Fetuin-A Level in Detecting Coronary Artery Disease.

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Abstract

Coronary artery disease (CAD) is the leading cause of death among cardiovascular diseases. Fetuin-A, a glycoprotein synthesized in the liver and secreted into the blood, plays roles in metabolic diseases and vascular calcification. Recent evidence also indicates that Fetuin-A is implicated in the formation of mineralo-organic nanoparticles, linking its role to nanostructural biomarkers of disease. Objectives: To determine whether Fetuin-A contributes to atherosclerosis, vascular calcification, and its potential as a nanostructural biomarker for CAD detection. The study involved 160 participants (SAP, STEMI, NSTEMI, and controls). Serum Fetuin-A, troponin T, CK-MB, lipid profile, and hsCRP were measured. Results showed that serum Fetuin-A levels were significantly reduced in CAD patients compared to controls ($p < 0.0001$). Conclusion: Reduced serum Fetuin-A levels may indicate severity and risk of CAD, suggesting Fetuin-A as a potential nanostructure-associated biomarker for early detection. This link between Fetuin-A and mineralo-organic nanoparticles is in line with findings on nanostructured biomedical applications.

Keywords: Serum Fetuin A, Biomarker, Coronary Artery Disease.

Introduction

Worldwide, ischemic heart illnesses account for a majority of fatalities; specifically, 20% of all deaths are caused by coronary artery disease (Leskelä et al., 2020). The most frequent type of coronary artery disease (CAD), atherosclerosis develops when several processes come together, including changes in lipid metabolism, activation of immune cells, activation of vascular endothelial cells, proliferation of smooth muscle cells (SMCs), calcification, angiogenesis, fibrous cap rupture, thrombosis, and more (Rognoni et al., 2015). Although these processes are useful as a defining feature to find atherosclerosis by imaging, some are important steps in lesion development (e.g., vascular inflammation) and others may be obvious (e.g., plaque calcification) (Jansen et al., 2016). Atherosclerosis was thought to be significantly triggered by inflammation. Multiple investigations have shown that atherosclerotic calcification is caused by inflammation within the artery wall (Iwai et al., 2019). A possible sign of atherosclerosis is the steady development in the presence of coronary artery calcification (CAC) with the passage of time (Otsuka et al., 2014). The vascular calcification process is complex and includes a trans differentiation of vascular smooth muscle cells (VSMCs) into bone progenitor-like cells. These cells up-regulate the development of Oste chondrogenesis marker and down-regulate the assembly of specific genes, losing their contractile ability, producing collagen matrix and forming vesicles (calcium–phosphorus-rich vesicles) could initiate mineralization process in the surface of intima (Vervloet and Cozzolino 2017). The liver is responsible for producing the glycoprotein known as Fetuin-A, which stands for Alpha-2-Heremans Schmid glycoprotein AHSG. Fetuin A is secreted in serum at a high concentration (50-100 g/l) during fetal life (Karajibani et al., 2019). Kai O. Pedersendari first discovered it in 1944 in bovine calves. Among its many roles, it maintains proper bone remodeling and calcium metabolism, prevents the phosphorylation of insulin tyrosine kinase and transformation of growth factor-.TGF-, acts as an inhibitor of arterial calcification, and induces the function of cytokines, which are markers of inflammation (Panjaitan 2020). Inflammation causes a drop in the serum concentration of fetain A, which is a negative acute-phase reactant. FA helps remove and transport waste that is pro-calcific and pro-inflammatory, much like a mineral chaperone. FA helps remove and transport waste that is pro-calcific and pro-inflammatory, much like a mineral chaperone (Robinson and Teran-Garcia 2016). According to (Reynolds et al., 2005), fetain A effectively inhibits circulation calcification and blocks around half of the ectopic calcification that can occur because of various disorders. Inflammation and calcification of the arterial wall were associated with CAD (Reynolds et al., 2005). The variables that promote or prevent calcium precipitation in the vasculature are involved in the development of atherosclerotic plaques. An essential process, calcification is a predictor of coronary artery disease in the

presence of calcified atherosclerotic plaques (Saad and Essa 2019). According to (Mori et al., 2012), fetain A plays a crucial function in CAD by aiding in the processes of inflammation and calcification. By binding to calcium phosphate during the early stages of crystal development, it inhibits both crystal growth and calcium deposition, hence inhibiting calcification. Patients with vascular calcification, where the amount of FA depends on the concentration of phosphate and calcium ions, also include it in the synthesis of mineralo-organic nanoparticles (NPs) (Ceylan et al., 2015). Recent studies also support the role of Fetuin-A in CAD. For example, demonstrated a significant correlation between low serum Fetuin-A and the risk of coronary atherosclerotic disease (Zhang et al., 2025). This link between Fetuin-A and mineralo-organic nanoparticles is in line with findings on nanostructured biomedical applications (Martel et al., 2018; Vashist et al., 2017). The objective of the present study is to evaluate the diagnostic role of Fetuin-A levels in detecting CAD, with emphasis on its role in vascular calcification and nanostructure-related mechanisms.

Method and Material

Subjects and study protocol

From September 2024 to February 2025, a total of 160 individuals taken part in the study. Patients who ran into the inclusion criteria and ran for medical care at the emergency departments of Ibn Al-bitar center (IBC) for cardiac surgery and Ibn Al-Nafes (IAN) cardiac specialty teaching hospital for their newly developed symptoms of ischemic heart disease (IHD) were included in the study. Electrocardiograms (ECG), echocardiography (ECHO), and cardiac enzyme testing validated the clinical diagnosis of stable angina or myocardial infarction (MI), based on the patient's history and symptoms of CAD. Inclusion requirements People on immunosuppressant medications, those with liver disorders, and those with myocarditis conditions such as pregnancy, diabetes mellitus (DM), malignant tumors (MT), and patients undergoing heart surgery Individuals suffering from renal failure.

Collecting samples and procedures

In the morning, while patients and controls were fasting, veins were tapped to draw approximately five milliliters of blood. The subjects' blood was taken into a gel tube and left to sit at room temperature for twenty minutes (20 minutes). Immediate evaluations of serum fetuin A, troponin T, CK-MB, Lipid profile, and hsCRP were performed after sera were separated by centrifugation (2000 xg) for 10 minutes following coagulation.

Results and Discussion

The study included 120 patients divided into three groups: those with STEMI, NSTEMI, and SAP, as well as 40 healthy controls who did not have any history of heart disease or medication treatment. There was a smoking rate assessment for every group that participated in the research; eight people, or 20% of the control group, smoked cigarettes. In the STEMI group, 26 people (65%) smoked, compared to the control group, which is significantly higher. When compared to the control group, group, 24 people in the NSTEMI group smoked cigarettes, which is a significant rise (60%). In the SAP group, 14 people (35%) of the total smoked cigarettes (Figure 1). These results complement literature findings such as those reported by (Zhang et al., 2025), confirming the diagnostic value of Fetuin-A in CAD

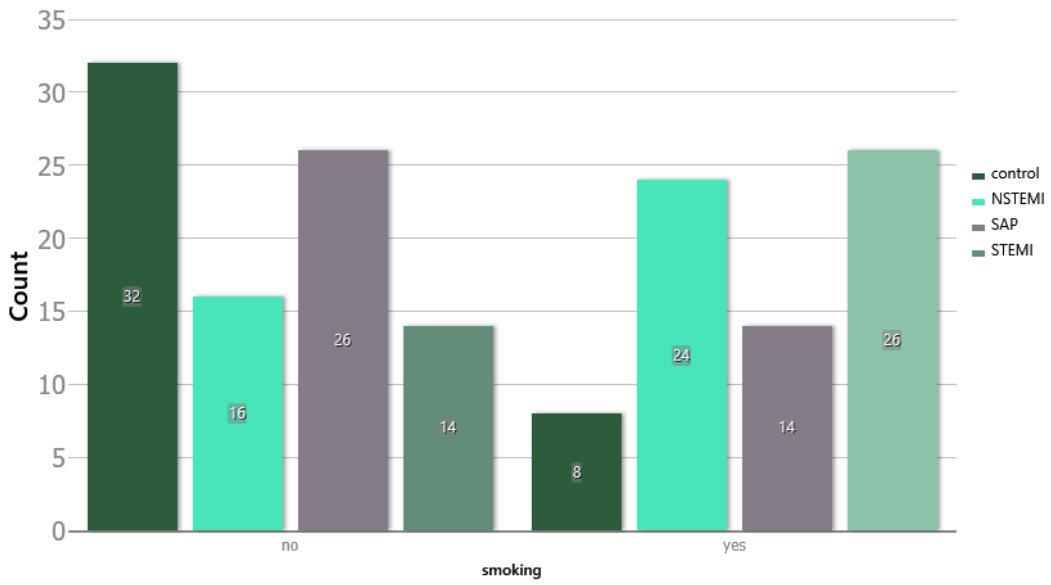

Fig. 1. Distribution of smoking status among CAD subgroups and healthy controls.

The study evaluated the lipid profiles and levels of cardiac markers, such as hsCRP, Troponin T, CKMB, and Fetuin A, for both the patients and the controls. Everything that was measured for each test, including the mean, standard deviation, skewness, kurtosis, median, and p-value (Table 1 & 2).

In the study, the mean and standard deviation (mean± SD) of serum Fetuin A levels in the SAP, STEMI, NSTEMI, and control groups were 60.37±16.84, 39.90±12.75, 21.19±7.54, and 166.61± 7.69 ng/ml, respectively. The groups in the current data showed a significant difference ($p < 0.0001$), as shown in Table (2). The results demonstrated that the ACS groups had reduced fetuin A levels compared to the

SAP and control groups. Regardless of the SAP group's fetuin A level was greater than that of the ACS group, compared to the control group, it was lower.

Table 1. **Blood biochemical analysis among CAD subgroups and controls.**

Statistic	No.	Mean±SD	Skewness	Kurtosis	Median±SEM	p-value
Cholesterol (mg/dl)						
SAP	40	221.95±40.32	-1.314	1.301	228.00±6.37	
STEMI	40	219.0±53.08	-0.523	-0.802	224.00±8.39	
NSTEMI	40	262.73±29.60	-0.582	-0.683	267.50± 4.68	**< 0.001**
Control	40	147.50±22.22	-0.288	-1.069	147.00±3.51	
Triglyceride (mg/dl)						
SAP	40	187.45±61.94	0.897	1.144	182.00±9.79	**< 0.001**
STEMI	40	208.10±30.55	0.788	1.476	206.00±4.83	
NSTEMI	40	226.95±40.97	0.554	-1.131	211.50±6.47	
Control	40	118.75±21.94	-0.379	-1.196	128.50±3.46	
HDL (mg/dl)						
SAP	40	44.10±12.05	-0.319	-0.808	46.50±1.90	**0.002**
STEMI	40	46.45±13.71	-0.312	-0.968	46.50±2.16	
NSTEMI	40	44.95±11.48	-0.228	-0.759	45.00±1.81	
Control	40	53.10±7.36	0.684	-0.674	50.50±1.16	
LDL (mg/dl)						
SAP	40	131.88±33.79	-0.211	-0.995	129.80±5.34	**< 0.001**
STEMI	40	137.53±45.68	-0.426	-0.882	153.50±7.22	
NSTEMI	40	175.97±37.89	0.614	0.208	170.70±5.99	
Control	40	78.26±20.56	-0.362	-1.388	82.95±3.25	
VLDL (mg/dl)						
SAP	40	36.71±9.85	0.633	0.235	35.60±1.55	**0.427**
STEMI	40	41.61±6.11	0.786	1.467	41.20±0.96	
NSTEMI	40	44.41±8.41	0.687	-0.872	41.80±1.33	
Control	40	35.43±53.10	4.070	14.748	25.00±8.39	

Table 2. **Cardiac ischemia markers among CAD subgroups and controls.**

Statistic	No.	Median	Mean ± SD	Skewness	Kurtosis	SEM	Pr > F*
hs CRP (mg/dl)							
SAP	40	1.35	1.55±0.94	0.42	-1.22	0.14	**< 0.0001**
STEMI	40	3.50	3.68±2.33	0.69	-0.28	0.36	
NSTEMI	40	2.90	3.27±2.28	1.06	0.59	0.36	
Control	40	0.77	0.66±0.26	-0.50	-1.33	0.04	
CKMB (U/L)							
SAP	40	23.37	26.25±20.17	3.57	15.87	3.19	**< 0.0001**
STEMI	40	46.10	48.99±10.44	0.21	-1.30	1.65	
NSTEMI	40	35.95	39.66±13.38	0.45	-0.78	2.11	
Control	40	20.10	18.12±6.19	-1.01	-0.07	0.98	
Troponin T (ng/ml)							
SAP	40	0.35	0.33±0.11	-0.73	-0.45	0.01	**< 0.0001**
STEMI	40	5.53	5.50±0.74	-0.02	-1.24	0.11	
NSTEMI	40	4.54	4.51±0.97	0.01	-0.35	0.15	
Control	40	0.06	0.10±0.08	1.70	1.68	0.01	
Fetuin A (ng/ml)							
SAP	40	57.00	60.37±16.84	0.86	-0.45	2.66	**< 0.0001**
STEMI	40	36.75	39.90±12.75	0.76	0.43	2.01	
NSTEMI	40	20.65	21.19±7.54	-0.10	-1.06	1.19	
Control	40	169.80	166.61±7.69	-0.46	-0.58	5.96	

Figure 2 and table 3 show that the Receiver Operator Characteristic (ROC) Curve, which was used to investigate Fetuin A as a biomarker for coronary artery disease (CAD) prediction, observed an extremely significant difference across all groups (p value <0.001).

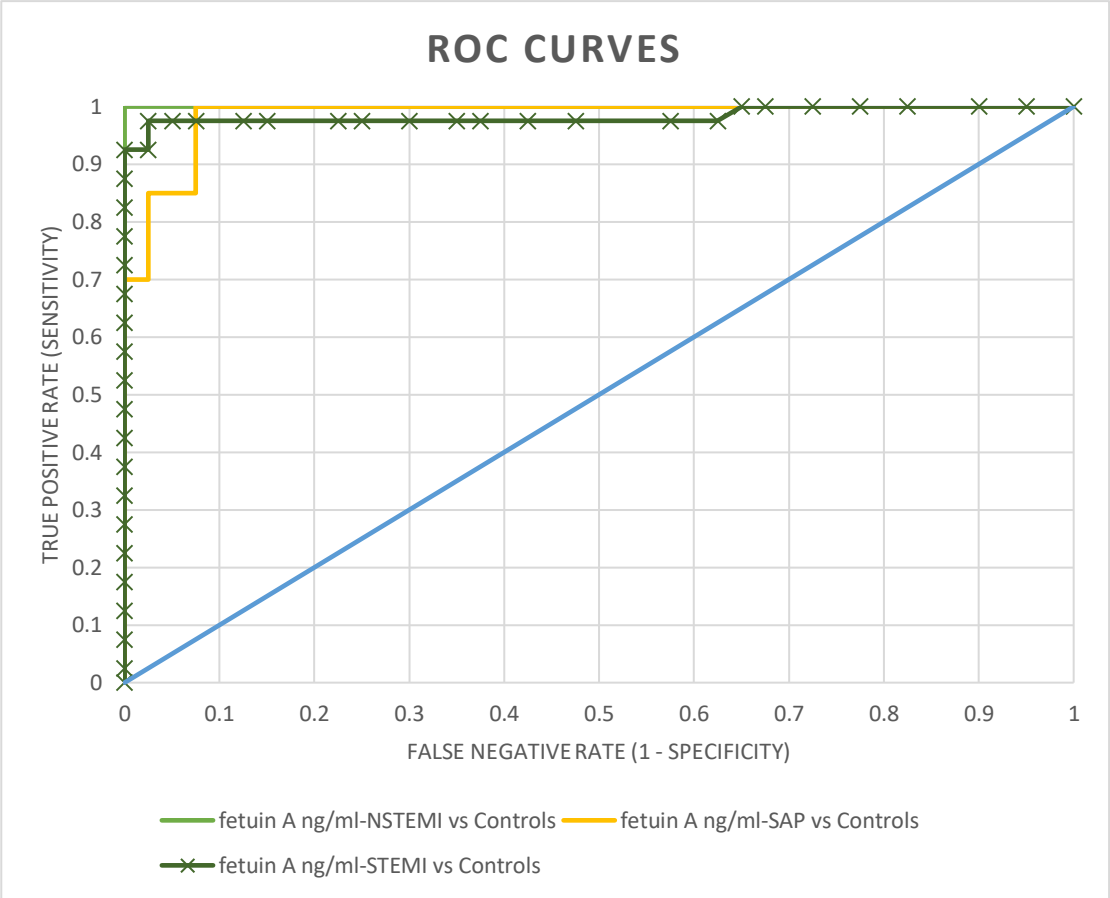

Fig. 2. ROC curve showing diagnostic ability of Fetuin-A levels to distinguish CAD patients from controls.

Table 3. **Diagnostic performance (sensitivity, specificity, accuracy) of Fetuin-A in CAD subgroups compared to controls.**

Group	Cut-off value	Specificity	Sensitivity	Area-under curve	Accuracy
SAP	90.6	85%	92.5%	0.985	0.88
STEMI	62.4	97.5%	92.5%	0.983	0.95
NSTEMI	32.4	100%	100%	1.00	1.00

Coronary artery disease (CAD) is a primary cause of death from cardiovascular disease (Joloudari et al., 2020). Vascular calcification and inflammation are disorders that increase the danger of cardiovascular disease (CVD). Arterial calcium deposits may change the stability of atherosclerotic plaques and increase vasomotor responses, both of which increase the risk of coronary artery disease (CAD) (Maddaloni et al., 2020). Figure 1 shows the results of the study, which revealed that 64 patients (53.3%) were smokers, and 56 patients (46.7%) were nonsmokers, with 8 patients (20%) in the control group being smokers. Figure 1 shows the results of the study, which revealed that 64 patients (53.3%) were smokers, and 56 patients (46.7%) were nonsmokers, with 8 patients (20%) in the control group being smokers. The results indicated significant differences between the patient groups when compared to the control group. Findings from this study confirmed those from earlier research showing smoking is a significant risk factor for CAD. Among people under the age of 50, the chance of developing an acute coronary syndrome is ten times higher for smokers than for nonsmokers (Joloudari et al., 2020; Maddaloni et al., 2020). Table (1) demonstrates a significant increase in the lipid profile ($p<0.0001$), apart from normal HDL levels and disappeared VLDL. Table (2) indicates significant variations in serum hsCRP levels between the control group and the patients investigated ($p < 0.0001$). Lower, moderate, and higher levels of hs-CRP (ranging from 1 to 3 mg/L) have been classified as cardiovascular risk, respectively. According to (Ding et al., 2019), high-sensitivity C-reactive protein (hsCRP) could be a useful biomarker to evaluate the severity of CAD to assist with risk stratification. According to (Muneeb et al., 2023), hsCRP is a biomarker that was shown to be higher in the ACS group than the SAP group, suggesting that it may be connected with plaque instability and the incidence of ACS. Consistent with earlier findings, patients in the SEMI and NSTEMI groups had significantly higher levels of CK-MB compared to those in the SAP group and the control group; this finding further supports the idea that acute coronary syndrome is associated with elevated CK-MB levels (Liu et al., 2020). A significant difference was seen between the groups that were evaluated according to the troponin T-test findings ($p <0.0001$). There was an increased risk of heart illnesses associated with elevated blood troponin levels in the SAP and ACS groups compared to the control group, according to the results.

Troponin T levels were also higher in the ACS group compared to the SAP group, and even higher in the SAP group compared to the control group. Consistent with earlier research, our findings show that troponin T levels in the blood are diagnostically useful and correlate with the severity of coronary artery disease (Pourmoghaddas et al., 2020). These findings confirmed those of earlier research showing that coronary artery syndrome was more inclined to develop in individuals with low serum Fetuin A levels (Saad and Essa 2019). It was also found that fetuin A, an anti-inflammatory protein that

plays a role in counter-regulating the immune response, was lower in the STEMI and NSTEMI groups. The inflammation process and cytokine production can be better predicted with a low Fetuin A level (Al-Hindi et al., 2010) states that the mechanism involving low Fetuin A levels is still unknown. According to (Li et al., 2019), the severity, frequency, and level of coronary artery disease were all correlated. There was a confirmed association between troponin T levels and cholesterol levels in patients with SAP, as shown by the positive correlation between the two variables (r =0.436, P = 0.005) in the SAP group's biochemical tests (Table 1). The current findings match those of earlier studies that found an association between stable angina patients' elevated troponin T and cholesterol levels and an increased risk of coronary artery disease (Salman et al., 2020). Serum hsCRP was positively correlated with CK-MB (r = 0.315, p = 0.047), demonstrating a link between the two biochemical markers in the STEMI group. Endothelial dysfunction, macrophage differentiation, plaque vulnerability, myocardial ischemia, development of MI, and subsequent releases of cardiac markers were all associated with increased hsCRP levels (Afrisham et al., 2021). In addition to the fact that CKMB and hs CRP are related, these biomarkers were linked to the likelihood and intensity of ACS (Afrisham et al., 2021). In STEMI patients, Fetuin A does not correlate with any other biomarkers (p value > 0.05). Due to its close relationship with increased mortality, arterial stiffening and calcification, and worsening of soft tissue calcification, fetuin A was found to significantly reduce and block systemic calcification (Babapour et al., 2021). This interpretation is consistent with nanotechnology-related studies in ETN, which emphasize the diagnostic potential of biomolecular nanostructures (Martel et al., 2018; Vashist et al., 2017).

Biochemical tests in the non-ST-elevation myocardial infarction (NSTEMI) group showed a positive correlation between serum hsCRP and troponin T (r = 0.340, p value 0.032), suggesting that these biomarkers, when used together, provide substantial prognostic information for the evaluation of ACS patients (Bouzidi et al., 2020). Patients with acute coronary syndrome (ACS) and elevated hsCRP levels had low serum fetuin A levels, according to the current research. It is possible to use these inflammatory markers to evaluate patients suspected of having ACS in the future (Al-Hindi 2010). Plaque rupture causes an aberrant rise of troponin T, and inflammation is a major factor in the prognosis of atherosclerosis. Consistent with other findings, this study found that HsCRP and troponin T were linked with the presence of CAD (Pourmoghaddas et al., 2020). These findings are in agreement with those of a previous study by (Narain et al., 2008), which found that high levels of serum cholesterol and low levels of serum Fetuin A were both linked to an increased risk of atherosclerosis and its subsequent complications. Inflammation causes an increase in the concentration of HsCRP, a positive acute phase

protein, and a reduction in the concentration of fetuin A, a negative acute phase protein (Al-Hindi 2010). According to the results, the serum Fetuin cut-off value Table (3) and figure (2) show that the concentration in the SAP group was 90.6 ng/ml, which had an 85% specificity and a 92.5% sensitivity. The cutoff value for serum fetuin was also mentioned. The concentration in STEMI was 62.4 ng/ml with 97.5% specificity and 92.5% sensitivity, while NSTEMI was 32.4 ng/ml with 100% specificity and sensitivity, as shown in table (3) and figure (2). Because of its high specificity and sensitivity, fetuin A has been shown to have a role in the pathogenesis of coronary artery disease by lowering serum levels in these patients.[11] found similar results in their own study.

Conclusion

The present study concludes that reduced serum Fetuin-A levels are strongly associated with coronary artery disease. Given its role in vascular calcification and nanoparticle formation, Fetuin-A could serve as a promising biomarker for early detection and risk stratification of CAD.

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