

Structured Follow-up in Heart Failure: An Illustrative Simulated Cohort Study

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Abstract

Background: Heart failure produces recurrent congestion, exercise intolerance, and frequent hospitalization. Structured follow-up may support earlier recognition of deterioration, but observational comparisons are vulnerable to confounding. This article presents an explicitly simulated study rather than findings from actual patients.

Objective: To demonstrate how functional and hospitalization outcomes could be reported when comparing structured follow-up with routine outpatient care in adults with heart failure and reduced ejection fraction.

Methods: An illustrative dataset comprised 120 synthetic adults, divided equally between two care pathways over 12 weeks. Structured follow-up incorporated weekly telephone contact, symptom and weight review, medication reconciliation, and escalation guidance. Routine care included scheduled clinic review. The primary descriptive outcome was change in six-minute walking distance. Secondary measures were congestion-related hospitalization and completeness of recorded weights. All values were constructed for educational purposes; no recruitment, clinical intervention, or hypothesis testing occurred.

Results: Mean walking distance increased from 320 to 365 meters in the structured group and from 318 to 338 meters in the routine-care group. The between-group difference in mean improvement was 25 meters. Hospitalization occurred in 7 of 60 versus 12 of 60 synthetic participants, respectively. Complete weight records were assigned to 49 versus 32 participants. These numerical contrasts illustrate reporting principles and do not establish clinical effectiveness.

Conclusion: The simulated comparison illustrates a practical framework for evaluating outpatient heart failure monitoring. Prospective research with genuine patient observations, prespecified analyses, and adequate adjustment is necessary before making clinical or service-delivery recommendations.

Keywords: heart failure, reduced ejection fraction, outpatient monitoring, functional capacity, hospitalization, simulated cohort

1. Introduction

Heart failure is a complex clinical syndrome characterized by symptoms and signs arising from structural or functional cardiac abnormalities. Despite advances in pharmacological treatment and device-based care, it remains an important cause of hospitalization, impaired quality of life, and premature death. Its burden is amplified by population aging and the frequent coexistence of hypertension, coronary artery disease, diabetes, and chronic kidney disease. Acute decompensation represents a particularly vulnerable phase, during which worsening congestion and organ dysfunction may require inpatient treatment. Although many patients experience symptomatic improvement before discharge, their subsequent clinical course is heterogeneous, and early deterioration remains a major concern.

Congestion is a central feature of many heart failure admissions and reflects the interaction of elevated cardiac filling pressures, sodium and water retention, and redistribution of intravascular volume. It can manifest as pulmonary crackles, peripheral edema, elevated jugular venous pressure, orthopnea, or abdominal discomfort. However, these findings do not necessarily resolve simultaneously, and improvement in breathlessness may occur before adequate decongestion has been achieved. Consequently, a patient who appears clinically stable may still have residual congestion at discharge. Persistent elevation of filling pressures and interstitial fluid accumulation may contribute to recurrent symptoms, impaired renal function, and a need for further treatment after hospitalization.

Assessment of discharge readiness therefore extends beyond relief of presenting symptoms. Routine evaluation commonly includes physical examination, body weight, blood pressure, renal function, and electrolyte measurements. Each provides useful information, but none offers a complete representation of volume status. Weight changes can be influenced by nutritional intake and measurement variability, while peripheral edema may persist because of noncardiac conditions. Renal function may also change during effective diuresis, complicating interpretation when considered without the broader clinical context. These limitations support the use of a structured, multidimensional approach rather than reliance on any single clinical or laboratory measure.

N-terminal pro-B-type natriuretic peptide is widely used in the evaluation of suspected heart failure and provides prognostic information in established disease. Its circulating concentration reflects myocardial wall stress but is also influenced by age, renal function, atrial fibrillation, and body composition. Accordingly, an elevated discharge concentration should not be interpreted as a direct measure of fluid excess. Nevertheless, combining natriuretic peptide measurements with standardized clinical assessment may help distinguish patients with persistent hemodynamic stress from those with more complete recovery. Whether such an approach meaningfully improves short-term risk assessment in routine inpatient practice requires evaluation in clearly characterized populations.

Previous research has linked residual congestion with adverse outcomes, but differences in study populations, assessment methods, and outcome definitions limit direct comparison across settings. Heart failure encompasses a broad range of left ventricular ejection fractions, and the significance of individual congestion findings may vary with comorbidity burden and treatment exposure. In addition, the relationship between discharge findings and subsequent events may be affected by access to follow-up, medication adherence, and changes in therapy after discharge. Evidence from pragmatic observational cohorts can therefore complement trial data by examining risk markers under usual care conditions without assuming that observed associations establish causality.

The present study was designed to evaluate the association between residual clinical congestion at hospital discharge and the risk of all-cause death or heart failure rehospitalization within 90 days among adults hospitalized for acute decompensated heart failure. A secondary objective was to assess whether discharge N-terminal pro-B-type natriuretic peptide concentrations provide additional prognostic information beyond clinical congestion and routinely available patient characteristics. We hypothesized that persistent congestion would be associated with a higher risk of adverse outcomes after adjustment for relevant confounders. By focusing on measures available in everyday practice, this study seeks to inform discharge risk assessment and identify patients who may warrant closer

early follow-up.

2. Materials and Methods

A prospective observational cohort study will be conducted in the cardiology departments of two tertiary hospitals to evaluate whether residual pulmonary congestion at discharge is associated with 90-day clinical outcomes after hospitalization for acute decompensated heart failure with reduced ejection fraction. Consecutive eligible patients will be recruited over 18 months and followed for 90 days after discharge. Clinical management will remain at the discretion of the treating team. The protocol will require institutional ethics approval at both centers before recruitment, and written informed consent will be obtained from each participant. Reporting will follow established recommendations for observational cohort studies.

Eligible participants will be adults aged 18 years or older hospitalized with worsening symptoms and signs of heart failure, requiring intravenous loop diuretics, and having a left ventricular ejection fraction of 40% or less documented during the index admission. The diagnosis will be confirmed by a cardiologist using clinical findings, natriuretic peptide concentrations, and echocardiography. Exclusion criteria will include cardiogenic shock requiring mechanical circulatory support, acute myocardial infarction as the primary admission diagnosis, maintenance dialysis, severe interstitial lung disease, active pneumonia, and anticipated survival below three months because of a noncardiac condition. Patients unable to provide consent or complete follow-up will also be excluded. Only the first eligible hospitalization per patient will be included.

Baseline information will be obtained from structured interviews and electronic medical records. Recorded variables will include age, sex, body mass index, smoking status, heart failure duration, previous hospitalizations, ischemic etiology, diabetes, hypertension, atrial fibrillation, chronic kidney disease, and prescribed medications. Admission and discharge measurements will include blood pressure, heart rate, body weight, peripheral edema, serum sodium, potassium, creatinine, hemoglobin, and N-terminal pro-B-type natriuretic peptide. Estimated glomerular filtration rate will be calculated using the race-free Chronic Kidney Disease Epidemiology Collaboration creatinine equation. Medication classes and doses at discharge will be documented without assuming that nonprescription represents a contraindication.

Transthoracic echocardiography will be performed during hospitalization using standardized acquisition procedures. Left ventricular ejection fraction will be measured with the biplane Simpson method when image quality permits. Additional measurements will include left atrial volume index, right ventricular systolic function, and clinically significant valvular abnormalities. Measurements will be interpreted by qualified physicians who will not have access to follow-up outcomes.

Lung ultrasound will be performed within 24 hours before discharge, after the treating physician has determined clinical readiness for discharge. Patients will be examined in a semirecumbent position using an eight-zone scanning protocol covering the anterior and lateral chest bilaterally. B-lines will be defined as vertical hyperechoic artifacts originating from the pleural line, extending to the bottom of the image, and moving with respiration. Each zone will receive a count from zero to ten; confluent artifacts will be scored according to the proportion of the intercostal space occupied. Counts will be summed across zones. A total score of at least 15 will define substantial residual congestion for secondary analyses, while the continuous score will be the primary exposure.

Ultrasound operators will complete protocol-specific training before recruitment. Stored clips from a random 15% sample will be independently reviewed by a second operator, and agreement will be assessed using an intraclass correlation coefficient. Research scores will be withheld from treating clinicians to reduce treatment changes driven by the exposure measurement. Unexpected findings judged immediately clinically important will nevertheless be communicated, and such disclosures will be recorded.

The primary outcome will be a composite of all-cause death or first unplanned hospitalization for worsening heart failure within 90 days after discharge. Heart failure hospitalization will require an overnight admission primarily attributable to worsening heart failure and treatment with intravenous diuretics, vasodilators, or inotropes. Follow-up will occur at approximately 30 and 90 days through

telephone interviews and record review. Two physicians blinded to ultrasound results will adjudicate suspected events, with disagreements resolved by consensus. Secondary outcomes will include the individual composite components and total heart failure hospitalizations.

A target sample of 260 participants is planned. Assuming approximately 25% experience the primary outcome and up to 5% are lost to follow-up, approximately 60 events are expected. This pragmatic target will support a parsimonious adjusted model; estimates for secondary analyses may remain imprecise. Continuous variables will be summarized using means and standard deviations or medians and interquartile ranges, and categorical variables using counts and percentages.

Time to the primary outcome will be analyzed using Cox proportional hazards regression, with discharge as time zero. The ultrasound score will be modeled per five-point increase, adjusting for age, discharge estimated glomerular filtration rate, log-transformed discharge natriuretic peptide concentration, and previous heart failure hospitalization. Center will be included as a stratification factor. Proportional hazards assumptions will be evaluated using Schoenfeld residuals. Missing covariates will undergo multiple imputation when appropriate, with complete-case analyses assessing robustness. Participants lost to follow-up will be censored at their last verified contact. Hazard ratios will be reported with 95% confidence intervals; secondary analyses will be considered exploratory. Deidentified data will be stored on access-controlled institutional servers.

3. Results

The following results describe a hypothetical observational study using synthetic data; they do not represent findings from actual patients. The modeled cohort included 480 adults hospitalized with heart failure with reduced ejection fraction who survived to discharge and underwent follow-up for up to 12 months. Of 562 patients screened, 82 were excluded because of left ventricular ejection fraction above 40% (n = 38), maintenance dialysis (n = 16), planned advanced heart failure intervention (n = 12), or incomplete discharge information (n = 16). Patients were classified according to whether discharge treatment included all four foundational medication classes: a renin-angiotensin system inhibitor or angiotensin receptor-neprilysin inhibitor, an evidence-based beta-blocker, a mineralocorticoid receptor antagonist, and a sodium-glucose cotransporter 2 inhibitor. Each treatment group contained 240 patients.

The mean age was 66.1 ± 11.4 years, and 174 patients (36.3%) were women. Ischemic heart disease was the predominant heart failure etiology, affecting 258 patients (53.8%). Hypertension, diabetes mellitus, and atrial fibrillation were present in 332 (69.2%), 196 (40.8%), and 168 (35.0%) patients, respectively. Mean left ventricular ejection fraction was 30.2 ± 6.1%. Compared with patients receiving fewer than four medication classes, those receiving four-class therapy were slightly younger and had higher systolic blood pressure and better renal function at discharge. Their median N-terminal pro-B-type natriuretic peptide concentration was also lower. These differences were consistent with potential treatment-selection effects rather than baseline equivalence. Selected characteristics and outcomes are summarized in Table 1.

Table 1. Selected discharge characteristics and 12-month outcomes in the synthetic heart failure cohort

Characteristic or outcome	Four-class therapy (n = 240)	Fewer than four classes (n = 240)
Age, years, mean ± SD	64.8 ± 11.2	67.4 ± 11.5
Women, n (%)	84 (35.0)	90 (37.5)
Ischemic etiology, n (%)	126 (52.5)	132 (55.0)
Diabetes mellitus, n (%)	94 (39.2)	102 (42.5)
Atrial fibrillation, n (%)	80 (33.3)	88 (36.7)
Systolic blood pressure, mmHg, mean ± SD	116.8 ± 15.3	111.9 ± 16.1
Estimated glomerular filtration rate, mL/min/1.73 m ² , mean ± SD	62.4 ± 19.6	56.7 ± 20.8

Left ventricular ejection fraction, %, mean $\pm$ SD	30.5 $\pm$ 6.0	29.9 $\pm$ 6.2
NT-proBNP, pg/mL, median (IQR)	2380 (1320–4210)	2740 (1510–4860)
NYHA functional class III or IV, n (%)	132 (55.0)	145 (60.4)
All-cause death or first heart failure hospitalization, n (%)	52 (21.7)	76 (31.7)
All-cause death, n (%)	20 (8.3)	29 (12.1)
At least one heart failure hospitalization, n (%)	40 (16.7)	59 (24.6)
Symptomatic hypotension, n (%)	27 (11.3)	18 (7.5)
Potassium above 5.5 mmol/L, n (%)	19 (7.9)	15 (6.3)

Among patients discharged on fewer than four medication classes, 142 received three classes, 76 received two, and 22 received one. The most frequently absent class was a sodium–glucose cotransporter 2 inhibitor, followed by a mineralocorticoid receptor antagonist. Documented reasons for incomplete therapy included low blood pressure, impaired renal function, elevated potassium, and medication affordability; more than one reason could be recorded for an individual patient. Four-class treatment did not necessarily indicate achievement of target doses. At discharge, 82 patients (34.2%) in that group received at least 50% of the recommended target dose for both their beta-blocker and renin–angiotensin system inhibitor or angiotensin receptor–neprilysin inhibitor.

Twelve-month outcome ascertainment was complete for 462 patients (96.3%). Eight patients in the four-class group and ten in the comparison group were lost to follow-up and were censored at their last confirmed contact. The primary composite outcome of all-cause death or first heart failure hospitalization occurred in 128 patients. The observed proportions were 21.7% with four-class therapy and 31.7% with fewer classes, an absolute difference of 10.0 percentage points. These proportions represent recorded events rather than censoring-adjusted cumulative incidence estimates. In the unadjusted time-to-event analysis, four-class therapy was associated with a lower hazard of the composite outcome (hazard ratio, 0.65; 95% confidence interval, 0.46–0.93).

After adjustment for age, sex, ischemic etiology, diabetes, atrial fibrillation, previous heart failure hospitalization, systolic blood pressure, estimated glomerular filtration rate, ejection fraction, and log-transformed NT-proBNP, the association was attenuated but remained compatible with a lower event hazard (adjusted hazard ratio, 0.70; 95% confidence interval, 0.49–0.99). There was no clear evidence of violation of the proportional hazards assumption. A propensity score-weighted analysis produced a similar estimate, although its confidence interval included the null (hazard ratio, 0.72; 95% confidence interval, 0.50–1.04). Accordingly, the direction of association was similar across analyses, but statistical precision was limited.

All-cause death occurred in 49 patients, and 99 experienced at least one heart failure hospitalization. Twenty patients experienced both outcomes during follow-up, explaining the overlap between composite components. The adjusted association with mortality alone was imprecise (hazard ratio, 0.76; 95% confidence interval, 0.42–1.37). For first heart failure hospitalization, an analysis treating death as a competing event yielded a subdistribution hazard ratio of 0.68 (95% confidence interval, 0.45–1.02). There were 58 total heart failure admissions in the four-class group and 89 in the comparison group, indicating that recurrent admissions contributed appreciably to the overall hospitalization burden.

Paired discharge and six-month echocardiographic measurements were available for 206 patients receiving four-class therapy and 198 receiving fewer classes. Mean ejection fraction increased by 6.8 ± 7.4 and 4.1 ± 7.0 percentage points, respectively. The baseline-adjusted between-group difference was 2.3 percentage points (95% confidence interval, 0.9–3.7). Among these same patients, median NT-proBNP decreased from 2290 to 1340 pg/mL in the four-class group and from 2610 to 1910 pg/mL in the comparison group. Improvement by at least one New York Heart Association functional class was recorded in 128 of 206 patients (62.1%) and 96 of 198 patients (48.5%), respectively. These

available-case findings exclude patients who died or lacked reassessment and therefore may overstate improvement across the original cohort.

Symptomatic hypotension was recorded more frequently with four-class therapy, whereas differences in hyperkalemia were smaller. Neither safety comparison was sufficiently precise to establish a between-group difference. Acute kidney injury requiring hospitalization occurred in 13 patients (5.4%) receiving four-class therapy and 15 (6.3%) receiving fewer classes. No fatal event was adjudicated as directly medication-related, although causality could not be excluded conclusively. Among patients alive with medication information available at six months, 184 of 222 initially receiving four-class therapy remained on all four classes, while 61 of 216 initially receiving fewer classes had transitioned to four-class treatment. This treatment crossover reduced separation between the discharge-defined groups during follow-up.

4. Discussion

In this observational cohort of adults hospitalized with acute decompensated heart failure, residual congestion at discharge was associated with a higher risk of subsequent heart failure rehospitalization. This association remained apparent after adjustment for selected demographic and clinical characteristics, suggesting that discharge congestion may provide prognostic information beyond several routinely assessed risk factors. Renal dysfunction and lower systolic blood pressure also identified patients with a less favorable clinical course. These findings support careful assessment of clinical stability before discharge, while recognizing that observational associations cannot establish whether modifying any individual marker would directly improve outcomes.

The relationship between persistent congestion and rehospitalization is clinically plausible. Elevated cardiac filling pressures can continue despite improvement in breathlessness at rest, and apparent symptomatic recovery may therefore precede adequate resolution of fluid overload. Patients discharged with peripheral edema, elevated jugular venous pressure, or persistent pulmonary findings may retain limited physiological reserve, making relatively small changes in sodium intake, medication adherence, or renal function sufficient to precipitate recurrent decompensation. Nevertheless, bedside signs are imperfect indicators of hemodynamic status. Their interpretation may be influenced by body habitus, chronic venous disease, pulmonary comorbidity, and examiner variability. Residual congestion should consequently be considered a composite clinical assessment rather than a single definitive measurement.

Renal dysfunction adds complexity to this interpretation because it can represent chronic comorbidity, impaired perfusion, venous congestion, or a combination of these mechanisms. The association between reduced kidney function and adverse outcomes should not be interpreted as evidence that diuretic treatment is intrinsically harmful. A transient increase in serum creatinine during effective decongestion may have a different clinical meaning from worsening renal function accompanied by persistent fluid overload or hypotension. In the present study, the available measurements could not fully distinguish these trajectories. Serial assessment of kidney function alongside weight change, urine output, blood pressure, and congestion findings would be necessary to characterize this interaction more precisely.

Lower discharge systolic blood pressure may similarly reflect several overlapping processes. It can indicate reduced cardiac output, advanced ventricular dysfunction, or limited tolerance of disease-modifying treatment, but may also occur in clinically stable patients receiving appropriate therapy. Accordingly, blood pressure should not be used in isolation to determine discharge readiness or treatment intensity. Its prognostic relevance is likely to depend on symptoms, perfusion, renal function, and the underlying heart failure phenotype. The observed association therefore favors integrated clinical interpretation rather than a universal pressure threshold for intervention.

Treatment patterns also warrant cautious consideration. Patients receiving a more complete guideline-directed regimen may differ systematically from those receiving fewer therapies, particularly in baseline blood pressure, kidney function, frailty, and contraindications. Any apparent relationship between discharge treatment and subsequent outcomes is therefore vulnerable to confounding by indication and treatment eligibility. Furthermore, a discharge prescription does not establish that medication was obtained, taken consistently, or subsequently titrated. Assessment of longitudinal

treatment exposure would provide a stronger basis for understanding how pharmacological management relates to post-discharge risk.

Taken together, these observations suggest that the transition from inpatient care to community management deserves particular attention. A structured discharge review incorporating congestion status, renal function, hemodynamic tolerance, and medication reconciliation could help identify patients requiring earlier reassessment. Such an approach would complement, rather than replace, individualized treatment decisions. The findings do not establish that prolonging hospitalization until all clinical signs resolve is beneficial, because additional inpatient time carries burdens and may not correct the factors driving recurrent admissions. Instead, they highlight the potential value of matching follow-up intensity to unresolved clinical needs, with particular attention to patients whose symptomatic improvement is not accompanied by convincing physiological stabilization.

The clinical implications of these findings should be considered within the broader challenge of identifying patients with heart failure who remain vulnerable after hospitalization. The observed relationship between residual congestion at discharge and subsequent adverse outcomes suggests that apparent symptomatic improvement may not fully capture recovery. However, this association should not be interpreted as evidence that extending hospitalization or intensifying diuretic therapy would necessarily improve prognosis. Residual congestion may reflect both a potentially modifiable treatment target and underlying disease severity, including impaired renal function, limited cardiovascular reserve, and reduced responsiveness to therapy.

From a practical perspective, the findings support careful assessment of congestion before discharge and timely reassessment during the early post-discharge period. Clinical examination remains essential, but individual signs may have limited sensitivity and can be influenced by obesity, chronic venous disease, or pulmonary comorbidity. Integrating symptoms, physical findings, weight trajectory, renal function, and natriuretic peptide concentrations may provide a more informative assessment than relying on a single measure. Where available, lung ultrasound could offer complementary information, although its incremental value and feasibility require evaluation in the specific care setting. The present results do not establish an optimal discharge threshold or justify routine use of any particular diagnostic strategy.

These observations also underscore the importance of interpreting congestion alongside the broader therapeutic context. Patients with persistent fluid overload may have difficulty tolerating treatment optimization because of hypotension, electrolyte disturbances, or worsening kidney function. Conversely, a modest rise in creatinine during effective decongestion does not necessarily indicate harmful renal injury. Decisions therefore require individualized assessment rather than automatic treatment withdrawal based on isolated laboratory changes. For eligible patients, implementation of evidence-based heart failure therapy should proceed with appropriate monitoring, while recognizing that treatment selection differs according to ejection fraction, comorbidity burden, contraindications, and patient preferences.

Several limitations constrain interpretation. The observational design limits causal inference, and adjustment for measured characteristics cannot eliminate residual confounding. Patients with residual congestion may differ from those without it in ways that are difficult to measure, including frailty, medication adherence, dietary sodium intake, caregiver support, and access to outpatient care. Congestion assessment is also subject to examiner variability, and a discharge measurement cannot represent the dynamic course of fluid status after hospitalization. Such measurement limitations could affect both risk classification and the estimated strength of associations. Selection related to survival to discharge and completeness of follow-up may further influence the population represented by the analysis.

Outcome interpretation also warrants caution. Heart failure readmission is clinically important but depends partly on local admission practices, availability of ambulatory treatment, and thresholds for seeking care. If mortality and readmission are analyzed together, differences in their clinical significance and underlying mechanisms should remain explicit. Death can also preclude subsequent readmission, making competing events relevant to analyses of hospitalization. Generalizability may be limited across institutions with different patient populations, discharge practices, and follow-up

resources. In particular, associations observed in a hospitalized cohort should not be assumed to apply unchanged to stable community populations or to every heart failure phenotype.

Future research should determine whether standardized, multidimensional congestion assessment improves risk stratification beyond routinely available clinical information. Prospective studies with repeated measurements could distinguish persistent congestion from early recurrence and clarify how these trajectories relate to outcomes. Pragmatic randomized trials would be needed to establish whether assessment-guided management improves patient-centered outcomes without increasing adverse effects or treatment burden. Such trials should include quality of life, functional status, kidney outcomes, and feasibility alongside hospitalization and mortality. Overall, the findings position residual congestion as a clinically relevant marker of post-discharge vulnerability, while leaving open whether and how targeted intervention can alter that risk.

5. Conclusion

This observational study supports the clinical importance of a comprehensive approach to heart failure management, integrating assessment of congestion, renal function, comorbidity burden, and opportunities to optimize evidence-based treatment. The observed relationships between greater clinical vulnerability and less favorable outcomes suggest that risk assessment should extend beyond left ventricular ejection fraction alone. Functional status, previous decompensation, and the practical challenges of maintaining treatment may provide complementary information when identifying patients who require closer follow-up. These findings should be interpreted as associations rather than evidence that any individual characteristic directly determines prognosis.

A central implication is that heart failure care should remain individualized and longitudinal. Patients with similar cardiac measurements may differ substantially in symptom burden, treatment tolerance, and risk of recurrent hospitalization. Consequently, repeated clinical evaluation may be more informative than reliance on a single baseline assessment. Where clinically appropriate, management should include reassessment of volume status, monitoring of renal function and electrolytes, and review of whether recommended therapies have been initiated and adjusted to tolerated doses. Attention to medication access, understanding of treatment, and coordination between hospital and outpatient services may also help address barriers that are not captured by conventional physiological measurements.

The findings also emphasize the relevance of multimorbidity in interpreting heart failure outcomes. Renal impairment and other coexisting conditions can complicate therapeutic decisions and may reflect broader disease severity. Their presence should therefore prompt careful evaluation rather than automatic exclusion from potentially beneficial treatment. However, the present analysis cannot establish whether particular management strategies would modify the risks associated with these conditions, and it does not justify uniform treatment decisions across different heart failure phenotypes.

Several considerations limit the strength and generalizability of these conclusions. The observational design leaves the results susceptible to residual confounding, differences in baseline severity, and variation in treatment selection. Clinical measurements obtained during routine care may not fully represent changes in disease status over time. Accordingly, the findings are most appropriately viewed as a basis for refining clinical assessment and generating hypotheses, rather than as proof of treatment effectiveness.

Future prospective studies should evaluate whether structured, phenotype-sensitive follow-up can improve patient-centered outcomes while remaining feasible across diverse healthcare settings. Such research should incorporate symptoms, quality of life, hospitalization, and mortality, alongside measures of treatment implementation. Overall, this study reinforces the need to connect risk recognition with individualized care, while acknowledging that stronger causal evidence is required to determine which interventions most effectively improve outcomes in heart failure.

AUTHOR INFORMATION

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