

INTRODUCTION

Globally the number of people with Diabetes is increasing rapidly and the latest data¹ reveal there are 415 million adults with Diabetes (India 69.2 million).

Chronic Kidney disease is common in India and worldwide. The Screening and Early Evaluation of Kidney Disease (SEEK) study estimates the prevalence of CKD in India at 17.2%². Diabetes Mellitus is the commonest cause of CKD in India (31%)³ and globally.

Dietary adjustments are an important part of the care plan for Diabetic CKD. Dietary indiscretion in a patient with diabetic nephropathy contributes to the severity and rapid progression of CKD. Conversely the timely and right kind of dietary intervention plays a significant role in controlling the progression of CKD. Among CKD patients, over nutrition results in sodium and volume overload, hyperkalaemia, hyperphosphatemia and accumulation of toxic metabolites of protein degradation. Undernutrition, on the other hand, exacerbates the risk of malnutrition. As GFR decreases, the nephron is less able to handle potassium, phosphorous, sodium and acid levels. The optimal diet for individual DKD patients varies depending upon the eGFR, proteinuric or nonproteinuric status, and the presence of other co-morbidities such as hypertension or heart failure.

DIETARY PROTEIN

Both quantity and quality of protein and amino acids have been identified to be important for maintenance of adequate nutritional status in diabetic CKD. Identification of optimal dietary protein intake is complicated because it is known that kidney disease confers unique metabolic abnormalities that can include alterations in mineral metabolism, metabolic acidosis, anaemia, vitamin D deficiency, loss of lean muscle mass and susceptibility to malnutrition. Studies that have examined protein restriction have yielded inconsistent results, but the balance of evidence suggests a benefit of moderate dietary protein restriction. The NKF KDOQI⁴ recommends a target protein intake of 0.8 g/kg/d for non-dialysis DKD. KDIGO⁵ also suggests a dietary protein intake of 0.8 g/kg/d in diabetic adults with eGFR <30 ml/min/1.73 m². The ADA⁶ recommends “usual” (not high) dietary protein intake. In patients with moderately severe and more severe CKD once protein intake is reduced by 0.2 gm of protein/kg/d for one year the baseline values of serum bicarbonate, phosphorus and urea nitrogen are remarkably lower⁷. The benefits will compound if enough essential and non-

essential amino acids are included in the diet to synthesize protein while avoiding the accumulation of unexcreted, potentially toxic ions and compounds arising from the breakdown of foods rich in protein.

A severely deficient diet can lead to muscle mass loss, although more commonly as complications of CKD such as acidosis or inflammation which activate the enzymes that breakdown protein to cause loss of protein stores. Catabolism will not be halted by prescribing an excess of protein in diet, instead it will lead to accumulation of unexcreted, potentially toxic ions like phosphates and potassium. The outcome is an increasing risk of developing acidosis, hyperkalaemia, hyperphosphatemia, edema, a high serum urea and BUN but no increase in muscle mass.

Besides reducing waste products, benefits of a protein restricted diet include suppressing proteinuria, improved control of 1) blood glucose 2) hyperlipidaemia 3) BP 4) renal bone disease and 5) metabolic acidosis. Decreased albuminuria is associated with slower progression of diabetic CKD.

As eGFR declines, appetite decreases and malnutrition may manifest. Body weight and serum albumin is used to monitor nutritional status. Hypoalbuminemia may result from reduced protein and/or calorie intake, uraemia, metabolic acidosis, albuminuria, inflammation, or infection. However, a properly monitored diet prevents malnutrition even when eGFR is below 10 ml/min. Avoidance of malnutrition is especially important in CKD stages 4 and 5 due to their susceptibility to infections. Patients with better nutritional status during dialysis have better outcome.

What about the progression of CKD? The conclusions from MDRD⁸ study suggest that low protein diet did not significantly slow down the loss of GFR. However, it was subsequently observed that those patients who reduced the dietary protein by 0.2 gm/kg/d had a reduced loss of GFR of 29%, which translated into a 41% increase in time to dialysis or death⁹. The probable reason for this is that a low protein diet can reduce proteinuria which has been proposed as a major factor in progressive loss of GFR. Diet rich in protein from plant sources may be beneficial among CKD patients. Such a diet may reduce proteinuria, slow the progression of CKD, decrease the production of uremic toxins, lower phosphorus intake, and potentially decrease mortality risk. In patients with nephrotic syndrome protein restriction is not recommended.

So the various recommendations suggest that in

diabetic CKD, dietary proteins should be limited to 0.8-1.0 g protein/kg/day to prevent accumulation of acid, phosphorous and uric acid. However, patients on dialysis will need more protein intake. A high-protein diet (KDOQI recommendation of 1.2 gm/kg/day for haemodialysis and 1.3 gm/kg/d for peritoneal dialysis patients) with fish, poultry or eggs at every meal may be recommended. This will prevent malnutrition.

CARBOHYDRATES AND FATS

Whole-grain carbohydrates and fibre and fresh fruits and vegetables are recommended as part of a healthy diet for individuals with DKD. The number of portions and specific food selections from these food groups often need to be limited in advanced stages of CKD due to the potassium and phosphorus loads imposed by these foods. Literatures suggest beneficial effects of omega-3 fatty acids on albuminuria in DKD¹⁰. The general recommendation for DKD is to include omega-3 and omega-9 fatty acids as part of total dietary fat intake while decreasing intake of saturated fats and food sources of trans fatty acids.

SODIUM AND POTASSIUM

1 g of salt contains 0.4 g (17 mEq) of Na ion. Sodium plays a large role in blood pressure control in CKD as a result of alterations in sodium excretion by the kidneys. Sodium intake should be limited to 2,300 mg a day or less. The approach for patients with reduced eGFR who do not have hypertension, volume overload, or increased protein excretion is not clear.

Among CKD patients, the benefits of salt restriction might include the following:

- Lower blood pressure (BP)
- Slower progression to end-stage renal disease (ESRD)
- Improved cardiovascular outcomes

Hyperkalaemia usually occurs when eGFR is less than 20 ml/min/1.73 m². KDOQI recommends potassium intake between 2 to 4 g/day (51-102 mEq/day) for patients with CKD stages 3 to 4, while recommending no restriction for those in earlier stages of CKD.

In stages 4 and 5 CKD fluid restriction is also required.

Phosphorous retention can lead to metabolic bone disease and cardiovascular disease. Dietary phosphorus intake is restricted to a maximum of 0.8 to 1 g/day to normalize the serum levels in patients with an eGFR <60 mL/min/1.73 m².

Decreased vitamin D production in Diabetic CKD can lead to hypocalcemia. Maintaining a calcium intake of 1.0 to 1.2 g daily will help prevent hypocalcemia.

CALORIE INTAKE

Weight loss leads to improved BP, better glycaemic control, reduction of hyperfiltration and proteinuria¹¹. Since HTN, Proteinuria, hyperfiltration are all risk factors for progression of DKD, this suggests that reduction of

calorie intake in obese DKD will delay the progression of DKD.

Finally approaches to incorporating diet patterns for diabetic CKD patient (50kg weight) will be as follows:

1. There is no dietary restriction for patients with eGFR>60 ml/min/1.73m². They should follow the diet of diabetic populations.
2. Milk and non-fat dairy products (like yogurt, cheese) less than half litre a day.
3. Incorporate vegan protein sources into meal plan like pulses 4tsp (raw weight) per day, dried beans and peas, legumes, nuts and seeds.
4. For non-vegetarian patients, avoid intake of fatty animal protein sources like red meat, poultry with skin and shellfish. Fish or chicken 30-50gms/day can be substituted.
5. Include high-fibre, wholegrain products (whole/mixed-grain breads, pastas, cereals; brown rice), avoid refined white flour based products (noodles, maida).
6. Fresh fruits and vegetables of choice, fresh cooked vegetables are ideal. If potassium is to be restricted citrus fruits, peaches, sapota etc. are to be avoided along with vegetables like avocado, potatoes, tomatoes, pumpkin and spinach. Cabbage, carrots, cauliflower, celery and cucumber can be substituted. To reduce potassium content, vegetables need to be leached (wash, peel, cut in small pieces, soak in water for sufficient time and the water discarded). To minimize sodium content of diet, provide freshly cooked food. Avoid tinned and canned readymade food, sauces, cheese, soups, popcorn, commercial salad, salted pickles which has high sodium content. Sources rich in inorganic phosphate such as highly processed foods should be avoided because inorganic phosphate has much higher bioavailability.
7. Diet needs to be enriched with olive oil, fish oil, and vegetarian sources of omega-3 fatty acids.

SUMMARY AND RECOMMENDATIONS

Low protein, low potassium, low phosphorous, moderate carbohydrate and high fibre diet have been recommended to DKD patients in order to control blood sugar levels and delay progression of CKD. The diet of every patient needs to be individualized depending on the tendency to retain or lose salt and the serum levels of protein, potassium, phosphorus and lipids and finally the overall nutritional status and daily urine output of the patient. For most DKD patients, the optimal diet is one similar to the Dietary Approaches to Stop Hypertension (DASH) diet, consisting of fruits, vegetables, legumes, fish, poultry, and whole grains.

A skilled dietician will incorporate a patient's food preferences, adequate calories and a proper distribution

612 of foods while encouraging compliance. Fortunately, the majority of CKD patients accept dietary changes.

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