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Comparison of Outcomes of Mild and Severe Community- and Hospital-Acquired Acute Kidney Injury

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Purpose: Acute kidney injury (AKI) has shown an increasingly common occurrence among hospitalized patients worldwide. We determined the incidence and compared the short- and long-term outcomes of all stages of community-acquired AKI (CA-AKI) and hospital-acquired AKI (HA-AKI), and identified predictors for such outcomes.
Materials and Methods: This observational, single-center, retrospective study identified patients admitted between January 2013 and December 2013 who developed CA-AKI or HA-AKI. Short- and long-term patient and renal outcomes were analyzed.
Results: AKI incidence was 14.3% (1882, CA-AKI 4.8% and HA-AKI 9.5%). The highest 30-day and 1-year mortality were recorded in the CA-AKI group. Thirty-day mortality rate was 11.4% in CA-AKI group and 5.7% in HA-AKI group (p<0.001). One-year mortality rates were 20.1% and 13.3%, respectively (p<0.001). More CA-AKI patients developed kidney failure with replacement therapy within 1 year (27, 4.3% vs. 18, 1.4% respectively, p<0.001).
Conclusion: In conclusion, patients with CA-AKI had worse short- and long-term outcomes compared to HA-AKI patients. AKI severity and discharge serum creatinine were significant independent predictors of 30-day and 1-year mortality.

Key Words: Acute kidney injury, community-acquired, hospital-acquired, mortality, outcomes

INTRODUCTION

Acute kidney injury (AKI), characterized by an abrupt decline in kidney function, is becoming an increasingly common and potentially fatal complication.[1] AKI may be present at admission or develop during hospitalization.[2] AKI is reported in 5%–15% of hospitalized patients,[3-6] and 7%–52% of critically ill patients.[7-12] AKI carries a significant risk of adverse outcomes, such as prolonged hospital stay, increased healthcare costs,[2,9,13-15] development of chronic kidney disease (CKD), and increased early and late mortality.[2,14-16]

Current studies on AKI have focused on short-term outcomes, such as in-hospital mortality;[15,17-21] and special groups of patients, such as those with severe,[10,19] post-operative,[22] or hospital-acquired AKI (HA-AKI),[3,19] and those in intensive care unit settings.[7,10-12,17,21,23] Little is known about community-acquired AKI (CA-AKI) and how it differs from HA-AKI. Few studies have compared their outcomes,[4-6,20,24-28] and even fewer studies have established clinical significance of less severe AKI.[29,30] The present study aimed to compare the incidence, short- and long-term outcomes of all stages of CA-AKI and HA-AKI, and identify the predictors of such outcomes.

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•The authors have no potential conflicts of interest to disclose.

MATERIALS AND METHODS

This is an observational, single-center, retrospective study performed at Seoul National University Bundang Hospital (SNUBH) involving all hospitalized adult AKI patients from January 2013 to December 2013. Patients with CKD stage 5, on kidney replacement therapy (KRT), or with history of kidney transplantation were excluded from this study.

The present study was approved by the SNUBH Institutional Review Board (B-1603-340-117) and performed in accordance with the Declaration of Helsinki. Since the study was retrospective and non-interventional in nature, informed consent was waived.

Baseline demographics and clinically relevant variables such as comorbidities and serum creatinine (SCr) at different time points were retrieved from electronic medical records. SCr was measured at the time of admission and during hospitalization whenever deemed necessary by the medical team handling the patients. Glomerular filtration rate was estimated using the chronic kidney disease epidemiology collaboration (CKD-EPI) creatinine equation. Other laboratory test results were also collected.

Primary outcomes were 30-day and 1-year mortality. Secondary outcomes were short- and long-term renal survival.

Definitions

AKI diagnosis was made in accordance with the 2012 Kidney Disease Improving Global Outcomes (KDIGO) AKI Guideline: 1) increase in SCr by ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours; or 2) increase in SCr to ≥1.5x baseline, which is known or presumed to have occurred within the prior 7 days.[31] AKI was staged according to the KDIGO criteria-stage 1: increase in SCr 1.5–1.9x the baseline OR ≥0.3 mg/dL (≥26.5 μmol/L) increase; stage 2: 2.0–2.9x the baseline; stage 3: 3.0x the baseline OR increase in SCr ≥4.0 mg/dL (≥353.6 μmol/L) OR initiation of KRT.[31] AKI was classified as CA-AKI when the initial SCr fulfilled the KDIGO AKI definition upon admission or within 24 hours of admission; and as HA-AKI when AKI diagnosis was made after 24 hours of admission.

Baseline SCr was the latest available SCr recorded within preceding 12 months. Initial SCr was the SCr upon AKI diagnosis. Maximum SCr was the highest SCr reached during AKI episode. Discharge SCr was the latest SCr measured prior to discharge from the hospital.

Short-term renal survival was defined as the dialysis independence at discharge, while long-term renal non-survival was the development of kidney failure with replacement therapy (KFRT) within 1 year of AKI diagnosis.

Statistical analysis

The median and interquartile range were reported for continuous variables, while frequency and percentage were reported for categorical data. Comparisons were made using Mann-Whitney U-test. Cox proportional hazard regression models were utilized to explore effects of variables and AKI severity on mortality. Known risk factors for adverse outcomes for kidney failure-age, sex, comorbidities, and baseline kidney function-were included in the adjusted model. Nephrology referral, defined as whether or not the patient was evaluated and managed by the Nephrology Service of SNUBH, was also included. Log-rank test was used to compare the survival estimated by Kaplan-Meier curves. To obtain the diagnostic discrimination abilities, the measures of kidney function at different time points were subjected to area under the curve (AUC) analysis of receiver operating characteristic (ROC) curves. The ROC analysis results were interpreted as follows: AUC 0.70–0.80, acceptable diagnostic accuracy and AUC 0.81–0.90, excellent diagnostic accuracy. For all analyses, p value<0.05 was considered statistically significant. SPSS version 20 (IBM Corp., Armonk, NY, USA) was used for all statistical analyses.

RESULTS

AKI incidence was 14.3% (1882/13192; CA-AKI 4.8% and HA-AKI 9.5%). One-third of AKI patients had CA-AKI (633/1882, 33.6%), while the rest developed AKI during hospital stay (1249/1882, 66.4%). More than two-thirds of patients in each group had stage 1 AKI (CA-AKI 421, 66.5% and HA-AKI 967, 77.4%). The remaining one-third of CA-AKI patients had more severe AKI (stages 2 and 3, 212, 33.5%). Severe AKI was less common in the HA-AKI group (stage 2 and 3, 282, 22.6%). Table 1 shows the baseline characteristics according to AKI classification.

The highest 30-day and 1-year mortality were recorded in the CA-AKI group. Thirty-day mortality rate was 11.4% in the CA-AKI group and 5.7% in the HA-AKI group (p<0.001). One-year mortality rates were 20.1% and 13.3%, respectively (p< 0.001). There was no significant difference in the short-term renal survival (CA-AKI 15, 2.4% and HA-AKI 15, 1.2%, p=0.056), but more patients in the CA-AKI group developed KFRT within 1 year (27, 4.3% vs. 18, 1.4% respectively, p<0.001). Table 2 summarizes the outcomes of CA-AKI and HA-AKI.

AKI severity was an independent predictor of short- and long-term mortality. An increasing mortality risk was noted along with increasing severity of AKI. Results of Cox proportional hazard regression model adjusted for age, sex, comorbidities–diabetes mellitus (DM), hypertension, angina, heart failure, malignancy, baseline SCr, and nephrology referral–are presented in Tables 3 and 4. Compared to CA-AKI stage 1 patients, CA-AKI and HA-AKI stage 2 and 3 patients had higher mortality risk after adjustments for age, sex, malignancy, baseline estimated glomerular filtration rate (eGFR), and nephrology referral (Table 3). When compared to patients with stage 1 CA-AKI, an increasing 30-day mortality adjusted hazard ratio were observed in stage 2 and stage 3 CA-AKI patients [adjust-

Table 1. Baseline Characteristics and Laboratory Parameters

Characteristics	CA-AKI (n=633)	HA-AKI (n=1249)	*p* value
Demographics			
Age (yr)	69 (54–78)	66 (53–75)	0.016
Sex			0.002
Female	285 (45.0)	655 (52.4)	
Male	348 (55.0)	594 (47.6)	
AKI stage			0.001
1	421 (66.5)	967 (77.4)	
2	128 (20.2)	178 (14.3)	
3	84 (13.3)	104 (8.3)	
Baseline SCr (mg/dL)	0.8 (0.5–0.9)	0.8 (0.6–1.1)	<0.001
Baseline eGFR (mL/min/1.73 m^2)	85.8 (80.0–114.4)	90.7 (60.0–104.4)	0.001
Initial SCr (mg/dL)	1.4 (0.4–0.9)	0.5 (0.5–1.4)	<0.001
Initial eGFR (mL/min/1.73 m^2)	48.5 (81.4–121.1)	104 (45.2–108.0)	<0.001
Comorbidities			
Angina	9 (1.4)	26 (2.1)	0.317
Myocardial infarction	6 (0.9)	10 (0.8)	0.742
Heart failure	24 (3.8)	31 (2.5)	0.111
Hypertension	35 (5.5)	61 (4.9)	0.548
Diabetes mellitus	59 (9.3)	80 (6.4)	0.022
Malignancy	217 (34.3)	361 (28.9)	0.017
Referral to nephrology	98 (15.5)	161 (12.9)	0.123
Surgery	117 (18.5)	690 (55.2)	<0.001
Hemoglobin (g/dL)	11.3 (10.4–13.3)	11.8 (9.9–13.1)	0.001
Albumin (g/dL)	3.6 (3.1–4.0)	3.6 (3.1–4.0)	<0.001
Maximum SCr (mg/dL)	1.5 (0.7–1.4)	0.9 (0.7–2.0)	0.002
Discharge SCr (mg/dL)	1.0 (0.6–1.1)	0.8 (0.6–1.4)	<0.001
Hospital stay (days)	7 (6–17)	11 (6–17)	<0.001

CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI; eGFR, estimated glomerular filtration rate; SCr, serum creatinine.
Data expressed as median, interquartile range, or frequency (percentage).

Table 2. Short- and Long-Term Outcomes of CA-AKI and HA-AKI

	CA-AKI (n=633)	HA-AKI (n=1249)	*p* value
30-day mortality	72/561 (11.4)	71/1178 (5.7)	<0.001
1-year mortality	127/506 (20.1)	166/1083 (13.3)	<0.001
KRT on discharge	15 (2.4)	15 (1.2)	0.056
KFRT	27 (4.3)	18 (1.4)	<0.001

CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI; KRT, kidney replacement therapy; KFRT, kidney failure with replacement therapy.
Data expressed as mortality/non-mortality (percentage of mortality).

ed hazard ratio (aHR) 4.07, 95% confidence interval (CI) 2.20–7.51, p<0.001 and aHR 8.38, 95% CI 4.68–15.01, p<0.001, respectively]. The same was observed for stage 2 and 3 HA-AKI patients (aHR 3.54, 95% CI 1.91–6.54, p<0.001 and aHR 7.83, 95% CI 4.41–13.87, p<0.001). However, HA-AKI stage 1 patients had a lower risk for 30-day mortality compared to CA-AKI stage 1 patients (aHR 0.66, 95% CI 0.37–1.17, p=0.151).

Compared to CA-AKI, HA-AKI patients had lower risk of 30-day and 1-year mortality (aHR 0.48, p<0.001 and aHR 0.64, p=0.007) (Table 4).

The probability of 1-year survival was estimated using Kaplan-Meier analysis (Fig. 1). CA-AKI patients had worse survival compared to HA-AKI patients regardless of AKI severity. There were significant differences in survival between the AKI types and among the AKI stages (p<0.001).

Predictors for mortality

Of all markers of kidney function analyzed, discharge SCr was the most consistent predictor of 30-day mortality among CA-AKI patients (HR 1.76, 95% CI 1.34–2.30, p<0.001) and HA-AKI patients (HR 4.78, 95% CI 2.29–9.99, p<0.001). Discharge SCr was associated with 1-year mortality risk among CA-AKI patients (HR 1.75, 95% CI 1.36–2.26, p<0.001) and HA-AKI patients (HR 2.19, 95% CI 1.41–3.41, p<0.001).

The area under the ROC curve for discharge SCr was 0.835 (Fig. 2), indicating that it could be a good predictor of 30-day mortality. Among CA-AKI patients, a discharge SCr cut-off of 1.425 mg/dL predicted 30-day mortality with 68.1% sensitivity and 72.4% specificity, while a discharge SCr of 1.135 mg/dL in HA-AKI patients could predict 30-day mortality with 74.6% sensitivity and 78.2% specificity (Supplementary Table 1, only online).

Table 3. Cox Regression Analysis for 30-Day Mortality Risks at Different Stages of CA-AKI and HA-AKI

	HR (95% CI)	*p* value	aHR (95% CI)	*p* value
CA-AKI stage 1	(Reference)		(Reference)	
HA-AKI stage 1	0.49 (0.26–0.93)	0.028	0.66 (0.37–1.17)	0.151
CA-AKI stage 2	3.41 (1.77–6.58)	<0.001	4.07 (2.20–7.51)	<0.001
HA-AKI stage 2	2.47 (1.24–4.91)	0.010	3.54 (1.91–6.54)	<0.001
CA-AKI stage 3	7.95 (4.37–14.46)	<0.001	8.38 (4.68–15.01)	<0.001
HA-AKI stage 3	4.95 (2.62–9.35)	<0.001	7.83 (4.41–13.87)	<0.001

HR, hazard ratio; CI, confidence interval; aHR, adjusted hazard ratio; CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI.
Model adjusted for age, sex, malignancy, angina, heart failure, hypertension, diabetes mellitus, baseline serum creatinine, and nephrology referral.

Table 4. Cox Regression Analysis for 30-Day and 1-Year Mortality Risks between CA-AKI and HA-AKI

	30-day mortality			1-year mortality		
	No. of events	aHR (95% CI)	*p* value	No. of events	HR (95% CI)	*p* value
CA-AKI (n=633)	72 (11.1)	(Reference)		127 (20.1)	(Reference)	
HA-AKI (n=1249)	71 (2.7)	0.48 (0.34–0.70)	<0.001	166 (13.3)	0.64 (0.46–0.89)	0.007

HR, hazard ratio; CI, confidence interval; aHR, adjusted hazard ratio; CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI.
Data expressed as n (percentage). Model adjusted for age, sex, malignancy, angina, heart failure, hypertension, diabetes mellitus, baseline serum creatinine, and nephrology referral.

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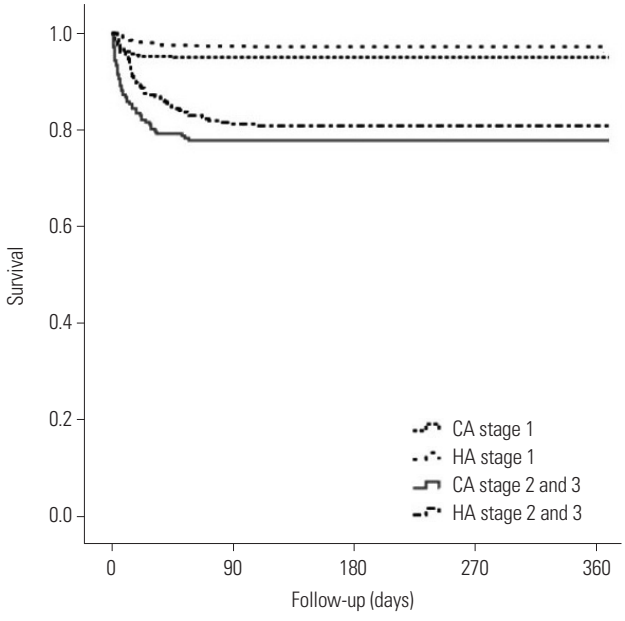
Renal recovery

As shown in Table 5, no difference was observed in short- and long-term renal survival between CA-AKI and HA-AKI (p=0.051 and 0.083 respectively). A significant predictor for short- and long-term renal non-recovery was discharge SCr (HR 2.54, 95% CI 2.31–2.79, p<0.001 and HR 2.55, 95% CI 2.36–2.76, p<0.001, respectively).

DISCUSSION

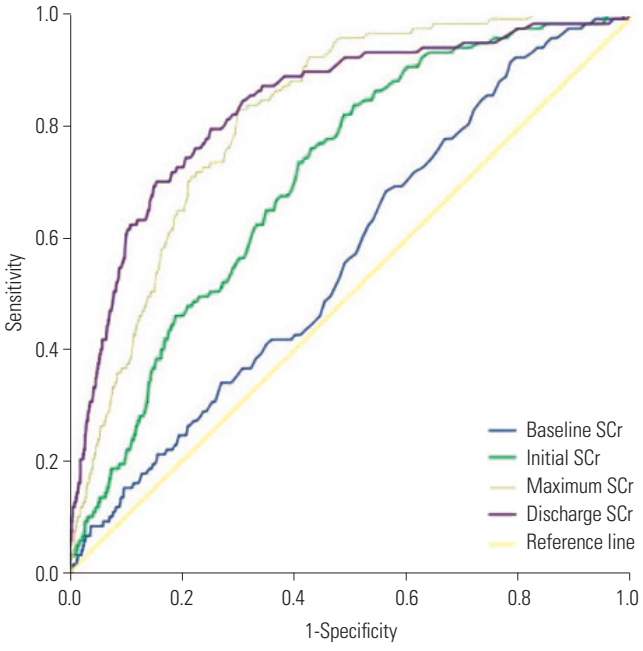
In the present study, approximately 14 of every 100 hospitalizations were complicated by AKI. The majority was HA-AKI (1249/1882, 66.4%). Xu, et al.,[20] in their analysis of 146148 hospitalizations in nine regional hospitals in China in 2013, reported a lower AKI incidence of 11.6% (HA-AKI 9.1% and CA-AKI 2.5%). This lower incidence can be attributed to their exclusion of CKD stage 4 and 5 patients. Comparable to our study, Aitken, et al.[5] reported a 15.5% incidence (HA-AKI 10.7% and CA-AKI 4.6%). These studies also showed higher prevalence of HA-AKI.

The results demonstrated that AKI conveyed a substantial risk of death and renal non-survival for at least 1 year beyond the diagnosis. The severe CA-AKI patients had the highest risk of mortality. Worse survival outcomes for CA-AKI can be explained by more severe AKI observed in this group. These patients also had lower serum albumin, which reflects a chronic condition, such as malnutrition or a severe acute disease process, both of which make patients more susceptible to complications and death. Moreover, more patients in the CA-AKI group had pre-existing DM and malignancy. Previous AKI studies found DM, malignancy, and increasing number of comorbidities to be related to increased mortality.[4,6,23]



Patients-at-risk					
CA-AKI stage 1	421	367	335	327	312
HA-AKI stage 1	967	904	866	848	824
CA-AKI stage 2&3	212	148	136	132	125
HA-AKI stage 2&3	282	207	197	193	188

Fig. 1. Kaplan-Meier survival curve comparing 1-year survival rates of patients with stage 1 and stage 2 and 3 community-acquired (CA)-AKI and hospital-acquired (HA)-AKI (log-rank test p<0.001). AKI, acute kidney injury.



	AUC	p
Baseline SCr	0.566	0.016
Initial SCr	0.709	<0.0001
Maximum SCr	0.816	<0.0001
Discharge SCr	0.835	<0.0001

Fig. 2. Receiver operating characteristic curves for SCr in AKI when predicting 1-year mortality. AUC, area under the curve; AKI, acute kidney injury; SCr, serum creatinine.

Table 5. Cox Regression Analysis for Renal Recovery between CA-AKI and HA-AKI

	Short-term renal recovery			Long-term renal recovery		
	No. of events	aHR	p value	No. of events	aHR (95% CI)	p value
HA-AKI (n=1249)	15 (1.2)	(Reference)		18 (1.4)	(Reference)	
CA-AKI (n=633)	15 (2.4)	2.14 (1.00–4.61)	0.051	27 (4.3)	1.76 (0.93–3.32)	0.083

HR, hazard ratio; CI, confidence interval; aHR, adjusted hazard ratio; CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI.
Data expressed as n (percentage). Model adjusted for age, sex, malignancy, angina, heart failure, hypertension, diabetes mellitus, baseline serum creatinine, and nephrology referral.

Although the development of AKI is known to increase the mortality risk, few studies have examined AKI severity as a mortality predictor.[2,14,20] A valuable finding of this study is that AKI severity corresponds to a proportional increase in short- and long-term mortality risk independent of age, sex, comorbidities, baseline renal function, and nephrology referral.

The results also showed that discharge SCr is a significant predictor of short- and long-term patient and renal outcomes. These findings place emphasis on continuous kidney function monitoring by assessing the clinical condition, urine output, and SCr levels during hospitalization. Measurements should be made to optimize renal recovery while patient is hospitalized through heightened attention to fluid and hemodynamic status, avoidance of nephrotoxic substances, and timely nephrology consultation. It is crucial to check kidney function prior to discharge, as long-term AKI management is more effective if patients who are most at risk for complications are identified at hospital discharge.

We reported a lower nephrology referral rate (258/1882, 13.8%) compared to the 72% reported by Aitken, et al.[5] The reason for this could be that the majority of AKI cases in this study were mild (73.8%). However, we found that even mild AKI or small increases in SCr can lead to significant adverse outcomes; therefore, an earlier nephrology referral should be practiced.

In conclusion, CA-AKI patients had worse survival compared to HA-AKI patients. Increased mortality risk was directly related to AKI severity. To date, data on mild AKI and its implications are scarce. The present study demonstrated that small increases in SCr can also lead to significant increase in mortality risk, and therefore, early intervention is warranted.

Given the long-term consequences of AKI, the goals should be early identification and management of AKI risk factors, as well as a timely diagnosis of AKI through accurate urine output and sensible renal function monitoring during hospitalization. Monitoring of kidney function should likewise extend beyond hospital discharge, as our data has shown long-term effects of AKI. Patients who develop AKI regardless of severity should be followed-up to improve long-term outcomes, and also be monitored for the development of CKD and its subsequent complications.

Most original articles on AKI have focused on highly-selected patients or specific conditions. The present study included a large number of hospitalized patients over a 1-year period; therefore, our results provide a better reflection of the true burden of AKI. Since we included all AKI patients regardless of severity, our findings may be generalizable to all AKI patients. A single laboratory was utilized, thereby limiting inter-laboratory errors. Outcome monitoring was done beyond hospitalization, and extended until 1 year after AKI diagnosis.

The limitations of our study include its retrospective and observational nature; as a result, this study could only predict the association between variables and outcomes, and not establish causal relationships. Also, due to its retrospective nature, this study could not completely identify patients with pre-existing CKD; however, having enrolled only patients with baseline SCr, we posed that no event happened during the interval between the latest SCr measurement prior to hospitalization and admission date, and used baseline SCr-based eGFR as marker for the diagnosis of pre-existing CKD. Moreover, we defined AKI based on SCr levels and excluded the urine output criteria, which might have led to an underestimation of AKI. There were no data on the exact causes of AKI, as well as the nature or extent of surgery and malignancy. However, we included some categorical data which roughly assumed potential causes of AKI, such as presence of infection and surgical history. Being a single-center study involving a tertiary hospital limits the external validity of this study, since behavior may vary according to the institution or region.

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AUTHOR CONTRIBUTIONS

Conceptualization: Ho Jun Chin, Ki Young Na, Dong-Wan Chae, and Sejoong Kim. Data curation: Kristianne Rachel Palanca Medina, Ji Won Ryu, and Eunjeong Kang. Formal analysis: Kristianne Rachel Palanca Medina, Ji Won Ryu, and Eunjeong Kang. Funding acquisition: Sejoong Kim. Investigation: Ji Won Ryu and Eunjeong Kang. Methodology: Kristianne Rachel Palanca Medina and Eunjeong Kang. Project administration: Sejoong Kim. Resources: Ho Jun Chin, Ki Young Na, Dong-Wan Chae, and Sejoong Kim. Software: Kristianne Rachel Palanca Medina and Sejoong Kim. Supervision: Sejoong Kim. Validation: Kristianne Rachel Palanca Medina, Jong Cheol Jeong, and Sejoong Kim. Visualization: Kristianne Rachel Palanca Medina and Sejoong Kim. Writing—original draft: Kristianne Rachel Palanca Medina and Jong Cheol Jeong. Writing—review & editing: all authors. Approval of final manuscript: all authors.

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