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Original Article

Comparison of Dyslipidemia Pattern Between Newly Diagnosed and Known Type 2 Diabetics

Hamza Nawaz[1], Roshan Tahir[1], Ehtisham Ul Haq[1], Abdul aziz[1], Zubair Ahmed[1]

[1] Pak Emirates Military Hospital Rawalpindi, Rawalpindi, Pakistan

*Corresponding author: Hamza Nawaz, drhamzaim@outlook.com

ABSTRACT

Background: Dyslipidemia is a major cardiovascular risk factor in type 2 diabetes mellitus and may already be substantially present at the time of diagnosis. Objective: To compare the pattern and severity of dyslipidemia between newly diagnosed and established type 2 diabetic patients. Methods: This prospective comparative study was conducted in the medical unit of Pak Emirates Military Hospital, Rawalpindi, from April to September 2023. A total of 120 patients with type 2 diabetes mellitus were recruited through consecutive sampling and allocated into two groups: 60 newly diagnosed and 60 with established disease. After a 10- to 12-hour fast, blood samples were obtained for fasting blood sugar, glycated hemoglobin, total cholesterol, triglycerides, HDL-C, LDL-C, and VLDL. Dyslipidemia was defined according to NCEP ATP III criteria. Data were analyzed in SPSS version 23 using independent-samples t-test and chi-square test, with $p<0.05$ considered significant. Results: Newly diagnosed patients had significantly higher total cholesterol (222.34 ± 42.98 vs 198.50 ± 36.29 mg/dL; p=0.001), triglycerides (216.26 ± 70.70 vs 180.64 ± 53.07 mg/dL; p=0.002), LDL-C (144.37 ± 36.93 vs 119.02 ± 31.05 mg/dL; p<0.001), VLDL (43.25 ± 14.14 vs 36.13 ± 10.62 mg/dL; p=0.002), and lower HDL-C (35.60 ± 6.78 vs 43.44 ± 9.61 mg/dL; p<0.001). Any dyslipidemia was present in 100.0% of newly diagnosed and 96.7% of established patients. Low HDL-C was the most prominent abnormality, affecting 90.0% versus 55.0%, respectively. Conclusion: Newly diagnosed type 2 diabetic patients exhibit a markedly worse lipid profile than established diabetics, supporting comprehensive lipid assessment at the time of diagnosis. Keywords: Type 2 diabetes mellitus, dyslipidemia, HDL-C, LDL-C, triglycerides, lipid profile.

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INTRODUCTION

Type 2 diabetes mellitus is one of the most important non-communicable diseases driving global morbidity and mortality, with its burden increasing across both developed and developing regions. The International Diabetes Federation estimated that more than 500 million adults were living with diabetes worldwide, underscoring the scale of the current metabolic health crisis (1). In Pakistan, the situation is particularly concerning, as national data have demonstrated a very high combined prevalence of diabetes and pre-diabetes, placing a substantial proportion of the adult population at risk of long-term vascular, renal, and neurologic complications (2). A major challenge within this disease spectrum is delayed diagnosis, whereby many individuals remain unaware of their diabetic status until metabolic derangements or target-organ damage have already developed (3). This diagnostic delay has important implications not only for glycemic control but also for the parallel cardiovascular risk profile that frequently accompanies type 2 diabetes.

Among the metabolic abnormalities associated with type 2 diabetes, dyslipidemia occupies a central position because it directly contributes to accelerated atherosclerosis and excess cardiovascular morbidity. Diabetic dyslipidemia is classically characterized by elevated triglycerides, reduced high-density lipoprotein cholesterol, and qualitative as well as quantitative abnormalities in low-density lipoprotein particles, particularly the predominance of smaller, denser, and more atherogenic subfractions (4). These lipid disturbances are not merely biochemical anomalies; they represent

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clinically meaningful drivers of coronary artery disease, cerebrovascular events, and peripheral vascular disease in an already high-risk population. Reports from Pakistan and neighboring countries indicate that dyslipidemia affects a substantial majority of patients with type 2 diabetes, with prevalence estimates ranging from approximately 70% to well above 90%, depending on the population studied and the thresholds applied (3,5). Such data reinforce the need to identify when during the natural history of diabetes these lipid abnormalities are most pronounced and most clinically actionable.

A particularly relevant but insufficiently resolved question is whether the lipid burden is more severe at the time of initial diagnosis or during the later course of established disease. From a pathophysiologic perspective, lipid derangements may begin long before overt diabetes is diagnosed, as progressive insulin resistance alters hepatic lipid metabolism, increases very-low-density lipoprotein production, reduces lipoprotein lipase activity, and disrupts reverse cholesterol transport (4,6). Conversely, individuals with known diabetes are more likely to have received medical treatment, dietary counseling, and lifestyle modification, all of which may partially attenuate lipid abnormalities over time (7). This creates a clinically important contrast between newly diagnosed patients, who may present with untreated and unrecognized metabolic risk, and previously diagnosed patients, in whom the lipid profile may be influenced by ongoing care. Understanding this distinction is essential because the timing of lipid assessment may alter opportunities for early cardiovascular risk reduction.

Although prior literature has examined dyslipidemia in diabetic populations, available evidence remains limited in several respects. Some studies have described dyslipidemia patterns among diabetic patients in general, while others have focused specifically on newly diagnosed cases, but relatively few have prospectively compared newly diagnosed and established type 2 diabetic patients within the same clinical framework (3,5,6). In Pakistan, this evidence gap is even more pronounced in military or tertiary-care institutional settings, where patient characteristics, treatment access, and referral patterns may differ from those in community cohorts or civilian hospitals (8). In addition, much of the existing literature has emphasized prevalence alone rather than directly contrasting the severity and distribution of individual lipid abnormalities across different stages of diabetic status. This leaves an important knowledge gap regarding whether early diabetes detection should automatically trigger aggressive lipid screening and management based on a demonstrably higher burden of dyslipidemia at diagnosis.

Given the well-established relationship between diabetic dyslipidemia and future cardiovascular disease, clarifying this comparison has immediate clinical relevance. If newly diagnosed patients exhibit a significantly more adverse lipid profile than patients with established disease, this would strengthen the rationale for comprehensive lipid assessment at the time of diagnosis and support earlier initiation of preventive strategies. It would also help clinicians prioritize specific lipid abnormalities, such as low high-density lipoprotein cholesterol or elevated triglycerides, that may be especially frequent at presentation. Therefore, the present study was designed to compare the pattern and frequency of dyslipidemia between newly diagnosed and known cases of type 2 diabetes mellitus in a tertiary-care military hospital setting. The study specifically evaluated total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very-low-density lipoprotein levels in both groups, with the objective of determining whether newly diagnosed patients carry a greater burden of lipid abnormality than those with established disease. We hypothesized that newly diagnosed patients with type 2 diabetes would demonstrate significantly more adverse lipid profiles and a higher frequency of individual dyslipidemic abnormalities than patients with known disease receiving ongoing care (1-8).

MATERIALS AND METHODS

This prospective comparative study was conducted in the medical unit of Pak Emirates Military Hospital, Rawalpindi, over a six-month period from April 2023 to September 2023. The study was designed to compare the pattern and magnitude of dyslipidemia between adults with newly diagnosed type 2 diabetes mellitus and those with established type 2 diabetes, using standardized biochemical assessment and prespecified diagnostic thresholds. A comparative observational design was selected because it allowed direct evaluation of naturally occurring differences in lipid profiles across two clinically relevant diabetic populations without altering routine management pathways. Ethical approval was obtained from the Institutional Ethical Review Committee before commencement of the study under reference No. A/28/ERC/10/4/23, dated 15 March 2023, and written informed consent was obtained from all participants prior to enrollment (9).

The sample size was estimated using the WHO formula for single-proportion estimation, $n = Z^2pq/d^2$, based on a previously reported dyslipidemia prevalence of 83.5% among Pakistani patients with type 2 diabetes (3,10). Using a 95% confidence level and a precision margin of 7%, the minimum required sample was calculated and then inflated to account for possible nonresponse or incomplete data, resulting in a final target sample of 120 participants. These were divided equally into two groups of 60 participants each. Consecutive sampling was used to recruit eligible patients presenting during the study period in order to reduce arbitrary selection and ensure inclusion of all accessible cases meeting the predefined criteria within the recruitment window.

Adults aged 30 to 75 years who fulfilled the diagnostic criteria for type 2 diabetes mellitus were considered eligible for inclusion. Participants in the newly diagnosed group were required to have received their diagnosis within the previous three months and to have no prior exposure to lipid-lowering medication, as this was necessary to preserve an untreated baseline lipid profile. Participants in the established diabetes group were required to have a documented diagnosis of type 2 diabetes for at least 12 months prior to enrollment. Patients with type 1 diabetes, gestational diabetes, chronic liver disease, chronic kidney disease, untreated thyroid dysfunction, pregnancy, or current steroid use were excluded because each of these conditions could independently alter lipid metabolism and confound the interpretation of diabetes-associated dyslipidemia.

After enrollment, all participants underwent a structured clinical evaluation performed using a standardized data collection form. Demographic and clinical variables recorded included age, sex, body mass index, blood pressure status, smoking status, family history of diabetes in first-degree relatives, duration of diabetes where applicable, fasting blood glucose, glycated hemoglobin, and complete fasting lipid profile. Body mass index was calculated as weight in kilograms divided by the square of height in meters. Hypertension and smoking status were recorded from clinical history and existing medical documentation at the time of assessment. To minimize information bias, the same predefined operational criteria were used for all participants, and biochemical measurements were obtained through the hospital's central laboratory following a uniform sample collection protocol.

For biochemical analysis, venous blood samples were collected after a 10- to 12-hour overnight fast. The fasting state was chosen to improve the reliability and comparability of triglyceride and related lipid measurements across both study groups. Serum samples were analyzed for fasting blood sugar, glycated hemoglobin, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very-low-density lipoprotein cholesterol using routine hospital laboratory procedures. Dyslipidemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III criteria. Total cholesterol of 200 mg/dL or higher was classified as elevated, triglycerides above 150 mg/dL were classified as high, high-density lipoprotein cholesterol below 40 mg/dL in men or below 50 mg/dL in women was considered low, and low-density lipoprotein cholesterol of 100 mg/dL or higher was considered raised. Participants meeting at least one of these criteria were categorized as having dyslipidemia (11). These threshold-based definitions ensured reproducibility and permitted clinically interpretable comparison of both overall dyslipidemia burden and the frequency of specific lipid abnormalities between the two groups.

Several steps were taken during design and analysis to reduce bias and improve internal validity. Restriction through eligibility criteria was used to exclude major secondary causes of dyslipidemia, such as thyroid disease, pregnancy, steroid exposure, and significant hepatic or renal impairment.

Standardized fasting blood collection and centralized laboratory processing were used to reduce measurement variability. Because age, body mass index, hypertension, smoking, and disease duration may influence lipid parameters, these variables were measured systematically and examined during statistical analysis as potential confounders. Although the study primarily aimed at group-level comparison, the analytic framework also emphasized careful interpretation of baseline clinical differences that could partly explain observed lipid variations. This strengthened the inferential value of the comparison, particularly in relation to differences between newly diagnosed untreated patients and those with longer disease duration who may have benefited from prior counseling or pharmacologic management.

All data were entered and analyzed using SPSS version 23. Continuous variables were summarized as mean with standard deviation, while categorical variables were presented as frequencies and percentages. Distributional assumptions for continuous variables were assessed before parametric testing, and between-group comparisons for normally distributed continuous data were performed using the independent-samples t-test. Categorical variables, including the frequency of individual lipid abnormalities and the overall presence of dyslipidemia, were compared using the chi-square test. A p-value of less than 0.05 was considered statistically significant. The primary analytical objective was comparison of lipid profile parameters and dyslipidemia frequency between newly diagnosed and established diabetes groups. Secondary evaluation included comparison of baseline clinical and biochemical characteristics relevant to cardiometabolic risk. Data were reviewed for completeness before analysis, and only records containing the variables required for the prespecified comparisons were included in the final dataset to preserve analytic consistency. All study procedures, variable definitions, and statistical thresholds were prespecified before final analysis to support reproducibility and data integrity (3,9-11).

RESULTS

A total of 120 participants were included, with 60 patients in the newly diagnosed type 2 diabetes mellitus group and 60 in the established diabetes group. The newly diagnosed group was significantly younger than the established group, with a mean age of 47.73 ± 9.60 years versus 53.25 ± 9.32 years, yielding a mean difference of -5.52 years (95% CI: -8.94 to -2.10; Cohen's d = -0.58; p=0.002). Body mass index was comparable between groups, with a negligible mean difference of 0.26 kg/m² (95% CI: -1.67 to 2.19; d=0.05; p=0.790). The sex distribution, hypertension frequency, smoking status, and family history of diabetes did not differ significantly, with all odds ratios crossing unity, indicating reasonable baseline comparability apart from age. The established group had a mean diabetes duration of 7.17 ± 3.67 years.

Table 1. Baseline Demographic and Clinical Characteristics with Comparative Inference

Variable	Newly Diagnosed (n=60)	Established T2DM (n=60)	Effect Estimate	95% CI	p-value
Age (years)	47.73 ± 9.60	53.25 ± 9.32	Mean difference = -5.52; d = -0.58	-8.94 to -2.10	0.002
BMI (kg/m²)	28.24 ± 4.83	27.98 ± 5.81	Mean difference = 0.26; d = 0.05	-1.67 to 2.19	0.790
Male sex, n (%)	36 (60.0)	30 (50.0)	OR = 1.50	0.73 to 3.09	0.271
Female sex, n (%)	24 (40.0)	30 (50.0)	—	—	—
Hypertension, n (%)	20 (33.3)	22 (36.7)	OR = 0.86	0.41 to 1.83	0.702
Smoking, n (%)	15 (25.0)	11 (18.3)	OR = 1.48	0.62 to 3.57	0.375
Family history of diabetes, n (%)	34 (56.7)	38 (63.3)	OR = 0.76	0.36 to 1.57	0.456
Duration of diabetes (years)	N/A	7.17 ± 3.67	—	—	—

The biochemical comparison showed a consistently more adverse metabolic profile in the newly diagnosed group. Fasting blood sugar was higher by 27.82 mg/dL (95% CI: 10.31 to 45.33; d=0.58; p=0.002). Although HbA1c was also higher in newly diagnosed patients, the between-group difference of 0.49% did not reach statistical significance (95% CI: -0.03 to 1.01; d=0.34; p=0.064). Lipid abnormalities were more pronounced in newly diagnosed patients across nearly all continuous lipid measures. Total cholesterol was higher by 23.84 mg/dL (95% CI: 8.87 to 38.81; d=0.60; p=0.001), triglycerides by 35.62 mg/dL (95% CI: 12.88 to 58.36; d=0.57; p=0.002), LDL-C by 25.35 mg/dL (95% CI: 11.98 to 38.72; d=0.74; p<0.001), and VLDL by 7.12 mg/dL (95% CI: 2.62 to 11.62; d=0.57; p=0.002). In contrast, HDL-C was markedly lower in newly diagnosed patients by 7.84 mg/dL (95% CI: -10.86 to -4.82; d=-0.95; p<0.001), representing the largest standardized effect among the lipid markers.

Table 2. Biochemical Comparison Between Newly Diagnosed and Established Type 2 Diabetes

Parameter	Newly Diagnosed (n=60)	Established T2DM (n=60)	Mean Difference	95% CI	Cohen's d	p-value
FBS (mg/dL)	184.84 ± 52.73	157.02 ± 42.35	27.82	10.31 to 45.33	0.58	0.002
HbA1c (%)	8.45 ± 1.53	7.96 ± 1.31	0.49	-0.03 to 1.01	0.34	0.064
Total Cholesterol (mg/dL)	222.34 ± 42.98	198.50 ± 36.29	23.84	8.87 to 38.81	0.60	0.001
Triglycerides (mg/dL)	216.26 ± 70.70	180.64 ± 53.07	35.62	12.88 to 58.36	0.57	0.002
HDL-C (mg/dL)	35.60 ± 6.78	43.44 ± 9.61	-7.84	-10.86 to -4.82	-0.95	<0.001
LDL-C (mg/dL)	144.37 ± 36.93	119.02 ± 31.05	25.35	11.98 to 38.72	0.74	<0.001
VLDL (mg/dL)	43.25 ± 14.14	36.13 ± 10.62	7.12	2.62 to 11.62	0.57	0.002
BMI (kg/m²)	28.24 ± 4.83	27.98 ± 5.81	0.26	-1.67 to 2.19	0.05	0.790

The categorical pattern of dyslipidemia further reinforced this gradient. Every patient in the newly diagnosed group met at least one criterion for dyslipidemia compared with 96.7% in the established group, although this absolute difference of 3.3 percentage points was not statistically significant (p=0.496). Raised total cholesterol was present in 65.0% of newly diagnosed patients versus 46.7% of established diabetics, corresponding to an odds ratio of 2.12 (95% CI: 1.02 to 4.42; p=0.040). Raised triglycerides were also more frequent in the newly diagnosed group at 85.0% versus 71.7%, with an odds ratio of 2.24 (95% CI: 0.91 to 5.53; p=0.078). The most pronounced separation was observed for depressed HDL-C, which affected 90.0% of newly diagnosed patients compared with 55.0% of established diabetics, producing a 35.0 percentage-point excess and an odds ratio of 7.36 (95% CI: 2.75 to 19.72; p<0.001). Raised LDL-C remained highly prevalent in both groups but still occurred more often among newly diagnosed patients at 90.0% versus 78.3%, with an odds ratio of 2.49 (95% CI: 0.88 to 7.07; p=0.079).

Table 3. Frequency of Individual Lipid Abnormalities with Association Estimates

Lipid Abnormality	Newly Diagnosed n (%)	Established T2DM n (%)	Absolute Difference (pp)	Odds Ratio	95% CI	p-value
Raised TC (≥200 mg/dL)	39 (65.0)	28 (46.7)	18.3	2.12	1.02 to 4.42	0.040
Raised TG (≥150 mg/dL)	51 (85.0)	43 (71.7)	13.3	2.24	0.91 to 5.53	0.078
Depressed HDL-C (M<40, F<50 mg/dL)	54 (90.0)	33 (55.0)	35.0	7.36	2.75 to 19.72	<0.001
Raised LDL-C (≥100 mg/dL)	54 (90.0)	47 (78.3)	11.7	2.49	0.88 to 7.07	0.079
Any Dyslipidemia	60 (100.0)	58 (96.7)	3.3	5.17*	0.24 to 110.01*	0.496

*Continuity correction applied because one cell contained zero.

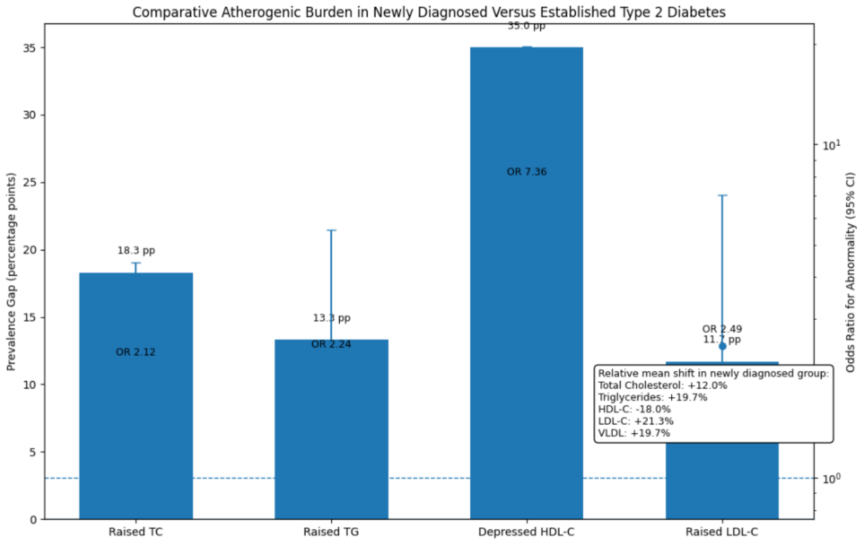

Figure 1 Comparative atherogenic burden is available here: Download Figure

The figure demonstrates that the newly diagnosed group carried a consistently heavier atherogenic burden than the established group, with the largest prevalence gap observed for depressed HDL-C at 35.0 percentage points and the strongest association estimate also centered on low HDL-C (OR 7.36, 95% CI 2.75–19.72). Raised total cholesterol and triglycerides showed smaller but still clinically relevant prevalence excesses of 18.3 and 13.3 percentage points, respectively, while raised LDL-C showed an 11.7 percentage-point difference. The embedded relative shifts further indicate that newly diagnosed patients had approximately 12.0% higher total cholesterol, 19.7% higher triglycerides, 21.3% higher LDL-C, and 19.7% higher VLDL, whereas HDL-C was 18.0% lower, highlighting that the most distinctive early dyslipidemia signature was not only generalized lipid elevation but a disproportionately severe HDL-C depletion.

DISCUSSION

The present study demonstrates that dyslipidemia was highly prevalent in both newly diagnosed and established type 2 diabetes mellitus, but the burden was consistently greater among newly diagnosed patients. This difference was evident not only in the proportion of patients with abnormal lipid parameters but also in the magnitude of derangement across the lipid profile. Newly diagnosed patients had significantly higher total cholesterol, triglycerides, LDL-C, and VLDL, alongside markedly lower HDL-C, indicating a more adverse and potentially more atherogenic metabolic phenotype at the time of presentation. The strongest distinction between the two groups was observed for HDL-C, where the mean difference was 7.84 mg/dL and the frequency of depressed HDL-C was 35.0 percentage points higher in the newly diagnosed group. This pattern suggests that the earliest clinically detected phase of type 2 diabetes may coincide with a particularly unfavorable cardiometabolic state, before the modifying influence of treatment, counseling, or risk-factor monitoring becomes established.

The near-universal prevalence of dyslipidemia in the study population is consistent with the broader literature showing that lipid abnormalities are among the most frequent metabolic comorbidities in type 2 diabetes. Earlier Pakistani work has documented dyslipidemia rates exceeding 80% in diabetic cohorts, while regional and international studies have similarly reported a high burden of abnormal triglycerides, reduced HDL-C, and raised LDL-C in both newly diagnosed and long-standing disease (12,13). The prevalence observed in the present study was higher than in several previous reports, which may be explained by differences in study setting, cardiovascular risk profile of the sampled population, and the lipid thresholds used to define abnormality. In particular, the use of LDL-C ≥100 mg/dL as a threshold aligns more closely with contemporary cardiovascular risk management in diabetes and increases sensitivity for identifying clinically relevant dyslipidemia in a high-risk population (14). This may partly account for the finding that 100% of newly diagnosed patients and 96.7% of established diabetics met at least one criterion for dyslipidemia.

The observation that newly diagnosed patients had worse lipid parameters than established diabetics is biologically plausible and clinically important. Type 2 diabetes is preceded by a prolonged period of insulin resistance and subclinical metabolic dysfunction during which hepatic overproduction of triglyceride-rich lipoproteins, impaired lipoprotein lipase activity, increased free fatty acid flux, and altered cholesterol transport progressively disturb lipid homeostasis. By the time diabetes is first diagnosed, many patients may therefore already have accumulated a substantial untreated atherogenic burden. In contrast, patients with established disease are more likely to have received medical advice, glycemic monitoring, statin therapy, dietary counseling, or other interventions that partly improve lipid values even when diabetes persists. Previous work from Iraq and China has also suggested that untreated or newly identified diabetic patients often exhibit more pronounced lipid disruption and lower treatment coverage than those already engaged in care pathways (15,16). The present findings support that interpretation and indicate that diagnosis may occur at a stage when cardiovascular risk is already materially elevated.

Among all lipid abnormalities, depressed HDL-C emerged as the most distinctive feature of newly diagnosed disease. This finding deserves particular emphasis because HDL particles play an important anti-atherogenic role through reverse cholesterol transport, antioxidant activity, anti-inflammatory effects, and endothelial protection. A 35.0 percentage-point excess in low HDL-C prevalence, together with an odds ratio of 7.36, indicates that low HDL-C may be the most clinically informative marker separating newly diagnosed diabetic patients in this setting. Similar observations have been reported in South Asian and African diabetic populations, where low HDL-C frequently appears as one of the dominant lipid abnormalities at presentation (13,17). This pattern may reflect the combined effects of insulin resistance, central adiposity, and disturbed triglyceride metabolism, all of which promote HDL depletion and qualitative HDL dysfunction. In practical terms, these data suggest that clinicians should not restrict attention to LDL-C alone when assessing newly diagnosed diabetic patients, as low HDL-C and hypertriglyceridemia may identify an especially adverse early phenotype.

Raised triglycerides and LDL-C were also notably frequent in both groups, but the absolute and relative burden remained greater among newly diagnosed patients. Triglycerides were elevated in 85.0% of newly diagnosed patients compared with 71.7% of established diabetics, while raised LDL-C was present in 90.0% and 78.3%, respectively. Although the categorical comparisons for triglycerides and LDL-C did not achieve conventional statistical significance, the corresponding continuous analyses showed meaningful between-group differences, with mean triglycerides higher by 35.62 mg/dL and LDL-C higher by 25.35 mg/dL in newly diagnosed patients. These findings suggest that the absence of significance in some categorical contrasts may be related more to threshold-based dichotomization and sample size constraints than to a true lack of clinical difference. Importantly, the effect size for LDL-C remained moderate to large, reinforcing its relevance as part of the early diabetic dyslipidemia spectrum. Similar relationships between poor metabolic control and worsening lipid burden have been described in observational diabetic cohorts from the Middle East and South Asia, where glycemic disruption, longer exposure to insulin resistance, and delayed treatment have each been associated with more severe lipid abnormalities (18,19).

Another clinically relevant observation was that the newly diagnosed group was significantly younger than the established diabetes group. This indicates that substantial dyslipidemia may already be present relatively early in the disease course and at a younger age, increasing the potential lifetime exposure to atherogenic risk. In populations such as Pakistan, where diabetes often develops earlier than in many Western settings, this has important implications for prevention strategies. A younger patient presenting with newly diagnosed diabetes and marked dyslipidemia may face decades of cumulative vascular risk unless risk-factor modification is initiated promptly. The coexistence of higher fasting blood glucose and worse lipid values in the newly diagnosed group further supports the concept of a clustered metabolic disturbance at first presentation rather than isolated hyperglycemia alone. Even though HbA1c did not differ significantly between groups, its numerically higher value in newly diagnosed patients is directionally consistent with this pattern and may have become statistically clearer in a larger sample.

From a clinical and public health perspective, these findings support the routine inclusion of a complete fasting lipid profile at the time of diagnosis of type 2 diabetes mellitus. Waiting for follow-up evaluation may delay recognition of a major modifiable cardiovascular risk factor during a period when the lipid burden appears greatest. Early identification of dyslipidemia may allow prompt initiation of lifestyle intervention, statin therapy where indicated, and closer cardiometabolic surveillance, thereby reducing the long-term probability of coronary, cerebrovascular, and peripheral vascular complications. The results also support a more integrated initial diabetic assessment in which glucose control and lipid risk are evaluated together rather than sequentially (20).

This study has several strengths, including its prospective comparative design, equal group allocation, use of standardized fasting biochemical assessment, and clinically interpretable comparison of both continuous lipid measures and categorical abnormalities. At the same time, certain limitations must be acknowledged. The sample was modest and drawn from a single tertiary-care military hospital, which may limit generalizability to broader civilian, rural, or primary-care populations. Consecutive sampling may also have introduced selection bias. In addition, age differed significantly between groups, and although major secondary causes of dyslipidemia were restricted through exclusion criteria, residual confounding from treatment status, dietary patterns, or unmeasured metabolic factors cannot be excluded. The study was cross-sectional in its comparative assessment and therefore cannot determine causality or the longitudinal evolution of lipid parameters after diagnosis. Larger multicenter studies using multivariable adjustment and follow-up-based designs would help clarify whether the greater lipid burden in newly diagnosed diabetes persists after accounting for treatment exposure and other confounders. Despite these limitations, the present data provide clinically coherent evidence that newly diagnosed type 2 diabetes is associated with a distinctly worse dyslipidemia profile at presentation and that early lipid evaluation should be considered an essential component of initial diabetes care (12-19).

CONCLUSION

Patients with newly diagnosed type 2 diabetes mellitus exhibited a significantly more adverse lipid profile than those with established disease, characterized by higher total cholesterol, triglycerides, LDL-C, and VLDL together with markedly lower HDL-C. Depressed HDL-C emerged as the most prominent distinguishing abnormality, while the overall prevalence of dyslipidemia remained extremely high in both groups. These findings indicate that substantial atherogenic risk is already present at the time of diabetes diagnosis and support routine comprehensive lipid assessment as part of the initial evaluation of all newly diagnosed patients so that timely lifestyle and pharmacologic interventions can be initiated without delay.

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