Cureus
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Open Access Original Article

Assessment, Initiation, Customization, Adjustment, and Reassessment (AICAR) Consensus Statement on Personalized Loop Diuretic Therapy and Decongestion in Heart Failure: A Modified Delphi Consensus Study

Review began 10/23/2026
Review ended 11/08/2026
Published 11/11/2026

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DOI: 10.7759/cureus.119505

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Abstract

Background: Congestion is a major driver of symptoms, hospitalization, and early readmission in heart failure. Despite the central role of loop diuretics, substantial variability persists in congestion assessment, dosing, administration, response monitoring, treatment escalation, renal safety, and discharge planning.

Objective: The study aimed to establish expert consensus on a personalized, response-guided approach to decongestion, primarily in hospitalized patients with acute heart failure, using the Assessment, Initiation, Customization, Adjustment, and Reassessment (AICAR) framework.

Methods: A multidisciplinary steering group developed evidence-informed statements across seven domains encompassing congestion assessment, loop-diuretic initiation, treatment customization, response-guided adjustment, renal safety and discharge planning, adjunctive therapies, and implementation. In Round 1, 33 experts rated 46 statements using a four-point Likert scale with an additional outside-scope option. Nineteen statements were retained without rerating, while 24 revised or consolidated statements underwent Round 2 reassessment. Respondent-level linkage confirmed participation of the same 33 experts in both rounds (100% retention). Agreement was defined as the proportion selecting agree or strongly agree, excluding outside-scope responses. Strong and very strong consensus were predefined as ⩾75% and ⩾90% agreement, respectively.

Results: In Round 1, 35 (76.1%) statements achieved very strong consensus, 10 (21.7%) strong consensus, and 1 (2.2%) did not reach consensus. All 24 reassessed statements achieved consensus in Round 2, with 21 (87.5%) attaining very strong consensus. The definitive 43-statement set comprised 37 (86.0%) statements with very strong and 6 (14.0%) with strong consensus. Mean agreement across the seven domains ranged from 90.4% to 92.1%. Comparatively lower agreement concerned acetazolamide use (83.3%), individualized loop-diuretic selection (84.8%), and torsemide preference for managing tolerance (84.8%). The overall AICAR framework achieved 90.9% agreement. Post hoc analysis identified minority disagreement despite attainment of consensus thresholds.

How to cite this article
Kumar S, Banerjee S, Pandey B, et al. (November 11, 2026) Assessment, Initiation, Customization, Adjustment, and Reassessment (AICAR) Consensus Statement on Personalized Loop Diuretic Therapy and Decongestion in Heart Failure: A Modified Delphi Consensus Study. Cureus 18(11): e119505. DOI 10.7759/cureus.119505

Conclusions: AICAR provides a consensus-derived organizational framework integrating multimodal congestion assessment, individualized loop diuretic therapy, early response-guided adjustment, renal safety, and discharge reassessment. Although panel continuity was confirmed, expert agreement does not independently validate specific therapeutic thresholds or establish clinical effectiveness. Prospective implementation and comparative outcome studies are warranted.

Categories: Cardiology, Nephrology, Cardiac/Thoracic/Vascular Surgery
Keywords: congestion, consensus statement, decongestion, diuretic resistance (dr), heart failure, loop diuretic, modified delphi, urinary sodium

Introduction

Heart failure remains a major cause of morbidity, mortality, impaired quality of life, and healthcare utilization. Across the spectrum of left ventricular ejection fraction, congestion is a dominant reason for clinical deterioration and hospital presentation, and diuretics are recommended to relieve fluid retention and symptoms [1,2].

Congestion is not synonymous with weight gain or peripheral edema. It reflects a multidimensional state involving increased cardiac filling pressures, venous hypertension, sodium retention, interstitial fluid accumulation, and organ congestion. These processes may coexist in different proportions, and clinical examination, natriuretic peptides, echocardiographic indices, inferior vena cava assessment and lung ultrasound provide complementary rather than interchangeable information [3-5].

The distinction between symptomatic improvement and complete decongestion is clinically important. Dyspnea may improve before filling pressures and venous congestion normalize, while residual intravascular or tissue congestion at discharge identifies patients at increased risk of death and rehospitalization [4-6].

Loop diuretics remain the principal pharmacological treatment for rapid decongestion. Nevertheless, their use varies widely in agent selection, oral-to-intravenous conversion, initial dose, frequency, bolus versus infusion, monitoring of urine output or natriuresis, escalation, sequential nephron blockade, interpretation of worsening renal function, and timing of transition to oral therapy [1-3].

Recent trials have clarified several components of this pathway without establishing a single universally applicable regimen. TRANSFORM-HF did not demonstrate superiority of torsemide over furosemide for mortality after heart failure hospitalization, and a mechanistic substudy did not confirm a consistent pharmacokinetic or pharmacodynamic advantage of torsemide at commonly used conversion ratios [7,8]. Acetazolamide improved early successful decongestion when added to standardized loop diuretic therapy in ADVOR, while hydrochlorothiazide increased weight loss and diuretic response in CLOROTIC at the cost of closer renal and electrolyte surveillance [9,10]. Empagliflozin initiated after clinical stabilization improved a hierarchical clinical benefit outcome in EMPULSE, whereas DAPA-RESIST showed that dapagliflozin should not be treated as a substitute for metolazone in established loop-diuretic resistance [11,12].

Early urinary sodium and urine output strategies have also gained attention. The PUSH-AHF randomized trial and the multicenter ENACT-HF implementation study support the feasibility of protocolized natriuresis-guided escalation, although definitive improvement in post-discharge clinical outcomes has not yet been established [13,14]. Ultrafiltration may remove fluid effectively in selected patients, but contemporary evidence does not support its routine use as first-line therapy [15]. In stable euvolemic outpatients, limited evidence suggests that cautious loop-diuretic withdrawal may be feasible in selected patients receiving optimized disease-modifying therapy [16].

These findings expose a practical gap between trial evidence, guideline recommendations, and the sequence of decisions required at the bedside. We therefore developed the Assessment, Initiation, Customization, Adjustment, and Reassessment (AICAR) framework to structure personalized decongestion while preserving clinical judgment. The objective of this modified Delphi study was to develop an evidence-informed, expert consensus framework to guide personalized loop diuretic therapy and decongestion in heart failure, drawing primarily on clinical experience within Indian tertiary care settings. The AICAR framework aims to facilitate structured clinical decision-making and contextual adaptation rather than serve as a formal clinical practice guideline or independently validated therapeutic protocol.

Materials And Methods

Study design and reporting approach

A modified Delphi consensus design was used because many practical aspects of loop diuretic therapy are clinically consequential but are incompletely resolved by randomized evidence. The approach combined evidence-informed statement generation, anonymous rating, predefined consensus thresholds, iterative statement refinement, and a stopping rule based on Round 2 consensus (Figure *1*). Reporting was structured with reference to the ACcurate COnsensus Reporting Document (ACCORD) guidance and the reporting

architecture of a contemporary cardiovascular Delphi consensus study [17,18].

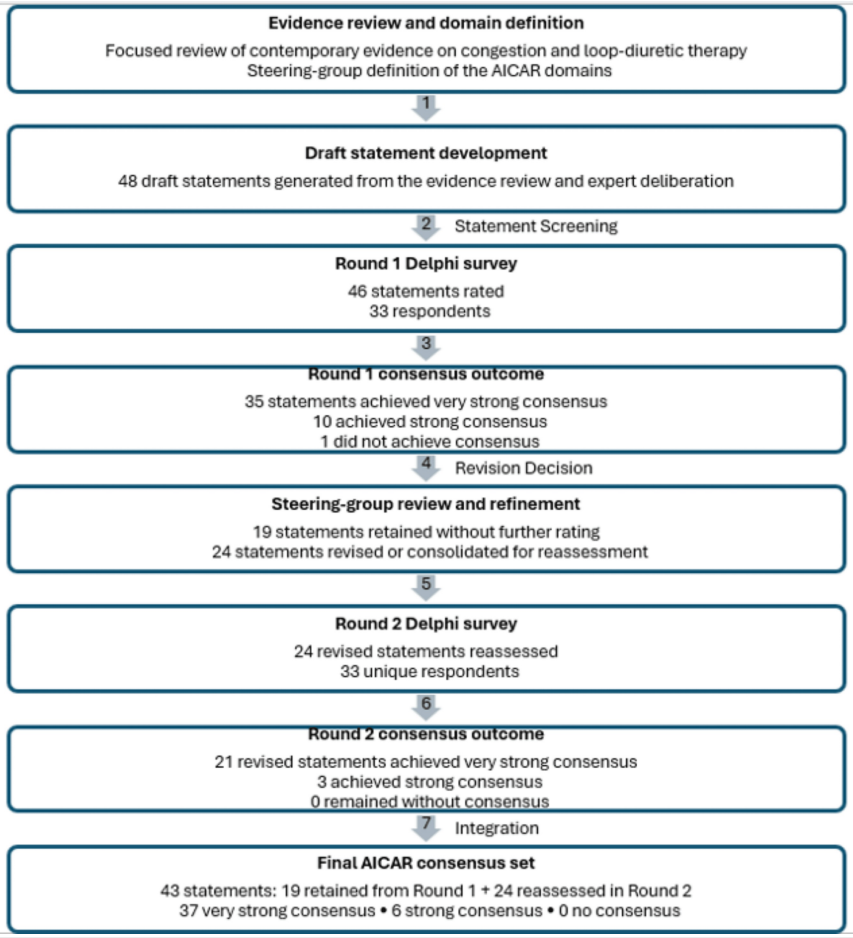


FIGURE 1: Modified Delphi study flow

Scope and conceptual framework

The scope included adult acute and chronic heart failure with clinically relevant congestion. The AICAR framework was specified before wider panel voting: Assessment of congestion; Initiation of loop diuretic therapy; Customization according to patient phenotype; Adjustment using early objective response; and Reassessment of congestion, renal safety, oral therapy, and follow-up. Additional statements addressed sequential nephron blockade, acetazolamide, sodium-glucose cotransporter 2 (SGLT2) inhibitors, mineralocorticoid receptor antagonists, ultrafiltration, implementation pathways, and patient education.

Evidence review

A focused literature review evaluated guideline recommendations, randomized trials, mechanistic studies, prospective cohorts, and contemporary consensus documents relating to congestion assessment, loop-diuretic pharmacology, urinary sodium, diuretic resistance, renal function changes, adjunctive decongestion, ultrafiltration, transition to oral therapy, and diuretic de-escalation. Randomized evidence was prioritized where available; mechanistic and observational evidence informed statements where trial evidence remained incomplete. The review was designed to support consensus development rather than to function as a systematic review or formal evidence-grading exercise.

Steering group and statement generation

A multidisciplinary steering group with expertise relevant to heart failure decongestion reviewed the evidence, defined the domains, and drafted the statements. The group considered clinical relevance, clarity, feasibility, redundancy, consistency with available evidence, and suitability for multidisciplinary voting. Forty-eight draft statements were initially generated. Following internal review, 46 statements entered Round 1.

Delphi panel and recruitment

Eligible respondents were healthcare professionals involved in the care of patients with acute or chronic heart failure, including cardiologists, internal medicine physicians, nephrologists, and clinical pharmacologists. Invitations were circulated through professional, academic, and clinical networks. Steering group members did not participate in wider panel voting. In the Indian healthcare setting, heart failure management is routinely undertaken by cardiologists across different subspecialties, as dedicated heart failure specialists and specialized heart failure services are not uniformly available. Furthermore, majority of the respondents were associated with high-volume tertiary care centers with substantial exposure to patients with acute and chronic heart failure. Participation was voluntary, and completion of the survey indicated consent.

Round 1

Round 1 comprised 46 statements across seven domains. Respondents selected "strongly disagree," "disagree," "agree," "strongly agree," or "outside my scope of practice." Thirty-three analyzable responses were included. Statement-level response distributions and agreements were reviewed by the steering group. Statements were retained, clarified, combined, or reframed according to the observed distribution, conceptual overlap, and evolving evidence.

Round 2 and stopping rule

Following Round 1, the steering group reviewed statement agreement, clarity, clinical relevance, and conceptual overlap. Nineteen statements meeting the predefined consensus threshold were retained without rerating, while 24 revised or consolidated statements were reassessed in Round 2 (July 2-16, 2026) using identical rating criteria. Retrospective linkage through original survey identifiers confirmed that all 33 respondents participated in both rounds (100% retention), with identifiers anonymized before analysis. All 24 reassessed statements achieved consensus, fulfilling the stopping criterion. The definitive 43-statement set combined Round 1 ratings for 19 retained statements and Round 2 ratings for 24 revised statements.

Disagreement and response dispersion analysis

To assess the robustness of threshold-based consensus, a post hoc descriptive analysis of response dispersion was performed for all 43 definitive statements. In addition to overall agreement, the proportions of strong agreement and active disagreement (disagree or strongly disagree) were calculated using eligible responses, excluding outside-scope ratings. The number of dissenting respondents was also recorded. Exploratory ordinal standard deviations were calculated from the four-point response scale to characterize rating dispersion. These analyses supplemented, but did not alter, the predefined consensus classifications.

Consensus definition and statistical analysis

Agreement was defined as the combined proportion selecting "agree" or "strongly agree." Responses marked "outside my scope of practice" were excluded from the denominator used to calculate agreement, but the category distribution itself was calculated against all submitted responses. Very strong consensus was defined as ⩾90% agreement, strong consensus as ⩾75% to <90%, and no consensus as <75%. Descriptive statistics were used without inferential comparison between rounds because only revised statements were rerated. For the definitive 43-statement analysis, the most recent applicable rating was used: Round 1 for 19 retained statements and Round 2 for 24 revised or consolidated statements. Data were summarized at statement and domain levels. Survey responses were analyzed in de-identified form. Email addresses were collected solely to distribute the survey and identify duplicate submissions and were removed before analysis. Respondent-level linkage was verified using anonymized participant identifiers to establish panel continuity and retention across rounds. Because only revised or consolidated statements underwent reassessment, the analyses focused on statement-level agreement rather than paired comparisons of individual rating changes. The final consensus classification was based on the most recent applicable rating for each statement.

Results

Delphi process and participation

The steering group developed 48 draft statements, of which 46 were evaluated by 33 experts in Round 1. Following review, 19 statements were retained unchanged, while 24 revised or consolidated statements proceeded to Round 2, yielding a definitive set of 43 statements. Retrospective respondent-level linkage confirmed participation of the same 33 experts in both rounds (100% retention). All 24 reassessed statements achieved the predefined consensus threshold, concluding the Delphi process after two rounds. The final analysis incorporated Round 1 ratings for 19 retained statements and Round 2 ratings for 24 revised or consolidated statements.

Respondent characteristics

A total of 33 healthcare professionals participated in the Delphi process. The panel was predominantly composed of cardiologists (n=26, 78.8%), followed by internal medicine physicians (n=4, 12.1%), nephrologists (n=2, 6.1%), and a clinical pharmacologist (n=1, 3.0%). Most respondents had substantial

clinical experience, with 30 (90.9%) participants reporting more than 20 years of practice; 2 (6.1%) had 10-15 years of experience, and 1 (3.0%) had 15-20 years of experience.

Twenty-one (63.6%) respondents worked in tertiary heart failure services, four (12.1%) in outpatient heart failure clinics, three (9.1%) in acute medical units, and three (9.1%) in academic or research settings. One respondent each worked in a district or general hospital cardiology service and in primary care. Twenty-two (66.7%) participants managed patients with acute or chronic heart failure daily, while nine (27.3%) did so several times per week. One participant managed such patients weekly, and one was not currently involved in direct patient care.

Thirty (90.9%) respondents regularly prescribed or recommended loop diuretic therapy, while two (6.1%) did so occasionally. Only one (3.0%) respondent reported no involvement in loop diuretic prescribing or recommendation. Overall, 93.9% managed patients with heart failure either daily or several times per week, and 97.0% were directly involved in loop diuretic decision-making (Table *1*). The Delphi panel comprised 33 healthcare professionals who participated in both survey rounds, with complete respondent-level linkage confirming continuity of panel composition throughout the consensus process.

Characteristic	Category	n (%)
Primary professional role	Cardiologist	26 (78.8)
	Internal medicine physician	4 (12.1)
	Nephrologist	2 (6.1)
	Clinical pharmacologist	1 (3.0)
Years in clinical practice	10-15 years	2 (6.1)
	15-20 years	1 (3.0)
	20 years	30 (90.9)
Main clinical setting	Tertiary heart failure service	21 (63.6)
	Outpatient heart failure clinic	4 (12.1)
	Acute medical unit	3 (9.1)
	Academic/research setting	3 (9.1)
	District/general hospital cardiology service	1 (3.0)
	Primary care	1 (3.0)
Frequency of heart failure management	Daily	22 (66.7)
	Several times per week	9 (27.3)
	Weekly	1 (3.0)
	Not currently involved in direct care	1 (3.0)
Loop diuretic prescribing or recommendation	Yes, regularly	30 (90.9)
	Yes, occasionally	2 (6.1)
	No	1 (3.0)

TABLE 1: Professional and clinical characteristics of the Delphi panel

Round 1 consensus

Consensus was achieved for 45 of 46 Round 1 statements (97.8%). Thirty-five statements achieved very strong consensus, ten achieved strong consensus, and one did not achieve consensus (Appendix 1). The only non-consensus statement proposed acetazolamide for selected hospitalized patients with acute heart failure and volume overload when early intensified decongestion was desired (71.9% agreement). The AICAR framework as a clinically useful structure for personalized decongestion achieved 90.9% agreement (Table *2*).

Domain	Statements, n	Very strong, n	Strong, n	No consensus, n	Mean agreement, %
Assessment of congestion	5	5	0	0	91.5
Initiation of loop diuretic therapy	6	5	1	0	91.2
Customization according to patient phenotype	6	4	2	0	90.4
Adjustment using early objective response	10	9	1	0	92.1
Reassessment, renal safety, and discharge planning	9	8	1	0	90.9
Combination and adjunctive decongestive therapies	4	3	1	0	91.1
Implementation and overall AICAR framework	3	3	0	0	90.9

TABLE 2: Definitive 43-statement consensus by domain

AICAR, Assessment, Initiation, Customization, Adjustment, and Reassessment.

Construction of the definitive 43-statement consensus set

All 24 revised statements reassessed in Round 2 achieved consensus: 21 achieved very strong consensus and 3 achieved strong consensuses. When these results were combined with the 19 Round 1 statements, all 43 definitive statements achieved consensus; 37 (86.0%) achieved very strong consensus and 6 (14.0%) achieved strong consensus. The six strong consensus statements were final Statement 11 on maintaining euvolemia with the lowest effective oral loop diuretic dose (87.5%), Statement 12 on individualized rather than standardized loop diuretic selection (84.8%), Statement 16 on clinical preference for torsemide to prevent or manage tolerance (84.8%), Statement 25 on selective fluid restriction in severe heart failure or hyponatremia (87.9%), Statement 35 on long-term loop diuretic therapy to maintain euvolemia and reduce worsening heart failure events (87.9%), and Statement 38 on acetazolamide for persistent volume overload despite intravenous loop diuretic therapy (83.3%). No definitive statement remained below the 75% threshold. For revised Statements 38-40, outside-scope responses yielded eligible agreement denominators of 30, 31 and 32, respectively (Figure *2*, Table *3*).

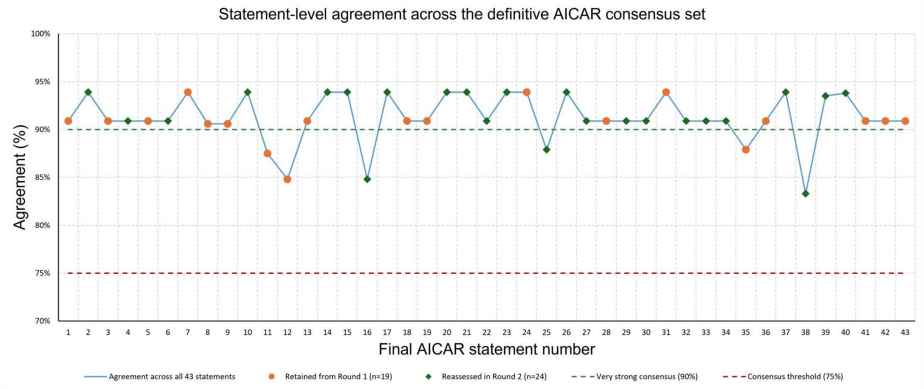


FIGURE 2: Agreement across the definitive 43-statement AICAR consensus set

AICAR, Assessment, Initiation, Customization, Adjustment, and Reassessment.

No.	Definitive consensus statement	Rating source	Strongly agree	Agree	Disagree	Strongly disagree	Outside scope	Agreement*	Consensus
1	Congestion should be assessed systematically in every patient with acute or chronic heart failure.	Round 1 retained	78.80%	12.10%	0.00%	9.10%	0.00%	90.90%	Very strong

2	Assessment of congestion should not rely on body weight alone. Use of biomarkers (NTProBNP/BNP) left atrial pressure (E/E' ratio) and IVC assessment by echocardiography and lung ultrasound can bolster clinical assessment.	Round 2 reassessed	81.80%	12.10%	0.00%	6.10%	0.00%	93.90%	Very strong
3	Clinical assessment of congestion should include symptoms, physical examination, renal function, electrolytes, medication exposure, and prior diuretic response.	Round 1 retained	63.60%	27.30%	0.00%	9.10%	0.00%	90.90%	Very strong
4	Assessment of residual congestion should be actively sought before hospital discharge; symptomatic improvement alone should not be considered euvolemia. Pre-discharge biomarker assay and echocardiography as and when indicated can be a good guide.	Round 2 reassessed	45.50%	45.50%	6.10%	3.00%	0.00%	90.90%	Very strong
5	Clinical features of right-sided or systemic venous congestion, including ascites, hepatic congestion, gut edema, and possible renal venous congestion, should be considered when evaluating diuretic response.	Round 1 retained	72.70%	18.20%	0.00%	9.10%	0.00%	90.90%	Very strong
6	Hospitalized patients with acute heart failure and clinically significant congestion should receive intravenous loop diuretics promptly unless contraindicated. Intravenous bolus fares at par with intravenous infusion in terms of symptomatic improvement and change in renal function.	Round 2 reassessed	69.70%	21.20%	3.00%	6.10%	0.00%	90.90%	Very strong
7	Initial loop diuretic dosing should account for prior diuretic exposure, kidney function, congestion severity, blood pressure, perfusion status, and likely oral absorption.	Round 1 retained	75.80%	18.20%	0.00%	6.10%	0.00%	93.90%	Very strong
8	In patients already taking chronic loop diuretics, the initial intravenous loop diuretic dose should generally be at least equivalent to the patient's outpatient total daily loop diuretic dose, after accounting for oral-to-intravenous conversion.	Round 1 retained	45.50%	42.40%	6.10%	3.00%	3.00%	90.60%	Very strong
9	In patients with severe congestion or chronic loop diuretic exposure, an initial intravenous loop diuretic dose higher than the outpatient oral equivalent may be appropriate when close monitoring is available.	Round 1 retained	51.50%	36.40%	3.00%	6.10%	3.00%	90.60%	Very strong
10	Intravenous loop diuretics should be favoured when oral absorption is unreliable, particularly with gut edema, ascites, severe right-sided congestion, or systemic venous congestion.	Round 2 reassessed	84.80%	9.10%	0.00%	6.10%	0.00%	93.90%	Very strong
11	In stable chronic heart failure, oral loop diuretics should be used to maintain euvolemia at the lowest effective dose.	Round 1 retained	69.70%	15.20%	9.10%	3.00%	3.00%	87.50%	Strong
12	The choice of loop diuretic and dosing strategy should be individualized rather than standardized for all patients.	Round 1 retained	48.50%	36.40%	9.10%	6.10%	0.00%	84.80%	Strong
13	Patients with chronic loop diuretic exposure often require higher initial doses during acute decompensation.	Round 1 retained	66.70%	24.20%	0.00%	9.10%	0.00%	90.90%	Very strong
14	Patients with chronic kidney disease may require higher loop diuretic doses because of reduced tubular delivery and require closer biochemical monitoring during intensified therapy.	Round 2 reassessed	78.80%	15.20%	0.00%	6.10%	0.00%	93.90%	Very strong
15	Although RCTs have not shown any significant difference in all-cause mortality and rates of hospitalization between torsemide and furosemide, torsemide has longer duration of action, requires less frequent dosing and its two to three times more potent than furosemide. Torsemide also has better and more predictable bioavailability and consistent absorption with oral therapy.	Round 2 reassessed	36.40%	57.60%	3.00%	3.00%	0.00%	93.90%	Very strong

16	While there is no exact statistical incidence rate for true pharmacological diuretic resistance (or tolerance) between torsemide and furosemide, torsemide is clinically preferred over furosemide to prevent and manage tolerance since it prevents the sodium “rebound” effect common with twice daily furosemide.	Round 2 reassessed	33.30%	51.50%	12.10%	3.00%	0.00%	84.80%	Strong
17	Strict sodium restriction, avoiding NSAIDs, multiple smaller doses of loop diuretics and sequential nephron blockades by combining thiazide like diuretic (Metolazone, hydrochlorothiazide, or chlorthalidone) with loop-diuretics can be effective steps in preventing or overcoming diuretic resistance.	Round 2 reassessed	66.70%	27.30%	0.00%	6.10%	0.00%	93.90%	Very strong
18	Older and frail patients require careful titration and frequent reassessment, but diuretic therapy should not be withheld solely because of age.	Round 1 retained	54.50%	36.40%	3.00%	6.10%	0.00%	90.90%	Very strong
19	In hospitalized patients with acute heart failure, diuretic response should be assessed early after intravenous loop diuretic therapy, ideally within 2–6 hours.	Round 1 retained	60.60%	30.30%	0.00%	9.10%	0.00%	90.90%	Very strong
20	Urine output should be monitored hourly after intravenous loop diuretic dosing. In hospitalized patients receiving intravenous loop diuretics for acute heart failure, spot urinary sodium should also be considered when available to guide early response assessment.	Round 2 reassessed	60.60%	33.30%	3.00%	3.00%	0.00%	93.90%	Very strong
21	A low spot urinary sodium after intravenous loop diuretic dosing, interpreted alongside urine output and clinical status, should prompt reassessment of the decongestive strategy. A spot urinary sodium level >50 – 70mmol/L should be targeted.	Round 2 reassessed	51.50%	42.40%	6.10%	0.00%	0.00%	93.90%	Very strong
22	Inadequate early natriuretic or diuretic response should prompt timely intensification rather than passive observation. If urinary spot sodium is <70mmol/L 2 hours after administration of loop diuretic or a urinary volume <150 ml/h, diuretic therapy should be doubled.	Round 2 reassessed	45.50%	45.50%	6.10%	3.00%	0.00%	90.90%	Very strong
23	When response is inadequate, clinicians should confirm that the intended dose and route were administered. Loop diuretics should be titrated to maximum daily intravenous dosage (Furosemide 200-600 mg/day, Torsemide 40-200 mg/day). Upfront addition of IV acetazolamide (500 mg IV once daily) or an oral thiazide (Metolazone 2.5-20 mg/day, Hydrochlorothiazide 25-100 mg/day titrated as per eGFR or Chlorthalidone 12.5 to 25 mg daily or intermittently) to be considered.	Round 2 reassessed	51.50%	42.40%	3.00%	3.00%	0.00%	93.90%	Very strong
24	When response even to multiple diuretics is inadequate, clinicians should assess dietary sodium intake, NSAID exposure, blood pressure, renal perfusion, adherence, and ongoing congestion.	Round 1 retained	66.70%	27.30%	3.00%	3.00%	0.00%	93.90%	Very strong
25	Salt and fluid restriction is a non-pharmacological strategy for managing heart failure specifically to prevent volume overload, pulmonary congestion and hospitalizations. Despite recent evidence suggesting personalized fluid restriction to avoid worsening thirst, current guidelines recommend restricting daily fluid intake to 1.5 – 2.0L/day for patients with severe heart failure or hyponatremia.	Round 2 reassessed	45.50%	42.40%	6.10%	6.10%	0.00%	87.90%	Strong
26	Renal function changes during decongestion should be interpreted clinically, balancing persistent congestion against a modest transient creatinine rise when decongestion is effective and perfusion is preserved.	Round 2 reassessed	69.70%	24.20%	0.00%	6.10%	0.00%	93.90%	Very strong
	True worsening (Acute Kidney Injury or Acute Tubular								

27	necrosis) of renal function must be differentiated from pseudo-worsening during decongestive therapy. In pseudo-worsening, patients have good diuretic response with progressive decongestion and normal renal injury biomarkers, and it is generally safe to continue or optimize HF therapy including diuretics. In true worsening, patients have worsening congestion and elevated renal injury biomarkers. (NGAL, KIM-1) and it usually requires reducing or stopping diuretics and RAAS blockers.	Round 2 reassessed	69.70%	21.20%	6.10%	3.00%	0.00%	90.90%	Very strong
28	Combination of intensified diuretic therapy requires close monitoring of renal function, electrolytes, blood pressure, and volume status.	Round 1 retained	72.70%	18.20%	0.00%	9.10%	0.00%	90.90%	Very strong
29	Decongestion should not be considered complete until an effective oral diuretic regimen, stable safety parameters, and a follow-up plan for laboratory monitoring, weight monitoring, symptom recognition, and clinical review are in place.	Round 2 reassessed	69.70%	21.20%	6.10%	3.00%	0.00%	90.90%	Very strong
30	Residual Congestion (RC) in ADHF is associated with adverse outcomes. Residual intravascular congestion (JVP >6 cmH20) is most common at discharge and is associated with higher mortality despite a good diuretic response during admission, while residual tissue congestion (Peripheral oedema Gr ≥2 or Pulmonary rales over ≥1/3 of lung fields, or both) is associated with poor diuretic response and the highest mortality.	Round 2 reassessed	60.60%	30.30%	6.10%	3.00%	0.00%	90.90%	Very strong
31	Stable decongestion should be used as an opportunity to initiate or optimize guideline-directed medical therapy whenever clinically appropriate.	Round 1 retained	60.60%	33.30%	3.00%	3.00%	0.00%	93.90%	Very strong
32	Transition from IV to oral loop diuretic in ADHF should occur once clinical euvolemia is reached. Dosing conversion ratios (IV-oral) are as follows: Furosemide 1:2, Bumetanide and Torsemide 1:1. Then total calculated oral dose is split into either once or twice daily dose depending on renal function and congestion severity. Rebound congestion/inadequate diuresis should be monitored. Torsemide is often preferred over furosemide in post-ADHF phase because of higher predictable bioavailability and longer duration of action.	Round 2 reassessed	54.50%	36.40%	6.10%	3.00%	0.00%	90.90%	Very strong
33	De-escalating loop diuretics in ADHF before or during GDMT initiation requires a delicate approach. Clinical euvolemia (clear lung fields, normal JVP, absence of oedema etc.) should be ensured prior to de-escalation. Transition before de-escalation from IV to oral loop diuretic should also be preferably achieved before GDMT (SGLT2i, RAAS blockers, and Beta-blocker) initiation. Potential re-congestion should be monitored by weight-monitoring and symptom.	Round 2 reassessed	51.50%	39.40%	6.10%	3.00%	0.00%	90.90%	Very strong
34	Concomitant use of Mineralocorticoid Receptor Antagonist (MRA) such as spironolactone or eplerenone with IV/oral loop diuretics in ADHF provides essential neurohormonal blockade and helps offset the potassium-wasting effects of loop diuretics without any significant increase in acute urine output or natriuresis even with higher-dose spironolactone.	Round 2 reassessed	72.70%	18.20%	3.00%	6.10%	0.00%	90.90%	Very strong
35	Long-term loop diuretic therapy should aim to maintain stable euvolemia and prevent recurrent congestion, which may reduce the risk of worsening heart failure events and rehospitalization.	Round 1 retained	60.60%	27.30%	12.10%	0.00%	0.00%	87.90%	Strong
36	Long-term loop diuretic when optimized scientifically may prevent re-congestion without causing progressive renal dysfunction.	Round 1 retained	51.50%	39.40%	6.10%	3.00%	0.00%	90.90%	Very strong

37	Thiazide or thiazide-like therapy (Metolazone, hydrochlorothiazide, chlorthalidone) should be considered for loop diuretic resistance when close safety monitoring is available. Thiazide-type add-on therapy should generally be used as short-course intensification rather than indefinite escalation.	Round 2 reassessed	51.50%	42.40%	0.00%	6.10%	0.00%	93.90%	Very strong
38	Acetazolamide should be considered (in absence of contraindications) in selected hospitalized patients with acute heart failure when early intensified decongestion is desired in cases of persistent significant volume overload despite IV loop diuretic therapy.	Round 2 reassessed	30.30%	45.50%	12.10%	3.00%	9.10%	83.30%	Strong
39	SGLT2 inhibitors should be initiated or continued in hospitalized heart failure patients once clinically stable and when not contraindicated. SGLT2 inhibitors should not be considered substitutes for loop diuretics in severe congestion or acute pulmonary edema.	Round 2 reassessed	66.70%	21.20%	0.00%	6.10%	6.10%	93.50%	Very strong
40	Ultrafiltration should not be used routinely as first-line decongestive therapy in acute heart failure. Ultrafiltration should be reserved for selected patients with refractory congestion after optimized pharmacologic therapy.	Round 2 reassessed	66.70%	24.20%	3.00%	3.00%	3.00%	93.80%	Very strong
41	Health systems and hospitals should consider standardized decongestion pathways for patients hospitalized with acute heart failure, adapted to local resources.	Round 1 retained	54.50%	36.40%	3.00%	6.10%	0.00%	90.90%	Very strong
42	Early response checkpoints after intravenous loop diuretic therapy should be incorporated into inpatient workflows where feasible.	Round 1 retained	54.50%	36.40%	3.00%	6.10%	0.00%	90.90%	Very strong
43	The AICAR framework — Assessment, Initiation, Customization, Adjustment, and Reassessment — is a clinically useful structure for organizing personalized decongestion in heart failure.	Round 1 retained	60.60%	30.30%	3.00%	6.10%	0.00%	90.90%	Very strong

TABLE 3: Definitive 43-statement response distributions and consensus classification

*The definitive set comprises 19 statements retained from Round 1 and 24 revised or consolidated statements reassessed in Round 2. Agreement was defined as the combined proportion of “agree” and “strongly agree” responses, excluding “outside scope” responses from the denominator. Response category percentages were calculated using all 33 responses in the respective round. Original statement wording is reproduced. For composite statements, agreement reflects the statement as a whole and does not establish independent endorsement or clinical validation of individual components, including drug doses, conversion ratios, or escalation thresholds, which require consideration of approved prescribing information, renal function, local formularies, and institutional protocols before implementation.

Disagreement and response dispersion

Post hoc analysis of the definitive 43-statement set identified minority disagreement despite universal attainment of the predefined consensus threshold. Six statements had at least four dissenting respondents, including those addressing acetazolamide use (5/30), individualized loop diuretic selection (5/33), torsemide preference (5/33), lowest effective oral dosing (4/32), fluid restriction (4/33), and long-term loop diuretic therapy (4/33). Six additional statements were notable for at least three strongly disagree responses. Exploratory ordinal standard deviations ranged from 0.61 to 0.89. These findings demonstrate variation in the intensity of expert opinion despite consistently high overall agreement (Appendix 2).

Assessment

All five definitive assessment statements achieved very strong consensus. Statement 2, which integrated natriuretic peptides, echocardiographic indices, inferior vena cava assessment, and lung ultrasound with clinical evaluation rather than reliance on body weight alone, achieved 93.9% agreement. Statement 4, requiring active assessment of residual congestion before discharge and explicitly distinguishing symptomatic improvement from euvolemia, achieved 90.9% agreement (Figure *3*).

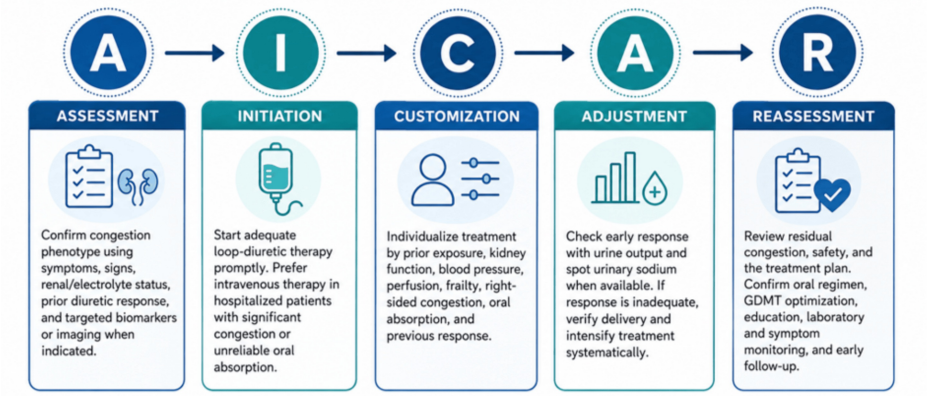


FIGURE 3: The AICAR clinical reasoning pathway

AICAR, Assessment, Initiation, Customization, Adjustment, and Reassessment; GDMT, guideline-directed management and therapy.

Initiation and customization

Within initiation and customization, prompt intravenous loop diuretic therapy for hospitalized patients with clinically significant congestion, with recognition that bolus and infusion strategies have comparable broad clinical effects, achieved 90.9% agreement (Statement 6). Intravenous administration when oral absorption is unreliable, achieved 93.9% (Statement 10), as did the combined statement that chronic kidney disease may require higher doses and closer biochemical surveillance (Statement 14). The panel agreed that randomized trials have not shown outcome superiority of torsemide over furosemide (93.9%; Statement 15); however, the more prescriptive preference for torsemide to prevent tolerance and sodium rebound reached strong rather than very strong consensus (84.8%; Statement 16) (Figure *3*).

Adjustment and diuretic resistance

The panel endorsed avoidance of non-steroidal anti-inflammatory drugs, sodium management, divided loop diuretic dosing, and sequential nephron blockade as strategies for diuretic resistance (93.9%; Statement 17). Hourly urine output monitoring with spot urinary sodium when available achieved 93.9% (Statement 20). A urinary sodium target of >50-70 mmol/L and protocolized dose escalation when urinary sodium remained <70 mmol/L at approximately 2 hours or urine output was <150 mL/hour achieved 93.9% and 90.9%, respectively (Statements 21 and 22). The detailed statement covering maximum loop diuretic doses and adjunctive dosing achieved 93.9% (Statement 23), although its compound structure requires local formulary verification before implementation. Selective fluid restriction achieved 87.9% (Statement 25), indicating greater uncertainty than most other response-guided statements (Figure *3*).

Reassessment, renal safety, and discharge

Contextual interpretation of renal function changes achieved 93.9% agreement (Statement 26). The panel supported distinguishing functional or “pseudo-worsening” renal changes during successful decongestion from true kidney injury (90.9%; Statement 27), while also endorsing a complete discharge package comprising an effective oral regimen, stable safety parameters, laboratory monitoring, weight and symptom surveillance, and a clear follow-up plan (90.9%; Statement 29). Recognition of residual intravascular and tissue congestion (Statement 30), careful intravenous-to-oral transition (Statement 32), loop diuretic de-escalation after euvolemia (Statement 33), and the disease-modifying role of mineralocorticoid receptor antagonists (Statement 34) each achieved very strong consensus (Figure *3*).

Adjunctive therapies

Short-course thiazide or thiazide-like therapy for loop diuretic resistance achieved 93.5% agreement (Statement 37). Acetazolamide achieved 83.3%, the lowest agreement in the definitive set (Statement 38). Initiation or continuation of an SGLT2 inhibitor after clinical stabilization, while not using it as a substitute for loop diuretics in severe congestion or pulmonary edema, achieved 93.5% (Statement 39). Reserving ultrafiltration for refractory congestion after optimized pharmacological therapy achieved 93.8% (Statement 40).

Key consensus recommendations

Assessment Should Be Multimodal

Do not infer congestion or euvolemia from body weight or symptomatic improvement alone. Integrate

symptoms, physical signs, renal and electrolyte status, medication exposure, prior diuretic response, and -- when clinically indicated -- natriuretic peptides, echocardiography, inferior vena cava assessment, or lung ultrasound.

Initiate Adequate Loop Diuretic Therapy Promptly

Use intravenous therapy in hospitalized patients with significant congestion when not contraindicated, particularly when gut edema, ascites, or systemic venous congestion makes oral absorption unreliable.

Customize Treatment to Phenotype

Account for prior loop diuretic exposure, kidney function, blood pressure, perfusion, frailty, right-sided congestion, oral absorption, and previous response. Agent selection should be individualized; current randomized evidence does not establish mortality superiority of torsemide over furosemide.

Measure Early Response

Record urine output after intravenous dosing and consider spot urinary sodium where available. Failure to achieve an adequate early response should trigger confirmation of drug delivery and prompt protocolized intensification rather than passive observation.

Treat Diuretic Resistance Systematically

Review sodium exposure, non-steroidal anti-inflammatory drugs, adherence, dose, route, perfusion, and ongoing congestion before sequential nephron blockade. Add-on thiazide-type therapy or acetazolamide should be selected with close renal, electrolyte, and hemodynamic monitoring.

Interpret Creatinine in Clinical Context

A modest transient elevation of creatinine during effective decongestion with preserved perfusion should not automatically prompt loop diuretic withdrawal. Persistent congestion, hypotension, oliguria, progressive biochemical deterioration, or other evidence of true kidney injury requires a different response.

Do Not Discharge Before Assessing Residual Congestion by Default

Before discharge, seek residual intravascular and tissue congestion, verify an effective oral regimen, document stable safety parameters, and provide a laboratory, weight, symptom, and clinical review plan.

Integrate Disease-Modifying Therapy

Once clinically stable, initiate or optimize guideline-directed therapy, including SGLT2 inhibitors and mineralocorticoid receptor antagonists where indicated. These agents complement but do not replace loop diuretics with severe acute congestion.

Reserve Ultrafiltration

Use ultrafiltration only in selected patients with refractory congestion after optimization of pharmacological therapy and multidisciplinary review.

Implement and Validate

Hospitals should adapt AICAR to local resources with defined response checkpoints, escalation safeguards, discharge criteria, and audit measures. Prospective validation is necessary before claiming outcome benefit.

Discussion

Principal findings

This modified Delphi study produced a definitive 43-statement consensus set spanning the complete pathway of personalized decongestion. Round 1 evaluated 46 statements; steering-group review retained 19 and revised or consolidated 24, and only those 24 were reassessed in Round 2. All 43 definitive statements reached consensus, with 37 achieving very strong and six achieving strong consensus. The principal contribution is not a new drug or isolated threshold but an explicit sequence of clinical reasoning: identify the congestion phenotype, initiate adequate therapy, customize to the patient, adjust early according to objective response, and reassess until decongestion is safe, durable, and linked to follow-up.

The high proportion of very strong consensus indicates that the AICAR sequence is broadly acceptable to

participating clinicians. The six statements with strong rather than very strong consensus nevertheless identify areas where practice remains less uniform: maintenance at the lowest effective oral dose, individualized agent and dosing selection, torsemide preference for tolerance, fluid restriction, long-term loop diuretic goals, and acetazolamide selection. The lowest agreement was observed for acetazolamide (83.3%), individualized loop diuretic selection (84.8%), and torsemide preference for preventing or managing tolerance (84.8%). These areas should remain visible rather than being obscured by the overall high consensus rate.

Although contemporary heart failure guidelines and clinical trials provide evidence-based recommendations for decongestion, translating these into sequential bedside decisions remains challenging, particularly in resource-variable settings. The AICAR framework addresses this implementation gap by integrating established evidence and expert opinion into five interconnected stages: Assessment, Initiation, Customization, Adjustment, and Reassessment. Its contribution is primarily organizational rather than therapeutic, providing a structured approach to congestion assessment, individualized treatment, response-guided adjustment, and discharge reassessment without introducing novel interventions or independently validated thresholds. Particularly relevant to the Indian healthcare context, AICAR accommodates variations in drug availability, monitoring resources, and specialist access. However, its clinical effectiveness, feasibility, and incremental benefit over existing decongestion pathways require prospective comparative validation.

Assessment: moving beyond weight and symptoms

The strongest assessment message is that no single marker defines congestion. In a pooled multimodality analysis, clinical, echocardiographic, and lung ultrasound signs each contributed prognostic information, supporting the AICAR recommendation to combine modalities according to availability and clinical need [4]. The EACVI consensus statement similarly positions lung ultrasound as an extension of clinical assessment rather than a standalone diagnostic or treatment target [5].

Randomized proof-of-concept data suggest that lung ultrasound-guided follow-up can increase diuretic adjustment and reduce urgent visits for worsening heart failure, although larger trials are required to establish effects on hospitalization and mortality [19]. The practical implication is proportionality: routine bedside assessment remains essential, while imaging and biomarkers are most useful when examination is equivocal, when subclinical pulmonary congestion is suspected, or when the trajectory does not match apparent symptomatic improvement.

The very strong agreement on pre-discharge assessment is reinforced by contemporary evidence on residual congestion. Distinct residual intravascular and tissue phenotypes are common at discharge and carry adverse prognostic implications, even when in-hospital diuretic response appears satisfactory [6]. AICAR therefore treats discharge as a reassessment point rather than an administrative endpoint.

Initiation: adequate dose, appropriate route, and early intent

Contemporary guidelines and position statements support prompt intravenous loop diuretic therapy in acute heart failure with congestion, with initial dosing guided by prior diuretic exposure and congestion severity [1-3]. The AICAR panel additionally emphasized impaired oral absorption in patients with gut edema, ascites, or systemic venous congestion, where intravenous administration may be preferable. Randomized evidence has not demonstrated consistent symptomatic or renal superiority of continuous infusion over intermittent bolus administration. Although bolus dosing offers simplicity and flexibility, continuous infusion may be considered in selected patients with inadequate response. The Delphi exercise did not independently assess preferences or phenotype-specific indications for either strategy. Accordingly, AICAR emphasizes individualized dosing and administration, guided by clinical response, hemodynamic status, and monitoring capacity. Adequate initial dosing and early reassessment are essential to avoid both delayed decongestion from undertreatment and electrolyte or hemodynamic complications from excessive dosing.

Customization and the torsemide-furosemide question

The consensus highlights a divergence between contemporary evidence and clinical prescribing preferences. Although TRANSFORM-HF demonstrated no mortality advantage of torsemide over furosemide, subsequent mechanistic investigations have further questioned its presumed pharmacological superiority [7,8]. Nevertheless, preferences based on perceived pharmacokinetic advantages, treatment response, and clinical experience persist. This evidence-practice gap provided an important rationale for the modified Delphi exercise, which sought to quantify expert agreement in areas of therapeutic uncertainty. Accordingly, the 84.8% agreement for Statement 16 reflects a preference among participating experts rather than established superiority of torsemide in preventing diuretic tolerance or rebound sodium retention. The statement should therefore not be interpreted as endorsing routine torsemide preference; loop diuretic selection should remain individualized according to clinical response, tolerability, patient characteristics, and available evidence.

Heart failure phenotype is another important consideration in treatment customization. Patients with heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF) may differ in ventricular compliance, filling pressures, and hemodynamic tolerance of volume reduction. Accordingly, decongestive therapy should account for congestion phenotype, blood pressure, renal function, systemic perfusion, and tolerance of fluid removal, irrespective of ejection fraction. However, the present consensus did not independently evaluate phenotype-specific diuretic strategies for HFrEF and HFpEF, highlighting an area requiring further investigation.

Adjustment: urinary sodium and response-guided escalation

The Adjustment component emphasizes early evaluation of diuretic response following intravenous administration. Observational evidence associates low early urinary sodium with inadequate diuresis and adverse outcomes [20]. PUSH-AHF demonstrated that urinary sodium-guided escalation increased 24-hour natriuresis without significantly reducing death or heart failure rehospitalization [13], while ENACT-HF demonstrated improved natriuresis and diuretic response through a protocolized approach, supporting its implementation feasibility [14].

Within AICAR, spot urinary sodium complements rather than replaces urine output and clinical assessment. Urinary sodium thresholds of approximately 50-70 mmol/L and urine output below 150 mL/hour may serve as early indicators of inadequate response, prompting reassessment and consideration of treatment intensification. However, these thresholds remain operational decision aids rather than universally validated predictors of clinical benefit and should be interpreted cautiously in patients with hemodynamic instability, advanced kidney dysfunction, recent diuretic exposure, urinary obstruction, or unreliable fluid measurements. Escalation decisions should integrate congestion severity, renal function, perfusion status, and observed treatment response rather than rely on fixed thresholds alone.

Although the detailed dose escalation statement achieved very strong consensus, its composite nature limits interpretation of individual dosing recommendations. Maximum loop diuretic doses, drug conversion ratios, and adjunctive treatment regimens require consideration of renal function, hemodynamic status, patient characteristics, drug availability, and institutional prescribing protocols. Accordingly, the consensus supports timely, monitored, response-guided escalation rather than universally applicable dosing algorithms.

Sequential nephron blockade and acetazolamide

The consensus on short-course thiazide-type add-on therapy is consistent with CLOROTIC, in which hydrochlorothiazide added to intravenous furosemide improved weight loss and diuretic response while increasing the need for surveillance of renal function and electrolytes [10]. Sequential nephron blockade is most appropriate after confirming dose delivery, adherence, and persistent congestion, and it should be accompanied by monitoring of sodium, potassium, magnesium, bicarbonate, blood pressure, and renal function.

Acetazolamide remained the most debated adjunct across both rounds. ADVOR demonstrated that acetazolamide 500 mg intravenously once daily, added to standardized loop diuretic therapy, increased successful decongestion within three days without a significant excess of prespecified safety outcomes [9]. Benefit was observed across the spectrum of ejection fraction. The lower Delphi agreement may reflect uncertainty regarding patient selection, bicarbonate status, kidney function, contraindications, availability, and the distinction between early combination therapy and rescue therapy after established resistance.

AICAR therefore supports acetazolamide in selected hospitalized patients rather than as mandatory universal therapy. The final decision should incorporate acid-base status, renal function, sulfonamide-related precautions, electrolyte risk, and the institutional capacity for monitoring.

SGLT2 inhibitors and mineralocorticoid receptor antagonists

EMPULSE demonstrated clinical benefits of empagliflozin initiated in clinically stabilized patients hospitalized with acute heart failure, irrespective of ejection fraction or diabetes status [11]. A prespecified analysis further demonstrated early and sustained improvements in congestion-related measures [21]. Conversely, DAPA-RESIST found that dapagliflozin was not superior to metolazone for congestion relief in loop diuretic-resistant heart failure, although it was associated with fewer biochemical disturbances [12]. These findings support the complementary role of SGLT2 inhibitors in decongestion and their initiation or continuation following clinical stabilization, without replacing or delaying adequate loop diuretic therapy or sequential nephron blockade when indicated. Notably, the Delphi consensus addressed SGLT2 inhibitors within broader hospitalized heart failure management rather than as an independently evaluated rescue strategy for diuretic resistance.

Mineralocorticoid receptor antagonists (MRAs) provide disease-modifying neurohormonal blockade and may counteract loop diuretic-associated potassium loss, although their acute natriuretic effects are limited. Their initiation or continuation should be guided by renal function, serum potassium, and clinical stability.

Accordingly, AICAR positions both SGLT2 inhibitors and MRAs as components of comprehensive heart failure management rather than substitutes for rapid decongestive therapy.

Renal function: congestion, functional change, and true injury

The consensus emphasizes that a modest rise in serum creatinine during effective decongestion should not automatically prompt diuretic reduction or withdrawal. Such changes may reflect hemodynamic alterations or hemoconcentration rather than structural tubular injury, particularly when congestion improves and systemic perfusion remains preserved. Accordingly, renal function should be interpreted alongside congestion status, hemodynamics, urine output, electrolyte balance, and biochemical trends [22].

Although the Round 2 statement identified neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1) as potential markers of tubular injury, their routine clinical utility remains uncertain. In the AKINESIS study, urinary NGAL did not outperform serum creatinine in predicting worsening renal function or adverse in-hospital outcomes, limiting its applicability for routine assessment [23]. Therefore, biomarker testing should not be considered mandatory or independently determine adjustments to diuretic or renin-angiotensin-aldosterone system inhibitor therapy.

Conversely, progressive creatinine elevation accompanied by persistent congestion, hypotension, oliguria, significant electrolyte disturbances, nephrotoxin exposure, or evidence of impaired organ perfusion warrants prompt evaluation for acute kidney injury and reassessment of treatment. Thus, AICAR advocates an integrated clinical and biochemical approach that distinguishes potentially reversible functional changes from suspected structural kidney injury, without overlooking clinically significant renal deterioration.

Fluid and sodium advice: from universal restriction to personalization

Fluid restriction achieved strong but not very strong consensus. This result is clinically coherent because the evidence differs by phenotype. Sodium moderation and avoidance of excessive intake are reasonable in patients prone to recurrent congestion, while strict fluid restriction may remain appropriate for severe congestion or dilutional hyponatremia [24]. However, the FRESH-UP randomized trial found that liberal fluid advice was not inferior to a 1.5-L restriction for health status in stable chronic heart failure and reduced thirst distress [25]. AICAR should therefore avoid a universal fixed fluid limit. Advice should be individualized to congestion, serum sodium, kidney function, diuretic intensity, climate, thirst burden, and self-management capacity, with reassessment rather than indefinite restriction by default [26].

Continuation versus discontinuation of low-dose loop diuretics

Continuation or withdrawal of low-dose loop diuretics in stable chronic heart failure remains debated. The present panel reached strong consensus that oral loop diuretics should generally be continued at the lowest effective dose required to maintain euvolemia. Nevertheless, withdrawal may be feasible in carefully selected, stable, euvolemic patients receiving optimized guideline-directed therapy. In a 2024 retrospective cohort, only 3 of 122 patients experienced worsening heart failure events within one year of loop diuretic discontinuation, although 15 required reinitiations of low-dose diuretic therapy during scheduled follow-up [27]. These findings therefore cannot be generalized beyond similarly low-risk patients. Accordingly, continuation should remain the usual approach, while cautious dose reduction or withdrawal may be considered at the treating physician's discretion, based on congestion history, renal function, natriuretic peptides, background therapy, and the availability of close clinical, weight, and symptom monitoring.

Transition to oral therapy, de-escalation, and guideline-directed treatment

An effective oral regimen is a discharge safety test. Transition should occur after meaningful clinical decongestion, with sufficient observation to detect inadequate absorption, rebound sodium retention, or recurrent symptoms. The conversion ratio used must reflect local practice and be validated by response rather than assumed to be exact, particularly considering emerging mechanistic data on torsemide and furosemide [8].

Loop diuretic reduction may be reasonable after stable euvolemia. In ReBIC-1, withdrawal of low-dose furosemide in selected stable, euvolemic outpatients receiving optimized therapy did not worsen dyspnea or increase short-term heart failure events [16]. These findings cannot be extrapolated to recently decompensated, persistently congested or high-dose patients; AICAR therefore links de-escalation to active surveillance for weight gain and symptoms.

Statement 33 reflected a stabilization-oriented approach to intravenous-to-oral diuretic transition before guideline-directed management and therapy (GDMT) optimization, rather than a mandatory therapeutic sequence. SGLT2 inhibitors and MRAs may be initiated or continued during intravenous diuresis in clinically stable patients, subject to renal, electrolyte, and other safety considerations. Successful decongestion and readiness for oral diuretics may indicate clinical stability for ARNI and beta-blocker initiation, although oral transition is not an absolute prerequisite. Beta-blocker initiation should generally follow adequate clinical

stabilization. Contemporary evidence supports timely in-hospital GDMT optimization, with STRONG-HF reinforcing the benefits of rapid treatment intensification and close follow-up [22,24]. Thus, GDMT initiation should be individualized according to therapeutic class and clinical stability without unnecessary delay.

Ultrafiltration

The very strong consensus against routine first-line ultrafiltration is consistent with contemporary evidence. A Cochrane review found uncertainty regarding clinically important benefits and highlighted heterogeneity in patient selection, timing, and comparator strategies [15]. Ultrafiltration may be considered when severe congestion persists despite optimized pharmacological therapy but requires vascular access, specialized expertise, careful fluid removal targets, and close hemodynamic and renal monitoring.

Implementation of the AICAR pathway

AICAR is intended to standardize decisions rather than impose a single drug sequence. A practical pathway should define the minimum congestion, the initial dose logic, timing of response checkpoints, escalation options, biochemical monitoring, criteria for adjunctive therapy, discharge euvolemia assessment, and follow-up responsibility. Local pathways should explicitly identify situations requiring senior cardiology, nephrology, or critical care review.

Implementation should be audited using process and outcome measures. Candidate process measures include documentation of congestion phenotype, time to first intravenous loop dose, urine output documentation, use of urinary sodium when available, time to escalation after inadequate response, electrolyte monitoring after combination therapy, residual congestion assessment, and completion of a discharge monitoring plan. Candidate outcomes include net fluid and weight change, congestion score, length of stay, worsening renal function with and without effective decongestion, 30- and 90-day rehospitalization, mortality, quality of life, and patient understanding of self-management.

Drug availability is an important consideration when adapting international decongestion strategies to the Indian setting. Intravenous bumetanide, although used in certain international heart failure pathways, may have limited routine availability in Indian practice. This may constrain the applicability of algorithms that depend on intravenous bumetanide as an alternative loop diuretic. The AICAR framework should therefore be adapted to locally available formulations while ensuring that dose conversions and escalation strategies are supported by appropriate prescribing safeguards.

Strengths and limitations

Strengths include an evidence-informed design, predefined consensus thresholds, an explicit outside-scope response option, transparent statement-level reporting, and coverage of the inpatient-to-outpatient decongestion pathway. The AICAR framework provides a structured, clinically memorable approach to decongestion. Importantly, respondent-level linkage confirmed participation of the same 33 experts across both rounds (100% retention), ensuring panel continuity. The modified Delphi design retained 19 unchanged statements and reassessed 24 revised or consolidated statements, although individual-level convergence could not be formally established because identical statements were not systematically rerated.

Several limitations warrant consideration. First, the relatively small panel was predominantly composed of experienced cardiologists, with limited representation from other healthcare disciplines. Although most respondents practiced in tertiary care settings, recruitment through professional networks may have introduced selection bias and limited generalizability. Second, several statements combined multiple clinical propositions, thresholds, or drug doses; agreement with composite statements does not establish independent endorsement or clinical validation of individual components. Third, the focused evidence review was not systematic and lacked formal certainty-of-evidence grading, limiting the strength of prescriptive recommendations. Finally, the predominantly Indian tertiary care perspective may restrict applicability to other healthcare settings. Expert consensus alone cannot establish clinical effectiveness, and AICAR remains an evidence-informed organizational framework requiring prospective evaluation of feasibility, safety, and clinical outcomes.

Research priorities

Future studies should compare AICAR-based pathways with usual care using pragmatic cluster randomized or stepped wedge designs. Priority outcomes include time to effective natriuresis, residual congestion at discharge, renal safety, length of stay, early rehospitalization, mortality, quality of life, and implementation cost. Research should also determine which patients derive the greatest benefit from urinary sodium-guided escalation, acetazolamide, thiazide-type add-on therapy, lung ultrasound-guided management, and cautious loop diuretic de-escalation.

Further consensus refinement should separate compound statements into single action recommendations, formally grade supporting evidence and include patient partners. Priorities are dose equivalence between

oral loop diuretics, operational definitions of true versus functional worsening renal function, safe fluid advice phenotypes, and the optimal sequencing of decongestion with in-hospital initiation of guideline-directed therapies.

Conclusions

The AICAR-modified Delphi consensus establishes a structured, clinically practical framework for personalized loop diuretic therapy and decongestion in heart failure. The panel supported systematic multimodal assessment, prompt and individualized loop diuretic therapy, early objective response monitoring, timely escalation, contextual renal safety interpretation, active detection of residual congestion, structured transition to oral therapy, and selective use of adjunctive treatments. The strongest remaining uncertainties concern routine torsemide preference, fluid restriction, and acetazolamide selection. AICAR should be implemented as a locally adapted clinical reasoning pathway, not as a substitute for prescribing safeguards or guideline-directed therapy, and should undergo prospective validation before claims of improved clinical outcomes are made.

Appendices

Appendix 1

No.	Round 1 statement	Strongly agree	Agree	Disagree	Strongly disagree	Outside scope	Agreement*	Consensus
1	Congestion should be assessed systematically in every patient with acute or chronic heart failure.	78.80%	12.10%	0.00%	9.10%	0.00%	90.90%	Very strong
2	Assessment of congestion should not rely on body weight alone.	69.70%	21.20%	0.00%	9.10%	0.00%	90.90%	Very strong
3	Clinical assessment of congestion should include symptoms, physical examination, renal function, electrolytes, medication exposure, and prior diuretic response.	63.60%	27.30%	0.00%	9.10%	0.00%	90.90%	Very strong
4	Residual congestion should be actively sought before hospital discharge.	66.70%	24.20%	3.00%	6.10%	0.00%	90.90%	Very strong
5	Symptomatic improvement alone should not be considered sufficient evidence of euvolemia.	66.70%	24.20%	3.00%	6.10%	0.00%	90.90%	Very strong
6	Clinical features of right-sided or systemic venous congestion, including ascites, hepatic congestion, gut edema, and possible renal venous congestion, should be considered when evaluating diuretic response.	72.70%	18.20%	0.00%	9.10%	0.00%	90.90%	Very strong
7	Hospitalized patients with acute heart failure and clinically significant congestion should receive intravenous loop diuretics promptly unless contraindicated.	78.80%	12.10%	0.00%	9.10%	0.00%	90.90%	Very strong
8	Initial loop diuretic dosing should account for prior diuretic exposure, kidney function, congestion severity, blood pressure, perfusion status, and likely oral absorption.	75.80%	18.20%	0.00%	6.10%	0.00%	93.90%	Very strong
9	In patients already taking chronic loop diuretics, the initial intravenous loop diuretic dose should generally be at least equivalent to the patient's outpatient total daily loop diuretic dose, after accounting for oral-to-intravenous conversion.	45.50%	42.40%	6.10%	3.00%	3.00%	90.60%	Very strong
10	In patients with severe congestion or chronic loop diuretic exposure, an initial intravenous loop diuretic dose higher than the outpatient oral equivalent may be appropriate when close monitoring is available.	51.50%	36.40%	3.00%	6.10%	3.00%	90.60%	Very strong
	Oral loop diuretic therapy may be unreliable in							

11	acute decompensated heart failure with bowel edema, ascites, or severe systemic congestion.	69.70%	18.20%	0.00%	9.10%	3.00%	90.60%	Very strong
12	In stable chronic heart failure, oral loop diuretics should be used to maintain euvolemia at the lowest effective dose.	69.70%	15.20%	9.10%	3.00%	3.00%	87.50%	Strong
13	The choice of loop diuretic and dosing strategy should be individualized rather than standardized for all patients.	48.50%	36.40%	9.10%	6.10%	0.00%	84.80%	Strong
14	Patients with chronic loop diuretic exposure often require higher initial doses during acute decompensation.	66.70%	24.20%	0.00%	9.10%	0.00%	90.90%	Very strong
15	Patients with chronic kidney disease may require higher loop diuretic doses because of reduced delivery of diuretic to the tubular site of action.	72.70%	18.20%	0.00%	9.10%	0.00%	90.90%	Very strong
16	Patients with chronic kidney disease receiving intensified diuretic therapy require closer biochemical monitoring.	66.70%	27.30%	3.00%	3.00%	0.00%	93.90%	Very strong
17	Intravenous loop diuretics should be favored in patients with gut edema, ascites, severe right-sided congestion, or unreliable oral absorption.	69.70%	21.20%	0.00%	9.10%	0.00%	90.90%	Very strong
18	Torsemide or bumetanide may be useful when oral furosemide absorption or response is unreliable.	48.50%	39.40%	9.10%	3.00%	0.00%	87.90%	Strong
19	Torsemide should not be routinely preferred over furosemide solely for mortality benefit after heart failure hospitalization.	30.30%	54.50%	9.10%	6.10%	0.00%	84.80%	Strong
20	Older and frail patients require careful titration and frequent reassessment, but diuretic therapy should not be withheld solely because of age.	54.50%	36.40%	3.00%	6.10%	0.00%	90.90%	Very strong
21	In hospitalized patients with acute heart failure, diuretic response should be assessed early after intravenous loop diuretic therapy, ideally within 2-6 hours.	60.60%	30.30%	0.00%	9.10%	0.00%	90.90%	Very strong
22	Urine output should be monitored after intravenous loop diuretic dosing.	72.70%	18.20%	0.00%	9.10%	0.00%	90.90%	Very strong
23	In hospitalized patients receiving intravenous loop diuretics for acute heart failure, spot urinary sodium should be considered when available to guide early response assessment.	36.40%	48.50%	12.10%	0.00%	3.00%	87.50%	Strong
24	A low spot urinary sodium after intravenous loop diuretic dosing, interpreted alongside urine output and clinical status, should prompt reassessment of the decongestive strategy.	36.40%	51.50%	6.10%	3.00%	3.00%	90.60%	Very strong
25	Inadequate early natriuretic or diuretic response should prompt timely intensification rather than passive observation.	66.70%	24.20%	3.00%	6.10%	0.00%	90.90%	Very strong
26	When response is inadequate, clinicians should confirm that the intended dose and route were administered.	51.50%	39.40%	3.00%	6.10%	0.00%	90.90%	Very strong
27	When response is inadequate, clinicians should assess dietary sodium intake, NSAID exposure, blood pressure, renal perfusion, adherence, and ongoing congestion.	66.70%	27.30%	3.00%	3.00%	0.00%	93.90%	Very strong
	When the initial loop diuretic response is poor,							

28	dose escalation should generally involve protocolized increases, such as dose doubling, rather than small incremental changes.	60.60%	27.30%	9.10%	3.00%	0.00%	87.90%	Strong
29	Renal function changes during decongestion should be interpreted in clinical context rather than prompting automatic diuretic de-escalation.	45.50%	45.50%	0.00%	9.10%	0.00%	90.90%	Very strong
30	The risk of persistent congestion should be weighed against a modest transient rise in creatinine when decongestion is effective and perfusion is preserved.	63.60%	27.30%	3.00%	6.10%	0.00%	90.90%	Very strong
31	Combination or intensified diuretic therapy requires close monitoring of renal function, electrolytes, blood pressure, and volume status.	72.70%	18.20%	0.00%	9.10%	0.00%	90.90%	Very strong
32	Decongestion should not be considered complete until the patient has an effective oral diuretic regimen, stable safety parameters, and a clear follow-up plan.	63.60%	30.30%	6.10%	0.00%	0.00%	93.90%	Very strong
33	Before discharge, patients should receive a follow-up plan that includes laboratory monitoring, weight monitoring, symptom recognition, and guidance on when to seek clinical review.	69.70%	24.20%	0.00%	6.10%	0.00%	93.90%	Very strong
34	Stable decongestion should be used as an opportunity to initiate or optimize guideline-directed medical therapy whenever clinically appropriate.	60.60%	33.30%	3.00%	3.00%	0.00%	93.90%	Very strong
35	Long-term loop diuretic therapy should aim to maintain stable euvolemia and prevent recurrent congestion, which may reduce the risk of worsening heart failure events and rehospitalization.	60.60%	27.30%	12.10%	0.00%	0.00%	87.90%	Strong
36	Long-term loop diuretic when optimized scientifically may prevent re-congestion without causing progressive renal dysfunction.	51.50%	39.40%	6.10%	3.00%	0.00%	90.90%	Very strong
37	Thiazide or thiazide-like therapy should be considered for loop diuretic resistance when close safety monitoring is available.	33.30%	54.50%	3.00%	9.10%	0.00%	87.90%	Strong
38	Thiazide-type add-on therapy should generally be used as short-course intensification rather than indefinite escalation.	33.30%	57.60%	6.10%	3.00%	0.00%	90.90%	Very strong
39	Acetazolamide should be considered in selected hospitalized patients with acute heart failure and volume overload, without contraindications, when early intensified decongestion is desired.	24.20%	45.50%	21.20%	6.10%	3.00%	71.90%	No consensus
40	SGLT2 inhibitors should be initiated or continued in hospitalized heart failure patients once clinically stable and when not contraindicated.	63.60%	27.30%	0.00%	6.10%	3.00%	93.80%	Very strong
41	SGLT2 inhibitors should not be considered substitutes for loop diuretics in severe congestion or acute pulmonary edema.	57.60%	27.30%	3.00%	9.10%	3.00%	87.50%	Strong
42	Ultrafiltration should not be used routinely as first-line decongestive therapy in acute heart failure.	45.50%	39.40%	6.10%	9.10%	0.00%	84.80%	Strong

43	Ultrafiltration should be reserved for selected patients with refractory congestion after optimized pharmacologic therapy.	54.50%	36.40%	0.00%	9.10%	0.00%	90.90%	Very strong
44	Health systems and hospitals should consider standardized decongestion pathways for patients hospitalized with acute heart failure, adapted to local resources.	54.50%	36.40%	3.00%	6.10%	0.00%	90.90%	Very strong
45	Early response checkpoints after intravenous loop diuretic therapy should be incorporated into inpatient workflows where feasible.	54.50%	36.40%	3.00%	6.10%	0.00%	90.90%	Very strong
46	The AICAR framework -- Assessment, Initiation, Customization, Adjustment, and Reassessment -- is a clinically useful structure for organizing personalized decongestion in heart failure.	60.60%	30.30%	3.00%	6.10%	0.00%	90.90%	Very strong

TABLE 4: Round 1 statement-level response distributions

*Agreement = strongly agree + agree, with “outside scope” excluded from the denominator

Appendix 2

Statement	Source	Strongly agree (n)	Agree (n)	Disagree (n)	Strongly disagree (n)	Outside scope (n)	Eligible (n)	Total agreement (n)	Dissent (n)	Agreement (%)	Dissent (%)	Strong agreement (%)	Ordinal SD (exploratory)	Published agreement (%)
1	Round 1 retained	26	4	0	3	0	33	30	3	90.9	9.1	78.8	0.886	90.9
2	Round 2 reassessed	27	4	0	2	0	33	31	2	93.9	6.1	81.8	0.758	93.9
3	Round 1 retained	21	9	0	3	0	33	30	3	90.9	9.1	63.6	0.891	90.9
4	Round 2 reassessed	15	15	2	1	0	33	30	3	90.9	9.1	45.5	0.725	90.9
5	Round 1 retained	24	6	0	3	0	33	30	3	90.9	9.1	72.7	0.891	90.9
6	Round 2 reassessed	23	7	1	2	0	33	30	3	90.9	9.1	69.7	0.82	90.9
7	Round 1 retained	25	6	0	2	0	33	31	2	93.9	6.1	75.8	0.771	93.9
8	Round 1 retained	15	14	2	1	1	32	29	3	90.6	9.4	46.9	0.734	90.6
9	Round 1 retained	17	12	1	2	1	32	29	3	90.6	9.4	53.1	0.82	90.6
10	Round 2 reassessed	28	3	0	2	0	33	31	2	93.9	6.1	84.8	0.75	93.9
11	Round 1 retained	23	5	3	1	1	32	28	4	87.5	12.5	71.9	0.788	87.5
12	Round 1 retained	16	12	3	2	0	33	28	5	84.8	15.2	48.5	0.862	84.8
13	Round 1 retained	22	8	0	3	0	33	30	3	90.9	9.1	66.7	0.892	90.9
14	Round 2	26	5	0	2	0	33	31	2	93.9	6.1	78.8	0.765	93.9

	reassessed													
15	Round 2 reassessed	12	19	1	1	0	33	31	2	93.9	6.1	36.4	0.664	93.9
16	Round 2 reassessed	11	17	4	1	0	33	28	5	84.8	15.2	33.3	0.744	84.8
17	Round 2 reassessed	22	9	0	2	0	33	31	2	93.9	6.1	66.7	0.782	93.9
18	Round 1 retained	18	12	1	2	0	33	30	3	90.9	9.1	54.5	0.814	90.9
19	Round 1 retained	20	10	0	3	0	33	30	3	90.9	9.1	60.6	0.889	90.9
20	Round 2 reassessed	20	11	1	1	0	33	31	2	93.9	6.1	60.6	0.702	93.9
21	Round 2 reassessed	17	14	2	0	0	33	31	2	93.9	6.1	51.5	0.608	93.9
22	Round 2 reassessed	15	15	2	1	0	33	30	3	90.9	9.1	45.5	0.725	90.9
23	Round 2 reassessed	17	14	1	1	0	33	31	2	93.9	6.1	51.5	0.698	93.9
24	Round 1 retained	22	9	1	1	0	33	31	2	93.9	6.1	66.7	0.698	93.9
25	Round 2 reassessed	15	14	2	2	0	33	29	4	87.9	12.1	45.5	0.827	87.9
26	Round 2 reassessed	23	8	0	2	0	33	31	2	93.9	6.1	69.7	0.78	93.9
27	Round 2 reassessed	23	7	2	1	0	33	30	3	90.9	9.1	69.7	0.74	90.9
28	Round 1 retained	24	6	0	3	0	33	30	3	90.9	9.1	72.7	0.891	90.9
29	Round 2 reassessed	23	7	2	1	0	33	30	3	90.9	9.1	69.7	0.74	90.9
30	Round 2 reassessed	20	10	2	1	0	33	30	3	90.9	9.1	60.6	0.744	90.9
31	Round 1 retained	20	11	1	1	0	33	31	2	93.9	6.1	60.6	0.702	93.9
32	Round 2 reassessed	18	12	2	1	0	33	30	3	90.9	9.1	54.5	0.74	90.9
33	Round 2 reassessed	17	13	2	1	0	33	30	3	90.9	9.1	51.5	0.736	90.9
34	Round 2 reassessed	24	6	1	2	0	33	30	3	90.9	9.1	72.7	0.818	90.9
35	Round 1 retained	20	9	4	0	0	33	29	4	87.9	12.1	60.6	0.702	87.9
36	Round 1 retained	17	13	2	1	0	33	30	3	90.9	9.1	51.5	0.736	90.9
37	Round 2 reassessed	17	14	0	2	0	33	31	2	93.9	6.1	51.5	0.776	93.9
38	Round 2 reassessed	10	15	4	1	3	30	25	5	83.3	16.7	33.3	0.763	83.3
	Round 2													

39	reassessed	22	7	0	2	2	31	29	2	93.5	6.5	71	0.794	93.5
40	Round 2 reassessed	22	8	1	1	1	32	30	2	93.8	6.3	68.8	0.701	93.8
41	Round 1 retained	18	12	1	2	0	33	30	3	90.9	9.1	54.5	0.814	90.9
42	Round 1 retained	18	12	1	2	0	33	30	3	90.9	9.1	54.5	0.814	90.9
43	Round 1 retained	20	10	1	2	0	33	30	3	90.9	9.1	60.6	0.82	90.9

TABLE 5: Dispersion analysis

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: All authors have confirmed that this study did not involve human participants or tissue. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Acknowledgements

The authors gratefully acknowledge the Association of Indian Cardiologists for Academics and Research (AICAR) for its academic support, coordination, and facilitation of this collaborative initiative.

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