Medical and Research Publications
International Open Access

Case Report **Journal of MAR Cardiology (Volume 4 Issue 5)**

Relation between Obesity and Myocardial Infarction

Shady Mohamed Akl El-Sayed [1]*, Azza Zakaria El-Eraky [2], Hanan Mohamed Kamal [2], Hesham Hegazy Ibrahim [2]

1. Cardiology consultant, Cardiovascular Medicine department, Ismailia General Hospital, Egypt.

2. Professor of Cardiology, Cardiovascular Medicine Department, Suez Canal University, Egypt.

Corresponding Author: Shady Mohamed Akl El-Sayed, Cardiology consultant, Cardiovascular Medicine department, Ismailia General Hospital, Egypt.

Received Date: August 18, 2022

Published Date: September 01, 2022

Abstract

Background: *Obesity is an excessive accumulation of fat that can impair health. Alterations in cardiovascular function have been observed in obese patients.*

Aim: *The aim of this study was to detect the potential effect of abdominal obesity on severity of myocardial infarction in patients with ST elevation myocardial infarction (STEMI).*

Patients and Methods: *This study was a cross sectional study that included 114 patients with acute STEMI who attended to Coronary Care Unit (CCU) at Mansoura International Hospital. Patients included in the study were divided into two groups: Group 1: patients who meet obesity criteria according to NCEP (n = 57) and Group 2: non obese patients (n = 57). The collected data included the medical history, clinical presentation, results of laboratory investigations, associated factors (DM, HTN, and Dyslipidemia), past history and family history of acute coronary syndrome. Results: Cardiac biomarkers were significantly higher in obese patients compared to non-obese patients. Conclusion: Presence of abdominal obesity is associated with greater myocardial necrosis after an acute coronary event.*

Keywords: *Obesity, Acute coronary syndrome, Cardiac biomarkers.*

Introduction

Abdominal obesity is the excessive accumulation of abdominal fat (visceral fat) resulting in an increase in waist size, over the past three decades, the worldwide prevalence of obesity has increased to alarming levels. In the USA, an estimated one-third of adults are obese. (1) The incidence rate of overweight has increased 2- fold and obesity more than 3-fold over the past 50 years. (2) In 2006, WHO estimates that the incidence of obesity has tripled over the past 20 years in developing countries, and projections predict that there will be 2.3 billion overweight (25–29.9 kg/m2) and 700 million obese (≥30.0 kg/m2) individuals worldwide in 2015. (3)

Abdominal obesity is one of the five clinical conditions (together with hypertriglyceridemia, higher cholesterol, lower high density lipoprotein)HDL(, fasting hyperglycemia and hypertension) that define

the existence of metabolic syndrome (MetS) according to the criteria established the National Cholesterol Education Program Adult Treatment Panel ((NCEP ATP III). (4)

Obesity and metabolic syndrome are major epidemics of the 21st century worldwide. (5,6) The metabolic syndrome (MetS) is characterized by a group of risk factors clustered in one individual, known to promote or increase the risk for development of diabetes mellitus and cardiovascular disease (CVD). (7, 8, 9)

Studies in patients with established vascular disease (coronary, cerebral and peripheral) demonstrate that the presence of metabolic syndrome correlates with the degree of underlying atherosclerosis. (10) This indicates that some of the clinical conditions that make up the metabolic syndrome-called not only increase the risk of cardiovascular disease, but once an event occurs, its presence is associated with greater severity and worse prognosis . (11)

Coronary artery diseases (CAD) are the most common cause of mortality in the world, myocardial infarction (MI) is the most common subtype of CAD, in addition, the prevalence of (MI) is increasing in the developing countries, In recent years, metabolic syndrome (MetS) is introduced as one of the major risk factors for CAD. (12)

In individuals with MetS, the risk of death from CAD is about two fold higher than normal and their risk of MI and stroke is 3 times than the normal population. (13) It has been reported that patients with symptomatic vascular disease and MetS have higher probabilities of suffering from extensive vascular damage. (14, 15)

Patients and methods

Study design:

This study was a cross-sectional study.

Study site

The study was conducted on patients with acute myocardial infarction (STEMI) in Coronary Care Unit (CCU) at Mansoura International Hospital.

Patient selection:

114 Patients with STEMI were randomly included in the study.

Included patients were divided into two groups:

- Group 1: patients who meet obesity criteria according to NCEP - ATPIII **(**BMI ≥ 30 kg/m2 and/or WC ≥ 88 cm in women or ≥ 102 cm in men**) (**n = 57**).**
- Group 2: non obese patients **(**n = 57**).**

Inclusion criteria:

- Age <18 years.
- Males and Females.
- Patients with STEMI diagnosed according to European Society of Cardiology **(**ESC**)** definition. **(**16**)**

Exclusion criteria:

- Patients with unstable angina or non ST elevation myocardial infarction **(**NSTEMI**).**
- Patients on hemodialysis.
- Patients with malignant diseases.
- Patients with old myocardial infarction **(**MI**).**

All patients included in the study were subjected to the following:

A detailed history was obtained from each patient with special emphasis on the following:

a. Age and sex.
b. History of chest pain: the most common symptom of acute myocardial infarction. **(**16**)**
c. Associated factors: Diabetes Mellitus, Hypertension and Dyslipidemia.
d. Family history of ischemic heart diseases **(**IHD**).**
e. Past history of acute coronary syndrome **(**ACS**).**

Clinical Examination

Anthropometric data: Body mass index **(**BMI**)** was calculated as the weight in kilograms divided by height in square meters, waist circumference **(**WC**)** was assessed using a tape measure over the top of the iliac crests with the patient standing naked and at the end of a normal expiration. Obesity was defined as BMI ≥ 30 kg/m2 and/or WC ≥ 88 cm in women or ≥ 102 cm in men. **(**4**)** Blood pressure and detailed physical examination to exclude heart and lung disease.

ECG: By using Keuz **(**ECG108**)** - 220 v ~ 240 y - 50 VA - 60 HZ Standard 12- Iead surface ECG. All ECGs were standardized to normal speed **(**25 mm/s**)** and sensitivity so that 1mv input produces 10 mm deflection. The American College of Cardiology, the American Heart Association **(**ACC/AHA**)** and the ESC concur that STEMI exists when the ECG of the patient presenting with acute chest pain shows:

(16**)** In the absence of left ventricular hypertrophy **(**LVH**)** and left bundle branch block **(**LBBB**)**: New ST elevation at the J point in 2 contiguous leads with ≥ 0.2 mV in men or ≥ 0.15mVinwomen in leads V2-V3 and/or ≥ 0.1 mV in other leads.

In the presence of LBBB or ST depression: New LBBB and symptoms suggestive of ACS. ST depression in leads V1–V3 indicates inferobasal myocardial ischemia **(**especially when the terminal T-wave is positive**)**. In suspected posterior **(**circumflex artery- related**)** or right ventricle-related infarction:

- ST elevation in V7 **(**at the left posterior axillary line**)**, V8 **(**at the left mid scapular line**)** and V9 **(**at the left paraspinal border**)**, using a cut-point > 0.05 mV.
- ST elevation in right precordial leads **(**V3R and V4R**)**, using a cut- off point > 0.05 mV and > 0.1 mV in men < 30 years. **(**16**)**

Laboratory data: Cardiac biomarkers: Myocardial infarction was diagnosed when levels of CK-MB above 25 U/L and Troponin T above 0.1 ng/ml **(**troponin +ve**)**. **(**16**)** CK-MB concentration was measured every 8 hours and Troponin T concentration was measured every 12 hours from admission to determine the maximum concentration would be reached in order to estimate the severity of myocardial infarction.

Results

The study included one hundred and fourteen patients who presented to Mansoura General Hospital with acute STEMI. Patients were divided into two groups: group 1 were obese and group 2 non obese patients according to NCEP criteria. The mean age of the study population was 57.13±9.61 **(**28 – 80 years**)**. Only 3 patients were between 18-39 years, 61 patients were between 40-59 years and 50 patients were above or equal to 60 years. 80 patients of the study population were males **(**70.18%**)** and 34 patients were females **(**29.82%**)**. **(**Table 1**)**

The mean of the BMI was higher in group 1 as expected than in group 2**(**30.77±0.52 versus 24.97±1.29**)**. Male patients in group 1 had significant higher WC than males in group 2**(**106.59± 2.48 versus 92.61±5.08**)**. Female patients in group 1 had significant higher WC than females in group 2**(**98.5±4.89 versus 85.86±1.41**)**. **(**Table 2**)** 62.28% of the study population was smokers **(**all of them were males**)**. 50% of the study population was hypertensive, 21.05% were diabetic and 24.56% were dislipidemic.14.04% of the study population has positive family history of CAD. HTN and DM were significantly high in group 1. **(**Table 3**)**

The mean value of maximum concentration of cardiac biomarkers was assessed among the study population. Cardiac biomarkers were significantly higher in obese group rather than non-obese group regardless they had normal or overweight BMI. **(**Table 4, Figure 1, 2**)**

According to BMI classification, there was a significant increase in cardiac biomarkers in obese group versus normal or overweight patients. **(**Table 5**)**

Cardiac biomarkers were significantly increased in males with WC ≥102cm versus Males with WC < 102. **(**Table 6, Figure 3, 4**)**

Cardiac biomarkers were significantly increased in female patients with WC ≥ 88cm versus female patients with WC < 88cm. **(**Table 7, Figure 5, 6**)**

The variables that showed statistically significant correlation with the concentrations of cardiac enzymes were BMI, WC, TGs, LDL-c and HDL-c **(**figures7-12**)**. The variable with the largest association was Male WC **(**r = 0.662, p < 0.001**)** for troponin - T **(**figure 4**)** and Female WC **(**r = 0.496, p < 0.001**)** for CK- MB **(**figure 5**)**. **(**Table 8**)**

In patients with HTN and DM, there were a significant relation between both and elevation of troponin T level. **(**Table 9**)**

A multiple linear regression analysis in order to obtain a statistical model predictor of myocardial enzyme elevation was performed. The results of the regression analysis showed that:

(a) The variables that significantly predicted CK-MB concentration among:

1. Male obese study group **(**group 1**)** was WC. **(**Table 10**)**
2. Female obese study group were **(**group 1**)** BMI and WC. **(**Table 11**)**
3. Male non obese study group **(**group 2**)** was BMI. **(**Table 12**)**

(b) The variables that significantly predicted Troponin T concentration among:

1. Non obese study group **(**group 2**)** was BMI. **(**Table 13**)**
2. Male obese study group**(**group 1**)** were WC and TGs. **(**Table14**)**
3. Female obese study group **(**group 1**)** was WC. **(**Table 15**)**
4. Male non obese study group **(**group 2**)** was BMI. **(**Table 16**)**

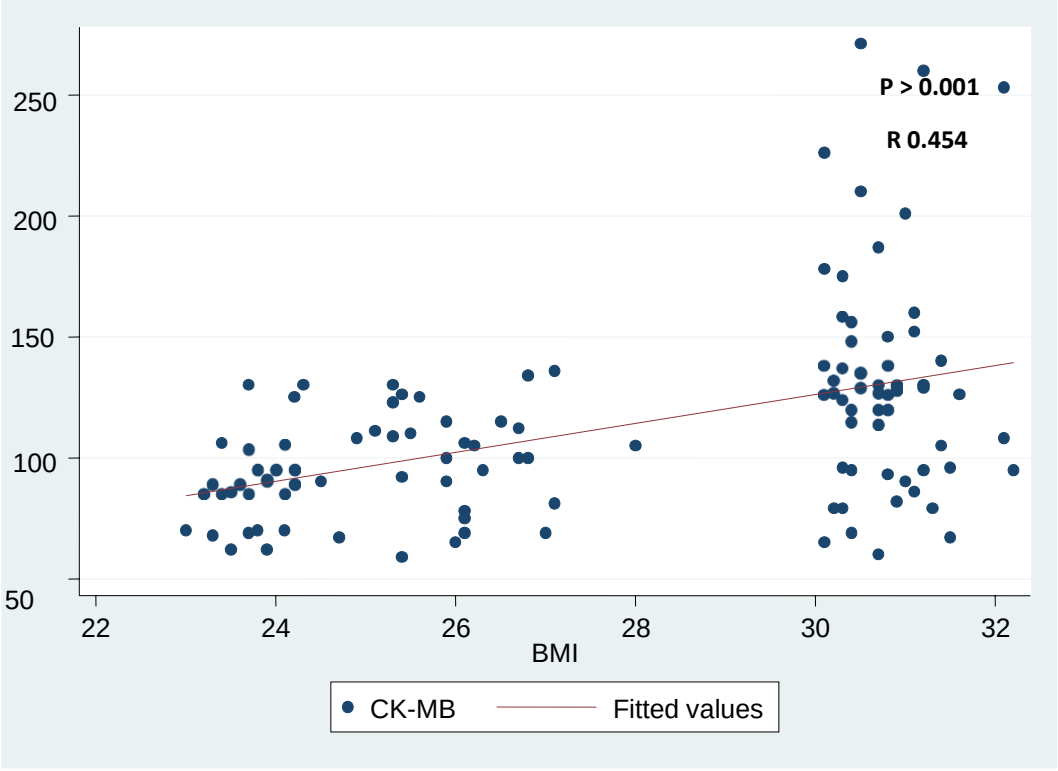

Figure 1: Correlation between CK-MB and BMI.

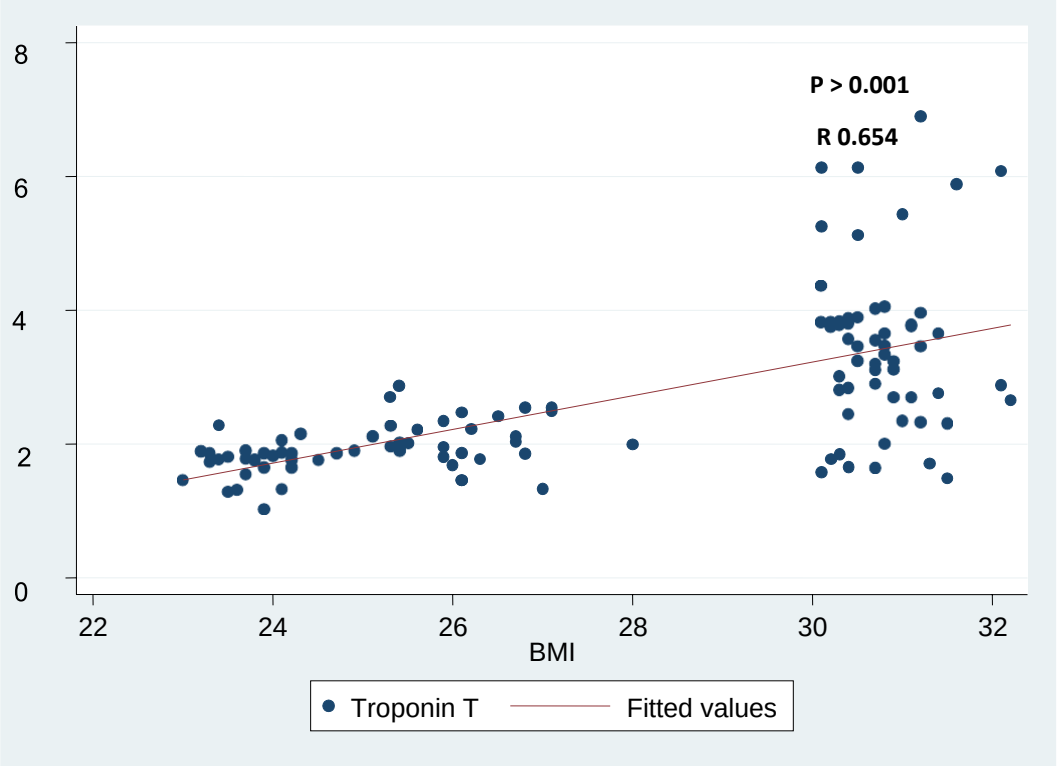

Figure 2: Correlation between Troponin T and BMI.

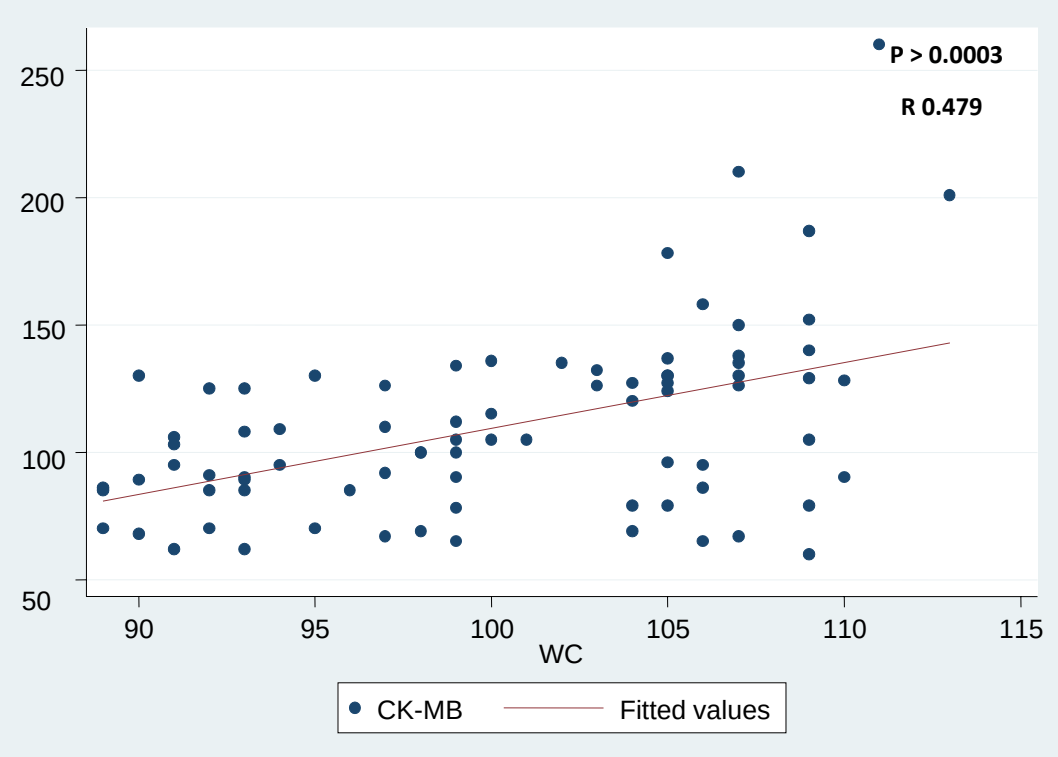

Figure 3: Correlation between CK-MB and male WC.

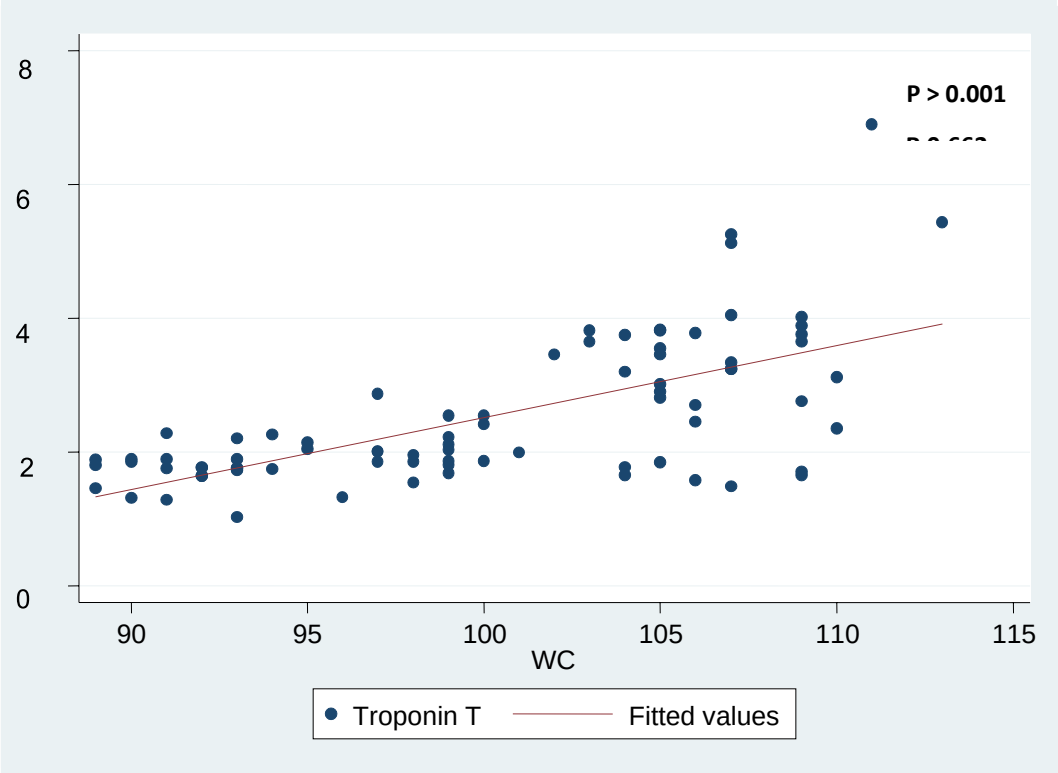

Figure 4: Correlation between Troponin T and male WC

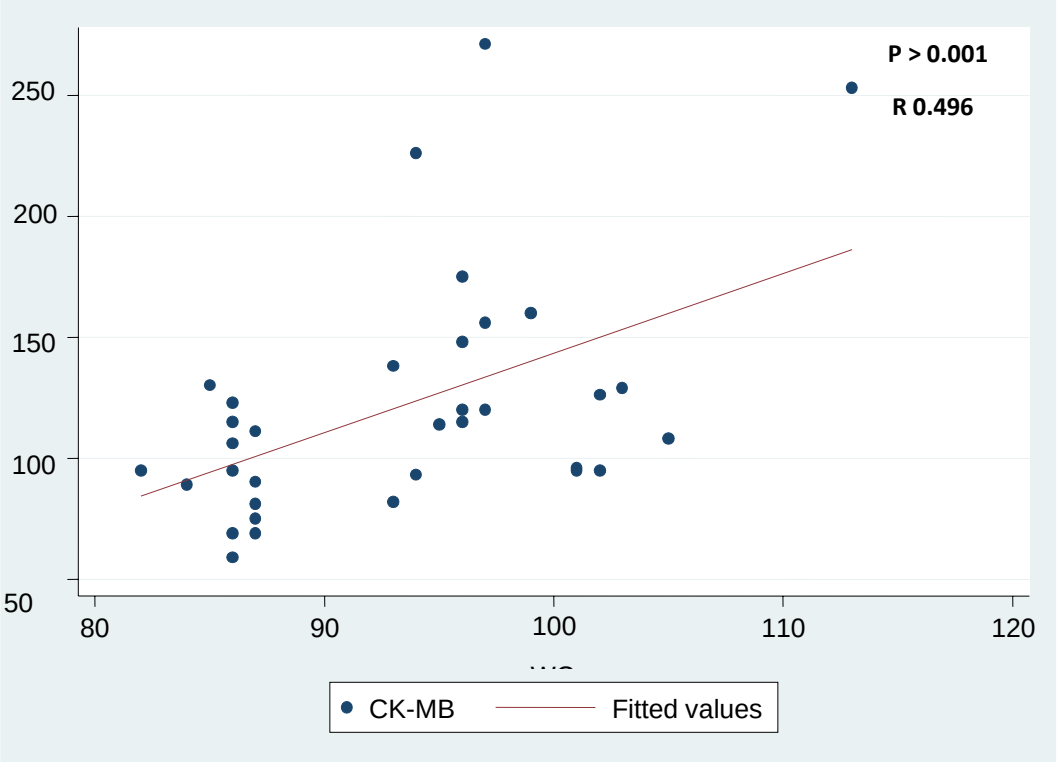

Figure 5: Correlation between CK-MB and female WC.

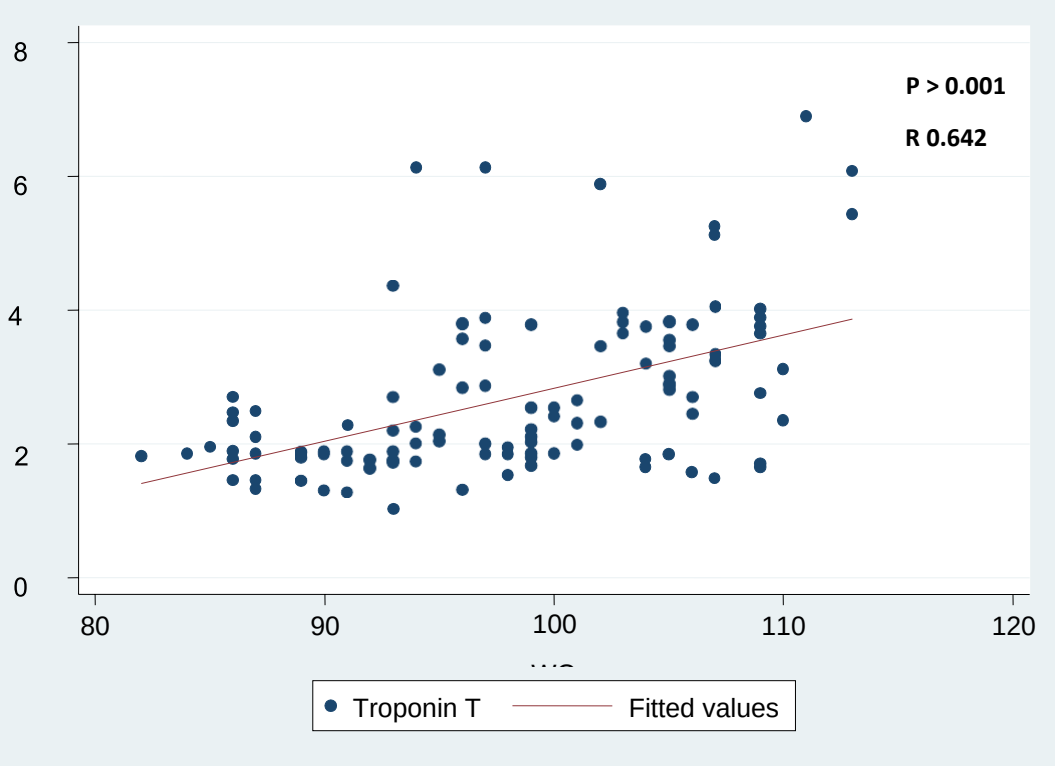

Figure 6: Correlation between Troponin T and female WC.

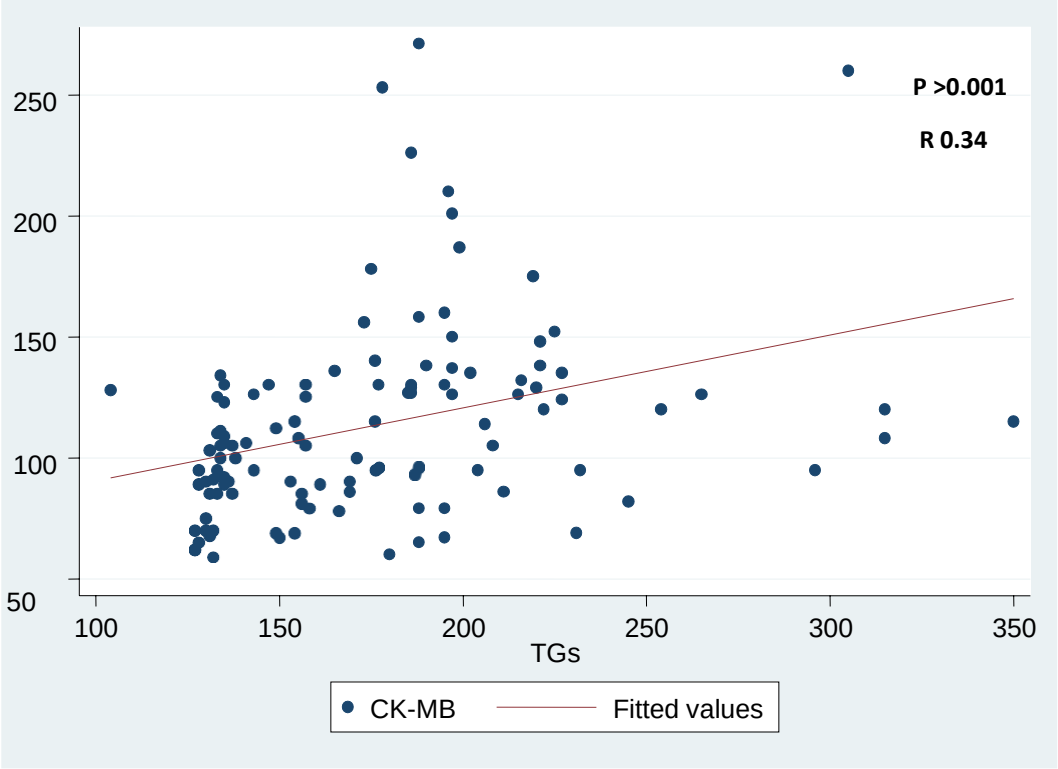

Figure 7: Correlation between CK-MB and TGs.

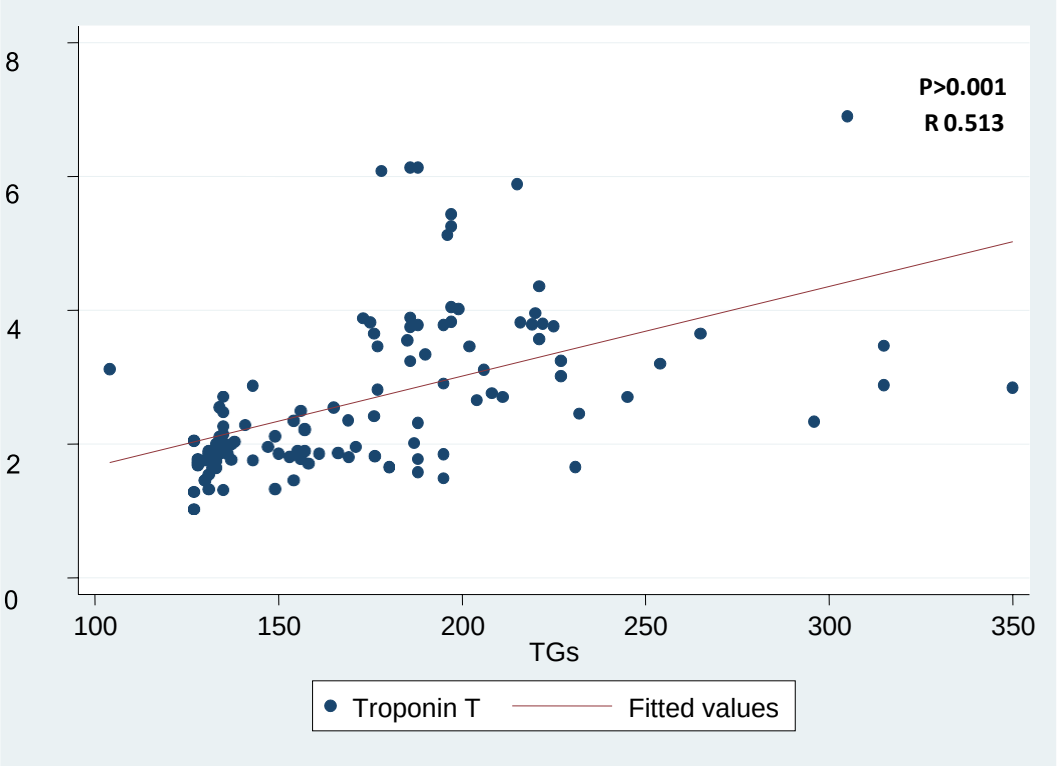

Figure 8: Correlation between Troponin T and TGs.

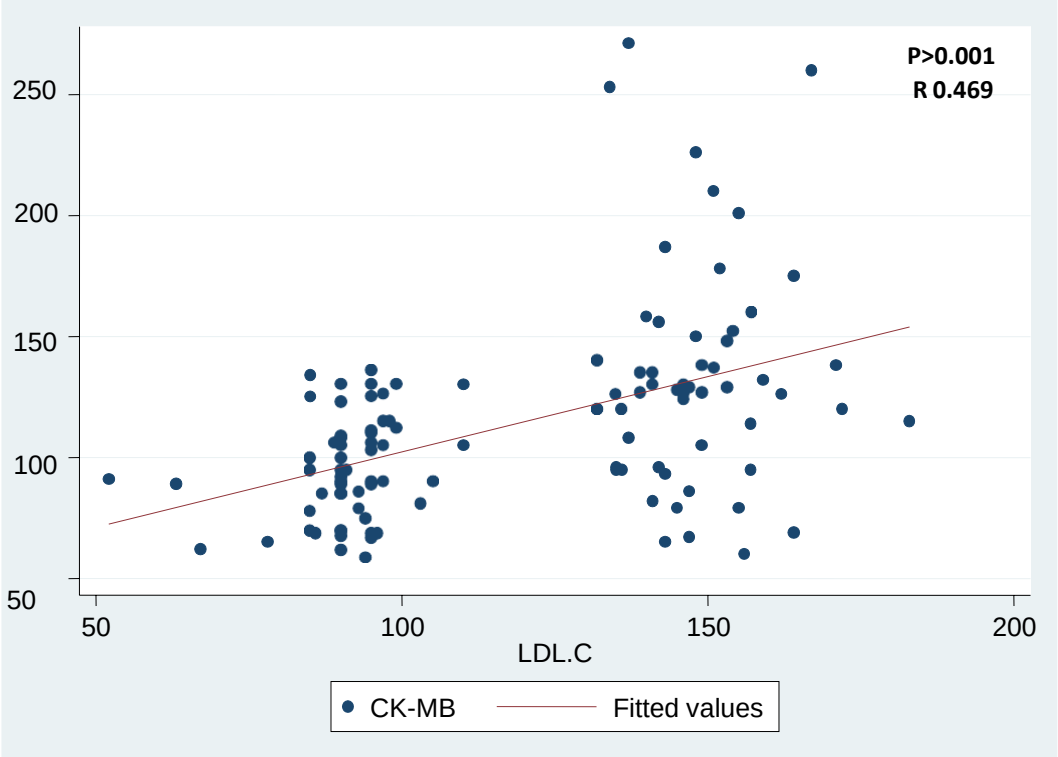

Figure 9: Correlation between CK-MB and LDL-C.

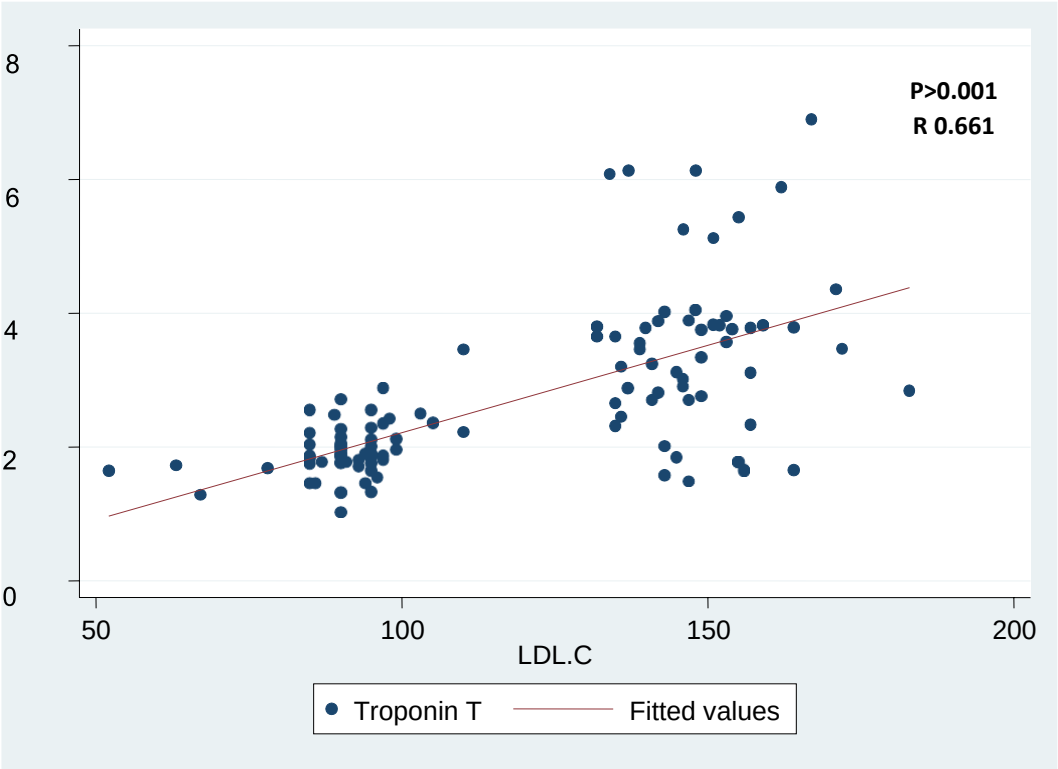

Figure 10: Correlation between Troponin T and LDL-C.

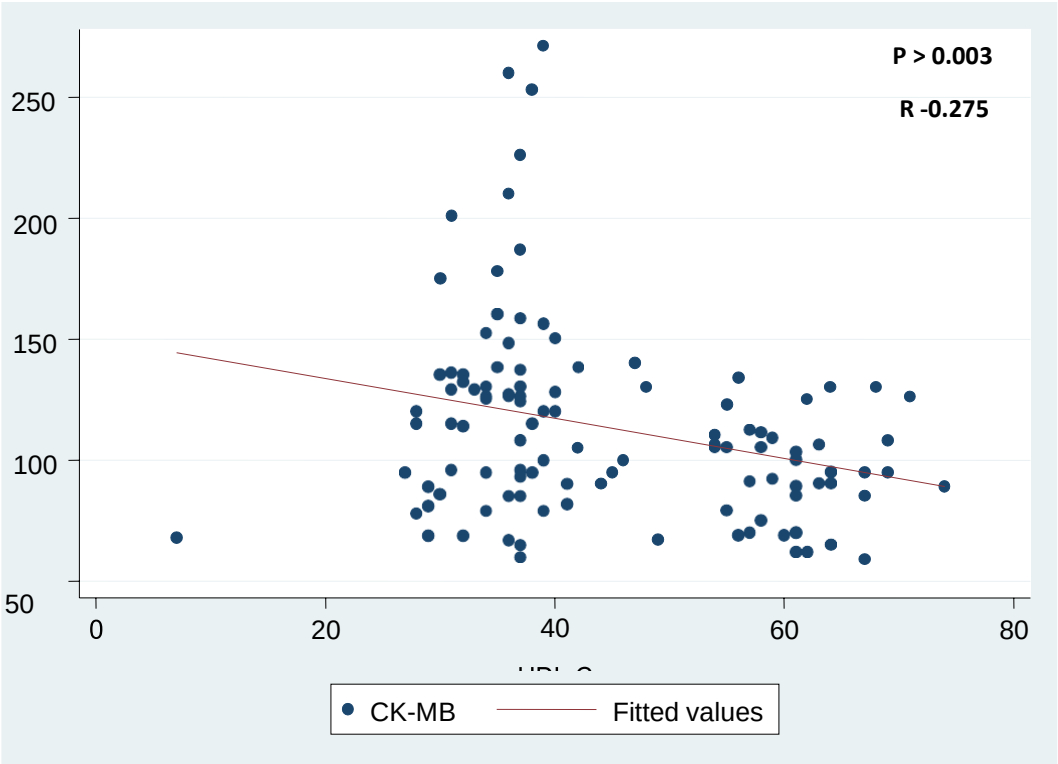

Figure 11: Correlation between CK-MB and HDL-C.

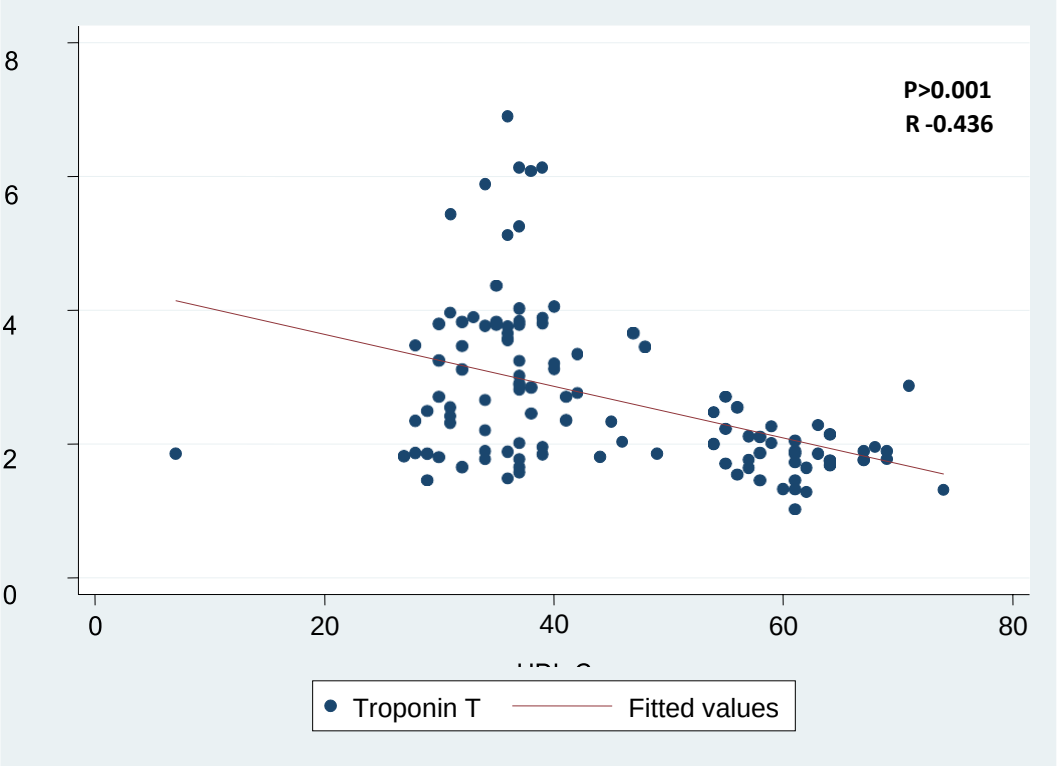

Figure 12: Correlation between Troponin T and HDL-C

Quantitative characteristics		Group 1 (obese)	Group 2 (non obese)	P value
		Mean±SD	Mean±SD	
Age		57.74±7.59	56.53±11.32	0.559
Qualitative characteristics		N (%)	N (%)	
Age group	18-39	0 (0%)	3 (5.26%)	0.189
	40-59	30 (52.63%)	31 (54.39%)	
	>=60	27 (47.37)	23 (40.35%)	
Qualitative		N (%)	N (%)	
characteristics				
Sex	Male	37 (64.91%)	43 (75.44%)	0.219
	Female	20 (35.09%)	14 (24.56%)	

Table 1: Demographic data of the patients (n=114).

Quantitative characteristics		Group 1 (obese)	Group 2 (non-obese)	P value
		Mean±SD	Mean±SD	
BMI (kg/m^2)		30.77±0.52	24.97±1.29	0.001*
WC (cm)	Overall	103.75±5.22	92.61±5.08	0.001*
	Male	106.59± 2.48	94.81±3.7	0.001*
	Female	98.5±4.89	85.86 ±1.41	0.001*

* P value is significant.

Table 2: Comparison of baseline characteristics among study groups (n = 114).

Qualitative characteristics	Group 1 (obese)	Group 2 (non obese)	P value
	N (%)	N (%)	
HTN	34 (59.65%)	23 (40.35%)	0.039*
DM	19 (33.33%)	5 (8.77%)	0.001*
Dyslipidemia	18 (31.58%)	10 (18.47%)	0.082
Family history ofCAD	6 (10.53%)	10 (17.54%)	0.281
Smoking	33 (57.89%)	38 (66.67%)	0.334

* P value is significant

Table 3: Frequency distribution according to risk factors of studied subjects (n=114).

Cardiac biomarkers	Group 1 (obese) Mean±SD	Group 2 (non obese) Mean±SD	P value
CK-MB (U/L)	131.58±47	95.25±20.87	0.001*
Troponin T (ng/ml)	3.47±1.24	1.91±0.37	0.001*

* P value is significant

Table 4: Cardiac biomarkers in study groups.

	BMI groups (kg/m^2)	CK-MB Mean±SD	Troponin T Mean±SD
Group 2 n = 29	Normal (< 25)	89.45±18.72	1.73±0.264
Group 2 n = 28	Overweight (25-29.9)	101.25±21.61	2.08±0.379
Group 1 n = 57	Obese (≥ 30)	131.58±47	3.466±1.238
P value		0.001*†	0.001*†‡

* P value is significant.

†P value is significant comparing obese vs. Normal and Obese vs.

Overweight groups.

‡P value is significant comparing Overweight vs. Normal (P 0.036).

Table 5: Comparison of BMI groups according to cardiac biomarkers

WC male groups	CK-MB Mean±SD	Troponin T Mean±SD
< 102cm (n = 43)	95.86±20.86	1.890±0.35
≥ 102cm (n = 37)	126.49±43.05	3.3±1.17
P value	0.0003*	0.001*

* P value is significant.

Table 6: Comparison of male WC groups according to cardiac biomarkers

WC female groups	CK-MB Mean±SD	Troponin T Mean±SD
< 88cm (n = 14)	93.36±21.58	1.96±0 .42
≥ 88cm (n = 20)	141±53.44	3.78±1.32
P value	0.001*	0.001*

* P value is significant.

Table 7: Comparison of female WC groups according to cardiac biomarkers:

		CK-MB		Troponin T	
		R(Pearson)	P value	R(Pearson)	P value
BMI		0.454	0.001*	0.654	0.001*
WC	Male	0.479	0.0003*	**0.662**	**0.001***
	Female	**0.496**	**0.001***	0.642	0.001*
TGs		0.34	0.001*	0.513	0.001*
LDL-C		0.469	0.001*	0.661	0.001*
HDL-C		-0.275	0.003*	-0.436	0.001*
SBP		0.078	0.41	-0.016	0.88
DBP		0.039	0.68	-0.034	0.717
FBS		0.075	0.43	0.192	0.41

*P value is significant

Table 8: Pearson correlation between cardiac biomarkers and patients quantitative variables

		CK-MB		Troponin T	
		Mean±SD	P value	Mean±SD	P value
HTN	+ve	117.88±37.13	0.057	2.86±1.06	0.004*
	-ve	108.95±43.56		2.51±1.31	
DM	+ve	123..13±43.94	0.19	3.21±1.42	0.016*
	-ve	110.82±39.44		2.54±1.1	

*P Value is significant

Table 9: Relation between cardiac biomarkers and HTN and DM

CK-MB	Coeff.	Std. Err.	P value
BMI	-15.56437	22.3126	0.491
WC	7.776667	3.395243	**0.029***
TGs	0.4364157	0.2310109	0.068
LDL-C	0.557326	0.6706237	0.412
HDL-C	0.6282138	2.030619	0.759
FBS	-415.3951	555.0008	0.46

* P value is significant.

Table 10: Multiple linear regression model for prediction of CK-MB levels among male obese study subjects.

CK-MB	Coeff.	Std. Err.	P value
BMI	-85.62302	27.76267	**0.008***
WC	11.55444	3.356172	**0.004***
TGs	-0.3642762	0.2476806	0.163
LDL-C	-0.239007	1.007914	0.816
HDL-C	0.1650978	2.645368	0.951
FBS	1764.923	744.7836	0.33

* P value is significant.

Table 11: Multiple linear regression model for prediction of CK-MB levels among female obese study subjects.

CK-MB	Coeff.	Std. Err.	P value
BMI	10.79238	4.028748	0.011*
WC	-2.337887	1.391145	0.101
TGs	0.3940791	0.2806695	0.169
LDL-C	0.3845149	0.3148106	0.23
HDL-C	0.3296034	0.2397501	0.177
FBS	-57.43885	78.37187	0.468

* P value is significant

Table 12: Multiple linear regression model for prediction of CK-MB levels among male non-obese study subjects

Troponin T	Coeff.	Std. Err.	P value
BMI	0.088563	0.038611	**0.026***
WC	0.004989	0.00927	0.593
TGs	0.0021768	0.0045106	0.631
LDL-C	0.008237	0.0054105	0.134
HDL-C	-0.0021854	0.0039406	0.582
FBS	-1.706155	1.187218	0.157

* P value is significant

Table 13: Multiple linear regression model for prediction of Troponin T levels among non-obese study subjects

Troponin T	Coeff.	Std. Err.	P value
BMI	-0.6195324	0.5984421	0.309
WC	0.2219044	0.0910632	**0.021***
TGs	0.0135312	0.0061959	**0.037***
LDL-C	0.011519	0.0179867	0.527
HDL-C	0.0093382	0.0544628	0.865
FBS	-6.051791	14.88557	0.687

* P value is significant.

Table 14: Multiple linear regression model for prediction of Troponin T levels among male obese study subjects.

Troponin T	Coeff.	Std. Err.	P value
BMI	-1.445223	0.7889295	0.088
WC	0.2350297	0.095372	**0.027***
TGs	-0.0121675	0.0070383	0.106
LDL-C	0.0177299	0.0286418	0.546
HDL-C	0.0166767	0.0751732	0.828
FBS	24.8422	21.16445	0.26

* P value is significant.

Table 15: Multiple linear regression model for prediction of Troponin T levels among female obese study subjects.

Troponin T	Coeff.	Std. Err.	P value
BMI	0.136405	0.066072	**0.046***
WC	-0.00875	0.022815	0.704
TGs	0.004042	0.004603	0.386
LDL-C	0.00716	0.005163	0.174
HDL-C	-0.00055	0.003932	0.89
FBS	-1.84411	1.285315	0.16

* P value is significant.

Table 16: Multiple linear regression model for prediction of Troponin T levels among male non-obese study subjects.

Discussion

Abdominal obesity is a condition that is considered to promote atherosclerosis and increases the risk of cardiovascular events. **(**17**)** Almost all of the major coronary heart disease risk factors, including lipid disorders **(**especially elevated triglycerides and low levels of high-density lipoprotein cholesterol**)**, glucose abnormalities, the metabolic syndrome, and diabetes mellitus, hypertension or left ventricular hypertrophy, and physical inactivity, are all adversely affected by overweight and obesity. **(**17**)**

Additionally, overweight and obesity may be independent risk factors for CHD and have adverse impacts on other cardiovascular disorders that may accompany CHD, including heart failure **(**HF**)**, atrial fibrillation, and risk for sudden cardiac death. **(**18**)**

In addition, obese patients have an increased burden of coronary artery disease and a higher incidence of acute coronary syndromes. **(**19**)**

Each abnormality promotes atherosclerosis independently, but when clustered to- gether, these metabolic disorders are increasingly atherogenic and enhance the risk of cardiovascular morbidity and mortality. **(**20**)**

CAD are the most common cause of mortality in the world, MI is the most common subtype of CAD, in addition, the prevalence of MI is increasing in the developing countries, In recent years, metabolic syndrome **(**MetS**)** is introduced as one of the major risk factors for CAD. **(**12**)**

The present study was conducted in order to find out the relation between presence of abdominal obesity and severity of myocardial infarction after an acute coronary event **(**STEMI**)**.

After estimation of maximum concentration of cardiac biomarkers, it was found that cardiac biomarkers were significantly high in obese group rather than non- obese group **(**P >0.001**)**.

That result showed a significant relation between obesity and increased levels of cardiac biomarkers which reflected severity of myocardial injury and that was consistent with a study done by Iglesias Bolaños P et al. in which they found that the presence of abdominal obesity was associated with greater myocardial necrosis after an acute coronary event. **(**21**)**

Furthermore, another study done by Dagenais GR, et al. concluded that Obesity, particularly abdominal adiposity, worsens the prognosis of patients with CVD, which concordant with our study. **(**22**)**

Nevertheless, a study done by Bucholz EM, et al. in which they concluded that relation between obesity and severity of myocardial infarction is paradox which disagreed with our study. **(**23**)**

In our study we found that both BMI and WC parameters for measuring obesity were significantly related to higher cardiac biomarkers **(**p>0.001**)** and that disagreed with the INTERHEART study which indicated however that the parameter of measuring obesity and had a best prediction of cardiovascular risk was not the traditional BMI, but waist circumference and waist – hip ratio. **(**24**)**

Furthermore, a study done by Wolk R, et al. found that BMI was independently associated with acute coronary syndromes which concordant with our study and reflected the importance of BMI in addition to WC. **(**25**)**

Obesity and distribution of body fat to the abdominal area have been linked to the promotion of plaque buildup and atherosclerosis. There are several other risk factors for CHD that are associated with obesity, including diabetes, insulin resistance, dyslipidemia, hypertension, LV hypertrophy, endothelial dysfunction, chronic inflammation, and obstructive sleep apnea. **(**26**)**

Obesity not only predisposes to insulin resistance and diabetes, but also contributes to atherogenic dyslipidemia. High levels of free fatty acids originating from visceral fat reach the liver through the portal circulation and stimulate synthesis of the triglyceride-rich lipoprotein VLDL by hepatocytes. The resulting elevation in VLDL can lower HDL cholesterol by augmenting exchange from HDL to VLDL by cholesteryl ester transfer protein. Adipose tissue can also synthesize cytokines such as tumor necrosis factor- α and interleukin-6. In this way obesity itself promotes inflammation and potentiates atherogenesis independent of effects on insulin resistance or lipoproteins. **(**27**)**

Sex: In our patients both sexes were represented with a significantly higher representation of the males **(**70.18%**)** compared to females **(**29.82%**)**. This was concordant with a study named Age and Gender Distribution in Patients with Acute ST Elevation Myocardial Infarction; A Survey in a Tertiary Care Government Hospital—NICVD, Karachi, Pakistan performed in Karachi, Pakistan which concluded that males are more at risk to have acute coronary event than females. However in that study percentage of males were81% and females 19%, this differences between them and our study may related to socioeconomic, genetic or associated risk factors. **(**28**)**

In a study performed by Borgeraas et al. Compared normal with obese subjects, they found obese men had an increased risk of AMI more than females and that was disagreed with our study **(**p = 0.219**)** which showed insignificant relation between incidence of AMI and subjects' sex. **(**29**)**

The role of major cardiovascular risk factors in the development of CHD is well established among men. Among women, the data are less extensive. Reasons for the sex difference in CHD risk are not fully understood. Even though in most populations, cardiovascular risk factor patterns are more favorable among women than among men. **(**30**)**

Atherosclerosis is also a cumulative process, starting at a fairly young age. Even though sex differences in serum cholesterol levels and blood pressure disappeared with age, it is possible that the cumulative effects of these risk factors on arteriosclerosis remain larger in men than in women. **(**31**)**

Age: The present study reported that Age ranges in group **(**1**)** from 57.74 ± 7.59 and age ranges in group **(**2**)** from 56.53 ± 11.32 and this was statistically insignificant between the two groups.

Our current results met the results of the study by Tavil et al. **(**32**)** who reported statistically no significant difference between the two groups as regard age. Similar results were reported by Lanz et al. **(**33**)** and Kragelund et al. **(**34**)**

Obesity and hypertension: Our study reported that in group **(**1**)** 59.65% of patients had essential hypertension, while in group **(**2**)** 40.35% of patients had essential hypertension which was statistically significant between the two groups.

Our results were concordant with the results of study done by Landsberg L, Aronne LJ, et al. in which they found a strong relationship between obesity and hypertension. **(**35**)** Recent data from NHANES indicate that the prevalence of hypertension among obese individuals, with a BMI <30 kg⁄m2, is 42.5% compared with 27.8% for overweight individuals **(**BMI 25.0–29.9 kg⁄m2**)** and 15.3% for those with BMI <25 kg⁄m2. **(**36**)**

Furthermore, Kragelund et al. **(**35**)** also reported that there was highly statistically significant increase in number of cases with hypertension in metabolic syndrome in comparison to cases without metabolic syndrome. In addition, Lanz et al. **(**33**)** found that hypertension was the single most frequent cardiovascular risk factor found in those with MS.

In a study performed by Peter W. F. Wilson et al. also found that obesity associated with increased relative and population attributable risk for hypertension and cardiovascular sequelae. **(**37**)**

Hypertension follows closely behind lipids on a list of classical risk factors for atherosclerosis. Increasing evidence supports the view that, like atherosclerosis itself, inflammation may participate in hypertension providing a pathphysiological link between these two diseases. Angiotensin II, in addition to its vasoconstrictor properties, can instigate intimal inflammation. **(**38**)**

Angiotensin II can also increase the expression by arterial smooth muscle cells of proinflammatory cytokines such as interleukin-6 and monocyte chemo-attractant protein-1 and of the leukocyte adhesion molecule, vascular cell adhesion molecule- 1 on endothelial cells. Some of the clinical benefits of angiotensin converting enzyme inhibitor therapy may derive from interrupting such proinflammatory pathways. **(**39**)**

Obesity and DM: The present study reported that the incidence of DM in Group **(**1**)** was 33.33% of patients, while incidence of DM in Group **(**2**)** was 8.77% of patients which was statistically significant between the two groups. Our results met the results of a study performed by Mokdad A. H et al. named Prevalence of obesity, diabetes, and obesity-related health risk factors in which they found a strong relationship between obesity and DM. **(**40**)**

More recently, investigators conducted a systematic review of 89 studies on weight related diseases and then did a statistical summary, or meta-analysis, of the data.

Of the 18 weight-related diseases they studied, diabetes was at the top of the risk list: Compared with men and women in the normal weight range **(**BMI lower than 25**)**, men with BMIs of 30 or higher had a sevenfold higher risk of developing type 2 diabetes, and women with BMIs of 30 or higher had a 12-fold higher risk. **(**41**)**

In the Islington Diabetes Survey, the prevalence of serious CAD increased from 9% in subjects with normal glucose tolerance to 17% in those with impaired glucose tolerance and 20% in those with diabetes. **(**42**)**

An Italian study carried out to evaluate MS and diabetes prognostic values in post- MI patients and found an increase in the risk of death and major cardiovascular events in both groups. Hyperglycemia and T2DM were independent predictive factors for recurrent ischemia and heart failure; Data from the 18-year Framingham. Study showed that the relative risk for CAD in diabetic men and women who were 45 to 74 years of age is 2.4 and 5.1 times greater, respectively, than for age- matched non diabetic men and women. **(**43**)**

Sustained hyperglycemia leads to the accumulation of non-enzymatically derived glycation products on proteins. In this process, glucose first forms chemically reversible Amadori type early glycation products with protein. Through a series of chemical rearrangements, some of these Amadori products are converted to advanced glycosylated end-products **(**AGE**)** as these are irreversibly bound to proteins, they accumulate continuously on long lived vessel wall proteins. The rate of accumulation is proportional to the time integrated blood glucose level over long periods of time. **(**43**)**

By binding surface receptors such as RAGE (receptor for AGE), these AGE- modified proteins can augment the production of proinflammatory cytokines and other inflammatory pathways in vascular endothelial cells. Beyond the hyperglycemia, the diabetic state promotes oxidative stress mediated by reactive oxygen species and carbonyl groups. As in the case of hypertension, inflammation links diabetes to atherosclerosis. (44)

A Pearson correlation between cardiac biomarkers and patients quantitative variables showed a strong relationship between increase cardiac biomarkers levels and BMI and WC plus high both TGs and LDL.c and low HDL.c levels. This results disagreed with a study done by Department of Endocrinology and Nutrition, Hospital Universitario de Getafe because in their study the found only BMI and WC had a strong relationship to cardiac biomarkers. (21)

A study performed at Manipal College of Medical Sciences, Pokhara, Nepal done by De Silva et al. found Subjects with chest pain and positive Troponin test (with confirmed cardiac event) were found to have significantly elevated levels of TC, TG, LDL and significantly reduced HDL levels when compared to the subjects who had only chest pain (Negative Troponin) and healthy controls which concordant with our results. (45)

Multiple prospective studies strongly suggest that elevated serum triglycerides are an independent risk factor for CAD. Other prospective studies show that a low level of HDL-C is an independent risk factor. (46, 47)

Two important mechanisms by which high-density lipoprotein (HDL) is thought to play a protective role against atherosclerosis which are first reverse cholesterol transport and second inhibition of LDL oxidation. (48, 49)

Our study showed that hypertensive patients who had STEMI had significant elevation in Troponin T level ($p > 0.004$) which reflected increasing in severity of myocardial necrosis and poor prognosis to those patients. That was consistent with several studies reported that a history of hypertension was associated with an increased rate of adverse outcomes after AMI such as stroke, heart failure, and cardiovascular death. (50) Increased incidence of AMI or sudden death in hypertensive patients may be related to several factors, such as endothelial damage, atherosclerosis, insulin resistance, left ventricular hypertrophy, and ventricular arrhythmias. (51)

Patients who had STEMI and were diabetic they showed marked elevation in Troponin T level ($p = 0.016$) and that means more myocardial injury and poor outcome, this result concordant with a study done by Wahab NN, Cowden EA, et al, which concluded that Hyperglycemia in AMI is associated

with poor outcome even among patients without known diabetes. This finding underlines the need for aggressive glucose management in this setting and may support a more vigorous screening strategy for early recognition of diabetes. **(**52**)**

Another studies showed poor prognosis, increased morbidities and mortality of patients who had STEMI and were diabetic in comparison with non-diabetic one which concordant with our result. **(**53-57**)**

A multiple linear regression analysis in order to obtain a statistical model predictor of myocardial enzymes elevation was performed. The results of the regression analysis showed that the variables significantly predicted CK-MB and Troponin T levels were BMI and WC. Several studies have attempted to relate obesity, measured as BMI, with prognosis and mortality after acute myocardial infarction. Presenting a higher BMI has not been shown to increase mortality after acute myocardial infarction. **(**58, 59**)**

However, patients with BMI <25 but WC> 88 cm in women and > 102 cm in men at increased risk of dying after suffering an acute myocardial infarction. **(**59**)** These findings support the results of our study on the relevance of abdominal fat distribution as predictor of severity of acute coronary event.

Conclusions

Presence of abdominal obesity is associated with greater myocardial necrosis after an acute coronary event.

Obesity is a risk factor of DM and HTN.

The effect of risk factors is additive, and the presence of multiple risk factors in the same patient can create a very high risk for increasing severity of an acute coronary event.

The waist circumference and body mass index are most important variables that significantly predict cardiac biomarkers' concentration **(**CK-MB, Troponin T**)**.

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