Effects of Dietary Lipids and Lovastatin during the Course of Nephropathies

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Dear Sir,

In a recent paper Samuelsson et al. [1] suggested that an altered metabolism of lipo-proteins containing apolipoprotein B (ApoB) has a significant effect on the progression of renal failure. Taking into account that hyper-lipoproteinemia and also glomerular ApoB deposition formation might play a role in this process and in the pathogenesis of some glomerular diseases, the attempt of a lipid-lowering therapy is justified. This study was designed to investigate the role of a reduction of serum lipid levels during the course of nephropathies with nephrotic hyperlipidemia.

Twelve patients (6 males and 6 females, age range 30-69 years) with focal-segmental glomerulosclerosis (FSGS), membranous nephropathy (MN), minimal-change disease (MCD), and nephrosclerosis (NScl) were studied. Admission criteria were proteinuria > 3.5 g/24 h, a decrease of the serum albumin level < 3.5 g/dl, hypercholesterolemia > 300 mg/dl, and the presence of ApoB depositions in the glomeruli. The renal function was normal in 6 patients; in the other 6, the serum creatinine level ranged from 1.6 to 3.7 mg/dl. Before starting the treatment (duration 12 months), all patients had persistent (6-120 months) nephrotic syndrome (NS), unsuccessfully treated using immunosuppressive drugs. The lipid-lowering treatment included a normal-protein, low-cholesterol diet enriched with polyunsaturated fatty acids combined with HMG the 3-hydroxy-methyl-glutaryl coenzyme A inhibitor lovastatin (Mevacor; 0.04-0.06 g/day). All other drugs, except calcium antagonists for 6 hypertensive patients, were excluded.

We observed the lipid-lowering effect after 3-4 months of treatment in 11 patients.

Remission of NS was observed in 7 cases (3 with FSGS, 3 with MN, 1 with NScl); in 3 other patients (1 with FSGS, 1 with MN, and

1 with MCD), the proteinuria significantly decreased and the serum albumin level significantly increased without complete remission of the NS. In 4 of these patients, the decrease of proteinuria was accompanied by an improvement of the renal function. In 2 cases (1 with FSGS, 1 with NScl), there was no improvement of the nephropathy after lipid-lowering treatment; the patient with NScl had decreased serum lipid levels, but renal failure progressed.

The difficulties in the treatment of immuno-suppressive resistant NS are well known. Previously, the treatment with lipid-lowering drugs has been shown to be effective in rats having FSGS [2,3]. Our experience can create a new clinical approach in the therapy of human NS due to noninflammatory nephropathies.

References

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