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RELATION BETWEEN PLASMA LEPTIN LEVEL AND LEFT VENTRICULAR FUNCTION IN OBESE FEMALES WITH INSULIN RESISTANCE

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Abstract

An association between obesity and cardiac mass has been recognized for almost two decades, whereas the precise nature of the association remains elusive Theoretical consideration have long suggested that it may be mediated at least in part by insulin resistance ***(Mc, Nutly ,2003).***Several studies have found an association between insulin resistance and left ventricular hypertrophy***.(Lacobellis et al,2003)***

In human, production of leptin(an adipocyte – derived peptide), has been linked to obesity, insulin and insulin sensitivity ***(Leyva et al, 1998*)** .It was considered that alteration in plasma concentration could constitute an additional component of metabolic syndrome of cardio-vascular risk***(Leyva et al, 1998***).

Aim of the work:

The aim of this work was to evaluate the relationship between obesity, insulin resistance, leptin and left ventricular mass and function in young obese females with insulin resistance.

Subjects and methods:

Sixty five premenopausal females aged 25-45 years with no history of diabetes or hypertension was participated in this study. Twenty were non obese and forty five were obese .Fasting serum glucose, insulin and leptin were assessed and homeostatic model assessment HOMA-IR score was calculated. According to HOMA-IR obese premenopausal females were divided into 2 subgroups: - **Subgroups 1**: (Insulin sensitive group or IS group) included 20 obese females with HOMA-IR <3.8.And **Subgroup 2**: (insulin resistance group or IR group) included 25 obese females with HOMA-IR≥3.8.Echocardiography was done for all females participated in the study to evaluate L.V mass and function.

Results:

Waist circumference (WC), serum insulin, serum leptin and HOMA-IR were significantly higher in obese group compared to non obese group ($p<0.05$, <0.05, <0.001 and <0.00l respectively) and between IR and IS subgroups ($p<0.05$, <0.05, <0.001 and <0.00l respectively).As regard Echocardiographic studies left ventricular mass(LVM) and left ventricular mass corrected t height $^{2.7}$ ($LVM/h^{2.7}$)were significantly higher in obese group compared to non obese group ($p<0.05$) and between IR and IS subgroups ($p<0.05$ for both),while the ratio between peak transmitral E and A wave velocity(E/A ratio) was lower in obese group compared to non obese group (<0.05), it was also lower in IR subgroup compared to IS subgroup ($p<0.05$).There was positive significant correlation between LVM and $LVM/H^{2.7}$ and serum insulin ($p<0.05$)and serum leptin ($p<0.05$) in IS subgroup while the correlation was highly significant between both and fasting leptin ($p<0.001$) in IR subgroup

Conclusion:

Obesity is a clinical syndrome associated with hyperinsulinemia, hyperleptinemia and insulin resistance Abnormalities of LV diastolic function and mass occur frequently in obese patients. Hyperleptinemia can be an early sign for left ventricular dysfunction in obese females.

Key words:
Leptin ,obesity, Left ventricular function, Left ventricular mass , insulin resistance

Introduction

The prevalence of obesity is increasing in both the developed and developing worlds ***(Wong et al. 2004)***
Recognition of the adverse cardiovascular complications of obesity and the increasing frequency of obesity has stimulated considerable research into the relationship between obesity and the cardiovascular system ***(Crisostomo et al 2001***).Obesity has been associated with heart failure ***(Kenchaiah et al (2002***) and individuals with serve obesity have long been recognized to have a form of cardiomyopathy attributed to chronic volume over load, characterized by left ventricular hypertrophy ***(Alpert (2001)*** and enlargement of LV ventricular mass that can be measured by echocardiography or angiography (***(Crisostomo et al 2001***). LV hypertrophy, indentifies patients at high risk of future morbid events, irrespective of age, blood pressure and LV ventricular function in relaxation. It has been proven to be independent predictive risk factor of cardiovascular morbidity and mortality (***(Crisostomo et al 2001***).
Obesity is also characterized by insulin resistance and compensatory hyperinsulinemia which may directly promote myocardial hypertrophy through the insulin-like growth factor -1 (IGF -1) receptors ***(Sasson, (1993).***
In humans production of leptin an adipocyte derived peptide has been linked to obesity, insulin and insulin sensitivity (***(Leyva et al, 1998***) .Several studies have show that leptin was increased in insulin- resistance states such as obesity ***(Paolisso et al, 1999).***The possibility that leptin plays a role in cardiovascular system is strengthened by the evidence that chronic leptin infusion has been shown to increase heart rate and blood pressure through stimulation of sympathetic nervous system activity ***(Paolisso et al, 1999).***

The aim of this work was to evaluate the relationship between obesity and insulin resistance, leptin, left ventricular mass and function in obese females with insulin resistance.

Subjects and Methods

Subjects

Sixty five premenopausal females, 25-45 years old with no history of diabetes or hypertension were participated in this study. Twenty were non obese with BMI<25Kg/m²and forty five were obese with BMI≥30Kg/m².All subjects included in the study had normal fasting glucose level (<110mg/dl), normal 2 h postprandial blood glucose level (<140mg/dl), normal resting arterial blood pressure (systolic<135mmhg; diastolic <85mmhg) for at least three measurements, normal serum level of total cholesterol (<220mg/dl) and triglysrides (<170 mg/dl).
None had any evidence of apparent cardiovascular, hepatic, renal, and respiratory or other metabolic disease by routine history, physical examination and laboratory screening tests.
Also, abnormal ECG findings except for those with non specific ST-T wave abnormalities or ECG criteria of LV hypertrophy were excluded.
All females were free from any medications known to affect glucose tolerance or LV mass (eg.diuritics, B blockers, corticosteroids, vasodilators as: calcium antagonists and angiotensine converting enzyme inhibitors).
Written informed consent was obtained from each female before participation in this study.

Methods

1- Anthropometric Measurements
Weight (to the nearest 0.1 kgm) and height to the nearest 0.5 cm) were measured while the subjects were fasting and wearing light clothes.
BMI was calculated as body weight (kgm) divided by height (in meter) square and was used as a marker of obesity.
Waist circumferences (in cm): were measured, as the widest diameter between the xiphoid process of the sternum and iliac crest.
2-Laboratory tests:
After an over night fast of at least 8 hours,5 ml of venous blood were withdrawn from patients

and control group. Blood was left to clot and serum was separated and used for assay of:

A-Fasting and 2 hours postprandial glucose level using chemistry autoanalyzer (Hitachi 911,Japan) and kits of Roche diagnostics, Germany.

B- Fasting insulin using DSL-10-1600 active enzyme, linked immuno-sorbant assay (ELISKA) KITS, for quantitative measurement of insulin, and an enzymatically amplified (one step) sandwich type immuno-assay.

C- Serum leptin serum leptin was assessed using Enzyme linked immunosorbant assay kit (Medizyme Leptin) supplied by Medipan Diagnostica, Germany.

D- Quantitative estimation of insulin resistance using Homeostatic Model Assessment (HOMA) score was done. HOMA is a reliable method for the assessment of in vivo insulin sensitivity in humans and its results were correlated with that obtained from the more sophisticated techniques such as the euglycemic clamb (10)

HOMA-IR: fasting insulin (μ/ml) × fasting glucose (mg/dl)/405.Insulin resistance was defined as HOMA-IR equal to or greater than 3.8 (11)
According to HOMA-IR, obese females were divided into 2 groups: **Subgroup 1**: Insulin sensitive (IS) group included 20 obese females with HOMAIR< 3.8.**Supgroup 2**: Insulin resistance (IR) group included 25 obese females with HOMAIR≥ 3.8.

Echocardiography:

Echocardiographic studies were performed at rest with the patient at steady state in the left lateral position, using commercially Philips EN-Visor using a 2.5 MHZ transducer.Two-dimensionaldirected M mode LV dimension measurement in accordance with American Society of echocardiography recommendations was used to measure the diameter and thickness of the left ventricle, in parasternal long axis, at the level of the tendinous cords of the mitral valve ***(Crisostomo et al 2001***).
The LV mass was calculated using Devereux and Reicheck formula {1.04 diastolic LV diameter + septal thickness + LV posterior wall thickness) ^{3}-(diastolic LV diameter) ^{3} - 13.6}, with Penn convention (***(Crisostomo et al 2001***), and was corrected to height$^{2.7}$ (LVM/ht$^{2.7)}$ ^{and} expected as units of (gm/m$^{2.7)}$ (***Sasson, 1993)*** .LV hypertrophy was defined as LVM>110 g/m^{3} and/or LVM/h$^{2.7}$ >47/h $^{2.7}$ (***(Lacobellis et al,2003)***.
To evaluate the LV systolic function, the ejection fraction of the LV (EF), and the shortening fraction was obtained.
The LV diastolic function was obtained by using the revolution ellipsoid method ***(Devereux et al ,1984).*** The LV diastolic function was evaluated through the ratio between peak transmitral E and A wave velocity, isovolumetric relaxation time (IVRT) of the LV and halftime E wave deceleration ***(Lebovitz et al, 1987).***

Statistical methods

Results were expressed as mean± standard deviation (SD).
Correlation between parameters was performed using spearman's rank correlation coefficient. SPSS program (version 14 windows) was used for data analysis.
P value less than 0.05 was considered significant, less than 0.001 considered highly significant and more than 0.05 was considered insignificant.

Results

(1)Anthropometric and Metabolic parameters

The summary data for anthropometric and metabolic parameters among non obese, obese, IS and IR subgroups are shown in tables (1 &2).No significant difference in age was found between obese and non obese groups (p>0.05), while BMI, WC and serum insulin were signicantly higher in obese groups compared to non obese groups (p<0.05).Only WC and serum insulin were significantly higher in IR subgroup compared to IS subgroup (p<0.05).
As regard serum leptin and HOMA-IR the difference was highly significant in obese groups compared to nonobese group (p<0.001) and between both IS and IR subgroups (p<0.001).

(2)Echocardiographic parameters:-

IR subgroup had higher LVM and LVM/H$^{2.7}$ comparing IS subgroup (p <0.05) and obese

group had significant higher LVM and $LVM/H^{2.7}$ comparing to non obese group ($p<0.05$) as shown in tables (3&4) .There was no significant difference in the parameters of systolic and diastolic functions of LV between the non obese, IS and IR subgroups ($p>0.05$ for all)except for E/A ratio which was lower in obese group compared to non obese group .E/A ratio was lower in IR subgroup than IS subgroup but this difference was statistically non significant.

As regard table (5) a univeriate analysis was performed in obese IS and IR subgroups LVM and $LVM^{2.7}$ were significantly correlated with serum leptin levels in both IS and IR subgroups while both were significantly correlated with serum insulin only in IS subgroup.

There was also significant negative correlation between LVM and $LVM/h^{2.7}$ and E/A ratio in both IS and IR obese subgroups.

Table (1) Anthropometric and metabolic parameters of obese group compared to non obese group:

	Nonobese group (n=20) mean±SD	**Obese group (n=45) mean±SD**	**P value**
Age	**32.6±9.8**	**36.16±4.6**	**>0.05**
BMI(kg/m^2)	**21.26±2.01**	**33.86±2.98**	**<0.05**
WC(cm)	**73.43±7.76**	**106.46±8.9**	**<0.05**
S. INSULIN µ U/ML	**9.5±1.85**	**20.5±12.9**	**<0.05**
S .LEPTIN ng/ml	**6.99±2.35**	**28.76±3.5**	**<0.001**
HOMA-IR	**1.24±0.26**	**3.5±0.8**	**<0.001**

Table (2) Anthropometric and metabolic parameters of obese IR group compared to IS subgroups

	IS subgroup n=20 mean±SD	IR subgroup n=25 mean ±SD	P value
Age	**34.7±8.2**	**35.6±5.2**	**>0.05**
BMI(kg/m²)	**29.233±1.72**	**36.13±3.7**	**>0.05**
WC(cm)	**101.6±10.23**	**110.7±10. 3**	**<0.05**
S. INSULIN µ U/ML	**17.08±6.8**	**24.04±8.62**	**<0.05**
S .LEPTIN ng/ml	**22.4±1.12**	**35.54±6.6**	**<0.001**
HOMA-IR	**2.9±0.87**	**4.14±1.09**	**<0.001**

Table (3) Echocardiographic parameters of obese group compared to non obese group:

		Non obese group n=20 mean ± SD	Obese group n=45 mean ± SD	P VALUE
LVM (gms)		**120.8±12.5**	**199.5±12.8**	**<0.05**
LVM/h$^{2.7}$ (gm/m$^{2.7}$)		**31.4 ± 3.2**	**50.21±2.9**	**<0.05**
Systolic function	**EF%**	**77±6**	**75±7**	**>0.05**
	FS%	**36±3**	**37±4**	**>0.05**
Diastolic function	**E/A ratio**	**1.56±0.43**	**1.02±0.7**	**<0.05**
	IVRT (m/sec)	**87±12**	**73±8**	**>0.05**
	EWD (m/sec)	**142±29**	**152±50**	**>0.05**

EF=ejection fraction SF=fractional shortening
E/A ratio=ratio between the E and A wave
IVRT=isovolumetric relaxation time of left ventricle
EWD=E wave deceleration time

Table (4) Echocardiographic parameters of obese IR group compared to IS group:

		IS subgroup n=20 mean ±SD	IR subgroup n=25 mean± SD	P VALUE
LVM (gms)		177.9±12.6	210.2±13.5	<0.05
LVM/$h^{2.7}$ ($gm/m^{2.7}$)		51.6±3.4	55.07±3.5	<0.05
Systolic function	EF%	72±7	74±5	>0.05
	FS%	36±5	38±7	>0.05
Diastolic function	E/A ratio	1.35±0.2	1.1±0.7	>0.05
	IVRT (m/sec)	71±12	70±9	>0.05
	EWD (m/sec)	146±42	160±50	>0.05

EF=ejection fraction SF=fractional shortening
E/A ratio=ratio between the E and A wave
IVRT=isovolumetric relaxation time of left ventricle
EWD=E wave deceleration time

Table (5) Univeriate correlation of BMI, WC, fasting insulin, fasting leptin and E/A ratio with LVM and LNM/$h^{2.7}$ in both IS and IR obese subgroups:

	LVM (g)				LVM/$h^{2.7}$ ($g/m^{2.7}$)			
	IS group		IR group		IS group		IR group	
	R	p	R	P	R	P	R	P
BMI (kg/m^2)	0.22	>0.01	0.23	>0.01	0.014	>0.01	0.016	>0.01
WC (cm)	0.37	>0.01	0.14	>0.01	0.16	>0.01	0.17	>0.01
S.INSLIN µU/ml	0.42	<0.00l	0.27	>0.01	0.48	<0.001	0.49	>0.0l
S.LEPTIN uU/ml	0.65	<0.001	0.56	<0.001	0.62	<0.001	0.49	<0.001
E/A ratio	0.46	<0.05	0.47	<0.05	0.42	<0.05	0.36	<0.05

Discussion

Obesity causes cardiovascular disturbances and the incidences of cardiovascular diseases are higher in obese patients than in lean subjects ***(Mohamed &Mohamed,2003***) .It is known that long standing obesity is associated with preclinical and clinical left ventricular dilatation with heart failure frequently being the ultimate cause of death in morbidly obese. ***(Marcus et al,2000)***

Systolic dysfunction and alteration of ventricular relaxation have also been described in obese subjects as compared to lean individuals ***(Crisostomo et al,2001),*** but it is now recognized that left ventricular diastolic function is an important determinate of the over all cardiac performance and clinical evidence of heart failure. In addition, impaired diastolic function and filling frequently occur well before systolic dysfunction in many cardiac disorders ***(Marcus et al,2000)***

Obesity is characterized by a great variability of insulin sensitivity degree, which could differently influence left ventricular mass. The association of insulin resistance with LV hypertrophy has been suggested, with conflicting results by several Echocardiographic reports ***(Lacobellis et al,2003)***). Nevertheless, the independent role of insulin sensitivity in inducing LV changes in obesity is not clear ***(Lacobellis et al,2003)***). Leptin plays a role in fat metabolism and correlates with insulin resistance and other markers of the metabolic syndrome, independent of total adiposity ***(Naiem & Kandil,2006).***

Recent observations suggest that the cardiovascular action of leptin may help to explain the link between excess fat mass and cardiovascular disease (***(Naiem & Kandil,2006).***).

The aim of our study was to evaluate a possible relationship between serum leptin and left ventricular mass and function in obese young females with insulin resistance.

In the present study, BMI and WC were expectably significantly higher in obese group compared to non obese group. On the other hand only WC was significantly higher in IR

compared to IS subgroups. Simpson, et al, ***(Simpson et al,2007)*** demonstrated that WC is a simple tool to diagnose insulin resistance and to identify those at greater risk for heart disease, diabetes, hypertension and dyslipidemia.

In the current study serum insulin was significantly higher in obese compared to non obese groups ($p<0.05$).Also it was signification higher in IR compared to IS subgroups.

Our results are in accordance with those found by **Kahn, et al,2006** who demonstrated that obesity is associated with increased risk of developing insulin resistance .They explained that as in obese individuals, adipose tissue release increased amount of non-estrified fatty acids, glycerol ,hormones ,pro-inflammatory cytokines and other factors that are involved in development of insulin resistance. When insulin resistance is accompanied by dysfunction of pancreatic islet-B cells, failure to control blood glucose levels results (**Kahn, et al,2006**)

In the current study, serum leptin was significantly higher in obese companied to non obese groups ($p<0.001$) and also it is significantly higher in IR compared to IS subgroups.

In agreement with our results, **Perego et al**,**2005** found that plasma serum leptin was significantly higher in obese normotensive female group compared to non obese normotensive group.

Also, **Sasson et al,1993** demonstrated that leptin levels are elevated in subjects with insulin resistance.

This may be due to the fact that insulin increases leptin production by human adipocytes in vitro ***(Naiem & Kandil,2006)***.In vivo studies in human have shown that while short term changes in plasma insulin fails to cause elevation in plasma leptin insulin concentrations, prolonged insulin infusion causes slight elevation. It is likely; therefore that the duration of exposure to hyperinsulinemia is important in sustaining a hyperleptinemic state ***(Naiem & Kandil,2006).***

HOMA-IR has been found to be a reliable reflection of insulin resistance with a good correlation with the euglycemic hyperinsulinemia glucose clamp procedure ***(Goodarzi,et al,2003).***

In our study HOMA-IR was found to be significantly higher in obese compared to non obese groups. Also it was significantly higher in IR companied to IS subgroups.

These findings are consistent with those of **Goodarzi et al, 2003**,**Uguar Atun et al**, **2005** and **Perego et al**, **2005.**

As regard both LVM and LVM/$h^{2.7}$ they were significantly higher in obese group compared to non obese groups.

Our results are in accordance with those found by **Crisotomo et al, 2001, Lacobellis et al,2003 and Wong et al,2004.**

A number of putative mechanisms may underline these morphological changes. Previously obesity was thought to cause pure eccentric left ventricular hypertrophy due to volume over load and it was assumed that concentric remodeling would only occur in the presence of hypertension. More recent studies have, however shown that relative wall thickness tends to increase along with body fat accumulation irrespective if blood pressure level***.(Karason et al,2003).***Recent studies have shown that adipose tissue is an active endocrine gland ,including a local rennin angiotensin system ***(Karisson et al,1998)) .***

Apart from expressing angiotensin peptide locally, it has been suggested that adipose tissue may contribute to circulating angiotensin II, an important regulator of blood pressure and a potent growth factor in myocardial tissue. This provides another potential mechanism by which excess body fat may directly contribute to LV hypertrophy ***(Karason et al,2003***).Another explanation is that obstructive sleep apnea is common in obese persons and may contribute to heart failure through several mechanisms, Increase in after load and wall stress associated with generation of negative intra-thoracic pressure during episodes of obstructive sleep apnea, as well as inflammatory cytokines and sympathetic activations are potential mechanisms ***(Lacobellis et al,2003).***

In our study LVM and LVM/ $h^{2.7}$ were significantly higher in IR compared to IS subgroups.

The link between insulin resistance and LV changes has been widely recognized with different results ***(Lacobellis et al,2003)***. Several studies have found an association between insulin resistance and LV hypertrophy, **Hiroyama et al, 2001 and Sandstorm et al ,2001,**whereas others have not **Galvane et al, 2000,Halmqvist et al ,2001 and Sandstorm et al,2000.** Nevertheless, the majority of these studies had selected a population of hypertensive or nondiabetic patients with a wide range of BMI ,which resulted in the main confounding

predictor of LVM ***(Lacobellis et al,2003)***. Our work differs from previous studies because of strict selection of obese subject. Our subject showed only excess fat without confounding factors on LVM such as hypertension, diabetes and dyslipidemia.Moreover, no significant differences of BMI age ,sex, lipid profile and blood pressure between IR and IS subgroups were found in our study, which pointed to the association between insulin resistance and LVM..

Several mechanisms can be involved to explain the effect of insulin resistance including an increase of LVM ***(Lacobellis et al,2003)***..The growth effect of insulin could be one of the mechanisms involved. Insulin acts directly or indirectly through the stimulation of growth factors such as insulin like growth factor 1, causing a pronounced LV hypertrophy in the rat ***(Deloughter et al,1999).***Moreover hyperinsulation can induce sodium retention, expansion of the extra cellular fluid volume, and ultimately LV hypertrophy ***(Verdeechia,et al,1999).***Insulin resistance could contribute to an increase of LVM also by enhancing kidney sodium reabsorption. The result is an extracellular volume expansion, which can leads to enlargement of LV diameters ***(Lacobellis et al,2003***).The fasting insulin levels and IR degree could also differently influence LVM. This is in agreement with our results as fasting serum insulin was significantly higher in IR compared to IS subgroups.

In the present study ,the systolic and diastolic functions were similar in obese and non obese groups except for a lower E/A ratio in obese group, Similar results were obtained by **Crisostomo et al ,2001.** The highly positive correlation between systolic blood pressure and LV mass increase , suggests that blood pressure may play a disproportionate role in the development of LV hypertrophy even in normotensive morbidly obese patients ***(Crisostomo et al,2001).*** This may be attributed to abnormal relaxation of left ventricle with increased dependence on left arterial contraction for filling ***(Mohamed & Mohamed,2003).***

In our study a positive correlation was found between both LVM and LVM/$h^{2.7}$and both serum insulin and serum leptin in IS group but as regard IR group a positive relation was found with only serum leptin and both LVM and LVM /$h^{2.7}$.

In agreement to our study **Naiem and Kandil**,**2006** had found a positive coloration between leptin and LVM/$h^{2.7}$ in obese group. **Barouch at al, 2003** has identified a novel direct link between leptin and cardiovascular remodeling.Most interestingly; exogenous administration of leptin in the primary leptin deficiency model reduces the ventricular hypertrophy very quickly. This suggests that the leptin receptors which are present in the myocardium may also have a primary remodeling effect ***(Naiem and Kandil*,*2006)***.

Many studies demonstrated that leptin is a hormone designed for periods of nutritional deficiency rather than nutritional excess. When there is a great decrease in amount of caloric intake in a host, there is a fewer fat stores and hence less leptin. The lower levels of leptin decrease the hypothalamic leptin receptor occupancy leading to a conservation of energy metabolism and an increase appetite for food. This is an important self preservation and survival strategy in insulin resistant patient there is a sitting of secondary leptin deficiency or leptin receptor resistance. This triggers a series of metabolic adaptations that are counterintuitive from nature's point of view. To protect the tissue from excessive leptin increases, the body has unregulated pathways that will lead to decrease receptor sensitivity .This therefore paradoxically produce a state of relative leptin deficiency, despite elevated leptin levels unfortunately, from the cell point of view this relative lake of leptin signaling will leads to hypertrophy ***(Barouch et al,2003).***

On the other hand, leptin has been demonstrated to induce proliferation, differentiation ad functional activation of hemopioetic and embryonic cells. Thus, it might play a role in the functional activation of the cell at the functional activation of the cell at the myocardial level also ***(Paolisso et al,1999).***

Leptin has been found to regulate many intracellular signaling pathways that are common to insulin signaling. It is known that at the hypothalamic levels insulin and leptin have overlapping effects in the control energy homeostasis ***(Perego et al,2005).***Recently it has been hypothiezed that positive cross link between insulin and leptin could play an important rule in peripheral tissue and in hyperinsulinemia associated cardiac hypertrophy.

In the current study there was also a significant negative correlation between LVM and LVM/$h^{2.7}$ and E/A ratio. Several studies have shown that LV diastolic filling becomes

progressively impaired as LV mass increase ***(Crisostomo et al ,2001).*****Zarich et al ,1991** studied 16 asymptomatic morbidly obese patients of both sexes, with age <50 years and they found that 50% of them had LV diastolic filling abnormalities. **Stoddard et al ,1992** studied 24 asymptomatic obese volunteers compared to lean control subjects and they found a prolonged of isovolumetric relaxation time in the obese group .They suggested that this index would be useful in the early detection of LV dysfunction in obesity (***(Crisostomo et al ,2001***)

Conclusion:

Obesity is a clinical syndrome associated with hyperinsulinemia, hyperleptinemia and insulin resistance .Abnormalities of LV diastolic function and mass occur frequently in obese patients. Hyperleptinemia can be an early sign for left ventricular dysfunction in obese females.

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العلاقه بين مستوى اللبتين ووظائف البطين الأيسر فى الأناث الأتى يعانين من السمنه بالأضافه الى مقاومة الأنسولين

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إن ملاحظة التلازم بين السمنة و تضخم القلب قد بدأت منذ حوالى عقدين تقريبا و إن كانت طبيعة وأسباب هذا التلازم لم تتضح بعد. كما وجد أن مستوى اللبتين فى الدم له علاقه بالسمنه و مستوى الإنسولين فى الدم. و يعتبر اللبتين عامل له علاقه بمتلازمة الأيض و مخاطر القلب.

الهدف من الدراسه:

إن الهدف من الدراسه يكمن فى تقييم العلاقه بين السمنة و كلا من مقاومة الإنسولين و مستوى اللبتين فى المصل و تضخم البطين الأيسر ووظائفه فى الإناث الأتى يعانين من السمنه بالأضافه الى مقاومة الأنسولين وقد تمت هذه الدراسه على مجموعه من الإناث ما بين 20 و45 عاما فى فتره ما قبل انقطاع الطمث و قد تم تقسيمهم الى ثلاث مجموعات : مجموعه ضابطة تضم 20 انثى ومجموعه حساسه للانسولين تضم 20 انثى و مجموعه مقاومه للانسولين وتضم 25 انثى وقد تم فحص وظيفة القلب بالترددات الصوتية لكل الإناث المشاركات بالدراسه.وقد أسفرت هذه الدراسه عن زيادة محيط الخصر و مستوى كلا من الإنسولين و اللبتين و هوما أر فى الأناث الأتى يعانين من السمنه بالمقارنة بالمجموعة الضابطة كما كانت اعلى فى المجموعة المقاومة بالمقارنة بالمجموعه الحساسه للانسولين . كما وجد ان هناك تاثير سلبى للسمنة على وظيفة و حجم البطين الأيسر فى الأناث الأتى يعانين من السمنه بالمقارنة بالمجموعة الضابطة كما كانت اكثر سوئا فى المجموعة المقاومة بالمقارنة بالمجموعه الحساسه للانسولين.

تعتبر السمنة متلازمة سريرية مصحوبة بزيادة فى نسبة مقاومة الإنسولين و مستوى اللبتين فى المصل و تضخم البطين الأيسر واضطراب وظائفه. يمكن اعتبار ارتفاع مستوى اللبتين كعلامة مبكرة لكشف اضطراب وظائف و تضخم البطين الأيسرفى الأناث الأتى يعانين من السمنه.