Letter to the Editor

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Effects of Weight Reduction and Angiotensin-Converting Enzyme Inhibition on IgA Nephropathy-Associated Proteinuria

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to 102 mm Hg in the 12 months after its initiation.

As demonstrated in patients with obesity-related hyperperfusion glomerulopathies [1], either ACE inhibitor therapy or weight loss reduced urinary protein excretion in this obese patient with IgA nephropathy. Previous studies [2] have demonstrated a significant antiproteinuric effect of ACE inhibi-

Dear Sir,

Observations over 8 years on a 45-year-old obese woman with IgA nephropathy add to those of Praga et al. [1].

Serial urinalyses had detected onset of microscopic hematuria in 1982 at age 32, and addition of very limited proteinuria from 1983 until evolution of nephrotic range proteinuria in early 1988. Renal biopsy findings at that time were fully consistent with IgA mesangial proliferative glomerulopathy. Brief episodes of gross hematuria accompanied intercurrent viral illnesses in 1987 and 1990. Serum creatinine has remained stable at 1.0 mg/dl (0.9-1.1 mg/dl; 80-97 µmol/l). The serum albumin is consistently normal.

Periods of weight reduction while following a 1,000 kcal/day diet in early 1988, and during consumption of a 420-kcal/day liquid dietary preparation between June and December 1989, and again between June and October 1990 were paralleled by prominent reductions in proteinuria and serum cholesterol (fig. 1). Subsequent intervals of weight gain were accompanied by resumption of previous levels of urinary protein excretion and hypercholesterolemia. Lova-statin (20-30 mg/day) in conjunction with a low cholesterol diet allowed reduction in serum cholesterol of approximately 100 mg/dl (2.59 mmol/l) over a period of 8 months. Addition of lisinopril 2.5 mg/day in September 1992 (5 mg/day since June 1993) induced a dramatic, sustained 60% reduction in proteinuria, despite continuing weight gain. Mean arterial pressure declined from 111 mm Hg during the year prior to angio-

tensin-converting enzyme (ACE) inhibition

Fig. 1. Variation in body mass index (BMI), proteinuria, and serum cholesterol over 8 years in a patient with IgA nephropathy.
tion in IgA nephropathy. Observations in this patient help to confirm the speculation of Praga et al. [1] that glomerular hyperperfusion and hyperfiltration universally present in the massively obese initiate or accentuate clinical proteinuria in the few of these patients with an additional predisposing factor, such as a solitary functioning kidney or incidental presence of a primary

References


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