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IJKD KIDNEY DISEASES

Association of Serum Uric Acid With Level of Blood Pressure in Type 2 Diabetic Patients

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This study was conducted to investigate the association between serum uric acid level and blood pressure in type 2 diabetes mellitus. Sixty patients with type 2 diabetes mellitus were enrolled to the study. None of the patients had a history of gout, were treated with allopurinol, or were treated with antihypertensive drugs previously. The mean duration of DM was 9.2 ± 4.9 years. The mean serum creatinine level was 0.98 ± 0.22 mg/dL, and the mean serum UA level was 4.4 ± 1.2 mg/dL. The mean protein level in 24-hour urine sample was 388 ± 22 mg/d. The mean systolic and diastolic blood pressure values were 133.0 ± 13.0 mm Hg and 84.0 ± 7.4 mm Hg, respectively. There was no significant difference in levels of serum uric acid, hemoglobin A1c, serum creatinine, proteinuria, or systolic and diastolic pressure between the men and the women. A significant positive correlation was seen between serum UA and systolic (r = 0.312, *P* = .02) and diastolic blood pressure (r = 0.297, *P* = .03). Results of this study suggest that serum uric acid had a strong association with levels of systolic and diastolic blood pressure in type 2 diabetic patients. More attention to the serum uric acid level and treatment of hyperuricemia could halt the progress of diabetic nephropathy.

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Brief Communication

Uric acid (UA), as the final oxidation product of purine catabolism, has been associated with various clinical conditions such as diabetes mellitus (DM) and atherosclerotic disease.[1] Recent studies suggest that UA is a relevant and independent risk factor for kidney disease, particularly in patients with hypertension.[1-3] It was shown that hyperuricemia, induced by a uricase inhibitor, triggered hypertension and impaired nitric oxide generation in the macula densa, while both hypertension and renal injury are reduced by inducing nitric oxide.[4-6] The mechanism by which uric acid may cause organ damage is not fully understood; however, there is increasing evidence that endothelial dysfunction is a mechanism whereby this substance may affect kidney function and structure.[7-9] The aim of this observational study was to determine whether baseline serum uric acid levels are associated with blood pressure in type 2 DM with preserved kidney function at baseline and without a previous history of cardiovascular disease or gout.

A cross-sectional study was carried out on 60 patients with type 2 DM. None of the patients had a history of gout and none was treated with allopurinol or antihypertensive drugs. Anthropometric measurements were collected, including height, body weight, and body mass index. Resting systolic blood pressures and the 5th phase diastolic blood pressures were measured three times, while the participants were seated, and the 2nd and 3rd measurements were averaged.[10,11]

Hypertension was defined as a blood pressure of 140/90 mmHg or higher or currently receiving antihypertensive treatment.[10,11] Venous blood samples were obtained in the fasting state for determination of serum levels of creatinine, uric acid, and hemoglobin A1c (reference range, 4% to 6%). In addition, 24-hour urine protein excretion was measured.

Results were expressed as mean ± standard deviation and comparisons were considered significant when two-sided *P* value was less than .05. The independent-samples *t* test was used for comparison of variables between men and women. The Spearman rho coefficient correlation was used for evaluating correlations among variables. For association of serum uric acid with levels of blood pressure, the partial correlation test was used with adjustment for age, duration of DM, and serum creatinine level.

Of the 60 participants, 56.7% were women. The mean of age was 57.0 ± 8.3 years. The mean duration of DM was 9.2 ± 4.9 years. The mean serum creatinine level was 0.98 ± 0.22 mg/dL, and the mean serum UA level was 4.4 ± 1.2 mg/dL. The mean protein level in 24-hour urine sample was 388 ± 22 mg/d (median, 303.5 mg/d). The mean systolic and diastolic blood pressure values were 133.0 ± 13.0 mm Hg and 84.0 ± 7.4 mm Hg, respectively. In this study, there was no significant difference in serum UA, HbA1c, or serum creatinine between the men and the women. Similarly, there was no significant difference in proteinuria and levels of systolic or diastolic pressure between the two groups. A significant positive correlation was seen between serum UA and systolic (r = 0.312, *P* = .02) and diastolic blood pressure (r = 0.297, *P* = .03).

In the present study, we found a significant positive correlation of serum UA with systolic and diastolic blood pressure. No significant difference was found in serum uric acid, HbA1c, or creatinine, between the men and women. Similarly, no significant difference was observed in proteinuria and levels of systolic or diastolic pressure between the men and the women. Increasing evidence supports a causal role for UA in hypertension and associated target organ damage in humans, especially in women.[1,4,7,12,13] Recent studies have shown that experimental hyperuricemia in rats induce hypertension.[1,4,7,12,13] The mechanism by which urate causes a deleterious effect may be related to oxidative stress and diminished production of nitric oxide and renal arteriolar damage due to proliferation of vascular smooth muscle cells.[4,7,11-17]

To find the association of serum UA and endothelial function, Zoccali and coworkers conducted a study on untreated hypertensive patients. They found that serum UA was inversely correlated with flow-mediated vasodilatation.[18] Diabetic nephropathy is now the major cause of end-stage renal disease.[6,17-20] Various epidemiological studies have also demonstrated that serum UA is associated with diabetic nephropathy, suggesting a potential role for uric acid in the disease pathogenesis.[1,4,7] Given these facts, we also could show that UA played a pathological role in the development of hypertension and in type 2 diabetic patients. Hypertension per se is an aggravating factor of diabetic nephropathy; thus, more attention to serum UA level and appropriate treatment of hyperuricemia could attenuate progression of diabetic nephropathy.

CONFLICT OF INTEREST

None declared.

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