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Hyponatremia as a Prognostic Factor in Heart Failure: A Narrative Review

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ABSTRACT

Introduction and purpose: Hyponatremia is the most frequent electrolyte abnormality in heart failure (HF) and reflects neurohormonal activation, congestion, renal dysfunction, and treatment intensity. This narrative review assesses whether hyponatremia is only a marker of advanced HF or an independent prognostic factor, and summarizes its value for risk stratification. **Brief description of the state of knowledge:** Across acute and chronic HF cohorts, low serum sodium at admission is consistently associated with longer hospitalization, higher in-hospital mortality, greater post-discharge mortality, and more frequent rehospitalization. Prognostic relevance is also seen for persistent hyponatremia, discharge hyponatremia, hospital-acquired hyponatremia, and worsening sodium trajectories during admission. These associations are present in both reduced and preserved ejection fraction and become stronger when hyponatremia coexists with renal dysfunction, severe congestion, or elevated natriuretic peptides. Cohort studies suggest that sodium improvement during hospitalization is linked to better short-term outcomes, but randomized evidence shows that sodium correction alone does not lower mortality. **Summary (conclusions):** Hyponatremia should be considered a readily available, low-cost prognostic marker in HF rather than an isolated laboratory abnormality. Its value is greatest when interpreted together with congestion, kidney function, and natriuretic peptide burden. Persistent or hospital-acquired hyponatremia identifies particularly vulnerable patients who may benefit from closer monitoring, careful volume reassessment, and optimized guideline-directed therapy. Future studies should determine whether phenotype-guided correction strategies can improve hard clinical outcomes.

Keywords: Heart Failure; Hyponatremia; Prognosis; Mortality; Hospitalization

INTRODUCTION

Heart failure (HF) remains a leading cause of hospitalization, repeated decompensation, and premature death despite major advances in guideline-directed medical therapy. Current European and North American HF guidelines recognize hyponatremia as a clinically important laboratory

abnormality that often accompanies advanced congestion, renal dysfunction, and severe neurohormonal activation [1,2].

From a pathophysiological perspective, low effective arterial blood volume in HF stimulates the sympathetic nervous system, the renin-angiotensin-aldosterone system, and non-osmotic arginine vasopressin release. The resulting preferential retention of free water lowers serum sodium concentration and produces dilutional hyponatremia; less commonly, sodium depletion may occur because of intensive diuretic therapy, low solute intake, or gastrointestinal losses [3,4].

General hyponatremia guidance emphasizes that serum sodium concentration must be interpreted in relation to chronicity, tonicity, extracellular volume, and the risks of overly rapid correction [5,6,7]. In HF, however, the most clinically relevant question is often not only how to correct sodium safely but also what a low sodium concentration signifies for prognosis.

Narrative reviews over the past two decades have consistently described hyponatremia as the most common electrolyte disorder in HF and as a marker of worse symptoms, longer hospital stay, higher readmission risk, and increased mortality [8,9,10,11]. The prognostic association was already suggested in earlier emergency and hospitalized HF cohorts, long before contemporary biomarker-guided management became routine [12,13,14].

Whether hyponatremia is merely a surrogate for disease severity or also identifies a biologically distinct, potentially modifiable high-risk phenotype remains unsettled. The purpose of this narrative review is to summarize the current state of knowledge on hyponatremia as a prognostic factor in acute and chronic HF, to compare evidence across clinical phenotypes and sodium trajectories, and to discuss the practical implications for bedside risk stratification.

DESCRIPTION OF THE STATE OF KNOWLEDGE

Pathophysiological background

In HF, serum sodium integrates several adverse processes at once: reduced cardiac output, impaired effective arterial filling, neurohormonal activation, renal hypoperfusion, and treatment burden. Classic physiological work linked worsening HF to activation of vasopressin, the renin-

angiotensin-aldosterone system, and sympathetic tone, thereby explaining why sodium falls as disease severity increases [3,4].

The pathophysiology is not uniform. Hypervolemic or dilutional hyponatremia is the dominant form in decompensated HF and reflects free-water retention that is disproportionate to sodium retention. Hypovolemic or depletional hyponatremia may emerge in overdiuresed patients, in those receiving thiazides or high natriuretic doses, or in the presence of poor oral intake and gastrointestinal losses [10,11]. Guidelines on hyponatremia therefore recommend a structured diagnostic approach based on symptoms, tonicity, urine studies, and volume assessment before treatment is intensified [5,6,7].

For prognosis, the key implication is that a single sodium value should never be interpreted in isolation. Low sodium can be the biochemical expression of severe congestion and neurohormonal stress, but it can also signal therapy-related sodium depletion. The adverse prognostic weight of hyponatremia is therefore best understood in combination with blood pressure, renal indices, natriuretic peptides, diuretic exposure, and the patient's trajectory during admission [8,9,10,11].

Admission hyponatremia in acute heart failure

Among hospitalized patients with worsening HF, the most influential evidence comes from the OPTIMIZE-HF registry. In 48,612 patients from 259 hospitals, admission hyponatremia was associated with longer hospital stay and higher in-hospital and early post-discharge mortality. After adjustment, each 3 mmol/L decrease in admission sodium below 140 mmol/L increased the risk of in-hospital death by 19.5%, follow-up mortality by 10%, and death or rehospitalization by 8% [15].

The ESCAPE trial extended this observation in severe systolic HF. Baseline hyponatremia remained independently related to 6-month mortality, even after accounting for clinical improvement during hospitalization [16]. Similarly, in the International Collaborative of NT-proBNP Study, hyponatremia present at admission in acutely decompensated HF was associated with higher 1-year mortality, with particularly strong prognostic value in patients who also had markedly elevated NT-proBNP concentrations [17].

These findings have been reproduced in Asian cohorts and other contemporary hospital series. In the COAST study, admission hyponatremia was present in 16.8% of hospitalized patients and independently predicted 12-month mortality (hazard ratio [HR] 1.72) [28]. In a cohort of 2,556

acute HF admissions, Lu et al. found admission hyponatremia to be associated with higher all-cause mortality (HR 1.43) and cardiovascular mortality (HR 1.50) [29].

Not all studies show the same magnitude of effect after adjustment. In a real-world cohort reported by Arevalo Lorido et al., hyponatremia independently predicted in-hospital complications but not 90-day mortality or readmission after multivariable adjustment, suggesting that comorbidity burden may attenuate or partly mediate the observed relationship in older, frailer populations [22]. This heterogeneity is clinically important: sodium is a strong prognostic variable, but its interpretation depends on the background risk profile of the cohort.

Persistent, discharge, and hospital-acquired hyponatremia

Serial sodium measurements frequently outperform a single admission value. In ESCAPE, persistent hyponatremia identified a particularly vulnerable phenotype: mortality at 6 months was 31% in patients with persistent hyponatremia versus 16% in normonatremic patients, and persistent hyponatremia independently predicted death (HR 1.82), HF rehospitalization (HR 1.52), and the composite of death or rehospitalization (HR 1.54) [16].

Hospital-acquired hyponatremia also carries adverse information. In 5,347 hospitalized patients with a diagnosis of HF, community-acquired hyponatremia and hospital-acquired hyponatremia were both associated with worse in-hospital outcomes and prolonged length of stay, but the trajectories were not identical, emphasizing that sodium decline during admission is itself clinically meaningful [23].

Progressive sodium deterioration is especially ominous when patients are normonatremic on admission. Konishi et al. showed that patients who developed hyponatremia by discharge after being normonatremic initially had a much lower 12-month cardiac event-free rate than those who remained normonatremic (22% vs 71%) [24]. Lu et al. likewise reported that the combination of admission hyponatremia and a further drop in sodium during hospitalization conferred incremental risk, with an adjusted HR of 2.26 for death compared with stable normonatremia [29].

Discharge sodium appears particularly relevant for post-hospital risk assessment. In a post hoc ESCAPE analysis, Omar et al. found that discharge hyponatremia among patients with normal admission sodium identified a subgroup with worse outcomes, although the prognostic signal was stronger when hyponatremia persisted from admission through discharge [26].

Not every temporary sodium fall carries the same implication. Verbrugge et al. distinguished transient decompensation-related hyponatremia from treatment-induced hyponatremia and persistent hyponatremia. Their analysis suggested that transient patterns can reflect different mechanisms and may be associated with an intermediate-risk phenotype rather than uniformly catastrophic prognosis [25]. This nuance helps explain why sodium must be interpreted as a dynamic clinical sign rather than as a binary laboratory threshold.

Table 1. Selected studies evaluating hyponatremia as a prognostic factor in heart failure

Study	Population and setting	Hyponatremia definition/pattern	Main prognostic message
OPTIMIZE-HF registry [15]	48,612 hospitalized HF patients	Admission Na <135 mmol/L; continuous analysis below 140 mmol/L	Longer stay and higher in-hospital and follow-up mortality; each 3 mmol/L decrease below 140 mmol/L increased in-hospital death by 19.5%.
ESCAPE trial [16]	433 patients with severe systolic HF	Baseline and persistent hyponatremia; threshold <=134 mmol/L	Persistent hyponatremia independently predicted 6-month death, HF rehospitalization, and death/rehospitalization.
International Collaborative of NT-proBNP Study [17]	628 patients hospitalized for acute decompensated HF	Admission Na <=135 mmol/L	Independent predictor of 1-year mortality, especially when NT-proBNP was markedly elevated.
Balling et al. [18]	3,465 chronic HF outpatients	P-sodium <136 mmol/L	Independent association with hospitalization/death and with all-cause mortality.

Study	Population and setting	Hyponatremia definition/pattern	Main prognostic message
Bettari et al. Duke Databank [19]	1,045 chronic HF patients with systolic dysfunction	Na <135 mmol/L	Independent predictor of all-cause death and cardiovascular death/rehospitalization.
Rusinaru et al. [20]	358 patients after first HFpEF hospitalization	Na <136 mEq/L; discharge trajectory assessed	Predicted overall and cardiovascular mortality; persistent hyponatremia had the poorest 7-year survival.
Bavishi et al. [21]	8,862 ambulatory veterans with HFrEF or HFpEF	Na <=135 mEq/L	Independent predictor of mortality in both HFrEF and HFpEF; hospitalization signal stronger in HFrEF.
Shchekochikhin et al. [23]	5,347 hospitalized patients with HF diagnosis	Community-acquired vs hospital-acquired hyponatremia	Both patterns were associated with worse in-hospital outcomes and longer length of stay.
Konishi et al. [24]	ADHF patients normonatremic on admission	Development of hyponatremia by discharge	Progression to hyponatremia during admission was linked to markedly worse 12-month cardiac prognosis.
Lu et al. [29]	2,556 acute HF admissions	Admission hyponatremia and in-hospital sodium drop	Baseline hyponatremia predicted long-term mortality, and the combination of low baseline sodium plus further sodium decline identified the highest-risk group.

Chronic ambulatory heart failure and evidence across ejection fraction phenotypes

Hyponatremia is not only a hospital problem. In ambulatory chronic HF, Balling et al. studied 3,465 outpatients and found hyponatremia in 17% of cases. After multivariable adjustment, low sodium remained associated with the composite of hospitalization or death (HR 1.2) and with all-cause mortality (HR 1.5) [18].

A similar message emerged from the Duke Databank for Cardiovascular Diseases. Among 1,045 patients with chronic HF and left ventricular systolic dysfunction, hyponatremia was independently associated with all-cause mortality (HR 1.42) and cardiovascular death or rehospitalization (HR 1.45) over long-term follow-up [19].

Evidence in HF with preserved ejection fraction (HFpEF) is especially important because prognostic markers are less well defined in this phenotype. Rusinaru et al. found hyponatremia in 25.4% of patients surviving a first hospitalization for HFpEF; it independently predicted both overall mortality and cardiovascular mortality, and persistent hyponatremia at discharge identified the worst long-term survival [20].

In a large ambulatory veterans cohort, Bavishi et al. demonstrated that hyponatremia predicted mortality in both HFrEF and HFpEF, although its association with hospitalization was stronger in HFrEF than in HFpEF [21]. More recent HFpEF-focused observational work has supported a similar relationship between lower sodium and adverse outcome, confirming that the prognostic signal is not confined to reduced ejection fraction [30].

Taken together, these data indicate that hyponatremia functions as a cross-phenotype marker of vulnerability in HF. The exact magnitude of risk differs across studies, but the direction of association is remarkably stable.

Interaction with congestion, kidney function, and natriuretic peptides

One reason sodium performs well prognostically is that it reflects the intersection of several adverse pathways. Patients with hyponatremia tend to have lower blood pressure, higher diuretic doses, worse renal indices, more severe symptoms, and more prominent biomarker activation [15,16,17,19,29].

The interaction with natriuretic peptides is particularly instructive. Mohammed et al. showed that hyponatremia predicted 1-year mortality mainly in the subgroup with more pronounced NT-proBNP elevation, implying that sodium contributes most when interpreted in the context of substantial wall stress and congestion [17].

Renal dysfunction amplifies the signal further. Both observational cohorts and contemporary reviews emphasize that a low sodium level accompanied by rising blood urea nitrogen, creatinine, or worsening renal function usually denotes a more advanced cardiorenal syndrome rather than an isolated electrolyte disturbance [11,23,39,40].

This multimarker interpretation also helps explain a common clinical puzzle: why some patients with mild hyponatremia appear stable while others do poorly. The answer is often that sodium concentration is not the final prognostic message; rather, it is the accessible surface marker of deeper hemodynamic and renal stress.

Does correction of hyponatremia improve prognosis?

Observational studies suggest that sodium improvement can accompany a better course. In the ACTIV in CHF trial, each 1 mmol/L increase in sodium during hospitalization was associated with lower 60-day mortality in a post hoc analysis [32]. A later meta-analysis of cohort studies also found that improvement of hyponatremia in acute decompensated HF was associated with lower mortality risk [35].

However, association is not the same as causation. The largest randomized evidence comes from EVEREST, in which oral tolvaptan improved dyspnea, body weight, and serum sodium but did not reduce long-term mortality or HF-related morbidity in the overall study population [33]. This is a crucial observation: sodium correction can be biologically and symptomatically meaningful without necessarily altering hard outcomes.

Subgroup analyses provide a more nuanced view. Hauptman et al., using EVEREST data, showed that hyponatremic patients had a more difficult in-hospital course than normonatremic patients, while tolvaptan improved sodium and short-term decongestion; a possible post-discharge benefit was suggested only in patients with more pronounced hyponatremia below 130 mEq/L [34]. Likewise, the rise in first follow-up sodium reported by Omar and Guglin was associated with

fewer rehospitalizations and fewer composite events, but this study remained observational and included a relatively small subgroup [27].

A broad 2023 meta-analysis confirmed that hyponatremia is associated with multiple adverse HF outcomes, including all-cause, cardiac, 30-day, 1-year, and in-hospital mortality [36]. Yet a separate literature on hyponatremia correction across diseases suggests that correction may be more useful as a treatment-stratification biomarker than as proof that sodium itself is the causal driver of risk [37,38].

The practical conclusion is that clinicians should not treat the laboratory number in isolation. When sodium improves because congestion is relieved, neurohormonal stress lessens, kidney perfusion stabilizes, and guideline-directed therapy can be optimized, prognosis may improve. When sodium is raised pharmacologically without changing the underlying HF biology, mortality may not change.

Clinical implications for bedside practice

Serum sodium is inexpensive, universally available, rapidly repeatable, and interpretable at every point of care. For these reasons, it deserves a place in routine HF risk stratification, especially at admission, during decongestion, and at discharge [1,2,39,40]. Adopting such a systematic monitoring approach aligns with the 2023 Focused Update of the ESC guidelines, which emphasizes the dynamic adjustment of therapy based on evolving clinical and biochemical markers [41].

A low sodium value should prompt three bedside questions. First, is the patient primarily congested and dilutional, or depleted and overdiuresed? Second, is the sodium level stable, improving, or worsening over time? Third, does hyponatremia coexist with low blood pressure, renal dysfunction, or markedly elevated natriuretic peptides? The answers to these questions determine whether hyponatremia functions mainly as a warning sign of severe HF biology or as a correctable treatment-related complication [10,11,39,40].

In practical terms, persistent hyponatremia, discharge hyponatremia, and hospital-acquired hyponatremia should trigger closer surveillance after discharge, careful reassessment of volume status, review of diuretic dosing and thiazide exposure, attention to renal function, and optimization of disease-modifying HF therapy whenever tolerated. This comprehensive surveillance is crucial

because, as seen in diverse clinical populations, electrolyte disturbances often reflect a broader systemic failure to maintain physiological stability under stress [42]. The prognostic value of sodium is greatest when it is interpreted as part of a phenotype rather than as a stand-alone abnormality.

Table 2. Clinical interpretation of hyponatremia patterns in heart failure

Pattern	Dominant mechanism	Typical prognostic implication	Practical bedside response
Admission hyponatremia	Usually dilutional; reflects congestion, low effective arterial volume, AVP/RAAS activation	Higher in-hospital mortality, longer stay, higher post-discharge mortality [15,17,28,29]	Assess congestion, blood pressure, kidney function, natriuretic peptide burden, and severity of HF.
Persistent hyponatremia	Ongoing neurohormonal activation and/or unresolved congestion; sometimes prolonged diuretic exposure	Very high post-discharge risk for death and rehospitalization [16,20]	Treat as a high-risk discharge phenotype; intensify follow-up and reassess decongestion strategy.
Discharge hyponatremia	Incomplete physiologic recovery or treatment-related sodium loss	Higher medium-term event risk, especially if hyponatremia was also present at admission [16,26]	Review discharge medications, residual congestion, kidney function, and early outpatient monitoring.
Hospital-acquired or worsening hyponatremia	Treatment-related sodium decline, ongoing HF progression, or evolving cardiorenal dysfunction	Signals adverse in-hospital course and worse subsequent outcomes [23,24,29]	Re-evaluate volume status, diuretic intensity, thiazide exposure, oral intake, and renal trajectory.
Transient decompensation hyponatremia	Early dilutional state that improves with successful decongestion	Intermediate risk; not equivalent to persistent hyponatremia [25]	Interpret in context of response to therapy and overall clinical improvement.

Pattern	Dominant mechanism	Typical prognostic implication	Practical bedside response
Severe hyponatremia (<130 mmol/L)	Advanced dilutional HF, marked neurohormonal activation, or mixed mechanism	Associated with very high risk; may identify subgroups with greater symptomatic benefit from vaptans [34]	Monitor correction carefully, avoid overly rapid sodium rise, and prioritize treatment of the underlying HF phenotype.

Discussion

The accumulated evidence supports a clear overall conclusion: hyponatremia is a robust prognostic factor in HF across acute and chronic settings. Its importance lies not only in the reproducibility of the association with mortality and rehospitalization, but also in the fact that it can be measured repeatedly without additional cost or technical burden.

At the same time, the literature does not support a simplistic view that hyponatremia is uniformly causal. The strongest studies suggest that sodium concentration captures the burden of congestion, neurohormonal activation, renal dysfunction, and aggressive diuretic exposure. This explains why dynamic patterns - persistent hyponatremia, discharge hyponatremia, and worsening sodium during hospitalization - often outperform a single baseline measurement.

Several limitations of the evidence base must be acknowledged. Most prognostic studies are observational and therefore susceptible to residual confounding. Definitions of hyponatremia vary, most commonly <135 or <136 mmol/L, and the timing of sodium sampling is not standardized. Many cohorts predate widespread use of angiotensin receptor-neprilysin inhibitors, sodium-glucose cotransporter-2 inhibitors, and contemporary congestion-monitoring strategies, so the exact calibration of sodium-based risk may evolve in newer populations.

Another limitation is biological heterogeneity. Studies often do not distinguish rigorously between dilutional and depletional hyponatremia, even though these states differ therapeutically and may differ prognostically. In addition, intercurrent hyperglycemia, liver dysfunction, and kidney disease may modify the meaning of a low sodium concentration.

Despite these limitations, the consistency of the overall signal across registries, single-center cohorts, outpatient networks, HFpEF cohorts, and meta-analyses strengthens confidence in the clinical relevance of hyponatremia. The most defensible interpretation is that hyponatremia is a powerful integrative biomarker of advanced HF physiology and treatment complexity, and that some but not all of its associated risk may be modifiable.

Future research should focus on contemporary HF populations treated with current guideline-directed regimens, repeated sodium measurements integrated with congestion biomarkers, and explicit phenotyping of dilutional versus depletional hyponatremia. Intervention studies should test whether phenotype-guided strategies aimed at correcting both sodium and the underlying hemodynamic abnormality can improve hard outcomes beyond simple normalization of the laboratory value.

Conclusions

Hyponatremia is a consistent and clinically useful prognostic marker in acute and chronic HF.
Admission hyponatremia predicts longer hospitalization, higher mortality, and more frequent rehospitalization, while persistent, discharge, and hospital-acquired hyponatremia identify especially high-risk patients.
The prognostic meaning of low sodium is strongest when interpreted together with congestion severity, renal dysfunction, blood pressure, natriuretic peptide burden, and sodium trajectory over time.
Correction of serum sodium may accompany clinical improvement, but current randomized evidence does not show that sodium normalization alone reduces long-term mortality.
In routine practice, hyponatremia should prompt structured reassessment of volume status and HF phenotype, optimization of guideline-directed therapy, and closer follow-up after discharge.

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