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Red blood cell distribution width/Albumin ratio as an independent predictor of diabetic peripheral neuropathy: A retrospective study

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Abstract

Background & Objective: Diabetic peripheral neuropathy (DPN) is a common chronic complication of diabetes characterized by the involvement of the peripheral nervous system. This study aimed to investigate the association between the red blood cell distribution width/albumin ratio (RAR) and the presence of diabetic peripheral neuropathy (DPN). *Methods:* The association between DPN, RAR, and other variables was examined using logistic regression analysis. To determine the predictive validity and optimal cutoff value of RAR for the presence of DPN, Receiver Operating Characteristic (ROC) curve analysis was performed to calculate the Area Under the Curve (AUC). Results: Compared to those without DPN, patients with DPN had a significantly higher prevalence of hypertension ($p=0.006$), a higher rate of smoking ($p=0.002$), and a longer duration of diabetes ($p<0.001$). Patients with DPN had significantly lower albumin levels ($p<0.001$), and significantly higher RDW ($p<0.001$) and RAR ($p<0.001$) values. As an independent variable, RAR was independently associated with higher odds of DPN (OR: 1.545, 95% CI: 1.235–1.914, $p<0.001$). The optimal cutoff value for RAR to predict the presence of DPN was determined to be 4.4 %/(g/dL) (Spec.: 99.2%, Sen.: 32.2%, AUC: 0.67).
Conclusion: Our findings suggest that RAR is an independent predictor of DPN and may serve as a complementary biomarker compared with RDW or albumin levels alone. Further prospective, multi-center studies with larger sample sizes are needed to more robustly establish the association between RAR and DPN.

Keywords: Red blood cell distribution width, albumin, diabetic peripheral neuropathy, type 2 diabetes mellitus

INTRODUCTION

Diabetes mellitus (DM) is now considered a global epidemic. The increasing prevalence of diabetes has led to a rise in the incidence of its chronic complications. The prevalence of diabetic peripheral neuropathy (DPN) among patients with diabetes is approximately 30%, and this rate increases over the course of the disease.[1] The most common form is distal symmetric polyneuropathy (DSPN), which is characterized by distally located pain and numbness in the lower extremities. Sensory and autonomic axons are typically affected more than motor axons.[2] Although the exact pathophysiology of DPN development is not fully elucidated, inflammation, oxidative stress, and mitochondrial damage are thought to occur as a result of a series of biochemical reactions affecting cellular metabolism, associated with glucotoxicity and hyperlipidemia.[3] These changes may lead to neuronal apoptosis and segmental demyelination within the peripheral nervous system, resulting in neuronal-glial damage and loss in both myelinated and unmyelinated fibers. Among the most important risk factors for the development of DPN are chronic hyperglycemia, central obesity, dyslipidemia, hypoinsulinemia, hypertension, age, and height.[4]

Diabetes mellitus (DM) has been shown to cause red blood cell dysfunction, associated

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with increased aggregation, microviscosity, and adhesiveness, as well as impairments in membrane structure.[5] In advanced diabetes, fragile erythrocytes become more susceptible to lysis, contributing to the increased severity of vascular complications.[6] Previous studies have associated high RDW values with the development of diabetic complications and an increased risk of mortality.[7,8] Furthermore, low serum levels of albumin, an antioxidant protein synthesized by the liver, have been observed in inflammatory and hypercoagulable states.[9] Recently, the RDW/albumin ratio (RAR), a novel inflammatory biomarker, has been utilized in assessing diabetic retinopathy, disease prognosis, and mortality risk, and has been shown to be a useful predictor.[10] Given the inadequacy of treatments targeting nerve damage in DPN, laboratory findings that enable the early detection of neuropathy are highly important. However, the role of the RAR in the development of DPN remains unclear. Therefore, in this study, we aimed to determine the predictive role of RAR for DPN development and to evaluate the association between them.

METHODS

Study design and participants

This retrospective study received ethical approval from our hospital's ethics committee on 25.03.2025 (protocol number: 384), and all procedures were conducted in accordance with the principles of the Declaration of Helsinki. Verbal consent was obtained since only previously recorded data were used. The study included 275 patients diagnosed with type 2 diabetes mellitus who presented to the outpatient neurology clinic between January 2022 and January 2025. Patients with type 2 diabetes were classified into two groups those with diabetic peripheral neuropathy (DPN) and those without DPN based on clinical symptomatic examination and the results of nerve conduction studies (NCS).

Patients were excluded from the study if they had type 1 diabetes mellitus or gestational diabetes. Additional exclusion criteria included known causes of polyneuropathy other than diabetes, such as: cervical and lumbar spondylopathy, rheumatic diseases, chronic kidney disease, oncological diseases, receiving chemotherapy treatment, immunological diseases, infections, thyroid disorders, use of medications known to affect thyroid function, chronic alcohol use, vitamin B12 deficiency, and hereditary neuropathy. Additionally, pregnant patients, individuals younger than 18 years, those with incomplete data, and patients who declined participation were not enrolled in the study. Data on patient age, sex, duration of diabetes, smoking status, history of chronic diseases (comorbidities), and biochemical parameters were recorded. Body Mass Index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). The RDW/albumin ratio (RAR) was calculated by dividing the red blood cell distribution width (RDW) by the serum albumin level.[10]

Definition of DPN

DPN is defined as the clinical and electrophysiological confirmation of peripheral neuropathy in patients with diabetes, after other causes of peripheral neuropathy have been excluded. In our clinic, prior to establishing a DPN diagnosis, patients underwent a neurological assessment that included evaluation of temperature, vibration, and pinprick sensation, as well as testing of ankle and knee tendon reflexes. Nerve conduction studies (NCS) were also performed. Patients were evaluated clinically and electrophysiologically by a neurologist with 10 years of experience in diabetic neuropathy. Ultimately, patients were classified as having DPN if they had a diagnosis of type 2 diabetes, presented with clinical symptoms and nerve conduction study (NCS) findings consistent with neuropathy, and had other potential causes of neuropathy ruled out.

Statistical analysis

Statistical analyses were performed using SPSS version 21. The Pearson Chi-Square test was used to analyze differences in categorical variables, such as sex, smoking status, presence of heart disease, hypertension, and stroke, between the groups with and without DPN. The Kolmogorov-Smirnov test was used to assess the normality of distribution for numerical (continuous) variables, including biochemical parameters and BMI values. As the continuous data were not normally distributed, the Mann-Whitney U test was employed to analyze differences between the groups based on median values. Logistic regression analysis was conducted to examine the association between DPN and RAR and other variables; Odds Ratios (OR) and their 95% Confidence Intervals (CI) were calculated. Fasting blood glucose was evaluated as an indicator of short-term glycemic status at the time of assessment, whereas HbA1c was included as a marker of long-term glycemic

control. Both parameters were initially analyzed to explore their univariate associations with diabetic peripheral neuropathy; however, only HbA1c was retained as an independent predictor in the multivariate logistic regression model. Finally, Receiver Operating Characteristic (ROC) curves and the Area Under the Curve (AUC) were utilized to determine the predictive validity and the optimal cutoff value of RAR for the presence of DPN. A p-value < 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 275 patients with type 2 diabetes mellitus were included in the study: 143 patients with DPN and 132 patients without DPN. The detailed participant enrollment process is shown in Figure 1.

The demographic characteristics of the study population are presented in Table 1. There were no significant differences between the DPN and non-DPN groups in terms of age or sex (p>0.05 for both). Regarding comorbidities, hypertension was significantly more prevalent among patients with DPN compared with those without DPN (p=0.006). In addition, the proportion of smokers was significantly higher in the DPN group (p=0.002). No significant between-group differences were observed for cardiovascular disease (p=0.076) or previous stroke history (p=0.055).

The groups were compared regarding biochemical parameters and BMI values. Patients with DPN had significantly higher fasting blood glucose levels (p=0.016) and HbA1c values (p=0.007), an indicator of long-term glycemic control, compared to those without DPN. Patients with DPN also exhibited significantly lower albumin levels (p<0.001), significantly higher

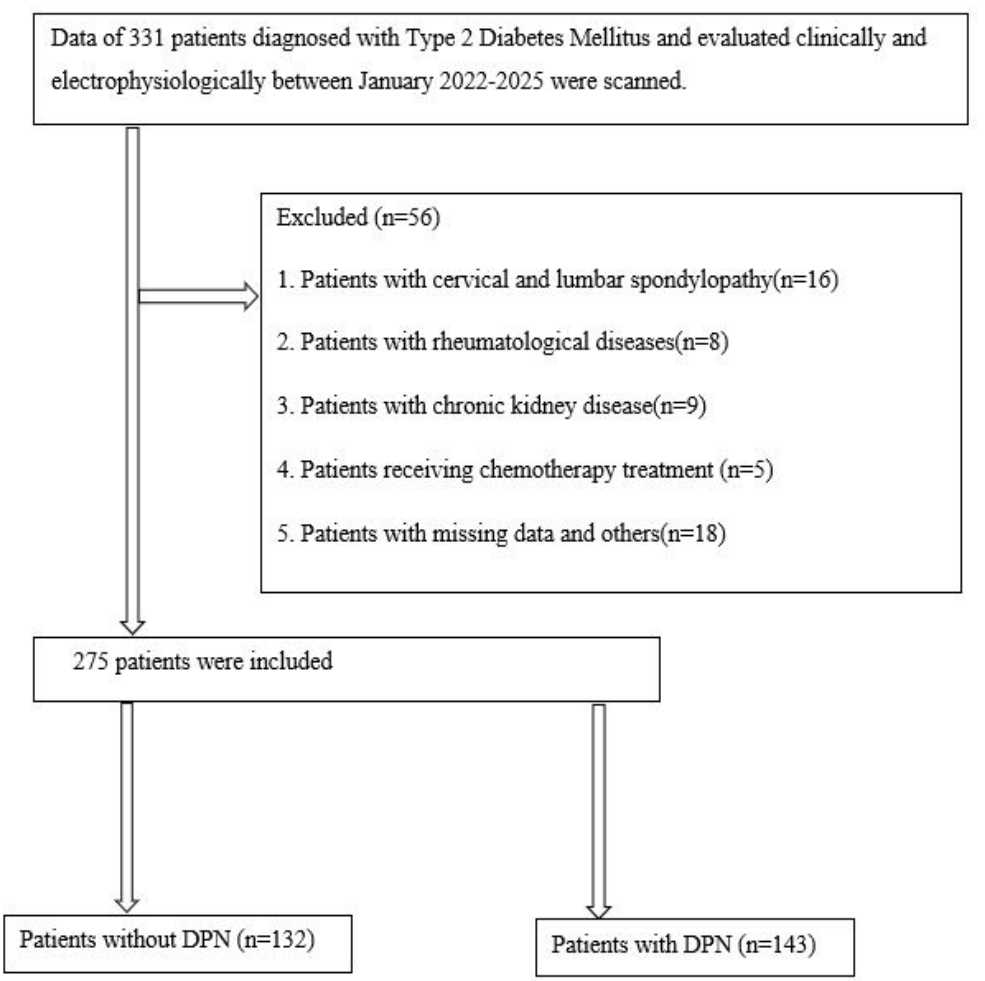

Figure 1. Flow chart of patients included in the study

Table 1: Demographic characteristics of patients with type 2 diabetes mellitus

	Without DPN(n=132)	With DPN(N=143)	t	p
Gender				
Male	62(%46.97)	72 (%48.95)	7.39	0.234
Female	70(%53.03)	73 (%51.05)		
Smoking status				
Smoker	19(%14.4)	49(%34.3)	13.51	**0.002**
Nonsmoker	113(%85.6)	94(%65.7)		
CVD				
Present	24(%37.5)	40(%62.5)	3.16	0.076
Absent	108(%51.2)	103(%48.8)		
HT				
Present	37(%36.6)	64(%63.4)	7.56	**0.006**
Absent	95(%54.6)	79(%45.4)		
Stroke				
Present	11(%31.4)	24(%68.6)	3.68	0.055
Absent	121(%50.4)	119(%49.6)		
Age	59.75±9.01	61.44±8.34	8405.5^{Z}	0.177
Disease Duration (year)	8.86±3.85	14.33±5.57	3983.0^{Z}	**<0.001**

Test Statistics: Pearson Chi-Square Test, Z: Mann Whitney U, DPN: Diabetic peripheral neuropathy, CVD: Cardiovascular diseases, HT: Hypertension

RDW values (p<0.001), and significantly higher RAR values (3.99 %/g/dL vs 3.31 %/g/dL, p<0.001) compared to the non-DPN group. (Table 2)

Multivariate logistic regression analysis demonstrated that RAR was independently associated with higher odds of diabetic peripheral neuropathy (OR: 1.545, 95% CI: 1.235–1.914, p<0.001). Each 1-unit increase in RAR was associated with 1.545-fold higher odds of DPN. In addition, diabetes duration was independently associated with higher odds of DPN (OR: 1.284 per year, 95% CI: 1.184–1.392, p<0.001),

Table 2: **Biochemical test results and differences by groups**

	Without DPN(n=132)	With DPN(n=143)	Z	P
FBG(mg/dl)	189.7±58.1	215.2±66.3	7366.0	**0.016**
HbA1c(%)	8.79±2.06	10.3±3.2	13609.5	**0.007**
Hemoglobin(g/dl)	13.8±1.9	13.6±1.4	10337.5	0.172
TG(mg/dl)	134.8±47.4	139.1±52.1	9097.2	0.605
HDL(mg/dl)	47.4±12.1	46.6±10.1	9978.1	0.412
LDL(mg/dl)	108.2±26.8	111.1±27.7	8885.0	0.401
RDW(%)	13.23±0.54	13.90±1.11	6063.5	**<0.001**
Albumin(g/dl)	4.11±0.72	3.72±0.91	11896.0	**<0.001**
RAR	3.31±0.58	3.99±1.16	6253.5	**<0.001**
BMI	30.7±4.5	30.4±5.1	9750.5	0.635

Z: Mann Whitney U, FBG: Fasting blood glucose, TG: Triglyceride, HDL: High density lipoprotein cholesterol, LDL: Low density lipoprotein cholesterol, RDW: Red Blood Cell Distribution Width, RAR: RDW/Albumin ratio, BMI: Body mass index

Table 3: Two-State Logistic Regression for the relationship between DPN and variables

Variables	β	S.E.	p	OR (95% CI)
Disease duration	0.250	0.042	**<0.001**	1.284(1.184-1.392)
RAR	0.435	0.102	**<0.001**	1.545(1.235-1.914)
HT	0.317	0.298	0.287	1.375(0.771-2.434)
Smoking status	0.203	0.371	0.583	1.225(0.605-2.582)
HbA1c(%)	0.126	0.069	**0.047**	1.134(1.012-1.285)

DPN: Diabetic peripheral neuropathy, S.E.: Standart Error, OR: Odds Ratio, *CI:Confidence interval RDW: Red Blood Cell Distribution Width, RAR: RDW/Albumin ratio*, *HT: Hypertension*

indicating a 28.4% increase in odds per additional year of diabetes. HbA1c was also associated with higher odds of DPN (OR: 1.134, 95% CI: 1.012–1.285, p=0.047) (Table 3).

We utilized ROC curve analysis to evaluate the predictive ability of RAR for the presence of DPN in patients with diabetes and to determine the optimal cutoff value. The optimal cutoff value for RAR to predict DPN development was found to be 4.4 %/(g/dL). (Specificity: 99.2%, Sensitivity: 32.2%, AUC: 0.67). This suggests that the RAR may serve as a useful discriminator for the development of DPN. Furthermore, at a cutoff value of RAR ≥ 4.4, a high specificity in minimizing false positives was demonstrated, reflected by a specificity of 99.2%. (Figure 2)

DISCUSSION

This study investigated the association between RAR levels and the development of polyneuropathy in patients with diabetes. In diabetic patients, higher RAR levels, longer duration of diabetes, and higher HbA1c values were found to be associated with the development of DPN. Our findings suggest that the RDW/albumin ratio may

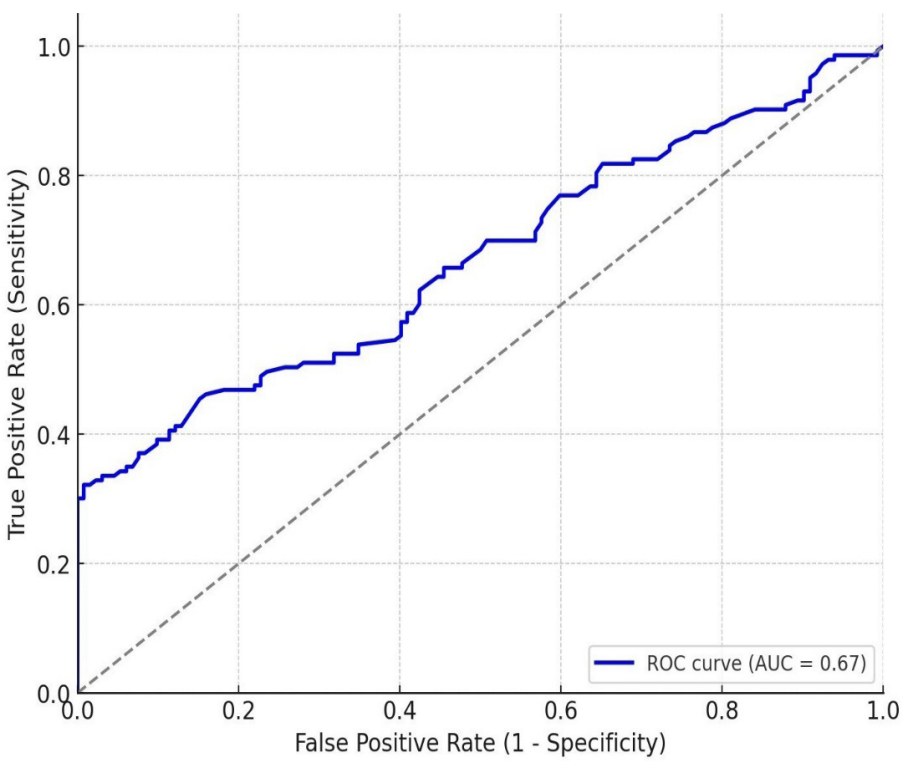

Figure 2. ROC curve analysis of the predictive value of RAR for the development of DPN

serve as a complementary biomarker compared with RDW or albumin levels alone for identifying the presence of DPN.

DPN is a chronic complication of diabetes characterized by involvement of the peripheral nervous system, which leads to a significant reduction in patients' quality of life.[11] In diabetic patients, the cumulative effect of long-term hyperglycemic exposure, hypertension, dyslipidemia, and smoking plays an important role in the development of microvascular complications such as polyneuropathy.[12] Recently, Wang *et al*., in a cross-sectional study involving a large population of patients with type 2 diabetes, demonstrated positive associations between DPN and both hypertension and diabetes duration.[13] Another study demonstrated that diabetic patients with polyneuropathy had higher systolic blood pressure levels and experienced more challenging blood pressure management, independent of a diagnosis of hypertension.[14] Smoking has previously been associated with an increased risk of developing type 2 diabetes, reduced metabolic control, and increased insulin resistance.[15] A meta-analysis involving thirty-eight prospective cohort studies reported an unadjusted odds ratio (OR) of 1.26 for the association between smoking and the development of DPN.[16] In contrast to these studies, Callaghan *et al*. did not find a significant association between the development of neuropathy and either hypertension or smoking.[17] In our study, hypertension and smoking were associated with the development of DPN in the initial analysis, but this association lost statistical significance after adjusting for other variables in the multivariate analysis. We suggest that the conflicting results observed in the literature may be attributable to variations in sample sizes, patient profiles, and methodological differences across studies. Furthermore, factors such as the duration of hypertension exposure, as well as smoking intensity and duration, could also have influenced the outcomes.

In our study, there was a positive correlation between longer diabetes duration and the development of DPN. After adjusting for all other potential factors in the logistic regression analysis, diabetes duration was identified as an independent risk factor for the development of DPN. Studies have consistently found diabetes duration to be an independent risk factor for DPN development, irrespective of age, even across diverse populations.[18] However, in the long term, a significant reduction in DPN prevalence has been observed in patients with tight metabolic control compared to those with poor glycemic control.[19] When evaluating the impact of diabetes duration, it is important to consider that achieving tight metabolic control can reduce the risk of DPN for both type 1 and type 2 diabetes.

The occurrence of DPN in both types of diabetes, and the observation that glycemic control slows neuropathy development particularly in type 1 diabetes, highlights the central role of hyperglycemia in its pathophysiology.[20] Glycated hemoglobin, or HbA1c, reflects the interaction between blood glucose concentration and erythrocyte lifespan and is the primary biomarker used to assess long-term glycemic control in individuals with diabetes.[21] Recently, in a retrospective cohort study, Cheng *et al.* demonstrated that diabetic patients with HbA1c levels > 7.7% had 3.15 times higher risk of developing polyneuropathy.[22] In our study, both fasting blood glucose and HbA1c levels were significantly higher in diabetic patients with DPN compared to those without DPN. Our regression analysis demonstrated that high HbA1c was identified as an independent predictor of DPN. Furthermore, no significant differences were observed between the groups regarding BMI and lipid parameters. While some studies have associated dyslipidemia and BMI with the risk of DPN[23], a meta-analysis involving 16 studies found no such association, consistent with our results.[24] It has been suggested that these conflicting findings may be attributable to population heterogeneity, ethnic differences, and variations in the prevalence of statin use across study periods or populations.

RDW reflects the size and heterogeneity of red blood cells in the circulation. Inflammatory and oxidative processes may lead to endothelial dysfunction, erythropoietin resistance, and reduced erythrocyte lifespan, thereby increasing RDW levels.[25] Albumin, a negative acute-phase reactant synthesized by the liver, has antioxidant and anticoagulant properties and also reflects nutritional status. Type 2 diabetes mellitus is characterized by chronic inflammation, and the resulting erythrocyte dysfunction and oxidative imbalance may influence both RDW and albumin levels.[26]

Accordingly, the RDW-to-albumin ratio (RAR) has been proposed as a composite biomarker that may better reflect these complex pathological processes than either parameter alone. In a large population-based study, RAR demonstrated superior predictive performance compared with RDW alone for diabetes prevalence and long-term

prognosis.[25] Similarly, Zhao *et al*. reported that an RAR value ≥2.9 was independently associated with diabetic retinopathy.[27]

In the present study, RDW and albumin alone were not independently associated with diabetic peripheral neuropathy in multivariate analysis; however, RAR emerged as an independent predictor. ROC analysis showed that an RAR cutoff value of 4.4 provided moderate discriminatory ability (AUC=0.67). Although this cutoff demonstrated limited sensitivity, its high specificity suggests potential utility for risk stratification and diagnostic support rather than for population-based screening. Lower cutoff values may improve sensitivity at the cost of increased false-positive rates. Given the clinical and pathophysiological differences between diabetic peripheral neuropathy and other microvascular complications, such as diabetic retinopathy, optimal cutoff values may vary according to the intended clinical application.

Consistent with previous reports showing weaker associations between RDW and neuropathy compared with other diabetic complications[28,29], our findings suggest that RAR may serve as a complementary biomarker reflecting the chronic inflammatory milieu associated with diabetic peripheral neuropathy. Nevertheless, further prospective studies are required to clarify its role in early diagnosis and risk assessment.

Several limitations of our study should be acknowledged. Firstly, the retrospective design limited our ability to control for all potential confounding variables and precludes the establishment of definitive causal relationships. Secondly, the relatively small sample size, the cross-sectional nature of the analysis, and the recruitment from a single center (leading to a potentially homogeneous study group) may limit the generalizability of our findings. Consequently, further studies with larger sample sizes, employing multi-center designs, and including diverse ethnic populations are warranted to confirm these results and enhance their generalizability. Thirdly, while our analyses demonstrated an association between DPN and RAR, the level of significance was not sufficiently strong to draw definitive conclusions, and the underlying mechanisms could not be fully elucidated. We believe that well-designed, large-scale, prospective cohort studies are necessary to further clarify the association between RAR and the risk of DPN development and to investigate the underlying causal mechanisms.

In conclusion, our findings suggest that RAR is an independent predictor of DPN and may serve as a more sensitive biomarker than assessing RDW or albumin levels alone. Additionally, diabetes duration and high HbA1c levels were confirmed as independent predictors of DPN. However, considering the limitations of our study, further prospective, multi-center studies with larger sample sizes are needed to more robustly establish the association between RAR and DPN.

DISCLOSURE

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Conflict of interest: None

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