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Letter

Should Fluid Balance Monitoring Take Priority Over Weight Monitoring in Type 2 Cardiorenal Syndrome?

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Patients with chronic heart failure are frequently admitted to inpatient wards and intensive care units for reasons beyond their cardiac condition, such as postoperative recovery or the management of neurological, infectious, or oncological complications. In these settings, when previously stable heart failure decompensates, fluid balance monitoring may provide greater prognostic value than weight monitoring. This is because weight gain alone does not adequately explain the development of decompensated heart failure [1]. For example, if a 70 kg patient retains 4.2 liters of fluid, which increases their body weight by 6%, only 350 mL of this fluid enters the intravascular compartment, and merely 105 mL contributes to the effective circulating blood volume, such modest arterial volume expansion rarely causes decompensation [1].

Instead, acute decompensated heart failure primarily results from a rapid shift of blood from the venous compartment (unstressed volume) into the arterial circulation (stressed volume), which dramatically elevates cardiac filling pressures [2]. This shift occurs when venous capacitance suddenly decreases in response to sympathetic and neurohormonal activation. Common triggers include hypoxia, hypercapnia, infection, acute hypovolemia (via diarrhea or hemorrhage), excessive physical exertion, or arrhythmias [2]. Majority of unstressed volume resides in the splanchnic venous bed and other abdominal capacitance vessels [3]. When adrenergic receptors in these vessels are stimulated, venous reservoir capacity rapidly contracts [3]. The resulting increase in preload raises central venous pressure and causes congestion in downstream abdominal organs, which explains why cardiorenal syndrome can develop without antecedent weight gain [2].

Of the five recognized subtypes of cardiorenal syndrome, this pathophysiological mechanism best characterizes Type 2. Accordingly, Type 2 cardiorenal syndrome should be managed by reducing cardiac filling pressures and renal venous pressure through decongestion and hemodynamic redistribution—not through aggressive volume removal aimed at weight reduction [3]. In addition to this decongestive treatment, pharmacological interventions such as angiotensin-converting enzyme inhibitors, angiotensin receptor-neprilysin inhibitors, sodium-glucose cotransporter-2 inhibitors, and splanchnic nerve modulation have also been shown to be effective in reversing this pathological hemodynamic redistribution [3].

This distinction is critical, as demonstrated by Fudim *et al.* [4] in their analysis of ultrafiltration patients via data from the Cardiorenal Rescue Study in Acute Decompensated Heart Failure (CARRESS-HF). They found that in patients with heart failure driven by venous redistribution without weight gain, ultrafiltration aimed at weight reduction decreased effective circulating volume, thereby increasing morbidity, mortality, and length of hospital stay. Patients presenting without weight gain were observed to have an ejection fraction (EF) exceeding 40%. This finding provides insight into the complex pathophysiology and management of heart failure with preserved ejection fraction (HFpEF). Conversely, the absence of adverse events from weight reduction in patients with EF below 40% suggests that heart failure progression in this population relates to weight gain from venous pooling rather than venous redistribution [4].

Marenzi *et al.* [5] further clarified these complex hemodynamic relationships by reporting that in oliguric cardiorenal syndrome, urine output increased tenfold following initial ultrafiltration. Taken together, these studies suggest that ultrafiltration in Type 2 cardiorenal syndrome should target reduction of renal venous pressure rather than weight loss per se. Indeed, urine output may serve as the earliest indicator of renal venous congestion. In patients with Type 2 cardiorenal syndrome, fluid balance monitoring may therefore be more informative than weight monitoring, though larger studies are required to confirm these observations.

With regard to monitoring techniques, studies comparing fluid balance monitoring with weight monitoring have shown a weak correlation between the two methods [6]. Which approach better guides fluid management decisions remains uncertain, leading to the current recommendation that clinicians employ both [7]. However, weight monitoring may be impractical in immobilized patients and those in intensive care units [8], whereas fluid balance monitoring can be readily performed, particularly in patients with urinary catheters.

An additional consideration is that patients admitted to intensive care units and inpatient wards can experience re-

duction in muscle, bone, and adipose tissue mass after the first week [9]. Reliance on weight monitoring alone may therefore mask persistent hypervolemia, as tissue loss offsets fluid retention and weight appears stable. This occult hypervolemia can increase morbidity and mortality, rendering fluid balance monitoring more valuable than weight monitoring in this context.

In hospitalized patients with a history of heart failure of any etiology, fluid balance monitoring should take precedence over weight monitoring for detecting Type 2 cardiorenal syndrome. Heart failure can develop in these patients even without weight gain or peripheral edema. Because venous redistribution and elevated renal venous pressure constitute the primary pathology, treatment should focus on reducing renal venous pressure rather than achieving weight loss. While alleviating renal venous congestion leads to rapid increases in urine output, thus achieving decongestion without compromising effective circulating volume, excessive ultrafiltration aimed at weight reduction may be detrimental. Further research is needed to determine optimal monitoring strategies for hospitalized patients with cardiorenal syndrome.

Key Points

• Heart failure patients who experience acute decompensation and renal impairment during hospitalization for non-cardiac reasons generally manifest Type 2 cardiorenal syndrome.

• Elevated renal venous pressure secondary to venous congestion is the predominant pathophysiological factor in Type 2 cardiorenal syndrome; therefore, therapy should target reduction of renal venous congestion and pressure rather than weight loss.

• In patients with Type 2 cardiorenal syndrome, increased urine output, rather than weight loss, may serve as the earliest indicator of reduced renal venous pressure.

• Focusing solely on weight targets may prolong hospitalization and increase morbidity and mortality, particularly in patients with heart failure with preserved ejection fraction (HFpEF).

• Because patients often require prolonged hospitalization for comorbidities, during which malnutrition can develop after one week, treatment planning based solely on weight monitoring may leave patients hypervolemic despite apparent weight stability.

• Studies comparing fluid balance monitoring with weight monitoring have yielded inconsistent results, with no clear superiority demonstrated for either method.

Availability of Data and Materials

Not applicable.

Author Contributions

All elements of the study and subsequent write-up were carried out by the author MA. The author read and approved the final manuscript. The author has participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflict of Interest

The author declares no conflict of interest.

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