

ORIGINAL ARTICLE

A NOVEL LINK BETWEEN ADIPOKINES AND LIPOPROTEIN (A) TO CONTEMPLATE THEIR DIAGNOSTIC ROLE IN PATIENTS WITH STEMI AND NSTEMI

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Background: Early detection of cardiac events allow better and cost-effective triage and well-timed management of these patients. Study was conducted to evaluate the prognostic significance of plasma adiponectin, resistin and lipoprotein (a) in a group of patients with ST segment elevation myocardial infarction and non-ST-segment elevation myocardial infarction. These parameters were also compared with other predictors like troponin T, troponin I and CK-MB. **Methods:** Present study was based on the 100 patients with AMI of whom 52 had a diagnosis of STEMI, and 48 had NSTEMI. Duration of study was January to June 2015. Patients having chest pain that was indicative of myocardial ischemia within first 12 hours after the onset of symptoms were included in the study. Adiponectin, Lp (a), resistin, troponin T and troponin I were estimated using ELISA method. Level of CK-MB was measured by Auto-analyser using standard kit of Merck. **Results:** Mean age of patients with STEMI was 52.32 years and with NSTEMI it was 48.17 years. Mean value of BMI of patients with STEMI was 28.33 and with NSTEMI was 25.22 Kg/m². Duration of chest pain in patients with STEMI was 8.64 and patients with NSTEMI it was 16.52 hours with highly significant difference ($p < 0.001$). History of smoking in patients with STEMI was more as compared to the patients with NSTEMI. Level of CPK, adiponectin and lipoprotein (a) was raised in patients with STEMI as compared to patients with NSTEMI but significant difference was only found in levels of CPK ($p < 0.001$) and in adiponectin ($p < 0.05$). Level of serum CK-MB, TnT, TnI and resistin was raised in patients with NSTEMI as compared to patients with STEMI but significant difference ($p < 0.001$) was only observed in serum troponin I concentration. **Conclusion:** It is concluded that the incidence of STEMI and NSTEMI is similar in our patients. However, the markers of STEMI are increased level of adiponectin and Lp(a) and the markers of NSTEMI are troponins especially troponin I, resistin and CKMB.

Keywords: Cardiac markers; STEMI; NSTEMI, Adipokines, Lipoprotein

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INTRODUCTION

Non ST segment elevation myocardial infarction (NSTEMI) incidence is observed in about two-thirds of patients and it is mainly due to necrosis of myocardium with release of biochemical markers Troponins T or I and creatine kinase-MB (CK-MB).^{1,2} In STEMI there is an elevation of creatine kinase MB fraction, troponin I or T above the cut off levels.³

Patient with chest pain initially seems well but is actually suffering from a disease process having an underlying severe problem. According to World Health Organization the diagnostic criteria of AMI is based on a typical chest pain evocative of myocardial ischemia, changes in ST-segment or new Q waves development and altered cardiac enzymes.⁴ However, if there is no chest pain or atypical pain it does not rule out STEMI or NSTEMI.⁵ Patients with chest pain for >20 minutes may indicate a serious issue.⁶

Obesity may affect STEMI diagnosis, cardiac catheterization, vascular access and also has

effect on therapies.⁷ Standard BMI for normal weight is 18.5–24.9 kg/m², for overweight is 25–29.9 kg/m² and for obese is BMI ≥ 30 kg/m².⁸

With increase in BMI, patients have extensive cardiovascular risk profile with onset of premature STEMI.⁹ BMI less than 18.5 kg/m² may be factor of increased mortality in all ages.¹⁰ Mortality rate was decreased for overweight and obese older patients after NSTEMI.¹¹ There is a contradiction in results obtained on the obesity paradox in patients with STEMI.³ To exclude AMI, measurement of cardiac enzymes used as biochemical markers is important with atypical chest pain or a non-diagnostic ECG.⁴ Cardiac troponins T and I are used to find out myocardial injury with symptoms associated with ischemia. It is proposed that these troponins are better markers as compared to CK-MB for the detection of myocardial necrosis. These are also important in finding the myocardial damage in a patient after many days of MI, after the onset of symptoms. Even a minimum rise in the levels of troponins shows an unfavourable prognosis and require proper therapy.¹²

The troponins are regulatory proteins present in skeletal and cardiac muscles and classified as troponin I (TnI), troponin T (TnT), and troponin C (TnC). They appear in the blood about 3–4 hours after myocardial infarction and are detectable for 10 days due to their long half-life. However, it is difficult to detect re-infarction with the help of these markers.¹³

cTnI is a marker of injury of cardiac muscle. It may be better in diagnosis of the myocardial infarction than CK-MB, myoglobin and isoenzyme of lactate dehydrogenase.¹⁴ CK-MB has shorter half-life and it may be useful for diagnosing reinfarction.¹⁵ Patients with increased troponin levels with negative CK-MB levels are classified as NSTEMI, even in the absence of diagnostic ECG changes.¹⁶

Increased levels of troponin were observed in 80% of patients with acute MI within 2–3 hours of onset as compared to 6–9 hours with CK-MB.¹⁷ However, it is found that cardiac markers are not related to the diagnosis of patients with ischemic chest pain and diagnostic ECGs with ST-segment elevation.^{18,19}

Resistin is a peptide hormone (cytokine) derived from adipose tissue. It increases the level of LDL and may be linked with increased risk of atherosclerosis.²⁰ It also takes part in the inflammatory response.²¹ Its concentrations are increased in patients with NSTEMI and STEMI and may play a role as an analytical marker.²²

Structurally lipoprotein (a) is similar to plasminogen and tissue plasminogen activator and leads to thrombogenesis. Lp (a) is cholesterol carrier and may contribute to atherosclerosis.²³ In addition, Lipoprotein (a) causes proliferation of smooth muscle cells by transporting oxidized phospholipids.²⁴ Lipoprotein (a) may be used as a strong predictor in screening for high blood lipid content²⁵, and can be used as the only marker for the presence of cerebrovascular disease²⁶ especially in unstable angina or NSTEMI.

Adiponectin is an independent risk factor for increased aortic stiffness in patients during the first few days after STEMI.²⁷ Baseline adiponectin concentrations were similar in patients suffering from ST-elevation myocardial infarction or NSTEMI and related with cardiac mortality and MI in patients referred for cardiac catheterization.²⁸

Both STEMI and NSTEMI continue to be a major public health problem all over the world particularly in developing countries this problem is increasing. There is a need to find out the earlier predictor of markers in both forms which may prevent fatality in society and may develop new strategies for the management of STEMI and NSTEMI patients. The study was conducted to

investigate the prognostic importance of plasma adiponectin, resistin and lipoprotein (a) in a group of patients with ST segment elevation myocardial infarction and non-ST-segment elevation myocardial infarction. Study also attempted to compare these parameters with other predictors like troponin T, troponin I and CK-MB.

MATERIAL AND METHODS

This cross-sectional study was based on 100 patients with AMI of whom 52 had a diagnosis of STEMI, and 48 had NSTEMI. Duration of study was January 2015 to June 2015. Patients with chest pain indicative of myocardial ischemia within 12 hours after the onset of symptoms presenting in Punjab Institute of Cardiology, Lahore, Pakistan were included in the study. Exclusion criteria were cardiac resuscitation, renal dysfunction, severe skeletal muscle damage or trauma, unstable angina or refusal to give informed consent. Ethical Review Committee of Department of Research, Training & Postgraduate Medical education Punjab Institute of Cardiology, Lahore, Pakistan approved the study protocol. Letter of consent was taken from each patient. Demographic characteristics of patients were noted by a thorough medical history and physical examination.

Adiponectin was measured by use of ELISA assay (Human Adiponectin ELISA). Lp (a) and resistin assay were analysed by ELISA (Ray Biotech. Inc). Troponin T and troponin I were estimated using ELISA method (Boehringer Mannheim, product 1289055). Level of CK-MB was measured by Auto analyser using standard kit of Merck.

Data was analysed by using SPSS 20.00. Variables were expressed as mean±SD. Comparison of variables in patients with STEMI and NSTEMI was carried out by using student 't' test. $p < 0.05$ was considered as a level of significance.

RESULTS

Demographic characteristics of patients with STEMI and NSTEMI are tabulated as table-1. Variation in the markers of disease in patients with STEMI and NSTEMI is tabulated (Table-2). It was observed that the level of CPK, adiponectin and lipoprotein (a) was elevated in patients with STEMI than patients with NSTEMI but significant difference was only observed in case of CPK ($p < 0.001$) and in case of adiponectin ($p < 0.05$). Level of serum CK-MB, TnT, TnI and resistin was increased in patients with NSTEMI as compared to patients with STEMI but significant difference ($p < 0.001$) was only found in serum levels of troponin I.

Table-1: Demographic characteristics of patients with STEMI and NSTEMI

Patients	Age (Yrs)	BMI (Kg/m ²)	Duration of chest pain (hrs)	H/O smoking
STEMI (52)	52.32±15.00	28.33±13.51	8.64±8.35	36 +ve 16 -ve
Non STEMI (48)	48.17±13.56	25.22±2.07	16.52±12.55**	28 +ve 20 -ve

Table-2: Variation in the markers of disease in patients with STEMI and NSTEMI

Markers	STEMI	NSTEMI
CPK (U/L)	595.77±547.58**	179.72±72.14
CK-MB (U/L)	130.25±67.38	197.90±193.56
Troponin t (pg/ml)	289.07±230.65	344.10±283.21
Troponin I (ng/ml)	0.54±0.47	7.71±4.09**
Adiponectin (µg/ml)	15.61±7.09*	8.59±5.87
Resistin	214.71±142.48	384.88±330.81
Lipoprotein (a) (mg/dl)	267.71±141.89	180±83.95

DISCUSSION

Mean age of patients with STEMI was 52.32 years. According to a study mean age of patients with STEMI is 63.3 years. However, study found that most of the patients were in the ages of 60, 70 and 80 years and the mortality rate was noted at the age of 80 years.²⁹

According to our study the mean age of patients with NSTEMI was 48.17 years. A study reported that NSTEMI in patients observed in age range of 30–54 years.³⁰ However, a study found that mean age of patients with NSTEMI was 70.3 years.³¹

Mean value of BMI of patients with STEMI was 28.33 and with NSTEMI was 25.22 Kg/m² with no significant difference. Data of another study shows that BMI greater than 25 kg/m² may be a risk factor of STEMI. It is suggested that decreasing BMI may have an avoidance for early incidence of STEMI.⁸ A study found a U-shaped curve between different BMI groups, with the reduced mortality in obese patients and the highest mortality in skinny patients.³ Another study found that age at time of myocardial infarction has an inverse relationship with BMI; the old age patients were usually underweight and normal-weight, whereas the youngest patients were obese. However obese patients show decreased of all-cause mortality after NSTEMI.¹¹ It is proposed that Patients with high BMI present with STEMI has less chance of coronary artery disease and there is improvement in systolic functioning of left ventricular and good quality of care. Apart from these compensatory mechanisms severe obesity is still related to high mortality rate.³² Patients experiencing acute events undergo catabolic changes, including inflammation and activation of neurohormonal systems, which

require additional energy reserves. Increased subcutaneous fat may provide resistance to the catabolic burden associated with acute cardiac events.³³

Duration of chest pain in patients with STEMI was 8.64 and patients with NSTEMI, it was 16.52 hours with highly significant difference ($p < 0.001$). It is reported that if patients are presently pain free, but had chest pain in the last 12 hours and ECG is abnormal or not available, the patient should be referred for emergency department. However, between patients with characteristic and uncharacteristic symptoms the incidence of STEMI and NSTEMI was similar⁵ (Zdzienicka 2007).

History of smoking in patients with STEMI was more as compared to the patients with NSTEMI. Data of a study revealed that the smoking index value is high in patients with STEMI.³⁴ It is suggested that stop smoking decrease the risk of AMI, and the usefulness of stopped smoking is linked with quantity smoked in both STEMI and NSTEMI.³⁵

We observed that the level of CPK, adiponectin and lipoprotein (a) was increased in patients with STEMI as compared to patients with NSTEMI but significant difference was only observed in case of CPK ($p < 0.001$) and in case of adiponectin ($p < 0.05$).

A study also found a significant difference between adiponectin levels in patients with STEMI and NSTEMI, although the pathophysiology is different in both forms. It is therefore thought that adiponectin may be directly related with atherosclerosis and thrombosis at the vascular wall.³⁶

It is reported that increased levels of plasma adiponectin in patients with STEMI is linked with cardiovascular mortality.³⁷ However, a study found non-significant difference in plasma adiponectin's levels between patients with STEMI and NSTEMI.³⁸ A study is in agreement with our study and found that Lipoprotein (a) or Lp (a) is increased in STEMI compared to NSTEMI patients but does not discriminate between non-cardiac chest pain and myocardial infarction.³⁹ However, a study observed that an increased level of Lp (a) is associated with NSTEMI.⁴⁰ A study reported that an increased level of lipoprotein(a), indicates 3–4 times increase in risk of MI.⁴¹ It is proposed that lipoprotein (a) enters and accumulates at sites of arterial injury.⁴² It may also take part in thrombus formation and decreased fibrinolysis through reserve plasminogen activation.⁴³ Lp(a) levels forecast secondary vascular problems in patients with STEMI and may give strategies of prevention in patients.⁴⁴

Levels of serum CK-MB, troponin T (TnT), troponin I (TnI) and resistin were raised in patients with NSTEMI than in patients with STEMI but significant difference ($p < 0.001$) was only found in case of serum troponin I.

A research also found an increased level of CK-MB, TnT and TnI in patients with NSTEMI as compared to patients with STEMI. However, a study observed that level of serum resistin was similar in patients with STEMI and NSTEMI.²⁰ Another study showed that the level of resistin was significantly increased in patients with NSTEMI.⁴⁵

Increased levels of serum TnI, TnT, and CK-MB may be the sign of necrosis of myocardial cells in MI which may be due to non-ischemic myocardial injury, usually observed in arrhythmia, heart failure, myocarditis, pulmonary embolism and surgical coronary procedures.⁴⁶

An increased level of troponin in patients with NSTEMI is related with approximately 4 times increased risk of cardiac mortality.⁴⁷ It is found that in patients with NSTEMI the initial rise of TnI is linked to death at six weeks, but CK-MB levels were not signs of unfavourable cardiac problems and did not possess any predictive significance.⁴⁸ Another study reported that increased level of troponin was an independent indicator of death in patients with chest pain and ST-segment elevation.⁴⁹

However, troponin estimations have some disadvantages. Levels of Troponin do not raise until 6 hours after the start of symptoms; therefore, it is necessary to repeat the test 8–12 hours after the appearance of symptoms. Second drawback is that the level of troponin continually increased for a period of 5–14 days after necrosis of myocardium, the benefit of these parameters in finding recurrent myocardial damage is imperfect.¹⁵

Though CK-MB is found in the myocardial muscles, it is also present in skeletal muscle and its false-elevations observed in different diseases including heavy exertion, trauma and myopathy. Therefore, its predictive value in patients with a NSTEMI is also limited.⁵⁰

CONCLUSION

It is concluded that the incidence of STEMI and NSTEMI is similar in our patients. However, the marker of STEMI are increased level of adiponectin and Lp (a) and the markers of NSTEMI are cardiac Troponins especially troponin I, resistin and CK.MB. Non-significant increase or decrease may be due to small number of patients included in this study. We didn't study the correlation of adiponectin level with Lp(a), a good indicator of cardiovascular mortality because of limited data. Some studies found that estimation of

high molecular weight (HMW) adiponectin may be a useful marker of STEMI but we determined only total form of adiponectin.

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AUTHOR'S CONTRIBUTION

SR, RK: Study design. AM: Supervised. SR, RK, UF: Write-up and proof reading. RK and SQ: Statistical analysis.

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