Uric Acid and Prognosis in Chronic Heart Failure

To the Editor:

We read with interest the article by Anker et al1 exploring the potential role of uric acid as a marker of prognosis in patients with chronic heart failure (CHF). We recently completed a 5-year follow-up of a study with the specific and predefined aim of exploring the prognostic role of electrical, autonomic, metabolic, and hemodynamic abnormalities in ambulant patients with CHF.2,3 Although Anker et al1 report some interesting and potentially important findings, we have some concerns, as follows.

(1) For a study exploring the utility of different variables in prognosis, one should establish a priori which variables are to be assessed, and all of these variables should be included in subsequent analyses. Was this performed in the present study, and if so, were the 7 variables in the multivariate analysis the only measurements selected?

(2) In a study such as this, patients should be recruited consecutively to remove any bias due to patient selection. In the derivation study of Anker et al,1 112 patients were recruited over 5 years, suggesting that a significant number of patients were excluded; details of these patients should be provided. Similarly, in the validation group, bias may be introduced because these particular patients had uric acid measured and may not be representative of the usual CHF population who did not have uric acid measured.

(3) In the United Kingdom Heart Failure Evaluation and Assessment of Risk Trial (UK-HEART), of 553 patients consecutively recruited with mild to moderate CHF, the range of creatinine was 60 to 340 μmol/L. We2 and others4,5 have shown that renal function assessed using creatinine or glomerular filtration rate is a strong, independent predictor of mortality in patients with CHF. Anker et al1 did not include patients with significantly increased creatinine in their study. This raises a number of important points. By excluding patients with high creatinine levels, one cannot automatically apply the findings to all CHF patients (particularly those with end-stage cardiac failure being considered for transplantation), many of whom will have substantially worse renal function than the patients described. Finally, did the relatively small number of highly selected patients lead to the failure of renal function to predict outcome?

The study of Anker et al1 is an important attempt to establish uric acid as a marker of adverse prognosis in CHF. However, its design and the highly selected population mean that until the results are validated prospectively in an adequately powered trial, one should be cautious about transferring these results into clinical practice.

Mark T. Kearney, MD
James Nolan, MD
For the UK-HEART Study Investigators

Department of Cardiology,
GKT School of Medicine
London
Cardiothoracic Centre,
North Staffordshire Infirmary
Stoke on Trent
United Kingdom
mark.earney@kcl.ac.uk

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Mark T. Kearney and James Nolan
For the UK-HEART Study Investigators

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