eGFR and Outcomes in Patients with Acute Decompensated Heart Failure with or without Elevated BUN

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Abstract

Background and objectives In patients with heart failure, the association of renal dysfunction and BUN levels with outcomes is unclear. The aim of our study was to investigate the association between the eGFR at discharge and outcomes in patients with heart failure with or without an elevated BUN level at discharge.

Design, setting, participants, & measurements Of 4842 patients enrolled in the Acute Decompensated Heart Failure Syndromes Registry, 4449 patients discharged alive after hospitalization for acute decompensated heart failure were investigated to assess the association of eGFR in the context of serum BUN level at discharge with all-cause mortality. The enrolled patients were divided into four groups on the basis of the discharge levels of eGFR (<45 or ≥ 45 ml/min per 1.73 m^2) and BUN (≥ 25 or <25 mg/dl). The median follow-up period after discharge was 517 (381–776) days.

Results The all–cause mortality rate after discharge was 19.1%. After adjustment for multiple comorbidities, an eGFR<45 ml/min per 1.73 m^2 was associated with a significantly higher risk of all-cause mortality in patients with a BUN≥ 25 mg/dl (hazard ratio, 1.58; 95% confidence interval, 1.33 to 1.88; $P<0.001$) but not in patients with a BUN<25 mg/dl (hazard ratio, 0.97; 95% confidence interval, 0.76 to 1.26; $P=0.84$) relative to those with an eGFR≥ 45 ml/min per 1.73 m^2 and a BUN<25 mg/dl. Among patients with an eGFR≥ 45 ml/min per 1.73 m^2, a BUN≥ 25 mg/dl was associated with a significantly higher risk of all-cause mortality than a BUN<25 mg/dl (hazard ratio, 1.34; 95% confidence interval, 1.04 to 1.73; $P=0.02$).

Conclusions We showed that elevation of BUN at discharge significantly modified the relation between eGFR at discharge and the risk of all-cause mortality after discharge, suggesting that the association between eGFR and outcomes may be largely dependent on concomitant elevation of BUN.

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Introduction

Indicators of renal function, such as serum creatinine and eGFR, are independent prognostic factors in patients with heart failure (HF) (1–7). However, serum creatinine is not sensitive enough to detect small decreases in GFR (8–10). Likewise, BUN is not as reliable a marker of renal function as the GFR, because urea levels are affected by protein intake, catabolism, and tubular reabsorption (2,11,12). However, on the basis of renal handling of urea, BUN more closely reflects neurohormonal activation than changes of creatinine or the GFR (1,11,12). Accordingly, baseline elevation of BUN is a sign of severe HF and has been shown to be a better prognostic indicator of mortality than the eGFR (12,13). Moreover, previous reports have suggested that an elevated BUN level combined with normal to mildly reduced renal function at admission is correlated with mortality in patients who have critical illness (14–17). However, the association of the BUN level in the compensated state with postdischarge outcomes in patients with HF is unclear. Recently, it has been suggested that patients hospitalized for HF are recognized to be at substantially higher risk for mortality in the period early after discharge, although the majority of patients with HF are judged to be stable at discharge (18,19). Therefore, we evaluated the association between eGFR at discharge and subsequent mortality in patients with acute decompensated HF with or without an elevated BUN level at discharge.

Materials and Methods

Study Design and Data Collection

The Acute Decompensated Heart Failure Syndromes (ATTEND) Registry is a nationwide hospital–based prospective, observational, multicenter cohort study that accumulated data on patients with acute decompensated HF admitted to 53 hospitals in all regions of Japan between April 1, 2007 and December 31, 2011. Patients are enrolled at their first admission

and then followed, so that data collection is patient based and not event based. The design and methods of this study as well as the patient profile have been described previously (20). Briefly, the ATTEND Registry Study has been designed to clarify the profile of patients with acute decompensated HF, including their demographic and clinical characteristics, treatment, in-hospital mortality, and postdischarge morbidity or mortality. Management of acute decompensated HF is not specified, and treatment is selected by the attending physician. The information obtained about each registered patient includes demographic data, medical history, baseline characteristics, initial evaluation, treatment, procedures, hospital course, and disposition. This study was conducted in accordance with the principles of the Declaration of Helsinki. Institutional review board approval was obtained at each participating medical center before the study, and all patients provided written informed consent at enrollment. The end point classification committee (two experienced cardiologists who were not study investigators) reviewed all end point data and asked the primary physician to confirm the cause of death if any problems were encountered.

Patients and Definitions

The ATTEND Registry Study enrolls consecutive eligible patients with a discharge diagnosis of acute decompensated HF. Inpatients with acute decompensated HF who meet the modified Framingham criteria are eligible for entry into the registry (21). Patients ages <20 years old, those with acute coronary syndrome, and others considered unsuitable for the study by their attending physicians were excluded. In addition, patients receiving hemodialysis were excluded from this analysis. Patients only qualify for enrollment if acute decompensated HF is the primary reason for admission. In this study, a preserved ejection fraction (EF) was defined as a documented EF >40% at admission, whereas a reduced EF was defined as an EF≤40% at admission. Patients were divided into four groups on the basis of their eGFR and BUN values at discharge from the hospital. To define renal insufficiency in the patients with HF, we compared three different formulas for estimating GFR, which were the Japanese–modified eGFR equation: $194\times$ (serum creatinine$^{-1.094}$) $\times$ (age$^{-0.287}$) $\times$ (0.739 for women); the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation: for men, $141\times$ minimum(creatinine/0.9, 1)$^{-0.411}\times$ maximum (creatinine/0.9, 1)$^{-1.209}\times 0.933^{age}\times 1.159$ (if black) and for women, $141\times$ minimum(creatinine/0.7, 1)$^{-0.329}\times$ maximum(creatinine/0.7, 1)$^{-1.209}\times 0.933^{age}\times 1.018\times 1.159$ (if black); and the simplified Modification of Diet in Renal Disease-4 (MDRD-4) equation: $186.3\times$ creatinine$^{-1.154}\times$ age$^{-0.203}\times 0.742$ (if a woman) $\times 1.212$ (if black) (22–25). Receiver–operating characteristics (ROC) analysis revealed that the Japanese–modified eGFR equation (area under the receiver–operating characteristics curve [AUC] =0.62) performed better for predicting all-cause death after discharge than the simplified MDRD-4 formula (AUC=0.61), whereas the Japanese–modified eGFR equation and the CKD-EPI equation (AUC=0.62) showed similar discrimination in predicting all-cause mortality after discharge. Therefore, in this study, we elected to use the Japanese–modified eGFR equation. Next, to define the optimum cutoff values of eGFR and BUN at discharge for predicting all-cause mortality after discharge, ROC analysis with the Youden index was performed. As shown in Figure 1, this analysis showed that the AUC for the eGFR at discharge was 0.62 and that a cutoff value of 43 ml/min per 1.73 m^2 had a sensitivity of 58% and a specificity of 63% for predicting all-cause mortality. However, ROC analysis showed that the AUC for BUN at discharge was 0.64 and that a cutoff value of 25 mg/dl had a sensitivity of 61% and a specificity of 61% for predicting all-cause mortality. Accordingly, the postdischarge mortality was evaluated in subgroups stratified by an eGFR at discharge ≥45 or <45 ml/min per 1.73 m^2 and a BUN level at discharge <25 or ≥25 mg/dl. That is, patients assigned to group 1 had an eGFR≥45 ml/min per 1.73 m^2 and a BUN<25 mg/dl, whereas group 2 had an eGFR≥45 ml/min per 1.73 m^2 and a BUN≥25 mg/dl, group 3 had an eGFR<45 ml/min per 1.73 m^2 and a BUN<25 mg/dl, and group 4 had an eGFR<45 ml/min per 1.73 m^2 and a BUN≥25 mg/dl. These four groups were used to investigate the association of eGFR at discharge with postdischarge mortality among

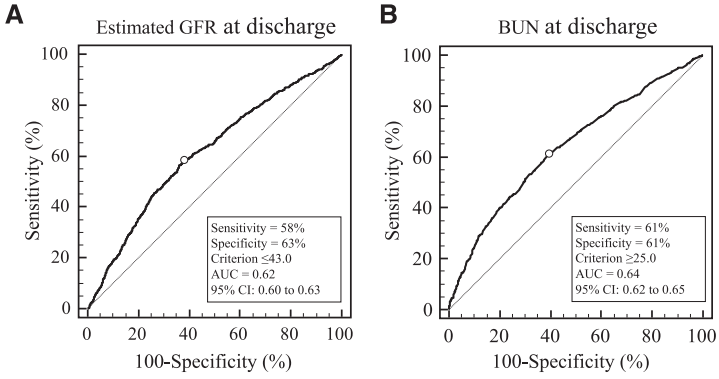


Figure 1. | Receiver-operating characteristic curves of eGFR at discharge and BUN at discharge predicting the all-cause mortality after discharge in patients with acute decompensated heart failure (HF). (A) Receiver-operating characteristic curve of eGFR at discharge. (B) Receiver-operating characteristic curve of BUN at discharge. Circles indicate the optimum cutoff values. AUC, area under the receiver–operating characteristic curve; 95% CI, 95% confidence interval.

Table 1. Baseline characteristics of all patients and four groups stratified by eGFR and BUN at discharge

Variables	Total	eGFR≥45 ml/min per 1.73 m^2		eGFR<45 ml/min per 1.73 m^2		*P* Value
		BUN<25 mg/dl (Group 1)	BUN≥25 mg/dl (Group 2)	BUN<25 mg/dl (Group 3)	BUN≥25 mg/dl (Group 4)	
No. of patients	4449	1981	418	569	1481	
Age, yr	72.6±13.8	68.4±14.8	73.0±13.3	74.9±12.4	77.1±11.0	<0.001
Sex, % men	58.3	61.5	67.2	48.2	55.2	<0.001
Etiology, %						
Ischemic	30.6	25.3	24.9	38.1	36.5	<0.001
Hypertensive	18.0	17.4	15.3	19.0	19.3	0.20
Valvular	18.9	17.3	23.2	17.2	20.5	0.01
Idiopathic dilated	13.2	16.2	14.8	9.5	10.1	<0.001
Medical history, %						
Prior hospitalization for heart failure	35.3	26.0	36.6	34.1	47.7	<0.001
Hypertension	69.5	64.3	62.7	76.1	76.0	<0.001
Dyslipidemia	37.3	35.0	30.1	43.3	40.4	<0.001
Diabetes	33.7	30.8	28.9	36.4	38.0	<0.001
Chronic obstructive pulmonary disease	11.8	11.1	16.5	10.2	12.0	0.01
Stroke	13.6	11.8	14.4	12.7	16.1	0.01
Atrial fibrillation at discharge, %	31.1	29.6	39.0	30.4	31.3	<0.001
Reduced ejection fraction (≤40%), %	53.4	55.2	58.9	49.9	50.7	0.003
Medications at discharge, %						
Loop diuretic	82.6	77.2	90.2	78.9	89.1	<0.001
Spironolactone or eplerenone	49.0	53.9	60.3	46.6	40.2	<0.001
Angiotensin–converting enzyme inhibitor	31.2	34.0	37.8	32.3	25.3	<0.001
Angiotensin II receptor blocker	46.6	44.3	43.3	47.6	50.2	0.003
β-Blocker	68.1	67.9	67.0	73.1	66.7	0.04
Body mass index, kg/m^2	21.6±4.2	21.9±4.4	20.4±4.3	22.0±4.4	21.3±3.8	<0.001
Systolic BP at discharge, mmHg	114.8±17.6	113.4±16.7	110.1±15.8	118.3±17.6	116.6±18.8	<0.001
Diastolic BP at discharge, mmHg	63.9±14.1	64.4±11.3	62.1±11.1	65.9±11.8	63.1±18.2	<0.001
Heart rate at discharge, beats per minute	70.2±11.9	70.9±12.0	71.2±11.6	70.4±11.8	69.0±11.7	<0.001
BUN at discharge, mg/dl	27.1±16.4	16.6±4.2	31.4±14.6	19.6±4.1	42.8±17.2	<0.001
Serum creatinine at discharge, mg/dl	1.35±1.20	0.83±0.21	0.95±0.19	1.67±2.04	2.03±1.33	<0.001
eGFR at discharge, ml/min per 1.73 m^2	49.2±23.2	67.5±18.2	56.9±12.9	35.3±8.7	27.8±10.0	<0.001
Serum sodium at discharge, mEq/L	138.4±4.0	138.9±3.7	137.4±3.9	139.0±3.6	137.8±4.4	<0.001
Hemoglobin at discharge, g/dl	12.0±2.3	12.8±2.2	12.5±2.3	11.5±2.0	11.1±2.1	<0.001
NYHA functional class at discharge, %						
I	94.3	96.0	91.4	95.7	92.4	<0.001
II	5.7	4.0	8.6	4.3	7.6	<0.001

Values are the means±SDs, proportions (percentages), or medians (interquartile ranges). NYHA, New York Heart Association.

patients with or without an elevated BUN level at discharge who were discharged alive after hospitalization for acute decompensated HF. The primary end point was all-cause mortality after discharge, and the secondary end point was cardiac death after discharge.

Statistical Analyses

Data are presented as means (SDs), medians with interquartile ranges, or proportions. ROC curves with Youden index were constructed to identify the optimum cutoff values of the eGFR and BUN at discharge for predicting postdischarge mortality. One-way ANOVA was used for a four-group comparison of continuous variables with a normal distribution, whereas the Kruskal–Wallis *H* test was used for skewed continuous variables or discrete variables. The chi-squared test was used to compare nominal-scale variables. Cumulative probabilities of event curves were estimated by the Kaplan–Meier analyses with a log-rank test. Univariate and multivariate Cox proportional hazards regression analyses were performed to assess the association of candidate variables with postdischarge mortality. The multivariate model included variables that were predictors of postdischarge mortality on univariate analysis as well as other factors known to influence postdischarge mortality, including age, sex, ischemic etiology, hypertension, diabetes, readmission for HF, left ventricular EF, atrial fibrillation, body mass index, systolic BP, serum sodium, hemoglobin, use of loop diuretic, use of spironolactone, use of angiotensin–converting enzyme inhibitor, and use of angiotensin II receptor blocker at discharge. The proportional hazards assumption was confirmed by the log (log survival function), and the influence of profile, interaction, and multicollinearity in these models was examined by regression diagnostic analysis. A P value of <0.05 (two tailed) was considered to indicate statistical significance. An independent statistical data center (STATZ Institute, Inc., Tokyo, Japan) performed all analyses using SAS system, version 9.3 software (SAS Institute Inc., Cary, NC).

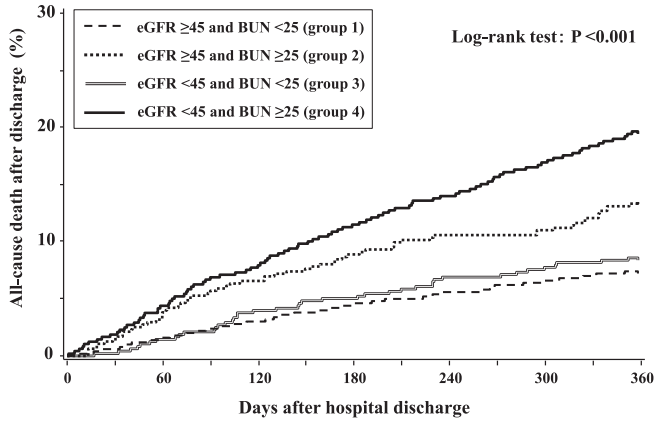
Results

Baseline Characteristics of All Patients and Groups Stratified by the eGFR and BUN

Among 4842 patients entered into the ATTEND Registry, 4530 patients were discharged alive after hospitalization for acute decompensated HF. Of these 4530 patients, 4449 (99.2%) patients were included in this analysis for whom data on the eGFR and BUN at discharge as well as postdischarge follow-up data were available (Table 1). The median follow-up period after discharge was 517 (381–776) days. The all–cause mortality rate and cardiac death rate after discharge were 19.1% and 10.4%, respectively. The baseline clinical characteristics of four groups of patients stratified by eGFR and BUN at discharge are shown in Table 1. There were no significant differences among the four groups with respect to hypertensive etiology and use of β-blockers at discharge. Patients with an eGFR$<$45 ml/min per 1.73 m^2 at discharge (groups 3 and 4) were significantly older; were more likely to be women; were more likely to have an ischemic etiology; were less likely to have an idiopathic dilated etiology; were more likely to have a history of hypertension, dyslipidemia, and diabetes; were more likely to have a preserved EF; were less likely to be using angiotensin–converting enzyme inhibitors at discharge; and had a higher systolic BP and lower hemoglobin at discharge than the patients with an eGFR$\geq$45 ml/min per 1.73 m^2 at discharge (groups 1 and 2).

Outcomes Stratified by the eGFR and BUN

Figure 2 shows Kaplan–Meier estimates of all-cause death after discharge for the four groups stratified by their eGFR and BUN values at discharge. According to univariate



Number at risk

eGFR ≥45 and BUN <25:	1981	1928	1888	1846	1778	1749	1718
eGFR ≥45 and BUN ≥25 :	418	397	384	369	341	337	323
eGFR <45 and BUN <25 :	569	556	537	525	504	495	486
eGFR <45 and BUN ≥25 :	1481	1398	1333	1271	1200	1146	1092

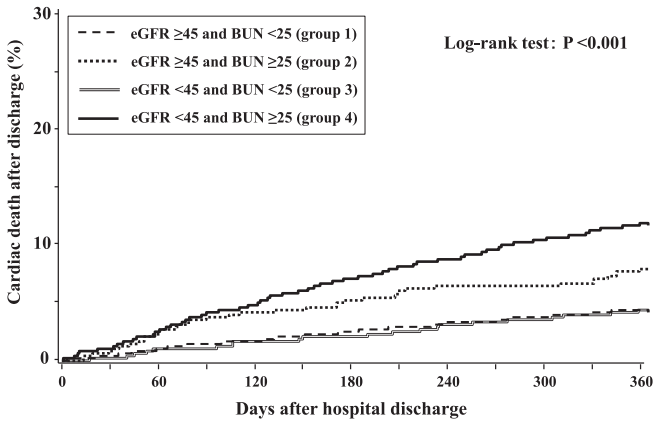
Figure 2. | Kaplan–Meier estimates of all-cause death after discharge in four groups stratified by the eGFR and BUN at discharge. Numbers at risk were given in the lower part.

Table 2. Unadjusted and adjusted risks of postdischarge mortality stratified by eGFR and BUN at discharge

End Point and Group	No. of Patients	No. of Patients with Events (%)	Unadjusted HR	95% CI	*P* Value	Adjusted HR	95% CI	*P* Value	*P* Value for Interaction[a]
All-cause death									0.004
eGFR≥45 and BUN<25	1981	245 (12.5)	1.00			1.00			
eGFR≥45 and BUN≥25	418	87 (20.8)	1.84	1.44 to 2.35	<0.001	1.34	1.04 to 1.73	0.02	
eGFR<45 and BUN<25	569	91 (15.8)	1.33	1.05 to 1.70	0.02	0.97	0.76 to 1.26	0.84	
eGFR<45 and BUN≥25	1481	439 (28.8)	2.71	2.32 to 3.17	<0.001	1.58	1.33 to 1.88	<0.001	
Cardiac death									0.002
eGFR≥45 and BUN<25	1981	130 (6.6)	1.00			1.00			
eGFR≥45 and BUN≥25	418	46 (11.0)	1.83	1.31 to 2.56	<0.001	1.31	0.96 to 1.85	0.08	
eGFR<45 and BUN<25	569	42 (7.4)	1.17	0.83 to 1.66	0.37	0.85	0.59 to 1.24	0.41	
eGFR<45 and BUN≥25	1481	246 (16.6)	2.94	2.37 to 3.63	<0.001	1.83	1.45 to 2.31	<0.001	

HR, hazard ratio; 95% CI, 95% confidence interval.
[a]*P* value for interaction was obtained from the model including a product term of eGFR as a continuous variable and BUN as a dichotomous variable.

analysis (Table 2), patients with either an eGFR<45 ml/min per 1.73 m^2 or a BUN≥25 mg/dl (groups 2–4) had a significantly higher rate of all-cause death than patients with an eGFR≥45 ml/min per 1.73 m^2 and BUN<25 mg/dl (group 1). After adjustment for multiple comorbidities and other clinical factors (Table 2), analysis of patients with an eGFR<45 ml/min per 1.73 m^2 (groups 3 and 4) showed that the hazard ratio (HR) was 1.58 (95% confidence interval [95% CI], 1.33 to 1.88; $P<0.001$) when they had a BUN≥25 mg/dl relative to those with an eGFR≥45 ml/min per 1.73 m^2 and BUN<25 mg/dl (group 1), whereas it was HR, 0.97 (95% CI, 0.76 to 1.26; $P=0.84$) when BUN was <25 mg/dl relative to those with an eGFR≥45 ml/min per 1.73 m^2 and BUN<25 mg/dl. However, among patients with an eGFR≥45 ml/min per 1.73 m^2, a BUN≥25 mg/dl was associated with a significantly higher adjusted risk of all-cause mortality than a BUN<25 mg/dl (HR, 1.34; 95% CI, 1.04 to 1.73; $P=0.02$). Figure 3 shows Kaplan–Meier estimates of cardiac death after discharge for the four groups stratified by their eGFR and BUN values at discharge. When multivariate analysis was done (Table 2), analysis of patients with a BUN≥25 mg/dl (groups 2 and 4) showed that the HR was 1.83 (95% CI, 1.45 to 2.31; $P<0.001$) when they had an eGFR<45 ml/min per 1.73 m^2 relative to those with an eGFR≥45 ml/min per 1.73 m^2 and BUN<25 mg/dl (group 1), whereas it was of borderline statistical significance (HR, 1.31; 95% CI, 0.96 to 1.85; $P=0.08$) when they had an eGFR≥45 ml/min per 1.73 m^2 relative to those with an eGFR≥45 ml/min per 1.73 m^2 and BUN<25 mg/dl. Additionally, when we examined the interaction between eGFR as a continuous variable and a BUN<25 or ≥25 mg/dl with respect to mortality, the association of eGFR with all-cause death or cardiac death was significantly greater in patients with a BUN≥25 mg/dl than in patients with a BUN<25 mg/dl ($P=0.004$ and $P=0.002$ for the interaction, respectively) (Table 2). We also examined the association of eGFR as a continuous variable with outcomes in patients with HF and low BUN levels. This analysis showed that, among patients with HF with a BUN<25 mg/dl at discharge (groups 1 and 3), a decrease in eGFR by 10 ml/min per 1.73 m^2 was not associated with a higher risk of all-cause mortality or cardiac death (adjusted HR, 0.99; 95% CI, 0.94 to 1.04; $P=0.65$ and adjusted HR, 0.96; 95% CI, 0.89 to 1.03; $P=0.25$, respectively). Next, to perform sensitivity analysis, we examined the association of eGFR and BUN at discharge with postdischarge mortality in subgroups stratified by an eGFR at discharge ≥30 or <30 ml/min per 1.73 m^2 and a BUN level at discharge <25 or ≥25 mg/dl. This analysis in subgroups stratified by an eGFR≥30 or <30 ml/min per 1.73 m^2 and a BUN level <25 or ≥25 mg/dl revealed similar results to those in subgroups stratified by an eGFR≥45 or <45 ml/min per 1.73 m^2 and a BUN level <25 or ≥25 mg/dl (Supplemental Table 1). Furthermore, when sensitivity analyses were performed using the other formulas to estimate GFR, the associations of eGFR using the simplified MDRD-4 formula or the CKD-EPI equation at discharge with postdischarge mortality in patients with or without elevated BUN levels at discharge were similar to that for GFR estimated by the Japanese-modified equation (Supplemental Tables 2 and 3).



Number at risk

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eGFR <45 and BUN ≥25 :	1481	1398	1334	1274	1200	1146	1092

Figure 3. | Kaplan–Meier estimates of cardiac death after discharge in four groups stratified by the eGFR and BUN at discharge. Numbers at risk were given in the lower part.

Discussion

A novel finding of this study was that an elevated BUN level at discharge significantly modified the relationship between eGFR at discharge and the higher risk of all-cause or cardiac death after discharge, suggesting that the association between eGFR and outcomes may be largely dependent on the presence of an elevated BUN level. The mechanism underlying this association is unclear, but some assumptions can be made. It is well known that creatinine is freely filtered by the glomerulus and not reabsorbed and that it undergoes tubular secretion. In contrast, urea is freely filtered and not secreted but is reabsorbed by the renal tubules (11). Accordingly, BUN is influenced by enhanced proximal and distal tubular reabsorption caused by neurohormonal activation, the urine flow rate, and the effect of arginine vasopressin on the urea transporter in the collecting duct (1,11,26). Thus, elevation of BUN may serve as an index of neurohormonal activation over and above any decline of eGFR (11). Therefore, even in patients with an eGFR≥45 ml/min per 1.73 m^2, elevation of BUN was associated with a higher risk of postdischarge mortality. This suggests that adoption of BUN as an index may overcome some of the limitations of eGFR. However, another prospective study is needed to investigate this finding.

It is well known that an elevated BUN level at admission is correlated with higher mortality among patients with acute decompensated HF (14,15,27). When HF is decompensated, an imbalance between vasoconstriction and the vasodilatory or natriuretic systems results in the retention of water and sodium, and this pathologic process is likely to contribute to elevation of BUN (11–15). However, when HF is compensated, vasoconstriction is counterbalanced by the vasodilatory and natriuretic systems (13–15). However, our findings showed that the serum BUN level measured in the compensated state (at discharge) is still a predictor of postdischarge mortality, regardless of the eGFR at discharge. Thus, in the compensated state as well as the decompensated state, elevation of BUN may not only reflect renal hypoperfusion and dysfunction but may indicate the cumulative effect of hemodynamic and neurohormonal changes. However, the relationship between sodium and water reabsorption, neurohormonal activation, and BUN is complex in patients with HF (12). Accordingly, additional investigation will be needed to clarify the mechanism underlying the association of the BUN level in the compensated state with worse outcomes in patients with HF.

Current guidelines recommended that an eGFR<60 ml/min per 1.73 m^2 should be considered abnormal (*i.e.*, indicating renal dysfunction) (1–6). We showed that patients with an eGFR<45 ml/min per 1.73 m^2 have a poor prognosis. This finding is in agreement with the results of some previous studies, which showed that patients with an eGFR<45 ml/min per 1.73 m^2 had significantly more adverse events (28,29). Regarding a cutoff value for BUN, it has been reported that a BUN>21–29 mg/dl at admission is associated with an increase of mortality after discharge, and therefore, our findings are in agreement with these previous studies that used similar cutoff values for BUN (14,15,30). However, additional investigation will be needed to clarify the predictive value of eGFR and BUN at discharge in patients who survive hospitalization for acute decompensated HF.

There are several limitations that need to be considered when interpreting the results of this study. First, eGFR and BUN were measured at a single time point (at discharge), and therefore, it is unknown whether these measured values were persistent or transient in this study population. Second, BUN incorporates the cumulative effects of several influences, including dietary protein intake, catabolism of endogenous proteins, and upper gastrointestinal bleeding.

Accordingly, BUN levels should be interpreted together with these settings in patients with HF. Third, because the prognosis may have been influenced by the definitions that we used, additional investigations will be needed to clarify the predictive values of eGFR or BUN at discharge in patients with HF.

In conclusion, among patients who survived hospitalization for acute decompensated HF, the eGFR at discharge was a risk factor for postdischarge mortality in association with elevation of BUN but not in patients with a low BUN at discharge. Accordingly, better understanding of the pathophysiologic features of HF that determine the eGFR and BUN levels at discharge is needed to allow development of targeted therapy that can improve the prognosis of patients who survive hospitalization for acute decompensated HF.

Acknowledgments

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Before the launch of the Acute Decompensated Heart Failure Syndromes (ATTEND) Registry, information on the objectives of this study, its social significance, and an abstract were provided to the society from UMIN (Clinical Trial Registration Identification no. UMIN000000736).

The Japan Heart Foundation had no role in the conduct of the study.

The ATTEND Registry investigators are listed in Supplemental Appendix.

Disclosures

None.

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