Urinary Excretion Rate of Guanidinoacetic Acid in Essential Hypertension

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Dear Sir,

Essential hypertension has been known to cause renal arteriolar lesion (benign nephrosclerosis) frequently after long-term duration, which may be associated with various glomerular and tubulo-interstitial changes [1, 2]. However, clinical findings show a poor correlation with pathological changes.

The production of guanidinoacetic acid (GAA), which is mainly produced in the kidney by the transamidation of glycine from argine, was known to be decreased in some renal diseases [3]. This study attempted to evaluate the urinary excretion of GAA in hypertensive patients (5 borderline hypertension and 25 hypertension) as well as 10 normal controls. They were subjected to creatinine clearance

(Ccr) during 1 h. All of them were withdrawn from all medication and given the same diet for 2 weeks prior to this study. The urinary excretion of β 2-microglobulin (BMG) and N-acetyl-D-glucosaminidase (NAG), which have been already evaluated as causes of early renal-damage in hypertension [4], was also measured. GAA were measured by enzymatic determination [5].

In hypertensive patients the urinary excretion rate of GAA was $41.9 \pm 17.1 \mu\text{g}/\text{min}$, which was markedly lower than in controls (81.8 ± 37.9 ; $p < 0.01$). However, there was no significant difference in Ccr, serum Cr, serum BMG, urinary excretion of BMG and NAG between these groups (table I). The urinary excretion rate of GAA had also a significant positive correlation with Ccr ($r = 0.62$; $p < 0.01$; fig. 1).

M/F = Male/female; p values vs. control: ** $p < 0.05$; *** $p < 0.01$.

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These findings suggest that the urinary excretion rate of GAA in hypertension may be a more important cause of renal damage than that of BMG and NAG.

150-
Control
Borderline
Hypertension
100-
50 J
References

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150 160
50 100
Urinary excretion rate of GAA($\mu\text{g}/\text{min}$)