Things We Do for No Reason™: Fluid Restriction for the Management of Acute Decompensated Heart Failure in Patients With Reduced Ejection Fraction

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Inspired by the ABIM Foundation's Choosing Wisely® campaign, the "Things We Do for No Reason™" (TWDFNR) series reviews practices that have become common parts of hospital care but may provide little value to our patients. Practices reviewed in the TWDFNR series do not represent clear-cut conclusions or clinical practice standards but are meant as a starting place for research and active discussions among hospitalists and patients. We invite you to be part of that discussion.

CLINICAL SCENARIO

The hospitalist enters admission orders for an 80-year-old woman with hypertension, coronary artery disease, and heart failure with reduced ejection fraction who presented to the emergency department with weight gain, lower extremity edema, and dyspnea on exertion. She has an elevated jugular venous pressure, crackles on pulmonary exam, and bilateral pitting edema with warm extremities. Labs show a sodium of 140 mmol/L and creatinine of 1.4 mg/dL. After ordering intravenous furosemide for management of acute decompensated heart failure (ADHF), the hospitalist arrives at the nutrition section of the CHF Admission Order Set and reflexively picks an option for a fluid-restricted diet.

BACKGROUND

Patients with ADHF, the leading cause of hospitalization for patients older than 65 years,[1] may present with signs and symptoms of volume overload: shortness of breath, lower-extremity swelling, and end-organ dysfunction. Before the 1980s, treatment of ADHF relied on loop diuretics, bedrest, and fluid restriction to minimize congestive symptoms.[2] Clinicians based this practice on early theories framing heart failure as primarily an issue of salt and water retention that could be counterbalanced by sodium and fluid restriction.[2]

Today, hospitalists understand heart failure with reduced ejection fraction (HFrEF) as a heterogenous disease with a shared pathophysiology in which reduced cardiac output, elevated systemic venous pressures, and/or shunting of blood away from the kidneys may all lead to decreased renal perfusion. These phenomena trigger the activation of the renin-angiotensin-aldosterone system (RAAS), leading to sodium and water retention and fluid redistribution.[2] As part of the modern day treatment regimen, providers continue to place patients on fluid-restricted diets. Guidelines support this practice.[3,4]

Since most of the existing literature on the topic of fluid restriction in ADHF relates to HFrEF (left ventricular ejection fraction [LVEF] <40%), as opposed to heart failure with a preserved ejection fraction (HFpEF, LVEF ≥50%), this review will focus on HFrEF patients. Limited existing data support extrapolating these arguments to HFpEF patients as well.[5]

WHY YOU MIGHT THINK FLUID RESTRICTION IS IMPORTANT IN THE MANAGEMENT OF ADHF IN HFREF PATIENTS

Longstanding conventional wisdom and data extrapolation from the chronic heart failure population has undergirded the practice of fluid restriction for ADHF. Current iterations of the American and European heart failure guidelines recommend fluid restriction of 1.5 to 2.0 L/day in severe ADHF as a management strategy.[3,4] The American guidelines recommend considering restricting fluid intake to 2 L/day for most hospitalized ADHF patients without hyponatremia or diuretic resistance. The guidelines base the recommendation on clinical experience and data from a single randomized trial evaluating the effects of sodium restriction on heart failure outcomes in outpatients recently admitted for ADHF.[4,6] This trial randomly assigned 232 patients with compensated HFrEF to either a normal or low-sodium diet plus oral furosemide. Researchers instructed both groups to adhere to a 1000 mL/day fluid restriction. The authors found a high incidence of readmissions for worsening congestive heart failure among a cohort of patients (n = 54) with a normal sodium diet who were excluded from randomization due to inability to adhere to the prescribed fluid restriction.[6] Notably, this study did not evaluate patients receiving treatment for ADHF and was not designed to investigate the role of fluid restriction for the treatment of ADHF.

A subsequent study by the same investigators looked more deliberately, although not singularly, at outpatient fluid restriction. This study randomly assigned 410 patients with compensated HFrEF into eight groups by fluid intake (1 L vs 2 L), salt intake (80 mmol vs 120 mmol), and furosemide dose (125 mg twice daily vs 250 mg twice daily). At 180 days, the group receiving the fluid-restricted diet with higher sodium intake and higher diuretic dose had the lowest risk of hospital readmission.[7]

Results from these studies of the chronic, compensated heart failure population, in conjunction with longstanding

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conventional wisdom, have influenced the management of patients hospitalized with ADHF.

WHY FLUID RESTRICTION IN THE MANAGEMENT OF ADHF IN HFREF PATIENTS MIGHT NOT BE HELPFUL

From a pathophysiologic perspective, fluid restriction in ADHF may counterproductively lead to RAAS activation.[8] Congestion develops when arterial underfilling leads to RAAS activation, triggering sodium and water retention.[2] Furthermore, RAAS activation, as measured by plasma levels of renin, angiotensin II, and aldosterone, correlates with prognosis and mortality in chronic HFrEF.[9] Analyses from one of the largest databases of biomarkers from ADHF suggest that RAAS is further upregulated during decongestive therapy.[10] While researchers have not studied the effects of fluid restriction on RAAS activation in ADHF patients, extrapolating from these data one may question whether fluid restriction in ADHF patients may further drive RAAS activation. Further activation may contribute to adverse incident outcomes such as worsening renal function.

The most relevant and compelling evidence against fluid restriction to date comes from Travers et al,[11] who conducted the first randomized controlled trial examining fluid restriction in ADHF patients. Their small study compared restricted (1 L fluid restriction) vs liberal (free fluid) intake in hospitalized patients with ADHF and demonstrated no difference in duration or daily dose of intravenous diuretics, time to symptomatic improvement, total daily fluid output, or average hospitalization weight loss between the two arms. Furthermore, researchers withdrew more patients in the fluid-restricted arm due to a sustained rise in serum creatinine, suggesting potential harm of this intervention.[11] The sample size (N = 67) and fluid-intake difference of only 400 mL between the two groups limited the study results.

In a subsequent randomized controlled trial, Aliti et al[12] examined the clinical outcomes of even more aggressive fluid restriction (800 mL/day) and sodium restriction (800 mg/day) versus liberal intake (at least 2.5 L fluid/day and approximately 3-5 g sodium/day) in hospitalized patients with ADHF (N = 75). While this study evaluated both fluid and sodium restriction, it produced relevant results. The study demonstrated no significant difference in weight loss, use of diuretics, or rehospitalization between the study arms.[12] At 30-day follow-up, researchers found that patients in the intervention group had more congestion and an increased likelihood of having a B-type natriuretic peptide (BNP) level greater than 700 pg/mL. In the subset of all patients with an elevated BNP level greater than 700 pg/mL at the end of the study, patients in the intervention group had a significantly higher rate of readmission (7 out of 22) compared with controls (1 of 20). Moreover, the fluid-restricted group had 50% higher perceived thirst values compared to the control group.[12] The sensation of thirst not only reduces quality of life, but, given that angiotensin II stimulates thirst, it may reflect RAAS activation.[13] For these reasons, clinicians should consider this side effect seriously, especially when the literature lacks evidence of the benefits from fluid restriction.

WHEN FLUID RESTRICTION IS HELPFUL IN THE MANAGEMENT OF DECOMPENSATED HEART FAILURE IN HFREF PATIENTS

Fluid-restrict patients who have chronic hyponatremia (Na <135 mmol/L) due to end-stage HFrEF in select circumstances. Hyponatremia develops in heart failure primarily because of the body's inability to excrete free water due to non-osmotic arginine vasopressin secretion.[4] Other processes contribute to hyponatremia, including increased free water intake due to angiotensin II stimulating thirst and decreased glomerular filtration rate limiting the kidney's ability to excrete free water. Since hyponatremia in heart failure primarily occurs due to derangements of free water regulation, limiting free water intake may help; the American College of Cardiology/American Heart Association and European heart failure guidelines explicitly recommend this strategy for patients with stage D heart failure.[3,4] However, no available randomized data support this practice, and observational data suggest that fluid restriction has limited impact on hyponatremia in ADHF.[14] Guidelines also suggest employing fluid restriction in patients with diuretic resistance as an adjunctive therapy.

Twenty-nine percent of patients with ADHF have comorbid chronic kidney disease (CKD).[15] Providers often prescribe patients with advanced CKD salt- and fluid-restrictive diets due to more limited abilities in sodium and free water excretion. However, no studies have examined the effects of fluid restriction alone without salt restriction in the CKD/ADHF population.

WHAT YOU SHOULD DO INSTEAD

In the present day of evidence-based pharmacologic therapies, research indicates that fluid-restriction does not help and potentially may harm. Instead, treat hospitalized HFrEF patients with ADHF with modern, evidence-based pharmacologic therapies and allow the patients to drink when thirsty.

RECOMMENDATIONS

- Treat patients with ADHF and reduced ejection fraction with evidence-based neurohormonal blockade and initiate loop diuretics to alleviate congestion.
- Allow patients with ADHF and reduced ejection fraction to drink when thirsty in the absence of hyponatremia.
- Consider initiating fluid restriction in patients with ADHF and concurrent hyponatremia and/or diuretic resistance. There is little evidence to guide setting specific limits on fluid intake.

CONCLUSION

The hospitalist starts the patient admitted for ADHF on an intravenous loop diuretic, continues her home beta blocker and angiotensin-converting enzyme inhibitor, and does not impose any fluid restriction. Her symptoms of congestion resolve, and she is discharged.

Hospitalists often treat patients with ADHF and reduced

ejection fraction with fluid restriction. However, limited evidence supports this practice as part of the management of ADHF. Fluid restriction may have unintended adverse effects of increasing thirst and worsening renal function and quality of life.

What do you do? Do you think this is a low-value practice? Is this truly a "Thing We Do for No Reason"? Let us know what you do in your practice and propose ideas for other "Things We Do for No Reason" topics. Please join in the conversation online at Twitter (#TWDFNR)/Facebook and don't forget to "Like It" on Facebook or retweet it on Twitter.

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