Role of ischemia-modified albumin in patients with acute decompensated heart failure

Ischemia-modified albumin (IMA) is a form of human serum albumin in which its N-terminal amino acids are modified because of ischemia (1). IMA is produced when ischemic stressors released from hypoxic heart tissue modify circulating albumin, thereby reducing its binding affinity to heavy metal ions such as cobalt (2, 3).

IMA was first identified in the early 1990s and has since been widely studied in patients presenting with myocardial ischemia (4). The ability of albumin to bind to cobalt is reduced in patients with myocardial ischemia, providing the basis for the albumin cobalt-binding test for detecting IMA (5). IMA is currently approved by the US Food and Drug Administration as a biomarker able to detect myocardial ischemia within minutes, while the levels of IMA continue to increase for 6–12 h (6). However, data on IMA levels in patients with heart failure (HF), especially acute HF, are still lacking.

In this issue, Çavuşoğlu et al. (7) report the results of the first study to investigate IMA levels in patients with acute HF treated with dobutamine or levosimendan. They found that patients presenting with acute HF had elevated levels of IMA and also suggested that in-hospital acute HF therapy significantly reduced serum IMA levels. Previous data on serum concentrations of IMA in patients with HF are limited. Dominguez-Rodriguez et al. (8) showed that IMA levels are related to the left ventricular ejection fraction in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention and in those who developed acute HF. IMA demonstrated a high sensitivity and negative predictive value for the diagnosis of HF. In agreement with this previous study, Çavuşoğlu et al. (7) showed that IMA is not only a promising biomarker for the diagnosis of acute HF but also useful for assessing the effect of inotropic therapy for acute HF.

However, limitations of IMA need to be considered. Firstly, HF is a complex clinical syndrome, the most common causes of which are coronary heart disease, hypertension, cardiomyopathy, valvular heart disease, and type 2 diabetes mellitus (9, 10). Sixty-two patients (88%) in the present study had ischemic HF. However, the possible effects of acute HF demographics on IMA levels remain unclear. Secondly, IMA was normally distributed in a group of apparently healthy volunteers and was not correlated with smoking, age, race, sex, or the Framingham risk score (11). However, IMA is dependent on the level of serum albumin (12). The impact of serum albumin on IMA levels is an important factor, even within the normal range (13), and given that hypoalbuminemia is common in patients with HF, the possible effects of hypoalbuminemia on IMA must be considered. Finally, although the study by Çavuşoğlu et al. (7) study suggested that IMA is a highly sensitive biomarker with diagnostic potential for the assessment of acute HF, problems with the stability of IMA and its lack of cardiac specificity have been reported (14, 15). The results of the current study suggest that the value of IMA lies in its ability to exclude, rather than confirm, the presence of acute HF, suggesting that its greatest value lies in patient reassurance.

Several issues relating to IMA remain unanswered. Although its main limitation is its low specificity, IMA is a highly sensitive negative marker. However, further clinical studies in larger numbers of patients are needed to address the utility, outcomes, and cost-effectiveness of IMA prior to its integration into clinical practice.

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Bir keskin neşter bir harap kalbim,

Opr. Dr. Kamil Furtun'un Aziz Hatırasına
(uyarlanmıştır)

My heart is a sharp lancet, ruined,

In valuable memory of Opr. Dr. Kamil Furtun
(adapted)