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Severity markers in patients with acute pancreatitis

Research Article

Abdullah Kisaoglu*, Bulent Aydinli, Gurkan Ozturk, Sabri Selcuk Atamanalp, Bunyami Ozogul, Mehmet Ilhan Yildirgan, Kamil Yalcin Polat

Department of General Surgery, Faculty of Medicine, Ataturk University, Erzurum, Turkey

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Abstract: To evaluate the effectiveness of serum levels of resistin and CD14 expression in monocytes, and high-sensitivity C-reactive protein (hsCRP) in early stages of acute pancreatitis and correct prediction of the severity of acute pancreatitis (AP) using scoring systems. The study involved 10 (29.41%) male and 24 (70.59%) female patients (total n=34) followed for AP diagnosis at the Department of General Surgery, Ataturk University Medical School between July 2008 and September 2009. In all the patients, Ranson and APACHE II scores, serum resistin, hsCRP, and monocyte CD14 expression levels were determined. The patients were divided into two groups as mild and severe AP groups. A control group was formed and the intergroup comparisons were made. Values ≥ 3 based on the Ranson scoring scale and values ≥ 8 in APACHE II scoring scale were considered to indicate severe AP. Evaluations were based on the values obtained on the 1st and 7th days for serum resistin and hsCRP levels and monocyte CD 14 expression. In 17 (50%) patients, severe AP was determined. No statistically significant differences were found between the mean serum resistin levels of AP groups, while the difference for the same parameter between the mild and severe AP groups and the control group was statistically significant. In the severe AP group, the mean 1st day and 7th day serum hsCRP levels were statistically significantly higher. The CD14 expression in monocytes was similar in all the groups. Serum hsCRP concentrations and Ranson and APACHE II scores and serum resistin and hsCRP concentrations on the 1st day were positively correlated. Serum hsCRP measurement is effective in determining the severity of acute pancreatitis. Serum resistin measurement may be a useful early marker in determining the inflammatory response in AP. However, CD14 expression in monocytes was not found to be a useful marker in the diagnosis and prediction of the disease severity in AP patients.

Keywords: *Acute pancreatitis • Severity • Resistin • hsCRP • CD14 expression in monocytes*

1. Introduction

Acute pancreatitis (AP) is a nonbacterial pancreas inflammation clinically characterized with acute abdominal pain and elevated serum and urinary levels of pancreatic enzymes [1-4]. In 80% of the patients, it presents in the form of acute edematous pancreatitis (AEP) that is self-limited and mild, rarely progresses with local and systemic complications, and can be improved with general supportive therapy. In the remaining 20 %, it is in the form of acute necrotizing pancreatitis with probable organ failures and high morbidity and mortality [5].

Early diagnosis and staging are main objectives in the initial evaluation and treatment of pancreatitis in patients with AP to differentiate those with severe disease and to establish proper treatment [6]. Clinical and laboratory studies have been conducted on numerous markers of pancreatitis as potential prognostic indicators; however, except a few, none have been commonly used in clinical practice [7,8]. The parameters that have been found effective in determining the severity of AP are the measurements of activation peptides such as C-reactive protein (CRP), alpha-2 macroglobulin, phospholipase A2, IL-6, and trypsinogen [2,3,8,9].

Clinical scoring systems are used in the determination of disease severity. Ranson criteria can distinguish mild form of pancreatitis from the severe form at nearly 80% sensitivity. APACHE II (Acute Physiologic and

* *E-mail: kisaoglu.a@gmail.com*

Chronic Health Evaluation II) scoring offers such advantages as being feasible for any medical condition and repeatable on admission or any time during hospitalization. These scoring systems clearly show the severity of the disease and reliably help predict potential complications [10].

C-reactive protein (CRP) is the main marker of inflammation [11]. High-sensitivity C-reactive protein (hsCRP) is a more sensitive marker than CRP which can show inflammation in otherwise healthy people, and useful in detecting cardiac problems [12].

Resistin is a hormone primary produced in adipose tissue. Recently it is suggested that resistin is not directly produced by adiposites, but rather by inflammatory cells. It is also stated that in humans, resistin acts as a mediator of insulin resistance associated with sepsis and inflammatory events [13].

CD14 is a monocyte receptor that responds against gram positive and negative bacterial components and fungi. It is expressed over the surface of neutrophils, monocytes, macrophages and fibroblasts with the stimulation of lipopolysaccharides [14]. It has been shown in experimental models, that CD14 antibodies decrease mortality of septic shock [15,16].

In this study, we aimed to evaluate the correlations of serum levels of resistin and CD14 expression in monocytes, and hsCRP and the severity of acute pancreatitis (AP) using scoring systems Ranson and APACHE II scoring systems.

2. Methods

This study was approved by institutional review board to perform. This prospective study involved 34 AP patients at the Department of General Surgery, Ataturk University Medical School between July 2008 and September 2009. Patients consisted the study group. The control group was composed of 20 healthy volunteers, age and gender matched with the patient group, who were otherwise healthy at the time of enrollment into the study based on their history and physical examination and applied to outpatient clinics of our hospital for diagnosis and treatment (Table 1).

Table 1. The Distribution of the Patient and Control Groups According to Age and Gender.

		Male		Female	
	n	%	Age ($\bar{x}\pm\sigma$)	%	Age ($\bar{x}\pm\sigma$)
Patient	34	29.41	57.30 ± 19.79	70.59	59.75 ± 14.42
Control	20	30	57.16 ± 9.55	70	58.85 ± 10.75

$\bar{x}$: Mean σ: Standard deviation

The following criteria were used in acute pancreatitis diagnosis [17,18]:

1. Acute upper abdominal pain suggesting the clinical progression
2. Hyperamylasemia of 5 items higher than the normal upper threshold
3. Characteristic computer tomography and/or sonography findings.

Patients fulfilling any of the below criteria were excluded from the study:

1. Malign disease
2. Chronic inflammatory disease
3. Diagnosed or suspected chronic pancreatitis
4. Organ failure.
5. Unstable coronary disease.
6. Liver failure.
7. Chronic obstructive pulmonary disease
8. Immunosuppressive diseases (because of their potential effects on immune response)
9. Presentation later than the first 24 hours

Ranson scores [19] were calculated based on the evaluations on presentation and after 48 hours for all the patients. APACHE II scores [7,20] were calculated based on the parameters used on presentation and when clinically needed during hospitalization.

In the light of the criteria established at 1992 Atlanta Acute Pancreatitis Symposium [20-22], Ranson scoring was made taking 3 points as the threshold and APACHE II scoring was made taking 8 points as the threshold.

Based on the same criteria, the patients were classified as mild AP and severe AP. patients, serum levels of resistin, hsCRP, and CD14 expression in monocytes were determined on the 1st and 7th days. Using CT and abdominal USG, all the patients in the mild and severe AP groups were evaluated for changes in the pancreas and other intraabdominal tissues.

2.1. Sampling and Evaluation

Venous blood samples of 10mL were obtained from the study and control groups and 8 mL of each sample was transferred into gel containing vacutainer tubes (plain), and 2 mL of the blood was transferred in to EDTA containing tubes for evaluation of CD14 in monocytes. CD14 levels in monocytes were determined at the Hematology Laboratory of Ataturk University Medical School. The samples that were put into biochemistry tubes were kept at room temperature for 30 minutes and centrifuged at 4000 rpm for 10 minutes. Serum samples separated were transferred into eppendorf tubes with a straw for evaluation of the hsCRP and resistin levels, and kept at -80 ^{o}C until the day of analysis.

Resistin level was determined using commercial kit (RayBio® Human Resistin, Cat#: ELH-Resistin-001, USA) by ELISA method. Serum hsCRP level was determined at the Microbiology laboratory of Ataturk University Medical School with BN II (Dade Behring, Marburg GmbH, Germany) by immunonefelometric method. In the literature, the reference values for healthy individuals are below 3mg/L. Using this instrument, 2147 healthy subjects were studied to determine the normal interval for serum CRP, and in 90%, mean:1.69 mg/L and in 95%, mean: 2.87 mg/L.

Peripheric blood monocyte phenotype was determined at the Hematology Laboratory of Ataturk University Medical School with flow cytometry instrument (CoulterEPICS XL-MCL, Beckman Coulter Inc., USA) using immunoflorescent antibodies. Data were analyzed with a commercial software program (Coulter System II Software version 2.0). CD14-PE marker was studied on the basis of CD45 and side scatter (SS).

Statistical analyses were conducted using SPSS 13.0 package program and the data were evaluated through Chi-square, Anova, Mann-Whitney U, and Wilcoxon signed-rank tests. Ranson scoring was used to assess linear regression between serum resistin and hsCRP levels and monocyte CD14 expression. APACHE II scoring was used for correlation analysis between serum hsCRP, resistin, and monocyte CD14 levels. The confidence interval was set at 95% and $p < 0.05$ was considered statistically significant.

3. Results

Of the 34 patients, 17 (50%) were classified as mild AP and 17 (50%) were classified as severe AP patients. Table 2 presents the results of the study (patient) group. When the mild and severe AP groups were compared, the Ranson and APACHE II scores of the severe AP group were found to be statistically significantly higher. The hsCRP levels measured on the 1st and 7th days were statistically significantly higher in the severe AP group than in the mild AP group. The mean serum hsCRP levels of the mild and severe AP patients measured on the 7th day were statistically significantly than the values of measured on the 1st day (respectively p=0.002 and p=0.039). Serum resistin, monocyte CD14 expression, and monocyte values of the mild and severe AP groups were compared, and no statistically significant differences were found. Although serum resistin level measured on the 7th day was lower than that measured

Table 2. The mean serum levels of hsCRP, Resistin, monocyte, and monocyte CD14 and Ranson and APACHE II scores of the mild and severe AP groups

	n	Mild AP group (n=17) ($\bar{x}\pm\sigma$)	Severe AP group (n=17) ($\bar{x}\pm\sigma$)	p
Ranson Score	34	1.11 ± 0.78	3.17 ± 0.72	0.000
APACHE II 1st day	34	3.47 ± 2.40	6.00 ± 2.31	0.004
APACHE II 3rd day	34	3.00 ± 2.34	6.64 ± 2.49	0.000
APACHE II 7th day	34	2.58 ± 2.18	5.17 ± 2.40	0.002
hsCRP 1st day (mg/L)	34	66.30 ± 60.80	137.20 ± 59.40	0.011
hsCRP 7th day (mg/L)	34	22.80 ± 32.80	103.80 ± 74.50	0.002
Resistin 1st day (ng/dl)	34	23.50 ± 12.30	26.48 ± 12.03	0.492
Resistin 7th day (ng/dl)	34	20.27 ± 9.71	19.60 ± 8.09	0.831
Monocyte CD14 1st day (%)	34	81.05 ± 14.51	82.82 ± 10.48	0.938
Monocyte CD14 7th day (%)	34	80.41 ± 7.46	79.94 ± 6.67	0.831
Monocyte 1st day (n/μl)	34	900.00 ± 738.24	1305.88 ± 1364.03	0.277
Monocyte 7th day (n/μl)	34	711.76 ± 231.52	1129.41 ± 1101.56	0.442

$\bar{x}$: Mean σ: Standard deviation

on the 1st day, the difference was not statistically significant (p=0.368 for mild AP group; p=0.093 for severe AP group).

The mean serum resistin levels of the mild and severe AP patients measured on 1st and the 7th day were statistically significantly higher than those of the control group. Comparisons of all the groups showed that the mean serum resistin levels on the 1st and 7th days were statistically significantly higher than those of the control group on the same days (p=0.002 and p=0.000 respectively). The mean serum CD14 levels of the mild and severe AP patients measured on 1st and the 7th day were statistically significantly higher than those of the control group. Comparisons of all the groups showed that the mean serum monocyte levels on the 1st and 7th days were statistically significantly higher than those of the control group (p=0.031 and p=0.015 respectively). The serum monocyte CD14 levels of the mild and severe AP groups measured on the 1st and 7th days were not statistically significantly different from those of the control group (Table 3).

The linear regression analyses between serum resistin, hsCRP and monocyte CD14 levels revealed a significant correlation between the serum resistin and hsCRP levels on the 1st day (Figure 1).

Correlation analyses among the parameters showed that serum hsCRP concentrations were positively correlated with Ranson and APACHE II scores, while resistin level was not correlated with these clinical scores. The serum resistin level measured on the 1st day was positively correlated with hsCRP level. Furthermore, Ranson and APACHE II scores were positively correlated; monocyte and monocyte CD14 expression levels were not correlated, but monocyte level measured on the 7th day was positively correlated with monocyte hsCRP expression (Table 4).

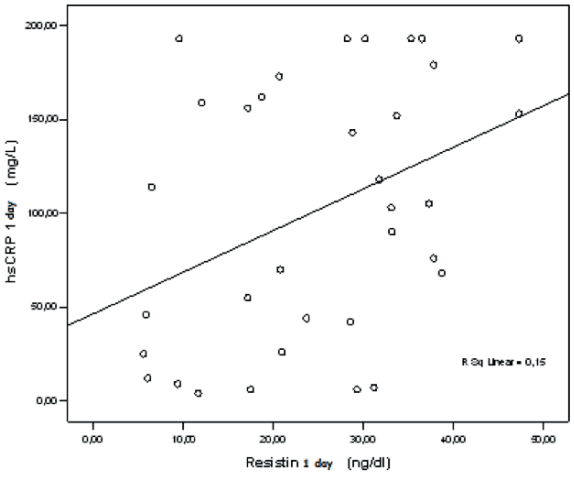

Figure 1. The linear regression between the serum resistin and hsCRP levels on the 1st day. (y=2.220x + 46.28 r=0.15)

In our study, 7 patients (20.6%) in the severe AP group developed local or systemic complications (organ failure in 3, necrosis in the pancreas in 2, and pseudocyst in the pancreas of 2). Moreover, 4 patients in this group suffered acute fluid retention.

4. Discussion

Acute pancreatitis (AP) may progress in various degrees of severity. Establishing the disease severity is important for determination of both prognosis and treatment approach. AP progresses with serious complication rates of 15-25% [23-25]. Acute fluid retention is seen in 30-50% of severe AP patients, spontaneously regressing in more than half [21]. In nearly 40% of all the AP patients that develop pancreas necrosis. Even in the best centers, the total mortality rate for edematous

Table 3. The mean serum resistin, monocyte, and monocyte CD14 levels of the mild and severe AP and control groups.

	Mild AP (n=17) ($\bar{x}\pm\sigma$)	Severe AP (n=17) ($\bar{x}\pm\sigma$)	Control (n=20) ($\bar{x}\pm\sigma$)	p
Resistin 1st day gün (ng/dl)	23.50 ± 12.30	26.48 ± 12.03	9.52 ± 3.29	0.003[a] 0.001[b]
Resistin 7th day (ng/dl)	20.27 ± 9.71	19.60 ± 8.09	9.52 ± 3.29	0.001[a] 0.002[b]
Monocyte 1st day (n/μl)	900.00 ± 738.24	1305.88 ± 1364.03	520.00 ± 196.28	0.056[a] 0.008[b]
Monocyte 7th day (n/μl)	711.76 ± 231.52	1129.41 ± 1101.56	520.00 ± 196.28	0.024[a] 0.021[b]
Monocyte CD14 1st day (%)	81.05 ± 14.51	82.82 ± 10.48	81.10 ± 5.06	0.635[a] 0.635[b]
Monocyte CD14 7th day (%)	80.41 ± 7.46	79.94 ± 6.67	81.10 ± 5.06	0.659[a] 0.495[b]

$\bar{x}$: Mean σ: Standard deviation a: Mild AP-Control group b: Severe AP-Control group

Table 4. Ranson, APACHE II score and serum hsCRP, resistin, and monocyte, monocyte CD14 correlation between levels.

	Ranson	APACHE II 1st day	APACHE II 3rd day	APACHE II 7th day	hsCRP 1st day	hsCRP 7th day	Monocyte CD14 1st day	MonocyteCD14 7th day	Resistin 1st day	Resistin 7th day	Monocyte 1st day	Monocyte 7th day
Ranson	r = 1	r = 0.537 p = 0.001	r = 0.703 p = 0.000	r = 0.575 p = 0.000	r = 0.432 p = 0.011	r = 0.535 p = 0.001	r = -0.143 p = 0.419	r = -0.027 p = 0.881	r = 0.188 p = 0.287	r = 0.165 p = 0.350	r = -0.007 p = 0.970	r = 0.207 p = 0.241
APACHE II 1st day	r = 0.537 p = 0.001	r = 1	r = 0.901 p = 0.000	r = 0.934 p = 0.000	r = 0.501 p = 0.003	r = 0.489 p = 0.003	r = 0.023 p = 0.896	r = -0.030 p = 0.866	r = -0.040 p = 0.822	r = 0.173 p = 0.328	r = 0.066 p = 0.712	r = 0.095 p = 0.592
APACHE II 3rd day	r = 0.703 p = 0.000	r = 0.901 p = 0.000	r = 1	r = 0.938 p = 0.000	r = 0.563 p = 0.001	r = 0.628 p = 0.000	r = -0.082 p = 0.644	r = -0.062 p = 0.729	r = 0.117 p = 0.511	r = 0.213 p = 0.227	r = 0.063 p = 0.723	r = 0.276 p = 0.114
APACHE II 7th day	r = 0.575 p = 0.000	r = 0.934 p = 0.000	r = 0.938 p = 0.000	r = 1	r = 0.411 p = 0.016	r = 0.530 p = 0.001	r = 0.007 p = 0.968	r = -0.012 p = 0.946	r = 0.009 p = 0.960	r = 0.157 p = 0.376	r = 0.029 p = 0.872	r = 0.135 p = 0.445
hsCRP 1st day (mg/L)	r = 0.432 p = 0.011	r = 0.501 p = 0.003	r = 0.563 p = 0.001	r = 0.411 p = 0.016	r = 1	r = 0.577 p = 0.000	r = 0.128 p = 0.469	r = 0.072 p = 0.686	r = 0.387 p = 0.024	r = 0.094 p = 0.598	r = 0.307 p = 0.077	r = 0.400 p = 0.019
hsCRP 7th day (mg/L)	r = 0.535 p = 0.001	r = 0.489 p = 0.003	r = 0.628 p = 0.000	r = 0.530 p = 0.001	r = 0.577 p = 0.000	r = 1	r = 0.020 p = 0.911	r = 0.196 p = 0.268	r = 0.177 p = 0.316	r = 0.041 p = 0.817	r = -0.145 p = 0.412	r = 0.534 p = 0.001
Monocyte CD14 1st day (%)	r = -0.143 p = 0.419	r = 0.023 p = 0.896	r = -0.082 p = 0.644	r = 0.007 p = 0.968	r = 0.128 p = 0.469	r = 0.020 p = 0.911	r = 1	r = 0.102 p = 0.568	r = -0.010 p = 0.955	r = -0.279 p = 0.110	r = 0.183 p = 0.300	r = 0.196 p = 0.266
Monocyte CD14 7th day (%)	r = -0.027 p = 0.881	r = -0.030 p = 0.866	r = -0.062 p = 0.729	r = -0.012 p = 0.946	r = 0.072 p = 0.686	r = 0.196 p = 0.268	r = 0.102 p = 0.568	r = 1	r = -0.027 p = 0.879	r = -0.138 p = 0.435	r = 0.078 p = 0.660	r = 0.119 p = 0.504
Resistin 1st day (ng/dL)	r = 0.188 p = 0.287	r = -0.040 p = 0.822	r = 0.117 p = 0.511	r = 0.009 p = 0.960	r = 0.387 p = 0.024	r = 0.177 p = 0.316	r = -0.010 p = 0.955	r = -0.027 p = 0.879	r = 1	r = 0.432 p = 0.011	r = 0.162 p = 0.361	r = 0.359 p = 0.037
Resistin 7th day (ng/dL)	r = 0.165 p = 0.350	r = 0.173 p = 0.328	r = 0.213 p = 0.227	r = 0.157 p = 0.376	r = 0.094 p = 0.598	r = 0.041 p = 0.817	r = -0.279 p = 0.110	r = -0.138 p = 0.435	r = 0.432 p = 0.011	r = 1	r = -0.194 p = 0.271	r = -0.038 p = 0.830
Monocyte 1st day (n/μL)	r = -0.007 p = 0.970	r = 0.066 p = 0.712	r = 0.063 p = 0.723	r = 0.029 p = 0.872	r = 0.307 p = 0.077	r = -0.145 p = 0.412	r = 0.183 p = 0.300	r = 0.078 p = 0.660	r = 0.162 p = 0.361	r = -0.194 p = 0.271	r = 1	r = -0.075 p = 0.673
Monocyte 7th day (n/μL)	r = 0.207 p = 0.241	r = 0.095 p = 0.592	r = 0.276 p = 0.114	r = 0.135 p = 0.445	r = 0.400 p = 0.019	r = 0.534 p = 0.001	r = 0.196 p = 0.266	r = 0.119 p = 0.504	r = 0.359 p = 0.037	r = -0.038 p = 0.830	r = -0.075 p = 0.673	r = 1

pancreatitis is 1% or below, for sterile necrosis, 5%, and for infected necrosis, 10-20% [26,27]. While mild pancreatitis regresses with supportive therapy, the mortality rate from severe necrotic pancreatitis varies from 20% to 40% [28-30]. AP may cause serious morbidity and mortality; thus, prediction and establishing the severity will lead to proper treatment and lower mortality through intensive care.

In this study, Ranson and APACHE II scoring systems, tools for determination of AP prognosis, were used and to determine the response to systemic inflammation and their role in predicting the disease severity, serum resistin, hsCRP, and CD14 expression in monocytes were measured.

Positive prognostic findings based on clinical evaluation systems for pancreatitis such as Ranson and Glasgow are used to predict potential morbidity and mortality [6]. These scoring systems are relatively successful in determining the disease progression; nevertheless, for an accurate evaluation, 48 hours should be completed. In the studies by Ranson in 1970s, a mortality rate of 40% was reported in the presence of 5 or 6 positive findings [7]. Higher Ranson scores in the severe AP group in our study suggests that this scoring system is useful in determining the prognosis and severity of the disease.

APACHE II is another scoring system that helps predict the disease severity according to the standard deviations of numerous physiological variables from the normal [7,20]. In this scoring system, scores are added for advanced age and multiple organ failures. Because it is more focused on age and comorbidity, it may reflect non-severe pancreatitis in the elderly as pancreatitis with poor prognosis, which is its most important disadvantage [7,31].In our study, a total of 7 patients developed local or systemic complications. Of these patients, 5 patients had Ranson and APACHE II scores higher than the threshold values. This indicates that AP patients with higher scores than the threshold of both scoring systems may have severe progression. In determining the severity of pancreatitis, Ranson scoring has 75% sensitivity and 68% specificity, while APACHE II scoring has 75% sensitivity and 92% specificity [32]. The objective of clinical evaluation scoring is to associate metabolic disorders and organ failures with morbidity and mortality [33]. Significantly higher APACHE II Scores in the severe AP group in the present study shows the effectiveness of this system in determining prognosis although the system is not specific for pancreatitis and is not easy to use.

Parallel to the extent of the damage in the pancreas and response of the organism, hepatocytes are stimulated by cytokines, acute phase response and the most important reactant of this response, serum CRP, increase [33]. CRP value over 150 mg/L indicates serious disease [34]. Consideration of 150 mg/L s the cut-off value in the first 48 hours after the onset of the symptoms offers more than 80% sensitivity and specificity, providing 86% accuracy in showing necrotic pancreas [35]. In our study, serum hsCRP values of 2 patients with necrotic pancreas measured on the 1st day were 193 mg/L in both, and on the 7th day, they were 193 mg/L and173 mg/L. Moreover, the serum hsCRP values of the 4 patients who developed local complications and fluid retention were above150 mg/L as measured on the 1st day. Gürleyik et al, in a prospective study involving 30 AP patients, the cut-off value for CRP was established as 150 mg/L for severe inflammation, and while no significant intergroup differences were found between the CRP levels measured in the first 24 hours, the values on the 48th hour were significantly higher in the severe AP group than in the mild AP group [33]. In our study, serum hsCRP levels were significantly higher in the severe AP group than in the mild AP group. The serum CRP levels on the 7th day were significantly reduced compared to the values on the 1st day and serum hsCRP concentrations were found to be positively correlated with Ranson and APACHE II scores.

In acute pancreatitis, local and systemic inflammatory response associated with acinar cell damage determines the severity of the effect of the attack on the organism [33]. Resistin has been shown to be expressed from human macrophages, and thus, it is thought to be associated with inflammatory conditions. Since resistin increases the production of adhesion molecules on arterial walls, it is claimed to have a direct proinflammatory effect on the vascular endothelial cells [36,37]. In recent years, it has been suggested that resistin does not generate directly from adipocytes but rather from the inflammatory cells that filters the fatty tissue [13]. In a study on 23 AP patients, the resistin level in severe AP patients was significantly elevated compared to the levels of mild and moderate AP patients. The authors reported that resistin level was associated with extrapancreatic necrosis, and resistin is positively correlated with systemic CRP levels [38]. Lesniowski et al determined higher serum resistin concentration in AP patients than in the controls [39]. In our study, the resistin levels measured on the 1st day were higher in the severe AP group than in the mild AP group. However, in the intra and intergroup comparisons between the AP groups for 1st and 7th day resistin levels, no statistically significant differences were found. Compared to the control group, however, the resistin levels of the mild and severe AP groups were statistically significantly higher. Correlation analyses between Ranson and APACHE II scores and

serum reisistin level indicated no correlations, while serum resistin level on the 1st day was positively correlated with hsCRP.

CD14 is a monocyte receptor that has a defensive response against fungi with its positive and negative compounds [40]. CD14 is expressed from the surfaces of neutrophils, monocytes, macrophages, and fibroblasts, which produce cytokines such as IL-1 and TNFα during LPS stimulation [14]. Antibodies developed against CD14 in experimental models are shown to decrease mortality from septic shock [15,16]. Yaegashi et al evaluated 66 patients with sepsis and 80 patients with SIRS and found higher sCD14 levels in the sepsis group than in the SIRS and healthy control groups. They have concluded that this finding shows specific elevation of sCD14 in patients with sepsis [41]. In another study, 30 AP patients were studied and their 1st, 3rd, and 7th day monocyte cell surface markers were compared among the groups and with the control group. When CD14 expression was quantitatively measured with MFI (mean fluorescence intensity), no statistically significant differences were found [42]. Rahman et al measured monocyte CD14 expression in the first 24 hours of the disease onset and determined no differences between AP and control groups [43].

In our study, inter and intragroup comparisons of the groups showed no statistically significant differences between the groups for monocyte CD14 groups. Similarly, while the serum monocyte levels of the mild and severe AP groups were not statistically significantly different, paired comparisons of the mild and severe AP groups with the control group revealed higher monocyte levels than in control group. Correlation analyses showed that monocyte and monocyte CD14 expression levels were not correlated, but monocyte level measured on the 7th day was positively correlated with monocyte hsCRP expression.

5. Conclusion

The findings of our study have shown that serum hsCRP measurement is effective in determining the severity of acute pancreatitis. Serum resistin measurement may be a useful early marker in determining the inflammatory response in AP but not in determining the disease severity. However, CD14 expression is not a useful marker in the diagnosis and prediction of the disease severity in AP patients.

Conflict of interest statement

Authors state no conflict of interest.

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