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A STUDY OF PREVALENCE OF METABOLIC SYNDROME IN THE MIDDLE AGED WOMEN OF GUNTUR CITY, A.P

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ABSTRACT

Introduction: Metabolic Syndrome (MetS) has received lot of attention because of it's growing association with obesity and atherosclerotic cardiovascular disease among women population. Central obesity, a physical finding plays a central role in the development of the Insulin Resistance (MetS). Central obesity appears to precede the other components of MetS, which increases with age. Women in this coastal belt of Andhra Pradesh prepare and consume delicious food items, mostly of rice and have tendency to become obese in forties. High carbohydrate intake and sedentary lifestyle with low physical activity are important known causes for development of MetS in adult women along with several considerations which are unique to women are pregnancy, use of oral contraceptives, and emotional stress.

Aim of the present study: The present study is undertaken among female population between the ages of 40-45 years in Guntur city and its surroundings in the state of Andhra Pradesh, India with an aim to identify the Metabolic Syndrome and to study it's prevalence with the use of measurement of following parameters 1.Physical a) Waist Circumference, b) Blood Pressure, 2. Biochemical a) Total Triglycerides, b) High Density Lipoprotein Cholesterol (HDL-C) and c) Glucose using fasting plasma sample.

Materials & Methods: This study was conducted in 112 women, out of which 61 were test group (obese i.e. with increased Waist circumference) and 51 were control group (non-obese i.e. normal waist circumference). Waist measurement is taken as a preliminary screening tool for differentiating test group and control group. Waist circumference was obtained at the level of the umbilicus using a plastic anthropometric tape. Systolic and diastolic blood pressures were obtained with a mercury sphygmomanometer by auscultatory method. Fasting plasma sample used for measurements of Glucose, Total Triglycerides, Total Cholesterol and HDL-cholesterol by using VITROS Dry Technology slide and DT 60 II chemistry systems.

Results: The overall risk analysis for Metabolic Syndrome in the 61 women test group, 36 are with 3 or more risk factors who are considered of having Metabolic Syndrome (MetS). So the prevalence for metabolic syndrome among women population with increased waist circumference of 40-45years is 59.02%. But in the 51 non-obese women control group, none with 3 or more risk factors were detected.

Conclusion: In this present study It is observed that central obesity, elevated fasting plasma triglycerides and decreased HDL-cholesterol are the prevalent components in test group with Metabolic Syndrome. Current study of detection and prevalence of MetS is very useful for prevention of chronic diseases in aged women. MetS can lead to diseases like Type II Diabetes mellitus (T2DM) and CVD in the aged. As the detection of MetS is possible in about 15 to 20 years before development of T2DM, CVD and their consequences, it is be possible to preserve health of aged women population.

INTRODUCTION

Metabolic syndrome(MetS) is a cluster of factors like central obesity, dyslipidemia, hyperglycemia and hypertension is known to pronounce risk for future development of type 2 diabetes mellitus and cardiovascular diseases (Sattar *et al.,* 2003) . Today cardiovascular disease is one of the main causes of mortality of women in the world (Lloyd-Jones *et al.,* 2009). Under current guidelines, revised in 2005 by the National Heart, Lung, and Blood Institute (NHLBI) and the American Heart Association (AHA) metabolic syndrome is diagnosed when an individual has at least 3 out of 5 conditions (Grundy *et al.,* 2005).

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After that The International Diabetes Federation (IDF) (Alberti *et al.,* 2006) consensus worldwide definition of the metabolic syndrome (2006) is: Central obesity (defined as waist circumference with ethnicity-specific values that is For south Asian, ≥90 cm in men or ≥80 cm in women) and any two of the following:

- Raised triglycerides: > 150 mg/dL, or specific treatment for this lipid abnormality.
- Reduced HDL-C: < 40 mg/dL in men, < 50 mg/dL in women, or specific treatment for this lipid abnormality.
- Raised blood pressure (BP): systolic BP > 130 or diastolic BP >85 mm Hg, or treatment of previously diagnosed hypertension.
- Raised fasting plasma glucose (FPG): >100 mg/dL, or previously diagnosed T2DM.

People with MetS have a fivefold greater risk of developingT2DM (Stern *et al.,* 2004). In MetS the clustering of metabolic abnormalities that occur in the same individual appear to confer a substantial additional cardiovascular risk over and above the sum of the risk associated with each abnormality (Sattar *et al.,* 2003; Golden *et al.,* 2002). The MetS is estimated to affect approximately 20–30% of the middle-aged population (Park et al., 2003), and prevalence appears to be increasing in the U.S. population with increasing obesity and sedentary lifestyle (Meigs, 2002) .r The risk of CVD attributed to the metabolic syndrome appears to be especially high in women, and it is estimated that half of all cardiovascular events in women are related to the metabolic syndrome (Wilson *et al.,* 1999). In the National Health and Nutrition Examination Survey (NHANES) 1988–1994 cohort, the prevalence of the MetS was lower in women than men (23.9 vs.27.8% [n : 5775]), whereas the prevalence was higher in women than men in the later 1999–2002 cohort (30.3 vs.28.0% [n : 1514]) (Ford, 2008). The psychosocial factors, including depression, can activate the HPA (Hypothalamo-pituitary-adrenal) axis producing hyper secretion of corticotrophin-releasing hormone, adrenocorticotropic hormone, and cortisol. This dysregulation of the HPA axis promotes fat deposition in visceral adipose tissue (Bjo¨ rntorp, 1991). In middle aged populations, depressive symptoms appear to be associated with an increased risk of the MetS more in women than in men (Raikkonen *et al.,* 2002; Raikkonen *et al.,* 2007) and higher morbidity among females from the MetS has been reported(14). Although in most existing research the assumption has been that depression predicts the MetS.

Predictive value for diabetes

Because glucose intolerance is embedded in the cluster of conditions, and such intolerance is a strong predictor of T2DM (Edelstein *et al.,* 1997), this association is conceivable. Also, central obesity, a component of the MetS, has been well characterized as enhancing the incidence of T2DM (Kaye *et al.,* 1991; Despre´ s, 1989) in women.

Predictive value for cardiovascular disease

Central obesity

Central obesity has been shown to increase the risk of Coronary Heart Disease (CHD) on its own (Lapidus et al., 1984; Clin Res Ed, 1984). This has been shown in both cross-sectional and longitudinal studies (Rexrode *et al.,* 1998; Yusuf *et al.,* 2004; Kim *et al.,* 2004; St-Pierre *et al.,* 2002). The IDF, by describing ethnic-specific central obesity measures, emphasizes that the risk of an expanded waist circumference varies among different ethnic groups, so that in many parts of the world have a lower cutoff of 35 inches (90cm) for men and 32 inches (80cm) for women than the NCEP cut off of 40 inches for men and 35 inches for women. A consensus conference held in Asia in 2000 strongly recommended a lower cut off of waist circumference given by IDF (World Health Organization, 2000), citing adequate evidence.

Hyperglycemia

Hyperglycemia is a continuously ascending independent risk factor for CHD, with no apparent threshold (Coutinho *et al.,* 1999; Gerich, 2003).

High blood pressure

High blood pressure is associated with an increased risk of CHD (Basile, 2003; Franklin *et al.,* 2001; Vasan *et al.,* 2001).Thomas and colleagues (Thomas *et al.,* 2005) reported in a 14-year study that hypertension was the most important risk factor associated with increased cardiovascular mortality among overweight and obese persons who have the MetS. A Japanese study also reported that hypertension was the strongest risk factor for the development of carotid plaque (Ishizaka *et al.,* 2005).

Dyslipidemia

The MetS is characterized by the development of a highly atherogenic lipid profile known as atherogenic dyslipidemia (NCEP, 2001; Ruotolo and Howard, 2002; Brunzell and Hokanson, 1999). This is characterized by hypertriglyceridemia, low HDL-cholesterol, increased numbers of small, dense, triglyceride-enriched low density lipoprotein (LDL) particles, increased remnant lipoproteins, and elevated apolipoprotein B concentrations (NCEP, 2001; Ruotolo and Howard, 2002). Because the LDL particles are rather low in cholesterol, LDL-C levels are often normal (Garvey *et al.,* 2003). Even though, free fatty acids (FFA) are not usually measured clinically because of their short half-life and the difficulty of assay, FFA plays an important role in the pathophysiology of the MetS. The products of free fatty acid oxidation can inhibit glucose transport activity. FFA are elevated in central obesity, leading to insulin resistance (Jensen *et al.,* 1989). The liver is central organ for the dyslipidemia and insulin resistance, because it remains more insulin sensitive and increases synthesis of fatty acids and triglyceride-rich very-low density lipoprotein (VLDL) particles (Ginsberg and Huang, 2000). This results in fasting hypertriglyceridemia, greater postprandial hyperlipemia, and elevations in triglyceride-rich remnant lipoproteins. All of these are associated with increased CVD risk. Numerous studies have documented a relationship of the MetS diagnosed by one of the MetS clusters and CHD (Isomaa *et al.,* 2001; Onat *et al.,* 2002; Kekalainen *et al.,* 1999; Alexander *et al.,* 2003; Malik *et al.,* 2004; Scuteri *et al.,* 2005). Although the absolute CVD risk associated with the MetS in youth is low it

has been shown that clusters of risk factors track into adulthood even more than single risk factors (Bao *et al.,* 1994; Chen *et al.,* 2007), suggesting that early identification may be beneficial in the amelioration of the diseases associated with this syndrome.

Aim of the present study

Metabolic syndrome is wide spread among women population in the world and increases with age. It can be detected with the help of measurement of following parameters:

1. Waist Circumference (WC),
2. Blood Pressure (BP),
3. Fasting Total Triglycerides (TTG),
4. High Density Lipoprotein Cholesterol (HDL-C) and
5. Fasting Plasma Glucose.

The present study is undertaken among female population between the ages of 40-45 years in Guntur City and surroundings to identify the MetS and risk of insulin resistance.

MATERIALS AND METHODS

There are currently two major definitions for metabolic syndrome and these are provided by the International Diabetes Federation (Alberti *et al.,* 2005; Campbell *et al.,* 2009) and the revised National Cholesterol Education Program (46), for the diagnosis of the MetS although the waist measurement is taken as a useful preliminary screening tool for differentiating control group from test group.

Control Group (With normal WC)

Women population who are healthy of age between 40 to 45 years with normal waist circumference (below 80cm), without hypertension, diabetes or any clinical abnormality of Guntur city were selected.

Test Group (With increased WC)

Women population of age between 40 to 45 years, with increased waist circumference ($\geq$ 80 cm) were selected as test group.

For all women both control and test groups the following data is recorded after taking consent from them.

Physical Parameters

a) Waist circumference (WC) was obtained at the level of the umbilicus with a plastic anthropometric tape.
b) Systolic and diastolic blood pressures were obtained with a mercury sphygmomanometer by auscultatory method following the American Heart Association protocol (Perloff *et al.,* 2001).

Chemical parameters

Fasting Blood samples were collected by venipuncture into heparinised bottle from each woman for the analysis of following;

a) Plasma Glucose
b) Lipid profile: i. Total Triglycerides, ii. Total Cholesterol, iii. HDL Cholesterol.

Estimation of Glucose

Principle and procedure

A drop (10µl) of test sample is deposited on the slide and evenly distributed by the spreading layer to underlying layers. After 5 min incubation oxidation of sample glucose occurred which is catalyzed by glucose oxidase to form hydrogen peroxide and gluconic acid. This reaction is followed by an oxidative coupling. The intensity of dye is measured by reflectance Spectrophotometry at 555nm.

Reaction sequence

$$\beta\text{-D Glucose} + O_2 + H_2O \xrightarrow{\text{Glucose oxidase}} \text{D-Gluconic acid} + H_2O_2$$

$$2H_2O_2 + \text{4-Amino antipyrine} + \text{1,7dihydroxynaphthalene} \longrightarrow \text{Red dye}$$

Reportable range for Glucose in method is: 20 – 450 mg / dl
Reference Normal interval for Glucose
Fasting Plasma Glucose level: 60 -100mg/dl

Estimation of Triglycerides

Principle and procedure

Estimation of triglycerides is performed using the VITROS Dry Technology slide and DT 60 II chemistry systems. VITROS TRIG DT slide is multilayered analytical element coated on a polyester support. The analysis is based on an enzymatic method. A drop (10µl) of test sample is deposited on the slide and evenly distributed by spreading layer to the underlying layers. The Triton X-100 surfactant in the spreading layer aids in dissociating the triglycerides from lipoprotein complexes present in the sample.

Reaction sequence

$$\text{Lipoproteins} \xrightarrow{\text{surfactant}} \text{triglycerides} + \text{proteins}$$

$$\text{Triglycerides} + H_2O \xrightarrow{\text{lipase}} \text{glycerol} + \text{fatty acids}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow[\text{glycerol kinase}]{Mgcl_2,} \text{L-}\alpha\text{ - glycerophosphate} + \text{ADP}$$

$$\text{L-}\alpha\text{ - glycerophosphate} + O2 \xrightarrow[\text{oxidase}]{\text{L}-\alpha-\text{glycerophosphate}} \text{DHAP} + H_2O_2$$

$$H_2O_2 + \text{leucodye} \xrightarrow{\text{peroxidase}} \text{dye} + 2H_2O$$

The density of dye formed is proportional to the triglyceride concentration present in the sample and is measured by reflectance spectrophotometer at 555nm after incubation of 5 min.

Table 3. Showing data of significance with P-Values

	PARAMETER	Waist circum-ference (WC) cms	Systolic B.P (SBP) mmHg	Diastolic B.P (DBP) mmHg	Fasting Plasma Glucose (FPG) mg%	Total triglycerides (TTG) mg%	Total cholesterol (TCH) mg%	HDL-cholesterol (HDL-C) mg%	VLDL-cholesterol (VLDL-C) mg%	LDL-cholesterol (LDL-C) mg%
CONTROL GROUP	Average	75.882	114.9	75.88	82.98	137.69	179.76	49.5294	27.45098	101.451
	SD±	3.2351	7.035	5.07	8.758	39.149	19.499	4.4558	7.728684	21.14929
OBESE GROUP	Average	87.361	118.48	76.3	93.13	163.28	202.11	42.23	32.7	127.18
	SD±	6.0856	10.698	6.581	8.912	76.417	29.856	5.343	15.31	22.499
	P Value	Highly signific-ant	Signific-ant	Not significa-nt	Highly significa-nt	Significant	Highly significant	Highly significant	Significant	Highly significant

Reportable dynamic range for Triglycerides:

15 – 400 mg / dl

Normal fasting reference interval for Triglycerides:

50 - 150mg / dl

Estimation of Cholesterol

Principle and procedure

A drop (10µl) of test sample is deposited on the slide and evenly distributed by the spreading layer to underlying layers. The Triton X-100 (TX 100) surfactant in the spreading layers aids in dissociating the cholesterol and cholesterol esters from lipoprotein complexes present in sample.

Reaction sequence

$$\text{Lipoprotein} \xrightarrow{TX\ 100} \text{cholesterol + cholesterol esters + proteins}$$

$$\text{Cholesterol esters + } H_2O \xrightarrow[\text{hydrolase}]{\text{cholesterol ester}} \text{cholesterol + fatty acids}$$

$$\text{Cholesterol + } O_2 \xrightarrow[\text{oxidase}]{\text{cholesterol}} \text{cholest-4-en3one + } H_2O_2$$

$$H_2O_2 + \text{leucodye} \xrightarrow{\text{peroxidase}} \text{dye + } 2H_2O$$

The density of dye formed is proportional to cholesterol concentration present in the sample and is measured by reflectance spectrophotometry at 55onm after incubation of 5 min.

Reportable range: 50 – 325mg / dl
Reference interval: Desirable <200 mg / dl
Border line high 200 – 239 mg / dl, High >240 mg / dl.

Estimation of HDL Cholesterol

Principle and procedure

HDL separated by precipitation of LDL and VLDL using dextrin sulphate and magnesium chloride provided in VITRO DT micro HDL tool. The HDL lipoproteins remain in the liquid portion of the tube after Centrifugation. The liquid portion is called the supernatant and is the portion analyzed. The non-HDL fractions form a pellet on the bottom of the tube and are discarded. A drop (10µl) of pretreated sample is deposited on the slide and is evenly distributed by spreading layer to underlying layers. The Triton X-100 (TX 100) surfactant in the spreading layers aids in dissociating the cholesterol and cholesterol esters from lipoprotein complexes present in the sample.

Reaction sequence

$$\text{HDL} \xrightarrow{\text{surfactant}} \text{cholesterol} + \text{cholesterol esters} + \text{proteins}$$

$$\text{Cholesterol esters} + H_2O \xrightarrow[\text{hydrolase}]{\text{cholesterol esters}} \text{cholesterol} + \text{fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow[\text{oxidase}]{\text{cholesterol}} \text{cholest-4-en-3-one} + H_2O_2$$

$$H_2O_2 + \text{leucodye} \xrightarrow{\text{peroxidase}} \text{dye} + 2H_2O$$

The density of dye formed is proportional to HDL -cholesterol concentration present in pretreated sample and is measured by reflectance spectrophotometry at 660nm after 5 min incubation.

Normal reference interval for HDL

males ≥40 mg/dl, females ≥50 mg/dl

RESULTS

The results of Waist Circumference (WC) in control group average is 75.88 with S.D. of ±3.24 compared to the results of WC in test group average is 87.36 with S.D. of ±6.09. The increase in WC in test group women is highly Significant ($p < 0.001$). The results of Systolic Blood Pressure (SBP) in control group women average is 114.90 with S.D. of ±7.03 compared to the results of SBP in test group women average is 118.48with S.D. of ±10.70 The increase in SBP in test group is Significant ($p < 0.05$) and the results of Diastolic Blood Pressure (DBP) in control group average is 75.88 with S.D. of ±5.07 compared to the results of DBP in test group average is 76.3 with S.D of ±6.58. The increase in DBP in test group is not Significant ($p > 0.1$). The results of Fasting Plasma Glucose(FPG) in control group average is 82.98 with S.D. of ±8.76 compared to the results of FPG in test group average is 93.13 with S.D. of ±.8.91 The increase in FPG in test group is highly Significant ($p < 0.001$).

The results of Total Triglycerides (TTG) in control group average is 137.69 with S.D. of ±39.15 compared to the results of TTG in test group average is 163.28 with S.D. of ±76.42. The increase in TTG in test group is Significant ($p <0.05$).The results of HDL cholesterol (HDL-C) in control group average is 49.53 with S.D. of ±4.46 compared to the results of HDLC in test group average is 42.23 with S.D. of ±5.34. The decrease in HDL-C in test group is highly Significant ($p < 0.001$). The overall risk analysis for Metabolic Syndrome in the 61 (sixty one) Obese women test group, 36 (thirty six) are with 3 or more risk factors who are considered of having Metabolic Syndrome. So the prevalence for metabolic syndrome among women population of 40-45years is 59.02%. But risk analysis for Metabolic Syndrome in the 51 (fifty one) non-obese women Control group, No single case metabolic syndrome with 3 risk factors was detected.

DISCUSSION

In this present study among women population of 40-45 years the prevalence metabolic syndrome is increased to 59.02% with increasing waist circumference when compared with contemporary age group of controls. The reason for increased waist circumference in India the genetic tendency of Asians to abdominal obesity (Yusuf *et al.,* 2001), in addition, high carbohydrate intake and sedentary lifestyle with low physical activity among adult population might be contributing factors (Sarrafzadegan et al., 2008; Kelishadi *et al.,* 2008).

Central obesity

Asians have an ethnic predisposition to adverse body fat distribution and metabolic syndrome (Yusuf *et al.,* 2001), hence, optimal cut off points for waist circumference have been established for South Asians (Alberti *et al.,* 2005), by using this cut off, the prevalence of the metabolic syndrome in this population is estimated to be 10-30% (Nestel *et al.,* 2007). Central obesity plays a central role in the development of the MetS and appears to precede the appearance of the other MetS components (Cameron Adrian *et al.,* 2008). In our present study we observe that central obesity and reduced HDL-cholesterol as the prevalent components of the Mets.

Many population-based studies of the Middle East (Khader *et al.,* 2007; Al-Lawati *et al.,* 2003; Azizi *et al.,* 2003, Sarrafzadegan *et al.,* 2008) as well as the current study, low HDL cholesterol followed by abdominal obesity has been the most common component of the MetS. In women, pregnancy-related circumstances must increased waist circumference increase with the number of pregnancies (Dennis *et al.,* 2005). Several considerations are unique to women with metabolic syndrome, including pregnancy, use of oral contraceptives, and polycystic ovarian syndrome (Entley-Lewis *et al.,* 2007). In the United States, MetS has a high prevalence in African Americans, particularly African American women, and this has been attributed to the higher prevalence of obesity, hypertension, and diabetes in this population (Clark *et al.,* 2007).

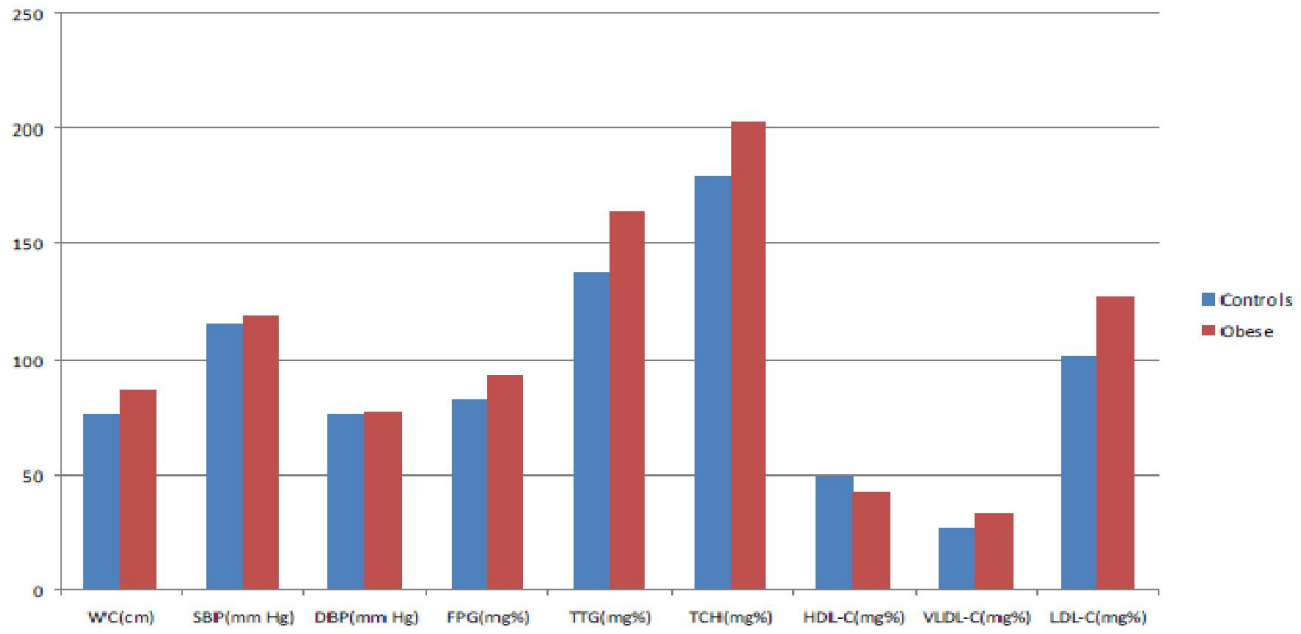

Figure 1. Bar diagram comparing averages of control group and obese group

In a cross-sectional study conducted in 1998-1999 of 200 female Puerto Ricans living in Connecticut, it was found that obesity was prevalent (Fitzgerald *et al.,* 2006).

Insulin Resistance

MetS is a detectable condition of insulin resistance. Insulin resistance is an underlying feature of the MetS (Grundy, 1999). In our present study also central obesity is significantly associated with impaired glucose condition which determines the insulin resistance condition. Proposed mechanisms of insulin resistance are centered on 3 themes: 1. Effects of mild to moderate hyperglycemia, 2. Effects of compensatory hyperinsulinemia, and 3. Effects of unbalanced pathways of insulin action. Hyperglycemia, largely postprandial and below diabetic levels, may lead to a variety of effects usually associated with T2DM. A more important mechanism may be compensatory hyperinsulinemia. The maintenance of normal postprandial glucose homeostasis requires that the pancreatic beta cells secrete a normal amount of insulin in response to the hyperglycaemic challenge and that the resultant hyperinsulinemia (Sattar *et al.,* 2003) stimulates glucose uptake by muscle, the tissue responsible for the disposal of 80% to 90% of the ingested glucose load, and (Lloyd-Jones *et al.,* 2009) suppresses endogenous glucose production, over 80% of which is derived from the liver.

In insulin-resistant conditions, the ability of insulin to augment glucose uptake and inhibit hepatic glucose production is impaired. Furthermore, adipocyte resistance to the antilipolytic effect of insulin and the consequent elevated plasma free fatty acid levels may play an important role in the development of insulin resistance in muscle and other target tissues. Dysfunctional energy storage to be the fundamental issue. The key abnormality is simply the presence of too much triglyceride, or body fat—that is, obesity. ―overflow hypothesis. Visceral adiposity can be measured by waist circumference, waist-hip ratio, or radiographic scans, and it correlates well with insulin resistance and other features of MetS. Excess triglyceride in myocytes and in abnormally large peripheral adipocytes appears to engender insulin resistance in these cells (Kelley and Mandarino, 2000). Insulin sensitivity in normal individuals varies by approximately 3-fold (Reaven, 1988). In insulin-resistant individuals, the pancreatic beta cells must secrete more insulin to maintain euglycemia. However, prolonged hyperfunction can lead to beta cell exhaustion. Insulin resistance appears to be the primary mediator of MetS (Lann and LeRoith, 2007). Metabolic syndrome is increasing in prevalence, paralleling an increasing epidemic of obesity. Dysfunctional adipose tissue also plays an important role in the pathogenesis of obesity-related insulin resistance (Goossens, 2008). MetS is a high-risk predictor of future T2DM. In the San Antonio Heart Study, the incidence of new-onset diabetes was double in persons with MetS than in those without the ATP III criteria of MetS at baseline (Lorenzo *et al.,* 2003). The worldwide increase in the prevalence of obesity in the recent decades is starting and is likely a cause of the rising incidence of insulin resistance and the MetS (Reaven, 1988; Ginsberg, 2000; Grundy, 2005), as well as CVD and T2DM (Ginsberg, 2000).

Dyslipidemia

The dyslipidemia of metabolic syndrome is characterized by elevated triglycerides (VLDL), low HDL, and small dense LDL, a triad that has been termed the atherogenic lipoprotein phenotype (Ginsberg, 2000). The major consequences of CETP (cholesteryl ester transfer protein) activity in MetS relate to triglyceride enrichment of HDL and LDL. When both of these lipoproteins are enriched with triglycerides, these are subjected to lipolysis by hepatic lipase. Upon lipolysis, the HDL and LDL become smaller. The lipolyzed HDL are cleared more rapidly from the circulation, resulting in reduced HDL and its apolipoprotein AI (apo-AI) concentrations. Higher mean TG and lower HDL-C values are seen in women with the MetS.

This pattern of lipid abnormality was documented in a recent Nigerian study (Wahab *et al.,* 2008). Our present study shows, Low HDL cholesterol is highly significant than triglyceride levels in association with central adiposity. Women with MetS having similar findings of elevated triglyceride and/or low HDL-C levels (Bittner, 2005). Such levels of HDL cholesterol and triglycerides appear to be more closely related to CHD risk among women (Brunner *et al.,* 1987; Bass *et al.,* 1993). Abnormal lipoproteins predict not only incident CHD among previously healthy women, but also recurrent events among those with prevalent CHD (Kannel, 1995; Shlipak *et al.,* 2003). The prevalence among African-American women was reported to be even higher still (Ford *et al.,* 2002; Steinbaum, 2004).

Hypertension

The kidneys play the key role in controlling blood pressure, notably through pressure natriuresis (Hall *et al*., 1980). Angiotensin and other neurohormonal mediators of hypertension have effects on intrarenal hemodynamics and renal electrolyte handling, and these factors determine long-term blood pressure control. Increased adipocyte mass leads to increased angiotensinogen production in adipose tissue, providing a potential mechanism for increased blood Pressure (Egan *et al., 2001).* Insulin resistance might also increase blood pressure via reduced nitric oxide– mediated vasodilatation, increased salt sensitivity or plasma volume expansion. Regular physical activity reduces obesity, increases HDL cholesterol, and decreases triglycerides (National Heart, Lung, and Blood Institute, 2001).

Summary and Conclusion

Cardiovascular disease remains the most widespread health Problem worldwide. Recently, Metabolic Syndrome has received lot of attention because of the growing prevalence of obesity and its association with atherosclerotic CVD among women population. Many longitudinal studies have confirmed that the Metabolic Syndrome is the risk factor for the subsequent development of CVD morbidity and mortality particularly in women. Metabolic Syndrome is a condition, which can be identified about two decades earlier than developing into CVD and by taking appropriate Lifestyle measures, the future development of T2DM and CVD may be prevented.

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