Nonsteroidal Anti-Inflammatory Drugs as Risk Factor for Renal Failure from Acute Uric Acid Nephropathy

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Dear Sir,

The risk for a rapidly occurring form of renal insufficiency due to acute uric acid nephropathy exists in patients affected by lympho- or myeloproliferative disorders as a result of rapid cell turnover, often during chemotherapy [1]. Our clinical experience shows, however, that the same pattern may appear in subjects who show no signs of neoplastic pathology and have no positive history of hyperuricemia or gout.

From January 1980 to November 1985 we observed 9 patients, all males between 20 and 46 years of age, with acute renal failure due to intratubular obstruction by uric acid crystals. They had been apparently healthy until the acute episode and had no preceding history of renal disease; only 2 cases suffered from episodic emission of gravel without hyperuricemia. The clinical symptoms consisted of lumbar pain and oliguria. Renal function, on admission, was reduced, in 4 cases by only 60% and returned to normal over 3 days, in the other 5 cases by 90–95% and more lasting; however, no dialytic treatment was necessary in view of the transitory nature of the oliguria.

The diagnosis of acute nephropathy due to uric acid was based on the exclusion of other possible causes and on the report of uric acid/creatinine ratio > 0.9 in random urine samples [2]. In the 5 more serious cases we carried out a renal biopsy which showed the presence of intratubular deposits of uric acid and focal tubular necrosis, mainly in the distal tract.

In all the patients, on return of normal renal function, we observed uricuria at the upper limits of the normal

range. A few hours before the appearance of acute symptoms, the patients had carried out intense physical activity. The 5 more serious cases had taken nonsteroidal anti-inflammatory drugs as symptomatic treatment for acute lumbago.

Intense physical activity, by stimulating an increase in the production of uric acid and a relative dehydration, may predispose to intratubular precipitation of uric acid in subjects who eliminate large quantities of it under normal conditions. Simultaneous treatment with nonsteroidal anti-inflammatory drugs aggravates the clinical picture, probably by inhibiting the synthesis of prosta-glandins which antagonize the action of ADH when diuresis is decreased.

References

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