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RESEARCH ARTICLE

The Role of 48-Hour CRP/Albumin Ratio in The Differential Diagnosis of Interstitial and Necrotizing Pancreatitis

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Abstract

Introduction: In this study, we aimed to examine the prognostic importance of the c – reactive protein (CRP)/albumin ratio in predicting acute necrotizing pancreatitis.

Methods: The study included 100 patients diagnosed with acute interstitial pancreatitis and 50 patients diagnosed with necrotizing pancreatitis between 2015 and 2020, all over the age of 18. **Results:** The CRP/albumin ratio was higher in the acute necrotizing pancreatitis group compared to the interstitial pancreatitis group (94.0 vs 34.0; p <0.001, respectively). Multivariable analysis revealed CRP/albumin ratio [OR: 1.075 (1.029-1.123), p = 0.001], source of infection [OR: 4.698 (2.078-10.620), p < 0.001], and lactate dehydrogenase [OR: 1.006 (1.002-1.010), p = 0.004] to be significantly predictive of developing necrotizing pancreatitis. A prediction value of CRP/albumin ratio >70.6 was found to be a significant marker in predicting necrotizing pancreatitis (Sensitivity: 76.0%; Specificity: 85.4%; AUC: 0.881; p < 0.001). In addition, to demonstrate the nonlinear relationship between CRP/albumin and the probability of necrotizing pancreatitis, cubic spline regression analysis was applied, showing that the probability of necrotizing pancreatitis increased in relation to the increase of the CRP/albumin ratio.

Conclusion: We conclude that the CRP/albumin ratio is a predictor of acute necrotizing pancreatitis. We believe that the CRP/albumin ratio is an inexpensive, sensitive, and easy-to-use predictor of acute necrotizing pancreatitis that can be used for both diagnosis and treatment follow-up.

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Introduction

Acute necrotizing pancreatitis is a severe form of acute pancreatitis associated with high morbidity and mortality that is characterized by the necrosis of the pancreatic parenchyma and/or the surrounding tissues.[1] It accounts for approximately 10% to 20% of all acute pancreatitis cases. About one-fourth of patients with necrotizing pancreatitis have a severe prognosis. The introduction of infection of the necrosis, gastrointestinal perforation, and bleeding into the clinical picture can increase the mortality rate up to 98%.[2] The prognosis in acute necrotizing pancreatitis cases is therefore fundamentally dependent on two factors: persistence of organ failure and secondary infection of pancreatic necrosis.

One of the important steps to reduce mortality in acute necrotizing pancreatitis is to understand the severity of the event and to plan the disease management accordingly before necrosis develops in and around the pancreatic parenchyma. This requires prognostically significant predictors of necrosis. Our review of the literature revealed that a large number of clinical and biochemical scoring systems have been assessed for this purpose. These primarily include the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, the Bedside Index of Severity in Acute Pancreatitis (BISAP), the Glasgow Pancreatitis Score, the Harmless Acute Pancreatitis Score (HAPS), the Japanese Severity Score (JSS), PANC3, the Pancreatitis Outcome Prediction (POP) score, the Ranson criteria, C-reactive protein (CRP), and systemic inflammatory response syndrome (SIRS).[3] However, among these scoring systems, only the Glasgow Pancreatitis Score and serum CRP levels provide pragmatic prognostic accuracy early on. Recently, the CRP/albumin ratio was used to predict the prognosis of acute edematous pancreatitis.[4]

Recent studies have used the CRP/albumin ratio as a prognostic marker for the severity of inflammation and mortality in chronic inflammatory diseases.[5-10] Since CRP is an acute-phase reactant that increases in inflammatory processes while albumin decreases in inflammatory processes, we believe that the CRP/albumin ratio can be used as a highly sensitive index for inflammatory diseases.

This study aims to investigate the prognostic significance of the CRP/albumin ratio in predicting acute necrotizing pancreatitis.

Materials and Methods

This retrospective study was conducted in the General Surgery Clinic of Ankara City Hospital between May 1 and June 1, 2020. The study was designed in accordance with the 2013 Brazil revision of the Helsinki Declaration and good clinical practice guidelines. The study was granted ethical approval by the Ankara City Hospital Ethics Committee.

The study included 100 patients diagnosed with acute interstitial pancreatitis and 50 patients diagnosed with necrotizing pancreatitis between 2015 and 2020, all over the age of 18. These cases were randomly included in the study in order of application.

The exclusion criteria were as follows: (a) being diagnosed with acute pancreatitis but refusing hospitalization, (b) being discharged during hospitalization of one's own accord, (c) having a preliminary acute pancreatitis diagnosis but dying before confirmation, (d) having non-pancreatitis-related infection foci, (e) severe malnutrition, (f) malignancy, (g) rheumatological or other diseases associated with chronic inflammatory processes, and (h) receiving immunosuppressive therapy for any reason.

Acute pancreatitis was diagnosed according to the National Pancreas Foundation criteria based on medical history, physical examinationfindings, amylase and lipase levels greater than 3 times the normal values, and/or computed tomography findings.[11] Pancreatitis diagnosis was confirmed using ultrasonography, computed tomography, or magnetic resonance imaging.

Clinical findings (respiratory rate, heart rate, Glasgow Coma Score, and body temperature), demographic findings (age, gender, comorbid conditions, antibiotic use, recurrent/chronic pancreatitis status, infection foci, length of hospital stay, disease severity, complications, and clinical outcome), laboratory findings [complete blood count, biochemistry, CRP, and erythrocyte sedimentation rate (ESR)], and radiological findings were retrospectively recorded from electronic and clinical files. Disease severity is classified as mild, moderate, or severe depending on the absence or presence of organ failure and local or systemic complications. Moderately severe acute pancreatitis is associated with transient organ failure that lasts <2 days, while severe acute pancreatitis is defined by organ failure that persists for ≥2 days.[12]

The CRP/albumin ratio was calculated by dividing the CRP level directly by the albumin level. The 48-hour laboratory parameters of the patients [white blood cells (WBCs), neutrophils, lymphocytes, monocytes, hemoglobin, platelets, CRP, ESR, blood glucose, urea, creatinine, sodium, potassium, calcium, albumin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma glutamyltransferase (GGT), lactate dehydrogenase (LDH), total bilirubin, direct bilirubin, amylase, lipase, and triglyceride] were recorded from patient files.

Statistical Analysis

Categorical variables were presented in frequency tables. Continuous variables were presented (mean, standard deviation (SD), or median, and interquartile ranges between 25% and 75%, as appropriate. Binary comparisons of numerical variables not conforming to a normal distribution were carried out using the Mann-Whitney U test. A p-value of less than 0.05 was accepted to indicate a statistical significance The receiver operating characteristic (ROC) curve was used to evaluate the performance of CRP albumin ratio. Additionally, to show the non-linear relationship between CRP alb ratio and necrotizing pancreatitis, we used cubic spline regression analysis and plotted the relationship between CRP albumin ratio and probability of necrotizing pancreatitis patients. Stata 15.0 Mac program was used for conducting the statistical analysis to evaluate the findings obtained from the study.

Results

Clinical and demographic characteristics of the study population are presented in detail in Table I. A total of 150 patients (100 with interstitial pancreatitis and 50 with necrotizing pancreatitis) were included in the study. The patients with interstitial pancreatitis (60.1 ± 18.0 years) were older than those with necrotizing pancreatitis (51.6 ± 17.2 years) (p = 0.007). The two groups were not significantly different in terms of gender distribution [interstitial pancreatitis, 56% female (n = 56); necrotizing pancreatitis, 44% female (n = 22)] (p = 0.17). The two groups were similar in terms of the presence of any comorbidity [interstitial pancreatitis, 48% (n = 48), necrotizing pancreatitis, 42% (n = 21)] (p = 0.49). As shown in Table I, necrotizing pancreatitis patients had significantly higher 48-hour respiratory rate (p = 0.001), 48-hour heart rate (p = 0.010), 48-hour Glasgow Coma Score (p = 0.008), 48-hour fever (p = 0.004), antibiotic use (p = 0.010), recurrent pancreatitis (p = 0.003), chronic pancreatitis (p = 0.004), duration of levels (p < 0.001), stay in intensive care unit (p < 0.001), and complication rates (p < 0.001) compared to interstitial pancreatitis patients. Four (8%) necrotizing pancreatitis and 2 (2%) interstitial pancreatitis patients died during hospitalization; this difference was not statistically significant. Other clinical and demographic characteristics of the study population are presented in Table I.

All laboratory findings are presented in Table II. CRP / albumin ratio was significantly higher in the necrotizing pancreatitis group compared to the interstitial pancreatitis group (94.0 vs 34.0; p <0.001, respectively). Several inflammatory mar-

Table 1. Clinical and demographic findings of the study population

Variable	Interstitial pancreatitis n(50)	Necrotizing pancreatitis n(100)	p
Age, (year)	60.1 (18.0)	51.6 (17.2)	0.007
Female, (%)	56 (56.0)	22 (44.0)	0.17
Radiological imaging, (%)			
No imaging	9 (9.0)	0 (0.0)	0.040
Computed Tomography	85 (85.0)	50 (100.0)	
Magnetic resonance	2 (2.0)		0 (0.0)
Ultrasonography	4 (4.0)	0 (0.0)	
Presence of comorbidity, (%)	48 (48.0)	21 (42.0)	0.49
48th – h breath count, (%)			
<21	92 (92.0)	36 (72.0)	0.001
>20	8 (8.0)	14 (28.0)	
Heart rate, (%)			
<91	95 (95.0)	41 (82.0)	0.010
>90	5 (5.0)	9 (18.0)	
48th – h GCS, (%)			
15	99 (99)	45 (90)	0.008
<15	1 (1)	5 (10)	
48th – h high fever, (%)			
Yes	89 (89.0)	35 (70.0)	0.004
No	11 (11.0)	15 (30.0)	
Antibiotic use, (%)	83 (84)	49 (98)	0.010
Infection source, (%)			
Not detected	24 (24)	2 (4)	<0.001
Pneumonia	3 (3)	2 (4)	
Urinary tract	5 (5)	0 (0)	
Gastroenteritis	2 (2)	1 (2)	
Biliary tract	63 (64)	5 (10)	
Infected pancreatic necrosis	0 (0)	7 (14)	
Peripancreatic abscess	2 (2)	5 (10)	
Pancreatic necrosis	0 (0)	8 (56)	
Recurrent pancreatitis, (%)	16 (16.0)	19 (38.0)	0.003
Chronic pancreatitis, (%)	5 (5.0)	10 (20.0)	0.004
Duration of hospitalization, (day)	8.9 (5.3)	28.2 (27.8)	<0.001
Intensive care requirement, (%)	12 (12.0)	18 (36.0)	<0.001
Severity, (%)			
Mild	39 (39.0)	0 (0.0)	<0.001
Middle	56 (56.0)	36 (72.0)	
Severe	5 (5.0)	14 (28.0)	
Complication, (%)			
Not detected	43 (90)	7 (28)	<0.001
Pseudocyst	4 (8)	2 (8)	
Wall of necrosis		0 (0)	8 (32)
Thrombosis	0 (0)	4 (16)	
Extra pancreatic complication		0 (0)	3 (12)
Mortality, (%)	2 (2.0)	4 (8.0)	0.077

Numerical variables were expressed as mean ± standard deviation or median (min-max).
Categorical variables were shown as numbers (%). * p <0.05 shows statistical significance.
Abbreviations: GCS: Glasgow Coma Scale

Table 2. Laboratory findings of the study population

Variable	Interstitial pancreatitis 100	Necrotizing pancreatitis 50	p
White Blood Cell Count, x10³/mL	10.7 (5.3)	15.2 (5.5)	<0.001
Hemoglobin, gr/dL	11.8 (1.7)	12.6 (2.1)	0.007
Neutrophil, x10³/mL	10.4 (4.3)	13.8 (4.7)	<0.001
Lymphocyte, x10³/mL	1.7 (3.3)	1.8 (1.3)	0.84
Monocyte, x10³/mL	0.7 (0.4)	1.0 (0.5)	<0.001
Platelet, x10³/mL	238.5 (72.3)	279.0 (99.6)	0.005
Blood glucose, mg/dL	144.6 (52.4)	177.2 (75.2)	0.002
Urea, mg/dL	23.4 (15.2)	30.3 (27.0)	0.048
48th – h Creatinine, mg/dL	0.8 (0.3)	1.0 (0.8)	0.10
ALT, U/L	216.3 (248.0)	98.4 (152.1)	0.002
AST, U/L	257.6 (302.2)	131.4 (215.4)	0.009
GGT, U/L	305.9 (311.5)	186.0 (246.0)	0.019
Amilase, U/L	1550.2 (1158.7)	1229.9 (1190.2)	0.12
LDH, U/L	235.4 (132.3)	480.2 (350.8)	<0.001
Totat biluribine, mg/dL	2.0 (2.1)	1.1 (0.7)	0.003
Direct biluribine, mg/dL	1.2 (1.7)	0.5 (0.4)	0.002
ALP, U/L	177.2 (154.5)	102.6 (45.8)	0.001
Albumin, g/dL	3.4 (0.4)	3.0 (0.6)	<0.001
CRP, mg/L	123.4 (89.9)	291.2 (114.7)	<0.001
CRP/albumin	34.0 (1.3 – 100.7)	94.0 (36.4 – 174.6)	<0.001
Eritrocyte Sedimentation Rate, mm/h	34.1 (23.9)	47.0 (32.1)	0.12
Lipase, U/L	685.9 (665.8)	420.3 (350.5)	0.15
Trygliceride, mg/dL	180.4 (257.6)	350.4 (386.0)	0.042
48th – h calcium mg/dL	8.5 (0.4)	8.3 (0.9)	0.69
Sodium, mEq/L	138.2 (3.6)	138.1 (4.7)	0.88
Potassium, mmol/L	4.0 (0.4)	4.1 (0.6)	0.27
Calcium, mg/dL	8.3 (0.5)	7.9 (0.8)	<0.001

Numerical variables were expressed as mean ± standard deviation or median (min-max).
Categorical variables were shown as numbers (%). * p <0.05 shows statistical significance.
Abbreviations: ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, GGT: Gamma Glutamyl Transferase, LDH: Lactate Dehydrogenase, ALP: Alkaline Phosphatase, CRP: C–Reactive Protein

kers were significantly higher in the necrotizing pancreatitis group compared to the interstitial pancreatitis group [WBCs ($p < 0.001$), neutrophils ($p < 0.001$), monocytes ($p < 0.001$), platelets ($p = 0.005$), LDH ($p < 0.001$), CRP ($p < 0.001$)], whereas ALT ($p = 0.002$), AST ($p = 0.009$), GGT ($p = 0.019$), and albumin ($p < 0.001$) were significantly lower. Multivariable analysis revealed CRP/albumin ratio [OR: 1.075 (1.029-1.123), p = 0.001], source of infection[OR: 4.698 (2.078-10.620), p < 0.001], and LDH [OR: 1.006 (1.002-1.010), p = 0.004] to be significantly predictive of developing necrotizing pancreatitis. As shown in Figure 1, A prediction value of CRP/albumin ratio >70.6 was found to be a significant marker in predicting necrotizing pancreatitis (Sensitivity: 76.0%; Specificity: 85.4%; AUC: 0.881; $p < 0.001$). In addition, to demonstrate the nonlinear relationship between the CRP/albumin ratio and the probability of necrotizing pancreatitis, cubic spline regression analysis was applied, showing that the probability of necrotizing pancreatitis increased in relation to the increasing CRP/albumin ratio (Table III) (Figure 2).

Table 3. Detection of risk factors associated with necrosis pancreatitis by multivariable regression analysis

Variables	Odds Ratio	Std. Err.	z	P>z	95% C.I. lower	95% C.I. upper
Age	.9598371	.0259399	-1.52	0.129	.9103188	1.012049
CRP/Albumin	1.074986	.0238381	3.26	0.001	1.029265	1.122738
Female	10.40612	16.14939	1.51	0.131	.4969342	217.9109
Presence of comorbidity	.5065307	.5006747	-0.69	0.491	.0729869	3.515333
Infection source	4.697751	1.955032	3.72	0.000	2.078026	10.62011
Recurrent pancreatitis	11.19728	18.32343	1.48	0.140	.453097	276.7159
Chronic pancreatitis	584.1837	1313.316	2.83	0.005	7.127623	47880
Lactat dehidrogenaza	1.006039	.0021074	2.87	0.004	1.001917	1.010178
Calcium	.4171726	.3710082	-0.98	0.326	.0729976	2.384093
48th – h breath count	9.116213	19.52286	1.03	0.302	.137063	606.3294
48th – h high fever	1.252869	1.710829	0.17	0.869	.0862114	18.20733

Abbreviations: CI: Confidence Intervals,

Discussion

In our study, the CRP/albumin ratio was higher in the acute necrotizing pancreatitis group compared to the interstitial pancreatitis group. Multivariable regression analysis revealed the CRP/albumin ratio to be an independent risk factor associated with necrotizing pancreatitis. Cubic spline regression analysis indicated that the probability of necrotizing pancreatitis increased in relation tothe increasing CRP/albumin ratio. To the best of our knowledge, this is the first study to investigate whether the CRP/albumin ratio plays a role in predicting acute necrotizing pancreatitis. The CRP/albumin ratio has been used as a prognostic and predictive marker in many diseases in the literature. Initial publications often examined CRP/albumin in the context of malignancy[10-13-17] and septic shock,[6,18] whereas later studies investigated the role of CRP/albumin in different disease groups.[19-22] CRP is very valuable in acute response in inflammatory conditions due to its short half-life. CRP levelsthe treatment of acute pancreatitis cases.[23] Wang et al. showed that low albumin and high CRP were associated with poor clinical outcome.[24] Since necrotizing pancreatitis is a poor outcome of acute pancreatitis, CRP and albumin may have prognostic significance in predicting necrotizing pancreatitis. Kaplan et al. examined the prognostic significance of the CRP/albumin ratio in 192 cases of acute pancreatitis.

They found the CRP/albumin ratio to be higher in acute pancreatitis patients who died compared to surviving patients. The CRP/albumin ratio was found to be an independent risk factor for mortality in acute pancreatitis. They also found a positive correlation between CRP/albumin ratios and Ranson scores (the prognostic significance of which has been demonstrated for acute pancreatitis), the Atlanta classification, hospitalization time, CRP, and ESR.4 In our study, the CRP/albumin ratio was higher in the group with necrotizing pancreatitis, which is a poor clinical outcome of acute pancreatitis, compared to the interstitial pancreatitis group. Hence, taking into account both our results and those of Kaplan et al.,[4] it can be inferred that for worse clinical outcome in acute pancreatitis, the CRP/albumin ratio and its sensitivity will be higher. This is supported by our cubic spline regression analysis finding that the probability of necrotizing pancreatitis increased in relation to the increasing CRP/albumin ratio. are often used at admission and in the follow-up of An important advantage of our study compared to the study by Kaplan et al.[4] is the high number of cases of necrotizing pancreatitis, a rare complication of acute pancreatitis, as well as the finding that the CRP/albumin ratio is useful in thedifferential diagnosis of interstitial pancreatitis and necrotizing pancreatitis. A further advantage 8-hour CRP and albumin levels, whereas Kaplan et al. used admission CRP and albumin levels to calculate CRP/albumin ratios.[2] Patients with acute pancreatitis commonly present with dehydration and intravascular volume reduction due to excessive volume depletion. This can translate into a dilutional increase in the molecules in the intravascular space, and this may affect the results of the study.

Yılmaz et al. evaluated the CRP/albumin ratio of patients with moderate to severe acute pancreatitis. They found that the CRP/albumin ratio was higher in the 60 patients with severe acute pancreatitis compared to the 204 patients with moderate acute pancreatitis.[25] However, the relationship between CRP/albumin ratio and poor prognostic factors has not been studied in detail.

Apart from these studies, we did not find any studies in the literature that examined the relationship between acute pancreatitis and the CRP/albumin ratio. One study evaluated the CRP/albumin ratio in patients with severe acute ulcerative colitis, a disease that progresses similarly to

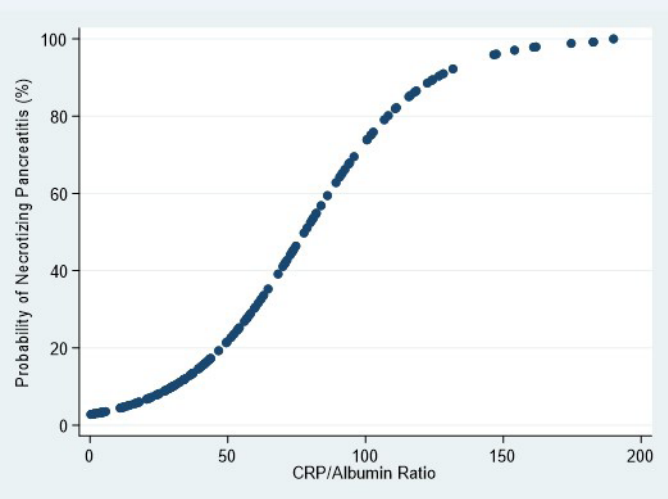

Figure 2. Cubic spline regression analysis for showing the probability of necrotizing pancreatitis increased in relation to the increasing CRP/albumin ratio

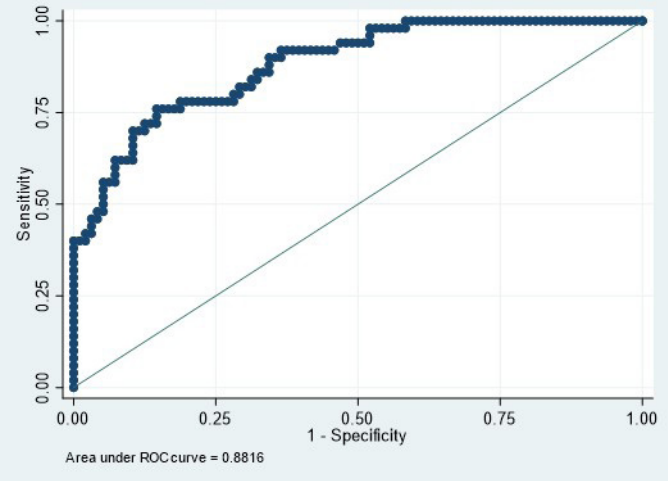

Figure 1. A prediction value of CRP/albumin ratio for predicting necrotizing pancreatitis

acute pancreatitis. The authors found that the CRP/albumin ratio can be used to differentiate severe ulcerative colitis from moderate and mild ulcerative colitis with a high value of the area under the curve.[26] A similar study investigated the role of the CRP/albumin ratio in Crohn's disease, a condition characterized by chronic inflammation, and found that the CRP/albumin ratio was associated with Crohn's disease activity.[19] The most important limitation of our study is its retrospective design. A prospective design may enable researchers to evaluate how the increased CRP/albumin ratio affects treatment response in necrotizing pancreatitis patients and the evaluation of its utility in treatment follow-up.A second limitation is that

albumin is a parameter closely associated with nutritional status. Although we excluded patients with any nutritional disorders indicated in their patient files from this study, there may have been some cases that were overlooked due to the retrospective design of the study. is that we calculated the CRP/albumin ratio using 4

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