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Dates: Received: 01 August, 2017; Accepted: 09 September, 2017; Published: 12 September, 2017

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Serum uric acid as a metabolic regulator of endothelial function in heart failure

Abstract

The development of heart failure (HF) associated with elevated level of serum uric acid (SUA). Additionally, the majority of individuals with traditional cardiovascular risk factors contributing in HF risk exhibited increased levels of SUA. Although SUA lowering drugs are widely used in patients with symptomatic hyperuricemia and gout beyond their etiologies, there is no agreement of SUA below target level 6.0 mg/dL in asymptomatic individuals with kidney injury and CV disease and data of ones in heart failure (HF) are sufficiently controversial. First SUA plays an important role in inducing oxidative stress, inflammation, neurohumoral activation, and endothelial dysfunction. Secondary, SUA may act as antioxidant contributing in restoring of vascular function. Moreover, SUA is able to epigenetically regulate a survival of endothelial precursors, mediate their mobbing and differentiation, as well as coordinate a turn-over effect of metabolic memory phenomenon into repair capability of cell precursors. However, elevated SUA level was found a predictor of adverse clinical outcomes of HF. The short communication is depicted the importance of new clinical data to confirm the emerging reparative ability of SUA in HF and its role as promising target for treatment in cardiac failure.

Keywords: Serum uric acid; Heart failure; Endothelial dysfunction; Biomarker

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observational studies have shown that elevated SUA levels were associated with reduced survival in in-patients and out-patients with several phenotypes of chronic HF, as well as in acute / actually decompensated HF [27-29].

In contrast, in recent meta-analysis lowering SUA levels under treatment did not predict improved surrogate clinical outcomes in HF [30]. Moreover, there was not convincing evidence regarding associations of SUA levels with increased risk of HF and other disease at higher risk of HF and HF mortality [31].

All these facts clarify that uric acid is multiple player with controversial activities that concurrently contribute in HF pathogenesis [32]. The modalities of uric acid cannot be discussed as unconditionally harmful effects supporting inflammation and cell death, but they may produce a favorable result on reparative activity of endothelium and restoring endothelium function and vascular integrity [33]. Whether SUA is a therapeutic target to reduce HF risk in vulnerable population is not fully clear and requires to be explained in the large clinical trials.

In conclusion, there was no strong and available evidence regarding only harmful effect of uric acid in vulnerable population patients at higher risk of HF as well as in individuals with established HF irrespective to left ventricular ejection fraction or isolating diastolic abnormality. The importance of new clinical data that confirm the emerging reparative ability of uric acid and its antioxidant activities across HF development require to be cleared in large investigations. The results of these trials would be intriguing and could open new perspective to use xanthine oxidase inhibitors as adjuvant care in HF management.

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Citation: Berezin AE (2017) Serum uric acid as a metabolic regulator of endothelial function in heart failure. Arch Clin Hypertens 3(1): 027-029.

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