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Corresponding Author

Sobia Ali

Assistant Professor, Department of
Biochemistry, Khyber Medical College,
Peshawar

Email: drsobiaali2@gmail.com

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Original Article

Association of C-Reactive Protein and Fibrinogen with Metabolic and Cardiovascular Risk Parameters among Diabetic Patients with and without Cardiac Disease

Bibi Hajira Ishaq¹, Muhammad Shabir², Zahid Sarfaraz³, Zafar Ali⁴, Muddasir Ahmed⁵, Sobia Ali⁶

1. Assistant Professor, Department of Biochemistry, Khyber Medical College, Peshawar
2. Dean, Postgraduate, Gandhara Medical University, Peshawar
3. Associate Professor Anatomy Department Khyber Girls Medical College Peshawar
4. Lecturer, Department of Biochemistry, Khyber Medical College, Peshawar
5. Assistant Professor, Northwest School of Medicine, Peshawar
6. Assistant Professor, Department of Biochemistry, Khyber Medical College, Peshawar

Abstract

Objective: To compare metabolic, lipid, inflammatory, and coagulation-related parameters between diabetic patients with and without cardiac disease and to evaluate correlations between C-reactive protein (CRP), fibrinogen, and selected clinical and biochemical parameters.

Methodology: This case-control analytical study was conducted in the Department of Biochemistry, Khyber Medical College, Peshawar, from October 2025 to March 2026. We included 200 diabetic participants: 100 with cardiac disease and 100 without. We evaluated group differences using appropriate comparative statistical tests and performed Pearson correlation analysis to assess relationships between CRP or fibrinogen and selected metabolic and lipid parameters. We considered $p < 0.05$ statistically significant.

Results: The overall mean age was 61.50 ± 9.82 years, and the mean BMI was 28.17 ± 3.23 kg/m². Cardiac diabetic patients had higher FBS (213.80 ± 91.62 vs. 175.61 ± 59.14 mg/dL), HbA1c (10.17 ± 3.45 vs. $8.31 \pm 3.04\%$), CRP (18.43 ± 4.22 vs. 12.67 ± 6.17), and fibrinogen (478.63 ± 165.98 vs. 413.44 ± 139.75) (all $p \leq 0.003$). Lipid abnormalities were more frequent in cardiac patients. CRP and fibrinogen showed significant correlations with lipid parameters and BMI across groups.

Conclusion: Cardiac diabetic patients demonstrated poorer glycaemic control and higher inflammatory and fibrinogen levels than diabetic patients without cardiac disease. The findings suggest that CRP and fibrinogen may reflect different components of the metabolic and cardiovascular risk profile in diabetes. Further prospective studies are required to determine their clinical and prognostic significance.

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Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia and an increased risk of both microvascular and macrovascular complications. Cardiovascular disease (CVD) remains one of the major causes of morbidity and mortality among individuals with diabetes. The increased cardiovascular burden associated with diabetes is multifactorial and involves complex interactions between hyperglycemia, insulin resistance, hypertension, dyslipidemia, obesity, oxidative stress, endothelial dysfunction, and chronic inflammation. These metabolic and vascular abnormalities contribute to progressive arterial injury and accelerate atherosclerotic cardiovascular disease [1,2]. Endothelial dysfunction is considered an important early manifestation of diabetic vascular injury. Persistent metabolic abnormalities, particularly hyperglycemia and atherogenic dyslipidemia, promote oxidative stress and inflammatory signaling, impairing endothelial function and vascular homeostasis.[3,4].In addition to conventional cardiovascular risk factors, increasing evidence suggests that inflammatory and hemostatic pathways contribute substantially to the development and progression of cardiovascular complications in patients with diabetes. Chronic inflammation may promote endothelial activation, leukocyte recruitment, vascular remodeling, and atherosclerotic plaque progression, whereas coagulation pathway activation may contribute to thrombus formation and adverse cardiovascular outcomes [5]. C-reactive protein (CRP) is an acute-phase protein synthesized primarily by the liver in response to inflammatory stimuli. It is widely used as a marker of systemic inflammation and has been extensively investigated in relation to cardiovascular risk. In patients with type 2 diabetes, increased CRP concentrations have been associated with adverse cardiovascular outcomes and mortality[6,7]. The persistent metabolic abnormalities characteristic of diabetes may contribute to a pro-inflammatory environment in which hyperglycemia, insulin resistance, obesity, and dyslipidemia interact with inflammatory pathways. Recent evidence further suggests that CRP-related inflammatory mechanisms may participate in endothelial injury, leukocyte recruitment, and progression of atherosclerotic disease [8]. Fibrinogen is another clinically relevant biomarker that links inflammation with hemostasis. It is a hepatic plasma glycoprotein that serves as a fibrin precursor during coagulation and also participates in platelet aggregation and vascular inflammatory processes. Elevated fibrinogen concentrations have been associated with cardiovascular disease and may reflect an underlying pro-inflammatory and prothrombotic state[9]. Large epidemiological studies have demonstrated associations between plasma fibrinogen levels and major cardiovascular outcomes. However, the extent to which fibrinogen acts as a causal mediator rather than a marker of cardiovascular risk remains uncertain[10]. The coexistence of diabetes and cardiovascular disease may therefore be associated with a greater inflammatory and hemostatic burden than diabetes alone. However, the relationships between glycemic control, lipid abnormalities, CRP, and fibrinogen may not be uniform across diabetic populations. Assessment of these biomarkers alongside conventional metabolic parameters may provide additional insight into the biological differences between diabetic patients with and without cardiac disease. In particular, examining correlations between CRP and fibrinogen and glycemic and lipid parameters may help clarify their relationship with the cardiometabolic profile. The present study compared demographic, clinical, metabolic, lipid, inflammatory, and hemostatic characteristics between diabetic patients with cardiac disease and those without documented cardiac disease. We focused on fasting blood glucose, glycated hemoglobin (HbA1c), lipid profile, CRP, and fibrinogen. We also performed Pearson correlation analysis to assess relationships

between CRP and fibrinogen and selected metabolic and cardiovascular risk parameters.

Materials and Methods

This case-control analytical study was conducted in the Department of Biochemistry, Khyber Medical College, Peshawar, from October 2025 to March 2026. It involved 200 diabetic participants. The study population was divided into two groups: 100 cardiac diabetic participants and 100 diabetic participants without cardiac disease. State the precise definition and diagnostic criteria used to classify participants as cardiac diabetic, as outlined in the original study protocol. Similarly, insert the study setting, duration, participant recruitment procedure, inclusion and exclusion criteria, and ethical approval information before submission, as these details were not provided in the supplied statistical tables.

Clinical and Biochemical Variables

The evaluated variables included age, systolic blood pressure, diastolic blood pressure, and BMI. Biochemical parameters included fasting blood sugar, HbA1c, total cholesterol, triglycerides, HDL, and LDL. Inflammatory and coagulation-related measurements included CRP and fibrinogen. Overall descriptive statistics showed a mean age of 61.50 ± 9.82 years, systolic blood pressure of 155.02 ± 22.03 mmHg, diastolic blood pressure of 93.43 ± 11.02 mmHg, and BMI of 28.17 ± 3.23 kg/m². Mean FBS was 194.70 ± 79.26 mg/dL, and mean HbA1c was $9.24 \pm 3.37\%$. The mean total cholesterol was 275.84 ± 137.05 , triglycerides 307.19 ± 175.96 , HDL 42.49 ± 15.97 , and LDL 173.83 ± 140.03 . Mean CRP was 15.55 ± 6.01 , and mean fibrinogen was 446.04 ± 156.49 . CRP remained significantly higher among cardiac diabetic participants across BMI categories, including normal weight (20.83 ± 5.26 vs. 10.12 ± 5.93 ; $p=0.026$), overweight (18.32 ± 4.89 vs. 10.16 ± 5.89 ; $p<0.001$), and obesity (18.26 ± 3.91 vs. 13.90 ± 6.00 ; $p<0.001$). Fibrinogen was significantly higher among overweight cardiac diabetic participants (492.77 ± 222.50 vs. 368.10 ± 139.99 ; $p=0.027$), while differences in normal-weight and obese participants were not significant. Among hypertensive participants, CRP (18.45 ± 4.14 vs. 12.69 ± 6.23 ; $p<0.001$) and fibrinogen (481.06 ± 160.37 vs. 417.28 ± 145.92 ; $p=0.008$) were significantly higher in the cardiac diabetic group. Among normotensive participants, CRP remained significantly higher (18.37 ± 4.68 vs. 12.53 ± 6.00 ; $p=0.002$), whereas fibrinogen did not differ significantly.

Correlation Analysis

In cardiac diabetic participants, CRP correlated positively with total cholesterol ($r=0.210$; $p=0.036$) and HDL ($r=0.202$; $p=0.044$). In diabetic participants, CRP was inversely correlated with HDL ($r=-0.245$; $p=0.014$). No other significant CRP correlations were identified. Fibrinogen showed a positive correlation with triglycerides in cardiac diabetic participants ($r=0.443$; $p<0.001$; $R^2=0.195$). Among diabetic participants, fibrinogen correlated positively with BMI ($r=0.241$; $p=0.016$). Other correlations between fibrinogen and the evaluated clinical or biochemical parameters were not statistically significant.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 27.0. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were presented as frequencies and percentages. Between-group

differences were assessed using appropriate comparative tests, and Pearson’s correlation coefficient was used to evaluate relationships between CRP, fibrinogen, and clinical and biochemical parameters. A p-value <0.05 was considered statistically significant.

Results

Baseline Characteristics

We included 200 participants: 100 with cardiac diabetes and 100 with diabetes. The overall mean age was 61.50±9.82 years (range, 45–90 years). Mean systolic and diastolic blood pressures were 155.02±22.03 and 93.43±11.02 mmHg, respectively, and mean BMI was 28.17±3.23 kg/m². Mean FBS and HbA1c were 194.70±79.26 mg/dL and 9.24±3.37%, respectively. Mean total cholesterol, triglycerides, HDL, and LDL were 275.84±137.05, 307.19±175.96, 42.49±15.97, and 173.83±140.03, respectively. Mean CRP and fibrinogen were 15.55±6.01 and 446.04±156.49, respectively.

Comparison Between Groups

Sex and residence distributions were comparable between groups. Overall, 99 (49.5%) participants were male and 101 (50.5%) female; 149 (74.5%) were from urban and 51 (25.5%) from rural areas. Obesity was present in 139 (69.5%) participants, with no significant difference between the cardiac diabetic and diabetic groups. Hypertension was present in 163 (81.5%) participants and was similarly distributed between groups (81.0% vs. 82.0%; p=0.427).Cardiac diabetic participants had significantly higher FBS (213.80±91.62 vs. 175.61±59.14 mg/dL; p<0.001) and HbA1c (10.17±3.45% vs. 8.31±3.04%; p<0.001). No significant between-group differences were observed in age, systolic or diastolic blood pressure, BMI, total cholesterol, triglycerides, HDL, or LDL.CRP was significantly higher in the cardiac diabetic group (18.43±4.22 vs. 12.67±6.17; p<0.001), as was fibrinogen (478.63±165.98 vs. 413.44±139.75; p=0.003).

Glycemic, Lipid and Biomarker Abnormalities

Overall, abnormal FBS, HbA1c, total cholesterol, triglycerides, HDL, LDL, CRP and fibrinogen were observed in 86.5%, 66.0%, 60.0%, 89.5%, 51.0%, 38.5%, 81.0% and 68.0% of participants, respectively. Abnormal HbA1c was more frequent in cardiac diabetic participants than in diabetic participants (74% vs. 58%; p=0.008). Similarly, abnormal triglycerides (97% vs. 82%; p<0.001), HDL (60% vs. 42%; p=0.005), CRP (99% vs. 63%; p<0.001), and fibrinogen (77% vs. 59%; p=0.003) were significantly more common in the cardiac diabetic group. Abnormal FBS, total cholesterol, and LDL did not differ significantly.

Table 1. Baseline characteristics of study participants

Characteristic	Total (N=200)	Cardiac diabetic (n=100)	Diabetic (n=100)	p-value
Age, years	61.50±9.82	62.52±8.95	60.49±10.56	0.144
Systolic BP, mmHg	155.02±22.03	153.30±22.92	156.75±21.07	0.269
Diastolic BP, mmHg	93.43±11.02	93.70±11.63	93.15±10.44	0.725

BMI, kg/m²	28.17±3.23	28.30±3.18	28.03±3.29	0.555
FBS, mg/dL	194.70±79.26	213.80±91.62	175.61±59.14	<0.001
HbA1c, %	9.24±3.37	10.17±3.45	8.31±3.04	<0.001
Total cholesterol, mg/dL	275.84±137.05	279.61±151.82	272.06±121.15	0.697
Triglycerides, mg/dL	307.19±175.96	324.37±142.60	290.02±203.23	0.168
HDL, mg/dL	42.49±15.97	40.67±16.54	44.32±15.25	0.106
LDL, mg/dL	173.83±140.03	176.09±154.09	171.58±125.14	0.820
CRP	15.55±6.01	18.43±4.22	12.67±6.17	<0.001
Fibrinogen	446.04±156.49	478.63±165.98	413.44±139.75	0.003

Values are presented as mean±SD. BP, blood pressure; BMI, body mass index; FBS, fasting blood sugar; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; CRP, C-reactive protein.

Table 2. Distribution of demographic and clinical characteristics

Characteristic	Cardiac diabetic, n (%)	Diabetic, n (%)	Total, n (%)
Gender			
Male	51 (51.0)	48 (48.0)	99 (49.5)
Female	49 (49.0)	52 (52.0)	101 (50.5)
Residence			
Urban	73 (73.0)	76 (76.0)	149 (74.5)
Rural	27 (27.0)	24 (24.0)	51 (25.5)
BMI category			
Normal	6 (6.0)	4 (4.0)	10 (5.0)
Overweight	22 (22.0)	29 (29.0)	51 (25.5)
Obese	72 (72.0)	67 (67.0)	139 (69.5)
Hypertension			
Hypertensive	81 (81.0)	82 (82.0)	163 (81.5)
Normotensive	19 (19.0)	18 (18.0)	37 (18.5)

Data are presented as numbers (percentages). BMI categories are reported according to the classification used in the study. The supplied analysis showed no statistically significant difference in hypertension between groups (p=0.427) and no significant difference in BMI categories.

Table 3. Glycaemic, lipid and inflammatory biomarker abnormalities

Parameter	Abnormal, n (%)	Normal, n (%)
Fasting blood sugar	173 (86.5)	27 (13.5)
HbA1c	132 (66.0)	68 (34.0)
Total cholesterol	120 (60.0)	80 (40.0)
Triglycerides	179 (89.5)	21 (10.5)
HDL	102 (51.0)	98 (49.0)
LDL	77 (38.5)	123 (61.5)

CRP	162 (81.0)	38 (19.0)
Fibrinogen	136 (68.0)	64 (32.0)

Data are presented as numbers (percentages). We defined abnormalities according to the predefined study classification. HDL, high-density lipoprotein; LDL, low-density lipoprotein; CRP, C-reactive protein.

Table 4. Comparison of biochemical abnormalities between cardiac diabetic and diabetic participants

Parameter	Cardiac diabetic, n (%)	
FBS		
Abnormal	89 (89.0)	
Normal	11 (11.0)	
HbA1c		
Abnormal	74 (74.0)	
Normal	26 (26.0)	
Total cholesterol		
Abnormal	57 (57.0)	
Normal	43 (43.0)	
Triglycerides		
Abnormal	97 (97.0)	
Normal	3 (3.0)	
HDL		
Abnormal	60 (60.0)	
Normal	40 (40.0)	
LDL		
Abnormal	34 (34.0)	
Normal	66 (66.0)	
CRP		
Abnormal	99 (99.0)	
Normal	1 (1.0)	
Fibrinogen		
Abnormal	77 (77.0)	
Normal	23 (23.0)	

Data are presented as numbers (percentages). P-values represent comparisons between cardiac diabetic and diabetic participants. FBS, fasting blood sugar; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; CRP, C-reactive protein.

Table 5. Pearson correlations of CRP with clinical and biochemical parameters

Parameter	Cardiac diabetic r	p-value	Diabetic r	p-value
Age	-0.096	0.340	-0.136	0.176
Systolic BP	0.049	0.631	0.108	0.283
Diastolic BP	0.027	0.789	0.116	0.250
BMI	-0.030	0.766	0.179	0.074
FBS	0.051	0.611	0.044	0.662
HbA1c	-0.132	0.191	0.150	0.137
Total cholesterol	0.210	0.036	0.066	0.511
Triglycerides	-0.055	0.585	-0.048	0.635
HDL	0.202	0.044	-0.245	0.014
LDL	0.189	0.060	0.106	0.292

Pearson correlation coefficients (r) are shown. *p<0.05 was considered statistically significant. BP, blood pressure; BMI, body mass index; FBS,

fasting blood sugar; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; CRP, C-reactive protein.

Table 6. Comparison of CRP according to BMI and hypertension status

Stratifying variable	Cardiac diabetic	Diabetic	p-value
BMI category			
Normal	20.83±5.26	10.12±5.93	0.026
Overweight	18.32±4.89	10.16±5.89	<0.001
Obese	18.26±3.91	13.90±6.00	<0.001
Hypertension status			
Hypertensive	18.45±4.14	12.69±6.23	<0.001
Normotensive	18.37±4.68	12.53±6.00	0.002

Values are presented as mean±SD. P-values represent between-group comparisons within each BMI or hypertension category. CRP, C-reactive protein; BMI, body mass index.

Table 7. Pearson correlations of fibrinogen with clinical and biochemical parameters

Parameter	Cardiac diabetic r	p-value	Diabetic r	p-value
Age	-0.102	0.095	-0.080	0.429
Systolic BP	0.008	0.934	0.047	0.644
Diastolic BP	0.165	0.102	0.057	0.573
BMI	0.134	<0.001	0.241	0.016
FBS	-0.063	0.530	0.116	0.248
HbA1c	0.058	0.003	0.041	0.683
Total cholesterol	0.119	0.240	0.165	0.100
Triglycerides	0.443	<0.001	0.078	0.443
HDL	-0.071	0.486	-0.012	0.907
LDL	0.047	0.644	0.144	0.153

Pearson correlation coefficients (r) are shown. *p<0.05; **p<0.01. BP, blood pressure; BMI, body mass index; FBS, fasting blood sugar; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein

Table 8. Comparison of fibrinogen according to BMI and hypertension status

Stratifying variable	Cardiac diabetic	Diabetic	p-value
BMI category			
Normal	409.00±126.63	297.50±42.72	0.086
Overweight	492.77±222.50	368.10±139.99	0.027
Obese	480.11±148.88	439.99±135.88	0.099
Hypertension status			
Hypertensive	481.06±160.37	417.28±145.92	0.008
Normotensive	468.26±192.50	395.94±109.02	0.167

Values are presented as mean±SD. P-values Represent between-group comparisons within each BMI or hypertension category.

Discussion

This study shows important differences between diabetic patients with cardiac disease and those without, particularly in glycaemic control, inflammation, and fibrinogen. Although several conventional clinical characteristics were similar between groups, cardiac diabetic participants had significantly higher FBS, HbA1c, CRP, and fibrinogen. These findings suggest that cardiovascular involvement among diabetic patients may be accompanied by a more pronounced metabolic and inflammatory burden. The relationship between glycaemic exposure and cardiovascular risk in diabetes has been extensively investigated, with both the magnitude and duration of hyperglycemia considered relevant to cardiovascular complications [11]. The first major finding was the difference in glycaemic control. Cardiac diabetic participants had substantially higher FBS and HbA1c than diabetic participants without cardiac disease. Mean FBS exceeded 200 mg/dL in the cardiac diabetic group, while mean HbA1c was above 10%. In contrast, the corresponding values in the non-diabetic group were lower, although still indicative of considerable glycaemic disturbance. These findings highlight the importance of chronic hyperglycemia as part of the overall cardiovascular risk profile in diabetes. Previous evidence indicates that the magnitude and duration of hyperglycemia are relevant to the development of cardiovascular complications in type 2 diabetes [12]. The categorical analysis supported this observation. Abnormal HbA1c was significantly more common among cardiac diabetic participants than among diabetic participants. However, abnormal FBS did not differ significantly between the groups. This difference may reflect that HbA1c represents longer-term glycaemic exposure, whereas FBS reflects glucose at a particular point in time. Therefore, the stronger group difference in HbA1c may indicate a more sustained metabolic disturbance among cardiac diabetic patients [11]. A second important finding was the high prevalence of obesity. Nearly 70% of the study population was classified as obese, and obesity was common in both groups. Although the proportion of obesity was somewhat higher among cardiac diabetic participants, the group difference was not statistically significant. This indicates that obesity alone did not distinguish the two groups in this sample. Nevertheless, obesity is closely linked to low-grade systemic inflammation, and previous evidence shows a positive association between BMI and circulating CRP concentrations [13]. Nevertheless, BMI showed an important relationship with fibrinogen among diabetic participants. We identified a positive correlation between BMI and fibrinogen in patients without cardiac disease. This finding suggests that increased adiposity may activate inflammatory or hemostatic pathways even before overt cardiac disease is present. Previous population-based evidence has shown associations between fibrinogen and BMI, as well as other metabolic abnormalities, supporting the biological plausibility of this observation [14]. The absence of a significant correlation in cardiac diabetic participants may reflect the influence of additional factors associated with established cardiovascular disease. Hypertension was also highly prevalent, affecting more than four-fifths of the study population. However, prevalence was almost identical in both groups. This suggests that hypertension, although highly prevalent and clinically important, did not differentiate cardiac diabetic participants from diabetic participants in the present sample. The absence of a between-group difference should therefore not be interpreted as evidence that hypertension is unimportant in cardiovascular risk; rather, its high prevalence in both groups may have reduced its discriminatory value in this particular population. The inflammatory findings were particularly notable. Mean CRP was substantially higher in cardiac diabetic patients than in diabetic patients without cardiac disease. Furthermore, almost all cardiac diabetic participants were categorized as having abnormal

CRP, compared with approximately two-thirds of diabetic participants. The difference remained evident when participants were stratified according to BMI and hypertension [15]. These findings strengthen the observation that the cardiac diabetic group had a greater inflammatory burden. Elevated CRP has been consistently associated with cardiovascular risk, and meta-analytic evidence in patients with type 2 diabetes has shown associations between higher CRP concentrations and cardiovascular and all-cause mortality. These CRP findings matter because inflammation is increasingly recognized as a component of cardiometabolic disease. However, the present study design does not allow determination of whether elevated CRP contributed to cardiac disease or resulted from it. CRP is a nonspecific inflammatory marker and can be affected by multiple clinical conditions [16]. Therefore, elevated CRP should be interpreted as evidence of an inflammatory state rather than proof of a specific cardiovascular mechanism. This cautious interpretation aligns with previous literature emphasizing the strong epidemiological association of CRP with cardiovascular disease while noting uncertainty about its direct causal role [15]. Interestingly, CRP correlations with glycaemic indices were not statistically significant. Despite the significantly higher FBS and HbA1c in cardiac diabetic participants, CRP did not demonstrate a significant linear relationship with either parameter within the individual groups. This indicates that group-level differences do not necessarily imply a direct linear relationship between two continuous variables. Other factors may contribute to inflammatory status independently of glycaemic control [16]. Thus, the higher CRP observed in the cardiac diabetic group should not be interpreted as being explained solely by differences in FBS or HbA1c. The lipid findings were also noteworthy. Triglyceride abnormality was extremely common overall and was particularly frequent among cardiac diabetic participants. Almost all cardiac diabetic participants had abnormal triglycerides according to the study's classification [17]. Although the continuous triglyceride values did not differ significantly between groups, categorical analysis demonstrated a significant difference. This may indicate that the distribution around the selected clinical threshold mattered even when mean values did not differ significantly. The close relationship between triglyceride abnormalities and other metabolic disturbances is consistent with previous research on fibrinogen and cardiometabolic risk factors [18]. Fibrinogen also distinguished the groups. Cardiac diabetic participants had significantly higher mean fibrinogen levels, and abnormal fibrinogen was also more frequent. This supports the possibility of increased inflammatory and prothrombotic activity among diabetic patients with cardiac disease. Fibrinogen is an acute-phase reactant and the principal precursor of fibrin, and it has been associated with thrombosis, platelet aggregation, and cardiovascular disease [19]. Large prospective studies have also demonstrated associations between higher plasma fibrinogen levels and coronary heart disease and stroke. However, the causal nature of these associations remains uncertain. The correlation between fibrinogen and triglycerides in cardiac diabetic participants was particularly important. The correlation coefficient was 0.443 ($p < 0.001$), representing the strongest relationship identified in the analysis. The corresponding scatter plot demonstrated a positive trend with R^2 of approximately 0.195. This suggests that approximately one-fifth of the variation in fibrinogen in this group was explained by its linear relationship with triglycerides, although most variation remained unexplained. The association is consistent with previous evidence showing relationships between fibrinogen concentrations and triglycerides and other established metabolic risk factors [20]. The association between fibrinogen and triglycerides may be biologically plausible because both may reflect aspects of metabolic and inflammatory dysregulation. However, the cross-sectional design does not allow causal interpretation. It would be inappropriate to conclude

that elevated triglycerides directly cause increased fibrinogen or vice versa. Previous large-scale analyses have similarly emphasized that, although fibrinogen is associated with cardiovascular outcomes, its causal relevance requires further investigation [21]. Another finding was the positive relationship between fibrinogen and BMI among diabetic participants without cardiac disease. This association suggests that adiposity may drive hemostatic activation before cardiovascular disease becomes clinically apparent. The finding is potentially relevant because obesity was highly prevalent in the overall study population. Previous population-level research has reported positive associations between fibrinogen and BMI, supporting the plausibility of this observation [13]. The relationship between HDL and CRP differed between groups. In cardiac diabetic participants, CRP demonstrated a weak positive correlation with HDL, whereas in diabetic participants without cardiac disease, the relationship was weakly inverse and statistically significant. Because the correlation coefficients were small, interpret these findings cautiously [8]. They may reflect differences in the underlying clinical characteristics of the two groups, treatment patterns, or other unmeasured factors. Previous studies have demonstrated that inflammatory and hemostatic biomarkers may correlate with several metabolic variables. Still, these relationships are not necessarily uniform across populations. Several conventional lipid variables did not demonstrate significant correlations with CRP or fibrinogen. Similarly, age, systolic blood pressure, and diastolic blood pressure generally showed no significant associations. These findings emphasize that inflammatory and hemostatic biomarkers may provide information not fully captured by conventional clinical measurements. Fibrinogen, in particular, is associated with several established cardiovascular risk factors and retains an epidemiological relationship with cardiovascular outcomes [17]. The scatter diagrams further demonstrate that many of the investigated relationships were weak. For example, CRP versus BMI, FBS, HbA1c, and triglycerides generally showed low R^2 values. Similarly, fibrinogen showed limited linear relationships with several metabolic variables. These observations matter because statistical significance should not automatically imply a strong clinical association. The magnitude of the correlation also matters. A statistically significant association can have limited explanatory or clinical importance when the correlation coefficient and corresponding R^2 are small. The study has several strengths. First, the sample included equal numbers of cardiac diabetic and diabetic participants, facilitating direct comparison [22]. Second, the analysis incorporated a broad range of clinical and biochemical variables rather than focusing only on glucose levels. Third, we evaluated both CRP and fibrinogen, allowing assessment of inflammatory and hemostatic characteristics. Finally, the study included correlation analyses and graphical assessment of relationships between biomarkers and metabolic variables. These features provide a broader assessment of the metabolic, inflammatory, and hemostatic characteristics of the two study groups. Several limitations should nevertheless be acknowledged [8]. The supplied tables do not provide sufficient information regarding the study setting, sampling method, diagnostic criteria for cardiac disease, duration of diabetes, medication use, or other potential confounding factors. These variables may substantially influence CRP, fibrinogen, lipid profile, and glycaemic control. In observational research, inadequate adjustment for confounding can bias association estimates [18]. The cross-sectional design also prevents establishing temporal or causal relationships. Accordingly, interpret the observed differences in CRP and fibrinogen as associations, not evidence that these biomarkers caused cardiac disease. Similarly, the association between triglycerides and fibrinogen cannot establish a directional relationship. Appropriate reporting and interpretation of cross-sectional observational studies require explicit consideration of

study design, potential bias, confounding, and limitations on causal inference [19]. Another limitation is the absence of multivariable analysis in the supplied results. Because age, BMI, hypertension, diabetes duration, glycaemic control, and lipid abnormalities may influence inflammatory biomarkers simultaneously, adjusted regression analysis could better assess independent associations. Multivariable regression is particularly useful in observational research when the objective is to estimate associations while accounting for potential confounding variables [11]. Future studies should therefore include larger, more representative samples; clearly defined cardiac diagnostic criteria; information on diabetes duration and medication use; and longitudinal follow-up. Multivariable regression models incorporating clinically relevant confounders would also help determine whether CRP and fibrinogen remain independently associated with cardiac disease after adjustment. Standardized reporting according to the STROBE recommendations would further improve transparency and facilitate interpretation and comparison with other observational studies. Overall, the present findings suggest that diabetic patients with cardiac disease had a greater burden of poor glycaemic control, systemic inflammation, and elevated fibrinogen than diabetic patients without cardiac disease. The particularly strong association between fibrinogen and triglycerides in the cardiac diabetic group and the association between fibrinogen and BMI in the non-cardiac diabetic group provide potentially relevant hypotheses for future investigation. Nevertheless, these findings should be interpreted as associations rather than causal relationships, and prospective studies with appropriate adjustment for clinical and treatment-related confounders are needed to determine the independent prognostic significance of CRP and fibrinogen.

Conclusion

This study demonstrated that diabetic patients with cardiac disease had significantly higher fasting blood glucose, HbA1c, CRP, and fibrinogen levels than diabetic patients without cardiac disease. Cardiac diabetic participants also showed a greater burden of abnormal triglycerides, HDL, CRP, and fibrinogen. Fibrinogen demonstrated a moderate positive correlation with triglycerides in cardiac diabetic participants, while BMI was positively correlated with fibrinogen among diabetic participants without cardiac disease. CRP showed weaker and variable relationships with lipid parameters. Overall, the findings indicate that cardiac involvement in diabetes is associated with poorer glycaemic control and a greater inflammatory and hemostatic burden. CRP and fibrinogen may therefore have potential value as complementary biomarkers for characterizing cardiometabolic risk. However, prospective studies with standardized cardiac definitions, larger samples and multivariable analyses are needed before their independent clinical or prognostic value can be established.

Ethical Approval

The Institutional Research Ethics Board (IREB) of Khyber Medical College, Peshawar, approved the study on **3 September 2025 (Approval No. 628/DME/KMC)**, and we kept participant information confidential throughout.

Informed Consent

We obtained written informed consent from all participants before enrollment. We informed participants about the study objectives, procedures, potential benefits, and their right to participate voluntarily or withdraw at any time without affecting their medical care. The

authors strictly maintained participants' confidentiality and privacy and did not disclose personal identifying information.

Conflict of Interest

The authors declare no conflict of interest related to this study's conduct, authorship, or publication.

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This study received no external funding. The authors conducted the research using available institutional resources. No funding agency was involved in the study design, data collection, analysis, interpretation, or manuscript preparation.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available because of privacy and confidentiality considerations for the study participants.

Authors' Contributions

Bibi Hajira Ishaq: Conceptualization, study design, methodology, data collection, data interpretation, and manuscript drafting.

Muhammad Shabir: Methodology, data collection, literature review, data interpretation, and critical revision of the manuscript.

Zafar Ali: Study supervision, methodology, data interpretation, and critical review of the manuscript.

Muddasir Ahmed, Zahid Sarfaraz: Conceptualization, data collection, literature review, data interpretation, and critical revision of the manuscript.

Sobia Ali: Data collection, literature review, data analysis, interpretation of results, and manuscript revision. Study supervision, conceptualization, methodology, critical review, and final approval of the manuscript.

All authors: Contributed to the conception/design or interpretation of the work, reviewed and approved the final version, and agreed to be accountable for all aspects of the work in accordance with ICMJE authorship criteria.

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