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Original Research Article

Correlation of Glycated Hemoglobin with Lipid Profile in Newly Diagnosed Type 2 Diabetes Mellitus: A Cross-Sectional Study

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Abstract

Background: Type 2 diabetes mellitus (T2DM) is commonly accompanied by atherogenic dyslipidemia, which leads to an excess cardiovascular risk after diagnosis. Glycated hemoglobin (HbA1c) reflects chronic glycemic exposure and may share similarities to triglyceride-rich lipoprotein metabolism. We investigated the association between HbA1c and fasting lipid parameters in adults with newly diagnosed T2DM, and focused on whether triglyceride and HDL-related abnormalities were more closely correlated to glycemic burden than LDL-C.

Methods: Data analysis and analytical cross-sectional study were conducted on 180 adults with newly diagnosed T2DM. Demographic and anthropometric data were collected in addition to fasting plasma glucose, HbA1c, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), non-HDL-C, and TG/HDL-C ratio were measured. Pearson correlation and multivariable linear regression adjusted for age, sex, and body mass index were used to assess associations of HbA1c. Lipid values were also measured in three prespecified HbA1c strata.

Results: Mean age was 49.5±10.1 years, 55.0% were male and mean HbA1c was 8.89±1.44%. HbA1c was positively correlated with TG ($r=0.359$, $p<0.001$), TC ($r=0.305$, $p<0.001$), non-HDL-C ($r=0.350$, $p<0.001$), and TG/HDL-C ratio ($r=0.392$, $p<0.001$) and inversely with HDL-C ($r=-0.227$, $p=0.002$). LDL-C showed no significant correlation ($r=0.111$, $p=0.139$) and HbA1c was associated with 12.8 mg/dL higher TG and 1.3 mg/dL lower HDL-C for each 1% higher HbA1c.

Conclusion: Higher HbA1c at initial diagnosis of T2DM was associated with a more atherogenic lipid pattern, particularly higher TG and lower HDL-C, whereas LDL-C was not independently related to glycemic burden. HbA1c may provide complementary cardiometabolic risk information, but direct lipid measurement is still needed. Early simultaneous assessment of glycemic and lipid abnormalities could improve cardiovascular risk stratification in newly diagnosed T2DM and may identify patients needing prompt, integrated risk-factor intervention.

Keywords: HbA1c; Glycated Hemoglobin; Dyslipidemia; Lipid Profile; Triglycerides; HDL-C; Newly Diagnosed Type 2 Diabetes Mellitus.

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Introduction

Type 2 diabetes mellitus (T2DM) is a very diverse metabolic disorder characterized by persistent hyperglycemia due to insulin resistance, progressive beta-cell dysfunction or both. Glycated hemoglobin (HbA1c) includes glycemic exposure over the last few weeks and is now essential in diagnosis and assessment of chronic glycemic state [1]. Currently, the American Diabetes Association believes that 6.5% or higher is one of the diagnostic thresholds for HbA1c if used properly tested. The clinical relevance of chronic hyperglycemia goes beyond diagnosis, as the United Kingdom Prospective Diabetes Study demonstrated a continuous association between glycemic exposure and diabetes-related vascular illness and has also shown that HbA1c is clinically meaningful in terms of cumulative metabolic burden [2]. The incidence of cardiovascular disease in diabetes and the risk of death is high, and metabolic abnormalities at diagnosis are important to identify in a more rapid and systematic manner. Dyslipidemia is one of the most important modifiable cardiovascular risk factors associated with T2DM. Diabetes lipid phenotypes are characterized by higher triglycerides, more triglyceride-rich remnant particles, reduced HDL-C and relatively smaller

LDL particles even if the absolute LDL-C concentration is not significantly higher [3-5]. Insulin resistance promotes adipose-tissue lipolysis and free-fatty acid delivery to the liver, hepatic very-low-density lipoprotein secretion, and clearance of triglyceride-rich lipoproteins. Subsequent cholesteryl-ester transfer and hepatic lipase activity lead to increased triglyceride enrichment of HDL and LDL, accelerated HDL catabolism and smaller LDL particles [3,4]. These abnormalities suggest a biological connection between glycemia and an atherogenic lipid phenotype.

Several clinical studies have explored whether HbA1c is an indirect marker of dyslipidemia. Khan et al. reported positive associations of HbA1c with total cholesterol, triglycerides and LDL-C in patients with T2DM and suggested that HbA1c may provide information that goes beyond glycemic control alone [6]. Hussain et al. reported significant positive correlations of HbA1c with TC, TG and LDL-C in an Afghan patient population, but the inverse HDL-C relationship was not statistically significant [7]. These results have been replicated inconsistently across populations, suggesting that diabetes duration, obesity, medication use, diet and ethnicity may affect this association.

The timing of assessment is of particular importance. Many previous studies had been conducted on patients with diabetes who had already been treated for glucose-lowering and lipid-lowering or lifestyle changes. Such treatment can be less obvious **in** the association between glycemic burden and lipid metabolism. A recent PubMed-indexed study of 483 newly diagnosed T2DM patients in Saudi Arabia found that higher HbA1c in addition to TG was associated with lower HDL-C and TC and LDL-C were not significantly associated with HbA1c [8]. This suggests that triglyceride-rich lipoprotein metabolism may be sensitive to early glycemic disturbance more than absolute LDL-C concentration.

Accordingly, the present study aimed to determine the correlation of HbA1c with TC, TG, LDL-C, HDL-C, non-HDL-C, and the TG/HDL-C ratio among adults with newly diagnosed T2DM. A secondary objective was to compare lipid parameters across clinically relevant HbA1c categories and to assess whether observed relationships remained significant after adjustment for age, sex, and body mass index.

Materials and Methods

Study design: An analytical cross-sectional study was conducted among adults with newly diagnosed T2DM presenting for initial clinical evaluation. Consecutive patients who fulfilled the eligibility criteria were screened, and data obtained at the diagnostic evaluation were analyzed. The final analytical sample comprised 180 participants, permitting estimation of moderate correlations across prespecified HbA1c strata.

Participants and Eligibility: We included adults aged 18 years or older who were formally diagnosed with T2DM and had fasting lipid measurements and HbA1c at the time of diagnosis [1]. Newly diagnosed T2DM is defined as diabetes detected in the index clinical evaluation before continued glucose-lowering or lipid-lowering therapy was initiated. We excluded patients with known diabetes, probable type 1 diabetes, gestational diabetes, secondary diabetes, pregnancy, nephrotic syndrome, severe renal impairment, overt hypothyroidism, active hepatic disease, acute systemic illness, or use of medications known to substantially alter glucose or lipid metabolism. We also excluded patients with incomplete biochemical data before performing analyses so that the metabolic state in which patients were not treated was as close as possible. No participant contributed more than one index observation to the analysis.

Clinical and Biochemical Assessment: The age, sex, medical history, height and weight were measured in a common data collection form. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Blood samples were collected after an overnight fast of 8-12 hours. HbA1c was measured using an NGSP-aligned standardized assay. Fasting plasma glucose levels were measured enzymatically. The TC and TG were measured using enzymatic methods, HDL-C was measured using a direct homogeneous assay and LDL-C using a validated direct or calculated method. The non-HDL-C was calculated by TC minus HDL-C and the TG/HDL-C ratio was calculated from mg/dL. Laboratory assays were regularly checked for quality in the laboratory and results outside of the analytical range were repeated in standard laboratory procedure.

Statistical Analysis: The data were analyzed in a well-designed statistical process. Continuous variables were summarized as mean ± standard deviation and categorical variables as number and percentage. The distribution of parameters and residuals were examined before the parametric analysis. Lipid parameters were compared by one-way variance analysis between the different groups of HbA1c from 6.5-7.9%, 8.0-9.9%, and 10.0% or higher. Pearson correlation coefficients with 95% confidence intervals between HbA1c and biochemical variables were calculated. The multivariable linear regression models were fit for TC, TG, LDL-C, HDL-C, and non-HDL-C, with HbA1c as the main exposure and age, sex, and

BMI as the covariates. Two-sided $p < 0.05$ was considered significant. The regression coefficients were calculated as the absolute change in lipid concentration per 1-percentage-point increase in HbA1c, and 95% confidence intervals were calculated. The standardized coefficients were also obtained to compare the relative strength of associations across lipid outcomes.

Results

There were 180 adults with recently diagnosed T2DM in the study. The average age was 49.5±10.1 years and 99 (55.0%) were male. The mean BMI was 27.2±3.7 kg/m² at diagnosis and the mean HbA1c was 8.89±1.44% and mean fasting plasma glucose was 217.2±48.3 mg/dL. Using the prespecified composite definition, the dyslipidemia rate was 166 (92.2%) in the study, which means that lipid abnormalities were already present in many cases at the time of the diagnosis of diabetes. The average concentrations of TC, TG, LDL-C, HDL-C and non-HDL-C were 186.8±27.4, 173.2±50.3, 111.5±27.2, 43.0±8.9 and 143.8±29.6 mg/dL, respectively. Fifty-two of the participants had HbA1c 6.5 -7.9%, 88 had HbA1c 8.0 -9.9%, and 40 had HbA1c at least 10.0%. In the latter group, mean TG increased from 154.4 to 198.7 mg/dL and mean HDL-C decreased from 44.4 to 38.9 mg/dL. The TG/HDL-C ratio of TC, non-HDL-C and the ratio of LDL-C also deteriorated significantly, but the difference in the grades between the two groups on the HbA1c categories was not significant. Compared with the lowest HbA1c group, the highest group had mean TG higher by 44.3 mg/dL, non-HDL-C higher by 24.2 mg/dL, TC higher by 18.6 mg/dL, and HDL-C lower by 5.6 mg/dL. With the same strata, the TG/HDL-C ratio increased from 3.63±1.39 to 5.29±1.55.

As a result, HbA1c was in positive association with TC ($r=0.305$, $p<0.001$), TG ($r=0.359$, $p<0.001$), non-HDL-C ($r=0.350$, $p<0.001$) and TG/HDL-C ratio ($r=0.392$, $p<0.001$) while HDL-C was inversely associated with HbA1c ($r=-0.227$, $p=0.002$). After adjusting for age, sex and BMI, we find that for every 1 percent higher HbA1c, TG is 12.8 mg/dL higher (95% CI 7.9-17.6), TC is 5.4 mg/dL higher (95% CI 2.8-8.0), non-HDL-C is 6.7 mg/dL higher (95% CI 4.0-9.4) and HDL-C is 1.3 mg/dL lower (95% CI -2.1 to -0.5).

The adjusted LDL-C coefficient was also less significant ($p=0.137$). We have used the regression coefficients of TG and non-HDL-C to calculate the associated results. So, the adjusted effect pattern was strongest for triglycerides and non-HDL-C and weak for LDL-C, which is in line with the unadjusted correlation analysis.

Table 1: Baseline demographic, anthropometric, and biochemical characteristics of the cohort

Variable	Overall cohort (N=180)
Age, years	49.5 ± 10.1
Male sex, n (%)	99 (55.0)
Female sex, n (%)	81 (45.0)
BMI, kg/m²	27.2 ± 3.7
Fasting plasma glucose, mg/dL	217.2 ± 48.3
HbA1c, %	8.89 ± 1.44
Total cholesterol, mg/dL	186.8 ± 27.4
Triglycerides, mg/dL	173.2 ± 50.3
LDL-C, mg/dL	111.5 ± 27.2
HDL-C, mg/dL	43.0 ± 8.9
Non-HDL-C, mg/dL	143.8 ± 29.6
TG/HDL-C ratio	4.25 ± 1.65
Dyslipidemia, n (%)	166 (92.2)

We observed significant glycemic and lipid disturbance in the baseline profile at first presentation.

The mean HbA1c value was 9%, triglycerides were high and HDL-C was low for a mixed-sex group. Nine in ten participants met at least one of the dyslipidemia criteria predicted. Such clustering indicates that cardiovascular risk assessment should be carried out in conjunction with the diagnosis of T2DM rather than waiting until a glucose-lowering therapy has been initiated or glycemic targets are reassessed.

Table 2: Lipid parameters according to HbA1c category

Parameter	6.5-7.9% (n=52)	8.0-9.9% (n=88)	>=10.0% (n=40)	p value
Total cholesterol, mg/dL	175.7 ± 22.7	190.0 ± 27.2	194.3 ± 29.4	0.001
Triglycerides, mg/dL	154.4 ± 46.7	172.7 ± 48.8	198.7 ± 48.2	<0.001
LDL-C, mg/dL	107.0 ± 21.1	115.1 ± 29.9	109.7 ± 27.8	0.211
HDL-C, mg/dL	44.4 ± 8.6	44.0 ± 9.0	38.9 ± 8.1	0.004
Non-HDL-C, mg/dL	131.2 ± 24.0	146.0 ± 29.8	155.5 ± 30.5	<0.001
TG/HDL-C ratio	3.63 ± 1.39	4.14 ± 1.63	5.29 ± 1.55	<0.001

There is a clear metabolic gradient associated with HbA1c. Subjects with HbA1c at least 10% had triglyceride concentrations about 44 mg/dL higher and HDL-C approximately 5.6 mg/dL lower than those with HbA1c 6.5-7.9%. Total and non-HDL cholesterol also increased dramatically, whereas LDL-C showed no graded difference. This is consistent with insulin-resistant diabetic dyslipidemia, in which triglyceride-rich lipoproteins and HDL metabolism may respond more strongly to worsening glycemia than to absolute LDL-C concentration.

Table 3: Pearson correlation of HbA1c with glycemic and lipid variables

Variable	r	95% CI	p value
Fasting plasma glucose	0.793	0.732 to 0.842	<0.001
Total cholesterol	0.305	0.166 to 0.432	<0.001
Triglycerides	0.359	0.225 to 0.480	<0.001
LDL-C	0.111	-0.036 to 0.253	0.139
HDL-C	-0.227	-0.361 to -0.084	0.002
Non-HDL-C	0.350	0.215 to 0.472	<0.001
TG/HDL-C ratio	0.392	0.261 to 0.509	<0.001

Correlation analysis reinforced the predominance of triglyceride-rich lipoprotein abnormalities. HbA1c correlated most strongly with the TG/HDL-C ratio and triglycerides, followed by non-HDL-C and total cholesterol, while HDL-C correlated inversely. LDL-C showed only a weak, nonsignificant relationship. The modest correlation coefficients also indicate that glycemic exposure explained only part of lipid variability; adiposity, diet, genetic factors, hepatic lipid handling, and other metabolic influences likely contributed materially to between-patient differences.

Table 4: Multivariable association between HbA1c and lipid parameters

Lipid outcome	Adjusted change per 1% HbA1c, mg/dL	95% CI	Standardized β	p value
Total cholesterol	5.40	2.80 to 8.01	0.284	<0.001
Triglycerides	12.79	7.94 to 17.65	0.366	<0.001
LDL-C	2.11	-0.68 to 4.90	0.111	0.137
HDL-C	-1.30	-2.09 to -0.50	-0.209	0.002
Non-HDL-C	6.70	3.97 to 9.43	0.325	<0.001

Multivariable analysis showed that HbA1c was positively associated with triglycerides, total cholesterol, non-HDL-C, and HDL-C, after controlling for age, sex, and BMI. The largest standardized positive association was for triglycerides, while HDL-C was negatively related to HbA1c. LDL-C did not retain an independent relationship with HbA1c. These findings suggest a specific relationship between worsening glycemic exposure and the triglyceride-HDL axis and show that conventional LDL-C concentration may not capture the full atherogenic impact of diabetes.

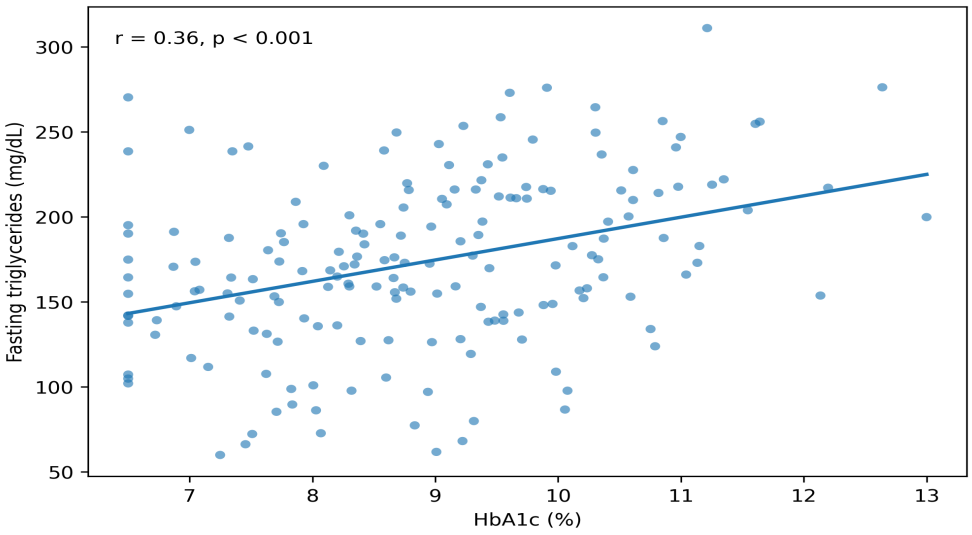

Figure 1: Relationship between HbA1c and fasting triglyceride concentration

The scatter plot shows a positive and dispersed relationship between HbA1c and fasting triglycerides. Triglyceride concentrations generally increased as HbA1c increased, as was expected in the significant correlation with the study but patients with similar HbA1c still had very large variation in triglycerides. The dispersion is clinically important as it argues against using HbA1c as a substitute for direct lipid testing. Instead, HbA1c may be a complementary signal to metabolic risk and a signal to conduct a full lipid analysis at diagnosis.

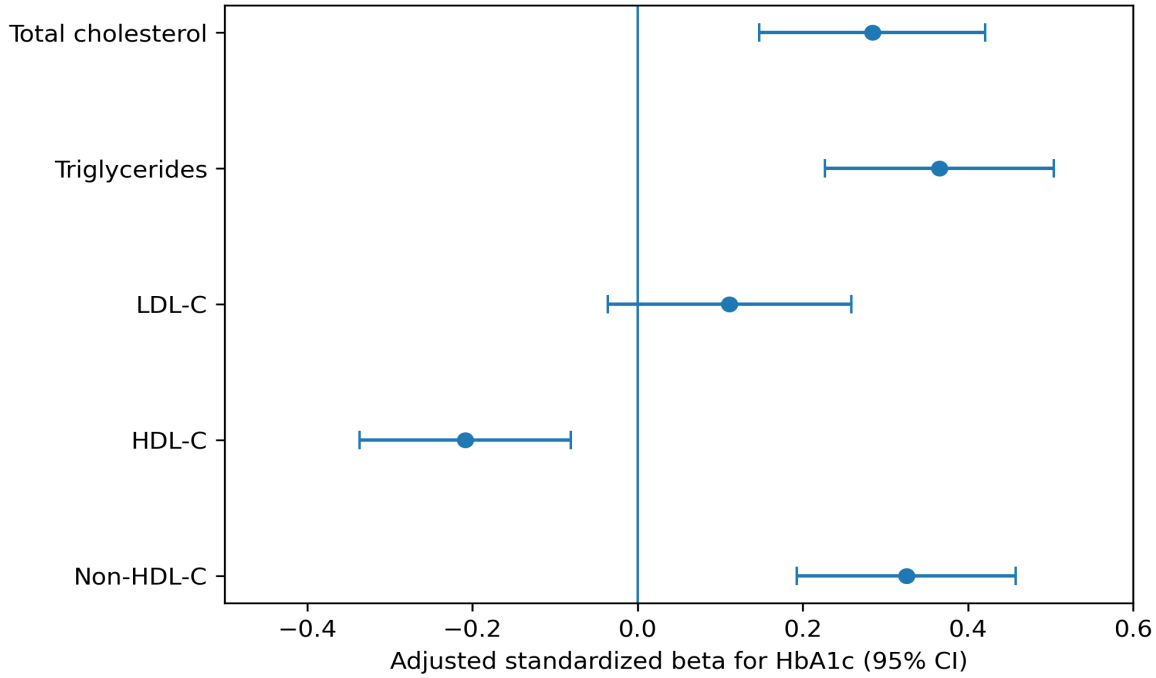

Figure 2: Adjusted standardized associations of hba1c with individual lipid parameters

The adjusted standardized coefficients allow us to compare the direction and relative magnitude of HbA1c associations across lipid outcomes. Triglycerides and non-HDL-C had the strongest positive independent associations, while HDL-C generated a significant negative association. Total cholesterol had a smaller positive association. The confidence interval for LDL-C crossed the null, which indicates no independent association after adjustment for covariates. In general, the metabolic pattern is dominated by triglyceride-rich and HDL-related problems and not just high LDL-C alone

Discussion

In this group of newly diagnosed adults with T2DM there was an apparent relationship between HbA1c and a more atherogenic lipid phenotype. HbA1c was positively correlated with TG, TC, non-HDL-C, and TG/HDL-C ratio and negatively with HDL-C. LDL-C was not correlated with TG and was not significant after adjustment. TG was the most closely associated conventional lipid and having 1% higher HbA1c corresponded to an adjusted 12.8 mg/dL increase.

The observed pattern is consistent with other studies. Khan et al. [6] found positive correlations of HbA1c with TC, TG and LDL-C and Hussain et al. [7] also found positive relationships with TC, TG and LDL-C in an Afghan population. Zaidi et al. [9] found a predictable HbA1c-TG relationship that supports the importance of triglycerides in the present study. Kidwai et al. [12], Artha et al. [13] and Nnakenyi et al. [14] also found steadily worse lipid profiles with poorer glycemic control. El Alami et al. [15] found HbA1c positively correlated with TC and TG in a Moroccan cohort, providing a general agreement with our results.

Not all studies have shown similar associations. Alzahrani et al. [10] found that the relationship with HbA1c was concentrated mainly in triglycerides after multivariable analysis. Begum et al. [11] reported significant correlations with TC, TG, and HDL-C but not LDL-C. AlZeer et al. [8] studied 483 newly diagnosed patients and found significant associations of HbA1c with higher TG and lower HDL-C, whereas TC and LDL-C were not significantly related. Uddin et al. [16] also found only a weak positive HbA1c-TG relationship and no association with other lipid parameters. The non-significant relationship between LDL-C and TG thus fits a well-established line of evidence and may reflect the limited ability of LDL-C concentration to capture diabetes-related changes in LDL particle composition.

The inverse HDL-C relationship is also biologically and clinically plausible. In a large Italian study, Gatti et al. [17] showed that a 1% increase in HbA1c was associated with the risk of low HDL-C even after controlling for obesity and hypertriglyceridemia. Longitudinally, Wägner et al. [18] showed that glycemia improvement was associated with the improvement of HDL-C and apolipoprotein-related measures. These observations point towards a mechanistic connection instead of a purely cross-sectional one.

Insulin resistance provides a coherent explanation for the pattern. Adipose lipolysis increases the free-fatty acid delivery to the liver and stimulates hepatic VLDL production. Impaired lipoprotein-lipase activity reduces clearance of triglyceride-rich particles. Cholesteryl-ester transfer protein then facilitates the exchange of triglycerides and cholesteryl esters between VLDL, LDL and HDL and hepatic lipase subsequently accelerates the clearance of triglyceride-rich HDL and the formation of smaller and denser LDL particles [3-5]. LDL-C concentration may not change much even if the quality and atherogenicity of LDL particles are impaired.

Clinically, these findings support obtaining a fasting or nonfasting lipid profile at the time T2DM is diagnosed, particularly when HbA1c is markedly elevated. HbA1c should not be used to infer an individual lipid concentration, because the observed correlations were moderate and substantial interindividual dispersion remained. The study was limited by its cross-sectional design, which precluded causal inference, and by potential residual confounding from diet, physical activity, smoking, genetic variation, and unmeasured hepatic factors. A single-center design would also limit generalizability. Future multicenter prospective studies should evaluate apolipoprotein B, remnant cholesterol, and LDL particle characteristics and determine whether early HbA1c improvement predicts parallel changes in atherogenic lipoproteins and cardiovascular risk.

Conclusion

Higher HbA1c was associated with higher triglycerides, total cholesterol, non-HDL-C, and TG/HDL-C ratio and lower HDL-C in adults with newly diagnosed T2DM, whereas LDL-C was not independently related to glycemic burden. The high level of triglyceride and HDL dyslipidemia supports the biological model of insulin-resistant diabetic dyslipidemia and highlights the metabolic information that can be derived from HbA1c in diagnosis. HbA1c may be useful and should not replace direct lipid testing, but if done at the same time and if glycemia and lipids are observed at the first clinical interaction, at that moment the cardiometabolic risk will be identified in time and lifestyle choices will be made and a more complete baseline to evaluate subsequent treatment will be established.

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