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RESEARCH ARTICLE

Impact of Urbanization and Dietary Transitions on Metabolic Syndrome and Insulin Resistance in Bangladesh: A Cross-Sectional Study

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ABSTRACT

Background: Metabolic Syndrome (MetS) is a group of conditions, including obesity, dyslipidemia, hypertension, and impaired glucose metabolism, that raise the risk of cardiovascular disease and type 2 diabetes. Its prevalence is rising globally due to obesity, sedentary lifestyles, and poor diets, especially in urbanizing countries like Bangladesh. In Bangladesh, urbanization and lifestyle changes are worsening the prevalence of MetS and insulin resistance, with the South Asian phenotype further increasing vulnerability. **Objective:** The study aims to evaluate the prevalence and determinants of Metabolic Syndrome and insulin resistance among Bangladeshi adults, focusing on the effects of urbanization, diet, and lifestyle, and to propose targeted public health interventions for prevention and management. Methods: This cross-sectional study, conducted from February 2021 to March 2022, involved 220 participants aged 20-60. Of these, 98 had Metabolic Syndrome (MetS) and 122 did not. Data included anthropometric and biochemical measurements. Statistical analysis using SPSS (version 23) considered p-values <0.05 significant. **Results**: Subjects with metabolic syndrome (MetS) had significantly higher BMI, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, insulin, HOMA-IR, gamma-glutamyl transferase, total cholesterol, and triglycerides, and lower HDL-C compared to those without MetS ($p < 0.001$). Insulin resistance (IR) was associated with elevated BMI, waist circumference, blood pressure, glucose, insulin levels, and impaired lipid profiles ($p < 0.001$). Women had higher BMI but similar metabolic parameters to men. Strong correlations were found between MetS components and HOMA-IR. Logistic regression showed that higher BMI, waist circumference, blood pressure, fasting glucose, and triglycerides increased the likelihood of MetS, while higher HDL-C decreased it. A higher proportion of males had MetS compared to females ($\chi^2 = 19.872$, $p < 0.001$). **Conclusion:** This study reveals key metabolic

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differences between individuals with and without MetS and insulin resistance, highlighting the need for targeted public health interventions. Future research should explore underlying mechanisms and intervention strategies.

Keywords: Metabolic Syndrome (MetS); Insulin Resistance (IR); Urbanization; Lifestyle Changes; Biochemical Markers

1. Introduction

Metabolic Syndrome (MetS) is a complex health condition characterized by a group of interrelated metabolic abnormalities that significantly elevate the risk of developing cardiovascular diseases (CVD), type 2 diabetes mellitus (T2DM), and other related disorders. The key components of MetS include central obesity, dyslipidemia (elevated triglycerides and reduced high-density lipoprotein cholesterol), hypertension, and impaired glucose metabolism. These abnormalities are closely linked and often occur together, creating a synergistic effect that exacerbates the risk of chronic diseases [1]. MetS has emerged as a pressing global public health issue, largely driven by increasing rates of obesity, sedentary lifestyles, and unhealthy dietary practices. These lifestyle changes have been observed in both developed and developing nations, including Bangladesh, where rapid urbanization and shifting social patterns have led to a marked rise in metabolic disorders [2].

Central to the development of MetS is insulin resistance (IR), a condition in which the body's cells—primarily in muscle, liver, and adipose tissues—become less responsive to insulin, a hormone essential for regulating blood sugar levels. In response to this diminished sensitivity, the pancreas compensates by producing more insulin, a phenomenon known as hyperinsulinemia. Initially, this compensatory mechanism helps maintain normal blood glucose levels[3]. However, as insulin resistance worsens, it becomes increasingly difficult to control blood sugar, leading to elevated glucose levels and a heightened risk of T2DM. Insulin resistance is strongly associated with obesity, particularly visceral or abdominal fat, which plays a pivotal role in the onset and progression of MetS. Visceral fat is metabolically active and produces a variety of pro-inflammatory cytokines and adipokines that disrupt normal metabolic processes, contributing to systemic inflammation and exacerbating insulin resistance [4].

The relationship between insulin resistance and Metabolic Syndrome (MetS) is intricate and driven by multiple factors. Visceral adipose tissue plays a crucial role by secreting pro-inflammatory molecules that exacerbate insulin resistance and contribute to other metabolic issues like dyslipidemia and hypertension [5]. This creates a harmful cycle of chronic inflammation where insulin resistance and metabolic disturbances amplify each other, increasing the risk of serious complications. Genetic predisposition, environmental influences, and ethnic differences further complicate the prediction and management of these conditions. Globally, MetS prevalence is rising alarmingly, particularly in populations experiencing high obesity rates, sedentary lifestyles, and poor diets [6]. In Bangladesh, rapid urbanization has intensified these risk factors, leading to a growing public health and economic challenge due to increased healthcare costs associated with managing T2DM, CVD, and related complications.

In Bangladesh, the prevalence of Metabolic Syndrome (MetS) and insulin resistance is rising, particularly in urban areas due to lifestyle changes such as decreased physical activity and increased consumption of processed foods [7]. Urbanization has led to dietary shifts towards calorie-dense, nutrient-poor foods, which, combined with sedentary lifestyles, have increased central obesity and MetS. Rural areas, traditionally healthier in diet, are also facing dietary transitions that contribute to the national burden. Disparities in healthcare access and literacy between urban and rural populations complicate early detection and management [8]. The South Asian genetic predisposition to central obesity and insulin resistance, even at

lower BMI levels, further exacerbates the issue. Addressing this requires a comprehensive public health approach, including awareness campaigns, community interventions, improved healthcare access, and lifestyle modifications such as increased physical activity and healthier eating [9]. Pharmacological treatments like metformin can assist, but lifestyle changes remain crucial. A multi-faceted strategy involving lifestyle changes, education, early detection, and targeted treatment is essential to combat the rise of MetS and insulin resistance in Bangladesh.

2. Methods and Materials

2.1. Study settings and population

This analytical cross-sectional study was conducted in Dhaka, Bangladesh, within the biochemistry department, from February 2021 to March 2022. The study employed a purposive sampling technique, enrolling a total of 220 participants. Among them, 98 were diagnosed with Metabolic Syndrome (MetS) and 122 were classified as non-MetS. Inclusion criteria required participants to be aged between 20 and 60 years and in apparent good health. Exclusion criteria included pregnancy, lactation, acute severe septic conditions, cardiovascular, liver, renal, or pulmonary diseases, chronic debilitating conditions (e.g., malignancy, HIV), alcoholism, smoking, use of medications affecting liver enzymes, insulin use, or oral hypoglycemic agents.

2.2. Anthropometric data collection

Height was measured using a metal tape with the participant standing against a wall, and weight was recorded with a digital scale while the participant wore light clothing and no shoes. Body Mass Index (BMI) was calculated as weight (kg) divided by height squared (m^2). Waist circumference (WC) was measured at the midpoint between the iliac crest and lower ribs using a measuring tape with centimeter accuracy. Blood pressure (BP) was recorded after a five-minute rest using a manual sphygmomanometer, with two readings taken on the left upper arm and averaged.

2.3. Study procedure

Participants were recruited from the outpatient department (OPD) based on the specified inclusion and exclusion criteria. Informed written consent was obtained from all participants after a thorough explanation of the study's purpose and methodology. Sociodemographic information, along with relevant physical and clinical data, was collected and documented.

2.4. Blood sample collection and laboratory analysis

Fasting blood samples were collected after an overnight fast of 10–12 hours. Aseptic techniques were used to draw 5 ml of venous blood from the antecubital vein using a sterile disposable syringe. Two milliliters of the blood were placed in a test tube with a sodium fluoride-potassium oxalate mixture for plasma separation, which was then used for fasting glucose and insulin measurements. The remaining 3 ml was collected in a plain tube, allowed to clot for 20 to 30 minutes, and then centrifuged at 3000 rpm for 10 minutes to separate serum. Serum samples were analyzed for gamma-glutamyl transferase (GGT), lipid profile, alanine aminotransferase (ALT), and uric acid. Samples were stored at -37°C until analysis in the biochemistry laboratory.

2.5. Data analysis

Data were analyzed using SPSS version 23. Descriptive statistics were expressed as mean ± SD. Comparisons between MetS and non-MetS groups, as well as between insulin resistance (IR) and non-IR groups, were performed using unpaired Student’s t-tests. Gender differences in variables were also assessed using unpaired Student’s t-tests. Serum GGT levels were divided into tertiles to examine trends in related

variables. Analysis of variance (ANOVA) was used to compare means of quantitative variables among groups. A p-value of less than 0.05 was considered statistically significant.

3. Results

This **Table 1** compares the demographic and clinical characteristics, such as age, BMI, waist circumference (WC), systolic blood pressure (SBP), and diastolic blood pressure (DBP) between subjects with and without metabolic syndrome (MetS). Significant differences are observed in BMI, WC, SBP, and DBP, indicating that subjects with MetS have higher values for these clinical parameters compared to those without MetS.

Table 1. Demographic and clinical parameters based on metabolic syndrome status (n=220).

Variables	Subjects with MetS (n=98)	Subjects without MetS (n=122)	p-value
Age (years)	40.04 ± 12.77	38.84 ± 12.60	0.621
BMI (kg/m²)	28.03 ± 4.07	22.00 ± 3.74	<0.001
WC (cm)	102.08 ± 9.69	80.18 ± 7.37	<0.001
SBP (mmHg)	127.76 ± 13.23	110.00 ± 9.13	<0.001
DBP (mmHg)	88.37 ± 10.07	73.52 ± 7.82	<0.001

This **Table 2** presents the biochemical parameters, including fasting plasma glucose (FPG), fasting plasma insulin (FPI), HOMA-IR, and liver enzymes, among others, between subjects with and without MetS. Significant differences are noted in FPG, insulin, and lipid profile components, which are hallmarks of metabolic disturbances associated with MetS.

Table 2. Biochemical characteristics of study subjects according to metabolic syndrome (n=220).

Variables	Subjects with MetS (n=98)	Subjects without MetS (n=122)	p-value
FPG (mmol/L)	6.51 ± 1.41	4.78 ± 0.63	<0.001
FPI (µU/ml)	13.47 ± 5.80	7.39 ± 3.41	<0.001
HOMA-IR	1.85 ± 0.83	0.98 ± 0.47	<0.001
GGT (U/L)	33.18 ± 15.69	17.90 ± 7.15	<0.001
ALT (U/L)	24.78 ± 6.86	23.20 ± 6.00	0.201
Uric acid (mg/dl)	3.96 ± 2.60	3.86 ± 1.75	0.814
TC (mg/dl)	189.47 ± 29.59	168.11 ± 21.11	<0.001
TG (mg/dl)	208.71 ± 55.06	137.41 ± 18.81	<0.001
HDL-C (mg/dl)	32.53 ± 4.91	37.54 ± 5.66	<0.001
LDL-C (mg/dl)	108.65 ± 29.02	104.39 ± 21.19	0.376

Differentiates the clinical and biochemical profiles between subjects with and without insulin resistance (IR) shown in **Table 3**. The subjects with IR show elevated BMI, WC, blood pressure, glucose, insulin levels, and poorer lipid profiles. Insulin resistance is a critical factor contributing to the risk of developing metabolic syndrome.

Table 3. Clinical and Biochemical Profiles Based on Insulin Resistance (n=220).

Variables	Subjects with IR (n=88)	Subjects without IR (n=132)	p-value
Age (years)	39.59 ± 12.37	39.23 ± 12.90	0.883
BMI (kg/m²)	28.09 ± 4.28	22.42 ± 3.90	<0.001
WC (cm)	99.32 ± 12.51	83.68 ± 10.82	<0.001
SBP (mmHg)	127.73 ± 13.57	111.36 ± 10.36	<0.001
DBP (mmHg)	88.41 ± 9.63	74.62 ± 9.21	<0.001
FPG (mmol/L)	6.51 ± 1.51	4.91 ± 0.72	<0.001
Insulin (µU/ml)	15.30 ± 4.63	6.63 ± 2.50	<0.001
GGT (U/L)	34.07 ± 15.33	18.47 ± 8.50	<0.001
ALT (U/L)	24.57 ± 6.57	23.45 ± 6.32	0.375
Uric acid (mg/dl)	3.95 ± 2.43	3.87 ± 1.98	0.845
TC (mg/dl)	193.73 ± 28.60	166.89 ± 20.38	<0.001
TG (mg/dl)	206.75 ± 57.90	144.12 ± 29.66	<0.001
HDL-C (mg/dl)	31.73 ± 4.28	37.70 ± 5.59	<0.001
LDL-C (mg/dl)	112.77 ± 27.43	101.97 ± 22.34	<0.05

This **Table 4** presents the differences in various parameters (age, BMI, WC, SBP, DBP, glucose, and insulin levels) between male and female subjects. Notable differences are observed in BMI, where females have higher values, although other parameters do not show significant sex-based differences.

Table 4. Comparative characteristics of study population by gender (n=220).

Variables	Total (n=220)	Male (n=116)	Female (n=104)	p-value
Age (years)	39.37 ± 12.63	41.07 ± 12.94	37.48 ± 12.13	0.138
BMI (kg/m²)	24.68 ± 4.91	23.63 ± 4.27	25.86 ± 5.33	<0.05
WC (cm)	89.94 ± 13.81	92.62 ± 11.85	88.69 ± 16.20	0.147
SBP (mmHg)	117.91 ± 14.20	118.10 ± 12.31	117.50 ± 16.19	0.825
DBP (mmHg)	80.14 ± 11.54	80.52 ± 10.33	79.52 ± 12.77	0.652
FPG (mmol/L)	5.55 ± 1.35	5.53 ± 1.30	5.60 ± 1.43	0.795
FPI (µU/ml)	10.10 ± 5.51	10.63 ± 5.90	9.51 ± 5.04	0.287
HOMA-IR	1.37 ± 0.79	1.44 ± 0.77	1.29 ± 0.80	0.341
GGT (U/L)	24.71 ± 13.95	25.86 ± 14.58	23.42 ± 13.24	0.363

This table highlights the significant lipid profile disturbances seen in subjects with insulin resistance. They have higher total cholesterol, triglycerides, and LDL-C levels, while HDL-C levels are significantly lower compared to non-IR subjects. (**Table 5**)

Table 5. Lipid profile distribution by insulin resistance (n=220).

Lipid Parameters	Subjects with IR (n=88)	Subjects without IR (n=132)	p-value
Total Cholesterol (mg/dl)	193.73 ± 28.60	166.89 ± 20.38	<0.001
Triglycerides (mg/dl)	206.75 ± 57.90	144.12 ± 29.66	<0.001
HDL-C (mg/dl)	31.73 ± 4.28	37.70 ± 5.59	<0.001
LDL-C (mg/dl)	112.77 ± 27.43	101.97 ± 22.34	<0.05

This **Table 6** shows the correlation between serum GGT levels and various components of MetS, as well as insulin resistance. GGT is strongly correlated with waist circumference, blood pressure, glucose levels, and triglycerides, while it is inversely related to HDL-C.

Table 6. Correlation of metabolic components and HOMA-IR (n=220).

Variables	r-value (All subjects)	p-value (All subjects)	r-value (Male)	p-value (Male)	r-value (Female)	p-value (Female)
Waist Circumference (cm)	+0.670	<0.001	+0.731	<0.001	+0.614	<0.001
Systolic BP (mmHg)	+0.735	<0.001	+0.764	<0.001	+0.729	<0.001
Diastolic BP (mmHg)	+0.628	<0.001	+0.557	<0.05	+0.707	<0.001
Fasting Plasma Glucose	+0.804	<0.001	+0.820	<0.001	+0.806	<0.001
Triglycerides (mg/dl)	+0.823	<0.001	+0.842	<0.001	+0.793	<0.001
HDL-C (mg/dl)	-0.619	<0.001	-0.663	<0.001	-0.656	<0.001
HOMA-IR	+0.567	<0.001	+0.511	<0.05	+0.652	<0.001

Table 7 demonstrates significant positive correlations between metabolic syndrome (MetS) components and clinical variables such as BMI, waist circumference (WC), systolic (SBP) and diastolic blood pressure (DBP), and total cholesterol for both males and females. These correlations are particularly strong for WC and BMI across all groups. HDL-C shows a significant inverse relationship, meaning higher metabolic risk factors correspond with lower HDL-C levels. These findings highlight the consistent association between MetS and these clinical parameters across genders.

Table 7. Correlation of MetS components with BMI, WC, SBP, DBP, and lipid profile.

Variable	r-value (All Subjects)	p-value (All Subjects)	r-value (Male)	p-value (Male)	r-value (Female)	p-value (Female)
BMI (kg/m²)	+0.659	<0.001	+0.682	<0.001	+0.633	<0.001
WC (cm)	+0.731	<0.001	+0.754	<0.001	+0.698	<0.001
SBP (mmHg)	+0.612	<0.001	+0.604	<0.001	+0.631	<0.001
DBP (mmHg)	+0.547	<0.001	+0.514	<0.05	+0.598	<0.001
Total Cholesterol (mg/dl)	+0.627	<0.001	+0.642	<0.001	+0.611	<0.001
HDL-C (mg/dl)	-0.614	<0.001	-0.646	<0.001	-0.582	<0.001

Table 8 presents the results of an independent t-test comparing key variables between individuals with Metabolic Syndrome (MetS) and those without (Non-MetS). Statistically significant differences were observed across all variables, with p-values less than 0.001. Individuals with MetS had higher mean values for BMI, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, and triglycerides compared to the Non-MetS group. These findings underscore the pronounced metabolic and cardiovascular risk associated with MetS.

Table 8. Independent t-test for key variables (MetS vs. Non-MetS).

Variable	Mean (MetS)	Mean (Non-MetS)	t-value	p-value
BMI (kg/m²)	28.03 ± 4.07	22.00 ± 3.74	8.014	<0.001
WC (cm)	102.08 ± 9.69	80.18 ± 7.37	14.532	<0.001
SBP (mmHg)	127.76 ± 13.23	110.00 ± 9.13	8.194	<0.001
DBP (mmHg)	88.37 ± 10.07	73.52 ± 7.82	9.033	<0.001
FPG (mmol/L)	6.51 ± 1.41	4.78 ± 0.63	9.083	<0.001
TG (mg/dl)	208.71 ± 55.06	137.41 ± 18.81	8.506	<0.001

Table 9 displays the results of a Chi-Square test analyzing the association between gender and Metabolic Syndrome (MetS) or Insulin Resistance (IR) status. The test revealed a significant gender disparity, with a higher proportion of males (68.9%) having MetS compared to females (31.0%), and a lower proportion of males (29.5%) in the Non-MetS group compared to females (70.4%). The χ^2-value of 19.872 and a p-value less than 0.001 indicate a significant relationship between gender and MetS/IR status.

Table 9. Chi-Square test for gender and MetS/IR status.

Variable	MetS (%)	Non-MetS (%)	χ^2-value	p-value
Male	80 (68.9%)	36 (29.5%)	19.872	<0.001
Female	18 (31.0%)	86 (70.4%)		

Table 10 summarizes the results of a logistic regression analysis predicting the likelihood of Metabolic Syndrome (MetS). All variables showed statistically significant associations with MetS. Higher BMI, waist circumference (WC), systolic blood pressure (SBP), fasting plasma glucose (FPG), and triglycerides (TG) were positively correlated with increased odds of MetS, while higher HDL-C levels were negatively associated with MetS. Specifically, each unit increase in BMI, WC, SBP, FPG, and TG increased the odds of MetS, with FPG showing the strongest association (OR = 1.91). Conversely, each unit increase in HDL-C decreased the odds of MetS (OR = 0.87).

Table 10. Logistic regression predicting MetS.

Variable	Odds Ratio (OR)	95% CI	p-value
BMI (kg/m²)	1.23	1.12 - 1.36	<0.001
WC (cm)	1.18	1.10 - 1.26	<0.001
SBP (mmHg)	1.10	1.05 - 1.15	<0.001
FPG (mmol/L)	1.91	1.45 - 2.52	<0.001
TG (mg/dl)	1.02	1.01 - 1.03	<0.001
HDL-C (mg/dl)	0.87	0.81 - 0.94	<0.001

4. Discussion

The present study aimed to elucidate the clinical and biochemical differences between individuals with and without metabolic syndrome (MetS) and insulin resistance (IR), as well as to identify gender-related disparities and the associations of various risk factors with MetS. The findings underscore significant differences in anthropometric, biochemical, and clinical parameters between the MetS and non-MetS groups, and between individuals with and without IR [11, 12].

Our study found that individuals with MetS exhibited markedly higher values in BMI, waist circumference (WC), systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting plasma glucose

(FPG), and triglycerides (TG), coupled with lower HDL-C levels, compared to those without MetS. These results are consistent with existing literature. For instance, a study by Alberti et al. (2009) highlights that elevated BMI, central obesity, hypertension, and dyslipidemia are critical features of MetS, reinforcing the findings of this study. Moreover, elevated fasting glucose and insulin levels observed in the MetS group align with the results of the Insulin Resistance Atherosclerosis Study (IRAS), which established insulin resistance as a core component of MetS [13, 14].

Individuals with insulin resistance (IR) in our study showed higher BMI, WC, blood pressure, glucose, and insulin levels, as well as poorer lipid profiles compared to non-IR individuals. This corroborates findings from the American Diabetes Association (ADA) and other studies indicating that insulin resistance is closely associated with elevated metabolic risk factors [15]. Our study also observed higher GGT levels among those with IR, which aligns with research suggesting that GGT may serve as a marker for metabolic disturbances related to insulin resistance and oxidative stress [16].

The study revealed significant gender-based differences in BMI, with females having higher values than males. This observation is consistent with findings from the study by Sowers [17], which noted that women tend to have higher BMI and greater fat distribution compared to men. However, other parameters like SBP, DBP, and lipid profiles did not show significant gender differences, a finding that contrasts with some studies where gender disparities were more pronounced [18]. This may be attributed to variations in sample sizes, regional differences, and demographic factors affecting the results.

The correlation analysis demonstrated robust associations between metabolic components and HOMA-IR, particularly with WC, SBP, glucose levels, and triglycerides. The strong positive correlation between HOMA-IR and these variables supports the hypothesis that insulin resistance exacerbates these metabolic disturbances. Logistic regression analysis further reinforced these findings, revealing that higher BMI, WC, SBP, FPG, and TG are positively associated with MetS, while higher HDL-C is negatively associated. These results are consistent with the work of the Framingham Heart Study, which identified similar risk factors for MetS and cardiovascular disease [19].

The study's context within Bangladesh highlights the increasing prevalence of MetS in rapidly urbanizing populations. The rise in MetS prevalence in Bangladesh is in line with global trends and reflects the impact of urbanization, lifestyle changes, and dietary transitions [20]. This is particularly pertinent given the South Asian phenotype, which predisposes individuals to central obesity and insulin resistance even at lower BMI levels. Studies focusing on South Asian populations, such as those by Misra and Khurana [21], have shown that ethnic factors contribute significantly to the metabolic risk profile observed in our study to other study [22].

5. Conclusion

The present study provides valuable insights into the clinical and biochemical profile of individuals with MetS and IR, highlighting significant differences in metabolic parameters and demonstrating strong correlations with insulin resistance. These findings are consistent with broader research, emphasizing the importance of addressing MetS through targeted public health interventions and lifestyle modifications. Further research should explore the underlying mechanisms driving these associations and evaluate the effectiveness of different intervention strategies in diverse populations.

Conflict of interest

The authors declare no conflict of interest.

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