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Serum Adiponectin Levels in Pre-Postmenopausal Metabolic Syndrome Women and the Correlation with Some Physio-Biochemical Parameters

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ABSTRACT

Menopause is known to compound cardiovascular disease risk factors, including body fat distribution change and blood pressure increase, because estrogen withdrawal influences systemic cardiometabolic functions . Higher concentrations of circulating adiponectin have been found in women in comparison with men , and the 'menopausal metabolic syndrome' in postmenopausal women has also been proposed as a new concept. The present study aims to elucidate the effect of menopause on the association of plasma adiponectin with metabolic syndrome(MetS).Total of 81 women(a premenopausal group (n = 42) and a postmenopausal group (n = 39)) ,with aged 35-65 years included in this study. About 5ml of fasting blood (8-12 h.) was collected from each women. To determine serum adiponectin ,FSH(Follicle-Stimulating Hormone),LH(Luteinizing Hormone) and Estradiol the quantitative sandwich enzyme immunoassay technique were used. Fasting blood glucose (FBG) , glycosylated hemoglobin (HbA1c) and Lipid profile were measured by an enzymatic colorimetric(GPO-POD) method.There were significant differences between pre and postmenopause in metabolic syndrome and control subject female. There were significant elevation ($P<0.05$) in Waist circumference (WC), Fasting blood glucose (FBG) , Hemoglobin A1C (glycosylated hemoglobin), Triglyceride (TG) while adiponectin level decline in metabolic patients than control group, whereas the results of BMI(Body Mass Index),Systolic(SBP) and diastolic(DBP) blood pressure ,Cholesterol , HDL(High density lipoprotein) , LDL(Low density lipoprotein) ,VLDL (Very low density lipoprotein) ,Estradiol, FSH and LH show no significant differences ($P > 0.05$) between metabolic groups and control. According to menopause the results show significant elevation ($P<0.05$) in WC and LH in postmenopause females than premenopause female and significant decrease ($P<0.05$) in Estradiol in postmenopause females than premenopause female , while other parameters show no significant differences ($p>0.05$) between postmenopause females than premenopause female.Correlation analysis showed positive correlation between adiponectin and FBG in premenopause control female also between Estradiol with LH in postmenopause metabolic female ($r= 0.72, P= 0.007$ and $r= 0.61, P= 0.003$), while there was an inverse correlation between Estradiol with FSH and LDL in premenopause metabolic female was found($r= - 0.49, P= 0.02$ and $r= - 0.47, P= 0.03$) .We conclude that Estradiol hormone withdrawal in postmenopause leads to higher body weight and visceral adipose tissue, but surprisingly does not change in adiponectin levels.

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INTRODUCTION

Metabolic syndrome (MetS) known as a clustering of cardiovascular risk factors associated with insulin resistance, hypertension, glucose intolerance, hypertriglyceridemia, and low levels of high-density lipoprotein cholesterol (HDL-C), is a major worldwide public health problem . Serum adiponectin, which is a 244-amino acid protein secreted specifically by adipose tissue, contains four differentiable domains that regulate lipid metabolism, glucose metabolism, and insulin sensitivity , and low circulating levels of serum adiponectin has been reported as a risk factor for the development of metabolic syndrome and cardiovascular disease(CVD) (Hennemam *et al*.,2009; Kawamoto *et al*.,2011).

Many groups have suggested that plasma adipokines are potential biomarkers for MetS, in particular plasma adiponectin.It was shown that MetS is more prevalent in men than in premenopausal women. After menopause, women experience a substantial increase in dyslipidemia and other MetS related risk factors, leading to a risk profile comparable with that seen in men. Thus, differences between pre- and postmenopausal women with

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regard to the prevalence of MetS and plasma adiponectin have been reported independent from each other. It has been suggested that plasma adiponectin is a promising biomarker for MetS(Nishizawa *et al*.,2002; Gavrila *et al*.,2003; Matsuzawa *et al*.,2004;Jurimae and Jurimae,2007 ;Hennemam *et al*.,2009).

Adiponectin increases fatty acid oxidation in adipose tissue, liver and muscle, enhancing insulin sensitivity, and inhibits inflammatory mediators and the expression of adhesion molecules within the vascular wall, lowering atherogenic risk (Kahn *et al*.,2005;Babaei *et al*.,2010;Lecke *et al*.,2011).

Adiponectin activates adenosine mono-phosphate activated protein kinase and peroxisome proliferator activated receptor-γ in the liver and skeletal muscle,thereby stimulating phosphorylation of acetyl-CoA carboxylase fatty acid oxidation thus decreasing tissue Triglyceride (TG) content in muscle and liver. These alterations increase insulin sensitivety.The expression levels of Adiponectin receptor R1/R2 are also decreased in obesity ,which reduces adiponectin sensitivity and finally leads to insulin resistance ,the so-called "vicious cycle".Adiponectin was demonstrated to strongly inhibit the expression of adhesion molecules,including intracellular adhesion molecule-1 vascular cellular adhesion molecule-1,and E-selectin(Garg *et al*.,2012).

The menopause transition begins with the onset of menstrual irregularities and ends with the last menstrual period, which is associated with unfavorable changes in body composition, abdominal fat deposition and general health outcomes; for this reason, it is mandatory to investigate the changes in these risk factors during the menopausal transition. Deleterious changes in inflammatory markers and adipokines correlate strongly with increased visceral adiposity at menopause (Davis *et al*.,2012).

Menopause is known to compound cardiovascular disease risk factors, including body fat distribution change and blood pressure increase, because estrogen withdrawal influences systemic cardiometabolic functions . Higher concentrations of circulating adiponectin have been found in women in comparison with men , and the ‘menopausal metabolic syndrome’ in postmenopausal women has also been proposed as a new concept (Kotani *et al*.,2011).

Estrogen deficiency is associated with an increase risk of cardiovascular diseases (CVD) in postmenopausal women. One important reason for this might be greater visceral fat and consequently insulin resistance. Estrogen deficiency accelerates the selective deposition of abdominal fat and tends to accumulate visceral fat. Recently, it has become evident that adiponectin, an adipocyte-secreted hormone, has a protective role against atherosclerosis and is inversely correlated with accumulation of abdominal fat mass and insulin resistance.Based on this information, we hypothesized that adiponectin levels and lipid metabolism are modified in the absence of estrogens, which promotes visceral fat accumulation and other clusters of metabolic syndrome components. Higher concentrations of adiponectin in women than in men suggest that estrogen might have a stimulatory impact on the production of adiponectin (Babaei *et al*.,2010;Natah *et al*.,2013) .Contradictory findings exist show no change in plasma adiponectin concentrations by estrogens(Sieminska *et al*.,2005).

Today, cardiovascular disease is one of the main causes of mortality of women in the world . Considering the metabolic changes that occur in women during post menopause (PM), there is an increase in risk factors of cardiovascular diseases (CVD) and incidence of these diseases in PM . Increased incidence of MetS during PM period has been shown in many studies throughout the world . The etiology of MetS is not clearly defined, but it is shown that the syndrome is associated with visceral obesity .Thus, the theory of metabolic changes during PM and increased abdominal obesity as a result of decrease in estrogen is one of the hypotheses which explain the increased incidence of the syndrome during this period(Heidari *et al*.,2010;Soni *et al*.,2010).

However, there are few studies exploring the relationship between adiponectin in menopausal status and MetS. The present study aims to elucidate the effect of menopause on the association of plasma adiponectin with MetS.

METHODS AND PATIENTS:

This study was performed in the Diabetic center in Mergan hospital, Babylon province in 2013.The study population was composed of a premenopausal group (n = 42) and a postmenopausal group (n = 39), with age average (35-65 year). A 5-ml blood sample was obtained from the antecubital vein after 8-12 hour fast. The serum was used for estimating FBG,HbA1c and lipid profile by biochemical kit using spectrophotometer techniques. Subjects were classified as premenopausal if they had regular menstrual periods, and postmenopausal if they reported no periods for at least 6 months. Body Mass Index was calculated by dividing the body weight over the square of the height (Kg/m^2) (WHO,2004). The waist circumference was measured while the subject standing up , at the narrowest point of the torso width-wise, usually just above the belly button ,which is ≥ 102 cm in male and ≥ 88 cm in female (WHO,2004). The serum total adiponectin concentrations were measured with an enzyme-linked immunosorbent assay. Systolic BP (SBP) and diastolic BP (DBP) were measured in the seated subject’s right-arm with an appropriate cuff size using a mercury sphygmomanometer after 5 minutes of rest. Individuals suffering from renal dysfunction, liver disease, congestive heart failure, retinopathy or cataract were excluded. Women on medications or hormone replacement therapy , pregnant women were also excluded from the study. MetS would be present in women with at least three of the following criteria: waist circumference ≥ 88 cm, blood pressure ≥ 130 mmHg or ≥ 85 mmHg, fasting blood glucose ≥ 100

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mg/dl, triglycerides ≥ 150 mg/dl, HDL cholesterol ≤ 50 mg/dl. The metabolic syndrome was defined according to Third Report of the National Cholesterol Education Program's Adult Treatment Panel (ATP III).

The analyses were performed using the statistical package for social sciences (SPSS version 17.0). Physiological and biochemical parameters data were analyzed using factorial experiment with completely randomized. Data were represented as mean ± SD. Bivaraite correlations were performed using the Pearson correlation coefficient .P value ($P<0.05$) was considered statistically significant .

RESULT:

There were significant differences between pre and postmenopause in metabolic syndrome and control subject female. There were significant elevation ($P<0.05$) in waist circumference (WC), Fasting blood glucose (FBG), Hemoglobin A1C (glycosylated hemoglobin), Triglyceride (TG) while adiponectin level decline in metabolic patients than control group, whereas the results of BMI(Body Mass Index),Systolic(SBP) and diastolic(DBP) blood pressure, Cholesterol, HDL(High density lipoprotein) , LDL(Low density lipoprotein) ,VLDL (Very low density lipoprotein) ,Estradiol, FSH and LH show no significant differences ($P > 0.05$) between metabolic groups and control. According to menopause the results show significant elevation ($P<0.05$) in WC and LH in postmenopause females than premenopause female and significant decrease ($P<0.05$) in Estradiol in postmenopause females than premenopause female , while other parameters show no significant differences ($p>0.05$) between postmenopause females than premenopause female Table (1).Correlation analysis showed positive correlation between adiponectin and FBG in premenopause control female also between Estradiol with LH in postmenopause metabolic female($r= 0.72, P= 0.007$ and $r= 0.61, P= 0.003$) , while there was an inverse correlation between Estradiol with FSH and LDL in premenopause metabolic female was found($r= - 0.49, P= 0.02$ and $r= - 0.47, P= 0.03$) Table (2) and Table (3).

DISCUSSION:

This study show the level of adiponectin decline in metabolic patients than control group Table (1) ,this may be due to adiponectin concentrations correlate negatively with insulin resistance and adiposity. Recent lines of evidence indicate that oligomers of adiponectin, especially the High Molecular Weight(HMW) form, may be more important for the prediction of MetS or insulin resistance than measurements of total adiponectin.Prediction of MetS, defined by the presence of a cluster of metabolic abnormalities, including impaired glucose metabolism, increased central adiposity, dyslipidemia, and hypertension, seems to be important because of its association with the subsequent development of type 2 diabetes and cardiovascular disease (Devaraj *et al*.,2008; Zhuo *et al*.,2010). The plasma adiponectin levels were lower in subjects with MetS when compared with subjects with no diagnosis of MetS (Santaniemi *et al*.,2006).

In the present study, we demonstrated that plasma concentrations of adiponectin, an adipocyte-derived plasma protein, were not different between pre- and postmenopausal women Table (1) in agreement with astudy of Nishizawa *et al*.,(2002) and Lecke *et al*.,(2011), while disagreement with Soni *et al*. 2011 ,Hasan *et al*.,2012).

Adiponectin was similar between premenopausal and postmenopausal overweight women, suggesting that high BMI inhibited the potential rise in adiponectin early in the menopause transition. Our results extend those of earlier work in mostly obese women with the metabolic syndrome (Hong *et al*., 2007) demonstrating that the adverse (suppressive) effect of body weight on adiponectin secretion is present even in healthy, overweight women who do not meet the clinical definition of obesity (Rouen *et al*.,2010).

One study reported that estrogen inhibits adiponectin(Babaei *et al*.,2010) ,while other studies have reported that there were no overt clinical effects of estrogen on adiponectin(Hong *et al*.,2007) ; thus, the changes in several sex hormones that occur with the onset of menopause may influence circulating adiponectin concentrations(Kotani *et al*.,2011) .

Menopause and aging in women are associated with changes in the metabolism of abdominal and gluteal adipose tissue. It was shown that adipose tissue is able to produce increasing levels of estrogen with age at the time of menopause. The parallel increased levels of expression of adiponectin in the gluteal fat might lead to enhanced insulin sensitivity, which could be a physiological prevention of decreasing insulin sensitivity by estrogen depletion (Mascarenhas-Melo *et al*.,2013).

There were significant elevation in waist circumferences between metabolic patients and control group (Table 1), which belong to that plasma adiponectin concentrations are negatively correlated with parameters of overall obesity as well as measures of central obesity .Central obesity is known to be associated with insulin resistance,and adiponectin may represent a link between central obesity and insulin resistance (Jurimae and Jurimae,2007).

In addition to confirming the association between body fat mass and adiponectin, we found that central obesity is an independent negative predictor of serum adiponectin. The relationship of adiponectin with WC appears to be stronger than with fat mass or BMI, indicating that central fat distribution is a better determinant of circulating adiponectin than total fat mass(Gavrila *et al*.,2003).

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There were no significant differences in blood pressure between metabolic patients and control group (Table 1), this may be due to hypertension is not strongly linked to the metabolic syndrome (Ford *et al*.,2002).

There were significant elevation in triglyceride between metabolic patients and control group (Table 1), this may be because an increased mass of stored triglyceride, especially visceral or deep subcutaneous adipose depot, leads to large adipocytes that are themselves resistant to the ability of insulin to suppress lipolysis; this result in increased release of circulating Free Fatty Acid(FFA) and glycerol, both of which may aggravate insulin resistance in skeletal muscle and liver. These FFA can influence insulin signaling pathway by activating of several serine/thrionine kinases, reducing the tyrosine phosphorylation on Insulin Receptor Substrate (IRS) and impairing IRS/phospho-inositide-3-kinase pathway .Alterations in menopause are known to increase the abdominal visceral fat content as well as blood concentrations of cholesterol, triacylglycerol, glucose, and insulin . Changes in visceral fat content were found to be associated with subsequent impaired glucose-insulin homeostasis(Hasan *et al*.,2012).

We demonstrated that TG significantly and independently strong associations with these MetS-associated variables in both men and women. The best marker of three MetS-associated variables was TG/ HDL-C ratio. The presence of hypertriglyceridemia, low HDL-C concentrations, and high TG/HDL-C ratios almost never occurred as isolated disorders, and were nearly always associated with insulin resistance because insulin affects TG and HDL-C metabolism (Kawamoto *et al*.,2011).

Several clinical reports have pointed to an association between plasma adiponectin and dyslipidaemia. Adiponectin increases tissue fat oxidation, leading to reduced levels of fatty acids and tissue triglyceride content, thus increasing insulin sensitivity. Paradoxically, plasma adiponectin concentrations are decreased in obese subjects, suggesting that hypoadiponectinemia is involved in the pathophysiology of obesity(Im *et al*.,2005; Jaleel *et al*.,2006;Resmini *et al*.,2008).

Adiponectin correlates negatively with serum triacylglycerol (triglyceride) and positively with serum HDL cholesterol in non-diabetic women or young healthy men. Subjects with low plasma adiponectin levels have low lipoprotein lipase activity . Hypoadiponectinaemia also associates with smaller LDL (low-density lipoprotein) particle size . In patients with Type II diabetes, plasma adiponectin levels correlate positively with HDL-cholesterol and negatively with triacylglycerols and apolipoprotein B-100 (Okamoto *et al*.,2006).

The present study revealed estradiol decline in postmenopausal while LH significantly increase in postmenopausal women (Table 1) , this may due to that Sex hormone binding globulin (SHBG) declines, free estradiol should increase. Therefore, in response to decreased SHBG, follicle-stimulating hormone levels may decrease to lower total estradiol production by the ovaries, thus keeping free estradiol relatively constant. Additionally, the molecular clearance rate of estradiol is positively associated with weight, also potentially reducing total estradiol levels. Also, there was a strongly negative and significant association between serum estradiol level and total cholesterol. Furthermore, the associations between serum estradiol concentrations were found to be significantly positive with HDL-cholesterol. This explains why the premenopausal women are more protected against atherosclerosis and coronary heart diseases(Ali and Al-Zaidi,2011).

In this study, correlation analysis showed positive correlation between adiponectin and FBG in premenopause control women(Table 2),which belong that adiponectin modulates glucose metabolism by having insulin-sensitizing effects . Adiponectin also decreases circulating free fatty acid concentrations and muscle triglyceride content by stimulating fatty acid oxidation in muscle via AMP-activated protein kinase (AMPK) (Santaniemi *et al*.,2006).

Correlation analysis showed an inverse correlation between estradiol with LDL (Table 3),due to that estrogen lowers LDL cholesterol by up regulating apo B100 receptors and increases HDL-cholesterol by inhibiting hepatic lipase activity (Im *et al*.,2005). Estrogen-induced alterations in serum lipids account for only approximately one-third of the observed clinical benefits of estrogen. The atheroprotective effects of estrogen were principally attributed to the hormones effects on serum lipid concentrations. Estradiol reduces the development of early lesions of atherosclerosis, through an improvement in the blood lipid profile, which reduces lipid deposits in the endothelium, with a decrease in total cholesterol and low-density lipoprotein cholesterol (LDL-C) and an increase in high-density lipoprotein cholesterol (HDL-C) in the plasma. The physiological process of estrogen works by binding to estrogen receptors (Ali and Al-Zaidi,2011).

Also there were an inverse correlation between estradiol and FSH in premenopausal woman while positive correlation between estradiol and LH in postmenopausal woman (Table 3) because the Menopausal transition is characterized by variations in cycle length and elevation in follicle-stimulating hormone (FSH) level(Yasui *et al*.,2012). With cessation of follicular genesis and menopause, FSH level rises by 10-20 fold, and LH by 3-5 fold. High levels of gonadotropines are maintained during two or three years after menopause. In later age, levels of gonadotropines decrease again, or remain only mildly elevated. LH stimulates ovaries to produce androgens, which persists until advanced years, so that ovary preserves its function of an endocrine organ. Of course, production of estradiols and progesterone by ovary ceases(Mesalic *et al*.,2008).

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Table 1: Comparison in levels of some physio-biochemical parameters between pre and postmenopause in metabolic syndrome and control subject female.

Groups / Parameter	Control N=40		Metabolic syndrome N=41		P value of group	P value of menopause
	Premen. (N=22) Mean± SD	Postmen. (N=18) Mean± SD	Premen. (N=20) Mean± SD	Postmen. (N=21) Mean± SD		
Adiponectin(µg/ml)	7.47±2.79	7.02±1.62	4.49±1.39	5.94±1.80	0.02*	0.55
BMI(kg/m2)	30.36±6.48	31.12±5.90	29.99±3.33	30.03±3.37	0.55	0.74
WC(cm)	91.75±10.64	102.75±11.23	100.6±6.22	104.19±8.86	0.03*	0.004*
SBP mm Hg	123.7±11.1	128.7±12.3	133.2±11.9	135.2±12.1	0.08	0.09
DBP mm Hg	81.05±7.8	79.6±9,3	83.7±8.8	82.1±9.2	0.87	0.65
FBG(mmol/l)	5.04±0.66	5.56±0.77	12.37±2.95	12.09±3.90	0.001*	0.87
HbA1c(%)	5.19±1.13	4.42±0.85	7.57±1.00	8.52±1.73	0.001*	0.80
Cholesterol(mmol/l)	4.19±0.86	4.83±1.06	4.80±1.64	5.48±1.31	0.09	0.07
TG(mmol/l)	1. 32±0.43	1.47±0.52	2.22±0.35	2.39±0.89	0.001*	0.56
HDL(mmol/l)	1.16±0.18	1.07±0.14	1.07±0.21	1.11±0.28	0.65	0.68
LDL(mmol/l)	2.54±0.47	3.09±0.08	2.91±0.34	3.53±0.20	0.22	0.07
VLDL(mmol/l)	0.41±0.09	0.66±0.03	0.91±0.05	1.08±0.02	0.07	0.41
Estradiol(pg/ml)	27.66±8.27	22.73±1.71	42.59±9.62	21.57±1.44	0.37	0.05*
FSH(mIU/ml)	28.19±7.95	29.32±7.93	30.95±5.13	35.45±7.19	0.53	0.69
LH(mIU/ml)	9.05±2.97	16.79±2.29	10.79±1.39	17.48±1.79	0.58	0.002*

BMI: Body mass index, WC: Waist circumference, BP: Blood pressure ,FBG: Fasting blood Glucose,HbA1c : Hemoglobin A1C (glycosylated hemoglobin), TG: Triglyceride, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, FSH: Follicle-stimulating hormone ,LH: Luteinizing hormone, Premen.: premenopause, Postmen.: postmenopause.*P value is significant ≤ 0 .05 level, SD :Standard Deviation.

Table 2: The correlation between adiponectin with some physio-biochemical parameters in pre and postmenopause female for both groups.

Groups	Index		BMI	WC	FBG	HbA1c	Col.	TG	HDL	LDL	VLDL	Estra.	FSH	LH
Con.	Adipo in Pre. (N=22)	r	0.42	0.01	0.72	0.06	0.08	-0.27	-0.009	0.09	0.16	-0.19	-0.33	-0.24
		p	0.17	0.95	0.007*	0.85	0.78	0.38	0.97	0.77	0.61	0.54	0.28	0.44
	Adipo in Post. (N=18)	r	0.49	-0.02	-0.20	-0.17	-0.04	-0.28	0.08	0.01	-0.28	0.13	0.29	-0.32
		p	0.21	0.95	0.62	0.68	0.92	0.49	0.83	0.98	0.49	0.75	0.48	0.43
MetS	Adipo in Pre. (N=20)	r	-0.06	0.07	-0.11	0.27	-0.01	0.31	-0.07	-0.22	0.19	0.08	-0.03	-0.22
		p	0.77	0.74	0.62	0.23	0.95	0.17	0.74	0.33	0.40	0.73	0.86	0.33
	Adipo in Post. (N=21)	r	0.16	0.15	-0.04	0.04	0.02	0.22	0.13	0.01	-0.21	-0.18	-0.04	-0.09
		p	0.47	0.49	0.83	0.83	0.90	0.33	0.56	0.93	0.34	0.41	0.85	0.69

Adipo :Adiponectin, Estra.: Estradiol, Pre.: premenopause, Post.: postmenopause, ,Con:Control, MetS:Metabolic syndrome. Correlation coefficient (r) ,* Correlation is significant ≤ 0.05 level (2-tailed).

Table 3: The correlation between estradiol with some physio-biochemical parameters in pre and postmenopause female for both groups.

Groups	Index		FSH	LH	Col.	TG	HDL	LDL	VLDL	BMI	WC
Con.	estradiol in Pre. (N=22)	r	0.12	-0.08	0.12	-0.30	0.05	0.17	-0.03	0.41	-0.23
		p	0.68	0.80	0.69	0.32	0.87	0.59	0.92	0.18	0.47
	estradiol in Post. (N=18)	r	-0.29	0.44	0.21	0.42	-0.01	0.12	0.43	-0.12	-0.47
		p	0.47	0.26	0.60	0.28	0.96	0.77	0.28	0.76	0.23
MetS	estradiol in Pre. (N=20)	r	-0.49	0.26	-0.32	-0.03	-0.15	-0.47	0.09	0.27	0.11
		p	0.02*	0.26	0.16	0.89	0.52	0.03*	0.69	0.24	0.61
	estradiol in Post. (N=21)	r	-0.007	0.61	0.03	0.14	0.04	0.02	-0.24	-0.09	0.10
		p	0.97	0.003*	0.87	0.53	0.85	0.92	0.28	0.67	0.65

Pre.: premenopause, Post.: postmenopause, Con:Control, MetS:Metabolic syndrome.
Correlation coefficient (r) ,* Correlation is significant ≤ 0.05 level (2-tailed).

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CONCLUSIONS:

We conclude that Estradiol hormone withdrawal in postmenopause leads to higher body weight and visceral adipose tissue, but surprisingly does not change in adiponectin levels.

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