Association Between LDL-to-HDL Cholesterol Ratio and Albuminuria in Patients With Type 2 Diabetes Mellitus: A Cross-Sectional Study

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Abstract

Background:
Dyslipidemia plays a role in the pathogenesis of diabetic kidney disease, yet the clinical utility of the LDL/HDL ratio for detecting early renal involvement remains uncertain. This study evaluated the association between LDL/HDL ratio and albuminuria in type 2 diabetes mellitus (T2DM) patients.

Methods:
A cross-sectional study was conducted using medical records of adults with T2DM at Siloam Hospital Lippo Village, Indonesia. Patients with complete lipid profiles and urine albumin–creatinine ratio (ACR) data were included. The LDL/HDL ratio was analyzed in relation to albuminuria. Receiver operating characteristic (ROC) analysis determined the optimal cutoff. The ratio was dichotomized using this cutoff, and associations with albuminuria were evaluated using chi-square tests and Poisson regression with robust variance to estimate adjusted prevalence ratios (aPRs).

Result:
A total of 95 patients were analyzed, of whom 46.3% had albuminuria. The LDL/HDL ratio correlated with ACR (ρ=0.528, $p<0.001$). ROC analysis showed good discrimination for albuminuria (AUC 0.796; 95%CI 0.704–0.888), with a bootstrap 95%CI 0.700–0.881. The optimal cutoff was 2.435, yielding 77.3% sensitivity and 74.5% specificity. Albuminuria was more frequent in patients with LDL/HDL ≥2.435 than in those below the cutoff (72.3% vs. 20.8%; $p<0.001$). LDL/HDL ≥2.435 was independently associated with albuminuria (aPR 3.03; 95%CI 1.82–5.26; $p<0.001$). Hypertension, older age, and higher HbA1c were also significant predictors.

Conclusion:
The LDL/HDL ratio showed acceptable discriminatory performance and was independently associated with albuminuria in patients with T2DM. This simple index may serve as an adjunctive marker for renal risk stratification, pending external validation in larger prospective studies.

Introduction

Over the past few decades, the prevalence of type 2 diabetes mellitus (T2DM) has increased substantially, particularly in developing countries, making it a major global public health concern. T2DM is a common metabolic disorder characterized by chronic hyperglycemia resulting from impaired insulin secretion and/or insulin resistance.[1] According to the World Health Organization, an estimated 422 million people worldwide were living with diabetes in 2021, with the majority of cases attributed to T2DM.[2] The rising burden of T2DM has been largely driven by

modifiable risk factors, including high-calorie diets, obesity, and physical inactivity.[3]

One of the most important contributors to morbidity and mortality in patients with T2DM is the development of chronic complications, particularly diabetic kidney disease (DKD).[4] DKD is characterized by progressive renal dysfunction, commonly identified by increased urinary albumin excretion, which is routinely assessed using the urine albumin–creatinine ratio (ACR). ACR is a well-established marker for detecting microalbuminuria and monitoring the progression of renal damage in patients with diabetes.

The pathogenesis of DKD is multifactorial. While chronic hyperglycemia is a key driver of renal injury, other metabolic and hemodynamic disturbances also contribute to disease progression. Excess free fatty acids, advanced glycation end products, and oxidative stress can activate inflammatory pathways, thereby exacerbating glomerular and tubular damage.[5]

Dyslipidemia is another common metabolic abnormality in T2DM, typically characterized by elevated triglyceride levels, increased low-density lipoprotein (LDL) cholesterol, and reduced high-density lipoprotein (HDL) cholesterol.[6] Beyond individual lipid components, lipid ratios have been increasingly used to reflect overall atherogenic burden. Among these, the LDL-to-HDL cholesterol ratio is a practical and informative marker of lipid imbalance and cardiovascular risk in patients with diabetes.

Lipid abnormalities tend to worsen with declining renal function. As kidney function deteriorates, triglyceride levels gradually increase, while HDL cholesterol levels decrease. In early stages of DKD, dyslipidemia is predominantly driven by hypertriglyceridemia. With progression to overt albuminuria, levels of triglycerides, LDL cholesterol, and small dense LDL cholesterol increase substantially, accompanied by a marked reduction in HDL cholesterol. In advanced DKD, lipid metabolism becomes further disrupted, with increased very-low-density lipoprotein cholesterol concentrations.[7–9]

Accumulating evidence suggests that dyslipidemia is not only associated with cardiovascular complications but may also contribute to the development and progression of DKD.[6] Excess circulating lipids are hypothesized to promote endothelial dysfunction, oxidative stress, and chronic inflammation, ultimately leading to renal injury. However, studies specifically examining the relationship between lipid ratios—particularly the LDL-to-HDL ratio—and urinary albumin excretion in patients with T2DM remain limited, as most prior research has focused primarily on cardiovascular outcomes.

Given the limited evidence directly linking the LDL-to-HDL ratio with ACR in patients with T2DM, further investigation is warranted to clarify the role of dyslipidemia in DKD. Evaluating this association may provide additional insight into renal risk stratification and support more targeted strategies for lipid management and the prevention of DKD progression.[10] Therefore, this study aimed to evaluate the association between the LDL-to-HDL cholesterol ratio and albuminuria, as assessed by the urine ACR, in patients with T2DM.

Material And Methods

Study Design and Setting

This study employed a cross-sectional design using secondary data obtained from medical records. The study was conducted at Siloam Hospital Lippo Village, Karawaci, Tangerang, Indonesia. Data were collected retrospectively between January and April 2025 from electronic and physical medical records of eligible patients. This study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline for cross-sectional studies.

Study Population

The target population consisted of adult patients aged 18 years or older with a

confirmed diagnosis of T2DM who received care at Siloam Hospital Lippo Village between January 2018 and January 2025 and had complete relevant clinical and laboratory data available for analysis.

Sampling Method

A non-probability purposive sampling approach was applied. Eligible patients were selected based on predefined inclusion and exclusion criteria. Ethical approval for this study was obtained from the Ethics Committee of the Faculty of Medicine, Pelita Harapan University (Approval No. 290/K-LKJ/ETIK/XI/2024). Permission to access medical records was granted by Siloam Hospital Lippo Village.

Given the retrospective cross-sectional design using anonymized secondary data, the requirement for informed consent was waived by the ethics committee, as the study involved no direct patient contact and posed minimal risk. All data were de-identified prior to analysis to ensure patient confidentiality, in accordance with institutional and international ethical standards.

Eligibility Criteria

Patients were included in the study if they had a confirmed diagnosis of T2DM and complete laboratory data for LDL cholesterol, HDL cholesterol, and urine ACR, all of which were obtained as part of routine clinical care. Patients were excluded if they had newly diagnosed T2DM (diagnosis duration <1 year from first documented diagnosis), unstable glycemic control (HbA1c fluctuation >2% within the preceding 3 months or documented treatment intensification for poor glycemic control), severe obesity (body mass index >35 kg/m²), heavy smoking history (≥10 pack-years), advanced chronic kidney disease (estimated glomerular filtration rate <30 mL/min/1.73 m²), or had documented acute infection requiring antimicrobial treatment or hospitalization at time of laboratory assessment.

Although all eligible patients during the study period were included, a minimum sample size was estimated using a two-proportion formula with α=0.05 and 80% power.

Data Collection and Outcomes

Data were extracted from electronic and physical medical records. The primary outcome was albuminuria, defined using urine ACR measurements and categorized as normoalbuminuria or albuminuria according to established clinical thresholds (ACR ≥30 mg/g). The primary exposure was the LDL/HDL ratio, calculated by dividing LDL cholesterol by HDL cholesterol concentrations measured in the same fasting blood sample.

Demographic variables included age and sex. Clinical variables included systolic and diastolic blood pressure, hypertension status (physician-diagnosed or documented antihypertensive use), smoking status, and medication use (angiotensin-converting enzyme [ACE] inhibitors/angiotensin receptor blockers [ARBs], metformin, insulin). Glycated hemoglobin (HbA1c) was included as a marker of glycemic control.

Laboratory measurements were performed as part of routine hospital clinical practice using standardized automated assays. The same institutional laboratory methods were applied to all patients, ensuring comparability of measurements across groups.

To reduce selection bias, predefined inclusion and exclusion criteria were applied uniformly. Information bias was minimized by using standardized laboratory measurements recorded prior to study conception. Data extraction was performed using structured forms, and all data were anonymized before analysis.

Statistical Analysis

Statistical analyses were performed using SPSS software (IBM Corp., Armonk, NY, USA) and R version 2025.09.0+387. Data normality was assessed using the Shapiro–Wilk test. Continuous variables

with normal distribution were expressed as mean ± standard deviation, whereas non-normally distributed variables were presented as median with interquartile range (IQR). Categorical variables were summarized as frequencies and percentages.

Baseline characteristics were compared between patients with normoalbuminuria and albuminuria using independent t-tests or Mann–Whitney U tests for continuous variables, as appropriate, and chi-square tests for categorical variables.

Given the non-normal distribution of the LDL/HDL ratio, Spearman rank correlation analysis was used as the primary analysis to evaluate the association between the LDL/HDL ratio and urine ACR. Receiver operating characteristic (ROC) curve analysis was subsequently conducted to assess the discriminatory performance of the LDL/HDL ratio for albuminuria and to determine the optimal cutoff value based on the Youden index. The area under the curve (AUC), sensitivity, specificity, and 95% confidence intervals (CIs) were reported. Internal validation of discriminatory performance was performed using stratified bootstrap resampling with 2,000 replicates to estimate bias-corrected 95% confidence intervals for the AUC. ROC curves of the LDL/HDL ratio, LDL cholesterol, and HDL cholesterol were compared using the DeLong method for correlated ROC curves.

For categorical analysis, the LDL/HDL ratio was dichotomized using the ROC-derived cutoff value, and its association with albuminuria was evaluated using the chi-square test.

To estimate the association between the LDL/HDL ratio and albuminuria while accounting for potential confounders, Poisson regression with robust variance estimation was used to calculate prevalence ratios (PRs) and 95% confidence intervals (CIs). The LDL/HDL ratio was entered as a categorical variable based on the ROC-derived cutoff to enhance clinical interpretability. The unadjusted model included only the LDL/HDL ratio category, while the multivariable model was adjusted for age, sex, hypertension, and HbA1c. A two-tailed p-value < 0.05 was considered statistically significant. No subgroup or interaction analyses were performed.

Only patients with complete data were included; therefore, no imputation for missing data was required. Because purposive sampling was used, analyses were interpreted as applicable to the study population without weighting. No additional sensitivity analyses were conducted.

Result

Baseline Characteristics

A total of 135 patients were initially identified through purposive sampling. Of these, 40 patients (29.6%) were excluded for not meeting inclusion criteria. Reasons for exclusion included newly diagnosed T2DM (<1 year), unstable glycemic control, severe obesity (BMI >35 kg/m²), heavy smoking, advanced chronic kidney disease (eGFR <30 mL/min/1.73 m²), and active infection. After applying eligibility criteria, 95 patients were included in the final analysis (Table 1). As this was a cross-sectional study using complete medical records, no follow-up was required and all eligible participants were analyzed.

Among the 95 patients with T2DM, 51 (53.7%) had normoalbuminuria and 44 (46.3%) had albuminuria. The overall mean age was 63 ± 9 years, and 48 patients (50.5%) were male.

Patients with albuminuria were significantly older than those with normoalbuminuria (65 ± 8.8 vs. 61 ± 8.6 years, p = 0.010). Systolic and diastolic blood pressures were also significantly higher in the albuminuria group (140.2 ± 18.1 vs. 123.4 ± 14.2 mmHg, p < 0.001; and 76.1 ± 12 vs. 71.7 ± 10.2 mmHg, p = 0.034, respectively). Accordingly, the prevalence of hypertension was markedly greater among patients with albuminuria (47.7% vs. 13.7%, p < 0.001).

With respect to metabolic parameters, median HbA1c levels were significantly higher in the albuminuria group (7.54 [IQR 6–8] vs. 6.60 [IQR 6–7], p = 0.023), although the proportion of patients achieving HbA1c <7% did not differ

significantly between groups (p = 0.135). Mean LDL and HDL cholesterol levels were comparable between groups; however, the LDL/HDL ratio was significantly higher among patients with albuminuria (median 3.33 [IQR 2–4] vs. 2.24 [IQR 1–3], p < 0.001).

Regarding medication use, insulin therapy was more frequent in the albuminuria group (31.8% vs. 11.8%, p = 0.017). Use of ACEis or ARBs and metformin tended to be higher among patients with albuminuria, although these differences did not reach statistical significance.

All included participants had complete data for the primary variables of interest; therefore, no missing data were present for LDL, HDL, HbA1c, or ACR.

Correlation Between LDL/HDL Ratio and Albumin-Creatinine Ratio

Given the non-normal distribution of the LDL/HDL ratio, Spearman correlation analysis was performed to assess its association with ACR (Table 2). A moderate positive correlation was observed between the LDL/HDL ratio and ACR (ρ = 0.528, p < 0.001), indicating that higher LDL/HDL ratios were associated with greater degrees of albuminuria.

ROC Curve Analysis

Receiver operating characteristic (ROC) analysis demonstrated that the LDL/HDL ratio had good discriminatory ability for albuminuria, with an apparent AUC of 0.796 (95% CI 0.704–0.888; p<0.001). The ROC curve is displayed in Figure 1. Internal bootstrap validation using 2,000 stratified replicates yielded a similar AUC estimate with a 95% CI of 0.700–0.881, supporting the stability of the model's discrimination. The optimal cutoff value was 2.435, with sensitivity of 77.3% and specificity of 74.5%.

In comparative ROC analyses, the LDL/HDL ratio demonstrated superior discrimination for albuminuria compared with LDL cholesterol alone (AUC 0.796 vs. 0.595; DeLong p<0.001) and HDL cholesterol alone (AUC 0.573; DeLong p<0.001) (Table 3).

Categorical Association Between LDL/HDL Ratio and Albuminuria

Using the ROC-derived cutoff of 2.435, patients were stratified into low and high LDL/HDL ratio groups (Table 4). Albuminuria was markedly more prevalent among patients with an LDL/HDL ratio ≥ 2.435 compared with those below the cutoff (72.3% vs 20.8%). Conversely, normoalbuminuria predominated in patients with LDL/HDL < 2.435 (79.2% vs 27.7%). Chi-square analysis demonstrated a statistically significant association between elevated LDL/HDL ratio and albuminuria (p < 0.001).

Multivariable Regression Analysis

In unadjusted Poisson regression analysis, patients with LDL/HDL ratio ≥ 2.435 had a significantly higher prevalence of albuminuria compared with those below the cutoff (PR 3.45; 95% CI 1.96–6.25; p < 0.001) (Table 5).

After adjustment for age, sex, hypertension, and HbA1c, LDL/HDL ≥ 2.435 remained independently associated with albuminuria. Patients with LDL/HDL ≥ 2.435 had a threefold higher prevalence of albuminuria compared with those below the cutoff (aPR 3.03; 95% CI 1.82–5.26; p < 0.001). Hypertension was also independently associated with albuminuria (aPR 2.13; 95% CI 1.47–3.02; p < 0.001). Increasing age (aPR 1.04 per year; p = 0.001) and higher HbA1c levels (aPR 1.09 per 1% increase; p = 0.009) were significant predictors, whereas sex was not associated with albuminuria.

No subgroup, interaction, or sensitivity analyses were performed. Because complete-case data were required for inclusion, no imputation procedures were necessary.

Discussion

Our findings demonstrate that the LDL/HDL ratio is a meaningful marker of renal involvement in T2DM. The strong

categorical association suggests that dyslipidemia characterized by elevated atherogenic lipoproteins relative to protective HDL is closely linked to glomerular injury.

The moderate correlation between LDL/HDL ratio and ACR further supports a dose–response relationship, indicating that worsening lipid imbalance parallels increasing severity of albuminuria. This observation is consistent with the concept that diabetic kidney disease (DKD) evolves along a continuum rather than through discrete thresholds.

Importantly, the persistence of this association after multivariable adjustment underscores the independent contribution of lipid imbalance to renal injury. While hypertension and age remain dominant drivers of DKD, our findings suggest that the LDL/HDL ratio provides additional risk stratification beyond these established factors.

A key finding of this study is the identification of an optimal LDL/HDL ratio cutoff of 2.435 for detecting albuminuria, with an AUC of 0.796, indicating good discriminatory ability. This suggests that the LDL/HDL ratio can serve as a clinically useful marker for identifying early renal involvement in patients with type 2 diabetes.

Previously proposed cutoffs provide useful context for interpreting this threshold. A ratio around 2.3 has been considered clinically appropriate for cardiometabolic risk and early renal involvement, demonstrating moderate sensitivity and specificity in vascular risk assessments.[11,12] Higher thresholds in the 2.5–2.7 range are frequently cited as clinically relevant for cardiometabolic and renal risk stratification, while values >2.7 have been proposed in some diabetic nephropathy cohorts but may reduce sensitivity and limit routine applicability.[12,13]

The ROC-derived cutoff of 2.435 identified in this study lies within the clinically recognized range but is slightly higher than the commonly used 2.3 threshold. This variation likely reflects population-specific metabolic characteristics, ethnic and regional differences in dyslipidemia patterns, and the use of albuminuria—rather than cardiovascular outcomes—as the primary endpoint. The observed sensitivity (77.3%) with acceptable specificity indicates that this threshold may improve discrimination for early renal involvement in Southeast Asian patients with type 2 diabetes. Collectively, these findings highlight the importance of population-specific cutoffs rather than relying on universal thresholds.

An important finding of the present study is that the LDL/HDL ratio significantly outperformed its individual lipid components in detecting albuminuria. Although LDL-C and HDL-C alone showed poor discrimination, combining these opposing lipid fractions into a ratio markedly improved diagnostic performance. This supports the concept that integrated lipid indices may better reflect the balance between atherogenic burden and protective lipid capacity than isolated lipid measures.

Pathophysiological Implications

An elevated LDL/HDL ratio reflects a state of lipid imbalance driven by insulin resistance, a hallmark of T2DM. Insulin resistance activates hormone-sensitive triglyceride lipase, increasing free fatty acid release from adipose tissue and stimulating hepatic triglyceride synthesis. This process raises circulating triglyceride levels and, via cholesterol ester transfer protein–mediated exchange, leads to a reciprocal reduction in HDL cholesterol.[14] The resulting dyslipidemia promotes ectopic lipid deposition in non-adipose tissues, including the kidney, where abnormal lipid accumulation contributes to lipotoxic injury.

In diabetic kidneys, triglyceride-rich lipid droplets accumulate within the glomeruli and tubulointerstitial compartments, activating inflammatory pathways and promoting transforming growth factor-β and reactive oxygen species production.[15,16] Podocytes are particularly susceptible to lipid overload; excessive lipid deposition induces mitochondrial oxidative stress, disrupts energy metabolism, and triggers cytoskeletal alterations that lead to foot process

effacement and podocyte loss.[6,15,17] These structural and functional changes compromise the glomerular filtration barrier, increasing permeability to albumin. In parallel, enhanced lipolysis of triglyceride-rich lipoproteins generates small dense LDL particles, which readily deposit in the renal basement membrane, undergo oxidative modification, and stimulate extracellular matrix expansion.[9]

Low HDL cholesterol further exacerbates renal injury by impairing antioxidant and anti-inflammatory defenses. Dysfunctional HDL reduces clearance of advanced glycation end products and promotes mesangial expansion and glomerular hypertrophy.[18] Oxidative stress, present even in early DKD, facilitates LDL oxidation, and oxidized LDL directly damages renal tubular epithelial cells, accelerating renal dysfunction and albuminuria.[19] Together, these mechanisms highlight how an increased LDL/HDL ratio captures the balance between pro-atherogenic and protective lipid fractions, providing a biologically plausible link between dyslipidemia and albuminuria in DKD.

Several issues regarding temporality warrant careful consideration. Because of the cross-sectional design, the direction of association between the LDL/HDL ratio and albuminuria cannot be established. Although dyslipidemia may contribute to renal injury, reverse causation is also plausible, whereby early DKD itself alters lipid metabolism through changes in lipoprotein handling, inflammation, and insulin resistance. Accordingly, the LDL/HDL ratio should be interpreted as a concurrent risk marker rather than a proven causal determinant or longitudinal predictor of albuminuria.

Comparison with Previous Studies

Previous studies evaluating the relationship between lipid parameters and albuminuria in patients with T2DM have reported heterogeneous findings. A study by Hwang et al. demonstrated that increased urinary ACR (>30 mg/g) was associated with an adverse lipid profile. In that study, albuminuric patients had higher mean total cholesterol, triglycerides, and LDL cholesterol, along with significantly lower HDL cholesterol compared with nonalbuminuric patients. Additionally, HbA1c levels were significantly higher in the albuminuria group, suggesting a combined effect of poor glycemic control and dyslipidemia on renal involvement.[20] These findings support the concept that lipid abnormalities may accompany or exacerbate DKD.

In contrast, other investigations have failed to demonstrate a significant association between conventional lipid parameters and albuminuria. Several studies reported no meaningful differences in LDL-C, HDL-C, or triglyceride levels between patients with and without albuminuria, indicating that isolated lipid measures may have limited sensitivity in capturing renal risk across different populations.[21,22]

Our findings add modest but clinically relevant evidence to the existing literature. Prior studies have linked dyslipidemia and various lipid ratios with albuminuria or DKD progression, but data specifically evaluating the LDL/HDL ratio in Southeast Asian patients with T2DM remain limited. Moreover, few studies have directly compared the discriminatory performance of the LDL/HDL ratio against its individual components in relation to albuminuria. By demonstrating significant superiority over LDL-C and HDL-C alone, the present study extends prior work and supports further evaluation of this inexpensive, routinely available marker.

Clinical Implications

From a clinical perspective, the LDL/HDL ratio is easily derived from routine lipid profiles and may serve as a practical adjunct marker for identifying patients at higher risk of diabetic kidney involvement. Early identification of such patients could facilitate more aggressive risk factor modification, including lipid-lowering therapy, blood pressure control, and optimization of glycemic management. However, given the cross-sectional design, the LDL/HDL ratio should be considered a risk marker rather than a causal factor at this stage.

The generalisability of our findings should be interpreted in light of the single-center, cross-sectional design and the specific characteristics of the study population. Participants were clinically stable adults with established type 2 DM, and patients with newly diagnosed disease, advanced chronic kidney disease, severe obesity, heavy smoking, or active infection were excluded; therefore, the results are most applicable to similar outpatient populations with preserved to moderately reduced renal function. The LDL/HDL ratio cutoff of 2.435 may be particularly relevant to Southeast Asian patients, given ethnic and metabolic differences in lipid patterns and insulin resistance, and may not be directly transferable to populations with different cardiovascular risk profiles or treatment patterns. As the study design precludes causal inference, the LDL/HDL ratio should be considered a concurrent risk marker rather than a predictive tool. Nevertheless, because it is derived from routine lipid testing, it has practical applicability across various clinical settings, pending validation in larger, multicenter, and prospective studies.

Strengths and Limitations

This study has several strengths. It is among the first in Southeast Asia to derive a population-specific LDL/HDL ratio cutoff for albuminuria in patients with T2DM. The use of ROC analysis enhances clinical applicability, while multivariable Poisson regression provides robust prevalence estimates.

Several limitations should be acknowledged. The cross-sectional, single-center design precludes causal inference and may limit generalizability. Residual confounding from unmeasured factors, including diabetes duration, dietary patterns, inflammatory markers, and unavailable statin or other lipid-lowering therapy data, cannot be excluded. The modest sample size may have affected cutoff precision; however, bootstrap validation demonstrated similar discrimination estimates, suggesting limited optimism in the reported AUC. Further prospective multicenter studies are warranted.

Future Directions

Prospective cohort studies are warranted to determine whether elevated LDL/HDL ratios predict the onset or progression of albuminuria over time. Further research should also explore whether targeted lipid-modifying interventions can attenuate renal risk independently of glycemic and blood pressure control. Incorporating lipid ratios into multifactorial risk models may enhance early risk stratification for DKD.

Conclusion

The LDL/HDL ratio demonstrated good discriminatory ability for detecting albuminuria in patients with type 2 diabetes, with an optimal cutoff of 2.435. Individuals with ratios above this threshold had a markedly higher prevalence of albuminuria, supporting the potential role of this lipid index as a simple and accessible marker for early renal involvement. These findings underscore the value of population-specific thresholds and suggest that the LDL/HDL ratio may aid may serve as an adjunctive marker for renal risk stratification pending external validation. Further prospective and multicenter studies are warranted to validate these results and assess their clinical applicability.

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Author's Statement

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