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Association between baseline intra-abdominal pressure and mortality risk in critically ill patients: A retrospective cohort study

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Article

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Abstract

Background:

Abnormally elevated intra-abdominal pressure (IAP) predisposes critically ill patients to multiple organ dysfunction. Although guidelines recommend routine IAP monitoring, the relative prognostic value of baseline IAP versus dynamic changes in IAP (ΔIAP) remains controversial. In complex critical care settings, medical interventions frequently introduce confounding by indication into ΔIAP measurements. Thus, identifying stable and independent IAP predictors of adverse outcomes is essential for optimizing early risk stratification. This study aimed to determine the independent predictive value of baseline IAP and ΔIAP for mortality across various time points and overall prognosis in critically ill patients, and to assess the incremental prognostic and clinical utility of adding baseline IAP to the standard SOFA score.

Methods:

We included 1,440 adult critically ill patients with documented baseline IAP from the MIMIC-IV database. Patients were stratified into three groups according to baseline IAP. Kaplan-Meier analysis and multivariable Cox proportional hazards models were used to assess the independent associations of baseline IAP and ΔIAP with 7-, 28-, 90-, and 365-day all-cause mortality. Optimal prognostic cutoffs were determined using receiver operating characteristic (ROC) curves and the maximum Youden index. Subgroup analyses were performed with interaction testing, and the likelihood ratio test (LRT), C-index, and net reclassification improvement (NRI) were calculated to quantify the incremental predictive value.

Results:

Multivariable Cox regression analysis identified high baseline IAP (> 20 mmHg) as an independent risk factor for 7-day mortality (HR:1.378). When treated as a continuous variable, each 1-mmHg increase in baseline IAP was independently associated with a 2.6% higher risk of 7-day mortality. Conversely, ΔIAP showed no independent predictive value for early mortality. Optimal cutoff analysis demonstrated that as the observation period extended from the early (7 days) to the medium- and long-term (28–365 days), the prognostic IAP threshold for mortality shifted from 19.5 mmHg to a stable 22.5 mmHg. Subgroup analyses verified the robust predictive performance of baseline IAP across diverse clinical subgroups. Furthermore, incorporating baseline IAP into the SOFA score significantly improved model goodness-of-fit (LRT P = 0.028) and risk reclassification capacity (NRI = 0.127).

Conclusions:

Baseline IAP is significantly associated with time-dependent mortality risk in critically ill patients, with early predictive utility superior to that of ΔIAP. Thus, it serves as a simple and robust predictor for early risk stratification in this population.

1. Background

Intra-abdominal pressure (IAP) refers to the pressure exerted by abdominal contents, including organs, fluid, and gas. Its abnormal elevation represents an important prognostic parameter in critically ill patients. According to the

2006 World Society of the Abdominal Compartment Syndrome (WSACS) consensus, intra-abdominal hypertension (IAH) is defined as a sustained IAP ≥ 12 mmHg. When IAH leads to new-onset organ dysfunction or failure, it progresses to abdominal compartment syndrome (ACS)[1], which is associated with significantly increased mortality. Thus, routine IAP monitoring has become standard practice for critically ill patients in modern intensive care units (ICUs).

Previous studies have demonstrated that elevated IAP is an independent risk factor for adverse outcomes in critically ill patients[2–3]. Its detrimental effects extend beyond intra-abdominal organ deterioration, exerting both direct and indirect impacts on multiple systemic organs[4]. Early clinical studies confirmed that elevated IAP impairs glomerular and tubular function, directly contributing to acute kidney injury (AKI)[5]. Concurrently, it compromises respiratory function by reducing pulmonary compliance and causing cranial displacement of the diaphragm, predisposing patients to pulmonary edema and atelectasis[6]. Furthermore, this diaphragmatic displacement mechanically compresses the heart and impedes systemic venous return. These combined effects decrease cardiac output, ultimately leading to visceral ischemia and hypoxia[7–8]. Thus, through multidimensional pathophysiological mechanisms, elevated IAP causes synergistic organ injury, driving the development of multiple organ dysfunction in critically ill patients.

In recent years, an increasing number of studies have investigated the association between IAP and clinical outcomes in critically ill patients; however, substantial knowledge gaps and controversies remain. Most studies have primarily examined the correlation between single IAP values and mortality, without clearly distinguishing the prognostic values of baseline IAP from that of IAP fluctuations (ΔIAP). Furthermore, IAP is highly susceptible to clinical interventions, including pharmacological therapy, abdominal decompression, and postural adjustments. As such, it remains unclear whether simple numerical changes in IAP can objectively reflect patient prognosis. Therefore, this retrospective cohort study was conducted to clarify the independent roles of baseline IAP and ΔIAP in prognostic assessment, after adjustment for relevant confounders. Ultimately, this study aims to address these gaps and provide more robust, evidence-based guidance for IAP monitoring and management in critically ill patients.

2. Methods

2.1 Study Design and Data Source

This study was a retrospective cohort analysis using data from the Medical Information Mart for Intensive Care IV (MIMIC-IV, version 3.1) database. Developed and maintained by the Laboratory for Computational Physiology at the Massachusetts Institute of Technology (MIT), the database includes comprehensive clinical data on patients admitted to the intensive care units (ICUs) at Beth Israel Deaconess Medical Center (BIDMC). The database was approved by the Institutional Review Boards (IRBs) of both MIT and BIDMC, and data usage complied with relevant ethical guidelines.

2.2 Study Population

Inclusion criteria: Critically ill patients admitted to the ICU with at least one valid baseline IAP measurement obtained within the first 24 hours of admission.

Exclusion criteria: (1) missing baseline IAP measurements or key clinical outcome data; (2) age < 18 or ≥ 90 years; (3) repeated ICU admissions (only the first admission was retained).

Patients were stratified into two groups according to their baseline IAP at ICU admission: a low IAP group (< 20 mmHg, n = 983) and a high IAP group (≥ = 20 mmHg, n = 457). This stratification aimed to investigate the associations of baseline IAP and ΔIAP with illness severity and clinical prognosis. The patient selection flowchart is presented in Fig. 1. Finally, 1440 critically ill patients were retained for the final analysis(Fig. 1).

2.3 Outcomes

Primary outcome: 7-day all-cause mortality.

Secondary outcomes: 28-, 90-, and 365-day all-cause mortality.

2.4 Variables and Parameters

2.4.1 Exposure Variables

Baseline IAP: The initial intra-abdominal pressure value measured within the first 24 hours of ICU admission.

Dynamic IAP changes (ΔIAP): The absolute change between the first and second consecutive IAP measurements.

2.4.2 Covariates and Clinical Parameters

The following clinical data were extracted from the MIMIC-IV database for confounding adjustment and baseline characterization:

(1) Demographic characteristics: age, sex, and body mass index (BMI).

(2) Comorbidities: diabetes, sepsis, acute kidney injury (AKI), cirrhosis, and history of abdominal surgery. The Charlson Comorbidity Index (CCI) was calculated to assess the comorbidity burden.

(3) Clinical interventions: mechanical ventilation, continuous renal replacement therapy (CRRT), and vasoactive agent use.

(4) Laboratory parameters: white blood cell (WBC) count, neutrophil count, lymphocyte count, platelet count, C-reactive protein (CRP), hepatic and renal function markers, electrolytes, and lactate.

(5) Disease severity and organ dysfunction scores: Sequential Organ Failure Assessment (SOFA), Acute Physiology and Chronic Health Evaluation III (APACHE III), and Logistic Organ Dysfunction System (LODS) scores.

Missing data for these covariates were handled by multiple imputation using the mice package in R.

2.5 Statistical Analysis

All statistical analyses were performed using R software (version 4.5.1). A two-sided P-value < 0.05 was considered statistically significant.

For baseline characteristic comparisons: Continuous variables were presented as medians with interquartile ranges (IQRs) and compared using the Kruskal-Wallis test. Categorical variables were expressed as frequencies (percentages) and compared using the chi-square test.

For survival analysis: Survival curves were constructed using the Kaplan-Meier method, and between-group differences in survival probability were assessed using the log-rank test.

For multivariable regression: Multivariable Cox proportional hazards regression models were used to evaluate the associations of baseline IAP and ΔIAP with all-cause mortality at each time point. Models were adjusted for potential confounders, including demographic characteristics, disease severity scores, and clinical interventions. Results were presented as hazard ratios (HRs) with corresponding 95% confidence intervals (95% CIs).

For optimal cutoff determination: Receiver operating characteristic (ROC) curves were used, and the maximum Youden index was calculated to identify the optimal cutoff values of baseline IAP for predicting mortality risk at each follow-up point.

For subgroup analysis: Subgroup analyses were performed by stratifying patients by age, sex, BMI, and SOFA score to evaluate the predictive performance of baseline IAP across different strata. Potential interactions between baseline IAP and these subgroup variables were also explored.

For predictive performance assessment: To evaluate the incremental predictive value of baseline IAP over the standard SOFA score, the goodness-of-fit between the base model (SOFA alone) and the augmented model (SOFA + IAP) was compared using the likelihood ratio test (LRT). The improvement in model discrimination was assessed by the change in the C-index. Furthermore, given that the C-index may lack sensitivity to model improvement, the continuous net reclassification improvement (NRI) and the integrated discrimination improvement (IDI), along with their 95% CIs, were also calculated. These metrics were used to quantify the comprehensive enhancement in risk reclassification and overall predicted probabilities following the incorporation of baseline IAP.

3. Results

3.1 Baseline Characteristics

Baseline patient characteristics differed significantly between the IAP groups (Table 1). Demographically, compared with the low IAP group (< 20 mmHg), the high IAP group (≥ 20 mmHg) had a higher proportion of males (68.3% vs. 57.0%, $P < 0.001$) and higher BMI levels (30.11 vs. 28.64 kg/m², $P < 0.001$), whereas their median age was significantly lower (59.00 vs. 62.00 years, $P = 0.010$).

Clinically, the incidence of acute kidney injury (AKI) in the high IAP group was significantly higher than that in the low IAP group (77.2% vs. 69.6%, $P = 0.003$). Concurrently, the median 24-hour urine output in the high IAP group was significantly lower (735.00 vs. 961.00 mL, $P < 0.001$). The high IAP group also had a significantly higher prevalence of acute pancreatitis (18.8% vs. 12.9%, $P = 0.003$). In contrast, no significant differences were observed between the two groups in the prevalence of diabetes, sepsis, and hypertension (all $P > 0.05$).

For disease severity scores, both APACHE III and SOFA scores were significantly higher in the high IAP group than in the low IAP group ($P = 0.014$ and $P = 0.037$, respectively), with the high IAP group having a median APACHE III score of 72.00. However, no significant intergroup difference was observed in the LODS score ($P = 0.075$).

For clinical interventions, the proportion of patients receiving CRRT was significantly higher in the high IAP group than that in the low IAP group (14.4% vs. 7.4%, $P < 0.001$). Conversely, the utilization rates of mechanical ventilation and vasoactive agents did not differ significantly between the two groups (all $P > 0.05$).

For laboratory findings, compared with the low IAP group, the high IAP group had significantly higher heart rate, WBC count, RDW, serum creatinine, BUN, serum potassium, total bilirubin, and lactate levels (all $P < 0.05$), whereas

their base excess levels were significantly lower (P = 0.002). Conversely, no significant differences were observed between the two groups in platelet count and AST/ALT levels (all P > 0.05).

BMI: Body mass index; BP: Blood pressure; MAP: Mean arterial pressure; WBC: White blood cell; PLT: Platelet; RDW: Red cell distribution width; BUN: Blood urea nitrogen; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; AKI: Acute kidney injury; CRRT: Continuous renal replacement therapy; SOFA: Sequential Organ Failure Assessment; APS III: Acute Physiology Score III; LODS: Logistic Organ Dysfunction System; LOS: Length of stay.

Table 1
Baseline characteristics of critically ill patients stratified by baseline IAP

Characteristic	Overall	Low IAP (< 20) N = 983	High IAP (≥ 20) N = 457	p-value[1]
Age (years)	61.00 (50.00, 72.00)	62.00 (51.00, 73.00)	59.00 (50.00, 70.00)	0.010
Gender, n (%)				< 0.001
F	568.0 (39.4%)	423.0 (43.0%)	145.0 (31.7%)	
M	872.0 (60.6%)	560.0 (57.0%)	312.0 (68.3%)	
BMI (kg/m²)	29.09 (25.23, 34.28)	28.64 (24.80, 33.51)	30.11 (26.20, 35.92)	< 0.001
Heart Rate (bpm)	99.00 (82.00, 115.00)	98.00 (81.00, 115.00)	102.00 (84.00, 117.00)	0.041
Respiratory Rate (bpm)	20.00 (16.00, 25.00)	20.00 (16.00, 25.00)	20.00 (16.00, 25.00)	0.612
Temperature (°C)	36.70 (36.30, 37.10)	36.70 (36.30, 37.10)	36.70 (36.20, 37.10)	0.204
Systolic BP (mmHg)	111.50 (97.00, 130.00)	112.00 (97.00, 130.00)	111.00 (97.00, 132.00)	0.758
Diastolic BP (mmHg)	63.50 (53.00, 76.00)	63.00 (52.00, 75.00)	65.00 (55.00, 77.00)	0.188
MAP (mmHg)	76.00 (64.00, 90.00)	75.00 (64.00, 90.00)	77.00 (63.00, 90.00)	0.786
24h Urine Volume (ml)	895.00 (376.00, 1649.00)	961.00 (409.00, 1725.00)	735.00 (270.00, 1462.00)	< 0.001
WBC count (×10⁹/L)	12.60 (8.30, 18.10)	12.30 (8.00, 17.80)	13.00 (8.90, 18.30)	0.032
PLT count (×10⁹/L)	179.00 (110.00, 264.00)	184.00 (117.00, 267.00)	172.00 (95.00, 259.00)	0.066
Hemoglobin (g/dL)	10.50 (8.80, 12.60)	10.40 (8.70, 12.50)	10.60 (8.90, 12.80)	0.121
RDW (%)	15.10 (13.90, 16.90)	15.00 (13.80, 16.80)	15.20 (14.10, 17.20)	0.028
Creatinine (mg/dL)	1.40 (0.90, 2.20)	130 (0.90, 2.10)	1.50 (1.00, 2.40)	< 0.001
BUN (mg/dL)	25.00 (16.00, 43.00)	24.00 (15.00, 43.00)	27.00 (17.00, 43.00)	0.022
Glucose (mg/dL)	142.00 (111.00, 184.00)	140.00 (111.00, 182.00)	146.00 (111.00, 188.00)	0.209
Potassium (mmol/L)	4.30 (3.80, 4.90)	4.20 (3.80, 4.80)	4.50 (4.00, 5.10)	< 0.001
Sodium (mmol/L)	138.00 (134.00, 141.00)	138.00 (134.00, 141.00)	138.00 (133.00, 141.00)	0.162
Calcium total (mg/dL)	7.90 (7.30, 8.60)	7.90 (7.30, 8.60)	7.90 (7.30, 8.60)	0.791
Albumin (g/dL)	2.80 (2.30, 3.30)	2.80 (2.30, 3.30)	2.80 (2.30, 3.30)	0.699
Total Bilirubin (mg/dL)	1.10 (0.50, 2.80)	1.00 (0.50, 2.50)	1.20 (0.60, 3.80)	0.004
AST (U/L)	67.00 (33.00, 176.50)	65.00 (33.00, 157.00)	71.00 (36.00, 208.00)	0.074

Characteristic	Overall	Low IAP (< 20) N = 983	High IAP (≥ 20) N = 457	p-value[1]
ALT (U/L)	36.00 (20.00, 100.00)	35.00 (19.00, 94.00)	39.00 (21.00, 115.00)	0.140
Lactate (mmol/L)	2.50 (1.60, 4.90)	2.40 (1.50, 4.50)	2.80 (1.70, 5.80)	< 0.001
PaO2 (mmHg)	89.50 (56.00, 168.00)	90.00 (56.00, 176.00)	89.00 (56.00, 151.00)	0.394
PaCO2 (mmHg)	40.00 (34.00, 48.00)	40.00 (34.00, 48.00)	40.00 (34.00, 48.00)	0.776
Base Excess	-4.00 (-9.00, 0.00)	-4.00 (-8.00, 0.00)	-5.00 (-10.00, 0.00)	0.002
Sepsis, n (%)	727.0 (50.5%)	499.0 (50.8%)	228.0 (49.9%)	0.758
AKI, n (%)	1037.0 (72.0%)	684.0 (69.6%)	353.0 (77.2%)	0.003
Acute Pancreatitis, n (%	213.0 (14.8%)	127.0 (12.9%)	86.0 (18.8%)	0.003
Diabetes, n (%)	369.0 (25.6%)	257.0 (26.1%)	112.0 (24.5%)	0.508
Hypertension, n (%)	570.0 (39.6%)	384.0 (39.1%)	186.0 (40.7%)	0.555
Mechanical Ventilation, n (%)	1,109.0 (77.0%)	762.0 (77.5%)	347.0 (75.9%)	0.505
Vasoactive Drugs, n (%)	876.0 (60.8%)	599.0 (60.9%)	277.0 (60.6%)	0.907
CRRT, n (%)	139.0 (9.7%)	73.0 (7.4%)	66.0 (14.4%)	< 0.001
SOFA Score	11.00 (8.00, 15.00)	11.00 (8.00, 15.00)	12.00 (9.00, 15.00)	0.037
APS III Score	68.00 (52.00, 89.00)	67.00 (50.00, 88.00)	72.00 (54.00, 91.00)	0.014
LODS Score	8.00 (6.00, 10.00)	8.00 (5.00, 10.00)	8.00 (6.00, 10.00)	0.075
Charlson Score	5.00 (3.00, 7.00)	5.00 (3.00, 7.00)	5.00 (3.00, 6.00)	0.151
Hospital Mortality, n (%)	555.0 (38.5%)	365.0 (37.1%)	190.0 (41.6%)	0.107
Survival time (days)	330.15 (8.79, 365.00)	364.16 (9.34, 365.00)	284.30 (6.14, 365.00)	0.313
LOS Hospital (days)	13.54 (6.10, 23.54)	13.65 (6.59, 23.97)	13.07 (5.23, 22.75)	0.105

BMI: Body mass index; BP: Blood pressure; MAP: Mean arterial pressure; WBC: White blood cell; PLT: Platelet; RDW: Red cell distribution width; BUN: Blood urea nitrogen; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; AKI: Acute kidney injury; CRRT: Continuous renal replacement therapy; SOFA: Sequential Organ Failure Assessment; APS III: Acute Physiology Score III; LODS: Logistic Organ Dysfunction System; LOS: Length of stay.

3.2 Association Between Baseline IAP and Mortality Risk

Multivariable Cox regression identified baseline IAP as an independent predictor of short-term mortality risk(Table 2). After adjustment for demographic characteristics, disease severity scores (SOFA, APACHE III, LODS), and clinical interventions, the high IAP group (> 20 mmHg) was significantly associated with a 37.8% higher risk of 7-day mortality (HR:1.378, 95%CI:1.015–1.871, P = 0.040). When treated as a continuous variable, each 1-mmHg

increase in baseline IAP was independently associated with a 2.6% higher risk of 7-day mortality (HR:1.026, 95%CI:1.005–1.047, P = 0.017).

Notably, the independent prognostic value of IAP was primarily concentrated in the early phase. Although univariate analysis indicated an association between IAP and medium- to long-term risk, after multivariable adjustment, neither the high IAP categorization nor the continuous IAP variable remained significantly associated with 28-, 90-, and 365-day mortality (all P > 0.05). These findings suggest that baseline IAP serves as a robust independent predictor of early mortality in critically ill patients, whereas long-term prognosis may be more comprehensively driven by overall illness severity and comorbidities.

Table 2
Multivariable Cox regression analysis for mortality risk at different time points Adjusted for: Age, Gender, BMI, SOFA, APACHE III, LODS, Vent, CRRT, Vasoactive

Variables	Univariate Analysis					Multivariate Analysis				
	β	S.E	Z	P	HR (95% CI)	β	S.E	Z	P	HR (95% CI)
7-day Mortality										
Low (≤ 20 mmHg)	-	-	-	-	1.00 (Reference)	-	-	-	-	1.00 (Reference)
High (> 20 mmHg)	0.400	0.154	2.604	0.009	1.491 (1.104–2.015)	0.321	0.156	2.056	0.040	1.378 (1.015–1.871)
Per 1 mmHg increase	0.028	0.010	2.891	0.004	1.029 (1.009–1.049)	0.025	0.011	2.392	0.017	1.026 (1.005–1.047)
28-day Mortality										
Low (≤ 20 mmHg)	-	-	-	-	1.00 (Reference)	-	-	-	-	1.00 (Reference)
High (> 20 mmHg)	0.136	0.120	1.135	0.256	1.145 (0.906–1.448)	0.068	0.121	0.563	0.573	1.070 (0.845–1.356)
Per 1 mmHg increase	0.014	0.008	1.901	0.057	1.015 (1.000–1.030)	0.010	0.008	1.297	0.195	1.010 (0.995–1.027)
90-day Mortality										
Low (≤ 20 mmHg)	-	-	-	-	1.00 (Reference)	-	-	-	-	1.00 (Reference)
High (> 20 mmHg)	0.137	0.107	1.287	0.198	1.147 (0.931–1.414)	0.068	0.108	0.631	0.528	1.070 (0.867–1.322)
Per 1 mmHg increase	0.015	0.007	2.207	0.027	1.015 (1.002–1.029)	0.010	0.007	1.450	0.147	1.010 (0.996–1.025)
365-day Mortality										
Low (≤ 20 mmHg)	-	-	-	-	1.00 (Reference)	-	-	-	-	1.00 (Reference)
High (> 20 mmHg)	0.148	0.083	1.774	0.076	1.159 (0.985–1.365)	0.091	0.084	1.084	0.278	1.096 (0.929–1.292)
Per 1 mmHg increase	0.013	0.005	2.312	0.021	1.013 (1.002–1.024)	0.009	0.006	1.517	0.129	1.009 (0.997–1.020)

3.3 Subgroup Analysis

Subgroup analysis demonstrated that the independent association between elevated baseline IAP and 7-day all-cause mortality was highly significant in the overall cohort (per 1-mmHg increase in IAP, HR:1.03, 95%CI:1.02–1.05, P < 0.001)(Fig. 2). Moreover, this prognostic value remained robust across most clinical strata.

Stratification by age and sex revealed that elevated IAP was consistently and significantly associated with increased mortality risk, irrespective of age (< 65 years: HR:1.03, P = 0.005; ≥ 65 years: HR:1.03, P = 0.008) or sex (male: HR:1.03, P < 0.001; female: HR:1.03, P = 0.038). Notably, no significant interactions were observed across these subgroups (all P for interaction > 0.05).

Stratification by sepsis further confirmed the consistency of the predictive value of IAP, regardless of whether patients had concurrent sepsis (sepsis group: HR:1.03, P = 0.008; non-sepsis group: HR:1.04, P = 0.002). Similarly, in subgroup analyses stratified by a history of abdominal surgery (surgery vs. non-surgery) and vasoactive agent use (use vs. non-use), baseline IAP remained an independent prognostic risk factor for 7-day all-cause mortality (all P < 0.05).

Of note, BMI stratification uncovered potential prognostic heterogeneity: among non-obese patients (BMI < 30 kg/m²), elevated IAP was significantly linked to elevated risk of 7-day all-cause mortality (HR:1.04, 95%CI:1.02–1.06, P < 0.001). By contrast, in the obese subgroup (BMI ≥ 30 kg/m²), although there was a trend toward heightened mortality risk, the association failed to reach statistical significance (HR:1.02, 95%CI:0.99–1.04, P = 0.176).

Regarding organ dysfunction and clinical interventions, this significant prognostic association persisted in patients with concurrent AKI (HR:1.03, 95%CI:1.01–1.05, P < 0.001) and those undergoing CRRT (HR:1.05, 95%CI:1.01–1.09, P = 0.006). Furthermore, mechanical ventilation stratification demonstrated that the positive association between raised IAP and mortality risk remained statistically significant regardless of mechanical ventilation use (use: HR:1.03, P < 0.001; non-use: HR:1.05, 95%CI:1.01–1.09, P = 0.016).

Figure 2 Subgroup analysis for the association between baseline IAP and 7-day all-cause mortality.

3.4 Survival Curve Analysis

Kaplan-Meier survival analysis revealed that baseline IAP levels were significantly associated with short-term prognosis in critically ill patients(Fig. 3). Stratified by a cutoff of 20 mmHg, the high IAP group (> 20 mmHg) and the low IAP group (≤ 20 mmHg) demonstrated a statistically significant difference in 7-day survival probabilities(P = 0.002), highlighting the crucial role of baseline IAP in risk stratification during the acute phase. However, as the follow-up period extended, the survival disparity between the two groups gradually attenuated. At the 28-day (P = 0.206), 90-day (P = 0.166), and 365-day (P = 0.086) observation points, the differences in survival trajectories failed to reach statistical significance (all P > 0.05). These findings indicate that the prognostic utility of baseline IAP is predominantly manifested in the early acute phase, with no substantial independent impact on medium- to long-term survival outcomes.

Figure 3 Kaplan-Meier survival curves for critically ill patients stratified by baseline IAP.

3.5 Optimal Cutoff Analysis

Optimal cutoff analysis was performed to determine optimal baseline IAP thresholds for predicting various prognostic endpoints in critically ill patients (Fig. 4). The analysis revealed that the optimal IAP cutoff for predicting short-term (7-day) mortality risk was 19.5 mmHg. For medium- to long-term prognosis, however, the optimal cutoffs demonstrated remarkable consistency: the prognostic thresholds for 28-, 90-, and 365-day mortality risks universally stabilized at 22.5 mmHg.

Figure 4 Optimal cutoff analysis of baseline IAP for predicting mortality at 7, 28, 90, and 365 days

3.6 Assessment of Predictive Performance

To quantify the incremental prognostic value of baseline IAP over the SOFA score for predicting early mortality risk, relevant incremental metrics were calculated (Table 3). The likelihood ratio test (LRT) revealed that incorporating baseline IAP into the SOFA model significantly improved model goodness-of-fit (χ^2= 4.85, P = 0.028). Concurrently, the C-index increased from 0.6686 to 0.6753, indicating a modest enhancement in model discrimination (Δ=+0.0067). Furthermore, reclassification analyses yielded an NRI of 0.127 (95% CI -0.024 to 0.279, P = 0.099) and an IDI of 0.003 (95% CI -0.001 to 0.007, P = 0.101).

IAP: Intra-abdominal pressure; SOFA: Sequential Organ Failure Assessment; NRI: Net reclassification improvement; IDI: Integrated discrimination improvement; CI: Confidence interval.

Table 3
Incremental predictive value of baseline IAP over the SOFA score

Statistical Metric	Value / Result	P Value
Model Fit (Likelihood Ratio Test)		
Chi-square	4.85	0.028
Discriminative Power (C-Index)		
SOFA Alone	0.6686	—
SOFA + IAP	0.6753	—
Net Improvement	+ 0.0067	—
Risk Reclassification (Continuous NRI)		
Total NRI (95% CI)	0.127 (-0.024–0.279)	0.099
Integrated Discrimination (IDI)		
IDI (95% CI)	0.003 (-0.001–0.007)	0.101

IAP: Intra-abdominal pressure; SOFA: Sequential Organ Failure Assessment; NRI: Net reclassification improvement; IDI: Integrated discrimination improvement; CI: Confidence interval.

3.7 Association Between Dynamic IAP Changes and Prognosis

In univariate analysis, ΔIAP exhibited no statistically significant associations with mortality at any time point (all P > 0.6)(Table 4). In the fully adjusted multivariable model, which incorporated demographic characteristics, disease severity scores, and clinical interventions, ΔIAP remained a non-significant predictor for both 7-day (HR:0.810, P = 0.123) and 28-day (HR:0.902,P = 0.306) mortality. Although a statistically significant inverse association was

observed between ΔIAP and long-term outcomes, specifically 90-day (HR:0.712, 95%CI:0.616–0.824, P < 0.001) and 365-day (HR:0.836, 95%CI:0.727–0.961,P = 0.012) mortality, these findings (HR < 1) contradict the well-established pathophysiological detriments of intra-abdominal hypertension and thus lack definitive clinical relevance. Taken together, these results suggest that the prognosis of critically ill patients is more closely linked to their baseline IAP at admission, rather than being driven by subsequent short-term fluctuations in ΔIAP.

Table 4
Association between dynamic IAP changes (ΔIAP) and mortality risk

Variables	Univariate Analysis					Multivariate Analysis				
	β	S.E	Z	P	HR (95% CI)	β	S.E	Z	P	HR (95% CI)
7-day Mortality										
ΔIAP	0.004	0.011	0.401	0.688	1.004 (0.983–1.027)	-0.210	0.137	-1.541	0.123	0.810 (0.620–1.059)
28-day Mortality										
ΔIAP	-0.003	0.008	-0.323	0.747	0.997 (0.981–1.014)	-0.103	0.101	-1.023	0.306	0.902 (0.740–1.099)
90-day Mortality										
ΔIAP	0.002	0.008	0.227	0.821	1.002 (0.987–1.017)	-0.340	0.074	-4.571	< 0.001	0.712 (0.616–0.824)
365-day Mortality										
ΔIAP	0.003	0.007	0.384	0.701	1.003 (0.989–1.017)	-0.179	0.071	-2.518	0.012	0.836 (0.727–0.961)

4. Discussion

Intra-abdominal pressure (IAP) represents an important prognostic parameter in critically ill patients. Its level reflects not only the physiological state of intra-abdominal organs but also directly influences the perfusion and function of multiple systemic organ systems[9]. Under normal physiological conditions, IAP is typically maintained between 0 and 5 mmHg. However, in critically ill states, its pathological elevation acts not merely as a surrogate marker of illness severity, but rather as an initiating factor that drives clinical deterioration[10]. Previous studies have demonstrated that persistently elevated IAP can lead to intra-abdominal organ hypoperfusion, thereby decreasing renal blood flow and heightening the risk of acute kidney injury. Furthermore, it can cause intestinal ischemia and trigger the cascade of inflammatory cytokines, ultimately leading to systemic inflammatory response syndrome (SIRS)[11]. Concurrently, elevated IAP impedes systemic venous return and elevates intrathoracic pressure, resulting in decreased cardiac output and increased respiratory resistance, which can ultimately culminate in multiple organ failure[12]. Despite current guidelines[13] recommending routine IAP monitoring for high-risk patients, the relative prognostic importance of baseline IAP versus ΔIAP remains highly debated. In complex critical care settings, relying solely on dynamic IAP changes is often hampered by the confounding effects of therapeutic interventions,

making it difficult to accurately reflect the patient's true degree of critical illness. Thus, identifying an early, stable, and independent IAP predictor of adverse outcomes is essential for guiding clinical decision-making.

To address the critical care need for accessible and accurate biomarkers to evaluate illness severity, this study investigated the prognostic value of baseline IAP for mortality risk and clinical outcomes in critically ill patients. Our findings demonstrate that baseline IAP serves as a reliable biomarker for risk stratification in this population, effectively compensating for the limitations of standard scoring systems, including the SOFA score, in evaluating intra-abdominal pathology. Survival analysis further confirmed a significant association between baseline IAP and short-term mortality. Notably, its predictive utility was significantly superior to that of ΔIAP, thereby offering a practical reference tool to guide individualized abdominal decompression strategies.

Analysis of baseline characteristics revealed significant clinical heterogeneity among critically ill patients stratified by IAP, with these disparities exhibiting a gradient pattern corresponding to IAP elevation.Regarding demographic characteristics, the high-IAP group exhibited a significantly higher BMI and a male predominance. This observation suggests that males with high BMI may constitute a susceptible population for IAH. This susceptibility is attributable not only to the direct mass effect of visceral fat accumulation but also to a reduced abdominal wall compliance[14]. Consequently, an elevated IAP in such individuals often signifies a more severe underlying clinical condition and a potential need for surgical intervention[15]. Furthermore, severely obese patients often present with chronically elevated IAP levels, suggesting that they may clinically exist in a state of chronic intra-abdominal hypertension[16].For organ function, the incidence of AKI in the high IAP group reached a staggering 77.2%, accompanied by a significant reduction in urine output. This finding corroborates the pathophysiological mechanism whereby elevated intra-abdominal pressure causes renal injury through mechanical compression of the renal parenchyma and impedance of venous return, leading to a precipitous drop in the effective glomerular filtration gradient[17–18]. Concurrently, patients in the high IAP group had significantly elevated serum lactate levels and decreased base excess. These metabolic alterations not only reflect severe systemic tissue hypoperfusion and anaerobic metabolism but also serve as early, sensitive indicators of visceral microcirculatory ischemia[19]. The perpetuation of such metabolic derangements, coupled with sustained IAH, creates a pathophysiological "cumulative risk" effect, ultimately leading to multiple organ failure and adverse clinical outcomes[18–19].Additionally, disease severity assessments indicated that both SOFA and APACHE III scores escalated in tandem with IAP, implying that patients with high IAP have more complex clinical conditions and a significantly increased demand for CRRT. These findings underscore that for patients with high baseline IAP, clinicians should promptly implement more aggressive organ support therapies and individualized, targeted interventions to improve prognosis.

Kaplan-Meier survival analysis further substantiates the utility of baseline IAP as a reliable biomarker for survival risk stratification in critically ill patients. In the 7-day survival analysis, the survival trajectory of the high IAP group showed a precipitous decline, and the overall divergence between the two survival curves was statistically significant ($P < 0.05$). This finding suggests that the severe detrimental impacts of IAH on critically ill patients are predominantly concentrated in the acute phase. As the follow-up duration extended, the statistical significance of the log-rank test progressively attenuated. Beyond the acute phase, the survival curves of the two groups tended to run parallel, indicating the absence of a substantial subsequent increase in mortality risk.

Multivariable Cox regression demonstrated that, even after adjustment for multiple confounders, baseline IAP remains a critical independent predictor of short-term (7-day) mortality risk. This implies that the severe pathophysiological insults inflicted by early IAH may trigger irreversible organ damage[20–21], thereby dictating early survival outcomes. However, with extended follow-up, baseline IAP lost its significant predictive value for 28-day

and longer-term survival. This indicates that once patients survive the acute phase, their long-term prognosis is predominantly dictated by the control of the primary disease, underlying comorbidities, and the recovery of organ function[22], with baseline IAP relinquishing its dominant predictive role.By contrast, our study confirmed that ΔIAP is not a reliable biomarker for predicting mortality risk. In both univariate analysis and the adjusted early (7-day and 28-day) multivariable models, ΔIAP failed to demonstrate significant predictive utility. Although the multivariable model revealed a significant inverse association between ΔIAP and long-term (90-day and 365-day) mortality risk, this contradicts the core pathophysiological mechanisms of IAH and lacks practical clinical relevance. This paradoxical phenomenon is fundamentally attributable to "confounding by indication" bias within the critical care setting[23]: essentially, the more critically ill and high-risk the patients are, the more likely clinicians are to implement aggressive decompression interventions, including paracentesis or laparostomy. As such, the high long-term mortality observed in this cohort is actually a continuation of the critical severity of their underlying diseases, rather than an independent predictive effect of the dynamic IAP changes.

Using optimal cutoff analysis, this study provides a robust quantitative basis for risk stratification in critically ill patients. The results indicated that the optimal cutoff for predicting early (7-day) mortality risk was 19.5 mmHg. Translated into clinical practice, this highlights 20 mmHg as a critical clinical threshold for identifying high-risk patients during the acute phase. This value aligns perfectly with the diagnostic criteria for Grade III IAH in the WSACS guidelines[24], indirectly reflecting the profound susceptibility of critically ill patients to organ hypoperfusion during the early stages of illness[25]. As the follow-up period extended, the optimal threshold for medium- to long-term mortality risk shifted upward and stabilized at 22.5 mmHg. This upward shift in the threshold suggests that once patients survive the acute phase, their physiological systems may develop a degree of compensation or tolerance to moderate IAH. As such, a more severe intra-abdominal pressure burden (≥ 23 mmHg) is required to reliably predict long-term outcomes. These findings not only validate the core value of baseline IAP in early risk prognostication but also advise clinicians to reference differentiated intervention thresholds when addressing various prognostic endpoints, thereby facilitating more precise, stratified management of critically ill patients.

Furthermore, subgroup analyses corroborated the absence of significant interactions between baseline IAP and any clinical subgroup variables (all P for interaction > 0.05). This indicates that the prognostic utility of this parameter is broadly applicable across critically ill patients with diverse clinical profiles. Of particular interest, BMI stratification uncovered potential prognostic heterogeneity: among non-obese patients (BMI < 30 kg/m², P < 0.001), elevated IAP was significantly linked to elevated risk of mortality, whereas the association failed to reach statistical significance in the obese subgroup (P = 0.176).Although the interaction test between these strata was negative (P = 0.150)—suggesting that this discrepancy may stem from an underpowered analysis driven by the smaller sample size of the obese subgroup—this trend, when contextualized with previous literature, poses a compelling hypothesis: obese patients often exhibit chronically elevated baseline IAP due to the persistent accumulation of intra-abdominal fat[26], and this prolonged mechanical tension may cause a chronic, compensatory remodeling of abdominal wall compliance[27], thereby elevating their tolerance threshold to acute surges in IAP. Given the inherent limitations of an observational design, this study cannot draw definitive conclusions regarding this phenomenon; the underlying pathophysiological mechanisms warrant validation in future prospective studies. Taken together, these findings not only reaffirm the robustness of the prognostic value of baseline IAP but also highlight its unique role in identifying high-risk subpopulations, including non-obese patients, providing a strong rationale for precise risk stratification in critical care.

Although the SOFA score is a classical tool for evaluating organ function in critically ill patients, it possesses an evaluation blind spot in intra-abdominal pathophysiological dimensions. Therefore, this study investigated the

incremental prognostic value of adding baseline IAP to the SOFA score. The results showed that incorporating baseline IAP into the SOFA model significantly improved model goodness-of-fit (P = 0.028), suggesting that baseline IAP acts as a mortality risk factor independent of the SOFA score. However, the incremental value analysis revealed that the C-index of the combined model increased by only 0.0067, indicating a modest improvement in discrimination. Furthermore, the IDI did not reach statistical significance(P = 0.101), failing to substantiate a substantial enhancement in predictive performance. Only the NRI showed a marginal positive trend (P = 0.099), hinting that baseline IAP may provide supplementary risk stratification reference for a subset of patients with occult intra-abdominal hypertension. In conclusion, while baseline IAP serves as a potential adjunctive indicator to the SOFA score, its current incremental clinical utility is limited and warrants further validation in large-scale, multicenter studies.

Based on these findings, baseline IAP emerges as a simple and robust biomarker for predicting acute-phase prognosis in critically ill patients. It can serve as a potential adjunctive indicator to the SOFA score, facilitating early risk stratification in this vulnerable population.However, several limitations of this study must be acknowledged. First, owing to its retrospective observational design, causality cannot be inferred. Furthermore, despite rigorous multivariable adjustment, the potential influence of unmeasured or residual confounders cannot be completely ruled out. Second, because the data were derived from a single-center database, the generalizability of our conclusions to other clinical settings may be limited.Future large-scale, multicenter prospective cohort studies are urgently needed to further validate the exact clinical utility of baseline IAP, thereby providing a more robust evidence-based foundation for the standardized management of intra-abdominal hypertension in critical care.

5. Conclusions

In conclusion, this study systematically investigated the prognostic value of baseline IAP in critically ill patients. Our findings demonstrate a significant, time-dependent dose-response relationship between baseline IAP and all-cause mortality, firmly establishing baseline IAP as an independent predictor of acute-phase mortality risk. Compared with ΔIAP, baseline IAP exhibited superior stability and reliability in its predictive performance. Furthermore, this prognostic utility remained highly robust across patient subgroups with diverse clinical profiles.

Abbreviations

IAP
Intra-abdominal pressure
ΔIAP
Dynamic changes in IAP
IAH
Intra-abdominal hypertension
ACS
Abdominal compartment syndrome
ICU
Intensive care unit
AKI
Acute kidney injury
WSACS
World Society of the Abdominal Compartment Syndrome

MIMIC-IV
Medical Information Mart for Intensive Care IV
MIT
Massachusetts Institute of Technology
BIDMC
Beth Israel Deaconess Medical Center
IRB
Institutional Review Board
BMI
Body mass index
CCI
Charlson Comorbidity Index
CRRT
Continuous renal replacement therapy
WBC
White blood cell
CRP
C-reactive protein
SOFA
Sequential Organ Failure Assessment
APACHE III
Acute Physiology and Chronic Health Evaluation III
LODS
Logistic Organ Dysfunction System
HR
Hazard ratio
CI
Confidence interval
ROC
Receiver operating characteristic
LRT
Likelihood ratio test
NRI
Net reclassification improvement
IDI
Integrated discrimination improvement
SIRS
Systemic inflammatory response syndrome

Declarations

Ethics approval and consent to participate

The data used in this study were obtained from the MIMIC-IV database. The establishment of this database was approved by the Institutional Review Boards (IRBs) of the Massachusetts Institute of Technology (MIT) and Beth

Israel Deaconess Medical Center (BIDMC). The requirement for individual informed consent was waived because the project did not impact clinical care and all protected health information was deidentified.

Consent for publication

Not applicable.

Availability of data and materials

The datasets analysed during the current study are available in the MIMIC-IV repository (https://physionet.org/content/mimiciv/). Access to this database is restricted and granted only to researchers who have completed the required credentialing (e.g., CITI training) and signed the data use agreement. The original contributions presented in this study are included in this article, and further inquiries or intermediate analysis datasets are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

JG and CLL analyzed the data and drafted the manuscript. RF, ZL, QB, LW, TB, and LK participated in data collection, statistical analysis, and literature review. FZ designed the study, obtained funding, and critically revised the manuscript. All authors read and approved the final manuscript.

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Authors' information

Not applicable.

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Figures

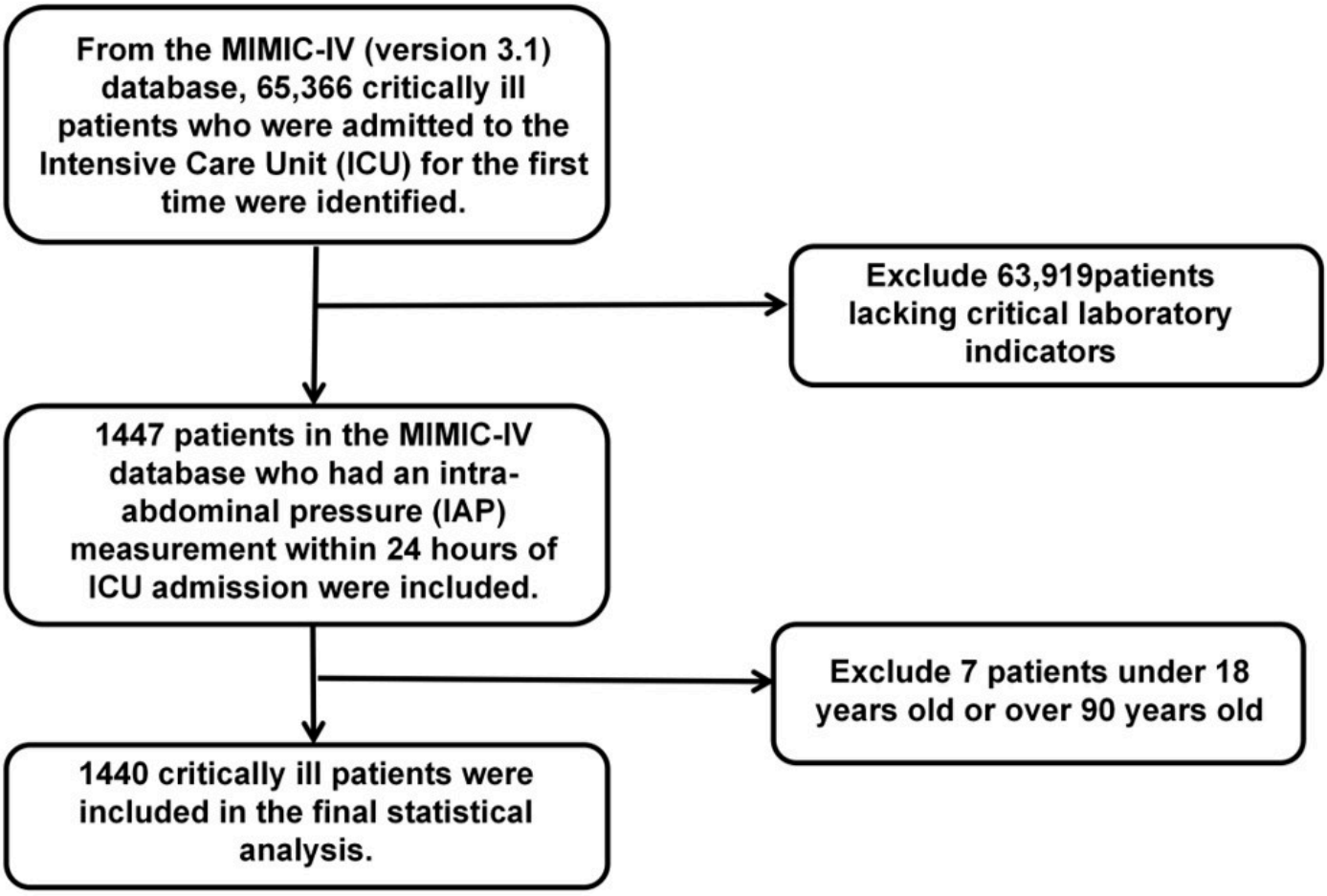


Figure 1

Flowchart of patient selection

Subgroup	n (%)	Events/Total	HR (95% CI)	P-value	P for interaction
All patients	1440 (100)	333/1440	1.03 (1.02 - 1.05)	<0.001	
Age: <65	827 (57.4)	166/827	1.03 (1.01 - 1.05)	0.005	0.939
Age: ≥65	613 (42.6)	167/613	1.03 (1.01 - 1.05)	0.008	
Gender: F	568 (39.4)	131/568	1.03 (1.00 - 1.05)	0.038	0.750
Gender: M	872 (60.6)	202/872	1.03 (1.01 - 1.05)	<0.001	
BMI: <30	811 (56.3)	186/811	1.04 (1.02 - 1.06)	<0.001	0.150
BMI: ≥30	629 (43.7)	147/629	1.02 (0.99 - 1.04)	0.176	
Sepsis: No	713 (49.5)	125/713	1.04 (1.01 - 1.06)	0.002	0.275
Sepsis: Yes	727 (50.5)	208/727	1.03 (1.01 - 1.05)	0.008	
AKI: No	403 (28.0)	53/403	1.04 (1.00 - 1.07)	0.055	0.291
AKI: Yes	1037 (72.0)	280/1037	1.03 (1.01 - 1.05)	<0.001	
Mech Vent: No	331 (23.0)	67/331	1.05 (1.01 - 1.09)	0.016	0.961
Mech Vent: Yes	1109 (77.0)	266/1109	1.03 (1.01 - 1.05)	<0.001	
CRRT: No	1301 (90.3)	267/1301	1.03 (1.01 - 1.04)	0.004	0.360
CRRT: Yes	139 (9.7)	66/139	1.05 (1.01 - 1.09)	0.006	
Vasoactive: No	564 (39.2)	78/564	1.04 (1.00 - 1.07)	0.029	0.738
Vasoactive: Yes	876 (60.8)	255/876	1.03 (1.01 - 1.05)	<0.001	
Abd Surgery: No	601 (41.7)	152/601	1.03 (1.00 - 1.05)	0.030	0.496
Abd Surgery: Yes	839 (58.3)	181/839	1.03 (1.01 - 1.05)	0.001	

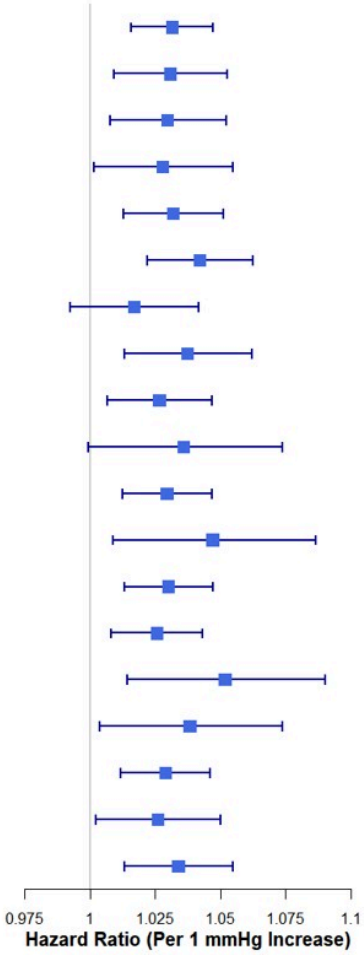


Figure 2

Figure 2

Subgroup analysis for the association between baseline IAP and 7-day all-cause mortality.

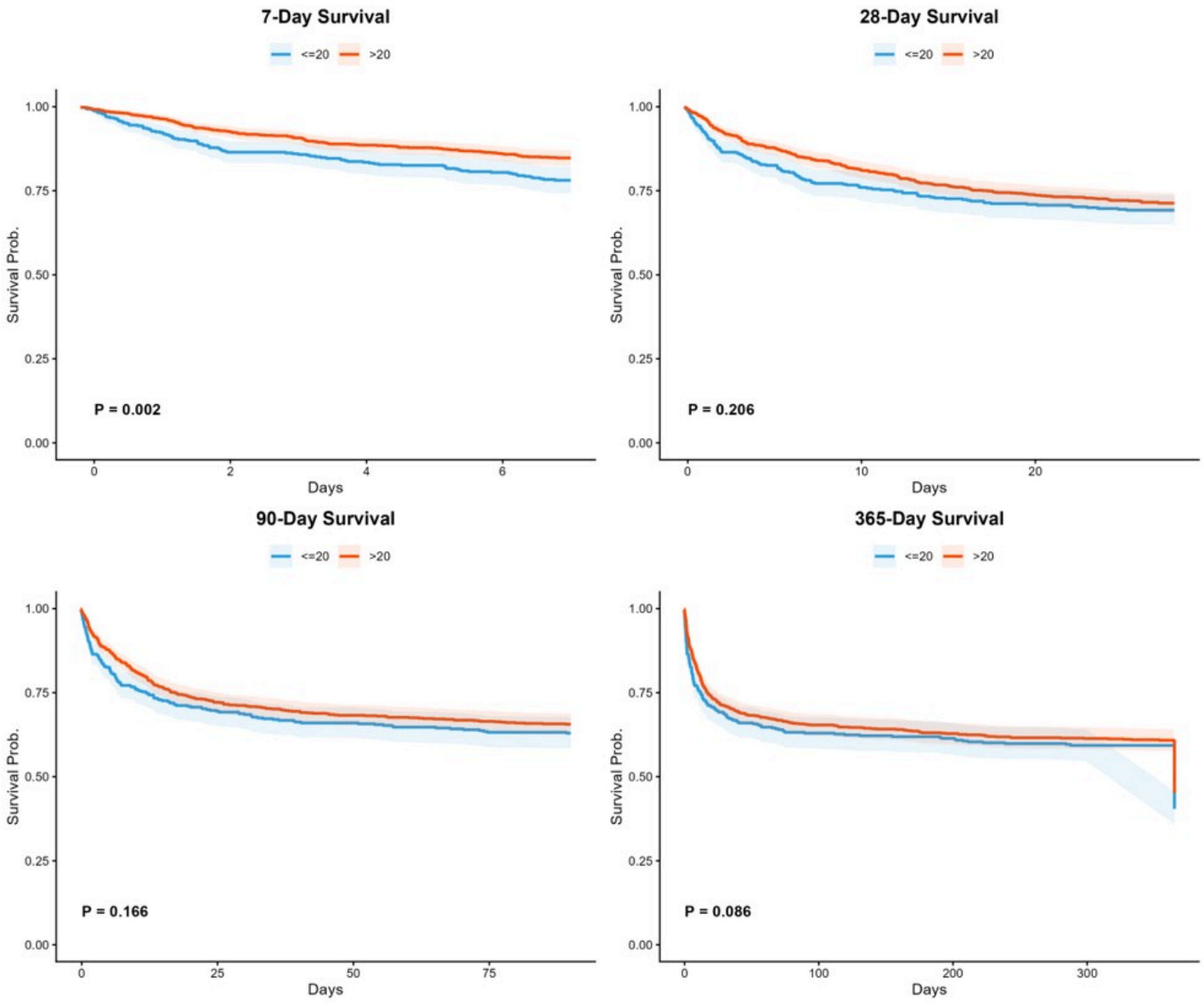


Figure 3

Figure 3

Kaplan-Meier survival curves for critically ill patients stratified by baseline IAP.

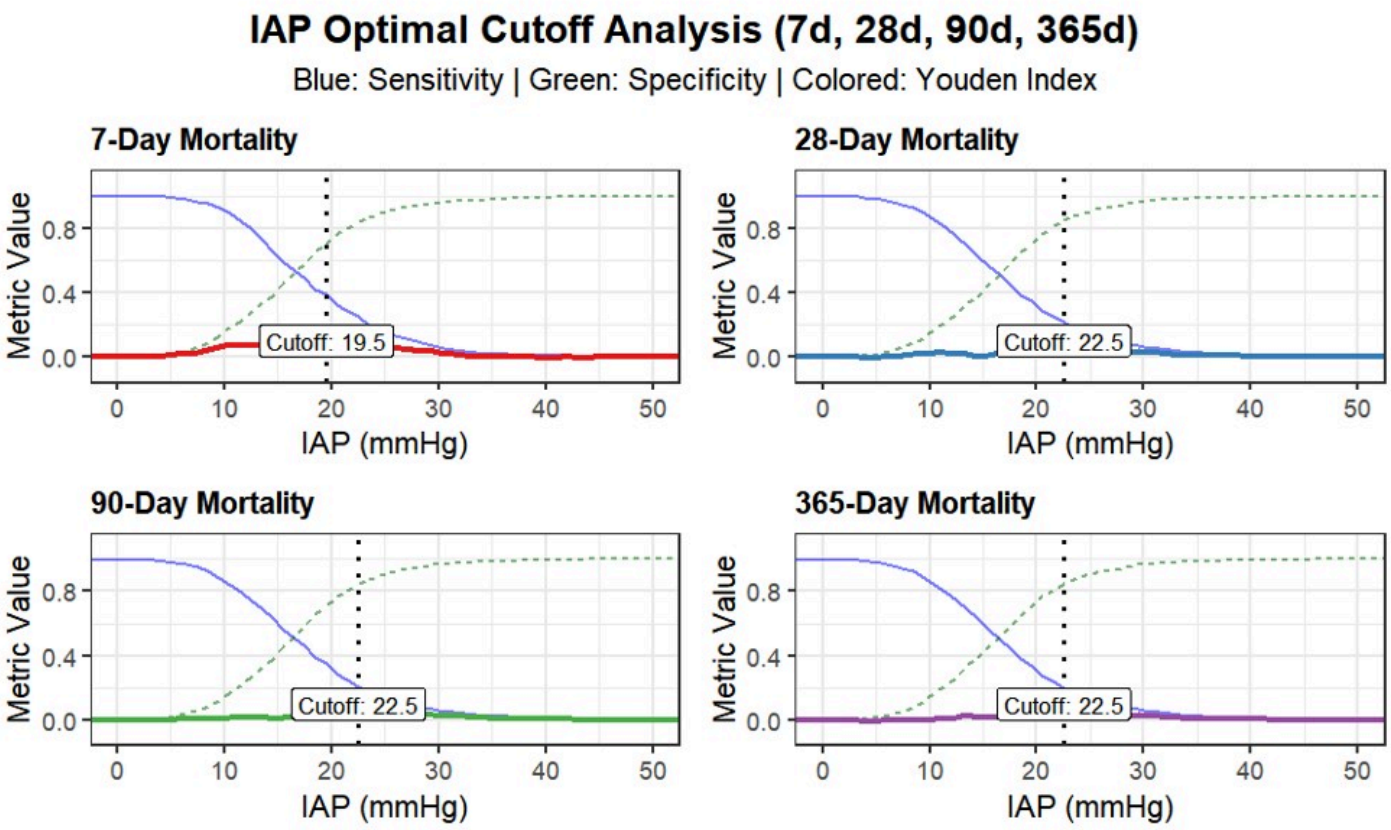


Figure 4

Figure 4

Optimal cutoff analysis of baseline IAP for predicting mortality at 7, 28, 90, and 365 days