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Evaluation Of E-Selectin Levels in Iraqi Patients with Acute Coronary Artery Syndrome

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Abstract:

Background: There is a great deal of mortality and morbidity associated with various cardiovascular diseases that comprise acute coronary syndrome (ACS). One crucial factor in the development of ACS is inflammation of the coronary plaque. Cell adhesion molecules play a key role in the inflammatory cascade. The vascular endothelium is directly affected by elevated levels of pro-inflammatory cytokines and other systemic inflammatory markers in ACS related to atherogenesis. This causes an increase in the expression of adhesion molecules, such as selectins.

Objective: This study aims to document the inflammatory response after acute coronary syndrome by evaluating the association between serum E-selectin levels and the risk and severity of acute coronary syndrome.

Materials and Methods: A case-control study involving 120 male subjects aged 41–70 years, who were divided into two groups: 60 ACS patients and 60 healthy individuals as a control. Serum E-selectin levels were measured using an ELISA technique.

Results: The study revealed a significant increase in serum E-selectin levels when comparing patients to the healthy control group (216.07±20.26 pg/ml Vs 179.74±53 pg/ml, $P \leq 0.0001$) respectively. The analysis of the receiver operating curve (ROC) for E-selectin showed a sensitivity of 85%, a specificity of 70%, a 95% confidence interval (CI) of 0.673-0.863, and the area under the curve (AUC) was 0.768. The cut-off point was set at 197.37 pg/ml or higher.

Conclusion: Elevated serum E-selectin levels in ACS patients suggest a potential role for adhesion molecules in the pathogenesis of ACS. Adhesion molecules could be considered as a biochemical marker for assessing ACS.

لدى المرضى العراقيين المصابين بمتلازمة الشريان التاجي الحادة E تقييم مستويات السليكتين

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الخلاصة

المقدمة :هناك نسبة كبيرة من الوفيات المرتبطة بأمراض القلب والأوعية الدموية المختلفة التي تشمل متلازمة الشريان التاجي الحادة .(ACS) أحد العوامل الحاسمة في تطور ACS هو الالتهاب في اللويحات التاجية. تلعب جزيئات الالتصاق الخلوي دورًا رئيسيًا في تسلسل الالتهاب. يتأثر بطانة الأوعية الدموية بشكل مباشر بمستويات مرتفعة من السيتوكينات المحفزة للالتهاب وغيرها من علامات الالتهاب الجهازية في ACS المتعلقة بتكون تصلب الشرايين. وهذا يسبب زيادة في تعبير جزيئات الالتصاق، مثل السليكتينات .

الهدف: تهدف هذه الدراسة إلى توثيق الاستجابة الالتهابية بعد متلازمة الشريان التاجي الحادة من خلال تقييم العلاقة بين مستويات السليكتين E في الدم وخطر وشدة متلازمة الشريان التاجي الحادة.

المرضى وطرق العمل :دراسة حالة-شاهد شملت 120 فرداً من الذكور الذين تتراوح أعمارهم بين 41-70 سنة، تم تقسيمهم إلى مجموعتين: 60 مريضًا بـ ACS و60 فردًا سليمًا كعينة تحكم. تم قياس مستويات السليكتين E في الدم باستخدام تقنية.ELISA

النتائج :كشفت الدراسة عن زيادة كبيرة في مستويات السليكتين E في الدم عند مقارنة المرضى بمجموعة التحكم السليمة 216.07±20.26) بيكوغرام/مل مقابل 179.74±53 بيكوغرام/مل، .($P \leq 0.0001$أظهر تحليل منحنى تشغيل المستقبل (ROC) للسليكتين E حساسية بنسبة 85%، وخصوصية بنسبة 70%، وفترة ثقة 95% (CI) بين 0.673-0.863، وكان مساحة تحت المنحنى .0.768 (AUC) تم تحديد نقطة القطع عند 197.37 بيكوغرام/مل أو أعلى.

الاستنتاج: تشير زيادة مستويات السليكتين E في الدم لدى مرضى ACS إلى دور محتمل لجزيئات الالتصاق في التسبب في متلازمة الشريان التاجي الحادة. يمكن اعتبار جزيئات الالتصاق كعلامة كيميائية حيوية لتقييم.ACS

1. Introduction

Acute coronary syndrome (ACS) is a condition characterized by reduced blood flow in the coronary arteries, leading to dysfunction or death of the heart muscle (Martelli et al., 2021), (Kimura et al., 2019). Common symptoms include chest pain resembling pressure, nausea, and sweating (Jasim et al., 2023). There are three subtypes of ACS: ST-elevation myocardial infarction (STEMI), unstable angina, and non-ST elevation myocardial infarction also known as NSTEMI, which can be distinguished by changes in the electrocardiogram (ECG) and blood tests (McGarry and Shenvi, 2021). STEMI occurs when a coronary artery is completely blocked, NSTEMI occurs when it is partially blocked, and unstable angina occurs when there is ischemia without cell damage or necrosis(Mihajlović et al., 2020), (Meyers et al., 2021). It is crucial to differentiate ACS from stable angina, which worsens with exertion or stress but improves with rest (Al-Tu'ma et al., 2016). Angina, whether new-onset or unstable, can strike suddenly, usually while at rest or with very little effort (Lindow et al., 2021). The adhesion molecules that selectins provide to the immune system of mammals are vital during tissue healing and the inflammatory response (Kristensen et al., 2022). Selectins are glycoproteins that serve as adhesion molecules that are vital to the immune system of mammals, particularly during inflamed response and tissue regeneration (Selvaraj et al., 2022). Cells in the cardiovascular system that bind to members of the selectin family of Ca^{2+}-dependent C-type lectins include the endothelial cell selectins (E-selectin and P-selectin) and the leukocyte selectin L-selectin., among many others (Barthel et al., 2007). Their interaction with cell surface glycans makes it easier for hematopoietic cells to adhere to vascular surfaces, which speeds up the process of circulating leukocytes rolling and delivering them to sites of inflammation (Hu et al., 2021), (Ganesh et al., 2021). Leukocytes adhere to endothelial cells in a series of steps known as the adhesion cascade, which relies on selectins (Ghazi et al., 2023). The adhesion molecule E-selectin, with a molecular weight of 115 kDa, is expressed exclusively by vascular endothelial cells (Cappenberg et al., 2022). E-selectin uses an N-terminal lectin domain of 119 residues with a 60-70% identity and approximately six cysteine-rich consensus repeats in its amino acid sequence to bind the oligosaccharide (McEver, 2015). Domains encoding epidermal growth factor and a calcium-dependent lectin at the N-terminus, the transmembrane domain, the intracellular cytoplasmic tail, and the chain of six consensus repeats make up this single-chain transmembrane glycoprotein (Tvaroška et al., 2020). Endothelial cells in the majority of tissues, such as skin microvessels and bone marrow, express E-selectin (Kappelmayer and Nagy Jr, 2017). On the other hand, lipopolysaccharide, interleukin-1b (IL-1b), and tumor necrosis factor-alpha (TNF-α) (Jubeli et al., 2012). stimulate E-selectin receptor expression. Various cancers, tumor angiogenesis, and metastasis have been linked to E-selectin expression (Noo et al., 2022).

The two important glycoprotein ligands that bind to selectin are E-selectin ligand-1 (ESL-1) for E-selectin and P-selectin glycoprotein ligand-1 (PSGL-1) for P-selectin expressed by cytokine-activated endothelial cells (Yeini and Satchi-Fainaro, 2022). E-selectin plays a crucial role in recruiting white blood cells to the injured areas. The cytokines IL-1b and TNF-α are released by macrophages in inflamed tissue, which stimulate nearby endothelial cells to over-express E-selectin (Spertini et al., 2019). Leukocytes in the blood that carry the appropriate ligand bind to E-

selectin weakly when subjected to the shear stress of blood flow. This causes the leukocytes to "roll" along the blood vessel's interior surface, creating and breaking brief interactions(Babar et al., 2019) **.** E-selectin may be a risk factor for acute coronary syndrome, especially since it is positively associated with the density of inflammatory cells, which are a major cause of plaque formation in the intima (Simon and Goldsmith, 2002)**.** This requires evaluating the association between E-selectin and the inflammatory response in ACS patients, and this is what the current study seeks to document.

2. Material, Patients and Method

Using a case-control research approach, data was collected from 120 male subjects obtained between Dec. 2022 and Nov. 2023. The subjects were aged between 41 to 70 years, and divided into two groups: 60 males with ACS patients and 60 apparently healthy males as a control group. The Sandwich-ELISA method was used to measure the levels of serum E-selectin (UNO/HUMAN/ Germany), and a SMART-120 chemistry analyzer was used to measure the levels of lipid profiles and other compounds in human serum. (AFLO / Germany, colorimetric enzymatic method). The BMI is expressed as kg/mˆ2, which is the result of dividing the weight (in kg) by the square of the height (in m) (Mirzaei and Khajeh, 2018)**.** The questionnaire gathered demographic data such as sex, age, smoking status, sedentary lifestyle, and family history of ACS for both patients and healthy controls. Exclusion criteria included subjects with chronic diseases like diabetes, cirrhosis, end-stage renal disease, acute heart failure, stroke, skeletal muscle injury, malignancy, endocrine dysfunction, and other inflammatory conditions .

2.1.Statistical Analysis

Data analysis was conducted using IBM's Statistical Package for Social Sciences, version 22.0 (SPSS, Chicago, Illinois, USA). scale variables for normally distributed data were displayed as mean ± standard deviation. The Shapiro-Wilk test was used to assess data distribution for normality. T-tests and analysis of variance tables were employed to compare the means of the biomarkers among different groups. A p-value below 0.05 was deemed statistically significant. Through the use of receiver operating characteristic (ROC) analysis, the ideal sensitivity and specificity threshold for critical cases was ascertained.

3. Results

Table 1 and Fig.1 and Fig.2. demonstrate the levels of serum E-selectin and other biomarkers between the study groups. E-selectin levels were significantly higher in patients compared to the control group, with a mean ± SD of 216.07±20.26 pg/ml in CAD patients and 179.74± 53.14 pg/ml in the control group. The patients in the study were overweight and had dyslipidemia, and BMI significantly differences in between the ACS patients and control groups. Additionally, there are significant differences in the levels of TC, TG, HDL-C, LDL-C, and VLDL-C, as presented in Table 1. The analysis of the receiver operating curve (ROC) for E-selectin showed a sensitivity of 85%, a specificity

of 70%, a 95% confidence interval (CI) of 0.673-0.863, and the area under the curve (AUC) was 0.768. The cut-off point was set at 197.37 pg/ml or higher. As shown in Table 2 and Fig.3.

Table 1: Comparison of Anthropometric and Clinical Parameters Between the Two Groups of ACS Patients and Controls.

Parameter	ACS Mean ± SD N = 60	Control Mean ± SD N = 60	P-value
Age, (year)	57.82±8.00	54.08±8.71	0.039
BMI, (kg/m^2)	27.40±3.75	23.70±1.80	≤ 0.0001
TC, (mg/dl)	247.38 ±44.36	176.50 ±14.50	≤ 0.0001
TG, (mg/dl)	221.29 ±82.93	128.53 ±29.67	≤ 0.0001
HDL-C, (mg/dl)	34.44 ±2.48	53.85 ±7.52	≤ 0.0001
LDL-C, (mg/dl)	170.58 ±39.97	98.03 ±42.80	≤ 0.0001
VLDL-C, (mg/dl)	42.07 ±17.75	21.70 ±5.76	≤ 0.0001
E-selectin, (pg/ml)	216.07±20.26	179.74±53.14	≤ 0.0001
T-test was significant at p ≤ 0.05; SD: standard deviation; S: significant; TC: Total cholesterol; LDL-C: Low-density lipoprotein cholesterol; TG: Triglycerides; HDL-C: High-density lipoprotein cholesterol; VLDL-C: Very low-density lipoprotein cholesterol BMI: Body mass index			

Table 2: Area Under the Curve (Auc), Optimal Threshold, Sensitivity, And Specificity Of E-Selectin Obtained by Roc Curve in Patients.

Parameter	Cut-off	Sensitivity	Specificity	AUC	P-value	95% CI	
E-Selectin (pg/ml)	197.37	0.85	0.70	0.768	0.0001	0.673	0.863
AUC: Area under the curve; CI: Confidence Interval							

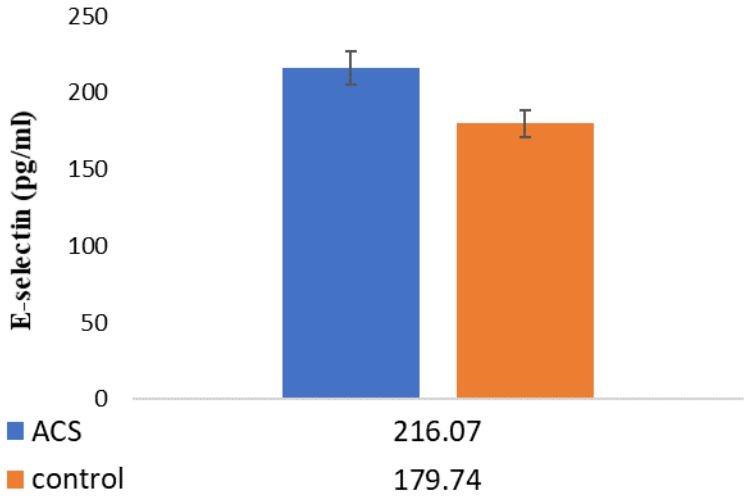

Figure 1: The Difference in The Mean Levels Of E-Selectin Between ACS And the Control Group (T-Test Was Significant at P ≤ 0.05).

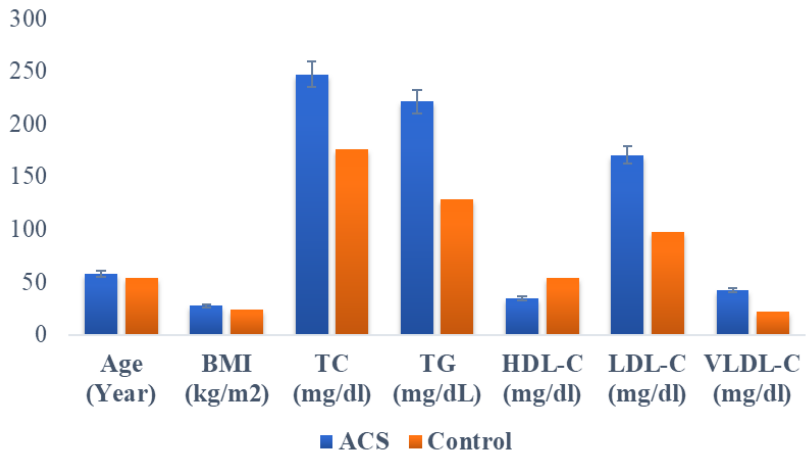

Figure 2: The Difference in Mean Levels of Age, BMI, And Lipid Profile Between the Two Groups of ACS Patients and Controls (T-Test Was Significant at $P \leq 0.05$)

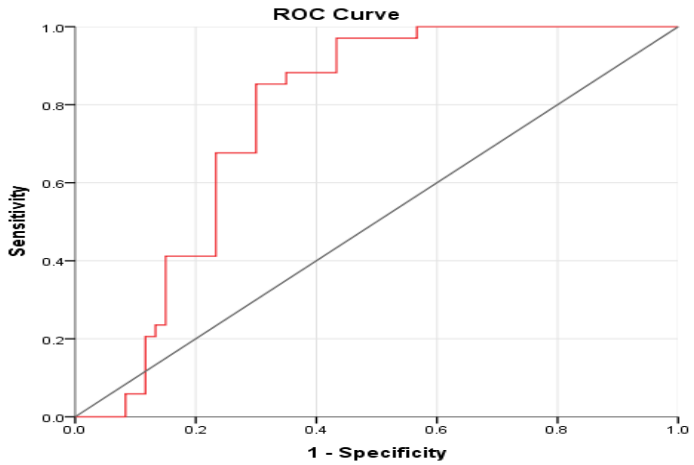

Figure 3: Receiver Operating Characteristic (ROC) Curve of Serum E-Selectin Level as Discriminators of Patients

4. Discussion

The results demonstrated that the patients' group exhibited a higher level of E-selectin. The current study demonstrated that E-selectin levels were higher in the patient group compared to the controls (ACS, 216.07 ± 20.26 pg/ml; controls, 179.74 ± 53.14 pg/ml; T-test, $P = 0.0002$). This is due to the inflammatory response and increased expression of pro-inflammatory cytokines such as IL-1b and TNF-α, which leads to the activation of endothelial cells and ultimately leads to increased E-selectin expression (Fang et al., 2011)**,** (Sutton et al., 2014)**.** Soluble E-selectin may exacerbate

inflammatory disease symptoms by activating β2 integrins and influencing leukocyte movement during the rolling process (Zhang, 2022). In this study, E-selectin was evaluated as a prognostic biomarker for patient groups in comparison to the healthy control group. De novo synthesis on endothelial cells can stimulate E-selectin production. E-selectin peaks reacting to inflammatory triggers, such as IL-1, lipopolysaccharide, or TNF-α, and then returns to baseline levels within 10 to 24 hours (Shephard, 2003). This supports the findings presented in this study, as samples are collected within a duration of 10 to 14 hours from the onset of symptoms in patients in this study. Prior human studies have been conducted on E-selectin concentrations in cardiovascular disorders. When inflammation occurs, leukocytes adhere to the walls of blood vessels through a cell adhesion molecule called E-selectin. E-selectin is necessary for effector T cells, B cells, neutrophils, monocytes, and natural killer cells to roll in the intima (Del Zoppo et al., 2000). Circulating leukocytes can cling to and cross the endothelial barrier because the vascular endothelium expresses E-selectin (González-Amaro and Sanchez-Madrid, 1999). 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 (Calabriso et al., 2023), (Granai et al., 2023). According to previous research, many adhesion molecules are found to increase when atherosclerotic conditions, whether chronic or acute, are present, consequently, an elevation in serum E-selectin is a distinct marker of endothelial activation. Recent evidence has connected inflammation to the pathophysiology of atherosclerosis-induced plaque rupture (ACS-induced) (Manuel Gomez et al., 2007).

In this study, we found that serum E-selectin levels rise when clinically significant atherosclerosis is present. Conventional histopathology has a hard time detecting a tiny number of cells. Histological detection of even a statistically significant increase in E-selectin-positive cells would be challenging, suggesting that an increase in serum E-selectin might be more indicative of a problem (Omar, 2022).

In addition, it seems that serum E-selectin is an extremely sensitive indicator of endothelial activation. It rises in preclinical disorders like dyslipidemia and falls sharply after intensive treatment with cholesterol-lowering medications (Matera et al., 2021). Increased expression of endothelial adhesion molecules may promote vascular damage, and the expression of E-selectin and L-selectin may be stimulated by oxidized LDL (Nomura et al., 2004). Nomura, S., et al., in their studies, showed hyperlipidemic diabetics exhibited significant decreases in E-selectin and L-selectin at 6 months after pitavastatin treatment. Therefore, pitavastatin could inhibit the progression of atherosclerosis by both the decrease in LDL-C and the elevation of adiponectin (Nomura et al., 2008).

5. Conclusion

Activated endothelial is essential for the development and advancement of atherosclerosis, and our present study demonstrates that E-selectin is a diagnostic marker for this process.

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