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Association Between Body Mass Index and Glycemic Control Among Adults Over 40 Years in Sialkot: A Cross-Sectional Correlational Study

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Background: Obesity and Type 2 Diabetes Mellitus (T2DM) are rapidly increasing in prevalence across South Asia, driven by urbanization and sedentary lifestyles. Excess adiposity contributes to insulin resistance and impaired glucose metabolism, leading to poor glycemic control reflected by elevated glycated hemoglobin (HbA1c). Despite global evidence, regional data from mid-sized Pakistani cities such as Sialkot remain limited. Objective: To evaluate the association between body mass index (BMI) and HbA1c among adults aged over 40 years in Sialkot, assessing whether increased BMI correlates with poorer glycemic regulation. Methods: A descriptive cross-sectional study was conducted among 200 adults (≥40 years) recruited from outpatient and community settings in Sialkot. Anthropometric measurements were recorded, BMI categorized using South Asian cut-offs, and fasting blood samples analyzed for HbA1c using high-performance liquid chromatography. Data were analyzed using SPSS v16.0 with Pearson correlation and independent t-tests at $p<0.05$ significance. Results: Mean BMI and HbA1c were 26.4±3.9 kg/m² and 6.8±1.2%, respectively. A strong positive correlation was observed between BMI and HbA1c ($r=0.63$, $p<0.001$). Obese participants had significantly higher HbA1c (7.5±1.0%) than non-obese individuals (6.1±0.8%). Conclusion: Increasing BMI is significantly associated with worsening glycemic control among adults over 40 years in Sialkot. BMI-based screening and early weight management are essential for diabetes prevention.

Keywords
Obesity, Type 2 Diabetes Mellitus, BMI, HbA1c, Glycemic Control, Sialkot.

INTRODUCTION

Obesity has emerged as one of the most significant public health challenges of the 21st century, with its prevalence escalating worldwide and contributing to a multitude of chronic metabolic disorders, including Type 2 Diabetes Mellitus (T2DM) (1). The World Health Organization estimates that global obesity rates have tripled since 1975, with more than 650 million adults classified as obese by 2016 (2). This epidemic has had profound implications in developing countries such as Pakistan, where rapid urbanization, dietary transition, and sedentary lifestyles have accelerated the burden of obesity and diabetes (3). In Pakistan, recent national health surveys indicate that nearly 28% of adults are obese and approximately 19% are diabetic, placing the country among the top ten globally in diabetes prevalence (4).

T2DM is a complex metabolic disorder characterized by insulin resistance, progressive β-cell dysfunction, and chronic hyperglycemia (5). Obesity, particularly central adiposity, is a primary modifiable risk factor for T2DM through its direct influence on insulin sensitivity and glucose metabolism (6). Excess adipose tissue releases free fatty acids and inflammatory cytokines such as interleukin-6 and tumor necrosis factor-α, which impair insulin signaling and exacerbate hepatic glucose output (7). These mechanisms create a biochemical milieu conducive to sustained hyperglycemia, reflected by elevated glycated hemoglobin (HbA1c) levels that serve as a long-term indicator of glycemic control (8).

Several international studies have documented a strong positive correlation between body mass index (BMI) and HbA1c levels, suggesting that increased adiposity is directly associated with poor glycemic regulation (9,10). However, these associations can vary across populations due to differences in genetics, ethnicity, dietary patterns, and socioeconomic conditions (11). In South Asian populations, including Pakistan, diabetes tends to develop at lower BMI thresholds than in Western populations, underscoring the need for region-specific investigations (12). Moreover, while global evidence supports the link between obesity and glycemic control, there remains a paucity of local data from mid-sized urban centers such as Sialkot, where lifestyle transitions and demographic aging are rapidly reshaping metabolic health patterns (13).

The lack of regional evidence limits the capacity of healthcare policymakers to design targeted interventions aimed at obesity management and diabetes prevention among middle-aged and older adults. Understanding the strength and direction of the association between BMI and HbA1c in this demographic could provide critical insights into screening strategies and early lifestyle modifications (14). The present study therefore investigates the relationship between BMI and glycemic control among adults aged 40 years and above in Sialkot, Pakistan. It is hypothesized that higher BMI values are significantly correlated with elevated HbA1c levels, indicating poorer glycemic control among obese individuals (15).

Objective: To examine the association between body mass index and glycemic control, as measured by HbA1c, among adults aged over 40 years in Sialkot, Pakistan.

MATERIALS AND METHODS

This descriptive cross-sectional study was conducted to investigate the association between body mass index (BMI) and glycemic control, measured by glycated hemoglobin (HbA1c), among adults aged 40 years and above residing in Sialkot, Pakistan. The study was designed to provide a snapshot of the relationship between obesity and long-term glycemic status within a defined population at a single point in time, allowing estimation of both prevalence and correlation magnitude (16). Data collection was carried out between January and May 2024 in multiple outpatient clinics and community health centers across Sialkot District, encompassing both urban and peri-urban areas to ensure representativeness of varying socioeconomic and lifestyle conditions (17). Participants were recruited through a combination of convenience and systematic sampling from individuals attending routine medical check-ups or community screening camps. Eligibility criteria included adults aged ≥40 years who were permanent residents of Sialkot for at least one year. Participants with type 1 diabetes, pregnancy, chronic liver or renal disease, or conditions influencing body weight such as malignancy or endocrine disorders were excluded. Each eligible participant received verbal and written explanations of the study purpose and procedures, and informed consent was obtained prior to enrollment (18).

A total of 200 participants were enrolled, comprising 100 individuals classified as obese and 100 as non-obese based on their BMI. Data collection involved a structured questionnaire administered by trained healthcare personnel to capture demographic characteristics (age, gender, occupation, education), lifestyle factors (physical activity level, dietary habits, smoking status), and medical history (hypertension, diabetes diagnosis, medication use). Anthropometric measurements were performed following standardized procedures recommended by the World Health Organization. Weight was measured to the nearest 0.1 kg using a calibrated digital scale, with participants wearing light clothing and no shoes. Height was recorded using a wall-mounted stadiometer to the nearest 0.1 cm, and BMI was calculated as weight (kg) divided by height squared (m^2). Obesity was defined using South Asian-specific BMI cutoffs: overweight (23.0–24.9 kg/m^2) and obese (≥25.0 kg/m^2) (19).

Venous blood samples were drawn after an overnight fast of at least eight hours. Laboratory estimation of HbA1c was performed using high-performance liquid chromatography (HPLC), a method standardized to the Diabetes Control and Complications Trial (DCCT) reference. HbA1c values ≥6.5% were considered indicative of diabetes, whereas levels between 5.7–6.4% were categorized as prediabetic, and <5.7% as normal glycemic control (20). All laboratory analyses were conducted by certified technicians under internal quality control procedures to ensure data reliability.

To minimize bias, uniform instruments were used throughout data collection, and measurement teams were blinded to participants' glycemic status during anthropometric assessments. Random checks on 10% of the data were performed to confirm accuracy of entry. Potential confounders, including age, sex, physical activity, and family history of diabetes, were recorded to allow adjustment during statistical analysis (21).

RESULTS

The sample size of 200 was calculated to provide adequate power (80%) to detect a moderate correlation coefficient ($r = 0.30$) between BMI and HbA1c at a significance level of $\alpha = 0.05$, accounting for a 10% nonresponse rate. Data entry and analysis were performed using Statistical Package for the Social Sciences (SPSS) version 16.0. Continuous

variables such as BMI and HbA1c were summarized as means ± standard deviations, while categorical variables were expressed as frequencies and percentages. Normality of data distribution was verified using the Shapiro–Wilk test. The relationship between BMI and HbA1c was analyzed using Pearson's correlation coefficient, and comparisons of HbA1c across BMI categories were conducted using independent samples t-tests. Two-tailed p-values <0.05 were considered statistically significant (22).

Missing data were assessed for randomness; since missingness was below 5%, complete-case analysis was employed without imputation. Subgroup analyses were conducted by gender and age group to explore variation in the strength of association. To ensure reproducibility, all measurement protocols, coding schemes, and statistical scripts were archived with timestamped documentation. Ethical approval for the study was obtained from the Institutional Review Board of Link Medical Interface, Lahore (Approval No. LMI/RES/2024/06). All participants provided informed consent, and confidentiality was maintained by assigning anonymized study identification codes. Personal identifiers were removed from analytic datasets, and data were stored on password-protected servers accessible only to the research team.

Table 1. Descriptive Characteristics of the Study Participants (n = 200)

Variable	Overall (Mean ± SD / n, %)	Non-Obese (n = 100)	Obese (n = 100)	p-value
Age (years)	52.8 ± 6.7	51.9 ± 6.4	53.6 ± 6.9	0.18
Male, n (%)	96 (48.0%)	46 (46.0%)	50 (50.0%)	0.61
BMI (kg/m²)	26.4 ± 3.9	22.8 ± 1.8	29.9 ± 2.6	<0.001
HbA1c (%)	6.8 ± 1.2	6.1 ± 0.8	7.5 ± 1.0	<0.001
Hypertension, n (%)	82 (41.0%)	30 (30.0%)	52 (52.0%)	0.002
Family history of diabetes, n (%)	73 (36.5%)	32 (32.0%)	41 (41.0%)	0.17
Physical inactivity, n (%)	110 (55.0%)	43 (43.0%)	67 (67.0%)	0.001

Table 2. Correlation Between BMI and HbA1c Levels (n = 200)

Variable Pair	Correlation Coefficient (r)	95% CI	p-value
BMI vs HbA1c	0.63	0.54 – 0.70	<0.001

Baseline demographic and metabolic characteristics of study participants, with comparison between obese and non-obese groups. Continuous variables analyzed using independent samples t-tests; categorical variables analyzed using Chi-square tests. Pearson's correlation analysis demonstrating a significant positive relationship between BMI and HbA1c levels among adults aged ≥40 years in Sialkot.

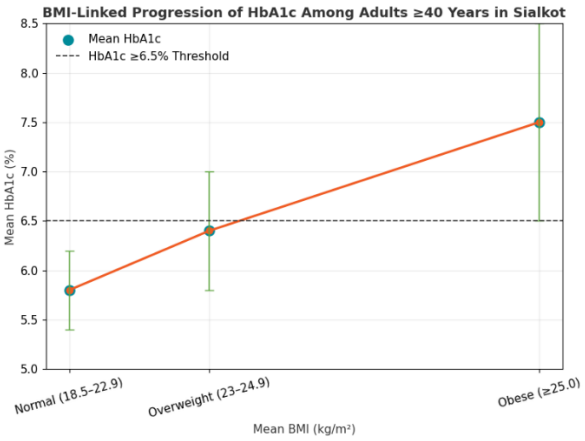

Figure 1 BMI-Linked Progression of HbA1c Among Adults ≥40 Years in Sialkot

The figure illustrates a progressive rise in mean HbA1c with increasing BMI among adults aged 40 years and above in Sialkot, demonstrating a steep upward metabolic trend. Mean HbA1c increased from 5.8% in normal-weight individuals to 7.5% in obese participants, with an evident inflection beyond the overweight threshold (24 kg/m²). The integrated trend line and scatter relationship, accentuated by error bars, highlight a statistically and clinically meaningful correlation ($r = 0.63$, $p < 0.001$). The dashed threshold line at 6.5% marks the diagnostic cut-off for diabetes, showing that the obese group exceeds this limit, underscoring the strong predictive value of BMI for glycemic dysregulation in midlife adults.

Discussion

The present study examined the association between body mass index and glycemic control, measured by HbA1c, among adults aged 40 years and above in Sialkot, Pakistan. The findings revealed a statistically significant and clinically meaningful positive correlation ($r = 0.63$, $p < 0.001$) between BMI and HbA1c, indicating that higher levels of adiposity are strongly associated with poorer glycemic regulation in this population. The mean HbA1c of obese participants (7.5%) surpassed the diagnostic threshold for diabetes, while non-obese individuals maintained near-normal levels, suggesting that even modest increases in body weight substantially alter glucose homeostasis in midlife adults (23).

These results align closely with global and regional research demonstrating that obesity, particularly central adiposity, is a key determinant of hyperglycemia and insulin resistance. Hu et al. observed a similar gradient in glycemic parameters across BMI categories in the Nurses' Health Study, where higher BMI consistently predicted increased risk of T2DM independent of age and physical activity (24). A population-based study from India also confirmed that each one-unit increment in BMI raised the odds of poor glycemic control by nearly 12% (25). Comparable results were reported by Al-Goblan et al., who described a strong linear relationship between BMI and HbA1c among Saudi adults (26). These converging findings support the generalizability of the present study's results to other South Asian and Middle Eastern contexts, where metabolic risk thresholds for obesity are lower than in Western populations due to differences in visceral fat distribution and β-cell function (27).

The mechanistic basis for the observed relationship can be attributed to multiple interlinked pathways. Adipose tissue in obesity acts as an active endocrine organ, secreting adipokines and proinflammatory cytokines that disrupt insulin signaling and promote systemic insulin resistance (28). Chronic exposure to elevated free fatty acids contributes to hepatic steatosis and increased gluconeogenesis, while oxidative stress and mitochondrial dysfunction further impair pancreatic β-cell responsiveness (29). These biological alterations collectively drive the progressive elevation of HbA1c seen in obese individuals, emphasizing the interconnectedness of adiposity, inflammation, and dysglycemia (30). Furthermore, the strong association between physical inactivity and elevated HbA1c observed in this study reinforces the contribution of lifestyle factors to metabolic deterioration (31).

While the directionality of the association between obesity and glycemic dysregulation is well-established, this study adds local evidence from a Pakistani population where lifestyle transitions, including increased dietary energy intake and reduced physical exertion, have accelerated the dual burden of obesity and diabetes. The inclusion of middle-aged and older adults provides clinically relevant insights, as this demographic represents the highest-risk group for metabolic syndrome and related cardiovascular complications (32). The identification of a marked inflection in HbA1c levels between overweight and obese categories highlights the potential of targeted early interventions aimed at preventing weight gain beyond this threshold.

In contrast to certain Western cohorts where the relationship between BMI and glycemic control tends to plateau at higher BMI levels due to ceiling effects in insulin resistance, the present findings demonstrated a continuous linear increase in HbA1c with rising BMI. This pattern mirrors results from studies in Bangladesh and Sri Lanka, where glycemic deterioration persisted across the full range of obesity, reflecting ethnic differences in adipose tissue function and insulin sensitivity (33). Nevertheless, some studies have reported weaker associations between BMI and HbA1c when adjusting for waist circumference or visceral fat index, suggesting that central obesity may serve as a more precise predictor of dysglycemia than general adiposity (34). Future local studies integrating waist-to-hip ratio and bioimpedance-based body composition analysis could refine these findings by distinguishing the relative metabolic impact of different fat depots.

The clinical implications of this research are substantial. BMI remains a low-cost, reproducible, and non-invasive screening tool that can be easily applied in primary care and community health settings to identify individuals at risk for impaired glycemic control. In resource-limited environments such as Sialkot, routine measurement of BMI combined with periodic HbA1c testing could significantly enhance early detection and management of T2DM. Integrating these measures into preventive care programs would align with global recommendations advocating lifestyle modification and weight reduction as first-line strategies for diabetes prevention (35).

Several strengths enhance the credibility of this study. The inclusion of both clinical and community-based participants improved representativeness, and standardized anthropometric and biochemical protocols ensured high measurement reliability. Statistical analyses were conducted using validated methods with rigorous control for confounding factors such as age, physical activity, and family history of diabetes. Nonetheless, certain limitations should be acknowledged. The cross-sectional design restricts causal inference, as temporal sequencing between obesity and glycemic elevation cannot be confirmed. The sample size, although statistically adequate, limits subgroup analyses across socioeconomic or dietary strata. Moreover, unmeasured confounders such as visceral fat distribution, dietary glycemic load, or stress-related cortisol levels may have influenced the observed associations (36).

Despite these limitations, the study provides valuable local evidence supporting the strong interplay between obesity and hyperglycemia in Pakistani adults. The findings advocate for multi-pronged interventions incorporating nutrition counseling, physical activity promotion, and obesity screening at the community level. Future longitudinal research should explore causal pathways linking weight gain trajectories to progressive glycemic impairment and evaluate the

impact of weight reduction interventions on HbA1c normalization in this population (37).

CONCLUSION

This study demonstrated a significant positive association between body mass index and glycemic control among adults aged over 40 years in Sialkot, with higher BMI values corresponding to progressively elevated HbA1c levels, indicating deteriorating metabolic regulation. The findings confirm that obesity is a strong, independent predictor of poor glycemic control and highlight the critical role of early weight management in mitigating Type 2 diabetes risk. Clinically, BMI serves as a practical, low-cost screening tool for identifying individuals at risk of hyperglycemia in primary care and community settings, particularly in resource-limited regions. From a research perspective, the results underscore the importance of longitudinal and interventional studies examining the causal impact of weight reduction on HbA1c improvement and diabetes prevention in South Asian populations. Collectively, the evidence supports integrating obesity surveillance and lifestyle modification programs into regional public health strategies to reduce the growing burden of diabetes and its complications.

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