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Renal Tubular Disorder in Kawasaki Disease

Tadashi ASAMI, Tokushi NAKANO and **Kaoru SAKAI**

Department of Pediatrics, Niigata University School of Medicine, Asahimachi 1-757, Niigata 951, Japan

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Summary. **Levels of urinary β-D-N-acetylglucosaminidase (NAG), known as a sensitive index of renal tubular disorder, were measured in six children with Kawasaki disease, or mucocutaneous lymphnode syndrome. The values were highly elevated during the first month of the disease in all of the patients, however, conventional renal function tests were normal. Although the patients had no renal symptoms except for slight proteinuria, these results might be considered as a warning of possible renal tubular disorder in the course of Kawasaki disease because some cases of acute renal failure have been reported in Japan.**

Kawasaki disease, or mucocutaneous lymphnode syndrome (MLNS), was first reported in 1967.[1] Since then, there have been many reports on the disease and its complications, such as coronary aneurysms, leading to myocardial infarction and unexpected death,[2] myositis,[3] and hydrops of the gall bladder.[4] However, renal involvement in Kawasaki disease draws little attention, although some cases of acute renal failure or nephrotic syndrome have been reported as the renal complication in Kawasaki disease in Japan. In order to clarify the renal involvements, our group measured serially the urinary β-D-N-acetylglucosaminidase (NAG) level, which is known as a sensitive index of renal tubular injury or disorder,[5,6] in patients with Kawasaki disease.

SUBJECTS AND METHODS

Six MLNS patients, ranging in age from 9 months to 4 years old, were measured for urinary NAG levels by our fluorometric method.[6] The values obtained from 237 age-matched healthy children were used as controls, and the levels lower than 60 u/ml were defined as a normal range of urinaly NAG by our method.[6]

RESULTS

All of the six patients showed elevated urinary NAG levels higher than 60 u/ml during a four-week period from the onset, and the sequential declines were noted, as demonstrated in Figure 1. These patients, with elevated urinary NAG level, had no signs and symptoms of renal complications except for slight proteinuria (20 to 40 mg/dl) or leukocyturia, which are commonly seen in this disease.

DISCUSSION

In this study neither a NAG/creatinine ration nor a total 24-hour urinary NAG excretion was used to evaluate the values because of three reasons: 1) urinary creatinine excretion varies widely according to body mass of the subjects, 2) partially collected urine samples were demonstrated to have rather narrow-ranged NAG levels,[6] providing a reliable normal range (below 60 u/ml), and 3) urinary NAG levels were serially determined on the same condition in the subjects.

To examine a possible derivation of urinary NAG from increased urinary leukocytes or epithelial cells, the following experiment was performed using 50 urine samples obtained from children with other diseases: A ten-ml sample of freshly-voided urine was centrifuged for five min at 1,500rpm. The supernatant was measured for NAG, and the leukocytes or epithelial cells in the centrifuged sediment were counted under a microscope and expressed as the numbers/high-power (×400) field (HPF). No significant correlations were found between the urinary NAG levels and the numbers of urinary leukocytes ($r=-0.05$, $n=50$) or epithelial cells ($r=-0.07$, $n=50$) as shown in Figure 2.

These results suggest that renal tubular cells are latently damaged by a still-unknown cause and are

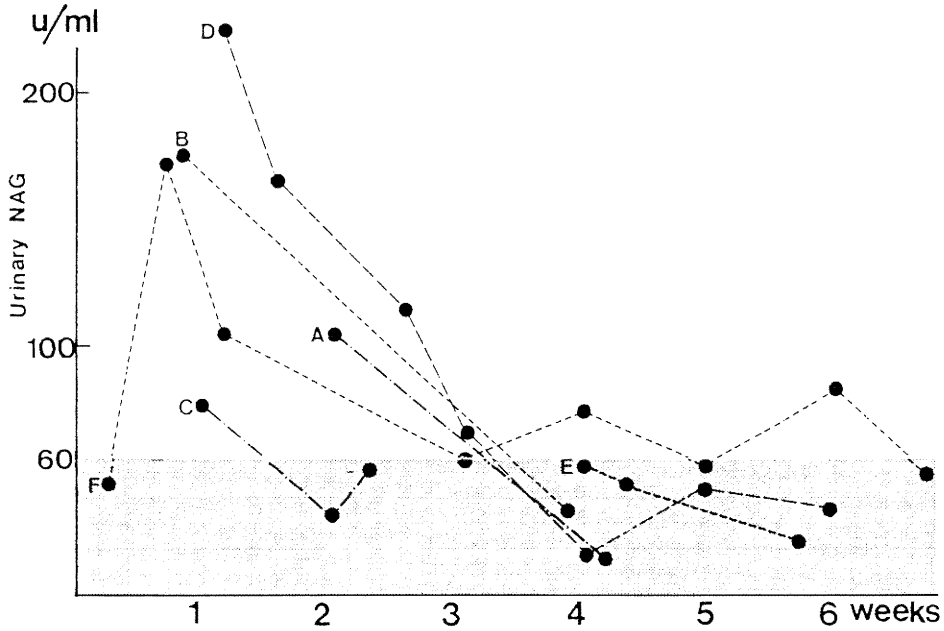

Fig. 1. Urinary NAG levels and the sequential changes in six patients (A-F) with Kawasaki disease.

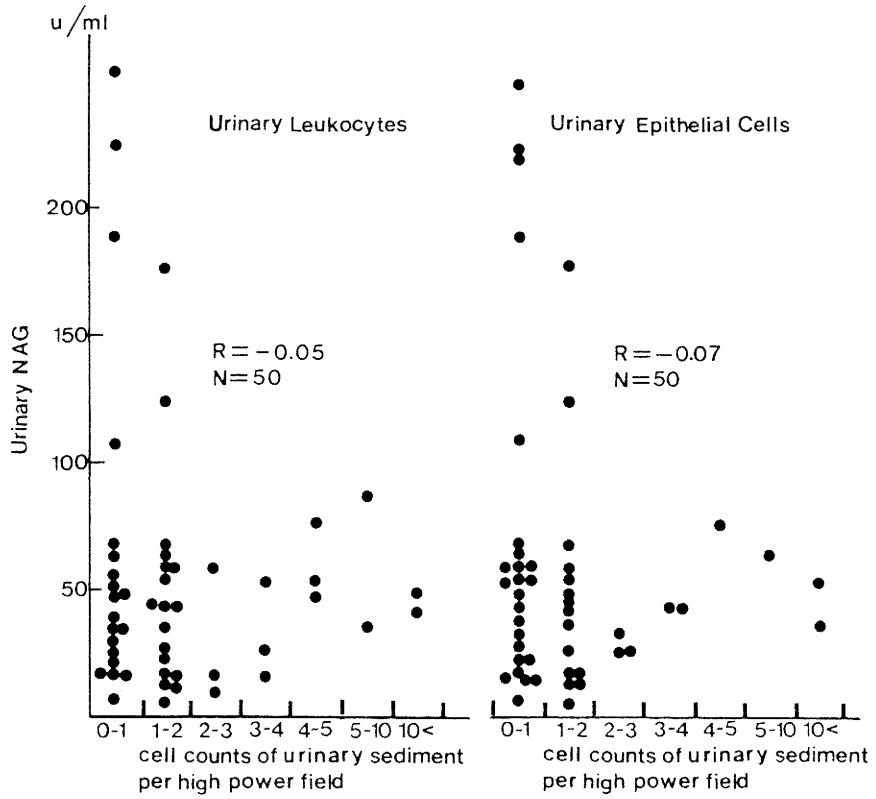

Fig. 2. Correlations between the urinary NAG levels and the urinary leukocyte and epithelial cell counts.

vulnerable to nephrotoxic agents in the early stages of Kawasaki disease. A possible development of acute renal failure, marked by the elevated urinary NAG level, should be kept in mind when treating children with Kawasaki disease, because some cases have been reported in Japan.

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