Cardiorenal syndrome

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TO THE EDITOR: I read with interest the thoughtful review of cardiorenal syndrome by Drs. Thind, Loehrke, and Wilt[1] and the accompanying editorial by Dr. Grodin.[2] These articles certainly add to our growing knowledge of the syndrome and the importance of treating volume overload in these complex patients.

Indeed, we and others have stressed the primary importance of renal dysfunction in patients with volume overload and acute decompensated heart failure.[3,4] We have learned that even small rises in serum creatinine predict poor outcomes in these patients. And even if the serum creatinine level comes back down during hospitalization, acute kidney injury (AKI) is still associated with risk.[5]

Nevertheless, clinicians remain frustrated with the practical management of patients with volume overload and worsening AKI. When faced with a rising serum creatinine level in a patient being treated for decompensated heart failure with signs or symptoms of volume overload, I suggest the following:

Perform careful bedside and chart review searching for evidence of AKI related to causes other than cardiorenal syndrome. Ask whether the rise in serum creatinine could be caused by new obstruction (eg, urinary retention, upper urinary tract obstruction), a nephrotoxin (eg, nonsteroidal anti-inflammatory drugs), a primary tubulointerstitial or glomerular process (eg, drug-induced acute interstitial nephritis, acute glomerulonephritis), acute tubular necrosis, or a new hemodynamic event threatening renal perfusion (eg, hypotension, a new arrhythmia). It is often best to arrive at a diagnosis of AKI due to cardiorenal dysfunction by exclusion, much like the working definitions of hepatorenal syndrome.[6] This requires review of the urine sediment (looking for evidence of granular casts of acute tubular necrosis, or evidence of glomerulonephritis or interstitial nephritis), electronic medical record, vital signs, telemetry, and perhaps renal ultrasonography.

In the absence of frank evidence of "overdiuresis" such as worsening hypernatremia, with dropping blood pressure, clinical hypoperfusion, and contraction alkalosis, avoid the temptation to suspend diuretics. Alternatively, an increase in diuretic dose, or addition of a distal diuretic (ie, metolazone) may be needed to address persistent renal venous congestion as the cause of the AKI.[3] In this situation, be sure to monitor electrolytes, volume status, and renal function closely while diuretic treatment is augmented. In many such cases, the serum creatinine may actually start to decrease after a more robust diuresis is generated. In these patients, it may also be prudent to temporarily suspend antagonists of the renin-angiotensin-aldosterone system, although this remains controversial.

Management of such patients should be done collaboratively with cardiologists well versed in the treatment of cardiorenal syndrome. It may be possible that the worsening renal function in these patients represents important changes in cardiac rhythm or function (eg, low cardiac output state, new or worsening valvular disease, ongoing myocardial ischemia, cardiac tamponade, uncontrolled bradycardia or tachyarrythmia). Interventions aimed at reversing such perturbations could be the most important steps in improving cardiorenal function and reversing AKI.

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IN REPLY: We thank Dr. Freda for his remarks and observations. Certainly, the clinical importance of this entity and the challenge it poses to clinicians cannot be overemphasized. We concur with the overall message and reply to his specific comments:

We completely agree that clinical data-gathering is of paramount importance. This includes careful history-taking, physical examination, electronic medical record review, laboratory data review, and imaging. As discussed in our article, renal electrolytes will reveal a prerenal state in acute cardiorenal syndrome, and other causes of prerenal acute kidney injury (AKI) should be ruled out. The role of point-of-care ultrasonography (eg, to measure the size and respirophasic variation of the inferior vena cava) as a vital diagnostic tool has been well described, and we endorse it.[1] Moreover, apart from snapshot values, trends are also very important. This is especially pertinent when the patient care is being transferred to a new service (eg, from hospitalist service to the critical care service). In this case, careful review of diuretic dosage, renal function trend, intake and output, and weight trend would help in the diagnosis.

Inadequate diuretic therapy is perhaps one of the most common errors made in the management of patients with acute cardiorenal syndrome. As mentioned in our article, diuretics should be correctly dosed based on the patient's renal function. It is a common misconception that diuretics are nephrotoxic: in reality, there is no direct renal toxicity from the drug itself. Certainly, overdiuresis may lead to AKI, but this is not a valid concern in patients with acute cardiorenal syndrome, who are fluid-overloaded by definition.

Another challenging clinical scenario is when a patient is diagnosed with acute cardiorenal syndrome but renal function worsens with diuretic therapy. In our experience, this is a paradoxical situation and often stems from misinterpretation of clinical data. The most common example is diuretic underdosage leading to inadequate diuretic response. Renal function will continue to decline in these patients, as renal congestion has not yet been relieved. This reiterates the importance of paying close attention to urine output and intake-output data. When the diuretic regimen is strengthened and a robust diuretic response is achieved, renal function should improve as systemic congestion diminishes.

Acute cardiorenal syndrome stems from hemodynamic derangements, and a multidisciplinary approach may certainly lead to better outcomes. Although we described the general theme of hemodynamic disturbances, patients with acute cardiorenal syndrome may have certain unique and complex hemodynamic "phenotypes" that we did not discuss due to the limited scope of the paper. One such phenotype worth mentioning is decompensated right heart failure, as seen in patients with severe pulmonary hypertension. Acute cardiorenal syndrome due to renal congestion is often seen in these patients, but they also have certain other unique characteristics such as ventricular interdependence.[2] Giving intravenous fluids to these patients not only will worsen renal function but can also cause catastrophic reduction in cardiac output and blood pressure due to worsening interventricular septal bowing. Certain treatments (eg, pulmonary vasodilators) are unique to this patient population, and these patients should hence be managed by experienced clinicians.

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