

Dynamic Monitoring of Procalcitonin for Early Prediction of Pancreatic Necrosis

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Research Article

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Abstract

Objective

To investigate the predictive value of procalcitonin (PCT) for pancreatic necrosis in acute pancreatitis (AP).

Methods

A retrospective analysis was conducted on the clinical data of 190 patients with AP admitted to our hospital between January 2021 and August 2022. Patients were divided into acute edematous pancreatitis (AEP) and acute necrotizing pancreatitis (ANP) groups based on the presence of pancreatic necrosis. Clinical parameters were compared between the two groups. Independent risk factors were identified using logistic regression analysis, and their predictive value for pancreatic necrosis was evaluated using receiver operating characteristic (ROC) curve analysis.

Results

Triglyceride (TG), 48-hour white blood cell (WBC) count, and 48-hour PCT were identified as independent risk factors for pancreatic necrosis. The area under the ROC curve (AUC) values for TG, 48-hour WBC, 48-hour PCT, Acute Physiology and Chronic Health Evaluation II (APACHE II), Bedside Index for Severity in Acute Pancreatitis (BISAP), and Ranson scores were 0.690, 0.581, 0.883, 0.652, 0.696, and 0.684, respectively. Among these, 48-hour PCT demonstrated the highest predictive value, with an optimal cutoff of > 0.23 ng/mL yielding a sensitivity of 84.6% and specificity of 76.8% (95% CI: 0.828–0.938), indicating good predictive performance.

Conclusion

PCT may serve as an effective, convenient, and rapid biomarker for early identification of pancreatic necrosis, warranting broader clinical application.

1. Introduction

Acute pancreatitis (AP) is an acute inflammatory disorder of the exocrine pancreas characterized by both local and systemic inflammatory responses. Pathologically, it is classified into acute edematous pancreatitis (AEP) and acute necrotizing pancreatitis (ANP) [1]. The majority of AP patients present with mild, self-limiting disease that typically resolves within one week [1]. However, approximately 20% of patients progress to ANP, which is associated with increased risks of organ failure and infectious complications, thereby contributing to elevated mortality [2]. Studies have reported an overall mortality

rate of 15–20% for ANP, with one-third of patients developing pancreatic infection during the disease course, further increasing mortality to 30–39% [3].

Currently, the diagnosis of ANP relies heavily on contrast-enhanced computed tomography (CECT). However, radiological changes typically become apparent only 72 hours after symptom onset, and early inappropriate use of CECT may prolong hospital stays. Moreover, not all patients are suitable candidates for this examination [4]. Therefore, identifying reliable and convenient biomarkers for early recognition of pancreatic necrosis is of critical importance for guiding clinical management.

Procalcitonin (PCT), the bioactive precursor peptide of calcitonin, is a rapidly responding acute-phase reactant. Given that pancreatic necrosis is directly associated with persistent inflammation, the relationship between PCT and pancreatitis has been extensively reported. Current research has primarily focused on the diagnosis of infected pancreatic necrosis, prediction of severe pancreatitis, and guidance for antibiotic therapy. However, studies on early identification of pancreatic necrosis remain limited and controversial. Bertsch et al. [5] and Hagjer et al. [6] both measured PCT on the first day of admission in AP patients. The former reported a sensitivity of 75% (95% CI: 0.35–0.97) and specificity of 57% (95% CI: 0.18–0.90) for predicting pancreatic necrosis, whereas the latter found a sensitivity of only 28% (95% CI: 0.15–0.45) and specificity of 81% (95% CI: 0.58–0.95). This study aimed to evaluate the accuracy of dynamic PCT monitoring for early prediction of pancreatic necrosis, providing evidence to support clinical decision-making.

2. Materials and Methods

2.1 Study Population

This retrospective study was approved by the Ethics Committee of Xiamen Hospital of Traditional Chinese Medicine (IRB number:2023-K023-01). The requirement of informed consent for publication of data was waived by the Xiamen Hospital of Traditional Chinese Medicine Review Board owing to the retrospective nature of the study. The study was conducted in accordance with the Declaration of Helsinki. We enrolled 304 adult patients (aged > 18 years) diagnosed with AP at our hospital between January 1, 2021, and August 31, 2022. Diagnosis of AP was based on at least two of the following three criteria: (1) abdominal pain consistent with AP; (2) serum amylase or lipase levels greater than three times the upper limit of normal; and (3) characteristic imaging findings of AP. The presence of pancreatic necrosis was determined by the absence of pancreatic parenchymal enhancement on CECT.

2.2 Inclusion and Exclusion Criteria

Inclusion criteria were: (1) definite diagnosis of AP; (2) hospital admission within 48 hours of symptom onset; and (3) complete clinical data.

Exclusion criteria were: (1) other pancreatic diseases such as chronic pancreatitis or pancreatic cancer; (2) incomplete data; (3) special populations including children, pregnant women, and lactating women;

(4) malignancy, immunodeficiency, long-term use of immunosuppressants or glucocorticoids, or severe hepatic/renal insufficiency; and (5) concurrent extrapancreatic infections at disease onset, including acute cholangitis, pulmonary infection, or urinary tract infection.

2.3 Data Collection

Demographic and laboratory data were extracted from electronic medical records, including age, sex, etiology, white blood cell (WBC) count, hematocrit, C-reactive protein (CRP), PCT, serum calcium, triglyceride (TG), total bilirubin (TBIL), alanine aminotransferase (ALT), aspartate aminotransferase (AST), D-dimer, lactate dehydrogenase (LDH), creatinine (Cr), blood urea nitrogen (BUN), and blood glucose (Glu). The Acute Physiology and Chronic Health Evaluation II (APACHE II) and Bedside Index for Severity in Acute Pancreatitis (BISAP) scores were calculated within 24 hours of admission. WBC, hematocrit, CRP, PCT, serum calcium, Cr, and BUN were reassessed at 48 hours, and Ranson scores were calculated at this time point. CECT was performed 72 hours after admission, and the Computed Tomography Severity Index (CTSI) was calculated. Patients were divided into AEP and ANP groups based on CECT findings. All data were independently collected and verified by two authors, and cases with missing data were excluded, resulting in a complete dataset for final analysis.

2.4 Statistical Analysis

Statistical analyses were performed using SPSS version 23.0. Continuous variables with normal distribution were expressed as mean $\pm$ standard deviation, while those with non-normal distribution were presented as median (interquartile range, IQR). Categorical variables were expressed as absolute numbers and percentages. Group comparisons were performed using Fisher's exact test, unpaired t-test, or chi-square test as appropriate. Independent risk factors for pancreatic necrosis were identified using binary logistic regression analysis. Predictive performance was evaluated using receiver operating characteristic (ROC) curves, and the area under the curve (AUC) was calculated. An AUC > 0.7 was considered indicative of good diagnostic value. Optimal cutoff values, sensitivity, and specificity were calculated using MedCalc software. A P-value < 0.05 was considered statistically significant.

3. Results

3.1 Baseline Characteristics

A total of 304 patients diagnosed with AP at Xiamen Hospital of Traditional Chinese Medicine between January 2021 and August 2022 were initially enrolled. After applying exclusion criteria—chronic pancreatitis ($n = 3$), pancreatic cancer ($n = 1$), other malignancies ($n = 2$), chronic renal failure ($n = 1$), incomplete data ($n = 65$), cholangitis ($n = 26$), and admission beyond 48 hours of symptom onset ($n = 16$)—190 patients were included in the final analysis. Of these, 151 (79.5%) were male and 39 (20.5%) were female. Etiologies included biliary ($n = 10$, 5.5%), alcoholic ($n = 12$, 6.3%), hypertriglyceridemic ($n = 110$, 57.8%), post-ERCP ($n = 2$, 1.0%), and other causes ($n = 56$, 29.4%).

Based on CECT findings, 138 patients (72.6%) were classified as AEP and 52 (27.4%) as ANP. Among ANP patients, 45 (86.5%) were male and 7 (13.5%) were female, with a mean age of 40.19 ± 11.62 years. In the AEP group, 106 (76.8%) were male and 32 (23.2%) were female, with a mean age of 41.64 ± 11.13 years. No significant differences were observed between groups in terms of sex or age (Table 1).

Table 1
Baseline characteristics of AEP and ANP groups

Characteristic	AEP (n = 138)	ANP (n = 52)	P-value
Sex			0.201
Male	106(76.8%)	45(86.5%)	
Female	32(23.2%)	7(13.5%)	
Age (years)	41.64 ± 11.13	40.19 ± 11.62	0.429
Etiology			
Biliary	9(6.5%)	1(1.9%)	
Hypertriglyceridemic	71(51.5%)	39(75.0%)	
Alcoholic	11(8.0%)	1(1.9%)	
Post-ERCP	2(1.4%)	0(0%)	
Other	45(32.6%)	11(21.2%)	

3.2 Clinical Parameters

Comparison of admission laboratory parameters revealed that WBC, CRP, PCT, TG, Glu, D-dimer, and AST were significantly higher in the ANP group than in the AEP group. At 48 hours, CRP, PCT, and serum calcium remained significantly elevated in the ANP group. APACHE II, BISAP, CTSI, and Ranson scores were all significantly higher in the ANP group compared with the AEP group (Table 2).

Table 2
Comparison of clinical parameters between AEP and ANP groups

Parameter	AEP (n = 138)	ANP (n = 52)	P-value
Admission parameters			
WBC (×10 ⁹ /L)	12.75 [10.70;15.70]	15.35 [12.33;17.85]	0.011
Hematocrit (%)	44.30 [40.20;46.50]	44.50[40.60;46.20]	1.000
CRP (mg/dL)	3.60 [0.68;20.90]	11.30 [2.20;61.20]	0.019
PCT (ng/mL)	0.05 [0.05;0.11]	0.12 [0.05;0.56]	0.005
LDH(U/L)	202.00[172.50;240.50]	205.00[177.25;273.00]	0.549
BUN (mmol/L)	4.20[3.40;5.00]	4.00 [3.15;4.60]	0.254
Cr (mmol/L)	73.95[65.76;81.18]	74.55 [67.40;87.48]	0.870
Serum calcium (mmol/L)	2.33 [2.23;2.40]	2.34[2.22;2.46]	0.846
Glu (mmol/L)	7.50 [6.10;11.09]	11.20 [7.98;15.63]	< 0.001
TBIL (mmol/L)	15.70 [13.10;21.33]	16.25 [11.45;22.76]	0.871
ALT (U/L)	26.00[18.00;42.25]	27.00 [18.25;41.50]	0.450
AST (U/L)	23.00 [17.75;33.25]	29.00 [20.25;40.75]	0.034
D-dimer(ng/mL)	144.5[98.00;264.00]	266.00[142.00;555.00]	0.001
TG(mmol/L)	4.86[1.31;10.57]	17.23 [4.01;38.36]	0.002
APACHE II (score)	3.00 [1.00;4.00]	4.00 [2.00;6.00]	0.045
BISAP (score)	1.00 [0.00;2.00]	2.00 [1.00;2.00]	< 0.001
48-hour parameters			
WBC (×10 ⁹ /L)	9.55[7.30;12.23]	10.40[7.83;14.53]	0.367
Hematocrit (%)	40.80 [37.98;43.53]	41.70 [37.05;44.65]	0.416
CRP (mg/dL)	106.30 [52.58;159.25]	230.60[150.76;303.60]	< 0.001
PCT (ng/mL)	0.10 [0.05;0.22]	0.78[0.32;1.87]	< 0.001
Footnote: WBC, white blood cell; CRP, C-reactive protein; PCT, procalcitonin; LDH, lactate dehydrogenase; BUN, blood urea nitrogen; Cr, creatinine; Glu, blood glucose; TBIL, total bilirubin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TG, triglyceride; APACHE II, Acute Physiology and Chronic Health Evaluation II; BISAP, Bedside Index for Severity in Acute Pancreatitis; CTSI, Computed Tomography Severity Index.			

Parameter	AEP (n = 138)	ANP (n = 52)	<i>P</i> -value
Admission parameters			
Serum calcium (mmol/L)	2.16 [2.07;2.23]	2.05 [1.93;2.19]	0.011
BUN (mmol/L)	2.80[2.08;3.33]	3.10 [2.60;4.00]	0.141
Cr (mmol/L)	72.35 [62.28;79.53]	68.90 [612.40;77.90]	0.253
Ranson (score)	1.00 [0.00;2.00]	2.00 [1.00;3.00]	0.004
CTSI (score)	1.00 [1.00;2.00]	2.00 [2.00;3.00]	< 0.001
Footnote: WBC, white blood cell; CRP, C-reactive protein; PCT, procalcitonin; LDH, lactate dehydrogenase; BUN, blood urea nitrogen; Cr, creatinine; Glu, blood glucose; TBIL, total bilirubin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TG, triglyceride; APACHE II, Acute Physiology and Chronic Health Evaluation II; BISAP, Bedside Index for Severity in Acute Pancreatitis; CTSI, Computed Tomography Severity Index.			

3.3 Logistic Regression Analysis

Univariate binary logistic regression identified 12 variables with statistically significant differences between groups: admission WBC, CRP, PCT, TG, LDH, Glu, D-dimer, and 48-hour WBC, CRP, PCT, serum calcium, and BUN. Multivariate analysis revealed that TG (OR = 1.040, 95% CI: 1.010–1.071, $P = 0.008$), 48-hour WBC (OR = 1.137, 95% CI: 1.026–1.259, $P = 0.014$), and 48-hour PCT (OR = 7.331, 95% CI: 2.702–19.892, $P < 0.001$) were independent risk factors for ANP (Table 3).

Table 3
Logistic regression analysis of independent risk factors for pancreatic necrosis

Parameter	Univariate analysis			Multivariate analysis		
	OR (95%CI)		P-value	OR (95%CI)		P-value
WBC ($\times 10^9/L$)	1.101	1.014–1.196	0.021			
CRP (mg/dL)	1.007	1.001–1.1012	0.017			
PCT (ng/mL)	18.262	3.250–102.62	0.001			
LDH (U/L)	1.133	1.001–1.005	0.031			
Glu (mmol/L)	1.089	1.056–1.215	< 0.001			
D-dimer(ng/mL)	1.001	1.000–1.002	0.027			
TG (mmol/L)	1.044	1.023–1.064	< 0.001	1.040	1.010–1.071	0.008
48-hour WBC ($\times 10^9/L$)	1.102	1.025–1.185	0.008	1.137	1.026–1.259	0.014
48-hour CRP (mg/dL)	1.006	1.003–1.010	< 0.001			
48-hour PCT (ng/mL)	8.294	3.460–19.880	< 0.001	7.331	2.702–19.892	< 0.001
48-hour serum calcium (mmol/L)	0.025	0.004–0.173	< 0.001			
48-hour BUN (mmol/L)	1.427	1.120–1.820	0.004			
Footnote: WBC, white blood cell; CRP, C-reactive protein; PCT, procalcitonin; LDH, lactate dehydrogenase; Glu, blood glucose; TG, triglyceride; BUN, blood urea nitrogen.						

3.4 ROC Curve Analysis

ROC curve analysis was performed to evaluate the predictive performance of TG, 48-hour PCT, 48-hour WBC, APACHE II, Ranson, and BISAP scores for pancreatic necrosis. The corresponding AUC values were 0.678, 0.878, 0.581, 0.654, 0.677, and 0.691, respectively (Fig. 1). Among these, 48-hour PCT demonstrated the highest predictive value. At an optimal cutoff of > 0.23 ng/mL, 48-hour PCT yielded a sensitivity of 84.6% and specificity of 76.8% (95% CI: 0.821–0.936), indicating good predictive performance (Table 4).

Table 4
ROC curve analysis for predicting pancreatic necrosis

Parameter	AUC	SE	95%CI	Cutoff	Sensitivity	Specificity	P-value
TG(mmol/L)	0.678	0.049	0.582– 0.774	> 10.72	61.5	76.8	< 0.001
48-hour PCT (ng/mL)	0.878	0.029	0.821– 0.936	> 0.23	84.6	76.8	< 0.001
48-hour WBC (×10 ⁹ /L)	0.581	0.052	0.480– 0.682	> 18.3	17.3	97.8	0.101
APACHEII	0.654	0.048	0.560– 0.749	> 4	46.90	78.7	0.002
Ranson	0.677	0.046	0.587– 0.767	> 1	53.1	71.7	< 0.001
BISAP	0.691	0.045	0.603– 0.767	> 1	57.1	74.8	< 0.001

4. Discussion

The incidence of AP is rising annually, and its clinical presentation is complex and variable. Although 80% of AEP cases are mild with very low mortality, the remaining 20% of ANP patients face increased mortality due to various complications, including multiple organ dysfunction, abdominal compartment syndrome, and infected pancreatic necrosis [7]. In the early phase of the disease, persistent organ failure resulting from systemic inflammatory response syndrome constitutes the primary cause of death. In the later phase, necrosis of pancreatic parenchyma and/or peripancreatic adipose tissue leads to ANP, with more patients succumbing to complications such as septic shock from infected pancreatic necrosis [8]. The extent of pancreatic necrosis is significantly associated with the risk of infection and organ dysfunction; studies have shown that pancreatic necrosis exceeding 50% is an important risk factor for organ dysfunction [9]. Early identification of ANP enables timely implementation of preventive measures including fluid resuscitation, intensive care, and organ support, potentially reducing mortality [10]. In this study, we compared the predictive performance of commonly used clinical scoring systems and biochemical markers for pancreatic necrosis. Our results demonstrated that TG, 48-hour WBC, and 48-hour PCT were independent risk factors for pancreatic necrosis, with PCT serving as a reliable single biochemical marker for early identification of pancreatic necrosis at 48 hours.

Compared with other etiologies, hypertriglyceridemic pancreatitis is more prone to persistent systemic inflammatory response syndrome, organ dysfunction, and severe disease [11]. Mallet et al. [12] found that TG dose-dependently exacerbates AP severity, increasing the incidence of local complications and organ failure. A prospective study from Spain reported that the incidence of ANP increases proportionally with TG levels, and admission TG ≥ 200 mg/dL was independently associated with pancreatic necrosis; however, its predictive ability was modest, with an AUC of only 0.601 (95% CI:

0.519–0.684) [13]. Our findings are consistent with these observations: TG was identified as an independent risk factor for ANP, but at a cutoff of > 10.72 mmol/L, the AUC was only 0.678 (95% CI: 0.582–0.774). This may be attributed to the rapid reduction in TG levels due to early fasting, fluid resuscitation, and other lipid-lowering interventions [14]. Furthermore, the relationship between TG and disease severity is influenced by the timing of measurement relative to symptom onset; after admission, supportive care and lipid-lowering therapy lead to a rapid decline in TG levels, and the correlation with disease severity may diminish or disappear within 24 hours to 5–7 days [15]. Therefore, early detection of TG levels at the onset of pancreatitis is crucial for accurate prediction of pancreatic necrosis.

As an important parameter of systemic inflammation, WBC is commonly used in the diagnosis and follow-up of pancreatitis. During the progression of pancreatitis to ANP and sepsis, the inflammatory response is predominantly mediated by neutrophils [16]. Dynamic monitoring of WBC may assist clinicians in assessing disease severity, but its sensitivity is relatively low [17]. Yilmaz et al. [18] reported that a WBC cutoff of > 15,200/ μ L predicted ANP with an AUC of 0.765 (95% CI: 0.614–0.916), with a sensitivity of only 66.6% and specificity of 87.6%. Another study found that WBC was an independent predictor of pancreatic necrosis development, with 48-hour WBC > 15.25×10^9 /L conferring a 3.5-fold increased risk of ANP [19]. Similarly, we found that 48-hour WBC was an independent risk factor for ANP, but with an AUC of only 0.581 (95% CI: 0.480–0.682) and low sensitivity and specificity, making it unsuitable for early identification of pancreatic necrosis. PCT may represent a more effective marker.

PCT is a general acute-phase reactant involved in infection and inflammatory processes, released by the thyroid, hepatocytes, and peripheral mononuclear cells. It is typically undetectable in healthy adults [17]. Early studies demonstrated significant differences in PCT levels between mild and severe AP cases, with elevated serum PCT (> 0.5 ng/mL) serving as a reliable early predictor of severe disease, mortality, and infected pancreatic necrosis [6]. Chen et al. [20] reported that PCT may serve as a biomarker for predicting ANP. In our study, we measured PCT at admission and at 48 hours in 190 AP patients. While PCT levels were significantly higher in the ANP group at both time points, admission PCT showed no independent predictive value. In contrast, 48-hour PCT was identified as an independent risk factor for pancreatic necrosis with superior predictive performance compared to APACHE II, Ranson, and BISAP scores. We attribute this to the fact that all enrolled patients presented within 48 hours of symptom onset, during which pancreatic inflammation may not have fully manifested and inflammatory cytokines may not have reached peak expression, thus making 48-hour PCT more discriminative than admission PCT.

Notably, the reported cutoff values for PCT in predicting pancreatic necrosis vary across studies. Staubli et al. [17] measured PCT at admission and at 48, 72, and 96 hours, taking averaged values, and found a cutoff of 0.75 ng/dL for identifying ANP (sensitivity: 100%; specificity: 98.86%). Bertsch et al. [5] reported that day-1 PCT > 0.5 ng/mL predicted pancreatic necrosis with 75% sensitivity (95% CI: 35–97%) and 57% specificity (95% CI: 18–90%). Chen et al. [20] reported a cutoff of 1.39 ng/dL (sensitivity: 60.9%; specificity: 75%). In contrast, our study identified a cutoff of 48-hour PCT > 0.23 ng/mL, with an AUC of 0.878, sensitivity of 84.6%, and specificity of 76.8% (95% CI: 0.821–0.936), which is lower than previously

reported values. We believe these discrepancies are primarily attributable to methodological differences: (1) varying time from symptom onset to admission across studies may influence test results; (2) other studies included a higher proportion of patients with infected pancreatic necrosis, whereas the majority of ANP cases in our cohort were sterile necrosis. Additionally, we excluded patients with concurrent extrapancreatic infections (e.g., acute cholangitis, pulmonary infection) to minimize confounding effects on PCT levels, which likely contributed to the lower cutoff value observed in our study.

We also evaluated the predictive performance of commonly used clinical scoring systems—APACHE II, Ranson, and BISAP—for pancreatic necrosis. The AUC values were 0.652, 0.696, and 0.684, respectively, indicating suboptimal predictive ability. These findings are consistent with the prospective study by Khanna et al. [21]. Moreover, the Ranson, APACHE II, and BISAP scores are computationally complex, requiring collection of multiple data points early in the disease course, which complicates the early risk stratification process. In comparison, PCT offers a more efficient and convenient marker worthy of clinical attention.

This study has several limitations. First, it is a retrospective study with a relatively small sample size, lacking multicenter, large-scale prospective validation. Second, due to the limited number of infected necrosis cases in our ANP cohort, we were unable to analyze differences in PCT levels between sterile and infected necrosis; this will be addressed in future studies with expanded sample sizes. Finally, to avoid confounding effects of extrapancreatic infections on PCT levels, we excluded patients with concurrent cholangitis, pulmonary infection, and urinary tract infection, resulting in a predominantly hypertriglyceridemic AP cohort. Therefore, the predictive performance of PCT for pancreatic necrosis may be diminished in the presence of infection, particularly in biliary pancreatitis.

5. Conclusion

PCT may serve as an effective, convenient, and rapid biomarker for early identification of pancreatic necrosis, warranting broader clinical application. However, its predictive value may be compromised in patients with concurrent extrapancreatic infections, particularly biliary pancreatitis. Future multicenter, large-scale prospective studies with subgroup analyses based on etiology are needed to further validate these findings.

Declarations

Ethics approval and consent to participate:

The study was approved by the Ethics Committee of Xiamen Hospital of Traditional Chinese Medicine and the ethical approval number was 2023-K023-01. The requirement of informed consent to participate was waived by the Xiamen Hospital of Traditional Chinese Medicine Review Board owing to the retrospective nature of the study. Our study adhered to the Declaration of Helsinki.

Consent for publication:

Not applicable.

Competing interests:

The authors report no competing interest.

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Author Contribution

Xi Xia Weng: Data collection, statistical analysis, manuscript writing. Jun Chao Zhang: Data collection, manuscript revision, study design, funding support.

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Availability of data and materials:

Not applicable.

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Figures

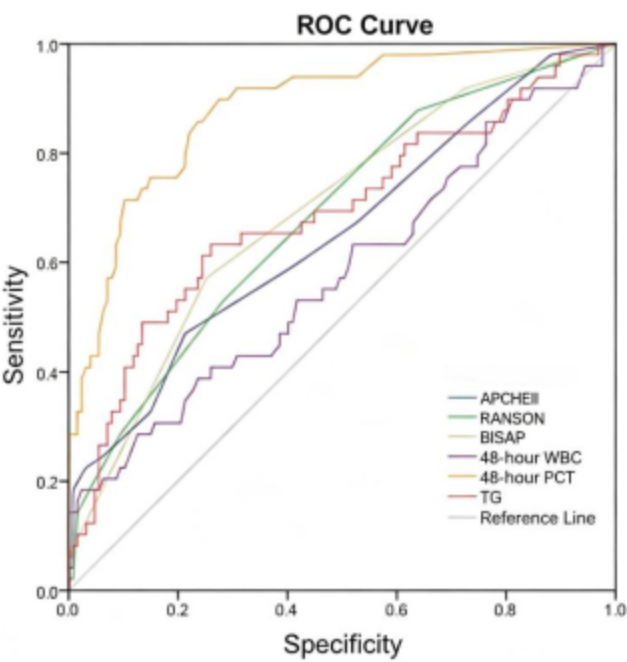


Figure 1

ROC curves for predicting pancreatic necrosis