J Clin Exp Cardiology 2018, Volume 9
DOI: 10.4172/2155-9880-C2-093

Kynurenine pathway metabolites are associated with mortality in patients with heart failure

Anders Lund
University of Bergen, Norway

Background: Circulating metabolites of tryptophan produced via the kynurenine pathway (kynurenines), are linked to all-cause and cardiovascular mortality in patients with coronary artery disease, but have not been extensively explored in patients with heart failure. We aimed to compare plasma levels of kynurenines between patients with heart failure and controls, and to investigate whether kynurenines predict mortality.

Methods & Results: The study included 202 patients with heart failure and 264 controls without heart disease (propensity score matched by age and gender). All participants underwent coronary angiography and cine ventriculography. Plasma kynurenines, pyridoxal 5'phosphate (PLP), C-reactive protein (CRP) and monocyte count were measured at baseline. Case-control differences were assessed by logistic regression and mortality-hazard by Cox regression. Results were adjusted for multiple testing. Ninety-four (47%) of the heart failure patients died during the follow-up period. Kynurenine (Kyn, [OR 1.65, p<0.001]) and the kynurenine-tryptophan-ratio (KTR, [OR 1.63, p<0.001]) were higher in patients with heart failure than in controls. KTR [HR 1.59, p=0.006], 3- hydroxykynurenine (HK, [HR 1.65, p<0.001]), and the ratio of HK to xanthurenic acid (HK/XA, [HR 1.67, p<0.001]) were all associated with increased mortality.

Conclusion: Metabolites of the kynurenine pathway, related to innate immune activation and apoptosis, are increased in heart failure and associated with mortality. Whether these associations reflect mainly underlying causes that activate the pathway or effects of the kynurenines themselves remains undecided.

lund.anders88@gmail.com

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