



Updates in Hospital Medicine

Updates in Hospital Medicine: Nephrology

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UPDATE I

“On-Demand” Dialysis Appears Safe and Possibly Beneficial to Dialysis-Requiring AKI

Hospitalists frequently manage patients with dialysis-requiring acute kidney injury (AKI-D). Standard practice typically defaults to scheduled thrice-weekly hemodialysis until recovery is present.^{1,2} However, frequent dialysis may inadvertently prolong renal injury through subclinical intradialytic hypotension and repetitive ischemic insults and/or inflammation.³ The recent LIBERATE-D trial investigated whether a conservative, indication-driven (“on-demand”) dialysis strategy improves kidney recovery compared to the conventional scheduled approach.⁴

This multicenter, unblinded randomized clinical trial enrolled 220 hemodynamically stable adults with AKI-D who had already initiated renal replacement therapy at four U.S. academic centers.⁴ Patients requiring vasopressors or with severe baseline chronic kidney disease (eGFR <15 mL/min/1.73 m²) were excluded. Using 1:1 randomization stratified by site and baseline eGFR, participants were assigned to a “on-demand” strategy, where dialysis occurred only for specific metabolic triggers (serum urea nitrogen >112 mg/dL, potassium >6 mmol/L, pH <7.15, refractory volume overload) or clinician judgment, versus a conventional strategy of scheduled thrice-weekly dialysis until recovery markers were met. The primary outcome was kidney function recovery at hospital discharge, defined as survival and dialysis independence for at least 14 consecutive days.⁴

Overall, at discharge, the “on-demand” dialysis group experienced higher rates of recovery (unadjusted odds ratio, 1.76 [95% CI, 1.02-3.03; P=0.04]). Seventy of 109 patients (64%) in the “on-demand” group achieved recovery compared with 55 of 109 patients (50%) in the conventional group (absolute risk difference 13.8%; 95% CI, 0.8%-26.8%; P=.04). This significance was lost after prespecified adjustments for baseline characteristics (baseline eGFR, age, sex, race, ethnicity), with adjusted odds ratio of 1.56 (95% CI, 0.86-2.84; P=0.15).

Beyond the primary recovery rate finding and the faster median time to recovery (2 days vs 8.5 days), the study’s secondary outcomes highlighted several clinical advantages. The “on-demand” group received significantly fewer weekly sessions (median 1.8 vs 3.1) and experienced fewer episodes of dialysis-associated hypotension (69 vs 97 events). There were no significant differences in mortality or severe adverse events (e.g., arrhythmias, severe electrolyte derangements), indicating that the reduced frequency did not compromise patient safety.

While the unadjusted results favor the “on-demand” approach, the findings have important caveats. Notably, the significance of the primary outcome was attenuated after prespecified adjustment for baseline characteristics, suggesting the effect size might be overestimated. Additionally, fidelity to trial protocol was challenging; “clinician judgment” rather than strict physiological criteria triggered nearly half (45.5%) of the dialysis sessions in the “on-demand” arm, highlighting the subjectivity involved in implementation. These findings apply strictly to hemodynamically stable patients, not the critically ill. Further validation in a larger, pragmatic trial is required to

confirm that “on-demand” dialysis definitively drives the observed recovery benefit.

Take-away: *For hemodynamically stable inpatients with AKI-D, consider partnering with nephrology on explicit trigger-based dialysis rather than automatic thrice-weekly treatments. This approach appears safe and may even accelerate kidney recovery, possibly by reducing dialysis-associated renal hypoperfusion. In addition to being clinically meaningful, the broad implementation of this approach may reduce overall healthcare resource utilization and lower the carbon footprint of dialysis for hospitalized patients.*

UPDATE 2

Bicarbonate for Critically Ill Patients with AKI and Metabolic Acidosis May Avert the Need for Dialysis

Metabolic acidosis is a common complication in patients hospitalized with acute kidney injury (AKI). Its etiology is often multifactorial and may include overproduction of lactic acid or other unmeasured anions, resulting in high anion-gap acidosis, or bicarbonate loss through the gastrointestinal or urinary tracts, leading to non anion-gap acidosis.⁵ Severe acidemia can lead to life-threatening cardiovascular complications, including impaired cardiac contractility, hypotension with a blunted response to vasopressors, and arrhythmias.⁶

Bicarbonate replacement to restore buffering capacity has become a common practice in patients with metabolic acidosis. However, the effect of bicarbonate therapy is controversial, including its impact on mortality and the need for kidney replacement therapy (KRT).⁷ The 2018 BICAR-ICU trial (n=389) found that bicarbonate therapy did not improve overall 28-day mortality or organ failure in severe metabolic acidosis.⁸ However, patients with KDIGO stage 2–3 AKI had lower mortality and less need for KRT.

This unexpected signal provided the impetus for BICAR-ICU 2, a recently published multicenter, randomized, open-label clinical trial.⁹ In this study, 627 adult patients with severe metabolic acidemia (pH ≤7.20) and moderate to severe AKI were randomized 1:1 to receive either sodium bicarbonate infusion or no bicarbonate (control group). Bicarbonate was administered as 4.2% intravenous aliquots of 125–250 mL, targeting a pH of 7.30 or higher, which was confirmed by blood gas analysis. The median age was 67 years, and approximately 60% were male.

The authors found that sodium bicarbonate treatment did not impact the primary outcome of day 90 all-cause mortality, which was 62% in both groups.⁹ Among 18 secondary outcomes, including length of stay and vasopressor use, only KRT use had a difference between the groups. KRT use was significantly lower in the bicarbonate group compared with the control group (35%

vs 50%; absolute difference –15.5%; 95% CI, –23.1 to –7.8). This reduction was largely driven by fewer acidosis-driven dialysis initiation (41% in the intervention group vs 64% in the control group). Key limitations include open-label design with cross-over, limited generalizability outside the ICU given the high mortality rate (62%), and the practical challenge of frequent blood gas monitoring in non-ICU patients without arterial lines.

Take-away: *Hospitalists may consider short-term bicarbonate therapy for patients with severe metabolic acidosis and moderate-to-severe AKI, as it appears safe. While it does not appear to impact mortality, it may help avert the need for KRT in this high-risk population. The cornerstone of treatment, however, remains addressing the underlying cause of acidosis.*

UPDATE 3

SGLT2 inhibitors should be continued in most patients that experience a UTI

Sodium-glucose cotransporter 2 (SGLT2) inhibitors were originally introduced as a glucose-lowering therapy for type 2 diabetes mellitus (DM).¹⁰ Their use has since expanded to non-diabetic patients with heart failure¹¹ and chronic kidney disease,¹² making them a frequently encountered medication class in hospitalized patients. Because SGLT2 inhibitors promote glucosuria, there has been concern about an increased risk of urinary tract infections (UTIs).^{13,14} In response, the U.S. Food and Drug Administration added warnings to SGLT2 inhibitor labeling regarding the risk of UTIs.¹⁵ Although no official guidelines recommend discontinuing SGLT2 inhibitors following a UTI, clinicians often choose to stop the medication in affected patients.¹⁶

The recent population-based index cohort study by Wu et al. (2025) sought to evaluate the clinical impacts of UTIs in patients with type 2 DM treated with SGLT2 inhibitors.¹⁷ The authors also used a target trial emulation to estimate outcomes for patients whose SGLT2 inhibitors were discontinued. Using a territory-wide database in Hong Kong, the authors identified 62,000 adult patients (mean age 63 years; 64% male) with type 2 DM who were prescribed SGLT2 inhibitors over a 7-year period.¹⁷ Of these patients, 3921 (6.4%) were diagnosed with at least one UTI. Patients who experienced UTI had significantly higher risks of primary cardiovascular composite outcomes (e.g., stroke, myocardial infarction, all-cause mortality) and renal composite outcomes (e.g., end-stage renal disease, 50% reduction in GFR, all-cause mortality) compared to patients without UTI (respectively, HR: 3.2, 95% CI: 2.9–3.5; HR: 2.5, 95% CI: 2.3–2.7).

After a UTI, approximately one-third (32%) of patients discontinued their SGLT2 inhibitors. Importantly, stopping these medications did not reduce the risk of re-

current UTI (HR: 1.0, 95% CI: 0.2-4.3). However, the patients who stopped SGLT2 inhibitors experienced significantly higher rates of both cardiovascular and renal composite outcomes (HR: 1.4 for both outcomes, 95% CI: 1.2-1.5). These findings were consistent across all subgroup analyses, including age, sex, diabetes duration, ACEIs or ARBs, smoking, glycemic control, and baseline renal function.

A major limitation of this study is that, despite target trial emulation, this observational dataset cannot eliminate the possibility that patients who discontinued SGLT2 inhibitors were systematically sicker at the time of UTI. Moreover, UTI and outcomes were ascertained from routine documentation and coding, and “discontinuation” was inferred from prescribing/refill data rather than directly observed use. Finally, findings from one regional health system may not apply to other populations or practice settings.

Take-Away: *This observational study suggests that SGLT2 inhibitors should generally be continued in most patients who experience a UTI. Discontinuation of these*

medications does not appear to reduce the risk of recurrent UTIs but is associated with worse cardiovascular and renal outcomes. Since patients who develop UTIs already represent a population with higher cardiovascular and renal risk, it is important to reap the protective benefits that SGLT2 inhibitors provide.

Disclosures/Conflicts of Interest

None

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