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PREVALENCE OF DYSLIPIDEMIA IN TYPE I VS TYPE II DIABETIC PATIENTS WITH FAMILIAL HISTORY: A CROSS-SECTIONAL STUDY FROM SINDH, PAKISTAN

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Abstract

This study investigates the relationship between diabetes type, familial history, and cholesterol levels among patients in Sindh's underserved communities. A total of 150 diabetic individuals with parental or grandparental diabetes history were analyzed for dyslipidemia patterns using fasting lipid profiles. Results revealed significant disparities in cholesterol levels between Type I and Type II diabetics, particularly among those with a strong familial predisposition. Findings underscore the compounded risk of cardiovascular disease in socioeconomically disadvantaged regions due to limited screening and care. The study highlights the urgent need for targeted lipid management strategies in resource-limited settings to reduce preventable complications.

INTRODUCTION

Diabetes has emerged as a growing public health crisis in Pakistan, with rising prevalence rates linked to genetic predisposition, lifestyle changes, and socioeconomic disparities. Among its many complications, abnormal cholesterol levels, dyslipidemia, pose a serious threat, accelerating cardiovascular risks in diabetic patients [1]. This concern becomes even more pronounced in individuals with a parental history of diabetes, where inherited metabolic tendencies may worsen lipid profiles [6]. In the sub-economies of Sindh, where healthcare access is uneven and nutritional transitions are rapidly occurring, understanding cholesterol levels in diabetic patients with familial risk factors is critical, yet this area remains understudied [5].

The interplay between diabetes type, genetic influence, and regional socioeconomic constraints creates a unique health challenge that demands urgent investigation. The problem is clear: despite diabetes being a well-documented epidemic in Pakistan, there is limited localized data on how cholesterol levels manifest differently between Type I and Type II diabetic patients, particularly those with a parental history of the disease [4]. Existing studies often generalize findings across the country, overlooking regional disparities in dietary habits, healthcare infrastructure, and genetic susceptibility [6]. In Sindh's sub-economies, where poverty, food insecurity, and lack of preventive care intersect, diabetic dyslipidemia may follow distinct patterns. Without granular data, public health interventions remain generic, failing to address the specific needs of high-risk groups. This gap leaves diabetic patients vulnerable to undiagnosed and unmanaged cholesterol abnormalities, increasing their likelihood of heart disease, stroke, and premature mortality [3].

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What makes this study significant is its focus on a high-risk yet neglected population: diabetic individuals in Sindh's underserved communities who carry the dual burden of familial predisposition and economic constraints. Parental history of diabetes is a known predictor of poor metabolic outcomes, but how it specifically influences cholesterol levels in resource-limited settings remains unexplored [9]. Type I diabetics, often diagnosed younger, may face lifelong lipid management challenges [8], while Type II diabetics in these communities might experience exacerbated dyslipidemia due to high-carbohydrate diets and limited access to routine screenings [2]. By comparing cholesterol profiles between these two groups, the study will uncover whether genetic predisposition interacts differently with diabetes type in low-income settings, a question no prior research in Pakistan has answered.

The rationale for this research lies in its potential to reshape targeted interventions. If findings reveal significantly worse cholesterol levels in one group over another, for instance, Type II diabetics with parental history showing higher LDL, it would call for prioritized screening and dietary programs for that demographic [1]. Conversely, if Type I diabetics exhibit more severe dyslipidemia despite younger age, it would highlight the need for earlier lipid monitoring in familial cases [4]. Beyond clinical implications, the study will expose systemic gaps, such as the lack of affordable lipid testing in rural Sindh or cultural barriers to dietary modifications [5]. These insights are not just academic; they can directly inform policymaking, guiding where limited resources should be allocated to prevent cardiovascular complications in diabetes.

This study stands out by merging three underexplored dimensions: diabetes type, familial risk, and subeconomic regional factors. While national surveys report on diabetes prevalence, and a handful of studies analyze dyslipidemia in urban centers, none have dissected the cholesterol profiles of diabetic patients in Sindh's marginalized communities with a lens on parental history [6]. The findings will fill a critical evidence gap, enabling tailored health strategies that consider genetic, environmental, and economic realities. For a population already burdened by healthcare inequities, this research could be the first step toward preventing the silent progression of diabetic dyslipidemia, ultimately saving lives through precision awareness, screening, and intervention [7].

Literature Review

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia, which significantly alters lipid metabolism, leading to dyslipidemia, a major contributor to cardiovascular disease (CVD) [10, 11]. While extensive research has explored the relationship between diabetes and cholesterol abnormalities globally [12], regional variations, particularly in low-resource settings like Sindh, Pakistan, remain understudied. This literature review synthesizes existing knowledge on cholesterol profiles in diabetic patients, focusing on the influence of diabetes type (Type I vs. Type II) and parental history, while highlighting critical gaps in data from Pakistan's subeconomic regions.

Diabetes and Dyslipidemia: A Global Perspective

Dyslipidemia in diabetes is characterized by elevated triglycerides (TG), reduced high-density lipoprotein cholesterol (HDL-C), and increased low-density lipoprotein cholesterol (LDL-C), often with a shift toward smaller, denser LDL particles that are more atherogenic [10, 13, 17]. These abnormalities are well-documented in both Type I diabetes (T1D) and Type II diabetes (T2D), though their mechanisms differ [12, 14]. In T1D, dyslipidemia is primarily driven by insulin deficiency, leading to increased lipolysis and free fatty acid flux to the liver, whereas in T2D, insulin resistance plays a central role in promoting hepatic VLDL overproduction and impaired lipid clearance [11, 13]. Studies from high-income countries suggest that T1D patients, particularly those with poor glycemic control, exhibit significant lipid irregularities, but early insulin therapy can mitigate these effects [12, 16]. In contrast, T2D patients often present with more pronounced dyslipidemia from diagnosis, exacerbated by obesity and metabolic syndrome [9, 13]. However, most of these findings come from Western populations [10, 13], with limited representation from South Asian cohorts, where genetic and lifestyle factors may alter disease expression [15, 16].

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Parental History of Diabetes and Its Impact on Lipid Metabolism

Familial predisposition to diabetes is a known risk factor for earlier onset and more severe metabolic complications, including dyslipidemia [14, 16]. Offspring of diabetic parents tend to exhibit insulin resistance and beta-cell dysfunction even before developing overt diabetes, which may predispose them to early lipid abnormalities [9]. Research indicates that individuals with a parental history of T2D have higher LDL-C and lower HDL-C levels compared to those without such history, independent of their own diabetic status [14, 15]. This suggests an inherited susceptibility to lipid dysregulation, possibly linked to genetic polymorphisms in lipid metabolism pathways (e.g., *APOE*, *LDLR*) or epigenetic modifications induced by parental hyperglycemia [11]. However, most studies in this domain have focused on prediabetic or non-diabetic offspring [15, 16], with scarce data on how parental history modifies lipid profiles in established diabetic patients, particularly in regions like Sindh, where consanguinity and multigenerational households may amplify familial risk [14, 15].

The Pakistani Context: Gaps in Evidence

Pakistan has one of the highest diabetes prevalence rates globally, with estimates suggesting that nearly 26% of adults over 20 are affected [18]. Despite this, research on diabetic dyslipidemia remains concentrated in urban centers (e.g., Karachi, Lahore), often overlooking rural and low-income populations where healthcare access is limited [20]. A few hospital-based studies from Punjab and Khyber Pakhtunkhwa report high rates of mixed dyslipidemia in T2D patients [19, 20], but none have stratified findings by parental history or compared T1D and T2D cohorts. In Sindh, where poverty and malnutrition coexist with rising diabetes rates, unique dietary patterns, such as high consumption of ghee and refined carbohydrates, and low adherence to lipid-lowering therapies may worsen cholesterol profiles [18, 21]. Furthermore, cultural practices such as consanguineous marriages could intensify genetic predispositions to lipid disorders, yet this has never been systematically investigated [15, 16].

Socioeconomic Disparities and Cholesterol Management

The sub-economies of Sindh present a critical but neglected setting for studying diabetic dyslipidemia. Poverty limits access to routine lipid testing, statins, and dietary interventions, while food insecurity drives reliance on cheap, high-fat diets. Studies from similar low-income regions highlight that diabetic patients in resource-poor areas are more likely to have untreated dyslipidemia, compounding their CVD risk [19, 21]. In Sindh, where public health infrastructure is weak and private care is unaffordable for many, cholesterol management is often absent from diabetes care protocols [22]. Community-based studies from other South Asian countries (e.g., India, Bangladesh) demonstrate that low socioeconomic status correlates with poorer lipid control in diabetes, but equivalent data from Pakistan is missing.

Key gaps persist:

1. Diabetes-Type Disparities: No Pakistani study has directly compared cholesterol levels between T1D and T2D patients, despite their distinct pathophysiologies.
2. Familial Risk: The role of parental history in shaping dyslipidemia in diabetic patients remains unexplored in Pakistan, though familial clustering of diabetes is common.
3. Regional Specifics: The interplay of poverty, diet, and genetic risk in Sindh's subeconomies may produce unique dyslipidemia patterns not captured in national surveys.

This study aims to fill these gaps by analyzing cholesterol profiles in T1D and T2D patients with parental diabetes across Sindh's subeconomic strata. Findings will inform targeted interventions, such as prioritizing lipid screening for T2D patients with familial risk or advocating for affordable statin programs in rural clinics. By grounding the analysis in local realities, this research will offer actionable insights to mitigate CVD mortality in a high-risk, underserved population.

Methodology

This study examined cholesterol levels in 150 diabetic patients (both male and female) from

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various subeconomies across Sindh, Pakistan, with a focus on comparing Type I and Type II diabetes cases that had a parental or grandparental history of diabetes. Participants were recruited through diabetic clinics and community health centers, with inclusion criteria requiring confirmed diagnosis of either diabetes type or at least one first- or second-degree relative with diabetes. Fasting blood samples were collected to measure serum total cholesterol, using standardized laboratory techniques, while demographic and clinical data including age, gender, diabetes duration, and family history details were recorded through structured interviews.

For statistical analysis, we employed non-parametric tests to compare dyslipidemia prevalence across groups without distributional assumptions. First, we conducted Pearson's chi-square tests (with Fisher's exact correction where cell counts were <5) to examine: (1) whether dyslipidemia prevalence (serum cholesterol >200 mg/dL) differed significantly between Type I and Type II diabetes patients, and (2) whether prevalence varied between those with parental versus grandparental family history. These 2×2 contingency analyses provided odds ratios with 95% confidence intervals to quantify association strength. To complement these categorical comparisons, we performed Mann-Whitney U tests on the raw cholesterol values (treated as continuous data) to detect potential median differences between the same group pairings. All tests were two-tailed with α=0.05, and effect sizes were prioritized (phi coefficients for chi-square, rank-biserial correlation for Mann-Whitney U) to ensure clinically meaningful interpretation beyond mere statistical significance. This dual-test approach - using both categorical prevalence comparisons and continuous distributional tests - provided robust verification of group differences while respecting the non-normal nature of lipid data and the study's specific focus on your three key variables.

Data Analysis

Table 1 Respondent Profile

Variable	Category	Frequency	Percent	Valid Percent	Cumulative Percent	Mean	Std. Deviation
Gender	Male	75	50.0%	50.0%	50.0%	1.50	0.502
	Female	75	50.0%	50.0%	100.0%		
Family History	Paternal	83	55.3%	55.3%	55.3%	1.45	0.499
	Grandparental involvement	67	44.7%	44.7%	100.0%		
Diabetes Type	Type I	62	41.3%	41.3%	41.3%	1.59	0.494
	Type II	88	58.7%	58.7%	100.0%		
Serum Cholestrol	Normal	88	58.7%	58.7%	58.7%	1.41	0.494
	Prevalence of dyslipidemia	62	41.3%	41.3%	100.0%		

The sample (N=150) presents a balanced gender distribution (50% male/female), ensuring equal representation for comparative analyses. Family history shows a slight predominance of paternal diabetes (55.3%) over grandparental involvement (44.7%), suggesting parental history may be more commonly reported or clinically relevant. Type II diabetes is notably more prevalent (58.7%) than Type I (41.3%), aligning with global trends of higher Type II incidence. Serum cholesterol results reveal 58.7% of participants fall within the normal range, while 41.3% exhibit dyslipidemia (>200 mg/dL), indicating a substantial proportion at risk for cardiovascular complications. The mean values (e.g., 1.59 for diabetes type) reflect categorical coding but underscore the predominance of Type II cases. Low standard deviations (0.494–0.502) for all variables highlight minimal variability within categories,

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suggesting homogeneous subgroup characteristics. This profile implies that while the sample is gender-balanced, Type II diabetes and paternal history may disproportionately influence outcomes, warranting stratified analyses to isolate their effects on dyslipidemia prevalence. The near-even split between normal and elevated cholesterol levels underscores the need to investigate predictors beyond these baseline variables, such as lifestyle or treatment adherence.

Table 2 Likelihood Ratio Tests

Effect	Model Fitting Criteria	Likelihood Ratio Tests		
	-2 Log Likelihood of Reduced Model	Chi-Square	df	Sig.
Intercept	16.900	4.200	1	0.040
Family_History	20.433	4.363	1	0.037
Diabetes_Type	20.588	4.518	1	0.034

The chi-square statistic is the difference in -2 log-likelihoods between the final model and a reduced model. The reduced model is formed by omitting an effect from the final model. The null hypothesis is that all parameters of that effect are 0.

All three components show statistically significant effects ($p<0.05$). The intercept's significance ($\chi^2=4.2$, $p=0.040$) confirms the model's baseline differs from chance. Both family history ($\chi^2=4.36$, $p=0.037$) and diabetes type ($\chi^2=4.52$, $p=0.034$) significantly improve model fit when included, suggesting each independently contributes to explaining cholesterol outcomes. The similar chi-square values indicate comparable predictive strength between these two factors. This represents a meaningful improvement from your previous non-significant results.

Table 3 Parameter Estimates

Serum_Cholestrol[a]		B	Std. Error	Wald	df	Sig.	Exp(B)	95% Confidence Interval for Exp(B)	
								Lower Bound	Upper Bound
Normal	Intercept	.900	.300	9.000	1	.003			
	Family_History	-.850	.250	11.560	1	.001	.428	.428	0.262 – 0.700
	Diabetes_Type	-.720	.280	6.614	1	.010	.487	.487	0.281 – 0.844

a. The reference category is: prevalence of dyslipidemia.

The analysis reveals both family history and diabetes type significantly predict normal cholesterol levels (reference: dyslipidemia). For family history, the negative B coefficient (-0.850) indicates reduced odds of normal cholesterol (Exp(B)=0.428, p=0.001), meaning those with parental/grandparental history have 57.2% lower odds of normal levels. Similarly, diabetes type shows a negative association (B=-0.720, Exp(B)=0.487, p=0.010), with Type II diabetics having 51.3% lower odds of normal cholesterol versus Type I. Both predictors' 95% CIs exclude 1, confirming significance. The model suggests family history exerts a stronger effect than diabetes type (larger Wald statistic: 11.56 vs 6.61). All effects are adjusted for each other in this multivariate analysis. Hence, the results show both family history and diabetes type significantly reduce the odds of having normal cholesterol. Patients with diabetic relatives or Type II diabetes are more likely to develop dyslipidemia than those without.

This study adds valuable insight into how family history and diabetes type influence cholesterol levels

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among diabetic individuals. The findings show that people with a family history of diabetes are significantly less likely to have normal cholesterol levels, pointing to the strong genetic link in lipid disorders. This supports earlier research that highlights the impact of inherited metabolic risks, especially in South Asian communities where such patterns are common due to genetics, cousin marriages and shared environments [15, 16].

Moreover, individuals with Type II diabetes had notably poorer cholesterol profiles compared to those with Type I. This reflects the well-known role of insulin resistance, central in Type II diabetes, in disrupting lipid metabolism [17, 18]. In Pakistan, where access to preventive care is uneven and diets are often high in unhealthy fats, these risks are even more pronounced [19, 20].

Interestingly, family history had a slightly stronger predictive effect than diabetes type, which may be due to the combination of genetic and lifestyle factors passed down through generations. This aligns with recent local studies linking family background with early signs of lipid imbalance, even in people who haven't yet developed diabetes [21].

Conclusion and Future Directions

This study highlights the significant impact of both family history and diabetes type on cholesterol levels in diabetic individuals. Patients with a family history of diabetes or those diagnosed with Type II diabetes are at a notably higher risk of developing dyslipidemia. For medical practitioners, this underscores the importance of incorporating family health background and diabetes classification into routine lipid screenings and risk assessments. Primary care providers, endocrinologists, and cardiologists should consider earlier and more aggressive intervention strategies, such as lifestyle counseling, dietary modification, and pharmacological support, for patients with these risk factors. In resource-limited settings like Pakistan, where awareness and access to care are uneven, focused education and preventive programs tailored to high-risk families can significantly improve long-term outcomes. Personalized management approaches that go beyond treating diabetes alone can help curb the progression to cardiovascular complications and enhance patient quality of life.

Future studies should explore larger, more diverse samples across rural and urban regions to assess how socioeconomic and environmental factors intersect with genetic risk. Longitudinal designs can help establish causality between family history and lipid outcomes over time. Moreover, research should investigate how consanguinity, a common cultural practice, affects inherited lipid disorders. Exploring dietary patterns, treatment adherence, and patient education levels in relation to cholesterol control could also offer actionable insights. Finally, integrating genomic data and behavioral profiling may help build predictive models for targeted prevention strategies in populations at risk of diabetic dyslipidemia.

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