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ORIGINAL ARTICLE

Association Between Diabetic Dyslipidemia and Vascular Tissue Damage: A Biochemical and Histopathological Evaluation

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ABSTRACT

Background: Type 2 diabetes mellitus is a major contributor to cardiovascular complications in diabetic dyslipidemia. However, there is no available data within Pakistan relating lipid abnormalities to actual histopathological vascular damage.

Objective: To evaluate the correlation between biochemical lipid profile disturbance with the vascular histopathological change in type 2 diabetes mellitus patients.

Methods: This cross-sectional study was conducted on 70 patients (50 diabetics, 20 non diabetic controls) who were patients of Elective vascular surgery in the Bakhtawar Amin Memorial Hospital, Multan & Sahara Medical College, Narowal from June 2022 to June 2023. Biochemically, fasting lipid profiles and HbA1c were assessed. H&E and Elastic Van Gieson staining were used to determine endothelial integrity, intimal-medial thickness, smooth muscle proliferation, and inflammatory infiltration in vascular tissues. Pearson correlation and multivariate regression were applied to the statistical analysis.

Results: Levels of LDL-C, triglycerides, total cholesterol, and HDL-C were significantly higher in diabetics than in controls ($p < 0.001$). Histopathological findings in diabetics showed a greater rate of endothelial disruption (76%), intimal thickening (66%), and smooth muscle hyperplasia (58%). In multivariate regression, LDL-C and triglycerides were independently related to intimal thickness ($p < 0.001$), while HDL-C was inversely related ($p < 0.01$). The variance in the vascular wall changes was explained by the regression model by 61%.

Conclusion: Histological evidence of vascular damage is strongly associated with diabetic dyslipidemia. In Pakistan, early lipid monitoring and management are crucial in preventing cardiovascular complications in type 2 diabetes, unless mandatory screening and timely treatment of lipid disorders are performed.

Keywords: Diabetic dyslipidemia, LDL-C, intimal thickening, endothelial disruption, vascular histopathology, Pakistan

INTRODUCTION

The disease burden of diabetes mellitus, especially type 2, is rapidly increasing in Pakistan and has become a critical public health issue. It is estimated that 33 million out of 8.5 million adults in Pakistan have diabetes, putting the country third in the world according to the International Diabetes Federation (IDF) 2021 estimates[1]. All these have contributed to the rapidly increasing prevalence of diabetes in urban as well as rural communities, including a sedentary lifestyle, urbanisation, poor dietary patterns, obesity, genetic predisposition, and poor public health awareness. The burden of this epidemic on the healthcare system has been substantial, and the incidence of diabetic complications, particularly cardiovascular diseases (CVD), which are the leading cause of death in diabetic people in the country, has risen[2].

Diabetic dyslipidemia, a unique abnormality of lipids characteristically seen in diabetic patients in Pakistan, is a critically neglected but important factor that contributes to the development of macrovascular complications in diabetes[3]. Typically, it is characterized by elevated triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), especially elevated small dense LDL particles, and low high-density lipoprotein cholesterol (HDL-C). These lipid abnormalities contribute to the pathogenesis of atherosclerosis, endothelial dysfunction, and vascular inflammation, which together accelerate vascular injury and increase morbidity of cardiovascular diseases[4].

Routine screening and management of dyslipidemia in diabetic patients in the Pakistani clinical setting is often inadequate because of a lack of standardized protocols, a lack of follow-up, economic constraints, and a lack of access to lipid-lowering medications[5]. In addition, histopathology and molecular research tools are generally not well utilized to link laboratory data to tissue-level changes, especially in cases involving vascular pathology. Although there are several works globally on biochemical aspects of dyslipidemia in diabetes, there is very little on the direct histopathological effects of dyslipidemia on the vascular tissues in diabetic individuals in Pakistan[6].

In order to develop a targeted, evidence-based strategy to prevent and manage diabetic vascular complications in the Pakistani population, it is necessary to understand how lipid profile abnormalities result in structural changes in the blood vessels[7]. Diabetics develop vascular tissue damage starting with endothelial dysfunction and leading to intimal thickening, smooth muscle proliferation, elastic fiber fragmentation, and inflammatory cell infiltration that can be reliably assessed by examination of vascular segments by histopathology. However, no data in Pakistan correlate these microscopic vascular changes with the biochemical lipid profiles in a well-structured clinical and laboratory setting[8].

This is a significant gap in our understanding of the local studies of diabetic macrovascular complications, lacking a good histopathological correlation. Since individuals in the population already have high cardiovascular risk as a result of genetic or environmental factors, establishing a biochemical histological link may allow for earlier diagnosis, better risk stratification, and earlier therapeutic interventions. Such study is particularly important in Pakistan, where the healthcare infrastructure is often overburdened and reactive, as well as where laboratory diagnostics are not uniformly available throughout regions[9].

To fill this gap, the current study was aimed to investigate the association between diabetic dyslipidemia and vascular tissue damage in a cohort of Pakistani patients undergoing vascular surgical procedures. This study correlates fasting lipid profiles with histopathological changes in vascular tissues such as endothelial injury, intimal-medial thickening, and inflammatory changes, as concrete, localized evidence of structural damage of lipid abnormalities in diabetes[10]. This biochemical and morphological

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correlation will not only provide a better understanding of the disease process but will also assist in the development of more effective, Pakistan-specific clinical guidelines for the management of diabetic dyslipidemia and reduction of vascular complications[11].

MATERIALS AND METHODS

Study Design and Duration: This is a one-year (June 2022 – June 2023) short-term cross-sectional study carried out in association with the Department of Pathology, Sahara Medical College, Narowal and Bakhtawar Amin Memorial Hospital, Multan, Pakistan. This study was to evaluate the association between diabetic dyslipidemia and vascular tissue damage on a combined biochemical and histopathological basis.

Sample Size and Power Analysis: On the basis of power analysis using G*Power software version 3.1, 70 patients were included in the study. For a medium to large effect size of 0.40, a significance level of 0.05, and a statistical power of 80%, the minimum required sample size was 64. To account for possible exclusions on poor tissue quality or incomplete data, we set the final sample size at 70 to provide enough power for subgroup comparisons and regression modeling.

Study Population and Sampling Technique: A consecutive purposive sampling technique was used in the study. Of the total of 70 patients recruited, 50 were diagnosed cases of type 2 diabetes mellitus for more than 5 years (Group A) and 20 were non diabetic controls (Group B) undergoing vascular surgeries for trauma or nonatherosclerotic conditions. Group classification was also based on glycemic history and HbA1c, and all participants were between 40 and 70 years of age.

Inclusion and Exclusion Criteria: The patients included were adults aged between 40 and 70 years, with type 2 diabetes mellitus (as per ADA criteria) for more than 5 years (Group A), and undergoing elective vascular surgical procedures for which excisable vascular tissue was available for analysis. Patients who gave written informed consent only were enrolled. They excluded patients if they had type 1 diabetes mellitus, suffered from myocardial infarction or stroke within the last six months, were on lipid-lowering therapy within the past three months, or suffered from chronic inflammatory, autoimmune, or malignant diseases. Also, tissue specimens deemed inadequate or degraded on histological processing were not included in the final evaluation.

Ethical Considerations: The research followed the ethical principles in the Declaration of Helsinki (2013). The current study obtained ethical approval from the Institutional Review Boards. All participants gave written informed consent after the purpose, procedures, potential risks, and benefits of the study were explained in their language.

Biochemical Investigations: Biochemical profiling of pre-operative fasted blood collection was performed in all participants. I collected early morning blood samples (10 mL) and analyzed them in the central laboratory by automated enzymatic colorimetric techniques on a Roche COBAS 6000 analyzer. For the following parameters, total Cholesterol (TC), triglycerides (TG), high density lipoprotein Cholesterol (HDL-C), and the latter calculated by the Friedewald equation, low density lipoprotein Cholesterol (LDL-C). Glycated Hemoglobin (HbA1c) was measured using high-performance liquid chromatography (HPLC) to categorize glycemic control, as well. Interpretation of lipid profile values was done according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria.

Tissue Collection and Histopathological Processing: Vascular tissue specimens of 1–2 cm length were excised from patients undergoing elective vascular surgeries and immediately fixed in 10% neutral buffered formalin. The tissue specimens were labeled and sent to the Histopathology Laboratory at Sahara Medical College. Paraffin embedding, sectioning at 4 microns, and routine H&E and EVG stains were used to process tissues for general morphological assessment and assessment of elastic fiber integrity and vessel wall architecture.

Histological Evaluation Parameters: The histopathological examination was to evaluate the endothelial integrity (intact vs denuded), intimal and medial thickness (measured using a calibrated ocular micrometer), smooth muscle cell hyperplasia, and inflammatory cell infiltration. The biochemical data were assessed by two independent senior histopathologists blinded to the biochemical data to minimize observational bias. Semi-quantitative visual field scoring was performed under high-power magnification (400x), and the level of intimal thickening and cellular proliferation was graded. The inflammatory infiltrates were graded as absent, mild, moderate, or severe.

Statistical Analysis: IBM SPSS Statistics version 25.0 was used to code and enter all collected data and analyze. These continuous variables were expressed as mean ± Standard Deviation (SD) age, lipid concentrations, HbA1c, and histological measurements. The frequencies and percentages were presented in categorical variables. Mean differences between diabetic and non-diabetic groups were compared with independent sample t tests. One-way ANOVA was used for multiple group comparisons, and Pearson's correlation coefficient was used to evaluate the effect of lipid parameters on vascular damage. In addition, multivariate linear regression analysis was carried out to find the significant predictors of histopathological damage, such as intimal thickening and smooth muscle hyperplasia. All tests were applied with a p-value < 0.05 considered statistically significant.

RESULTS

We enrolled a total of n=70 subjects, 50 of whom were made up of subjects with type 2 diabetes mellitus (Group A) and 20 were non-diabetic subjects (Group B). The mean age of the diabetic group was 58.6 ± 6.2 years, and 56.1 ± 5.8 years for the non diabetic group. Despite this difference not being statistically significant ($p = 0.09$), diabetic patients had a significantly higher body mass index (BMI) of 28.3 ± 2.5 kg/m² than controls ($p < 0.05$). There was no significant difference in the gender distribution in both groups; the diabetic group showed a male predominance (30 males, 20 females) while controls had 12 males and 8 females.

Biochemically, diabetic individuals had marked dyslipidemia. Diabetics (225.4 ± 34.2 mg/dL) had significantly higher total cholesterol levels than controls (182.6 ± 25.3 mg/dL, $p < 0.01$). Furthermore, mean triglyceride levels were significantly higher in diabetics (212.3 ± 40.1 mg/dL) than in non diabetics (139.5 ± 30.6 mg/dL, $p < 0.001$). Mean LDL-C values in the diabetic group were 147.6 ± 28.4 mg/dL and 98.3 ± 19.6 mg/dL in the non diabetic group ($p < 0.001$). On the other hand, the HDL-C levels were significantly reduced in diabetics (36.2 ± 5.8 mg/dL) than non diabetics (49.1 ± 6.4 mg/dL, $p < 0.001$). HbA1c measured as an index of glycemic control was significantly different between the groups; 8.5±1.0% in diabetics and 5.4±0.6% in non-diabetics ($p < 0.001$). Table 1 summarizes the comparative biochemical and demographic characteristics.

Table 1: Demographics and Biochemical Profile of Study Participants

Parameter	Diabetic Group (n=50)	Non-Diabetic Group (n=20)	p-value
Age (years)	58.6 ± 6.2	56.1 ± 5.8	0.09
Gender (M/F)	30/20	12/8	–
BMI (kg/m²)	28.3 ± 2.5	26.4 ± 2.1	<0.05
HbA1c (%)	8.5 ± 1.0	5.4 ± 0.6	<0.001
Total Cholesterol (mg/dL)	225.4 ± 34.2	182.6 ± 25.3	<0.01
Triglycerides (mg/dL)	212.3 ± 40.1	139.5 ± 30.6	<0.001
LDL-C (mg/dL)	147.6 ± 28.4	98.3 ± 19.6	<0.001
HDL-C (mg/dL)	36.2 ± 5.8	49.1 ± 6.4	<0.001

Excised vascular tissues were subjected to histopathological examination, and the diabetic and non diabetics exhibited significant differences. In 76% of diabetic specimens, endothelial disruption was observed, whereas in 10% of control samples, such changes were seen ($p < 0.001$). Intimal thickening greater than 200 µm was found in 66% of diabetics and only 5% of controls ($p <$

0.001). Diabetic tissues had 58% smooth muscle hyperplasia, compared to 5% in non diabetic tissues ($p < 0.001$). Diabetic specimens had been found to have 52% inflammatory cell infiltration and none in the control group ($p < 0.001$). The results are summarized in Table 2.

Table 2: Histopathological Findings in Vascular Tissues

Histopathological Finding	Diabetic Group (n=50)	Non-Diabetic Group (n=20)	p-value
Endothelial Disruption	38 (76%)	2 (10%)	<0.001
Intimal Thickening (>200 μm)	33 (66%)	1 (5%)	<0.001
Smooth Muscle Hyperplasia	29 (58%)	1 (5%)	<0.001
Inflammatory Cell Infiltration	26 (52%)	0 (0%)	<0.001

Pearson correlation coefficients were calculated to quantify the relationship between lipid abnormalities and vascular damage. Intimal thickness showed a strong positive correlation with LDL-C ($r = 0.68$, $p < 0.001$), which indicates a major contribution of LDL-C to structural vascular change. Intimal thickness was also positively correlated with triglycerides ($r = 0.59$, $p < 0.01$). On the contrary, HDL-C was statistically significantly negatively correlated with intimal thickness ($r = -0.47$, $p < 0.01$), which may suggest a protective effect. Table 3 shows the correlation matrix.

Table 3: Correlation Between Lipid Parameters and Intimal Thickening

Lipid Parameter	Correlation with Intimal Thickness (r)	p-value
LDL-C	0.68	<0.001
Triglycerides	0.59	<0.01
HDL-C	-0.47	<0.01

A multivariate linear regression analysis was performed to further explore the independent effect of lipid parameters and demographic variables on vascular structural damage. In this context, the independent variables were LDL-C, triglycerides, HDL-C, age, and BMI, and the dependent variable was intimal thickness. However, independent predictors of increased intimal thickness were LDL-C ($B = 0.42$, $p < 0.001$) and triglycerides ($B = 0.36$, $p < 0.01$). A protective association was still found in HDL-C with a significant negative coefficient ($B = -0.28$, $p < 0.01$). Age ($p = 0.07$) and BMI ($p = 0.12$) did not have statistically significant predictive value. The model had a good fit with an adjusted R^2 of 0.61, which is 61% of the variability in intimal thickening explained. Table 4 presents the detailed regression output.

Table 4: Multivariate Regression Analysis for Predictors of Intimal Thickening

Predictor Variable	Unstandardized Coefficient (B)	Standard Error	Standardized Beta (β)	p-value
LDL-C	0.42	0.09	0.52	<0.001
Triglycerides	0.36	0.11	0.43	<0.01
HDL-C	-0.28	0.10	-0.39	<0.01
Age	0.05	0.06	0.18	0.07
BMI	0.03	0.04	0.12	0.12

Finally, this study shows a strong association between diabetic dyslipidemia and histological structural vascular damage. High levels of LDL-C and triglycerides were associated with positive correlation of endothelial disruption, intimal thickening, and smooth muscle proliferation, and low levels of HDL-C were inversely associated with vascular injury. After adjusting for age and BMI in multivariate analysis, these relationships remained significant, indicating that lipid abnormalities are an independent contributor to the pathogenesis of vascular damage in diabetic patients. These results demonstrate that dyslipidemia in type 2 diabetes is not only a biochemical disturbance but also a contributing cause of the structural deterioration of vascular tissue. These results have implications that are clinically relevant and call for early lipid control and vascular screening in diabetic populations, especially in the Pakistani healthcare setting, where cardiovascular risk is high.

DISCUSSION

These findings provide strong histopathological evidence for a link between diabetic dyslipidemia and the structural damage of the vessel, a relationship that has been widely theorized but rarely demonstrated with tissue correlation in the Pakistani population[12]. Total cholesterol, triglycerides, and LDL cholesterol were significantly higher and HDL cholesterol markedly lower in patients with type 2 diabetes. The histological changes associated with these lipid abnormalities included endothelial disruption, intimal thickening, smooth muscle proliferation, and inflammatory infiltration[13].

This is consistent with previous global studies describing the atherogenic potential of LDL and triglycerides in the diabetic milieu. The high positive correlations found between LDL-C and intimal thickness ($r = 0.68$) as well as triglycerides ($r = 0.59$) support the hypothesis that these lipids are directly involved in early structural remodeling of arterial walls[14]. HDLC also showed a protective inverse correlation ($r = -0.47$), indicating that it is involved in the maintenance of vascular integrity. The observations are consistent with the 'response to injury' hypothesis of atherosclerosis and are characterized by lipid infiltration, followed by endothelial damage and inflammatory cascades, and vascular remodeling[15].

The presence of inflammatory infiltrates and smooth muscle hyperplasia in over half of diabetic samples, but in almost none of control samples, indicates the existence of an active proliferative and immune-modulated process caused by chronic metabolic stress. These findings are clinically significant, since they demonstrate that vascular injury is not an epiphenomenon of macrovascular disease but is an early and ongoing pathological process in poorly controlled diabetes[16].

This is particularly important for Pakistan at a time when there is a rapid increase in the prevalence of noncommunicable diseases, and diabetes and cardiovascular disease are key contributors. Despite the awareness of dyslipidemia, lipid screening and intensive lipid-lowering therapy are still underutilized in clinical practice. The strong histopathological associations identified here underscore the need to revise screening protocols and treatment thresholds in diabetic populations, particularly in primary care and tertiary vascular centers[17, 18].

This study has limitations in terms of a small sample size from two institutions, which may limit generalizability. In addition, the inflammatory cytokines or oxidative stress markers were not directly quantified. Further elucidation of the mechanistic pathways will be dependent on future studies with molecular markers[19].

CONCLUSION

This is an important biochemical and histological study that demonstrates that diabetic dyslipidemia, especially elevated LDL-C and triglyceride levels, is strongly linked to vascular tissue damage. In histopathological analysis, these lipid abnormalities independently predict intimal thickening, endothelial disruption, and smooth muscle proliferation. The results highlight the relevance of early lipid profile monitoring and management in diabetic individuals to avoid irreversible vascular injury. In light of the increasing incidence of cardiovascular and metabolic diseases in Pakistan, the incorporation of vascular assessment in routine diabetic care can substantially benefit patients' outcomes as well as prevent long-term complications.

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Data Availability Statement: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Authors contribution:
TH: Conceptualization, Study Design, Supervision
QY: Literature Review, Data Collection
GEN: Manuscript writing, Critical Analysis, Statistical analysis, English Editing
IUH: Statistical Analysis, Data Interpretation
SF: Data Curation, Formatting, References
MSZKS: Final Review, Technical Editing, Approval of Manuscript

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