Letter by Palazzuoli et al Regarding Article, “Is Worsening Renal Function an Ominous Prognostic Sign in Patients With Acute Heart Failure? The Role of Congestion and Its Interaction With Renal Function”

To the Editor:

We congratulate Dr Metra and colleagues in helping us further understand the problem of worsening renal function, better termed acute kidney injury (AKI), in heart failure (HF) patients.1 This study adds to the literature in that residual congestion at the time of discharge is an additional risk to AKI as an in-hospital complication. Approximately 20% to 40% of patients admitted with HF have an associated development of AKI, defined on the basis of conventional criteria. In a meta-analysis evaluating the impact of AKI in HF at 6 months follow-up, there was a strong association to an unfavorable prognosis (odds ratio 1.62). In less advanced HF stages, chronic kidney disease is associated with left ventricular systolic volume and higher functional class.2 It is likely that many of the patients in the study by Metra and colleagues had resolving AKI because it appears the baseline and peak serum creatinine at any time was used to generate change in serum creatinine and, thus, the definition of AKI.

Irrespective of any measure of congestion, changes in renal function are consistently related to higher short and longer term HF mortality. In a retrospective analysis of 949 patients from OPTIME-CHF Trial, Klein et al3 investigated the relation between the admission values and changes in blood urea nitrogen (BUN) and estimated glomerular filtration rate, and the rate of death within 60 days after discharge. Independent of values at admission, an increase of >10 mg/dL in BUN during hospitalization at any time was associated with a worse 60 day survival; BUN (per 5 mg/dL increase) had a hazard ratio of 1.08 (95% CI, 1.01–1.16).1 In the POSH study, among 248 patients with acute decompensated heart failure and systolic dysfunction, one third developed AKI during hospitalization. Of note, only in those with complicated courses during hospitalization (development of shock or cardiac ischemia) did AKI have a significant association with mortality.3 Rusinaru et al4 demonstrated in patients admitted for HF and preserved left ventricular systolic function that reduced baseline eGFR and AKI predicted long term mortality. Finally, the same authors of the present paper previously reported that an increase of 0.3 mg/dL in creatinine was an independent predictor of death or HF rehospitalization (hazards ratio 1.47).

The findings of Metra et al suggest persistent hemodynamic compromise in addition to AKI identify high risk patients over the next several weeks to months. Furthermore, it may be renal venous congestion in these circumstances that is the common pathophysiologic link leading to cardiorenal death. It appears to take only 1 clinical feature to identify a persistently congested patient. It is conceivable that real time monitoring of body water content using bioelectric impedance vectorial analysis combined with measurement of blood natriuretic peptides could lead to not only better assessments of congestion but also indicators of the need for additional therapy.5

In addition, ultrasound lung comets are a simple echographic tool recognizing extravascular lung water, associated with left ventricular diastolic or systolic dysfunction as well as pulmonary congestion. The complement of new molecular targets to identify AKI, such as neutrophil gelatinase associated lipocalin, kidney injury molecule-1, L-type fatty acid binding protein, and cystatin-C will help further understand the patient with combined cardiorenal failure. The present paper from Dr Metra and colleagues makes it clear that an assessment of congestion is a critical piece to improved inpatient HF management and discharge planning. We look forward to future research that integrates clinical, physiological, and biochemical parameters in an attempt to ideally treat each HF patient, avoid AKI, and reduce mortality.

Disclosures

None.

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References

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