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ASSOCIATION OF SERUM OMENTIN-1 AND LEPTIN WITH ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

Introduction: Obesity, the most common nutritional disorder in industrialized countries, is associated with an increased mortality and morbidity of cardiovascular disease (CVD). **Objectives:** The main objective of the study is to find the association of serum Omentin-1 and Leptin with acute myocardial infarction. **Material and methods:** The data was collected from Doctors Hospital and Research Center, Lahore during January 2020 till June 2020. The data was collected after the permission of ethical committee of hospital. The data was collected from patients who suffering from acute myocardial infarction and visited OPD of the hospital regularly. The general information of the patients include age, BMI, BP, hypertension, smoking and previous history of myocardial infarction. **Results:** The data was collected from 50 patients. The mean age of the patients was 26 ± 3.9 years, with a maximum number of patients (60.2%) being within the age of 25-30 years and the rest in the age group of 20-25 years. The youngest patient was 20.1 years old. AMI in very young patients was highly prevalent in urban population (63.9%). **Conclusion:** It is concluded that Serum Omentin levels were significantly lowered while no change was found in serum leptin levels. Serum omentin-1 levels were independently associated with the MI.

KEYWORDS: CVD, BMI, BP, MI.

INTRODUCTION

Obesity, the most common nutritional disorder in industrialized countries, is associated with an increased mortality and morbidity of cardiovascular disease (CVD). Obesity is a chronic, multifactorial, and complex disease resulting from a long-term positive energy balance, in which both genetic and environmental factors are involved.[1] It was recently suggested that some forms of obesity are associated with chronic low-grade inflammation. Acute myocardial infarction is one of the main diseases leading to death worldwide and is caused by atherosclerosis and other metabolic syndromes that induce inflammatory and immunological reactions that are caused by adipokines like Visfatin.[2]

Omentin-1 is secreted from omentum and visceral fat also, it is found in the heart, lung, placenta, and ovary, it produced protective roles in regulating inflammatory and immunological response to induction of insulin sensitivity and downregulation of tumor necrosis factor (TNF), and it inhibits free radicals and superoxide formations that play an important role in the vascular inflammation and smooth muscle remodeling; so it is regarded as protective adipokine in IHD.[3] Moreover, omentin-1 levels are inversely correlated with obesity, body mass index, hemoglobin, cholesterol, and type 2 diabetes mellitus.[4]

Omentin-1, an adipocytokine released from visceral fat tissue, is associated with metabolic syndrome, diabetes and hypertension. A previous study found that that omentin-1 was down regulated by insulin and glucose. The influence of the omentin-1 on vascular health had been suggested.[5] Previous studies revealed that omentin-1 might have role in atherosclerosis in patients with metabolic syndrome, coronary artery disease (CAD), cardiovascular dysfunction in diabetes mellitus, and peripheral artery disease (PAD).

Literature indicates that omentin-1 has protective and leptin have a damaging role in atherosclerosis, but still there is inconsistency in their role in the AMI. Moreover, these parameters have not been discussed much in AMI patients, so there is a scarcity of available data about these parameters' involvement in MI in our local population.[6]

Objectives

The main objective of the study is to find the association of serum Omentin-1 and Leptin with acute myocardial infarction.

MATERIAL AND METHODS

The data was collected from Doctors Hospital and Research Center, Lahore during January 2020 till June 2020. The data was collected after the permission of ethical committee of hospital. The data was collected from patients who suffering from acute myocardial infarction and visited OPD of the hospital regularly. The general information of the patients include age, BMI, BP, hypertension, smoking and previous history of myocardial infarction. Blood sample was collected after fasting of night and then centrifuged it at 4000rpm for the separation of serum. Omentin and Leptin levels were measured by using commercially available ELISA kits.

The data was collected and analysed using SPSS version 19. All the values were expressed in mean and standard deviation.

RESULTS

The data was collected from 50 patients. The mean age of the patients was 26 ± 3.9 years, with a maximum number of patients (60.2%) being within the age of 25-30 years and the rest in the age group of 20-25 years. The youngest patient was 20.1 years old. AMI in very young patients was highly prevalent in urban population (63.9%). Smoking was the most common risk factor (77.4%), hyper lipidemia being the second common risk factor (78.5%), whereas 46.8% of the patients had a family history of premature CAD.

Table 01: Baseline characteristics of the patients.

Variables	n (%)
Age (year)	26.0 ± 3.9
Sex (male/female)	1061/95 (95.1/4.9)
Background (urban/rural)	714/452 (63.9/36.1)
Smoking	877 (78.5)
Hypertension	229 (20.5)
Diabetes	191 (17.2)
Family history of premature CAD	522 (46.8)
Obesity	437 (39.1)
Hyperhomocysteinemia	214 (19.2)
Physical inactivity	432 (38.7)
Substance abuse (cannabis)	52 (4.6)
Stressful life events	330 (29.6)
Dyslipidemia	236 (21.2)
Total cholesterol (mg/dl)	193.7 ± 36.4
LDL-C (mg/dl)	123.2 ± 26.1
TG (mg/dl)	177.1 ± 57.4
HDL-C (mg/dl)	33.2 ± 7.3
Non-HDL-C (mg/dl)	158.0 ± 14.9

Table 02: Serum omentin-1 and Leptin level in acute MI in the patients.

Biochemical parameters	Mean ± SD	95% CI Upper–lower limits	P-value
S. omentin-1 pg/mL	27.13 ± 1.55	27.7088–26.5512	<0.0001
S. Leptin-I pg/mL	21.202 ± 3.483	21.4395–20.9645	<0.0001
FBG mg/dL	118.96 ± 9.704	122.5835–15.3365	<0.0001
Total cholesterol mg/dL	140.96 ± 30.091	152.1962–129.7238	0.0475
TG mg/dL	138.66 ± 10.366	142.5307–134.7893	<0.0001
HDL mg/dL	51.23 ± 5.882	53.4264–49.0336	0.0058
VLDL mg/dL	27.64 ± 2.082	27.7088–26.5512	<0.0001
LDL mg/dL	62.08 ± 3.834	62.2667–61.8933	<0.0001
AI	0.072 ± 0.008	0.075–0.069	0.0003
SPB mmHg	123.16 ± 9.042	126.5363–119.7837	<0.0001
DPB mmHg	75.50 ± 1.643	76.1135–74.8865	<0.0001
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Furthermore, there is a significant difference in most of biochemical parameters regarding serum omentin-1 and laptin between the patients that received metformin and the patients that did not receive metformin prior to occurrences of acute MI.

DISCUSSION

Serum omentin-1 is significantly negatively linked with weight, BMI, waist circumference, W/H ratio, HbA, hsCRP and leptin in MI patients while no significant association with TC, TG, LDL-C, and HDL-C was found. Our results are similar to many studies that found lower omentin-1 levels in CAD subjects, and it was negatively linked with BMI and waist circumference and independently.[7]

Omentin-1 is a novel adipokine primarily released from visceral adipose tissue. So far, conflicting results were reported from studies investigating the association between omentin-1 and different cardiovascular endpoints.[8] On the one hand studies promoted omentin-1 as cardio-protective adipokine, showing cross-sectional associations of omentin-1 with several cardiometabolic parameters e.g. inverse association between omentin-1 and carotid artery intima-media thickness in patients with metabolic syndrome and decreased omentin-1 associated with cardiovascular dysfunction in patients with type 2 diabetes.[9]

Of the many positive and negative acute-phase reactants, perhaps the most recognized is CRP, which is a member of the pentraxin family that attaches to the plasma membrane of damaged cells causing cell death through activation of the complement cascade[10] More than 20 prospective epidemiologic studies have demonstrated that high-sensitivity CRP is an independent predictor of myocardial infarction, stroke, peripheral arterial disease, and sudden cardiac death, even in apparently healthy individuals.[11] Clearly, CRP is one of the strongest markers of chronic inflammation, and it has been reported that it also directly participates in the coronary and aortic atherosclerosis that leads to cardiac events.[12]

CONCLUSION

It is concluded that Serum Omentin levels were significantly lowered while no change was found in serum leptin levels. Serum omentin-1 levels were independently associated with the MI.

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