

Original Article

Microalbuminuria in Metabolic Obesity: A Cross-Sectional Study in a Selected Tertiary Care Hospital of Bangladesh

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Abstract

Background: Overweight and obese individuals may have no cardiometabolic risk whereas normal weight individuals may present with cardiometabolic risk. The term 'Metabolic obesity' has been floated to identify hidden metabolic risks irrespective of BMI. The pathophysiology of metabolic obesity can be explained by microvascular dysfunction and microalbuminuria is a well-known marker of microvascular dysfunction. **Objective:** The objective of this study was to find out the association of microalbuminuria with metabolic obesity in Bangladeshi adult subjects. **Materials and Methods:** This cross-sectional analytical study included 200 individuals who attended outpatient department in Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka from March 2018 to February 2019. The study subjects were divided into metabolically obese (metabolically unhealthy) group and metabolically non-obese (metabolically healthy) group by metabolic syndrome (MetS) criteria. Metabolic syndrome was defined according to the South Asian Modified-National Cholesterol Education Program (NCEP). Microalbuminuria was defined as a urinary albumin to creatinine of 30 to 300 mg/gm. Demographic profile, BP, height, weight, waist circumference etc. were measured and fasting blood glucose, serum triglyceride, serum HDL-C were estimated and albumin to creatinine ratio (ACR) was calculated. Statistical analysis was done using SPSS version 22.0. **Results:** The frequencies of metabolically obese (metabolically unhealthy) group and metabolically non-obese (metabolically healthy) group were 128 (64%) and 72 (36%) respectively. Mean values for age (p value 0.001), body mass index (p value 0.027), waist circumference ($p < 0.001$), systolic blood pressure (SBP) ($p < 0.001$) and diastolic blood pressure (DBP) ($p < 0.001$), fasting blood glucose ($p < 0.001$) and triglycerides ($p < 0.001$) were significantly higher in the metabolically obese group compared to metabolically non-obese group. Among the study subjects, the prevalence of microalbuminuria was 32.5% and prevalence of microalbuminuria was found very high (38.3%) in metabolically obese group, whereas microalbuminuria in metabolically non-obese group was found 22.2%, which was

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statistically significant (p value 0.02). Our results showed that diastolic BP ($p < 0.001$), systolic BP ($p < 0.001$), fasting blood sugar ($p < 0.001$) and triglyceride ($p < 0.008$) were significantly correlated with microalbuminuria. In the logistic regression analysis, diastolic BP (p value 0.015) and FBS (p value 0.039) were significantly associated with microalbuminuria. After harmonization of statistical analysis, our study indicated that elevated blood pressure and fasting blood sugar had strong association with microalbuminuria and are likely to be critical components that lead a substantial number of subjects to the prestage of metabolic obesity in the Bangladeshi adult population. **Conclusion:** Our study concludes that the prevalence of microalbuminuria is significantly high in metabolic obesity (metabolically unhealthy) in Bangladeshi adult population. Association of microalbuminuria with metabolic obesity is mainly attributed to high diastolic blood pressure and fasting blood glucose.

Key words: Metabolic obesity; Metabolic syndrome (MetS); Microalbuminuria; Cardiometabolic risks; BMI; NCEP

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Introduction

Obesity is frequently associated with preventable death and has emerged as a major health care challenge in the 21st century.¹ Overweight and obesity are leading causes of hypertension, cardiovascular disease (CVD), type 2 diabetes and several other morbidities.² Body mass index (BMI) is a simple index of weight-for-height that is used to assess weight status and to define overweight and obesity in adults as an international consensus.³ However, paradoxically among traditional obesity and overweight, there lies a subset of population without the expected metabolic disturbance of their excess body weight. These patients circumvented the classic models of metabolic and cardiovascular risk, and are known as the “metabolically healthy obese” (MHO) and “metabolically healthy overweight” (MHOW). On the contrary, there are individuals, who despite having “normal” weight, show an increased burden of these risks. These individuals are called “metabolically obese, normal weight individuals” (MONW).⁴ The term metabolic obesity has been floated to resolve this issue. Metabolic obesity stands for the individuals with unhealthy metabolic profile irrespective of BMI.⁵ Pajunen et al and Goday et al mentioned about metabolically healthy and

metabolically unhealthy phenotypes in different BMI groups (normal weight, overweight, obese).^{6,7} They used the components of metabolic syndrome (waist circumference, serum triglyceride, HDL-C, fasting blood sugar, blood pressure) for this classification. Individual having ≥ 3 components abnormal is regarded as metabolically unhealthy and individual having 0–2 components abnormal is regarded as metabolically healthy.^{6,7} So, finally six metabolic phenotypes are identified – metabolically healthy normal weight (MHNW), metabolically obese normal weight (MONW), metabolically healthy overweight (MHOW), metabolically obese overweight (MOOW), metabolically healthy obese (MHO) and metabolically unhealthy obese (MUO).⁸

Out of six metabolic phenotypes, metabolically obese normal weight, metabolically obese overweight, metabolically healthy obese and metabolically unhealthy obese are categorized as obesity phenotypes.⁹ Among the obesity phenotypes, MONW, MOOW and MUO were selected as ‘**metabolic obesity**’.⁸ Metabolic obesity, in fact, tends to speak out metabolically unhealthy state. So, in any of lean or overweight or obese individuals, metabolic obesity

may identify those at risk for cardiovascular disease better than the BMI-based definition of obesity. Metabolic obesity can more accurately scrutinize metabolic profile by assessing the different components of metabolic syndrome while BMI-based obesity may misinterpret some of the hidden metabolic risks. With the rapidly epidemic growth of the obesity in whole world, a better understanding of the risk factors for the complications of obesity is critical. Now-a-days, microalbuminuria has been recognized as the most important risk factor for the increased morbidity and mortality in the obese population.¹⁰⁻¹³

Microalbuminuria has currently been defined as urinary albumin : creatinine (ACR) of 30–300 mg/gm of creatinine, if measured in a spot urine collection.¹⁴ Microalbuminuria (MAU) has been recognized as an early sign of renal damage, chronic kidney disease (CKD) and an independent predictor of end-stage renal disease and cardiovascular mortality and morbidity.¹⁵ Microalbuminuria is a cardiovascular risk indicator in diabetes, hypertensive patients, and the general population, and has been shown to be associated with endothelial dysfunction and to be predictive for coronary artery disease, myocardial infarction, stroke, and all-cause mortality.¹⁶ Several studies have disclosed that metabolic syndrome is independently associated with an increased risk for chronic kidney disease (CKD) and microalbuminuria among the general population of western countries.^{17,18} Several epidemiological studies have been conducted globally to determine the relationship between microalbuminuria and components of metabolic syndrome by an early morning or random spot urine sample and a significant positive correlation was found among them.¹⁹⁻²¹ Therefore, it is logical to speculate the link between metabolic obesity, microalbuminuria, renal damage and other morbidity and mortality. Since the inception of the term metabolic obesity, all obesity-related cardio metabolic complications are now ascribed to metabolic obesity

rather than traditional obesity. Therefore, researchers and health care providers are now more concerned with metabolic obesity rather than the traditional BMI-based obesity because metabolic obesity is more inclusive to address and screen out the people at risk. Therefore, this study was designed to find out the association of microalbuminuria with metabolic obesity in a tertiary care hospital of Bangladesh.

Materials and Methods

This cross-sectional analytical study was conducted from March 2018 to February 2019. By non-probability purposive sampling, a total of 200 study subjects of both sexes, aged between 20 to 60 years, were selected from the outpatient department of Bangabandhu Sheikh Mujib Medical University. The subjects with BMI less than 18.5 kg/m², pregnancy, previous history of stroke, ischemic heart disease, chronic liver disease, chronic kidney disease and malignancy were excluded. Purpose and procedure of the study were explained in details and informed written consent was taken from each study subject. Initial evaluation of the individuals by history and clinical examination were performed and recorded in the preformed data collection sheet. Demographic profile and BP, anthropometric parameters including height, weight, and waist circumference were measured in all study subjects and BMI was calculated. Then fasting blood specimens were collected to estimate plasma glucose, serum triglyceride, and serum HDL-C. Urine specimen was collected for measurement of ACR. The study subjects were grouped into three body mass index classes (normal weight, overweight and obese with BMI 18.5–22.9, 23–27.4 and ≥ 27.5 kg/m² respectively) and also further categorized into metabolically unhealthy or healthy phenotypes by presence or absence of metabolic syndrome respectively. According to the South Asian modified NCEP criteria, metabolic syndrome was considered to be present if three or more of the following five criteria were found: abdominal obesity (waist

circumference ≥ 90 cm in men, ≥ 80 cm in women), BP (systolic ≥ 130 mm Hg and/or diastolic ≥ 85 mm Hg), pre-existing diabetes or fasting blood glucose ≥ 100 mg/dL or ≥ 5.6 mmol/L, serum triglyceride ≥ 150 mg/dL, plasma HDL-C < 40 mg/dL in men, < 50 mg/dL in women. Thus, using these criteria all the subjects finally were categorized into six metabolic phenotypes. The study subjects were designed into two groups: Metabolically obese (Metabolically unhealthy) group and Metabolically non-obese (Metabolically healthy) group. Metabolically obese (Metabolically unhealthy) group includes individuals having ≥ 3 components of metabolic syndrome, and includes the metabolic phenotypes MONW, MOOW and MUO. Metabolically non-obese (metabolically healthy) group includes individuals having 0–2 components of metabolic syndrome, and includes the metabolic phenotypes MHO, MHNW and MHOW. Microalbuminuria was evaluated in different phenotypes (MONW, MOOW, MUO) of metabolic obesity and was also compared with metabolically non-obese group. Association of microalbuminuria with metabolic obesity was evaluated.

Statistical analysis

Collected data were entered, checked, edited and then processed with the help of the software Statistical Package for Social Sciences (SPSS) version 22.0. Results were expressed as mean \pm SD. Unpaired Student's 't' test was used to compare differences in baseline characteristics between the metabolically obese group (metabolically unhealthy) and metabolically non-obese (metabolically healthy) group. Chi-square test was used for the comparison of qualitative data. Pearson's correlation was performed to correlate selective risk factors with microalbumin levels. We evaluated the association between metabolic syndrome components and microalbuminuria using logistic regression analysis. p value < 0.05 was considered as statistically significant.

Results

MONW, MOOW, MUO, MHO, MHOW and MHNW

were 12 (6%), 46 (23%), 70 (35%), 26 (13%), 26 (13%) and 20 (10%) respectively. Among them, MUO and MOOW phenotypes were found to predominate with lowest frequency in MONW phenotype (Table I). In our study, the frequencies of metabolically obese (metabolically unhealthy) group and metabolically non-obese (metabolically healthy) group were 128 (64%) and 72 (36%) respectively (Table II). In our study, out of total population of 200, aged older than 20 years, about 154 subjects were found as obesity phenotypes, of which MONW, MOOW, MHO and MUO were 12 (7.8%), 46 (29.9%), 26 (16.9%) and 70 (45.4%) respectively (Table III). Mean values for age (p value 0.001), body mass index (p value 0.027), waist circumference ($p < 0.001$), systolic blood pressure (SBP) ($p < 0.001$), diastolic blood pressure (DBP) ($p < 0.001$), fasting blood glucose ($p < 0.001$) and triglycerides ($p < 0.001$) were significantly higher in the metabolically obese group compared to metabolically non-obese group. HDL-C level was significantly lower ($P < 0.001$) in the metabolically obese group compared to metabolically non-obese group (Table IV). Among the study subjects, the prevalence of microalbuminuria was 32.5%. Our study showed that prevalence of microalbuminuria was found very high (38.3%) in metabolically obese group whereas microalbuminuria in metabolically non-obese group was found 22.2%, which was statistically significant (p value 0.02) (Table V). In the metabolically obese (metabolically unhealthy) group, we found microalbuminuria more frequent (59.18%) in MUO (Table VI). Our results showed that diastolic BP ($p < 0.001$), systolic BP ($p < 0.001$), fasting blood sugar ($p < 0.001$) and triglyceride ($p < 0.008$) significantly correlated with microalbuminuria (Table VII). In the logistic regression analysis, diastolic BP (p value 0.015) and FBS (p value 0.039) were significantly associated with microalbuminuria (Table VIII).

Table I: Frequency of metabolic phenotypes among the study subjects (n=200)

Metabolic phenotypes	Frequency	Percentage	95% CI
MONW (Obesity phenotype)	12	6.0	3.0–9.5
MOOW (Obesity phenotype)	46	23.0	17.0–29.0
MUO (Obesity phenotype)	70	35.0	28.5–41.0
MHO (Obesity phenotype)	26	13.0	8.5–18.0
MHOW	26	13.0	8.5–18.0
MHNW	20	10.0	6.5–14.5

MONW = Metabolically obese normal weight, MOOW = Metabolically obese overweight,

MUO = Metabolically unhealthy obese, MHO = Metabolically healthy obese,

MHOW = Metabolically healthy overweight, MHNW = Metabolically healthy normal weight

Table II: Frequency of metabolically obese (metabolically unhealthy) group and metabolically non-obese (metabolically healthy) group among the study subjects (n=200)

Groups	Frequency	Percentage	95% CI
Metabolically obese	128	64.0	57.0 –70.5
Metabolically non-obese	72	36.0	29.5–43.0

Table III: Frequency of different obesity phenotypes among adult obese individuals (n=154)

Obesity phenotypes	Frequency	Percentage	95% CI
MONW	12	7.8	3.9–12.3
MOOW	46	29.9	22.7–37.7
MUO	70	45.4	37.7–53.9
Healthy obese (MHO)	26	16.9	11.0–23.4

Table IV: Comparison of anthropometric measurements and biochemical parameters between metabolically obese (metabolically unhealthy) group and metabolically non-obese (metabolically healthy) group (n=200)

Variables	Metabolically obese (n=128) Mean±SD	Metabolically non-obese (n=72) Mean±SD	p values
Age (years)	45.37±10.78	40.17±10.98	0.001
WC (cm)	95.92±9.55	88.68±9.52	<0.001
BMI (kg/m ²)	28.48±5.21	26.76±5.26	0.027
FBS (mmol/L)	8.20±3.23	6.05±2.22	<0.001
TG (mg/dL)	202.13±109.52	128.93±67.81	<0.001
HDL (mg/dL)	37.91±7.34	42.14±9.29	<0.001
SBP (mm Hg)	129.73±16.71	114.86±14.82	<0.001
DBP (mm Hg)	85.43±13.47	79.24±10.54	<0.001

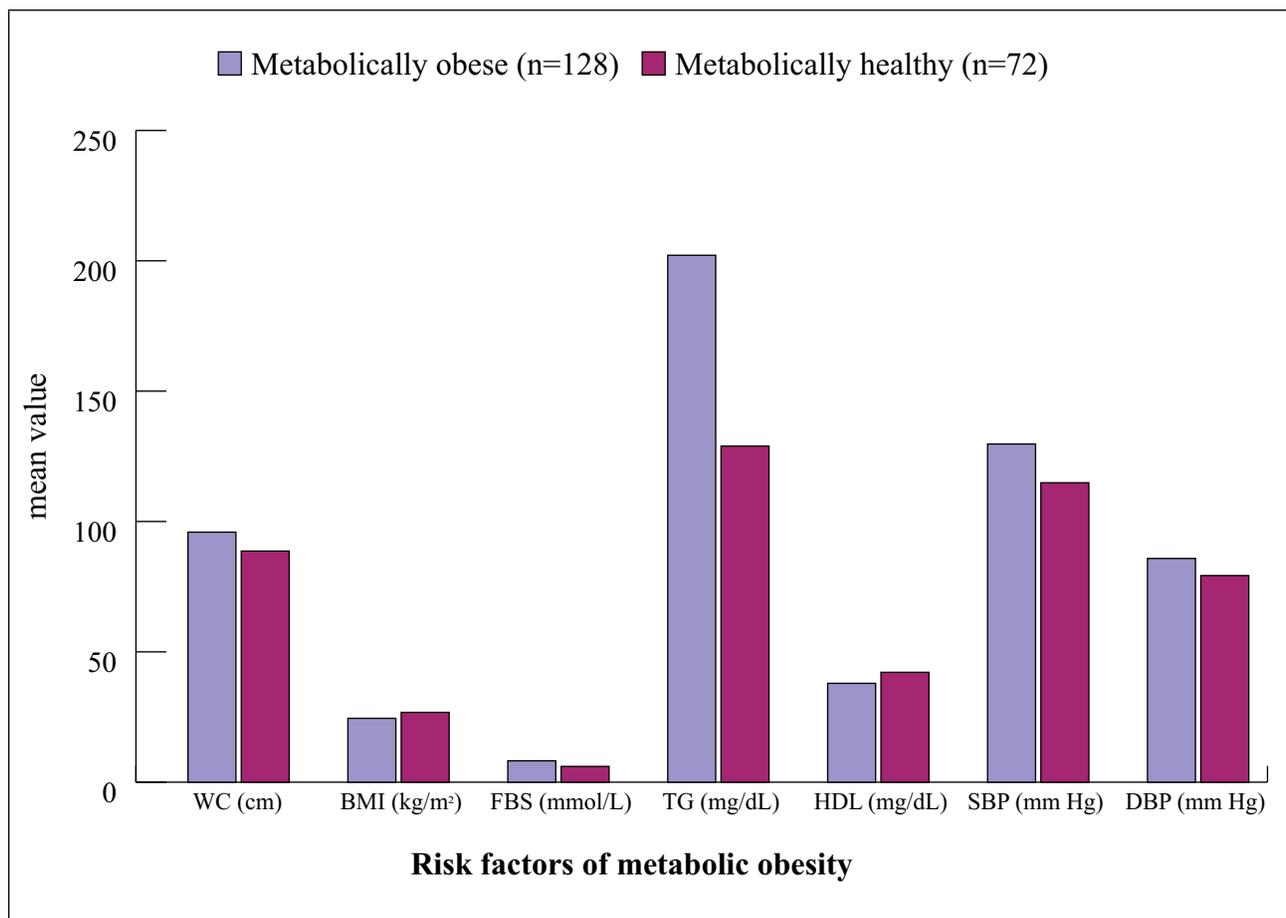


Fig 1. Multiple bar diagram showing anthropometric and biochemical parameters between metabolically obese (metabolically unhealthy) and metabolically non-obese (metabolically healthy) group

Table V: Comparison of microalbuminuria between metabolically obese (metabolically unhealthy) group and metabolically non-obese (metabolically healthy) group among the study subjects (n=200)

Parameters	Metabolically obese (n=128) Number (%)	Metabolically non-obese (n=72) Number (%)	Total (n=200)	p value
Microalbuminuria	49 (38.3)	16 (22.2)	65 (32.5%)	0.02
No microalbuminuria	79 (61.7)	56 (77.8)	135 (67.5%)	

Table VI: Comparison of microalbuminuria in different phenotypes of metabolic obesity (metabolically unhealthy) (n=49)

Metabolic obesity phenotypes	Microalbuminuria		95% CI
	Number	Percentage	
MONW	3	6.12	0.0-50.0
MOOW	17	34.70	23.9-50.0
MUO	29	59.18	30.0-52.9

Table VII: Correlation of microalbuminuria with selective risk factors (n=128)

Microalbuminuria (MAU)	Selective risk factors	Pearson's correlation	
		r values	p values
ACR	WC (cm)	0.084	0.346
	FBS (mmol/L)	0.446	0.001
	Triglyceride (mg/dL)	0.235	0.008
	HDL cholesterol (mg/dL)	0.096	0.279
	SBP (mm of Hg)	0.518	0.001
	DBP (mm of Hg)	0.549	0.001

ACR–Albumin to creatinine ratio, WC–Waist circumference, BMI–Body mass index, FBS–Fasting blood sugar, TG–Triglycerides, HDL-C–High density lipoprotein cholesterol, SBP–Systolic blood pressure, DBP–Diastolic blood pressure

Table VIII: Association of components of metabolic syndrome with microalbuminuria (n=200)

Variables	β	S.E.	p values	OR	95% CI	
WC (cm)	0.014	0.017	0.419	1.014	0.981	–1.047
FBS (mmol/L)	0.120	0.058	0.039	1.127	1.006	–1.263
TG (mg/dL)	–0.004	0.002	0.055	0.996	0.992	–1.000
HDL (mg/dL)	–0.015	0.022	0.489	0.985	0.944	–1.028
SBP (mm Hg)	0.018	0.017	0.274	1.019	0.986	–1.053
DBP (mm Hg)	0.058	0.024	0.015	1.059	1.011	–1.110

Discussion

Metabolically obese (metabolically unhealthy) group includes MONW, MOOW and MUO. Metabolically non obese (metabolically healthy) group includes MHO, MHNW and MHOW. This group distribution was also done by Pajunen et al⁶ and Goday et al⁷ in their studies where they brought out the metabolically healthy and unhealthy phenotypes in different BMI groups. Metabolic obesity stands for the individuals with unhealthy metabolic profile irrespective of BMI. We used the metabolic syndrome (MetS) criteria of the South Asian Modified-National Cholesterol Education Program and WHO Asian BMI cut-off to identify metabolic healthy and unhealthy phenotypes, of which MHO, MHOW, MHNW, MONW, MOOW and MUO were 26 (13%), 26 (13%), 20 (10%), 12 (6%), 46 (23%) and 70 (35%) respectively. Among them, MUO and MOOW phenotypes were

found to predominate with lowest frequency in MONW phenotype. In our study, the frequencies of metabolically obese (metabolically unhealthy) group and metabolically non-obese (metabolically healthy) group were 128 (64%) and 72 (36%) respectively. Azizi et al²² reported in their study of an urban area in Tehran that out of a total population of 10,368 aged older than 20 years, about 33% were diagnosed as metabolically unhealthy according to ATP III criteria. As there are currently no international unified criteria for defining metabolic obesity, it is difficult to compare these results with the studies from other countries.

In our study, out of total population of 200, aged older than 20 years, about 154 subjects were found as obesity phenotypes, of which MONW, MOOW, MHO and MUO were 12 (7.8%), 46 (29.9%), 26 (16.9%)

and 70 (45.4%) respectively. These data are supported by Pajunen et al⁶ and Wildman et al⁸. Pajunen et al⁶ conducted a cross sectional study on general population aged 45-74 years in Finland and found the prevalence of obesity phenotypes as follows: MOOW 28.5%, MUO 21.3%, MONW 7.2%, MHO 3.3%. Wildman et al⁸ found this prevalence among US population as follows: MUO 20.9%, MOOW 17%, MHO 9.7% and MONW 8.1%.

In this study, age, waist circumference, body mass index (BMI), fasting blood sugar, triglyceride, systolic blood pressure and diastolic blood pressure levels were significantly higher and HDL-C level was significantly lower in the metabolically obese group (metabolically unhealthy) than in the metabolically non-obese (metabolically healthy) group. Our findings correlated with the study done by Wildman et al⁸ in US populations where they found that individuals without metabolic syndrome had lower fasting blood glucose, better lipid profile, lower CVD score and less estimated liver fat compared to subjects with metabolic syndrome.

Among the study subjects, the prevalence of microalbuminuria was 32.5%. However, Lin et al²³ showed that the prevalence of microalbuminuria was 11.5% in the population of Taiwan, ≥ 40 years and Hao et al²⁴ found that the prevalence of microalbuminuria was 13.7% in 2321 Japanese. The discrepancy in the prevalence of microalbuminuria across this population might be attributable to the differences in the characteristics of study participants, such as age, race, life style and cardiovascular risk factors.

Our study showed that prevalence of microalbuminuria was found very high (38.3%) in metabolically obese group, whereas microalbuminuria in metabolically non-obese group was found 22.2%, which was statistically significant (p value 0.02). Our findings on the association of microalbuminuria with metabolic obesity were in line with the results of recently published studies. The prevalence of microalbuminuria was consistently higher (p<0.0001) in persons with the metabolic syndrome than those without in Chinese (20.3% vs 2.0%),²⁵ in Japanese (20.8% vs 12.2%)²⁴ and in US Americans (13.7% vs 4.8%)¹⁷.

In the metabolically obese (metabolically unhealthy) group, we found frequency of microalbuminuria in MONW, MOOW and MUO were 6.12%, 34.70% and 59.18% respectively. A recent study by Choi et al²⁶ reported that MUO group was at increased risk of developing microalbuminuria than MONW and MOOW groups. Our study suggested that metabolic disorder was associated with the development of microalbuminuria which was consistent with the findings of previous studies that emphasized the importance of metabolic health.²⁷⁻²⁹

Studies have yielded mixed results for the association between metabolic obesity and microalbuminuria. Chen et al³⁰ reported a positive association between elevated BP, reduced HDL-C, elevated TG and microalbuminuria in a cross sectional study of US adults. Hao et al²⁴ reported a positive association between high FBS, high BP, obesity and microalbuminuria in a cross sectional study of 2321 adults. Based on the impact of various components of metabolic syndrome on chronic kidney disease, a study conducted by Song et al³¹ presented the concept of dividing all five components of metabolic syndrome into a critical arm including elevated BP, reduced HDL-C and elevated FBS and a noncritical arm including elevated TG and obesity. Therefore, the impact of the components of metabolic syndrome on microalbuminuria should be interpreted judiciously. Our results showed that diastolic BP (p<0.001), systolic BP (p<0.001), fasting blood sugar (p<0.001) and triglyceride (p<0.008) were significantly correlated with microalbuminuria. In the logistic regression analysis, diastolic BP (p value 0.015) and FBS (p value 0.039) were significantly associated with microalbuminuria. After harmonization of statistical analysis, our study indicated that elevated blood pressure and fasting blood sugar had strong association with microalbuminuria and are likely to be critical components that lead a substantial number of subjects to the prestage of metabolic obesity in the Bangladeshi adult population.

Limitations

Study population was selected from Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka

and the study was conducted in a short period of time with a small sample size. So, the results of the study may not reflect the exact picture of the whole country.

First, its cross-sectional design made it difficult to infer causality between metabolic obesity and microalbuminuria.

Second, microalbuminuria was assessed only once, which could have resulted in some misclassifications of microalbuminuria.

Third, we did not consider subjects receiving lipid-lowering therapy to have high triglyceride or low HDL-C levels, because we could not ascertain the exact purpose of this medication given the clinical situation that lipid-lowering agents can be prescribed to decrease low-density lipoprotein cholesterol or triglyceride levels or to increase HDL-C levels.

Fourth, our study had a relatively small sample size. Only 49 persons had microalbuminuria among 128 metabolic obese persons. However, this shortfall might be overcome by intensive and accurate measurement of all the variables under study.

Recommendation

This study was conducted in 200 healthy adult individual. It is recommended to conduct a study with a larger sample size. These suggest that the prevention of several chronic diseases is necessary if the signs of metabolic disorder are evident regardless of normal body weight. One of the important implications of our study is that we probably should consider to screen microalbuminuria in persons with pre-hypertensions or pre-diabetes and to identify those at high renal and cardiovascular risk. More comprehensive and intensive management of metabolic obesity at its early stage should be started to prevent the progression of renal injury and cardiovascular complication.

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