Lipoprotein(a) Predicts the Risk of Thrombogenic Complications in Nephrotic Syndrome

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Dear Sir,

Hypercoagulability is not a rare finding in nephrotic patients under the administration of adrenal steroids. The deposition of fibrinogen in the renal biopsy specimen and the increase in plasma and/or urinary fibrinogen degradation products are often noted during the course of nephrosis. In the last 2 years we have seen 3 nephrotic patients with thrombogenic complications. All of them exhibited high concentrations of plasma lipoprotein(a) [Lp(a)], even when they were in the remission stage. Lp(a) is a cholesterol-rich plasma lipoprotein containing apo(a) and apo B-100, and is said to be associated with the development of atherosclerosis [1]. It is also known to have thrombogenic properties because it has close structural homology with plasminogen [2]. Several investigators have demonstrated that Lp(a) is an independent risk factor for coronary heart disease (CHD) [3]. As reported by Armstrong et al. [4], when the plasma level of Lp(a) is above 30 mg/dl the risk for CHD increases two fold. We have measured plasma Lp(a) levels in patients with nephrotic syndrome, and investigated whether the increase in Lp(a) predicts the risk of thrombogenic complications in nephrotic patients under the adrenal steroid therapy.

Plasma samples were taken from 32 normal volunteers, 20 nephrotic patients without thrombogenic complications (9 of them were in the remission stage), 1 nephrotic patient with acute myocardial infarction (case 2), and 2 nephrotic patients with cerebral infarction (cases 1 and 3). The plasma Lp(a) level was measured by the enzyme-linked immunoassay kits (Biopool AB) [5]. Low density lipoprotein (LDL) cholesterol was calculated using the Friedwald approximation. As shown in table 1, Lp(a) and LDL cholesterol levels (mean $\pm$ SD)

were significantly elevated in nephrotic patients. These values were more increased in the nephrotic stage. The plasma Lp(a) levels of 3 complicated patients remained above 45 mg/dl even in the remission stage.

Our results suggest that plasma Lp(a) may predict the risk of thrombogenic complications in nephrosis. Thereby, we propose that the simultaneous use of anticoagulant drugs should be

indispensable for nephrotic patients under the steroid therapy, especially in case of increased Lp(a) levels.

References

Utermann G: The mysteries of lipoprotein(a). Science 1989;246:904-910. Scott J: Thrombogenesis linked to atherogenesis at last? Nature 1989;341:22-23.
Seed M, Hoppichler F, Reaveley D, McCarthy S, Thompson GR, Boerwinkle E, Utermann G: Relation of serum lipoprotein(a) concentration and apolipoprotein(a) phenotype to coronary heart disease in patients with familial hypercholesterolemia. N Engl J Med 1990; 322:1494-1499. Armstrong VW, Cremer P, Eberle E, Manke A, Schulze F, Wieland H, Seidel D: The association between serum Lp(a) concentrations and angiographically assessed coronary atherosclerosis : Dependence on serum LDL levels. Atherosclerosis 1986;62:249-257. Gaubatz JW, Cushing GL, Morrisett JD: Quantitation, isolation, and characterisation of human lipoprotein(a). Methods Enzymol 1986; 129:167-189.

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