TREATMENT OF HYPERURICEMIA IN ESSENTIAL HYPERTENSION

To the Editor:

The 2 recent articles linking an elevated serum uric acid level with an increased risk of developing hypertension are of great interest.1,2 The application of the key finding in these 2 studies, that is, a graded increase in risk of developing essential hypertension based on the serum uric acid level, is likely to be more far reaching than earlier reports because of the larger sample size, longer follow-up period, and the comprehensive adjustment for confounding variables, such as age, body mass index, smoking, plasma lipid levels, serum glucose concentration, and renal function. This will lead to the inevitable discussion of whether treatment to lower serum uric acid can favorably impact on the natural history of essential hypertension and the complications of a sustained high blood pressure level. Before clinicians embark on this course of action, caution would be warranted based on studies that I conducted on the effect of allopurinol treatment in the spontaneously hypertensive rat.3 I demonstrated that administration of allopurinol (100 mg/kg of body weight per day) for 15 weeks had no impact on the development of hypertension in this strain. Moreover, compared with the control Wistar–Kyoto rat strain, allopurinol was associated with significant nephrotoxicity characterized by impaired somatic and kidney growth, azotemia, and significant tubulointerstitial injury. Therefore, I recommend that uric acid–lowering therapies be assessed systematically in well-designed clinical trials with sufficient long-term observation to detect the effect of treatment on blood pressure. In addition, adequate safety precautions should be incorporated into the protocol design for detection of renal dysfunction and other unanticipated adverse events to insure that the risk:benefit ratio justifies continuation of the test therapy.

Disclosures

None.

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