BMJ Open Relationship between Braden Scale scores and acute kidney injury among patients with acute coronary syndrome: a multicentre retrospective cohort study

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ABSTRACT

Objectives To evaluate the impact of pressure ulcer events assessed by the Braden Scale (BS) on acute kidney injury (AKI) in patients with acute coronary syndrome (ACS).

Design A multicentre retrospective cohort study.

Setting Chest pain centres from seven tertiary hospitals in China.

Participants We analysed 3185 patients with ACS from the Retrospective Evaluation of Acute Chest Pain study. The patients were divided into three groups (B1, B2 and B3) according to their BS scores (≤ 12 vs $13-14$ vs ≥ 15 , respectively) at admission.

Outcome measures AKI was defined according to the criteria of the 2012 Kidney Disease: Improving Global Outcomes. Multivariate logistic analysis was used to evaluate the relationship between the BS score and AKI.

Results There were 461 patients (14.5%) with ACS who had the complication of AKI. Patients with a lower score on the BS had a higher incidence of AKI ($p < 0.001$). Multivariate logistic regression analysis showed that adjusted ORs of the BS score for AKI were 2.242 (B1 vs B3: 95% CI: 1.643 to 3.060, $p < 0.001$) and 1.566 (B2 vs B3: 95% CI: 1.186 to 2.069, $p = 0.002$). The receiver operating characteristic curve analysis showed that the area under the curve of the BS score was 0.719 (95% CI: 0.702 to 0.736; $p < 0.001$) for AKI.

Conclusions The BS score was independently associated with AKI. It may be a useful tool to identify those who may benefit from further prediction and prevention of AKI in patients with ACS.

Trail registration number ChiCTR1900024657 (<http://www.chictr.org.cn/>). The satge reates to results.

INTRODUCTION

Acute coronary syndrome (ACS), the acute manifestation of ischaemic heart disease, remains a major cause of morbidity and mortality worldwide¹ and is responsible for more than 1 million hospital admissions in the USA annually.² Although the mortality of patients with ACS has declined in recent years, the in-hospital mortality rate remains at 2.5%–4.2%.^{3–5} The identification of high-risk

STRENGTHS AND LIMITATIONS OF THIS STUDY

- ⇒ This is the first study to investigate the relationship between Braden Scale (BS) scores and acute kidney injury (AKI) in patients with acute coronary syndrome (ACS).
- ⇒ As a routine nursing evaluation programme, the BS tool should be considered by health professionals to stratify the risk of AKI for patients with ACS.
- ⇒ The causality between BS scores and AKI needs to be questioned because some patients already developed AKI before admission.
- ⇒ The creatinine level at first admission was set as the baseline creatinine level, which may reduce the detection rate of AKI.

patients with ACS is important and facilitates therapeutic decision-making.⁶

Acute kidney injury (AKI) is a common complication of ACS that may be due to haemodynamic impairment and the use of contrast agents.⁷ According to a previous study, the incidence of AKI in patients with ACS ranges from 6.3% to 36.6%,⁸ and AKI has a detrimental effect on the prognosis of patients, including prolonged length of hospital stay, higher incidence of cardiovascular events and twofold–threefold mortality.^{9–11} In view of the high incidence of AKI and its negative influence on the prognosis of patients with ACS, it is crucial to identify high-risk patients who may develop AKI so that medical staff can predict and prevent the occurrence of AKI in patients with ACS.

Frailty is a clinical syndrome, which results in the decline of body recovery, reserve capacity and resistance to stress.¹² The overall frailty status of patients reportedly correlated with renal functional reserve^{13–15} and was significantly associated with a high incidence of AKI.¹⁶ Therefore, early assessment and screening of frailty are critical to the assessment of AKI.

The Braden Scale (BS) is widely used in routine nursing evaluation programmes to predict pressure ulcer events in patients.¹⁷ Because the BS examines several factors that could contribute to assessing frailty, such as nutrition, cognition, activity and function, it was recommended as a frailty identification tool.¹⁸ In our previous study, the low BS score was reportedly associated with death in patients with acute myocardial infection¹⁹; however, the relationship between the BS scores and AKI remains unclear. Clarifying the relationship may help nurses in daily nursing services identify patients with a high risk of AKI, avoid the use of nephrotoxic drugs, limit the dosage of contrast medium and make rational allocations of nursing management for high-risk patients with AKI.^{20 21} Therefore, this study aimed to evaluate the relationship between the BS score and AKI in patients with ACS undergoing primary percutaneous coronary intervention (PCI).

MATERIALS AND METHODS

Study design

In this multicentre retrospective cohort study, data from the Retrospective Evaluation of Acute Chest Pain (REACP) study were analysed to evaluate whether the BS could predict AKI in patients with ACS undergoing primary PCI. The REACP study enrolled patients with acute chest pain at chest pain centres from seven tertiary hospitals in China and registered them at www.chictr.org.cn (identifier: ChiCTR1900024657).

Study population

From January 2017 to February 2019, 14 460 patients visited the acute chest pain centre, and 3337 adult patients were diagnosed with ACS and underwent primary PCI. Of these patients, 122 patients did not have relevant data and failed to return for follow-up, 150 patients were treated with thrombolysis and 218 patients left the hospital within 24 hours. Finally, 2847 patients were selected and divided into B1, B2 and B3

groups, according to the sum of their BS scores: ≤ 12 , 13–14 and ≥ 15 on admission, respectively (figure 1).

Data collection and definition

We obtained data from the database of the REACP study. Data on vital signs, medical history, coronary angiography, medications, laboratory examination and imaging findings were collected by the physicians. Bedside echocardiography was performed by a professional technician within 24 hours after admission, and the left ventricular ejection fraction (LVEF) was calculated using the biplane Simpson method (Philips E33 Medical Systems, Bothell, Washington, USA). The Global Registry of Acute Coronary Events (GRACE) score and Gensini score were calculated according to previous studies.^{22–25} The Mehran risk score includes hypotension, use of intra-aortic balloon pump, congestive heart failure, age, anaemia, diabetes mellitus, contrast media volume and estimated glomerular filtration rate (eGFR).²⁶ All data were reviewed by two trained clinical research coordinators.

At the emergency room, the protocol for the nurse is to use the BS on each patient at admission with re-evaluation every 24 hours. Six subscales constitute the BS, which includes sensation, communication, water, activity, nutrition and friction, and shear force. Each subscale has 1–4 points, excluding friction and shear force that have 1–3 points. Based on this scoring system, the total points can range from 6 to 23.¹⁷ According to the scientific interpretation of the BS in 2005, to identify the patient's risk of developing pressure ulcers, the scores were as follows: 15–23, mild or no risk; 13–14, moderate risk; and ≤ 12 , high risk.²⁷

Staging of AKI was based on the criterion of Kidney Disease: Improving Global Outcomes.²⁸ AKI stage 1 criteria are serum creatinine level >26.5 mmol/L (0.3 mg/dL) within 48 hours, an increase in serum creatinine to 1.5-fold–1.9-fold of the baseline value, or urine output <0.5 mL/kg/hour for 6–12 hours. The criteria for AKI stage 2 are an increase in serum creatinine to 2.0-fold–2.9-fold of the baseline value or urine output <0.5 mL/kg/hour for 12 hours. AKI stage 3 criteria are serum creatinine level >353.6 mmol/L (4.0 mg/dL), an increase in serum creatinine more than 3.0-fold of the baseline value, urine output <0.3 mL/kg/hour for 24 hours or anuria for 12 hours. The first serum creatinine value measured on admission is considered the baseline serum creatinine.

Endpoint and follow-up

The study population underwent a median of 11.9 months (5.0–20.4 months) of follow-up. Trained physicians interviewed patients using structured telephone questionnaires. The primary endpoint was AKI.

Statistical analysis

The categorical variables were presented as numbers (percentages) and compared by the χ^2 test. The

Figure 1 Study flow chart. PCI, percutaneous coronary intervention.

Table 1 Demographics, triage vital signs, comorbidities and laboratory findings of patients with ACS in different BS group

Variables	B1 (n=361)	B2 (n=584)	B3 (n=1902)	P value
Age, years	73.97±11.91	72.25±11.23	62.96±12.55	<0.001
Males, n (%)	120 (33.2)	202 (34.6)	374 (19.7)	<0.001
BMI, kg/m ²	22.98±3.69	23.36±3.5	24.56±3.2	<0.001
Hypertension, n (%)	216 (59.8)	332 (56.8)	996 (52.4)	0.012
Diabetes, n (%)	137 (38)	180 (30.8)	480 (25.2)	<0.001
Hyperlipidaemia, n (%)	39 (10.8)	63 (10.8)	214 (11.3)	0.935
COPD, n (%)	24 (6.6)	29 (5)	48 (2.5)	<0.001
SBP, mm Hg	123.83±28.25	127.68±25.87	128.49±23.15	0.004
DBP, mm Hg	73.41±18.22	75.84±16.24	79.12±15.16	<0.001
Heart rate, beats/min	86.68±22.62	83.34±19.38	79.24±17.07	<0.001
Killip class >1, n (%)	244 (67.6)	319 (54.6)	725 (38.1)	<0.001
STEMI, n (%)	209 (57.9)	332 (56.8)	996 (52.4)	0.990
LVEF	48.71±13.31	52.34±11.76	54.67±11.25	<0.001
Laboratory findings				
WBC, 10 ⁹ /L	9.67 (7.3–12.31)	9.2 (6.93–12.32)	8.86 (6.91–11.29)	0.001
Platelet count, 10 ⁹ /L	184.62±77.08	187.76±80.5	183.9±72.02	0.551
Blood glucose, mmol/L	10.43±5.33	9.24±4.46	8.53±3.95	<0.001
Creatinine, µmol/L	97 (75.75–145.25)	83 (69–110)	77 (65–92)	<0.001
eGFR	60.12±28.14	67.87±26.25	83.07±23.49	<0.001
BUN, mmol/L	9.58±6.89	7.92±4.96	6.29±3.42	<0.001
Triglycerides, mmol/L	1.19 (0.84–1.62)	1.19 (0.84–1.73)	1.41 (0.97–2.12)	<0.001
Total cholesterol, mmol/L	4.11±1.31	4.3±1.15	4.48±1.26	<0.001
HDL, mmol/L	1.13±0.38	1.19±0.38	1.13±0.33	0.002
LDL, mmol/L	2.51±1.13	2.63±1.01	2.79±1.1	<0.001
NT-proBNP, pg/mL	3099 (1171–9137)	1616 (392–4307)	566 (154–1767)	<0.001
CTnT, pg/mL	967.9 (189–3554)	728 (100–2941)	458 (55–2099)	<0.001
PCI				
Urgent, n (%)	229 (63.8)	390 (66.8)	1263 (66.6)	0.566
Contrast dose, mL	131.88±30.33	125.21±27.03	120.04±23.16	<0.001
Stent	1 (1–2)	1 (1–2)	1 (1–2)	0.208
Three-vessel disease, n (%)	100 (27.7)	117 (20.0)	298 (15.7)	<0.001
Procedure time, min	41.92±10.66	41.14±10.25	39.85±9.85	<0.001
Risk score				
GRACE score	178.55±43.95	163.39±38.6	137.43±35.12	<0.001
Gensini score	80 (34–124)	66 (37–103)	57 (30–90)	<0.001
Mehran risk score	15.47±5.15	13.85±5.15	11.17±5.01	<0.001

ACS, acute coronary syndrome; BMI, body mass index; BS, Braden Scale; BUN, blood urea nitrogen; COPD, chronic obstructive pulmonary disease; CTnT, cardiac troponin T; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; GRACE, Global Registry of Acute Coronary Events; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-brain natriuretic peptide; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; STEMI, ST-segment elevation myocardial infarction; WBC, white blood cell count.

continuous variables were reported as medians (25th–75th) or means±SDs, according to non-normal and normal distribution and compared using the Mann-Whitney U test or analysis of variance, respectively.

The logistic regression model was used to evaluate whether BS scores are associated with AKI. All variables were included in a univariate model, and the significant variables were re-entered into a multivariable model. The area under the receiver operating

Figure 2 The incidence of AKI in patients with different BS score. AKI, acute kidney injury; BS, Braden Scale.

characteristic (ROC) curves was established to evaluate the ability of the BS scores to predict AKI. The cumulative survival rates among the three groups of patients (B1 vs B2 vs B3) with or without AKI were compared by the Kaplan-Meier curve. To explore the indirect effect of the BS scores on all-cause mortality through the bypath of AKI, path analysis established by structural equation modelling was performed.²⁹ The results of the path analysis were analysed by standardised regression coefficients (β) to describe the direct and indirect effects on all-cause mortality, and the proportion of the mediating effect was calculated by dividing the regression coefficient of the indirect path by the total regression coefficient. A two-tailed p value of <0.05 was considered significant. Data were analysed using SPSS Statistics V.20.0 and R for Windows V.3.5.0.

Patient and public involvement

The patients were not involved in the design of or data collection in this study.

RESULTS

Baseline patient characteristics

The mean age of the 2847 patients with ACS was 66.26 ± 13.08 years, and 2151 (75.5%) of them were men. There were 361 patients (12.7%), 584 patients (20.5%) and 1902 patients (66.8%) assigned to B1, B2 and B3 groups, respectively. Baseline characteristics of the patients in the B1 group were that they were older and more likely to have complications, such as hypertension, diabetes and Killip class $\geq$ II, and have higher baseline renal function indicators, cardiac biomarkers, consumption of contrast agent, GRACE score, Gensini score and Mehran risk score ($p < 0.05$, table 1). The baseline characteristics of patients divided into non-AKI, AKI stage 1, AKI stage 2 and AKI stage 3 are compared in online supplemental table 1.

The relationship between the BS and AKI

With the increase in the BS scores, the incidence of AKI gradually decreased ($p < 0.001$). Patients with lower BS

Table 2 Univariate and multivariate logistic regression analysis of Braden Scale for acute kidney injury

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	P value	OR	95% CI	P value
Braden Scale	–	–	<0.001	–	–	<0.001
B1 vs B3	5.881	4.530 to 7.636	<0.001	2.242	1.643 to 3.060	<0.001
B2 vs B3	2.934	2.299 to 3.745	<0.001	1.566	1.186 to 2.069	0.002
Age (years)	1.069	1.059 to 1.079	<0.001	1.056	1.044 to 1.068	<0.001
Male	1.606	1.292 to 1.995	<0.001	0.836	0.647 to 1.081	0.173
Systolic blood pressure (mmHg)	0.988	0.981 to 0.994	<0.001	1.001	0.994 to 1.009	0.739
Heart rate (beats/min)	1.011	1.006 to 1.017	<0.001	1.000	0.994 to 1.007	0.915
Statin	0.439	0.335 to 0.576	<0.001	0.585	0.424 to 0.807	0.001
Diabetes	2.564	2.088 to 3.149	<0.001	2.123	1.675 to 2.691	<0.001
Killip class	2.072	1.878 to 2.286	<0.001	1.640	1.461 to 1.842	<0.001
White blood cell count ($10^9/L$)	1.070	1.044 to 1.096	<0.001	1.061	1.028 to 1.096	<0.001
Cardiac troponin T (per 100 pg/mL)	1.008	1.005 to 1.011	<0.001	1.005	1.001 to 1.009	0.010

Figure 3 ROC curve analysis according to BS score for AKI in patients with ACS. ACS, acute coronary syndrome; AKI, acute kidney injury; AUC, area under the curve; BS, Braden Scale; ROC, receiver operating characteristic.

scores had a higher incidence of AKI (38.5% vs 23.9% vs 9.6%) and AKI stage 1 (20.5% vs 13.4% vs 6.4%), AKI stage 2 (9.1% vs 5.5% vs 1.4%) and AKI stage 3 (8.9% vs 5.0% vs 1.8%) (figure 2).

In univariate logistic regression analysis, BS score, age, male sex, systolic blood pressure, heart rate, statin level, diabetes, Killip class, white blood cell count and cardiac troponin T level were significantly related with AKI. After adjusting all significant factors, the multivariate logistic regression model showed that decreased BS scores were independent predictors for AKI (B1 vs B3: OR: 2.242, 95% CI: 1.643 to 3.060, $p=0.002$; B2 vs B3: OR: 1.566, 95% CI: 1.186 to 2.069, $p<0.001$, table 2).

In the ROC curve analysis, the BS scores showed a significant prediction efficiency for AKI (area under the curve (AUC): 0.719; 95% CI: 0.702 to 0.736; $p<0.001$, figure 3). The cut-off value of BS scores was set at 12, yielding a high sensitivity of 90.7% and a specificity of 30.2% for AKI. Moreover, when the cut-off value of BS scores was set at 15, the sensitivity and specificity for AKI were 45.6% and 77.2%, respectively. Moreover, BS scores improved the AUC of Mehran risk score from 0.765 (95% CI: 0.742 to 0.788) to 0.785 (95% CI: 0.763 to 0.807), which was significantly greater than that of Mehran risk score alone ($p<0.001$).

Subgroup analysis

The OR for AKI was calculated after grouping the patients by age, sex, systolic blood pressure, heart rate, statin level, diabetes, Killip class, white blood cell count and cardiac troponin T. A lower BS score was still

associated with a higher incidence of AKI in patients with ACS (table 3).

The interaction among BS groups, AKI and mortality

Overall, 126 patients (4.4%) died in the hospital, and 345 (12.1%) total deaths were recorded during the follow-up period. Patients with lower BS or AKI have higher all-cause mortality and longer length of stay ($p<0.001$) in the hospital and higher all-cause mortality during the follow-up period ($p<0.001$, online supplemental tables 2 and 3).

Kaplan-Meier analysis showed that the cumulative survival rate of patients with lower BS scores or AKI was lower than that of patients without these factors in patients with ACS (B1+AKI vs B2+AKI vs B1+non-AKI vs B3+AKI vs B2+non-AKI vs B3+non-AKI: 35.2% vs 60.4% vs 67.4% vs 77.9% vs 81.0% vs 92.5%, figure 4).

The BS score was significantly associated with both AKI and all-cause mortality during the follow-up period according to path analysis ($p<0.001$). In the structural equation model, the indirect impact of the BS score on long-term all-cause mortality mediated by AKI accounted for 30% (figure 5).

DISCUSSION

Our study found that the incidence of AKI gradually increased with the decrease of BS scores in patients with ACS. According to multivariate logistic regression analysis, BS scores were independently related to AKI in patients with ACS. In subgroup analysis, this association

Table 3 Logistic regression analysis of AKI in different subgroups according to BS

Variables	B1 vs B3	P value	B2 vs B3	P value
	OR (95% CI)		OR (95% CI)	
Age, years				
≤67	8.351 (4.729 to 14.746)	<0.001	4.513 (2.725 to 7.474)	<0.001
>67	3.424 (2.533 to 4.628)	<0.001	1.718 (1.292 to 2.284)	<0.001
Gender				
Male	6.635 (4.832 to 9.112)	<0.001	3.520 (2.619 to 4.730)	<0.001
Female	3.867 (2.431 to 6.149)	<0.001	1.710 (1.106 to 2.645)	0.016
DBP, mm Hg				
≤77	5.573 (3.934 to 7.895)	<0.001	2.566 (1.834 to 3.589)	<0.001
>77	5.877 (3.935 to 8.777)	<0.001	3.308 (2.316 to 4.725)	<0.001
Heart rate, beats/min				
≤78	6.438 (4.280 to 9.684)	<0.001	2.621 (1.790 to 3.837)	<0.001
>78	5.378 (3.820 to 7.570)	<0.001	3.074 (2.231 to 4.237)	0.004
WBC, 10 ⁹ /L				
≤9.00	4.316 (2.905 to 6.412)	<0.001	2.845 (2.008 to 4.031)	<0.001
>9.00	7.394 (5.187 to 10.54)	<0.001	3.021 (2.145 to 4.255)	<0.001
CTnT, pg/mL				
≤556	4.155 (2.763 to 6.248)	<0.001	2.483 (1.739 to 3.546)	<0.001
>556	7.336 (5.166 to 10.416)	<0.001	3.371 (2.403 to 4.729)	<0.001
Killip class				
I	3.629 (2.210 to 5.957)	<0.001	2.099 (1.384 to 3.185)	<0.001
II–IV	5.418 (3.904 to 7.519)	<0.001	2.946 (2.150 to 4.037)	<0.001
GRACE score				
≤143	3.797 (1.978 to 7.287)	<0.001	2.383 (1.378 to 4.122)	0.002
>143	3.952 (2.919 to 5.35)	<0.001	2.029 (1.524 to 2.700)	<0.001
Statin admission				
No	5.804 (3.118 to 10.806)	<0.001	2.616 (1.364 to 5.018)	<0.001
Yes	5.435 (4.058 to 7.281)	<0.001	2.933 (2.253 to 3.819)	<0.001
Acute myocardial infarction				
STEMI	5.012 (3.420 to 7.346)	<0.001	3.075 (2.189 to 4.320)	<0.001
NSTE-ACS	7.044 (4.897 to 10.131)	<0.001	2.876 (2.012 to 4.111)	<0.001

The continuous variables were grouped by median.
 AKI, acute kidney injury; BS, Braden Scale; CTnT, cardiac troponin T; DBP, diastolic blood pressure; GRACE, Global Registry of Acute Coronary Events; NSTE-ACS, non-ST-segment elevated acute coronary syndrome; STEMI, ST-segment elevated myocardial infarction; WBC, white blood cell count.

was consistent in patients with different characteristics. Furthermore, the BS score achieved a significant incremental predictive value beyond the Mehran risk scores, which is a widely validated contrast-induced nephropathy assessment index.^{30 31} Therefore, in clinical nursing practice, the BS cannot only be used to assess the risk of pressure ulcer^{19 32} but can also be used as a tool for the risk assessment of AKI.

According to a meta-analysis, the incidence of AKI in patients with ACS was as high as 15.8%, which was similar to our findings in this study, and the incidence of AKI in the present study was 16.2%.⁸ Considering that numerous studies have confirmed the adverse effects of AKI on the prognosis of patients with ACS, predicting the incidence

of AKI is a key measure for early risk stratification of patients with ACS.

As in previous studies, both BS scores and AKI were risk factors for death^{10 19}; therefore, we aimed to further clarify the interaction between BS scores, AKI and death. The Kaplan-Meier survival analysis was constructed, and the results showed that the cumulative survival rate of ACS patients with lower BS scores and/or AKI was lower than that of patients with ACS without these factors. More importantly, the path analysis by the structural equation model showed that the approximately 30% indirect impact of BS scores on long-term all-cause mortality was mediated by AKI. It was suggested that BS scores were

Figure 4 Kaplan-Meier survival curve of long-term all-cause mortality in different BS score patients with or without AKI. AKI, acute kidney injury; BS, Braden Scale.

closely related to AKI, and their interaction increases the occurrence of mortality events.

Although the BS is a widely used tool for the routine nursing assessment of pressure ulcer events, it evaluates several indicators related to frailty, such as nutrition, cognition and function. Therefore, recent studies recommended it as a tool to identify frailty.¹⁸ However, there may be other mechanisms to explain the relationship between BS scores and AKI, given that a low BS score does not necessarily indicate frailty. In fact, patients show lower cognition, function and activity in the acute stage of ischaemic events, which may be affected by the severity of the disease itself. Even though the BS score reflects the frailty from some aspects, the BS score on admission does not represent the BS score for patients under the usual state. As shown, patients with lower BS scores have higher Killip class and cardiac marker levels, lower LVEF and diastolic blood pressure, and higher incidence of cardiogenic shock, reflecting a more severe haemodynamic impairment. In addition, patients with lower BS

scores have higher frequency of multiple vessel lesions and more severe coronary stenosis, which may explain the increased consumption of contrast agents. Finally, higher baseline indicators for inflammation, thrombosis and stress may also be related to patients with lower BS scores. All of these may be potential contributors to AKI. Moreover, several studies have shown that the assessment of peripheral arterial disease is helpful to predict contrast-induced AKI.^{33 34} At the same time, peripheral artery disease has been proved to be an independent factor of pressure ulcer and related to BS scores.^{35–37} Given that the BS consists of activity, movement and perception, it may reflect the severity of peripheral arterial disease and indirectly assess the risk of AKI events. Therefore, the BS score prediction of AKI events may be due to several factors.

For patients at risk of AKI assessed by the BS score, nurses should administer nephrotoxic drugs cautiously, reduce the dose of contrast agents rationally, ensure adequate hydration and maintain renal perfusion.^{20 21} In addition to increasing the preventive measures for AKI, nurses should prevent the adverse impact of frailty on AKI based on comprehensive interventions aimed at improving the prognosis of patients with ACS and improving BS scores, such as adequate nutrition intake, provision of social support, maintenance of cognitive function and ability to perform daily activities, which may ameliorate the vicious circle between frailty and AKI.^{38 39}

CONCLUSIONS

The BS score at admission was independently associated with AKI for patients with ACS. The BS may be a useful and simple tool to identify the risk of AKI among patients with ACS. In addition, more than 30% of the effects of the BS score on mortality were mediated by AKI, which suggests that frailty and AKI are two important intervention targets to improve the prognosis of patients with ACS.

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Contributors ZL, DL, ZZ and ZW conceived and planned the study. ZL, BL and YJ performed the analysis. ZL, BL, ZZ and ZW wrote the original draft with input from all authors. ZL, BL and YJ visualised the results. ZL, BL, YJ and DL contributed to the interpretation of the results. XL, ZZ and ZW did critical revision of the article. ZZ and ZW supervised the entire process. All the authors read and approved the final manuscript. All authors take responsibility for the integrity of the data and accuracy of the data analysis. ZL was responsible for the overall content as guarantor. ZL accepts full responsibility for the finished work and/or the conduct of the study, had access to the data, and controlled the decision to publish.

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Competing interests None declared.

Patient consent for publication Not applicable.

Figure 5 Direct and indirect effects of admission BS score on AKI and long-term all-cause mortality. β coefficient was calculated by the standard regression equation. AKI, acute kidney injury; BS, Braden Scale.

Ethics approval The study complied with the Declaration of Helsinki, and the Human Ethical Committee of West China Hospital of Sichuan University approved the study protocol.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. Data are available upon reasonable request. The data are available upon request from the corresponding author (303680215@qq.com).

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