indian diet is especially suitable for the management of chronic renal failure, and not much modification is needed. To most patients, I prescribe a very simple menu which is as follows:

- Avoid high potassium sources like coconut water, dried fruits and nuts, and soft drinks. Only one fresh fruit per day is advisable (in the case of a large fruit like papaya, one slice). If salad is consumed, it is best to avoid fruit on that day.
- No restriction on cooked vegetables, rice, wheat, oils, sugar. Suitable restriction must be added for the rare patient with hyperlipidaemia.

If the patient is oedematous, there should be no salt in the food and none should be added at the table, and the liquid intake should be restricted to 300 ml. This is in addition to the milk which has been permitted as indicated earlier. It is important to place a limitation on all liquids and not only on water, as otherwise considerable amounts of water may enter the diet in the form of gravies. The Indian diet being extremely sloppy in consistency. It should not be forgotten that metabolism of solids will add approximately 1 litre of water to the body daily. Once the patient is free from oedema, salt and liquid intake may be gradually increased till the limits of tolerance are reached.

The patient with predominant interstitial changes and also in a salt losing state may, however, require salt supplements and large quantities of water.

Special problems in children: It is difficult to find the suitable diet for children with chronic renal failure, and there is no unanimity of opinion on what should be done. Children are less likely to accept diet restrictions, and if they do not get what they want, they are likely to stop eating and get undernourished. All calculations regarding the protein and calorie requirements mentioned for adults are complicated in children by the fact that we must make allowance for the proteins needed for growth. It is fairly well accepted now to give the child the recommended dietary allowance for his or her size and age. One could give 15 kcalories per cm height, and 1.5 to 2 gm protein per 100 kcal. What I usually do is to let the child take a normal diet except to avoid dangerous doses of potassium.

The nephrotic syndrome: The old idea of forcing a high protein diet on nephrotic patients has now been abandoned. A high protein intake leads to hyperfiltration and increased proteinuria, and is therefore self-defeating in nephrotics. I would normally allow the patient to continue on her normal protein intake. Lipids need to be regulated.

Again, the dietary management of the nephrotic syndrome in children poses problems. A high protein diet increases proteinuria, and makes it more difficult to control the nephrotic state. However, one cannot restrict proteins in a child, because one has to provide enough for growth. Fortunately, most cases of the nephrotic syndrome in children are due to minimal lesion nephropathy which responds to steroids in a short time and do not require modification of diet. For others, it has been suggested that we give 3 to 4 gm of protein/kg body weight/24 hours. My usual practice, however, is to give a normal diet and adjust only the salt intake as called for.

**RENAL STONES**

The most common renal stones are those containing calcium, usually in the form of oxalate. Calcium phosphate stones are usually associated with renal tubular acidosis, and struvite stones with infection. But there is not much role for dietary management. Uric acid stones are to some extent dependent on the diet. I will therefore consider dietary management of calcium oxalate and uric acid stones.

Calcium oxalate stones: Calcium is one of the ingredients of the stone. It seems obvious that the more the calcium in the diet, the more will be excreted in the urine, and therefore the greater will be the tendency to stone formation. However, this is simplistic reasoning. Intestinal absorption of calcium is tightly regulated, and depends to a great extent on the calcium content of the body. If the body is replete with calcium, absorption is low, and correspondingly urinary excretion is low too. Some stone formers have specific defects of tubular excretion or intestinal absorption of calcium. Calcium restriction in the latter could lead to deficiency in the body, and to secondary hyperparathyroidism. The only clear indication for dietary restriction of calcium would be intestinal hypercalciuria. Patients consuming large amounts of milk or milk products may be advised to restrict themselves to a cup of milk and one of curds each day.
The possible harm from a low calcium intake is that calcium forms a complex with oxalate in the bowel which is not readily absorbed, and thus the dietary oxalate is kept in the bowel and passed in the faeces. In the absence of calcium, oxalate is more readily absorbed, and secondary hyperoxaluria may result.

Oxalate is the end product of the metabolism of a number of substances. About 40 per cent comes from ascorbic acid, and 50 per cent from glycolate, glycine, and hydroxyproline. These are components of proteins, and high protein intake would therefore increase their excretion in the urine. Only about 10 per cent of urinary oxalate is directly derived from dietary oxalate containing foods, and therefore the scope for reducing urinary oxalate excretion by dietary restriction of oxalate-rich substances is small. It would be wise to avoid excessive intake of the following items especially rich in oxalate, namely leaves including the numerous varieties of edible greens and tea, nuts, chocolate, and beetroot. It is not necessary to ban these items entirely.

As we have already seen, diet is responsible for only 10 per cent of oxalate in the urine. An excessive intake of ascorbic acid is similarly inadvisable, and so very large amounts of citrus fruits and juices should be avoided. Again, a total embargo is not called for as the pathway of the conversion of ascorbic acid to oxalate is readily saturated. It is to be noted that tomato does not figure anywhere on this list. Every stone former in the country, and many people with other renal diseases, are being deprived of the use of this tasty component of our diet, for no valid reason. It is neither very high in oxalate content nor in ascorbic acid.

There is a close relationship between renal tubular reabsorption of sodium and calcium. When the body is replete with sodium, tubular reabsorption of water from the proximal convoluted tubule is diminished, along with all the solutes in it. There will therefore be more calcium in the urine. Conversely, in a sodium depleted patient, sodium along with calcium will be reabsorbed more from the proximal tubule. In the distal tubule, reabsorption of calcium is largely charge-related. If more sodium is available for reabsorption, the contents of the tubular lumen will be relatively negative, and this will tend to retain the positively charged calcium ion in the lumen.

While there is no evidence to link a high sodium intake with the development of calcium containing renal stones, it is certainly helpful to restrict sodium intake moderately when attempting to treat patients, as this lowers the amount of calcium in the urine.

A very high protein intake is associated with renal stone formation. Protein increases glomerular filtration, and therefore calcium will increase. There is increased acid production with increased protein intake, and this reduces citrate excretion in the urine. More citrate is absorbed from the proximal tubule when acid excretion is high, and more is utilised for gluconeogenesis in the cell. Urinary citrate chelates calcium and forms a readily soluble product which takes calcium away from stone formation.

Protein also increases urinary uric acid excretion, and uric acid is a promoter of calcium oxalate precipitation in the urine. For all these reasons, it is advisable to restrict animal protein intake in stone formers.

The single most important dietary manipulation of benefit in stone formers is the consumption of large volumes of water. Water dilutes all the salts in the urine, and takes them out of the stage of unstable supersaturation in which stones are likely to form. The larger the liquid intake, especially at night, the less the chance of stone formation. It should be noted that the patient should actively force liquid intake during the night. My advice is to drink a glass of water every hour, two on retiring to sleep, and two more when he wakes up to pass urine.

There is a theoretical objection to drinking large amounts of water. There are inhibitors of stone formation in the urine, and they may be diluted by the water and thus be less effective. However, clinical evidence indicates that people are much more likely to form stones in hot dry regions in the absence of enough water to drink, and less likely if they drink water copiously.

Uric acid stones: The tendency to form uric acid stones is not related as well to the actual amount of uric acid as it is to the pH of the urine. Uric acid itself is relatively insoluble, while sodium urate is far more soluble. So-

trium urate forms in an alkaline urine and the relative amount of uric acid is directly proportional to the acidity of the urine. Vegetables give rise to alkali in the body, and protein of animal origin to acid, as already discussed. Meat intake is harmful to the uric acid stone former, both because it contributes a large amount of uric acid from purine metabolism and because it renders the urine acidic, as already discussed. The best treatment for a uric acid stone former is to become a vegetarian! Among meats, offal (internal organs like liver, brain and pancreas) and red meats are especially harmful, white meat relatively less so.

As with calcium stones, a large liquid intake is also beneficial.

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References