

Beyond Worsening Renal Function: Prognostic Value of Dynamic Renal Trajectories in Acute Heart Failure

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Abstract

Introduction. Renal dysfunction is common in patients with acute heart failure (HF) and is associated with a worse prognosis. Dynamic assessment of renal trajectories during hospitalization may provide additional prognostic information.

Objectives. To evaluate the prognostic impact of renal function trajectory during hospitalization in patients with HF, as well as its association with mortality, readmission, and the composite endpoint.

Methods. Retrospective observational study including 1,398 consecutive patients hospitalized for acute HF between 2016 and 2025. Renal function trajectories were defined according to the relative change in creatinine between admission and discharge: improvement ($\geq 20\%$ decrease), worsening ($\geq 20\%$ increase), or stability ($\pm 20\%$). The outcomes of interest were all-cause mortality, HF readmission, and the composite endpoint.

Results. Renal improvement occurred in 17.0% of patients, worsening in 22.0%, and stable renal function in 61.0%. Patients with renal improvement had a worse baseline profile, with higher NT-proBNP levels and lower hemoglobin.

During a follow-up of up to 10 years, there were 520 deaths (37.2%), 546 readmissions (39.1%), and 777 composite events (55.6%). Mortality was significantly higher in the improvement and worsening groups compared with stable group (44.3% and 42.7% vs 33.3%; $p = 0.0009$).

In multivariable analysis, renal worsening was independently associated with higher mortality (HR 1.29; 95% CI 1.03–1.61; $p = 0.028$), whereas renal improvement showed no significant association (HR 1.06; 95% CI 0.83–1.37; $p = 0.635$). Age, NT-proBNP, and hemoglobin were consistent predictors across all models.

Conclusions. Renal function trajectories during hospitalization have differential prognostic implications in acute HF. Renal worsening is independently associated with higher mortality, whereas renal improvement does not confer prognostic benefit. These findings suggest that renal improvement may reflect transient hemodynamic changes rather than true functional recovery.

INTRODUCTION

Renal dysfunction is a common finding in patients with heart failure (HF) and is consistently associated with a worse prognosis [1–3]. In this context, dynamic changes in renal function during hospitalization have attracted increasing interest, particularly the so-called renal improvement or worsening, as potential markers of clinical course [4–6].

Traditionally, worsening renal function has been associated with an increased risk of adverse events, including mortality and readmissions [2,3]. However, the clinical significance of renal improvement is less clear. Although it could be interpreted as a favorable sign, several studies suggest that these

changes may reflect transient hemodynamic modifications or hemoconcentration phenomena rather than true recovery of renal function [7,8].

On the other hand, most studies have evaluated renal function in isolation or at single time points, without considering its trajectory during hospitalization [1,9]. This dynamic approach could provide additional prognostic information, allowing the identification of patterns with different clinical significance [4,5,10].

In this context, the aim of the present study was to analyze the prognostic impact of different renal function trajectories during hospitalization in patients with HF, assessing their association with mortality, readmission, and the composite endpoint. Additionally, we investigated whether these trajectories retain independent prognostic value after adjustment for relevant clinical and laboratory variables.

MATERIALS AND METHODS

A retrospective observational study was conducted including consecutive patients hospitalized for acute HF at a tertiary care center between January 1, 2016, and December 31, 2025.

Readmission was defined as any HF-related rehospitalization. A total of 2,062 patients with a diagnosis of HF were identified. For the analysis, planned admissions for diagnostic evaluation or invasive treatment (n=423), patients lost to follow-up (n=189), and those with a single laboratory determination or with measurements obtained more than 48 hours before discharge (n=52) were excluded. The final cohort consisted of 1,398 patients.

Clinical, laboratory, therapeutic, and echocardiographic variables were collected during hospitalization. Data were systematically recorded in a structured database at discharge by cardiologists with expertise in HF. Information on mortality and readmissions was obtained through clinical record review and outpatient follow-up.

Renal function was assessed dynamically using two time points:

- Baseline value: serum creatinine at admission
- Final value: last available determination within 48 hours prior to discharge

The relative change in creatinine was calculated using the formula: $(\text{final creatinine} - \text{baseline creatinine}) / \text{baseline creatinine} \times 100$

Patients were classified into three renal function trajectories:

- Renal improvement: $\geq 20\%$ decrease
- Renal worsening: $\geq 20\%$ increase
- Stable renal function: change between -20% and $+20\%$

The $\pm 20\%$ relative change in creatinine was selected as the classification threshold based on prior studies evaluating dynamic renal trajectories in HF [5,6], to identify clinically relevant changes while

minimizing biological variability. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation.

NT-proBNP levels and other biomarkers were assessed at admission and at discharge (<48 hours) to evaluate their impact in the multivariable analysis.

The study was approved by the local ethics committee and conducted in accordance with the principles of the Declaration of Helsinki. Due to its retrospective design, informed consent was waived.

Statistical Analysis

Continuous variables were expressed as mean $\pm$ standard deviation or median (interquartile range), according to their distribution, assessed using the Shapiro–Wilk test. Categorical variables were expressed as frequencies and percentages.

Comparisons between groups were performed using ANOVA or the Kruskal–Wallis test for continuous variables, and the chi-square test for categorical variables. Survival analysis was conducted using Kaplan–Meier curves, compared with the log-rank test.

Cox regression models were used to evaluate the association between renal function trajectories and clinical outcomes. Multivariable models were constructed using a priori variable selection based on clinical relevance, including demographic variables, comorbidities, and baseline laboratory parameters, avoiding the use of automated selection procedures. Multivariable Cox models included the following covariates, selected a priori based on clinical relevance: age, sex, diabetes mellitus, atrial fibrillation, baseline eGFR, NT-proBNP at admission, hemoglobin, left ventricular ejection fraction category, and de novo versus chronic heart failure. Missing data were handled using complete case analysis; the proportion of missing values was less than 5% for all variables included in the models. The number of events per variable was ≥ 10 in all multivariable analyses.

A p-value of less than 0.05 was considered statistically significant. The statistical programs used were IBM SPSS Statistics Version 27® and Stata® Statistics version 16.1. number of serie 501606323439

RESULTS

Clinical Characteristics

Among the included patients, 238 (17.0%) showed renal improvement, 307 (22.0%) renal worsening, and 853 (61.0%) maintained stable renal function.

Patients with renal worsening were older compared with the other groups ($p < 0.001$). Regarding comorbidities, patients with renal improvement had a higher prevalence of diabetes ($p = 0.040$), whereas dyslipidemia was more frequent in the worsening group ($p = 0.047$).

Patients with stable renal function more frequently presented with de novo HF ($p=0.025$), while length of hospital stay was longer in the renal improvement group ($p<0.001$) (Table 1).

Baseline Analytical and Echocardiographic Profile

Patients with renal improvement had a significantly worse baseline profile, with higher levels of urea, creatinine, and uric acid, and lower glomerular filtration rate ($p<0.001$ for all comparisons). They also showed higher NT-proBNP levels at admission ($p<0.001$), as well as lower sodium and hemoglobin levels ($p<0.001$). No significant differences were observed in bilirubin, liver enzymes, or CA125 among the groups.

From an echocardiographic perspective, differences were observed in left ventricular ejection fraction ($p=0.018$), with a higher proportion of preserved left ventricular ejection fraction (LVEF) in the renal worsening group (Table 2).

Dynamic Analytical Changes During Hospitalization

Significant changes were observed in urea, creatinine, and glomerular filtration rate attributable to the distribution of the study groups (Figure 1).

The renal improvement group showed a significant reduction in NT-proBNP levels and an increase in hemoglobin ($p<0.001$), whereas the worsening group exhibited higher NT-proBNP levels ($p<0.001$). No relevant changes were observed in glutamic pyruvic transaminase (GPT) or CA125 levels (Figure 2).

Mortality, Readmission, and Survival Curves

During the 10-year follow-up, there were 520 all-cause deaths (37.2%), 546 HF readmissions (39.1%), and 777 composite events (55.6%).

Mortality was significantly higher in the renal improvement and worsening groups compared with the stable group ($p=0.0009$). Readmission showed a trend toward differences between groups but did not reach statistical significance ($p=0.059$). The composite endpoint was more frequent in the renal improvement group, followed by the worsening group and the stable group ($p<0.001$) (Table 3, Figure 3).

Kaplan–Meier curves showed significant differences between groups for mortality ($p<0.001$), readmission ($p=0.007$), and the composite endpoint ($p<0.001$). The stable renal function group had better survival compared with the other groups, while no significant differences were observed between the improvement and worsening groups (Figure 4).

Multivariable Analysis

In the multivariable analysis, renal worsening was independently associated with a higher risk of mortality compared with the stable group ($p=0.028$), whereas renal improvement showed no significant association ($p=0.635$).

In the readmission analysis, renal trajectory was not independently associated with the risk of events. Similarly, no independent association was observed between renal trajectory and the composite

endpoint.

Other clinical variables and analytical biomarkers (NT-proBNP and hemoglobin) were associated with mortality, readmissions, and the composite endpoint (Table 4, Figure 5).

DISCUSSION

In this observational study including a large cohort of patients hospitalized for acute HF, dynamic renal function trajectories during hospitalization and their prognostic impact were analyzed. The main findings can be summarized in three key aspects: first, worsening renal function was independently associated with an increased risk of mortality. Second, renal improvement did not translate into a prognostic benefit, showing mortality rates similar to the worsening group. Finally, renal trajectories were not independently associated with readmission or the composite endpoint after multivariable adjustment. These results suggest that the dynamic evolution of renal function during hospitalization has complex and non-linear prognostic implications, and its interpretation should be contextualized within the patient's overall clinical status.

A relevant finding of the study is that patients with renal improvement had a more unfavorable baseline profile, characterized by greater renal dysfunction, higher NT-proBNP levels, and lower hemoglobin. This pattern suggests that renal improvement does not necessarily represent true functional recovery, but may instead reflect an initial state of greater congestion or renal hypoperfusion. In this context, the decrease in creatinine during hospitalization could be related to the resolution of renal venous congestion or to hemodynamic changes induced by diuretic therapy. In this regard, previous studies have shown that venous congestion plays a key role in renal dysfunction in HF, even more than cardiac output [11,12]. Therefore, changes in creatinine should be interpreted in the context of decongestion and hemodynamic shifts, rather than as an isolated marker of intrinsic renal function.

On the other hand, renal improvement was not associated with better prognosis. In fact, patients with renal improvement had mortality rates similar to those with renal worsening and higher than those with stable renal function. This finding challenges the classical interpretation of decreased creatinine as a favorable sign. A possible explanation is that renal improvement reflects hemoconcentration phenomena, fluid redistribution, or transient changes in renal perfusion rather than true structural recovery. In this sense, renal improvement may be a marker of patients with a higher baseline congestion burden, requiring more intensive therapy during hospitalization. These findings reinforce the idea that reductions in creatinine should not be interpreted in isolation as a therapeutic goal, but rather in conjunction with other markers of decongestion, such as NT-proBNP or hemoglobin. This result is consistent with previous studies suggesting that decreases in creatinine may reflect hemoconcentration or transient hemodynamic changes during decongestion, rather than true renal recovery [7,13], although the present data do not allow direct mechanistic conclusions.

The absence of significant differences between renal improvement and worsening in survival curves reinforces the hypothesis that both phenomena may represent different manifestations of the same

underlying pathophysiological state. In line with the literature, worsening renal function was associated with a higher risk of mortality [2,14]. However, the lack of association with readmissions suggests that it may act more as a marker of systemic severity than as a direct determinant of recurrent events.

A particularly relevant finding is that the group with stable renal function had the best prognosis across all analyses. This suggests that renal stability may be a marker of hemodynamic balance and lower clinical severity. In contrast, both improvement and worsening of renal function may reflect states of greater physiological instability. From a clinical perspective, this finding emphasizes the importance of avoiding both deterioration and excessive fluctuations in renal function during hospital management. The lack of significant differences between renal improvement and worsening in terms of survival suggests that both trajectories may represent different expressions of the same underlying pathophysiological process, as described in recent studies focusing on renal trajectories [5,6].

NT-proBNP and hemoglobin showed a consistent role as prognostic predictors in all analyzed models [15–17]. NT-proBNP, as a marker of myocardial stress and congestion, supports the hypothesis that volume overload plays a central role in the relationship between renal function and prognosis. Hemoglobin, on the other hand, may reflect hemoconcentration phenomena or nutritional status, both of which are related to clinical outcomes. The integration of biomarkers with renal trajectory could improve risk stratification, although in this study renal trajectory alone showed limited value beyond mortality. These findings are in line with current studies highlighting the relevance of biomarkers in risk stratification in heart failure [16,17].

The results of this study have several relevant clinical implications. They challenge the simplistic interpretation of renal improvement as a sign of good prognosis and they reinforce the need to interpret changes in creatinine within the overall clinical context. In addition, they suggest that therapeutic goals in acute HF should focus on hemodynamic stabilization and effective decongestion rather than strict short-term preservation of renal function. Finally, they support the use of a multidimensional assessment combining renal trajectories with biomarkers and clinical parameters for improved risk stratification. The importance of such multidimensional approaches has been emphasized in previous publications. Overall, these results support the need for an integrated evaluation that combines renal trajectories, biomarkers, and clinical variables, as suggested by recent studies in the field [18].

This study has several limitations that should be considered. Its retrospective design may introduce selection and information biases. The definition of renal trajectories was based on relative changes in creatinine, which may be influenced by multiple factors such as volume status, diuretic therapy or muscle mass and direct measures of congestion or invasive hemodynamic parameters were not available, limiting the pathophysiological interpretation of the findings. Systematic data on medication adjustments during hospitalization (as inotrope use) were not available. Furthermore, we did not distinguish between transient and persistent changes in renal function. So, although multivariable adjustments were performed, residual confounding cannot be excluded.

Heart failure pharmacotherapy changed substantially during the study period (2016–2025), adjustment for calendar year in a sensitivity analysis did not materially alter the results. Nevertheless, a potential influence of temporal treatment changes on renal trajectories cannot be completely excluded.

The exclusion of 52 patients without a discharge creatinine measurement may have biased the results, as these patients could represent a higher-risk subgroup.

Despite these limitations, this study includes a large and well-characterized cohort of consecutive patients hospitalized for acute heart failure, providing strong external validity and real-world representativeness. Moreover, the analysis is based on a dynamic assessment of renal function using serial measurements during hospitalization, overcoming the limitations of traditional static approaches and offering a more comprehensive view of patient pathophysiology. Finally, the study provides a novel and clinically relevant message by challenging the traditional interpretation of renal improvement as a marker of good prognosis, thereby contributing to a better understanding of the role of renal function in acute HF.

Conclusions

In patients hospitalized for acute HF, dynamic renal function trajectories show heterogeneous prognostic value. Renal worsening is independently associated with higher mortality, whereas renal improvement does not confer prognostic benefit.

Stable renal function is associated with the best prognosis, suggesting that fluctuations in creatinine reflect states of greater clinical instability.

These findings highlight the need to interpret changes in renal function within the context of the overall clinical and hemodynamic status, avoiding isolated interpretation of creatinine changes.

Declarations

Funding Sources

This study was not supported by any sponsor or funder.

Ethics Declaration:

Ethics approval and consent to participate

This study protocol was reviewed and approved by Research Ethics Committee of Hospital Universitari i Politècnic La Fe, approval number 2026-0495-1. This study was conducted using anonymized retrospective data obtained from hospital archive records. Individual informed consent was waived due to the retrospective nature of the study and the use of fully anonymized data. As this was a

retrospective, non-interventional observational study, prospective clinical trial registration was not applicable, and the study was not registered in a clinical trial registry.

Conflict of Interest Statement

The authors declare no conflicts of interest relationships relevant to the content of this article.

Data availability

Data are not publicly available due to institutional and patient privacy restrictions, but may be obtained from the corresponding author upon reasonable request and with approval from Instituto de Investigación Sanitaria La Fe.

Author contributions

B.G.C., R.L.-V. and L.A.-B. contributed to the conception and design of the study, as well as the analysis and interpretation of the data; wrote the manuscript; supervised the project; and approved its submission. B.G.C., V.D.T., and J.M.S. contributed to the acquisition and analysis of the data. V.V.S.A. and M.P.F. contributed to the data analysis and provided critical revisions. A.S.-O. and C.V.A.S. contributed to the acquisition of data and provided technical support. L.M.D. contributed to the conception and design of the study and provided critical revisions of the article for important intellectual content. All authors read and approved the final manuscript.

Data Availability Statement

The data underlying this article cannot be shared publicly due to the privacy of individuals that participated in the study. The data will be shared on reasonable request to the corresponding author.

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Tables

Table 1. Clinical Characteristics					
	Renal improvement n: 238	Renal worsening n: 307	Intermediate changes n: 853	p-value	All patients n: 1398
Age (years)*	72.1 ± 12.6	74.6 ± 11.8	70.9 ± 13.2	<0.001	71.9 ± 12.8
Sex (male)	154 (64.6)	170 (55.4)	518 (60.8)	0.083	842 (60.2)
HT	45 (19.0)	76 (24.6)	194 (22.8)	0.136	315 (22.5)
DL	93 (39.2)	145 (47.2)	368 (43.2)	0.047	606 (43.3)
DM	119 (50.0)	131 (42.8)	421 (49.4)	0.040	671 (48.0)
Smoking	53 (22.3)	88 (28.7)	263 (30.8)	0.192	404 (28.9)
Alcohol	11 (4.6)	20 (6.5)	61 (7.2)	0.493	92 (6.6)
COPD	34 (14.1)	43 (13.9)	115 (13.5)	0.989	192 (13.7)
SAHS	29 (12.0)	41 (13.4)	116 (13.6)	0.912	186 (13.3)
Obesity	44 (18.6)	49 (16.1)	100 (11.7)	0.194	193 (13.8)
Hypothyroidism	13 (5.5)	21 (6.7)	55 (6.5)	0.735	89 (6.4)
AF	110 (46.4)	136 (44.2)	397 (46.5)	0.812	643 (46.0)
Stroke	21 (8.8)	31 (10.1)	25 (2.9)	0.065	77 (5.5)
PVD	38 (16.1)	41 (13.4)	158 (18.5)	0.155	237 (14.8)
De novo HF	65 (27.3)	105 (34.2)	315 (36.9)	0.025	485 (34.7)
Ischemic etiology	102 (42,8)	135 (43,9)	317 (37,1)	0.376	554 (39,6)
Length of stay*	13.7 ± 14.3	11.9 ± 10.5	8.2 ± 6.8	<0.001	9.6 ± 8.8
Continuous variables: normal distribution (Shapiro–Wilk test). Mean ± standard deviation. Compared using ANOVA. Categorical variables: n (%). Compared using chi-square test. Abbreviations: AF: atrial fibrillation; COPD: chronic obstructive pulmonary disease; DL: dyslipidemia;					

DM: diabetes mellitus; HF: heart failure; PVD: peripheral vascular disease; SAHS: sleep apnea-hypopnea syndrome.

Table 2. Baseline analytical and echocardiographic profile					
	Renal improvement n: 238	Renal worsening n: 307	Intermediate changes n: 853	p-value	All patients n: 1398
Urea (mg/dL)#	86.0 [62.0-123.0]	73.0 [52.0-103.0]	63.0 [45.0-88.0]	<0.001	67.0 [48.0-95.0]
Creatinine (mg/dL)#	1.8 [1.3-2.5]	1.5 [1.1-2.0]	1.2 [0.9-1.6]	<0.001	1.3 [1.0-1.9]
GFR(ml/min)#	32.0 [22.0-47.0]	40.0 [28.0-55.0]	52.0 [36.0-68.0]	<0.001	46.0 [30.0-63.0]
Bilirrubina (mg/dL)#	0.8 [0.5-1.2]	0.8 [0.5-1.2]	0.7 [0.5-1.1]	0.103	0.8 [0.5-1.1]
AST (U/L)#	28.0 [20.0-42.0]	27.0 [20.0-40.0]	25.0 [19.0-37.0]	0.066	26.0 [19.0-38.0]
ALT (U/L)#	24.0 [16.0-36.0]	23.0 [16.0-34.0]	22.0 [15.0-32.0]	0.087	23.0 [15.0-33.0]
NT-proBNP (pg/mL)#@	780 [420-1450]	620 [310-1100]	480 [220-920]	<0.001	530 [260-1050]
Sodium (mmol/L)#	135 [132-138]	136 [133-139]	137 [134-140]	<0.001	136 [133-139]
Potassium (mmol/L)#	4.6 [4.2-5.1]	4.4 [4.0-4.8]	4.3 [3.9-4.7]	<0.001	4.4 [4.0-4.8]
Hemoglobin (g/dL)#	11.2 [9.8-12.6]	11.8 [10.4-13.1]	12.4 [11.0-13.6]	<0.001	12.1 [10.7-13.4]
Platelets (10 ³ /μL)#	210 [165-270]	220 [170-280]	215 [170-275]	0.349	218 [170-275]

Uric acid (mg/dL)#	8.2 [6.5–10.1]	7.5 [6.0–9.2]	6.8 [5.5–8.5]	<0.001	7.2 [5.8–9.0]
TSAT (%)#	22.0 [15.0–30.0]	23.0 [16.0–31.0]	24.0 [17.0–32.0]	0.742	23.0 [16.0–31.0]
Ferritin (ng/mL)#	180 [90–350]	160 [80–300]	140 [70–260]	0.001	150 [80–290]
CA125 (U/mL)#	38 [20–85]	35 [18–80]	32 [17–75]	0.587	34 [18–78]
Echocardiography					
LVEF				0.018	
Normal	77 (32.4)	132 (43.0)	340 (39.9)		549 (39.3)
Mild dysfunction	17 (7.1)	27 (8.8)	53 (6.2)		97 (6.9)
Moderate dysfunction	35 (14.7)	48 (15.6)	137 (16.1)		220 (15.7)
Severe dysfunction	108 (45.4)	99 (32.2)	322 (37.7)		529 (37.8)
Missing	1 (0.4)	1 (0.3)	1 (0.1)		3 (0.2)
RVEF				0.210	
Normal	130 (54.6)	187 (60.9)	457 (53.6)		774 (55.4)
Mild dysfunction	25 (10.5)	25 (8.1)	99 (11.6)		149 (10.7)
Moderate dysfunction	32 (13.4)	42 (13.7)	102 (12.0)		176 (12.6)
Severe dysfunction	16 (6.7)	16 (5.2)	74 (8.7)		106 (7.6)
Missing	35 (14.7)	37 (12.1)	121 (14.2)		193 (13.8)
#Continuous variables: non-normal distribution (Shapiro–Wilk test). Median and interquartile range. Compared using Kruskal–Wallis test. @:10 Categorical variables: n (%). Compared using chi-square test. The percentage of missing values is shown for each variable. Echocardiographic assessment: LVEF estimated using the biplane Simpson method (Normal: ≥50%, mild dysfunction: 40–49%, moderate dysfunction: 35–39%, severe dysfunction: <35%). RVEF estimated by visual assessment. Abbreviations: ALT: alanine aminotransferase; AST: aspartate aminotransferase; CA125: cancer antigen 125; GFR: glomerular filtration rate; NT-proBNP: N-terminal pro-B-type Natriuretic peptide; LVEF: left ventricular ejection fraction; RVEF: right ventricular ejection fraction; TSAT: transferrin saturation.					

Table 3. Mortality and readmission					
	Renal improvement n: 238	Renal worsening n: 307	Intermediate changes n: 853	p- value	All patients n: 1398
Exitus	105 (44.3)	131 (42.7)	284 (33.3)	0.0009	520 (37.2)
Readmission	109 (46.4)	113 (36.9)	324 (38.3)	0.059	546 (39.1)
Composite endpoint	161 (67.6)	174 (56.7)	442 (51.8)	<0.001	777 (55.6)
Categorical variables: n (%). Compared using the chi-square test.					

Table 4. Multivariable Analysis of Mortality, Readmission and Composite Event						
Variable	Mortality HR (95% CI)	p	Readmission HR (95% CI)	p	Composite Event HR (95% CI)	p
Improvement vs stable	1.06 (0.83– 1.37)	0.635	1.12 (0.88–1.43)	0.348	1.13 (0.93–1.38)	0.224
Worsening vs stable	1.29 (1.03– 1.61)	0.028	1.05 (0.84–1.31)	0.655	1.13 (0.94–1.36)	0.210
Age (per year)	1.04 (1.03– 1.05)	<0.001	1.02 (1.01–1.03)	<0.001	1.03 (1.02–1.04)	<0.001
Sex	1.17 (0.96– 1.43)	0.123	1.30 (1.06–1.59)	0.010	1.15 (0.98–1.36)	0.088
Hypertension	0.82 (0.64– 1.06)	0.123	0.94 (0.75–1.17)	0.633	0.83 (0.68–1.02)	0.066
Diabetes	1.22 (1.00– 1.48)	0.052	1.16 (0.92–1.46)	0.118	1.22 (1.04–1.42)	0.017
NT-proBNP (baseline)	1.00 (1.00– 1.00)	<0.001	1.00 (1.00–1.00)	0.004	1.00 (1.00–1.00)	<0.001
Hemoglobin	0.94 (0.90– 0.99)	0.010	0.96 (0.92–0.99)	0.036	0.97 (0.94–1.00)	0.071

HR: Hazard Ratio; CI: Confidence Interval.

Figures

Figure 1. Dynamic changes in renal parameters during hospitalization

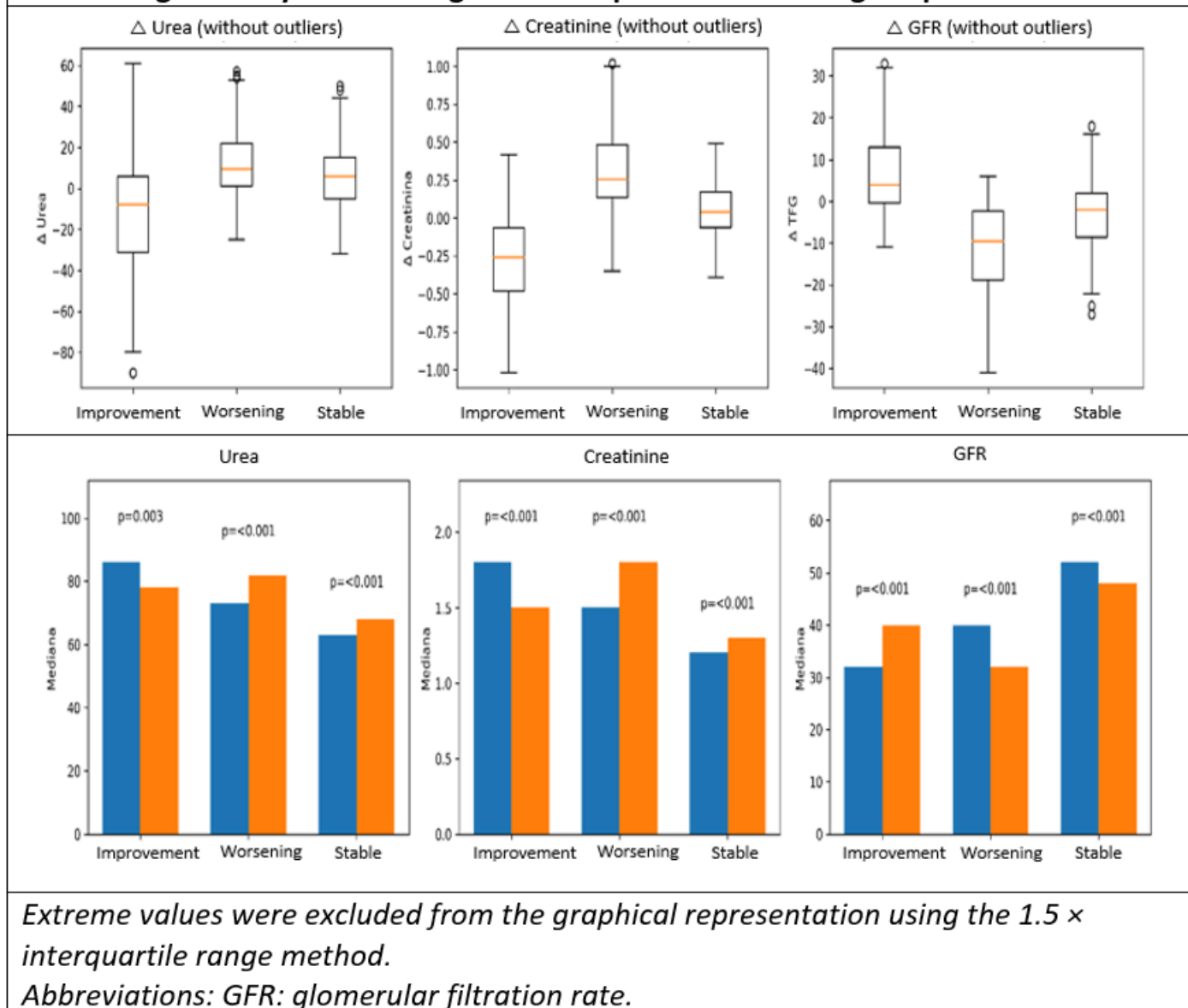


Figure 1

See image above for figure legend.

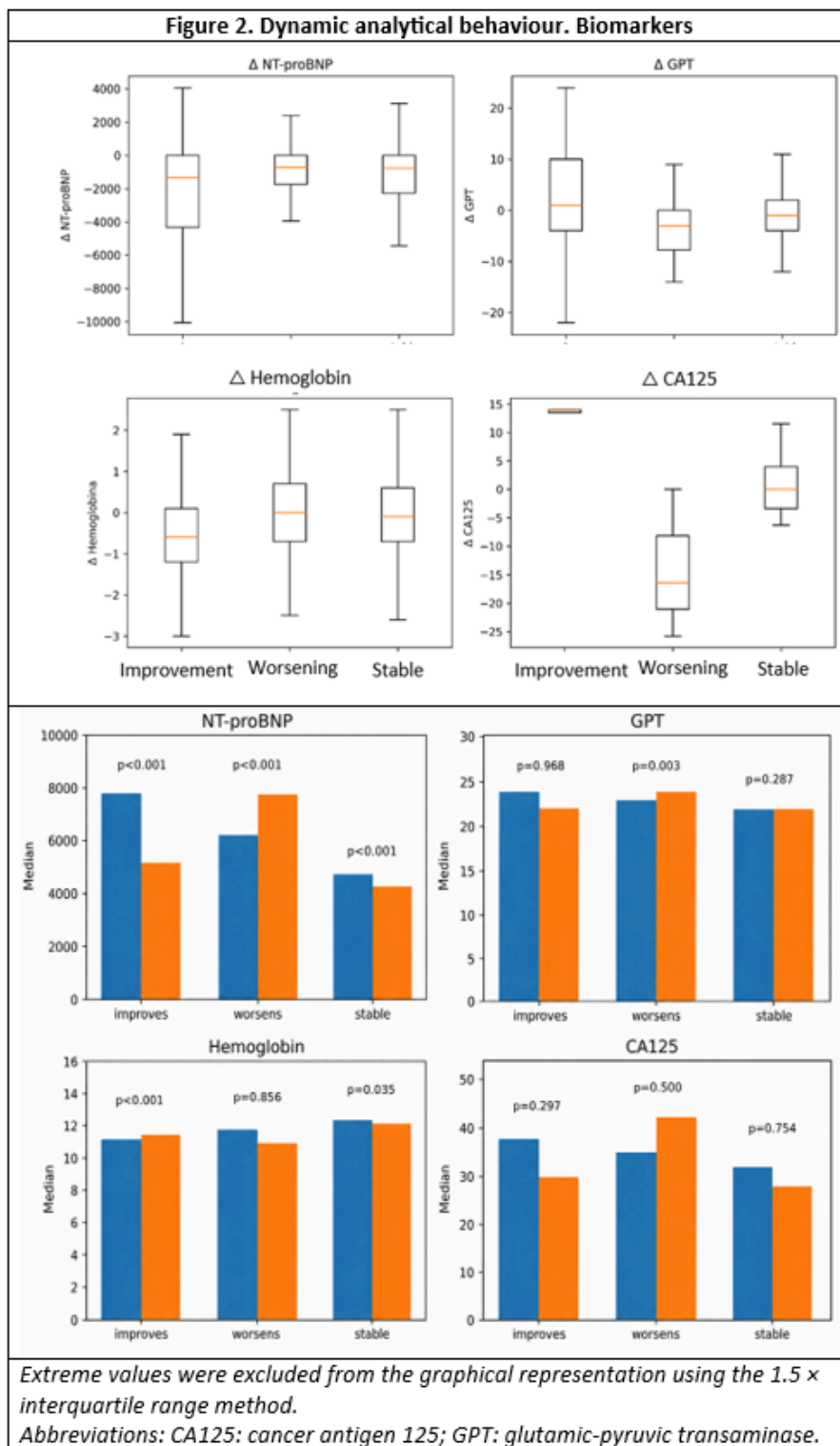
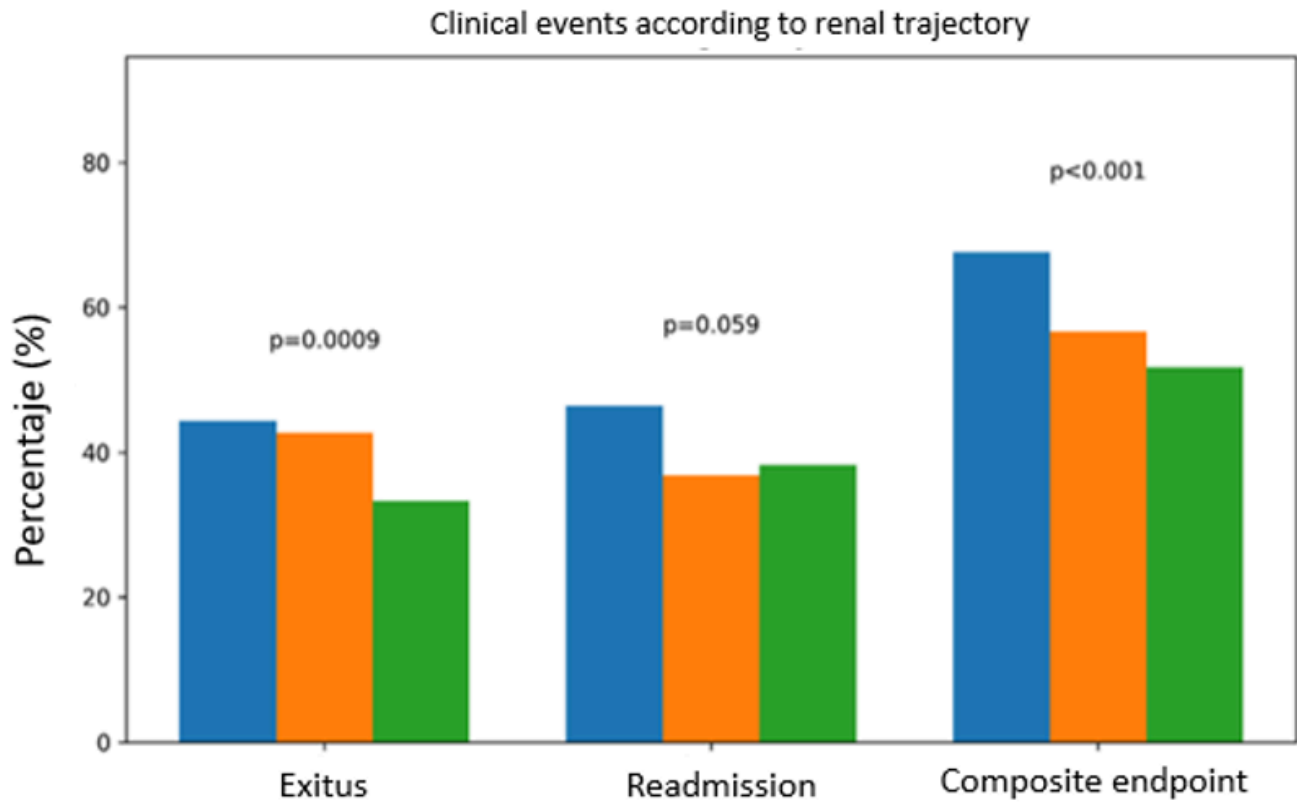


Figure 2

See image above for figure legend.

Figure 3. Mortality and readmission



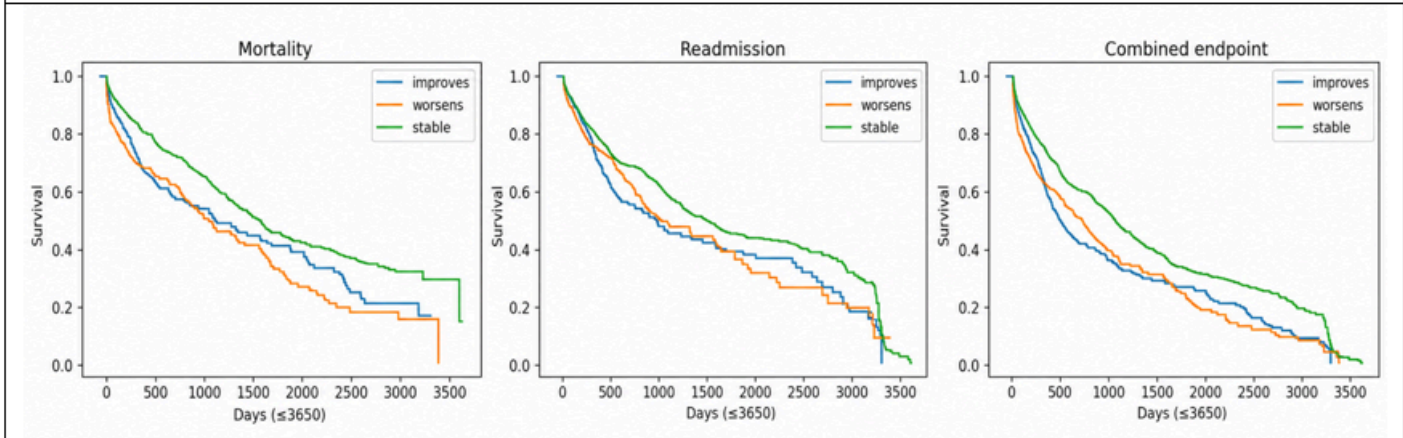
Compared using the chi-square test.

Blue: renal improvement. Orange: renal worsening. Green: intermediate changes.

Figure 3

See image above for figure legend.

Figure 4. Survival curves



Kaplan–Meier curves compared using the log-rank test.

Mortality: $p < 0.001$. (Improvement vs worsening: $p = 0.246$; Improvement vs stable: $p = 0.0013$; Worsening vs stable: $p < 0.001$)

Readmission: $p = 0.007$. (Improvement vs worsening: $p = 0.891$; Improvement vs stable: $p = 0.0073$; Worsening vs stable: $p = 0.023$)

Composite endpoint: $p < 0.001$. (Improvement vs worsening: $p = 0.841$; Improvement vs stable: $p < 0.001$; Worsening vs stable: $p < 0.001$)

Figure 4

See image above for figure legend.

Figure 5. Multivariable analysis

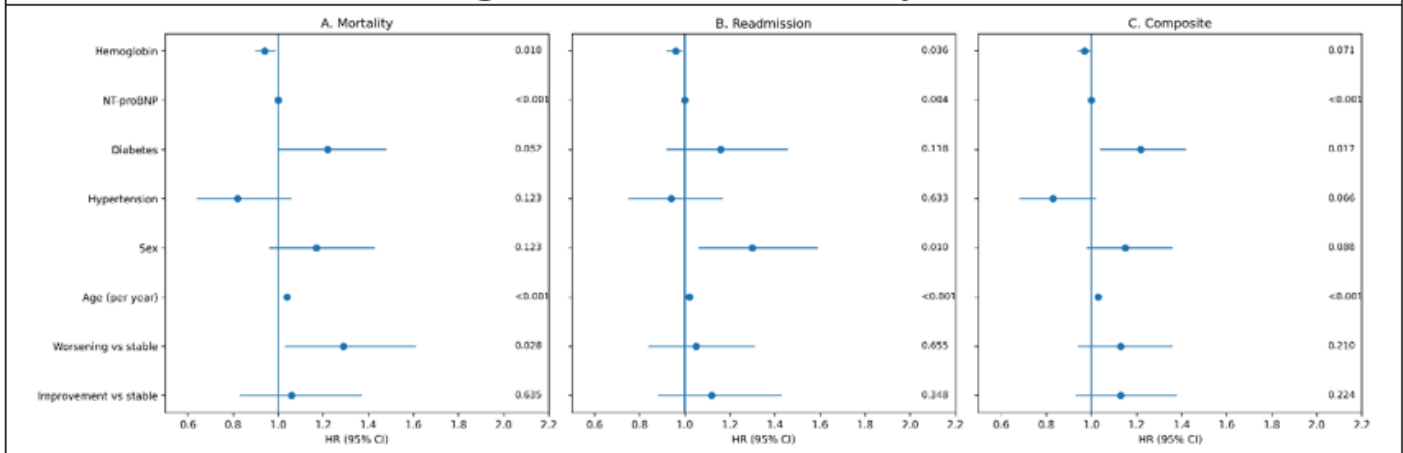


Figure 5

See image above for figure legend.

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- [GraphicalAbstract.jpg](#)