Abstract

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Human Tissue Kallikrein Activity in Angiographically Documented Chronic Stable Coronary Artery Disease.

[Article in English, Portuguese]

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Abstract

AbstractBackground:Human tissue kallikrein (hK1) is a key enzyme in the kallikrein-kinin system (KKS). hK1-specific amidase activity is reduced in urine samples from hypertensive and heart failure (HF) patients. The pathophysiologic role of hK1 in coronary artery disease (CAD) remains unclear.

OBJECTIVE: To evaluate hK1-specific amidase activity in the urine of CAD patientsMethods:Sixty-five individuals (18-75 years) who underwent cardiac catheterism (CATH) were included. Random midstream urine samples were collected immediately before CATH. Patients were classified in two groups according to the presence of coronary lesions: CAD (43 patients) and non-CAD (22 patients). hK1 amidase activity was estimated using the chromogenic substrate D-Val-Leu-Arg-Nan. Creatinine was determined using Jaffé's method. Urinary hK1-specific amidase activity was expressed as μM/(min · mg creatinine) to correct for differences in urine flow rates.

RESULTS: Urinary hK1-specific amidase activity levels were similar between CAD [0.146 μM/(min ·mg creatinine)] and non-CAD [0.189 μM/(min . mg creatinine)] patients (p = 0.803) and remained similar to values previously reported for hypertensive patients [0.210 μM/(min . mg creatinine)] and HF patients [0.104 μM/(min . mg creatinine)]. CAD severity and hypertension were not observed to significantly affect urinary hK1-specific amidase activity.

CONCLUSION: CAD patients had low levels of urinary hK1-specific amidase activity, suggesting that renal KKS activity may be reduced in patients with this disease.

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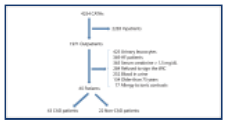

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