We present a patient with the metabolic syndrome who demonstrated microalbuminuria, an index of cardiovascular risk (1), at the time of presentation. The patient was treated with an intensive regimen of dietary and exercise changes, and all diagnostic criteria for the metabolic syndrome resolved, followed by resolution of the microalbuminuria.

**HISTORY AND EXAMINATION** — A 43-year-old man presented to his primary care physician for a “new patient evaluation.” Xerostomia, polydipsia, and polyuria, which had been attributed to chronic rhinosinusitis, were noted. A normal fasting blood glucose level had been recorded just before the onset of these symptoms, ~5 years prior the visit. Physical examination and laboratory evaluation revealed the presence of metabolic syndrome, encompassing the following diagnoses: central obesity, hypertension, type 2 diabetes, hypertriglyceridemia, and low HDL (data below). The patient was started on aspirin at 81 mg once per day and glipizide extended release at 5.0 mg once per day. He was also told to lose weight through a program of exercise and dietary interventions.

**INVESTIGATION** — Compliance with the 1,500-calorie American Diabetes Association diet was self-taught and achieved through portion-control, and a change in content to decrease caloric density (i.e., increase in uncooked whole fruits and vegetables, especially carrots, and decrease in meats, juices, and processed carbohydrates). Dietary fat remained at ~35% of total calories but with a shift from animal to vegetable sources (walnuts, almonds, peanut butter, legumes, and plant stanols). Exercise was begun with 1 day off on odd weeks and 2 consecutive days off on even weeks. The exercise regimen was implemented at the local health club and consisted of running on a treadmill (or occasionally outdoors), hyper-elliptical cross-training, and upper-body resistance training. The initial regimen resulted in a burn of 600 calories per day in two sessions of 60-min duration. After 6 weeks of glipizide use, palpitations and lightheadness attributed to hypoglycemia were noted, and the medication was discontinued. Exercise intensity was increased as tolerated (in very small steps every 2 weeks), eventually reaching 1,000 calories in a single session lasting 90–120 min. As the calorie burn was increased, the diet was liberalized to maintain a BMI of 23.0 kg/m². The regimen of aspirin, diet, and exercise was continued, with evaluation by the primary care internist at 3-month intervals.

Components and correlates of the metabolic syndrome responded to the regimen over 27 months as follows: fasting blood glucose decreased from 380 to 95 mg/dl (normalized within 3 months), fasting serum triglycerides decreased from 320 to 41 mg/dl (normalized within 3 months), blood pressure decreased from 140/90 to 105/65 mmHg (normalized within 9 months), HDL cholesterol increased from 37 to 61 mg/dl (normalized within 9 months), LDL cholesterol decreased from 210 to 88 mg/dl (normalized within 9 months), and BMI decreased from 33.0 to 23.0 kg/m² and decreased to <26.0 within 3 months (target of 23.0 achieved at 15 months). Later normalization (within 24 months) of microalbuminuria was noted (from 70 to 9.0 mg protein/g urinary creatinine).

**CONCLUSIONS** — The current report is the first documented instance of metabolic syndrome–related microalbuminuria (2) resolving with changes in exercise (3) and diet alone; the early 6-week oral hypoglycemic trial is unlikely to have affected outcomes at 27 months. The recent landmark study of Gaede et al. (4) clearly demonstrates a significant decrease in vascular risk and proteinuria resulting from a therapeutic approach integrating changes in exercise and diet with medications to correct the metabolic syndrome elements in people with type 2 diabetes and proteinuria. However, Gaede et al. did not explore either the relative importance of lifestyle changes as
opposed to medications in the beneficial outcomes or the time-course of resolution of the metabolic syndrome components and correlates.

The late response of this case of microalbuminuria is consistent with the concept that in diabetes and in the metabolic syndrome, the level of albumin in the urine integrates the degree of microvascular injury resulting from hyperglycemia, hypertension, and dyslipidemia. Cholesterol abnormalities and blood pressure also responded relatively slowly. The slow progress was despite immediate changes in exercise and dietary behavior and relatively rapid changes in body mass, although the target body mass was achieved slowly. These findings indicate that even suboptimal weight loss is likely to result in some improvement in the metabolic syndrome, at least if it is associated with increased exercise. Furthermore, the slow response of body mass, hypertension, hypercholesterolemia, and microalbuminuria may be typical and could contribute in some cases to the failure of compliance with lifestyle interventions in the long term. Practitioners should be aware of the slow pace of improvement in these important indexes of vascular health and consider reinforcing the longer-term benefits of lifestyle modification to encourage patients to continue to comply with the regimen.

References