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Correlation of Glycated Albumin Levels and the Glycated Albumin/HbA1c Ratio with Diabetic Retinopathy

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Abstract

Background: Diabetic retinopathy is one of the microvascular complications of Diabetic Mellitus (DM). Diagnosis of diabetic retinopathy based on clinical manifestations of retinal vascular disorders, assessed using the Early Treatment Diabetic Retinopathy Study (ETDRS) severity scale. Fluctuations in blood glucose levels are thought to play a role in the progression of DM complications, and glycated albumin appears to reflect greater fluctuations in blood glucose levels. The aim of this study was to determine the difference and correlation between glycated albumin levels and glycated albumin/HbA1c ratio (GA/HbA1c ratio) with the degree of severity of diabetic retinopathy.

Methods: The research design was cross-sectional. The subjects of the study were DM patients who were examined for glycated albumin, HbA1c, and GA/HbA1c ratio calculation, and for clinical examination of retinal vascular disorders using the ETDRS severity scale.

Result : The median glycated albumin level in the non-diabetic retinopathy group was lower than the diabetic retinopathy group (18.8 vs 24.3; p=0.018), while the median GA/HbA1c ratio appeared to be lower than the diabetic retinopathy group but did not differ significantly (2.4 vs 3.4; p=0.238). The level of glycated albumin and the GA/HbA1c ratio did not appear to be linear with the severity of diabetic retinopathy. There was a significant weak positive correlation between glycated albumin levels and diabetic retinopathy (r=0.304; p=0.006), while the correlation between the GA/HbA1c ratio and diabetic retinopathy was not significant (r=0.218; p>0.176).

Conclusion : There is a significant difference in glycated albumin levels between non-diabetic non-retinopathy and diabetic retinopathy. There is a significant positive correlation between glycated albumin levels and diabetic retinopathy. It needs further study with a larger sample and a balanced distribution of non-diabetic retinopathy and diabetic retinopathy groups to be used independently.

Keywords: glycated albumin, glycated albumin/HbA1c ratio, ETDRS, diabetic retinopathy

1. INTRODUCTION

Hyperglycemia is a hallmark of Diabetes Mellitus (DM) (1). The increase in cases and prevalence of DM is occurring more rapidly in low- and middle-income countries (1,2,3), which can be accompanied by an increase in the number of DM patients with complications of retinopathy. Diabetic retinopathy is the leading cause of blindness in people aged 20-74 years in the United States, and people with DM have a 25-fold greater risk of developing blindness. Blindness results from progressive diabetic retinopathy and macular oedema (4).

The long-term goal of DM management is to prevent and slow the progression of microangiopathy complications (1), including

using fasting blood glucose, 2-hour postprandial blood glucose (2JPP) and Adult Haemoglobin A1C (HbA1C) (1,5). Glucose levels fluctuate widely, and results can be affected by stress, delayed serum tests, and physical activity, so alternative markers, such as glycated albumin, are needed to assess glycemic control (5).

HbA1c levels reflect average glycemic control over 2-3 months, (4,6), but do not accurately reflect glycemic control in conditions of short-term changes in plasma glucose levels and in some conditions, such as anaemia and haemoglobin variants. Glycated albumin more accurately reflects short-term changes in plasma glucose levels and postprandial glucose levels (7,8) is not affected by haemoglobin metabolism disorders7 and serum protein levels because glycated albumin calculates the ratio between glycated albumin levels and total serum albumin5. With a half-life of serum albumin of about 2 weeks, glycated albumin reflects shorter glycemic control than HbA1c (2-4 weeks previously), so it better reflects fluctuations in glycemic status changes after DM management and is also more sensitive to reflect endogenous insulin secretion (5,6).

2. LITERATURE REVIEW

Diabetes mellitus is a metabolic disease characterised by hyperglycemia caused by abnormalities in insulin secretion, insulin action, or both (1). Chronic hyperglycemia induces apoptosis of vascular endothelial cells by increasing mitochondrial superoxide production. Acute and chronic fluctuations in blood glucose levels can increase oxidative stress (3,9). Excessive glucose metabolism produces free radicals. Several metabolic pathways that can cause metabolic stress in people with type 2 diabetes include the polyol pathway, the hexosamine pathway, activation of protein kinase C (PKC), and advanced glycation end-products (AGEs) (3,10).

Diabetic retinopathy is a microvascular complication of diabetes and is a leading cause of vision loss in the working-age population (11, 12). Symptoms of diabetic retinopathy include sudden changes in vision, blurred vision, gradual vision loss, eye pain, double vision, floating vision, and difficulty seeing at night. In some people with early diabetic retinopathy, there are no symptoms before major bleeding occurs in the eye (13). The diagnosis of diabetic retinopathy is based on clinical manifestations of retinal vascular abnormalities (11).

Duration of suffering from DM, hyperglycemia, age over 40 years, hypertension and dyslipidemia are risk factors for non-proliferative diabetic retinopathy (NPDR) (14). In patients with type 1 DM, 25%, 60%, and 80% experienced diabetic retinopathy after 5, 10, and 15 years, respectively (15), while in type 2 DM, the incidence of diabetic retinopathy after 10 years was 18% and increased to 52% after 20 years (16). The severity of diabetic retinopathy increases with the longer duration of suffering from DM. The number of sufferers of diabetic retinopathy, NPDR and proliferative diabetic retinopathy (PDR), in the group suffering from DM for more than 5 years is more than in the group suffering from DM <5 years (17). Hyperglycemia is the main factor in the development of DM complications. Diabetes mellitus involves several overlapping and interrelated pathways that result in several complications, including diabetic retinopathy and diabetic macular edema (18).

One method for assessing the severity of diabetic retinopathy is a fundus examination, which is graded using the Early Treatment Diabetic Retinopathy Study (ETDRS) severity scale, a multicenter study designed to evaluate argon laser photocoagulation and aspirin treatment in the management of patients with nonproliferative or early proliferative diabetic retinopathy (19). Diabetic retinopathy is divided into early (NPDR) and advanced (PDR) stages (10, 11, 15, 20).

Early stages include mild, moderate, and severe, which may or may not be accompanied by the development of diabetic macular oedema (10). In NPDR, increased vascular permeability and capillary occlusion are found during retinal vessel observation. Microaneurysms, haemorrhages, and fatty deposits (hard exudates) can be detected using fundus photography, although patients may be asymptomatic (11, 20).

In PDR, severe visual impairment can occur due to vitreous haemorrhage or traction retinal

detachment (9). The most common causes of vision loss in patients with diabetic retinopathy are PDR and diabetic macular oedema, characterised by macular swelling due to fluid accumulation in the macula triggered by damage to the blood-retinal barrier. Diabetic macular oedema can occur at any stage of diabetic retinopathy and causes visual distortion and decreased visual acuity (10, 11).

The pathophysiology of diabetic retinopathy includes hyperglycemia, retinal microvasculopathy, inflammation, and retinal neurodegeneration. Hyperglycemia causes retinal microvascular damage. Multiple metabolic pathways are involved in hyperglycemia-induced vascular damage, including the polyol pathway, AGE accumulation, the PKC pathway, and the hexosamine pathway (11). The initial response of retinal blood vessels to hyperglycemia is vascular dilation and altered blood flow (21). Hyperglycemia also triggers pericyte apoptosis, which damages the capillary support structure and leads to microaneurysm formation, the earliest clinical sign of diabetic retinopathy (11). Inflammation plays a key role in the pathogenesis of diabetic retinopathy, which begins with leukostasis. Long-term, low-grade inflammation is found equally across all severity levels of diabetic retinopathy. Elevated pro-inflammatory cytokines and chemokines are found in serum, vitreous, and aqueous fluid samples from patients with diabetic retinopathy (11,22). This leukostasis causes leukocyte-endothelial adhesion mediated by adhesion molecules. Increased endothelial cell adhesion molecules are found in diabetes, and their plasma levels correlate with the severity of diabetic retinopathy (11).

Hyperglycemia causes increased mitochondrial fragmentation and cell apoptosis (23). Increased pro-apoptotic molecules, mitochondrial dysfunction, and increased reactive oxygen species (ROS) are involved in retinal degeneration in diabetic retinopathy. Retinal neurodegeneration is an early stage in the development of diabetic retinopathy (11).

The risk of diabetic retinopathy can be reduced by early detection and strict control of blood glucose levels, blood pressure, and lipid profiles (10). Hyperglycemia will cause haemoglobin, albumin, lipoproteins, and other tissue proteins to undergo non-enzymatic glycosylation (3). Glycosylation is a response to increased blood glucose levels that changes the chemical structure and function of proteins by adding glucose to the amine group of proteins, including haemoglobin and albumin (24). The percentage of glycosylated proteins is used to estimate average glycemic control in the evaluation of diabetes. Glycosylated haemoglobin (HbA) reflects the blood glucose level according to the lifespan of erythrocytes (120 days). Among glycated proteins, HbA is the gold standard for blood glucose control in patients with type 2 diabetes. In recent years, glycated albumin has been introduced as an index of medium-term diabetes control. Glycated albumin is an albumin containing lysine that binds to glucose. Albumin has a shorter half-life of 12-19 days, making it an alternative marker of glycemic control. Glycated albumin occurs when glucose molecules combine with protein molecules to form ketoamines through the process of glycation (3).

In several previous studies, the GA/HbA1c ratio has been shown to correlate with diabetic retinopathy (25). Elevated glycated albumin levels and the GA/HbA1c ratio correlated with diabetic retinopathy (26). However, in several other studies, elevated HbA1c levels correlated with diabetic retinopathy in prediabetic patients, but not with glycated albumin and the GA/HbA1c ratio (27).

3. MATERIALS AND METHODS

The subjects underwent fundus examination with an ophthalmoscope using the ETDRS severity scale to assess diabetic retinopathy, as well as glycated albumin and HbA1c levels. Data analysis was performed to determine differences in glycated albumin levels and the GA/HbA1c ratio between subjects without diabetic retinopathy and those with diabetic retinopathy. Statistical calculations used a significance level of $p<0.05$ and a 95% confidence interval.

Data collection was conducted at the Department of Clinical Pathology and Laboratory Medicine, Faculty of Medicine, Public Health, and Medicine, Gadjah Mada University (FKKMK) - Dr Sardjito General Hospital, Yogyakarta. Inclusion

criteria for this study were patients with diabetes aged >20 years. Exclusion criteria included obesity, proteinuria, type 1 diabetes, drug-induced diabetes, kidney failure, and incomplete data. Subjects signed an informed consent form. This study was a non-experimental study with a cross-sectional design, no specific interventions, and was part of a collaborative study with ethical clearance from the Medical and Health Ethics Research Committee of the Faculty of Medicine, Public Health, and Nursing, Gadjah Mada University (FKKMK UGM), number KE/FK/0431/EC/2019. Based on previous research, 6, the minimum sample size for this study was 32 participants.

Glycated albumin testing was performed at the Pramita Clinical Laboratory using an Abbott Architect CI 4100 instrument with GA-L reagent from Asahi Kasei Pharma Corporation, based on the ketoamine oxidase (KAOD) principle and a reference value of 11-16%. HbA1c testing was performed at the Integrated Laboratory Installation of Dr Sardjito General Hospital using an ADAMS A1c Lite instrument with dual-wavelength colourimetry (420/500 nm) and high-performance liquid chromatography (HPLC). The GA/HbA1c ratio was calculated by dividing glycated albumin by HbA1c. Instrument and method feasibility tests were conducted, including calibration, precision, and accuracy tests on both devices. Glycated albumin samples were pooled and run in a single test. Sample storage was based on the stability of the glycated albumin samples28. HbA1c testing to calculate the GA/HbA1c ratio was performed immediately after sample collection.

4. RESULTS

The study subjects were 81, consisting of 18 men and 63 women, with an age range of 21–84 years (median 61 years, mean 61 years), and a duration of diabetes mellitus (DM) of 2–24 years (median 5 years, mean 7 years). Three subjects (3.7%) were aged <40 years, 30 (37.0%) were aged 40–59 years, and 50 (59.3%) were aged ≥60 years. Twenty-two (27.2%) were aged <5 years, 40 (49.3%) were aged 5–10 years, and 19 (23.5%) were aged >10 years, with a median of 5 years.

Of the 81 study subjects, glycated albumin, GDP, GD2JPP, diabetic retinopathy status, and macular oedema status were examined in all subjects, but HbA1c was measured in only 40 (**Table 1**). Kolmogorov-Smirnov normality tests on age, DM duration, GDP, GD2JPP, and glycated albumin data, as well as Shapiro-Wilk tests on HbA1c data and the GA/HbA1c ratio, indicated non-normal distributions ($p<0.05$).

Table 1. Characteristics of research subjects

Characteristics	n	(%)	Median (Min-Max)	Mean
Age (years)			61 (21-84)	61
- <40	3	3,7		
- 40-59	30	37,0		
- ≥60	48	59,3		
Duration of DM (years)			5 (2-24)	7
- <5	22	27,2		
- 5-10	40	49,3		
- >10	19	23,5		
Glycated albumin (%)	81		19,1 (11,4-59,0)	21,7
HbA1c (%)	40		7,4 (4,5-13,5)	8,0
GA/HbA1c ratio	40		2,5 (1,3-5,8)	2,8
Fasting glucose (mg/dl)	81		120 (80-320)	180
Post prandial glucose (mg/dl)	81		238 (90-435)	244

A total of 74 people (91.4%) experienced non-diabetic retinopathy, and 7 people (8.6%) experienced diabetic retinopathy. The median glycated albumin level in non-diabetic retinopathy was 18.8 (11.4-59.0) and in diabetic retinopathy was 24.3 (21.1-35.6). There was a significant difference in glycated albumin levels between the non-diabetic retinopathy and diabetic retinopathy

groups (median 18.8 vs 24.3; p=0.018). The median glycated albumin level in the non-diabetic retinopathy group appeared lower than in the diabetic retinopathy group (**Table 2**).

Table 2. Median glycated albumin levels in the non-diabetic retinopathy and diabetic retinopathy groups

Characteristics	n	Median glycated albumin (%)	p
Non-diabetic retinopathy	74	18,8 (11,4-59,0)	0,018*
Diabetic retinopathy	7	24,3 (21,1-35,6)	

*) Mann-Whitney test, significant if p<0.05

The median GA/HbA1c ratio for non-diabetic retinopathy was 2.4 (1.3-5.8) and for diabetic retinopathy was 3.4 (3.3-3.6). There was no significant difference in the median GA/HbA1c ratio between non-diabetic retinopathy and diabetic retinopathy (**Table 3**).

Table 3. Median GA/HbA1c ratio in non-diabetic retinopathy and diabetic retinopathy groups

Characteristics	n	Median GA/HbA1c ratio	p
Non-diabetic retinopathy	38	2,4 (1,3-5,8)	0,238
Diabetic retinopathy	2	3,4 (3,3-3,6)	

*) Mann-Whitney test, significant if p<0.05

Based on the severity of diabetic retinopathy, 74 people (91.4%) had non-diabetic retinopathy, 5 people (6.2%) had NPDR, and 2 people (2.4%) had PDR. The median glycated albumin level in non-diabetic retinopathy was 18.8 (11.4-59.0), NPDR 24.3 (22.6-35.6), and PDR 24.0 (21.1-26.8). The median GA/HbA1c ratio in non-diabetic retinopathy was 2.4 (1.3-5.8), whereas in NPDR and PDR, only 1 subject had a GA/HbA1c ratio of 3.6 and 3.3, respectively. The median glycated albumin level in non-diabetic retinopathy was lower than in NPDR and PDR (**Table 4**).

Table 4. Proportion of subjects based on the severity of diabetic retinopathy

Characteristics	n	Median (min-max) glycated albumin	n (%)	Median (min-max) GA/HbA1c ratio
Non-diabetic retinopathy	74	18,8 (11,4-59,0)	38	2,4 (1,3-5,8)
NPDR	5	24,3 (22,6-35,6)	1	- (3,6)
PDR	2	24,0 (21,1-26,8)	1	- (3,3)

Glycated albumin levels were divided into median glycated albumin groups <19.1% and ≥19.1%, GA/HbA1c ratio was divided into median GA/HbA1c ratio groups <2.5% and ≥2.5%, and diabetic retinopathy was divided into non-diabetic retinopathy and diabetic retinopathy groups. In **Table 5**, there was no significant correlation between glycated albumin levels, HbA1c levels, or the GA/HbA1c ratio and diabetic retinopathy; however, there was a weak positive correlation between glycated albumin levels and diabetic retinopathy (r=0.304; p=0.006).

Table 5. Correlation of glycated albumin levels and GA/HbA1c ratio with diabetic retinopathy

Characteristics	r	p
Correlation of glycated albumin levels with diabetic retinopathy	0,304	0,006*
Correlation of GA/HbA1c Ratio with Diabetic Retinopathy	0,218	0,176

*) Spearman correlation test, significant if p<0.05

5. DISCUSSION

In this study, glycated albumin levels were significantly lower in non-diabetic retinopathy compared to diabetic retinopathy. The GA/HbA1c ratio in non-diabetic retinopathy was also lower than in diabetic retinopathy, but not statistically significant. As a previous study has shown that glycated albumin levels and the GA/HbA1c ratio in non-diabetic retinopathy are lower than in the diabetic retinopathy group (18.2±3.6 vs 19.7±4.0; p<0.0001 and 2.55±0.34 vs 2.66±0.31; p<0.001) (24). Mean glycated albumin levels and the GA/HbA1c ratio were higher in the diabetic retinopathy group compared to the non-diabetic retinopathy group, but not in HbA1c (6).

In this study, glycated albumin levels showed a weak positive correlation with diabetic retinopathy, whereas the GA/HbA1c ratio did not. This can be caused by differences in the number of subjects between groups and by the small sample size in this study. Small samples will decrease statistical power. Studies with small numbers of subjects often fail to detect correlations that actually exist in the wider population. In addition, it can also be caused by variations in the duration of diabetes. Diabetic retinopathy is strongly influenced by disease duration. The incidence of diabetic retinopathy may not appear clinically, even though the GA/HbA1c ratio shows blood sugar fluctuations. In a previous study, the incidence of diabetic retinopathy in type 2 DM was 18% after 10 years and 52% after 20 years (16). An increased glycated albumin level was significantly correlated with the presence of diabetic retinopathy, and measuring GA levels in addition to HbA1c was beneficial as a marker for retinopathy, especially in patients with moderate glycemic control (26). A prospective cohort study within the Atherosclerosis Risk in Communities (ARIC) study also showed a strong, significant correlation between glycated albumin levels and diabetic retinopathy (29).

A previous study found that higher levels of glycated albumin in the diabetic retinopathy group support the notion that poorly controlled blood sugar levels, reflected in higher glycated albumin levels, most probably facilitated the development of diabetic retinopathy. Diabetic retinopathy is significantly associated with elevated glycated albumin levels. Measuring glycated albumin levels in addition to HbA1c was beneficial as a marker for retinopathy, especially in patients with moderate glycemic control. Glycated albumin may be a more valuable glycation index than HbA1c for checking glycemic control in type-2 diabetics who have very fluctuating and poorly controlled glycemic conditions (30).

The development of macroangiopathic complications of diabetes is more closely related to postprandial hyperglycemia than to average glucose levels, and HbA1c levels are used as a marker of average glucose levels, not postprandial glucose levels. Glycated albumin reflects both postprandial and average glucose levels. The GA/HbA1c ratio is higher in patients with type 1 diabetes than in those with type 2 diabetes. Blood glucose levels in patients with type 1 diabetes tend to be unstable, with greater glucose fluctuations. This indicates that glycated albumin is better than HbA1c in reflecting the amplitude of glucose fluctuations. Postprandial hyperglycemia is associated with the development of another microangiopathic complication of diabetes, namely diabetic retinopathy. DM complications due to postprandial hyperglycemia are more related to glycated albumin than to HbA1c (8).

Glycated albumin is an index of glycemic control that reflects short-term blood glucose status, specifically over the previous 2-4 weeks. It is independent of serum protein levels because it calculates the glycated albumin-to-total serum albumin ratio. It can detect glycemic fluctuations and changes more quickly than HbA1c (5).

Ketoamine glycated albumin is formed by a specific bond between albumin and glucose through a non-enzymatic oxidation reaction. Therefore, measuring glycated albumin is more specific than fructosamine, which measures the total amount of glycosylated serum protein, which can be influenced by total serum protein levels. Changes in glycated albumin are more sensitive and not parallel to those in fructosamine, making it a suitable indicator of short-term glycemic control (31).

Diabetic complications result from chronic hyperglycemia and acute fluctuations in blood

glucose levels. Previous research has shown a role for glycemic variability, or postprandial glucose excursions, in micro- and macroangiopathic complications. This suggests that even with the same HbA1c level, diabetic complications can occur at different levels depending on the patient's blood glucose fluctuation status (26). Fluctuations in blood glucose levels trigger oxidative stress and accelerate the worsening of endothelial dysfunction9. Therefore, specific indicators for monitoring blood glucose fluctuations may help predict the development of diabetic complications. Glycated albumin also plays a role in atherogenesis by inducing the production of inflammatory mediators in the vascular wall, suggesting that glycated albumin is closely linked to vascular complications in diabetic patients (26).

Advanced glycated end-products, a heterogeneous group of molecules formed by non-enzymatic glycation reactions between reducing sugars and free amino groups of proteins, lipids, and nucleic acids (32), are considered a key factor in the pathogenesis of diabetic retinopathy (10, 33). Although AGE formation is a natural process associated with ageing, and nearly all proteins in the body carry a glycation load, the rate of AGE formation is accelerated in diabetes due to persistent hyperglycemia and oxidative stress. Several previous studies have shown a correlation between AGE levels and the clinical progression of diabetic macular oedema. Vascular endothelial growth factor (VEGF) secretion is the final target of AGEs in most tissues, including the retinal pigment epithelium, leading to neovascularisation with a tendency to leak and disrupting the blood-retinal barrier. Furthermore, VEGF also damages the retinal pigment epithelial barrier, impairing its function (32).

In this study, differences in glycated albumin levels and the GA/HbA1c ratio between each subgroup of diabetic retinopathy severity were not significant. Glycated albumin levels and the GA/HbA1c ratio in the non-diabetic retinopathy group appeared lower than those in the NPDR and PDR groups, but in the more severe retinopathy group (PDR), they appeared lower than those in the milder retinopathy group (NPDR). Both insignificant statistical findings, where the GA/HbA1c ratio in non-diabetic retinopathy is lower than in diabetic retinopathy, which was discussed previously, and differences in glycated albumin levels and the GA/HbA1c ratio between each subgroup of diabetic retinopathy severity, are due to the difference in the number of subjects in the non-diabetic retinopathy group and the retinopathy group being too different. Using a smaller sample increases the risk of assuming a false premise. Unbalanced designs are common in research, but this condition has significant effects, including reduced homogeneity of variance and decreased statistical power. However, we believe that when the number of subjects between groups is equal, the GA/HbA1c ratio can yield a significant result. A previous study found that mean glycated albumin levels in the non-diabetic retinopathy group were lower than those in the NPDR and PDR groups. 27. Other studies stated that glycated albumin levels and the GA/HbA1c ratio were linearly different at each level of diabetic retinopathy severity and could be used for the investigation and progression of diabetic retinopathy, thus strengthening the opinion that fluctuations in blood glucose levels are related to the development and progression of diabetic retinopathy (25).

In this study, there was no significant correlation between glycated albumin levels and HbA1c. This is thought to be the cause of the absence of a difference in the GA/HbA1c ratio between the non-diabetic retinopathy and diabetic retinopathy groups and the absence of a significant correlation between the GA/HbA1c ratio and diabetic retinopathy, in addition to being caused by the small number of samples in the diabetic retinopathy group.

The main limitation of this study was the limited number of samples, with 81 glycated albumin data and 40 GA/HbA1c ratio data. Furthermore, this study could not provide a cut-off point for glycated albumin's ability to predict diabetic retinopathy due to significantly different sample sizes between the non-diabetic retinopathy and diabetic retinopathy groups. earch obtained 5 cut-off points for glycated albumin, namely 2 cut-off points used as limits

that indicate the ability of glycated albumin in screening for poor glycemic control by emphasizing the sensitivity value without sacrificing the specificity value, namely ≥18.7% and ≥19%, 2 cut-off points used as limits that indicate the ability of glycated albumin in diagnosing poor glycemic control by emphasizing the specificity value without sacrificing the sensitivity value, namely ≥21.4% and 22.4%, and 1 cut-off point used as the optimal limit that indicates the balanced ability of glycated albumin both in terms of screening and diagnostics, namely 20.4% (5).

As a result of this study, mean glycated albumin levels had a weak positive correlation with diabetic retinopathy. Median glycated albumin levels were lower in the non-diabetic retinopathy group than in the diabetic retinopathy group, but this difference was not linearly related to the severity of diabetic retinopathy.

6. CONCLUSION

There was a significant difference in glycated albumin levels between non-diabetic retinopathy and diabetic retinopathy patients. There was no significant difference in the GA/HbA1c ratio between non-diabetic retinopathy and diabetic retinopathy patients.

There was a significant weak positive correlation between glycated albumin levels and diabetic retinopathy. There was no significant correlation between the GA/HbA1c ratio and diabetic retinopathy.

7. RECOMMENDATION

Glycated albumin can serve as an additional marker to assess short-term glycemic control and to prevent diabetic retinopathy. Further research is being conducted with a larger sample and a balanced distribution of non-diabetic retinopathy and diabetic retinopathy groups to enable independent use. Istijo, S.A. 2015. Konsensus Pengelolaan dan Pencegahan Diabetes Mellitus Tipe 2 di Indonesia, PERKENI.

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