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Original article

# Development of a nomogram for predicting kidney replacement therapy and hyperkalemia in acute kidney injury

## *Desarrollo de un nomograma para predecir la terapia de reemplazo renal y la hipercalemia en la lesión renal aguda*

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ABSTRACT

*Background:* Acute kidney injury (AKI) is prevalent among hospitalized patients and is frequently complicated by hyperkalemia (HyperK), kidney replacement therapy (KRT), and major adverse kidney events (MAKE). Early prediction of these outcomes remains a clinical priority.
*Objective:* To develop and internally validate nomograms using routinely collected clinical variables to predict the risk of HyperK, KRT, and MAKE, including death and ≥25 mL/min/1.73 $m^2$ reduction in eGFR in hospitalized AKI patients.
*Methods:* This retrospective cohort study included 753 adult AKI patients without initial HyperK, evaluated at a tertiary referral center from 2020 to 2024. Logistic regression models identified predictors of HyperK and MAKE, stratified by sex. Model performance was assessed via AUC, calibration, and predictive metrics. Nomograms were constructed based on final multivariate models.
*Results:* During follow-up, 24% of patients developed HyperK. Independent predictors included vasopressor use, shock, urinary obstruction, low hemoglobin, and higher baseline potassium. The HyperK model demonstrated moderate discrimination (AUC 0.68) but a high negative predictive value (97%). Sex-stratified nomograms for MAKE, KRT, and mortality showed strong performance (AUCs 0.74–0.98), with highest accuracy observed in KRT models for both sexes (AUC 0.96). Predictors varied by sex but commonly included volume overload, acid–base disorders, uremia, and elevated creatinine.
*Conclusion:* We developed pragmatic and accessible nomograms capable of predicting HyperK, KRT, and MAKE in AKI patients using standard clinical data. These tools offer timely, personalized risk stratification and may support clinical decision-making in diverse hospital settings.

*Palabras clave:*
Lesión renal aguda
Nomograma
Terapia de reemplazo renal
Episodio renal adverso mayor

RESUMEN

*Antecedentes:* La lesión renal aguda (LRA) es prevalente entre los pacientes hospitalizados, complicándose a menudo por hipercalemia (HyperK), terapia de reemplazo renal (TRR), y episodios renales adversos mayores (MAKE). La predicción temprana de estos resultados sigue siendo una prioridad clínica.

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*Objetivo:* Desarrollar y validar nomogramas a nivel interno utilizando variables clínicas recopiladas rutinariamente para predecir el riesgo de HyperK, KRT y MAKE, incluyendo muerte y reducción ≥25 ml/min/1,73 $m^2$ de TFGe en pacientes hospitalizados con LRA.
*Métodos:* Este estudio retrospectivo de cohorte incluyó 753 pacientes adultos con LRA sin HyperK inicial, evaluados en un centro de referencia terciario de 2020 a 2024. Los modelos de regresión logística identificaron factores predictivos para HyperK y MAKE, estratificados por sexo. El desempeño del modelo fue evaluado mediante AUC, calibración y métricas predictivas. Se construyeron los nomogramas sobre la base de los modelos multivariante finales.
*Resultados:* Durante el seguimiento, el 24% de los pacientes desarrollaron HyperK. Los factores predictivos independientes incluyeron uso de vasopresores, shock, obstrucción urinaria, baja hemoglobina y potasio basal más elevado. El modelo de HyperK demostró una discriminación moderada (AUC 0,68), aunque un valor predictivo negativo alto (97%). Los nomogramas estratificados por sexo para MAKE, KRT y mortalidad reflejaron un desempeño fuerte (AUC 0,74-0,98), con una mayor precisión observada en los modelos para LRA en ambos sexos (AUC 0,96). Los predictores variaron por sexo, pero incluyeron comúnmente sobrecarga de volumen, trastorno ácido–base, uremia y elevación de creatinina.
*Conclusión:* Desarrollamos nomogramas pragmáticos y accesibles capaces de predecir HyperK, LRA y MAKE en los pacientes de LRA utilizando datos clínicos estándar. Dichas herramientas ofrecen una estratificación del riesgo oportuna y personalizada, y pueden respaldar la toma de decisiones clínica en diversos ámbitos hospitalarios.

## Background

Acute kidney injury (AKI) occurs in 1 in 3 hospitalized patients,[1] increases the risk of mortality[2] and is associated with the deterioration of physiological functions of practically all organs,[3] even more so when AKI warrants the initiation of kidney replacement therapy (KRT).[4] Among the pathophysiological alterations associated with this high mortality rate are electrolyte dysregulations resulting from kidney dysfunction; hyperkalemia (HyperK) is the most studied electrolyte disturbance in this context. It occurs more frequently in patients with AKI, being 20 times more likely compared to the general population.[5] It occurs in up to 20% of cases, in this context is easily explained because kidney function is the most important determinant for potassium excretion,[6] but also because of the usual etiologies that accompany AKI, which also increase the risk of HyperK.[7] Patients with AKI who develop HyperK are intuitively those with the highest disease burden, have more comorbidities, manifested by higher severity scores, higher serum creatinine values, higher uremic toxin load, more volume overload, more severe metabolic acidosis.[8] HyperK associated with AKI is one of the most feared complications, it is associated with increased mortality[9] it is often observed secondary to urinary obstruction, dehydration, rhabdomyolysis, nephrotoxic drugs,[10] for this reason, it is one of the most common reasons to urgently start KRT.[11] Clinical trials and meta-analyses recommend initiating KRT in patients with AKI and HyperK refractory to medical management.[12] Therefore, we consider that anticipating the event of HyperK in patients with AKI is relevant, as it would have the potential to alert the clinician about its incidence, raise awareness, promote serum potassium monitoring, perform actions that could prevent potassium elevation, and eventually treat it in a timely manner, thereby limiting adverse events. To our knowledge, the possibility of anticipating AKI complications such as HyperK, the initiation of KRT, and mortality has not been deeply explored. In order to reduce this information gap, we propose the creation of simple nomograms of common variables taken during hospitalization, which have the capacity to predict the incidence of HyperK, initiation of KRT, deterioration of kidney function, and mortality during the following days in hospitalized patients with AKI.

## Methods

### *Study population and data collection*

We performed a retrospective cohort study at Hospital Civil de Guadalajara Fray Antonio Alcalde, Mexico, between August 2020 and June 2024. All included patients were hospitalized and were consulted by the nephrology service and met KDIGO serum creatinine criteria (sCr) for AKI were initially screened. Patients were eligible for inclusion if they were ≥18 years old, had a known baseline sCr measurement within the preceding six months, and had available follow-up laboratory data during hospitalization. Patients were excluded if they had HyperK at the time of the first nephrology consultation, were receiving KRT, had undergone kidney transplantation, were pregnant, or had incomplete clinical or laboratory data precluding outcome assessment. Clinical characteristics, demographic data, and laboratory values were retrospectively recorded via automated data extraction from our institutional electronic health record system. The diagnosis of AKI was made using the serum creatinine (sCr) KDIGO criteria, urine output criteria were not applied due to incomplete and inconsistent documentation of hourly urine output in the electronic medical record. Chronic kidney disease (CKD) was defined as an estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 $m^2$ for more than three months.[13] We selected the MAKE outcomes to improve the consistency of AKI outcomes reported across studies. MAKE outcomes were defined as death, a new requirement for kidney replacement therapy (KRT), or worsening kidney function by a ≥25% decline in the eGFR from baseline.[14] We selected the MAKE criteria during hospitalization, as it appeared most appropriate given that the majority of AKI patients initiate KRT and die during this timeframe.[13] We included patients with known baseline sCr levels, defined as the most recent sCr value in the last six months before hospitalization, and who had sCr levels in the following days to assess MAKE analyses in our study. We describe contributing to AKI, e.g., sepsis, shock, comorbidities, nephrotoxic among others. We recorded biochemistry values such as hemoglobin, platelets, leukocytes, glucose, urea, sCr, sodium, potassium, chlorine, phosphate, and calcium. We defined baseline diuretics as the use of thiazides and furosemide.

The population included in the present study was limited to patients with AKI who did not have HyperK at the first nephrology evaluation. We monitored patients with more than three serum potassium tests to track their clinical course and detect HyperK. The index potassium value was that obtained at the first visit, this had to be within the normal range (3.5–5.5 mEq/L). We defined HyperK as serum potassium >5.5 mEq/L, since this value is associated with mortality in AKI.[15] We excluded pregnant women, patients aged <18 years, patients undergoing KRT, kidney transplant recipients, and patients with HyperK at the first visit. All participants gave their written informed consent. The indications for KRT included fluid overload that was resistant to diuretics, severe HyperK, severe

metabolic acidosis, and uremic manifestations, such as encephalopathy, pericarditis, and seizures.[16] Shock was defined as the presence of sustained hypotension requiring vasopressor support to maintain a mean arterial pressure ≥65 mmHg, as documented in the medical record. Shock and vasopressor use were recorded as distinct clinical variables because not all patients receiving vasoactive support met criteria for distributive shock; some patients had cardiogenic shock and required inotropic agents without vasopressors. Fluid overload was diagnosed based on clinical assessment, including the presence of peripheral edema, pulmonary congestion on physical examination or imaging, positive fluid balance, and/or the need for diuretic escalation as recorded by the treating team.

The study was approved by the Hospital Civil de Guadalajara Fray Antonio Alcalde Institutional Review Board (CE 180/24). The results were reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines[17] and the transparent reporting of a multivariate prediction model for individual prognosis or diagnosis (TRIPOD) statement.[18]

### *Objectives*

The primary objective was to develop a nomogram to predict HyperK in AKI patients during follow-up based on the clinical variables collected at round visits. The secondary objectives were to determine the development of HyperK in men and women separately, the components of the MAKE composite variable, including GFR reduction >25 mL/min/1.73 $m^2$, death and KRT during follow-up.

### *Statistical methods*

Descriptive statistics were employed to summarize demographic, clinical, and laboratory characteristics. Categorical variables were expressed as frequencies and percentages and compared using chi-square or Fisher's exact test. Continuous variables were reported as medians with interquartile ranges (IQR) and analyzed using the Mann–Whitney *U* test due to non-normal distributions. To identify independent predictors of HyperK (serum potassium ≥5.5 mmol/L), KRT initiation, mortality and MAKE, multivariate logistic regression models were constructed. The events-per-variable (EPV) ratio was calculated for each multivariable model, and all primary models met or exceeded the recommended minimum EPV threshold of 10, reducing the risk of overfitting. Candidate variables were selected based on univariate analysis ($p < 0.20$) and clinical relevance. A backward stepwise selection strategy was used to derive the final models. Adjusted odds ratios (OR) and 95% confidence intervals (CI) were reported for each predictor. Model performance was evaluated using multiple metrics. Discrimination was assessed via the area under the receiver operating characteristic curve (AUC), while calibration was examined through Hosmer–Lemeshow goodness-of-fit tests and calibration plots. Additional model diagnostics included the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) to assess model parsimony. To assess clinical utility, several performance indicators were calculated: accuracy, sensitivity, specificity, F1 score, Youden index, and negative predictive value (NPV). Confusion matrices were constructed to quantify classification errors. Notably, the KRT model exhibited high discriminative power (AUC 0.96) and balanced performance across key indicators. Based on the final multivariate models, nomograms were developed to enable bedside estimation of individual risk probabilities for each outcome. These graphical tools assign weighted point values to each predictor, facilitating personalized clinical decision-making. All statistical analyses were conducted using R software (version 4.4.2) and Stata, with a two-sided *p*-value <0.05 considered statistically significant.

## Results

Between August 2020 and June 2024, a total of 1171 patients hospitalized with AKI were evaluated by the nephrology department. Among these, 755 patients (64.4%) did not exhibit HyperK at the time of the first nephrology consultation. After excluding 416 individuals who did not meet inclusion criteria and two pregnant women, a final sample of 753 patients was included for analysis. During the follow-up period, 182 patients (24%) developed HyperK, as depicted in Fig. 1.

### *Primary objective: prediction of hyperkalemia*

Baseline characteristics stratified by the development of HyperK are presented in Table 1. The mean age was 53.0 years (39.0, 66.0) and 38.8% (292) were male. Patients who developed HyperK showed a more severe clinical profile, with significantly higher baseline serum potassium levels (median 4.7 vs. 4.2 mmol/L), more frequent vasopressor use (40.1% vs. 24.5%), higher incidence of shock

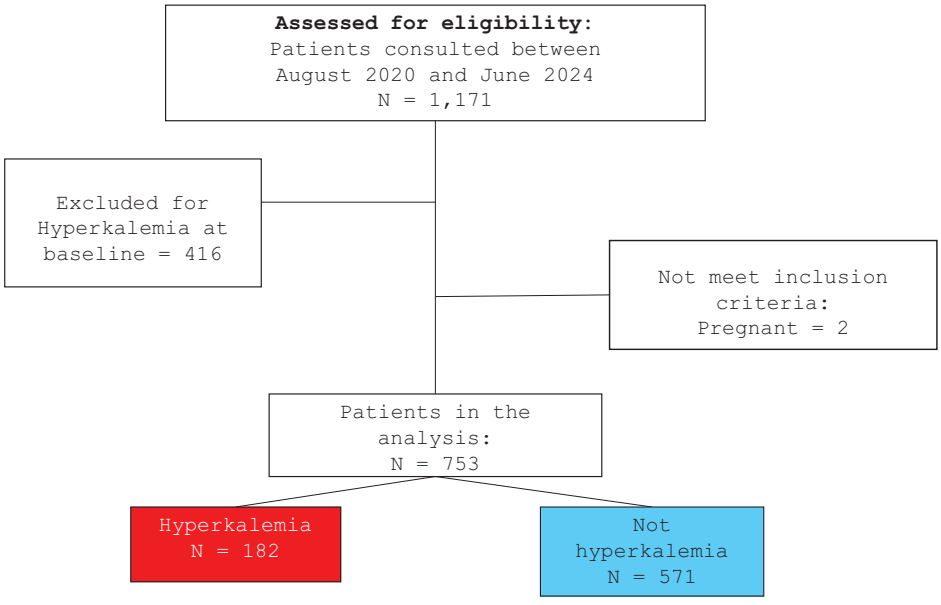



**Fig. 1.** Flow chart of the cohort.

**Table 1**
Baseline demographic and clinical characteristics of patients with AKI according to the development of HyperK.

| Variable | Total ($n$ = 753) | HyperK ($n$ = 182) | Non-HyperK ($n$ = 571) | $p$ |
|---|---|---|---|---|
| *Demographics, comorbilities and treatments* | | | | |
| Age, years | 53.0 (39.0, 66.0) | 53.5 (41.2, 66.0) | 53.0 (38.5, 66.0) | 0.467 |
| Male | 292 (38.8) | 71 (39.0) | 221 (38.7) | 1.000 |
| DM | 237 (31.5) | 58 (31.9) | 179 (31.3) | 0.927 |
| HTN | 252 (33.5) | 57 (31.3) | 195 (34.2) | 0.528 |
| CHF | 72 (9.6) | 18 (9.9) | 54 (9.5) | 0.88 |
| CKD | 148 (19.7) | 24 (13.2) | 124 (21.7) | 0.013 |
| CVE | 35 (4.6) | 5 (2.7) | 30 (5.3) | 0.224 |
| BMI | 25.2 (22.8, 28.7) | 26.1 (23.4, 29.4) | 25.0 (22.6, 28.4) | 0.029 |
| Antihipertensives | 222 (29.5) | 51 (28.0) | 171 (29.9) | 0.642 |
| Diuretics | 258 (34.3) | 69 (37.9) | 189 (33.1) | 0.244 |
| *AKI etiology and acute complications* | | | | |
| Shock | 129 (17.1) | 48 (26.4) | 81 (14.2) | 0.000 |
| Vasopressors | 213 (28.3) | 73 (40.1) | 140 (24.5) | 0.000 |
| Sepsis | 370 (49.1) | 85 (46.7) | 285 (49.9) | 0.496 |
| Hypovolemia | 163 (21.6) | 40 (22.0) | 123 (21.5) | 0.918 |
| Cardiorenal | 101 (13.4) | 21 (11.5) | 80 (14.0) | 0.454 |
| Nephrotoxic | 25 (3.3) | 7 (3.8) | 18 (3.2) | 0.638 |
| Urinary obstruction | 94 (12.5) | 27 (14.8) | 67 (11.7) | 0.303 |
| Metabolic acidosis | 73 (9.7) | 29 (15.9) | 44 (7.7) | 0.002 |
| Fluid overload | 87 (11.6) | 30 (16.5) | 57 (10.0) | 0.023 |
| Uremia | 97 (12.9) | 31 (17.0) | 66 (11.6) | 0.058 |
| *Biochemical variables* | | | | |
| Hemoglobin, g/L | 9.2 (7.6, 10.9) | 9.5 (7.8, 12.3) | 9.2 (7.6, 10.8) | 0.090 |
| Platelets, U/L | 191 (120, 290) | 176 (107, 286) | 192 (122, 290) | 0.440 |
| Leucocytes, U/L | 12 (8, 17) | 12 (8, 18) | 12 (8, 17) | 0.657 |
| Glucose, mg/dL | 112.0 (86.0, 146.0) | 115.4 (88.2, 165.5) | 110.0 (85.5, 142.0) | 0.127 |
| Urea, mg/dL | 143.0 (88.9, 199.3) | 137.0 (83.2, 198.7) | 145.0 (90.7, 199.2) | 0.369 |
| Creatinine, mg/dL | 3.5 (2.2, 5.5) | 3.3 (2.2, 5.1) | 3.6 (2.2, 5.6) | 0.213 |
| Sodium, mEq/L | 136.0 (131.0, 141.0) | 135.4 (131.0, 140.0) | 136.0 (131.0, 141.0) | 0.768 |
| Potassium, mEq/L | 4.4 (3.8, 4.9) | 4.7 (4.3, 5.1) | 4.2 (3.7, 4.7) | 0.000 |
| Chloride, mEq/L | 103.0 (97.8, 108.0) | 103.0 (98.0, 108.0) | 103.0 (97.6, 108.0) | 0.967 |
| Calcium, mg/dL | 7.8 (7.3, 8.3) | 7.8 (7.3, 8.4) | 7.8 (7.3, 8.3) | 0.231 |
| Phosphorus, mg/dL | 5.3 (3.9, 6.6) | 5.3 (3.8, 6.7) | 5.2 (3.9, 6.6) | 0.974 |

Categorical variables are expressed as number and percentage ($n$, %), and continuous variables as median and interquartile range (IQR) or mean ± standard deviation (SD), as appropriate. BMI, body mass index; CHF, chronic heart failure; CKD, chronic kidney disease; CVE, cerebrovascular event; DM, diabetes mellitus; HTN, hypertension; IQR, interquartile range; MAP, mean arterial pressure; SD, standard deviation.

(26.4% vs. 14.2%), and greater prevalence of volume overload (16.5% vs. 10.0%). Interestingly, CKD was less prevalent in the HyperK group (13.2% vs. 21.7%). Other differences included higher rates of acid–base disorders (15.9% vs. 7.7%) and a modestly elevated BMI (26.1 vs. 25.0 kg/m$^2$), $p$ for all $<0.05$. To predict the development of HyperK, a multivariate logistic regression model identified five independent predictors: vasopressor use (OR 1.62; 95% CI: 1.00–2.62), presence of shock (OR 1.73; 95% CI: 1.00–2.97), urinary tract obstruction (OR 1.87; 95% CI: 1.04–3.37), lower hemoglobin levels (OR 0.59; 95% CI: 0.36–0.98), and elevated baseline potassium (OR 2.45; 95% CI: 1.42–4.19), Table 2. Despite slightly better AIC and BIC values in the univariate model, the multivariate model was selected for its superior clinical applicability (AIC 795.01; BIC 850.49). The model's diagnostic performance, summarized in Supplemental Table 1, showed an accuracy of 77%, sensitivity of 58%, specificity of 78%, and a high negative predictive value of 97%, though with a modest AUC of 0.68 (Fig. 2). These findings support the model's use as a triage tool to rule out HyperK in low-risk individuals. Based on the multivariate model, a nomogram was developed to estimate individualized HyperK risk (Fig. 3). This tool assigns weighted point values to each significant predictor, allowing clinicians to compute a total score and translate it into a predicted probability. For example, a patient presenting with vasopressor use, urinary obstruction, low hemoglobin, and borderline potassium values would accumulate a higher score, reflecting a higher estimated risk of HyperK. The point allocation for each variable is detailed in Supplemental Table 1. The nomogram's high negative predictive value and ease of use make it particularly suitable for rapid clinical screening.

*Secondary objectives: prediction of MAKE and component outcomes by sex*

*In male patients*

To assess the development of MAKE and its individual components in male patients, four distinct multivariate models and corresponding nomograms were constructed (Supplemental Fig. 1A–D). The MAKE

**Table 2**
Factors associated with HyperK in AKI patients by univariable and multivariable analyses.

| Variables | Univariate logistic regression | | Multivariate logistic regression | |
|---|---|---|---|---|
| | OR (95% CI) | $p$ | OR (95% CI) | $p$ |
| CKD | 0.54 (0.33, 0.86) | 0.013 | 0.60 (0.34, 1.05) | 0.077 |
| NSAID | 0.70 (0.46, 1.05) | 0.093 | 0.68 (0.42, 1.10) | 0.118 |
| Diuretics | 1.23 (0.87, 1.74) | 0.234 | 1.35 (0.89, 2.05) | 0.157 |
| Vasopressor | 2.06 (1.44, 2.93) | 0.000 | 1.62 (1.00, 2.62) | 0.050 |
| Shock | 2.16 (1.43, 3.24) | 0.000 | 1.72 (1.00, 2.96) | 0.050 |
| Urinary obstruction | 1.31 (0.79, 2.10) | 0.271 | 1.86 (1.04, 3.36) | 0.036 |
| Metabolic acidosis | 2.27 (1.36, 3.73) | 0.001 | 1.44 (0.78, 2.71) | 0.245 |
| Fluid overload | 1.78 (1.09, 2.85) | 0.018 | | |
| Uremia | 1.57 (0.97, 2.47) | 0.056 | 1.50 (0.87, 2.60) | 0.139 |
| BMI | 1.02 (0.99, 1.05) | 0.089 | 0.69 (0.46, 1.04) | 0.077 |
| Weight | 1.00 (0.99, 1.01) | 0.065 | | |
| Hemoglobin | 1.03 (0.99, 1.09) | 0.114 | 0.59 (0.35, 0.97) | 0.040 |
| Potassium | 2.20 (1.68, 2.90) | 0.000 | 2.44 (1.42, 4.19) | 0.001 |
| Constant | 0.36 (0.19, 0.64) | 0.001 | | |

BMI, body mass index; CKD, chronic kidney disease; NSAID, non-steroidal anti inflammatory drug.

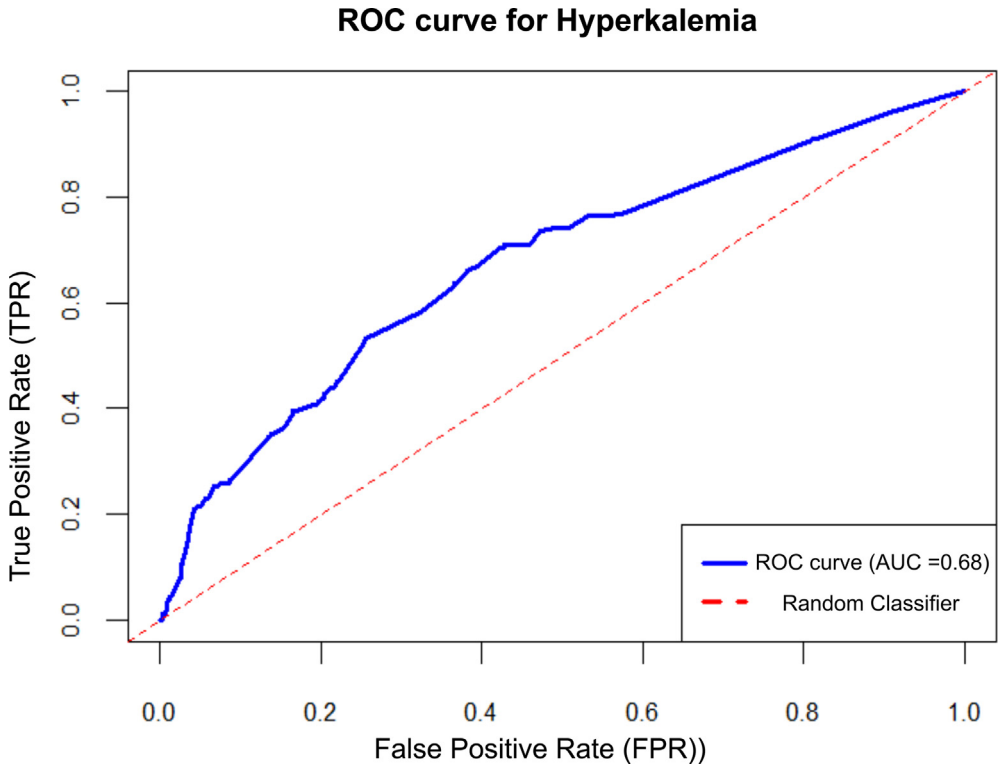
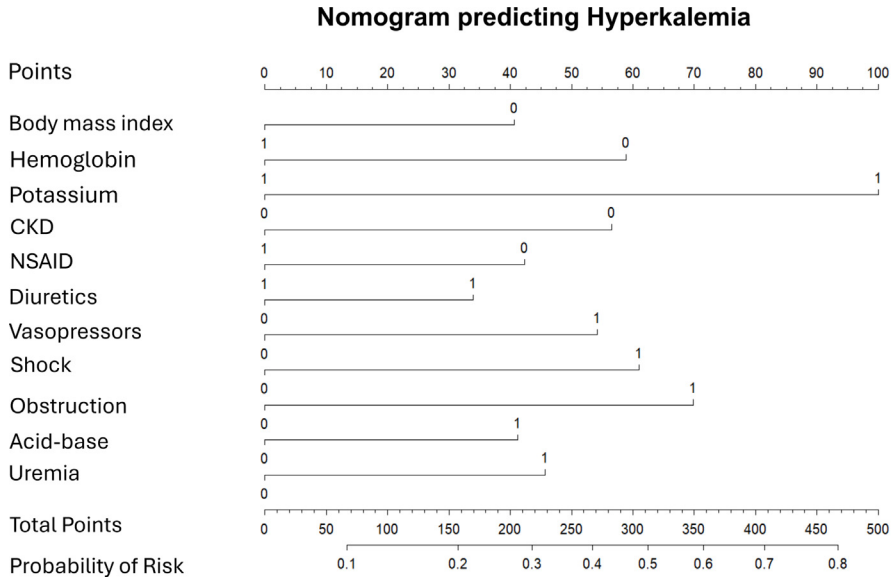



**Fig. 2.** ROC curve predict HyperK in all patients.




| Total points | Risk probability |
|---|---|
| 112 | 0.15 |
| 157 | 0.2 |
| 188 | 0.25 |
| 218 | 0.3 |
| 242 | 0.35 |
| 267 | 0.4 |
| 290 | 0.45 |
| 312 | 0.5 |
| 335 | 0.55 |
| 358 | 0.6 |
| 382 | 0.65 |
| 407 | 0.7 |
| 437 | 0.75 |
| 467 | 0.8 |

**Fig. 3.** Nomogram predicting HyperK.

model identified elevated serum creatinine at admission (OR 4.28; 95% CI: 2.34–7.84), uremia (OR 11.76; 95% CI: 1.40–90.99), and cardiopathy (OR 10.01; 95% CI: 1.10–82.40) as strong independent predictors, while non-steroidal anti-inflammatory drugs (NSAID) use was associated with lower risk (OR 0.41; 95% CI: 0.22–0.75). This model demonstrated excellent sensitivity (96.5%), good accuracy (82.9%), and an AUC of 0.80. The associated nomogram translates these variables into an individualized risk estimate (Supplemental Fig. 1A), providing a reliable tool for early detection of high-risk patients. The in-hospital mortality model identified vasopressor use (OR 2.34; 95% CI: 1.30–4.21), elevated serum creatinine (OR 2.11; 95% CI: 1.08–4.11), and older age (OR 1.89; 95% CI: 1.06–3.37) as significant contributors to death. Despite low sensitivity (19.8%), the model achieved high specificity (95.8%) and an AUC of 0.77, making the mortality nomogram (Supplemental Fig. 1B) particularly effective in identifying survivors. The need for KRT prediction model incorporated nine predictors. The strongest were acid–base disturbances (OR 87.20; 95% CI: 5.71–12.85), volume overload (OR 24.95; 95% CI: 1.42–38.00), HyperK (OR 10.07; 95% CI: 10.87–88.22), uremia (OR 5.22; 95% CI: 2.45–11.70), and hypernatremia (OR 4.63; 95% CI:

8.00–34.40). Additional factors included elevated creatinine (OR 7.39; 95% CI: 1.57–34.54), respiratory rate (OR 4.18; 95% CI: 1.26–13.91), serum glucose (OR 3.19; 95% CI: 1.02–9.99), and low platelet count (OR 0.27; 95% CI: 0.08–0.93). The nomogram (Supplemental Fig. 1C) achieved excellent performance (AUC 0.98; accuracy 94.8%), confirming its utility for clinical decision-making. For the prediction of eGFR decline ≥25 mL/min/1.73 $m^2$ significant predictors included baseline creatinine (OR 3.70; 95% CI: 2.14–6.40), acid–base disturbances (OR 5.38; 95% CI: 1.53–19.20), serum chloride (OR 2.21; 95% CI: 1.10–4.44), and uremia (OR 2.53; 95% CI: 1.00–6.44). The nomogram (Supplemental Fig. 1D) achieved an AUC of 0.75 and a sensitivity of 93.8%, offering reliable identification of patients at risk of functional deterioration.

*In female patients*

Among women, the MAKE nomogram (Supplemental Fig. 2A) was developed from a model that identified volume overload (OR 13.43; 95% CI: 1.08–141.96), elevated urea (OR 5.75; 95% CI: 1.90–17.37), creatinine (OR 3.27; 95% CI: 1.46–7.31), and increased heart rate (OR 2.99; 95% CI: 1.17–7.66) as major predictors. NSAID use was again associated with lower risk (OR 0.37; 95% CI: 0.16–0.85). This model demonstrated an AUC of 0.79, high sensitivity (96.7%), and a robust F1 score (0.91), suggesting excellent detection capacity. The mortality model (Supplemental Fig. 2B) highlighted the following independent predictors: shock (OR 11.01; 95% CI: 3.61–33.60), hyperbilirubinemia (OR 26.97; 95% CI: 1.12–58.25), sepsis (OR 5.00; 95% CI: 2.14–11.66), age (OR 2.74; 95% CI: 1.16–6.47), cerebrovascular disease (OR 7.56; 95% CI: 1.55–37.12), and elevated heart rate (OR 5.07; 95% CI: 1.40–18.43). Protective associations were observed for lower BMI (OR 0.41; 95% CI: 0.19–0.91) and diabetes (OR 0.36; 95% CI: 0.15–0.85). The model achieved an AUC of 0.84, with excellent specificity (95.3%). The need for KRT model (Supplemental Fig. 2C) identified acid–base disorders (OR 34.00; 95% CI: 25.68–39.47), volume overload (OR 33.81; 95% CI: 32.86–293.38), HyperK (OR 22.26; 95% CI: 17.50–24.93), uremia (OR 34.33; 95% CI: 8.03–133.00), and elevated platelet count (OR 4.26; 95% CI: 1.17–15.59) as key predictors. The nomogram performed exceptionally well (AUC 0.96; accuracy 95.2%; F1 score 0.92). Finally, the model for eGFR decline ≥25 mL/min/1.73 $m^2$ in women included baseline creatinine as the only statistically significant predictor (OR 3.39; 95% CI: 1.75–6.58). Although simplified, the nomogram (Supplemental Fig. 2D) showed solid predictive performance (AUC 0.74), and sensitivity of 93.9%, making it a practical tool for early identification of renal deterioration.

## Discussion

In this cohort, we developed and validated predictive nomograms for HyperK, and MAKE in hospitalized patients with AKI. Using routinely collected clinical variables at the time of nephrology consultation, we constructed logistic regression models stratified by sex and evaluated their predictive performance across multiple outcomes. The nomograms demonstrated strong discriminative capacity, particularly among women. While the model's capacity to predict HyperK was modest, its high negative predictive value suggests that it may be especially useful to identify patients at low risk. These findings underscore the clinical relevance of early risk stratification using predictive tools to guide timely intervention in AKI management and support individualized decision-making in clinical practice. Sex-stratified analyses were conducted a priori to account for known biological and clinical differences between men and women in AKI. Stratification allowed the development of sex-specific prediction models with greater clinical interpretability and bedside applicability.

This baseline characteristics highlights that while many variables were comparable between AKI patients who developed HyperK had higher rates of metabolic acidosis, volume overload, shock, vasopressor use, and slightly elevated BMI. These findings suggest that HyperK in AKI may be a marker of more severe or complex clinical presentations, emphasizing the need for closer monitoring and targeted interventions in this subgroup. The final multivariable model identifies hemodynamic instability (including shock and vasopressor use) and obstructive uropathy as key independent clinical predictors of HyperK in patients with AKI. Vasopressor use likely reflects a more severe hemodynamic compromise contributing to impaired renal potassium excretion[19] as well as shock consistent with impaired perfusion, acidosis, and cell lysis.[20] Obstruction significantly increased the odds of HyperK, possibly due to decreased tubular excretion and back-pressure on tubular function.[10] Our model demonstrates modest discriminative ability performance is better than chance (AUC 0.68), from a clinical standpoint, this model is more reliable for excluding HyperK than for confirming it. It is very high negative predictive value (≈97%) means that when the model classifies a patient as low risk, true HyperK is unlikely, useful in high-volume emergency or critical care settings to prioritize laboratory confirmation. However, the low precision means that many model-flagged "at-risk" patients will not actually be hyperkalemic; thus, model-positive results must be validated with serum potassium measurement before treatment decisions. Given the moderate AUC and only fair sensitivity, clinicians should not rely on the model to catch all hyperkalemic cases, especially in hemodynamically unstable patients or those receiving vasoactive drugs, where the clinical threshold for testing should remain low.

Several studies have evaluated the risk of developing HyperK in the context of AKI. In patients presenting to the emergency department, the prevalence of HyperK in those with AKI was 13%. Risk factors included AKI stage, use of potassium-sparing diuretics, ACE inhibitors, and underlying CKD. Furthermore, HyperK was associated with increased in-hospital mortality and a longer hospital stay.[9] Our group demonstrated in a prospective cohort that the trajectory of serum potassium during hospitalization for AKI has a significant prognostic impact; the transition from normokalemia to HyperK was associated with an increased risk of in-hospital mortality.[21] In patients with AKI and sepsis, elevated admission potassium levels (≥4.5 mmol/L) were associated with a significant increase in 30-day ICU mortality.[22] Unlike previous studies that primarily focused on the prevalence, clinical correlates, and prognostic implications of HyperK in AKI, our manuscript advances the field by constructing and validating predictive nomogram specifically designed to estimate the individual risk of developing HyperK, our approach integrates routinely available clinical variables into predictive models with demonstrable discriminative power, supporting early triage and targeted intervention in AKI patients.

Multiple studies have evaluated the risk of developing MAKE in patients with AKI. Recent meta-analyses and systematic reviews identify heterogeneous risk of MAKE after AKI, which depends on factors such as the stage and duration of AKI, recovery of renal function, and underlying comorbidities. However, the association is consistent across all clinical scenarios evaluated.[23–25] In recent years, several nomograms have been developed to predict the risk of MAKE in patients with AKI; in patients with sepsis and type 2 diabetes, a nomogram has demonstrated excellent discriminatory ability to predict MAKE at 30 days, with an AUC of 0.91 and good calibration.[26] While recent studies, such as that by Xin and colleagues, have developed nomograms with excellent discriminative capacity for predicting 30-day MAKE in specific high-risk populations, our study expands the applicability of predictive modeling by including a broader cohort of hospitalized patients with AKI, regardless of etiology. Unlike Xin's model, which incorporates biomarkers such as cystatin C, HDL, and apolipoprotein E, parameters not routinely

available in all clinical settings, our nomograms rely on widely accessible clinical and biochemical variables present at the time of nephrology consultation. Although the AUC of our MAKE prediction model was slightly lower (0.79 in women and 0.80 in men), the models demonstrated good calibration and strong sensitivity, supporting their practical use in real-time clinical decision-making. Furthermore, our study uniquely stratified predictions by sex and concurrently addressed related outcomes such as HyperK and KRT initiation, offering a more comprehensive tool for risk stratification in AKI.

Predicting the need for KRT in patients with AKI remains largely based on classic clinical and laboratory criteria as established in international clinical trials and guidelines.[27,28] Several new biomarkers have been evaluated to predict the need for KRT, including NGAL (blood and urine), cystatin C, interleukin-18, and the TIMP-2 × IGFBP7 product, with areas AUCs ranging from 0.67 to 0.86.[29,30] Their clinical utility remains limited due to moderate discriminative performance (AUC 0.67–0.86), lack of standardized thresholds, and confounding by comorbidities. In contrast, our study offers a pragmatic and robust alternative by constructing nomograms based solely on routinely available clinical data collected at the time of nephrology consultation. Our model achieved excellent predictive accuracy for KRT initiation (AUC 0.96), with high sensitivity (88.7%) and specificity (98.5%), surpassing the performance typically reported for biomarker-based models.

There are multiple nomograms and mortality prediction models in patients with AKI, especially in the context of critically ill patients and those undergoing CRRT. The nomogram developed by Wang et al. integrates variables at the start of CRRT, showing excellent discriminatory capacity to predict 90-day mortality (C-index 0.810).[31] Other models, such as Zeng et al., with good performance in predicting 28-, 56-, and 84-day mortality in patients with AKI on CRRT (AUC 0.77–0.80).[32] In specific populations, such as cirrhotic patients with AKI predicting 90- and 180-day mortality.[33] In addition, nomograms exist for subgroups such as sepsis-associated AKI and for predicting long-term survival in the ICU.[34–36] These models often rely on complex variables such as SOFA scores, phosphate levels, and serial laboratory data collected after CRRT initiation. In contrast, our study offers a broader and more pragmatic approach by developing prediction tools applicable to general hospitalized AKI patients prior to KRT initiation. Our nomograms rely exclusively on routinely available clinical and biochemical variables and demonstrated excellent discriminative performance, particularly for predicting KRT (AUC 0.96) and MAKE (AUC 0.79–0.80). Moreover, by stratifying analyses by sex and incorporating outcomes beyond mortality, including HyperK and eGFR decline, our models provide a comprehensive and actionable framework for early risk stratification, without the need for ICU-specific scores or delayed variables.

Our results should be interpreted with their limitations, the most relevant of which is the single-center design, which may limit the generalizability of the predictive models to other healthcare settings with different patient populations, resource availability, and clinical practices. The lack of external validation, although the models were internally validated, they have not yet undergone external validation in independent cohorts. Information regarding exposure to renin angiotensin aldosterone system inhibitors was not available in our dataset; therefore, we were unable to account for their potential influence on the development of HyperK. The exclusion of patients without nephrology consultation, which may introduce selection bias and limit applicability in broader AKI populations. The absence of dynamic or time-dependent variables, the models rely exclusively on static variables obtained at the time of consultation. They do not incorporate dynamic laboratory trends. The omission of urine output criteria may have led to misclassification of AKI severity, particularly in patients with oliguric or anuric presentations, and could have influenced the estimation of HyperK risk. The lack of stratification by KDIGO AKI stages represents an important limitation, as this classification is closely related to disease severity and the initiation KRT. The limited inclusion of biomarkers, while the manuscript emphasizes using routinely available variables, the exclusion of novel or emerging biomarkers (e.g., NGAL, TIMP-2 × IGFBP7, cystatin C) may reduce comparative predictive precision relative to more sophisticated models. Stratified analyses by CKD stage were descriptive and underpowered, limiting our ability to draw definitive conclusions regarding differential HyperK risk across CKD stages. The imbalanced outcome frequencies (e.g., low mortality rate), which may affect the reliability and calibration of those specific models.

This study possesses strengths. Clinically, the predictive nomograms were developed using routinely available variables at the time of nephrology consultation, allowing for immediate applicability in real-world settings without the need for specialized biomarkers or advanced technologies. Unlike previous models focused solely on mortality or dialysis initiation, our study addresses a broader spectrum of clinically relevant outcomes, including HyperK, KRT, and MAKE. Additionally, the stratified analysis by sex accounts for potential biological and clinical differences between male and female patients, enhancing the precision of risk prediction. The study also employed robust internal validation through a comprehensive set of performance metrics, including accuracy, predictive values, F1 score, and confusion matrices.

## Conclusion

We developed and internally validated clinically applicable nomograms for predicting HyperK, the need for KRT, and MAKE in patients with AKI. By relying solely on readily available clinical and laboratory data at the time of nephrology consultation, these models offer accurate, timely, and personalized risk stratification. Their strong discriminative performance and ease of use support their potential integration into routine clinical practice to enhance decision-making and improve outcomes in AKI management.

## CRediT authorship contribution statement

JSCI designed the study, analysed the data, and wrote and supervised the manuscript. JSCA, GRG, GNB, RMG, AMGG, LAV, GJAM, JAGF recollected data, analysed, and wrote the manuscript. GGG, PCL, EJS wrote and supervised the manuscript. All authors agree to be accountable for all aspects of the work.

## Ethics approval and consent to participate

The study was approved by the Hospital Civil de Guadalajara Fray Antonio Alcalde Institutional Review Board (CE 180/24). All participants gave their written informed consent. The study was conducted following the ethical principles outlined in the Declaration of Helsinki.

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## Conflict of interest

JSCI participates as a speaker for AstraZeneca, Boehringer, Bayer, Novo Nordisk, Vantive, and Amgen. PCL and EJS are employees of

Astra Zeneca México. The remaining authors report that they have no conflicts of interest.

## Data availability

The files and data are in the physical and electronic archives of the Civil Hospital of Guadalajara Fray Antonio Alcalde and can be requested with prior authorization. All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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## Appendix A. Supplementary data

Supplementary data associated with this article can be found in the online version available at https://doi.org/10.1016/j.nefro.2026.501489.

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