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Individual Lipids and Lipid Ratios in Type-2 Diabetic Patients: Association with Glycemic Control Status

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Abstract

The amount of glycosylated hemoglobin (HbA_{1c}) reflects the long-term glycemic control of patients with diabetes. HbA_{1c} also predicts the risk for the development of diabetic complications such as cardiovascular disease (CVD). Patients with type-2 diabetes and the characteristic of dyslipidemia are frequently found. Also, dyslipidemia plays as an independent risk factor for CVD. This study was aimed to evaluate the relationship between glycemic control status with serum individual lipid profiles and lipid ratios in patients with type-2 diabetes. This cross-sectional study consisted of 80 patients. Depending on the HbA_{1c} level, the patients were divided into two groups, good glycemic control group ($HbA_{1c} < 7.0\%$, $n = 15$) and poor glycemic control group ($HbA_{1c} \geq 7.0\%$, $n = 65$). The association of HbA_{1c} with individual lipids (TC, TG, HDL-C, LDL-C, Non-HDL-C) and lipid ratios (TC/HDL-C, TG/HDL-C, LDL-C/HDL-C, monocyte/HDL-C) were analyzed. The value of individual lipids and lipid ratios did not correlate with HbA_{1c} level (p-value ≥ 0.05). Parameters of individual lipids and lipid ratios were not independently associated with poor glycemic control, which was analyzed by logistic regression. ROC analysis found both LDL-C and LDL-C/HDL-C were not accurate to be used as a prognostic indicator of poor glycemic control in patients with type-2 diabetes ($p = 0.155$, $p = 0.297$, respectively). The present study found that there was no association between individual lipids and lipid ratios with glycemic control status.

Keywords: Type-2 diabetes; Glycosylated hemoglobin; Individual lipids; Lipid ratios.

1. INTRODUCTION

Diabetes mellitus (DM), a lifelong metabolic disease, remains a major global problem in medicine. It is characterized by a hyperglycemic condition, which is caused by either insulin deficiency or insulin resistance or both. The chronic hyperglycemic condition may lead to impairment at the molecular level and result in the development of complications, that is, diabetic nephropathy (DN) or cardiovascular disease (CVD) [1, 2].

Glycosylated hemoglobin (HbA_{1c}) served as an indicator for long-term glycemic control. It reflects as cumulative 2-3 months of blood glucose [3]. As the gold standard for glycemic control determined by Diabetes Complications and Control Trial, the HbA_{1c} value of $\leq 7\%$ served as a critical value to reduce vascular complications' risk [4]. Also, the increase in HbA_{1c} is considered an independent risk factor for coronary heart disease (CHD) in patients with diabetes [5].

Several studies pointed that patients with type-2 diabetes could have altered lipid parameters at various degrees. A study conducted by Giansanti *et al*. [6] in 1999 reported significant hypercholesterolemia and hyperlipidemia in diabetic patients with CVD than without CVD. Another study by Mahato *et al*. [7] also found an association between glycemic control and lipid parameters. Several lipid profiles in type-2 diabetic patients with $HbA_{1c} > 7$ were significantly higher compared with diabetic patients with $HbA_{1c} \leq 7$ [7]. It is an important data since it increases the rate of mortality in patients. In addition to the measurement of lipid parameters with individual lipid profile, current data pointed that lipid ratio is better compared to individual lipids to reflect morbidity and severity of coronary heart disease (CHD) [8, 9].

Data regarding the association between glycemic control, which is determined by HbA_{1c} levels, and individual lipid profiles or lipid ratios are scant in literature. This study aimed to determine the relationship between glycemic control with serum individual lipid profiles and lipid ratios in patients with type-2 diabetes.

2. METHODS

2.1. Study Design and Patient Selection

A total of 80 patients (43 males and 37 females) with type-2 diabetes were included in this study, which was carried out at Sanglah General Hospital, Denpasar, Bali, Indonesia. Mean age of this study was 54.45 ± 9.34 years. Subjects were classified into two groups according to their HbA_{1c} levels (good glycemic control or GGC: $HbA_{1c} < 7\%$, $n = 15$ and poor glycemic control or PGC: $HbA_{1c} \geq 7\%$, $n = 65$). Patients with conditions that affect the lipid profile, such as history or ongoing thyroid disease and obstructive liver disorders (obtained from anamnesis or medical record), were excluded from this study.

This study has been evaluated and approved by the local Ethics Committee of Udayana University/Sanglah General Hospital, Denpasar, Bali, Indonesia.

2.2. Data Collection

Variables of age, gender, height and weight measurement, systolic and diastolic blood pressure, duration of diseases, medication history, and disease history were obtained by anamnesis, physical examination, and medical reports analysis. Body mass index was measured by Quetelet index with the formula of weight/height^2.

2.3. Biochemical and Hematological Evaluation

Biochemical and hematologic parameters (i.e., serum creatinine, blood urea nitrogen, random blood glucose, HbA_{1c}, total cholesterol, triglyceride, HDL-C, LDL-C, and absolute monocyte count) were obtained from medical reports of the patients. Non-HDL cholesterol was calculated by subtraction of total cholesterol with HDL-C. Monocyte-to-HDL ratio was measured by divided absolute monocyte count with HDL level.

2.4. Statistical Analysis

All data were analyzed with a computer program. Each variable was analyzed descriptively. Shapiro–Wilk and Kolmogorov–Smirnov test was used to determine normality of data. Mean ± standard deviation (SD) and median (minimum–maximum) were used to present data with and without normal distribution, respectively. To determine statistical significant difference, parametric statistic (independent *t*-test) and nonparametric statistic (Mann–Whitney U-test) were used as appropriate. Correlation analysis was performed with Pearson (parametric) or Spearman (nonparametric). Multivariate analysis with logistic regression was performed to determine variables, which associated with poor glycemic control in patients with diabetes. Receiver-operating curve (ROC) analysis was used to predict poor glycemic control in patients with diabetes. Statistical significance of data was set at *p*-value of less than 0.05.

3. RESULTS

The demographic, clinical, and laboratory features of 80 patients with DM involved in this study are depicted in Table 1. No differences were found between all variables with respect to glycemic control status ($p \geq 0.05$), except for the variable of random

Table 1: Demographic, clinical, and laboratory features of the study groups.

Parameters	$HbA_{1c} > 7\%$ ($n = 15$)	$HbA_{1c} \geq 7\%$ ($n = 65$)	*p*-value
Age (years)	56 (41-79)	52 (39-79)	0.381*
Gender (female/male)	5/10	38/27	
Disease duration (months)	72.5 (6-240)	60 (1-240)	0.348*
Systolic BP (mmHg)	120 (100-150)	120 (80-180)	0.685*
Diastolic BP (mmHg)	80 (70-90)	80 (70-100)	0.586*
BMI (kg/m^2)	22.22 (18.73-24.97)	23.43 (16.86-35.79)	0.239*
BUN (mg/dL)	29 (11-68)	14 (2.1-141.4)	0.05*
Creatinine serum (mg/dL)	1.38 (0.7-6.8)	1.2 (0.37-17)	0.02*
RBG (mg/dL)	136 (97-265.44)	235 (57-630)	0.018*
TC (mg/dL)	174 (78-270)	143 (48-376)	0.427*
TG (mg/dL)	134 (80.13-204.32)	127 (42.71-620.57)	0.99*
HDL-C (mg/dL)	34.32 ± 20.24	30.79 ± 13.55	0.527**
LDL-C (mg/dL)	126 (15-202.04)	74.85 (13-230)	0.155*
TC/HDL-C	5.74 (3.45-11.49)	4.97 (1.86-56.33)	0.586*
TG/HDL-C	3.75 (1.99-15.64)	4.28 (0.9-40.33)	0.57*
LDL-C/HDL-C	3.71 (1.49-5.2)	2.6 (0.36-7.46)	0.297*
Non-HDL-C (mg/dL)	149 (63-204)	110.5 (31-335.2)	0.463*
Monocyte (10^3/μL)	1 (0.23-1.49)	0.63 (0.19-3.56)	0.477*
Monocyte/HDL-C	0.02 (0.01-0.1)	0.019 (0.01-0.14)	0.794*

*Mann–Whitney U-test [median (min–max)].
**Independent *t*-test (mean ± standard deviation).
BMI: body mass index; BP: blood pressure; BUN: blood urea nitrogen; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; LDL/HDL-C: low-density lipoprotein to high-density lipoprotein cholesterol ratio; Monocyte/HDL-C: monocyte to high-density lipoprotein cholesterol ratio; RBG: random blood glucose; TC: total cholesterol; TC/HDL-C: total cholesterol to high-density lipoprotein cholesterol ratio; TG: triglycerides; TG/HDL-C: triglycerides to high-density lipoprotein cholesterol ratio.

Table 2: Bivariate correlation results between HbA_{1c} level with individual lipids and lipid ratios parameters in patients with type-2 diabetes.

Parameters	*r*	*p*-value
TC (mg/dL)	−0.122	0.368*
TG (mg/dL)	−0.048	0.72*
HDL-C (mg/dL)	−0.128	0.344**
LDL-C (mg/dL)	−0.087	0.344**
TC/HDL-C	−0.015	0.914*
TG/HDL-C	0.094	0.49*
LDL-C/HDL-C	0.005	0.969*
Non-HDL-C (mg/dL)	−0.061	0.653**
Monocyte/HDL-C	0.084	0.543*

*Spearman correlation analysis.
**Pearson correlation analysis.

Table 3: Multivariate logistic regression results on poor glycemic control in patients with type-2 diabetes ($HbA_{1c} \geq 7\%$).

Parameters	Coefficient	OR	95% CI		*p*-value
			Lower	Upper	
TC (mg/dL)	0.003	1.003	0.966	1.041	0.871
TG (mg/dL)	0.023	1.023	0.988	1.060	0.197
HDL-C (mg/dL)	0.07	1.072	0.907	1.268	0.415
LDL-C (mg/dL)	−0.064	0.938	0.878	1.002	0.059
TC/HDL-C	−0.091	0.913	0.584	1.427	0.690
TG/HDL-C	−0.017	0.984	0.586	1.651	0.950
LDL-C/HDL-C	1.285	3.614	0.535	24.417	0.187

blood glucose and serum creatinine. Variable of random blood glucose tends to increase along with poor glycemic control, while creatinine serum level decreased ($p < 0.05$).

The correlation between HbA_{1c} level with individual lipids and lipid ratios (Table 2) was analyzed using Spearman or Pearson correlation analysis. As shown in Table 2, the HbA_{1c} level was not significantly correlated with individual lipids and lipid ratios.

Multivariate analysis with logistic regression presented in Table 3. Individual lipids and lipid ratios had no significant effect on poor glycemic control in patients with type-2 diabetes.

ROC analysis of LDL-C and LDL-C/HDL-C as prognostic indicators of poor glycemic control were presented in Figures 1 and 2, respectively. ROC analysis in Figure 1 showed that the area under the curve (AUC) was very weak, which means that LDL-C was not accurate to be used as a prognostic indicator of poor glycemic control (AUC, 0.348; 95% CI, 0.117-0.580; $p = 0.155$).

ROC analysis in Figure 2 showed that the area under the curve (AUC) was very weak, which means that ratio of LDL-C and HDL-C was not accurate to be used as a prognostic indicator of poor glycemic control (AUC, 0.389; 95% CI, 0.187-0.591; $p = 0.297$).

4. DISCUSSION

Diabetes mellitus (DM), a lifelong metabolic disease, is caused by either insulin deficiency or insulin resistance or both [1, 2]. To determine the glycemic control, HbA_{1c} serves as the gold standard as it reflects 2-3 months of blood glucose levels [4]. Previous study found alteration in lipid parameter in relation to HbA_{1c} [7]. This comes as an important matter, since it affects the morbidity and mortality of patients, as it is closely related with diabetic complication in terms of CVD.

The present study found that individual lipids and lipid ratios did not differ significantly between good glycemic control and poor glycemic control group. The value of individual lipids and lipid ratios also did not correlate with HbA_{1c}. Parameters of individual lipids and lipid ratios were not independently associated with poor glycemic control, which was analyzed by logistic

Figure 1: Receiver-operating characteristics (ROC) analysis of LDL-C toward poor glycemic control.

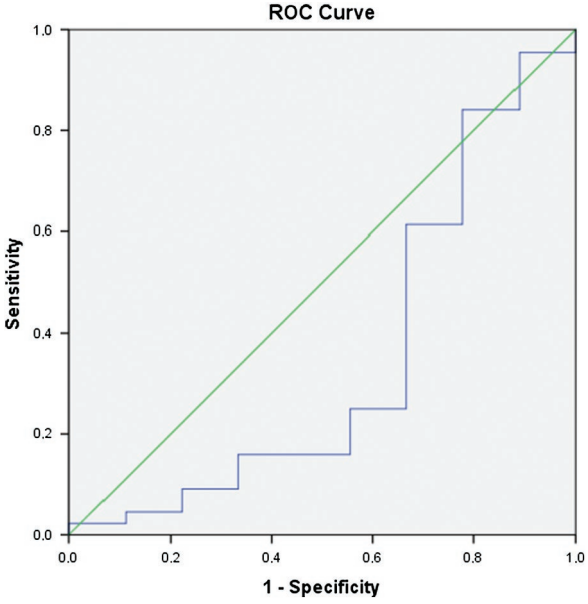
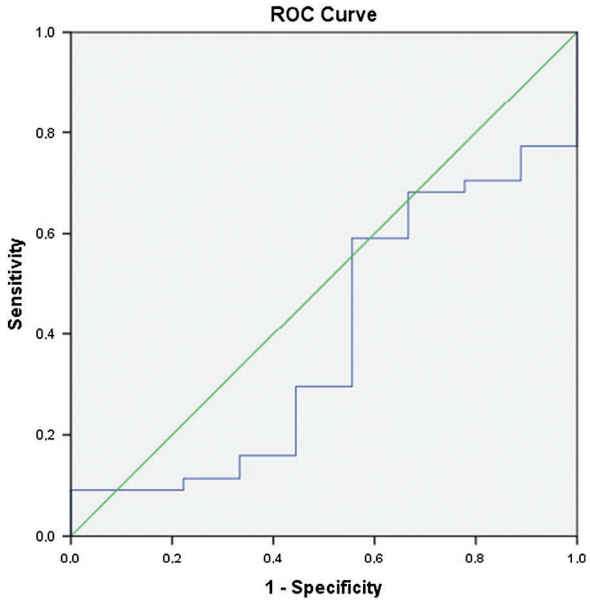


Figure 2: Receiver-operating characteristics (ROC) analysis of LDL-C/HDL-C toward poor glycemic control.



regression. A prognostic indicator of poor glycemic control ($HbA_{1c} \geq 7$) in patients with type-2 diabetes using either LDL-C or LDL-C/HDL-C was not accurate.

The glycemic control is delineated by the amount of HbA_{1c}. In addition, fasting and postprandial blood glucose levels were closely related with HbA_{1c}. The Diabetes Complications and Control Trial (DCCT) determined HbA_{1c} as the best beneficial marker of glycemic control [10]. HbA_{1c} is also able to predict the risk for the emergence of complications in patients with diabetes. The increase in HbA_{1c} is currently considered as an independent risk factor for CVD in an individual with diabetic condition or not. The research found that HbA_{1c} demonstrated significant correlations with lipid abnormalities [7].

The prevalence of lipid abnormalities tends to increase in people with type-2 diabetes, hence contributes to the risk of CVD. Although the pathophysiology of diabetic dyslipidemia remains unclear, the insulin resistance and relative insulin deficiency influence the change in lipid metabolism, since insulin is greatly involved in modulating lipid metabolism [11]. Insulin resistance is related to increased expression of microsomal transfer protein and excessive flux of free fatty acids in the liver. This mechanism is driving the overproduction of very low-density lipoproteins (VLDL) secretion [12]. The intracellular hormone-sensitive lipase, activated by insulin deficiency or resistance, induced the formation of nonesterified fatty acids (NEFA) from triglycerides stored in adipose tissue. High circulating levels of NEFA increase hepatic triglyceride and apolipoprotein B production [13].

This research found that individual lipids (TC, TG, HDL-C, LDL-C, non-HDL-C) and lipid ratios (TC/HDL-C, TG/HDL-C, LDL-C/HDL-C, monocyte/HDL-C) were not significantly different between good or poor glycemic control group, as shown in Table 1. TC, TG, HDL-C, LDL-C, non-HDL-C were not correlated with HbA_{1c} level ($p = 0.368$, $p = 0.72$, $p = 0.344$, $p = 0.344$, $p = 0.653$, respectively). Similarly, the lipid ratios of TC/HDL-C, TG/HDL-C, LDL-C/HDL-C, monocyte/HDL-C were also not correlated with HbA_{1c} level ($p = 0.914$, $p = 0.49$, $p = 0.969$, and $p = 0.543$, respectively). Multivariate analysis with logistic regression found that individual lipids and lipid ratios did not appear to be a significant determinant of poor glycemic control (Table 3).

This result is in contrast with several other studies. Mahato *et al.* [7] found that HbA_{1c} demonstrated positive and significant correlations with TC, LDL-C, LDL-C/HDL-C ratio, non-HDL-C and TC/HDL-C ratio. Compared to the subjects with HbA_{1c} less than or equal to 7.0%, subjects with HbA_{1c} value more than 7.0% had a higher value of TC, LDL-C, LDL-C/HDL-C ratio, non-HDL-C and TC/HDL-C ratio significantly [7]. Yan Z *et al.* [14] also reported that with the increased level of HbA_{1c}, several lipid parameters (LDL-C, TC/HDL-C and LDL-C/HDL-C) had a significantly increasing trend ($p < 0.05$). However, similar to this study, they did not observe a significant correlation of HbA_{1c} with TG, TC or HDL-C [14].

Yan Z *et al.* [14] reported that compared with individual lipid indexes, the changes of lipid ratio could reflect impaired lipid metabolism at an earlier stage, and the most sensitive indicator is LDL-C/HDL-C ratio. However, in contrast to their study, this study revealed that LDL-C and LDL-C/HDL-C could not be used as a predictor of poor glycemic control, as shown in ROC analysis ($p = 0.155$ and $p = 0.297$, respectively). Lipid measures were known to have some predictive power to predict the HbA_{1c} levels. It may be presumed that lipid measures were either related to the poor blood glucose control or also responded to lifestyle measures [15]. There were other stronger parameters to predict the HbA_{1c} level in poorer glycemic control group, such as baseline HbA_{1c} level, glucose lowering agents, higher BMI, younger age, using basal insulin alone, higher insulin dose,

and female [16]. The higher short-term HbA_{1c} levels (6 months) was predicted by longer diabetes duration and lower total cholesterol level at baseline. But cholesterol level was not a predictor of long-term (18 months) HbA_{1c} in patients with diabetes initiating oral antiglycemic agent (metformin). The parameter of the duration of diabetes may be considered as a preferable predictor of HbA_{1c} levels after 6 and 18 months [17].

Besides the main findings, this study revealed that creatinine serum and random blood glucose were significantly different based on HbA_{1c} glycemic control ($p = 0.02$ and $p = 0.018$, respectively). The previous study pointed that HbA_{1c} may be used as a beneficial biomarker to determine the risk of renal disorders and was significantly correlated with creatinine serum in impaired glucose tolerant [18]. Furthermore, poor glycemic control, characterized by higher HbA_{1c} levels, was closely related with chronic kidney disease (CKD) in patients with diabetes. The hyperglycemic condition may activate the growth factors, lead to alteration in creatinine serum levels through the development of intraglomerular hypertension and renal injury [19].

A significant linear-positive correlation is reported between levels of HbA_{1c} and random blood sugar**.** Glycosylated hemoglobin reflects the patient's average blood glucose in the last 120 days. In the condition of high blood glucose, glucose molecules bound to the beta chain of hemoglobin in red blood cells. The longer duration of hyperglycemia, the higher the glycosylated hemoglobin, since more glucose will attach to hemoglobin [20].

This study possesses several limitations. First, this was a cross-sectional study. Second, the number of subjects was relatively small. Thus, further study with larger number of samples is recommended.

5. CONCLUSION

There was no association between individual lipids (TC, TG, HDL-C, LDL-C, non-HDL-C) and lipid ratios (TC/HDL-C, TG/HDL-C, LDL-C/HDL-C, monocyte/HDL-C) with glycemic control status. LDL-C and LDL-C/HDL-C also did not predict poor glycemic control ($HbA_{1c} \geq 7$) in patients with type-2 diabetes.

Author Contributions

Maratni NPT, Sindhughosa DA, Mardewi IGA, and Manuaba IBAP designed the study; Maratni NPT, Sindhughosa DA, and Mardewi IGA collected the data; Maratni NPT, Sindhughosa DA, Mardewi IGA, Manuaba IBAP, and Saraswati MR analyzed and interpreted the data; Maratni NPT, Sindhughosa DA, and Mardewi IGA drafted the manuscript; Saraswati MR critically reviewed the manuscript for important intellectual content and revised the manuscript; All authors read and approved the final manuscript.

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None.

Conflict of Interest

All authors declare that there is no conflict of interest regarding the publication of this manuscript.

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