

## Lipid Profile of Postmenopausal Women with Central Obesity

Monowara Khanam<sup>1</sup>, Md. Aminul Haque Khan<sup>2</sup>, Md. Rezwanur Rahman<sup>3</sup>,  
Selima Akhter<sup>4</sup>, Md. Mozammel Hoque<sup>5</sup>

### Abstract

**Background:** Following menopause there are changes in values of lipid profile parameters. Abdominal obesity has also been linked to significant metabolic abnormalities including changes in lipid parameter values. So, we designed this study to observe the pattern of lipid profile parameters in postmenopausal central obese women. **Objective:** To assess the lipid profile status of postmenopausal women with central obesity. **Materials and Methods:** This cross sectional study was carried out in the department of Biochemistry, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh during the period of January 2005 to December 2005. Seventy four postmenopausal women with central obesity and age matched 56 nonobese postmenopausal women were included in the study. Central obesity was defined having waist hip ratio more than 0.8. All statistical analyses were done by SPSS 12.0. *p* values <0.05 were considered significant. **Results:** Statistically no significant difference was observed between the central obese women and nonobese women in total cholesterol and LDL-cholesterol levels. But HDL-cholesterol was found lower and triacylglycerol was found higher in postmenopausal central obese women. **Conclusion:** Dyslipidaemia is a feature of postmenopausal women with central obesity.

**Key words:** Central obesity, Postmenopausal, Nonobese

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### Introduction

Postmenopausal women have an increased tendency for gaining weight. The decline of endogenous oestrogen, together with physical inactivity is probably the major cause of this phenomenon. Postmenopausal overweight and obesity lead to increased rates of hypertension, diabetes mellitus, coronary heart disease (CHD) and all-cause mortality.<sup>1</sup> Following menopause, adverse changes in lipid profile occur and the levels of several coagulation factors increase.<sup>2</sup>

The lipid profile is a group of tests that are often done together to determine risk of CHD. It includes total cholesterol (TC), HDL-cholesterol, LDL-cholesterol and triacylglycerol (TAG). It is used to

guide health care providers in decision making as to how a person at risk should be treated. The scenario of the lipid profile is considered along with other known risk factors of CHD to develop a plan of treatment and follow-up.<sup>3</sup>

It is well known that in pre-menopausal women the incidence of cardiovascular events is lower than in men of the same age and after menopause cardiovascular morbidity and mortality in women become similar to that of man indicating that female sex hormones play a relevant protective role upon the vasculature. The prevalence of obesity and overweight is also higher in postmenopausal women than that in men of comparable age.<sup>4</sup>

1. Assistant Professor, Department of Biochemistry, National Institute of Kidney Diseases and Urology, Dhaka

2. Professor, Department of Biochemistry, Enam Medical College, Savar, Dhaka

3. Associate Professor, Department of Biochemistry, Delta Medical College, Dhaka

4. Associate Professor, Department of Biochemistry, National Institute of Kidney Diseases and Urology, Dhaka

5. Professor, Department of Biochemistry, Bangabandhu Sheikh Mujib Medical University, Dhaka

Correspondence Monowara Khanam, Email: monowara59@gmail.com

Obesity is a term commonly used to describe individual with increased body fat. It is associated with an increased risk of atherosclerosis, diabetes mellitus and gall bladder disease. Normal fat content of body is considered to be 12–18% of body weight in men and 18–25% of body weight in women. Obesity is commonly said to be present when body fat content is more than 20% and 25% of body weight in men and women respectively. A value that correlates better with body fat is the body mass index (BMI).<sup>5</sup> Individuals with BMI between 25 and 29.9 are overweight, and BMI  $\geq 30$  are defined as obese.<sup>6</sup>

There are two major types of fat distribution in adult obese.<sup>7</sup>

1. Some adults store their fat mainly around the hips and thigh, which gives them a pear shape known as gynoid distribution; this is a characteristic of women.
2. The second type found in both sexes is the storage of fat primarily in the abdomen, producing an 'apple' shape known as android distribution.

Excess fat located in the central abdominal area of the body is called android, 'apple-shaped' or upper body obesity and is associated with a greater risk for hypertension, insulin resistance, diabetes, dyslipidaemia and coronary heart disease. Android fat distribution is defined by waist to hip ratio (WHR) more than 0.8 for women and more than 1 for men.<sup>6</sup>

Through the effect of menopausal transition, the morbidity and mortality of cardiovascular diseases in women are increased. Lack of the oestrogen protection is presumed to be the major reason. However, several other physiological changes (such as aging effect, increased body weight or android pattern of body fat distribution, decreasing resting metabolic rate and physical activity etc.) which develop during menopause may also influence the risk of cardiovascular disease. Among these factors the android pattern of body fat distribution seems to be the major issue. The android body fat distribution, glucose intolerance, hyperlipidaemia and hypertension appear to be clustered together in the same subject.<sup>8</sup>

The metabolic phenotype of postmenopausal women, which includes an increased tendency for body fat deposition in the abdominal region, suggests that insulin resistance may underlie the characteristic features of postmenopausal dyslipidaemia. Adverse effects of insulin resistance on lipid metabolism, with

consequent effects on circulating TAG concentrations, may be the primary metabolic defects that lead to low HDL-cholesterol and increased prevalence of small dense LDL which are the key features of the atherogenic lipoprotein phenotype. Greater tendency for central fat deposition after the menopause may be particularly relevant to the higher incidence of CHD in postmenopausal women. Although central obesity has been shown to be a strong risk factor of CHD for both men and women, studies in women generally produce values for relative risk that are higher than those found in men.<sup>9</sup>

The possibility that raised TAG is a key feature of the lipid disturbance that leading to increased risk of CHD after the menopause is supported by the fact that raised TAG is more strongly associated with CHD risk in women than men.

The Framingham Study<sup>10</sup> showed that in women the strongest predictor of CHD risk was a TAG level greater than 1.7 mmol/L and HDL-cholesterol values less than 1.3 mmol/L. In a prospective follow-up study (over a 20 years period) on 1462 women a strong association between serum TAG concentrations and CHD mortality was reported. The dominance of TAG as a risk factor in postmenopausal women and its link with central obesity and insulin resistance is also supported by the observation that women with non-insulin-dependent diabetes mellitus have a 4-5 fold increase in risk of CHD, compared with a 2-3 fold increase in men.<sup>9</sup>

Obesity is commonly regarded as an important contributor to the development of hyperlipidaemia on the basis that cross-sectional studies have documented statistically significant, although modest, associations between lipid levels and various estimates of obesity. Vague et al<sup>11</sup> introduced the 'masculine differentiation index' to distinguish between 'android' and 'gynoid' obesity. He found that a predominance of fat in the upper body (android obesity) was associated with metabolic disturbances, while a predominance of fat in the lower body (gynoid obesity) was associated only with problems such as abdominal pressure and locomotor difficulty. Following menopause fat is increasingly deposited in the upper body region which is associated with low HDL-cholesterol, high apolipoprotein-B and high triglycerides.<sup>12</sup>

Abdominal obesity has been linked to significant metabolic abnormalities including insulin resistance, hyperinsulinaemia, and elevated TAG levels as well as increased incidence of hypertension, glucose intolerance and diabetes mellitus. Abdominal adiposity as measured by waist-hip ratio (WHR), is an independent risk factor for CHD in men and perhaps also in women.<sup>13</sup>

Waist circumference and WHR are important indicators of cardiovascular risk even after adjustment for BMI. The increased visceral fat mass associated with increased waist circumference is largely the result of overall obesity, whereas in case of an increased WHR, the increase in visceral fat is due to other factors as well.<sup>14</sup>

The prevalence of obesity is increasing worldwide. Cardiovascular disease (CVD) remains the major cause of death in postmenopausal women. Before menopause, women are relatively protected from ischaemic heart disease and thromboembolism by their circulating oestrogen, but this protection is lost after menopause.<sup>2</sup>

Therefore it is important to study lipid profile in postmenopausal women with central (abdominal) obesity. So this study was designed to observe the pattern of lipid profile parameters in the postmenopausal central obese women.

### Materials and Methods

This cross sectional study was done in the department of Biochemistry, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh during the period of January 2005 to December 2005. Seventy four postmenopausal women with central obesity and age matched 56 nonobese postmenopausal women were included in the study. The subjects were selected by nonrandom purposive sampling from outpatient departments of BSMMU, Dhaka, Dhaka Medical College Hospital, Dhaka, Bashail and Shakhipur Upazilla Health Complexes, Tangail and different

places of Dhaka City. Permission for the study was taken from the concerned authorities. All the subjects included in the study were informed of the purpose of the study. Written consent was taken after detailed explanation about the study.

Central obesity was defined having WHR >0.8. Postmenopausal women with diabetes mellitus, alcoholism, chronic renal failure, nephritis, nephrotic syndrome, myocardial infarction, hypertension, and hypothyroidism were excluded from the study.

After selection of the subjects, 4 mL of blood specimen was collected from each of them in fasting condition with all aseptic precautions for estimation of serum TC, TAG and HDL-cholesterol levels. LDL-cholesterol level was calculated by applying Friedewald's formula.

All statistical analyses were done by SPSS 12.0. Unpaired Student's t test was done to compare the values between groups. p values <0.05 were considered significant.

### Results

The mean age of the postmenopausal women with central obesity was 55.80 (40–80) years and that of nonobese controls was 57.70 (40–76). Table I shows the comparison of serum lipid parameters between postmenopausal centrally obese cases and nonobese controls. Statistically there was no difference between the two groups in total cholesterol and LDL-cholesterol levels. But HDL-cholesterol was significantly lower in centrally obese postmenopausal women compared to postmenopausal nonobese controls and TAG levels were found significantly higher in the centrally obese cases compared to controls.

Table I: Comparison of serum lipid parameters between postmenopausal centrally obese cases and non-obese controls

Lipid parameters (mg/dL)	Cases (n=74) Mean ± SD	Controls (n=56) Mean ± SD	t values	p values
Total cholesterol	197.1 ± 50.6	184.9 ± 41.2	1.47	> 0.05
Triacylglycerol	149.4 ± 65.6	120.7 ± 46.3	2.91	< 0.01
HDL-cholesterol	29.6 ± 6.3	33.5 ± 7.2	3.32	< 0.01
LDL-cholesterol	137.1 ± 50.1	128.0 ± 41.5	1.10	> 0.05

## Discussion

Obesity is a well documented separate risk factor for metabolic and vascular disease, which may reduce life expectancy for overweight people.<sup>15</sup> Menopause tends to be associated with an increased risk of obesity and a shift to an abdominal fat distribution with associated increase in health risk.<sup>16</sup>

In this study we have measured serum lipid profile in 74 cases of postmenopausal central obese women and 56 nonobese control postmenopausal women. The mean serum HDL-cholesterol level was found to be significantly low and mean serum TAG level was found to be significantly high in central obese cases compared to that of controls. With respect to TC and LDL-cholesterol two groups did not differ. This finding supports other similar studies.<sup>17-19</sup>

Serum TC and LDL-cholesterol of two groups did not differ significantly, but the values of TC and LDL-cholesterol had trend to increase in central obese cases. It may be due to small sample size in our study. Carr<sup>20</sup> observed that elevated LDL-cholesterol is not a feature of dyslipidaemia in case of postmenopausal women with abdominal obesity. Our study is similar with this observation.

Many longitudinal studies have shown that TAG level increases with transition through the menopause and the increase in TAG also appears early in the postmenopausal period. Poehlman et al<sup>21</sup> found that prospective transition to postmenopause was associated with a 16% increase in TAG. It was observed in most studies that total HDL-cholesterol level falls slightly with menopause, whereas other blood lipids had no change.<sup>22</sup> Increasing TAG with menopause may be related to the fact that TAG levels are highly correlated with increasing abdominal fat content and insulin resistance.<sup>22</sup>

In this study WHR was used as indicator of central obesity. In a small study of Swedish men, it was observed that a high waist-to-hip ratio after adjustment for age and BMI was associated with an increased visceral fat area and a decreased thigh muscle area.<sup>23</sup>

The study done by Seidell et al<sup>24</sup> observed that larger waist and smaller hip circumferences than what was predicted on the basis of BMI and age are

both independently related (but in opposite direction) to risk factors such as low HDL-cholesterol, high triacylglycerol and high insulin concentration.

In this study of central obese cases we have found increased TAG, decreased HDL-cholesterol without significant differences in TC and LDL-cholesterol. Central obesity is associated with a threatening combination of metabolic abnormalities that includes dyslipidaemia (low HDL-cholesterol and high TAG), insulin resistance, glucose intolerance and hypertension which have been referred to metabolic syndrome. Individuals with this syndrome have a significantly increased risk for developing diabetes mellitus and cardiovascular disorders. So increased TAG and low HDL-cholesterol are risk factors for central obese postmenopausal women.

From the findings of the present study, it can be concluded that dyslipidaemia is a feature of postmenopausal women with central obesity. However, further prospective studies with large sample size should be carried out to evaluate the degree of dyslipidaemia of centrally obese postmenopausal women. Extensive studies should be done on central obesity in men, women and children as obesity is a global problem now-a-days. We recommend creating awareness regarding metabolic complications of central obesity in postmenopausal women as well as in general population.

## References

1. Berry EM, Brejezinski A, Dubnov G. Weight control and the management of obesity after menopause: the role of physical activity. *Maturitas* 2003; 44(2): 89-101.
2. Basurto L, Hernandez M, Zarate A. The metabolic syndrome in postmenopausal women: clinical implication. *Gac Med Mex* 2003; 139(6): 625-628.
3. Libby P. Prevention and treatment of atherosclerosis. In: DL Kasper, Fauci AS, Longo DL, Braunwald E, Hauser SL, Jameson JL (eds). *Harrison's principles of internal medicine*. 16<sup>th</sup> edn. New York: McGraw-Hill, 2005: 1430-1433.
4. Ruppelli A. Hypertension and obesity after the menopause. *J Hypertens* 2002; 20(2): S26-28.
5. Gannong WF. Energy balance, metabolism and nutrition. In: *Review of medical physiology*. 20<sup>th</sup> edn. New York: McGraw-Hill Companies, 2001: 271-306.

6. Obesity. In: Champe PC, Harvey RA, Ferrier DR (eds). Lippincott's illustrated reviews: Biochemistry. 3<sup>rd</sup> edn. Philadelphia: Lippincott Williams & Wilkins, 2005: 347-354.
7. Frier BM, Truswell AS, Shepherd J, de Looy A, Jung R. Diabetes mellitus, and nutritional and metabolic disorders. In: Haslett C, Chilver ER, Hunter JAA, Boon NA (eds). Davidson's principles and practice of medicine. 18<sup>th</sup> edn. London: Churchill Livingstone, 1999: 471-542.
8. Chang CJ, Wu CH, Yao WJ, Yang YC, Wu JS, Lu FH. Relationship of age, menopause and central obesity on cardiovascular disease risk factor in Chinese women. *International Journal of Obesity* 2000; 24: 1699-1704.
9. Loverove JA, Sliva R, Wright JW, Williams CM. Adiposity, insulin and lipid metabolism in post-menopausal women. *International Journal of Obesity* 2002; 26: 475-486.
10. Castelli WP. The triglyceride issue: a view from Framingham. *Am Heart J* 1988; 112: 432-437.
11. Vague J. The degree of masculine differentiation of obesities: a factor determining predisposition to diabetes, atherosclerosis, gout, and uric calculous disease. *Am J Clin Nutr* January 1956; 4(1): 20-34.
12. Gower BA, Nagy TR, Goran MI, Toth MJ, Poehlman ET. Fat distribution and plasma lipid-lipoprotein concentrations in pre- and postmenopausal women. *International Journal of Obesity* 1998; 22: 605-611.
13. Rexrode KM, Carey VJ, Hennekens CH, Walters EE, Colditz GA, Stampfer MJ et al. Abdominal adiposity and coronary heart disease in women. *JAMA* 1998; 280(21): 1843-1848.
14. Rexrode KM, Carey VJ, Hennekens CH, Walters EE, Colditz GA, Stampfer MJ et al. Abdominal adiposity and coronary heart disease in women. *JAMA* 1999; 281: 2284-2285.
15. Silberbauer K. Cardiovascular sequelae and risk of obesity. *Acta Med Austriaca* 1998; 25(4-5): 133-135.
16. Lovejoy PC. The menopause and obesity. *Prim Care* 2003; 30(2): 317-325.
17. Bruzell JD, Hokanson JE. Low-density and high-density lipoprotein subspecies and risk for premature coronary artery disease. *Am J Med* 1999; 107: 165.
18. Bikkina M, Larson MG, Levy D. Prognostic implications of asymptomatic ventricular arrhythmias: the Framingham Heart Study. *Ann Intern Med* 1992; 117: 990-996.
19. Lee ET, Cowan LD, Welty TK, Sievers M, Howard WJ, Oopik A et al. All-cause mortality and cardiovascular disease mortality in three American Indian populations, aged 45-74 years, 1984-1988: the Strong Heart Study. *Am. J. Epidemiol.* 1998; 147(11): 995-1008.
20. Carr MC, Brunzell JD. Abdominal obesity and dyslipidemia in the metabolic syndrome. *The Journal of Clinical Endocrinology & Metabolism* 2004; 89(6): 2601-2607.
21. Poehlman ET, Toth MJ, Gardner AW. Changes in energy balance and body composition at menopause: a controlled longitudinal study. *Ann Intern Medicine* 1995; 123: 673-675.
22. Carr MC. The emergence of the metabolic syndrome with menopause. *J Clin Endocrinol Metab* 2003; 88(6): 2404-2411.
23. Seidell JC, Bjorntorp P, Sjostrom L, Sannerstedt R, Krotkiewski M, Kvist H. Regional distribution of muscle and fat mass in men - New insight into the risk of abdominal obesity using computed tomography. *Int J Obes* 1989; 13: 289-303.
24. Seidell JC, Perusse L, Despres JP, Bouchard C. Waist and hip circumferences have independent and opposite effects on cardiovascular disease risk factors: the Quebec Family Study. *American Journal of Clinical Nutrition* 2001; 74(3): 315-321.