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Original Article

The Importance of Serum Omentin-1 and Visfatin Levels in Determining Acute Pancreatitis Activation

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ABSTRACT

Background Acute pancreatitis is a disease that can lead to serious mortality and morbidity. Therefore, the use of inflammatory markers is of great importance in determining the prognosis of the disease. Omentin-1 and visfatin are newly discovered adipokines associated with inflammation. In this study, we aimed to demonstrate the importance of omentin-1 and visfatin in diagnosing and activating acute pancreatitis.

Methods Serum samples from 52 patients diagnosed with acute pancreatitis who presented to the Emergency Department of Çanakkale Onsekiz Mart University Health Practice and Research Hospital between July 2022 and May 2023 were analyzed for serum omentin-1 and visfatin levels, along with routine laboratory tests, during both the initial and remission periods. Disease severity was calculated using the Modified Glasgow Prognostic Score. Correlation analysis was conducted among study variables.

Results The marker with the highest sensitivity and specificity in predicting active disease was found to be C-reactive protein (CRP). The sensitivity of serum omentin-1 levels in determining active disease was 84.62%, with a specificity of 73.17%. Serum visfatin levels had a sensitivity of 76.92% and a specificity of 78.05% in determining active disease. According to the Modified Glasgow Prognostic Scoring System, omentin showed the highest sensitivity (82.61%) in distinguishing mild-moderate cases from severe cases, while visfatin had the highest specificity (86.21%).

Conclusion In our study, serum levels of omentin-1 and visfatin negatively correlated with disease diagnosis and severity.

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INTRODUCTION

Acute pancreatitis (AP) is a reversible inflammatory disease affecting the pancreas and surrounding tissues, initiated by activated pancreatic enzymes due to various etiological factors. The majority of cases are attributed to gallstones and alcohol.[1] In addition, rarer causes such as hypertriglyceridemia, medications, post-ERCP, and obstruction of the pancreatic duct can also lead to acute pancreatitis.[2] The diagnosis of AP is established with typical abdominal pain, serum amylase-lipase levels three times higher than normal, and the presence of accompanying imaging findings. Diagnosis is made with the presence of at least two of these criteria.[3]

The severity of the disease varies from self-limiting mild cases to multiple organ failure, and although its course is uncertain, it can be severe enough to result in death. It is unclear which aetiology of pancreatitis will be more severe or mild. For this reason, certain scoring systems have been developed to determine the prognosis.[4] Some of these scoring systems include Ranson, The Acute Physiology and Chronic Health Evaluation (APACHE II), The Bedside Index for Severity in Acute Pancreatitis (BISAP), Glasgow, and Systemic Inflammatory Response Syndrome (SIRS). Since some scoring systems are far from practical in practice, it has been predicted that some biomarkers should be used to determine the severity of acute pancreatitis. CRP is one of these biomarkers.[1,4]

Adipokines are molecules synthesized from adipose tissue and surrounding connective tissue, exerting autocrine, endocrine, and paracrine effects. Adipokines play crucial roles in nutrition and energy modulation, inflammation, lipid, and glucose metabolism.[5] Omentin-1 is an adipokine with anti-inflammatory function; it reduces the expression of C-reactive protein and tumour necrosis factor. It increases insulin-induced glucose uptake and nitric oxide synthesis in yellow adipose tissue, thus preventing the development of diabetes and ischemic heart disease.[6] Omentin-1 is a protein primarily synthesized from visceral adipose tissue, vascular cells, colon, and lungs. It has emerged as a biomarker for various conditions such as insulin resistance, diabetes, inflammatory diseases, polycystic ovary syndrome, and preeclampsia. Decreased levels of serum omentin-1 have been associated with these conditions.[7] Conversely, Visfatin is secreted from various tissues, including adipose tissue, placenta, myometrium, bone marrow, liver, lungs, muscles, heart, macrophages, and neutrophils. In individuals with obesity and high body mass index, adipocytes undergo hypertrophy and hyperplasia, leading to increased secretion of various adipokines, including visfatin. Visfatin, which exists in both intracellular and extracellular forms, has been found to play a role in significant metabolic events such as obesity, insulin resistance, increased inflammation, and angiogenesis. High serum concentrations of visfatin activate immune cells and contribute to chronic inflammation in adipocytes.[8,9] Visfatin is an adipokine that increases the expression of TNF-alpha, IL-1, IL-6, and adhesion molecules in the epithelium and can be used as a non-invasive, easily measurable marker to diagnose disease activity and severity.[10] Acute pancreatitis is an inflammatory disease that can lead to severe organ failure and death, and we can predict which patients will have a worse prognosis with some scoring systems. These scoring systems can be challenging to calculate in practice. We designed this study to predict the severity of patients with easier methods by looking at inflammatory markers such as omentin-1 and visfatin.

MATERIAL AND METHODS

This prospective study included 52 patients diagnosed with acute pancreatitis who presented to the Emergency Department of Çanakkale Onsekiz Mart University Health Practice and Research Hospital and were admitted to the Gastroenterology Department for treatment between July 2022 and May 2023. The study's Inclusion criteria were patients admitted to the Gastroenterology Department for treatment and follow-up, aged 18 years or older, and provided signed informed consent. Pregnant patients, those under 18, and those with malignancies were excluded from the study. A control group comprising 41 individuals who visited our hospital's Internal Medicine or Gastroenterology Clinic for routine check-ups and had no chronic diseases or active infections was also included.

Ethics committee approval was sufficient for the use of patient data. Informed consent was signed before peripheral blood was taken from the patient and control groups to study omentin-1 and visfatin levels. Demographic data such as age, gender, chronic diseases, alcohol and smoking habits, and body mass index were recorded for both patients and the control group, along with the length of hospital stay, development of complications, and aetiology of pancreatitis in the patient group. Modified Glasgow

Prognostic Scores (Modified Imrie Score) were calculated within 48 hours of admission. Patients with an Imrie score below 3 were classified as having mild to moderate pancreatitis, while those with a score of 3 or higher were classified as having severe pancreatitis.

Laboratory tests performed at admission, before discharge, and for the entire control group were documented. Venous blood samples were collected from patients at admission and before discharge to measure serum omentin-1 and visfatin levels. Samples obtained from patients and the control group were centrifuged at 1,500 g for 10 minutes. The centrifuged samples were stored at -40°C in a refrigerator. Serum omentin levels were measured using the BT LAB Human Omentin ELISA kit (Catalogue no E5814Hu; Bioassay Technology Laboratory, Zhejiang, China), while serum visfatin levels were measured using the BT LAB Human Visfatin ELISA kit (Catalogue no E0025Hu; Bioassay Technology Laboratory, Zhejiang, China). A multiscan FC microplate reader (Thermo Scientific Finland) was used for the analysis of the ELISA kits.

Statistical Analysis

Statistical data analysis was performed using SPSS Version 26 (Statistical Package for Social Sciences). Demographic data were expressed as mean and standard deviation for numerical variables, and as number (n) and percentage (%) for categorical variables. Normality testing for numerical variables was conducted using the Shapiro-Wilk test. For comparisons between two groups, the Student's t-test was used for numerical data, and the Chi-square test was used for categorical variables. Correlation analysis between omentin, visfatin, and other inflammatory parameters used in the study was conducted using the Pearson correlation test. Receiver operating characteristic (ROC) analysis was employed to determine the optimal cut-off values for omentin, visfatin, and other inflammatory parameters that could identify acute pancreatitis and severe cases. Univariate logistic regression analysis calculated odds ratios of independent clinical parameters to predict acute pancreatitis.

RESULTS

The study included 52 patients admitted with a diagnosis of AP and 41 healthy individuals. Among the patient group, 30 (57.7%) were female and 22 (42.3%) were male, while in the control group, 32 (78%) were female and 9 (22%) were male. No significant difference was observed between the groups in determining active disease statistically (p=0.054). The

Table 1. The demographic and clinical characteristics of the individuals in the study

	Acute pancreatitis (n: 52)	Control (n: 41)
Age (year)	63.6±16.3	32.4±8.4
Gender n (%)		
Female	30 (57.7)	9 (22.0)
Male	22 (42.3)	32 (78.0)
Alcohol addiction n (%)	3 (5.8)	17 (41.4)
Smoking n (%)	14 (26.9)	14 (34.1)
Additional diseases n (%)		
Diabetes Mellitus	17 (32.7)	-
Hypertension	37 (63.5)	-
Coronary artery disease	14 (26.9)	-
Other	9 (17.3)	-
Length of hospitalization (day)	5.8±4.1	-
Complication		
Yes	5 (9.6)	-
No	47 (90.4)	-
Aetiology n (%)		
Biliary	39 (75.0)	-
Alcohol	3 (5.8)	-
Hyperlipidemia	4 (7.7)	-
Other	6 (11.5)	-
mGKS* score n (%)		
Mild- modarete	29 (55.8)	-
Severe	23 (44.2)	-

*mGKS: Modified Glasgow Prognostic Score.

patient group's mean age was 63.6±16.3 years, while the mean age of the control group was 32.4±8.4 years. Age was statistically significant in determining active disease between the groups (p<0.001). Complications developed in 9.6% of the patient group. According to the Modified Glasgow Prognostic Score, 29 (55.8%) patients were in the mild-moderate group and 23 (44.2%) patients in the severe group. All demographic data of the patients were summarized in Table 1.

The mean serum visfatin levels in the patient group during the active period were 24±21.3 ng/mL, during remission, 38.3±32.9 ng/mL, and in the control group, 51.7±35.7 ng/mL. The distribution of serum visfatin levels is summarized in Figure 1. A significant statistical difference was observed between the active period and the control group in serum visfatin levels. However, as seen in Figure 1, the distribution of serum visfatin levels during remission and in the control group was similar, and no significant statistical difference was found between the two groups (p=0.063).

In the patient group, the mean serum omentin-1 levels during the active period were 48.8±37.6 ng/L, during remission, 71.6±57.1 ng/L, and in the control group, 88.6±57.7 ng/L. When compared between the active period and the control group, a significant statistical difference was found in serum omentin-1 levels. However, as shown in Figure 2, the distribution of serum omentin-1 levels during remission and in the control group was similar. No significant statistical difference was found between the two groups regarding omentin-1 levels (p=0.157). The difference between inflammatory parameters during the active period and the control group is detailed in Table 2. There were significant statistical differences found between the active period and the control group in white blood cell (WBC), haemoglobin, platelet-lymphocyte ratio (PLR), neutrophil-lymphocyte ratio (NLR), CRP, omentin-1, and visfatin levels.

Table 3 showed patients' omentin, visfatin levels,

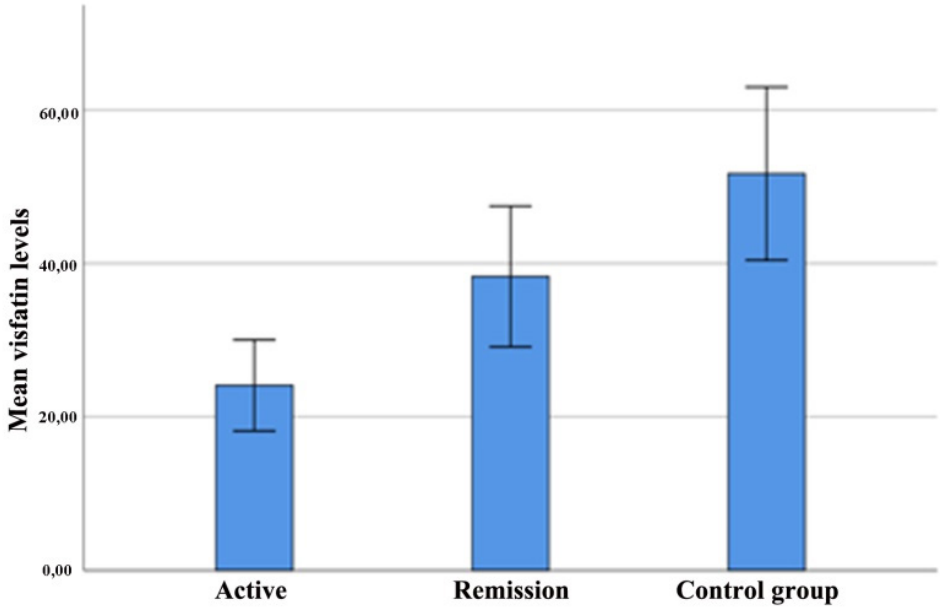

Figure 1. Distribution graph of serum Visfatin levels in active, remission, and control groups

Table 2. Acute pancreatitis, active phase and control group, inflammatory parameters

	Active period	Control group	T score	*P-value*
WBC (/mm³x10³)	12.7±4.3	6.8±1.8	8.727	<0.001
Haemoglobin (g/dL)	11.9±2	13.1±1.1	-3,666	<0,001
PLT(/mm³x10³)	244±133	267±55	-1,086	0,281
NLR	18,8±48	1.8±0.5	2.514	0.015
PLR	261±223	126±27	4.314	<0.001
CRP (mg/L)	118.4±93.4	1.7±1.1	9.009	<0.001
Omentin (ng/L)	48.8±37.6	88.6±57.6	-3.824	<0.001
Visfatin (ng/mL)	24±21.3	51.7±35.7	-4.371	<0.001

WBC: white blood cell, NLR: neutrophil-lymphocyte ratio, PLR: platelet-lymphocyte ratio, CRP: C-reactive protein, PLT: platelet.

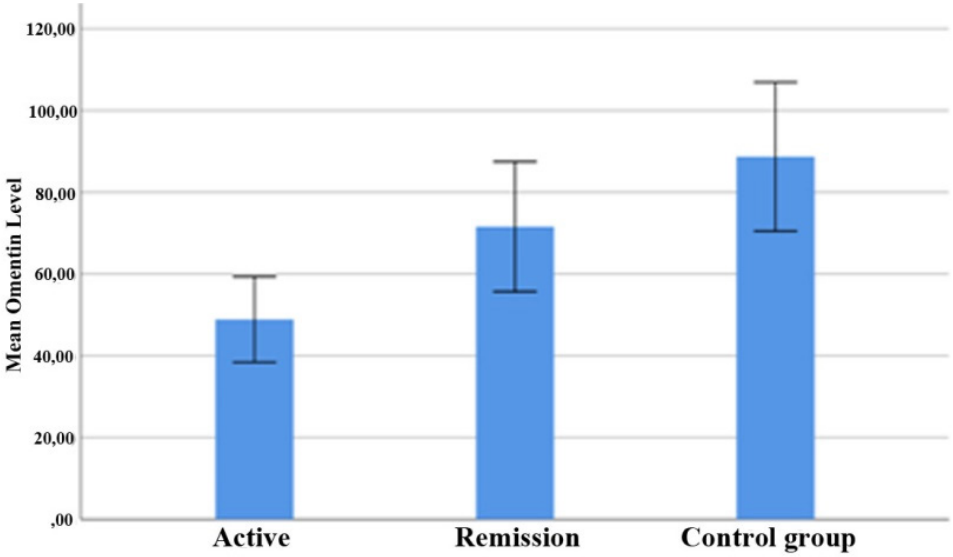

Figure 2. Distribution graph of serum Omentin-1 levels in active, remission, and control groups

and other inflammatory parameters in the mild-moderate and severe groups. Significant statistical differences were found between the two groups in visfatin, omentin-1, CRP, WBC, and NLR values. However, no significant statistical differences were found between the mild-moderate and severe patient groups in platelet (PLT) and PLR values. The correlation analysis of omentin, visfatin, CRP, and WBC values in acute pancreatitis is summarized in Table 4. In our study, omentin and visfatin correlate negatively with CRP and WBC. The ROC analyses of omentin, visfatin, and other inflammatory parameters in distinguishing severe cases among patients with acute pancreatitis, based on the Modified Glasgow Prognostic Scoring System, are summarized in Table 5. The sensitivity of serum omentin levels in distinguishing patients with acute pancreatitis was found to be 84.62% with a specificity of 73.17%, while the sensitivity of visfatin levels was 76.92% with a specificity of 78.05% (Figure 3). A univariate logistic regression analysis was conducted to establish the role of omentin, visfatin, and certain markers in determining acute pancreatitis (Table 6). It was not added to the table because it was not a significant parameter in the multivariate analysis.

DISCUSSION

Acute pancreatitis is characterized by the activation of pancreatic enzymes within the pancreatic parenchyma

Table 3. Comparison of omentin, visfatin, and other study variables based on disease severity calculated with modified Glasgow scoring

	Mild- modarete AP	Severe AP	T score	*P-value*
CRP (mg/L)	85.9±85.2	159.5±88.6	-3.042	0.004
WBC(/mm^{3}x10^3)	11.2±3.4	14.7±4.7	-2.983	0.005
PLT (/mm^{3}x10^3)	270.3±161.1	212.3±80.8	1.576	0.121
PLR	227.9±185.1	303.5±162.1	-1.218	0.229
NLR	7.8±7.3	32.9±71.4	-1.888	0.045
Omentin (ng/L)	63.1±45.1	30.9±9.5	3.747	0.001
Visfatin (ng/mL)	31.7±26.1	14.5±4.2	3.506	0.001

WBC: white blood cell, NLR: neutrophil-lymphocyte ratio, PLR: platelet-lymphocyte ratio, CRP: C-reactive protein, PLT: platelet.

Table 4. Correlation analysis between study variables in acute pancreatitis

	Omentin		Visfatin		CRP		WBC	
	R	*P-value*	R	*P-value*	R	*P-value*	R	*P-value*
WBC	-0.394	<0.001	-0.445	<0.001	0.650	<0.001	-	-
CRP	-0.361	0.001	-0.412	<0.001	-	-	0.650	<0.001
Omentin	-	-	0.863	<0.001	-0.361	0.001	-0.394	<0.001
Visfatin	0.863	<0.001	-	-	-0.412	<0.001	-0.445	<0.001

WBC: white blood cell, CRP: C-reactive protein.

Table-5. Overall accuracy and ROC analyses of omentin and visfatin with other conventional inflammation markers to determine acute pancreatitis and differentiate mild cases from severe cases according to the modified Glasgow Prognostic score.

	AUC	Cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Acute pancreatitis vs. controls						
CRP	0.987	5.1	100.00	97.30	98.08	100.00
WBC	0.918	8.5	84.62	80.56	86.27	78.38
NLR	0.947	2.4	86.54	83.33	88.24	81.08
Omentin	0.791	50.3	84.62	73.17	80.00	78.95
Visfatin	0.775	21.4	76.92	78.05	81.63	72.73
Mild vs severe AP						
CRP	0.737	99.5	73.91	72.41	68.0	77.78
WBC	0.723	11.6	78.26	65.52	64.29	79.17
NLR	0.760	7.4	78.26	65.52	64.59	79.17
Omentin	0.868	40.5	82.61	79.31	76.0	85.19
Visfatin	0.826	18.35	78.26	86.21	81.82	83.33

WBC: white blood cell, NLR: neutrophil-lymphocyte ratio, CRP: C-reactive protein, AUC: area under the curve, PPV: positive predictive value, NPV: negative predictive value.

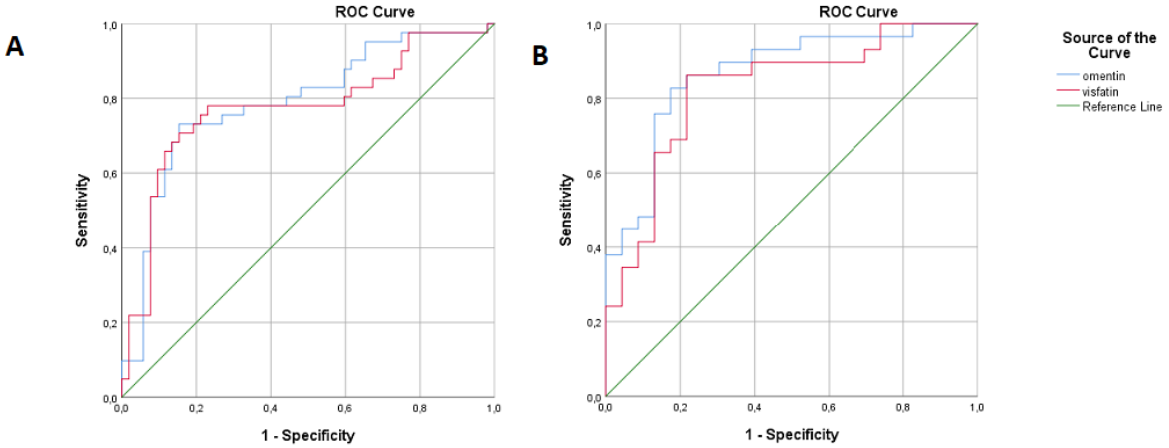

Figure 3. ROC analysis of omentin and visfatin in identifying severe patients with acute pancreatitis (A) and by Modified Glasgow Prognostic Score (B)

Table 6. Univariate logistic regression analysis of specific markers in the diagnosis of acute pancreatitis

	OR	%95 CI	*P-value*
Age	1.193	1.107-1.286	<0.001
Gender (female reference)	0.390	0.149-1.017	0.054
WBC	2.234	1.575-3.167	<0.001
CRP	3.130	1.340-7.315	0.008
NLR	5.564	2.234-13.859	<0.001
Visfatin	0.961	0.941-0.981	<0.001
Omentin	0.975	0.961-0.990	0.001

WBC: white blood cell, NLR: neutrophil-lymphocyte ratio, CRP: C-reactive protein, OR: odds ratio.

due to various etiological factors, leading to local and systemic inflammation. Given its increasing global incidence, prolonged hospitalizations, and potential long-term consequences such as endocrine and exocrine pancreatic insufficiency, the disease carries significant socio-economic implications. In our study, we aimed to identify novel markers that could assist in diagnosing the disease and determining its severity.

The mean age of our patient group was 63.6±16.3 years, with 57.7% of the patients being female. In a study conducted by Bardakçı et al.11, the mean age of patients was 68.6 years, with 61% being female, while Xiao et al.[12] did not find a significant statistical difference in gender. In a study by Samanta et al.[13], demographic data revealed that age was only significant for mortality in univariate regression analysis. In contrast, in our study, age was found to be more significant.

According to a study evaluating 700 patients by Hong et al.[14], the aetiology of AP was

found to be gallstones, idiopathic, alcohol, and hypertriglyceridemia in descending order. Similarly, in our study, etiologies included biliary pancreatitis, other causes, and alcohol, consistent with the literature. Katuchova et al.[15] found obesity to be a risk factor for both local and systemic complications. At the same time, Martinez et al.[16] reported a higher incidence of severe disease and mortality in obese individuals. Our study found that body mass index (BMI) was above 25 in 73% of the patient group, consistent with the literature, indicating that it is an independent risk factor for AP.

White blood cell count (WBC) is an important marker in scoring systems such as Ranson, Imrie, and SIRS in acute pancreatitis. In a study by Huang et al.[17], the WBC value was statistically significant in distinguishing between mild and severe pancreatitis. Consistent with the literature, our study also found statistically significant differences in WBC values between the mild-moderate and severe patient groups. Additionally, significant differences in WBC values were found between the active period and remission period, as well as between the active period and the control group, in line with the literature. C-reactive protein (CRP) is a positive acute-phase reactant synthesized by the liver, which rises within hours in cases of inflammation and infection.[18,19] High levels of CRP have been correlated with the development of pancreatic necrosis and severe pancreatitis in many studies. In a study by Khanna et al.[20] involving 72 patients, CRP demonstrated high accuracy in predicting severe disease, with an AUC of 0.91 (95% confidence interval). In our study, significant statistical differences were found in CRP values between the active patient group and the control group and between the active patient group and the remission group. The correlation analysis between CRP, WBC, omentin, and visfatin revealed that CRP correlated with other inflammatory markers.

In acute pancreatitis, vascular endothelial dysfunction and increased vascular permeability occur due to tissue damage in the pancreatic tissue, resulting in leukocyte migration.[21] Improvement in the prognosis of acute pancreatitis has been associated with decreased neutrophil count.[22] Recent studies have also reported that lymphopenia is associated with disease severity and has independent prognostic value in various diseases, including acute pancreatitis.[23-27] The neutrophil-to-lymphocyte ratio (NLR) is a marker calculated from the neutrophil and lymphocyte counts, which helps determine the severity and prognosis of acute pancreatitis.[28] In a study by Gençdal et al.[29] involving 435 patients, significant statistical differences were found in NLR values between mild and severe patient groups according to the Ranson score. Our study found significant statistical differences in NLR values between the active patient, remission, and control groups. Consistent with the literature, NLR was found to be a practical and highly sensitive marker that provides significant results in diagnosing acute pancreatitis and determining its severity.

Adipose tissue is now considered an endocrine organ because it produces many bioactive molecules called adipokines. Omentin and visfatin are some of these adipokines. It is known that these adipokines produced from adipose tissue play a role in critical metabolic events such as inflammation, immunity, vascular hemostasis, lipid metabolism, and insulin sensitivity.[30] While no studies investigating the relationship between visfatin and omentin with acute pancreatitis were found in the literature, many studies examine the association of these two adipokines with other inflammatory diseases. Our study aimed to use visfatin and omentin-1 as new markers for diagnosing and determining the severity of acute pancreatitis.

Visfatin is a newly discovered adipokine produced mainly by visceral adipose tissue in internal organs, also known as pre-B cell colony-enhancing factor and nicotinamide phosphoribosyltransferase.[31] In our study, significant statistical differences were found in visfatin levels between active and control patients. In contrast, no significant difference was found between the remission period and the control group. According to the Modified Glasgow Scoring, significant statistical differences were detected between the mild-moderate and severe severity patients. Visfatin showed a negative correlation between diagnosing the disease and determining its severity. While no study investigating visfatin levels in acute pancreatitis was found in the literature, there are studies examining visfatin levels in different diseases. In these studies, visfatin levels positively correlated with diagnosing and determining the disease's severity. No study correlating with our results was found in the literature. The discrepancy in visfatin's results from the literature may be attributed to examining serum visfatin levels instead of tissue samples and using different ELISA kits.

Omentin, also known as intelectin-1 and intestinal

lactoferrin receptor, is one of the newly discovered adipokines.[10,32] Omentin is a protective adipokine that increases insulin sensitivity and protects against atherosclerosis and cardiovascular diseases. By inhibiting NF-kappaB, omentin suppresses inflammation and plays an important role in immunity.[8] Our study indicates that serum omentin-1 levels in patients with acute pancreatitis are significantly decreased compared to the control group. There is a significant statistical difference between the two groups. In a study by Yin et al. involving 192 patients with inflammatory bowel disease (IBD), serum omentin-1 levels in the patient group were found to be significantly lower compared to healthy individuals ($p<0.001$). When comparing the patient groups with mild-moderate and severe pancreatitis in our study, there was a significant statistical difference in serum omentin-1 levels. Serum omentin-1 levels were statistically significantly lower in the active patient group compared to the remission group. Similarly to our study, Yin et al.[33] found lower serum omentin-1 levels in the active patient group In our study, serum omentin-1 shows a negative correlation compared to CRP and WBC in detecting active disease. Furthermore, in the univariate logistic regression analysis, omentin-1 and inflammatory markers such as NLR, WBC, and CRP yielded significant results in detecting active disease. This analysis associates low serum omentin-1 levels with active disease ($p=0.001$, OR: 0.975, 95% CI: 0.961-0.990).

This study demonstrated the value of omentin-1 and visfatin in showing the severity of acute pancreatitis, but there were several limitations. First, groups of similar age and gender could not be selected. Because our pancreatitis patients were older, the healthy control group patients who came to the internal medicine outpatient clinic were younger. This was the most important limitation of our study. In addition, it was a single-centre study; omentin-1 and visfatin levels vary according to obesity, insulin resistance, and body fat distribution, and omentin-1 and visfatin levels were measured from a serum sample, not from pancreatic tissue. Additionally, including other inflammatory markers in the study may contribute to a more comprehensive assessment of the disease.

CONCLUSIONS

Thus, serum omentin-1 levels can be used as a useful biomarker to distinguish the active patient group from healthy individuals in diagnosing the disease. Additionally, its lower levels in the severely ill group can guide clinicians in determining the prognosis of the disease. Our study demonstrated that omentin-1 levels have anti-inflammatory effects, consistent with the literature. No studies in the literature have investigated serum visfatin levels in acute pancreatitis. However, various studies on other inflammatory diseases have examined visfatin levels, showing a positive correlation with disease severity. In our study, unlike other inflammatory diseases, visfatin levels showed a negative correlation in diagnosing and determining the severity of the disease.

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Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical Statement

The study received ethical approval from the Clinical Research Ethics Committee of Çanakkale Onsekiz Mart University Faculty of Medicine on November 3, 2022, under approval number 2022/13-12.

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Authors' Contribution

Study Conception: OK, YK, DB; Study Design: OK, DK, YB; Materials: DK, MD; Data Collection: DK, MD, HYC; Analysis and interpretion: DK, MD, HYC, Literature Review: OK, DK; Critical Review: OK, DK, YB; Manuscript writing: OK, DK, YB.

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