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CARDIOMETABOLIC STATUS OF MENOPAUSAL WOMEN IN NKWELLE-EZUNAKA, ANAMBRA STATE

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ABSTRACT

Background: Menopause results from ovarian aging or ovarectomy and is accompanied by hormonal and metabolic changes that contribute to cardiovascular disease (CVD), a leading cause of mortality.

Aim of study: To assess the cardiometabolic status of menopausal women in Nkwelle-Ezunaka using Body Mass Index (BMI), Waist Hip Ratio (WHR), Diastolic Blood Pressure (DBP), Systolic Blood Pressure (SBP), Total Cholesterol (TC), Triglycerides (TG), Low Density Lipoprotein Cholesterol (LDL-C), High Density Lipoprotein Cholesterol (HDL-C), Insulin Resistance, Fasting Plasma Glucose (FPG) and Fasting Insulin (FI).

Materials and Methods: This cross-sectional study involved ninety females (45 menopausal, and 45 premenopausal) recruited by simple random sampling technique. Blood pressure was measured using the auscultatory method, BMI calculated using weight and height measurements, while WHR was obtained using waist and hip circumference measurements. Levels of TC, TG, LDL-C, HDL-C, IR, FPG and FI were determined spectrophotometrically using standard methods. Independent t-test was used for the statistical analysis of data.

Results: A significantly higher mean values of BP, WHR, TC, TG and LDL-C ($p<0.05$) and lower mean values of FI (6.80 ± 4.46) and IR (1.49 ± 1.15) were observed in menopausal women compared to premenopausal women (12.90 ± 15.33)(3.95 ± 5.45) with $p<0.05$ in both cases. No significant differences were found in the mean values of BMI, HDL-C and FPG in menopausal women compared with the premenopausal women ($p> 0.05$**)**.

Conclusion: Waist Hip Ratio, Diastolic Blood Pressure, Systolic Blood Pressure, Total Cholesterol, Triglycerides, Low Density Lipoprotein Cholesterol, may be better indicators of unhealthy cardio-metabolic status than Body Mass Index, High Density Lipoprotein Cholesterol and Fasting Plasma Glucose. Menopausal women appear to be more predisposed to cardiovascular disease than the premenopausal women.

Keywords: *Menopause, Body Mass Index, Waist Hip Ratio, Fasting Plasma Glucose, Insulin Resistance, dyslipidaemia*

INTRODUCTION

Menopause is clinically diagnosed when a healthy woman has not menstruated for up to one year.[1] The average level of total oestrogen (E2) during a woman's fertile life is 100–250 pg/ml but the concentration of E2 in circulation declines up to 10 pg/mL postmenopause.[2] The dramatic decrease in ooestrogen production in menopause may alter glucose and lipid metabolism and lead to probable changes in insulin sensitivity, lipid metabolism and body mass index resulting in cardiovascular morbidity and mortality.[3] Central obesity which is quite common in menopausal women caused by dyslipidaemia is also strongly associated with cardiovascular risk with or without BMI adjustments.[4] Also, before menopause, the prevalence of hypertension (HTN) in women is much lower than in men; however, this prevalence increases significantly in menopausal women and equates to that in men.[5] Sex

hormones are said to be responsible for the sex differences in the regulation of blood pressure[6] because it affect systems that are considered to play an important part in the development of hypertension, such as renin angiotensin aldosterone system, endothelin, nitric oxide (NO) system and immune system.[6]

Menopause may also deteriorate lipid profile making IT more atherogenic than that of their premenopausal counterpart.[7] The total cholesterol (TC), triglycerides (TG) and low-density lipoprotein cholesterol (LDL-c) may increase, and these changes may be accompanied by a decrease in high-density lipoprotein cholesterol (HDL-c).[8] This partially explains the increased cardiovascular risk in postmenopausal women, particularly among those with an earlier onset of menopause.[9, 10]

The reduction in oestrogen may also predispose menopausal women to developing insulin resistance possibly due to the critical role oestrogen plays in carbohydrate metabolism.[11] In support of this hypothesis, it has been shown that surgically induced menopause increases the risk of developing insulin resistance and metabolic syndrome more[12] probably because oestrogen decline could also affect insulin production by pancreatic β cells and insulin disposal in muscles, which are conditions that further exacerbate the risk of diabetes.[13] The homeostasis model assessment of insulin resistance index (HOMA-IR) is the most commonly used proxy for insulin resistance as it correlates strongly with the results of euglycemic-hyperinsulinemic clamps[14] being used as the gold standard of insulin resistance evaluation.[15]

Studies across the world have thrown light on the increased cardiometabolic risks in menopausal women as compared to their corresponding premenopausal.[11, 16-19] Therefore, this study aim to use some measures of cardiometabolic status (SBP, DBP, BMI, WHR, TC, TG, LDL-C, HDL-C, FPG and FI) to evaluate the cardiometabolic status of menopausal women in Nkwelle-Ezunaka, Anambra state.

MATERIALS AND METHODS

Study area

This research was conducted in Nkwelle-Ezunaka, Anambra state. Nkwelle-Ezunaka is one of the five towns in Oyi Local Government Area of Anambra state[20], located about 8.5 kilometers northeast of Onitsha, Anambra state. It is bordered by nine neighbouring towns; Nteje and Umunya to the east, Nsugbe and Umueri to the north, Onitsha and Obosi to the west and Nkpor, Ogidi and Ogbunike to the south. Nkwelle-Ezunaka has a vast land rich in farming and is a fast developing sub-urban area in Nigeria.

Study design

This cross-sectional study was designed to assess cardio-metabolic disorders in menopausal women. A total of 90 female subjects within the age range of 19 to 55 years were recruited for using random sampling techniques. This included 45 menopausal women and 45 premenopausal women. A random pick of 2 areas was made with an average of 45 individuals mobilized for the study from each selected area. Participants were interviewed via structured questionnaires and physical assessment.

Sample size: Sample size was determined using Daniel[21] sample size formula given as[22]:

$$N = \frac{z^2 P(1-P)}{d^2}$$

N= sample size, z = confidence interval, p= expected prevalence or proportion and d =

precision. Here confidence interval of 1.96 and precision of 0.05 was used and menopausal prevalence of 3.96%.

N = (1.96)^2 X 3.96% (1-3.96%)/0.05^2
N = 3.8416 X 3.96/100 (1-3.96/100)/0.05^2
N=3.8416 X 0.0396(1-0.0396)/0.0025
N = 3.8416 X 0.0396(0.604)/0.0025
N= 3.8416 X 0.0396 (241.6)
N= 3.8416 X 9.567
N = 36.7
N= 36.7
N = 37

Thus, a minimum sample size of 37 was determined using menopausal prevalence rate[18] of 3.96% but a total of 90 subjects were recruited for the study.

Ethical consideration

The ethical approval was obtained from Ethics Review Committee, Nnamdi Azikiwe University Teaching Hospital, Nnewi (NAUTH/CS/66/VOL.16/VER.3/306/2021/080). The study participants were enlightened on the purpose of the study and allowed to choose to verbally volunteer.

Inclusion criteria

Apparently healthy premenopausal and menopausal females within the age range of 19 to 65 years.

Exclusion criteria

Individuals on hormonal treatments, those with history of cardiovascular diseases, diabetes and malignant tumors and individuals outside the age range of 19 to 65 years.

Determination of blood pressure

Blood pressure measurement was measured using the auscultatory method[23]. Using a suitably calibrated mercury sphygmomanometer, the volunteers were allowed to rest for five minutes and the blood pressure was taken in the sitting position. A cuff was wrapped around the subject's upper arm and inflated; the brachial artery was occluded as the cuff gradually deflated. Blood flow was re-established, accompanied by tapping or thumping sounds that can be detected with a stethoscope held over the brachial artery. The first tapping or thumping sound signified the systolic pressure and the point at which the tapping ceased was taken as the diastolic pressure. Systolic pressure and diastolic pressure greater than 140 mm/Hg and 90 mm/Hg respectively indicated high blood pressure while systolic pressure of 90mm/Hg and 60mm/Hg of diastolic pressure was regarded as low blood pressure.

Determination of waist and hip circumference and waist to hip ratio

Waist circumference was measured[24] at the midpoint between the lower margin of the least palpable rib and the top of the umbilicus, with the tape around the body in a horizontal position. Participant stood upright with both feet together and both arms relaxed by their side; after finding the lower edge of the participant's last rib on their side and the upper edge of the umblicus, the waist circumference was measured horizontally between these two points. Prior to the measurement the participants were asked to exhale gently. Hip circumference was also measured horizontally in a standing position by putting the participant's feet apart and arms at their chest using the same tape measure at the most prominent area of the buttock when seen sideways. Waist-to-hip ratio was calculated as

waist circumference (cm) to hip circumference (cm).

Determination of body mass index

Body mass index[25] was calculated using the formula:

BMI = weight/height^2

Laboratory Methods:

Determination of total cholesterol

Evaluation of total cholesterol was done using enzymatic method as described by Manafa et al.[26]

Determination of triglyceride levels

The assessment of TG was done using enzymatic method as described by Ihim et al[27].

Determination of high density lipoprotein

Assessment of high density lipoprotein was done using the method as described by Gulsen et al[28].

Evaluation of low density lipoprotein levels

The Friedewald equation was used to calculate low density lipoprotein as described by Boqun et al[29], given as:

Total cholesterol = VLDLchol +LDLchol+HDLchol

TG is an estimate of VLDLchol

LDLchol = [Total chol]– [HDLchol] –[TG]/5

Assessment of insulin resistance

The insulin resistance index of each subject was determined by homeostatic model assessment (HOMA) according to the method described by Hashemipour et al[30]. An insulin resistance score was computed with the formula:

$$\frac{\text{fasting plasma glucose (mmol/l) x fasting serum insulin (mU/l)}}{22.5}$$

Low HOMA-IR values indicated high insulin sensitivity while high HOMA-IR showed low insulin sensitivity (insulin resistance).

Data Analysis

Obtained data was summarized using mean and standard deviation, and analysed using the Independent t-test. Results were deemed significant at $p<0.05$.

RESULTS

There was a significantly higher level of mean age in the test subjects compared with the control (54.19 ± 5.14 vs 29.58 ± 11.13; $p<0.05$). The mean systolic and diastolic blood pressure values of the test subjects were significantly higher compared with the control (146.21 ± 18.85 vs 132.38 ± 26.20; $p<0.05$) (89.38 ± 10.17 vs 81.19 ± 14.17; $p<0.05$) respectively while the mean waist-hip ratio of the test group showed a significantly higher level compared with the control (0.90 ± 0.07 vs 0.85 ± 0.05; $p<0.05$). However, there was no significant difference in the body mass index of the test subjects compared with the control (30.14 ± 5.55 vs 28.23 ± 4.97; $p<0.05$). Table1 summarizes these findings.

However, a significantly higher mean level of total cholesterol was observed in the test subjects compared with the control (223.56 ± 37.18 vs 191.20 ± 51.07; $p<0.05$). Also, there were significantly higher mean levels of low density lipoprotein and triglycerides in the test subjects compared with those of the control (114.28 ± 17.85 vs 105.25 ± 23.54 ;$p<0.05$) and (151.07 ± 30.12 vs 128.36 ± 51.25; $p<0.05$) respectively while the mean levels of insulin and insulin resistance were significantly lower in the test subjects compared with the control (6.80 ± 4.46 vs 12.90 ± 15.33; $p<0.05$) and (1.49 ± 1.15 vs

3.95 ± 5.45; p < 0.05) respectively. However, there was no significant difference in the mean levels of fasting plasma glucose (FPG) (87.17 ± 18.11 vs 84.35 ± 13.29 p> 0.05) and high density lipoprotein (HDL) (49.64 ± 15.95 vs 45.67 ± 16.87 p> 0.05) in the test group compared with the control group (Table 2).

Table 1: Levels of some anthropometric variables of control and test group (mean ± SD)

Parameters	Test group (Menopausal women)	Control group (premenopausal women)	t-test	p-value
Age (year)	54.19 ± 5.14	29.58 ± 11.13	-13.660	0.000
SBP (mmHg)	146.21± 18.85	132.38 ± 26.20	-2.198	0.032
DBP (mmHg)	89.38 ± 10.17	81.19 ± 14.17	-2.412	0.019
BMI(kg/m^2)	30.14 ± 5.55	28.23 ± 4.97	-1.504	0.137
WC/HC	0.90 ± 0.07	0.85 ± 0.05	-4.486	0.000

*Statistically significant at p<0.05

Table 2: Levels of lipid profile, FPG, insulin and insulin resistance in control and test groups (mean ± SD).

Parameters	Test	Control	t-test	p-value
TC(mg/dl)	223.56 ± 37.18	191.20 ± 51.07	-3.456	0.001
TG(mg/dl)	114.28 ± 17.85	105.25 ± 23.54	-2.060	0.042
HDL-C(mg/dl)	49.64 ± 15.95	45.67 ± 16.87	-1.146	0.255
LDL-C (mg/dl)	151.07 ± 30.12	128.36 ± 51.25	-2.587	0.011
FPG(mg/dl)	87.17 ± 18.11	84.35 ± 13.29	-0.836	0.405
FI (mIU/L)	6.80 ± 4.46	12.90 ± 15.33	2.613	0.011
HOMA-IR	1.49 ± 1.15	3.95 ± 5.45	3.026	0.003

*Statistically significant at p<0.05

Key:

TC= total cholesterol TG= triglycerides, HDL-C = high density lipoprotein cholesterol, LDL-C= low density lipoprotein cholesterol, FPG = fasting plasma glucose, FI = fasting insulin, HOMA-IR = homeostatic model assessment of insulin resistance.

DISCUSSION

Ovarian atrophy and hormonal changes in menopausal women may increase the risk of diabetes, dyslipidemia and cardiovascular disease (cardiometabolic disorders).[31, 32] This is possibly due to the important roles oestrogen plays in the maintenance of lipid and glucose homeostasis[33] and its imminent cardioprotective effects. In this study the measures of adiposity in menopausal women in Nkwelle-Ezunaka metropolis were evaluated. The findings of this study revealed increased systolic and diastolic blood pressure of menopausal women (test) compared with premenopausal women (control). Aging in both males and females can be identified by an increase in blood pressure but the incidence of hypertension in women after menopause is greater than in males.[34] The sharp rise in BP after menopause may be both a direct effect of hormonal changes on the vasculature and metabolic changes with ageing.[35] These hormonal changes especially oestrogen decline also affect the rennin-angiotensin–aldosterone system (RAAS) that regulate sodium and water intake, output and consequently blood pressure. Conversely, oestrogen exerts inhibitory effects on classical RAAS pathway resulting in overall vasodilatory and antihypertensive response.[36] But its deficiency due to menopause may contribute to over activity of the rennin aldosterone angiotensin system (RAAS). This over activity of the RAAS has been implicated in the pathogenesis of a number of cardiovascular disease entities, including hypertension.[37] The activation of RAAS is not the sole contributor of hypertension in menopause rather a mediator.[34] Another mechanism contributing to hypertension in postmenopausal women is an increase in sympathetic activation that could be due to increased body weight and redistribution of body fat as well as increased leptin levels.[38] Obesity may be another causal factor of hypertension in menopausal females.[34] Obesity, especially visceral obesity, is the integral part of the group of metabolic syndrome which comprise insulin resistance (type 2 diabetes), dyslipidemia and waist circumference greater than 35 inches for men, all of which are known to cause hypertension.[39] The incidence of obesity is close to 40% in menopausal women.[34] Additionally, androgen production continues in menopausal women and may increase arterial stiffness and vascular inflammation leading to endothelial dysfunction and increased BP. However, findings are inconsistent in the role of oestrogen/androgen on hypertension in menopausal women.[40] Severity of menopausal symptoms also plays a role. It has been reported that women who experience vasomotor symptoms such as hot flashes have higher awake and asleep blood pressure when compared to women without hot flashes.[40] Menopausal women are also more likely to have a non-dipping BP pattern which is associated with poorer cardiovascular outcomes and more target organ damage in women compared to men.[41] The impact of increased blood pressure is different for men and women. It has been shown that for a comparable 10mmHg increase in systolic blood pressure, women experience a 25% increase in cardiovascular disease risk while men's risk is only 15% higher.[42] Sex-specific differences in blood pressure (BP) have been noted since the early 1900's when women were first observed to have lower BP compared to men of a similar age.[40] Blood pressure, and consequently hypertension prevalence, is lower in women from adolescence until menopause or the fifth decade of life.[43,44]

Despite the higher prevalence of hypertension in men, a study of 32,833 individuals (17,733 women or 54%) followed for over four decades, demonstrated that women actually have a steeper increase in BP as early as the third decade that continues throughout the life course.[44] These differences persisted even after adjustment for multiple cardiovascular risk factors. Taken together, these sex differences in BP across the life course may have important implications for the diagnosis and treatment of hypertension in men and women, though currently there are no sex-specific guidelines for the diagnosis or treatment of hypertension.[40]

Anxiety and depression may also play a role in increasing blood pressure. Menopause has been proven to predispose women to various psychological health problems, including depression and anxiety.[45] Several menopausal symptoms such as hot flashes, night sweats, and insomnia, may contribute to increased risk of depressive and anxiety symptoms.[46,47,48] Menopausal women with depression and anxiety have a higher risk of developing hypertension.[49] Sympathetic activity can be upregulated with anxiety and chronic mental stress, which may lead to hypertension.[50] Increased BP was also seen due to enhanced levels of anxiety in a small Spanish cohort study.[40] The onset of hypertension can cause a variety of symptoms, such as palpitations, hot flushes, headaches, chest pain, pain between the shoulder blades, tiredness and sleep disturbances, which are often attributed to menopause.[51]

Our findings align with the reports of Eghbali-Babadi et al [52] who found high systolic and diastolic blood pressure to be highly prevalent in menopausal women in Iran. Okeahialam *et al*[53] also observed lower anthropometric indices, systolic and diastolic blood in 218 premenopausal females compared 270 menopausal, they postulated that menopause comes with worse CVD profile.[53]

Our study also showed a higher waist hip ratio (WHR) in menopausal women compared with premenopausal women. This possibly implies that more menopausal women had central adiposity than premenopausal women. Waist hip ratio measures body fat distribution and values above 0.85 for females would indicate central body fat distribution. High WHR is considered to be a risk factor for cardiovascular diseases, hypertension and diabetes.[54] Ooestrogen deficiency in menopause may have a direct effect on lipid metabolism and body fat composition and distribution with a transition from gynecoid (apple) to android (pear) body shape and increased abdominal and visceral fat accumulation associated with increased CVS and metabolic risks.[55-58] The visceral distribution of adipocytes is postulated to increase inflammation, an important trigger for cardiovascular and metabolic disease.[57] Abdominal fat is considered an endocrine organ able to produce many adipokines and substances that are associated with hypertension, dyslipidaemia, insulin resistance, type 2 diabetes and metabolic syndrome.[57]

The mechanism of central adiposity in menopause is not fully understood though it is postulated that genetic and environmental factors play a role.[59] These factors determine adipose tissue mass and distribution by modulating energy balance and lipid related enzyme activitities.[60] Subcutaneous adipose tissue serves as long term lipid storage while visceral adipose tissue is metabolically more active and acts as an acute response supplier of systemic fatty acids.[61] Menopause is characterized by low oestrogen level and high levels of follicle stimulating

hormone (FSH). The rise in the level of FSH was shown by Kohrt and Wierman to have an independent effect on regulation of energy homeostasis[62]. FSH promotes lipid biosynthesis and is positively associated with leptin and negatively with adiponectin levels in cellular and animal models.[63] It might explain why the use of oestrogen replacement therapy which does not completely suppress FSH levels, may fail to prevent the fat changes in menopause.[57] A meta-analysis suggests that aging is the main contributor of increased overall adiposity while menopause contributes to adipose tissue accumulation in the waist area.[64] Sleep problems like insomnia, sleep apnea, are core menopausal symptoms which can disrupt metabolism as proven by several studies and can lead to central obesity[65,66,67]. Women have been suggested to have a 41% higher risk of developing insomnia than men[68]. A study by Chaput et al [69] who checked the effects of sleep duration on visceral fat found that changing sleep duration from less than 6hrs to 7 to 8 hrs was inversely associated with visceral adipose tissue gain. In essence subcutaneous and visceral fat mass is tightly associated with sleep duration.[70] Poor sleep has been shown to increase sugar cravings which factors in central obesity.

Short sleep duration increases a woman's stress level likewise the negative impact menopausal symptoms exerts on a woman's mental health, hiking a woman's stress level.[71] The high stress level experienced by menopausal women has endocrine consequences due to the increased effects of glucocorticoids and cortisol leading to loss of muscle and bone mass and visceral fat accumulation[72, 73] resulting in central obesity. These problems will physically strain the women as well as exert massive burden on the mental health of the women.[74] A study by Jayabharathi, 2016 found that 75% menopausal women had high to very high level of stress.[75] Menopause is overall a time of increased stress, including the experience of stressful life events like a divorce or the loss of a loved one[76]. Due to the close interaction of the reproductive and the stress axes[77], stress can act as a precipitating or perpetuating factor for disorders like depression or insomnia and central obesity.[68]

Physical inactivity can also be a factor in the occurrence of central obesity in menopausal women. Notably, physical activity levels tend to decline during and following menopause, which ultimately exacerbates metabolic dysfunction.[78] Although the extent to which physical inactivity contributes to metabolic shifts during the menopause is not fully known, it is noteworthy that the typical gain in central adiposity during the menopause is linked to an approximately 40% reduction in physical activity.[79]

Unhealthy diet might also be a causative factor of central obesity, as both exercise training and adopting/maintaining healthy dietary patterns following the menopause are essential in mitigating visceral fat accumulation and preserving metabolic health.[80] This is in concordance with study by[81] who found 77.7 % prevalence of central obesity among 273 menopausal women aged 45 to 65 years. Also a significant difference in WHR between premenopausal and menopausal women[16] was demonstrated but portrays that it as a better predictor of subclinical atherosclerosis. This also agrees with Selvaraj et al[82] that did a population and family-based epidemiological study of 2181 adults aged 37 to 65 years (perimenopausal and menopausal age) and discovered 80 % of these women had central obesity. A combination of hormonal shifts and chronological aging are primarily what paved the way to a cluster of

metabolic abnormalities associated with the menopause[79].

This study also revealed significantly increased total cholesterol and LDL cholesterol in menopausal women compared with control while there was no significant difference in HDL. Physiological decline in oestrogen levels during menopause plays a major role in abnormal lipid metabolism such as elevated low-density lipoprotein concentration (Dyslipidaemia).[83] Also glucose spikes caused by disordered carbohydrate metabolism as a result of oestrogen deficiency can also exacerbate dyslipidaemia.[84] The lipid panel test revealed significant increased levels of total cholesterol, LDL-cholesterol and triglyceride in menopausal women compared to premenopausal and surprisingly a significant increase in HDL-Cholesterol. Also the findings of this study agrees with that of Inaraja et al [85] who did a retrospective observational study of 13517 laboratory analysis (3,073 premenopausal and 10,444 postmenopausal lab results) of 275 women from gynecology unit of hospital Quiro'n Salud, Madrid (2007-2018) and found a significantly higher levels of total cholesterol, LDL-cholesterol and triglyceride in menopausal women than premenopausal women while HDL-cholesterol levels were significantly lower in all cases. Similarly a study by[86] found that after menopause, women had higher levels of triglycerides and LDL.

After menopause, total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-c) usually increase, and these changes are accompanied by a decrease in high-density lipoprotein cholesterol (HDL-c) and an increase in triglycerides (TG).[8] In addition to these major lipid abnormalities, modifications in size and density of these lipoprotein particles are expected to happen after the loss of ovarian hormonal production. This partially explains the increased cardiovascular risk in postmenopausal women, particularly among those with an earlier onset of menopause.[9] Age and sex are primary physiological factors that have a strong influence on blood lipid levels.[63] With increasing age, lipid levels increased among both men and women[87,88] , but prevalence of dyslipidemia was significantly higher among women in midlife than men.[89] This suggests that the menopausal transition (MT) may contribute substantially to dyslipidemia in women in midlife.[90] Previous works about the relationship between the MT and lipid profiles were controversial. Some studies report that there was no change in lipid profiles before and after menopause, suggesting the possible effect of only chronological aging[91,92]. Several studies have revealed a significant association between the MT and lipids, as evidenced by substantial changes in lipid profiles after the final menstrual period (FMP)[93], Matthews *et al*. showed an association between MT and lipids profiles, with age at menopause playing an important role in lipid changes during the MT[94] and Di Francesco *et al*. suggested they experience serum lipid changes owing to a significant increase in the sex hormone oestrogen.[95] Their low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), and triglycerides (TG) increase and high-density lipoprotein cholesterol (HDL-C) decreases.[10] In Korea, dyslipidemia among women increased with age and showed a significant difference before and after menopause. The prevalence was 27.6% in women aged ≤40 years, 55.9% in women aged 40–59 years, and 64.6% in women aged ≥60 years.[10] In particular, the prevalence of high LDL-C was more than six times higher in those in their 50s when compared with those in their 30s.[96] This probably causes a significant increase

in the incidence of fatal cardiovascular disease.[95] Women with HDL-C levels < 50 mg/dL have a 30% increased risk of death from cardiovascular disease, and those with a TC level between 200 and 399 mg/dL have a 65% increased risk of death.[97]

An interesting observation in our study is the higher insulin levels and insulin resistance in premenopausal women compared with the menopausal group which contradicts the studies of Fonseca *et al*. that evaluated the association between insulin resistance and metabolic syndrome in 150 women 40-65 years treated at a gynecology outpatient clinic.[98] They found that menopausal women had higher prevalence of insulin resistance[98] and that of Kirtikar *et al*. who studied cardiometabolic risk in premenopausal and postmenopausal women who had fasting insulin of 23±12.3 mIU/L against that of premenopausal group less than 3mIU/L.[99] This contradiction could be due to limited sample size or a possible rise of metabolic syndrome in the premenopausal women as postulated in a study by Isaki *et al*. that examined 401 young women for insulin resistance and found (32) 8% and only 6 out of the 32 were overweight which they attributed to metabolic syndrome.[100] A combination of hormonal shifts and chronological aging are primarily what pave the way to a cluster of metabolic abnormalities associated with the menopause.[79]

CONCLUSION/RECOMMENDATION

Blood pressure, waist-hip ratio, total cholesterol, low density lipoprotein cholesterol and triglycerides increased significantly in menopausal women compared with premenopausal women. However no significant difference was observed in BMI, HDL-C and FPG of the menopausal group compared with the premenopausal females. This suggest that waist-hip ratio, blood pressure, total cholesterol, low density lipoprotein cholesterol and triglycerides are better indicators of cardiometabolic status than BMI, HDL-C and FPG and are therefore recommended as valuable tools in the assessment of adiposity.

Also fasting insulin levels and insulin resistance decreased significantly in menopausal women compared with the premenopausal group.

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