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ORIGINAL RESEARCH

# The Predictive Value of Liver/Spleen CT Value, BISAP Score Combined with Serum sPD-L1 for Hyperlipidemic Acute Pancreatitis

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**Objective:** To explore the predictive value of liver/spleen CT Value, bedside index for severity in acute pancreatitis (BISAP) score combined with serum soluble programmed death ligand 1 (sPD-L1) within 24 hours of onset for the severity tendency of hyperlipidemic acute pancreatitis (HTG-AP).

**Methods:** A retrospective study was conducted on the clinical data of 102 patients with mild to moderate HTG-AP. These patients were divided into the progression group (24 cases) and the stable group (78 cases) based on whether they progressed to severe HTG-AP. The liver/spleen CT values, BISAP scores, serum sPD-L1 levels and blood urea nitrogen (BUN) levels were compared within 24 hours of onset in the two groups. The COX regression model was used to analyze the influencing factors of the tendency of HTG-AP severity. The predictive efficacy of each indicator was evaluated through the Receiver operating characteristic curves (ROC).

**Results:** The BUN, BISAP score, serum sPD-L1 levels, and the proportion of moderate HTG-AP in the progression group were all higher than those in the stable group, while the liver/spleen CT values were lower than the stable group (all $P<0.05$). Liver/spleen CT values were protective factors (HR=0.245, 95% CI: 0.098–0.613) for the tendency towards severe HTG-AP, while BISAP scores (HR=4.536) and serum sPD-L1 (HR=4.345) were risk factors (all $P<0.05$). The combined prediction of the three indicators for the severity of HTG-AP had an AUC of 0.826, with a sensitivity of 0.89 and a specificity of 0.79, and the efficacy was superior to that of a single indicator (all $P<0.05$).

**Conclusion:** Liver/spleen CT values, BISAP scores and serum sPD-L1 have certain predictive value for the tendency of severe deterioration in patients with HTG-AP within 24 hours of onset. When these three factors are combined, the predictive efficacy is even better.

**Keywords:** acute pancreatitis, hyperlipidemia, fatty liver, BISAP score, soluble programmed death ligand 1, severity

## Introduction

Hypertriglyceridemic acute pancreatitis (HTG-AP) is one of the common acute abdominal conditions in clinical practice. With hypertriglyceridemia as the main cause, the onset of the disease is sudden and the progression is rapid. Patients with HTG-AP often experience self-digestion of the pancreas due to abnormal activation of pancreatic enzymes, resulting in severe abdominal pain, nausea, vomiting, and other symptoms. In severe cases, it can lead to multiple organ failure, with a relatively high mortality rate.[1] It is worth noting that there is a significant difference in the prognosis of HTG-AP patients. Some patients may rapidly progress to a severe condition, while others remain relatively stable Therefore, early identification of the tendency towards severe deterioration and timely intervention are crucial for improving the prognosis.[2] From the perspective of the pathological mechanism, hypertriglyceridemia can induce and aggravate pancreatic damage through multiple pathways. Excessive triglycerides are decomposed into free fatty acids under the action of lipase. These free fatty acids have strong cytotoxicity and can directly damage pancreatic acinar cells and vascular endothelium, triggering local



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inflammatory responses. Meanwhile, the accumulation of free fatty acids can also lead to pancreatic microcirculation disorders, aggravating tissue ischemia and hypoxia and forming an "inflammation-ischemia" vicious cycle, which further exacerbates pancreatic necrosis.[3,4] However, at present, there is a lack of precise early prediction methods in clinical practice, and traditional assessment methods are unable to quickly identify high-risk patients.

In recent years, the value of imaging indicators, inflammatory scores, and immune molecule markers in predicting disease progression has gradually gained attention. Among them, liver/spleen CT values can indirectly indicate the inflammatory status of the pancreas and the entire body by reflecting the degree of liver fat deposition and the degree of inflammation in the spleen.[5] The BISAP score, as a comprehensive assessment tool, can quickly determine the severity of pancreatic inflammation through bedside indicators such as blood urea nitrogen and consciousness status.[6] The soluble programmed death ligand 1 (sPD-L1) activates T cells (especially Th17 cells), releasing pro-inflammatory factors such as IL-6 and IL-17, thereby intensifying the local and systemic inflammatory responses in the pancreas and promoting the progression of the disease to a more severe state. Although sPD-L1 is widely used in cancer immunotherapy, its core function is to participate in the immune response by regulating T cell activity.[7] In HTG-AP, persistent inflammation is the core driving factor for disease progression. The sPD-L1 protein can inhibit the recognition and clearance of inflammatory damage by T cells, thereby exacerbating the local and systemic inflammatory responses in the pancreas.[8] Therefore, the changes in its level are associated with the pathogenesis of the severity of HTG-AP and have the theoretical basis for being used as a predictor. Although these indicators are respectively related to the inflammatory progression and organ damage of HTG-AP at different levels, the predictive efficacy of a single indicator is limited. Liver/spleen CT values only reflect the inflammatory state related to liver fat deposition. The BISAP score focuses on clinical macroscopic assessment but lacks molecular-level information. The sPD-L1 can reflect the degree of immune inflammation, but it is greatly influenced by the individual's underlying immune status.

Given that a single indicator is insufficient to comprehensively capture the complex mechanisms of disease progression, integrating multiple dimensions of indicators for prediction may enhance efficiency. This study aims to explore the predictive value of liver/spleen CT values, BISAP score and serum sPD-L1 for the tendency of HTG-AP to become severe within 24 hours of onset, with the expectation of providing theoretical support for early clinical intervention.

# Materials and Methods

## Clinical Materials

The study was approved by the hospital's medical Ethics Committee and was complies with the Declaration of Helsinki. A retrospective study was conducted on 102 patients with mild to moderate HTG-AP admitted in our hospital from January 2021 to December 2023. Among them, there were 57 males and 45 females, aged 31–59 years, with an average age of 45.51 ± 6.09 years. Inclusion criteria: (1) Patients who met the clinical diagnosis of HTG-AP as follows:[9] accompanied by HTG-AP abdominal pain; serum lipase and/or amylase ≥ three times of the upper limit of normal; imaging changes that met HTG-AP. (2) The patient's triglyceride level was ≥ 11.3 mmol/L, and the chyle like serum triglyceride level was 5.65–11.3 mmol/L. (3) The time from onset to admission of the patient was less than 6 hours. (4) The patient was over 18 years old. (5) The patient's clinical diagnosis and treatment data were complete. Exclusion criteria: (1) Patients with pancreatitis caused by other factors such as alcohol and biliary tract diseases; (2) Patients with severe bleeding tendency; (3) Patients with malignant tumors of the digestive tract; (4) Patients with coagulation dysfunction; (5) Patients with mental illnesses; (6) Pregnant and lactating women; (7) Patients with recurrent HTG-AP. The selection process for the general data was shown in Figure 1.

## Methods

### Clinical Materials

The clinical data of patients were collected within 24 hours of onset, including age, gender, drinking history, history of diabetes, history of hypertension, white blood cell count (WBC), neutrophil count (NEUT), platelet count (PLT), hematocrit (HCT), blood urea nitrogen (BUN).

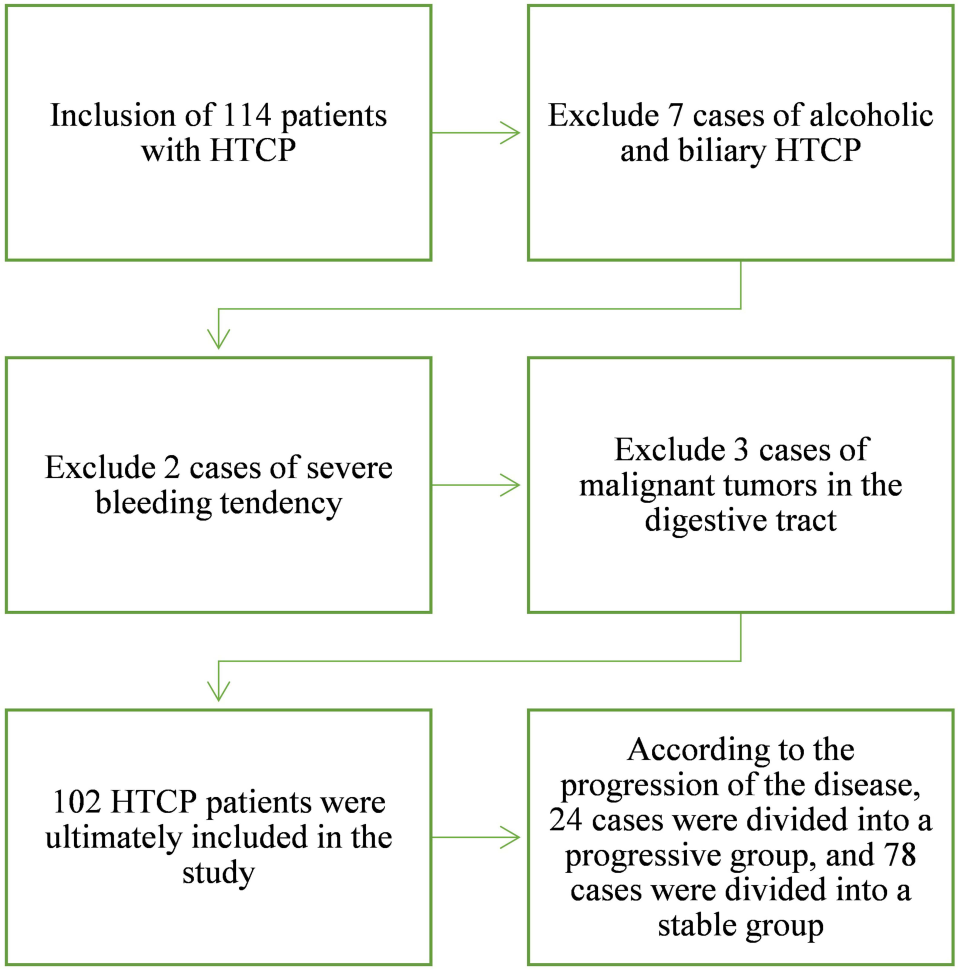


**Figure 1** Diagram of data screening process.

## HTG-AP Severity Assessment

According to the *2012 Atlanta Consensus on Acute Pancreatitis*,[10] the clinical diagnosis of HTG-AP was mild for those without organ failure or systemic or local severe complications, moderate for those with transient organ dysfunction and systemic or local complications lasting ≤ 48 hours, and severe for those with persistent organ dysfunction lasting > 48 hours. According to whether the outcome progressed to severe HTG-AP, patients were divided into a progression group and a stable group. Following the strict grouping approach as recommended in the *2012 Atlanta Consensus on Acute Pancreatitis*:[10] Patients with severe HTG-AP who progressed to "with persistent organ dysfunction for > 48 hours" were included in the progression group (24 cases); Patients with mild HTG-AP (classified as "without organ failure") or moderate HTG-AP (classified as "with transient organ dysfunction for ≤ 48 hours") who remained in the "no organ failure" state after treatment were included in the stable group (78 cases).

## BISAP Scoring Evaluation

The BISAP score includes serum urea nitrogen (BUN), consciousness status, systemic inflammatory response syndrome, age, and pulmonary infiltration on chest X-ray. Scoring criteria:[11] Serum BUN < 25 mg/dL was scored 0 points and serum BUN ≥ 25 mg/dL was scored 1 point. A clear state of consciousness earned 0 points, and changes (such as drowsiness, confusion, blurred consciousness, etc) earned 1 point. The presence of systemic inflammatory response syndrome (such as body temperature ≥ 38°C or ≤ 36°C, heart rate ≥ 90 beats/minute, respiratory rate ≥ 20 beats/minute, white blood cell count ≥ 12,000/μ L or ≤ 4000/μ L) was scored 1 point, and the absence was scored 0 points. Age<60 years old earned 0 points and age ≥ 60 years old earned 1 point. A chest X-ray showing lung infiltration (such as pulmonary effusion) was scored 1 point, and no chest X-ray abnormalities (no lung infiltration or effusion) were scored 0 points. The higher the total score was, the more severe the acute pancreatitis was. The BISAP score was independently assessed by two physicians with over 5 years of experience in emergency medicine (blind to the outcome of whether the patient progressed to a critical condition). If the results of the two scores were inconsistent (the difference was ≥ 1 point), the third doctor with the professional title of deputy chief physician or above would review and obtain the final consistent result. The intraclass correlation coefficient (ICC) was used to evaluate the inter-rater consistency, and the ICC was 0.89 (95% CI: 0.82–0.94).

### Examination of Serum sPD-L1

After admission, 3 mL of peripheral venous blood was collected from the patient. The blood sample was centrifuged at low temperature for 14 minutes under a centrifugal force of 1500 g and a radius of 13.0 cm. Then, the supernatant was collected and detected for the serum sPD-L1 level using ELISA method. The reagent kit was purchased from Shanghai Baililai Biotechnology Co., Ltd. (batch number: BLL103031E).

### Examination of Liver/Spleen CT Values

The patient's abdomen was scanned by CT. Two axial planes were selected, each containing 2 right liver planes, 1 left liver plane, and 1 spleen plane for measurement of the region of interest. An area of 200–300 $mm^2$ was selected to avoid blood vessels and artifacts. Three consecutive tests were performed on all intervals and the median was adopted. The severity evaluation criteria for fatty liver[9] included liver/spleen CT values ≤ 0.5 for severe, 0.5–0.7 for moderate, and >0.7–1.0 for mild. Liver/spleen CT values were measured independently by two radiologists with more than 8 years of experience in abdominal imaging diagnosis (single blind to patient grouping and clinical outcomes), using the same selection criteria for the region of interest. The intra-class correlation coefficient (ICC) was used to evaluate the inter-observer consistency. The ICC of liver/spleen CT value measurement was 0.91 (95% CI: 0.86–0.95), and the consistency was good. If the difference between the two measurements was more than 0.1, the measurement was repeated by the third chief physician radiologist, and the result was taken as the final value.

## Statistical Analysis

SPSS.24 was used to for data analysis. The measurement data was expressed as mean ± standard deviation ($\bar{x} \pm s$) and compared using *t*-test. Enumeration data were represented as [cases (%)] and compared using $\chi^2$ test. The COX regression model was used to estimate the risk ratio and 95% confidence interval (CI) of HTG-AP severity tendency risk. Receiver Operating Characteristic (ROC) curves were drawn to evaluate the predictive value of liver/spleen CT values, BISAP scores, and serum sPD-L1 for the progression of HTG-AP severity. The Bonferroni method was used to correct for multiple comparisons to control the risk of false positive results. $P<0.05$ indicated a statistically significant difference.

# Results

## Comparison of Clinical Data Between Two Groups

Among 102 patients with HTG-AP, 24 cases progressed to severe HTG-AP (the progression group), accounting for 23.53%. 78 patients did not progress (the stable group). There was no significant difference in age, gender, drinking history, diabetes, hypertension and blood routine indexes (WBC, NEUT, PLT and HCT) between the two groups ($P>0.05$). The BUN, BISAP score, serum sPD-L1 level and the proportion of moderate HTG-AP in the progression group were higher than those in the stable group, and the Liver/spleen CT values were lower than that in the stable group ($P<0.05$, Table 1).

## Analysis of the Influence of Liver/Spleen CT Values, BISAP Scores, and Serum sPD-L1 on the Tendency Towards Severe HTG-AP

The development of HTG-AP as severe was taken as the dependent variable (yes=1, no=0), and the liver/spleen CT value, BISAP score, and serum sPD-L1 were taken as independent variables. The assigned values were shown in Table 2. Multivariate Logistic regression model was used to analyze the influence of each index on the tendency of HTG-AP to

**Table 1** Comparison of Clinical Data

| Factors | The Progressive Group (n=24) | The Stable Group (n=78) | $\chi^2$/t | *P* |
|---|---|---|---|---|
| Age ($\bar{x} \pm s$, year) | 45.62±6.09 | 47.58±5.92 | 1.409 | 0.162 |
| Gender (cases/%) | | | 0.557 | 0.454 |
| Male | 15 (62.50) | 42 (53.85) | | |
| Female | 9 (37.50) | 36 (46.15) | | |
| Drinking history (cases/%) | | | 1.255 | 0.263 |
| Yes | 12 (50.00) | 29 (37.18) | | |
| No | 12 (50.00) | 49 (62.82) | | |
| Combined diabetes (cases/%) | | | 1.569 | 0.210 |
| Yes | 6 (25.00) | 11 (14.10) | | |
| No | 18 (75.00) | 67 (85.90) | | |
| Combined hypertension (cases/%) | | | 0.258 | 0.612 |
| Yes | 3 (12.50) | 7 (8.97) | | |
| No | 21 (87.50) | 71 (91.03) | | |
| Blood routine | | | | |
| WBC ($\bar{x} \pm s$, $\times 10^9$/L) | 15.42±3.05 | 13.97±3.62 | 1.776 | 0.079 |
| NEUT ($\bar{x} \pm s$, $\times 10^9$/L) | 12.91±3.83 | 11.75±3.41 | 1.415 | 0.160 |
| PLT ($\bar{x} \pm s$, $\times 10^9$/L) | 259.38±80.05 | 252.72±69.31 | 0.397 | 0.692 |
| HCT ($\bar{x} \pm s$) | 44.15±4.61 | 43.52±5.92 | 0.478 | 0.634 |
| BUN ($\bar{x} \pm s$, mg/dL) | 26.41±2.27 | 22.08±3.14 | 6.261 | 0.001 |
| Liver/spleen CT values ($\bar{x} \pm s$) | 0.67±0.25 | 0.81±0.29 | 2.132 | 0.035 |
| BISAP scoring ($\bar{x} \pm s$, score) | 2.31±0.45 | 1.42±0.37 | 9.780 | 0.001 |
| Serum sPD-L1 ($\bar{x} \pm s$, ng/mL) | 109.41±10.61 | 102.39±11.25 | 2.708 | 0.009 |
| Severity (cases /%) | | | 10.743 | 0.001 |
| Light | 8 (33.33) | 55 (70.51) | | |
| Moderate | 16 (66.67) | 23 (29.49) | | |

**Table 2** Variable Assignment

| Factors | Variable | Assignment |
|---|---|---|
| Severity tendency of HTG-AP | Y | Yes=1, no=0 |
| Liver/spleen CT value | X1 | Measured values |
| BISAP scores | X2 | Measured values |
| Serum sPD-L1 | X3 | Measured values |

become severe. The results showed that liver/spleen CT values (HR=0.245, 95% CI: 0.098–0.613) were protective factors ($P$<0.05) for the tendency towards severe HTG-AP. BISAP scores (HR=4.536, 95% CI: 1.811–11.361) and serum sPD-L1 (HR=4.345, 95% CI: 1.735–10.883) were risk factors ($P$<0.05, Table 3 and Figure 2).

**Table 3** Analysis of the Influence of Liver/Spleen CT Values, BISAP Scores, and Serum sPD-L1 on the Tendency Towards Severe HTG-AP

| Factors | Parameter Estimation | Standard Error | $\chi^2$ | *P* | *OR* | 95% CI |
|---|---|---|---|---|---|---|
| Liver/spleen CT value | −1.382 | 0.671 | 4.286 | 0.038 | 0.251 | 0.067–0.629 |
| BISAP scores | 1.536 | 0.628 | 5.941 | 0.015 | 4.643 | 1.328–11.472 |
| Serum sPD-L1 | 1.492 | 0.645 | 5.317 | 0.021 | 4.410 | 1.286–10.975 |

**Notes**: In this study, the sample size of the progression group was small (n=24), accounting for 23.53% of the total sample size, which may limit the statistical power of the multivariate model and have the potential risk of overfitting. The results need to be interpreted with caution in combination with clinical practice.

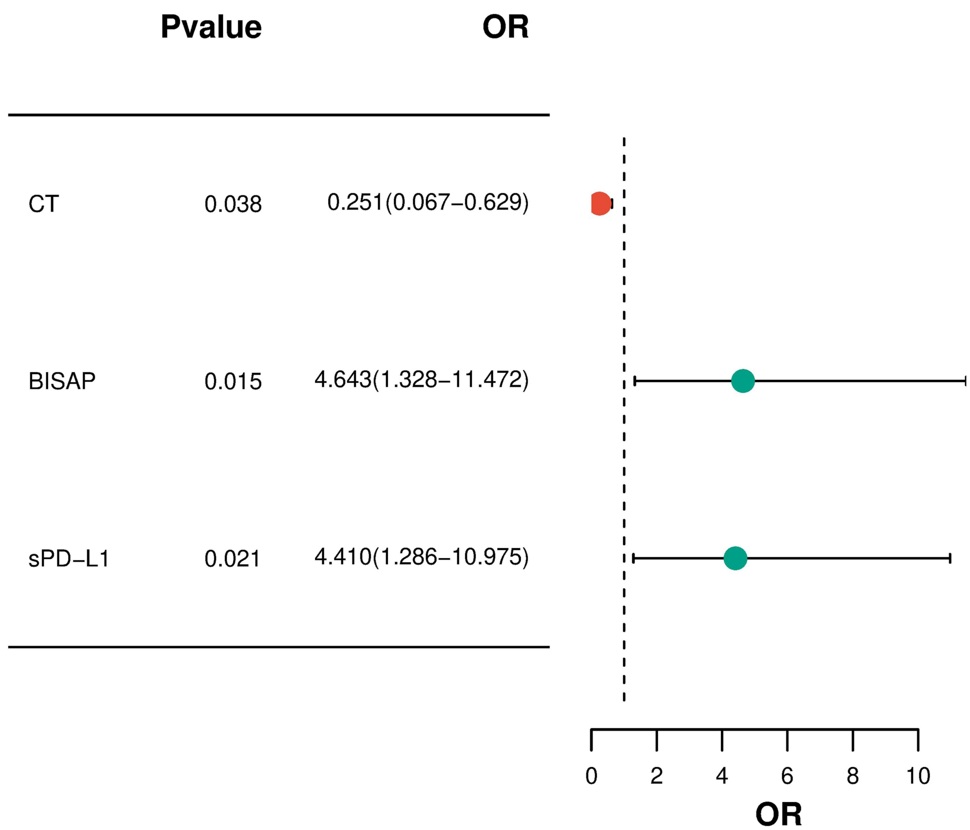


**Figure 2** Forest plot of multivariate Logistic regression model analysis.

## Analysis of the Predictive Value of Liver/Spleen CT Values, BISAP Scores, and Serum sPD-L1 for the Severity Tendency of HTG-AP

After Bonferroni correction for multiple comparisons, the efficacy of liver/spleen CT values, BISAP score, and serum sPD-L1 alone and in combination in predicting the severity of HTG-AP was as following: liver/spleen CT values: the cut-off value was 0.74, the sensitivity was 0.78, the specificity was 0.73, and the AUC was 0.716 (95% CI: 0.605–0.814) ($P<0.05$); BISAP score: the Cut-off value was 1.86, the sensitivity was 0.71, the specificity was 0.74, and the AUC was 0.732 (95% CI: 0.639–0.837) ($P<0.05$); serum sPD-L1: the Cut-off value was 105.53 ng/mL, the sensitivity was 0.76, the specificity was 0.78, and the AUC was 0.751 (95% CI: 0.683–0.841) ($P<0.05$). Combination of the three: the sensitivity was 0.89, the specificity was 0.79, and the AUC was 0.826 (95% CI: 0.731–0.915) ($P<0.05$). The predictive efficacy of single index was lower than that of the combination of the three (all $P<0.05$, corrected for multiple comparisons). (Table 4 and Figure 3).

**Table 4** Analysis of the Predictive Value of Liver/Spleen CT Values, BISAP Scores, and Serum sPD-L1 for the Severity Tendency of HTG-AP

| Indicators | Cut-Off | Sensitivity | Specificity | *P* | AUC | 95% CI | |
|---|---|---|---|---|---|---|---|
| | | | | | | Upper Limit | Lower Limit |
| Liver/spleen CT value | 0.74 | 0.78 | 0.73 | 0.001 | 0.716 | 0.605 | 0.814 |
| BISAP scores | 1.86 | 0.71 | 0.74 | 0.001 | 0.732 | 0.639 | 0.837 |
| Serum sPD-L1 | 105.53 ng/mL | 0.76 | 0.78 | 0.001 | 0.751 | 0.683 | 0.841 |
| Combined detection | - | 0.89 | 0.79 | 0.001 | 0.826 | 0.731 | 0.915 |

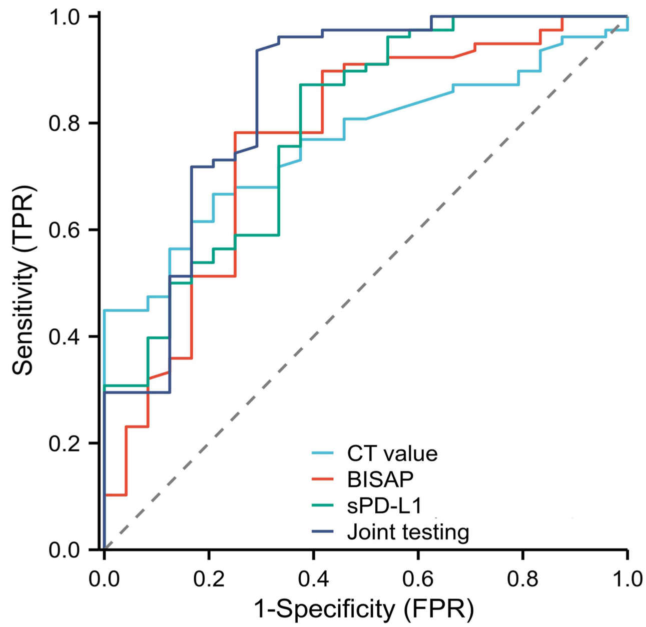


**Figure 3** ROC curves of liver/spleen CT values, BISAP scores, and serum sPD-L1 predicting the severity tendency of HTG-AP.

# Discussion

In recent years, with the increasing proportion of people with hyperlipidemia, the incidence of HTG-AP has been on the rise year by year.[12] Research has shown that the onset of HTG-AP is related to the action of triglyceride metabolites - free fatty acids - on pancreatic cells. Abnormal activation of pancreatic enzymes, pancreatic microcirculation disorders, calcium overload and other physiological and pathological reactions lead to secondary persistent inflammatory infiltration and severe abdominal pain.[13–15] At present, the treatment management strategies for HTG-AP in clinical practice are constantly improving. The implementation of symptomatic treatment plans such as anti-infection, nutritional support, lipid-lowering, and analgesia has effectively reduced the mortality rate of HTG-AP patients. However, the acute progression and variable condition of HTG-AP make it difficult for some patients to achieve the expected clinical treatment goals in disease control, resulting in poor prognosis.[16,17] In current research, indicators for efficiently assessing the severity and progression risk of HTG-AP include comprehensive evaluation criteria such as Acute Physiology and Chronic Health Status Score II,[18] Modified CT Severity Index,[19] and Ranson Score.[20] The above indicators can effectively evaluate the prognostic risk of HTG-AP, but there are shortcomings such as poor timeliness and cumbersome operation, which have significant limitations in clinical application. Therefore, screening for simple and feasible high-sensitivity biological factors to reasonably predict the risk of severe progression of HTG-AP has become a key area that clinical practitioners urgently need to explore.

In this study, 24 out of 102 patients with HTG-AP progressed to severe HTG-AP, accounting for 23.53%, indicating that patients with mild to moderate HTG-AP have a higher probability of progressing to severe HTG-AP. Therefore, improving the monitoring and evaluation of HTG-AP patients in the early stages of onset remains a key focus of clinical research in clinical practice. In this study, compared to the stable group, the advanced group had higher BISAP scores, serum sPD-L1, and lower liver/spleen CT values. The result indicated that changes in BISAP scores, serum sPD-L1, and liver/spleen CT values might be related to the progression of HTG-AP patients. Further regression analysis of risk factors confirmed that BISAP score, serum sPD-L1, and liver/spleen CT values were influencing factors for the tendency towards severe HTG-AP. It could be seen that the decrease in liver/spleen CT values and the increase in BISAP score and serum sPD-L1 were closely related to the tendency towards severe HTG-AP.

HTG-AP patients are accompanied by abnormal elevation of triglycerides and other lipids. The liver is an important organ for storing and decomposing fat, and excessive accumulation of lipids such as triglycerides can lead to the occurrence and aggravation of fatty liver.[21,22] Research has shown that HTG-AP patients with combined fatty liver have

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a higher risk of multiple organ failure and death, and a poorer prognosis.[23,24] The decrease in liver/spleen CT values of HTG-AP patients in the progression group in this study also indicated the above viewpoint. Fatty liver is a chronic inflammatory disease of the liver. Research has shown that fatty liver can promote the synthesis and release of various chronic inflammatory mediators such as heparinase and interleukin, and promote the progression of liver inflammation.[25] Other scholars have found that the inhibitory effect of fatty liver on trypsin can exacerbate the inflammatory response of pancreatitis.[26] Meanwhile, as a lipid metabolism organ, the accumulation process of lipids such as triglycerides in the liver itself is not conducive to the symptom relief of HTG-AP. Therefore, the decrease in liver/spleen CT values in patients with HTG-AP is one of the biological signals indicating a tendency towards severe HTG-AP.

Persistent inflammatory response runs through the occurrence and development process of HTG-AP.[27] Research has shown that free fatty acids can promote the release and chemotaxis of local inflammatory factors and activate inflammatory immune factors such as neutrophils and T lymphocytes through pathological pathways such as activating protease C and damaging pancreatic acinar cells.[28] sPD-L1 is a soluble immune checkpoint molecule, which can inhibit the activity of T cells and promote severe pancreatitis.[29] In this study, the serum sPD-L1 expression level was higher in HTG-AP patients in the progression group, indicating that it may be a potential biological factor for the severe development of HTG-AP. The study has found that sPD-L1 is also abnormally increased in hyperlipidemic tissue chronic inflammation, which is related to the systemic inflammatory response caused by HTG-AP.[30] Lipids and inflammatory factors in HTG-AP may affect the expression of sPD-L1, which can be monitored to assess the severity and progression risk of HTG-AP.

The BISAP score is a comprehensive assessment system that evaluates the severity of HTG-AP through multiple dimensions such as urea nitrogen, level of consciousness, and systemic inflammatory response syndrome. The increase in BISAP score indicates an increased risk of HTG-AP progression.[31] Therefore, in subsequent clinical practice, liver/spleen CT values, sPD-L1, and BISAP scoring indicators can be referred to for risk assessment of early HTG-AP patients within 24 hours of onset, providing support for clinical treatment decisions. The ROC analysis in this study showed that liver/spleen CT values, BISAP scores, and serum sPD-L1 alone or in combination had high sensitivity and specificity in predicting the severity tendency of HTG-AP. This suggested that all of the above indicators could be used for predicting the severity tendency of HTG-AP patients within 24 hours of onset. However, as a single center research, this study still had limitations. Further studies with large sample and multicenter are needed to explore the relationship between liver/spleen CT values, BISAP scores, and serum sPD-L1 and the progression of HTG-AP, providing theoretical support for prognostic management.

## Limitation

This study was a single-center retrospective study. The small number of severe cases (n = 24) relative to total (n = 102) may reduce the statistical power of multivariate Logistic regression analysis, increase the risk of type II error, and lead to limited stability and reliability of regression coefficients. Representative CT images were not provided to visually show the association between liver/spleen CT values and the tendency of HTG-AP to become severe, and there was a lack of imaging evidence to support it. There were some limitations in the statistical analysis method, which may affect the accuracy of the results. This prediction model has not been validated by an external cohort, and its generalization ability and clinical applicability need to be further confirmed. In the future, multi-center, large-sample prospective studies are needed to supplement imaging data, optimize statistical methods, and conduct external cohort validation to further verify the predictive value of each indicator.

## Conclusion

In general, liver/spleen CT values, BISAP scores, and serum sPD-L1 have certain predictive value for the severe tendency of HTG-AP patients within 24 hours of onset, and the combined use of the three has better predictive efficacy, which provides a reliable basis for early identification of high-risk patients and formulation of intervention strategies.

## Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

## Ethics Approval and Consent to Participate

This study was approved by The Ethics Committee of the First Affiliated Hospital of Guangxi Medical University, and was complies with the Declaration of Helsinki. Informed consent was obtained from participants for the participation in the study and all methods were carried out in accordance with relevant guidelines and regulations.

## Consent to Participate

The patients participating in the study all agree to publish the research results.

## Disclosure

The authors declare that they have no competing interests.

## References


1. Kiss L, Fur G, Pisipati S, et al. Mechanisms linking hypertriglyceridemia to acute pancreatitis. *Acta Physiol*. 2023;237(3):e13916. doi:10.1111/apha.13916
2. Chen EX, Tu YS, She ZF, et al. The clinical characteristic of alcohol-hyperlipidemia etiologically complex type of acute pancreatitis. *Eur Rev Med Pharmacol Sci*. 2022;26(19):7212–7218. doi:10.26355/eurrev_202210_29913
3. Qiu M, Zhou X, Zippi M, et al. Comprehensive review on the pathogenesis of hypertriglyceridaemia-associated acute pancreatitis. *Ann Med*. 2023;55(2):2265939. doi:10.1080/07853890.2023.2265939
4. Mathai MJ, Reddy MV, Shetty V. Analysis of the accuracy of the modified CT severity index in predicting clinical outcomes in acute pancreatitis: a cross-sectional study. *Cureus*. 2024;16(3):e56123. doi:10.7759/cureus.56123
5. Gan M, He X, Zheng X. FDG PET/CT imaging of liver and spleen histiocytic sarcoma. *Clin Nucl Med*. 2024;49(3):272–273. doi:10.1097/RLU.0000000000005043
6. Gupta D, Mandal NS, Arora JK, et al. Comparative evaluation of harmless acute pancreatitis score (HAPS) and bedside index of severity in acute pancreatitis (BISAP) scoring system in the stratification of prognosis in acute pancreatitis. *Cureus*. 2022;14(12):e32540. doi:10.7759/cureus.32540
7. Yu X, Pan Y, Fei Q, et al. Serum soluble PD-1 plays a role in predicting infection complications in patients with acute pancreatitis. *Immun Inflamm Dis*. 2021;9(1):310–318. doi:10.1002/iid3.394
8. Serratì S, Di Fonte R, Porcelli L, et al. Circulating extracellular vesicles are monitoring biomarkers of anti-PD1 response and enhancer of tumor progression and immunosuppression in metastatic melanoma. *J Exp Clin Cancer Res*. 2023;42(1):251. doi:10.1186/s13046-023-02808-9
9. Yokoe M, Takada T, Mayumi T, et al. Japanese guidelines for the management of acute pancreatitis: japanese Guidelines 2015. *J Hepatobiliary Pancreat Sci*. 2015;22(6):405–432. doi:10.1002/jhbp.259
10. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013;62(1):102–111. doi:10.1136/gutjnl-2012-302779
11. Kim DH, Lukens FJ, Ko D, et al. Modified Bedside Index for severity in acute pancreatitis (BISAP) score validation in the national inpatient sample database. *Adv Med Sci*. 2023;68(2):208–212. doi:10.1016/j.advms.2023.05.004
12. Gupta M, Liti B, Barrett C, et al. Prevention and management of hypertriglyceridemia-induced acute pancreatitis during pregnancy: a systematic review. *Am J Med*. 2022;135(6):709–714. doi:10.1016/j.amjmed.2021.12.006
13. Cao L, Chen Y, Liu S, et al. Early plasmapheresis among patients with hypertriglyceridemia-associated acute pancreatitis. *JAMA Network Open*. 2023;6(6):e2320802. doi:10.1001/jamanetworkopen.2023.20802
14. Lin XY, Zeng Y, Zhang ZC, et al. Incidence and clinical characteristics of hypertriglyceridemic acute pancreatitis: a retrospective single-center study. *World J Gastroenterol*. 2022;28(29):3946–3959. doi:10.3748/wjg.v28.i29.3946
15. Alexander VJ, Karwatowska-Prokopczuk E, Prohaska TA, et al. Volanesorsen to prevent acute pancreatitis in hypertriglyceridemia. *N Engl J Med*. 2024;390(5):476–477. doi:10.1056/NEJMc2306575
16. Gligorijevic N, Stefanovic-Racic M, Kershaw EE. Medical management of hypertriglyceridemia in pancreatitis. *Curr Opin Gastroenterol*. 2023;39(5):421–427. doi:10.1097/MOG.0000000000000956
17. Aguilar-Salinas CA, Gomez-Diaz RA, Corral P. New therapies for primary hyperlipidemia. *J Clin Endocrinol Metab*. 2022;107(5):1216–1224. doi:10.1210/clinem/dgab876
18. Capurso G, Ponz DLPR, Lauri G, et al. Clinical usefulness of scoring systems to predict severe acute pancreatitis: a systematic review and meta-analysis with pre and post-test probability assessment. *United Eur Gastroenterol J*. 2023;11(9):825–836. doi:10.1002/ueg2.12464
19. Xu WH, Li XH, Yu NJ, et al. Comparison of the imaging and clinical characteristics between initial and recurrent alcoholic acute pancreatitis: a retrospective cross-sectional study. *Am J Drug Alcohol Abuse*. 2023;49(4):431–439. doi:10.1080/00952990.2023.2211221
20. Wang X, Zhu L, Tao K, et al. Red cell distribution width to serum albumin ratio as an early prognostic marker for severe acute pancreatitis: a retrospective study. *Arab J Gastroenterol*. 2022;23(3):206–209.
21. Gouni-Berthold I, Schwarz J, Berthold HK. Updates in drug treatment of severe hypertriglyceridemia. *Curr Atheroscler Rep*. 2023;25(10):701–709. doi:10.1007/s11883-023-01140-z
22. Stroes E, Alexander VJ, Karwatowska-Prokopczuk E, et al. Olezarsen, acute pancreatitis, and familial chylomicronemia syndrome. *N Engl J Med*. 2024;390(19):1781–1792. doi:10.1056/NEJMoa2400201
23. Kirkpatrick CF, Sikand G, Petersen KS, et al. Nutrition interventions for adults with dyslipidemia: a clinical perspective from the national lipid association. *J Clin Lipidol*. 2023;17(4):428–451. doi:10.1016/j.jacl.2023.05.099
24. Deshotels MR, Hadley TD, Roth M, et al. Genetic testing for hypertriglyceridemia in academic lipid clinics: implications for precision medicine-brief report. *Arterioscler Thromb Vasc Biol*. 2022;42(12):1461–1467. doi:10.1161/ATVBAHA.122.318445

25. Gubensek J. The role of apheresis and insulin therapy in hypertriglyceridemic acute pancreatitis-a concise review. *BMC Gastroenterol*. 2023;23(1):341. doi:10.1186/s12876-023-02957-3
26. Yang G, Zhang X. Trimethylamine N-oxide promotes hyperlipidemia acute pancreatitis via inflammatory response. *Can J Physiol Pharmacol*. 2022;100(1):61–67. doi:10.1139/cjpp-2021-0421
27. Hajibandeh S, Jurdon R, Heaton E, et al. The risk of recurrent pancreatitis after first episode of acute pancreatitis in relation to etiology and severity of disease: a systematic review, meta-analysis and meta-regression analysis. *J Gastroenterol Hepatol*. 2023;38(10):1718–1733. doi:10.1111/jgh.16264
28. Wang L, Xu T, Wang R, et al. Hypertriglyceridemia acute pancreatitis: animal experiment research. *Dig Dis Sci*. 2022;67(3):761–772. doi:10.1007/s10620-021-06928-0
29. Mucileanu A, Chira R, Mircea PA. PD-1/PD-L1 expression in pancreatic cancer and its implication in novel therapies. *Med Pharm Rep*. 2021;94(4):402–410. doi:10.15386/mpr-2116
30. Sun S, Chen Y, Liu Z, et al. Serum-soluble PD-L1 may be a potential diagnostic biomarker in sepsis. *Scand J Immunol*. 2021;94(1):e13049. doi:10.1111/sji.13049
31. Karabuga B, Gemcioglu E, Konca KE, et al. Comparison of the predictive values of CRP, CRP/albumin, RDW, neutrophil/lymphocyte, and platelet/lymphocyte levels in determining the severity of acute pancreatitis in patients with acute pancreatitis according to the BISAP score. *Bratisl Lek Listy*. 2022;123(2):129–135. doi:10.4149/BLL_2022_020