

Serum Uric Acid Levels: A Potential Biomarker for the Acute Myocardial Infarction in Hypertensive Patients

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Editorial

Cardiovascular diseases (CD) are one of the main causes of death in the world, among them is ischemic cardiac disease (ICD). ICD is believed to cause 26 million deaths worldwide by 2030 [1,2]. Among ICD, acute myocardial infarction (AMI) is the most common cause of morbidity and mortality in the world [2,3]. AMI is characterized by a long period, and severe, of cardiac ischemia (e.g. thromboembolism) that promotes large myocardial damage. The treatment of cardiac ischemia is aggravated by reperfusion (e.g. revascularization) which compromises cardiac function, especially the excitation-contraction coupling mechanism [2-4].

The diagnosis of AMI is performed through the analysis of several parameters, among them are the measurements of biochemical serum markers of cardiac lesion. Currently, the most used in the medical clinic for this purpose is the total creatine kinase (CK), and creatine kinase MB fraction (CK-MB), as well as troponins I and T proteins (TnI and TnT) [5].

The increased concentration of uric acid (UA) in serum, a metabolite of purines degradation produced by activity of xanthine oxidase enzyme, serves as an independent risk factor for cardiovascular disease [6-11]. Indeed, the UA has been related to inflammation, endothelial dysfunction and deterioration of left ventricular function via changing calcium sensitivity of myofilaments, alterations of myocardial energetic metabolism, thus serving as major prognostic marker predicting mortality, and needful for heart transplantation in patients with progressive heart failure [12,13]. In addition, the UA has been used alone, or in combination with other parameters (e.g. serum biomarkers and clinical), as a predictor for coronary heart disease, and managing of the cardiac patient [14]. Moreover, UA has been related to the increased of blood pressure of the children and adolescents [15], and adults [16,17].

Our group currently studies the alterations of excitation-contraction coupling mechanisms of cardiac cells submitted to ischemia-reperfusion injury in animal model of hypertension, by using the model of spontaneously hypertensive rats (SHR) and normotensive Wistar rats (NWR), with emphasis in serum markers of cardiac injury [18]. We found that hypertension does not increase serum levels of UA in SHR group when compared to NWR. However, we observed increased serum levels of UA in the SHR group submitted to cardiac ischemia and reperfusion, compared to NWR, suggesting a possible relevance of this marker in human hypertensive patients in the condition of cardiac ischemia and reperfusion.

As conclusion, we suggest that UA could be used in combination with other cardiac injury markers for the diagnosis of acute myocardial infarction in hypertensive patients.

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