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ORIGINAL RESEARCH

Predictive Value of Troponin I, Creatinine Kinase Isoenzyme and the New Japanese Severity Score in Severe Acute Pancreatitis

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Purpose: To evaluate troponin I, creatine kinase isoenzyme, and the new Japanese Severity Score(JSS) for predicting Severe Acute Pancreatitis-Associated myocardial Injury(SACI).

Patients and Methods: This retrospective study included 136 patients with Severe Acute Pancreatitis, hospitalized in grade-III hospital from June 1, 2015, to October 31, 2022; selected using convenience sampling method and divided into SACI occurrence (n =34) and SACI non-occurrence (n =102) groups. New JSS evaluated predictive value of each SACI index. Binary logistic regression model compared risk factors and constructed a prediction model. Area under receiver operating characteristic curve (AUC) and Hosmer–Lemeshow goodness of fit test evaluated model's prediction efficiency and calibration ability.

Results: The incidence of SACI was 25%. Univariate analysis found that troponin I and creatine kinase isoenzyme were significantly different ($P < 0.05$) and independent risk factors for SACI. The new JSS, troponin I, and creatine kinase isoenzyme were included in the prediction model. The prediction model had a good calibration ability, and its predicted value and the actual observed value were not significantly different (Hosmer–Lemeshow $\chi2 = 5.408$, $P = 0.368$). AUC of the model was 0.803 (95% CI: 0.689–0.918), and the optimal threshold of the prediction model was 0.318 with the maximum Youden index (0.488). The AUC for internal validation was 0.788 (95% CI: 0.657–0.876), and external validation was 0.761 (95% CI: 0.622–0.832).

Conclusion: Troponin I and creatine kinase isoenzymes combined with the new JSS have a high predictive value for SACI, improving the early prediction and treatment of at-risk patients.

Keywords: acute pancreatitis, new Japanese severity score, myocardial injury, prediction model, pancreatic heart syndrome

Introduction

Acute pancreatitis is characterized by oedema, haemorrhage, and necrosis due to digestion of the pancreatic tissue caused by multiple pathogenic factors. According to the 2012 New Atlanta AP Classification Standards,[1,2] pancreatitis is defined as mild acute pancreatitis(MAP), moderately severe acute pancreatitis(MSAP), or severe acute pancreatitis (SAP). Studies have shown that MAP and MSAP account for a large proportion of cases, and following active treatment, patients usually recover quickly.[3,4] However, SAP accounts for approximately 20–30% of cases of AP, and the hospital mortality rate is approximately 15–30%.[3,5] SAP-associated myocardial injury (SACI), also known as "pancreatic heart syndrome", is a serious complication of SAP disease progression characterized by a high mortality rate. The clinical manifestations are varied and include abnormal cardiac function, toxic myocarditis, pericarditis, pericardial effusion, and heart failure.[1,6–8] There is an urgent need for specific evaluation tools with good applicability to enable nurses to play an important role in the early identification, rescue, and management of patients with SACI; early identification of high-risk patients with SACI; and provide predictive care and improve the prognosis of patients with high-risk factors. However, existing studies mainly focus on risk factor analysis and evaluation and improvement of scoring systems, without

Received: 31 January 2024
Accepted: 30 May 2024
Published: 6 June 2024

effective combination, and therefore, they lack specificity.[3] Nurses often identify SACI through electrocardiograph monitoring and other empirical means, but there is a lack of reliable and convenient risk prediction tools for SACI. In addition, nursing work and research predominantly focus on the rescue, treatment coordination, and follow-up care of patients with SACI, and not on the potential role of nurses in early identification and intervention of high-risk patients with clinical SACI. In order to further advance clinical nursing work, the focus of this study was to construct a prediction model for the risk of myocardial injury in patients with SAP during hospitalization, and combine this model with independent risk factors to construct a predictive nursing plan for patients with SACI. Early prediction and recognition of SACI are key to establishing a corresponding early warning program, successful treatment, and improving nursing quality and prognosis.[9,10]

Most of the current studies on pancreatitis severity and early prediction are based on machine learning. Most of them are retrospective. Li and colleagues developed an inflammation-based model to identify SAP. They selected diabetes mellitus, fatty liver, high white blood cell count, C-reactive protein, and other factors to build this model.[11] And the model comprising fatty liver, procalcitonin, and C-reactive protein-to-lymphocyte ratio exhibits satisfactory diagnostic performance for SAP. Moreover, researchers employed artificial intelligence in conjunction with scikit-learn, XGBoost, and catboost Python packages for modeling to predict the severity of acute pancreatitis.[12] The XGBoost classifier emerged as the most effective model, with the six most influential features being the respiratory rate, body temperature, abdominal muscular reflex, gender, age, and glucose level. Sun and other scholars employed 11 blood molecules, including prothrombin time, serum potassium, and lymphocyte percentage, to forecast the severity of acute pancreatitis.[13] They then compared the constructed model with APACHE II (Acute Physiology and Chronic Health Evaluation II), BISAP, and Ranson score, which demonstrated that the model exhibited greater accuracy and broader applicability. The majority of existing predictive models incorporate examination indicators during the patient's hospitalization into the model construction process, with a comparison being made to previous models. However, few researchers have combined the scoring criteria with the examination indicators for predictive model construction. Otherwise, the current prediction model on SACI could not be retrieved.

The new Japanese Severity Score (JSS) was proposed and improved by Japanese researchers based on more than a decade of clinical trials and studies in Asian populations.[14] According to studies, the new JSS system is more professional, simple, and effective than the APACHE II, Ranson score, and 18 other scoring systems for evaluating SAP in Asian people.[15] However, since the application scope of the new JSS is primarily limited to Japan, continued research in form of clinical studies are warranted combined with multi-factors to introduce it in China. Therefore, based on the new JSS, combined with the independent risk factors troponin I and creatine kinase isoenzyme of SACI, this study constructed an early warning assessment tool aimed at providing guidance for prediction and early nursing intervention of SACI.

Materials and Methods

Patients

A total of 136 patients with SAP who were hospitalized in a grade-III hospital from June 1, 2015, to October 31, 2022 were selected to be included in the study using the convenience sampling method. The inclusion criteria were: (1) Meet the 2012 New Atlanta AP classification standard; (2) Age ≥18 years old; (3) Hospitalization duration ≥24 hours. The exclusion criteria were: (1) Patients with incomplete clinical data; (2) Patients with chronic diseases of the heart, liver, kidney, and other important organs, and malignant tumor's; (3) Pregnant and lactating women; (4) Patients with history of immunosuppressant and hormone drug use within 1 month before the onset of disease; (5) Patients with arrhythmias and patients who develop other organ infections during admission.

Tools

The new JSS consists of nine evaluation indicators and CT grading. The evaluation indicators include: (1) Residual alkali ≤ −3 mmol/L or shock (systolic blood pressure < 10.66 kPa); (2) PaO_2 ≤ 7.99 kPa (indoor air) or respiratory failure (ventilator assisted ventilation); (3) Blood urea nitrogen (BUN) ≥ 14.28 mmol/L (40 mg/dL), creatinine ≥ 176.8 μmol/L

(2.0 mg/dL), or oliguria (urine volume after intravenous fluid resuscitation; < 400 mL/d); (4) Lactate dehydrogenase ≥ 2 times the upper limit of normal value; (5) Platelet count ≤ 100×109 /L; (6) Serum calcium ≤ 1.88 mmol/L (7.5 mg/dL); (7) C-reactive protein ≥ 150 mg/L; (8) Diagnostic criteria for systemic inflammatory response syndrome (SIRS) ≥ 3 terms (a. body temperature > 38°C or < 36°C. b. heart rate > 90 beats/min. c. Respiratory rate > 20 breaths/min or PaCO2 < 32 torr (1 torr = 1 mmHg = 0.133 kPa). d. White blood cells > 12×109 /L, WBC < 4×109 /L, or > 10% juvenile erythrocytes); (9) Age ≥ 70 years. The CT grading includes: (1) Inflammation spread to the prerenal space of the extrapancreatic tissue (0 points), the mesocolon root (1 point), and the tissue below the lower pole of the kidney (2 points). (2) The pancreas under low enhancement on CT is divided into three segments: the head, body, and tail of the pancreas. Lesions confined to one segment or only involving peripancreatic tissue score 0 points, extending to two segments score 1 point, and covering two whole segments or more score 2 points. The scoring system for the CT grading is: (1) + (2) = total score. According to the obtained total score, a total score of 0 or 1, is grade 1; total score = 2, is grade 2; total score ≥ 3, is grade 3. According to the new JSS standard, if the nine evaluation indicators are ≥ 3 points, or the CT grading is > 2, the severity of the disease is graded as "critical".

Outcome Index

In this study, myocardial injury was the outcome index, and troponin I > 0.03 ng/mL was defined as myocardial injury according to the normal reference value of troponin I in our hospital.[16]

Data Collection

Two graduate students trained and qualified in scale application, data collection, and other related subjects were appointed as full-time data collection personnel for this study. The electronic medical records of patients diagnosed with "severe acute pancreatitis" were selected from the medical record inquiry system of the hospital, and two graduate students were included as eligible patients in strict accordance with the inclusion and exclusion criteria of this study. In order to improve the accuracy and comprehensiveness of this study, after consulting relevant literature and the electronic medical record system, the relevant data for this study, including laboratory examination, imaging examination, admission record, disease course record, and discharge summary, were collected. The data was double-checked and the following data was collated in an Excel file: (1) Sociodemographic data: patient's age, sex, history, and hospital admittance blood pressure, heart rate, body temperature, and respiratory rate. (2) Laboratory and imaging data of the patient within 24 hours after admission. Laboratory indicators included: Leukocyte and platelet count, D-dimer, activated partial thromboplastin time, thrombin time, fibrinogen, blood glucose, amylase, high density lipoprotein, low density lipoprotein, CRP, LDH, serum calcium, triglyceride, troponin I, creatine kinase isoenzyme, total cholesterol, procalcitonin, BUN, oxygen partial pressure, and base surplus. Radiographic indicators included: pancreatic and/or peripancreatic inflammation, mild exudation, single fluid accumulation, multiple fluid accumulation, pancreatic and fat necrosis, pancreatic abscess, pleural effusion, and abdominal effusion.

Data Analysis

SPSS 25.0 was used for statistical analysis of the data. For measurement data satisfying a normal distribution, an independent sample *t* test was used for comparison between groups, expressed as mean ± standard deviation ($\bar{x}$±S); The Mann–Whitney *U*-test was used to represent the non- normal distribution, and the quartile [P50 (P25, P75)] was used to represent the count data (eg, %), The chi-squared test was used for comparison between groups; Binary logistic regression analysis was used for multivariate analysis, and the area under receiver operating characteristic curve (AUC) was used to test the prediction effect of the model, and the AUC of the model was compared with the new JSS. According to the prediction efficiency rule: 0.5 ≤ AUC < 0.7 indicates the prediction efficiency is low; 0.7 ≤ AUC < 0.9 indicates the prediction efficiency is medium; 0.9 ≤ AUC ≤ 1.0 indicates the prediction efficiency is high. When the Youden index reaches the maximum, it is regarded as the critical value of the prediction model. The prediction model of this study was internally verified using the Bootstrap repeated sampling method (sampling times = 500), and externally verified using new sample data (cases admitted after the data collection deadline of this study, from June to October 2022). All test results were statistically significant with P < 0.05.

Results

A total of 136 patients were included in this study, including 98 men (72.06%) and 38 women (27.94%). The average age was 48.30 ± 16.13 years. The new JSS ranged from 0 to 8, with an average of 3.33 ± 1.92. SACI occurred in 34 cases (25%).

Single-Factor Analysis of SACI

Patients with SACI and those without SACI were classified into the occurrence group and the non-occurrence group, respectively. The univariate results of SACI showed that the intergroup comparison of new JSS (P = 0.039), respiratory rate (P = 0.008), WBC count (P = 0.025), platelet count (P=0.037), activated partial thrombin time (P = 0.026), troponin I (P = 0.000), creatine kinase isoenzyme (P = 0.011), and residual alkali (P = 0.028) were statistically significant, while other indicators were not statistically significant (P > 0.05), as shown in Table 1.

Table 1 Results of Univariate Analysis of SACI (n=136)

Variable	Constituencies		Statistics	*P*
	Non-SACI(n=102)	SACI(n=34)		
Age(year)	44.00(36.00, 57.25)	45.50(35.00, 66.25)	−0.128[a]	0.898
Gender				
Female	31(81.58%)	7(18.42%)	1.217[c]	0.270
Male	71(72.45%)	27(27.55%)		
New JSS	3.00(2.00, 4.00)	4.00(3.00, 6.00)	−2.062[b]	0.039
Temperature(°C)	36.50(36.30, 37.00)	36.70(36.28, 37.25)	−0.672[b]	0.501
Heart Rate(HR)	97.38±23.53	99.82±20.03	−0.543[a]	0.588
Respiratory Rate(RR)	22.00(18.75, 25.00)	24.50(20.75, 26.50)	−2.635[b]	0.008
SBP(mmHg)	137.58±21.19	142.94±22.22	−1.263[a]	0.209
DBP(mmHg)	88.00(78.00, 97.00)	90.50(76.75, 103.25)	−0.704[b]	0.482
Alcohol	34(75.56%)	11(24.44%)	0.011[c]	0.916
Smoke	40(72.73%)	15(27.27%)	0.254[c]	0.614
Hypertension	25(69.44%)	11(30.56%)	0.806[c]	0.369
Hyperlipidemia	8(88.89%)	1(11.11%)	0.992[c]	0.319
WBC(×10^9/L)	13.54±4.74	15.65±4.49	2.268[a]	0.025
Platelet(×10^9/L)	223.07±85.32	188.09±77.86	2.112[a]	0.037
D-Dimer(mg/L)	1.94(0.73, 4.83)	2.52(0.56, 5.17)	−0.159[b]	0.874
Prothrombin Time(s)	12.30(11.10, 13.60)	12.40(11.00, 14.10)	−0.415[b]	0.678
APTT(s)	27.00(23.05, 30.15)	28.80(25.90, 37.80)	−2.230[b]	0.026
Thrombin Time(s)	17.90(16.40, 19.70)	18.10(16.70, 21.50)	−0.788[b]	0.431
Fibrinogen(g/L)	4.45±1.92	3.83±2.32	1.490[a]	0.139
Troponin I(ng/mL)	0.01(0.01, 0.03)	0.05(0.04, 0.30)	−9.036[b]	0.000
Blood Glucose(mmol/L)	9.52(7.30, 12.98)	9.43(7.03, 14.46)	−0.168[b]	0.867
Amylase(U/L)	404.05(145.73, 932.60)	497.90(142.05, 1015.85)	−0.318[b]	0.751
HDL(mmol/L)	1.21(0.84, 2.04)	1.19(0.90, 3.69)	−0.440[b]	0.660
LDL(mmol/L)	2.62±1.43	2.29±1.91	1.055[a]	0.293
C-reactive protein(mg/L)	113.40(36.00, 168.35)	42.00(6.88, 132.38)	−1.734[b]	0.083
Lactate Dehydrogenase(U/L)	300.10(232.50, 413.90)	309.95(226.58, 633.35)	−0.841[b]	0.400
Serum Calcium(mmol/L)	2.09±0.26	2.12±0.32	−0.568[a]	0.571
Triglyceride(mmol/L)	5.49(1.14, 18.16)	4.09(1.09, 20.67)	−0.366[b]	0.714
Creatine Kinase Isoenzyme(ng/mL)	3.52(1.75, 15.40)	8.41(4.49, 30.15)	−2.551[b]	0.011
Total Cholesterol(mmol/L)	5.81(4.21, 11.92)	5.17(3.59, 12.62)	−0.781[b]	0.435
Procalcitonin(ng/mL)	0.59(0.28, 1.60)	0.75(0.13, 3.03)	−0.449[b]	0.654
PaO_2(mmHg)	76.50(64.00, 102.75)	75.50(64.50, 92.25)	−0.451[b]	0.652
Base Excess	−1.10(−4.40, 0.75)	−3.70(−9.93, −0.15)	−2.194[b]	0.028

Notes: [a]*t* value; [b]*Z* value; [c]χ^2.

Abbreviations: New JSS, new Japan Severity Score; SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cells; APTT, activated partial thromboplastin time; HDL, high density lipoprotein; LDL, low density lipoprotein; PaO_2, arterial oxygen partial pressure.

Multi-Factor Analysis of SACI

Multi-factor analysis was used to determine whether SACI occurs as the dependent variable (assignment: occurrence = 1, non-occurrence = 0). Factors with statistically significant differences in the univariate analysis in Table 1 (new JSS, respiratory rate, WBC count, platelet count, activated partial thromboplastin time, troponin I, creatine kinase isoenzyme, and residual alkali) were taken as independent variables for binary logistic regression analysis. Collinearity test results showed that the variance inflation factors of all indicators were less than 10, indicating no collinearity. Logistic stepwise regression results showed that new JSS, leukocyte count, troponin I, and creatine kinase isoenzyme were independent risk factors for SACI, as shown in Table 2.

Prediction Model and Effect Evaluation

According to the results of multi-factor analysis, the prediction model was constructed as follows: Logit (SACI) = −5.825 + (0.536 * JSS) + (0.025 * creatine kinase isoenzyme) + (0.096 * leukocyte count) + (0.035 * troponin I). The AUC of the model was 0.803 (95% CI: 0.689–0.918), the specificity was 0.885, the sensitivity was 0.710, the negative predictive value was 0.912, and the positive predictive value was 0.647. The optimal threshold of the prediction model was 0.318 with the maximum Youden index (0.488). The AUC for internal validation was 0.788 (95% CI: 0.657–0.876) and external validation was 0.761 (95% CI: 0.622–0.832) (Figure 1).

Comparison Between the Prediction Model and the New JSS Prediction Model

Compared with our prediction model, the AUC of the new JSS prediction model was 0.697 (95% CI: 0.631–0.873); specificity, 0.847; sensitivity, 0.580; positive predictive value, 0.529; negative predictive value, 0.872; the maximum Youden index (0.382) to determine the optimal threshold of the new JSS prediction model was 4.5. There was a statistically significant difference in the AUC between the prediction model and the new JSS prediction model ($P < 0.05$) (Figure 2).

Discussion

Analysis of SACI Related Risk Factors

New JSS

The results of this study demonstrated that the new JSS exhibited a specificity of 0.847, a sensitivity of 0.580, a positive predictive value of 0.529, and a negative predictive value of 0.872. These values were found to have an independent predictive value for SACI, similar to the results of a large multicenter European study.[14] The new JSS was developed based on the Asian population. The related research findings indicated that the new JSS score has a good predictive value for in-hospital mortality in patients with SAP and helps in the early assessment of hospital admission.[15] And study has shown that the new JSS is very useful and easier to use than traditional scores in predicting prognosis, such as Ranson score and APACHE II.[17] Serum calcium, LDH, CRP, advanced age, SIRS, and other indicators in the new JSS are risk factors for SACI,[14] which comprehensively reflect the basic status of patients. Consequently, this study constructed a clinical prediction model of SACI based on the new JSS.

Leukocyte Count

SAP is a life-threatening condition, and the WBC levels must be monitored closely during the occurrence and development of the disease.[18,19] Binary logistical regression analysis demonstrated that WBC levels were an independent risk factor for SACI. This is closely related to the condition and prognosis of patients with SAP, as shown by Zeng L et al.[20] SAP is an abdominal

Table 2 Results of Multivariate Analysis of SACI

	B	*SE*	*Wald*	*P*	OR(95% CI)
New Japan Severity Score	0.536	0.158	11.543	0.001	1.709(1.254, 2.327)
Creatine Kinase Isoenzyme	0.025	0.010	6.032	0.014	1.026(1.005, 1.046)
White Blood Cells	0.096	0.044	4.822	0.028	1.101(1.010, 1.200)
Troponin I	0.035	0.015	5.477	0.019	1.036(1.006, 1.067)
Constant Variable	−5.825	2.067	7.941	0.005	0.003

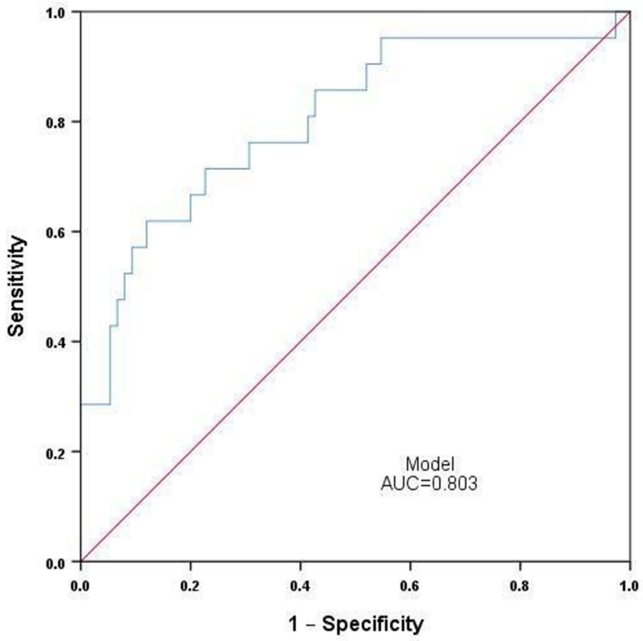

Figure 1 Effect analysis of the prediction model in this study.
Abbreviation: AUC, area under receiver operating characteristic curve.

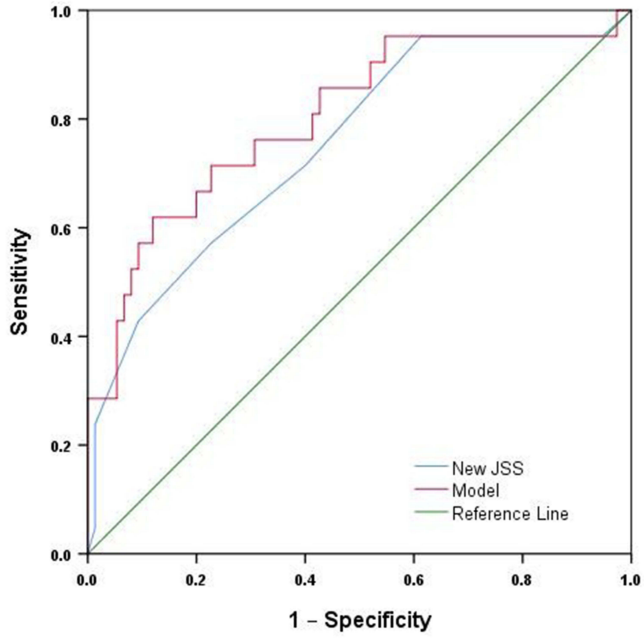

Figure 2 Comparison between the prediction model and the new JSS prediction model.

emergency with extensive inflammation and necrosis of pancreatic tissue accompanied by distal organ damage. SAP may rapidly manifest as a local inflammatory amplification reaction, enhanced expression of inflammatory factors, and SIRS,[21–23] resulting in the development of complicated and severe disease. The result is SIRS and MODS. Studies have shown that oxidative stress caused by the inflammatory response is closely related to the increased risk of occurrence.[24,25] Based on this, it is recommended that medical staff should reinforce dynamic assessment and monitoring of WBC levels in patients with SAP, in order to facilitate effective intervention in the clinical progression of SACI.

Markers of Myocardial Injury

Traditional markers of myocardial injury have high sensitivity and specificity for simple myocardial injury, but their sensitivity and specificity for SAP complicated myocardial injury are uncertain. The results of this study demonstrated that monitoring troponin I and creatine kinase isoenzymes in patients with SAP could provide prediction for SACI, affect dynamic assessment of disease severity, and the recovery of patients, as also shown by Luo Y. et al.[25] The results of this study demonstrated that troponin I and creatine kinase isoenzymes possess certain predictive value for SACI. In the event of myocardial injury, both troponin I and creatine kinase isoenzymes may increase, thereby allowing medical staff to monitor the dynamic evolution of myocardial injury markers in patients with SAP.[22] This allows for the implementation of appropriate interventions. Consequently, it is imperative that the status of patients be promptly and systematically assessed. Furthermore, the specificity of these two markers in SACI prediction must be further explored. In addition, the assays of troponin I and creatine kinase isoenzymes must be improved as a priority.[26] Finally, assay results must be closely monitored to accurately screen out high-risk patients in a timely manner. This will enable the achievement of clinical titres that would assist with the early diagnosis, treatment, and rehabilitation of patients with SACI.[27,28]

The SACI Prediction Model

In this study, SACI risk prediction model was constructed based on the new JSS combined with selected high-risk factors. The analysis of the application of the model shows that the present model has a good predictive value. Internalization of the model confirms that the present model has good reproducibility. The results of this study indicate that use of the SACI prediction model is more effective than use of the new JSS alone, providing a more reliable means of identifying patients with SACI. Therefore, medical staff can screen out high-risk patients according to Logit (SACI) score, and transfer patients with SAP with symptoms of SACI to the intensive care unit immediately for close 24-hour monitoring,[29] because close monitoring is particularly important in rapid diagnosis and timely management. Concurrently, targeted treatment and nursing intervention measures should be implemented, which has important practical significance for improving the prognosis of such patients.[29,30]

Significance of Constructing SACI Prediction Model for Nursing Work

During the development of SAP, the influence of various factors, such as bioactive inflammatory factors, can significantly change the ultrastructure of the heart, and clinical manifestations such as myocardial cell oedema, myocardial hypertrophy, and myocardial interstitial collagen deposition may occur,[25,31] thus inducing SACI. At present, although great progress has been made in testing, imaging, treatment, and nursing, the molecular mechanism of SACI is still unclear. The research on SACI is predominantly focused on risk factor analysis or analysis of the risk value of a single factor for SACI, with a lack of relevant studies based on a risk factor specificity and sensitivity prediction model. To the best of our knowledge, there are currently no systematic and standard treatment and nursing programs, or predictive prevention and treatment programs.[26] Therefore, in order to effectively implement predictive nursing, the construction of a prediction model for the risk of myocardial injury in patients with SAP during hospitalisation has become the focus of research, with the combination of this model with independent risk factors to construct a preliminary predictive nursing plan for SACI as the core. To improve the initiative of clinical nurses in accurately evaluating and predicting patients that may develop SACI,[32,33] this SACI prediction model with independent risk factors was constructed to help clinical nurses identify high-risk factors for SACI as early as possible and provide timely intervention. We aimed to improve the predictability and precision of clinical nursing work, explore potential nursing strategies, and save treatment time, so as to achieve the goal of improving the overall health outcome of SACI patients.

Foresight

A survey in China showed that although medical staff can have a good grasp of cardiac rehabilitation knowledge, low education level, low title level and short working hours have more negative attitudes toward the implementation of cardiac rehabilitation.[34] This also suggests that future model updates should pay attention to the characteristics of model users to form a concise, convenient, and universal predictive model for early detection of cardiac injury and cardiac

rehabilitation.[26,35] Existing studies have shown that cardiac injury in rats with pancreatitis can be ameliorated by abdominal puncture drainage or traditional Chinese medicine, but other therapeutic options have not been demonstrated. Based on the factors included in the predictive model constructed in this study, researchers can take targeted therapeutic measures in the future to expand the therapeutic options for SACI.

Limitations

Although this study constructed a prediction model for SACI, it still has limitations. First, this study used the new JSS to construct the model, but did not use common scores such as APACHE II and Ranson score to construct the model, which can be constructed according to different scores and compared with the predictive effect in subsequent studies. Second, only troponin I was selected as the evaluation standard of myocardial injury in this study, and all the indicators for predicting myocardial injury were not compared, which needs to be further included in subsequent studies. Finally, this study was conducted only in the same hospital, and the predictive effect of the modified model needs to be further investigated in different hospitals.

Conclusion

New JSS, leukocytes, troponin I, and creatine kinase isoenzymes are independent risk factors for SACI, and in this study we constructed a prediction model for SACI based on these variables and verified the validity of the model with data. When compared with the use of the new JSS alone, this study found that the predictions of the constructed prediction model had a better predictive effect. Future studies can conduct multi-sample, multi-center studies based on this model to validate the predictive effect of this model.

Abbreviations

JSS, Japanese Severity Score; SAP, Severe Acute Pancreatitis; SACI, Severe Acute Pancreatitis-Associated myocardial Injury; AUC, Area under receiver operating characteristic curve.

Data Sharing Statement

Data used in this study can be requested from the corresponding author with a justification.

Ethics Approval and Informed Consent

This retrospective study was reviewed and approved by the Ethics Committee of Binzhou Medical University (Lun Yan Batch No. 2023-003). The Ethics Committee did not require signed informed consent from patients because all data were collected from an electronic case database and all patients were discharged from the hospital at the time of data collection. The study team adhered to the Declaration of Helsinki and ensured that the study data were not disclosed to third parties.

Acknowledgments

We thank the intensive care unit staff for their help with data collection.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This research was funded by the Natural Science Foundation Project of Shandong Province (ZR2022MH117) and the China Social Welfare Foundation Love Fund (HLCXKT-20230124).

Disclosure

The authors declare no conflict of interest.

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