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RESEARCH ARTICLE

Dong et al: Braden score and 30-day mortality in AP

Braden score at ICU admission predicts 30-day mortality in acute pancreatitis

Lihong Dong[1*], Hong Wang[2], Xixiang Yang[3], Xiaolin Zhu[1], Chen He[1]

[1]Department of Critical Care Medicine, The First Affiliated Hospital of Kunming Medical University, Kunming, China;

[2]Department of Critical Care Medicine, The Sixth Affiliated Hospital of Kunming Medical University, Yuxi, China;

[3]Second Clinical College, Ningxia Medical University, Yinchuan, China.

*Correspondence to Lihong Dong: 2375126670@qq.com.

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ABSTRACT

The Braden score, a bedside assessment tool for evaluating the risk of pressure ulcers and frailty, may identify vulnerabilities pertinent to outcomes in acute pancreatitis (AP). However, its prognostic significance in this context remains uncertain. This study aimed to determine whether the Braden score at admission predicts all-cause mortality in intensive care unit (ICU) patients with AP and whether it provides additional value to existing clinical models. In a retrospective single-center cohort study utilizing data from MIMIC-IV v3.1 (2008–2022), we included 1,985 adults diagnosed with AP. We analyzed the Braden score as both a continuous variable and a dichotomous variable (high-risk: ≤15 vs. low-risk: >15), with 30-day mortality as the primary endpoint (with secondary endpoints at 90, 180, and 360 days). Our methodology encompassed Kaplan–Meier analysis, multivariable Cox regression, restricted cubic splines, receiver operating characteristic curves, and calibration assessments. By the 30-day mark, a total of 230 deaths were recorded (11.6%). Each 1-point increase in the Braden score correlated with a 7.7% reduction in mortality risk (HR 0.923, 95% CI 0.873–0.976; p=0.005). Furthermore, patients categorized as low-risk experienced lower mortality rates compared to high-risk patients (HR 0.688, 95% CI 0.501–0.945; p=0.021). The discrimination capability at 30 days was moderate (AUC 0.67, 95% CI 0.63–0.71), with an optimal cutoff score of 15 (sensitivity 61%, specificity 65%) and good calibration; however, performance diminished over longer durations. Incorporating the Braden score into a baseline clinical model enhanced predictive accuracy (AUC 0.712 vs. 0.647; NRI 0.235; IDI 0.040; all p<0.001). The Braden score at ICU admission is independently associated with 30-day mortality in patients with AP, providing moderate, well-calibrated predictions and significant incremental value. This supports its application as an early and straightforward tool for risk stratification, pending prospective validation.

Keywords: Braden score, acute pancreatitis, risk of death, MIMIC-IV database.

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory disease of the pancreas marked by premature activation of multiple digestive enzymes, resulting in self-digestion of the pancreas. As the condition progresses, it results in systemic inflammatory responses and even organ failure(1). AP is a prevalent gastrointestinal disease, showing an annual incidence of 13-45 cases per 100,000 people(2). Over the last 20 years, the incidence and hospitalization rates of AP have continued to rise, placing a heavy burden on patients, families, and the healthcare system(3). The prognosis of AP depends on its severity. About 75-80% of patients experience slow progression and can be cured with intravenous infusion and supportive care(4, 5). However, nearly 20% of patients develop moderate or severe AP, along with pancreatic or peripancreatic tissue necrosis and even organ failure, resulting in an overall mortality rate of 20% to 40%(6, 7). Therefore, identifying potent prognostic indices to stratify high-risk populations with poor outcomes holds critical clinical significance.

Currently, the Ranson criteria(8), Balthazar grading(9), APACHE-II(8), Sequential Organ Failure Assessment (SOFA)(10), and bedside index for severity in AP(11) are commonly used scoring systems for predicting AP severity and prognosis. These scores assist in better understanding the course of AP. Nonetheless, most are complex and require time to collect sufficient data, which enhances the mortality risk due to missing the optimal treatment window. In addition, current research has identified several biomarkers associated with AP prognosis, including procalcitonin (PCT), C-reactive protein, interleukin-6, red blood cell distribution width, albumin, creatinine (Cr), blood urea nitrogen (BUN), and serum calcium (Ca)(12-16). However, due to the complex pathophysiological state of patients, the correlation between these single indicators and AP mortality risk is unsatisfactory(17). Thereby, there is a pressing need for simpler, faster, highly reproducible, and sensitive indices to measure the all-cause mortality (ACM) risk of AP.

The Braden scale is extensively used for evaluating pressure ulcer risk(18) and identifying frailty(19). It includes six dimensions: sensory perception, moisture, mobility, activity, nutritional status, and friction/shear force. Since the Braden score is easy to gain and does not need laboratory data, it is widely applied in medical, surgical, and intensive care settings(20, 21). As research has progressed, the applicability of the Braden score has expanded to effectively forecast adverse clinical outcomes in critically ill patients, including MI, ischemic stroke, delirium, COVID-19, traumatic brain injury, sepsis, and cardiac patients in the intensive care unit (ICU)(22-29). Although the Braden score was originally developed to evaluate pressure ulcers, its ability to evaluate patients' overall frailty has sparked broader interest in its clinical applications. This may be related to its multidimensional assessments (e.g., mobility and nutritional status), which may be critical to the initiation and progression of AP(30-32). However, currently, no studies confirm the link between Braden scores and ACM risk in AP. Therefore, this study aims to explore the link between Braden scores and ACM risk to provide a simple, early risk assessment tool for AP patients, and further reveal the link between mobility/nutritional status in the Braden score and AP prognosis.

MATERIALS AND METHODS

Study population

Data used in this study were sourced from the MIMIC-IV database (3.1 version), a large public database developed by the Computational Physiology Laboratory at the Massachusetts Institute of Technology, which covers detailed records of all patients admitted to Beth Israel Deaconess Medical Center from 2008 to 2022(33). To ensure patient privacy, personal data were all de-identified, with patient identifiers replaced by random codes, thereby exempting the study from ethical approval and informed consent. The first author LhD completed the Collaborative Institutional Training Initiative course and passed the Conflict of Interest and Data or Specimen Research

Only exams (ID: 14326940), gaining authorization to access the database and extract the relevant variables required. Our study obeyed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines(34).

According to the ICD-9 code 577.0 and the ICD-10 codes K85-K85.92, ICU admission data for AP patients were harvested. Patients meeting the following criteria were excluded: (1) Patients younger than 18 years old at the time of initial admission; (2) Patients not admitted to the ICU; (3) For patients with repeated admissions for AP, only data from the first admission were retained; (4) Patients with missing Braden assessment records (Fig 1).

Braden score assessment

The Braden score was developed in 1987 by American nurses Barbara Braden and Nancy Bergstrom and is a widely used clinical tool for evaluating patients' risk of pressure ulcers(35). The Braden score was assessed by the ICU nurse using the ward's standardized Pressure Ulcer Risk Screening Form after the patient's admission. Before assessment, the nurse must complete online training and pass the assessment. Here, the Braden score at ICU admission was used as the exposure factor, which included six key components: sensory perception, moisture, mobility, activity, nutritional status, and friction/shear force(36). Scores for each dimension ranged from 1 to 4, except for friction/shear, which ranged from 1 to 3. The total score ranged from 6 to 23 points, with lower scores indicating a greater risk of pressure ulcers(37). A cutoff value of 15 was utilized to allocate participants into low-risk group (Braden score > 15) and high-risk group (≤ 15) under clinical experts' experience and previous articles(25, 29).

Outcome variables

The primary outcome was ACM risk at 30 days. Secondary outcomes included the ACM risk at 90 days, 180 days, and 360 days. The time origin for all survival analyses was defined as the ICU admission date. Patients were followed from ICU admission until the earliest occurrence of any of the following events: (1) death from

any cause; (2) the prespecified follow-up period (30 days, 90 days, 180 days, or 360 days); or (3) the last recording in the MIMIC-IV database. Death events were identified through the "dod" (date of death) variable in the MIMIC-IV database, which integrated hospital records and external state-level death registry data. Patients surviving without recorded deaths during follow-up were censored at the earlier end of the follow-up period or their last database recording. This approach ensured consistent identification of in-hospital and out-of-hospital deaths while minimizing informative censoring due to follow-up loss.

Data extraction

Data were extracted using PostgreSQL software (version 17) and Navicat Premium software (version 17.2.3) through structured query language. The following variables were obtained: 1) Demographic data: age, sex, marital status, and ethnicity; 2) Vital signs: heart rate, respiratory rate (RR); 3) Comorbidities were determined based on ICD-9 or ICD-10: mild or severe liver disease, kidney disease, malignant tumors, chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), peripheral vascular disease (PVD), myocardial infarction (MI), hypertension (HP); 4) Laboratory indicators: white blood cell count (WBC), platelet count (PLT), hemoglobin (HGB), hematocrit (HCT), prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio (INR), lactate, albumin (ALB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatinine (Cr), BUN, total bilirubin (TB), calcium (Ca), and blood glucose (BG); 5) Clinical treatment: drugs (norepinephrine, statins), mechanical ventilation (MV); 6) Disease scores: Braden score, SOFA score, and Glasgow Coma Scale (GCS). For data with multiple measurements, we extracted the values measured on the first day of ICU admission.

Methods for outliers and missing value

To address potential bias caused by sample exclusion, variables with missing values over 20% were eliminated, and variables with missing data less than 20% were imputed using the random forest imputation method (MissForest)(38). Variables with

outliers were handled using the winsorize method, with 1% and 99% as the cutoff points(39, 40).

Statistical analysis

The normality of continuous variables was determined by the Shapiro-Wilks test. For normal distribution, continuous variables were delineated as mean ± standard deviation, and skewed distributions were delineated as median (interquartile range [IQR]). Normally distributed continuous variables were analyzed by t-tests, while skewed variables were analyzed utilizing Mann-Whitney U tests. Categorical data were portrayed as percentages (%) and processed via the chi-square test or Fisher's exact test. Patients were classified into high-risk and low-risk groups as per the Braden score. Survival curves were drawn using the Kaplan-Meier (KM) method, and intergroup comparisons were made utilizing the log-rank test. Cox regression models were leveraged to determine the link between Braden scores and endpoints, generating hazard ratios (HR) and 95% confidence intervals (CI). Three models were created: Model 1 (unadjusted model), Model 2 (control for age, sex, marital status, and race), and Model 3 [considered demographic information, vital signs, laboratory indicators (albumin, AST, BUN, lactate, Ca, Cr, BG, HCT, PLT, PT, PTT, TB, WBC), comorbidities, clinical treatment, and GCS score]. To avoid multicollinearity, the variance inflation factor (VIF) was estimated for each variable, and variables with VIF > 5 were excluded. We found that ALT (VIF = 6.02), HB (VIF = 24.15), and INR (VIF = 25.04) all exceeded 5 and thus were excluded. Meanwhile, although the initial VIFs for AST, HCT, and PT exceeded 5, their values decreased to 1.63, 1.34, and 1.83, respectively, after excluding the aforementioned high-VIF variables. This indicated that the high correlations among these variables were primarily driven by the excluded variables (Supplementary Fig 1-2). Subsequently, restricted cubic spline (RCS) curves were employed to study the potential linear relationship between Braden scores and ACM risk (The three nodes correspond to the 10th, 50th, and 75th percentiles of the Braden score, with the time origin being ICU admission). Receiver

operating characteristic (ROC) analysis was leveraged to examine the predictive power of Braden scores for ACM risk at 30, 90, 180, and 360 days after ICU admission, to determine the sensitivity and specificity, and to calculate the area under the curve (AUC). The net reclassification improvement (NRI) and integrated discrimination improvement (IDI) were calculated to assess the additional predictive value of the Braden scale for ACM risk in AP patients. A calibration curve was plotted to assess the consistency between model predictions and actual observations. Subgroup analyses were implemented to inspect the relationship in different subgroups: age, sex, marital status, ethnicity, mild or severe liver disease, kidney disease, malignant tumors, COPD, CHF, PVD, MI, HP, norepinephrine, statins, and MV. The log-likelihood ratio test was adopted to assess the interaction between the Braden score and variables. All data processing, analysis, and graph generation were performed using R software 4.4.3. p-value < 0.05 implied statistical significance.

Declaration on exploratory analysis

All subgroup analyses and comparisons of long-term endpoints at 90, 180, and 360 days were exploratory. No adjustments were made for multiple testing. These results were used only to generate hypotheses and identify potential signals and should not be considered definitive conclusions. Further validation in independent prospective cohorts is required.

RESULTS

Baseline traits

According to the established criteria, 1,985 AP patients were included. The basic clinical traits are outlined in Table 1. The high-risk group mainly consisted of older people and Caucasians (P < 0.001). Additionally, high-risk populations had lower initial laboratory values for ALB, HGB, HCT, PLT, and Ca levels at admission, while AST, BUN, lactate, Cr, BG, INR, PT, PTT, TB, and WBC levels were higher. Furthermore, high-risk patients exhibited higher SOFA scores (P < 0.001), indicating

more severe illness, and were more likely to require norepinephrine support and MV ($P < 0.001$). No prominent differences were discerned in sex, heart rate, RR, ALT, mild or severe liver disease, kidney disease, malignant tumors, COPD, CHF, PVD, MI, HP, and statins ($P > 0.05$).

KM survival curve

Among 1,985 AP patients, 230 died within 30 days, 324 died within 90 days, 375 died within 180 days, and 451 died within 360 days. The KM curve showed remarkable differences in ACM risk between the two groups at 30 days, 90 days, 180 days, and 360 days (Fig 2). High-risk patients had greater ACM risk than low-risk patients at these time points (all log-rank $P < 0.001$).

Relationship between the braden scale and prognosis in AP patients

Cox regression models found that when the Braden score was included as a continuous variable, each 1-unit rise was markedly linked with reduced ACM risk. Specifically, for 30-day ACM risk, the HR and their 95% CI in the three models were as follows: 0.81 (0.772-0.851), 0.822 (0.781-0.866), and 0.923 (0.873-0.976) (all $p < 0.05$). Consistent results were discerned for 90-day, 180-day, and 360-day ACM risk. When the Braden score was treated as a dichotomous variable, the low-risk group was remarkably linked to reduced 30-day ACM risk compared to the high-risk group (Model 1: HR, 0.376 [95% CI 0.282-0.502] $P < 0.001$; Model 2: HR, 0.422 [95% CI 0.315-0.565] $P < 0.001$; Model 3: HR, 0.688 [95% CI 0.501-0.945] $P = 0.021$). However, after adjusting for all confounders, no marked link was found between Braden scores and ACM risk at 90 days, 180 days, and 360 days (Table 2). The results of the HRs and CIs for each confounding factor are detailed in Supplementary Fig 3.

RCS analysis (Fig 3) showed that Braden scores were significantly linearly related to ACM risk at 30 days (P for nonlinear = 0.155) and 90 days (P for nonlinear = 0.637).

However, no nonlinear association was observed at 180 and 360 days, but the Braden score showed a significant linear protective trend at both 180 and 360 days.

Prognostic value of braden scores for AP patients

ROC curves (Fig 4) (Table 3) indicated that the Braden score demonstrated significant predictive advantage, with the 30-day AUC [67.02% (95% CI: 63.44-70.61)] significantly superior to the 90-day [63.51% (95% CI: 60.22-66.81)], 180 days [61.43% (95% CI: 58.29-64.57)], and 360 days [60.13% (95% CI: 57.18-63.07)]. In addition, we obtained the optimal cutoff value of 15 for the Braden score, with the most significant sensitivity (61.04%) and specificity (64.71%) at 30 days. Additionally, the Braden score demonstrated good calibration in predicting the 30-day mortality risk, and predicted probabilities aligned well with observed probabilities, without significant systematic deviation (Fig 5). This further confirms the favorable predictive capability of the Braden score for ACM risk in AP patients, highlighting its important clinical utility. After adding Braden to Model 2 (which included conventional variables, such as age, sex, marital status, and ethnicity), the AUC increased, and this increase was statistically significant (Table 4). To assess the model's ability to reclassify risk, the NRI and IDI were calculated. The inclusion of the Braden score increased the NRI for Model 2 and improved the IDI (Table 4), suggesting that incorporating the Braden score may enhance the predictive model's accuracy and risk reclassification capability.

Subgroup analyses

We further explored the potential association between the Braden score and ACM risk at 30, 90, 180, and 360 days across different AP patient cohorts. After stratification by age, sex, marital status, race, and comorbidities, exploratory analysis suggested potential signals of an association between the Braden score and 30-day mortality risk in subgroups of patients aged <60 years, females, Caucasians, married individuals, and those with renal disease (none adjusted for multiple comparisons). Additionally,

the Braden score showed interaction effects with mild liver disease, severe liver disease, peripheral vascular disease, hypertension, norepinephrine use, and mechanical ventilation ($P < 0.05$), though these findings remained exploratory. For the long-term endpoints at 90, 180, and 360 days, only malignant tumors, norepinephrine, and mechanical ventilation demonstrated potential interaction signals ($P < 0.05$). Fig 6 illustrates these hypothesis-generating findings, which require further validation in independent cohorts.

Sensitivity analysis

To validate the robustness of the strategy for handling missing values, we conducted a sensitivity analysis: ① After excluding variables with a missing rate >20%, the analysis of complete cases without imputation (n=1585, 79.8%) showed that each 1-point increase in the Braden score was associated with 30-day mortality HR = 0.931 (95% CI 0.870–0.996, p = 0.037), consistent with the primary estimate (Supplementary Table 1). ② After excluding variables with a missing rate >10%, the remaining variables with a missing rate ≤10% (31 variables in total) were imputed using the same MissForest algorithm, followed by the primary Cox regression model. Results showed that the HR for 30-day mortality associated with the Braden score was 0.909 (95% CI 0.861–0.959, p < 0.001), highly consistent with the full text imputation results (Supplementary Table 2). This indicates that the primary conclusions are unaffected by imputation strategies or missing data proportions.

DISCUSSION

This is the first cohort study to delve into the link between Braden scores at admission and outcomes in AP. A retrospective analysis was conducted utilizing a large public medical database. The Braden score is an independent predictor of 30-day ACM risk in patients with AP, and this finding remains significant after adjusting for potential confounders. Our study unveiled that the Braden score was linearly correlated with ACM risk in AP patients. KM survival analysis confirmed that high-risk patients had

greater ACM risk at 30 days, 90 days, 180 days, and 360 days. In addition, the Braden score was a reliable predictor of ACM risk in AP, with higher AUC values at 30 days than at 90 days, 180 days, and 360 days. Subgroup analyses substantiated the robustness. Therefore, this study explored an early, simple, and efficient assessment tool for evaluating the ACM risk in AP patients.

There has been growing attention on the association between the Braden Scale and disease outcomes in the ICU, which has become a critical area of research. For example, Ting et al. reported that the Braden score was greatly associated with mortality risk in critically ill septic patients(27). Tang et al. stated that for critically ill patients with ischemic stroke, the Braden score demonstrated strong predictive performance for 30-day mortality risk, with an AUC of 0.71(23). Shang et al. demonstrated that a Braden score below 16 can effectively predict the delirium risk in critically ill surgical patients(41). Yang et al. further emphasized that Braden score was notably linked with ACM risk in critically ill individuals with non-traumatic subarachnoid hemorrhage(42). Consistently, our findings highlight the potential utility of Braden scores in assessing AP prognosis and further reveal the link between Braden scores and the prognosis of pancreatitis.

The Braden score is considered an effective index for evaluating patients' risk of pressure ulcers. Our study further expands its utility to assess ACM risk in AP patients. The significant value of the Braden score may stem from its comprehensive reflection regarding the patient's overall health status across six dimensions. A lower Braden score typically indicates greater risk and issues in these areas. Potential mechanisms may explicate the link. First, patients with wet exposure, sensory impairment, and reduced mobility are more likely to be bedridden for long periods and have difficulty moving, thereby increasing the risk of pressure ulcers and deep vein thrombosis. An international study involving 1,117 ICU wards confirmed a strong correlation between low Braden scores and pressure ulcer incidence, with mortality risk increasing as pressure ulcer severity worsened(43). Additionally,

Gaurav et al. demonstrated that AP patients had a high incidence of limb deep vein thrombosis(44), possibly due to prolonged bed rest and inflammatory cascades(45). The mechanism of venous thrombosis involves reduced venous return pressure, a hypercoagulable state of blood, and systemic inflammatory responses, leading to vascular endothelial damage(46). Thrombosis is closely associated with AP severity, and combining thrombosis and inflammatory biomarkers can predict short-term outcomes in AP patients(47). Additionally, nutritional status is a key dimension of the Braden score. AP patients are in a state of high catabolism, with significant consumption of proteins and glycogen, often accompanied by malnutrition and impaired immune function, thereby increasing susceptibility to infections and inflammatory responses and raising the risk of mortality(48). Furthermore, malnutrition can alter the composition of the intestinal epithelial barrier function and increase intestinal mucosal permeability, thus resulting in intestinal bacterial translocation, pancreatic tissue necrosis, infection, and multiple organ dysfunction syndrome (MODS)(49). In addition, prolonged bed rest, impaired motor function, malnutrition, and persistent inflammation in AP patients can lead to significant muscle wasting. Multiple studies have demonstrated that sarcopenia is a poor prognostic factor for AP, increasing mortality risk among ICU-admitted AP patients and serving as a pronounced predictor of mortality risk(50-52). As mentioned above, the Braden score provides a more comprehensive approach that combines functional and nutritional aspects to assess patient status from multiple angles, which makes it a valuable supplementary bedside tool for identifying mortality risk in AP patients, facilitating early clinical intervention and improving prognosis.

The pathophysiological mechanisms of AP are complex, involving autoactivation of pancreatic enzymes, oxidative stress, and immune dysregulation, which leads to the release of damage-associated molecular patterns (DAMPs). This initiates an inflammatory cascade, which ultimately evolves to systemic inflammatory response syndrome (SIRS) and MODS (53-55). Patients with low Braden scores often exhibit

reduced mobility and malnutrition, which may exacerbate oxidative stress and immune dysregulation, intensify pancreatic inflammatory responses, and trigger MODS, thereby increasing mortality risk. However, this study demonstrates only statistical associations. The causal pathways require validation through prospective cohort or experimental studies.

Our research found that the Braden score was a good predictor of 30-day mortality, but its predictive ability declined over longer periods (90, 180, and 360 days). This is because the Braden score reflects the instantaneous frailty status at admission, which is closely associated with early hospital complications (pressure ulcers, DVT, hospital-acquired infections). Hence, the 30-day mortality prediction is reliable. Once patients enter the chronic phase, long-term mortality is much more dependent on dynamic factors, such as pancreatic necrosis infection, recurrent exacerbations, new-onset diabetes/exocrine insufficiency, cardiovascular events, persistent inflammation, and progression of sarcopenia-frailty(56). Consequently, the predictive power of the Braden score diminishes over time. Clinically, combining the Braden score with indicators that can be retested 3–6 months post-discharge (SOFA trend, CRP/albumin ratio, HbA1c, residual necrosis on imaging, gait speed, or handgrip strength) and chronic disease burden (frailty index, readmission frequency) in a joint model may enhance long-term predictive accuracy.

Exploratory subgroup analyses suggested that the association between Braden scores and 30-day mortality was relatively stronger in patients <60 years old, females, and those with chronic kidney disease (Fig 6). These observations are purely hypothesis-generating and have not undergone multiple corrections. Hence, they require validation in external cohorts. One possible explanation is that the baseline organ reserve in the aforementioned population has not yet been depleted by advanced age or severe comorbidities, and nutritional-functional status may still be a significant contributor to short-term outcomes. Younger or female patients experience a more rapid decline in muscle mass and immune reserve, and the nutritional-activity deficits

reflected by low Braden scores may be more readily converted into early adverse events. Chronic kidney disease itself is frequently accompanied by protein consumption, anemia, and immunosuppression, which overlap significantly with the nutritional and friction-shear dimensions of the Braden scale, potentially increasing its sensitivity. Conversely, in critically ill patients with decompensated liver disease, we observed a diminished discriminatory effect of the Braden score. We hypothesize that when pancreatitis coexists with severe liver disease, pancreatic enzymes entering the liver via the portal vein may exacerbate hepatic injury and trigger systemic inflammatory responses(57, 58). Additionally, factors such as hypoalbuminemia, ascites, and hepatic encephalopathy may contribute to consistently low Braden scores in the nutrition/hydration subscale, thereby diminishing its additional discriminatory value. Similarly, in critically ill patients requiring mechanical ventilation or norepinephrine support, this association nearly disappeared. This suggests that once patients enter the stage of overt multiple organ failure, baseline frailty indicators may be overshadowed by the extreme severity of their condition. Organ failure itself, rather than skin-activity risk, then dominates short-term prognosis, thereby diminishing the Braden scale's discriminatory power(59, 60)a. Therefore, Braden score ≤15 has limited value as a standalone alert threshold in populations requiring intensive organ support. In clinical practice, it should be combined with dynamic indicators such as SOFA and lactate for comprehensive assessment. For mild-to-moderate AP or the aforementioned high-risk subgroups, the Braden score may serve as a simple, early risk stratification tool.

One of the main strengths of this study is that it first proposes that Braden score is an independent predictor of ACM risk in AP. The MIMIC-IV database offers extensive and diverse population data, which enables us to perform comprehensive adjustments, adjust potential confounders, and ensure the results' reliability. Early assessment using the Braden score can identify high-risk AP individuals who are likely to have poor outcomes, enabling timely intervention and improved prognoses. Compared to

other complex scores, Braden scores offer the advantages of simplicity, cost-effectiveness, and ease of calculation, and can be applied in diverse healthcare settings and regions with limited resources.

Although we provide valuable evidence for the prognostic value of Braden scores in AP, it is imperative to admit certain shortcomings. First, the single-center retrospective design restrains the inference of any causality. Though we performed multivariate adjustments and subgroup analyses, residual confounders may remain, which could undermine the prognostic outcomes. Therefore, it is necessary to conduct prospective multicenter studies. Second, given data limitations, we cannot perform subgroup analyses by AP etiologies, nor obtain relevant information on imaging examinations. Future studies need to include detailed etiological data. Third, our analysis focused on the initial Braden score at admission, and its dynamic changes over time were beyond the scope of our assessment. Further research is warranted to investigate the prognostic value of dynamic Braden scores to clarify its clinical utility. Fourth, this study was based on single-center ICU data from the MIMIC-IV database, and the results were only applicable to the AP population receiving intensive care. The generalizability to general wards or other healthcare settings requires further validation. Fifth, although the number of patients with missing Braden scores is negligible (n = 9, 0.45%), we cannot entirely rule out the possibility that this minimal exclusion may introduce selection bias if the missing values correlate with unmeasured severity or frailty indicators. Sixth, 230 patients died within 30 days in this study. Ultimately, 33 covariates were included in Model 3, with an EPV of about 6.9, slightly below the conventional threshold of ≥10. Although VIF-based exclusion methods were employed, potential overfitting risks remain, necessitating validation in an independent cohort.

CONCLUSION

Our study expands the application value of the Braden score in predicting outcomes for AP patients, suggesting that the Braden score may serve as a simple, early

supplementary indicator for risk stratification and identification of patients with higher mortality risk. This study is a single-center retrospective analysis, and the findings are only at the hypothesis-generating stage. Future prospective multicenter cohorts are required to confirm the clinical value of the Braden score as a simple bedside supplementary tool.

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TABLES AND FIGURES WITH LEGENDS

Table 1. Baseline characteristics of the study population

Variable	Overall (*n*=1985)	High-risk groupa (*n*=1024)	Low-risk group (*n*=961)	*p*
Personal characteristics				
Age_group (%)				<0.001*
<60	1004 (50.6)	477 (46.6)	527 (54.8)	
>=60	981 (49.4)	547 (53.4)	434 (45.2)	
Gender (%)				0.76
Male	1126 (56.7)	577 (56.3)	549 (57.1)	
Female	859 (43.3)	447 (43.7)	412 (42.9)	
Marital (%)				0.003*
Single	632 (31.8)	326 (31.8)	306 (31.8)	
Divorced/Widowed	333 (16.8)	162 (15.8)	171 (17.8)	
Married	851 (42.9)	426 (41.6)	425 (44.2)	
Unknow	169 (8.51)	110 (10.7)	59 (6.14)	
Race (%)				<0.001*
White	1258 (63.4)	641 (62.6)	617 (64.2)	
No White	509 (25.6)	237 (23.1)	272 (28.3)	
Unknow	218 (11.0)	146 (14.3)	72 (7.49)	
Vital signs and laboratory tests				
Heart rate (beats/min)	94.0 [80.0;110]	94.0 [80.0;111]	93.0 [81.0;109]	0.236
Respiration rate	19.0 [16.0;24.0]	19.5 [16.0;24.0]	19.0 [16.0;23.0]	0.393

(beats/min)				
Albumin (g/dL)	3.10 [2.60;3.60]	2.90 [2.50;3.42]	3.30 [2.80;3.80]	<0.001*
ALT (IU/L)	38.0 [19.0;94.0]	38.0 [19.0;102]	38.0 [20.0;88.0]	0.431
AST (IU/L)	52.0 [27.0;132]	58.0 [29.0;150]	48.0 [25.0;115]	<0.001*
BUN (mg/dL)	18.0 [12.0;33.0]	21.0 [13.0;37.0]	17.0 [11.0;29.0]	<0.001*
Calcium (mg/dL)	8.10 [7.60;8.70]	8.00 [7.40;8.60]	8.20 [7.70;8.80]	<0.001*
Creatinine (mg/dL)	1.00 [0.70;1.70]	1.10 [0.70;1.80]	0.90 [0.70;1.50]	<0.001*
Glucose (mg/dL)	126 [102;167]	129 [103;174]	123 [99.0;158]	0.002*
HGB (g/dL)	10.7 [9.10;12.3]	10.5 [9.00;12.1]	11.0 [9.30;12.5]	<0.001*
HCT (%)	32.4 [27.7;37.1]	32.0 [27.4;36.8]	33.0 [28.2;37.7]	0.003*
INR	1.30 [1.10;1.60]	1.30 [1.20;1.60]	1.20 [1.10;1.50]	<0.001
Lac (mmol/L)	1.80 [1.30;2.70]	1.90 [1.30;2.90]	1.70 [1.20;2.50]	<0.001
PLT (K/uL)	185 [126;260]	179 [121;256]	191 [137;262]	0.017
PT (sec)	14.3 [12.6;17.1]	14.7 [13.0;17.6]	13.7 [12.3;16.4]	<0.001
PTT (sec)	30.6 [27.2;36.8]	31.3 [27.6;38.4]	29.8 [26.9;34.9]	<0.001
Total bili (mg/dL)	0.80 [0.40;2.20]	0.90 [0.50;2.42]	0.80 [0.40;2.00]	0.001
WBC (K/uL)	11.0 [7.40;16.3]	11.9 [8.10;17.4]	10.2 [6.90;15.3]	<0.001
Comorbidities				
Mild liver disease (%)				0.374
No	1235 (62.2)	627 (61.2)	608 (63.3)	
Yes	750 (37.8)	397 (38.8)	353 (36.7)	
Renal disease (%)				0.72
No	1367 (68.9)	701 (68.5)	666 (69.3)	

Yes	618 (31.1)	323 (31.5)	295 (30.7)	
Severe liver disease (%)				0.111
No	1653 (83.3)	839 (81.9)	814 (84.7)	
Yes	332 (16.7)	185 (18.1)	147 (15.3)	
Malignant cancer (%)				0.531
No	1609 (81.1)	836 (81.6)	773 (80.4)	
Yes	376 (18.9)	188 (18.4)	188 (19.6)	
Chronic pulmonary disease (%)				0.682
No	1373 (69.2)	713 (69.6)	660 (68.7)	
Yes	612 (30.8)	311 (30.4)	301 (31.3)	
Congestive heart failure (%)				0.797
No	1378 (69.4)	714 (69.7)	664 (69.1)	
Yes	607 (30.6)	310 (30.3)	297 (30.9)	
Peripheral vascular disease (%)				0.97
No	1650 (83.1)	852 (83.2)	798 (83.0)	
Yes	335 (16.9)	172 (16.8)	163 (17.0)	
Myocardial infarct (%)				0.791
No	1624 (81.8)	835 (81.5)	789 (82.1)	
Yes	361 (18.2)	189 (18.5)	172 (17.9)	
Hypertension (%)				0.469
No	794 (40.0)	418 (40.8)	376 (39.1)	

Yes	1191 (60.0)	606 (59.2)	585 (60.9)	
Norepinephrine (%)				<0.001*
No	1323 (66.6)	608 (59.4)	715 (74.4)	
Yes	662 (33.4)	416 (40.6)	246 (25.6)	
Statins (%)				0.719
No	945 (47.6)	492 (48.0)	453 (47.1)	
Yes	1040 (52.4)	532 (52.0)	508 (52.9)	
Mechanical ventilation (%)				<0.001*
No	1054 (53.1)	397 (38.8)	657 (68.4)	
Yes	931 (46.9)	627 (61.2)	304 (31.6)	
Scores				
GCS	15.0 [15.0;15.0]	15.0 [14.0;15.0]	15.0 [15.0;15.0]	<0.001*
SOFA	5.00 [2.00;8.00]	6.00 [3.00;9.00]	4.00 [2.00;6.00]	<0.001*

[a]In our study, the low-risk group was defined as a Braden score>15 and the high-risk group was defined as a Braden score≤15. *Significant difference between two groups ($p<0.05$). Abbreviations: GCS: Glasgow Coma Scale; SOFA: Sequential organ failure assessment; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; HGB: Hemoglobin; HCT: Hematocrit; PLT: Platelet count; PT: Prothrombin time; PTT: Partial thromboplastin time; WBC: White blood cell count.

Table 2. Univariate and multivariate Cox regression models of Braden score with mortality in patients with acute pancreatitis

Outcome	Model 1			Model 2			Model 3		
	HR	**95% CI**	***p***	**HR**	**95% CI**	***p***	**HR**	**95% CI**	***p***
30-day mortality									
Continuous	0.810	0.772-0.851	<0.001	0.822	0.781-0.866	<0.001	0.923	0.873-0.976	0.005
Category									
High-risk （Braden score≤15）	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Low-risk （Braden score＞15）	0.376	0.282-0.502	<0.001	0.422	0.315-0.565	<0.001	0.688	0.501-0.945	0.021
90-day mortality									
Continuous	0.848	0.814-0.883	<0.001	0.858	0.823-0.895	<0.001	0.943	0.901-0.988	0.013
Category									
High-risk （Braden score≤15）	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Low-risk （Braden score＞15）	0.482	0.382-0.607	<0.001	0.530	0.419-0.670	<0.001	0.791	0.614-1.019	0.07
180-day mortality									
Continuous	0.869	0.837-0.902	<0.001	0.881	0.847-0.916	<0.001	0.957	0.918-0.999	0.045
Category									

High-risk （Braden score≤15）	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Low-risk （Braden score＞15）	0.549	0.445-0.678	<0.001	0.607	0.491-0.751	<0.001	0.875	0.694-1.103	0.3
360-day mortality									
Continuous	0.884	0.855-0.915	<0.001	0.893	0.862-0.925	<0.001	0.958	0.922-0.996	0.03
Category									
High-risk （Braden score≤15）	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Low-risk （Braden score＞15）	0.607	0.502-0.734	<0.001	0.658	0.543-0.797	<0.001	0.905	0.734-1.114	0.3

Model 1. unadjusted; Model 2. adjusted for admission age group, gender, marital, race; Model 3. adjusted for admission age group, gender, marital, race, heart rate, RR, albumin, aspartate transaminase, urea nitrogen, calcium, creatinine, glucose, hematocrit, lactate, platelet, PT, PTT, total bilirubin, WBC, MLD, renal disease, severe liver disease, malignant cancer, COPD, congestive heart failure, peripheral vascular disease, myocardial infarct, HP, norepinephrine, statins, MV, GCS. Abbreviations: HR: Hazard ratio; CI: Confidence interval; RR: Respiratory rate; PT: Prothrombin time; PTT: Partial thromboplastin time; WBC: White blood cell; MLD: Mean lung density; COPD: Chronic obstructive pulmonary disease; HP: Hypertension; MV: Mechanical ventilation; GCS: Glasgow Coma Scale.

Table 3. Information of ROC curves

Variables	AUC (%)	95% CI	Threshold	Specificity	Sensitivity
Status 30d	67.02	63.44-70.61	15	0.6471	0.6104
Status 90d	63.51	60.22-66.81	15	0.6504	0.5521
Status 180d	61.43	58.29-64.57	15	0.6501	0.5239
Status 360d	60.13	57.18-63.07	15	0.6512	0.4989

Abbreviations: ROC: Receiver operating characteristic; AUC: Area under the curve; CI: Confidence interval.

Table 4. The performance indicators of multivariate models (including the Braden model and the model without the Braden component) in predicting the all-cause mortality risk of AP patients

	AUC		Net reclassification improvement		Integrated discrimination improvement	
	Index (95% CI)	*p* value for Δ AUC	Index (95% CI)	*p* value	Index (95% CI)	*p* value
30d mortality with Braden	0.712（0.675-0.749）	*p* <0.001	0.235（0.161-0.291）	*p* <0.001	0.040（0.023-0.064）	*p* <0.001
30d mortality without Braden	0.647（0.609-0.685）					
90d mortality with Braden	0.687（0.654-0.719）	*p* <0.001	0.193（0.128-0.248）	*p* <0.001	0.034（0.018-0.054）	*p* <0.001
90d mortality without Braden	0.643（0.611-0.676）					
180d mortality with Braden	0.683（0.653-0.714）	*p* <0.001	0.160（0.100-0.209）	*p* <0.001	0.027（0.013-0.045）	*p* <0.001
180d mortality without Braden	0.651（0.620-0.681）					
360d mortality with Braden	0.666（0.637-0.695）	*p* <0.001	0.134（0.081-0.186）	*p* <0.001	0.025（0.012-	*p* <0.001

					0.041）	

Abbreviations: AUC: Area under the receiver operating characteristic curve; CI: Confidence interval.

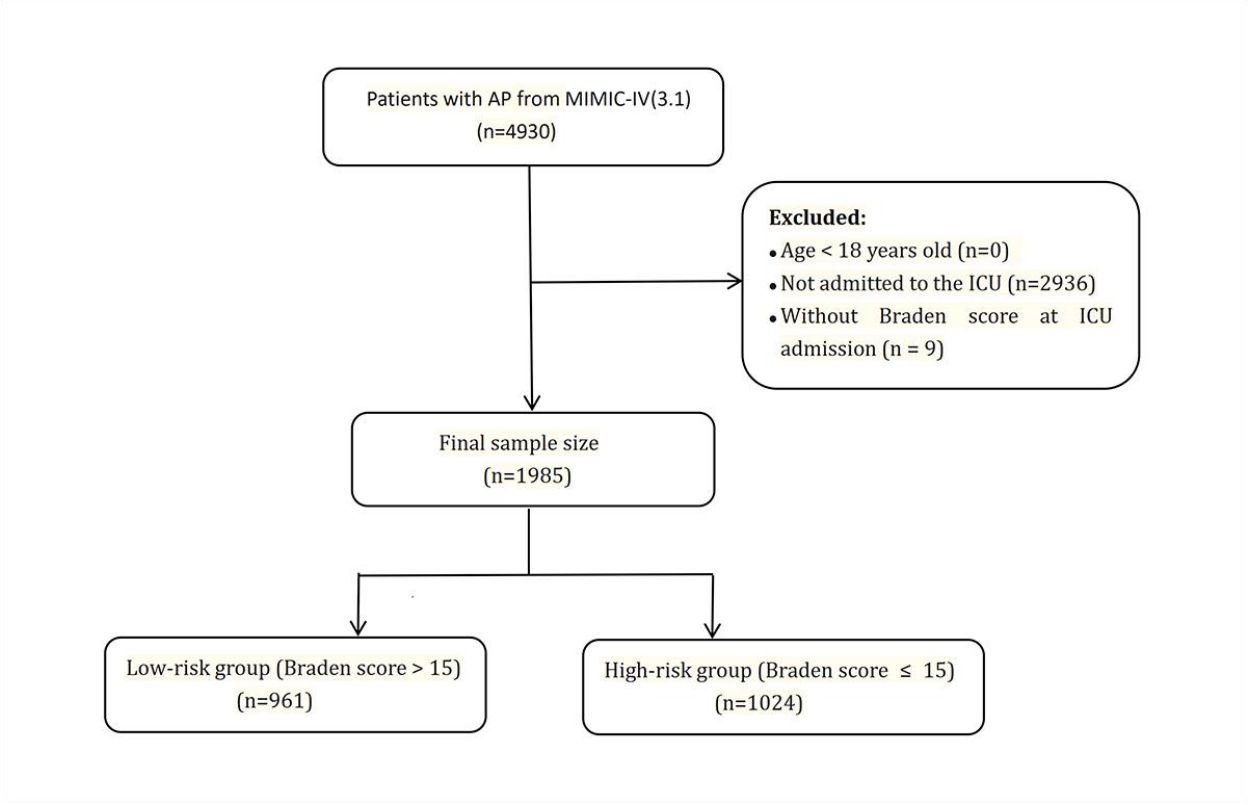

Figure 1. Flowchart for participants selection

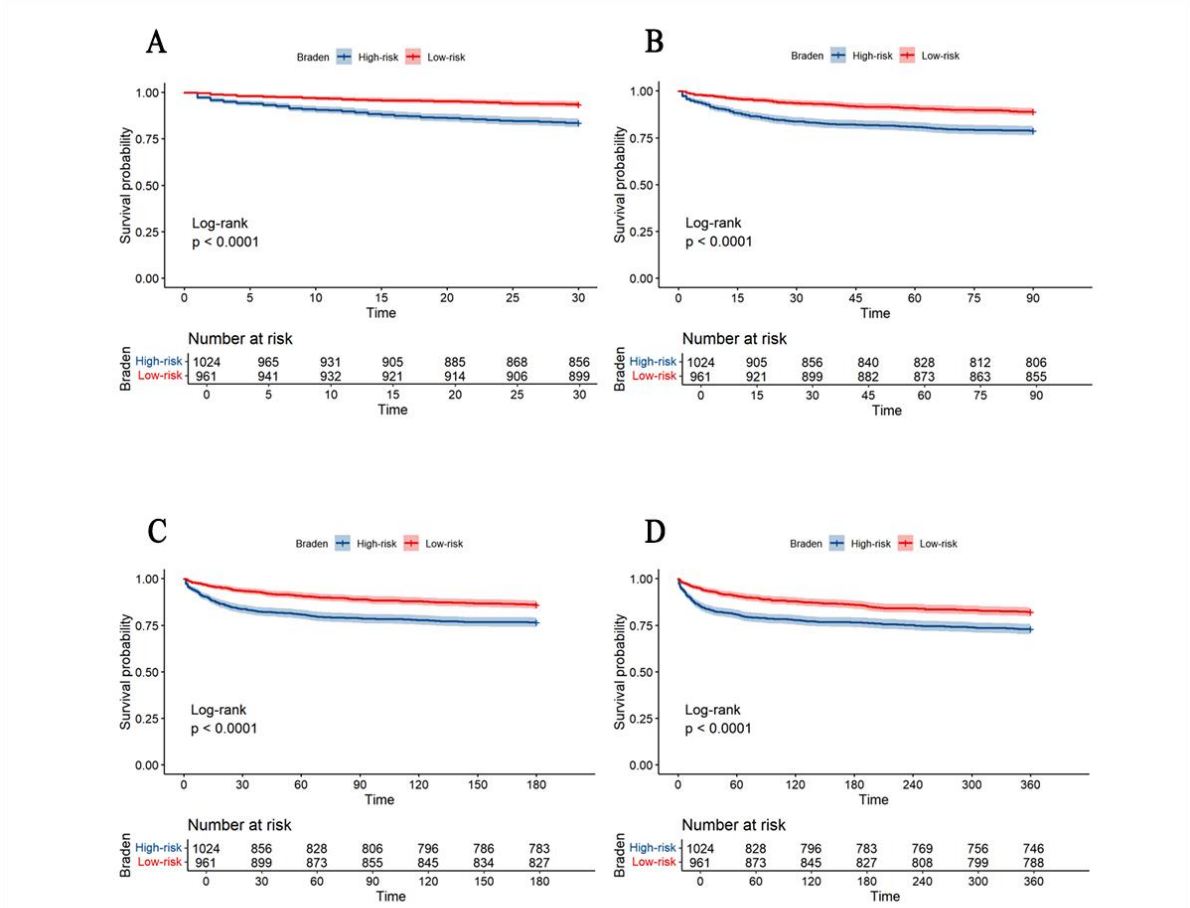

Figure 2. Kaplan–Meier survival curves for ACM by Braden risk category in AP. In the overall cohort (*n* = 1,985), 230, 324, 375, and 451 deaths occurred within 30, 90, 180, and 360 days, respectively. At each time point, high-risk patients (Braden score ≤15) had higher ACM than low-risk patients (Braden score >15); all log-rank P < 0.001. (A) 30-day, (B) 90-day, (C) 180-day, and (D) 360-day mortality. Numbers at risk are shown beneath each plot. Abbreviations: AP: Acute pancreatitis; ACM: All-cause mortality.

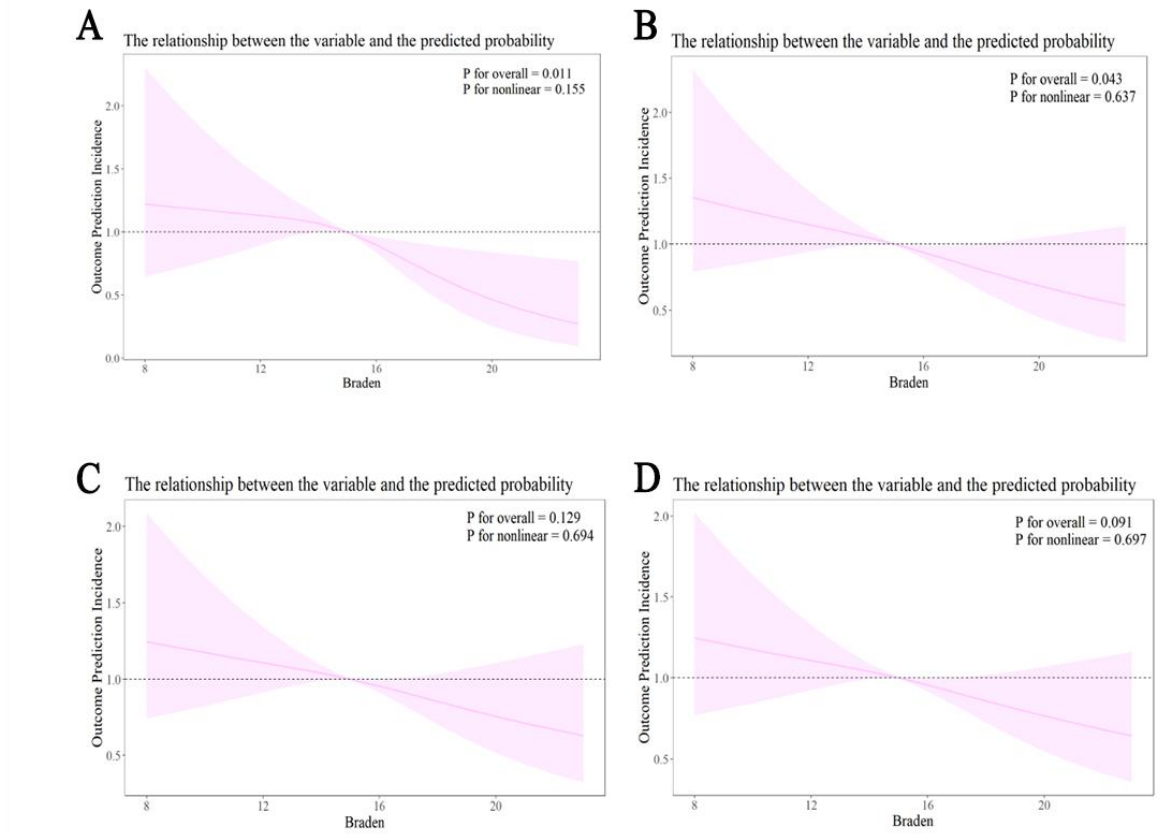

Figure 3. RCS models showing the dose–response relationship between admission Braden score and all-cause mortality risk in acute pancreatitis patients at (A) 30-day, (B) 90-day, (C) 180-day, and (D) 360-day follow-up. The black dashed line represents the HR, with the shaded area indicating the 95% confidence interval. Abbreviations: RCS: Restricted cubic spline; HR: Hazard ratio.

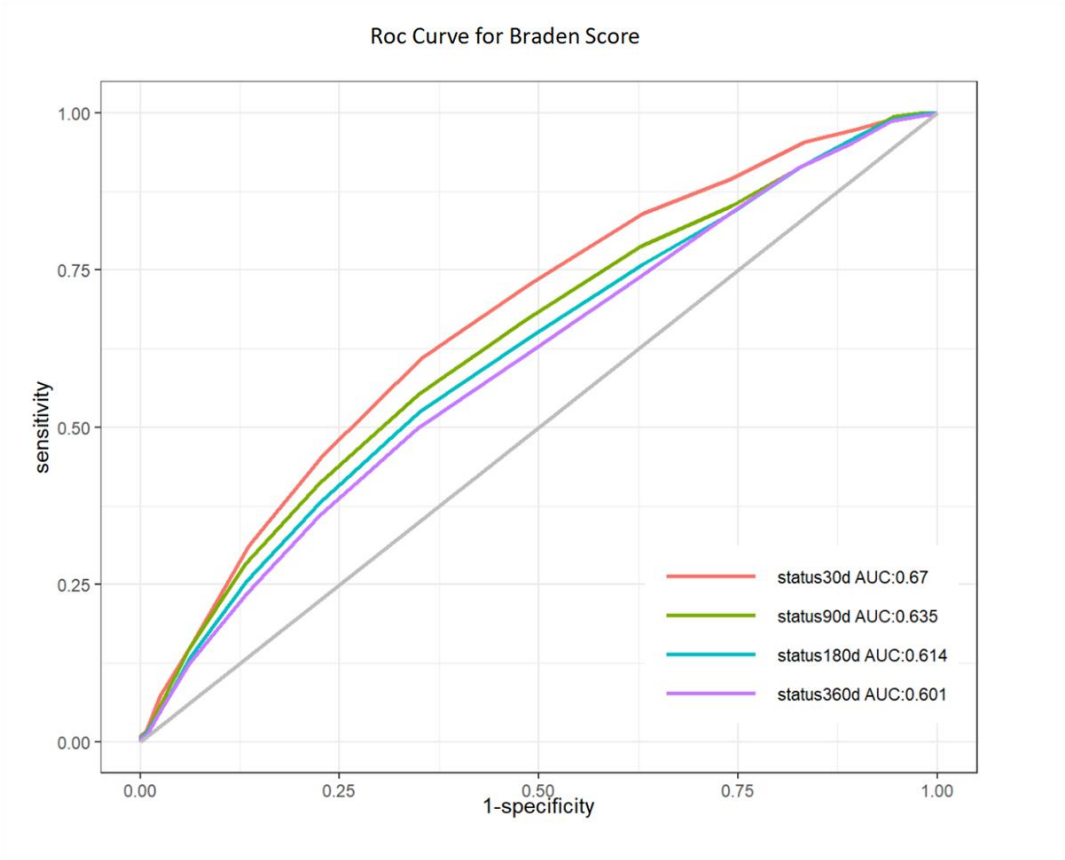

Figure 4. ROC curve for Braden Scale's predictive accuracy. ROC curve demonstrating the Braden Scale's efficacy in predicting 30-day mortality, 90-day mortality, 180-day mortality, 1-year mortality, with the calculated AUC. Abbreviations: ROC: Receiver operating characteristic; AUC: Area under the curve.

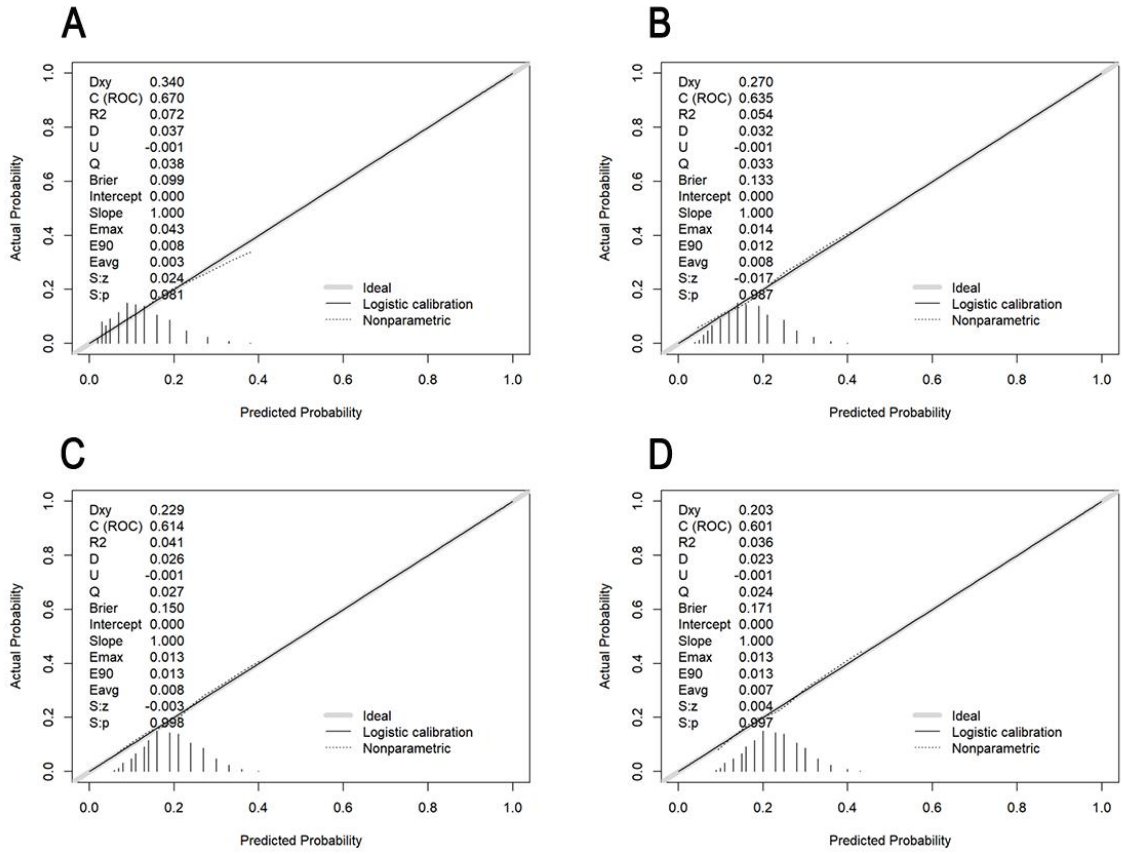

Figure 5. Calibration of the Braden score for all-cause mortality in AP at (A) 30, (B) 90, (C) 180, and (D) 360 days. Predicted probabilities closely matched observed probabilities, exhibiting no significant systematic deviation. Abbreviations: ROC: Receiver operating characteristic; AP: Acute pancreatitis.

A

Variables	N	NA	HR(95%CI)	p for interaction
Age(years)				0.936
<60	1004		0.907(0.824-0.998)	
>60	981		0.931(0.863-1.004)	
Gender				0.795
Male	1126		0.927(0.853-1.006)	
Female	859		0.917(0.843-0.996)	
Marital				0.209
Single	632		0.98(0.872-1.101)	
Divorced/Widowed	333		0.924(0.774-1.101)	
Married	851		0.881(0.804-0.964)	
Unknow	169		0.874(0.689-1.109)	
Race				0.522
White	1258		0.924(0.861-0.991)	
No White	509		0.908(0.797-1.035)	
Unknow	218		0.89(0.729-1.085)	
Mild liver disease				0.039
No	1235		0.9(0.827-0.979)	
Yes	750		0.94(0.866-1.02)	
Renal disease				0.184
No	1367		0.938(0.878-1.002)	
Yes	618		0.821(0.727-0.927)	
Severe liver disease				0.004
No	1653		0.91(0.85-0.975)	
Yes	332		0.958(0.863-1.063)	
Malignant cancer				0.429
No	1609		0.905(0.844-0.97)	
Yes	376		0.975(0.869-1.093)	
Chronic pulmonary disease				0.465
No	1373		0.92(0.863-0.982)	
Yes	612		0.96(0.844-1.093)	
Congestive heart failure				0.846
No	1378		0.897(0.838-0.96)	
Yes	607		0.948(0.848-1.061)	
Peripheral vascular disease				0.034
No	1650		0.901(0.847-0.958)	
Yes	335		1.087(0.915-1.291)	
Myocardial infarct				0.172
No	1624		0.893(0.837-0.953)	
Yes	361		0.994(0.862-1.147)	
Hypertension				0.041
No	794		0.858(0.789-0.933)	
Yes	1191		0.988(0.908-1.075)	
Norepinephrine				0.001
No	1323		0.765(0.688-0.852)	
Yes	662		0.966(0.899-1.038)	
Statins				0.416
No	945		0.913(0.848-0.984)	
Yes	1040		0.913(0.828-1.007)	
Mechanical ventilation				0.001
No	1054		0.827(0.751-0.912)	
Yes	931		0.982(0.912-1.058)	

B

Variables	N	NA	HR(95%CI)	p for interaction
Age(years)				0.953
<60	1004		0.926(0.857-1)	
>60	981		0.948(0.891-1.008)	
Gender				0.201
Male	1126		0.935(0.874-1)	
Female	859		0.951(0.889-1.017)	
Marital				0.077
Single	632		0.997(0.909-1.093)	
Divorced/Widowed	333		0.929(0.809-1.065)	
Married	851		0.939(0.872-1.011)	
Unknow	169		0.818(0.695-0.963)	
Race				0.99
White	1258		0.952(0.899-1.009)	
No White	509		0.949(0.856-1.052)	
Unknow	218		0.862(0.725-1.027)	
Mild liver disease				0.831
No	1235		0.955(0.894-1.019)	
Yes	750		0.922(0.859-0.99)	
Renal disease				0.406
No	1367		0.958(0.908-1.011)	
Yes	618		0.875(0.795-0.963)	
Severe liver disease				0.268
No	1653		0.952(0.901-1.006)	
Yes	332		0.917(0.833-1.01)	
Malignant cancer				0.005
No	1609		0.904(0.853-0.958)	
Yes	376		1.053(0.963-1.151)	
Chronic pulmonary disease				0.949
No	1373		0.934(0.886-0.984)	
Yes	612		0.987(0.89-1.096)	
Congestive heart failure				0.629
No	1378		0.926(0.877-0.978)	
Yes	607		0.971(0.883-1.068)	
Peripheral vascular disease				0.067
No	1650		0.929(0.884-0.977)	
Yes	335		1.046(0.911-1.201)	
Myocardial infarct				0.158
No	1624		0.922(0.875-0.973)	
Yes	361		0.992(0.882-1.115)	
Hypertension				0.017
No	794		0.888(0.827-0.952)	
Yes	1191		1.001(0.937-1.07)	
Norepinephrine				0.007
No	1323		0.841(0.779-0.908)	
Yes	662		0.99(0.931-1.052)	
Statins				0.082
No	945		0.933(0.876-0.994)	
Yes	1040		0.955(0.887-1.028)	
Mechanical ventilation				0.002
No	1054		0.865(0.805-0.93)	
Yes	931		1.008(0.947-1.073)	

C

Variables	N	NA	HR(95%CI)	p for interaction
Age(years)				0.9
<60	1004		0.952(0.885-1.024)	
>60	981		0.952(0.9-1.008)	
Gender				0.541
Male	1126		0.959(0.902-1.02)	
Female	859		0.955(0.896-1.018)	
Marital				0.068
Single	632		0.997(0.916-1.084)	
Divorced/Widowed	333		0.914(0.802-1.042)	
Married	851		0.969(0.906-1.036)	
Unknow	169		0.838(0.721-0.975)	
Race				0.855
White	1258		0.969(0.919-1.023)	
No White	509		0.961(0.873-1.059)	
Unknow	218		0.848(0.717-1.002)	
Mild liver disease				0.82
No	1235		0.976(0.921-1.036)	
Yes	750		0.927(0.866-0.992)	
Renal disease				0.751
No	1367		0.966(0.919-1.016)	
Yes	618		0.912(0.835-0.996)	
Severe liver disease				0.307
No	1653		0.965(0.918-1.015)	
Yes	332		0.932(0.852-1.02)	
Malignant cancer				0.001
No	1609		0.912(0.863-0.963)	
Yes	376		1.079(0.994-1.171)	
Chronic pulmonary disease				0.591
No	1373		0.946(0.901-0.994)	
Yes	612		1.013(0.922-1.113)	
Congestive heart failure				0.474
No	1378		0.945(0.899-0.994)	
Yes	607		0.972(0.892-1.058)	
Peripheral vascular disease				0.019
No	1650		0.941(0.898-0.986)	
Yes	335		1.026(0.905-1.164)	
Myocardial infarct				0.156
No	1624		0.936(0.892-0.983)	
Yes	361		1.006(0.907-1.115)	
Hypertension				0.043
No	794		0.908(0.852-0.969)	
Yes	1191		1.007(0.947-1.071)	
Norepinephrine				0.025
No	1323		0.873(0.815-0.935)	
Yes	662		0.998(0.942-1.058)	
Statins				0.011
No	945		0.943(0.888-1.001)	
Yes	1040		0.984(0.921-1.051)	
Mechanical ventilation				0.001
No	1054		0.881(0.825-0.941)	
Yes	931		1.023(0.964-1.085)	

D

Variables	N	NA	HR(95%CI)	p for interaction
Age(years)				0.437
<60	1004		0.958(0.899-1.021)	
>60	981		0.946(0.898-0.996)	
Gender				0.445
Male	1126		0.947(0.896-1)	
Female	859		0.975(0.919-1.034)	
Marital				0.008
Single	632		1.015(0.943-1.093)	
Divorced/Widowed	333		0.93(0.832-1.039)	
Married	851		0.947(0.89-1.007)	
Unknow	169		0.844(0.735-0.97)	
Race				0.851
White	1258		0.961(0.915-1.01)	
No White	509		0.966(0.89-1.048)	
Unknow	218		0.886(0.759-1.033)	
Mild liver disease				0.865
No	1235		0.965(0.915-1.017)	
Yes	750		0.942(0.887-1)	
Renal disease				0.637
No	1367		0.963(0.919-1.008)	
Yes	618		0.943(0.874-1.017)	
Severe liver disease				0.766
No	1653		0.968(0.925-1.013)	
Yes	332		0.934(0.859-1.015)	
Malignant cancer				0.003
No	1609		0.923(0.879-0.97)	
Yes	376		1.047(0.974-1.126)	
Chronic pulmonary disease				0.211
No	1373		0.941(0.899-0.985)	
Yes	612		1.008(0.931-1.091)	
Congestive heart failure				0.123
No	1378		0.942(0.899-0.987)	
Yes	607		0.989(0.919-1.064)	
Peripheral vascular disease				0.023
No	1650		0.941(0.902-0.981)	
Yes	335		1.026(0.919-1.145)	
Myocardial infarct				0.143
No	1624		0.942(0.901-0.985)	
Yes	361		1.019(0.928-1.119)	
Hypertension				0.177
No	794		0.938(0.885-0.994)	
Yes	1191		0.981(0.928-1.037)	
Norepinephrine				0.046
No	1323		0.892(0.84-0.946)	
Yes	662		1(0.947-1.056)	
Statins				0.002
No	945		0.947(0.896-1)	
Yes	1040		0.99(0.935-1.049)	
Mechanical ventilation				0.003
No	1054		0.892(0.842-0.945)	
Yes	931		1.012(0.958-1.069)	

Figure 6. Forest plots showing subgroup analyses of the association between admission Braden score (low-risk: >15 vs. high-risk: ≤15) and all-cause mortality in acute pancreatitis patients at (A) 30-day, (B) 90-day, (C) 180-day, and (D) 360-day follow-up. Hazard ratios (HRs) and 95% confidence intervals (CIs) were derived from Cox proportional hazards models adjusted for relevant covariates. Subgroups were stratified by demographic and clinical characteristics. Interaction p-values between Braden score and each variable are shown. All subgroup comparisons are exploratory and were not adjusted for multiple testing.

SUPPLEMENTAL DATA

Supplemental data are available at the following link:
https://www.bjbms.org/ojs/index.php/bjbms/article/view/13115/4024