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Risk factors for acute kidney injury in critically ill patients with torso injury

A retrospective observational single-center study

Young Hoon Sul, MD, PhD[a,b], Jin Young Lee, MD[a,*], Se Heon Kim, MD[a], Jin Bong Ye, MD[a], Jin Suk Lee, MD[a], Su Young Yoon, MD[c], Jung Hee Choi, MD[d]

Abstract

Acute kidney injury (AKI) is common in trauma patients and associated with poor outcomes. Identifying AKI risk factors in trauma patients is important for risk stratification and provision of optimal intensive care unit (ICU) treatment. This study identified AKI risk factors in patients admitted to critical care after sustaining torso injuries.

We performed a retrospective chart review involving 380 patients who sustained torso injuries from January 2016 to December 2019. Patients were included if they were aged >15 years, admitted to an ICU, survived for >48 hours, and had thoracic and/or abdominal injuries and no end-stage renal disease. AKI was defined according to the Kidney Disease Improving Global Outcomes definition and staging system. Clinical and laboratory variables were compared between the AKI and non-AKI groups (n = 72 and 308, respectively). AKI risk factors were assessed using multivariate logistic regression analysis.

AKI occurred in 72 (18.9%) patients and was associated with higher mortality than non-AKI patients (26% vs 4%, $P < .001$). Multivariate logistic regression analysis identified bowel injury, cumulative fluid balance >2.5 L for 24 hours, lactate levels, and vasopressor use (adjusted odds ratio: 2.953, 2.058, 1.170, and 2.910; 95% confidence interval: 1.410–6.181, 1.017–4.164, 1.019–1.343, and 1.414–5.987; $P = .004$, .045, .026, and .004, respectively) as independent risk factors for AKI.

AKI in patients admitted to the ICU with torso injury had a substantial mortality. Recognizing risk factors at an early stage could aid risk stratification and provision of optimal ICU care.

Abbreviations: AKI = acute kidney injury, ICU = intensive care unit, ISS = injury severity score, KDIGO = Kidney Disease Improving Global Outcomes, RRT = renal replacement therapy.

Keywords: acute kidney injury, intensive care, risk factors, torso injury

1. Introduction

Acute kidney injury (AKI) is common in critically ill trauma patients and is associated with a high mortality rate, prolonged length of hospital stay, and increased costs.[1,2] Several studies have reported on the risk factors and outcomes of AKI.[2–4] The risk of AKI in trauma patients depends on multiple factors, such as hemorrhage, hypoperfusion, reperfusion injury, rhabdomyolysis, direct injury to the kidney, exposure to nephrotoxic substances, and sepsis.[1–4]

AKI is a heterogeneous condition ranging from mild kidney function impairment to the need for renal replacement therapy (RRT). Therefore, consensus definitions of AKI have been

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[a] Deparment of Trauma Surgery, Trauma Center, Chungbuk National University Hospital, Cheongju, Republic of Korea, [b] Department of Trauma Surgery, College of Medicine, Chungbuk National University, Cheongju, Republic of Korea, [c] Department of Cardiovascular and Thoracic Surgery, Trauma Center, Chungbuk National University Hospital, Cheongju, Republic of Korea, [d] Department of Anesthesiology and Pain Medicine, Chungbuk National University Hospital, Cheongju, Republic of Korea.

[*] Correspondence: Jin Young Lee, Departement of trauma surgery, Trauma Center, Chungbuk National University Hospital, Cheongju, Republic of Korea (e-mail: client80@hanmali.net).

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developed to include all AKI severities and allow for comparison between studies.[5] One such definition is the risk, injury, failure, loss of kidney function, and end-stage kidney disease criteria,[6] which was later modified by the Acute Kidney Injury Network[7] and the Kidney Disease: Improving Global Outcomes (KDIGO) organizations who then developed the current widely accepted KDIGO criteria.[8]

The identification of AKI risk factors in trauma patients is important for risk stratification and the provision of optimal intensive care unit (ICU) treatment. Thoracic or abdominal trauma is often life-threatening, and abdominal injury increased the odds ratio for AKI.[5] Although many studies have reported on the incidence and risk factors for AKI in trauma patients admitted to a critical care unit,[1–5] there was marked heterogeneity regarding the patient characteristics and trauma centers in these studies. Therefore, we aimed to identify the risk factors for AKI in patients admitted to ICU after torso injury in a single trauma center.

2. Methods

2.1. Study design and data collection

This retrospective observational cohort study was conducted at a single, level I trauma center at OO University Hospital, South Korea. Overall, 428 trauma patients were admitted to the ICU after sustaining thoracic and/or abdominal injuries from January 2016 to December 2019. Finally, after meeting the following inclusion criteria, 380 trauma patients were included in this study: >15 years old, survived for >48 hours, and no end-stage renal disease. Patients were divided into 2 groups: AKI and non-AKI (n = 72 and 308, respectively).

This study was approved by the institutional review board of Chungbuk University Hospital (2020-02-006-003). The review board waived the requirement for informed consent due to the retrospective nature of the study.

2.2. Study variables and definitions

The baseline characteristics included age, sex, underlying disease, Glasgow coma scale score, and trauma-related variables, such as the injury severity score (ISS), revised trauma score, abbreviated injury scale, and mechanism of injury. Clinical and laboratory variables were collected and analyzed after arrival at the hospital. Clinical outcomes included length of ICU or hospital stay, ventilator days, occurrence of pneumonia, and in-hospital mortality.

Hypotension was defined as a systolic blood pressure of <90 mm Hg. In a case with abnormal findings of focused assessment with sonography for trauma[9] in any areas, was defined focused assessment with sonography for trauma positive. Thoracic and/or abdominal injuries were identified by formal reading of computed tomography scans such as hemothorax, pneumothorax, rib fractures, abdominal solid organ injury, and intestinal injuries. During the first 24 hours from arrival at the hospital, the quantity of any fluids administered was extracted from the medical records, excluding the number of blood transfusions. The cumulative fluid balance for 24 hours was calculated by subtracting all outputs from the total volume of the infused fluid for 24 hours. A cut-off value of 2.5 L for the cumulative fluid balance for 24 hours was estimated using the area under the curve. An emergency operation was defined as a thoracic- and/or abdominal-related operation performed within the initial 24 hours. AKI diagnosis was made based on the current KDIGO criteria[8] using the serum creatinine (SCr) and urine output criteria. The baseline SCr level was used as the initial laboratory result. In most of the patients, the baseline SCr level was not known, but SCr increase $\geq$ 0.3 mg/dL within 48 hours was the first criteria for diagnosis[8]. Therefore, we studied patients who survived sufficiently during an observational period for AKI diagnosis.

2.3. Statistical analyses

Statistical analyses were performed using IBM SPSS Statistics 23.0 (IBM Corp, Armonk, NY). Categorical data are presented as numbers (%), and they were compared using the χ^2 or Fisher exact test. Continuous variables are expressed as mean ± standard deviation or median and interquartile ranges and were compared between the groups using the Student *t* test or Mann–Whitney *U* test. Factors that were significantly associated with AKI ($P < .05$) in the univariate analysis were included in the multivariate analysis along with the age, ISS, hypotension, presence of hemothorax or intestinal injury, number of packed red blood cell transfusions, and cumulative fluid balance during the initial 24 hours, lactate levels, and vasopressor use. Multivariate regression analysis was performed with a logistic regression model using the maximum likelihood method and backward stepwise selection. Goodness-of-fit was assessed using the Hosmer–Lemeshow test.

3. Results

3.1. Baseline characteristics of the patients

The baseline characteristics of the patients are listed in Table 1. Of the 380 patients included in the study, 72 (18.9%) had AKI. The mean age was 53 years, and the patients in the AKI group were significantly older than those in the non-AKI group (57 years vs 52 years, $P = .040$). The AKI group showed a higher rate of diabetes mellitus than did the non-AKI group; however, this was not significant. Blunt injury occurred in most patients, and car accidents were the most common mechanism of injury. Trauma scores such as the ISS and revised trauma score showed that the patients were more severely injured in the AKI group than in the non-AKI group. The mean abbreviated injury scale score for the chest was significantly higher in the AKI group than in the non-AKI group ($P = .011$).

3.2. Clinical and initial laboratory variables

The clinical and initial laboratory variables of the patients are presented in Tables 2 and 3. The proportions of patients with hypotension, hemothorax, and intestinal injury (47% vs 21%, $P < .001$; 47% vs 32%, $P = .019$; and 33% vs 9%, $P < .001$, respectively) were all greater in the AKI group than in the non-AKI group. In the initial 24 hours, the patients in the AKI group were more likely to receive blood transfusions and received a greater volume of fluid infusions (79% vs 50%, $P < .001$ and 6.5 L vs 4.2 L, $P < .001$, respectively) than did those in the non-AKI group. In the initial 48 hours, the patients in the AKI group were more likely to have vasopressors, diuretics, and glycopeptides administered (65% vs 20%, $P < .001$; 44% vs 24%, $P = .031$; and 15% vs 7%, $P = .031$, respectively) than were those in the

Table 1

Baseline characteristics of patients with torso injury.

	Total (n = 380)	AKI (n = 72)	Non-AKI (n = 308)	*P* value
Age (yrs), mean ± SD	53 ± 17.4	57 ± 20.1	52 ± 16.6	.040
Male, n (%)	271 (71)	50 (69)	221 (72)	.772
Underlying disease, n (%)				
HTN	106 (28)	26 (36)	80 (26)	.108
DM	39 (10)	12 (17)	27 (9)	.054
CAoD	10 (3)	3 (4)	7 (2)	.408
CVA	13 (3)	5 (7)	8 (3)	.073
Blunt trauma, n (%)	372 (98)	72 (100)	300 (97)	.324
Injury mechanism, n (%)				.566
Pedestrian TA	69 (18)	15 (21)	54 (18)	
In car TA	113 (30)	23 (32)	90 (29)	
Motorcycle TA	45 (12)	9 (13)	36 (12)	
Fall	97 (26)	11 (15)	86 (28)	
Others	56 (15)	14 (19)	42 (14)	
GCS, median (IQR)	15.0 (10.0–15.0)	14 (8.0–15.0)	15.0 (11.0 – 15.0)	.010
Hypotension, n (%)	99 (26)	34 (47)	65 (21)	<.001
ISS, mean ± SD	24.9 ± 9.8	29.1 ± 11.5	24.0 ± 9.1	.001
RTS, median (IQR)	7.550 (5.967–7.840)	6.375 (4.297–7.840)	7.840 (6.375–7.840)	<.001
AIS score, mean ± SD				
Chest	2.8 ± 0.69	3.0 ± 0.63	2.8 ± 0.70	.024
Abdomen	2.7 ± 0.78	2.8 ± 0.81	2.7 ± 0.77	.438

AIS = abbreviated injury scale, AKI = acute kidney injury, CAoD = coronary artery occlusive disease, CVA = cerebrovascular accident, DM = diabetes mellitus, GCS = Glasgow coma scale, HTN = hypertension, Hypotension = systolic blood pressure <90 mm Hg, IQR = interquartile range, ISS = injury severity score, RTS = revised trauma score, SD = standard deviation, TA = traffic accident.

non-AKI group. However, hydroxyethyl starch administration was not significantly different between the 2 groups. No significant differences were noted in electrolyte levels; however, hemoglobin, creatinine, base deficit, and lactate levels differed significantly between the 2 groups (Table 3).

3.3. Clinical outcomes

The median length of hospital stay did not differ between the 2 groups; however, the length of ICU stay and duration of mechanical ventilation (11.3 days vs 6.8 days, $P < .00$ and 17 days vs 3 days, $P < .001$, respectively) were longer in the AKI group than in the non-AKI group. While emergency operations were performed more frequently in the AKI group, there was no difference in the proportion of patients who underwent angioembolization between the 2 groups. In our cohort, 43 (84%) of the 51 patients who had intestinal injury and 41 (19.6%) of the 209 patients identified with solid abdominal organ injury underwent emergency surgery (see Table, Supplemental Digital Content, http://links.lww.com/MD/G292, which lists emergency operations in abdominal intestinal and solid organ injury). The in-hospital mortality was several folds higher in the AKI group than in the non-AKI group (26% vs 4%,

Table 2

Comparison of clinical parameters between patients with acute kidney injury and those without acute kidney injury.

	Total (n = 380)	AKI (n = 72)	Non-AKI (n = 308)	*P* value
FAST positive, n (%)	121 (32)	29 (46)	92 (34)	.080
Hemothorax, n (%)	132 (35)	34 (47)	98 (32)	.019
Pneumothorax, n (%)	160 (42)	37 (51)	123 (40)	.085
Rib fracture (s), n (%)	292 (77)	58 (81)	234 (76)	.442
Abdominal solid organ injury, n (%)	209 (55)	37 (51)	172 (56)	.513
Intestinal injury, n (%)	51 (13)	24 (33)	27 (9)	<.001
Transfusion 24 h, n (%)	210 (55)	57 (79)	153 (50)	<.001
pRBC transfusions (U) 24 h, median (IQR)	1 (0–4)	4 (1–8)	0 (0–3)	<.001
FFP transfusions (U) 24 h, median (IQR)	0 (0–3)	3 (0–9)	0 (0–2)	<.001
Total volume of infused fluid 24 h (L), median (IQR)	4.6 (3.4–6.6)	6.5 (4.6–8.6)	4.2 (3.3–6.0)	<.001
Cumulative fluid balance at 24 h (L), median (IQR)	1.9 (0.7–3.6)	3.6 (2.2–5.6)	1.6 (0.5–2.9)	<.001
Cumulative fluid balance 24 h > 2.5 L	148 (39)	52 (72)	96 (31)	<.001
HES administered 24 h, n (%)	117 (31)	27 (38)	90 (29)	.202
Drug exposure during 48 h, n (%)				
Vasopressor	108 (28)	47 (65)	61 (20)	<.001
Diuretics	105 (28)	32 (44)	73 (24)	.001
Glycopeptide	32 (8)	11 (15)	21 (7)	.031
IV contrast media	363 (96)	71 (99)	292 (95)	.215

AKI = acute kidney injury, FAST = focused assessment with sonography for trauma, FFP = fresh frozen plasma, HES = hydroxyethyl starch, IQR = interquartile range, IV = intravenous, pRBC = packed red blood cell.

Table 3

Comparison of laboratory findings between patients with acute kidney injury and those without acute kidney injury.

	Total (n = 380)	AKI (n = 72)	Non-AKI (n = 308)	*P* value
Hemoglobin (g/dL), median (IQR)	12.9 (11.2–14.2)	11.9 (10.3–13.8)	13.0 (11.5–14.4)	.017
BUN (mg/dL), median (IQR)	15.9 (12.7–20.2)	16.9 (13.2–22.2)	15.8 (12.5–19.4)	.091
Creatinine (μmol/L), median (IQR)	0.87 (0.74–1.05)	0.96 (0.76–1.21)	0.85 (0.73–1.01)	.002
CPK (g/dL), median (IQR)	373 (225 –676)	455 (229–738)	345 (222–654)	.215
Sodium (mmol/L), median (IQR)	139 (137–141)	139 (137–141)	139 (137–141)	.523
Potassium (mmol/L), median (IQR)	3.9 (3.6–4.2)	3.9 (3.6–4.3)	3.9 (3.5–4.2)	.391
Chloride (mmol/L), median (IQR)	107 (105–110)	108 (106–110)	107 (105–110)	.187
pH, median (IQR)	7.36 (7.30–7.40)	7.33 (7.25–7.39)	7.37 (7.32–7.41)	<.001
Base deficit (mmol/L), median (IQR)	–4.0 (–7.6 to –1.8)	–7.4 (–9.6 to –2.9)	–3.7 (–6.8 to –1.7)	<.001
Lactate (mmol/L), median (IQR)	3.0 (2.0–4.4)	4.2 (2.6–6.2)	2.8 (1.8–4.1)	<.001

AKI = acute kidney injury, BUN = blood urea nitrogen, CPK = creatine phosphokinase, IQR = interquartile range.

$P < .001$), and RRT was performed in 13 (18%) patients in the AKI group (Table 4).

3.4. Logistic regression analysis for AKI risk factors

The multivariate logistic analysis identified intestinal injury, cumulative fluid balance >2.5 L for 24 hours, lactate levels, and vasopressor use as independent risk factors for AKI (adjusted odds ratio [OR] 2.953, 95% confidence interval [CI] 1.410–6.181, $P = .004$; OR 2.058, 95% CI 1.017–4.164, $P = .045$; OR 1.170, 95% CI 1.019–1.343, $P = .026$; OR 2.910, 95% CI 1.414–5.987, $P = .004$, respectively; Table 5).

4. Discussion

In our study, we analyzed patients with torso injuries admitted to ICU and found an 18.9% incidence of AKI, with 18% of AKI patients receiving RRT and a 6.5-fold increase in in-hospital mortality relative to that observed for non-AKI patients. AKI is a common complication associated with an independent risk of mortality.[4] In a recent systemic review and meta-analysis, AKI was reported in approximately 24% of trauma patients admitted to ICUs.[5]

Advanced age is associated with AKI in trauma patients.[1–3,5] Patients aged >65 years are mainly admitted to trauma ICUs for low-energy falls and for admissions relating to their comorbidities, which may affect baseline renal function.[10] However, a recent study on trauma patients aged ≥75 years found that AKI was associated with the magnitude of injury and shock, rather than the patient's comorbidities, as well as the risk of death.[11] In our study, age was analyzed as a potential risk factor; however, we did not find that it was a significant factor.

While our study did not identify solid organ injury as a risk factor, intestinal injury was identified as an independent risk factor for AKI. A recent systemic review and meta-analysis reported uncertainty regarding whether direct trauma to the kidneys and/or urinary tract affects kidney function.[5] Surgery remains a leading cause of AKI in hospitalized patients; the incidences for cardiac, general, and thoracic surgery were reported as 18.7%, 13.2%, and 12.0%, respectively.[12] Specifically, in trauma patients, packing of the abdomen during damage control surgery in severe intra-abdominal injuries may also affect kidney function.[5] Sepsis in the ICU is a major cause of AKI, as it results in both hypoperfusion and inflammatory insults.[13] It may be expected that many intestinal injury patients might develop sepsis; however, our study did not evaluate postoperative sepsis. This was one of its limitations.

The renal responses to hypoperfusion are afferent arteriole dilation and efferent arteriole vasoconstriction to maintain glomerular filtration in addition to neurohormonal responses to expand the intravascular volume.[12] Fluid administration is the most common initial therapy for restoring stroke volume and improving renal perfusion in hypovolemic trauma patients.[14] However, fluid therapy has a growing number of potential adverse effects [15] Interstitial edema and subsequent dysfunction of the heart, lungs, and gastrointestinal tract are well known.[16] These effects are pronounced in encapsulated organs, such as the liver and kidneys, which lack the capacity to accommodate

Table 4

Clinical outcomes of patients with or without acute kidney injury.

	Total (n = 380)	AKI (n = 72)	Non-AKI (n = 308)	*P* value
LoH (d), median (IQR)	36.7 (19.8–67.2)	36 (15.5–73.2)	37 (20.0–65.0)	.138
LoICU (d), median (IQR)	8.5 (3.6–18.8)	11.3 (5.4–20.6)	6.8 (3.1–17.3)	<.001
Mechanical ventilation, n (%)	177 (47)	53 (74)	124 (40)	<.001
DoMV (d), median (IQR)	4.0 (1.3–8.5)	7 (3.5–15.0)	3.0 (1.0–7.5)	<.001
Emergency operation, n (%)	82 (36)	31 (43)	51 (16)	<.001
Angioembolization, n (%)	166 (44)	33 (46)	133 (43)	.693
Pneumonia, n (%)	63 (17)	24 (33)	39 (13)	<.001
RRT, n (%)		13 (18)		
In-hospital mortality, n (%)	31 (8)	19 (26)	12 (4)	<.001

AKI = acute kidney injury, DoMV = duration of mechanical ventilation, IQR = interquartile range, LoH = length of hospital stay, LoICU = length of intensive care unit stay, RRT = renal replacement therapy.

Table 5

Univariate and multivariate analyses evaluating risk factors for acute kidney injury.

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Age (yrs)	1.018 (1.003–1.034)	.021	1.016 (0.998–1.035)	.078
ISS	1.052 (1.026–1.079)	<.001		
Hypotension (SBP <90 mm Hg, yes/no)	3.345 (1.954–5.726)	<.001		
Hemothorax (yes/no)	1.917 (1.139–3.229)	.014		
Intestinal injury (yes/no)	5.204 (2.773–9.764)	<.001	2.953 (1.410–6.181)	.004
pRBC transfusions (U) 24 h	1.234 (1.152–1.322)	<.001		
Cumulative fluid balance 24 h > 2.5 L	5.742 (3.249–10.146)	<.001	2.058 (1.017–4.164)	.045
Lactate (mmol/L)	1.311 (1.171–1.467)	<.001	1.170 (1.019–1.343)	.026
Use of vasopressors (yes/no)	7.612 (4.347–13.331)	<.001	2.910 (1.414–5.987)	.004

CI = confidence interval, ISS = injury severity score, OR = odds ratio, pRBC = packed red blood cell, SBP = systolic blood pressure.
P value for the Hosmer–Lemeshow goodness-of-fit test was .638.

additional volume.[15] Our study showed that a positive fluid balance (>2.5 L) for 24 hours was independently associated with the development of AKI. To our knowledge, no randomized controlled studies have reported that a positive fluid balance is either beneficial or necessary in AKI or during acute illness in general.[15] Goal-directed fluid restriction may decrease AKI in surgical patients.[17] Therefore, fluid responsiveness should be investigated as an endpoint for posttraumatic resuscitation to prevent unnecessary fluid administration and subsequent AKI.[14]

A higher blood lactate level after arrival at the hospital indicates the importance of cumulated metabolic debts owing to tissue hypoperfusion, regardless of the presence of hemorrhagic or non-hemorrhagic shock,[18] and blood lactate levels are reported to be associated with AKI.[4,19,20] We also identified the blood lactate level after arrival as an independent risk factor for AKI.

The volumes of resuscitation fluids and blood products administered in the first 24 hours were greater in the AKI group than in the non-AKI group. They were also more likely to be treated with vasopressors. Previous retrospective studies highlighted an association of vasopressor use with mortality in trauma patients.[21,22] To our knowledge, no previous studies have examined the association of AKI with vasopressor use in trauma patients.[20] Norepinephrine use may increase renal perfusion and the glomerular perfusion rate in patients with circulatory failure[23,24]; however, in an experimental study, norepinephrine failed to increase renal microcirculation in a septic pig.[25] A recent study reported that early vasopressor use was not associated with mortality in trauma patients with hemorrhagic shock.[26] As renal blood flow and perfusion may vary between individuals, evaluation of renal blood flow and its distribution and resistive index have been proposed for identifying the optimal mean arterial pressure target for each patient.[24] The optimal individual mean arterial pressure may avoid excessive vasopressor use.

This study has several limitations. First, this was a retrospective, single-center study. Our cohort was very specific to thoracic and/or abdominal injury trauma patients. The retrospective design and specific cohort of cases resulted in selection bias, and our results cannot be generalized to other general trauma patients. Second, postoperative sepsis was not investigated; therefore, the association of sepsis with AKI could not be analyzed. Third, the fluid resuscitation strategy could not be controlled because of the retrospective collection of data, and our study did not include different infused fluids and different amounts of hydroxyethyl starch infusions. Fourth, in the absence of a true baseline serum creatinine level in most patients, the first laboratory results at the hospital were used as a baseline, which might have led to misclassification.

In summary, the development of AKI in patients admitted to the ICU after torso injury was significantly associated with intestinal injury, a cumulative fluid balance >2.5 L for 24 hours, initial serum lactate levels, and vasopressor use. Recognizing risk factors at an early stage could aid risk stratification and the provision of optimal ICU care.

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Author contributions

Conceptualization: Young Hoon Sul, Jin Suk Lee, Se Heon Kim, Jung Hee Choi, Jin Young Lee.
Data curation: Young Hoon Sul, Jin Bong Ye, Jin Suk Lee, Se Heon Kim, Su Young Yoon, Jung Hee Choi, Jin Young Lee.
Formal analysis: Jin Bong Ye, Jung Hee Choi, Jin Young Lee.
Writing – original draft: Jin Young Lee.
Writing – review & editing: Young Hoon Sul, Jin Bong Ye, Jin Suk Lee, Se Heon Kim, Su Young Yoon, Jung Hee Choi, Jin Young Lee.

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