Obesity and metabolic syndrome among Egyptian adolescents

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Abstract:

A considerable increase in the prevalence of metabolic syndrome (MS) has been reported in parallel to the increasing frequency of childhood obesity and type 2 diabetes mellitus. This study provides current estimates of the metabolic syndrome and some of its individual components in obese Egyptian adolescents.

A total of 93 persons were submitted to this study and were classified into two groups. The first one included 53 obese patients without MS. Second group included 40 obese patients with MS. Both were compared with control group (40 healthy persons). All were cross matched regarding age, sex and race. The studied subjects were investigated for serum C-reactive protein (CRP), adiponectin and homocysteine (Hcy). In addition, some lipid parameters via total cholesterol (TC), high density lipoprotein (HDL-c), low density lipoprotein (LDL-c) and triacylglycerol (TAG) were also determined to reflect the presence of dyslipidemia.

Our study showed that there was a highly significant elevation of homeostatic model assessment- insulin resistant (HOMA-IR), CRP, homocysteine and lipid profile in patients without MS and with MS, while there was a highly significant decrease in adiponectin in the same patients.

The results of this study suggest that Surrogate markers of insulin resistance such as fasting insulin, HOMA-IR, adiponectin and glucose tolerance test, could be a useful addition to routine evaluations of overweight children in order to alert clinicians to potential increased risk in those with IR even without MS.

Key words: *Obesity, insulin resistance, HOMA-IR, adolescent, homocysteine.*

1-Introduction

Obesity and overweight and have been shown to be associated with increased prevalence of type II diabetes, gastro-oesophageal reflux, hypertension, dyslipidemia, and certain cancers[1] .Obesity is associated with an increased risk of mortality, particularly from cardiovascular diseases [2]. Recently, several landmark articles have shown a reduction in mortality for morbidly obese individuals who underwent bariatric surgery compared with controls without surgical intervention [3, 4].

Metabolic syndrome is defined as a condition in which a person has at least three of the five heart disease risk factors: abdominal obesity with a waist circumference of ≥ 102 cm for men and ≥ 88 cm for women; Abnormal lipids profile included a total cholesterol level ≥ 200 mg/dl or triacylglycerol (TAG) level ≥150 mg/dl ; HDL cholesterol level < 40 mg/dl for men and < 50 mg/dl for women; high blood pressure, with a systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg or taking antihypertensive medication; elevated fasting plasma glucose concentration ≥ 125 mg/dl, have a haemoglobin $A1_C$ level ≥ 6.0%, or use antidiabetic medication(s) such as insulin or oral hypoglycaemic agents [5] .

Among Egyptian adolescents the prevalence of obesity was 20.5%, the prevalence of MS was 7.4% ***NNI*** [6]**.** This study provides current estimates the metabolic syndrome and some of its individual components in obese Egyptian adolescents.

2-Subjects and Methods

2-1Subjects:

This study included 93 patients together with 40 healthy control subjects. All were randomly selected from Egyptian preparatory schools in Giza governorate. A full medical history was taken with special attention to any associated medical problems. The age, gender, weight, height and body mass index (BMI) were recorded. The exclusion criteria were: renal and liver disease, bronchial asthma, diabetes insipidus, thyroid disease and cancer.

In this study the subjects were divided into two groups:

Group-I:

Included, 53 obese patients without metabolic syndrome (33 males – 20 females) with ages range from 11-17 (mean, 14.38 ±SD 1.54) years.

Group-II:

Included, 40 obese patients with metabolic syndrome (19 males - 21 females), ages ranged from 10-17 (mean, 14.43 ±SD 1.46) years.

Control Group:

Included, 40 healthy subjects with age ranged from 11-18 (mean, 15.81 ±SD 1.4) years.

2-2 Blood Specimens:

From each subject ten ml of venous blood samples were collected in the morning after an overnight fasting and was kept in clean glass tube without additives to clot at 37 ^oC for 20 minutes, and then centrifuged at 3000 rpm for

10 minutes. The serum were then separated and stored at -70 °C to be thawed only once on demand for the determination of biochemical investigations.

2-3 Chemicals and Reagents:

Chemicals used in the present study were of high analytical grade, purchased from Spinreact (Germany), STANBIO (Germany), ABC diagnostic (Egypt), Bicon (Germany), Randox (USA), R D system(USA), Gamma trade Co. (Biosurce international, California) and Euroclone (Italy).

2 -4 Parameters to Be Investigated:

I- Sera were subjected to the following investigation:

- Quantitative determination of serum glucose by ***Trinder*** [7].
- Quantitative determination of insulin according to the method of ***Lang et al,.*** [8] using Roche Elecsys 2010.
- The homeostasis model assessment (HOMA-IR) method, the index of insulin resistance was calculated on the basis of fasting values of plasma glucose and insulin. (HOMA-IR) = (fasting glucose in mmol/L x fasting insulin in uU/ml/22.5) according to ***EL Sammak et al.,*** [9]
- Quantitative determination of triacylglycerol by ***Young and Pestaner*** [10]
- Quantitative determination of serum total cholesterol by ***Trinder*** [7].
- Quantitative determination of serum high density lipoprotein cholesterol (HDL-c) by ***Friedewald*** [11].
- Quantitative determination of low density lipoprotein cholesterol (LDL-c) by ***Freidwald*** [11].
- Risk ratio LDL-c/HDL-c was Calculated.
- Quantitative determination of serum C-reactive protein concentration was carried out according to the method of ***Roberts*** [12].
- Quantitative determination of serum homocysteine concentration was carried out according to the method of ***Lawrence*** [13], using ELISA technique.
- Quantitative determination of serum adiponectin concentration was carried out according to ***LINCO Research*** [14].

II- Control subjects and patients were subjected to the following anthropometric measurements:

The height and weight were measured by well trained staff, and body mass index (BMI) was calculated according the following ***formula Quetelet*** [15].

BMI = weight (kg)/Height (meter) ^{2}.

Statistical Analysis

All data was entered and analysed using Statistical Package for Social Sciences version 15.0 (SPSS, Inc., Chicago, IL, USA). For continuous data, descriptive analyses used means and standard deviation (SD).

The *t* test was used to compare between two independent means.

3-Results:

The goal of this work is to investigate the status of obesity in Egyptian adolescents with metabolic syndrome and to assess the potential relationship between some biochemical parameters in obese patients and whether these parameters are changed in those patients.

This aim will be achieved by determination of fasting blood glucose, insulin, HOMA-IR, lipid profile, homocysteine also CRP & adeponectin which reflect the inflammatory process. Moreover, control subjects and patients were subjected to anthropometric measurements.

1- Measurements of blood pressure and BMI related to age in obese and control groups:

The results presented in table (1) and fig. (1) revealed that, Diastolic blood pressure was significantly decreased by -3.06% ($P<0.05$) in group I when compared with control group while, DBP group showed significant increase by 3.79% in II vs. I. Also BMI was highly significantly increased in groups I and II by 32.73% and 40.55% respectively ($P<0.001$) when compared with control group while, the BMI showed non- significant change in group II vs. I.

2- The concentration of blood glucose, insulin and HOMA-IR of obese and control groups:

Data recorded in table (2) and fig. (2) showed that, fasting blood glucose was highly significantly increased in groups I and II by 31.63% and 32.29% respectively ($p<0.001$) when compared with control group while, non-significant changes in group II vs. I was observed.

Moreover, insulin was also highly significantly increased in group I and II by 45.80% and 70.96% respectively ($P<0.001$) when compared with control group while, it showed significant increase in group II vs. I by 17.25% ($P<0.05$).

HOMA-IR was calculated and showed highly significant increase in group I and II by 45.47%, 74.71% respectively ($P<0.001$) when compared with control group in addition to highly significant increase in group II vs. I by 29.24% ($P<0.001$).

3- Concentrations of lipids profile and risk factor of obese and control groups:

The results presented in table (3) and fig. (3) showed Highly significant increase of triacylglycerol in group I and II by 29.9% and 38.24% respectively ($p<0.001$) when compared with control group while, non-significant in group II vs. I was observed. While Cholesterol level was highly significantly increased in group II by 20.03% ($p<0.001$) when compared with control group while, it showed highly significantly increased in group II vs. I by 15.73% ($p<0.001$). Similarly LDL-c was highly significant increase by 25.33% ($p<0.001$) in group II and significant decrease by -10.71% ($p<0.05$) in group I when compared with control group while, in group II vs. I LDL-c showed highly significant change by 40.36%.

4- Concentrations of homocysteine, CRP and adiponectin in obese and control groups:

Table & fig. (4) Revealed highly significant increase of homocysteine in group I by 27.41% ($p<0.001$) while, non-significant change was observed in group II when compared with control group. Comparison of group II vs. I showed significant decrease by -13.07% ($p<0.05$).

There is a highly significant increase of CRP in group I and II by 88.63% and 85.35% respectively ($p<0.001$) when compared with control group. While, non-significant changes in group II vs. I was recorded. But highly significant decrease of adiponectin in group I and II by -33.90% and -34.48% respectively ($p<0.001$) when compared with control group. While, non-significant changes in group II vs. group I was recorded.

Discussion

Childhood obesity is reaching epidemic proportions and represents the most important chronic disease in this age group [16]. In the USA 15.8% of children between 6 and 11 years and 16.1% of adolescents have a body mass index (BMI) in the range of overweight[17] .

The metabolic syndrome called (insulin resistance syndrome and X syndrome) is a common pathophysiologic condition with implications for the development of many chronic diseases. Obesity beginning in childhood often precedes the hyperinsulinemic state. The metabolic syndrome is rapidly increasing in prevalence with rising childhood obesity and sedentary lifestyles worldwide. Although chronic diseases are now well recognized as a growing problem for low- and middle-income countries, limited data are available for these countries, and the developing world has been largely ignored in health strategies[18].

In our study, diastolic blood pressure (DBP) was significant decreased in patient group I compared with control group, while significant increased in group II *vs*. I, which agreement with ***McGill H et.al,*** [19] & **Lurbe *et. al.,*** [20] who have shown that the extent of early atherosclerosis of the aorta and coronary arteries is directly associated with levels of lipids, blood pressure, and obesity in childhood and adolescence.

Sinaiko et al. [21] showed a lack of significant correlations for blood pressure with fasting insulin (adjusted for BMI), insulin resistance, triglycerides, HDL-c, and LDL- c. However, when the MS factors (triglycerides, HDL-c, fasting insulin, and BMI) were considered together as a cluster and comparisons made between children with high and low blood pressure, the cluster score was significantly higher in the high blood pressure group.

Obesity is characterized by an excess of adipose tissue. There are different types of fat and different locations for fat in the body. Visceral fat has been shown to be a better indicator of obesity associated disorders than the amount of total fat. The most commonly used measurement for determining obesity is the BMI, which is calculated as the weight ((kg)/height m^2) [22].

In the present study, the level of BMI was significant increase in patient groups compared to control group. These results are in agreement with results of ***Weiss et al.*** [23]who showed that, as the degree of obesity increases the prevalence of BMI increases, with obesity occurring in 38.7% of moderately obese (mean BMI 33.4 kg/m^2) and 49.7% of severely obese (mean BMI 40.6 kg/m2) in children and adolescents.

Current study showed that, FBS and insulin were highly significantly increased in patient groups ($P<0.001$) compared with control group. These results are in agreement with results of ***Raitakari et al.,*** [24]who reported that, insulin concentration was higher in children who subsequently showed clustering of high triglycerides, low HDL-C, and high systolic blood pressure levels at follow-up in the longitudinal Cardiovascular Risk in Young Finns Study.

Ten & Maclaren [25] state that, one reason for the elevated glucose may be that IR is generally associated with increased level of insulin production. The ability of insulin insensitive individual's beta cells to compensate for IR by production extra quantities of insulin in response to a glucose load may decrease with age.

In addition, ***Lambert et al.,*** [26]., reported a deleterious effect of prolonged presence of elevated insulin levels may take time to manifest themselves with regard to increase in circulating glucose levels, blood pressure and triglycerides, he also reported that there is association between IR and MS risk factors in the adolescents but not in the younger age groups.

Currently there are no widely accepted values to define IR in either normal or overweight children. Hence, markers of IR such as homeostatic model assessment (HOMA) as described by ***Haffner et al.***, [27] have been used as a surrogate measure of IR in epidemiological studies to identify associated risk factors.

Our study shows the correlations of HOMA as a continuous variable with the individual MS criteria and BMI. Glucose and insulin correlated significantly with HOMA for all groups. Similar results have been reported by ***Dhuper et al.***, [28] who demonstrated that, among individual MS components, all were significantly associated with increasing HOMA in the 12-19 age group, whereas in the young age groups only glucose and BMI showed statistically significant association, suggesting that, the associations between IR, obesity and CVD risk factors likely increase with age.

National Cholesterol Education Program 2002 classified such conditions as atherogenic dyslipidemia and it is commonly present in obese persons. In the present study dyslipidemia was observed, the major abnormalities were highly significant changes of TAG, total cholesterol, LDL-c and LDL/HDL ratio in all studied groups compared to control

group while, HDL-c was not significantly decrease in all studied groups.

Similar observation was reported by ***Wanner& Quaschningnn*** [29] who found the common characteristics of the lipid profile include an elevation of serum triacylglycerol, a decrease in the high- density lipoprotein cholesterol, and some elevation in the low-density lipoprotein cholesterol. All of which have been associated with increased atherosclerotic risk. Dyslipidemia is closely related to an increased risk of cardiovascular diseases including atherosclerosis, hypertension and myocardial infarction. ***Verh*** [30] showed that the metabolic syndrome is associated Hyperinsulinemia and impaired glycaemic control, independent of lipid levels, were associated with increased in vivo LDL oxidation, as reflected by the higher prevalence of high oxidized LDL. High levels of oxidized LDL were associated with increased risk of future myocardial infarction,

Consisted with our results also ***Csabi et al.,***[31] who noticed that, abnormal lipid profiles also are found in children with obesity and insulin resistance**.** ***Freedman et al.,*** [32] stated that, data from the Bogalusa Heart Study have shown that overweight children have significantly higher levels of total cholesterol, LDL-c, and triglycerides and lower HDL-c levels than normal-weight children. The hypertriglyceridemic waist phenotype has been proposed in adults as a predictor of the MS. [33, 34]

Elevated plasma homocysteine is an independent risk factor for cardiovascular diseases such as arteriosclerosis [35]), *coronary* heart disease, stroke and myocardial infarction [36].

The vascular damage, a major condition associated with elevated plasma Hyc levels may be due to protein Homocysteinylation. The Homocysteinylated proteins accumulate

in the vascular wall surfaces resulting in the destruction of the endothelial cells [37]

In addition, ***Glowinska et al.,*** [38] stated that, an increased concentration of Hcy was found, as did in our study. Some findings, however, show that there is dependence between the concentration of Hcy in children, and CVD.

Isomaa et al.,[39] stated that, obesity is also characterized by a whole inflammatory with an increase in circulating level of pro-inflammatory cytokines such as C-reactive protein interleukin-1-β (IL-1β), interleukine-6 (IL-6), and a decrease in anti-inflammatory cytokine levels such as adiponectin [40]**.**

The present results showed a highly significant increase in patient groups in CRP compared with control group while, no significant change of group II *vs.* I, agree with finding of ***Balkau & Charles*** [41] who found that, the serum level of CRP was highly significant increase in obese children and adolescent.

Initial analysis of our data indicated that, adiponectin was associated with clinical and biochemical parameter development apart from the degree of obesity. In our study, serum adiponectin was a highly significantly decreased in patient groups compared with control group, while was no significant change in group II *vs.* I.

Our results are in agreement with ***Trujillo & Scherer*** [42] who reported that, serum levels of adiponectin are decreased in obese and type 2 diabetes subjects. The low serum levels of adiponectin have been proposed to be a predictor for the development of insulin resistance and diabetes.

Several studies in animal models and human, have reported that adiponectin plasma levels are inversely correlated with the increase in pro-inflammatory cytokines and markers such as C-reactive protein [43]**.**

The results obtained in our study improved that obesity is an important factor for the development of insulin resistance and dyslipidemia in adolescents, which increase the prevalence of metabolic syndrome.

Conclusion

Insulin resistance is very common among children and adolescents with obesity, significantly associated with high blood pressure and high triglycerides levels. Obesity as malnourishment is an alarming problem of public health even in developed countries. The metabolic syndrome is a common and complex disorder combining obesity, dyslipidemia, hypertension, and insulin resistance. It is a primary risk factor for diabetes and cardiovascular disease.

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Table (1): Results of statistical analysis of age, SBP, DBP and BMI in obese and control groups.

Group	**Age** (Years)	**SBP**	**DBP**	**BMI**
		mm Hg		
Control:				
Range	(11.8-18.4)	(120-140)	(80-90)	(18.4-25.1)
Mean	15.81	130.00	81.75	22.02
SD	1.40	3.58	3.32	1.42
Group I:				
Range	(11.3-17.4)	(100-170)	(60-95)	(22.4-49.4)
Mean	14.38	129.66	79.25	29.23
SD	1.54	11.56	6.38	6.37
%Change	-	-1.51	-3.06	32.73
P<	-	NS	0.05	0.001
Group II:				
Range	(10.9-17.1)	(120-170)	(65-95)	(22.2-49.4)
Mean	14.43	131.63	82.25	30.95
SD	1.46	12.53	6.88	6.94
%Change	-	1.25	0.61	40.55
P<	-	NS	NS	0.001
Group II vs I:				
% Change	-	1.52	3.79	5.89
P<	-	NS	0.05	NS

NS: non-significant (P>0.05); P<0.05: significant;
P< 0.01 & 0.001: highly significant.

Table 2: Results of statistical analysis of FBS, insulin and HOMA-IR in obese and control groups.

Parameters / Group	FBS mg/dl	Insulin □mol/L	HOMA-IR
Control: Range Mean SD	 (67-108) 87.27 10.58	 (6.6-14) 9.62 2.17	 (38.4-80.9) 55.6045 12.76
Group I: Range Mean SD %Change P<	 (81-147) 114.87 16.39 31.63 0.001	 (8.9-27.8) 14.03 4.25 45.80 0.001	 (39.7-148.1) 80.89 25.21 45.47 0.001
Group II: Range Mean SD %Change P<	 (80.6-170) 115.45 19.12 32.29 0.001	 (7.9-34.6) 16.45 6.59 70.96 0.001	 (45.9-211.8) 97.148 43.50 74.71 0.001
% Change II vs I P<	0.50 NS	17.25 0.05	29.24 0.001

NS: non-significant (P>0.05); ***P<0.05: significant;***
P< 0.01 & 0.001: highly significant.

- **Table 3:** Results of statistical analysis of lipids profile in obese and control groups.

Group	TAG	TC	HDL-c	LDL-c	LDL-c /HDL-c Ratio
	mg/dl				
Control: Range Mean SD	 (64-136) 97.93 22.07	 (111-230) 165.25 25.89	 (34-61) 48.31 6.66	 (58-161) 109.76 22.47	 (0.25-0.74) 0.46 0.11
Group I: Range Mean SD %Change P<	 (84-175) 127.21 30.16 29.90 0.001	 (130-259) 171.40 24.32 -16.31 NS	 (34.6-64.6) 47.48 7.07 -1.71 NS	 (58-133) 98.01 18.84 -10.71 0.05	 (0.26-0.83) 0.51 0.12 29.67 NS
Group II: Range Mean SD %Change P<	 (83-199) 135.38 37.40 38.24 0.001	 (110-297) 198.35 40.97 20.03 0.001	 (33-62) 47.18 6.73 -2.32 NS	 (65-188) 137.57 34.64 25.33 0.001	 (0.24-0.72) 0.37 0.13 -19.34 0.001
Group II vs I: % Change P<	 6.42 NS	 15.73 0.001	 -0.63 NS	 40.36 0.001	 -26.89 0.001

NS: non-significant (P>0.05); P<0.05: significant;
P< 0.01 & 0.001: highly significant.

Table 4: Results of statistical analysis of homocysteine, CRP and adiponectin in obese and control groups.

Parameters / Group	H. cysteine □U/L	CRP mg/L	Adiponectin ng/ml
Control:			
Range	(6.1-24.1)	(0.98-3.79)	(42-94.7)
Mean	14.64	2.00	68.57
SD	4.25	0.63	14.76
Group I:			
Range	(9.3-36.8)	(2.2-6.2)	(29.5-85.5)
Mean	18.65	3.76	45.33
SD	7.38	0.98	14.14
%Change	27.41	88.63	-33.90
P<	0.001	0.001	0.001
Group II:			
Range	(8.8-27.2)	(2.3-7.5)	(26.5-85.5)
Mean	16.22	3.70	44.93
SD	4.68	1.15	15.43
%Change	10.76	85.35	-34.48
P<	NS	0.001	0.001
Group II vs I:			
% Change	-13.07	-1.74	-0.88
P<	0.05	NS	NS

NS: non-significant (P>0.05); P<0.05: significant;
P< 0.01 & 0.001: highly significant.

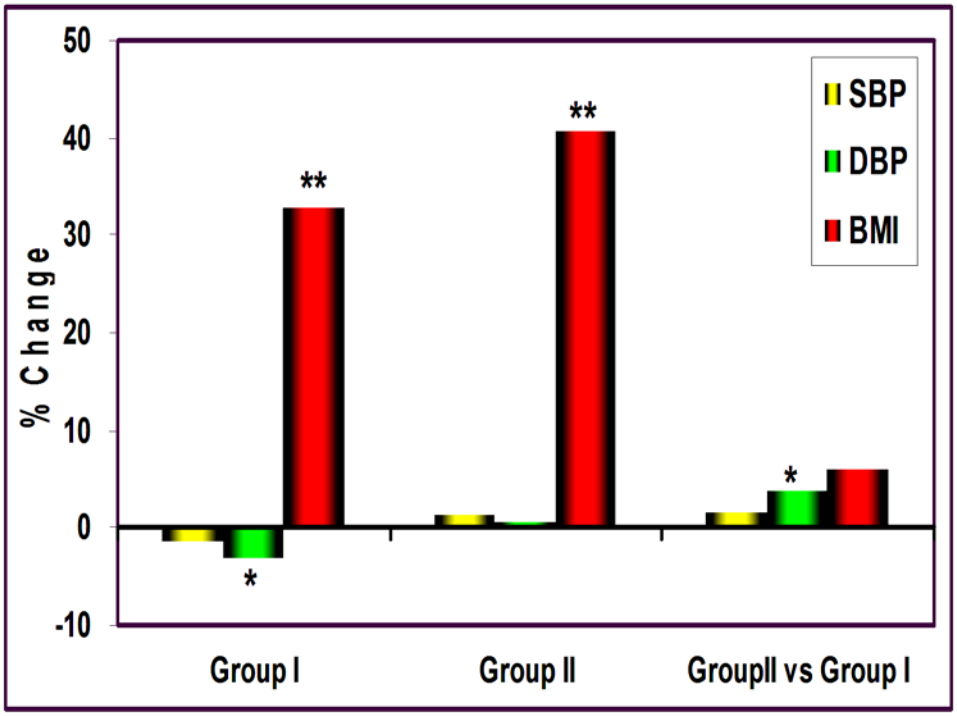

Fig. (1): % Change of SBP, DBP and BMI in different studied groups.

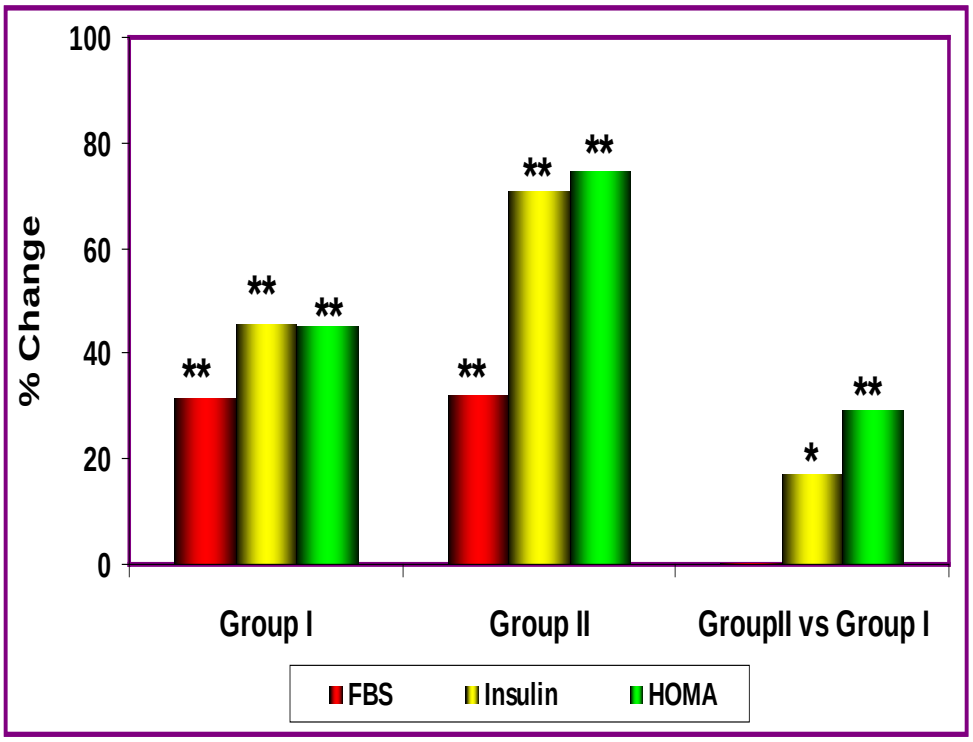

Fig. (2): % Change of FBS, insulin and HOMA-IR in different studied groups.

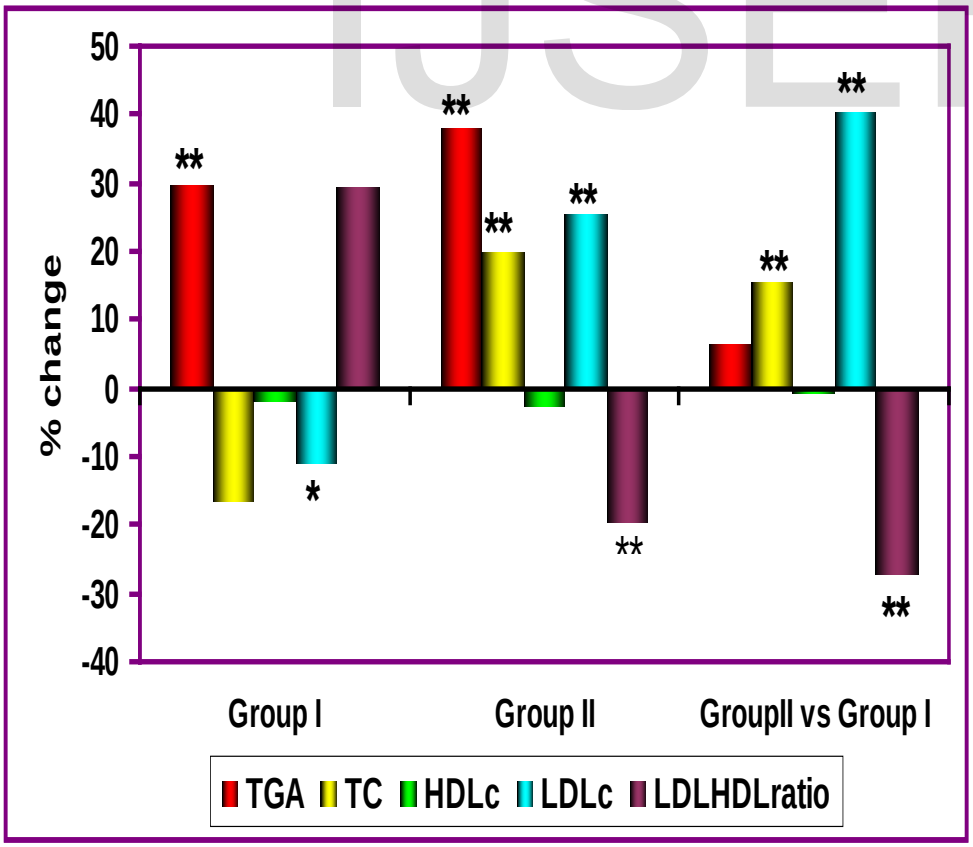

Fig. (3): % Change of lipids profile in different studied groups.

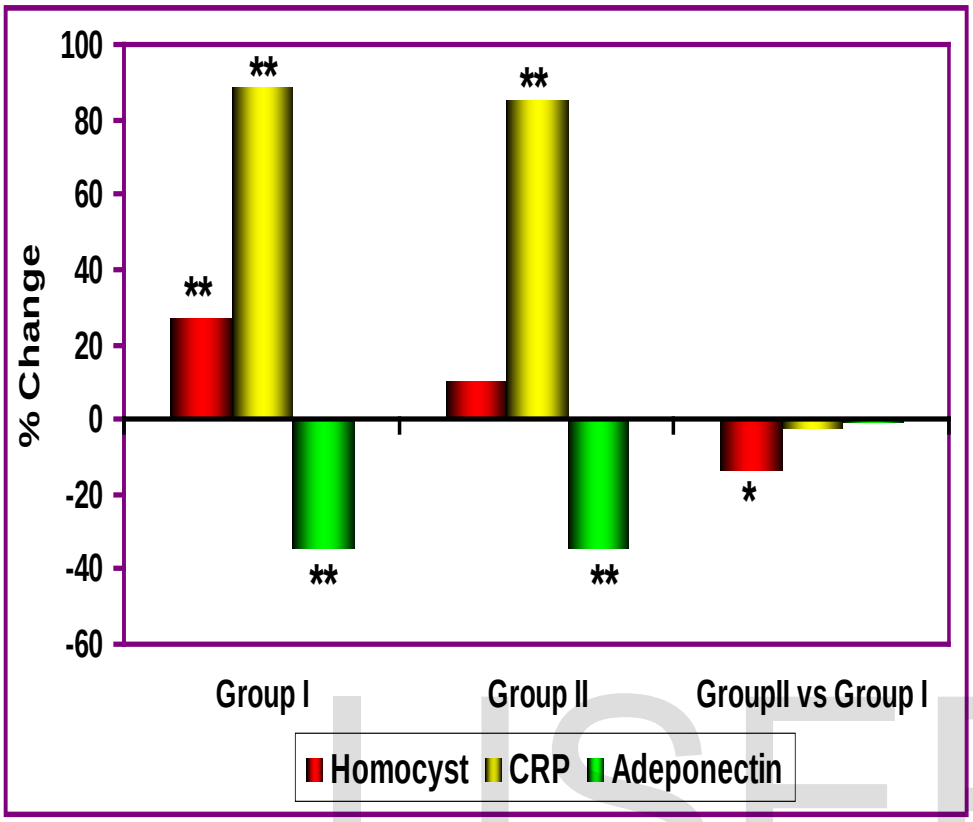

Fig. (4): % Change of homocysteine, CRP and adiponectin in different studied groups.

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