

Medical Journal of Babylon Vol. 13- No. 4:750 - 754, 2016





Original Research Article

Correlation of Fetuin A level with ECG types of Acute Myocardial Infarction

Haydar Hashim Al-Shalah* Oday Al-Salihi Dina Ayed Mohammed College of Medicine, University of Babylon, Hilla, IRAQ

Accepted 8 January, 2017

Abstract

Acute myocardial infarction is one of the commonest disease with serious complications and increasing morbidity and mortality. Coronary atherosclerosis plays a crucial role in the underlying pathophysiology. Fetuin-A is a protein which is closely linked with increased risk of cardiovascular disease, and secreted mainly by the liver, produces subclinical inflammation and insulin resistance. This study was aimed to evaluate the association of serum fetuin A level with ECG types of acute myocardial infarction.

This is a case–control study included 88 subjects divided into two groups; 44 patients with acute myocardial infarction, 36 of them with STEMI and 7 with NSTEMI. The other 44 were apparently healthy individuals taken as a control. Serum fetuin-A levels were measured by using ELISA technique. There was significant differences in serum fetuin-A levels between patients and control, (p value ≤ 0.05). The mean differences between serum fetuin-A levels and both type of AMI (STEMI and NSTEMI), were also significant(p value ≤ 0.05) where higher values observed in STEMI. This study concluded that fetuin-A level was significantly correlated with type of AMI and higher values of fetuin-A were observed in STEMI.

Key Words: Fetuin-A, AMI, ELISA technique, STEMI, NSTEMI, ECG.

الخلاصة

احتشاء العضلة القلبية الحاد سبب المضعفات المرضيه والموت بشكل واسع في العالم. احتشاء العضلةالقلبية موت الخلايا القلبية التي تحدث بعد انتزالها عن الاوكسجين لفتره طويله. حيث ان تصلب الشرايين هو الاكثر شيوعا لامراض الشرايين التاجيه. ويعد الفتوين آ البروتين الذي له علاقه وثيقه بتطور امراض القلب والشرايين ويتم افرازه عن طريق الكبد ومن خلال التجارب التي تم آجراءها على الحيوانات لوحظ ان لها علاقه مع السكري النوع المقاوم للأنسولين والالتهابات الثانوية. وتهدف هذه الدراسة لتقييم مستوى الفتوين وعلاقته مع آنواع التخطيط العضلي للقلب للمرضى الذين يعانون من آحتشاء عضلة القلب الحاد.

حيث تضمنت هذه الدراسة ٨٨ عينه تحوي ٤٤ من المرضى الذين يعانون من آحتشاء عضلة القلب الحاد(٣٦ حالة هم STEMI و ٧ حالات هم NSTEMI) و ٤٤ من الأصحاءحيث تم قياس مستوى الفتوين في مصل الدم بتقنية الآليزا.وبمقارنة مجموعة الاصحاء بمجموعة المرضى لوحظ قلة مستوى الفتوين للمرضى حيث (P<0.05)،حيث لوحظ هذا في كلا النوعين STEMI و NSTEMI,ولكن نزول الفتوين في نوع NSTEMI يكون واضحا واعلى مما هو عليه في NSTEMI وهذا يدل على آن هنالك علاقة وثيقة بين نزول مستوى الفتوين وآنواع الآحتشاء العضلي الحاد وخصوصا في نوع STEMI.

^{*}E-mail:alshalah2010@yahoo.com

Introduction

he term acute myocardial infarction (AMI) should be reserved to the state of myocardial necrosis and theclinical features that associated with Fetuin A[1]. Annually, about three and four million people are estimated to have an acute ST-elevation MI (STEMI) and non-ST-elevation MI (NSTEMI) respectively. The disease is seen predominantly in developed countries, however it becomes increasingly more common in developing countries [2].

In ECG finding, MI is classified as STEMI and NSTEMI, existence of a ST segment elevation on ECG or Q-wave are associated with poor prognosis [3].

There is an evidence of high risk for recurrent ischemic conditions in patients with pre-existing AMI which suggests that these population may obtain some benefit from intensive secondary prophylaxis [4].

Fetuin-A (FA) inhibit tyrosine kinaseenzyme which might lead to insulin resistance in the target tissues. The reference interval of serum FAin healthy adult individuals ranges between(0.4-1) mg/mL, however at the end ofchildhood, the serum FA were higher than or similar to adult levels. On electrophoresis, FA dominating the alpha-2 region[5].

There is an evidence of association between FA and cardiovascular disease (CVD) resulting from insulin resistance syndrome. This association is also occurs with other markers of CVD such as high sensitive C-reactive protein (CRP). They have been found that FA is positively correlated with hCRP and higher value are associated with increased risk of MI [6].

Materials and Methods

Subjects: This is a case–control study performed between the first of December

2015 and the first of March 2016 included 88 subjects divided into two groups; 44 patients with AMI,36 of them with STEMI and 7 with NSTEMI. The other 44 were apparently healthy individuals taken as a control. Patients were diagnosed consultant physician at Marjan Medical City/Hilla. The patients' Meanage ± SD was 61.29±10.85 which matched with control group (Meanage±SD was 56.72±9.86), furthermore the socio-demographic status between two groups was matched also. The practical part of the study was achieved at the laboratories of Department of Pathology and Clinical Biochemistry/ Babylon College of Medicine.

Ethical considerations

Legal agreements from research related offices had been taken , in addition , verbal acceptance from all participants involved in this study was undertaken.

Sample collection

From each subject enrolled in the study. about 5 ml of blood was obtained by vein puncture. The aspirated blood was put in gel separating tube, centrifuged at 6000 X g for 10 minutes. The obtained serum was stored in eppendo rf and kept freezing until time of analysis.

Methods

Fetuin-A level in serum was measured by sandwich enzyme-linked immune-sorbent assay (ELISA) using a kit provided by Biorbyt / USA.

Statistical Analysis

The obtained data were analyzed by computer using SPSS program, version 19^{th} . Descriptive data were expressed as (mean \pm SD), while level of significance between variables was determined by Chi square (X^2) and (t test). The selected level of significant for P values was less than (0.05).

Results

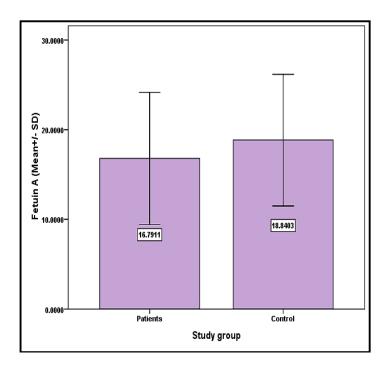


Figure 1: Comparison of patients and control by FA level. p value ≤ 0.05 is significant

The mean \pm SD was 16.9 \pm 3.6 ng/ml and 18.3 \pm 4.41 ng/ml respectively. There wasasignificant difference in the level of FA between patients and control group (p value<0.05).

Table-1shows that the mean of FA in patients with STEMI was higher than its mean in NSTEMI. The mean \pm SD was 17.23 \pm 2.92 and 13.97 \pm 5.89 respectively. There was a significant difference in the mean value between STEMI and NSTEMI (p value < 0.05).

Table 1: Mean difference of Fetuin A level by ECG type of Myocardial Infarction

MI	Groups	No	Mean	SD	P value
	STEMI	36	17.23	2.92	0.030*
	NSTEMI	7	13.97	5.89	

p value ≤ 0.05 is significant

Discussion

From pathophysiological point of view, AMI is commonly defined as a death of cardiomyocyte due to a prolonged ischemia resulting from an acute imbalance between oxygen demand supply resulting fromatherosclerotic process which is in turn regarded as the hallmark of this pathophysiology[7, 8]. FA has a positive

effect in prevention of systemic calcification and inflammatory process. In the present study, The level of FA was low in patients compared with control and this agreed with Merx et al who concluded the same observation. Decreased concentration of fetuin A will produce a continuous inflammatory process which in turn affect cardiac functions by causing cardiac

calcifications and fibrosis and hence lead to evolvement of CVD [9]. Furthermore, theanti-inflammatory property of FA in opposing nonspecific immune\response causesmacrophage deactivation, where low level of FA will produce large amounts of toxic substances such as tumor necrosis factor and consequently increase the risk of acute coronary syndrome recurrence [10]. AMI is classified based on ECG finding into

AMI is classified based on ECG finding into STEMI and NSTEMI[3]. The mechanism of ST segment elevation or depression, dependent upon pathological type of AMI whether transmural or sub-endocardial. ST segment elevation occurs in transmural AMI which involve the whole thickness of heart muscle while depression appears on ECG when small area of sub-endocardial wall is affected. In either case, the current flow from the depolarized ischemic area to normal regions result in the appearance of ST segment elevation depression[11,12]. In the current study most of patients involved were STEMI. The level of FA was higher in patients with STEMI than NSTEMI.

Human FA formed and secreted by liver, kidneys and many organ of human being. It exerts its effect throughout toll like receptors which are widely distributed in different tissues. It is thought that FA has proinflammatory and anti- inflammatory effects [13]. The higher values of FA in STEMI might be related to over expression in FA synthesis in STEMI which involve the whole thickness of myocardium or may be linked to wide spread inflammatory process in STEMI compared to NSTEMI.

Conclusions

Fetuin-A level was significantly correlated withtype of AMIand highervalues of FA were observed in STEMI

References

Bloomfield P.,Bradbury A, Grubb N.R.,NewbyD.E,chapter 18 In:Boon N.A., Colledge N. R.,Walker B.R. 22 nd Edition, Davidson's Principle and Practice of Medicine,

- UK: Churchill Livingstone Elsevier(2014):pp590.
- 2. WhiteH D, Chew DP. Acute myocardial infarction. Lancet 2008; 372(9638): 570–584.
- 3. Moe KT, Wong P. "Current trends in diagnostic biomarkers of acute coronary syndrome" .Ann. Acad. Med. Singap. 2010; 39 (3): 210–215.
- 4.Jernberg T, Hasvold P, Henriksson M, Hjelm H, Thuresson M, Janzon M. Cardiovascular risk in post-myocardial infarction patients: nationwide real world data demonstrate the importance of a long-term perspective. Eur Heart J 2015; 36: 1163–1170.
- 5. Fisher E, Stefan N, Saar K et al., "Association of AHSG gene polymorphisms with fetuin-A plasma levels and cardiovascular diseases in the EPIC-potsdam study." Circulation: Cardiovascular Genetics2009;2 (6): 607–613.
- 6. Tawfik PH , HafezSS , Mahmoud NH , El Sayed HM. Serum Fetuin A, HS-CRP and Homocysteine as Biochemical Markers of Cardiovascular Complications in Chronic Dialysis Patients .ActaMedica International 2015; 2 (1):57-64.
- 7. Myocardial infarction redefined a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. Eur Heart J 2000;21:1502–1513.
- 8.Van de Werf F, Bax J, Betriu A, et al. "Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology".Eur. Heart J 2008; 29 (23): 2909–2945
- 9.Merx MW, Schafer C, Westenfeld R, et al. Myocardial stiffness, cardiac remodeling, and diastolic dysfunction in calcification- prone Fetuin-A-deficient mice. J Am SocNephrol 2005; 16: 3357-3364.
- 10.Lim P., Moutereau S., Simon T., Gallet R., Probst V., Ferrieres J., et al. Usefulness of Fetuin-A and Protein C-Reactive Concentrations for Prediction of Outcome in Acute Coronary Syndromes (from the French Registry of Acute ST-Elevation Non-ST-Elevation Myocardial Infarction [FAST-MI]), Amer Cardiol, 2013;111(1): 31-37.

Al-Shalah et al.

MJB-2016

- 11. DeWood MA, Spores J, Notske R, *et al.*Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med 1980;303:897-902
- 12.Reznik AG. "[Morphology of acute myocardial infarction at prenecrotic stage]". Kardiologiia2010; 50 (1): 4–8.
- 13. Mukhopadhyay S, Mondal SA, Kumar M, Dutta D. "Pro-inflammatory and anti-inflammatory attributes of fetuin-A: a novel hepatokine modulating cardiovascular and glycemic outcomes in metabolic syndrome". Endocr Pract. 2014; 20 (12): 1345–1351.