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STUDY OF FETUIN A AS BIOMARKER FOR CORONARY ARTERY DISEASES IN SOME EGYPTIAN PATIENTS

By

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ABSTRACT

Background: Fetuin A protein is Alpha 2-Heremans Schmid glycoprotein (AHSG). It acts as negative acute phase reactant synthesized by the liver cells, it is responsible for preventing calcium and phosphate precipitation in the blood by increasing their solubility and inhibiting calcium crystal growth. Deposits of calcium phosphates (hydroxyapatites) in the muscular layer of the blood vessels appear to play not only a significant role in stiffening arteries but also for the induction of an early phase of coronary arteriosclerosis. Fetuin A has been considered to play a crucial role in the protection from vascular calcification by solubilizing calcium and phosphorus in serum**. Objective:** the aim of this study was to determine the level of fetuin A in some Egyptian patients with coronary artery disease and its correlation with the severity of the disease. **Subjects and methods:** sixty Egyptian persons were involved in this study, twenty of them were healthy as a control, other twenty of them were patients with stable angina, the last twenty were patients with acute myocardial infarction. Serum fetuin A, CRP, CK-MB, troponine I, creatnine, lipid profile and blood glucose estimation were performed. In addition, exercise ECG was done for all individuals. **Results:** serum fetuin A level decreased in the two groups with coronary artery diseases than the control; it also markedly decreased in patients with myocardial infarction than both those with stable angina and control. **Conclusion:** This study showed that decreased serum fetuin A level was correlated with the development of coronary artery disease. Fetuin A might be clinically valuable for reflecting the progression of coronary artery disease.

Key words: Fetuin A, coronary artery disease, vascular calcifications.

INTRODUCTION

Coronary artery disease is a complex chronic inflammatory disease characterized by remodeling and narrowing of the coronary arteries supplying oxygen to the heart. The World Health Organization (WHO) has revealed that coronary artery disease has become the leading cause of mortality worldwide **(Aslan and Dogan, 2011).** According to the latest WHO data published in May 2014, coronary heart disease deaths in Egypt reached 23.14% of total deaths **(WHO, 2014).**

Deposits of calcium phosphates (hydroxyapatites) in the muscular layer of the blood vessels appear to play not only a significant role in stiffening arteries, but also for the induction of an early phase of coronary arteriosclerosis, i.e atherosclerotic calcification **(Lanza, 2007).**

Fetuin A is a multifunctional protein that can modulate and inhibit vascular

calcification in both local and systemic manner. Fetuin A can locally inhibit calcifications in the walls of the blood vessels. There, it is internalized by vascular smooth muscle cells (VSMCs) and incorporated into the matrix vesicles from apoptotic and viable VSMCs. This results in inhibition of mineral nucleation. It also inhibits VSMC apoptosis and enhances phagocytosis of vesicles, thus further limiting their ability to mineralize the vasculature **(Stenvinkel et al., 2005).**

Fetuin A also acts systemically by binding excess mineral and forming fetuin–mineral complex. That is called calciprotein particles (CPPs). Fetuin A is essential for the formation and stabilization of calciprotein particles. CPPs must be cleared from circulation to prevent local deposition and pathological calcification. Fetuin A containing CPPs facilitate the clearance of mineral debris and are cleared by the reticuloendothelial system, namely Kupffer cells of the liver and marginal zone macrophages of the spleen via scavenger receptor-AI/II (SR-AI/II) and eventually leaves the body through the kidney **(Herrmann et al., 2012).**

Fetuin A, a negative acute-phase protein, is implicated as an anti-inflammatory mediator, that participates in macrophage deactivation, antifibrotic activity, and inhibition of apoptosis of vascular smooth muscle cells **(Li and Zhu, 2011)**. **Merx et al. (2005)** reported promotion of cardiac fibrosis, calcification, notably impaired diastolic function, and tolerance to ischemia as well as catecholamine resistance in the hearts of fetuin A knockout mice.

SUBJECTS AND METHODS

The subjects in the current study were sixty who were selected from the Coronary Care Unit and out patients clinics in Al-Hussein Hospital, Al-Azhar University, Cairo, Egypt. Investigations were carried out at Al-Azhar Faculty of Medicine for Girls, Biochemistry Department. The protocol of this study was approved by the medical ethics committee in the Faculty of Medicine for Girls, Al-Azhar University and written informed consent was obtained from all participants.

The subjects were divided into three equal groups: -

- **Group I (Control group)** included healthy individuals (11 males and 9 females) with age ranged from 45-65 years with mean age (52.6±7.4). They had no symptoms or signs of cardiac ischemia which were confirmed by normal electrocardiography (ECG) during exertion.
- **Group II (Stable angina)** included patients (12 males and 8 females) with age ranged from 41-64 years with mean age (57.1±6.8). They were suffering from stable angina. All these patients had exertion or emotional stress associated with electrocardiographic (ECG) horizontal or down sloping ST segment depression.
- **Group III (Myocardial infarction)** included patients (15 males and 5 females) with age ranged from 45-68 years with mean age (55.7±7.1). All these patients were admitted to the Coronary Care Unit with a clinical diagnosis of acute myocardial infarction (MI). They were diagnosed

according to the criteria of the world health organization (WHO) that require at least two of the following three elements to be present for the diagnosis of acute MI: (1) A history of ischemic-type chest discomfort, (2) Changes on serially obtained ECG, (3) A rise and fall in serum cardiac markers **(Pedoe et al., 1994).**

Exclusion criteria:- Patients with malignancies, osteoporosis, systemic or local infection, hepatic and renal diseases (creatinine levels >2.0 mg/dL) and patients receiving systemic glucocorti-coids or immunosuppressants were excluded from the study. All previous diseases can affect serum fetuin A level (**Oktay et al., 2010)**.

Specimen Collection:

Five milliliters (ml) of venous blood was taken into plain collection tubes and allowed to clot 20 minutes before centrifugation. Serum was stored at –20^{o}C until utilized.

All studied groups were subjected to the following:-

1. Full history taking including history of CAD, smoking and diabetes.
2. General examination including BMI.
3. Laboratory investigations which included:

(1) Fasting plasma glucose according to Spinreact kit **(Young, 2001)**.

(2) Serum creatinine according to Diamond kit **(Henry, 1974)**.

(3) Serum lipid profile which included serum cholesterol was measured according to spinreact kit **(Burtis and Ashwood, 1999)**, serum triglycerids according to the method of **Bjorksten (1972),** serum high density lipoprotein (HDL) according to the method of **Burtis and Ashwood (1999)** by spinreact kit, estimation of serum low density lipoprotein according to Fiedewald Formula **(LDL) =** Total cholesterol – (HDL + triglyceride/5).

(4) Cardiac troponin I by rapid test device **(Mehegan and Tobacman, 1991)**.

(5) Serum C-reactive protein by Latex serology detection of C-reactive protein **(Fisher and Nakamura, 1976)**.

(6) Serum CK-MB by colorimetric kit supplied by Chrono Lab **(Gerhardt, 1979)**.

(7) **Serum fetuin A level by ELISA** (Enzyme Linked Immuno Sorbent Assay) Kit supplied by WKEA MED SUPPLIES CORP. 1325. USA **(Olivier et al., 2000)**.

Statistical analysis: All statistical analysis were performed by SPSS V17 using descriptive statistics. Mean ± SD for out the outcome variable of interest were computed. One way analysis of variance (ANOVA) with repeated measures. Chi square was used for comparison of dependent variables. $P<0.05$ was considered to be significant. Receiver Operating Curve (sensitivity and specificity) was used.

RESULTS

As regards the clinical assessment there were non significant differences between studied groups in age, sex distribution, BMI, family history of CAD, number of smokers and non – smokers. Number of diabetics and non- diabetics. All these risk factors were cross matched between groups to abolish their effect on serum fetuin A level.

Table (1): Clinical data of all studied groups:

Groups / Variables	Control (N=20)	Stable angina (N=20)	Myocardial infarction (N=20)	Tests	
				X^2/t	P-value
Sex					
Female	9(45.0%)	8(40.0%)	5(25.0%)	1.866	0.393 N.S
Male	11(55.0%)	12(60.0%)	15(75.0%)		
Age					
Mean±SD	52.6±7.4	57.1±6.8	55.7±7.1	2.090	0.133 N.S
BMI (Kg/m2)					
Mean±SD	30.0±3.8	31.2±3.8	30.7±2.4	0.606	0.549 N.S
Family history of CAD					
Negative	15(75.0%)	12(60.0%)	14(70.0%)	1.078	0.583 N.S
Positive	5(25.0%)	8(40.0%)	6(30.0%)		
Smoking					
Negative	14(70.0%)	10(50.0%)	10(50.0%)	2.172	0.338 N.S
Positive	6(30.0%)	10(50.0%)	10(50.0%)		
Hypertension					
Negative	15(75.0%)	13(65.0%)	14(70.0%)	0.476	0.788 N.S
Positive	5(25.0%)	7(35.0%)	6(30.0%)		
Diabetes Mellitus					
Negative	15(75.0%)	10(50.0%)	13(65.0%)	2.727	0.256 N.S
Positive	5(25.0%)	10(50.0%)	7(35.0%)		

P value is significant at P <0.05.

As regard laboratory finding there were no significant differences in plasma glucose level, serum creatinine level but there were significant differences in serum TAG, HDL, LDL, CK-MB levels, also there was significant difference in number of CRP positive and CRP negative between groups. There was significant difference in number of cardiac troponin I positive and negative between groups.

Table (2): Laboratory data of all studied groups:

Groups / Variables	Control (N=20)	Stable angina (N=20)	myocardial infarction (N=20)	Tests	
				X^2/t	P-value
CRP (number %)					
Negative	18(90.0%)	15(75.0%)	0(0.0%)	37.576	<0.001*
Positive	2(10.0%)	5(25.0%)	20(100.0%)		
Troponin					
Negative	20(100.0%)	20(100.0%)	0(0.0%)	60.000	<0.001*
Positive	0(0.0%)	0(0.0%)	20(100.0%)		
FPS (mg/dl)	99.3±27.3	117.6±39.6	117.6±42.8	1.627	0.205
Creatinine (mg/dl)	0.9±0.2	1.0±0.1	0.9±0.2	1.256	0.293
TG (mg/dl)	110.7±18.5	210.8±22.3	222.7±37.5	100.908	<0.001*
T. cholest. (mg/dl)	135.5±19.6	221.6±21.0	244.0±27.8	123.338	<0.001*
HDL-C (mg/dl)	57.0±6.6	49.9±2.8	42.8±6.6	32.303	<0.001*
LDL-C (mg/dl)	56.1±20.4	129.6±17.3	156.7±22.4	133.330	<0.001*
CK-MB (U/L)	3.4±1.5	7.5±3.9	74.0±15.9	347.966	<0.001*

P value is highly significant at P <0.05.

As regards the fetuin A level it was significantly decreased in all patient groups than the control group. It also was significantly decreased in patients with stable angina than control group, while the group with myocardial infarction has significant decreased serum fetuin A level than the two other groups.

Table (3): Fetuin A level in control group and coronary artery disease group.

Groups	Fetuin A (ng/ml)		T-test	
	Range	Mean ± SD	T	P-value
Control (n=20)	542.8 - 900.5	703.42 ± 110.25	6.593	<0.001*
Patients (n=40)	225.5 - 616.5	446.62 ± 105.21		

P value is significant at P <0.05.

Table (4): Fetuin A level in all studied groups.

Groups	Fetuin A (ng/ml)		ANOVA	
	Range	Mean ± SD	F	P-value
Control (n=20)	542.8 - 900.5	703.42 ± 110.25	69.233	<0.001*
Stable angina (n=20)	418.6 - 616.5	519.59 ± 58.26		
Myocardial infarction (n=20)	225.5 - 520.3	373.64 ± 90.09		
Tukey's test				
Control & Stable angina	**Control & Myocardial infarction**	**Stable angina & Myocardial infarction**		
<0.001*	<0.001*	<0.001*		

Table (5): ROC curve between control and patients with CAD as regard fetuin A.

ROC curve between control and patients as regard fetuin A (ng/ml)					
Cut off	**Sensitivity**	**Specificity**	**PPV**	**NPV**	**Accuracy**
<=536.42 *	**82.5**	**100.0**	**100.0**	**74.1**	**97.4%**

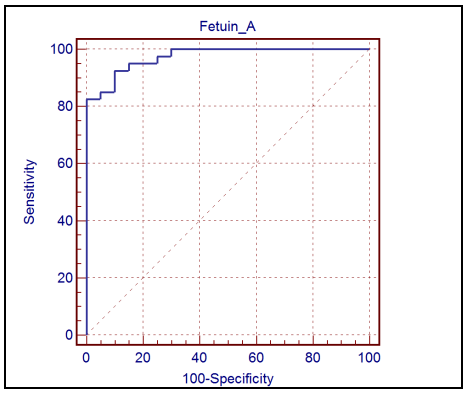

Figure (1): ROC curve between control and patients with CAD

Regarding the diagnostic accuracy of serum fetuin A as a predictor and a diagnostic marker for CAD, we analyzed the receiver operator characteristic curve (ROC) **table (5), figure (1)** and showed that cut off value was (536.42ng/ml**)**, serum fetuin A showed 82.5% sensitivity, 100% specificity with accuracy 97.4%, PPV (positive predictive value) is 100.0% and NPV (negative predictive value) is 74.1%.

Table (6): ROC curve between control and stable angina group as regard fetuin A

ROC curve between control and stable angina as regard fetuin A (ng/ml)					
Cut off	**Sensitivity**	**Specificity**	**PPV**	**NPV**	**Accuracy**
<=585.55 *	**85.0**	**90.0**	**89.5**	**85.7**	**94.8%**

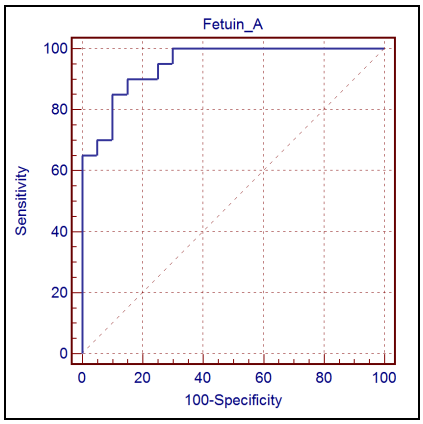

Figure (2): ROC curve between control and stable angina group.

Regarding the diagnostic accuracy of plasma fetuin A as a predictor and a diagnostic marker for stable angina, we analyzed the receiver operator characteristic curve (ROC) **table (6), figure (2)** showed that cut off value was (585.55ng/ml**)**, serum fetuin A showed 85.0% sensitivity, 90.0% specificity with accuracy 94.8%, PPV (positive predictive value) is 89.5% and NPV (negative predictive value) is 85.7%.

Table (7): ROC curve between control and myocardial infarction group as regard Fetuin A

ROC curve between control and myocardial infarction as regard fetuin A (ng/ml)					
Cut off	**Sensitivity**	**Specificity**	**PPV**	**NPV**	**Accuracy**
<=520.3 *	**100.0**	**100.0**	**100.0**	**100.0**	**100.0%**

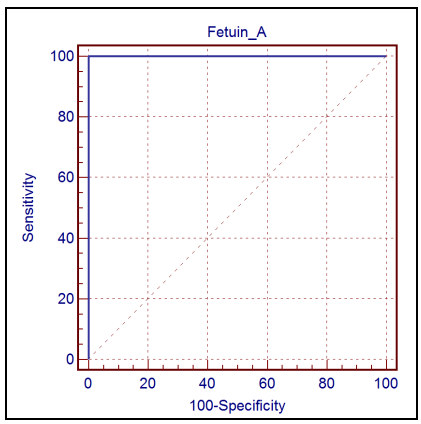

Figure (3): ROC curve between control and myocardial infarction group.

Regarding the diagnostic accuracy of serum fetuin A as a predictor and a diagnostic marker for myocardial infarction, we analyzed the receiver operator characteristic curve (ROC) **table (7), figure (3)** showed that cut off value was (520.3ng/ml**)**, serum fetuin A showed 100.0% sensitivity, 100.0% specificity with accuracy 100.0%, PPV (positive predictive value) is 100.0% and NPV (negative predictive value) is 100.0%.

DISCUSSION

Vascular calcification is found in the majority of advanced atherosclerotic lesions that contribute to the overall morbidity of atherosclerosis by decreasing the elasticity of the vessels results in reduced vascular compliance and impaired myocardial perfusion **(Afsar et al., 2012).**

Fetuin A is a liver-derived potent systemic inhibitor of calcification and a negative acute phase reactant. It prevents calcium and phosphate precipitation in the serum and protects from arterial media calcification by inhibiting vascular smooth muscle cell (VSMC) apoptosis. Fetuin A prevents basic calcium particle nucleation in the supersaturated extracellular environment **(Schafer et al., 2005).** It plays an important role in preventing calcium deposition and inhibition of unwanted (vascular) calcification. As fetuin A level decreases, calcium deposition increases which leads to narrowing of coronary artery result in CAD, aortic calcification and stenosis **(Afsar et al., 2012).**

In this study common risk factors, such as age, sex, family history of CAD, BMI, smoking status, hypertension and diabetes mellitus were similarly distributed in all studied groups to abolish their effect on fetuin A level.

The present study showed that there was a statistically significant difference in mean **fetuin A** level between patients affected by coronary artery disease (myocardial infarction and stable angina) in comparison to control group. The control group showed significant higher level.

This result agreed with **Amal et al. (2014)** who found that fetuin A level significantly decreases in patients with coronary artery diseases compared to the control group. **Cigdem et al. (2012) and Hans-Josef et al. (2015)** also reported that fetuin A level decreases in patients with acute coronary syndrome when compared to the control group.

The present study also showed that there was a statistically significant difference in mean **fetuin A** level between studied groups. The myocardial infarction group showed the most lower level of fetuin A.

This result agreed with **Oktay et al. (2010)** who reported that fetuin A levels significantly decrease in patients with myocardial infarction when compared to patients with stable angina and patients without coronary disease.

So, the more lower fetuin A level, the more was the bad prognosis. These results matched with the physiological role of fetuin A in protection against cardiovascular events by functioning to keep calcium and phosphorus solubilized in serum, thus preventing hydroxyapatite deposition in vessel walls **(Merx et al., 2005).**

Fetuin A inhibits self amplification of inflammatory response by improving the uptake of spermine which is essential for the inhibition of the synthesis of various pro-inflammatory cytokines from activated macrophages and monocytes. A low fetuin A concentration may increase the ongoing inflammatory process and also the overproduction of cardiotoxic cytokines such as tumor necrosis factor which exposes patients to a high risk of recurrence of acute coronary syndrome **(Lim et al., 2013).**

Ze-Lin et al., (2014) reported that fetuin A level decreases in patients with coronary artery disease compared to control group and hypothesized that decreased serum fetuin A concentrations may directly limit cardiac functions by effectively promoting cardiac calcification and thus influence coronary artery disease progression.

Receiver operator characteristic curve (ROC) for determining the diagnostic accuracy of serum fetuin A as a predictor and a diagnostic marker for CAD, stable angina and myocardial infarction, with high validity and over all accuracy.

Pascal et al. (2013) analyzed the receiver operator characteristic curve (ROC) curve for determining the validity of fetuin A for prediction of outcome in acute coronary syndrome and showed that the validity of fetuin A as a predictor of death in acute coronary syndrome.

So, there was a significant connection between lower serum levels of fetuin A and coronary artery disease. Thus, fetuin A might be clinically valuable for reflecting the progression of coronary artery disease.

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خلفية البحث : فتوين "أ" هوالبروتين ألفا "2" هيرمان سكا يمد بروتين سكري ويعمل كبروتين سالب التفاعل (مثبط للإلتهاب) ويصنع في خلايا الكبد، فتوين "أ" يمنع تريب الكالسيوم والفوسفات في جدار الأوعية الدموية عن طريق زيادة ذوبانهم في الدم حيث أن ترسيب أملاح فوسفات الكالسيوم (هيدروكسي اباتيت) في جدار الأوعية الدموية تسبب تصلب الشرايين وتلعب دورا كبيرا في تطور مرض الشريان التاجي. و وجود فتوين "أ" في الدم يمنع تكلس الأوعية الدموية وتصلبها عن طريق زيادة ذوبان الكالسيوم والفوسفات فى الدم.

الهدف من البحث : معرفة مستوى فتوين "أ" فى مصل مرضى الشريان التاجى من المصريين وعلاقته بتطور المرض.

المرضى و طرق البحث: شملت هذه الدراسة 60 شخصاً من المصريين منهم 20 شخصا متطوعا و20 شخصا مريض بالذبحة الصدرية المستقرة و 20 شخصا آخرين مصابين بإحتشاء عضلة القلب. وتم عمل الآتى لجميع المرضى و أيضا للمجموعة الضابطة: تم أخذ التاريخ المرضي، عمل فحص إكلينيكي كامل يشمل رسم قلب بالمجهود و الفحوصات المعملية التي شملت الفحوصات المعملية الروتينية مثل سكر صائم ،مستوى الكرياتينين فى الدم ،تحاليل دهون شاملة،انزيمات القلب (كرياتينين كينيز،بروتين التروبونين)،البروتين التفاعلى "سى"وكذلك قياس مستوى فتوين "أ" فى المصل بواسطة الإليزا.

النتائج :حدث نقص فى مستوى فتوين "أ" فى مصل دم الأفراد الذين يعانون من إحتشاء عضلة القلب مقارنة بمرضى الذبحة الصدرية المستقرة ومجموعة الأصحاء وكذلك نقص فتوين "أ" فى مرضى الذبحة الصدرية المستقرة عن مجموعة الأصحاء.

الاستنتاج : فتوين "أ" يمكن أن يستخدم لمتابعة حالة مرضى الشريان التاجي كما يمكن أن يستخدم لتقييم التطور المرضى للمصابين بأمراض الشريان التاجي.