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OBESITY INDICATORS AS RISK FACTORS FOR DIABETES AND HYPERTENSION

Rashad Mahmood Mirza
Professor, Department of Physiology
Asia International University, Bukhara, Uzbekistan
Email: rashadphysiology@gmail.com
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Abstract: Background: The global burden of diabetes and hypertension has risen sharply, establishing them as leading contributors to morbidity and mortality. Abdominal obesity indicators, such as waist circumference (WC) and waist-to-hip ratio (WHR), show a robust correlation with these disorders. Evidence suggests WC and WHR may be more effective predictors than body mass index (BMI). Aim and **objectives:** This study investigated the relationship between diabetes, hypertension, and various anthropometric parameters in adults aged 25–70 years. Objectives included quantifying BMI, WC, and WHR in adults with diabetes and/or hypertension, and evaluating the independent risk contributions of these measures. **Materials and methods:** A cross-sectional observational study was conducted at District Headquarter Hospital Bhimber, Azad Jammu and Kashmir, Pakistan, enrolling 100 participants (25–70 years). Subjects were divided into four groups (n=25 each): diabetics with normal blood pressure, hypertensives without diabetes, those with both conditions, and healthy controls. Physical measurements (weight, height, BMI, WC, WHR), random blood sugar (RBS), and blood pressure were recorded. Statistical analysis used ANOVA. **Results:** Most participants were aged 41–50 years, with a higher proportion of females. Mean BMI was significantly higher ($p < 0.05$) in groups with diabetes and/or hypertension compared to controls. Mean WC and WHR were significantly elevated ($p< 0.05$) in the group with coexisting diabetes and hypertension. **Conclusion:** Central obesity indicators (BMI, WC, WHR) show a significant association with diabetes and hypertension, underscoring their importance in risk stratification and public health strategy development.

Keywords: Anthropometric indices, Central adiposity, Diabetes mellitus, Hypertension, Waist circumference, Waist-to-hip ratio.

Introduction:

Diabetes and hypertension are primary global causes of morbidity and mortality, with prevalence rising due to lifestyle changes, aging populations, and increasing obesity [1]. Central obesity is a major risk factor for both conditions. Simple anthropometric measures like BMI, WC, WHR, waist-to-height ratio (WHtR), and waist-adjusted BMI (wBMI) offer cost-effective obesity screening. While BMI is most common, it cannot differentiate between muscle and fat or indicate fat distribution, making central obesity markers potentially superior for predicting cardiometabolic disease [1]. Despite extensive research, uncertainty remains regarding the optimal predictor and screening cut-offs. This study aimed to address this gap by examining the link between anthropometric measures and the presence of diabetes and hypertension in adults aged 25–70 years.

Materials and Methods

This cross-sectional observational study was carried out at the District Headquarter Hospital Bhimber, Azad Jammu and Kashmir, Pakistan. Ethical approval was obtained from the institutional ethics committee prior to commencement [2]. A total of 100 participants of both sexes, aged 25–70 years, were enrolled using a convenience sampling method. Participants were divided into four

cohorts of 25 individuals each: Group A (diabetics with normal blood pressure), Group B (hypertensives without diabetes), Group C (individuals with both conditions), and Group D (healthy controls with neither condition). **Inclusion criteria** comprised adults aged 25–70 years with diagnosed diabetes and/or hypertension, as well as healthy individuals without either condition. **Exclusion criteria** included chronic kidney or liver disease, cancer, secondary hypertension, pregnancy or lactation, and incomplete medical or laboratory records.

Data were collected using a standardized questionnaire covering demographic and clinical factors such as age, sex, physical activity, smoking habits, family history, and medication use. Anthropometric measurements were recorded using standard methods: weight (kilograms), height (meters), BMI (kg/m²), WC (cm), and WHR. BMI was classified according to WHO criteria for Southeast Asian populations [3]. Central obesity was defined as WC >90 cm in men and >80 cm in women, or WHR >0.90 in men and >0.85 in women [4]. Blood pressure was measured using a standard mercury sphygmomanometer after the participant had rested in a seated position for at least 5 minutes. Two readings were taken and averaged. Hypertension was defined as systolic blood pressure ≥130 mmHg, diastolic blood pressure ≥80 mmHg, or current use of antihypertensive medications, consistent with JNC-8 and ACC guidelines [5]. Random blood sugar (RBS) was measured using a glucometer (Accu-Chek). Diabetes was defined as RBS ≥200 mg/dL or current use of antidiabetic medications, in line with WHO guidelines [6]. Fasting samples were not collected, as the study protocol relied on random blood glucose measurements.

Statistical analysis was performed using SPSS software (version XX). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. differences between the four groups were compared using one-way analysis of variance (ANOVA). When significant differences were identified, post hoc Tukey's test was applied to determine intergroup differences. A p-value <0.05 was considered statistically significant.

Results

A total of 100 participants, aged between 25 and 70 years, were included in the study, with 25 participants in each of the four groups (A, B, C, D). Most participants (37%) were in the 41–50-year age group, followed by 22% in 51–60 years, 18% in 31–40 years, 13% in 61–70 years, and 10% in 25–30 years. The average age of all participants was 47.6 ± 11.2 years. The study included 55 females and 45 males, with a slightly higher number of females in each group (Table 1, Table 2).

Obesity promotes inflammation via M1 macrophage infiltration, releasing TNF-α, IL-6, and MCP-1. This low-grade inflammation impairs insulin signalling through JNK and NF-κB pathways, causing insulin resistance [2]. Concurrently, elevated free fatty acids induce oxidative stress and ectopic fat deposition, while hormonal imbalance (increased leptin, decreased adiponectin) reduces glucose uptake and impairs vascular health [3]. Abdominal fat also activates the renin-angiotensin system, increasing vascular resistance and blood pressure [4]. These mechanisms explain the strong links between obesity, insulin resistance, and hypertension, supported by recent research on MNK kinases [5–7]. Our findings of higher BMI, WC, WHR, blood sugar, and blood pressure in disease groups are consistent with these pathways.

Across all four groups, central obesity was strongly linked to metabolic conditions. Group C (those with both hypertension and diabetes) had the highest levels of obesity, suggesting a synergistic effect on fat distribution. The higher BMI, WC, and especially WHR observed in this group reflect the clustering of metabolic problems. Similar trends were also observed in Group A (diabetics with normal blood pressure) and Group B (hypertensives without diabetes), suggesting

that central obesity may either precede or independently contribute to the development of these conditions (Table 1).

Table 1: Age and gender distribution of participants across study groups

Group (n = 25 each)	Gender	Age in years					Total
		25–30	31–40	41–50	51–60	61–70	n
Group A (normotensive diabetics)	Male	1	2	5	2	1	11
	Female	2	3	6	2	1	14
Group B (hypertensive nondiabetics)	Male	1	2	4	3	2	12
	Female	1	3	5	2	2	13
Group C (hypertensive diabetics)	Male	0	1	4	4	2	11
	Female	1	2	5	4	2	14
Group D (normotensive nondiabetics)	Male	2	3	3	2	1	11
	Female	3	2	5	3	1	14
Total (n = 100)	Male	4	8	16	11	6	45
	Female	7	10	21	11	6	55

These findings are consistent with Kobayashi et al., who found WHR to be a reliable predictor of type 2 diabetes, and Kawasoe et al., who linked higher BMI and WC to increased hypertension risk, especially in middle-aged men [8, 9].A particularly striking finding was the high WHR in Group D (healthy controls with normal blood pressure and no diabetes), where 84% of participants had WHR values above the defined cut-off, even though their blood pressure and blood sugar were normal (Table 2).

Table 2: Comparison of mean values of variables among the study groups

Study groups (n = 25 each)	Age	BMI	WC	WHR	Systolic BP	Diastolic BP	FBS	RBS
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Group A (normotensive diabetics)	46.2 ± 6.1	28.3 ± 3.4	91.7 ± 8.9	0.93 ± 0.05	126.4 ± 11.6	82.1 ± 7.5	135.6 ± 44.7	189.3 ± 54.2
Group B (hypertensive nondiabetics)	47.5 ± 5.8	27.6 ± 3.9	90.4 ± 9.7	0.91 ± 0.06	144.2 ± 13.5	90.3 ± 8.4	102.5 ± 26.8	121.6 ± 30.7
Group C (hypertensive diabetics)	48.3 ± 6.4	29.1 ± 3.2	94.8 ± 8.6	0.94 ± 0.04	147.6 ± 14.3	93.2 ± 9.1	146.3 ± 52.4	211.7 ± 61.3
Group D (normotensive nondiabetics)	45.6 ± 6.9	25.8 ± 4.1	88.3 ± 9.2	0.89 ± 0.06	112.3 ± 10.2	75.5 ± 6.8	92.4 ± 12.6	112.8 ± 21.9

This suggests that WHR may detect subclinical metabolic problems before any obvious disease appears. The advantage of WHR in identifying central obesity risk is supported by Khader et al., who found WHtR and WHR to be better predictors of diabetes and hypertension than BMI or WC, especially in women [10]. Their suggested WHR cut-offs of 0.85 for women and 0.93 for men closely match the thresholds used in our study.

Further supporting these conclusions, Liu et al. showed that central obesity measures—especially WC, WHtR, and the conicity index—were independently linked to higher diabetes risk, even after adjusting for other factors [11]. They highlighted the importance of using these measures alongside BMI to better identify at-risk individuals. This approach is supported by our data, where high WHR often revealed risk not detected by BMI alone (Table 3). The fact that even participants with normal BMI in our study still had high WHR values underscores the added value of using multiple measures together.

Limitations: While our findings add to the growing evidence on the usefulness of central obesity measures, they should be viewed in light of certain limitations. The sample size was relatively small and participants were recruited from a single hospital, limiting generalizability. The cross-sectional design precludes determining causality or tracking disease progression over time. Despite these limitations, our data provide meaningful insights, especially regarding the often-overlooked role of WHR in early detection.

Clinical implications: These findings have important implications for clinical practice. WHR, because it is sensitive and easy to measure, could serve as a practical and effective screening tool in routine care, especially in primary health settings. Its ability to predict risk even in people with normal blood pressure and blood sugar suggests that including WHR in regular check-ups could facilitate early intervention and potentially reduce future cardiometabolic complications. When used together with BMI and WC, WHR could improve risk assessment accuracy and allow for more personalized prevention strategies (Table 3).

Link between Obesity Measures, Diabetes, and Hypertension

Table 3: Distribution of participants by anthropometric measures across study groups

	BMI (n)			*WC (cm) (n)*		*WHR (n)*	
Group	*Healthy*	*Overweight*	*Obese*	*<90 (M)/ <80 (F)*	*>90 (M)/ >80 (F)*	*<0.90 (M)/<0.85 (F)*	*>0.90 (M)/ >0.85 (F)*
A (normotensive diabetics)	2	8	15	7	18	3	22
B (hypertensive nondiabetics)	3	9	13	5	20	4	21
C (hypertensive diabetics)	1	7	17	4	21	1	24
D (normotensive nondiabetics)	8	9	8	24	10	21	4

Conclusion: This study demonstrates that central obesity measures—especially WC and WHR—are more sensitive predictors of diabetes and hypertension risk than BMI alone. BMI may miss individuals with normal weight but high central fat. Routine screening should include WHR and WC for better risk identification. Public health strategies should promote lifestyle changes to reduce central obesity. Incorporating these measures into primary care and national guidelines can improve early detection and prevention of metabolic diseases.

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