Abstract: 1465

Serum uric acid levels and xanthine oxidase activity in chronic heart failure patients with chronic kidney disease

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Background. Recent experimental and clinical data suggest that there might be a direct pathophysiological role for increased xanthine oxidase (XO) activity and hyperuricemia in the progression of heart failure (HF).
Purpose. To evaluate the impact of hyperuricemia and XO activation in HF patients with chronic kidney disease (CKD).
Methods. Baseline characteristics were: 112 patients with chronic HF (among them 51 men and 61 women), mean age – 72.5±8.6 years. All patients were divided into 2 groups: within CKD (n=72) and non-CKD (n=40) participants. We used enzymatic colorimetric test, PAP – method with antilipid factor to evaluate serum uric acid (SUA) levels. Asymptomatic hyperuricemia was defined as SUA levels >7.0 mg/dl in men and >6.0 mg/dl in women. XO activity was determined by a coupled enzyme assay, which resulted in a colorimetric (570 nm)/fluorometric (lex= 535/lem=587 nm) product, proportional to the hydrogen peroxide generated. One unit of XO was defined as the amount of enzyme that catalyzed the oxidation of xanthine, yielding 1.0 mmole of uric acid and hydrogen peroxide per minute at 25 °C.
Results. In patients group 62.5% had asymptomatic hyperuricemia. The mean SUA levels for matched patients were (8.47±0.23) mg/dL, in the group without hyperuricemia – (5.38±0.19) mg/dL. SUA correlated negatively with left ventricular ejection fraction (LVEF) (r=-0.3, p<0.05). Patients with functional class New York Heart Association (NYHA) III have significantly higher SUA levels compared to patients with NYHA II: (8.5±0.39) mg/dL and (6.88±0.25) mg/dL respectively (p<0.01). The kidney function significance in the development of the xanthine metabolism violations proves the revealed inverse correlation between estimated glomerular filtration rate (eGFR) and XO activity (r=-0.7, p<0.05) as well as SUA levels in patients with chronic HF (r=-0.3, p<0.05). Patients with concomitant CKD had higher XO activity levels compared to non-CKD patients: (7.51±0.77) mU/ml vs (4.69±0.77) mU/ml respectively (p<0.01). The mean SUA levels were not significantly different: (7.63±0.27) mg/dl vs (7.46±0.39) mg/dl respectively (p=0.73). Comparison of mean GFR in patients with and without hyperuricemia revealed significantly lower GFR in patients with asymptomatic hyperuricemia: (59.9±2.95) ml/min/1.73m2 and (76.6±6.05)ml/min/1.73 m2 respectively (p<0.01). Data also showed that patients with eGFR>60 ml/min/1.73 m2 have significantly higher SUA levels and XO activity compared to those with eGFR<60 ml/min/1.73 m2: (8.21±0.29) mg/dl vs (6.73±0.31) mg/dl (p<0.001) and (8.72±0.8) mU/ml vs (4.15±0.56) mU/ml respectively (p<0.001).
Conclusion. Uric acid itself rather than up-regulated XO activity in patients with chronic HF is associated with LVEF and the progression of HF functional class. But the presence of concomitant CKD in chronic HF patients can modify the xanthine metabolism towards oxidase pathway.