ECHO IDAHO

Managing Heart Failure in Primary Care

# Outpatient Decongestion Strategies in Heart Failure

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12/3/25



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# Disclosures/References

**No industry disclosures.**

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# Objectives

- Review patient selection for outpatient decongestion in heart failure
- Outline bedside assessment of volume status
- Delineate outpatient pharmacologic decongestion and uptitration strategies
- Discuss monitoring, follow-up, and escalation criteria for the inpatient setting
- Apply concepts to a practical case example

# Why Outpatient Decongestion?

- Heart failure is the leading cause of US hospitalization and readmission for patients over 65
- Many exacerbations are driven primarily by congestion without shock or hypoxia and without underlying high-risk provocation
- Stable patients in exacerbation can be managed safely at home
- Effective outpatient diuresis can evolve to a patient-centered strategy to intervene early (on the basis of weight and subtle symptoms) for future exacerbations
- Outpatient management can:
    - Reduce hospitalizations and costs
    - Improve patient engagement and quality of life

# Defining Outpatient Decongestion

- Treatment of symptomatic pulmonary and systemic venous congestion in a patient with known or suspected HF can be achieved at home in patients stable enough to remain out of the hospital
  - Decongestion tools:
    - Oral loop diuretics (initiation or dose escalation)
    - Short courses of adjunctive thiazide-type diuretics
    - Clinic or day-hospital IV loop diuretics if available
  - Should always be embedded within guideline-directed medical therapy (GDMT)

# Patient Selection – Who Is Appropriate?

- Hemodynamically stable:
  - No shock, SBP typically ≥ 90–100 mmHg
  - Warm extremities, no cool/clammy skin– no indirect evidence for early cardiogenic shock
  - Respiratory status:

    No severe resting dyspnea or need for high-flow O2
  - Cardiac rhythm

    No significant tachycardia (new atrial fibrillation?) or bradycardia

Other considerations:

- Able to take oral meds and maintain PO intake
- Reliable for daily weights and rapid follow-up
- Support at home or easy access to care

# Key Exclusion Criteria– Who is not appropriate?

High risk presentations:

- Hypotension or signs of poor perfusion
- Chest pain suggestive of ACS
- New syncope, rapid AF with hemodynamic compromise, unexplained tachy- or bradycardia
- Severe hypoxia or respiratory distress
- Acute kidney injury, severe electrolyte derangements, or confusion
- Social factors: unreliable follow-up, inability to understand or adhere to plan

# Initial Assessment – History & Vitals

- Clarify the antecedent trajectory:
    - Onset and progression of dyspnea, orthopnea, PND, edema, weight gain
    - Triggers: dietary indiscretion, missed meds, infection, arrhythmia, ischemia
- Vital signs and weight:
    - BP, HR, RR, $SpO_2$ (rest and with minimal exertion)
    - Compare current weight to dry weight if known
- History counts: Review HF phenotype and EF, prior responses to diuretics, baseline renal function

# Physical Exam – Volume Status

- Neck veins: JVP height, waveform; hepatojugular reflux
- Lungs: rales, wheeze, dullness suggesting effusions
- Heart: S3, murmurs (MR, AS), rhythm
- Peripheral: edema (pitting, distribution), ascites, hepatomegaly
- Perfusion: capillary refill, extremity temperature, mental status
- Point-of-care ultrasound (if available): IVC size/collapsibility, B-lines

# Baseline Testing Before or During Decongestion

Laboratory tests:

- BMP (Na, K, $CO_2$, BUN, creatinine), Mg
- Consider BNP/NT-proBNP to support diagnosis or track trend

Additional diagnostics as indicated:

- CBC (anemia, infection), troponin if ischemia suspected
- LFTs if marked hepatic congestion
- TFTs if additional signs or symptoms suggestive
- Chest x-ray for pulmonary congestion, effusions
- Echocardiogram if HF phenotype / EF unknown or significantly changed

# But why diuretics for decongestion?

*"The effects of diuretics on morbidity and mortality are uncertain*. As such, diuretics should not be used in isolation but always combined with other GDMT for HF that reduces hospitalizations and prolongs survival."

| COR | LOE | Recommendations |
|---|---|---|
| 1 | B-NR | 1. In patients with HF who have fluid retention, diuretics are recommended to relieve congestion, improve symptoms, and prevent worsening HF (1-5). |
| 1 | B-NR | 2. For patients with HF and congestive symptoms, addition of a thiazide (e.g., metolazone) to treatment with a loop diuretic should be reserved for patients who do not respond to moderate- or high-dose loop diuretics to minimize electrolyte abnormalities (6). |

# Why diuretics?

- Randomized controlled trials with diuretics have shown their effects to increase urinary sodium excretion, decrease physical signs of fluid retention, and improve symptoms, QOL, and exercise tolerance, without reduction in mortality.
- Nonrandomized OPTIMIZE-HF (Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure) registry revealed reduced 30-day all-cause mortality and hospitalization for HF with diuretic use compared with no diuretic use after hospital discharge for HF.

# Oral Loop Diuretics – Core Strategy

- Loop agents: furosemide, bumetanide, torsemide
  - Typical outpatient starting dose (furosemide):
    - Furosemide 20–40 mg once or twice daily; higher in CKD
    - 40 mg furosemide = 1 mg bumetanide = 20 mg torsemide
  - Short-term intensification for congestion:
    - Double usual total daily oral dose
    - Split doses (e.g., morning + early afternoon) to reduce rebound $Na^+$ retention
    - Bioavailability and half life affect response
  - Monitor response: weight change, urine output within hours of administration, overall congestive symptom relief

| Drug | Initial Daily Dose | Maximum Total Daily Dose | Duration of Action |
|---|---|---|---|
| **Loop diuretics** | | | |
| Bumetanide | 0.5–1.0 mg once or twice | 10 mg | 4–6 h |
| Furosemide | 20–40 mg once or twice | 600 mg | 6–8 h |
| Torsemide | 10–20 mg once | 200 mg | 12–16 h |
| **Thiazide diuretics** | | | |
| Chlorthiazide | 250–500 mg once or twice | 1000 mg | 6–12 h |
| Chlorthalidone | 12.5–25 mg once | 100 mg | 24–72 h |
| Hydrochlorothiazide | 25 mg once or twice | 200 mg | 6–12 h |
| Indapamide | 2.5 mg once | 5 mg | 36 h |
| Metolazone | 2.5 mg once | 20 mg | 12–24 h |

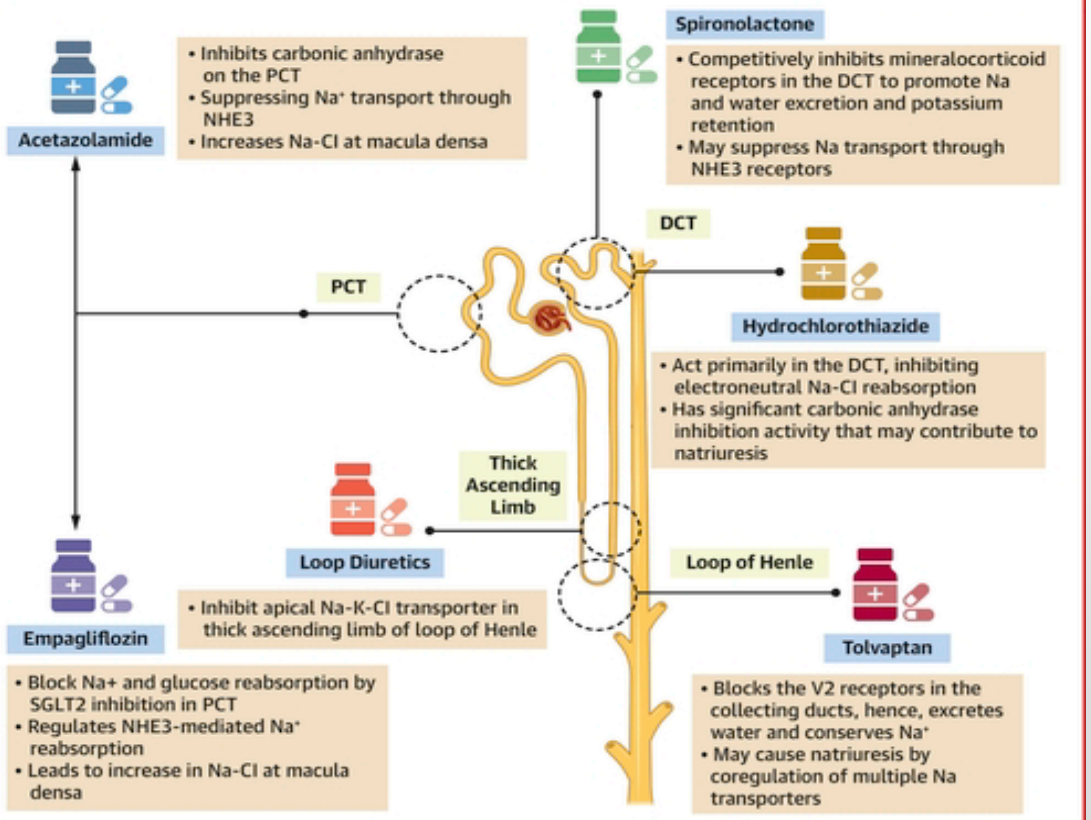
# Managing Diuretic Resistance (Outpatient)

- Recognize signs of diuretic resistance
  - Minimal weight loss or symptom improvement despite adequate loop dosing (goal 0.5 - 1kg daily–need to address if unmet)
  - Check contributing factors:
    - High $Na^+$ intake, NSAIDs, poor medication adherence
    - Hypotension limiting dose, significant CKD
  - Strategies:
    - Increase loop dose within safe range
    - Switch to higher-bioavailability loop (e.g., bumetanide/torsemide)
    - Consider a single supervised IV loop dose in clinic/day-hospital with labs

# Sequential Nephron Blockade

- Thiazide-type add-on (e.g., metolazone, chlorthalidone, HCTZ):
  - Low dose given 30–60 min before loop diuretic
  - Use cautiously and briefly due to risk of severe hyponatremia and hypokalemia (especially metolazone)
- Mineralocorticoid receptor antagonist spiro/eplerenone):
  - Primarily for mortality benefit in HFrEF; reduces risk of hypokalemia and yields mild additional natriuresis
- Acetazolamide: Blocks carbonic anhydrase, limits HCo3/Na/H20 resorption in the proximal tubule
- Tolvaptan: Blocks vasopressin receptor to increase free water diuresis

**CENTRAL ILLUSTRATION: Mechanism of Diuretics Evaluated in Acute Heart Failure Trials**

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**Tariq Jamal Siddiqi et al. *J Am Coll Cardiol HF* 2025; 13:14-27.**



JACC Heart Failure

# SGLT2 Inhibitors in HF and Congestion

- Agents: dapagliflozin, empagliflozin (benefit in HFrEF and HFpEF)
  - Mechanisms:
    - Mild osmotic diuresis and natriuresis
    - Reduced HF hospitalization and CV death in trials
  - Practical points:
    - Usually started once reasonably euvolemic and hemodynamically stable
    - Avoid initiation in severe AKI or dialysis-dependent patients
    - Educate about genital infections, volume symptoms, and sick-day rules

# Monitoring Renal Function & Electrolytes

- Before intensifying diuretics: check baseline BMP and Mg
  - Recheck within 3–7 days (or sooner with higher-risk patients):
    - Creatinine/BUN: small transient rise may be acceptable if congestion improves
    - Potassium: watch for hypokalemia with high-dose loop or thiazides
    - Sodium: avoid or promptly address significant hyponatremia
  - Adjust diuretics, RAAS blockade, and MRAs based on lab trends and symptoms
  - Urine sodium may not be practical for guiding outpatient decongestion

# Continue Guideline-Directed Therapy

- Maintain evidence-based GDMT whenever possible (ACEi/ARB/ARNI, beta-blocker, MRA, SGLT2i)
- Consider temporary dose reduction or holding RAAS blockers if:
  - Symptomatic hypotension
  - Marked rise in creatinine or $K^+$
- Beta-blockers are usually continued unless clear low-output state or cardiogenic shock
- MRA can be continued for augmented diuresis and potassium support, with follow up of electrolytes/renal function
- SGLT2i can be continued with follow up of renal function

# Non-Pharmacologic Decongestion Support

- Dietary sodium restriction (e.g., ~2g/day, individualized)
- Moderate fluid restriction in markedly hypervolemic or hyponatremic patients– 2 - 2.5L/day
- Daily weights at home:
    - Same scale, after awakening, similar clothing
- Physical activity as tolerated; avoid prolonged bed rest when stable
- Address sleep apnea and other comorbid conditions
- Continue to monitor for signs/symptoms of evolving infection or other exacerbation trigger(s)

# When to Escalate to ED or Inpatient Care

- Immediate evaluation if:
  - Rest dyspnea, tachypnea, or new need for high-flow $O_2$ / NIV
  - Chest pain, suspected ACS, or new neurologic deficits
  - SBP < 90 mmHg with signs of poor perfusion
  - Syncope, sustained ventricular arrhythmias, rapid AF with instability
  - Confusion, agitation, or inability to maintain oral intake
  - Rapid rise in creatinine, severe electrolyte abnormalities, or failure of outpatient diuresis

# Documentation & Follow-Up Planning

**Document:**

- Baseline symptoms, weight, vital signs, exam findings
- Exact diuretic regimen and any temporary or permanent dose changes or weight-based regimen
- Safety parameters and explicit instructions for escalation in care for patient and family

**Schedule follow-up:**

- Phone or telehealth check within 48–72 h after major changes
- In-person review and labs within 1 week (earlier if high risk)
- Update long-term HF management and advance care planning as appropriate

# Key Takeaways & Practical Algorithm

1. Confirm HF-related congestion and rule out high risk signs
2. Select hemodynamically stable, reliable patients for outpatient decongestion approach
3. Intensify loop diuretic therapy (2 - 2.5 times baseline) ± carefully monitored combination strategies
4. Monitor weight, symptoms, vitals, renal function, and electrolytes closely– communicate by phone/tele-chart at 2–3 day interval
5. Maintain or optimize GDMT whenever feasible
6. Have clear thresholds for escalation to ED or inpatient care
7. Embed decongestion within a broader HF disease-management program, including early detection and patient-centered strategy

# Questions?