Central obesity and prevalence of metabolic syndrome in post-menopausal women

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Illustration V  
Illustration I
Central obesity and prevalence of metabolic syndrome in post-menopausal women

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Abstract

The study was aimed to determine the prevalence of metabolic syndrome and its components in the pre and post menopausal rural and urban women.

Study Design: The study included 100 postmenopausal women in the age range of 45-60 years selected from the rural and urban population equally. An equal number of premenopausal women in the age range of 25-40 years were also selected for comparison. The adult treatment panel 3 (ATP3) criteria was used to classify subjects as having metabolic syndrome.

Results: A borderline high BMI, higher waist to hip ratio, higher systolic and diastolic blood pressure, higher fasting blood glucose and dyslipidemia were observed in the postmenopausal group as compared to premenopausal counterparts. 68% of the rural and 74% of the urban post menopausal subjects were having >88 cm of waist circumference. Abdominal obesity was also observed in 27% of the rural and 31% of the urban premenopausal subjects. The prevalence of metabolic syndrome was found to be higher in postmenopausal subjects. In the rural and urban groups it was 41% and 43% respectively in comparison to 20% and 27% of the premenopausal subjects.

Conclusion: Abdominal obesity plays a central role in connecting the metabolic syndrome with the metabolic alterations of menopause and can be a strong predictor of impending metabolic syndrome.

Introduction

Women are protected from Ischemic Heart Disease (IHD) in comparison to men due to the anti-atherogenic effect of oestrogen released from the ovaries while the regular menstruation is maintained. The risk of cardiovascular diseases increases with the onset of menopause. The metabolic syndrome (MS), a cluster of risk factors including obesity, glucose intolerance, dyslipidemia, and hypertension that increase the risk for cardiovascular disease and type 2 diabetes mellitus [1,2], is more prevalent in men than the age matched premenopausal women [3,4,5]. However, after menopause the prevalence is markedly increased among women than men [6], particularly over the age of 60 [7, 8]. Many cross-sectional studies have shown an increased risk of metabolic syndrome in postmenopausal women which varies from 32.6% to 41.5% [9, 10]. Changing hormonal milieu with decreasing oestrogen and alteration of its ratio with testosterone has been implicated as a causal factor for the emergence of MS at menopausal transition [11, 12]. Besides menopausal hormonal changes, aging also plays a role in clustering of cardio-vascular risk factors [13] however, various studies have reported the association of postmenopausal status independent of normal aging with an increased risk of the MS [14,15]. The relative importance of factors that influence cardiovascular risk in postmenopausal women are unknown. Alterations in lipid metabolism with oestrogen deficiency are thought to be a substantial component of CVD risk in postmenopausal women [16], but there are also direct effects of oestrogen deficiency on body fat distribution (central obesity), insulin action, the arterial wall, and fibrinolysis that may influence cardiovascular risk. These factors contribute to an increased prevalence of the metabolic syndrome in postmenopausal women compared with premenopausal women [17] and this postmenopausal worsening of the metabolic profile may contribute to the future risk of CVD.

Thus, identification of postmenopausal women at high risk for MS has important implications for the reduction of CVD burden.

The study was aimed to
1. Determine the prevalence of metabolic syndrome and its components
2. Assess the complications associated with MS and
3. Assess the impact of life style and urbanization on prevalence of metabolic syndrome in the pre and post menopausal women.

Methods
Menopause was defined as at least 12 consecutive months of amenorrhea with no other medical cause. The premenopausal women were regularly menstruating, non-pregnant, and non-lactating with no use of hormonal contraception for at least 1 year. Women who were amenorrheic due to hysterectomy or cessation of periods other than by a natural cause were identified and excluded from the study. The proven cases of secondary hypertension, pregnant women and those with diabetes mellitus, ischemic heart disease, liver disease, gastro intestinal disorders, renal disease or any other acute or chronic disease, were also excluded from the study.

The women mostly were visited in the outpatient department of the general hospital because of hot flashes, mood swing, vaginal dryness, sleep disturbances, night sweat, forgetfulness, urinary symptoms, pain with intercourse, palpitations, anxiety, joint and muscle pain, depression and irritability.

A questionnaire was completed for each patient including demographic information, menopausal status, medical history, reproductive history, drug history, family history, physical examination and clinical laboratory data. An Informed consent was obtained from each participant.

Weight (kg) to the nearest 0.2 kg was measured with a calibrated (ADD weighing) scale. The height (in meters) of the subjects was determined with a stadiometer to the nearest 0.5 cm. The BMI was calculated as the weight (kg) divided by the height (m) squared (kg/m²).

Using a flexible metric tape the waist circumference (in centimetres) was assessed at a point midway between the lowest rib and the iliac crest with the subject standing. Waist-to-hip ratio (WHR) was calculated by waist circumference divided by hip circumference.

The Blood Pressure (BP) of each participant was measured, using the auscultatory method with a standardized calibrated mercury column-type sphygmomanometer and a BP above 130/90 mm Hg was regarded as hypertension.

Biochemical Analyses
All blood specimens were drawn at 8:00 a.m. after a 12-h fast. Samples were centrifuged within 1 hour and the sera frozen immediately at −20°C. Fasting plasma glucose was determined by the glucose oxidase method (Boehringer Mannheim, Mannheim, Germany). Serum lipid and lipoprotein cholesterol levels were measured in fresh serum samples. Serum total cholesterol and triglyceride levels were determined enzymatically (Boehringer Mannheim). Serum HDL cholesterol level was determined enzymatically after precipitation of LDLs and VLDLs with dextran sulphate MgCl₂ [18].

In all the patients besides blood biochemistry, 12 lead ECG was also performed along with complete clinical examination of the patient. A detailed case record was prepared for each patient on a preformed study sheet. Comparison of groups was performed using student’s t test. Data was presented as Mean ±S.D.

Weight status:
Underweight was defined as a BMI< 18.5 kg/m², normal BMI as >18.5-24.9 kg/m², overweight as BMI between 25-29.9 kg/m², obese as BMI>30-39.9 and BMI > 409 kg/m² was considered as extreme obesity [19].

Metabolic syndrome definition:
Postmenopausal women were considered to have metabolic syndrome if they had any three or more of the following criteria, according to the NCEP: ATP III criteria [20]:
1. Central obesity: Waist circumference >88 cm
2. Hypertriglyceridemia: Triglycerides ±150 mg/dL or specific medication
3. Low HDL cholesterol: < 50 mg/dL or specific medication
4. Hypertension: Blood pressure ±130 mm systolic or ±85 mm diastolic or specific medication
5. Fasting plasma glucose ±110 mg/dL or specific medication or previously diagnosed type 2 diabetes.

Results
The study subjects were distributed in to 2 main groups (I and II) and subgroups (A and B) according to their menstrual and rural or urban status respectively [Illustration-I].

The base line characteristics of the study subjects (premenopausal and post menopausal- rural and urban combined) are shown in Illustration-II

The mean age of the postmenopausal subjects was 57.25±0.80 years as compared to 34.48±0.74 years of that of pre menopausal subjects. BMI (Body mass index) was found to be higher in the post menopausal (Illustration-II) group as compared to premenopausal. Statistically insignificant variations of BMI were also observed between rural and urban groups of both premenopausal and postmenopausal subjects (Illustration-III).

A similar trend was observed in the waist to hip ratio (WHR) (Illustration-II), WHR value in total (rural and
urban combined) postmenopausal women and premenopausal was 0.91±0.08 and 0.079±0.05 respectively. The difference between the values was highly significant (p< 0.001).

Systolic and diastolic blood pressure, fasting blood glucose and lipid parameters were found to be higher in the postmenopausal group as compared to premenopausal counterparts. The difference between each of the two values was highly significant in all the parameters (p< 0.001), except diastolic blood pressure where statistically significant difference (p< 0.01) was observed.

Illustration-III shows the base line characteristics of study subjects in different groups (Rural and urban)
In each of the assessments the urban subjects were having higher levels than their age matched rural subjects (Illustration-III) but the difference was insignificant statistically.

The prevalence of individual components of metabolic syndrome as per NCEP: ATPIII criteria has been highlighted in Illustration-IV.

Discussion

In our study, the prevalence of metabolic syndrome was found to be higher in postmenopausal subjects. In the rural and urban groups it was 41% and 43% respectively in comparison to 20% and 27% of the premenopausal subjects (Illustration-V).

Our findings were consistent with many of previous studies [21-25], where post-menopausal women were found to be at higher risk of MS than pre-menopausal women. There was a disagreement between our study and some other studies done in Iran, western India, Argentina and Ecuador with a prevalence of 69%, 55%, 22% and 41.5% respectively [26-29]. These differences in prevalence of metabolic syndrome in different studies might be due to different investigation criteria of the syndrome, socioeconomic and environmental differences, genetic factors and lifestyle.

In our study BMI although higher in the post menopausal subjects ( Illustration-II) was not suggestive of general obesity but the waist circumference and waist to hip ratio were more conclusive of prevalence of central obesity amongst post menopausal subjects (Illustration-III and IV). Normal BMI in the presence of central obesity in post menopausal women has been reported by many studies. Menopause is believed to be associated with weight gain, most studies do not reveal increases in BMI independent of normal aging [30, 31]. It is estimated that middle-aged women gain approximately 0.55 kg (-1 lb)/yr, there does not appear to be an independent effect of menopause on body weight. However, even in the absence of weight gain, body fat distribution changes across the menopause [32, 33].

68% of the rural and 74% of the urban post menopausal subjects were having>88 cm of waist circumference. Cross-sectional [34] and longitudinal studies [35] have shown that the menopausal transition is associated with a preferential increase in abdominal adiposity, independent of the effect of age and total body adiposity. Visceral fat accumulation is thought by many to be the major determinant of the metabolic syndrome. Women with high amounts of visceral fat have an excess of cardiovascular mortality and associated metabolic abnormalities. High waist to hip ratio has also been reported to be linked to a higher risk of breast cancer [36].

In the present study abdominal obesity was also observed in 27% of the rural and 31% of the urban premenopausal subjects (Illustration-IV). This could be due to difference in dietary habits, physical inactivity, socioeconomic or genetic background.

A significantly higher level of metabolic risk factors including blood pressure, triglyceride and fasting blood glucose were observed among post-menopausal women than pre-menopausal women. HDLc was also found to be significantly lower in the postmenopausal group. In agreement with the results of our study, many previous studies have reported higher prevalence of hypertension [18, 20, and 37], hypercholesterolemia [20], hypertriglyceridemia [38], low HDL [17, 20] and elevated fasting blood glucose [17, 20] among post menopausal women than pre-menopausal women.

A high amount of abdominal fat is associated with increased insulin resistance, free fatty acid (FFA) levels, and decreased adiponectin. These factors contribute to increased secretion of apolipoprotein B (apo B)-containing particles, leading to hypertriglyceridemia and increased hepatic lipase (HL) activity resulting in a predominance of small dense LDL particles and a reduction in large antiatherogenic HDL2 particles. A similar pattern of lipid abnormalities emerges with menopause [39].

Our study revealed high fasting glucose levels in the post menopausal subjects (both rural and urban) and the differences were statistically highly significant (Illustration II and III). 68% of the rural and 72% of the urban postmenopausal women were having higher than 100 mg/dl of the fasting blood glucose values (Illustration-IV). The subjects with higher than 88 cm
Waist circumference were having higher fasting blood glucose levels. A parallel rise was observed in these two parameters.

Abdominal obesity is closely associated with increased insulin resistance, compensatory hyperinsulinemia, and increased risk of type 2 diabetes, independent of an individual’s total body fat content [40]. Insulin resistance, with inadequate compensatory hyperinsulinemia, diminishes the normal suppression of FFA arising from adipose tissue by insulin. The increased levels of FFA may impair peripheral glucose uptake, increase hepatic gluconeogenesis, and reduce hepatic clearance of insulin [41]. Several groups have shown increased fasting insulin [42, 43] and increased fasting glucose levels [44, 45] in postmenopausal compared with premenopausal women, which would imply worsened insulin resistance with the menopause.

Metabolic syndrome was also observed in 23.5% of the premenopausal subjects (rural and urban combined-Illustration-V) and 56% (combined) of the premenopausal subjects were having waist circumference >88 cm (Illustration-IV). Thus it implies that abdominal obesity per se is the leading factor for metabolic syndrome. Current evidence implies that multiple risk factors for CVD emerge in the postmenopausal period, but features of the metabolic syndrome may be present even before menopause [39]. Moreover, Asian Indians, in general, are prone to have MS at a younger age and have severe morbidity and mortality consequences as compared to Caucasians [46, 47, and 48].

Rural and urban variations in the components of metabolic syndrome in both pre and post menopausal subjects of our study were in accordance with the reports of other studies [49, 50]. The differences can be attributed to socioeconomic status, a sedentary lifestyle, and poor diet quality. Diet quality and physical activity are higher in rural compared to urban subjects. Physical activity appears protective for obesity, high blood pressure, and low HDL-c [51].

Metabolic syndrome not only increases the cardiovascular risk, each of its components have been found to be associated with increasing risk for breast cancer, obesity, particularly central obesity, could induce chronic low-grade inflammation [52], which is another known risk factor of breast cancer and can increase the likelihood of epigenetic alterations such as aberrant DNA methylation [53, 54]. Aberrant DNA methylation plays a crucial role in breast carcinogenesis [55].

Hyperinsulinemia and hyperglycaemia are biomarkers for insulin resistance [56]. Both of these disorders are critical to the initial development and progression of breast cancer. Goodwin et al. [57] firstly reported that in both premenopausal and postmenopausal women, insulin levels were correlated with breast tumour stage, nodal stage and tumour grade, and related to an increased risk of distance recurrence and a shorter survival regardless of the BMI. As two important components of the MS [58], higher TG and lower HDL-C levels in serum were found to be more common in patients with malignant diseases including breast cancer compared with non-cancer subjects [59, 60]. Low HDL-C is further related to increased levels of several other hormones including estrogens, insulin, and IGF-1, all of which can stimulate cancer development [61]. The positive association between low HDL-C and breast cancer risk may reflect the relative importance and mutual dependence of different pathways in the progression of breast cancer, particularly among postmenopausal women. For postmenopausal women, bio-available estrogens, the major stimulus for breast carcinogenesis, are mainly formed in fat tissue or in the granulosa cells of the ovarian follicle through the aromatization of androstenedione and testosterone instead of direct ovarian oestrogen production [62]. Results from both animal models [63, 64] and human studies [65] have implicated that hypertension may increase the response to carcinogens and initiate the process of carcinogenesis. Thus each of the individual components of MS is associated with a risk for breast cancer.

**Conclusion(s)**

Abdominal obesity plays a central role in connecting the metabolic syndrome with the metabolic alterations of menopause and can be a strong predictor of impending metabolic syndrome. Metabolic syndrome not only increases the risk for cardiovascular diseases in post menopausal women but it is a potential risk factor of breast cancer as well. The MS and individual metabolic disorder can be prevented and modified. Hence a close attention is needed not only after but before menopause also for weight management, increasing physical activity and adopting healthy life style not only to prevent the onset of metabolic syndrome but to improve the quality of life as well.

**Abbreviation(s)**

IHD (Ischemic heart disease), MS (Metabolic syndrome), CVD (Cardiovascular disease), ATP
(Adult treatment panel, BMI (body mass index), WHR (Waist to hip ratio), TG (triglycerides), HDLc (High density lipoprotein cholesterol).

Acknowledgement(s)

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46. Misra A, Khurana L. The metabolic syndrome in
The study subjects were distributed in to two main groups – Group I comprising of 100 postmenopausal (rural) and Group II (premenopausal). Both the groups I and II were further sub classed as - A (rural) and B (urban). There were 50 study subjects in each of the groups.
**Illustration 2**

Base line characteristics of study subjects

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Post menopausal (Total Rural and Urban)</th>
<th>Premenopausal (Total Rural and Urban)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
<td>100</td>
<td>100</td>
<td>-</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>57.25±0.80</td>
<td>34.48±0.74</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Body Mass Index(BMI)Kg/m2</td>
<td>24.43 ± 2.34</td>
<td>22.69 ±2.23</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Waist to Hip ratio (WHR)</td>
<td>0.91 ± 0.08</td>
<td>0.79 ± 0.05</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic B.P.(mmHg)</td>
<td>141.46±15.62</td>
<td>116.94 ± 6.96</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic B.P.(mm Hg)</td>
<td>80.40 ± 8.67</td>
<td>76.78 ± 5.29</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Fasting blood glucose(mg/dl)</td>
<td>118 ±6.51</td>
<td>79±4.56</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>S. Total Cholesterol(mg/dl)</td>
<td>224.05±48.89</td>
<td>162.37± 25.67</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>S. Triglycerides (mg/dl)</td>
<td>137.22±40.31</td>
<td>111.85±19.38</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VLDLc (mg/dl)</td>
<td>27.44±8.06</td>
<td>22.37±3.88</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LDLc(mg/dl)</td>
<td>155.40±49.08</td>
<td>89.47±25.81</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDLc(mg/dl)</td>
<td>41.21±6.87</td>
<td>50.53±6.01</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Continuous data were presented as mean ±standard deviation of mean (SDM). VLDL-C: Very Low Density Lipoprotein-Cholesterol, LDL-C: Low Density Lipoprotein-Cholesterol, HDL-C: High Density Lipoprotein-cholesterol
Illustration 3

Base line characteristics of study subjects in different groups (rural and urban)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Rural</th>
<th>Urban</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Post menopausal</td>
<td>Premenopausal</td>
<td>Post menopausal</td>
</tr>
<tr>
<td>Number of subjects</td>
<td>50</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>Body Mass Index (BMI) Kg/m2</td>
<td>24.04 ±2.36</td>
<td>22.34 ±1.96</td>
<td>24.83 ± 2.27</td>
</tr>
<tr>
<td>Waist to Hip ratio (WHR)</td>
<td>0.903 ± 0.079</td>
<td>0.776 ± 0.039</td>
<td>0.926 ±0.083</td>
</tr>
<tr>
<td>Systolic B.P.(mmHg)</td>
<td>139.84 ± 14.62</td>
<td>117.00 ± 6.24</td>
<td>141.46 ± 15.62</td>
</tr>
<tr>
<td>Diastolic B.P.(mm Hg)</td>
<td>80.24 ± 8.79</td>
<td>77.12±5.42</td>
<td>80.24 ± 8.79</td>
</tr>
<tr>
<td>Fasting blood glucose(mg/dl)</td>
<td>110 ±26.34</td>
<td>87±15.63</td>
<td>119±25.61</td>
</tr>
<tr>
<td>S. Total Cholesterol(mg/dl)</td>
<td>220.08±44.03</td>
<td>160.54 ± 23.07</td>
<td>228.02 ±53.47</td>
</tr>
<tr>
<td>S. Triglycerides (mg/dl)</td>
<td>132.10 ± 37.70</td>
<td>111.30± 18.93</td>
<td>142.34±42.52</td>
</tr>
<tr>
<td>VLDLc (mg/dl)</td>
<td>26.42± 7.54</td>
<td>22.26 ± 3.79</td>
<td>28.47± 8.50</td>
</tr>
<tr>
<td>LDLc(mg/dl)</td>
<td>151.46±44.34</td>
<td>88.14± 23.32</td>
<td>159.33±53.56</td>
</tr>
<tr>
<td>HDLc(mg/dl)</td>
<td>42.20±6.55</td>
<td>50.14±5.65</td>
<td>40.22±7.11</td>
</tr>
</tbody>
</table>

Continuous data were presented as mean ±standard error of mean (SEM). VLDL-C: Very Low Density Lipoprotein-Cholesterol, LDL-C: Low Density Lipoprotein-Cholesterol, HDL-C: High Density Lipoprotein-cholesterol
Illustration 4

Prevalence of components of metabolic syndrome in different study subjects as per NCEP: ATPIII criteria

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Rural (number and percentage of subjects having individual metabolic syndrome component)</th>
<th>Urban (percentage of subjects having individual metabolic syndrome component)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Post menopausal n (%)</td>
<td>Pre menopausal n(%)</td>
</tr>
<tr>
<td>Waist circumference &gt;88 cm</td>
<td>34(68)</td>
<td>27(54)</td>
</tr>
<tr>
<td>Triglycerides ±150 mg/dL</td>
<td>12(24%)</td>
<td>5(10)</td>
</tr>
<tr>
<td>HDL cholesterol: &lt;50 mg/dL</td>
<td>14(28)</td>
<td>4(8)</td>
</tr>
<tr>
<td>Systolic Blood pressure±130 mm Hg</td>
<td>25(50)</td>
<td>5(10)</td>
</tr>
<tr>
<td>Diastolic Blood pressure±85 mm Hg</td>
<td>10(20)</td>
<td>2(4)</td>
</tr>
<tr>
<td>Fasting plasma glucose ±100 mg/dL</td>
<td>34(68)</td>
<td>12(24)</td>
</tr>
</tbody>
</table>

A larger number of post-menopausal subjects were having components of metabolic syndrome as compared to pre-menopausal counterparts. Statistically insignificant variations were observed amongst rural and urban subjects with urban subjects having higher values than the rural ones.
Illustration 5

Prevalence of metabolic syndrome in rural and urban post menopausal and pre menopausal subjects

As per NCEP: ATPIII criteria a total of 42% (rural and urban combined) - 41% and 43% rural and urban groups respectively of the postmenopausal subjects were having metabolic syndrome. In comparison metabolic syndrome was observed in 23.5% (rural and urban combined) – 20% and 27% in rural and urban groups respectively of the premenopausal subjects.