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Elevated IAP in critically ill patients associated with increased AKI incidence: a cohort study from the MIMIC-IV database

ShengHui Miao[1,4], Mingkun Yang[2,4], Wen Li[2] & Jing Yan[3]✉

Elevated intra-abdominal pressure can engender a spectrum of adverse physiological repercussions in patients, but further research is needed to ascertain whether elevated intra-abdominal pressure exerts significant effects on renal function. The study used MIMIC-IV database to identify critical patients with IAP monitoring. Patients were categorized into Low-IAP and High-IAP groups based on the results of the restricted cubic splines curve, with HR = 1 set at IAP = 16 mmHg. The primary outcome of the study was the occurrence of AKI within 72 h of ICU admission, and secondary outcomes including the rate of CRRT utilization and 28-day all-cause mortality. Cox proportional hazards regression analysis was employed to clarify the relationship between IAP and AKI. A total of 1746 patients were included in our study. Restricted cubic spline analysis demonstrated an increased risk of AKI with higher IAP. Multivariable Cox proportional hazards analysis uncovered a notable correlation between elevated IAP and AKI incidence (HR: 1.40(1.14–1.71)). After adjusting for confounding factors, patients with elevated IAP remained significantly related with AKI (HR: 1.23(1.01–1.52)). The Kaplan–Meier survival curves indicated a significant superior 28-day survival rate for Low-IAP group (the log-rank test p-value was 0.001) and the cumulative risk curve showed a higher demand for CRRT in the High-IAP group (the log-rank test p-value was 0.0028). Augmented intra-abdominal pressure (above 16 mmHg) is significantly associated with a higher incidence of acute kidney injury (AKI) in critically ill patients, along with an increased need for continuous renal replacement therapy (CRRT) and a higher 28-day mortality rate.

Keywords Intra-abdominal pressure, Acute kidney injury, MIMIC-IV database, Cohort study

Abbreviations

AKI	Acute kidney injury
AUC	The area under the curve
CCI	Charlson Comorbidity Index
CCU	Coronary care unit
CHF	Chronic heart failure
CI	Confidence interval
CKD	Chronic kidney disease
CRRT	Continuous renal replacement therapy
HR	Hazards ratio
IAP	Intra-abdominal pressure
ICU	Intensive care unit
MIMIC	The Medical Information Mart for Intensive Care database
MV	Mechanical ventilation
RCS	Restricted cubic splines
SAPSII	Simplified acute physiology scores II

[1]The Fourth Affiliated Hospital, International Institutes of Medicine, Zhejiang University School of Medicine, Yiwu 322000, China. [2]Department of Second Clinical Medical College, Zhejiang Chinese Medicine University, Hangzhou 310053, Zhejiang, China. [3]Zhejiang Hospital, Zhejiang University School of Medicine, Lingyin Road 12, Hangzhou 310013, Zhejiang, China. [4]These authors contributed equally: ShengHui Miao and Mingkun Yang. ✉email: yanjing201801@163.com

SD	Standard deviations
SICU	Surgery intensive care unit
SOFA	Sequential Organ Failure Assessment
SQL	Structured Query Language
WBC	White blood cell

Elevated intra-abdominal pressure (IAH) is a common yet serious concern in critically ill patients within intensive care units (ICUs)[1]. A significant global study involving 491 patients across 15 ICUs revealed that 34% of patients had IAH at admission, and nearly half developed IAH during their ICU stay[2]. According to the current consensus from the World Society of the Abdominal Compartment Syndrome (WSACS), IAH is defined as an intra-abdominal pressure exceeding 12 mmHg[3]. A pressure above 20 mmHg is indicative of abdominal compartment syndrome (ACS), the most severe circumstance of IAH[3]. There is a substantial link between IAH and mortality, often leading to multiple organ dysfunction[4,5]. Therefore, diligent monitoring of intra-abdominal pressure and associated organ dysfunction is crucial for managing high-risk patients effectively. This approach is essential for improving outcomes and mitigating the adverse effects of IAH and ACS in the ICU setting.

The kidney, particularly susceptible to heightened intra-abdominal pressure, endures compromised function as elevated pressure escalates renal venous pressure and diminishes perfusion pressure. This cascade effects a reduction in glomerular filtration rate, manifesting as oliguria and tubular dysfunction[6,7]. Therefore, acute kidney injury, as a prevalent condition within the ICU, may be one of the significant complications caused by elevated intra-abdominal pressure.

Despite this, research exploring the link between intra-abdominal pressure and AKI remains scant, predominantly featuring observational studies with limited sample sizes[8–10]. Notably, a recent prospective cohort study among critically ill obstetric patients failed to establish a significant correlation between early AKI and intra-abdominal pressure[9], casting doubt on the assumed connection. This study aims to explore the relationship between intra-abdominal pressure and the occurrence of AKI.

Methods

Data source

The study utilized data obtained from the Medical Information Mart for Intensive Care database (MIMIC-IV 2.2, https://physionet.org/content/mimiciv/2.2/), a publicly accessible repository housing clinical data from 73,181 adult patients admitted to intensive care units (ICUs) at the Beth Israel Deaconess Medical Center in Boston, Massachusetts, USA, spanning the years 2008 to 2019[11]. Approval for database access was granted by the Institutional Review Boards of MIT and Beth Israel Deaconess Medical Center. Given the anonymized nature of the database and its standardized data, this study was exempt from separate ethics approval in accordance with the principles outlined in the Declaration of Helsinki regarding patient populations.

Study population

As showed in Fig. 1, Patients in the MIMIC-IV who received the monitoring of bladder pressure were eligible for inclusion. Subsequently, we employed the boxplot function to generate a 3σ plot, facilitating the removal of outlier measurements. Following this, we utilized the boxplot function to construct a 3σ plot, facilitating the exclusion of abnormal measurement values exceeding three standard deviations, which were excluded and subsequently imputed. Moreover, we exclusively considered data from the initial ICU admission of each patient, and individuals who did not undergo monitoring within the first 24 h of ICU admission were also omitted from our study. Following the screening process above, the remaining patients had an ICU length of stay exceeding 24 h, and all individuals were over 18 years of age. Finally, we computed the average bladder pressure measured within the first day of ICU admission for each patient. We divided patients into two groups based on the threshold determined by the inflection point identified through restricted cubic spline (RCS) analysis (16 mmHg).

Variables and outcome

We utilized structured query language (SQL) to extract patients' baseline characteristics with particular consideration given to factors already confirmed to increase the risk of AKI occurrence[12,13], including demographic information such as age, gender, and weight. Disease severity scores comprised the SOFA score, SAPS II score, and CCI score. Comorbidities included chronic heart failure, chronic kidney disease and diabetes. And we documented the prevalence of sepsis. The use of life support treatment on the first day was assessed, including invasive mechanical ventilation (MV) and vasopressor administration. Additionally, laboratory parameters such as hemoglobin levels, white blood cell counts and serum creatinine were included in the analysis. All baseline indicators were extracted as the average values within the first 24 h of ICU admission. The missing values for all variables did not exceed 7%, and were imputed using the "Mice" package in R software with random forest imputation method[14].

The primary outcome was whether patients were diagnosed with acute kidney injury (AKI) within 72 h of ICU admission, based on the criteria of Kidney Disease: Improving Global Outcomes (KDIGO)[15]. AKI is defined by either an absolute increase in Scr exceeding 0.3 mg/dL (26.5 μmol/L) or a relative increase by more than 50% from baseline within 48 h, and it also can be diagnosed when urine output is less than 0.5 mL/kg/h (in adults) for 6 h or longer.

Secondary outcomes comprised the 28-day mortality rate, occurrence of AKI within one week, utilization of CRRT within 72 h and one-week.

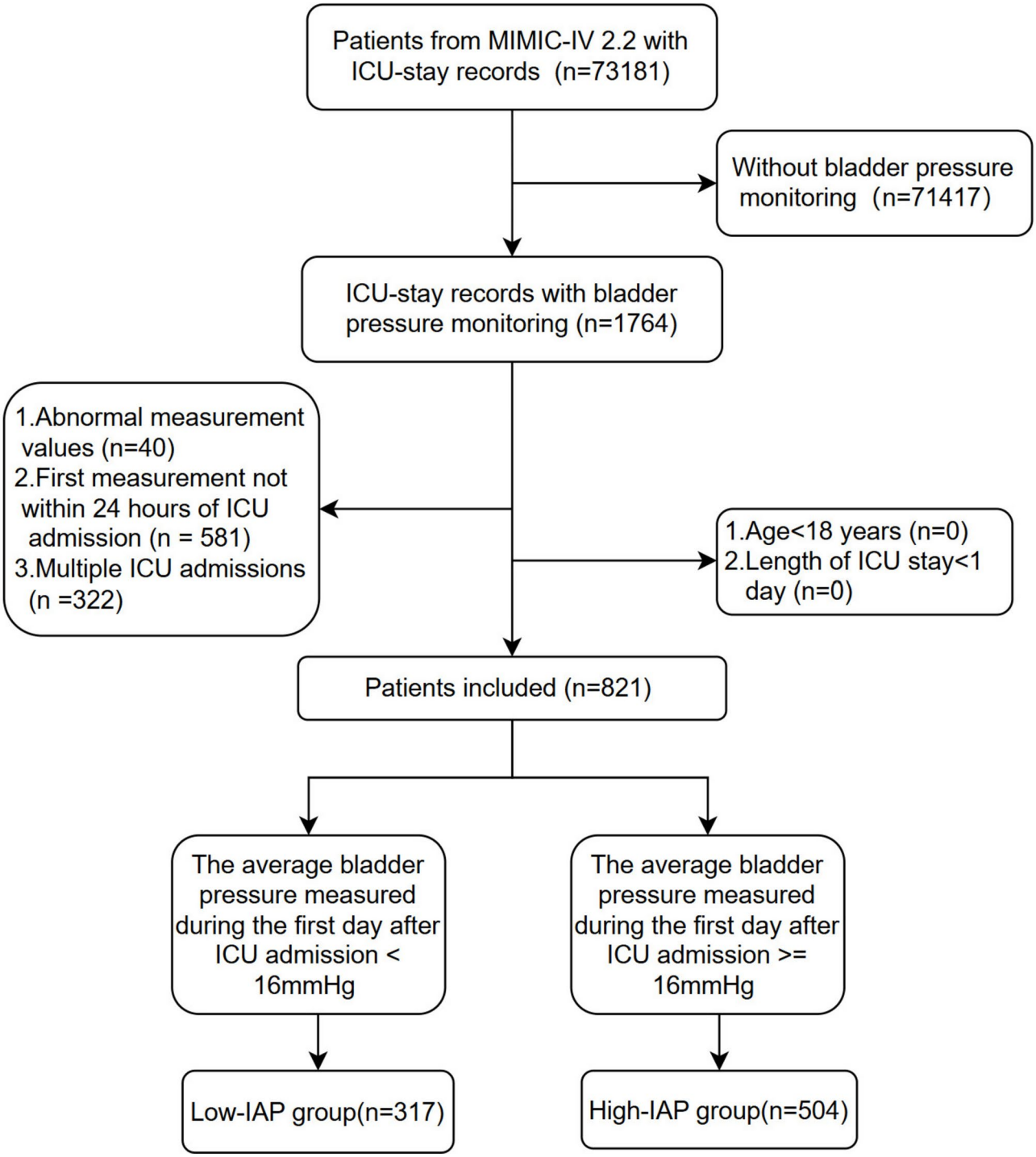

Fig. 1. Flow chart of patient selection. MIMIC, the Medical Information Mart for Intensive Care database; ICU, Intensive care unit; IAP, Intra-abdominal pressure.

Statistical analysis

Continuous variables are expressed as means (standard deviations), while categorical variables are presented as total counts and percentages. Group comparisons were conducted using the X2 test or Fisher's exact test for categorical variables, and Student's t-test or the Mann–Whitney U test for continuous variables, as deemed appropriate.

We constructed Cox proportional hazards regression models to assess the relationship between IAP and AKI occurrence, with HR (95% CI) as the outcome measure[16]. Certain models were adjusted for specific variables. Both clinical and prognostic variables were included in the multivariable models: Crude model: Unadjusted; Adjusted model 1: Adjusted for demographic variables including age, gender, and weight; Adjusted model 2: Adjusted for

all variables, including age, gender, weight, ICU type, SOFA score, SAPSII score, Charlson Comorbidity Index (CCI), chronic heart failure (CHF), diabetes, chronic kidney disease (CKD), sepsis, vasopressor use, invasive mechanical ventilation (MV), white blood cell (WBC) count, red blood cell (RBC) count and creatinine(Cr).

The cumulative risk curves depict the incidence of AKI within 72 h and the utilization of CRRT within 72 h for both the Low-IAP and High-IAP groups. Kaplan–Meier curves illustrate the 28-day mortality rates for patients in both groups. Subgroup analyses were conducted by stratifying the study population based on age, gender, ICU type, SOFA score, chronic heart failure (CHF), chronic kidney disease (CKD), diabetes, sepsis, use of invasive mechanical ventilation, and vasopressor therapy.

Statistical analyses were conducted using the R programming language (version 4.3.3). Statistical significance was defined as $p < 0.05$.

Result

Cohort characteristic

A total of 1746 patients underwent intra-abdominal pressure (IAP) monitoring in the MIMIC-IV database. Ultimately, 821 patients met the inclusion criteria, with 317 patients categorized in the Low-IAP group and 504 patients in the High-IAP group. Compared to the Low-IAP group, the High-IAP group had a greater proportion of males (58.0% vs 66.1%, $p = 0.025$), higher body weight (83.04 vs 87.85, $p < 0.001$) and SOFA scores (9.0 vs 10.0, $p < 0.001$) (Table 1).

Primary outcome

As depicted in Fig. 2a, the restricted cubic splines (RCS) regression model was employed to illustrate that the risk of AKI increased non-linearly with rising IAP (P for non-linearity = 0.002), selecting IAP 16 mmHg as the reference level, represented by a vertical dashed line, the horizontal dashed line indicates a hazard ratio of 1.0. Before IAP reaches 16 mmHg, it acts as a protective factor against AKI occurrence. However, as it continues to increase, this protective effect gradually diminishes. After reaching 16 mmHg, further increases in IAP elevate

	Low-IAP	High-IAP	P value
n	317	504	
Baseline characteristics			
Age (Year)	61.49 (16.58)	61.87 (14.75)	0.751
Male (%)	184 (58.0)	333 (66.1)	0.025
ICU-type (%)			0.131
CCU	46 (14.5)	79 (15.7)	
SICU	185 (58.4)	259 (51.4)	
Other	86 (27.1)	166 (32.9)	
Weight (Kg)	83.04 (19.68)	87.85 (20.45)	0.001
SOFA	9.0 (5.0 to 12.0)	10.0 (6.0 to 13.0)	0.001
SAPSII	48.0 (35.0 to 61.0)	50.0 (38.0 to 62.0)	0.092
CCI (%)	5.0 (2.0 to 7.0)	4.0 (3.0 to 7.0)	0.252
CHF (%)	47 (14.8)	85 (16.9)	0.499
CKD (%)	54 (17.0)	91 (18.1)	0.78
Diabetes (%)	91 (28.7)	122 (24.2)	0.177
Sepsis (%)	266 (83.9)	430 (85.3)	0.656
Vasopressor (%)	93 (29.3)	163 (32.3)	0.408
Invasive MV (%)	242 (76.3)	383 (76.0)	0.976
Hemoglobin(g/L)	9.27 (2.01)	9.22 (2.11)	0.825
WBC ($*10^9$)	15.94 (7.97)	15.68 (7.78)	0.618
Creatinine(mg/dl)	1.1 (0.7 to 2.0)	1.3 (0.9 to 2.1)	0.050
Outcome			
AKI (%)	142 (44.8)	274 (54.4)	0.009
Stage1 (%)	71 (22.4)	137 (27.2)	0.05
Stage2 (%)	43 (13.6)	82 (16.3)	0.03
Stage3 (%)	28 (8.8)	55 (10.9)	0.01
CRRT (%)	89(28.1)	188(37.2)	0.012
28-day mortality (%)	83(26.2)	186(36.9)	0.008

Table 1. Baseline characteristics and outcomes of the low- and high-IAP groups. ICU, Intensive care unit; CCU, Coronary care unit; SICU, Surgery intensive care unit; SOFA, Sequential Organ Failure Assessment; SAPSII, simplified acute physiology scores II; CCI, Charlson Comorbidity Index; CHF, Chronic heart failure; CKD, Chronic kidney disease; WBC, white blood cell; AKI, acute kidney injury; CRRT, continuous renal replace therapy.

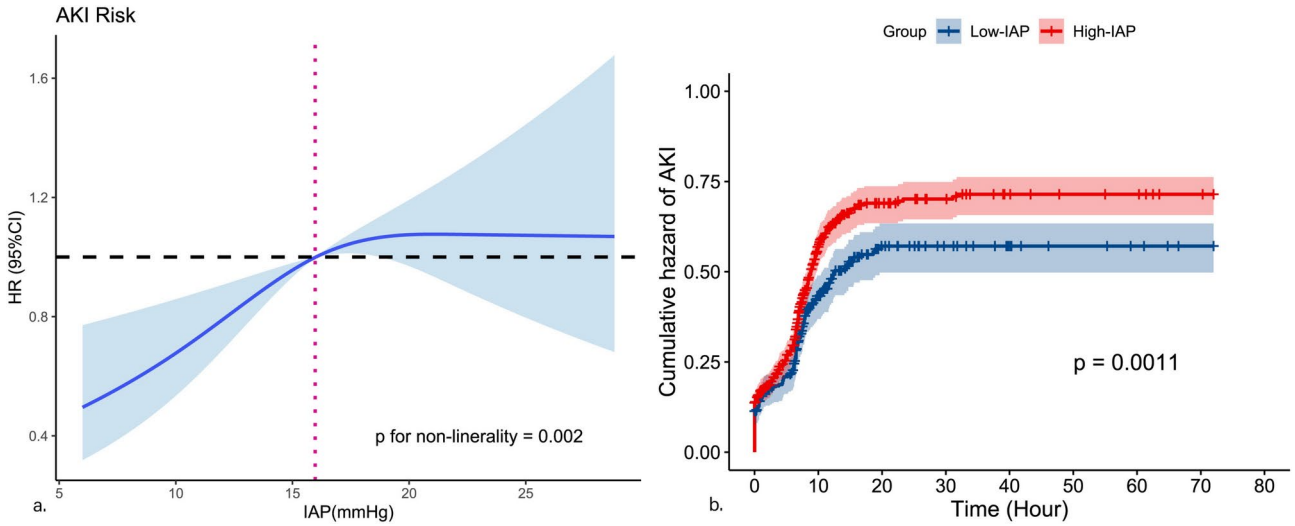

Fig. 2. (**a**) Restricted cubic spline for AKI within the first 72 h of ICU admission. HR, hazard ratio; AKI, Acute kidney injury; IAP, Intra-abdominal pressure; (**b**) the cumulative risk curve for AKI within the first 72 h of ICU admission. AKI, Acute kidney injury.

	Crude model (HR 95%CI)	P value	Adjusted model 1 (HR 95%CI)	P value	Adjusted model 2 (HR 95%CI)	P value
Low-IAP Group	Reference		Reference		Reference	
High-IAP Group	1.40 (1.14–1.71)	0.001	1.27 (1.04–1.6)	0.021	1.23 (1.01–1.52)	0.039

Table 2. Cox proportional hazard ratios (HR) for AKI occurrence. Model 1 adjusted by age, sex and weight; Model 2 adjusted by all variables.

the risk of AKI occurrence. Subsequently, the curve gradually enters a plateau phase, indicating that with further elevation of intra-abdominal pressure, the risk of AKI occurrence is only a minimal increase.

Nevertheless, the area under the curve (AUC) for IAP did not meet the desired threshold of adequacy (AUC for occurrence of AKI within 72 h: 0.550, 95% CI: 0.53–0.61, p = 0.007), and we identified the optimal threshold to be 14.5 mmHg using the Youden index method.

Cumulative risk curves for AKI occurrence within 72 h of ICU admission were plotted using a threshold of IAP 16 mmHg, based on the inflection point identified by the Restricted Cubic Spline curve. It was observed that the high-IAP group exhibited a significantly higher risk of early AKI occurrence following ICU admission compared to the low-IAP group (log-rank P = 0.0011). Subsequently, the trend in both groups gradually plateaued (Fig. 2b).

The multivariate Cox regression analyses revealed a significant detrimental effect of elevated IAP on AKI occurrence (Table2). No adjustments were made for any confounding factors in crude model (HR: 1.40(1.14–1.71), p = 0.001). In Adjusted model 1, adjustments were made for demographic parameters (HR: 1.27(1.04–1.6), p = 0.021), while in model 2, adjustments were made for all potential influencing factors (HR: 1.23 (1.01–1.52), p = 0.039).

Secondary outcomes

Researchers plotted cumulative risk curves to assess the utilization of continuous renal replacement therapy (CRRT) within 72 h of ICU admission for both groups, corresponding to the occurrence of AKI (Fig. 3a). The results revealed a higher demand for CRRT treatment in the high-IAP group (p = 0.0028). Additionally, Kaplan–Meier survival curves were constructed, and the log-rank test was employed to examine the influence of intra-abdominal pressure on 28-day all-cause mortality (Fig. 3b). It was observed that the low-IAP group demonstrated notably higher cumulative survival rates in comparison to the high-IAP group (p = 0.001).

Subclass analysis

The risk stratification value of the increased IAP for primary endpoints was further analyzed in multiple subgroups of the enrolled patients, including age, gender, ICU-type, SOFA, CHF, CKD, diabetes, sepsis, first-day vasopressor and invasive MV use (Fig. 4). In the subgroup of individuals under 65 years old (HR: 1.4 (1.14–1.71)), males (HR: 1.47 (1.13–1.91)), those admitted to the CCU (HR: 1.84 (1.04–3.24)), those admitted to the SICU(HR: 1.42 (1.07–1.89)), those with a SOFA score greater than 6 (HR: 1.26 (1.01–1.57)), those with a SOFA score less than 6 (HR: 1.82 (1.04–3.2)), those without concurrent CHF (HR: 1.4 (1.12–1.75)), those without concurrent CKD (HR: 1.4 (1.11–1.77)), those without concurrent diabetes (HR: 1.41 (1.1–1.8)), those with

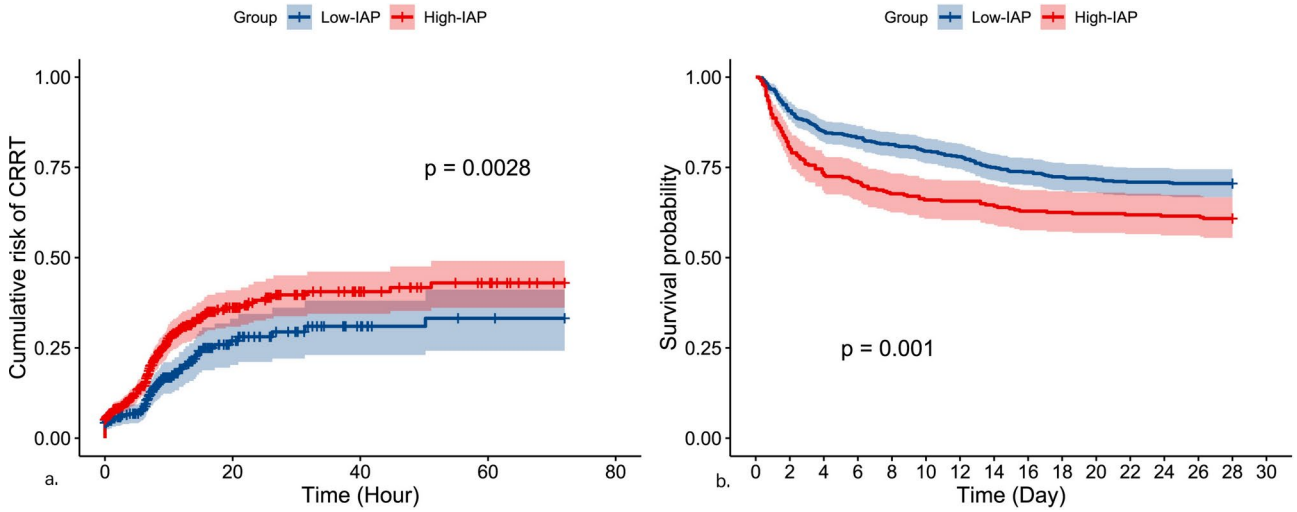

Fig. 3. (**a**) The cumulative risk curve for CRRT utilization within the first 72 h of ICU admission. CRRT, Continuous renal replacement therapy; (**b**) Kaplan–Meier survival analysis curves for 28-day all-cause mortality.

concurrent sepsis (HR: 1.41 (1.14–1.75)), those administered vasopressors (HR: 1.48 (1.14–1.93)), and those not using invasive MV (HR: 1.45 (1.16–1.82)), elevated IAP remained significantly associated with the risk of AKI occurrence within 72 h.

Discussion

This study, utilizing the MIMIC-IV database, investigated the potential link between elevated IAP and the risk of AKI. Our findings indicate a significant association between increased IAP and the likelihood of developing AKI. Even after adjusting for potential confounders, this association remains robust, with the high IAP group exhibiting a hazard ratio (HR) of 1.23 (95% CI: 1.01–1.52) compared to the normal IAP group. Additionally, analysis using RCS revealed a nonlinear relationship between elevated IAP and the incidence of AKI. These results underscore the importance of vigilant monitoring and management of intra-abdominal pressure.

There is increasing awareness regarding the monitoring of IAP in critically ill patients, particularly due to its association with a spectrum of organ dysfunctions, including AKI. According to the World Society of Abdominal Compartment Syndrome (WSACS) criteria, normal intra-abdominal pressure should not exceed 12 mmHg, and intra-abdominal pressure exceeding 20 mmHg is considered indicative of abdominal compartment syndrome (ACS)[3]. While an increase in IAP is often linked with organ dysfunction, clear clinical evidence delineating this correlation remains elusive. Current research, primarily involving small-scale prospective studies, presents inconsistent findings on the relationship between elevated IAP and AKI risk. In a small-sample (n = 60) retrospective study by Demarchi et al.[8], a correlation was found between increased IAP following abdominal surgery and a higher incidence of AKI with a calculated optimal threshold of 7.68 mmHg using the Youden index. The initial IAP measurement at ICU admission was a predictor of AKI, with an area under the receiver operating characteristic curve (AUC) of 0.669 ($p < 0.029$). Similarly, Mazzeffi et al.[17] reported that 35 out of 42 patients (83.3%) undergoing cardiac surgery experienced elevated IAP at least once during the perioperative period, suggesting that IAH could heighten AKI risk in this group—a finding consistent with our own results. Conversely, a prospective study[9] involving 50 obstetric patients observed no significant association between IAP and early AKI. Despite the prevalence of IAH and AKI in this cohort, the AUC for IAP in predicting early AKI was only 0.499 (95% CI: 0.325–0.673) with a p-value of 0.992, indicating no predictive value. Our study further explores this relationship through careful adjustment for related confounders. In our fully adjusted model, the high IAP group showed a HR of 1.23 (1.01–1.52) with a p-value of less than 0.05, indicating that elevated IAP is an independent risk factor for AKI, representing a 1.23-fold increased risk of the outcome in the high IAP group compared to the low IAP group. Specifically, high IAP increased the risk of AKI by 25%. These findings emphasize the need for vigilant monitoring and management of intra-abdominal pressure to mitigate the risk of AKI in critically ill patients. In addition, we utilized restricted cubic spline regression to assess the nonlinear association between IAP and AKI. The results of the RCS model revealed a nonlinear escalation in the risk of AKI as IAP increased (nonlinear $P = 0.002$). As IAP continues to increase, the associated risk curve for AKI stabilizes. Additionally, we observed that a significant elevation in intra-abdominal pressure (IAP > 16 mmHg) appears to be necessary to substantially increase the risk of AKI occurrence.

In present, the pathophysiology of renal injury induced by elevated IAP remains not fully understood, but several mechanisms have been proposed. Firstly, as the post- glomerular intrarenal vascular network operates under low pressure[18], increased IAP can compress or occlude renal veins and tubules, thereby elevating glomerular capillary output pressure. This results in increased proximal tubular pressure and reduced renal arterial inflow, ultimately diminishing the transcapillary hydrostatic pressure gradient essential for glomerular filtration. Consequently, this can lead to a reduced glomerular filtration rate, causing oliguria and renal tubular

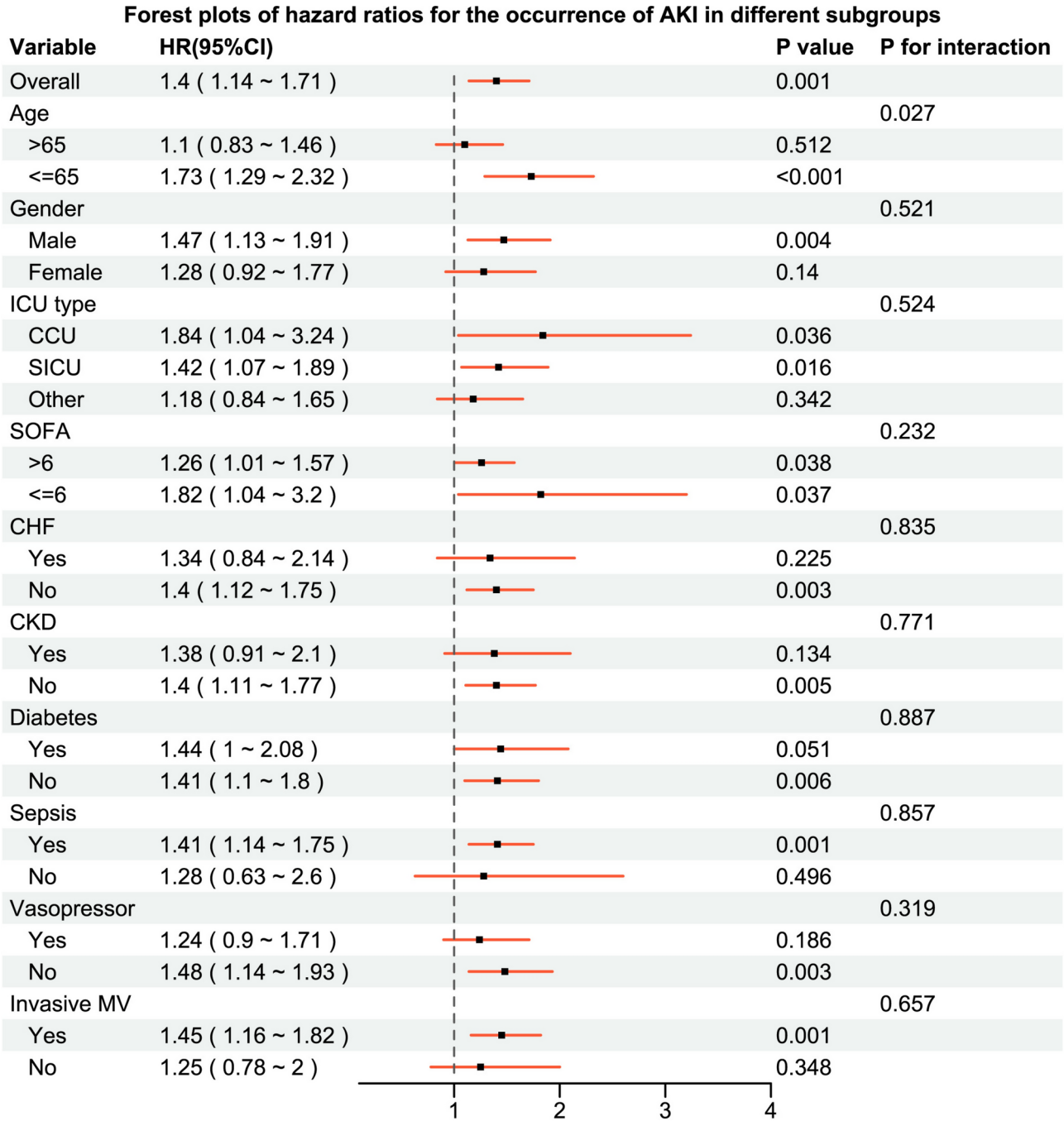

Fig. 4. Forest plots of hazard ratios for the incidence of acute kidney injury (AKI) in different subgroups. HR, hazard ratio; ICU, intensive care unit; CCU, coronary care unit; SICU, surgery intensive care unit; SOFA, sequential organ failure assessment; CHF, chronic heart failure; CKD, chronic kidney disease; MV, mechanical ventilation.

dysfunction[19]. For instance, Doty et al.[20] observed in pigs that increasing renal venous pressure to 30 mmHg led to a substantial drop in GFR from 26 to 8 ml/min, with the changes being partially or completely reversible upon reduction of the renal venous pressure. Secondly, further increases in IAP can impact cardiovascular function by reducing cardiac preload, impairing myocardial contractility, and increasing cardiac afterload, all of which contribute to decreased cardiac output. This interplay may trigger cardiorenal syndrome, further exacerbating renal deterioration[21,22]. Additionally, reduced cardiac function activates compensatory mechanisms, leading to elevated levels of catecholamines, renin, and angiotensin-aldosterone, along with an increase in inflammatory cytokines, which may further impair renal function[23–25]. In summary, heightened IAP can adversely affect renal function through both indirect systemic effects and direct renal impacts, underscoring the complex interdependencies between increased abdominal pressure and renal health.

This study presents several notable advantages. First, it is pioneering in utilizing data from the MIMIC database to investigate the correlation between IAP and AKI. The large sample size employed exceeds those of previous observational studies, lending greater reliability to the findings. Second, throughout the model-building process, we accounted for potential confounding factors, enhancing the robustness of our conclusions. Moreover, this study advances our understanding by exploring the nonlinear relationship between IAP and the incidence of AKI using RCS regression, thereby providing a more nuanced analysis of how variations in IAP influence AKI risk.

This study also has certain limitations. Firstly, it is retrospective in nature, and although we employed the multivariable regression method to minimize the influence of confounding factors and enhance the robustness of our results, these findings need to be validated through further prospective cohort studies. Secondly, patients who did not undergo intra-abdominal pressure measurement were excluded from the study. Consequently, our study population may inherently consist of individuals considered at potential risk for intra-abdominal hypertension, which could introduce a degree of selection bias. Finally, considering IAP is a dynamic parameter, relying on a single measurement may not adequately capture the relationship between its fluctuations over time and the incidence of AKI. A more comprehensive approach that accounts for the dynamic changes in IAP may provide deeper insights into its impact on AKI.

Conclusion

In summary, our research indicated that mildly elevated IAP within the range of 12–16 mmHg does not substantially raise the risk of AKI development. However, higher IAP levels are associated with increased rates of AKI, CRRT requirement, and 28-day mortality.

Data availability

Our data was obtained from MIMIC-IV2.2, This data can be found here: MIMIC-IV v2.2 (physionet.org), thus no more permission was required.

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Author contributions

Shenghui Miao and Mingkun Yang co-led this study. They conceptualized the research aims, planned analyses, and guided the literature review. Shenghui Miao extracted data from the MIMIC-IV database. Wen Li assisted with data processing and statistical analysis. Shenghui Miao and Mingkun Yang drafted the initial manuscript. Jing Yan provided feedback and approved the final manuscript. All authors reviewed and approved the final manuscript.

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Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

Use of the database was approved by the Institutional Review Boards of MIT and Beth Israel Deaconess Medical Center. As the database is anonymized and contains standardized data, this study did not require separate ethics approval per the Declaration of Helsinki. Therefore, this manuscript is exempt from the requirement for ethical approval statement and informed consent. The participants in the study have all passed the official ethics test and are qualified to access the database.

Additional information

Correspondence and requests for materials should be addressed to J.Y.