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Fluid balance trajectories and outcomes in acute heart failure complicated by acute kidney injury: Insights from the MIMIC-IV database

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Abstract

Background:

Acute heart failure (AHF) complicated by acute kidney injury (AKI) is associated with poor outcomes. Fluid management is central to treatment but remains challenging due to the bidirectional heart–kidney interaction. While most studies focus on static measures of fluid status, the prognostic value of dynamic fluid balance trajectories has not been fully elucidated.

Methods:

We performed a retrospective cohort study using the Medical Information Mart for Intensive Care IV (MIMIC-IV) database. Patients with AHF and AKI were identified, and their daily fluid balance during the first week of ICU admission was modeled using the elbow method and silhouette coefficient. Outcomes were assessed using Kaplan–Meier survival curves and Cox proportional hazards regression.

Results:

A total of 817 patients were included (median age 71.8 years; 52.1% male). Three distinct fluid balance trajectories were identified: sustained negative balance (SNB, 55.7%), positive-to-negative balance (SPNB, 40.6%), and sustained positive balance (SPB, 3.7%). The SPB group had significantly higher 28-day and 365-day mortality compared with the SNB and SPNB groups (30.0% vs. 15.0%; 53.3% vs. 35.1%; both $P < 0.05$). In multivariate analysis, the SNB group showed a 36% lower risk of death compared with the SPB group (HR 0.64, 95% CI 0.44–0.94; $P = 0.021$). Independent predictors of mortality included AKI stage 3 (HR 1.55; $P = 0.003$), lower body weight, hyponatremia, and elevated blood urea nitrogen. Patients with AKI stage 3 had a 28-day mortality of 26.8%, significantly higher than those with stage 1–2 AKI ($P < 0.001$).

Conclusions:

Dynamic fluid balance trajectories are strongly associated with outcomes in patients with AHF and AKI. Sustained positive balance confers the highest risk, whereas achieving or maintaining negative balance is linked with improved survival. These findings highlight the potential role of trajectory-based fluid monitoring to guide individualized fluid management.

Introduction

Acute heart failure (AHF) is a major global health burden, with profound pathophysiological abnormalities that substantially impair patient quality of life[1,2]. Acute kidney injury (AKI) frequently complicates AHF and is strongly associated with increased morbidity, mortality, and accelerated

progression to chronic kidney disease[3,4]. Approximately 20% of patients with acute cardiac conditions develop AKI or worsening renal function, with the prevalence rising to nearly 50% among patients admitted to cardiac intensive care units[5]. The coexistence of AHF and AKI often triggers a vicious cycle, in which deterioration of one organ exacerbates dysfunction of the other, resulting in dual organ failure and poor prognosis[6,7].

The concept of cardiorenal syndrome (CRS) was introduced to describe this bidirectional interaction between the heart and kidneys[8]. Acute CRS represents the most severe manifestation, in which therapeutic options for AHF are frequently limited by renal deterioration. Traditional markers, such as urine output and serum creatinine, are limited in prognostic utility: urine output is easily affected by diuretic use, while elevations in serum creatinine often occur late in the course of AKI and reflect established injury rather than early risk[9–11].

The hemodynamic interdependence between the heart and kidneys underscores the importance of precise fluid management in AHF with AKI. Renal perfusion is critically dependent on cardiac function, while cardiac preload is directly influenced by sodium and water balance maintained by the kidneys[12,13]. Accordingly, guideline-directed therapies for AHF emphasize careful monitoring of renal hemodynamics and volume status[14].

In this context, dynamic assessment of fluid balance trajectories may provide a more accurately and timely indicator of prognosis compared with static or single-point measurements. This approach can mitigate the confounding effects of medications on urine output and the lag inherent in creatinine-based detection[15]. Group-based trajectory modeling (GBTM), supported by statistical tools such as the elbow method and silhouette coefficient, allows the classification of patients into subgroups with similar longitudinal patterns of fluid balance, capturing the dynamic evolution of volume status[16].

Therefore, in the present study, we applied trajectory analysis to classify fluid balance patterns in patients with AHF complicated by AKI using data from the MIMIC-IV database. We further investigated the associations between these fluid balance trajectories and patient prognosis, as well as key clinical risk factors. Our goal was to provide novel insights into the prognostic implications of dynamic fluid monitoring and to support early identification and targeted management of high-risk patients with acute cardiorenal dysfunction[17].

Methods

Data Source

This study was conducted using the Medical Information Mart for Intensive Care IV (MIMIC-IV, version 2.0), a large, freely accessible, de-identified critical care database developed by the Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center, covering ICU admissions between 2008 and 2019[10,18]. The database includes patient demographics, laboratory test results, vital sign

measurements, medications, and clinical notes[19]. Access was obtained through PhysioNet (certification number: 64981500). Since all data were de-identified, informed consent was waived.

Study Population

We included adult patients (≥ 18 years) admitted to the ICU with a diagnosis of acute heart failure (ICD codes) who subsequently developed AKI during hospitalization. Exclusion criteria were: ICU stay < 72 hours; End-stage renal disease (ESRD); AKI present prior to or within 24 hours of ICU admission; 10% missing values for key variables; Multiple admissions (only the first admission considered). Only patients with ≥ 3 consecutive days of fluid balance records within the first 7 ICU days were included for trajectory analysis.

Definitions

Fluid balance (FB)

FB= (Total fluid input − Total fluid output)/Admission body weight (kg)[20].

Fluid input included oral intake, intravenous fluids (resuscitation, maintenance), blood products, drug infusions, and enteral/parenteral nutrition. Fluid output included urine, drainage, ultrafiltration, and estimated gastrointestinal loss. Insensible losses were not considered.

Acute kidney injury (AKI):

Defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 criteria[21]. Given the high rate of missing urine output data, AKI was determined solely based on serum creatinine (Scr). The lowest Scr value within 7 days before ICU admission was used as baseline.

Covariates

Potential confounders were selected based on prior literature[21,22] and biological plausibility. These included:

Demographics: age, sex;

Severity indices: Charlson Comorbidity Index (CCI), Systemic Inflammatory Response Syndrome (SIRS), SOFA score, APSIII score;

Laboratory parameters: WBC, hemoglobin, platelets, blood urea nitrogen, serum creatinine, troponin, lactate, sodium, potassium.

Outcomes

Primary outcomes were:

1)28-day mortality

2)1-year mortality

Secondary outcomes included:

1)Requirement of mechanical ventilation

2)Use of continuous renal replacement therapy (CRRT)

3)Requirement of vasoactive agents

Statistical Analysis

Missing data:

Variables with > 10% missingness were excluded. Remaining missing values were imputed using the K-nearest neighbor (KNN) algorithm[23,24].

Trajectory clustering:

Daily FB from day 3 to day 7 was used for clustering. The elbow method[25] and silhouette coefficient were applied to determine the optimal number of clusters, with three clusters identified as optimal (Supplementary Fig. 1). Fisher's discriminant analysis[26] was performed to validate cluster separability.

Comparisons:

Baseline characteristics among clusters were compared using ANOVA or Kruskal–Wallis tests for continuous variables, and χ^2 or Fisher's exact tests for categorical variables.

Survival analysis:

Kaplan–Meier curves were plotted for 28-day and 1-year mortality, with comparisons made using the log-rank test.

Software:

Statistical analyses were performed using R (v4.2.2) and Stata (v15.1). Figures were generated using GraphPad Prism 9.

Results

Baseline Characteristics

A total of 817 patients with acute heart failure complicated by AKI were included in the analysis. The median age was 71.8 years (interquartile range [IQR] 61.5–80.6 years), and 52.1% were male. The distribution of AKI severity was as follows: Stage 1: 200 patients (24.4%), Stage 2: 449 patients (55.0%), and Stage 3: 168 patients (20.6%). The overall mortality rates at 28 days and 365 days were 14.9% and 35.1%, respectively.

Clinical Characteristics of Survivors vs. Non-Survivors

We compared clinical and demographic characteristics between survivors and non-survivors at 365 days (Table 1). Significant differences were observed in age, gender, weight, WBC, platelet count, creatinine, BUN, troponin-T, BNP, sodium, hemoglobin, APACHE III, SOFA, SIRS, CCI, AKI stage, length of ICU and hospital stay, and treatment-related variables (mechanical ventilation, CRRT, vasopressor use). Mortality at both 28 and 365 days differed significantly between the groups. The fluid balance trajectories of survivors and non-survivors during the first week in the ICU are shown in Fig. 1. Notably, most non-survivors demonstrated a sustained negative fluid balance in the early ICU period.

Table 1
Baseline characteristics compared between survivors and nonsurvivors. at 365 days

Characteristics	Overall(n = 817)	Survivors (n = 530)	Nonsurvivors (n = 287)	P Value
Age, yr,Mean ± SD	70.3 ± 14.3	67.5 ± 14.5	75.4 ± 12.5	< 0.001
Gender[male,n(%)]	426(52.1)	287(54.2)	139(48.4)	0.124
Weight(kg)	81.3 ± 25.1	84.7 ± 26.3	75.2 ± 21.4	< 0.001
Wbc(k/uL)	12.4 ± 6.95	12.6 ± 6.08	13.0 ± 8.43	0.383
Platelet()	224 ± 113	221 ± 99.7	219 ± 122	0.783
Creatinine(mg/dL)	1.1 ± 0.717	1.04 ± 0.56	1.11 ± 0.53	0.099
Bun(mg/dL)	26.6 ± 18.1	23.4 ± 14.5	30.4 ± 17.6	< 0.001
Troponin-T	1.3 ± 3.38	1.38 ± 3.08	1.03 ± 2.49	0.244
Bnp	25.8 ± 15.9	8226 ± 8667	11355 ± 9960	0.066
Sodium	138 ± 5.11	138 ± 4.80	138 ± 5.62	0.051
Hb(g/dL)	10.7 ± 2.11	10.9 ± 2.15	10.3 ± 1.98	< 0.001
Apsiii	45.5 ± 16.5	43.5 ± 15.7	49.1 ± 17.4	< 0.001
Sofa	6.71 ± 3.54	6.32 ± 3.21	7.44 ± 3.99	< 0.001
Sirs	2.66 ± 0.861	2.64 ± 0.89	2.70 ± 0.82	0.295
CCI	5.66 ± 2.18	5.09 ± 1.91	6.71 ± 2.25	0.945
Aki_score	1	143(27.0)	57(19.9)	< 0.001
	2	303(57.2)	146(50.9)	
	3	84(15.8)	84(29.3)	
Length of stay[d,M(IQR)]				
Hospital	16.9 ± 17.7	16.3 ± 17.0	18.1 ± 19.0	0.157
ICU	7.8 ± 6.68	7.2 ± 5.7	8.9 ± 8.0	< 0.001
Treatments				
Mechventilator	478(58.5)	312(58.9)	166(57.8)	0.776

This table summarizes demographic data, comorbidities, disease severity scores, laboratory parameters, treatment interventions, and outcomes stratified by 365-day survival status. Statistically significant differences were observed in variables including age, gender, weight, WBC, platelet count, creatinine, BUN, troponin-T, BNP, sodium, hemoglobin, APACHE III, SOFA, SIRS, CCI, AKI score, length of stay (hospital and ICU), mechanical ventilation, CRRT, vasopressor use, and both 28-day and 365-day mortality (all P < 0.05).

Characteristics	Overall(n = 817)	Survivors (n = 530)	Nonsurvivors (n = 287)	P Value
Crrt	26(3.2)	6(1.1)	20(7.0)	< 0.001*
vasopressors	471(57.6)	310(58.5)	161(56.1)	0.509

This table summarizes demographic data, comorbidities, disease severity scores, laboratory parameters, treatment interventions, and outcomes stratified by 365-day survival status. Statistically significant differences were observed in variables including age, gender, weight, WBC, platelet count, creatinine, BUN, troponin-T, BNP, sodium, hemoglobin, APACHE III, SOFA, SIRS, CCI, AKI score, length of stay (hospital and ICU), mechanical ventilation, CRRT, vasopressor use, and both 28-day and 365-day mortality (all P < 0.05).

Identification of Fluid Balance Trajectories

Using the elbow method and silhouette score, the optimal number of clusters was determined to be k = 3 (Supplementary Material 1). Patients were categorized into three fluid balance trajectory subphenotypes (Fig. 2):Class 1: Shift from positive to negative balance (SPNB, n = 332, 40.6%);Class 2: Sustained negative balance (SNB, n = 455, 55.7%)Class 3: Sustained positive balance (SPB, n = 30, 3.7%)

Clinical Characteristics by Fluid Balance Trajectory

Univariate analysis revealed that ICU stay, hospital stay, weight, APACHE III, SOFA, SIRS scores, mean WBC, platelet counts, AKI stage, use of mechanical ventilation, CRRT, and vasopressors differed significantly among the three subphenotypes (Table 2).

Table 2
Baseline characteristics compared between three fluid balance trajectory.

Characteristics	Overall(n = 817)	Sustained negative balance(n = 455)	Sustained positive balance(n = 30)	Shift from positive to negative balance(n = 332)	P value
Age, yr,Mean ± SD	70.3 ± 14.3	70.8 ± 13.7	67.0 ± 16.7	69.9 ± 14.8	0.308
Gender	426(52.1)	229(50.3)	17(56.7)	180(54.2)	0.492
Weight	81.3 ± 25.1	85.0 ± 26.0	82.7 ± 27.7	76.1 ± 22.6	< 0.001
Wbc	12.4 ± 6.95	11.9 ± 6.44	16.3 ± 15.2	13.5 ± 6.50	< 0.001
Platelet	224 ± 113	229 ± 99	223 ± 171	208 ± 111	0.026
Creatinine	1.1 ± 0.717	1.08 ± 0.45	1.11 ± 0.84	1.04 ± 0.64	0.547
Bun	26.6 ± 18.1	26.8 ± 15.0	23.8 ± 12.4	24.7 ± 17.3	0.158
Troponin-T	1.3 ± 3.38	1.14 ± 2.47	0.27 ± 0.35	1.47 ± 3.44	0.158
Bnp	25.8 ± 15.9	9689 ± 9707	8256 ± 7569	8564 ± 8208	0.285
Sodium	138 ± 5.11	138 ± 5.33	137 ± 4.60	138 ± 4.85	0.817
Hemoglobin	10.7 ± 2.11	11.0 ± 2.23	10.3 ± 1.98	10.4 ± 1.92	0.671
Apsiii	45.5 ± 16.5	43.3 ± 14.6	54.7 ± 18.8	47.7 ± 18.2	< 0.001
Sofa	6.71 ± 3.54	5.89 ± 3.42	9.63 ± 4.62	7.58 ± 3.24	< 0.001
Sirs	2.66 ± 0.861	2.56 ± 0.87	3.10 ± 0.92	2.75 ± 0.83	< 0.001
Charison	5.66 ± 2.18	5.68 ± 2.13	5.67 ± 2.23	5.63 ± 2.24	0.945
Aki_score	1	108(23.7)	2(6.7)	90(27.1)	< 0.001
	2	257(56.5)	9(30.0)	183(55.1)	
	3	90(19.8	19(63.3)	59(17.8	
Length of stay[d,M(IQR)]					
Hospital	16.9 ± 17.7	16.1 ± 17.2	29.6 ± 34.9	16.9 ± 15.7	< 0.001
* Exact probability method (Fisher test)					
This table presents the distribution of demographic features, severity scores, laboratory values, and treatment interventions across the three trajectory subphenotypes: shift from positive to negative balance (SPNB), sustained negative balance (SNB), and sustained positive balance (SPB). Significant group differences were found in hospital and ICU length of stay, body weight, APACHE III, SOFA, SIRS, average WBC and platelet counts, AKI stage, mechanical ventilation, CRRT, and vasopressor support (all P < 0.05).					

Characteristics	Overall(n = 817)	Sustained negative balance(n = 455)	Sustained positive balance(n = 30)	Shift from positive to negative balance(n = 332)	P value
ICU	7.8 ± 6.68	7.3 ± 5.7	14.0 ± 14.4	7.9 ± 6.6	< 0.001
Treatment					
Mechventilator	478(58.5)	215(47.3)	22(73.3)	241(72.6)	< 0.001
Crrt	26(3.2)	11(2.4)	8(26.7)	7(2.1)	< 0.001*
vasopressors	471(57.6)	210(46.2)	24(80.0)	237(71.4)	< 0.001
* Exact probability method (Fisher test)					
This table presents the distribution of demographic features, severity scores, laboratory values, and treatment interventions across the three trajectory subphenotypes: shift from positive to negative balance (SPNB), sustained negative balance (SNB), and sustained positive balance (SPB). Significant group differences were found in hospital and ICU length of stay, body weight, APACHE III, SOFA, SIRS, average WBC and platelet counts, AKI stage, mechanical ventilation, CRRT, and vasopressor support (all P < 0.05).					

Survival Analysis

Kaplan–Meier survival analysis demonstrated significant differences in both 28-day and 365-day survival across the three groups (Figs. 4 and 5). The SPB group exhibited the highest mortality (365-day mortality 53.3%, 28-day mortality 30.0%), significantly higher than the other two groups (P < 0.05).

In Cox regression analysis, compared with the SNB group, the hazard ratios (HRs) for the SPNB and SPB groups at 365 days were 1.69 (95% CI 1.01–2.83, P = 0.044) and 0.78 (95% CI 0.61–0.999, P = 0.049), respectively. At 28 days, the HRs were 2.85 (95% CI 1.35–6.04, P = 0.006) and 0.98 (95% CI 0.63–1.52, P = 0.919). Full details are shown in Supplementary Material 2.

Further stratification by AKI stage showed that patients with AKI stage 3 had the poorest 28-day survival (mortality 26.8%, P < 0.05). Mortality rates for AKI stages 1 and 2 were ~ 4% and 12.5%, respectively, with no significant difference between them (Fig. 3).

Independent Predictors of Mortality

Multivariate Cox regression identified fluid balance trajectory (SNB vs. SPB) and AKI stage as independent predictors of 365-day mortality. After adjustment, the HRs were 0.64 (95% CI 0.44–0.94, P = 0.021) for the SNB group and 1.55 (95% CI 1.16–2.08, P = 0.003) for higher AKI stage. Other significant predictors included weight, serum sodium (first measurement), and average BUN, although their effect sizes were smaller (HRs: 0.979, 0.974, and 1.01, respectively). Detailed results are presented in Fig. 6 and Table 3.

Table 3
Multivariate Cox proportional hazards regression analysis of predictors of 365-day mortality

	HR	lower95	upper95	P value
cluster SPNB	0.796	0.322	1.97	0.621
cluster SNB	0.641	0.44	0.936	0.021
los_hospital	0.992	0.979	1	0.196
weight	0.979	0.97	0.998	< 0.001
apsiii	1.01	0.997	1.02	0.167
sofa	1.06	0.981	1.15	0.137
sirs	1.05	0.84	1.3	0.684
sodium	0.974	0.95	1	0.05
wbc	1.01	0.951	1.06	0.837
platelet	1	0.998	1.01	0.321
bun	1.01	1.01	1.02	< 0.001
troponin_t	0.965	0.903	1.03	0.291
aki_score	1.55	1.16	2.08	0.003
mechventilator	1.12	0.714	1.75	0.627
crrt	1.16	0.458	2.94	0.754
vasopressors	0.746	0.476	1.17	0.202

This table summarizes independent predictors of 365-day mortality after adjustment for confounders. Both fluid balance trajectory (SNB vs. SPB) and AKI score remained strong predictors of long-term outcomes. Additional significant predictors included body weight, sodium level, and average BUN, though their hazard ratios were of smaller magnitude.

Discussion

In this study, we applied the elbow method combined with the silhouette coefficient[27] to classify dynamic fluid balance trajectories in 817 ICU patients with acute heart failure complicated by AKI. Three distinct patterns were identified: sustained positive balance (SPB), sustained negative balance (SNB), and positive-to-negative balance (SPNB). Patients in the SPB group demonstrated significantly higher short- and long-term mortality, and AKI grade 3 emerged as an independent predictor of death. These findings indicate that persistent positive fluid balance carries important prognostic implications in acute cardiorenal syndrome (CRS) and highlight the critical need to refine fluid management strategies in this population.

Although fluid management has long been a cornerstone of critical care[28], most prior studies have not specifically addressed CRS trajectories. Our work provides a novel framework to objectively classify fluid balance patterns and links these patterns to outcomes. Consistent with prior research in sepsis and post-cardiac arrest patients, we observed that positive cumulative fluid balance was associated with worse survival[22,29]. Importantly, studies in septic shock have also suggested a nonlinear relationship between fluid balance and mortality, with both inadequate and excessive resuscitation being harmful[20,30]. The present results extend this concept to CRS, showing that persistent overload worsens prognosis, while early transition to a negative balance may mitigate adverse effects.

The differences among subtypes provide additional clinical insight. Patients in the SNB trajectory had lower APACHE III and AOFAS scores, reflecting milder illness and more favorable outcomes. By contrast, patients in the SPB group not only had higher severity scores but also required greater use of mechanical ventilation, vasoactive drugs, and CRRT, all of which indicate severe clinical status and highlight the difficulty of fluid removal in unstable patients. The SPNB group showed intermediate outcomes, supporting the hypothesis that appropriate adjustment of fluid strategy during the course of illness can improve survival. These findings align with sepsis studies showing that fluid accumulation beyond the initial resuscitation phase is particularly detrimental, and timely correction may reduce risk[31,32]. Notably, the interaction analysis revealed a synergistic effect between AKI grade 3 and SPB trajectory, reinforcing the idea that fluid overload and renal dysfunction amplify each other, thereby accelerating progression to multiorgan failure[33,34].

From a pathophysiological perspective, these results are consistent with the evolving understanding of CRS. While reduced cardiac output and renal hypoperfusion were once considered the primary drivers of renal dysfunction, accumulating evidence highlights the equally important roles of venous congestion and maladaptive neurohormonal activation[35–37]. These mechanisms promote sodium and water retention, increase cardiac preload, and exacerbate both renal and cardiac injury[37–39]. Our findings strengthen this paradigm by linking sustained positive balance with poor outcomes, suggesting that congestion, rather than hypoperfusion, may represent the dominant mechanism of harm in acute CRS[40,41].

The clinical implications of our study are considerable. Dynamic modeling of fluid balance trajectories provides superior prognostic value compared with static measurements and enables earlier recognition of high-risk patients. Continuous monitoring of fluid balance trends rather than single-point evaluation may allow clinicians to identify patients drifting into a persistently positive balance and intervene accordingly. In addition, simple and accessible laboratory markers such as serum sodium and urine protein were independently associated with mortality, offering pragmatic tools for early risk stratification. Taken together, these findings may help guide individualized fluid management strategies, optimize the timing of diuretics or CRRT, and ultimately improve outcomes in this high-risk population[42–44].

Nevertheless, several limitations must be acknowledged. This was a single-center retrospective analysis, which may limit generalizability, and the small proportion of SPB patients reduced the statistical power of subgroup analyses. Important clinical details such as diuretic regimens, echocardiographic data, and hemodynamic monitoring were not available, precluding deeper mechanistic exploration. Additionally, biomarkers of renal injury such as NGAL were lacking, which restricted evaluation of subclinical kidney damage. These limitations underscore the need for prospective multicenter studies integrating standardized fluid protocols, advanced monitoring, and biomarker-based stratification to further validate our findings[45].

In conclusion, this study demonstrates that sustained positive fluid balance trajectories are strongly associated with adverse outcomes in acute CRS, with AKI grade 3 representing an independent and synergistic predictor of mortality. By introducing trajectory-based analysis, we provide novel dynamic prognostic indicators and identify a potential therapeutic window for individualized fluid adjustment. Future research should focus on defining threshold values for fluid trajectories, tailoring management strategies to AKI subtypes, and integrating biomarkers to enable precision fluid therapy in critically ill patients with CRS[45].

Use of artificial intelligence

No artificial intelligence was used during preparation of this manuscript.

Declarations

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CRediT authorship contribution statement

Weixiao Feng: Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization. Jiaming Gao: Writing – review & editing, Methodology, Formal analysis, Data curation, Conceptualization. Weiwei Feng: data curation & investigation. Jinyuan Mei: data curation & investigation. Yujie Song: Methodology & Validation, Visualization. Shuixiang Deng: project administration & resources. Yu He: Resources & Software. Shengjie Feng: Writing – review & editing, Supervision, Conceptualization, project administration.

Declaration of competing interest

There is no conflict of interest.

Data availability statement

The datasets generated during the current study are available in the mimic IV. All data files are provided in excel format and the analysis code is licensed under MIT License. Alternatively, the data are available from the corresponding author upon reasonable request.

Author Contribution

Weixiao Feng: Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization. Jiaming Gao: Writing – review & editing, Methodology, Formal analysis, Data curation, Conceptualization. Weiwei Feng: data curation & investigation. Jinyuan Mei: data curation & investigation. Yujie Song: Methodology & Validation, Visualization. Shuixiang Deng: project administration & resources. Yu He: Resources & Software. Shengjie Feng: Writing – review & editing, Supervision, Conceptualization, project administration.

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Figures

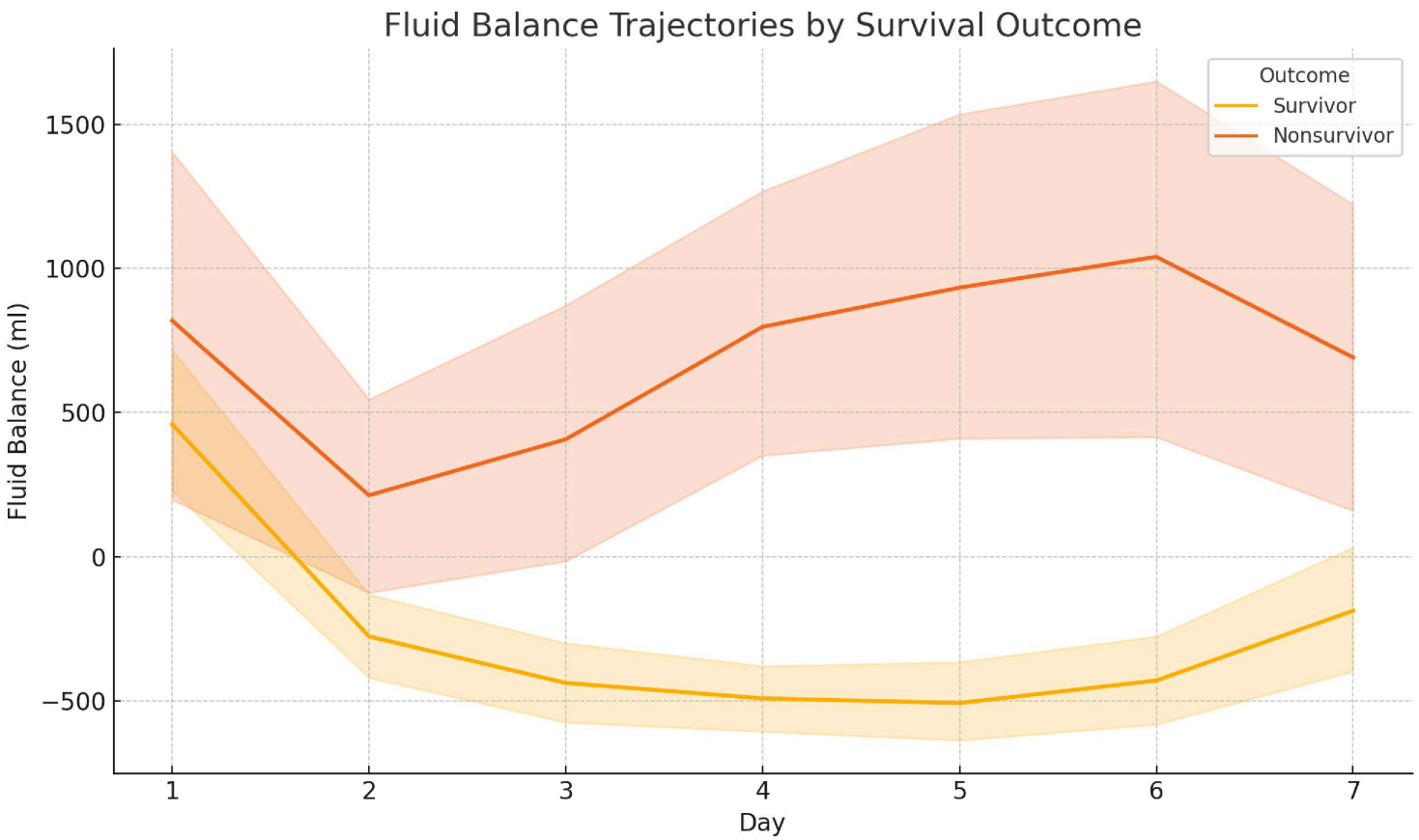


Figure 1

Dynamic changes in fluid balance between survivors and non-survivors

This figure illustrates the dynamic changes in cumulative fluid balance during the first week of ICU stay, stratified by survival status at 365 days. Survivors generally maintained a more stable fluid balance, while non-survivors tended to present with a persistent negative balance throughout the observation period.

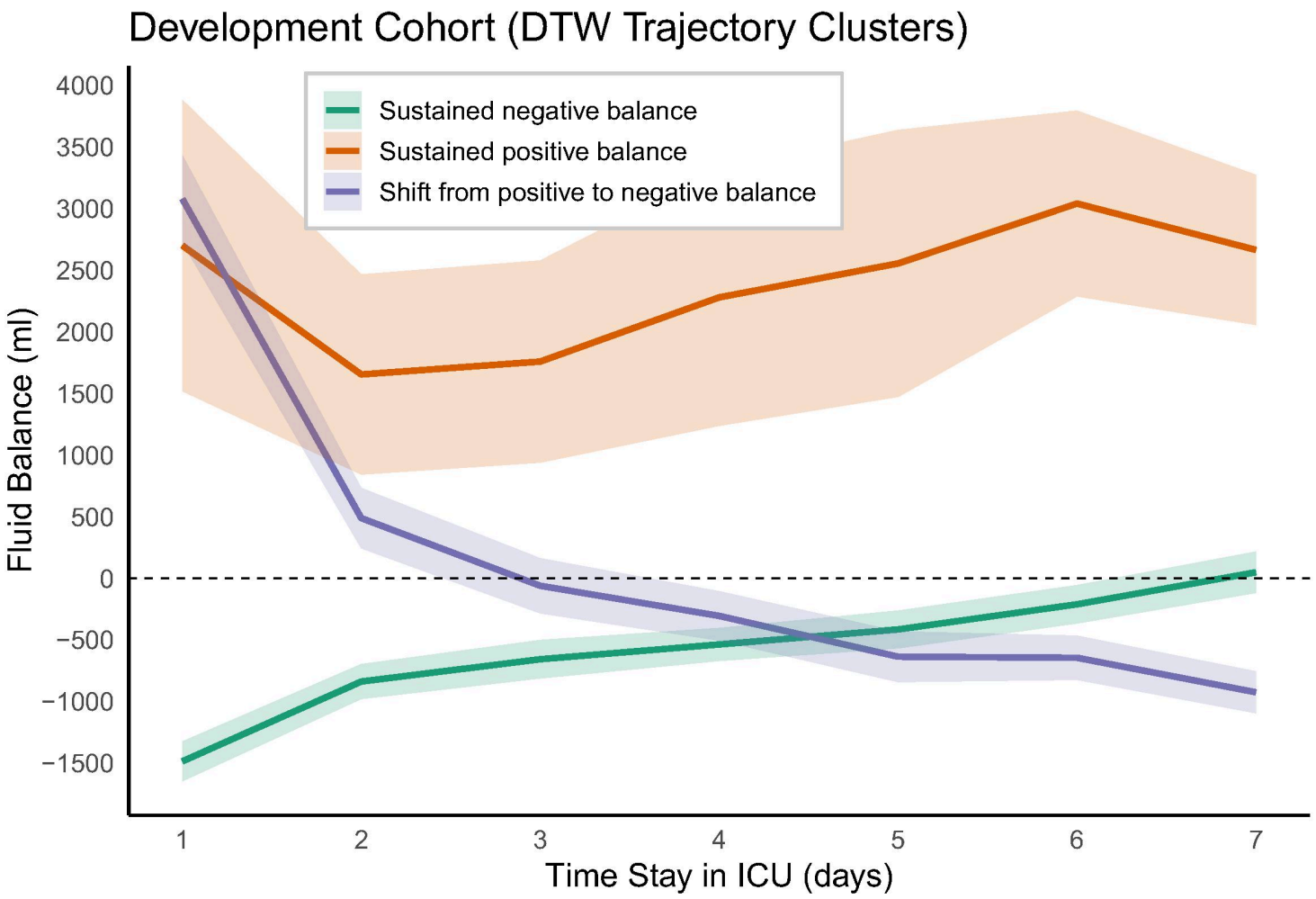


Figure 2

Fluid balance trajectory classification results

Latent class trajectory modeling identified three distinct subphenotypes of fluid balance over the first week: Class 1 (shift from positive to negative balance, SPNB), Class 2 (sustained negative balance, SNB), and Class 3 (sustained positive balance, SPB). The trajectories represent the mean fluid balance with 95% confidence intervals.

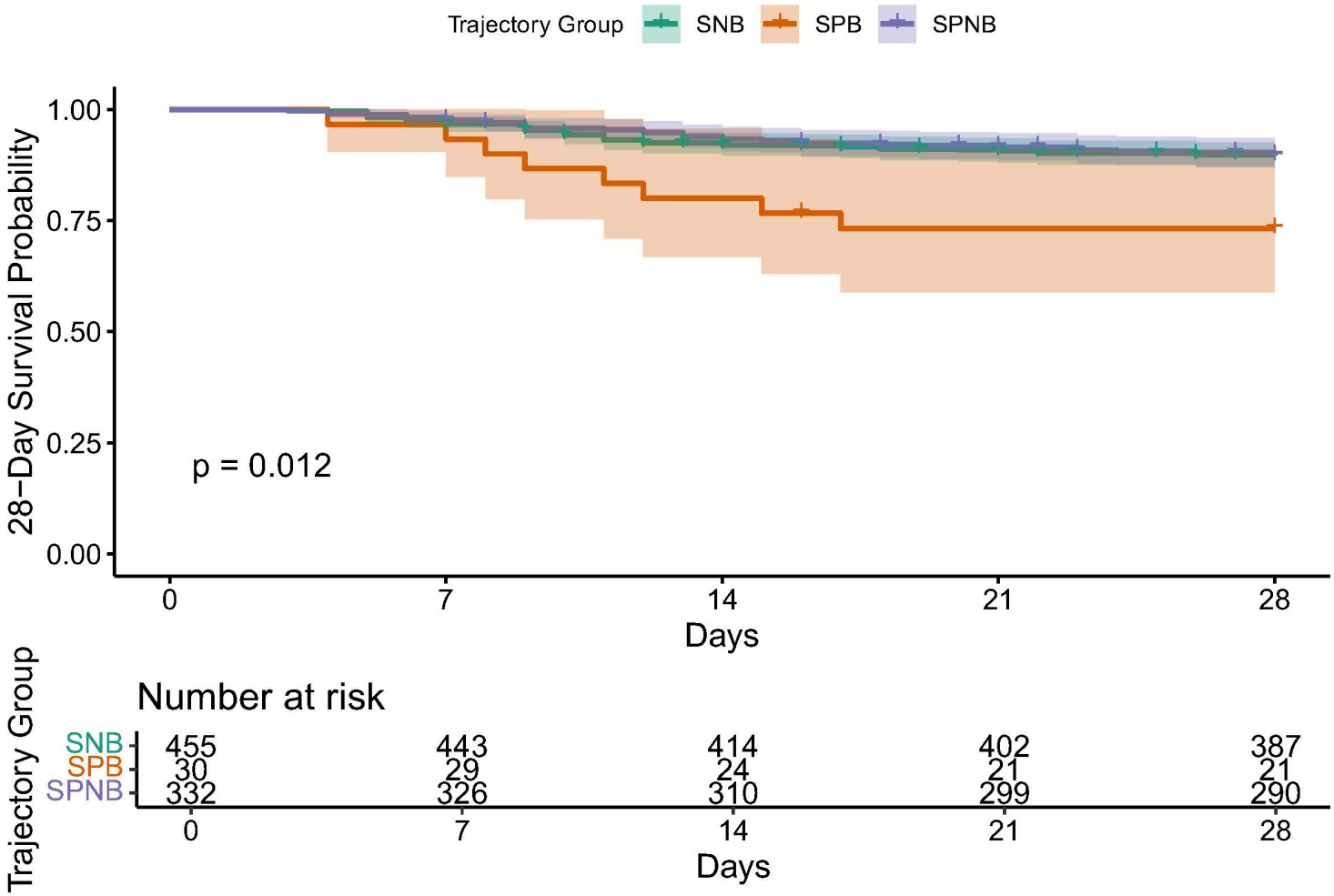


Figure 3

Kaplan–Meier survival curves of different trajectory groups

Kaplan–Meier curves display 365-day survival probabilities among patients with different fluid balance trajectories. Patients in the SPB group showed the poorest survival, while those in the SNB group had the most favorable outcomes (log-rank P < 0.05).

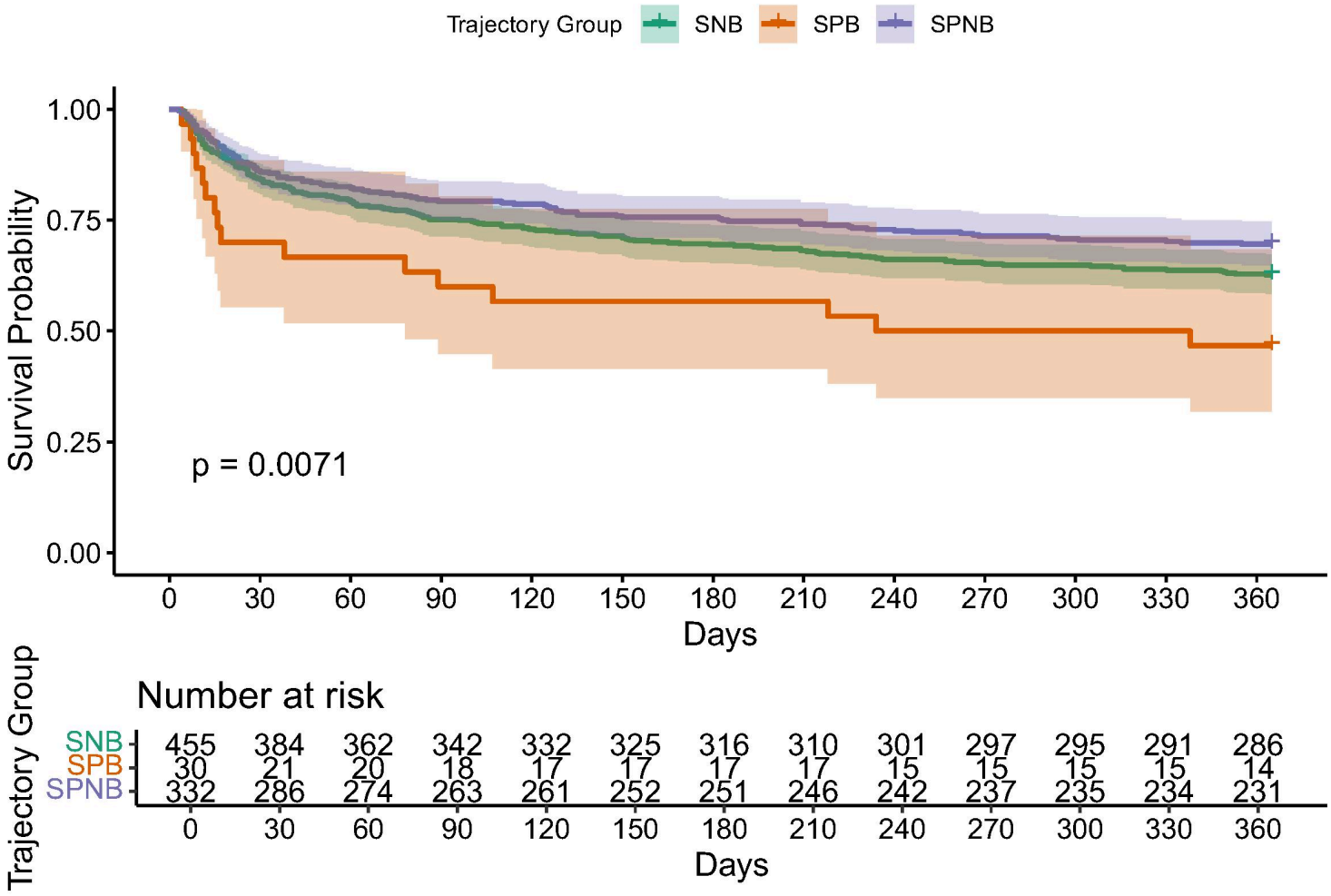


Figure 4

28-day survival curves stratified by AKI stages

Kaplan–Meier survival analysis stratified by AKI stages demonstrates significantly lower 28-day survival among patients with AKI stage 3 compared to stages 1 and 2 (log-rank $P < 0.05$).

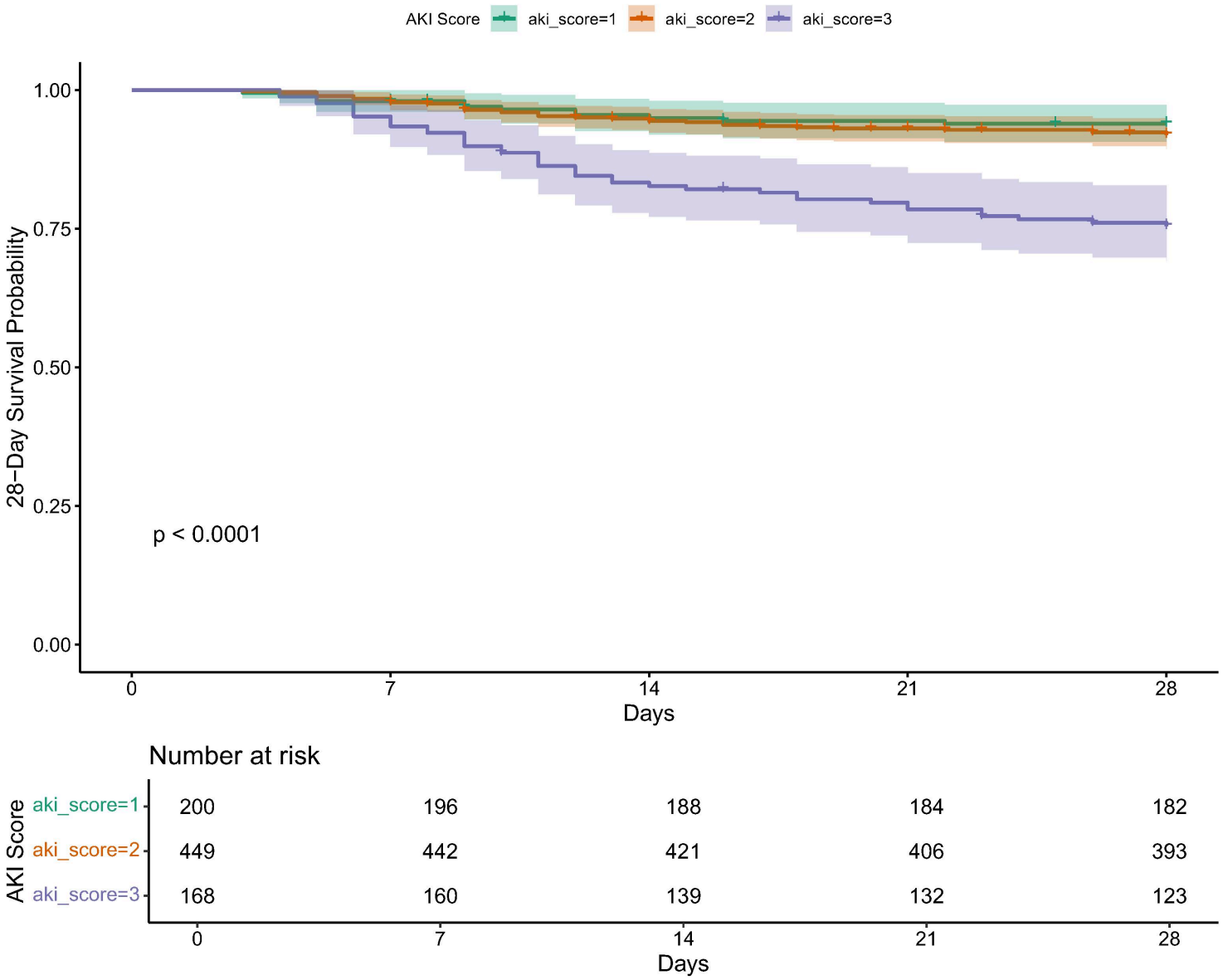


Figure 5

Forest plot of Cox proportional hazards regression analysis

Multivariate Cox regression analysis identifies independent predictors of 365-day mortality. The forest plot highlights fluid balance trajectory (SNB vs. SPB) and AKI stage as the strongest prognostic factors, while other covariates such as weight, sodium, and BUN showed smaller but significant contributions.

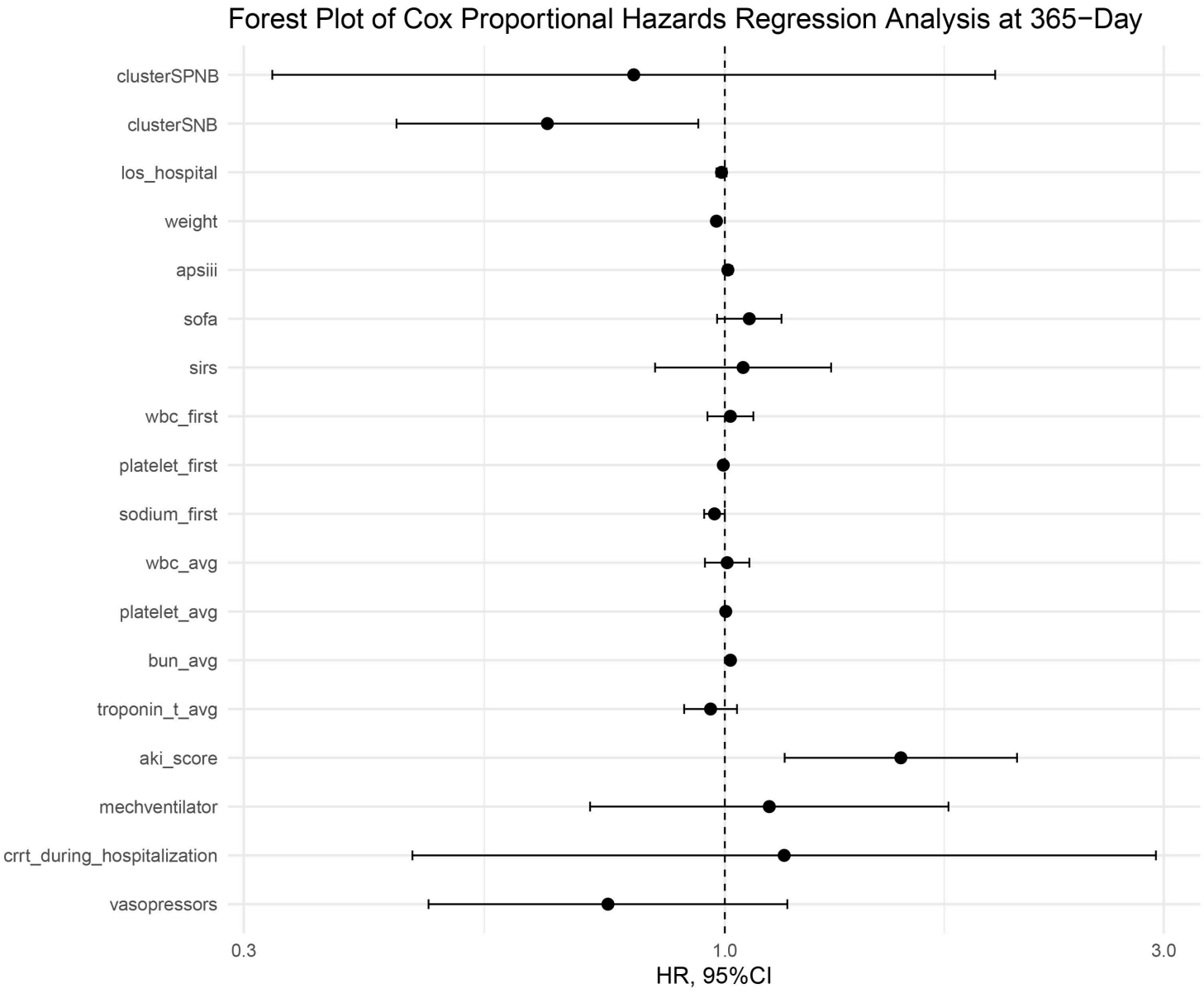


Figure 6

Legend not included with this version.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Supply1.pdf
- Supply2.xlsx
- CRSdata.xlsx