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Review Article

Nutritional management of diabetic nephropathy

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ABSTRACT

Diabetic nephropathy (DN) is characterized by albuminuria, which is usually accompanied by hypertension, progressive rise in proteinuria. There are several approaches to delay progression of diabetic nephropathy towards end stage renal failure (ESRD). Current approaches include a) control of blood glucose; b) low-protein diet; c) control of hypertension; restriction of dietary salt, phosphorus and potassium in advanced cases d) control of hyperfiltration, usually through angiotensin-converting enzyme inhibitors or angiotensin-receptor blocking agents. Nutrition management is fundamental for the prevention of diabetic nephropathy to ESRD.

Diabetic nephropathy (DN) is the leading cause of end stage renal failure (ESRD) in the past two decades.[1,2] It is characterized by albuminuria, which is usually accompanied by hypertension, progressive rise in proteinuria (albuminuria >0.5 g/24 h), and decline in renal function. Fig. 1 illustrates interaction between metabolic and hemodynamic pathways in the pathophysiology of diabetic nephropathy.[1,2] DN carries a 20- to 40-fold increased risk for cardiovascular (CV) mortality. To delay progression of DN towards ESRD following measures are recommended a) control of blood glucose; b) low-protein diet; c) control of hypertension; restriction of dietary salt, phosphorus and potassium in advanced cases d) control of hyperfiltration, usually through angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blocking (ARB) agents.

Nutrition management is fundamental for the prevention of DN. Therefore goals of medical nutritional therapy which will be dealt in this article are:

1. Maintenance of near normal blood glucose levels (glycemic control) by controlling food intake and exercise
2. Achieving optimal serum lipids and blood pressure to reduce the risk of cardiovascular disease (CVD)
3. Management of body weight
4. Maintaining biochemical parameters and fluid status
5. Prevention of long term complications
6. Prevention of malnutrition and strategies to control diabetic gastroparesis.

1. Maintenance of glycemic control

The Diabetes Control and Complications Trial (DCCT 1993) and the United Kingdom Prospective Diabetes Study (UKPDS) have shown that intensive insulin therapy can significantly reduce the risk of the development of microalbuminuria and overt nephropathy.[3] Target HbA1C should be <7.0%. However,

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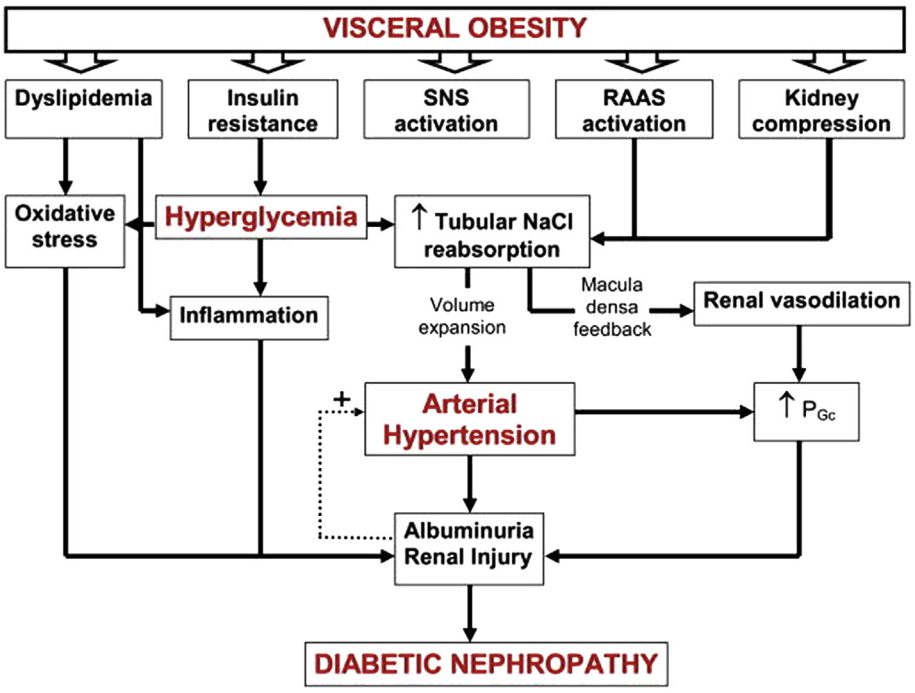

Fig. 1 – Interaction between metabolic and hemodynamic pathways in the pathophysiology of diabetic nephropathy. SNS: sympathetic nervous system; RAAS, renin angiotensin aldosterone system; P_{Gc}, intraglomerular capillary pressure (Christine Maric and John E. Hall. Obesity, metabolic syndrome and diabetic nephropathy. *Contrib Nephrol*. 2011; 170: 28–35).

the values may be falsely elevated or decreased in patients with chronic kidney disease (CKD) because of reduced red blood cell life span, blood transfusion, iron deficiency, accelerated erythropoiesis due to administration of erythropoietin stimulating agents, and metabolic acidosis. According to KDOQI Guideline 11 for management of DN, all diabetic dialysis patients should follow the American Diabetes Association (ADA) guidelines.[4,5] ADA recommendation is based on evidence from the Diabetes Control and Complications Trial (DCCT).[6] Several trails on intensive versus conventional control of blood glucose levels using insulin therapy and oral hypoglycemic medications in order to control complications like retinopathy and nephropathy[7–9] concluded that careful control of blood glucose has a significant effect on decreasing the complications of diabetes. Tight glycemic control can have potential problems for patients on dialysis. Care should be taken to prevent hypoglycemic episodes in patients on dialysis who experience significant nausea or gastrointestinal complaints. If with good glycemic control nutritional status improves and patient gains body weight (which is not due to volume overload), then clinical dry weight should be readjusted for the purpose of postdialysis weight targets. For non-dialysis diabetic CKD patients, treatment should be initiated with oral hypoglycemic drugs rather than insulin. If estimated glomerular filtration rate (eGFR) <30 mL/min per 1.73 m^2 metformin should be avoided because of an increased risk of lactic acidosis. According to ADA, after five years of diagnosing DM microalbuminuria must be tested annually. Microalbuminuria is also associated with elevated HbA1C levels (>8.1).

Amount and type of carbohydrate (CHO) in food influences blood glucose levels.[10] There are two approaches to manage CHO intake; 1. keep amount of CHO constant and 2. CHO counting. A fixed CHO meal plan is useful for patients who use only diet to control their blood sugar or who are on fixed doses of insulin or other hypoglycemics. In this approach amount of CHO intake is kept constant and the timing of food intake is fixed. Patients who follow CHO counting method, adjust insulin according to intake. However, this approach requires good understanding of CHO amount in food item. Food exchange list is often handy in such situations so that patients can trade between different groups of foods. Table 1 shows the exchange list for meal planning. Patients who are on peritoneal dialysis, glucose contained in the dialysate (peritoneal dialysate) may increase the requirement of hypoglycemic agents.

Fat and protein intake also have to be monitored in this approach. According to ADA CHO should be derived primarily from whole grains and non-fat or low dairy products.[12,13] However, with decreasing kidney function food choices and portions may need to be adjusted to maintain serum potassium and phosphorus within normal ranges. If patients can be educated on importance of glycemic index (Table 2) and glycemic load it will help them in having better control of blood sugar. Low GI is value under 55, moderate GI is 56–69 and high GI is greater than 70. Fiber intake can reduce blood sugars in diabetic patients. ADA recommends fiber intake of 14 g/1000 kcal which can be increased up to 35 g. Table 3 and 4 show low sodium and potassium vegetables and fruits. Milk and milk products are high in phosphorus (1 g of protein bring