

GENDER-BASED DIFFERENCES IN CARDIOMETABOLIC RISK PROFILES AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS WITH AND WITHOUT CORONARY HEART DISEASE

Sobia Ali¹, Bibi Hajira Ishaq¹, Madiha Hameed², Zahid Sarfaraz Khan³, Kashif Ur Rehman Khalil⁴, Ubaidullah⁵

¹Department of Medical Biochemistry, Khyber Medical College, Peshawar - Pakistan

²Department of Dentistry, Combined Military Hospital, Peshawar - Pakistan

³Department of Biochemistry, Khyber Girls Medical College, Peshawar - Pakistan

⁴Department of Community Medicine, Khyber Medical College, Peshawar - Pakistan

⁵Department of Biochemistry, Gandhara Medical University, Peshawar - Pakistan

ABSTRACT

Objective: To compare gender-based differences in cardiometabolic risk profiles among patients with type 2 diabetes mellitus with and without coronary heart disease

Materials and Methods: This comparative cross-sectional study was conducted at Khyber Medical College and Khyber Teaching Hospital and included 200 patients with type 2 diabetes mellitus: 100 with coronary heart disease and 100 without. Clinical and laboratory data included age, BMI, obesity, hypertension, blood pressure, fasting blood glucose, HbA1C, total cholesterol, triglycerides, HDL-C, LDL-C, CRP, and fibrinogen. Statistical analyses included independent-samples t-tests, Mann-Whitney U tests, Pearson chi-square tests, and binary logistic regression.

Results: Females had a higher mean BMI than males ($p < 0.001$) and a higher prevalence of obesity ($p < 0.001$). Median triglycerides were also higher among females (271 versus 240 mg/dL, $p = 0.037$). Although females had higher mean HDL-C, low HDL-C by gender-specific thresholds was more common among females ($p < 0.001$).

Conclusion: Females had a greater burden of obesity and adverse triglyceride and HDL-C patterns. Glycemic control and triglyceride reduction may be more important determinants of CHD than gender alone.

Keywords: Coronary heart disease; Type 2 diabetes mellitus; gender differences; obesity; triglycerides; HbA1c; HDL cholesterol.

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INTRODUCTION

Diabetes mellitus type 2 is a significant metabolic disease that is marked by chronic hyperglycemia; apart from the glucose disturbance, affected people often also develop obesity and hypertension, dyslipidemia and inflammation of the blood vessels, and atherosclerotic cardiovascular disease. ^{1, 2} High blood sugar, high triglycerides, low HDL cholesterol, hypertension, and increased adiposity cause endothelial dysfunction, oxidative stress, inflammation, and accelerated coronary ath-

erosclerosis. ³ Therefore, current evidence indicates that all cardiovascular risk factors should be evaluated, not only blood glucose control. ⁴

Gender is now being recognized as an important biological and clinical predictor of the presentation of diabetes and cardiovascular outcomes. ⁵ Men and women have a different body fat distribution, insulin sensitivity, lipid metabolism, hormonal exposure, vascular function, and age of onset for diabetes. ⁶ There tends to be more total body fat and subcutaneous body fat in women and more visceral fat in men. The benefits of protection from metabolism noted in many premenopausal women, however, may not be present once menopause is reached, as menopause is associated with lower estrogen levels leading to central obesity, insulin resistance, dyslipidemia and higher vascular risk. ⁷ Other gender-related differences in cardiometabolic health could be affected by other factors, such as socio-cultural factors, physical activity, access to health care, treatment intensity, and adherence. ⁸ Athero-

Correspondence

Dr. Bibi Hajira Ishaq,

Assistant Professor

Department of Medical Biochemistry,
Khyber Medical College, Peshawar-Pakistan

Cell: +92-333-9378213

Email: Zahid.dr@gmail.com

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genic dyslipidemia is a classic feature.⁹ The interpretation of HDL-C is especially relevant for gender-based research efforts, as females generally have higher absolute HDL-C levels, but higher thresholds for low HDL-C.¹⁰

Limited evidence is available on gender-based cardiometabolic differences when comparisons are conducted both in the overall population and separately among patients with and without coronary heart disease. Many previous studies have concentrated on inflammatory biomarkers, cardiovascular mortality, or general diabetes complications without simultaneously evaluating glycemic status, obesity, blood pressure, and complete lipid profiles. The objective was to compare the cardiometabolic risk profiles of male and female patients with type 2 diabetes mellitus. After controlling for age, BMI, hypertension, HbA1c, triglycerides, and HDL-C, the study also evaluated gender differences within patients with and without coronary heart disease and assessed whether gender was independently associated with CHD.

MATERIALS AND METHODS

This comparative cross-sectional study was conducted at Khyber Medical College and Khyber Teaching Hospital in Peshawar. The study continued for three months, from August 12 to November 1, 2025. The Khyber Medical College Institutional Review Board in Peshawar, Pakistan, approved the study under reference number 560/DME/KMC, dated August 12, 2025.

We calculated the sample size to compare two independent proportions using a 95% confidence level, 80% statistical power, and a 1:1 allocation ratio. With expected coronary-risk proportions of 50% among males and 72% among females (a 44% relative increase), we estimated a minimum required sample size of 152 participants (76 per gender group).¹¹ The calculated sample size was increased to account for incomplete records, subgroup comparisons, and multivariate regression analysis. Thus, the final analysis included 200 participants.

We assessed adult patients with an established diagnosis of type 2 diabetes mellitus who attended medical, diabetes, or cardiology services during the study period. Participants were divided into two groups based on documented coronary heart disease status: CHD and non-CHD. The primary objective was to compare cardiometabolic risk profiles between male and female participants in the overall sample and separately within the CHD and non-CHD diabetic groups.

The sampling technique used was non-probability consecutive sampling. The subjects included were patients of 18 years and above, diagnosed with T2DM, of both genders. In the 'cardiac-diabetic group, participants needed to have a medical record documenting a diagnosis of CHD. We excluded patients with incomplete clinical or laboratory data, as well as those with type 1 diabetes

mellitus, pregnancy, acute severe infection, advanced hepatic disease, end-stage renal disease, malignancy, or conditions likely to significantly affect lipid or inflammatory measurements. We also excluded patients who could not be assessed because they required emergency treatment.

We used a structured data collection form. Data collected from each participant included age, gender, coronary heart disease (CHD) status, hypertension status, body mass index, and blood pressure (systolic and diastolic). We recorded body weight on a calibrated scale and height (without shoes) in meters. Hypertension status was determined by documented medical history, current antihypertensive therapy, or clinical diagnosis.

Venous blood samples were collected under aseptic conditions after an overnight fast. Laboratory measurements included FBG, HbA1c, TC, TG, HDL-C, LDL-C, CRP, and fibrinogen. The institutional laboratory used standard operating procedures and equipment for all biochemical assays. Because the main focus of the analysis was variation in blood pressure, glycemic status, lipids, obesity, and coronary risk by gender, CRP and fibrinogen were examined as secondary variables.

We used IBM SPSS Statistics version 26 to enter and analyze the data. We used the Shapiro-Wilk test to assess normality of continuous variables. We used the Mann-Whitney U test or the independent t-test to compare continuous variables. We used the chi-square test to assess categorical data. We conducted gender-based comparisons overall and by CHD status. After controlling for age, BMI, hypertension, HbA1c, TG, and HDL-C, we used binary logistic regression to evaluate the independent relationship between gender and CHD. We considered $p < 0.05$ significant and reported adjusted odds ratios with 95% CIs.

RESULTS

Female participants had a significantly higher mean BMI than male participants ($p < 0.001$). The prevalence of obesity was also significantly higher among females (43.6%) than among males (17.2%) ($p < 0.001$). However, no gender-based differences were found in the prevalence of coronary heart disease, hypertension, systolic blood pressure, diastolic blood pressure, or age.

The median triglyceride level was significantly higher in females than in males ($p = 0.037$). HDL-C was also significantly higher among females (p -value was less than 0.001). Frequencies of low HDL-C were significantly higher in 'females than in males' ($p < 0.001$). Fasting blood glucose, HbA1C, poor glycemic control, total cholesterol, hypertriglyceridemia, LDL-C, CRP, and fibrinogen did not differ significantly.

Among CHD patients, women showed higher obesity, BMI, TC, TG, and low HDL-C prevalence than men.

In the non-CHD group, women were older and had higher BMI, obesity, SBP, FBG, HDL-C, and low HDL-C prevalence, whereas hypertension and DBP were similar between genders

A logistic regression overall model was statistically significant with $p < 0.001$. The Nagelkerke R^2 explained around 28.4% of the variance in CHD, and the model cor-

rectly classified 71.0% of the participants. The Hosmer–Lemeshow test was not significant, $\chi^2(8) = 8.929$, $p = 0.348$ (good model fit). Adjusted odds of CHD were 35% lower in females than in males, although this result was not significant ($p = 0.232$). An increase of 1 mg/dL of triglycerides was significantly associated with a small increase in CHD odds ($p = 0.001$).

Table No 1: Demographic and clinical characteristics of participants according to gender

Characteristics	Male (n=99)	Female (n=101)	Test statistic	p-value
Age, years	61.34 ± 8.74	63.33 ± 8.61	t = -1.617	0.108
BMI, kg/m ²	27.58 ± 2.73	29.31 ± 2.60	t = -4.600	<0.001
Obesity, n (%)	17 (17.2)	44 (43.6)	$\chi^2 = 16.429$	<0.001
Hypertension, n (%)	52 (52.5)	51 (50.5)	$\chi^2 = 0.083$	0.774
Systolic blood pressure, mmHg	142.64 ± 16.10	146.20 ± 15.79	t = -1.579	0.116
Diastolic blood pressure, mmHg	85.64 ± 8.94	85.27 ± 9.08	t = 0.290	0.772
Coronary heart disease, n (%)	51 (51.5)	49 (48.5)	$\chi^2 = 0.180$	0.671

Table No 2: Glycemic, lipid and inflammatory parameters according to gender

Parameter	Male (n=99)	Female (n=101)	Test statistic	p-value
Fasting blood glucose, mg/dL	215.43 ± 38.15	217.97 ± 33.97	t = -0.497	0.620
HbA1c, %	9.16 ± 1.77	9.09 ± 1.76	t = 0.310	0.757
Poor glycemic control, n (%)	88 (88.9)	89 (88.1)	$\chi^2 = 0.029$	0.864
Total cholesterol, mg/dL	241.21 ± 47.23	252.83 ± 50.61	t = -1.678	0.095
Triglycerides, mg/dL	240 (IQR 112)	271 (IQR 156)	U = 4147.50	0.037
Hypertriglyceridemia, n (%)	90 (90.9)	92 (91.1)	$\chi^2 = 0.002$	0.965
HDL-C, mg/dL	41.02 ± 8.92	46.42 ± 8.62	t = -4.349	<0.001
Low HDL-C, n (%)	42 (42.4)	68 (67.3)	$\chi^2 = 12.527$	<0.001
LDL-C, mg/dL	147.18 ± 40.55	145.86 ± 38.05	t = 0.238	0.812
CRP, mg/L	14.93 ± 5.09	15.05 ± 4.53	t = -0.171	0.865
Fibrinogen, mg/dL	425.69 ± 100.29	453.23 ± 99.44	t = -1.950	0.053

Table No 3: Gender-based differences within patients with and without coronary heart disease

Parameter	Male (n=99)	Female (n=101)	Test statistic	p-value		
Age, years	8.08 ± 63.49	8.51 ± 63.43	0.970	8.92 ± 59.06	8.78 ± 63.23	0.021
BMI, kg/m ²	2.86 ± 27.77	2.17 ± 29.41	0.002	2.61 ± 27.38	2.96 ± 29.22	0.001
Obesity, n (%)	15.7) 8)	44.9) 22)	0.001	18.8) 9)	42.3) 22)	0.011
Hypertension, n (%)	58.8) 30)	44.9) 22)	0.164	45.8) 22)	55.8) 29)	0.321
Systolic BP, mmHg	15.42 ± 145.71	15.95 ± 145.78	0.982	16.32 ± 139.38	15.79 ± 146.60	0.027
Diastolic BP, mmHg	9.99 ± 86.43	8.76 ± 85.90	0.777	7.68 ± 84.79	9.41 ± 84.67	0.945
Fasting blood glucose, mg/dL	39.53 ± 231.45	35.08 ± 222.88	0.255	28.24 ± 198.42	32.54 ± 213.35	0.016
HbA1c, %	1.48 ± 9.99	1.68 ± 9.56	0.179	1.64 ± 8.28	1.72 ± 8.64	0.296
Total cholesterol, mg/dL	44.80 ± 250.53	49.60 ± 273.04	0.019	48.20 ± 231.31	44.09 ± 233.79	0.789
Triglycerides, mg/dL	253.0 (IQR 102.0)	321.0 (IQR 219.0)	0.016	232.5 (IQR 140.25)	244.0 (IQR 111.25)	0.528
HDL-C, mg/dL	9.82 ± 40.45	8.75 ± 45.18	0.013	7.92 ± 41.63	8.41 ± 47.58	<0.001
Low HDL-C, n (%)	45.1) 23)	71.4) 35)	0.008	39.6) 19)	63.5) 33)	0.017
LDL-C, mg/dL	38.57 ± 154.04	37.71 ± 158.57	0.554	41.72 ± 139.90	34.62 ± 133.88	0.434

Table No 4: Multivariable logistic regression analysis of factors associated with coronary heart disease

Predictor	β coefficient	Adjusted OR	%95 CI for AOR	p-value
Female gender versus male	0.428–	0.652	1.315–0.323	0.232
Age, per year	0.029	1.030	1.070–0.992	0.127
BMI, per kg/m ²	0.075	1.078	1.218–0.955	0.226
Hypertension, yes versus no	0.232–	0.793	1.539–0.409	0.493
HbA1c, per %1 increase	0.503	1.653	2.034–1.344	<0.001
Triglycerides, per 1 mg/dL increase	0.005	1.005	1.009–1.002	0.001
HDL-C, per 1 mg/dL increase	0.023–	0.977	1.013–0.942	0.212

DISCUSSION

The current investigation found significant gender differences in the cardiometabolic profiles of T2DM patients, despite a similar overall incidence of coronary heart disease between males and females. Female participants had a significantly higher mean BMI and a much higher prevalence of obesity than men. They also had higher triglyceride and HDL-C levels, although the prevalence of low HDL-C, based on gender-specific thresholds, was considerably higher in females. These data suggest that men and women may have different cardiometabolic risk distributions, even if their observed burden of coronary disease is comparable. According to current research, women with type 2 diabetes may have a greater relative increase in cardiovascular risk than men due to variations in adiposity, hormonal state, vascular biology, and risk-factor treatment (12-14).

The most notable clinical difference was the much higher obesity prevalence in females. Obesity prevalence in females (43.6%) was substantially greater than in males (17.2%) in both the CHD and non-CHD groups. This finding is consistent with current data, which show that at the time of clinical assessment, women with type 2 diabetes have a higher BMI and more overall adiposity than men.^{12, 15} Genetic, hormonal, and social factors influence gender disparities in body composition. Women have a higher ratio of total body fat and SAF to VOF than men. Women have a higher ratio of total body fat and SAF to VOF than men. After menopause, falling estrogen levels may lead to increased central fat accumulation, insulin resistance, and worsening metabolic profile.¹⁶ There could be other explanations for women's higher prevalence of overweight and obesity, such as differences in physical activity, household chores, and access to preventive programs and structured activities. Because the obesity gap was maintained in both study groups, weight control should remain a significant emphasis of cardiovascular protection for diabetic women, regardless of whether CHD has been diagnosed.

The lipid results revealed considerable gender variations. The median triglyceride concentration was substantially higher in females than in males, particularly in the CHD group, where it was 321 mg/dL in females and 253 mg/dL in males. Females had a greater mean HDL-C

concentration, but they were more likely to be classified as having low HDL-C based on gender-specific thresholds. Another large primary-care research of type 2 diabetes patients found that women had greater HDL-C levels than men, although the differences were dependent on age, menopausal status, and statin use.¹⁷ This is because the level of HDL-C that is measured in women should not necessarily be regarded as adequate lipid control.^{18, 19} This is because HDL-C levels in women do not always indicate proper cholesterol control. Triglycerides, HDL-C, and triglyceride indices have been linked to insulin resistance, coronary atherosclerosis, and poor cardiovascular outcomes.²⁰⁻²² The current findings support analyzing the overall lipid profile and using gender-specific cutoff values rather than the absolute value of HDL-C.

The subgroup analysis indicated gender disparities by CHD status. Among patients with established CHD, females had higher BMI, obesity prevalence, total cholesterol, triglycerides, HDL-C, and low-HDL prevalence than males. Female patients without CHD were older than male patients without CHD and had higher BMI, obesity prevalence, systolic blood pressure, fasting glucose, HDL-C, and low-HDL prevalence. Based on these findings, it is proposed that female patients may develop several of these adverse metabolic characteristics before the clinical diagnosis of coronary disease.

According to EUROASPIRE surveys, women with dysglycemia and coronary artery disease are more likely than men to be older, obese, and to have not met prescribed blood pressure, cholesterol, or physical activity target.²³ Gender inequalities in the prevalence, treatment, and control of cardiovascular risk factors persist in other recent research.²⁴ These disparities could be attributed to age, menopause, medication, disease type, access to health care, and local lifestyle choices. This should be done in addition to, not instead of, an individual assessment of modifiable cardiovascular risks, and it should be gender-sensitive.

After adjusting for age, BMI, hypertension, HbA1c, triglycerides, and HDL-C, gender did not predict CHD independently in multivariable analysis. HbA1c and triglycerides, on the other hand, were significant independent predictors of CHD. The adjusted risk of CHD for

each 1% increase in HbA1c was 65.3%, demonstrating the importance of continued glycemic control in CHD risk. Recent studies have linked poor HbA1c control and CHV with the severity of coronary disease, cardiovascular events, restenosis, and mortality.^{25, 26}

The study has a few limitations. The cross-sectional design does not account for time or causality. Diabetes duration, smoking, diabetes treatment, menopause, physical activity, dietary habits, waist size, and socioeconomic status were not reported, potentially leading to residual confounding. The single-center architecture and very tiny subgroups may potentially contribute to limited generalizability. Larger prospective studies are required to assess the efficacy of gender-specific weight loss, hyperglycemia, and lowering of triglyceride-rich lipoproteins for clinically relevant reductions in coronary events.

CONCLUSION

Female patients with type 2 diabetes had a larger burden of obesity, higher triglyceride concentrations, and a higher prevalence of low HDL-C than male patients, according to gender-specific criteria. These disparities were observed in people with and without coronary heart disease. After controlling for significant cardiometabolic variables, gender was not found to be an independent predictor of CHD. Higher HbA1c and triglyceride levels were revealed as the primary independent risk factors for CHD. Cardiovascular risk assessment in type 2 diabetes should therefore include gender-sensitive interpretations of obesity and lipid abnormalities, along with a strong clinical focus on glycemic control, triglyceride reduction, and comprehensive management of modifiable risk factors.

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Authors Contribution:

Following authors have made substantial contributions to the manuscript as under

Authors	Conceived & designed the analysis	Collected the data	Contributed data or analysis tools	Performed the analysis	Wrote the paper	Other contribution
Ali S	✓	✓	✗	✗	✓	✗
Ishaq Bh	✓	✗	✓	✓	✓	✗
Hameed M	✓	✓	✗	✗	✗	✓
Khan Zs	✓	✗	✓	✓	✓	✗
Khalil Kur	✓	✓	✗	✗	✗	✓
Ubaidullah	✓	✗	✓	✓	✓	✗

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Ethical Approval:

This Manuscript was approved by the Ethical Review Board of IREB of Khyber Medical College, Peshawar. Vide No. 560/DME/KMC. Dated: 12 08 2025



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