Clinical Value of Energy Spectrum Computed Tomography Imaging in Evaluating the Severity of Acute Pancreatitis

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Abstract: Objective: The value of spectral computed tomography in evaluating the severity of acute pancreatitis (AP). Methods: We selected 54 patients with AP (treated between June 2024 and November 2025) and divided them into mild, moderate, and severe AP groups by employing the Revised Atlanta classification (RAC) and iodine concentration (IC) and standardized iodine concentration (NIC) values were recorded. These parameters were combined with procalcitonin, interleukin-6, haematocrit, and D-dimer levels to predict the condition of severity AP. Results: Differences in procalcitonin, interleukin6, and hospital stay were significant ($P < 0.05$). IC and NIC also differed significantly between groups ($P < 0.05$). ROC analysis showed AUCs of 0.661, 0.748, 0.554, 0.534 for procalcitonin, interleukin6, Ddimer, and haematocrit, respectively. IC achieved an AUC of 0.834, with an optimal cutoff of 2.7 mg/mL for diagnosing moderatesevere AP. Conclusion: Spectral CT has clinical guiding value in the diagnosis of AP.

***Keywords*:** acute pancreatitis; spectral spectrum computed tomography; severity

1. Introduction

Acute pancreatitis (AP) is a common acute abdominal emergency. Most cases are mild and self-limiting, but about 20% progress to severe acute pancreatitis (SAP), with a mortality rate up to 30% and complications including pancreatic necrosis, systemic inflammation, and multiple organ failure[1]. In SAP, 20%–40% develop infected pancreatic and peripancreatic necrosis, with mortality of 19.8% for sterile necrosis and 35.2% for infected necrosis[2]. Early severity assessment is critical for prognosis and management. Computed tomography (CT) is widely used for diagnosis and grading, but early findings may not fully reflect pancreatic necrosis[3]. Contrast-enhanced CT can detect obvious ischemia or necrosis, but cannot quantitatively evaluate pancreatic microcirculation or identify incomplete ischemic necrosis[4].

Spectral CT enables wateriodine decomposition by separating iodine signals, generating materialspecific images. As iodine is the key component of contrast agents, spectral CT sensitively and objectively quantifies iodine concentration (IC). By assessing local tissue iodine uptake, it directly reflects tissue blood perfusion at the microcirculatory level[5].

We aimed to explore the application value of spectral CT imaging in the assessment of AP and provide diagnostic and therapeutic strategies for assessment of AP severity in clinical practice.

2. Methods

2.1 General Information

We collected data of 54 patients with AP admitted to our hospital between June 2024 and October 2025, including 27 men and 27 women, aged 24-68 years, with a mean age of 46.11±11.64 years.

Inclusion criteria: (1) AP diagnosed by clinical manifestations, serum pancreatic enzymes, and imaging; (2) Age 18–70 years; (3) Stable vital signs and complete clinical data.

Exclusion criteria: (1) Postoperative, traumatic, or tumor-induced pancreatitis; (2) Cirrhosis, chronic obstructive pulmonary disease, silicosis, respiratory/heart failure, chronic pancreatitis, or autoimmune diseases; (3) Iodine allergy or CT contraindications; (4) Concurrent extrapulmonary infections or heart/liver/kidney dysfunction.

All patients included in this study provided informed consent and signed an informed consent form. This study was conducted in compliance with the principles of medical ethics and was approved by the Ethics Committee of our hospital. We employed, the Chinese Guidelines for the Diagnosis and Treatment of AP and the Revised Atlanta Classification (RAC) [6] to categorize our study cohort into the following groups: MAP group (no organ failure, local complications, n = 29), MSAP group (transient organ dysfunction ≤ 48 h and/or local complications, n = 5), SAP group (persistent organ dysfunction > 48h, n = 20). They were further divided into mild group (29 cases) and moderate to severe groups (25 cases; 5 patients with moderate AP).

2.2 Inspection methods

Venous blood was collected within 12 hours of admission to measure procalcitonin, interleukin6, Ddimer, and haematocrit. All patients underwent upper abdominal spectral CT scan (Siemens SOMATOM Force, Germany) with breath instructions during scanning.

2.3 Image analysis

Two experienced imaging deputy directors transferred images to Syngo.Via. Arterial-phase ROIs were placed in the center of the pancreatic head, body, and tail (1/2–2/3 of the regional pancreatic volume), avoiding blood vessels, pancreatic ducts, and liquefactive necrosis. An abdominal aorta ROI was set at the same level as the lesion. Pancreatic mean IC and abdominal aorta IC were measured to calculate normalized iodine concentration (NIC = pancreatic mean IC / abdominal aorta IC). Disputes were resolved by joint discussion of the two physicians.

2.4 Statistical analysis

We employed IBM SPSS25.0 statistical software for data analysis. Measurement data that conform to a normal distribution are represented by mean ± standard deviation ($x \pm s$), while those that do not conform are represented by median and interquartile range [M (P25, P75)]. The comparison between groups of normally distributed quantitative data was conducted using t-test or analysis of variance. Non-parametric tests (Mann Whitney U) were used to compare non-normally distributed quantitative data between the two groups. The comparison of countable data was conducted using the Chi square test ($\chi 2$). We employed binary logistic regression analysis to identify independent risk factors for AP, plotting receiver operating characteristics (ROC) to evaluate the severity grading efficacy of AP, and determining the threshold through the maximum Jordan index. Statistical significance of the measured difference was set at $P < 0.05$.

3. Results

3.1 General information

There was no statistically significant difference in baseline data such as sex, etiology, age, BMI, D-dimer, and haematocrit levels between the two groups of patients (all $P > 0.05$, Table 1). The difference in hospitalization days, procalcitonin, interleukin-6 levels between the MAP and the MSAP + SAP groups was highly significant ($P < 0.001$, Table 1).

Table 1. Patient characteristics

Parameters	MAP group (n = 29)	MSAP+SAP group (n = 25)	$\chi 2/t$	P-Value
Sex			1.862	0.172
Men	12 (41.38%)	15 (60.00%)		
Women	17 (56.62%)	10 (40.00%)		
Etiology			3.184	0.359
Biliary origin	13 (44.83%)	10(40.00%)		
Alcoholic	10 (34.48%)	8 (32.00%)		
Hypertriglyceridemia	6 (20.69%)	6(24.00%)		
Other	0(0.00%)	1 (4.00%)		
Age (years)	48.48±10.93	43.36±12.04	1.638	0.107
BMI (kg/m^2)	25.01±3.54	26.88±4.66	-1.674	0.100
Length of hospital stay (d)	10.72±3.05	18.96±10.14	-4.167	<0.001
Procalcitonin (μg/L)	1.25±0.67	1.60±0.60	-2.006	0.050
Interleukin-6 (pg/ml)	101.24±50.24	142.97±42.17	-3.275	0.002
D-dimer (ng/mL)	2171.95[1657.98;2531.26]	2595.56[1856.99;2683.69]	-0.685	0.493
Hematocrit (L/L)	0.43±0.09	0.44±0.09	-0.482	0.632

Abbreviations: MAP: mild acute pancreatitis, MSAP: moderate severe acute pancreatitis; SAP: severe acute pancreatitis, BMI: body mass index; Note: Significance at P-value ≤ 0.05

3.2 Comparison of IC and NIC parameters between two groups of patients with AP

Both groups of patients with AP showed statistically significant differences in IC and NIC ($P < 0.05$, Table 2).

Table 2. Differences in IC and NIC parameters between two groups of patients with AP

Parameter	MAP group	MSAP + SAP group	t	P-value
IC (mg/mL)	2.89±0.50	2.09±0.72	4.805	<0.001
NIC	0.32±0.44	0.26±0.07	3.565	0.001

Abbreviations: IC: Iodine concentration value; NIC: standardized iodine concentration

Note: Significance at P-value ≤ 0.05.

3.3 Establishment of an early clinical diagnosis prediction model for SAP and plotting the Receiver Operating Characteristic (ROC) curve

ROC analysis showed AUCs of procalcitonin, IL6, Ddimer, and HCT for AP severity grading were 0.661, 0.748, 0.554, 0.534, with sensitivities 56.0%, 84.0%, 64.0%, 48.0%, specificities 75.9%, 62.1%, 58.6%, 62.1%, and cutoffs 1.745 ng/mL, 100.610 pg/mL, 2310.66 ng/mL, 0.44 L/L.IC and NIC yielded AUCs of 0.824 and 0.746, sensitivities 79.3% and 84.0%, specificities 65.5% and 72.0%, with optimal cutoffs 2.7 mg/mL and 0.295 for moderatesevere AP (Table 3, Figures 1, 2).

Table 3. Sensitivity, specificity, Area under the ROC curve, and optimal cutoff values for each indicator

Indicator	AUC	Optimal cutoff value	Sensitivity (%)	Specificity (%)	95%CI
Procalcitonin	0.661	1.745 ng/mL	56.0	75.9	0.515-0.807
Interleukin-6	0.748	100.610 pg/mL	84.0	62.1	0.616-0.879
D-dimer	0.554	2310.66 ng/mL	64.0	58.6	0.398-0.711
Haematocrit	0.534	0.44 L/L	48.0	62.1	0.378-0.689
IC	0.834	2.7 mg/mL	79.3	84.0	0.719-0.949
NIC	0.746	0.295	65.5	72.0	0.616-0.877

Abbreviations: AUC: Area under the ROC curve of each patient indicator

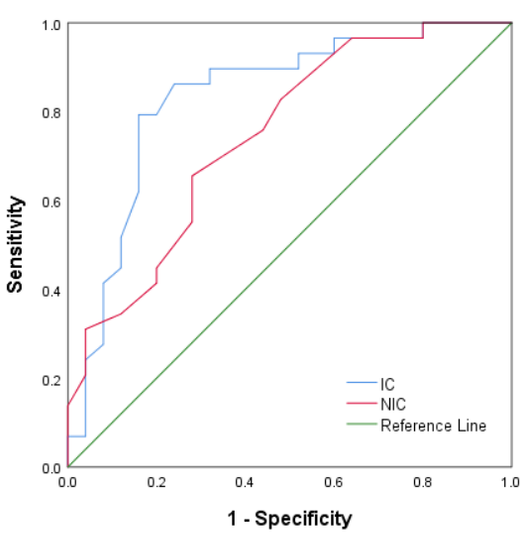

Figure 1. ROC curve for predicting the severity of acute pancreatitis using IC and NIC

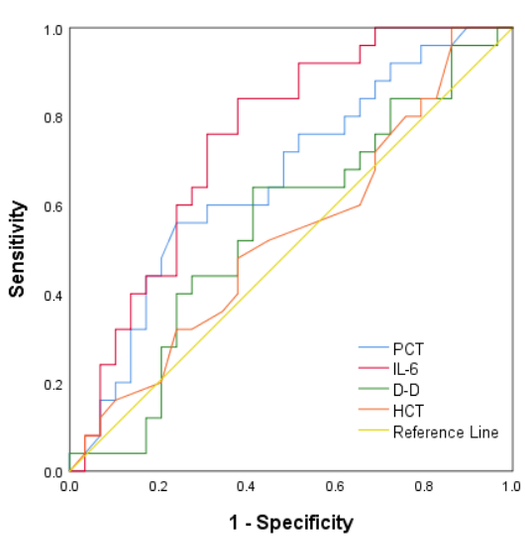

Figure 2. ROC curve for predicting the severity of acute pancreatitis using PCT, IL-6, D-dimer, and HCT

4. Discussion

The progression of AP is driven by pancreatic microcirculatory disturbance and systemic inflammatory response. The pancreas lacks effective collateral circulation, making it vulnerable to ischemiahypoxia[8]. Enzyme activation, endothelial injury, vasospasm, and ischemiareperfusion injury synergistically reduce microcirculatory perfusion[9], leading to necrosis, SIRS, multiple organ failure, and high mortality[10].

Traditional assessments have limitations. Conventional CT only enables qualitative visual evaluation and cannot quantify perfusion[4]. Serological markers such as PCT and IL6 reflect inflammation but are affected by confounding factors and show lower diagnostic efficacy than spectral CT[11]. Nevertheless, clinicians should still monitor AP patients—especially coagulation disorders and pancreatic necrosis in suspected severe cases[12].

Spectral CT, with wateriodine separation, allows quantitative assessment of pancreatic microcirculation by measuring IC and NIC, thus achieving a leap from "qualitative description" to "quantitative analysis"[5]. In this study, IC in the mild AP group (2.89 ± 0.50 mg/mL) was significantly higher than in the moderatesevere group (2.09 ± 0.72 mg/mL). IC and NIC were independent protective factors.

This study first demonstrated the early predictive value of spectral CTderived IC for moderately severe acute pancreatitis (MSAP). IC superior to conventional serological and imaging scores. Unlike previous DECT studies reporting an IC threshold of 1.63 mg/mL for SAP[13], the present higher threshold improves early identification of highrisk MSAP patients. IC < 2.7 mg/mL correlated with more severe complications, confirming a negative correlation between IC and disease severity.

This study also found that spectral CT compensates for the false-negative limitation of MCTSI. Eleven patients initially graded as mild AP by MCTSI but progressing to moderate-severe AP all had initial IC < 2.7 mg/mL, indicating quantitative iodine parameters can detect early microcirculatory abnormalities missed by visual assessment. This is consistent with Hamada et al.[14] conclusion that iodine concentration analysis allows objective evaluation of AP severity even with atypical imaging findings. This provides important early warning to avoid delayed intervention and facilitate timely intensive therapy.

5. Conclusion

Spectral CT can quantitatively measure pancreatic IC, avoiding the subjectivity of conventional CT and interference from serological markers. It objectively reflects pancreatic microcirculatory perfusion, providing a reliable index for acute pancreatitis severity grading and prognosis assessment, especially for high-risk patients with early atypical imaging. IC < 2.7 mg/mL indicates a significantly higher risk of progression to moderatesevere AP and requires intensive monitoring. Combining IC with MCTSI, procalcitonin, and IL6 helps establish a multidimensional model to improve diagnostic accuracy.

In conclusion, spectral CT has important clinical value in AP evaluation. Its quantitative parameters help guide individualized treatment and optimize intervention timing, deserving wide clinical application. Future multicenter, large-sample studies with etiological subgroup analysis and long-term follow-up are needed to verify its diagnostic threshold and improve its role in AP whole-course management.

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