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Original Article

Comparison of Interleukin-6, C-Reactive Protein, Procalcitonin, and the Computed Tomography Severity Index for Early Prediction of Severity of Acute Pancreatitis

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Background/Aims: Acute pancreatitis (AP) is a common gastrointestinal disease associated with hospitalization. With the increase in its incidence, AP has become a greater burden on healthcare resources. Early identification of patients with mild AP can facilitate the appropriate use of resources. We aimed to investigate the ability of inflammatory markers, including interleukin-6 (IL-6), procalcitonin, and C-reactive protein (CRP), as well as various scoring systems to differentiate mild AP from more severe diseases.

Methods: We retrospectively investigated patients hospitalized with AP, for whom severity assessment and clinical course confirmation were possible. Inflammatory markers were measured at admission, and CRP levels were measured 24 hours after admission (CRP2). Predictive values were calculated using the area under the receiver operating characteristic curve (AUROC) and logistic regression model analysis.

Results: Of 103 patients with AP, 42 (40.8%) were diagnosed with mild AP according to the revised Atlanta classification. Based on the AUROC, IL-6 (0.755, p<0.001), CRP2 (0.787, p<0.001), and computed tomography severity index (CTSI) (0.851, p<0.001) were useful predictors of mild AP. With standard cutoff values, the diagnostic sensitivity, specificity, and accuracy were 83.3%, 62.3%, and 70.9% for IL-6 (<50 pg/mL), and 78.6%, 63.9%, and 69.9% for CRP2 (<50 mg/L), respectively. The AUROC of IL-6 and CRP2 were significantly higher than those of other inflammatory markers and were not significantly different from that of CTSI.

Conclusions: IL-6, CRP2, and CTSI are helpful for early differentiation of AP severity. Among inflammatory markers, IL-6 has the advantage of early prediction of mild pancreatitis at the time of admission. **(Gut Liver 2023;17:629-637)**

Key Words: Acute pancreatitis; Severity; Interleukin-6; C-reactive protein

INTRODUCTION

Acute pancreatitis (AP) is an acute inflammatory disease of the pancreas and one of the most common gastrointestinal diseases for hospitalization.[1] The global incidence of AP is approximately 20 to 40 per 100,000 population and has been rising in recent decades.[2-4] With the increase in the incidence of AP, the number of hospitalizations and medical costs have increased markedly; as a result, the economic burden and consumption of health care resources due to AP are substantial.[5]

The inflammatory process in AP can affect regional and remote organ systems. Most patients with AP have interstitial pancreatitis, a mild form of the disease, and the overall mortality is reported to be approximately 5%. However, approximately 20% of patients have necrotizing pancreatitis or organ failure, and mortality rates are reported to be up to 50% in these patients.[6] Therefore, various efforts have been made to predict the severity of AP for early stratification of high- or low-risk patients and to initiate adequate treatment.

To predict the severity of AP, several scoring systems,

including clinical, radiographic, and laboratory findings, have been proposed: the Acute Physiology and Chronic Health Evaluation II (APACHE II), the Bedside Index for Severity in Acute Pancreatitis (BISAP), and the Ranson score.[7-9] However, there are inconveniences and limitations in practical use to predict AP severity given that many parameters require measurement, and evaluation requires 48 to 72 hours.

Efforts have also been made to predict AP severity using parameters that can be easily measured. The computed tomography severity index (CTSI) has been proposed for evaluation of AP severity using computed tomography (CT) findings.[10] C-reactive protein (CRP) and procalcitonin have been suggested as predictive markers for severity in various studies.[11] Interleukin-6 (IL-6), a multifunctional cytokine released by macrophages in response to tissue injury,[12] has also been proposed as an early predictor of severity in AP.[13]

To date, studies performed for the prediction of AP severity have focused on severe AP and mortality, and studies on the prediction of mild AP are lacking. Through early recognition of mild AP, unnecessary massive hydration can be avoided, and oral feeding can be provided early. In addition, the consumption of healthcare resources can be reduced through early outpatient clinic-based treatment rather than long-term hospitalization.

Therefore, we aimed to evaluate and compare the accuracy of inflammatory markers, such as CRP, IL-6, and procalcitonin, in predicting mild AP. In addition, we investigated the accuracy of various scoring systems for predicting mild AP and compared them with that of inflammatory markers.

MATERIALS AND METHODS

1. Patients and study design

This retrospective study was conducted with patients hospitalized for AP in a tertiary hospital from January 2018 to December 2021. The inclusion criteria were as follows: (1) age ≥18 years; (2) patients who met the diagnostic criteria for AP;[1] (3) patients who underwent laboratory tests, including IL-6, procalcitonin, and CRP at admission (CRP1), and additional measurement of CRP 24 hours after admission (CRP2); (4) patients who had sufficient laboratory and imaging test results to assess severity; and (5) patients whose clinical courses could be clearly identified until the completion of treatment. A total of 103 patients who met the inclusion criteria were retrospectively investigated.

AP severity was assessed according to the revised Atlanta classification.[14] Severe AP was defined as persistent organ failure (≥48 hours). Moderately severe AP was defined as transient organ failure (<48 hours) or the presence of local complications. Mild AP was defined as the absence of organ failure or local or systemic complications. Systemic inflammatory response syndrome was defined by two or more of the following criteria: heart rate of >90 beats/min, body temperature of <36°C or >38°C, white blood cell count of <4,000 or >12,000/mm^3, and respiration of >20/min or pCO_2 <32 mm Hg. Organ failure was defined according to the Modified Marshall scoring system as follows: respiratory failure (PaO_2/FiO_2 >200); acute renal failure (serum creatinine >1.9 mg/dL); or cardiovascular failure (systolic blood pressure of <90 mm Hg any time during the course of AP).[15] Patients were treated according to the accepted standard management of AP. Patients with gallstone pancreatitis or cholangitis due to common bile duct stones underwent endoscopic retrograde cholangiopancreatography, and other causal factors were immediately corrected after identifying the etiology.

This study was approved by the Gangnam Severance Hospital Institutional Review Board (IRB number: 3-2020-0164) and was conducted in accordance with the principles set forth in the Declaration of Helsinki. The requirement for informed consent was waived because of the retrospective nature of the study, and the analysis used anonymous clinical data.

2. Clinical parameters and measurement of inflammatory markers

Most of the patients underwent CT on the day of admission. Blood samples for laboratory tests were obtained at admission and at 24 and 48 hours after admission. Various scoring systems, including Ranson criteria, the APACHE II score, BISAP, and CTSI, were calculated based on laboratory and imaging studies. CRP level was measured using an immunoturbidimetry method on an AU5822 analyzer (Beckman Coulter, Brea, CA, USA). Procalcitonin and IL-6 were measured using a chemiluminescence immunoassay on Vitro XT7600 (Ortho Clinical Diagnostics, Raritan, NJ, USA) and Cobas e801 (Roche Diagnostics GmbH, Mannheim, Germany), respectively.

3. Study outcomes and statistical analysis

Continuous variables are expressed as mean±standard deviation if the data were normally distributed or else as median (interquartile range). The one-way analysis of variance was used to compare the differences in clinical characteristics among the three groups.

The area under the receiver operating characteristic curve (AUROC) was used to assess the predictive accuracy

of various inflammatory markers such as IL-6, CRP, and procalcitonin, and scoring systems such as BISAP, Ranson score, CTSI, and APACHE II score for predicting mild AP. The univariate logistic regression analysis was used to examine the association between each predictor and mild AP, and the results were expressed as odds ratios (ORs) and 95% confidence intervals (CIs).

We selected the best predictors for mild AP based on the AUROC results. Next, the predictive accuracy according to commonly used cutoff values of the selected predictors was assessed, and AUROC values were compared between predictors using the DeLong method.[16]

Statistical analyses were performed using IBM SPSS Statistics for Windows version 25.0 (IBM Corp., Armonk, NY, USA). Differences were considered statistically significant at $p<0.05$.

RESULTS

1. Baseline characteristics of the study population

During the study period, 352 AP patients visited the hospital. After investigating the medical records, we excluded 249 patients from the analysis due to the following

Table 1. Baseline Characteristics of the Study Population

Variable	Mild (n=42)	Moderately severe (n=53)	Severe (n=8)	p-value
Age, yr	50.48±20.34	47.49±17.46	51.63±19.94	0.688
Male sex	22 (52.4)	43 (81.1)	5 (62.5)	0.011
Body mass index, kg/m^2	24.13±3.72	23.78±3.97	24.89±6.20	0.748
Hypertension	8 (19.0)	14 (26.4)	4 (50.0)	0.159
Diabetes mellitus	6 (14.3)	16 (30.2)	3 (37.5)	0.103
Etiology				0.088
Biliary	17 (40.5)	15 (28.3)	1 (12.5)	
Alcoholic	9 (21.4)	27 (50.9)	3 (37.5)	
Hypertriglyceridemia	4 (9.5)	4 (7.6)	1 (12.5)	
Idiopathic	8 (19.1)	5 (9.4)	3 (37.5)	
Malignancy	1 (2.4)	1 (1.9)	0	
Pancreatic divisum	3 (7.1)	1 (1.9)	0	
White blood cell, /mm^3	9,450±3,544	13,574±5,320	12,724±7,110	<0.001*
Hematocrit, %	41.6±6.0	44.5±5.6	41.0±8.9	0.045
Creatinine, mg/dL	0.80±0.23	0.91±0.47	1.97±1.34	<0.001†,‡
Amylase, U/L	999±1,597	973±1,290	362±357	0.475
Lipase, U/L	7,345±14,083	4,799±6,978	1,186±605	0.231
Accompanying inflammatory conditions				
Cholangitis	17 (40.5)	18 (34.0)	2 (25.0)	0.644
Enteritis	7 (16.7)	9 (17.0)	4 (50.0)	0.075
Inflammatory markers				
CRP1, mg/L	24.98±52.59	50.94±96.27	73.91±102.35	0.165
CRP2, mg/L	38.91±55.95	116.57±122.65	157.51±121.02	<0.001*,†
Interleukin-6, pg/mL	261.56±843.07	356.14±743.42	674.30±940.32	0.406
Procalcitonin, ng/mL	1.46±6.79	5.20±26.37	2.10±3.61	0.638
CTSI	2 (0–4)	4 (2–8)	3.5 (1–4)	<0.001*,†
BISAP	0 (0–3)	0 (0–2)	1 (0–4)	0.169
Ranson score	1 (0–4)	2 (0–5)	3.5 (1–5)	0.001§
APACHE II score	6 (0–14)	7 (0–19)	15 (4–42)	0.007†,‡
Length of hospital stay, day	6.12±2.96	11.70±7.87	29.50±21.31	<0.001§
Organ failure	0	9 (17.0)	8 (100)	<0.001§
Intensive care unit admission	0	4 (7.5)	7 (87.5)	<0.001§
Mortality	0	0	2 (25.0)	0.005†,‡

Data are presented as mean±SD, number (%), or median (interquartile range).
CRP1, C-reactive protein at admission; CRP2, C-reactive protein 24 hours after admission; CTSI, computed tomography severity index; BISAP, Bedside Index for Severity in Acute Pancreatitis; APACHE II, Acute Physiology and Chronic Health Examination II.
*Mild versus moderately severe was significant; †Mild versus severe was significant; ‡Moderately severe versus severe was significant; §All pairwise comparisons were significant.

reasons: patients with insufficient laboratory tests (n=142), difficulty in assessing the severity through medical records (n=87), and cases where the clinical courses could not be clearly identified (n=20). The baseline characteristics of the patients are summarized in Table 1. Of the 103 patients, 42 were diagnosed with mild AP, 53 were diagnosed with moderately severe AP, and eight were diagnosed with severe AP. The mean age did not differ among the three groups, but the proportion of male patients was significantly higher in the moderately severe AP group (p=0.011). The most common etiology of AP was alcoholic (39/103, 37.9%), followed by biliary (33/103, 32.0%), idiopathic (16/103, 15.6%), hypertriglyceridemia (9/103, 8.7%), and others (6/103, 5.8%; two patients were associated with malignancy, and four had AP due to pancreatic divisum). There was no significant difference in etiology and accompanying inflammatory conditions such as cholangitis and enteritis based on the severity of the group. At the time of admission, patients with mild AP had significantly lower white blood cell counts than those with moderately severe AP. Patients with severe AP showed significantly higher serum creatinine levels than those with mild or moderately severe AP.

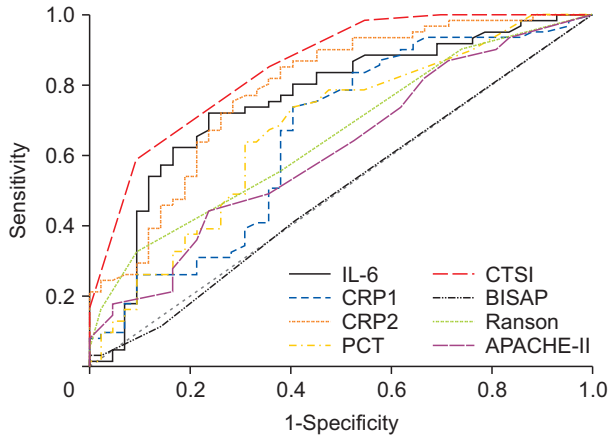

Fig. 1. Receiver operating characteristic curve of various factors for distinguishing mild acute pancreatitis from moderately severe and severe acute pancreatitis.
IL-6, interleukin-6; CRP1, C-reactive protein at admission; CRP2, C-reactive protein 24 hours after admission; PCT, procalcitonin; CTSI, computed tomography severity index; BISAP, Bedside Index for Severity in Acute Pancreatitis; APACHE II, Acute Physiology and Chronic Health Evaluation II.

The values of the inflammatory markers measured at the time of admission were not significantly different among the three groups. The CRP2 level was significantly lower in the mild AP group than that in the other two groups (mild vs moderately severe, p=0.001; mild vs severe, p=0.009). Among the various scoring systems, CTSI was significantly different between mild AP and other groups (mild vs moderately severe, p<0.001; vs severe, p=0.022), and the APACHE II score was significantly different between severe AP and other groups (severe vs mild, p=0.003; vs moderately severe, p=0.009). The Ranson scores were significantly different between all three groups, whereas the BISAP scores were not different between the groups.

The length of hospital stay, rate of organ failure, and intensive care unit admission rate increased significantly according to AP severity, and mortality (n=2) occurred only in the severe AP group.

2. The prognostic value of inflammatory markers and scoring systems for predicting mild AP

To assess the accuracy of the inflammatory markers and scoring systems for the prediction of mild AP, we measured the AUROC for each predictor (Fig. 1). Based on the AUROC, CTSI (AUROC, 0.851; 95% CI, 0.777 to 0.925; p<0.001), CRP2 (AUROC, 0.787; 95% CI, 0.696 to 0.878; p<0.001), and IL-6 (at admission: AUROC, 0.755; 95% CI, 0.656 to 0.854; p<0.001) were confirmed as strong predictors of mild AP.

To confirm the predictive accuracy of inflammatory

Table 2. Prognostic Value of Inflammatory Markers and Scoring Systems for Predicting Mild Acute Pancreatitis

Variable	AUROC	95% CI	p-value	OR	95% CI	p-value
Interleukin-6 (<50 pg/mL)	0.728	0.629–0.827	<0.001	8.261	3.155–21.632	<0.001
CRP1 (<50 mg/L)	0.572	0.461–0.683	0.218	2.631	0.881–7.860	0.083
CRP2 (<50 mg/L)	0.713	0.611–0.814	<0.001	6.500	2.633–16.044	<0.001
Procalcitonin (<0.5 ng/mL)	0.581	0.470–0.691	0.166	2.439	0.923–6.445	0.072
CTSI (≤2)	0.748	0.646–0.849	<0.001	10.400	4.029–26.843	<0.001
BISAP (≤1)	0.486	0.372–0.600	0.809	0.778	0.242–2.504	0.674
APACHE II (≤7)	0.567	0.455–0.680	0.247	1.742	0.778–3.902	0.177
Ranson (≤2)	0.616	0.508–0.724	0.046	4.634	1.452–14.793	0.01

AUROC, area under the receiver operating characteristic curve; CI, confidence interval; OR, odds ratio; CRP1, C-reactive protein at admission; CRP2, C-reactive protein 24 hours after admission; CTSI, computed tomography severity index; BISAP, Bedside Index for Severity in Acute Pancreatitis; APACHE II, Acute Physiology and Chronic Health Evaluation II.

markers and scoring systems according to commonly used cutoff values for mild AP, AUROC measurements and logistic regression analysis were performed (Table 2). As a result, a CTSI of ≤2 (OR, 10.400; 95% CI, 4.029 to 26.843; p<0.001), IL-6 of <50 pg/mL (OR, 8.261; 95% CI, 3.155 to 21.632; p<0.001), CRP2 of <50 mg/L (OR, 6.500; 95% CI, 2.633 to 16.044; p<0.001), and Ranson score of ≤2 (OR, 4.634; 95% CI, 1.452 to 14.793; p=0.01) had significantly elevated ORs for the prediction of mild AP versus moderately severe and severe AP. However, AUROC value did not increase when each of these three markers was combined (Supplementary Table 1).

3. Comparison of different inflammatory markers and CTSI in predicting mild AP versus moderately severe and severe AP

We compared the AUROC for the prediction of mild AP using the following inflammatory markers: CRP1, CRP2, procalcitonin, and IL-6 (Fig. 2). It was confirmed that the AUROCs of IL-6 and CRP2 were significantly higher than those of the other inflammatory markers. When comparing the AUROC of these two inflammatory

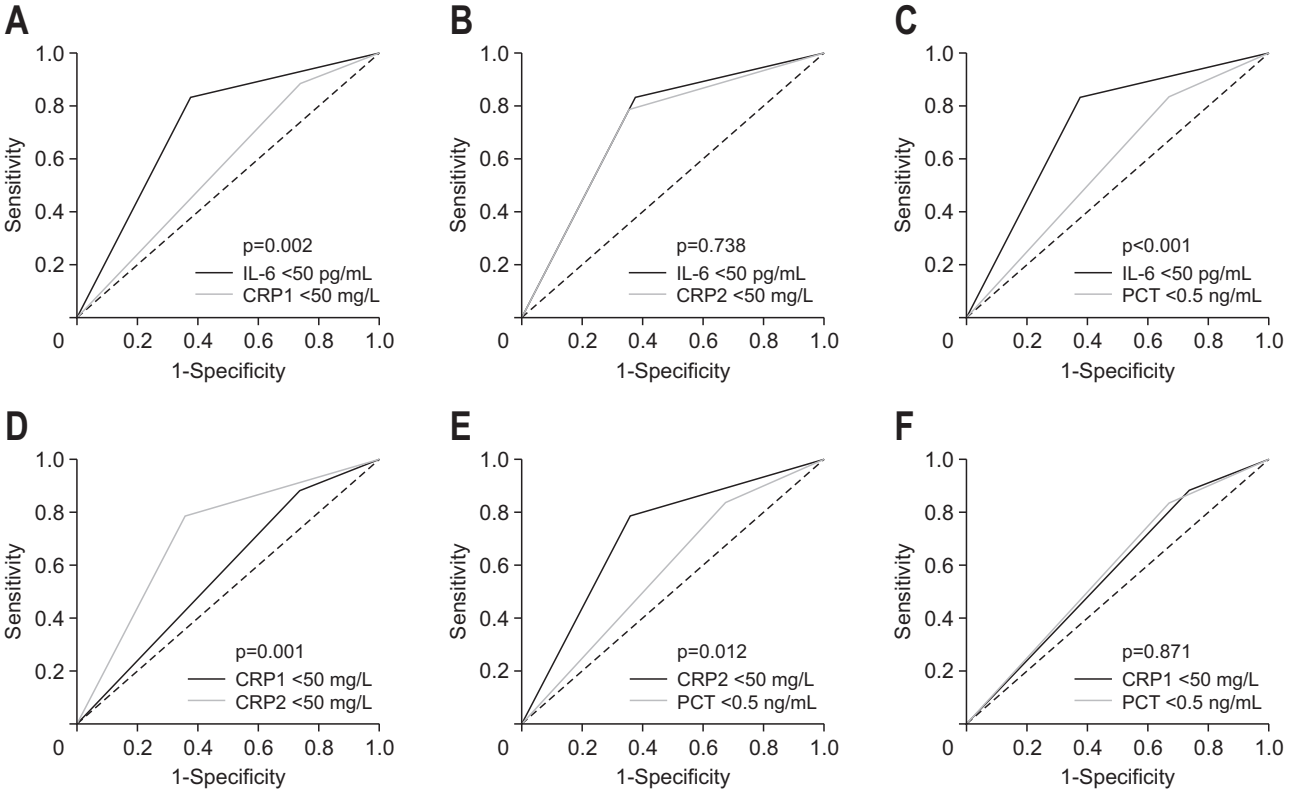

Fig. 2. (A-F) Comparison of area under the receiver operating characteristic curve (AUROC) between inflammatory markers (according to the commonly used cutoff values). IL-6 and CRP2 had significantly greater AUROCs than other markers.
IL-6, interleukin-6; CRP1, C-reactive protein at admission; CRP2, C-reactive protein 24 hours after admission; PCT, procalcitonin.

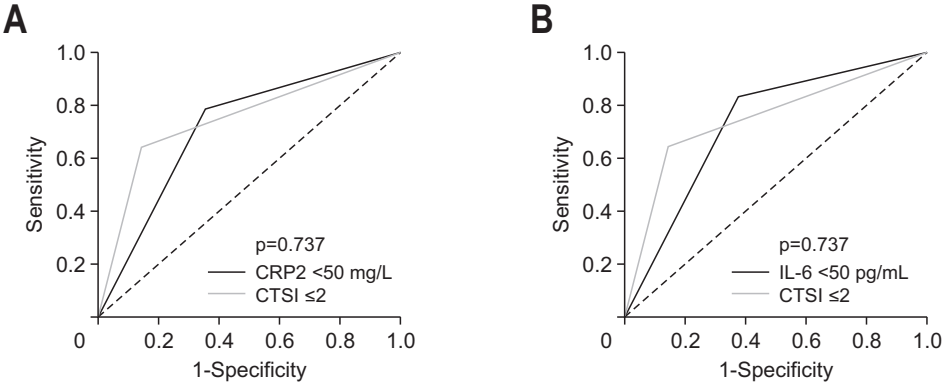

Fig. 3. Comparison of area under the receiver operating characteristic curve between (A) CRP2, (B) IL-6 and CTSI (≤2). There was no statistically significant difference.
CRP2, C-reactive protein 24 hours after admission; CTSI, computed tomography severity index; IL-6, interleukin-6.

markers with CTSI, there was no statistically significant difference (Fig. 3).

DISCUSSION

In this study, it was confirmed that serum IL-6, CRP2, and CTSI were useful for predicting mild AP. These parameters were relatively easy to measure and more accurate than other inflammatory markers and complex scoring systems. Although early recognition of patients most likely to have a mild course can provide practical guidance in the early management of AP, studies on this are still lacking. Therefore, our study may be useful to physicians treating patients with AP in emergency departments or inpatient clinics.

In patients with AP, immediate and adequate fluid resuscitation is very important to prevent pancreatic necrosis and organ failure.[17-19] However, there are concerns that massive hydration can be associated with respiratory complications or abdominal compartment syndrome, so clinical guidelines recommend goal-directed therapy in fluid resuscitation using mean arterial pressure and urine output as clinical parameters.[20,21] To restore oral or enteral nutrition, fasting and "bowel rest" have traditionally been recommended. Recently, it was reported that early oral or enteral nutrition can prevent systemic complications and reduce morbidity and mortality.[22,23] However, when adopting this early refeeding strategy, the physician's concern arising from the traditional viewpoint and the patients' persistent pain can be hurdles in clinical practice. If mild AP can be predicted in the early course of the disease, massive fluid resuscitation can be avoided, and early refeeding can be safely performed more actively without concern.

Prophylactic antibiotic use is not recommended in all patients with AP without evidence of infection, even in those with severe AP and necrotizing pancreatitis. However, the frequency of antibiotic use is high because many patients with AP show leukocytosis and/or fever at diagnosis, and other infectious diseases cannot be completely excluded.[24] One retrospective study reported global overuse of antibiotics (31% to 82% of frequency) in AP management.[25] Administration of antibiotics, massive hydration, and "bowel rest" inevitably require a considerable period of hospitalization, which in turn carries a significant burden on the health care system.[26] Thus, early recognition of mild AP can also diminish unnecessary antibiotic use and reduces the medical burden.

Both IL-6 and CRP are known as inflammatory markers that predict severe AP and are useful in the early phase of AP. However, between these two markers, there was a difference in the time to reach the peak level after the onset of AP. According to the previous study that measured the plasma concentrations of two inflammatory markers in the early phase of AP, the peak level of IL-6 appeared between 24 and 36 hours, and CRP reached its peak level at 36 and 48 hours after symptom onset.[27] Other studies have also reported that the useful measurement point to predict severe AP of IL-6 is earlier than CRP.[28-30] In the present study, IL-6 and CRP2 levels showed similar predictive values in predicting mild AP.

When comparing patients with IL-6 levels of <50 pg/mL and ≥50 pg/mL among patients confirmed with mild AP, patients with low IL-6 levels tended to have shorter hospital stays (5.89 days vs 7.29 days, p=0.258) and lower CRP levels (15.05 mg/L vs 74.65 mg/L at admission, p=0.137; 27.90 mg/L vs 93.93 mg/L at 24 hours, p=0.070). In addition, the BISAP, Ranson, and APACHE II scores were significantly lower (Supplementary Table 2). Not all patients with low IL-6 levels at admission showed a mild course of disease. Although mortality was not identified in patients with low IL-6 levels, two patients underwent intensive care unit care, and one patient showed severe AP with persistent organ failure lasting more than 48 hours. One patient who received transient intensive care unit care had a high CTSI score of 4 at the time of admission and required a drainage procedure for the pseudocyst. One patient with persistent organ failure had a high BMI (29 kg/m^2) and a high BISAP score of 4 (presence of systemic inflammatory response syndrome, impaired mental status, pleural effusion, and elevated blood urea nitrogen). Therefore, even if IL-6 levels are low, careful monitoring is required when other worrisome findings exist.

At the time of admission, 37 of 103 patients had accompanying cholangitis and 20 patients had enteritis. Patients with cholangitis showed higher total bilirubin (2.41 mg/dL vs 1.59 mg/dL, p=0.068), IL-6 (578 pg/mL vs 210 pg/mL, p=0.070) and procalcitonin (6.36 ng/mL vs 1.80 ng/mL, p=0.386) than patients without cholangitis. Patients with enteritis showed higher white blood cell (12,144/mm^3 vs 11,749/mm^3, p=0.812), total bilirubin (2.04 mg/dL vs 1.83 mg/dL, p=0.731), IL-6 (345 pg/mL vs 341 pg/mL, p=0.985), and CRP2 (100.3 mg/L vs 85.1 mg/L, p=0.578) levels. Although there were no statistically significant differences, accompanying other inflammatory conditions can affect to inflammatory markers such as IL-6 and CRP. Therefore, it is necessary to consider other accompanying inflammatory conditions when interpreting the laboratory values.

Procalcitonin is known as an early marker of systemic bacterial infection and sepsis.[31] In previous studies, procalcitonin was useful in predicting severe AP or infected

necrosis of the pancreas.[32,33] In this study population, procalcitonin also showed a high AUROC value for predicting severe AP (AUROC, 0.813; 95% CI, 0.724 to 0.903, data not shown). However, while predicting mild AP, procalcitonin showed a lower predictive value than CRP or IL-6. This is thought to be due to the clinical characteristics of mild AP, which is rarely related with bacterial infections.

CT is the most important imaging test for the diagnosis of AP and evaluation of complications. Although there is a possibility that CT performed in the early phase of AP may underestimate necrosis or severity,[34] it is unavoidable to initially perform CT because most AP patients visit the emergency department with epigastric pain, which requires a differential diagnosis. In this study, 91.7% (100/103) of patients had undergone CT on the day of the hospital visit (of the remaining three patients, two patients underwent CT on day 2 of hospitalization and one patient on day 3 of hospitalization due to renal insufficiency and poor compliance), and CTSI was calculated based on these findings. There may be limitations in predicting severe AP, but CTSI was identified as an accurate parameter in predicting mild AP, along with IL-6 and CRP2. Among the patients who showed favorable findings for both CTSI (≤2) and IL-6 (<50 pg/mL), only one patient required intensive care unit care (mentioned above), and two patients showed necrotizing features. All other patients showed favorable clinical courses, and no mortality was identified.

It is important to note that fluid resuscitation and close monitoring that take place in the hospital cannot be overlooked even in mild AP patients. Evidence that fluid resuscitation is not required in a certain group of AP patients is still lacking. Additionally, there are still limitations when predicting mild AP using inflammatory markers individually or in combination with CTSI and there is a possibility of misdiagnosing moderately severe and severe AP as mild AP. Therefore, inflammatory markers should not be used solely for predicting severity. Monitoring of other clinical parameters and adequate initial management is important to prevent fatal disease course. In this context, a previous study had proposed a course of treatment for mild AP patients that included initial management in the emergency department for 12 to 24 hours.[35] If further studies are conducted on the appropriate discharge time for patients with mild AP, in addition to our findings, more precise guidelines can be established and decision-making for discharge will become easier.

This study has some limitations. First, this was a retrospective study conducted in a single institution; therefore, there may be bias in patient selection and interpretation of results. Particularly, there is a possibility of selection bias because IL-6 and CRP2 were not routinely administered to all patients. The exact time interval between symptom onset and blood collection for laboratory examination could not be measured. Second, there was no external validation set, owing to the limited number of patients. In addition, the timing of the CT scan was not constant because most patients visited the hospital through emergency department, and there was a possibility that a moderately severe AP was overestimated. Despite these limitations, this study has several strengths. Inflammatory markers, such as IL-6, CRP, and procalcitonin, and various parameters necessary for the scoring systems were systematically measured and compared in all patients. In addition, follow-up was completed in all patients until AP treatment was completed. Based on these strengths, we attempted to overcome the limitations of the retrospective study and provide practical information.

In conclusion, assessment of IL-6 at admission and CRP2 showed acceptable performance in discriminating mild AP from more severe cases. These inflammatory markers showed similar predictive values to CTSI and were more useful than other scoring systems. Although AP severity should not be predicted through these markers alone, early identification of patients expected to have mild AP can be made possible through the assessment of these markers, so that these patients can be discharged early and subsequently managed in an outpatient clinic. In particular, because the predictive value of IL-6 measured during hospitalization is similar to that of CTSI and CRP2, it can facilitate clinical decision-making.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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AUTHOR CONTRIBUTIONS

Study concept and design: I.R.C., S.I.J., J.H.C. Data acquisition: M.Y.D., S.Y.H. Data analysis and interpretation: I.R.C., M.Y.D., J.H.C. Drafting of the manuscript: I.R.C. Critical revision of the manuscript for important

intellectual content: S.I.J., J.H.C. Statistical analysis: I.R.C. Obtained funding: J.H.C. Administrative, technical, or material support: M.Y.D., S.Y.H. Study supervision: S.I.J., J.H.C. Approval of final manuscript: all authors.

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SUPPLEMENTARY MATERIALS

Supplementary materials can be accessed at https://doi.org/10.5009/gnl220356.

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