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To analyse the correlation between UAER and eGFR and the risk factors for reducing eGFR in patients with type 2 diabetes

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Abstract

Objective To analyse the correlation between urinary albumin excretion rate (UAER) and estimated glomerular filtration rate (eGFR) and the risk factors for reducing eGFR in patients with type 2 diabetes mellitus (T2DM).

Methods A total of 431 T2DM patients admitted between January 2019 and March 2020 were selected and divided into two groups according to eGFR level. Comparing the differences between baseline data and clinical indicators, multivariate logistic regression was used to analyse the risk factors of eGFR reduction and to analyse the association between UAER and eGFR.

Results In total, 167 patients were included in the study group and 264 patients were included in the conventional group. The study group participants were older, had longer diabetes duration, and had higher fatty liver, peripheral vascular disease (PVD), hypertension prevalence, and mean body mass index ($P < 0.05$). The levels of various indicators were lower than those of the conventional group ($P < 0.05$). Additionally, PVD, nocturnal systolic blood pressure, fatty liver, and beta-2-microglobulin (β 2-MG) were independent risk factors for eGFR decline, with high density lipoprotein (HDL) and fasting C-peptide (CP) as protective factors. There was no obvious correlation between UAER and eGFR.

Conclusion Peripheral vascular disease, systolic blood pressure, fatty liver, and beta-2-microglobulin are risk factors for decreased eGFR levels in patients with T2DM, which should be applied for control DKD. HDL and fasting CP have important effects on maintaining eGFR, and blood pressure and fasting CP can be used as new targets for subsequent diabetic kidney disease treatment.

Keywords Diabetic kidney disease, Urinary albumin excretion rate, Esitimated glomerular filtration rate, Association, Risk factors

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Introduction

Diabetic kidney disease (DKD) is a common and serious complication in patients with diabetes and one of the main causes of end-stage renal disease. With the increasing global prevalence of diabetes, the incidence of DKD is also increasing rapidly, placing heavy health and economic pressures on patients. The occurrence of DKD is closely related to many factors, including hyperglycaemia, hypertension, and dyslipidaemia, which together contribute to the progressive decline of glomerular filtration function [1].

Glomerular filtration rate (GFR) is an important indicator for evaluating renal function, and a decline in this rate indicates a decline in renal function. The estimated GF rate (eGFR) is one method commonly used in clinical evaluation. Urinary albumin excretion rate (UAER) is an important indicator of the early detection of DKD that reflects the degree of kidney injury and its prognosis [2]. However, the relationship between UAER and eGFR, as well as the specific risk factors affecting eGFR decline are inconsistent [3]. In addition, to date, many studies have focused on the effect of a single factor on renal function and, accordingly, lack comprehensive and systematic multivariate analysis [4].

In recent years, research has shown that factors such as fatty liver, peripheral vascular disease (PVD), hypertension, and dyslipidaemia may be associated with the deterioration of renal function in patients with DKD. For example, fatty liver is closely associated with insulin resistance and inflammatory response, which may accelerate the deterioration of renal function. Furthermore, PVD may adversely affect the kidneys [5] through its effect on blood circulation throughout the body. Moreover, hypertension and dyslipidaemia have been recognised as key risk factors for the progression of DKD that lead to glomerulosclerosis and a loss of kidney function [6, 7] through multiple pathways. Diabetic kidney disease [8] can occur in nearly 30% of patients with type 2 diabetes mellitus (T2DM). Existing data investigations show that the two main factors inducing DKD are hyperglycaemia and hemodynamic abnormalities [9].

This study aimed to explore the association between UAER and eGFR by analysing the clinical data of patients with DKD and identifying independent risk factors affecting eGFR decline using multivariate logistic regression analysis. Specifically, this study analyses the effects of factors such as age, diabetes duration, fatty liver, PVD, hypertension, and body mass index (BMI) on eGFR. These analyses will aim to provide a scientific basis for the early diagnosis and precision treatment of patients with DKD in a bid to improve patient outcomes and reduce the incidence of DKD-related complications.

Data and methods

General information

A total of 431 patients with T2DM were enrolled from January 2019 to March 2020 and evaluated for the study. Inclusion criteria: ① patients meeting the diagnostic criteria of diabetes in the China T2DM Prevention Guidelines 2020 edition; ② patients between 18 and 75 years old with complete medical records; ③ patients with normal kidney function based on an eGFR of ≥60 mL/min/1.73 m^2, which is indicative of stage 1 or 2 chronic kidney disease; ④ patients with no blood system or mental disease, and who presented good communication abilities; ⑤ patients who could complete the study independently, showed high enthusiasm for treatment, and cooperated with the physician's treatment. Exclusion criteria: ① patients with other severe renal diseases (e.g. nephritis, nephrotic syndrome); ② patients who had recently received medication affecting their renal function (e.g. non-steroidal anti-inflammatory drugs, certain antibiotics); ③ patients with severe cardiovascular, liver, or other systemic diseases; ④ women who were pregnant or lactating; ⑤ patients who could not complete study follow-up or lacked complete medical records.

Sample size calculation

The sample size was calculated based on a power analysis to detect a clinically significant difference in eGFR between the two groups, with a significance level of 0.05 and power of 80%. To account for potential dropouts, we increased the sample size to 431 participants. A post-hoc power analysis was conducted to ensure the adequacy of the sample size. The power to detect differences in primary outcomes was 80%, indicating that the study had sufficient statistical efficacy to draw valid conclusions.

Methods

Group method

Cockcroft-Gault formula was used to calculate the GFR [10, 11]. For male patients, the formula was as follows: eGFR = (140 - age) weight (kg) / (0. 818 creatinine (μ mol / l), and the female formula was as follows: eGFR= [(140 - age) weight (kg) / (0. 818 creatinine (μ mol / l))] 0.85. The patients were divided into two groups according to the eGFR level: < eGFR 90 ml/min for the study group and eGFR > 90 ml/min for the conventional group.

Index detection

(1) Basic information about the patients was collected for each group, including age, duration of diabetes, BMI, admission blood pressure, peripheral vascular disease, hypertension, fatty liver, and other conditions. (2) Venous blood samples were collected from the patients during a fasting state in the morning and blood samples for blood routine measurement, glycosylated haemoglobin,

biochemistry, 5 ml each of morning urine and fasting venous blood, detection, and analysis of biochemical indicators using an automated biochemical analyser. Fasting insulin (FINS), 2 h FINs, fasting C-peptide (FCP), 2 h C-peptide (CP) were detected by enhanced chemiluminescence immunoassay, and patients were collected for 24 h urine and calculated their UAER. According to the UAER level, the results were divided into normal (<30 mg/24 h), micro (30–299 mg/24 h), and large (300 mg/24 h) amounts. The UAER was measured three times for each participant over a 24-h period. The average of these three measurements was used as the final UAER value for analysis to ensure accuracy and reduce variability.

Observed indicators

The following indicators were collected and analyzed: (1) the difference in baseline data between the study and conventional groups; (2) the difference in clinical indicators between the study and conventional groups; (3) the risk factors of eGFR decline in patients with DKD using logistic regression analysis; (4) correlation between UAER and eGFR using Spearman and Pearson analyses; (5) logistic univariate and multivariate regression analysis between UAER and eGFR.

Statistical methods

All statistical tests were two-sided and $P<0.05$ was considered to be statistically significant. The SPSS 24.0 statistical software (IBM, Armonk, NY, USA) were used to conduct the analyses. Measurement data of normal distribution are described as mean±standard deviation (mean±SD); Group comparisons were performed using the independent sample t-test. Non-normal data are described as median and interquartile spacing (M [Q1, Q3)]); The Mann-Whitney U rank sum test was used for comparison between groups; Count data are described in cases and composition ratios (n, [%]). Chi-square test or Fisher's exact probability method. The correlation between UAER and eGFR was analyzed using the Person and spearman methods, and the risk factors for decreased eGFR were analysed by multivariate logistic regression.

Table 1 Basic characteristics of the population[($\bar{x}\pm s$), n(%)]

variant	description($n=431$)
age/years, (Mean ± SD	55.96 ± 12.39
male/female	261/170
Duration of T2DM (years, Mean ± SD)	7.16 ± 2.32
BMI, Mean ± SD	21.86 ± 4.50
eGFR (mL/min/1.73 m^2, Mean ± SD)	130.47 ± 49.36
Peripheral vascular disease, n(%)	316(73.32)
Peripheral neuropathy, n(%)	201(46.64)
Diabetic retinopathy, n(%)	83(19.26)
hypertention, n(%)	225(52.20)
Coronary heart disease, n(%)	59(13.70)
Fatty liver, n(%)	280(64.97)
Insulin medication history, n(%)	186(43.1%)

Note: T2DM, Type 2 Diabetes Mellitus; eGFR, estimated glomerularfiltration rate

Results

General information about the cohort

The study included a total of 431 patients diagnosed with T2DM, with a mean age of 55.96±12.39 years. Of the cohort, 261 were male and 170 were female, with a mean BMI of 21.86±4.50 kg/m^2. The duration of diabetes ranged from 1 to 20 years, with a mean disease duration of 7.16±2.32 years. The mean eGFR for all participants was 130.47±49.36. Among the patients, 225 had hypertension, 83 presented with diabetic retinopathy, 59 had coronary heart disease, 280 were diagnosed with fatty liver, and 316 had PVD. A total of 186 patients had a history of insulin therapy (Table 1).

Analysing the risk factors for eGFR decline in patients with DKD

Baseline data comparison of the difference between the study and conventional groups

The age, proportion of peripheral vascular disease, proportion of hypertension, proportion of fatty liver, BMI, and the course of disease in the study group were significantly higher compared with the conventional group, and the differences between groups were statistically significant ($P<0.05$) (see Table 2).

Comparison of the difference in clinical indicators between the study and conventional groups

The levels of red blood cells, haemoglobin, HbA1c, fasting blood glucose (FBG), triglyceride (TG), low density lipoprotein (LDL), systolic blood pressure (SBP), serum creatinine (Scr), Apoliprotein A1 (APoA 1), beta-2-microglobulin (β 2-MG), and 2-h postprandial blood glucose (2-h PBG) in the study group were significantly higher than in the study group; APoB, 2 hINS, HDL, FINS, FCP, and 2-h CP in the study group were significantly lower than in the conventional group, and the data between the groups were statistically significant ($P<0.05$) (see Table 3).

Logistic regression analysis of the risk factors for eGFR decline in patients with DKD

Study factors were adjusted for age, sex, BMI, peripheral vascular disease, hypertension, coronary heart disease, fatty liver, and disease duration. The results that corresponded to $P<0.01$ after the disease course was analysed for the multivariate logistic regression model group, and the results showed that PVD, systolic blood pressure, fatty liver, and beta-2-microglobulin were independent

Table 2 Baseline data differences between the study group and the conventional group [($\bar{x} \pm s$), n(%)]

factors	Conventional group	Study group	t/x2	*P*
age	50.61 ± 10.79	64.41 ± 9.77	-13.42	< 0.001
male/female(n/n)	164/100	97/70	0.698	0.403
BMI, Mean ± SD	19.81 ± 2.26	23.15 ± 5.05	-9.369	< 0.001
Duration of T2DM (years, Mean ± SD)	5.16 ± 1.02	8.16 ± 1.32	-26.489	< 0.001
Peripheral vascular disease, n(%)	171(64.77)	145(86.83)	25.432	< 0.001
Peripheral neuropathy, n(%)	118(44.70)	83(49.70)	1.029	0.31
Diabetic nephropathy, n(%)	46(17.42)	37(22.16)	1.473	0.225
hypertention, n(%)	122(46.21)	103(61.68)	9.804	0.002
Fatty liver, n(%)	121(45.45)	102(61.08)	9.52	0.002

Note: T2DM, Type 2 Diabetes Mellitus; $P < 0.05$, with a statistically significant difference

risk factors for eGFR decline in patients with DKD, while HDL and FCP were protective factors for eGFR decline in patients with DKD (see Table 4).

Analysis of the relationship between UAER and eGFR

The relationship between UAER and eGFR was analysed by Spearman correlation and Pearson correlation. The results showed no obvious correlation between UAER and eGFR ($P > 0.05$). The results of univariate analysis showed that the UAER level did not influence the change in eGFR level; however, logistic multivariate analysis conducted after adjusting for age and sex showed that the UAER level did not influence a change in eGFR level. The results of multivariate logistic analysis after adjusting for age, gender, BMI, peripheral vascular disease, hypertension, coronary heart disease, fatty liver, and disease course factors showed that UAER was not an influencing factor of eGFR (see Tables 5 and 6).

Discussion

Patients with DKD often develop kidney injuries, and eGFR is a good indicator for assessing kidney function and kidney injury. When kidney injuries, eGFR will be reduced [12] accordingly. The concentration of haemoglobin (red blood cells) is correlated with blood rheology. The concentration of both haemolobin and blood rhelogy increases substantially, the blood viscosity will increase accordingly, which will slow down the speed of blood flow, greatly increase the risk of thrombosis, affect the blood flow of patients and increase the risk of thrombosis. Therefore, in patients with DKD, the risk of atherosclerosis and thrombosis is significantly higher than in those with NDKD, and the risk of coronary heart disease, peripheral vascular disease, and hypertension is higher [13]. The age, peripheral vascular disease, hypertension, diabetes, BMI, fatty liver, duration of the disease were significantly higher in the study group compared with the conventional group, and the differences in the data of the two groups were statistically significant ($P < 0.05$). Consistent with the conclusions of the above study [13], with age, physical decline and prolonged disease duration led to further development of the disease, so the group with a higher eGFR reduction in DKD patients had a higher mean age and disease duration, which is consistent with the findings of this study. As a result, among the patients with DKD, the group with an eGFR reduction had a higher mean age and disease duration, which is consistent with the results of the current study.

The results of this study showed that the levels of RBC, haemoglobin, HbA1c, FBG, TG, LDL, SBP, Scr, APoA 1, beta-2-microglobulin, and 2-h PBG in the study group were significantly higher than those of the conventional group; APoB, HDL, FINS, FCP, and 2-h CP levels in the study group were significantly lower than those in the conventional group. The differences between the groups were statistically significant ($P < 0.05$). The results of the multivariate analysis showed that PVD, systolic blood pressure, fatty liver, and beta-2-microglobulin were independent risk factors for eGFR decline in patients with DKD, while HDL and FCP were protective factors for eGFR decrease in patients with DKD. In the disease development process of patients with DKD, blood rheology is important. This is primarily manifested as blood hypercoagulability and a slow blood flow rate, and these conditions could lead to the formation of a microthrombus [13, 14]. A common complication among patients with DKD is hyperlipidaemia, which can cause atherosclerosis and abnormal changes in GFR, increase the risk of fatty liver and peripheral vascular disease, and, as a result, aggravate kidney injuries and the overall condition of patients with DKD, leading to a reduction in eGFR level [15]. Huang et al. [16] found that the HDL of patients with early stage DKD was significantly different compared with T2DM patients in the clinical stage and suggested that metabolic disorders could aggravate the problem of lipid peroxide accumulation to some extent; this could subsequently affect the integrity of endothelial cells, leading to further damage to these cells and their function. In addition, the large-scale deposition of lipid peroxide will seriously damage glomerular mesangial cells, affecting the movement and contraction of cells, and aggravate kidney damage in patients, leading

Table 3 Differences in clinical indicators between the study group and the conventional group [($\bar{x} \pm s$), n(%)]

group	Red cell	hemoglobin(g/dl)		HbA1c(%)	2hPBG(mmol/L)	FINS(μU/mL)	
Conventional group	4.15 ± 0.49	133.94 ± 14.83		8.18 ± 2.02	17.71 ± 1.71	8.65 ± 4.02	
Study group	4.80 ± 0.69	143.31 ± 16.84		9.72 ± 1.93	19.62 ± 1.03	7.45 ± 4.12	
t	-11.419	-6.060		-7.844	-13.015	2.990	
P	< 0.001	< 0.001		< 0.001	< 0.001	0.003	
group	2hINS(μU/mL)	FCP(ng·ml-1)		2hCP(ng·ml-1)	FBG(mmol/L)	UAER	
Conventional group	10.65 ± 4.12	2.29 ± 0.92		6.13 ± 2.12	7.71 ± 1.79	15.00 ± 9.35	
Study group	8.35 ± 4.21	1.98 ± 0.72		5.61 ± 2.12	12.62 ± 2.02	14.50 ± 9.32	
t	5.599	3.696		-2.481	-26.382	0.542	
P	< 0.001	< 0.001		0.013	< 0.001	0.588	
group	UAERgroup, n(%)			SBP(mmHg)	Serum creatinine(μmol/L)	TC(mmol·L-1)	
	normal	micro	macro				
Conventional group	203(76.89)	54(20.45)	7(2.65)	138.30 ± 5.80	58.30 ± 10.80	4.59 ± 1.27	
Study group	124(74.25)	34(20.36)	9(5.39)	145.30 ± 5.80	72.10 ± 9.32	4.42 ± 1.20	
t/x2	0.390			-12.207	-13.613	1.383	
P	0.532			< 0.001	< 0.001	0.167	
group	TG(mmol·L-1)	LDL(mmol·L-1)		HDL(mmol·L-1)	ApoA1(g/L)	ApoB(g/L)	β2-MG(mg·L-1)
Conventional group	1.28 ± 0.69	2.16 ± 0.69		1.17 ± 0.32	1.40 ± 0.12	0.99 ± 0.32	2.49 ± 0.42
Study group	1.86 ± 0.86	2.52 ± 0.63		1.03 ± 0.24	1.47 ± 0.22	0.91 ± 0.12	2.95 ± 0.54
t	-7.715	5.455		4.855	-4.265	3.095	-9.897
P	< 0.001	< 0.001		< 0.001	< 0.001	< 0.001	< 0.001

Note: $P<0.05$, with a statistically significant difference

Table 4 Logistic regression analysis of risk factors for decreased EGFR in diabetic nephropathy patients

Features	β	S.E	Wald	*P*	OR	95%CI	
						lower	upper
Intercept	-4.1133	1.0125	16.5041	< 0.001	-	-	-
PVD	0.8220	0.3205	6.5768	0.010	2.275	1.214	4.264
HDL	-0.9522	0.2604	13.3707	< 0.001	0.386	0.232	0.643
Fatty Liver	2.0564	0.4805	18.3154	< 0.001	7.818	3.048	20.050
β2-MG	1.3823	0.2186	39.9703	< 0.001	3.984	2.595	6.115
FCP	-0.2836	0.1057	7.2030	0.007	0.753	0.612	0.926
Systolic pressure	1.4531	0.2233	23.3812	< 0.001	4.398	3.272	7.723

Note: PVD, peripheral vascular disease; HDL, high density lipoprotein; β 2-MG, beta-2-microglobulin; FCP, fasting C-peptide

Table 5 Rank correlation test between UAER and eGFR

Variable	spearman		pearson	
	r	*P*	*r*	*P*
UAER	0.042	0.378	-0.006	0.902

Note: UAER, Urinary Albumin Excretion Rate

to a reduction in eGFR [17]. Yu Junnan [18] confirmed that changes in blood routine and renal function index would reduce eGFR in patients with DKD. In this study, the APoA1 level in the study group was higher than in the conventional group, while the APoB level was lower than compared with the conventional group; these results are inconsistent with the results of existing studies. Additional data obtained from Sarahi Nunez [19] showed that CP, as one of the active substances has different physiological effects from that of insulin. Fasting CP is a protective factor for reduced renal function after PCI; here, the mechanism of action may be that CP can reduce renal albumin filtration rate and reduce its glomerular hyperperfusion status. In the glomerular system, CP and insulin-driven glucose metabolism-related protein play an important role in promoting eGFR to restore normal level, another glomerular microvascular PKC-B, RAGE expression level by CP level to some extent, and CP of PKA expression, and make the urinary protein secretion level in a certain extent, indirectly avoid the accumulation of glomerular extracellular matrix, effectively reduce glomerular hypertrophy, glomerular membrane expansion problems, to improve renal function. Li et al. [20] A double-blind clinical study combining insulin and CP in patients with diabetes found that after 4 weeks of administration, the eGFR decreased by 6% in the patient group with type 1 diabetes but remained unchanged in the control group. Three months after administration, the eGFR decreased by 6% in the group of patients with type 1 diabetes, while remaining unchanged in the control group, confirming that CP could reduce the renal albumin filtration rate and reduce its hyperperfusion status. In the glomerular system, CP combined with insulin-driven proteins, and related to glucose metabolism, is important for preventing the development of DKD and for promoting the restoration of normal levels of eGFR.

The analysis of beta-2-microglobulin as an independent risk factor for decreased renal function after PCI in patients with CHD may indicate that lymphocytes, platelets, and multinucleated leukocytes are the main secretory sources of beta-2-microglobulin. The secretion and synthesis of β-2 microglobulin are in dynamic equilibrium. Secreted beta-2-microglobulin can freely pass in the glomeruli in the state of normal renal function. With normal renal function, most of the beta-2-microglobulin is absorbed by the proximal renal tubules during this process of dynamic equilibrium. Thus, the exclusion rate is low. If the amount of beta-2-microglobulin in serum exceeds the normal level, decreased GF function is indicated. If the amount of beta-2-microglobulin in the urine exceeds the standard level, renal tubular injury is indicated, leading to decreased renal function and a lower eGFR. Fu Mei et al. [21] showed that the serum beta-2-microglobulin in cases of nephrotic syndrome was significantly higher compared with healthy patients, and beta-2-microglobulin showed a significant negative correlation with renal function indicators. Mei et al. states thddonat there is a close correlation between

Table 6 Logistic regression analysis of UAER and eGFR

Variable	Univariate analysis		Multivariate analysis*1		Multivariate analysis *2	
	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
UAER						
normal	Ref		Ref		Ref	
Micro	1.031(0.635–1.672)	0.902	0.846(0.459–1.562)	0.594	1.466(0.705–3.046)	0.306
Macro	2.105(0.765–5.794)	0.150	2.607(0.742–9.157)	0.134	4.056(0.848–19.392)	0.079

Note: UAER, Urinary Albumin Excretion Rate. * 1 to adjust age and sex; * 2 to adjust age, sex, BMI, peripheral vascular disease, hypertension, coronary heart disease, fatty liver, course of disease

serum beta-2-microglobulin and renal function indicators, which can reflect the disease state of patients with nephrotic syndrome to some extent. This result is consistent with the outcomes of the present study.

Long-term hypertension will lead to a change in blood flow in the kidneys, leading to a reduction in the resistance of glomerular vessels, as well as abnormally increased blood flow, leading to a long-term high pulse pressure in glomeruli, eventually damaging the renal vascular endothelial function and glomerulus, causing renal function damage and reducing the eGFR level. Wang Pu et al. [22] showed that the decline of renal function in elderly postural hypertension was more obvious, which is consistent with the results of this study. The results of this study provide new insights into the correlation between UAER and eGFR in patients with DKD, as well as the risk factors affecting a decline in eGFR.

This study still has some limitations. ① Limited sample size: Although 431 patients were included in this study, the sample size was relatively small, which may have affected the generalisability and representativeness of the results. A larger study could help to further validate the findings of this study. ② Single-centre study: This study was conducted in a single medical centre and may include a selection bias. A multicentre study will help to improve the external validity and universality of the results. ③ Cross-sectional study design: This study adopted a cross-sectional design, which can only reveal the correlation between variables and cannot determine causal relationships. The cross-sectional nature of the data limited our ability to assess long-term outcomes or construct predictive models. Additionally, the lack of longitudinal follow-up limited our ability to determine the long-term impact of these predictors on patient outcomes. Future studies should focus on collecting more detailed data over a longer period to clarify the causal relationship between UAER and eGFR changes. ④ The applied eGFR formula: In this study, the C-aGFR4 was used to estimate eGFR. Although this formula has been verified in the Chinese population, different formulas can be adjusted and verified within different populations.

Conclusion

In conclusion, PVD, systolic blood pressure, fatty liver, and beta-2-microglobulin are independent risk factors for eGFR decline in patients with DKD. In subsequent clinical treatment, PVD, blood pressure, beta-2-microglobulin, peripheral vascular disease, and fatty liver disease should be a focus of prevention. Furthermore, HDL and FCP are protective factors for normal GF rate in patients with DKD, and blood pressure and FCP can be used as new targets for the subsequent treatment of DKD.

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Author contributions
Li HH conceived and designed the study. Han LW and Gao X collected the data and helped the date analysis and statistics. All authors took part in drafting the manuscript. All authors read and approved the final manuscript.

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Data availability
All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Beijing Chao-Yang Hospital, Capital Medical University, and all participants gave informed consent and signed.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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