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TNF-α and E-Selectin as Valuable Biomarkers in Patients with Acute Coronary Artery Syndrome

Ghassan Jabbar Auda[1], Ali Mohammed Barakat[1], Hassan Sarhan Sachet[1], Raed Fanoukh Aboqader[2*], Adnan Taan Thamer [3]

1. Department of Microbiology and Immunology, College of Medicine, Al-Ayen Iraqi University, AUIQ, An Nasiriyah, Iraq.
2. Department of Internal Medicine and Clinical Immunology, College of Medicine, Al-Ayen Iraqi University, AUIQ, An Nasiriyah, Iraq.
3. Department of Internal Medicine and Intervention Cardiology, College of Medicine, Al-Ayen Iraqi University, AUIQ, An Nasiriyah, Iraq.

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***Corresponding**: Raed Fanoukh Aboqader **Address**: College of Medicine, Al-Ayen Iraqi University, AUIQ, An Nasiriyah, Iraq.
Email: raed.fanoukh@alayen.edu.iq

ABSTRACT

Coronary artery disease (CAD) remains the leading cause of mortality worldwide, especially in developing countries, with dyslipidemia being a major risk factor. This study aimed to evaluate lipid parameters and inflammatory biomarkers—E-selectin and tumor necrosis factor-alpha (TNF-α)—to understand their roles in the pathogenesis of acute coronary syndrome (ACS). A case-control design was used, involving 120 participants: 60 patients diagnosed with ACS and 60 healthy controls, enrolled between January and December 2024. Blood samples were analyzed to assess lipid profiles, including total cholesterol, triglycerides, HDL, LDL, and VLDL, using a SMART-120 chemistry analyzer. Serum levels of TNF-α and E-selectin were measured using enzyme-linked immunosorbent assay (ELISA). Results showed significant differences in lipid profiles between ACS patients and controls, supporting the impact of dyslipidemia on ACS development. E-selectin levels were significantly elevated in ACS patients (213.26 ± 2.72 pg/mL) compared to controls (175.11 ± 2.71 pg/mL), with $P < 0.0001$. Similarly, TNF-α levels were higher in patients (83.20 ± 3.88 pg/mL) than controls (45.65 ± 1.79 pg/mL), also with $P < 0.0001$. ROC curve analysis demonstrated that E-selectin had 96% sensitivity and specificity at a cutoff of 73.44 pg/mL, while TNF-α had 93% sensitivity and 86% specificity at a cutoff of 188.65 pg/mL. Both biomarkers positively correlated with body mass index ($r = 0.572$, $P < 0.0001$).The findings suggest that TNF-α and E-selectin are potential diagnostic biomarkers for ACS and play key.

Keywords: Acute coronary artery syndrome, E-selectin, TNF-alpha, hyperlipidemia

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Introduction

Acute coronary syndrome (ACS) is a critical condition characterized by reduced blood flow in the coronary arteries, leading to heart muscle dysfunction or death. Patients commonly experience alarming symptoms, including chest pain described as pressure, along with nausea and excessive sweating (1). Given the rising prevalence of aging, alongside the escalating rates of obesity and diabetes mellitus within the global populace, it is hypothesized that the incidence of morbidity associated with atherosclerosis and its clinical repercussions will rise. This trend is anticipated to exert a considerable detrimental influence on the socio-economic conditions and overall quality of life within our society (2).

ACS can be classified into three subtypes: ST-elevation myocardial infarction (STEMI), unstable angina, and non-ST elevation myocardial infarction (NSTEMI). McGarry and Shenvi (2021) discuss how these subtypes are distinguished through electrocardiogram (ECG) changes and blood tests. STEMI results from complete coronary artery blockage, NSTEMI from partial blockage, and unstable angina involves ischemia without cell damage (3). Until the relatively recent past, the prevailing paradigm regarding atherosclerosis was primarily centered around the notion of a straightforward process involving the deposition of lipids within the vascular wall, which was subsequently accompanied by a reactive proliferation of vascular smooth muscle cells (VSMCs) that ultimately resulted in the narrowing of the arterial lumen, known as luminal stenosis (4).

Selectins, complex glycoproteins essential for the immune system, play a crucial role in tissue healing and inflammation, as highlighted by Kristensen et al. E-selectin, P-selectin, and L-selectin interact with endothelial cells and contribute to the immune response (5). The interaction between selectins and cell surface glycans facilitates leukocyte adherence to vascular surfaces, promoting their delivery to inflammation sites. This adherence follows a systematic "adhesion cascade," reliant on selectins, as described previously (6). E-selectin is a potential risk factor for ACS due to its association with inflammatory cell density, which contributes to plaque development. Its expression is stimulated by lipopolysaccharide, interleukin-1β (IL-1β), and tumor necrosis factor-alpha (TNF-α). E-selectin is also linked to various diseases, including cancer and metastasis, underscoring its pathological relevance (7, 8). Endothelial cells in multiple tissues, such as skin microvessels and bone marrow, express E-selectin. Two key glycoprotein ligands that interact with selectin are E-selectin ligand-1 (ESL-1), which binds to E-selectin, and P-selectin glycoprotein ligand-1 (PSGL-1), which binds to P-selectin, both expressed by cytokine-activated endothelial cells (6). TNFα promotes inflammation through gene activation, cell proliferation, and blood coagulation. Endothelial dysfunction is a critical phase in atherosclerosis progression, and while TNFα partially explains the higher atherosclerosis event frequency in overweight patients, E-selectin is essential for the rolling of effector cells (B and T cells), monocytes, neutrophils, and natural killer cells in the intima (9, 10).

The connection between TNF-alpha and E-selectin is established in numerous inflammatory disorders. TNF-alpha is recognized for its ability to promote the expression of E-selectin on endothelial cells, thereby enhancing the recruitment and adhesion of leukocytes. This process is crucial to the inflammatory mechanisms associated with ACS. Research examining the impact of TNF-alpha antagonism in obese individuals with metabolic dysregulation revealed that such antagonism notably decreases E-selectin levels, highlighting the regulatory influence of TNF-alpha on E-selectin expression. This highlights the need to examine the relationship between E-selectin and TNFα in patients with ACS, which is the focus of the current study, and the correlation between factors that can affect ACS. While E-Selectin's role in ACS has been studied, the combined evaluation of TNF-α and E-Selectin in hospitalized patients, correlated with BMI, age, and all of the lipid profiles, remains unpublished, therefore we aimed to highlight the exact status of the abovementioned molecules in a case-control study.

Methods

This case-control study involved 120 individuals 60 patients with ACS and 60 healthy controls—from outpatient clinics in Nassiyria Province, Iraq, between January and December 2024. A comprehensive questionnaire gathered demographic data, including gender and age for both groups. Patients aged >18 years who were self-confessed in the Department of Cardiology were studied. Appropriate age and sex-matched healthy participants without any prior history

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of ACS were included as controls. We intended to study 60 patients with severe acute chest pain and suspected ACS. The diagnosis and confirmation of ACS was established through positive ECG alterations, enzyme level increases, and angiographic evaluations.

Patients exhibiting typical symptoms along with ST elevation of ≥2 mm in leads V1-V3 or ≥1 mm in other leads, ST depression indicative of posterior myocardial infarction, new left bundle branch block, or established myocardial infarction characterized by Q waves of ≥0.04 seconds in leads V1-V6 or II, aVL, aVR, or elevated cardiac enzyme levels were included. A minimum of two of these criteria were required for a diagnosis of STEMI in the study. For Non-STEMI inclusion, at least two of the following criteria were necessary: 1.Characteristic chest pain, 2.ST depression greater than 1 mm, 3.T wave inversion, and 4.Positive troponin levels (11). Strict exclusion criteria removed individuals with chronic conditions such as diabetes, liver cirrhosis, end-stage renal failure, acute heart failure, strokes, musculoskeletal injuries, cancer, endocrine disorders, and other inflammatory diseases that could affect study outcomes. Besides, patients who were taking medications for cardiovascular disease or other major diseases were excluded from the study. Participants' ages ranged from 39 to 72 years and were divided into two groups: 60 males diagnosed with ACS and 60 healthy males serving as controls. Body Mass Index (BMI) was calculated in kg/m² by dividing weight in kilograms by height in meters squared.

Blood sampling

Blood samples (5 mL) were obtained through a 17-gauge cannula with careful attention to ensure smooth extraction of blood to avoid artefactual platelet activation ex vivo and anticoagulated with D-phenylalanyl-L-propyl-L-arginine chloromethyl-ketone (PPACK). During vasomotor assessments, venous blood was withdrawn simultaneously from each arm and collected into tubes containing a potassium EDTA 1:9 ratio for cytokine assays. EDTA samples were centrifuged at 1000 g for 10 min at 20°C. Platelet-free plasma was decanted and stored at -80°C before assay.

Analysis of E-selectin, TNFα and related parameters

Sera samples were collected to evaluate various lipid profiles, including total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C), using a SMART-120 chemistry analyzer and a kit supplied by KHB IVD Blood Lipid Clinical Chemistry Analyzer Test Kit Biochemistry Reagent with CE (China). Low-density lipoprotein cholesterol (LDL-C) was calculated using Friedewald's and Fredrickson's formula. E-selectin (RayBio® Human E-selectin ELISA Kit; Acession No: P16581) and TNFα (RayBio® Human TNF alpha ELISA Kit; Acession No: P01375) were measured in sera samples using the Sandwich-ELISA technique with RayBio® kits according to the manufacturer's instructions by sandwich ELISA method.

The assay used two monoclonal antibodies against two different epitopes of human TNF-alpha and E-Selectin. For each factor, the wells coated with antihuman monoclonal antibody samples to be measured or standards were incubated. After washing, a peroxidase-conjugated anti-human monoclonal antibody was added into the microwell and incubated. After another washing, the peroxidase substrate was mixed with the chromogen and allowed to incubate for an additional period of time.

An acid solution was then added to each well to terminate the enzyme reaction and to stabilize the developed color. The optical density (OD) of each well was measured at 450 nm using a microplate reader. The concentration of TNF-alpha and E-Selectin were calibrated from a dose-response curve based on reference standards. After generating the standard curves for each factor in each well, it was converted from the mean fluorescence intensity by using the linear portion of the standard curves, and the results were expressed as pg/mL

Statistical Analysis

Data analysis was conducted utilizing IBM's Statistical Package for the Social Sciences, version 27.0 (SPSS, Chicago, Illinois, USA) and R Studio 9.4. The Shapiro-Wilk test was applied to assess the normality of the data distribution. T-tests and analysis of variance (ANOVA) tables were employed to compare the mean values of biomarkers across different groups. By employing receiver operating characteristic (ROC) analysis, the Area Under the Curve (AUC), optimal threshold, sensitivity, specificity, and the 95% confidence interval lower and upper limits of E-Selectin were determined in critical scenarios. The association between ACS and lipid profiles, TNF-alpha, and E-selectin was investigated

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using Pearson's correlation coefficient. A binary logistic regression model was utilized to control for body mass index (BMI) and dependent variables. Scale variables that exhibited a normal distribution were expressed as mean ± standard deviation. A p-value of less than 0.05 was deemed statistically significant.

Results

Baseline demographical characteristics of patients with ACS and control

Based on the obtained results, the mean ± SD age in the ACS group and control group recorded 58.3 ± 10.45 years and 56.23 ±10.86 years, respectively, with a difference that was not statistically significant (P=0.914) (Table 1). As shown in Table 1, the mean ± SD of BMI in the control group was 23.07 ± 0.66 kg/m^2, while the ACS group recorded 28.06 ± 0.83 kg/m^{2,} which was statistically significant (P<0.05). The lipid profile and correlation with the other variables

According to the findings acquired, TC levels in the ACS group and the control cohort were assessed at 227.95 ± 18.35mg/dl and 181.43 ± 8.5mg/dl, respectively, with the observed differences reaching statistical significance (P<0.05). Furthermore, notable disparities were identified in the TG concentrations between the two studied groups; specifically, the ACS group exhibited levels of 269.53 ± 17.78 mg/dl, while the control group demonstrated values of 168.08 ± 12.95 mg/dl (P<0.001). Conversely, HDL-C levels in the ACS group (32.63 ± 2.17mg/dl) were markedly lower than those in the control group (55.68 ± 2.27mg/dl) (P<0.001). Additionally, both LDL-C and VLDL-C levels in the ACS patients were elevated compared to the control group, with these differences being statistically significant (P<0.05) (Table 1).

Table 1. Comparative analysis of anthropometric and clinical parameters by Independent T-test among two groups: ACS patients versus control subjects.

Factors	ACS	Control	P- value
Age (year)	58.3 ± 10.45	56.23 ± 10.86	P=0.914
BMI ((kg/m^2)	28.06 ± 0.83	23.07 ± 0.66	P<0.05
Total Cholesterol (mg/dl)	227. 95 ± 18.35	181.43 ± 8.5	P<0.001
Total Triglyceride(mg/dl)	269.53 ± 17.78	168.08 ± 12.95	P<0.05
HDL-C (mg/dl)	32.63 ± 2.17	55.68 ± 2.27	P<0.001
LDL-C(mg/dl)	173.92 ± 7.45	102.89 ± 6.76	P<0.05
VLDL-C(mg/dl)	43.75 ± 2.06	24.07 ± 2.01	P<0.05

The level of TNF-alpha and E-Selectin in the two groups

The findings indicate that E-selectin concentrations were markedly elevated in patients when compared to the control group, with mean ± SD values of 213.26 ± 2.72 pg/mL for patients with ACS and 175.11 ± 2.71 pg/mL for the control group (P< 0.001). Additionally, the outcomes demonstrated that TNF-alpha concentrations were markedly elevated in patients when compared to the control group, with mean ± SD values of 83.20 ± 3.88 pg/mL for patients with ACS and 45.65 ± 1.79 pg/mL for the control group (P<0.001)(Table 2 Figure 1).

The correlation and regression between the independent and dependent variables

A positive correlation was observed between BMI with higher E-Selectin (r=0.572, P<0.0001) and TNF-alpha (r=0.576, P<0.0001) levels (Table 3 and Figure 2). Additionally, a positive correlation was observed among increasing age and high BMI and TC, TG, VLDL-C, and LDL-C, while the correlation between BMI and age with HDL-C was negative (Table 3). The study population consisted of overweight individuals with dyslipidemia, revealing significant differences in BMI between the ACS patients and the control group. The alteration of lipid profiles serum levels in two

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groups was statistically significant and a positive correlation was observed between BMI with higher TC (r = 0.852, P<0.001), TG (r = 0.904, P<0.001), LDL-C (r = 0.939, P<0.001), and VLDL-C (r = 0.942, P<0.001) levels. However, a negative correlation was achieved between higher BMI and enhanced HDL-C level (r = -0.933, P<0.001) (Table 3 and Figure 2).

As illustrated in Table 4, in the weighted least-squares regression analysis, ACS and BMI were the independent variables influencing E-Selectin concentration (R^2 = 0.45, β- β-coefficient=-0.67, P<0.001; R^2=0.32, β- β-coefficient=0.57, P<0.001, respectively). Additionally, in the linear regression analysis, ACS and BMI were the independent variables influencing TNF-alpha concentration (R^2 =0.45, β- β-coefficient = -0.67, P<0.001; R^2 =0.331, β- β-coefficient =0.57, P<0.001, respectively). Additionally, in the linear regression analysis, ACS and BMI were the independent variables influencing TNF-alpha concentration (R 2 = 0.45, β- β-coefficient = -0.67, P < 0.001; R 2 = 0.331, β- β-coefficient = 0.57, P < 0.001, respectively). The independent variables' effect on the profile lipid data is described in Table 3 and Figure 2.

The receiver operating characteristic (ROC) analysis

The ROC analysis for E-selectin indicated a sensitivity of 96% and a specificity of 96%, with a 95% CI ranging from 0.726 to 0.965 and an area under the curve (AUC) of 0.846. The established cut-off point was determined to be 73.44 pg/mL or greater with a P value <0.0001, as depicted in Table 5 and Figure 3. The ROC analysis for TNF-alpha indicated a sensitivity of 93% and a specificity of 86%, with a 95% CI ranging from 0.724 to 0.973 and an AUC of 0.846. The established cut-off point was determined to be 188.65 pg/mL or greater with a P value <0.0001, as depicted in Table 5 and Figure 3.

Table 2. Evaluation of E-Selein and TNF-alpha using an Independent T-test in patients with ACS compared to a control group.

	ACS	Control	P- value
TNF-a (pg/mL)	83.20 ± 3.88	45.65 ± 1.79	P< 0.0001
E-Selectin(pg/mL)	2.76 ± 213.26	2.71 ± 175.10	P< 0.0001

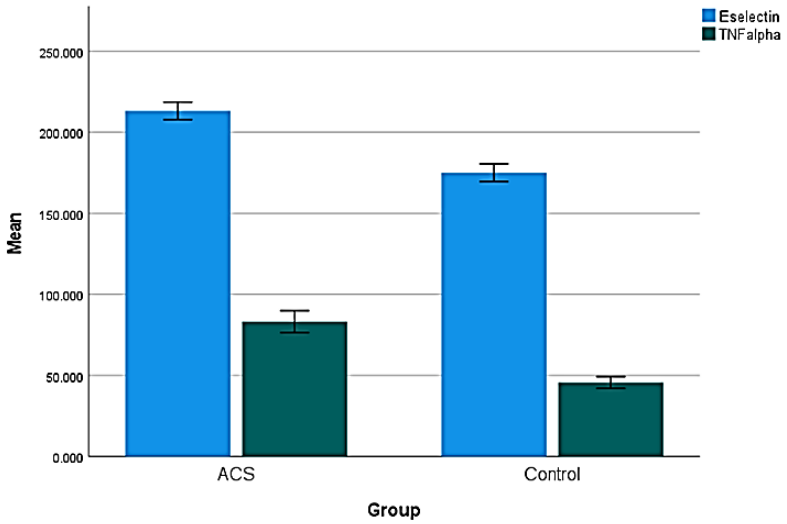


Figure 1. Comparison of TNF-alpha and E-Selectin in ACS and Control group.

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Table 3. Correlation among various variables which was carried out using Pearson's correlation coefficient.The asterisks replicate significant differences with *: p<0.05, **: p<0.01, ***: p<0.001, and ****: p<0.0001.

	BMI	Age	TC	TG	HDL-C	LDL-C	VLDL-C	E-Selectin	TNF-α
BMI	1	0.090	0.852**	0.904**	-0.933**	0.939**	0.942**	0.572**	0.576**
Age	0.090	1	0.092	0.102	-0.054	0.121	0.081	0.142	0.056
TC	0.852**	0.092	1	0.805**	-0.823**	0.841**	0.831**	0.506**	0.606**
TG	0.904**	0.102	0.805**	1	-0.918**	0.942**	0.944**	0.695**	0.680**
HDL-C	-0.933**	-0.054	-0.823**	-0.918**	1	-0.943**	-0.946**	-0.622**	-0.623**
LDL-C	0.939**	0.121	0.841**	0.942**	-0.943**	1	0.970**	0.641**	0.672**
VLDL-C	0.942**	0.081	0.831**	0.944**	-0.946**	0.970**	1	647**	0.611**
E-Selectin	0.572**	0.142	0.506**	0.695**	-0.622**	0.641**	0.647**	1	0.460**
TNF-α	0.576**	0.056	0.606**	0.680**	-0.623**	0.672**	0.611**	0.460**	1

Table 4. Multivariate stepwise regression analysis for lipid profiles, E-Selectin, and TNF-alpha as dependent variables and BMI and disease as independent variables.

Dependent variable	Independent variable	R^2	β	P-value
TC	BMI	0.72	0.85	P<0.001
	ACS	0.73	-0.856	P<0.001
TG	BMI	0.81	0.90	P<0.001
	ACS	0.91	-0.95	P<0.001
HDL-C	BMI	0.87	-0.93	P<0.001
	ACS	0.92	-0.96	P<0.001
LDL-C	BMI	0.88	0.93	P<0.001
	ACS	0.96	-0.98	P<0.001
VLDL-C	BMI	0.84	0.94	P<0.001
	ACS	0.96	-.098	P<0.001
E-Selectin	BMI	0.321	0.571	P<0.001
	ACS	0.45	-0.67	P<0.001
TNF-alpha	BMI	0.331	0.57	P<0.001
	ACS	0.45	-0.67	P<0.001

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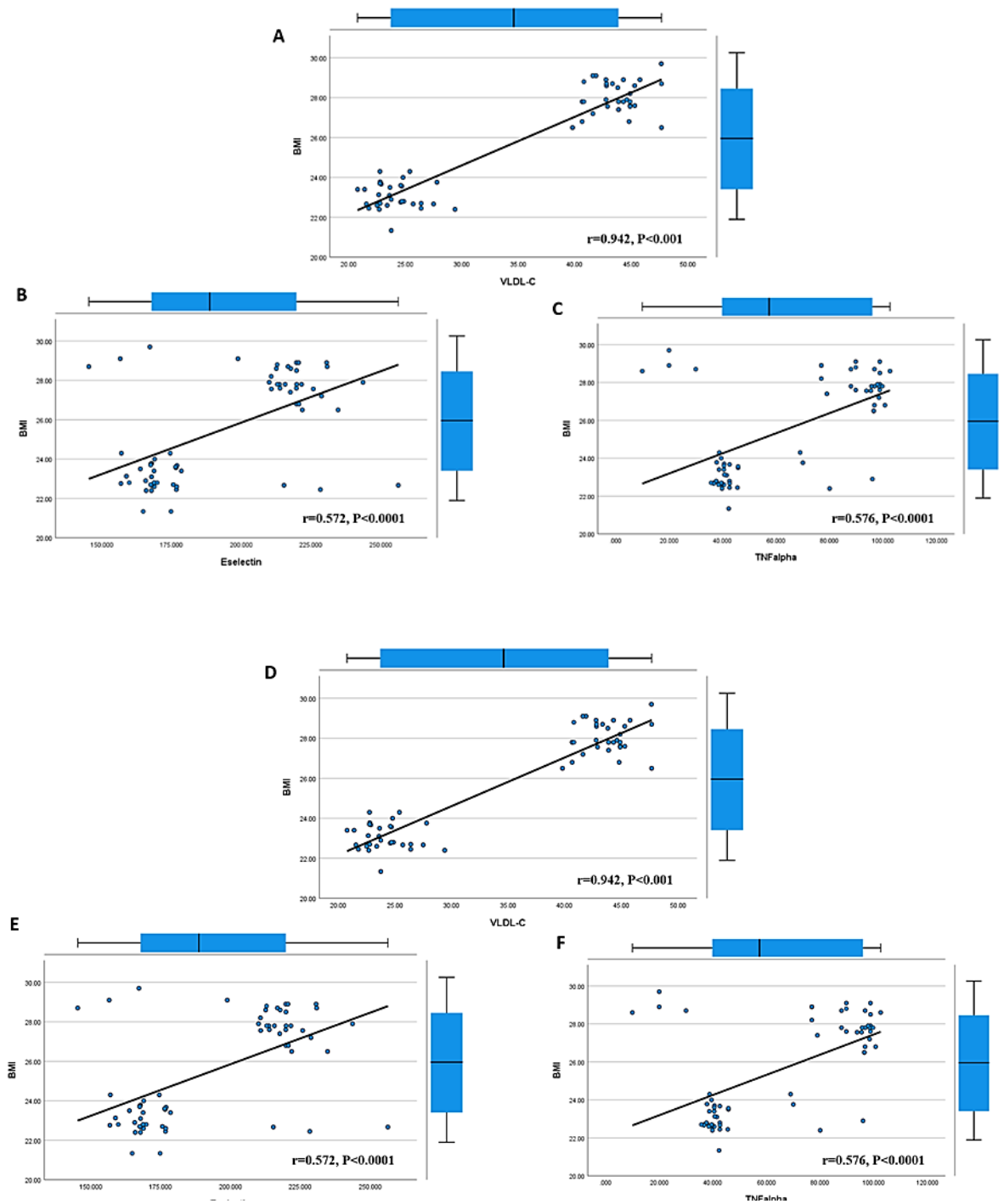


Figure 2. Regression variable plot for BMI as the independent variable and lipid profiles (A), E-Selectin (B), and TNF-alpha(C) as dependent variables. The alteration of lipid profiles serum levels in two groups was statistically significant and a positive correlation was observed between BMI with higher TC (r=0.852, P<0.001), TG (r=0.904, P<0.001), LDL-C (r=0.939, P<0.001), and VLDL-C (r=0.942, P<0.001) (D) levels. However, a negative correlation was achieved between higher BMI and enhanced HDL level (r= -0.933, P<0.001) . Based on the figure, a positive correlation was observed between BMI with higher E-Selectin (E) (r=0.572, P<0.0001) and TNF-alpha (F) (r=0.576, P<0.0001) levels.

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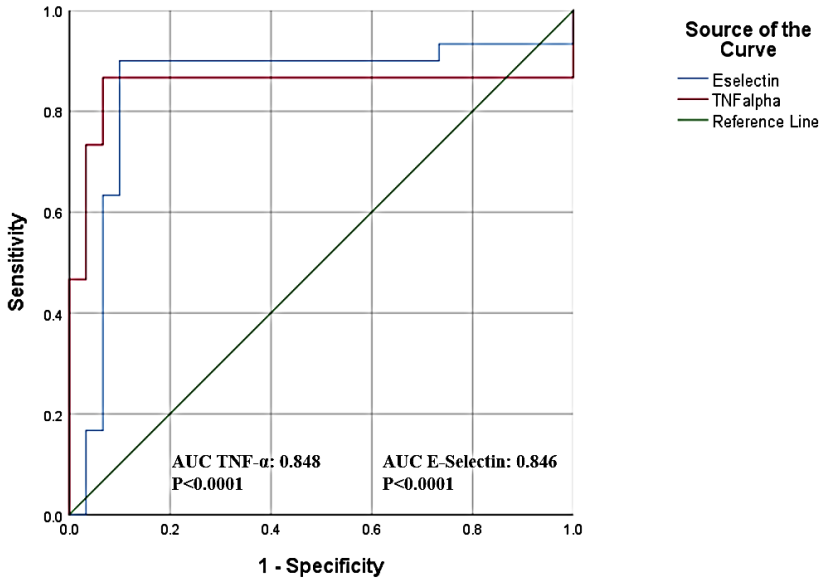


Figure 3. Receiver Operating Characteristic (ROC) Curve of E-selectin and TNF-alpha for discrimination of patients than control.

Table 5. Area Under the Curve (AUC), Best Threshold, Sensitivity, Specificity, and 95% CI Lower and Upper of E-Selectin and TNF-alpah obtained by Roc Curve for discrimination of patients than control.

Parameter	Cut-off	Sensitivity	Specificity	AUC	P-value	95% CI	
E-Selectin (pg/mL)	188.65	0.96	0.96	0.846	<0.001	0.726	0.965
TNF-alpha(pg/mL)	73.44	0.93	0.86	0.848	<0.001	0.724	0.965

AUC: Area under the curve; CI: Confidence Interval

Discussion

The correlation between TNF-alpha and E-selectin in ACS patients is well-supported by evidence from multiple studies. These biomarkers do not only serve as indicators of inflammation and endothelial dysfunction but also correlate with disease severity and outcomes. The impaired function of the endothelium is the first step in atherosclerosis development.Previously, it was shown that TNFα has a role in obesity. TNFα promotes inflammation through gene activation, proliferation of cells, and coagulation of blood. Impaired function of the endothelium is the first step in atherosclerosis development; for this reason, high TNFα was linked with obesity and a high chance of atherosclerosis (9, 10). The results of the presented study demonstrated that the patients' group exhibited a higher level of E-selectin. This is due to the inflammatory response and increased expression of cytokines that are pro-inflammatory like TNF-α, which leads to the activation of endothelial cells and ultimately leads to increased E-selectin expression. When inflammation occurs, leukocytes adhere to the walls of blood vessels through a cell adhesion molecule called E-selectin(12).

In this research, E-selectin and TNF-alpha were considered as potential prognostic biomarkers for the patient group compared to the healthy control group. This supports the fact that E-selectin and TNF-alpha are responsible for atherosclerosis during inflammatory reactions. Based on Luo et al., reported in 2022, sustained activation of TNF-α has been observed in

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individuals with stable angina and ACS in contrast to healthy individuals. Additionally, both IL-1β and TNF-α have been implicated in promoting the adhesion of monocytes to endothelial cells within atherosclerotic plaques (13).

Atban et al. concluded that TNF-α levels significantly increased in patients with ACS (6.77 pg/mL) compared to a control group (4.5 pg/mL), indicating its potential role as a biomarker for this condition. The highest levels of TNF-α were associated with myocardial infarction rather than unstable angina, suggesting that TNF-α may be more indicative of the severity of myocardial damage. Our research also highlighted a correlation between elevated TNF-α levels and a higher incidence of complications in ACS patients (7). Koukkunen et al. reported a 3.5-fold elevation in TNF-α level among patients diagnosed with ACS. They proposed two primary sources contributing to these observations: the first being the 'inflammation' factor, which encompasses C-reactive protein (CRP), fibrinogen, and interleukin-6 (IL-6), and the second being the 'injury' factor, which includes troponin-T and TNF-α (14).

In the present study, we observed about 2-fold enhancement in TNF-alpha level in ACS group in comparison to the control group which mean ± SD values of 83.37 ± 3.88 pg/mL for patients with ACS and 45.65 ± 1.79 pg/mL for the control group (P< 0.0001). Previous studies have observed elevated circulating levels of inflammatory markers such as TNF-α in ACS patients and a direct correlation with an increase in the severity of ACS and in-hospital mortality (15). Emara et al. in 2020 reported that the best cutoff level of TNF-α was 155 ng/L, where sensitivity was 90.62%, specificity was 94.12%, positive predictive value was 93.5%, negative predictive value was 91.4%, and diagnostic accuracy was 92.42%. The TNF-α level showed a significant positive correlation with fasting blood glucose (FBG), creatinine, and HbA1c in ACS patients (16). The ROC analysis for TNF-alpha in our study indicated a higher sensitivity of 93% and a specificity of 86%, with a 95% CI ranging from 0.724 to 0.973, and an AUC of 0.846 with a cut-off point of 188.65 pg/mL.

Circulating leukocytes can cross the endothelial barrier because the vascular endothelium expresses E-selectin. The fact that E-selectin is exclusively expressed by endothelial cells in the intima linked to atherosclerotic lesions makes it unique and critically important. When the amount of soluble E-selectin in the bloodstream mirrors its expression on endothelial cells, there is systemic inflammation and endothelial activation. An increase in E-selectin is thought to specifically indicate endothelial activation, reactive protein, and dysfunction (17, 18). Hateb et al., reported that the receiver operating curve for E-selectin showed a sensitivity of 85%, a specificity of 70%, a 95% CI of 0.673-0.863, and the AUC was 0.768. The cut-off point was set at 197.37 pg/ml (19). Furthermore, serum E-selectin appears to serve as a highly sensitive marker for endothelial activation. The upregulation of endothelial adhesion molecules may contribute to vascular injury, and the expression of E-selectin and L-selectin can be induced by oxidized LDL-C (12, 20). Nomura, S. et al. demonstrated that hyperlipidemic diabetic patients showed marked reductions in E-selectin and L-selectin levels six months post pitavastatin therapy (21).

Based on previous results, circulating low-density lipoprotein cholesterol has the capacity to bind to receptors on endothelial cells, where it undergoes modification or oxidation. In the arterial intima, oxidized LDL-C serves as a potent trigger for the migration and accumulation of inflammatory cells. Following their migration, monocytes differentiate into macrophages, which utilize scavenger receptors to uptake oxidized LDL-C, transforming into foam cells. The lipid irregularities were characterized by diminished HDL levels and elevated TC and LDL-C levels(22). Based on the Yang et al.'s study, in patients with ACS, the mean baseline values for TC, LDL-C, HDL-C, non-HDL cholesterol, apolipoprotein B, and remnant cholesterol were 4.61 mmol/L, 2.83 mmol/L, 1.12 mmol/L, 3.38 mmol/L, 96 mg/dL, and 0.65 mmol/L, respectively. After 12 months, LDL-C-C levels decreased significantly, with only 36.6% achieving the target of <1.8 mmol/L, highlighting the need for improved lipid management in this population (23).

In patients diagnosed with acute myocardial infarction (AMI), the mean total cholesterol level was 175 mg/dl, LDL-C was 109.53 mg/dl, HDL-C was 42.14 mg/dl, and triglycerides were 168.85 mg/dl. Notably, 46.25% of patients had low HDL cholesterol (< 40 mg/dl), indicating atherogenic lipid profiles as a common risk factor in ACS patients. This highlights the need to address lipid levels in prevention strategies for AMI (24). Yaqoob et al. in 2023 exhibited significantly higher levels of TC and TG compared to

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healthy individuals, with mean TC at 263.91 mg/dl and mean TG at 210.71 mg/dl. Additionally, a positive correlation exists between obesity (BMI > 25 kg/m²) and dyslipidemia, with higher levels of LDL-C and VLDL-C observed in obese individuals (25, 26).

The results of our study highlights the role of inflammation in the development of atherosclerosis, linking elevated levels of TNF-α and E-selectin to endothelial dysfunction. This understanding can guide future research and therapeutic strategies aimed at targeting inflammation in ACS.

The study demonstrates that both TNF- α and E-selectin can serve as reliable biomarkers for diagnosing ACS, with high sensitivity and specificity. This can lead to earlier and more accurate identification of patients at risk, facilitating timely interventions. Regular monitoring of TNF-α and E-selectin levels in patients with dyslipidemia or other risk factors for coronary artery disease can help track disease progression and the effectiveness of therapeutic interventions. This proactive approach may improve patient outcomes by allowing for adjustments in treatment plans based on biomarker levels. A positive correlation was detected between enhancing BMI and higher age with TNF-α, E-selectin, TC, TG, LDL-C, and VLDL-C, indicating that higher BMI is associated with increased levels of these factors. Overall, the study underscores the importance of TNF-α and E-selectin in clinical settings, potentially leading to improved diagnostic and therapeutic approaches for managing ACS and its associated risks.

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