Determination of Glomerular NAG Fraction in Urine Samples of Patients with Diabetic Nephropathy Using the NAG Test Kit

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Dear Sir,

A study on glomerular N-acetyl- β -D-glucosaminidase (NAG) in the urine of patients with diabetic nephropathy is described. It is generally considered that high levels of urinary NAG excretion might be related to the degree of renal tubular disturbance in patients with diabetes mellitus, hypertension or proteinuria. Recently, the Ames NAG test® kit which involves a simple procedure and high reproducibility has been newly developed [1]. It has the advantage of being able to detect lower levels of urinary NAG activity because of its high optimal density. It is easy to clarify the levels of the glomerular (A) and tubular (B) NAG fraction in the urine since the A fraction is inactivated after incubation at 60°C for 10 min [2]. Urinary samples were obtained from 25 patients with non-insulin-dependent diabetes mellitus (NIDDM), 20 patients with chronic glomerulonephritis (CGN) and 20 age-matched healthy adults. Among the 25 patients with NIDDM, 16 patients were diagnosed as having diabetic nephropathy according to continuous proteinuria and/or renal biopsy. The samples were centrifuged at 3,000 rpm for 5 min. Fresh or heat-inactivated (60°C, 10 min) urine samples were used in this study. Urinary NAG activity was measured by the Ames NAG test® kit as described by Poci et al. [1]. Duplicate tests were performed. There was no significant difference in the levels of total NAG in these patients and healthy adults between the Ames NAG test® kit and the ordinary m-cresolsulfon-phthaleiny-NAG (MCP) method. The levels of total NAG activity of patients with NIDDM were significantly higher than those of patients with CGN or healthy adults ($p < 0.05$). The levels of the NAG A fraction in patients with NIDDM were significantly higher than those in patients with CGN or healthy adults ($p < 0.05$ and $p < 0.01$, respectively). The levels of the NAG A fraction in patients with diabetic nephropathy were significantly higher than those in NIDDM patients without nephropathy ($p < 0.01$). There was no significant difference in the levels of the NAG B fraction in the urine of patients with NIDDM, CGN and healthy adults. Furthermore, the levels of the NAG A fraction were

correlated with the levels of proteinuria in patients with diabetic nephropathy. It was suggested that the levels of the NAG A fraction in the urine samples might be a better reflection of glomerular damage than the measurement of total urinary NAG activity in patients with NIDDM.

References

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