DOI: https://doi.org/10.53350/pjmhs22168627

ORIGINAL ARTICLE

Interplay Between Adiponectin, Resistin, Lipoprotein (A) and Prognosis in Middle to old age Female Cases with ST / Non ST Elevation Myocardial Infarction

SHAZIA RASHID[1], JAVED ANVER QURESHI[2], RUKHSHAN KHURSHID[3], ISMAT TAHIRA[4], SOFIA SHOUKAT[5], UZMA FARYAL[6]
[1]*IMBB/CRIMM, University of Lahore*
[2]*Professor IMBB/CRIMM, University of Lahore*
[3]*Assistant Professor Biochemistry, Shalamar Medical and Dental College Lahore*
[4]*General Practitioner, Chaudhary Clinic Lahore.*
[5]*Assistant Professor Biochemistry, Ayub Medical College Abbottabad.*
[6]*HOD Biochemistry department, Women Medical and Dental College Abbottabad*
Corresponding author: Shazia Rashid, Email: shazia.rashid9@gmail.com, Cell: +92 3086990960

ABSTRACT

Acute myocardial infarction (AMI) refers to ST-elevated myocardial infarction and non ST elevation myocardial infarction, is the known presentation of coronary artery disease. Study was planned to explore the interplay of the adipokines, resistin / lipoprotein (a) and prognosis in middle to old age female patients with ST / Non ST Elevation Myocardial Infarction.

Material and Methods: A cross-sectional study was conducted on 150 middle to old age female patients with acute myocardial infarction (AMI). Consented patients were divided into 2 groups based on ST and Non ST elevation. Duration of study was six months from December 2015 to May 2016. Levels of adiponectin, lipoprotein (a) and resistin were measured. 50 healthy subjects matched for age and gender also participated in study.

Results: Mean age of patients with NSTEMI was 58.89 while with STEMI was 50.59 years. Decreased levels of serum adinopectin, resistin and lipoprotein A was observed in female patients with NSTEMI in comparison with these parameters of STEMI, but significantly high level was seen in context of resistin. Positive correlation of age with serum adiponectin and resistin and a negative correlation of age with serum lipoprotein (a) was in female patients with STEMI and NSTEMI.

Conclusion: Study found a direct interaction of adiponectin and resistin with strong prognosis of ST and weak prognosis of Non ST elevation of myocardial infarction; whereas lipoprotein (a) showed a strong indirect interaction with age in women with both STEMI and NSTEMI.

Keywords: Adiponectin, lipoprotein (a), resistin, STEMI and NSTEMI.

INTRODUCTION

Myocardial infarction is (MI) a main issue of cardiovascular disease. MIs are categorized into ST elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI)[1]. Globally, over and above 3.0 million individuals have STEMI and 4.0 million have NSTEMI each yr[2]. STEMIs happen twice as in male as female[3].The mortality rate decreased significantly over time among patients with non-ST-segment. In contrast, mortality rate did not change among cases with STEMI[4].

STEMI is defined as the combination of symptoms related to poor oxygenation of the heart with elevation of the ST segments on the electrocardiogram followed by an increase in proteins in the blood related to heart muscle's death[5]. It includes thrombolysis and percutaneous coronary intervention[6]. NSTEMI demarcated as a progress of necrosis of muscle of heart without the changes in ECG. It usually occurs by developing a partial or complete occlusion of a major coronary or minor coronary artery previously affected by atherosclerosis and can be verified by increase values of cardiac markers in circulation. NSTEMI is not as much severe in comparison to STEMI[4]. Main jeopardy factors are high values of serum cholesterol, diabetes, hypertension and smoking. Other factors are aging, family history, sedentary life style, high BMI, excess intake of carbohydrates, diets lack of fruits, vegetables, fruit & polyunsaturated fatty acids[7].

Adipose tissue release many cytokines like adiponectin, lipoprotein (a) or LP (a) and resistin that play an important role in progression of obesity-linked diseases including atherosclerotic vascular ailments[8]. Adiponectin (a cytokine) is secreted from adipocyte. Adiponectin possesses anti-atherosclerotic effects, arbitrated through pleiotropic inflections of different cells of vassals[9]. In STEMI cases, high values plasma adiponectin is related with CV mortality[10].

Lipoprotein (a) consists of a cholesterol-rich LDL particle apolipoprotein(a), attached via a disulfide bond[11]. Levels of Lp(a) lipoprotein are elevated especially increased in oxidative stress. High values of circulating lipoprotein(a) are jeopardy factor for multiple vascular ailments, like CHD, venous thrombosis and ischaemic stroke due to intimal deposition of cholesterol[12].

Human resistin is a novel adipokine mainly produced from monocytes and macrophages[13].It is therefore suggested as an inflammatory marker and it may be involved in promoting atherosclerosis via lipid accumulation in macrophages in patients with STEMI[14]. Investigation of novel biomarkers in cardiac disease may help to highlight their function in the pathogenesis of heart problem especially MI with ST and non ST segment elevation.

MATERIAL AND METHODS

Total 150 female patients aged from 40 to 70 years from Pakistan Institute of Cardiology (PIC) Lahore presenting with AMI were involved in the project study. Of the cases, 40% were identified with STEMI and 60% with NSTEMI. Diagnostic criteria for STEMI were founded by the European Society of Cardiology/ American College of Cardiology's redefinition of guidelines of MI[15]. The duration of study was Dec 2015 to May 2016. Subjects including patients were categorized into 2 groups; group one included 100 patients with STEMI and group two include 50 patients with NSTEMI. The patients with heart failure, valvular heart disease, hepatic or renal dysfunction, chronic inflammatory diseases, surgery, trauma, diabetics taking drugs like thiazolidinediones or insulin were excluded from the study. Insulin was taken as the inhibitor of serum resistin.

Levels of adiponectin, resistin and Lp(a) were estimated in blood of patients drawn within 24.0 hourrs after beginning of MI. Plasma adiponectin was estimated by the technique of ELISA (Human Adiponectin ELISA). Circulating resistin and Lp (a) were analyzed by ELISA (Ray Biotech. Inc).The patients in the study were enrolled after approved diagnosis of AMI and rule out of other patients with diabetes mellitus, renal disease or muscle dystrophy.

The protocol of study was permitted by Institution Ethical Committee, Department of Training, Research & Postgraduate, Punjab Institute of Cardiology, Lahore. Written informed consensus was obtained before enrollment from all the participants. A comprehensive questionnaire documenting information on demographic data, medical and family history of coronary artery disease was also completed for each subject.

Statistical Analysis: Data was entered and analyzed by SPSS 20.0. Age and other biochemical variables were accessible as a mean ± SD. Significant differences between variables of the cases with STEMI and NSTEMI were calculated by student 'test. Pearson correlation among between biochemical variables was carried out. Significant P values less than 0.05.

RESULTS

Table 1: Variation of adinopectin, lipoprotein & resistin in female cases with NSTEMI and STEMI. Values are expressed as mean ± SD

Subjects	Age (years)	Adinopectin (µg/ml)	Resistin (ng/ml)	Lipoprotein (a) (mg/dl)
NSTEMI (50)	58.89±12.19	10.47±6.46	12.06±0.92	29.36±3.45
STEMI (100)	50.59±7.62	13.90±1.11	26.12±1.38**	32.76±1.53

**P<0.001= Significant difference

Table 2: Correlation of age with serum adiponectin, resistin and lipoprotein (a) in female patients with STEMI

Variables	Correlation coefficient (r)	P value
Adiponectin	0.5	<0.05
Resistin	0.31	>0.05
Lipoprotein (a)	-0.51	<0.05

Table 3: Correlation of age with serum resistin, adiponectin, & lipoprotein (a) in cases with NSTEMI

Variables	Correlation coefficient (r)	P value
Adiponectin	0.08	>0.05
Resistin	0.47	<0.05
Lipoprotein (a)	-0.36	>0.05

Variation in the levels of adinopectin, lipoprotein a and resistin in cases with NSTEMI and STEMI is presented in Table 1. Mean age of patients with NSTEMI was 58.89 while with STEMI was 50.59 years. Decreased levels of serum adinopectin, resistin and lipoprotein A were observed in female patients with NSTEMI in comparison with these parameters of STEMI, however the circulating resistin was significantly associated with age (P <0.05). Positive correlation of age with serum adiponectin and circulating resistin was in female cases with STEMI & NSTEMI. However significant difference was only observed between age of female patients with STEMI and their circulating adiponectin. Also the other positive correlation significant of age with serum resistin was observed in female patients with NSTEMI. Negative correlation of age with serum lipoprotein (a) was observed in female patients with STEMI and NSTEMI. However level of serum lipoprotein a was significantly correlated with age (P<0.05) in female STEMI cases (Table 2 & 3).

DISCUSSION

Mean age of cases with NSTEMI was 58.89 years and with STEMI was 50.59 years. Our study is in accord with a study which described increased level of adiponectin in relationship with old age[16]. Our study observed increased level of adiponetin in STEMI cases in comparison with NSTEMI. A research study reported that higher values of adiponectin may be an early predictor of adiponectin resistance of adiponectin or due to primary inflammation [17]. Controversial results observed by another study demonstrated increased prevalence of cardio vascular problem due to higher serum adiponectin that may act as a marker for greater homeostatic dysregulation and related apoptosis[18]. Our study observed a strong direct association of circulating adiponectin with age in women with STEMI and weak correlation was observed in women with NSTEMI. A study suggested that adiponectin was associated positively with age, female sex and may have a role with its genetic variants in developing MI in different age groups[19]. We agreed with studies that observed significant correlation of adiponectin with middle age women with STEMI and NSTEMI and stated that higher adiponectin levels associated with heart failure-related wasting in middle age[20].

Level of lipoprotein (a) was non significantly elevated in STEMI cases in comparison with NSTEMI cases. Negative correlation of age with serum lipoprotein (a) was observed in female STEMI & NSTEMI. Some studies also found that Lp (a) is not directly related with age and gender [21]. We observed significantly raised values of resistin in STEMI cases in comparison with NSTEMI. A study also found an increase level of resistin and reported that it was significantly correlated with major unfavorable cardiac proceedings in cases of acute coronary syndrome [22]. We observed a positive correlation of age with circulating resistin in female patients with STEMI and NSTEMI. We agreed with study that observed significantly high level of serum resistin in middle age MI patients and reported that increase level of serum resistin during the acute phase of STEMI is a good predictor of size of myocardial infarction [23].

CONCLUSION

Study found a direct interaction of adiponectin and resistin with strong prognosis of ST and weak prognosis of Non ST elevation of myocardial infarction. Whereas lipoprotein (a), showed a strong indirect interaction with age in women with both STEMI and NSTEMI. Additional studies are required to find the interplay of adiponectin, resistin and lipoprotein (a) with pathophysiology of STEMI and NSTEMI on large number of patients that may help in the improvement of therapeutic tactic against MI with / without the ECG change.

Author's Contribution: SR, RK: Study design. JA, RK: Supervised. SR, IT, UF: Write-up. IT, UF, SS: Proof reading. IT, SS: Statistical analysis.

REFERENCES

1. Moe KT, Wong P.Current trends in diagnostic biomarkers of acute coronary syndrome. Ann. Acad. Med. Singap 2010; 39(3):210-5.
2. White HD, Chew DP, Chew F. Acute myocardial infarction. Lancet 2008; 372(9638):570-84.
3. O'Gara PT, Kushner FG, Ascheim DD, Casey DE, Chung MK, de Lemos JA et al. ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am CollCardiol2013; 61(4):78-140.
4. de Winter RJ, Tijssen JGP. Non–ST-Segment Elevation Myocardial Infarction Revascularization for Everyone? J Am CollCardiolIntv 2012;5(9):903-905.
5. Bates ER, Menees DS. Acute ST-elevation myocardial infarction. Current Opinion in Critical Care 2012; 18(5):417-23.
6. Mehta PK, Wei J, Wenger NK. Ischemic heart disease in women: A focus on risk factors. Trends in Cardiovascular Medicine 2014; 25(2):140-151.
7. LauDC, DhillonB, YanH, SzmitkoPE, VermaS. Adipokines: Molecular links between obesity and atherosclerosis. Am J Physiol Heart CircPhysiol2005;288(5):H2031-41.
8. Bernardes G, Oliveira D, França JID, Piegas LS. Serum Adiponectin and Cardiometabolic Risk in Patients with Acute Coronary Syndromes. Arq Bras Cardiol 2013; 101(5):399-409.
9. Ntaios G, Gatselis NK, Makaritsis K, et al. Adipokines as mediators of endothelial function and atherosclerosis. Atherosclerosis 2013; 227(2):216-221.
10. De Roeck L, Vandamme S, Everaert BR, Hoymans V, Haine S, Vandendriessche T et al. Adiponectin and ischemia-reperfusion injury in ST segment elevation myocardial infarction.Eur Heart J Acute Cardiovasc Care2016;5(1):71-6.
11. Dangas G, Mehran R, Harpel PC, et al. Lipoprotein(a) and inflammation in human coronary atheroma: association with the severity of clinical presentation. J Am CollCardiol1998; 32(7):2035-2042.
12. Yang RZ Huang Q, Xu A, McLenithan JC, Eisen JA, Shuldiner AR et al. Comparative studies of resistin expression and phylogenomics in human and mouse. BiochemBiophys Res Commun2003; 310 (3):927-935.
13. Jung HS, Park KH, Cho YM, Chung SS, Cho HJ, Cho SY, et al.Resistin is secreted from macrophages in atheromas and promotes atherosclerosis. Cardiovasc Res 2006;69(1):76-85.

14. Panduranga P, Al-Zakwani I, Sulaiman K, Al-Habib K, Al Swaidi J, Al-Motarreb A. Clinical Profile and Mortality of ST-Segment Elevation Myocardial Infarction Patients Receiving Thrombolytic Therapy in the Middle East. Heart Views 2012; 13(2):35-41.
15. Thygesen K, Alpert JS, White HD, Jaffe AS, Apple FS, Galvani M, et al. On behalf of the Joint ESC/ACCF/AHA/WHF. Task Force for the redefinition of myocardial infarction. Universal definition of myocardial infarction. Eur Heart J 2007; 28(20):2525-2538.
16. Cavusoglu E, Ruwende C, Chopra V, Yanamadala S, Eng C, Clark LT et al. Adiponectin is an independent predictor of all-cause mortality, cardiac mortality, and myocardial infarction in patients presenting with chest pain.Eur Heart J 2006;27(19):2300-9.
17. Krasnodębski P, Opolski G, Karnafel W. Plasma adiponectin levels in acute myocardial infarction and during the postinfarction recovery period in patients with type 2 diabetes mellitus. Kardiol Pol. 2011;69(9):924-30.
18. Braunwald E. Unstable angina: An etiologic approach to management. Circulation1998;98(21):2219
19. Ambroziak M, Kolanowska M, Bartoszewicz Z, Budaj B. Adiponectin gene variants and decreased adiponectin plasma levels are associated with the risk of myocardial infarction in young age. Gene 2018; 642: 498-504. doi.org/10.1016/j.gene.2017.11.064.
20. Springer J, Anker S, Doehner W. Adiponectin Resistance in Heart Failure and the Emerging Pattern of Metabolic Failure in Chronic Heart Failure.Circulation: Heart Failure. 2010;3:181–182 doi.org/10.1161/CIRCHEARTFAILURE.110.945063
21. Pare G, Caku A, McQueen M, Anand SS, Enas E, Clarke R, Boffa M. Lipoprotein(a) Levels and the Risk of Myocardial Infarction Among 7 Ethnic Groups. Circulation 2019; 139(12):1472-1482 https://doi.org/10.1161/CIRCULATIONAHA.118.A034311
22. Erer B, Sayer N, SinanGüvenç T and Eren M. Prognostic Value of Serum Resistin Levels in Patients with Acute Coronary Syndrome. Kardiologiapolska 2013; 72(2):247-250
23. Zhao X,Wang Y, Liu C, Zhou P, Sheng Z, Li Y et al. Association between variation of Troponin and Prognosis of Acute Myocardial Infarction before and after Primary percutaneous coronary intervention. J Inter Cardiol. Volume 2020, Article ID 4793178, 13 pages https://doi.org/10.1155/2020/4793178