**Original Article**

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# Evaluating the Impact of BMI on HbA1c and Inflammatory Markers in Diabetic Men and Women

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*Funding Source: None*
*Conflict of Interest: None*

*Received: Mar 11, 2024*
*Accepted: July 04, 2024*

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**ABSTRACT**

**Objective:** This study is focused on the impact of BMI on Glycosylated hemoglobin (HbA1c) and the inflammatory biomarkers that are ESR, monocyte to lymphocyte ratio (MLR), platelet count, total leukocyte count and ferritin levels in diabetic men and women.

**Methodology:** This cross sectional study design was used to collect 160 samples (80 males and 80 females) between ages 25 and 75 years from Pakistan Railway hospital Rawalpindi from January 2024 to April 2024. Patients were assigned to 3 groups depending upon their BMI (Group I= up to 24.9 kg/m2, Group II=25-29.9 kg/m2 and Group III= ≥30kg/m2). Biochemical analysis involved measuring HbA1c, ESR, complete blood counts, including the differential cell count for estimating MLR and serum ferritin levels. Data was analyzed using SPSS 21.

**Results:** The mean BMI value of men was 26.84 ± 4.02, and women was 28.31±6.25 (p=0.001). The number of participants with normal BMI were 64 (40.0%), 77 (48.1%) were overweight and 19 (11.9%) were obese. A correlation of BMI with HbA1c ((r=0.213, p=0.007) and ESR(r=0.252, p=0.001) was positive while with monocyte to lymphocyte ratio (r=-0.162, p=0.44) was negative and a lack of correlation with total leukocyte count (r=-0.025, p=0.756) as well as with platelet count (r=0.144, p=0.069). Also a gender based significant correlation (p=0.001) was observed between BMI and ferritin levels.

**Conclusion:** This study concludes a significant correlation between BMI, HbA1C and inflammatory biomarkers (ESR, ferritin) with a distinct pattern of differences between the two genders showing higher HBA1 c levels in all BMI categories among women, which calls for gender specific treatment strategies.

Key words: Diabetes Mellitus, Body mass index, inflammation, ESR, monocyte to lymphocyte ratio (MLR), platelet count, leukocyte count, ferritin.

**Keywords:** Oral precancer, early predictor, TSA, LBSA.

*Cite this article as: Ayub S, Khan WU, Zainub A, Asim M, Noureen F, Ghulam B, Saeed M.* Evaluating the Impact of BMI on HbA1c and Inflammatory Markers in Diabetic Men and Women. *Ann Pak Inst Med Sci. 2024; 20(SUPPL-1):614-619. doi. 10.48036/apims.v20iSUPPL-1.1070*

## Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia due to defects in insulin secretion, insulin action, or both. [1] According to the International Diabetes Federation (IDF), diabetes represents a significant global health challenge, with increasing prevalence and associated complications leading to substantial morbidity and mortality.[2] The global prevalence of diabetes has been steadily increasing

over the past few decades. As of 2023, approximately 537 million adults aged 20-79 years are living with diabetes, a number projected to rise to 643 million by 2030 and 783 million by 2045. [2]

The major risk factors for diabetes include age, family history, race/ethnicity and physical inactivity and obesity. [1] Body Mass Index (BMI kg/m2) is a critical modifiable risk factor that has been extensively used to indicate adiposity, categorizing individuals as normal (18.5-24.9), underweight (below 18.5), overweight (25.0- 29.9) and obese (30.0 and above). [3] There have been several studies indicating a strong association between BMI and diabetes, making BMI one of the most reliable predictors of type 2 diabetes. [4]

Glycosylated (HbA1c) is the most accurate and reliable criterion for the diagnosis of Diabetes. [5] Patients are considered as diabetic with HbA1c levels ≥ 6.5% , while HbA1c ranges 5.7% - 6.4% indicates prediabetics. [6] Insulin sensitivity and body fat distribution are influenced by sex hormones i.e. estrogen and testosterone. [7] These hormonal differences between genders may affect the HbA1c levels and inflammatory responses. [8]

Chronic low-grade inflammation is a hallmark of diabetes, contributing to insulin resistance and β-cell dysfunction. Inflammatory biomarkers not only indicate systemic inflammation but also play a role in the pathogenesis of diabetic complications, including cardiovascular disease and nephropathy. [9] Many studies have shown that the CBC and ratio derived from the cells such as Monocyte to lymphocyte ratio, Platelet count, total leukocyte count have been associated with many diseases. [10] These studies lack the information regarding the impact of BMI on these inflammatory markers.

The present study aims to focus on the impact of BMI on HbA1c levels and on inflammatory markers that are ESR, monocyte to lymphocyte ratio, platelet count, total leukocyte count and ferritin levels in diabetic men and women. By understanding the effect of BMI on HbA1c and inflammation in both men and women, personalized treatment strategies can be developed. Interventions might need to be deployed differently for men and women targeting HbA1c control and inflammation reduction.

# Methodology

It was a cross-sectional study conducted at Pakistan Railway Hospital, Rawalpindi after getting approval from institutional Ethical Review Committee. The study extended from January 2024 to April 2024 using non-probability convenient sampling. A total of 160 diabetic patients (80 males, 80 females), age between 25 to 75 years, were enrolled in the study. Known patients of hemoglobinopathies, endocrine disorders, renal and hepatic disorders and pregnant women were excluded from this study

Complete history of study participants was documented through a structured questionnaire. All the participants underwent measurement of blood pressure, weight and height. The height and body weight were measured with the subjects standing in light clothes without shoes. Body mass index (BMI) was calculated as weight (kg)/square of height ($m^2$). Patients were divided into three groups according to their BMI. Group-I included patients with BMI up to 24.9 kg/m2. Group-II included patients with BMI of 25-29.9 kg/m2. Obesity was defined as having a BMI≥30kg/$m^2$ , these patients were placed in group-III.

For biochemical analysis, whole blood samples were drawn in plastic vacutainers using EDTA for measuring HbA1c (%) by chemiluminescent microparticle immunoassay (CMIA) on Architect from ABBOTT Diagnostic, Complete blood counts by Midray BC 3000 Plus fully automated Haematology Analyser and ESR (mm/hour) by Wintrobe Method. MLR was calculated as the ratio of monocytes to lymphocytes from the differential white blood cells count. For estimation of serum ferritin, the blood sample was allowed to stand at room temperature for two hours and then centrifuged for 10 minutes at 2200 RPM for serum separation. Separated serum was transferred to small sterile tubes and stored at - 20 $^0$C prior to biochemical estimation of ferritin levels (ng/ml) by chemiluminescent microparticle immunoassay (CMIA) on Architect from ABBOTT Diagnostic.

SPSS 21 software was used for data analysis. For each variable, data was represented as range (sample minimum and maximum) and mean ± SD. Patients were divided into three groups (I,II,II) on the basis of BMI. Clinical and biochemical parameters were compare among the three BMI categories using one-way ANOVA. *P* values below 0.05 were considered statistically significant. The study population was further stratified according to gender, and mean ± SD was computed against each BMI category of males and female separately. Pearson correlation analysis was performed to find correlation between BMI and the independent variables in diabetic males and females

doi. 10.48036/apims.v20iSUPPL-1.1070

# Results

A total of 160 participants (80 males and 80 females) were enrolled. The average age of participants was 50.01 ± 11.97 (females 48.95±17.73 and males 51.07±12.18). Demographic characteristics and biochemical parameters of participants are shown in Table I. The mean BMI in the whole population was 25.19 ± 3.97 (Min: 16.30, Max: 33.00), mean HbA1c was 7.45 ± 1.70 (Min: 4.50, Max:12.00), and mean duration of diabetes was 7.82 ± 8.10 (Min: 1.00, Max:35.00). Serum ferritin for men was 140.22 ± 77.06 and 99.05±43.50 for women (p=0.001). The mean BMI value of men was 26.84 ± 4.02, and women was 28.31±6.25 (p=0.001). The number of participants with normal BMI were 64 (40.0%), 77 (48.1%) were overweight and 19 (11.9%) were obese.

**Table I: Demographic characteristics and means of biochemical parameters of participants. (n=160)**

| Parameters | Minimum - Maximum | Mean ± SD |
|---|---|---|
| Age (years) | 25.00 - 75.00 | 50.01 ± 11.97 |
| Height (meters) | 1.24 - 1.88 | 1.60 ± .14 |
| Weight (kg) | 18.40 - 99.0 | 69.18 ± 15.37 |
| BMI (kg/m2) | 16.30 - 33.00 | 25.19 ± 3.97 |
| Duration of diabetes (years) | 1.00 - 35.00 | 7.82 ± 8.10 |
| HbA1c (%) | 4.50 - 12.00 | 7.45 ± 1.70 |
| Ferritin (ng/mL) | 28.00 - 310.00 | 119.63 ± 65.70 |
| Hb (g/dL) | 7.00 - 18.20 | 12.91 ± 2.76 |
| ESR (mm/hr) | 3.00 - 30.00 | 10.67 ± 6.01 |
| TLC (10*3/microL) | 4.00 - 14.00 | 9.03 ± 1.93 |
| Platelet (10*3/microL) | 146.00 – 463.00 | 256.06 ± 67.14 |
| Monocyte/Lymphocyte ratio | 0.50 - 16.30 | 3.10 ± 2.93 |

In Table II, patients were studied according to their BMI and divided into 3 groups: Normal category patients with mean BMI 21.82 ± 2.10, overweight patients with mean BMI 24.82 ± 7.91 and obese patients with means BMI 30.93 ± 1.02. Demographic and biochemical parameters were studied in both males and females (Table III). According to the results of ANOVA test, a significant difference was observed between different groups of BMI with respect to mean weight, BMI, HBA1c, serum ferritin, Hb levels (p<0.001).

Correlations were made between the variables studied (Table IV), identifying a statistically significant positive relationship between BMI and weight (males: r = 0.33, p = 0.002; females: r = 0.89, p <0.001), serum ferritin (males: r = 0.164, p = 0.066; females: r = 0.34, p = 0.002) and HbA1c (males: 0.38, p < 0.001: females: r = 0.47, p <0.001). In addition, ESR showed significant positive correlation with BMI among females only (r = 0.33, p = 0.002). There was no significant relationship between platelets count and BMI in males and females (r = 0.06, p = 0.19 and r = 0.22, p = 0.07 respectively). Similarly, no correlation was observed between BMI and age of the patient along with total duration of diabetes and mean monocyte-to-lymphocyte ratio. A positive correlation between BMI and weight and negative correlation between BMI and monocyte-to-lymphocyte ratio, is shown on the scatterplot distribution (Figures 1 and 2)

# Discussion

This study gives an analysis of the relationship between body mass index (BMI), glycated haemoglobin (HbA1c) levels and other inflammatory indicators in male and female diabetic patients. For diagnosis and management purpose of diabetes HbA1c marker is significant indicator of long-term hyperglycemia expressing blood glucose levels over past three months.[11]

Our study showed, positive correlation between BMI and HbA1c i.e (r=0.213, p=0.007), suggesting that high BMI is linked with raised HbA1c levels. The study by Kristina

**Table II: Demographic characteristics and biochemical parameters of patients according to BMI.**

| Parameters | Normal (n=64) | Overweight (n=77) | Obese(n=19) | p value |
|---|---|---|---|---|
| | Mean ± SD | Mean ± SD | Mean ± SD | |
| Age (years) | 50.03 ± 14.25 | 50.92 ± 9.61 | 46.26 ± 12.07 | 0.317 |
| Height (meters) | 1.58 ± .14 | 1.62 ± 0.14 | 1.58 ± 0.14 | 0.243 |
| Weight (kg) | 61.09 ± 5.99 | 70.73 ± 17.05 | 90.15 ± 5.25 | <0.001* |
| BMI (kg/m2) | 21.82 ± 2.10 | 24.82 ± 7.91 | 30.93 ± 1.02 | <0.001* |
| Duration of diabetes (years) | 8.68 ± 9.82 | 7.15 ± 5.98 | 7.63 ± 9.32 | 0.535 |
| HbA1c (%) | 6.67 ± 1.34 | 7.75 ± 1.69 | 8.87 ± 1.52 | <0.001* |
| Ferritin (ng/mL) | 102.28 ± 46.61 | 119.58 ± 64.98 | 178.26 ± 89.47 | <0.001* |
| Hb (g/dL) | 12.11 ± 2.66 | 13.87 ± 2.61 | 11.78 ± 2.51 | <0.001* |
| ESR (mm/hr) | 9.56 ± 5.63 | 11.19 ± 6.25 | 12.31 ± 5.94 | 0.124 |
| TLC (10*3/microL) | 8.73 ± 2.06 | 9.26 ± 1.73 | 9.11 ± 2.20 | 0.275 |
| Platelet (10*3/microL) | 243.07 ± 62.88 | 259.81 ± 67.03 | 284.57 ± 74.13 | 0.047* |
| Monocyte/Lymphocyte ratio | 3.63 ± 2.51 | 2.85 ± 3.27 | 2.30 ± 2.55 | 0.132 |

**Table III: Basic characteristics and biochemical parameters of patients according to gender.**

| | Males (n=80) | | | Females (n=80) | | |
|---|---|---|---|---|---|---|
| | **Normal** | **Overweight** | **Obese** | **Normal** | **Overweight** | **Obese** |
| **N** | 27 | 47 | 06 | 37 | 30 | 13 |
| **Age (years)** | 52.55 ± 14.89 | 50.70 ± 10.34 | 47.33 ± 13.30 | 48.18 ± 13.68 | 51.26 ± 8.49 | 45.76 ± 12.0 |
| **Height (meters)** | 1.53 ± 0.16 | 1.61 ± 0.16 | 1.45 ± 0.11 | 1.62 ± 0.11 | 1.63 ± 0.12 | 1.64 ± 0.12 |
| **Weight (kg)** | 62 ± 5.03 | 67.95 ± 21.06 | 88.83 ± 7.96 | 60.43 ± 6.59 | 75.10 ± 5.09 | 90.76 ± 3.7 |
| **BMI (kg/m2)** | 22.55 ± 1.65 | 23.81 ± 9.76 | 30.95 ± 1.25 | 21.28 ± 2.24 | 26.39 ± 2.90 | 30.92 ± 0.95 |
| **Duration of diabetes (years)** | 9.40 ± 10.12 | 6.85 ± 5.98 | 6.00 ± 3.89 | 8.16 ± 9.69 | 7.63 ± 6.03 | 8.38 ±11.0 |
| **HbA1c (%)** | 6.54 ± 0.98 | 7.60 ± 1.69 | 8.68 ± 2.12 | 6.7 ± 1.57 | 7.99 ± 1.70 | 8.96 ± 1.26 |
| **Ferritin (ng/mL)** | 119.63 ± 57.12 | 136.60 ± 71.38 | 261.16 ± 100.1 | 89.62 ± 32.49 | 92.93 ± 42.16 | 140 ± 53.02 |
| **Hb (g/dL)** | 12.63 ± 2.76 | 14.47 ± 2.61 | 12.31 ± 3.37 | 11.72 ± 2.55 | 12.92 ± 2.37 | 11.53 ± 2.13 |
| **ESR (mm/hr)** | 10.07 ± 6.38 | 11.97 ± 6.23 | 11.83 ± 5.70 | 9.18 ± 5.07 | 9.96 ± 6.18 | 12.53 ± 6.26 |
| **TLC (10*3/microL)** | 8.75 ± 1.47 | 9.29 ± 1.79 | 9.11 ± 1.47 | 8.72 ± 2.43 | 9.21 ± 1.66 | 9.10 ± 2.52 |
| **Platelet (10*3/microL)** | 236.33 ±53.88 | 259.48 ± 73.00 | 281.50 ± 109.51 | 248 ± 69.01 | 260 ± 57.64 | 286 ±56.92 |
| **Monocyte/Lymphocyte ratio** | 3.89 ±2.63 | 2.85 ± 3.05 | 2.11 ±2.64 | 3.44 ± 2.44 | 2.86 ± 3.65 | 2.39 ±2.61 |

**Table IV: Ccorrelation between BMI and the independent variables in diabetic males and females.**

| **Variables** | **Total** | | **Males** | | **Females** | |
|---|---|---|---|---|---|---|
| | **r** | **p** | **r** | **p** | **r** | **p** |
| Age (years) | -0.14 | 0.86 | -0.11 | 0.33 | -0.02 | 0.86 |
| Height (meters) | -0.03 | 0.68 | 0.05 | 0.61 | 0.06 | 0.55 |
| Weight (kg) | 0.85 | <0.001* | 0.33 | 0.002* | 0.89 | <0.001* |
| Duration of diabetes (years) | -0.002 | 0.98 | -0.16 | 0.14 | -0.002 | 0.98 |
| HbA1c (%) | 0.21 | 0.007* | 0.38 | <0.001* | 0.47 | <0.001* |
| Ferritin (ng/mL) | 0.17 | 0.03* | 0.35 | 0.001* | 0.34 | 0.002* |
| Hb (g/dL) | -0.03 | 0.66 | 0.15 | 0.17 | 0.52 | 0.64 |
| ESR (mm/hr) | 0.25 | 0.001* | 0.12 | 0.26 | 0.33 | 0.002* |
| TLC (10*3/microL) | -0.02 | 0.75 | 0.122 | 0.28 | 0.85 | 0.45 |
| Platelet (10*3/microL) | 0.14 | 0.06 | 0.19 | 0.09 | 0.22 | 0.07 |
| Monocyte/Lymphocyte ratio | -0.16 | 0.44 | -0.19 | 0.08 | -0.13 | 0.23 |

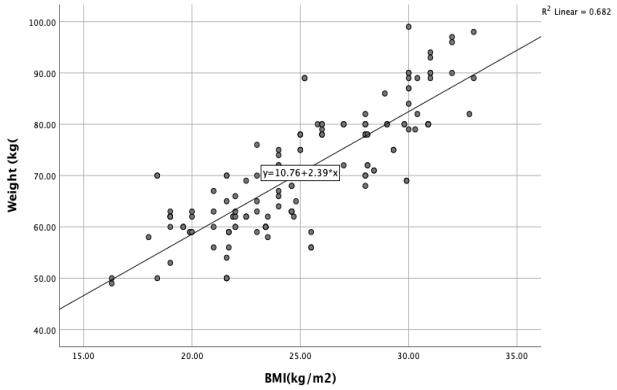


**Figure 1. Correlation between BMI and weight.**

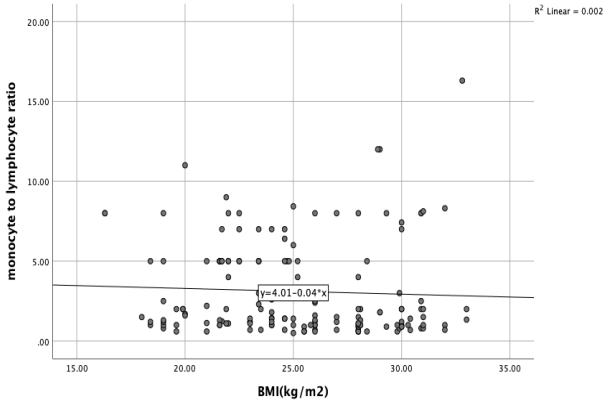


**Figure 2. Correlation between BMI and monocyte-to-lymphocyte ratio.**

S et al in which eight years cohort was taken and the findings are in line with our study.[12] The findings similar to our study was also highlighted that higher BMI elevates the risk of insulin resistance, raising blood glucose levels and in turn elevated HbA1c levels. [13,4]

According to our study, both gender revealed a significant rise in HbA1c levels with rising BMI. However, pronounced increase in HbA1c as BMI increased from normal to obese categories in women was more noticeable (Normal: 21.28 ± 2.24, Overweight: 26.39 ± 2.90, Obese: 30.92 ± 0.95, p<0.001), compared to men (Normal: 22.54 ± 1.65, Overweight: 23.81 ± 9.76, Obese: 30.95 ± 1.25, p=0.002). Demonstrated results proposed that women may be more vulnerable to adverse effects of obesity. In systemic review by Tiziana Ciarambino men tend to accumulate more visceral fat, while women typically have a higher proportion of

subcutaneous fat. Insulin resistant is common in men due to increased visceral fat. But following menopause women prevalence of insulin resistance equals men. These findings are in contrast to our study which reveals distribution of body fat and insulin sensitivity are affected by Sex hormones such as estrogen and testosterone contributing to gender variations hence affecting management of diabetes. [14]

The findings in our study indicating significant positive correlation between BMI and ESR (r=0.252, p=0.001) revealing that higher the BMI the increased levels of ESR are expected suggesting greater inflammatory activity as ESR is chronic inflammation indicator. These findings are in consistent with the study with the understanding that increased adiposity induces chronic low-grade inflammation. Adipose tissue produces pro-inflammatory cytokines like TNF-α, IL-6, and CRP, contributing to elevated ESR levels. [15]

Negative correlation between BMI and the monocyte to lymphocyte ratio (r=-0.162, p=0.44) was found in our study suggesting that higher BMI can affect the balance of white blood cells, which signals towards a shift in the immune response. A lower monocyte to lymphocyte ratio with high BMI represents reflect a chronic inflammatory state. The work of Karakaya et al and Figueroa et al were objectively comparable to our study but yielded contrasting results. They demonstrated Monocyte to lymphocyte ratio was much higher in obese patients with insulin resistance. [16,17]

The general marker of inflammation is Total leukocyte count and our study showed lack of a noticeable correlation between BMI and total leukocyte count (r=-0.025, p=0.756) suggesting that BMI can affect inflammatory markers like ESR and the monocyte to lymphocyte ratio, but it may not affect overall leukocyte count. The results of our study are contradictory to study which emphasizes the correlation between total leukocyte count and BMI in the setting of diabetes. [18]

Obesity-induced inflammation can lead to platelet activation, contributing to the development of atherosclerosis and other cardiovascular diseases. While our study did not find a statistically significant correlation between BMI and platelet count (r=0.144, p=0.069). Recent study have shown that platelet count and obesity has complex relationship. Barrachina et al reveals hyperactivity in platelets from obese patients causing atherothrombosis risk increase. [19] Few mentioned studies indicate that diabetic patients show increased platelet number and activation causing aggregation of platelets and clot formation. In patients with diabetes, hyperglycemia and insulin resistance increase platelet number and activation, causing heightened platelet aggregation and the formation of resistant clots and these findings are not consistent with our study. [20,21,22]

Significant correlation was shown in our study between serum ferritin levels and BMI, discerning between genders. In the results of our study, serum ferritin for obese men was 261.16 ± 100.1 ng/mL and for obese women was 140.53 ± 53.02 ng/mL, with a significant difference (p=0.001). It also revealed that as BMI raised the level of serum ferritin also raised suggesting positive link between storage of iron and adiposity. Through an extensive literature review numerous studies have shown findings similar to our study showing positive relationship of ferritin with increased BMI and poor glycemic control. A study by Lianlong Yu et al analyzed 8163 participants and demonstrated similar findings to our study. [23] Similarly, Akhtar et al stated that for functioning of beta cells for insulin secretion iron is needed, but increasing iron and ferritin (storage form of iron ) levels causes oxidative damage hence, dysfunction of beta cells of pancreas, thus consequent hyperglycemia. [24]

Glycemic control and the emergence of complications are significantly influenced by the duration of diabetes. Our study showed no noteworthy correlation between BMI and the duration of diabetes (r=0.002, p=0.98). Similar results were demonstrated by a cross sectional study conducted on 409 diabetic individuals and suggested that BMI correlation with the duration of the diabetes is not well established. [25]

# Conclusion

This study demonstrated significant correlations between BMI and HbA1c, as well as inflammatory markers (ESR, ferritin) indicating that higher BMI is associated with elevated HbA1c levels. Gender differences were also seen with distinct patterns with women showing higher HbA1c levels across all BMI categories and men revealing a stronger correlation between BMI and inflammatory markers, emphasizing the need for gender-specific treatment strategies.

LIMITATIONS: This study has certain limitations, the small sample size and cross sectional study limits the generalization of the results to larger population, so multicentric studies on large cohort should be done. Longitudinal studies should be planned to establish long-term impact of BMI on HbA1c and

inflammatory markers. Research on the function of adipokines, cytokines, and other inflammatory mediators is necessary to understand how obesity affects glycemic control in diabetes patients and causes systemic inflammation.

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