Development and Validation of a Model to Predict Acute Kidney Injury in Hospitalized Patients With Cirrhosis

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OBJECTIVES: **Acute kidney injury (AKI) is a common complication in hospitalized patients with cirrhosis which contributes to morbidity and mortality. Improved prediction of AKI in this population is needed for prevention and early intervention. We developed a model to identify hospitalized patients at risk for AKI.**

METHODS: **Admission data from a prospective cohort of hospitalized patients with cirrhosis without AKI on admission (n = 397) was used for derivation. AKI development in the first week of admission was captured. Independent predictors of AKI on multivariate logistic regression were used to develop the prediction model. External validation was performed on a separate multicenter cohort (n = 308).**

RESULTS: **In the derivation cohort, the mean age was 57 years, the Model for End-Stage Liver Disease score was 17, and 59 patients (15%) developed AKI after a median of 4 days. Admission creatinine (OR: 2.38 per 1 mg/dL increase [95% CI: 1.47–3.85]), international normalized ratio (OR: 1.92 per 1 unit increase [95% CI: 1.92–3.10]), and white blood cell count (OR: 1.09 per 1 × 10⁹/L increase [95% CI: 1.04–1.15]) were independently associated with AKI. These variables were used to develop a prediction model (area underneath the receiver operator curve: 0.77 [95% CI: 0.70–0.83]). In the validation cohort (mean age of 53 years, Model for End-Stage Liver Disease score of 16, and AKI development of 13%), the area underneath the receiver operator curve for the model was 0.70 (95% CI: 0.61–0.78).**

DISCUSSION: **A model consisting of admission creatinine, international normalized ratio, and white blood cell count can identify patients with cirrhosis at risk for in-hospital AKI development. On further validation, our model can be used to apply novel interventions to reduce the incidence of AKI among patients with cirrhosis who are hospitalized.**

INTRODUCTION

Acute kidney injury (AKI) is an important complication of decompensated cirrhosis that drives poor outcomes (1,2). AKI is present in 20% (3) of patients with cirrhosis admitted to the hospital, and it develops in up to 19% after admission (4). Its development is independently associated with high short-term mortality and leads to significant morbidity after discharge from the hospital (5,6).

Given the significant burden and prognostic implications of AKI, accurate risk stratification in this population is crucial for efforts to prevent, recognize, and treat AKI to improve outcomes.

Although a rise in serum creatinine (Scr) is the cornerstone of AKI diagnosis (7), early recognition is hindered as acute changes in Scr are often delayed due to the time required for creatinine to accumulate and equilibrate. In addition, factors such as ascites, malnutrition, and sarcopenia which are common in hospitalized patients with cirrhosis contribute to spurious creatinine-based estimates of renal function and can prevent the early recognition of patients at risk (8). Novel renal biomarkers such as cystatin C and neutrophil gelatinase-associated lipocalin may allow for improved risk assessment (9), but these research tools are expensive and remain far from clinical use (2). Thus, there is a major

unmet need for an accurate tool to predict AKI in hospitalized patients with cirrhosis.

An AKI prediction model using readily available objective clinical parameters could be valuable to identify patients at high risk for AKI. Accurate identification of high-risk patients will allow for early intervention and appropriate allocation of hospital resources to improve patient care and outcomes. Furthermore, development of a prediction model could aid clinical trial design for AKI prevention by identifying patients at highest risk.

Thus, this study develops a simple, objective model to predict AKI development in hospitalized patients with cirrhosis. To identify those at risk promptly, we examined data collected within the first 24 h of hospitalization from a large prospective cohort of hospitalized patients. Further validation of the predictive model was performed on an independent external cohort.

PATIENTS AND METHODS

Study design

We created a model to predict in-hospital AKI using a prospective cohort of patients with cirrhosis who were nonelectively admitted to Indiana University Hospital. The Indiana University Hospital is the largest referral and academic tertiary care hospital in the state of Indiana. External validation of the prediction model was performed using a multicenter retrospective cohort of hospitalized patients with cirrhosis. The diagnosis of cirrhosis was made based on clinical parameters involving laboratory tests, endoscopic or radiologic evidence of cirrhosis, evidence of decompensation (hepatic encephalopathy, ascites, variceal bleeding, jaundice), and liver biopsy if available. Those who met inclusion criteria (see section on "Inclusion Criteria") were included for the analysis. The derivation and validation of the prediction model was designed according to the TRIPOD guidelines (10). The protocol was approved by the Institutional Review Board at our center.

Inclusion criteria. The study inclusion criteria were the following:

1. Availability of a baseline Scr as defined by the International Club of Ascites (7).
2. Availability of the following laboratory studies within 24 h of admission: complete blood count, basic metabolic profile, hepatic panel, and international normalized ratio (INR).

Patients excluded from the analysis were those who did not meet the inclusion criteria as well as the following: those who had previously undergone liver or kidney transplant, had AKI on admission, and had acute or chronic requirement of hemodialysis at the time of admission, and those with missing laboratory measurements within 24 h of admission.

Definition of AKI. AKI was defined by the Kidney Disease Improving Global Outcomes guideline (11), which has been endorsed by the International Club of Ascites (7) for patients with cirrhosis. Because urine output documentation can be unreliable (7), only the rise in Scr $\geq$ 0.3 mg/dL or $\geq$ 1.5$\times$ baseline was used to define the in-hospital development of AKI. The primary outcome was AKI development up to 7 days after admission. AKI beyond 7 days was not included in the primary outcome because of the practical difficulty in predicting AKI later in the hospitalization based on the admission data. Stages of AKI were defined by Kidney Disease Improving Global Outcomes (11) staging system which has also

been endorsed by the International Club of Ascites: stage 1, increase in Scr > 0.3 mg/dL or increase in Scr $\geq$ 1.5–2$\times$ baseline; stage 2, increase in Scr > 2$\times$–3$\times$ increase from baseline; stage 3, increase in Scr >3$\times$ from baseline or Scr > 4.0 mg/dL with an acute increase $\geq$ 0.3 mg/dL or initiation of hemodialysis.

Derivation cohort

Patients who were prospectively enrolled from June 2014 to October 2018 and met the inclusion criteria were included for the analysis. The etiology of cirrhosis, history of complications related to cirrhosis, demographics, reason for hospitalization, daily vital signs, and laboratory data (complete blood count, metabolic panel, and hepatic panel) from the day of admission to day 14 of hospitalization were collected. In addition, the status of infections (on admission and during the hospitalization), phenotype of AKI (prerenal azotemia, acute tubular necrosis, hepatorenal syndrome type 1, and other), precipitants of AKI (large-volume paracentesis, excessive use of diuretics, spontaneous bacterial peritonitis (SBP), non-SBP infections, portal hypertensive-related bleeding, intravenous contrast, and other), and 30-day mortality details were collected. The etiology of cirrhosis was categorized into viral hepatitis C (HCV), HCV and alcohol, alcohol, nonalcoholic steatohepatitis, and other. Cirrhosis severity was calculated using the Model for End-Stage Liver Disease (MELD) (12) and Child-Turcotte-Pugh (13) scores. Acute on chronic liver failure (ACLF) and its severity (grades 1–3) were defined by the CLIF Consortium Organ Failure Score (14).

Validation cohort

Patients who were admitted nonelectively to 4 safety-net hospitals (Baylor College of Medicine Medical Center, Boston University Medical Center, Eskenazi Medical Center, and John Stroger Hospital of Cook County Health) in 2012 were screened for inclusion criteria. Details of this cohort have been previously described (15). Data collected were analogous to the derivation cohort, with the exception of phenotype of AKI, ACLF details, precipitants of AKI, and mortality.

Context of use

The current study was designed to create a prediction model for AKI development in hospitalized patients with cirrhosis. The potential decisions to be made based on the model are the intensity of kidney function monitoring, potential AKI prophylaxis with volume administration, inclusion criteria for preventive clinical trial design, and avoidance of certain nephrotoxins.

Statistical analysis

Patients were stratified based on the development of AKI or not, and their characteristics on admission were compared. Continuous variables were presented as mean $\pm$ SD and median with interquartile range (IQR) where appropriate. Categorical variables were presented as percentages. Differences across the groups with respect to categorical variables were analyzed using χ^2 test and Fisher Exact test, whereas continuous variables were analyzed using the nonparametric Kruskal-Wallis test and student t test. A 2-sided nominal P value of less than or equal to 0.05 was considered significant.

Derivation of the AKI prediction model. Variables of clinical significance were considered for the multivariate analysis. These were baseline Scr, admission Scr, white blood cell count (WBC),

mean arterial pressure, INR, admission sodium, admission total bilirubin, admission albumin, presence of infection on admission, ascites, age, and gender (Table 1). The variables were ordered based on univariate significance by fitting a logistic regression model and added into the multivariate model using a forward selection procedure. Highly correlated variables were compared and included only once. Model selection was based on minimizing Akaike information criterion and maximizing area underneath the receiver operator curve (AUROC) or concordance c-statistic, with priority given to the lowest Akaike information criterion. The final model was then applied to the validation cohort, and AUROC analysis was performed for the prediction of AKI. The diagnostic performance of the model in both derivation and validation cohorts was evaluated using sensitivity, specificity, positive predictive value, negative predictive value, and diagnostic accuracy at 90% sensitivity, 90% specificity, and the maximum Youden index. The final model was then compared with the MELD-Na and the CLIF Consortium Acute Decompensation (CLIF-AD) (16) scores via DeLongs method. All analyses were performed in SAS version 9.4.

RESULTS

Derivation cohort
A total of 627 patients were enrolled during the study period. After excluding those who did not meet the inclusion criteria (229 patients in total: 191 had AKI on admission and 38 had missing admission labs), a total of 397 patients were included for the analysis (59 with AKI development [14%] and 338 without AKI development).

Patient characteristics. The mean age of the entire cohort was 57.3 ± 10.8 years. Fifty-seven percent were men, with a mean MELD score of 17.2 ± 6.1. Nonalcoholic steatohepatitis (33%), alcohol (27%), and HCV (15%) were the most common etiologies of cirrhosis. Those who developed AKI had a significantly higher baseline Scr, admission Scr, admission total bilirubin, admission INR, admission blood urea nitrogen, admission WBC, and admission MELD score compared with the non-AKI group (Table 1). The presence of infection was found to be significantly higher in the AKI group compared with the non-AKI group (21% and 11%, respectively; $P = 0.023$). The AKI group had more admissions for ascites/anasarca and fewer admissions for portal hypertensive-related bleeding (Table 1).

One hundred seventy-five patients had ACLF on admission, of which 31 patients developed AKI. There were no differences between the 2 groups for the presence of ACLF on admission (54% AKI development vs 42% no AKI development; $P = 0.120$) (Table 1). However, there were differences across ACLF grades between the 2 groups ($P = 0.001$), with a higher percentage of patients having ACLF grade 3 who developed AKI (34%) vs patients who did not develop AKI (18%; $P = 0.003$).

AKI development details and hospitalization outcomes. The median (IQR) time to AKI development was 4 (3–5) hospital days. Ninety-five percent of patients had stage 1 AKI at the time of diagnosis, followed by 5% for stage 2. The most frequent precipitant for AKI identified was non-SBP infections (32% overall), followed by excessive use of diuretics (19%). SBP was found to be the cause of AKI in 7% of patients.

In those who had stage 1 AKI at the time of diagnoses, 68% stayed at stage 1 and the rest progressed to stages 2 and 3 (16% and 10%, respectively). The most frequent phenotype of AKI was prerenal at 52%, followed by acute tubular necrosis at 19% and type 1 hepatorenal syndrome at 3%. Ten percent of patients with AKI required hemodialysis during the hospitalization. Compared with the non-AKI group, patients who developed AKI had significantly higher in-hospital mortality (16% vs 2%; $P < 0.001$) and 30-day mortality (28% vs 6%; $P < 0.001$).

Validation cohort
A total of 733 patients were screened for inclusion criteria. Four hundred twenty-five patients were excluded (170 patients with AKI on admission and 255 patients with missing admission data), leaving 308 patients for the analysis (41 with AKI development [13%] and 267 without AKI). Most of them were men (73%), and the mean MELD score was 15.9 ± 5.9. Similar to the derivation cohort, patients who developed AKI had higher baseline Scr, admission Scr, admission total bilirubin, admission INR, admission blood urea nitrogen, admission WBC, and admission MELD score compared with the non-AKI group (see Table 1, Supplementary Digital Content, http://links.lww.com/CTG/A92). In addition, the presence of infection was found to be significantly higher in the AKI development group (42%) compared with the non-AKI group (15%) ($P \leq 0.0001$). Comparisons between the derivation and validation cohorts can be found in Supplementary Table 2 (Supplementary Digital Content, http://links.lww.com/CTG/A92).

The median (IQR) time to AKI development was 2 (1–3) days, and 73%, 20%, and 8% had stage 1, 2, and 3 at the time of AKI diagnosis. Thirty-one percent of patients diagnosed at stage 1 progressed to higher stages of AKI, and 63% of patients with stage 2 AKI progressed to stage 3. Seven percent of patients required hemodialysis.

AKI prediction model
The admission variables that were found to be significant for AKI development on univariate analysis in the derivation cohort are listed in Table 2. On multivariate analysis, admission Scr (OR: 2.38 per 1 mg/dL increase [95% CI: 1.47–3.85]), INR (OR: 1.92 per 1 unit increase [95% CI: 1.92–3.10]), and WBC (OR: 1.09 per 1 × 10^9/L increase [95% CI: 1.04–1.15]) were independently associated with AKI. Using these variables, a logistic regression model was created for prediction:

$$\text{AKI Prediction Score}:$$
$$-4.59 + 0.089 \times WBC + 0.87 \times Scr + 0.65 \times INR.$$

$$\text{Predicted probability of AKI}$$
$$= \frac{\exp(-4.59 + 0.089 \times WBC + 0.87 \times Scr + 0.65 \times INR)}{1 + \exp(-4.59 + 0.089 \times WBC + 0.87 \times Scr + 0.65 \times INR)}.$$

The AUROC for prediction was found to be 0.77 (95% CI: 0.70, 0.83; $P < 0.0001$) (Figure 1), and the Akaike information criterion was 298. The AKI prediction model was found to have a numerically higher AUROC than MELD-Na score (0.71 [95% CI: 0.63, 0.79]) and CLIF-AD score (0.73 [95% CI: 0.66, 0.80]), although the differences were not statistically significant ($P = 0.08$ vs MELD-NA and $P = 0.13$ vs CLIF-AD).

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Table 1. Comparisons of admission characteristics stratified by AKI status

Characteristic	Development of AKI (n = 59)	Non-AKI (n = 338)	*P* value
Mean age (±SD)	56.6 (10.8)	57.6 (11)	0.305
Sex, n (% male)	35 (59)	186 (55)	0.540
Race, n (% white)	58 (98)	316 (94)	0.344
Etiology of cirrhosis, n (%)			
Alcohol	20 (34)	90 (27)	
HCV	7 (12)	53 (15)	0.687
HCV and alcohol	3 (5)	30 (9)	
NASH	21 (36)	111 (33)	
Other	8 (13)	54 (16)	
Baseline creatinine, mg/dL (±SD)	1.3 (0.7)	1.0 (0.5)	<0.001
CTP stage, n (%)			
A	1 (2)	20 (6)	
B	13 (22)	118 (35)	0.033
C	45 (76)	197 (59)	
MELD (±SD)	21.2 (7.9)	16.5 (5.5)	<0.001
Presence of ACLF, n (%)	31 (53)	144 (43)	0.120
ACLF grade, n (%)			
1	4 (7)	5 (1)	
2	7 (12)	79 (23)	
3	20 (34)	60 (18)	0.001
Ascites, n (%)			
None	5 (9)	82 (24)	
Controlled	9 (15)	99 (29)	<0.001
Uncontrolled	45 (76)	157 (46)	
Sodium, mmol/L (±SD)	130.9 (7.5)	133.5 (5.5)	0.002
Creatinine, mg/dL (±SD)	1.3 (0.6)	1.0 (0.5)	<0.001
BUN, mg/dL (±SD)	28.8 (18.2)	19.7 (12.4)	<0.001
Albumin, g/dL (±SD)	2.7 (0.6)	2.9 (0.6)	0.022
INR (±SD)	1.9 (0.7)	1.6 (0.5)	<0.001
Total bilirubin, mg/dL (±SD)	6.1 (5.9)	4 (4.5)	0.002
WBC, $\times 10^9$ (±SD)	9.4 (7.2)	6.6 (4.3)	<0.001
Mean arterial pressure (±SD)	82.7 (14.1)	85.4 (14.6)	0.186
Presence of infection, n (%)	12 (21.4)	32 (10.6)	0.023
Reason for admission, n (%)			
Overt hepatic encephalopathy	14 (25)	88 (26)	
PHTN-related bleed	3 (5)	59 (18)	0.042
Ascites/anasarca	16 (27)	52 (15)	
Other	14 (23)	69 (20)	
Liver unrelated	12 (20)	70 (21)	

ACLF, acute on chronic liver failure; AKI, acute kidney injury; BUN, blood urea nitrogen; CTP, Child-Turcotte-Pugh; HCV, hepatitis C virus; INR, international normalized ratio; MELD, Model for End-Stage Liver Disease; NASH, nonalcoholic steatohepatitis; PHTN, portal hypertension; WBC, white blood cell count.

The diagnostic performance of the AKI prediction model is shown in Table 3. With the sensitivity and specificity fixed at 90%, the negative predictive value was high at 96% and 88%, respectively. The diagnostic accuracy was highest with a cutoff > −1.21 (specificity at 90%) at 82%. When the logistic regression AKI prediction model from the derivation cohort was applied to the validation cohort, the

Table 2. Independent predictors for acute kidney injury prediction

	Univariate		Multivariate	
Admission predictor	Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value
Creatinine	2.32 (1.48–3.65)	<0.001	2.38 (1.47–3.85)	<0.001
WBC	1.10 (1.04–1.15)	<0.001	1.09 (1.04–1.15)	0.001
INR	2.07 (1.31–3.27)	0.002	1.92 (1.19–3.10)	0.008
Baseline creatinine	2.16 (1.39–3.35)	0.001		
Sodium	0.93 (0.89–0.98)	0.002		
Total bilirubin	1.07 (1.02–1.13)	0.003		
Albumin	0.55 (0.33–0.92)	0.022		
Presence of infection	2.31 (1.11–4.82)	0.026		
Ascites				
Controlled vs none	1.06 (0.35–3.19)	0.2308		
Uncontrolled vs none	3.20 (1.29–7.95)	0.0008		
Mean arterial pressure	0.99 (0.97–1.01)	0.186		
Age	0.99 (0.96–1.01)	0.305		
Male	1.19 (0.68–2.09)	0.541		

CI, confidence interval; INR, international normalized ratio; WBC, white blood cell count.

AUROC was 0.70 (95% CI: 0.61, 0.78; $P < 0.0001$) (Figure 2). Similar to the derivation cohort, a score of > -1.21 had a diagnostic accuracy at 82%, with a specificity of 90%.

DISCUSSION

In this study, we have derived and externally validated a predictive model for the development of AKI in hospitalized patients with cirrhosis. Our model is based on 3 objective measures that are widely available in clinical practice (Scr, INR, and WBC) and can therefore be applied in a variety of hospitalized settings. The AUROC for prediction of AKI in the derivation cohort was 0.77, and the AUROC in an independent validation cohort was 0.70.

The pathophysiology of AKI in patients with cirrhosis is complex and often related to renal vasoconstriction secondary to the effective hypovolemia that occurs in advanced cirrhosis and portal hypertension (17). Furthermore, it is increasingly recognized that
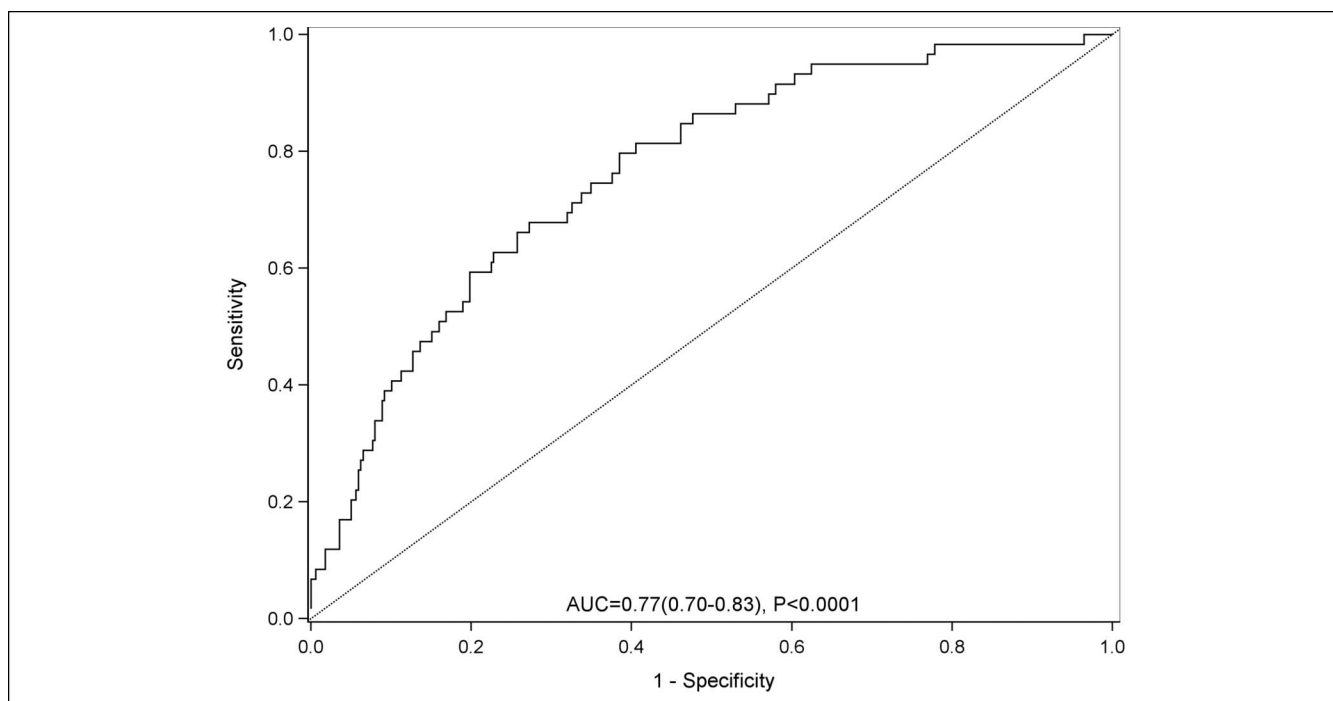
Figure 1. Area underneath the receiver operating curve for the prediction of acute kidney injury in the derivation cohort.

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Table 3. Diagnostic performance of the AKI prediction model

Selected cutoffs	Raw score	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Diagnostic accuracy (%)
Derivation cohort						
90% sensitivity*	−2.38	90	39	20	96	46
Maximum Youden index	−1.82	63	74	29	92	72
90% specificity*	−1.21	31	90	37	88	82
Validation cohort						
90% sensitivity*	−2.38	81	44	18	94	49
Maximum Youden Index	−1.82	46	78	25	91	74
90% specificity*	−1.21	29	90	32	89	82

NPV, negative predictive value; PPV, positive predictive value.
*Fixed at sensitivity and specificity closest to 90% but ≥90%.

AKI is associated with systemic and intrarenal inflammation (18–21). Patients with advanced cirrhosis have higher levels of circulating endotoxins, possibly due to bacterial translocation across the intestinal wall (22,23), thus promoting the production of many proinflammatory cytokines, substances that cause peripheral and splanchnic vasodilation (24,25), as well as intrarenal endothelial dysfunction and cell damage (20,26). Each of these pathophysiologic elements is indirectly captured by the variables in our predictive model. For example, a high WBC, a surrogate of systemic inflammation, is a known independent predictor of mortality in patients with advanced cirrhosis and is a driver for the development of ACLF (19,27). An elevated WBC has also been associated with renal dysfunction in patients with SBP (28,29) and alcoholic hepatitis (30). The other 2 components of the risk score were INR and Scr. INR is a well-known marker of progressive liver failure and therefore worsening portal hypertension (12,13) which has been linked to renal impairment (17,31,32). Furthermore, despite its limitations, we found Scr to have the highest OR of 2.38 for 1 mg/dL increase. Our findings are in line with other studies (33–35), where higher Scr is independently associated with AKI development and poor renal outcomes.

There are several limitations to our study. First, although urine output is not considered a part of the diagnostic criteria for AKI by the International Club of Ascites (7), we were not able to compare our risk score with early urine output. Urine output should be evaluated in future studies because it has been shown to be sensitive for incident AKI (36). Similarly, we were unable to compare our model with novel kidney biomarkers (e.g., cystatin C, neutrophil gelatinase-associated lipocalin) for risk assessment. Thus, future studies are also needed to assess these kidney
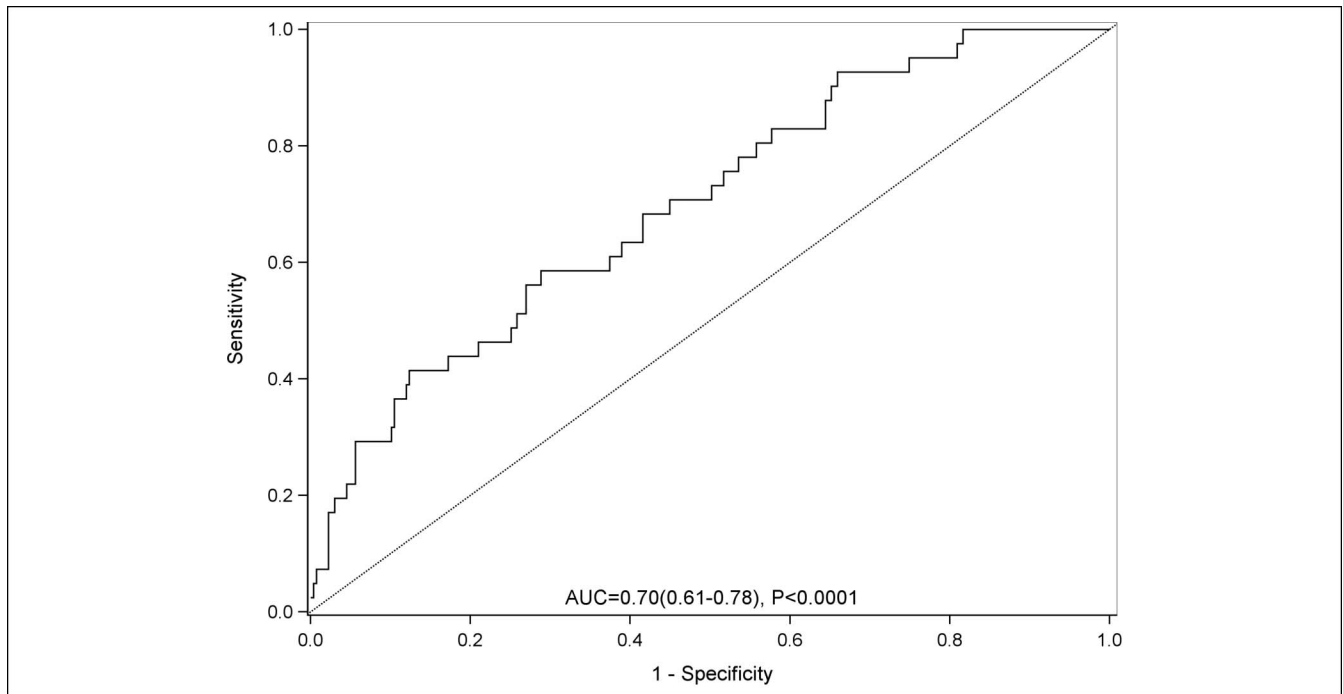
Figure 2. Area underneath the receiver operating curve for the prediction of acute kidney injury in the validation cohort.

biomarkers to substantiate their roles as clinical tools and to assess if the addition of biomarkers enhances prediction. Last, because only 5% of patients had AKI stage 2 or higher at the time of diagnosis, we were unable to determine if our model is applicable to predict higher stages of AKI. In contrast to these weaknesses, the study benefits from the use of a multicenter external validation cohort, with numerous significant differences compared with the derivation cohort. These differences highlight the potential generalizability of the model, which may be applied in diverse settings to predict AKI.

The ability to risk stratify patients early during their hospitalization has several important implications for clinical management. For example, a patient without AKI on admission with an INR of 2.5, WBC of 15, and Scr 2.0 would have an AKI risk score of 0.11, corresponding to a 7-day risk of AKI of 53% with >80% diagnostic accuracy. By contrast, a patient with an INR of 1.7, WBC of 10, and Scr of 0.8 would have an AKI risk score of −1.89, corresponding to a much lower risk of 13%. Hence, knowledge of individual patient risk for AKI may allow the treating physician to personalize the intensity of laboratory monitoring and to be mindful in the use of intravenous diuretics or iodinated contrast. Furthermore, risk stratification could provide a standardized approach for future clinical trials focusing on AKI prevention. Therefore, though not yet proven, the model has a clear potential to influence therapeutic or prognostic decisions in the future. An example of this would be early administration of albumin or early hepatorenal syndrome-directed therapy (albumin, terlipressin, etc.) in a patient at high risk to develop hepatorenal syndrome. Our model of AKI prediction calculator is available at http://gihep.com/calculators/hepatology/aki-in-hospitalized-patients-with-cirrhosis/.

In conclusion, in this large prospective study of hospitalized patients with cirrhosis, the development of in-hospital AKI is common and confers a poor prognosis. Our model for AKI prediction is based on 3 widely available laboratory parameters; it captures the pathophysiologic elements of AKI, and it may identify patients at high risk for AKI. The use of this model may allow for closer monitoring and early treatment to prevent AKI in those at risk. Ultimately, future studies are required to demonstrate whether this risk score can be used to improve clinical outcomes.

CONFLICTS OF INTEREST

Study Highlights

WHAT IS KNOWN

✓ In-hospital AKI is a common complication of cirrhosis which leads to significant morbidity and mortality.

✓ There are no existing tools to predict in-hospital AKI in this population. Creation of a prediction model may help guide treatment and improve outcomes.

WHAT IS NEW HERE

✓ Admission laboratory values (white blood cell count, creatinine, and international normalized ratio) were used to derive the AKI prediction model. The model had an AUROC of 0.77 (95% CI: 0.70–0.83; $P < 0.001$) for AKI prediction.

✓ When validated in a separate multicenter cohort, the AUROC for AKI prediction was 0.70 (95% CI: 0.61–0.78; $P < 0.001$).

✓ Our AKI prediction model may allow for prevention and timely treatment of AKI to improve patient outcomes.

TRANSLATIONAL IMPACT

✓ Our AKI prediction model may allow for prevention and timely treatment of AKI to improve patient outcomes.

✓ Furthermore, risk stratification via our AKI model could provide a standardized approach for future clinical trials focusing on AKI prevention.

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