Microalbuminuria in the course of familial Mediterranean fever

Sir,
In their interesting paper, Lobato et al. [1] studied the importance of microalbuminuria as a predictor of symptomatic disease and clinical nephropathy in familial transthyretin related amyloidosis. They demonstrated that microalbuminuria represents a first stage of clinical amyloid nephropathy and that it could be premonitory of neuropathy. They recommend the systematic and regular search for urinary albumin excretion in symptomatic gene carriers and patients, instead of renal biopsy, as a non-invasive method for estimating the progression of amyloidosis.

Previously, it has been shown that in cases of essential hypertension and diabetic nephropathy, early renal glomerular damage can be detected by microalbuminuria [2]. Saatci et al. [3] previously showed increased urinary \( \beta \)-2-microglobulin excretion and microalbuminuria during familial Mediterranean fever (FMF) attacks. FMF is characterized by recurrent fever and serositis. The most important complication of the disease is AA amyloidosis. Proteinuria and renal failure may complicate the clinical course. We have measured the microalbuminuria and urinary glycosaminoglycan (GAG) levels as predictors of amyloidosis in patients with FMF in an attack-free period. We found that mean urinary GAG levels of the patients with FMF secondary amyloidosis were lower than those of FMF patients without amyloidosis [4]. When we examined the duration of disease in the FMF group without amyloidosis, we saw that median GAG levels were significantly lower in patients having the disease for >10 years compared with those having the disease for <3 years. Also, microalbuminuria levels tended to increase with the duration of the disease. In FMF patients with low GAG levels, colchicine dose was increased and urinary GAG and microalbuminuria levels were re-examined after 2 weeks. In these patients, urinary GAG levels increased significantly with increments in the colchicine dose. Microalbuminuria decreased along with the increase in urinary GAG when colchicine dose was increased.

These results suggest that urinary GAG and microalbuminuria may be predictors of amyloidosis in FMF patients. We also suggest that effective colchicine dosing may be monitored by following urinary GAG excretion and microalbuminuria. Our results are in agreement with the recommendation by Lobato et al.

Conflict of interest statement. None declared.

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High protein diets may be hazardous for the kidneys

Sir,
With the promise of rapid weight loss and better health, many patients are turning to high protein, low carbohydrate diets. ‘Low carb’ diet books are topping the New York Times Bestseller lists week after week, and are being touted as quick fixes to today’s epidemics of obesity and type 2 diabetes. It must be acknowledged that these diets improve weight, sugars and cholesterol in the short term, but at what risk for a patient with renal disease?

From a nephrologist’s point of view, there are two major concerns raised by such high protein, low carbohydrate diets. The first is worsening of proteinuria in patients with renal disease and the second is the development of nephrolithiasis in otherwise healthy people. Diabetic nephropathy is the leading cause of end-stage renal disease (ESRD) worldwide. Weight loss and improved glycaemic control are critical to attenuation of renal disease progression in these patients, but a highly significant predictor of progression is proteinuria [1,2]. Many animal and human studies of diabetic and other renal diseases have demonstrated that high protein diets exacerbate proteinuria and accelerate renal disease progression [3,4]. In addition, diets with high sodium and purine intakes are associated with increased risk of nephrolithiasis [5]. The current emphasis on high protein, low carbohydrate diets poses a dilemma: patients may benefit from weight loss and improved metabolic parameters but, if the potential cost is more rapid progression towards ESRD or the development of kidney stones, is the trade-off worth it?

We suggest that renal patients should be referred to a registered dietitian prior to considering these diets, and that meal plans should be carefully ‘tailored’ to meet individual needs. To maximize benefit and minimize potential harm, food choices should be modified to include low sodium, lower protein (0.8–1 g/kg/day) options, avoidance of processed foods, focus on more healthy fats, gradual inclusion of ‘good’ carbohydrates and adequate hydration.

At present, long-term data on cardiovascular and renal outcomes in patients following high protein, low carbohydrate diets are lacking. Until we have evidence that the benefits in terms of weight loss, glycaemic control and lipids outweigh potential adverse renal consequences, we suggest that physicians exercise caution in encouraging patients with renal disease to follow these diet plans without appropriate supervision.

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