

Research Progress on the Value of Inflammatory Markers Combined with the TyG Index in the Early Prediction of the Severity of Acute Pancreatitis

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Abstract

Introduction: Acute pancreatitis (AP) is a common acute gastrointestinal emergency with an increasing global incidence. Some patients may rapidly progress to severe acute pancreatitis (SAP), complicated by pancreatic necrosis, multiple organ dysfunction syndrome (MODS), and other conditions, leading to a significant increase in mortality. Early and accurate assessment of AP severity is crucial for formulating individualized treatment plans, reducing complications, and improving prognosis. However, traditional prediction methods have inherent limitations: the Ranson score requires 48-hour dynamic monitoring, which restricts early assessment; the APACHE II score involves complex indicators; the BISAP score has the risk of missed diagnosis of local severe lesions; and CTSI/MCTSI rely on imaging examinations with delayed imaging changes. None of these can meet the needs of early and rapid assessment. Based on the pathological basis of the “inflammation-metabolism” interaction in the pathogenesis of AP (inflammation exacerbates insulin resistance, while metabolic disorders further promote inflammation), the combined application of inflammatory markers and the triglyceride-glucose (TyG) index provides a new approach for predicting AP severity. **Methods:** This study conducted a literature review by searching Chinese and English literatures published from 2018 to 2025 in databases such as China National Knowledge Infrastructure (CNKI), Wanfang Data Knowledge Service Platform, and PubMed. A total of 19 valid studies were included, and the relevant research progress was systematically summarized and analyzed. **Results:** Inflammatory markers can effectively reflect the body's inflammatory state. For

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example, the area under the curve (AUC) of C-reactive protein (CRP) at 24 hours after admission for predicting severe acute pancreatitis (SAP) is 0.787; the systemic immune-inflammation index (SII) has an AUC of 0.809 - 0.920 for predicting SAP, which is superior to single indicators such as neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR). As a marker of insulin resistance, the TyG index is significantly higher in SAP patients than in mild AP patients (mean difference = 0.61, 95% confidence interval: 0.46 - 0.76) and is an independent predictor of SAP (odds ratio [OR] = 1.835 - 7.14). Importantly, the combined models integrating inflammatory markers and the TyG index perform better. For instance, the AUC of the CRP + NLR + TyG model for predicting SAP is 0.882 (sensitivity = 77.2%, specificity = 88.5%); in hyperlipidemic AP (HLAP), the model combining CT findings with blood biomarkers (such as calcium, CRP) has an AUC of 0.957 in the training group and 0.930 in the validation group, which is superior to traditional methods like the Ranson score. These combined models use routine clinical indicators, featuring low cost and fast turnaround, and have good potential for clinical translation, such as integration into electronic medical records for emergency triage. **Discussion:** The combined application of inflammatory markers and the TyG index has important value in the early prediction of AP severity, making up for the shortcomings of traditional methods. However, most existing studies are single-center retrospective studies with limited sample sizes, and there is a lack of data on special populations (such as pregnant women and elderly patients). Future research should focus on multicenter prospective studies to verify external validity, optimize models for specific subtypes (such as HLAP) and populations, and explore their long-term prognostic value to accelerate clinical application.

Keywords

Acute Pancreatitis, Inflammatory Markers, TyG Index, Severity, Early Prediction

1. Introduction

Acute pancreatitis (AP) is a common acute gastrointestinal emergency in clinical practice, with an increasing incidence year by year. Its condition is complex and variable. Mild cases may present as self-limiting inflammation, while severe cases can progress to severe acute pancreatitis (SAP), accompanied by complications such as persistent organ failure, pancreatic necrosis, and infection, with a relatively high mortality rate [1]. Early and accurate identification of the severity of AP is crucial for optimizing treatment strategies and improving patient prognosis. In clinical practice, scholars have developed various scoring systems for assessing the severity of AP, such as Acute Physiology and Chronic Health Evaluation (APACHE) II, Bedside Index of Severity in Acute Pancreatitis (BISAP), Ranson's score, and modified Computed Tomography Severity Index (modified CTSI)

[1] [2]. Among them, modified CTSI shows high accuracy in predicting SAP, pancreatic necrosis, organ failure, and ICU admission [1] [2], while CTSI is also superior to APACHE II in evaluating persistent organ failure, death, and the need for intervention for necrosis [2]. However, these scoring systems based on physiological indicators or imaging have certain limitations: although APACHE II has high sensitivity, its calculation is complex; BISAP is easy to operate but has the problem of low sensitivity [3] [4]; CT-related indices, although accurate, rely on imaging examinations, which are not suitable for early frequent evaluation, especially in resource-limited areas where they are difficult to be widely applied.

With the in-depth study of the pathological mechanism of AP, the core role of inflammatory response in disease progression has gradually become clear, so inflammatory markers have become a research hotspot in predicting the severity of AP. At present, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), red cell distribution width (RDW), C-reactive protein (CRP), procalcitonin (PCT), and interleukin-6 (IL-6) have all been confirmed to be related to the severity of AP [5]-[9]. In addition, systemic immune-inflammation index (SII) and systemic inflammation response index (SIRI), by integrating multiple inflammation-related cell counts, show good accuracy in predicting SAP and acute kidney injury (AKI) [10]-[12]. Although a single inflammatory marker has certain value in evaluating the severity of AP, its specificity and stability still need to be improved due to individual differences and dynamic changes in inflammation [13].

In recent years, the triglyceride-glucose (TyG) index, as a surrogate indicator of insulin resistance, has gradually been applied to predict the severity of AP due to its close association with metabolic disorders and inflammatory responses. The calculation formula of TyG index is \ln [fasting triglycerides (mg/dL) \times fasting blood glucose (mg/dL)/2], and its increase can reflect metabolic imbalance and potential inflammatory status in the body [14] [15]. A number of studies have shown that the TyG index is significantly increased in SAP patients and is an independent risk factor for predicting SAP [14] [15]; at the same time, the TyG index is closely related to adverse prognoses such as ICU admission, death, and AKI, and its combined application with other indicators (such as venous excess ultrasound score, SII, etc.) can further improve the prediction efficiency [16]-[18].

In view of the limitations of a single inflammatory marker or TyG index in the early prediction of AP severity, exploring the value of their combined application has become an important direction of research in recent years. Existing studies have shown that the combination of inflammatory markers (such as CRP, NLR) and TyG index can significantly improve the accuracy of SAP prediction [19], providing a new idea for early clinical identification of high-risk patients. This article aims to systematically review the research progress of the combination of inflammatory markers and TyG index in the early prediction of AP severity, summarize their clinical application value and existing problems, so as to provide a reference for optimizing AP risk stratification strategies.

2. Methods

2.1. Literature Search Strategy

This study employed a literature review approach. Relevant Chinese and English literature published between 2018 and 2025 was retrieved from databases such as China National Knowledge Infrastructure (CNKI), Wanfang Data Knowledge Service Platform, and PubMed. Full Boolean search terms, Chinese: (急性胰腺炎) AND (炎症指标 OR 炎症标志物) AND (TyG 指数 OR 甘油三酯-葡萄糖指数) AND (严重程度预测 OR 早期预测 OR 联合预测价值); English (“acute pancreatitis”) AND (“inflammatory markers” OR “inflammatory indicators”) AND (“TyG index” OR “triglyceride-glucose index”) AND (“severity prediction” OR “early prediction” OR “combined predictive value”).

2.2. Study Selection and Quality Assessment

Initial screening removed duplicates, followed by title/abstract review and full-text evaluation (**Figure 1**). Two independent reviewers assessed study quality using the Newcastle-Ottawa Scale (NOS) for cohort studies (score ≥ 6 deemed high quality). Discrepancies were resolved by consensus.

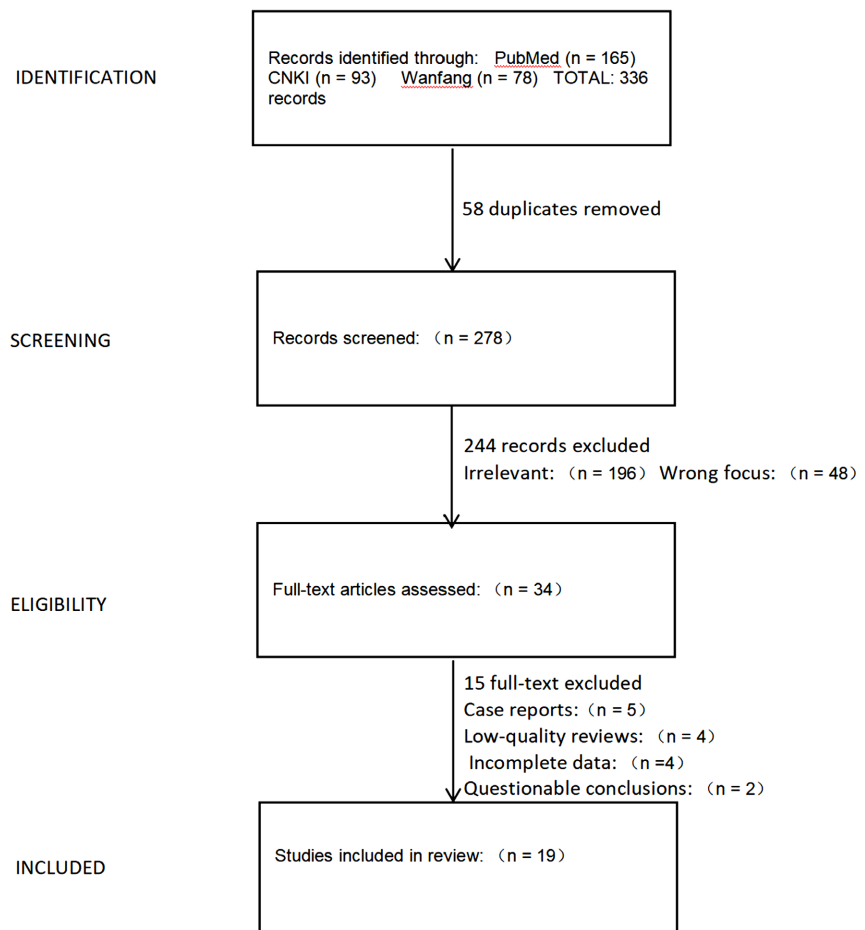


Figure 1. PRISMA flow diagram of literature selection.

The inclusion criteria were as follows: studies focusing on the prediction of acute pancreatitis (AP) severity; the association between inflammatory indicators, or the TyG index, and AP; and the construction of prediction models combining inflammatory indicators with the TyG index. The exclusion criteria included case reports, review articles (except for high-quality ones), studies with incomplete data, and those with questionable conclusions.

Ultimately, more than 19 valid pieces of literature were included. By summarizing, analyzing, and synthesizing the content of this body of literature, this study sorts out the research progress in the early prediction of AP severity using a combination of inflammatory indicators and the TyG index.

2.3. Data Synthesis

Data from eligible studies were extracted into a standardized table, including title, year, design, biomarkers, AUC, and cut-off values.

3. Results

I) Application and Limitations of Traditional Methods for Predicting the Severity of Acute Pancreatitis

Common traditional methods for predicting the severity of acute pancreatitis (AP) include the Ranson score, APACHE II score, BISAP score, and CTSI (including the modified MCTSI). The specific performance data are as follows:

Ranson score: Evaluates 11 indicators at admission and 48 hours after admission. It demonstrates good accuracy in predicting severe acute pancreatitis (SAP). The higher the score, the greater the disease severity; a score ≥ 3 indicates an elevated risk of complications and mortality. A limitation is that the Ranson score requires 48-hour dynamic monitoring, which restricts its application in early assessment.

APACHE II score: Integrates acute physiological indicators, age, and chronic health status. This scoring system is suitable for dynamic assessment of ICU patients. The higher the score, the higher the in-hospital mortality rate. A limitation is that the scoring system involves complex indicators, making dynamic updates challenging in clinical practice.

BISAP score: Based on 5 indicators (such as blood urea nitrogen, altered mental status, etc.), it is simple to calculate and suitable for rapid triage. A limitation is that the simplified nature of its indicators may result in missed detection of local severe lesions.

CTSI and MCTSI: Assess pancreatic inflammation and necrosis through contrast-enhanced CT. CTSI shows good accuracy in predicting persistent organ failure, death, and infected necrosis. Among these, the modified CTSI outperforms the unmodified version in predicting SAP. Limitations of this method include reliance on imaging equipment, delayed imaging changes relative to clinical symptoms, and subjective assessment.

II) Value of Inflammatory Markers in Predicting the Severity of AP

1) Basic Inflammatory Markers

CRP: Its levels correlate with the severity of AP. The AUC of CRP measured at 24 hours after admission (CRP2) for predicting SAP is 0.787. It serves as an independent predictor of SAP (OR = 1.011), and when combined with the neutrophil-to-lymphocyte ratio at 48 hours (NLR48h) and CRP at 48 hours after admission (CRP48h), the AUC for predicting SAP reaches 0.89.

White blood cells and neutrophils: Their counts are elevated in SAP patients and are associated with pancreatic necrosis and multiple organ dysfunction syndrome (MODS). Neutrophil extracellular traps (NETs) and intracellular signals (such as phosphorylated STAT3 [pSTAT3]) are linked to disease severity.

Monocytes: Activated monocytes release bioactive substances that affect organ function, and their signaling pathways (such as phosphorylated NF- κ B [pNF- κ B]) can predict persistent organ failure.

2) New Systemic Inflammatory Indicators

Systemic immune-inflammation index (SII): Calculated as (platelets \times neutrophils)/lymphocytes, this index is significantly elevated in SAP patients. Its AUC for predicting SAP is 0.809, and for predicting acute kidney injury (AKI) is 0.820. In another study, the AUC of SII for predicting SAP reaches 0.920, which is superior to that of NLR and PLR.

Neutrophil-to-lymphocyte ratio (NLR): This ratio is elevated in SAP patients (AUC = 0.722 for predicting SAP, AUC = 0.851 for predicting mortality). It is an independent predictor of SAP (OR = 1.078), and when combined with CRP, it can improve the accuracy of severity stratification.

Systemic inflammation response index (SIRI): Calculated based on (neutrophils \times monocytes)/lymphocytes, its AUC for predicting SAP is 0.782, and for predicting AKI is 0.776.

Platelet-to-lymphocyte ratio (PLR): Its predictive value is lower than that of NLR (AUC = 0.621 for predicting SAP), but when combined with BISAP, it can enhance the identification of severe cases.

III) Predictive Value of TyG Index in AP

The triglyceride-glucose (TyG) index is calculated as $\ln [(fasting\ triglycerides\ [mg/dL] \times fasting\ blood\ glucose\ [mg/dL])/2]$. It reflects insulin resistance and is associated with the severity of AP:

The TyG index in SAP patients is significantly higher than that in mild acute pancreatitis (MAP) patients (mean difference [MD] = 0.61, 95% CI: 0.46 - 0.76).

It is an independent predictor of SAP (OR = 1.835; OR = 7.14). The cut-off value is 8.76 for non-hyperlipidemic AP and 11.81 for hyperlipidemic AP.

After incorporating the TyG index into the prediction model, the AUC increases from 0.738 to 0.830.

It is associated with complications (such as infection, AKI) and mortality: the TyG-BMI index (TyG \times body mass index) in the highest quartile increases the risk of ICU and in-hospital mortality.

IV) Predictive Models and Efficacy of Inflammatory Markers Combined with TyG Index

1) Theoretical Basis

Inflammation and metabolism form a positive feedback loop: inflammatory factors exacerbate insulin resistance, while metabolic disorders promote inflammation. The TyG index reflects metabolic status, and inflammatory markers indicate inflammatory activity. Their combination enables comprehensive prediction of AP severity.

2) Construction and Efficacy of Combined Models

CRP + NLR + TyG: The AUC for predicting SAP is 0.882 (sensitivity = 77.2%, specificity = 88.5%).

SII + Nutritional Risk Index (NRI) + TyG: The AUC for predicting SAP is 0.705.

BISAP + Red Blood Cell Distribution Width (RDW): The AUC for predicting SAP is 0.872, which is better than that of a single indicator.

SOFA + RDW: The AUC for predicting 28-day mortality is 0.976.

3) Comparison with Other Methods

Combined models (such as CRP + NLR + TyG, with an AUC = 0.882) are comparable to single indicators like BISAP or RDW but have mechanistic advantages in metabolism-related AP (such as hyperlipidemic AP).

V) Clinical Translation Prospects of Prediction Models

Combined models use conventional indicators (such as CRP, triglycerides), featuring low cost and rapid turnaround. They can be integrated into electronic medical records for automatic calculation, facilitating emergency triage. For example, models combining CT findings and blood biomarkers have been proven to have clinical application value in HLAP (**Table 1**).

Table 1. Evidence table: Article titles, years, design, sample sizes, biomarkers, cut-offs, and AUCs for each included study were summarized.

Article Title	Year	Study Design	Sample Size	Biomarkers/Scoring Systems	Cut-offs	AUC (Outcome)
[1] Comparison of APACHE II, BISAP, Ranson's score and modified CTSI	2018	Prospective	50	APACHE II, BISAP, Ranson's score, modified CTSI	Optimal cut-offs evaluated (not specified)	modified CTSI: 0.919 (severe AP), 0.993 (pancreatic necrosis/ICU admission); APACHE II: 0.834 (severe AP), 0.831 (organ failure)
[2] Evaluation of MCTSI and CTSI	2021	Prospective cohort	149	MCTSI, CTSI, APACHE-II	Not specified	CTSI: 0.749 (persistent organ failure), 0.793 (death), 0.862 (intervention against necrosis), 0.883 (infected necrosis)
[3] BISAP score for predicting SAP (meta-analysis)	2016	Meta-analysis	1972 (9 studies)	BISAP	2 and 3 (subgroup analysis; 3 with higher specificity)	0.77 (severe AP)
[4] BISAP, NLR, CRP, BUN for AP outcomes	2021	Retrospective	Not specified	BISAP, NLR48h, CRP48h, BUN, hematocrit48h	Not specified	NLR48h + CRP48h: 0.89 (severe AP); BISAP: "fair" (severe AP)

Continued

[5] Severity stratification with NLR, PLR, RDW, and scores	2019	Retrospective	406	NLR, PLR, RDW, BUN, SOFA, BISAP, Ranson, APACHE II; BISAP + RDW, SOFA + RDW	Not specified	SAP: BISAP (0.841), BISAP+RDW (0.872); Mortality: SOFA (0.968), SOFA+RDW (0.976)
[6] PCT, CRP, IL-6, NLR, TyG for AP severity	2025	Retrospective	137 AP, 30 controls	PCT, CRP, IL-6, NLR, TyG; CRP + NLR, CRP + TyG, CRP + NLR + TyG	Not specified	CRP + NLR + TyG: 0.882 (severe AP)
[7] PCT for AP severity (meta-analysis)	2006	Meta-analysis	9 studies	PCT	Not specified	Overall: 0.91; High-quality studies: 0.94 (severe AP)
[8] Inflammation-based model for SAP	2024	Retrospective	253 (60 SAP)	SII, NLR, PLR, LMR, NPR, SIRI, PAR, CAR, CLR, TyG; Model (fatty liver, PCT, CLR)	Not specified	Model: 0.795 (severe AP)
[9] IL-6, CRP, PCT, CTSI for AP severity	2023	Retrospective	103	IL-6, CRP2 (24 h), PCT, CTSI	IL-6 < 50 pg/mL; CRP2 < 50 mg/L	IL-6: 0.755; CRP2: 0.787; CTSI: 0.851 (mild vs severe AP)
[10] Leukocyte signaling pathways for AP severity	2021	Prospective	174 AP, 31 controls	pSTAT1, pSTAT6, pNF- κ B, pAkt, pSTAT3 (leukocytes)	Not specified	Not reported
[11] SII and SIRI for AP outcomes	2022	Retrospective	332	SII, SIRI	Not specified	SII: 0.809 (SAP), 0.820 (AKI); SIRI: 0.782 (SAP), 0.776 (AKI)
[12] SII for AP severity	2021	Retrospective	101 (28 SAP)	SII, NLR, PLR	SII \geq 2207.53	SII: 0.920 (severe AP)
[13] NLR, PLR, RDW for AP severity	2023	Prospective cohort	131 (21 SAP)	NLR, PLR, RDW; combinations	NLR 13.5; PLR 202.7; RDW 13.1%	NLR: 0.82; PLR: 0.72; RDW: 0.73 (severe AP)
[14] TyG index and SAP (meta-analysis)	2025	Systematic review/ meta-analysis	2262 (10 studies)	TyG index	Not specified	Not reported (mean differences reported)
[15] TyG index for AP prognosis	2023	Retrospective	353 (47 SAP)	TyG index	8.76 (non-HTG/AAP); 11.81 (HTG/AAP)	Not explicitly reported (independent predictor for SAP)
[16] VExUS + TyG for AKI in hyperlipidemic AP	2024	Retrospective	110 (23 AKI)	TyG index, VExUS score; combination	Not specified	Combination: High (sensitivity 100%, specificity 95.65 for AKI)
[17] TyG index for SAP	2020	Multicenter	373	TyG index	Not specified	Model with TyG: 0.830 (SAP) vs without: 0.738
[18] TyG-BMI for mortality in critically ill AP	2024	Retrospective	419	TyG-BMI	243 (threshold for in-hospital mortality)	Not reported
[19] SII, NRI, TyG for HTGAP severity	2025	Retrospective	300	SII, NRI, TyG; combination	Not specified	Combination: 0.705 (SAP)

4. Discussion

Early prediction of the severity of acute pancreatitis (AP) is crucial for optimizing clinical management and improving patient outcomes. This review summarizes the research progress in the application of inflammatory markers combined with the triglyceride-glucose (TyG) index in this field. The literature findings not only highlight the advantages and limitations of individual markers but also demonstrate the value of their combined use.

Early studies focused on traditional severity scoring systems, such as APACHE II, BISAP, Ranson scores, and computed tomography (CT)-based indices (CTSI and modified CTSI). A 2018 study comparing these systems found that the modified CTSI had the highest area under the curve (AUC) in predicting severe acute pancreatitis (SAP), pancreatic necrosis, organ failure, and ICU admission. In contrast, APACHE II exhibited higher sensitivity and negative predictive value, rendering it particularly valuable in resource-limited settings [1]. A 2021 study further confirmed that CTSI and modified CTSI outperformed APACHE II in predicting persistent organ failure, mortality, and the need for necrotic intervention, with CTSI slightly surpassing modified CTSI. This underscores the role of radiological indices in assessing local complications [2].

However, traditional scoring systems have limitations. For example, a 2016 study evaluating BISAP showed that it had low sensitivity (64.82%) but high specificity (83.62%) in predicting SAP, indicating that it may miss some severe cases when used alone. This highlights the need for supplementary markers, such as inflammatory indices [3]. A 2021 study comparing BISAP with the 48-hour neutrophil-to-lymphocyte ratio (NLR48h) and 48-hour C-reactive protein (CRP48h) found that the combined use of NLR48h and CRP48h yielded an AUC of 0.89, with a sensitivity of 68% and specificity of 92%. This suggests that inflammatory markers can enhance predictive accuracy beyond what a single scoring system can achieve.

Inflammatory markers, including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), red blood cell distribution width (RDW), and systemic immune-inflammation index (SII), have shown potential in assessing AP severity. A 2019 study reported that when RDW was combined with BISAP, the AUC for SAP prediction increased to 0.872, while RDW alone demonstrated strong discriminative ability for both SAP and mortality [5]. A 2025 study identified CRP and NLR as independent predictors of SAP, and their combination with the TyG index yielded an AUC of 0.882, further supporting the effectiveness of a multi-marker strategy [6]. Reference [7] analyzed procalcitonin (PCT) and found it had moderate sensitivity (74%) but high specificity (83%) for SAP; high-quality studies showed that its sensitivity could be improved to 89%, suggesting its potential in early identification of infectious complications.

Novel inflammatory indices have also gained attention. A 2024 study developed an inflammatory model integrating fatty liver, PCT, and the C-reactive protein-to-lymphocyte ratio (CLR), which achieved an AUC of 0.795 for SAP prediction,

highlighting the value of integrating clinical and inflammatory parameters [8]. A 2023 study compared interleukin-6 (IL-6), 24-hour CRP (CRP2), and CTSI, finding that the AUCs of IL-6 and CRP2 were comparable to that of CTSI, with IL-6 offering the advantage of early assessment upon admission [9]. Additionally, a 2021 study exploring intracellular signaling pathways in leukocytes found that phosphorylated STAT3 (pSTAT3) and pSTAT1 levels correlated with AP severity, providing insights into the molecular mechanisms underlying inflammation-driven disease progression [10].

A 2022 study evaluated the systemic inflammatory response index (SIRI) and SII in relation to AP severity and acute kidney injury (AKI). It showed that SII had an AUC of 0.809 for SAP and 0.820 for AKI [11]. A 2021 study further confirmed that SII (AUC 0.920) outperformed NLR and PLR in predicting SAP, with high sensitivity (92.9%) and specificity (87.7%), making it a reliable inflammatory marker [12]. A 2023 study reported that NLR (AUC 0.82), PLR (AUC 0.72), and RDW (AUC 0.73) could individually predict SAP, but their combination did not improve performance, indicating that a single marker may be sufficient in specific contexts [13].

The TyG index, a surrogate marker for insulin resistance, has emerged as a key metabolic-inflammatory marker in AP. A 2025 meta-analysis showed that the TyG index was significantly elevated in SAP patients (mean difference = 0.61) and could predict ICU admission and mortality [14]. A 2023 study identified the TyG index as an independent risk factor for SAP (OR 1.835), with different cut-off values in hypertriglyceridemic AP (HTGAP) and non-HTGAP, emphasizing its applicability across AP subtypes [15]. A 2024 study demonstrated that combining the TyG index with the venous excess ultrasound (VExUS) score achieved 100% sensitivity and 95.65% specificity in predicting AKI in HTGAP patients, highlighting the synergistic effect of metabolic and hemodynamic markers [16].

The TyG index can also enhance the performance of existing models. A 2020 study found that adding the TyG index to an SAP prediction model increased the AUC from 0.738 to 0.830, confirming its incremental value [17]. A 2024 study further showed that the TyG-BMI index (TyG combined with body mass index) could predict in-hospital and ICU mortality in critically ill AP patients, with a threshold effect at 243 [18]. Additionally, studies have reported that in HTGAP, combining SII, the nutritional risk index (NRI), and the TyG index increased the AUC for SAP prediction to 0.705, underscoring the advantages of integrating immune, nutritional, and metabolic parameters [19].

In summary, inflammatory markers and the TyG index each offer unique perspectives for assessing AP severity: inflammatory markers reflect systemic immune responses, while the TyG index captures metabolic disturbances (closely linked to AP pathogenesis, particularly in HTGAP). Their combination integrates information from both aspects, improving predictive accuracy. However, challenges remain, including variability in cut-off values, limited data on early (<24 hours) prediction, and the need for validation in diverse populations. Future re-

search should focus on multicenter studies to standardize markers, explore the dynamic changes of combined indices, and integrate them into clinical decision-making tools to optimize early risk stratification of AP.

5. Conclusion

Traditional scoring systems for predicting acute pancreatitis (AP) severity have inherent limitations, while inflammatory markers and the triglyceride-glucose (TyG) index show distinct value in reflecting immune responses and metabolic disturbances, respectively. Their combined application integrates multi-dimensional information, significantly improving the accuracy of early prediction for severe AP (SAP) and adverse outcomes such as acute kidney injury (AKI) and mortality, with particular advantages in hyperlipidemic AP. However, challenges including inconsistent cut-off values and limited early-stage validation remain. Future research should focus on standardizing markers, exploring dynamic changes, and promoting clinical translation to optimize AP risk stratification.

6. Limitations and Future Directions

In contemporary research, the combined application of inflammatory markers and the TyG index in predicting the severity of acute pancreatitis is encumbered by several limitations, including inconsistent cut-off values, paucity of early-stage data, constraints in study design, and insufficient representation of heterogeneous populations, with the dynamic variations of combined indices remaining inadequately elucidated. Going forward, it is imperative to establish unified criteria through multicenter investigations, prioritize ultra-early validation, integrate multi-dimensional datasets, develop automated analytical tools, and explore dynamic regulatory patterns. Such initiatives will enhance the clinical utility of these combined models and refine the early risk stratification of acute pancreatitis.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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