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Original Article

Predictive value of lipid levels in coronary heart disease in elderly hypertensive patients

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Abstract: Objective: To evaluate the predictive value of lipid levels for coronary heart disease (CHD) risk in elderly hypertensive patients and to establish a prediction model. Methods: Data from 428 elderly hypertensive patients attending the First Affiliated Hospital of Anhui Medical University between January 2021 and December 2023 were retrospectively collected. Patients were categorized into CHD and non-CHD groups based on the presence of comorbid CHD. Risk factors were identified using logistic regression, and a clinical prediction model was constructed. Model discrimination and calibration were assessed using receiver operating characteristic (ROC) curves and the Hosmer-Lemeshow test. Decision curve analysis (DCA) was used to assess the clinical application value of the model. Results: Advanced age, smoking, hypertension duration >10 years, and abnormal total cholesterol (TC) were independently associated with an increased risk of CHD in elderly hypertensive patients. In addition, there was a trend linking abnormalities in low-density lipoprotein cholesterol (LDL-C) and lipoprotein(a) [Lp(a)] with higher CHD risk in this population. The developed clinical prediction model showed good discrimination (AUC=0.71) and calibration (P=0.907). The model's accuracy, sensitivity, specificity, positive predictive value, and negative predictive value were 0.69, 0.72, 0.60, 0.82, and 0.46, respectively. Conclusion: Abnormal lipid levels are independent predictors of increased CHD risk in elderly hypertensive patients. The prediction model developed in this study holds clinical value in assessing CHD risk, enabling early identification of high-risk patients and the development of individualized preventive strategies.

Keywords: Coronary atherosclerotic heart disease, lipid levels, hypertension, predictive value

Introduction

Coronary atherosclerotic heart disease (CHD), a typical form of ischemic heart disease, is one of the leading causes of death and disability worldwide [1]. CHD is prevalent in middle-aged and elderly populations, with high morbidity and mortality rates. Previous studies have estimated 197 million cases of CHD globally, with 9.1 million deaths [2]. Hypertension is a critical risk factor for CHD, not only accelerating atherosclerosis but also increasing the risk of cardiovascular events [3]. As the economy develops and the population ages, the incidence of CHD in elderly hypertensive patients continues to rise, posing a significant threat to public health [4]. Abnormal blood lipid levels play a pivotal role in the pathogenesis of atherosclerosis, with a strong correlation between lipid disturbances and the development of CHD [5]. This study aims to evaluate the predictive potential of lipid levels for the risk of combined coronary atherosclerosis in elderly individuals with hypertension, providing a scientific basis for clinical prevention and treatment.

The principal pathological mechanism of CHD is coronary atherosclerosis, which develops through a multifactorial, multistep process [6]. This process is characterized by lipid metabolism abnormalities, endothelial dysfunction, inflammation, thrombosis, and other contributing factors [7]. Hypertension, an important risk factor for CHD, not only causes endothelial damage by increasing the impact of blood flow on the vascular wall, but also accelerates atherosclerosis through inflammatory responses, oxidative stress, and other mechanisms [8]. Lipid levels are key indicators of lipid metabolism, and numerous studies have confirmed

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that dyslipidemia is a significant factor in the development of CHD [9]. Hypercholesterolemia leads to lipid deposition in arterial walls and the formation of atherosclerotic plaques, which can cause lumen stenosis, myocardial ischemia, and even myocardial infarction [10]. The elderly often suffer from multiple chronic conditions, such as hypertension and diabetes, which exacerbate the risk of coronary atherosclerosis. Therefore, predicting the risk of combined coronary atherosclerosis in elderly hypertensive populations based on lipid levels is crucial for early identification of high-risk patients and for developing personalized preventive and therapeutic strategies.

In recent years, significant progress has been made in understanding the relationship between lipid levels and coronary atherosclerosis. Epidemiological studies have established that lipid levels are positively correlated with the incidence of CHD, with elevated total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) being key risk factors for CHD [11]. Conversely, lower levels of high-density lipoprotein cholesterol (HDL-C), a protective factor against atherosclerosis, also increase CHD risk [12]. Regarding hypertension's relationship with coronary atherosclerosis, studies have shown that the incidence of atherosclerosis is significantly higher in hypertensive individuals than in those with normal blood pressure [3]. Furthermore, hypertension and dyslipidemia often coexist, and their interaction further increases the risk of coronary atherosclerosis [13]. However, research on predicting the risk of combined CHD in elderly hypertensive populations based on lipid levels remains insufficient, particularly in terms of prediction accuracy across different age groups, genders, and lipid levels. More in-depth investigations are required.

Given the high prevalence of hypertension and dyslipidemia in the elderly and their impact on CHD risk, this study aims to explore the potential for predicting secondary CHD in elderly hypertensive patients based on lipid levels. A retrospective design will be employed to analyze the relationship between lipid levels and coronary atherosclerosis, develop a prediction model, and evaluate its predictive accuracy using data on demographics, lipid levels, and blood pressure of elderly hypertensive patients. It is anticipated that this study will clarify the role of lipid levels in predicting CHD risk in elderly hypertensive populations and provide a novel clinical assessment tool. Additionally, the findings are expected to offer a scientific foundation for future cardiovascular disease prevention strategies, particularly in co-managing hypertension and dyslipidemia.

Materials and methods

Study subjects

This study employed a retrospective design to collect data from elderly hypertensive patients who were examined and treated at The First Affiliated Hospital of Anhui Medical University between January 2021 and December 2023. Data from 428 hypertensive patients were retrieved using the hospital's electronic medical record system, with exclusions made for those who did not meet the inclusion criteria. These patients were divided into two groups: those with CHD and those without (non-CHD). The inclusion criteria for this study were as follows: (a) patients diagnosed with hypertension according to the Chinese Guidelines for Hypertension Prevention and Treatment [14]; (b) patients aged ≥65 years; (c) patients with complete clinical data. The exclusion criteria were: (a) incomplete basic information or medical records; (b) patients experiencing hypertensive crises; (c) patients with severe comorbid conditions such as tumors, autoimmune diseases, or organ failure; (d) patients with mental illness, psychological disorders, or cognitive impairment. This study was approved by the Ethics Committee of The First Affiliated Hospital of Anhui Medical University.

Data collection

Patient information was obtained from the hospital's medical record system and on-site inquiries. The collected data included both general and clinical information. General data comprised age, sex, body mass index (BMI), smoking habits, alcohol consumption, exercise duration, family history of CHD, family history of hypertension, duration of hypertension, presence of diabetes, and hyperuricemia. Clinical data included TC, TG, HDL-C, LDL-C, and Lp-a levels.

Grouping standard

CHD diagnosis was based on typical clinical symptoms, along with evidence of myocardial ischemia or coronary artery obstruction confirmed through electrocardiograms, coronary angiography, or other diagnostic methods [15]. The CHD diagnosis was made jointly by two clinicians. Patients were categorized as follows: the CHD group was assigned a value of 1, and the non-CHD group was assigned a value of 0. Lipid levels were classified into normal or abnormal groups based on the following thresholds:

TC <6.2 mmol/L: normal; ≥6.2 mmol/L: abnormal; TG <2.3 mmol/L: normal; ≥2.3 mmol/L: abnormal; LDL-C <4.1 mmol/L: normal; ≥4.1 mmol/L: abnormal; HDL-C ≥1.0 mmol/L: normal; <1.0 mmol/L: abnormal; Lp(a) <300 mg/L: normal; ≥300 mg/L: abnormal.

Clinical prediction model

Logistic regression analysis was used to identify risk factors for CHD. Variables with a *p*-value <0.05 in univariate logistic regression were included in the multivariate logistic regression model, and the resulting clinical prediction model was presented as a nomogram. The model's discrimination was assessed using receiver operating characteristic (ROC) curves and the area under the curve (AUC). Calibration was evaluated using calibration curves and the Hosmer-Lemeshow test. The accuracy, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were used to assess the model's performance. Additionally, decision curve analysis (DCA) was performed to evaluate the clinical utility of the model.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation, and differences between groups were analyzed using the t-test or Mann-Whitney U test, as appropriate. Categorical variables were described as frequencies and percentages, with differences analyzed using the χ^2 test or Fisher's exact test. Logistic regression models were employed to analyze risk factors for CHD and construct the clinical prediction model. All statistical analyses were performed using SPSS version 27.0. Statistical tests were two-tailed, and a *p*-value <0.05 was considered statistically significant.

Results

Comparison of general information

The mean age of the non-CHD group was 69.77±3.60 years, while the mean age of the CHD group was 71.16±4.29 years (P=0.002) (**Table 1**). There were 85 (27.12%) smokers in the non-CHD group and 56 (45.90%) smokers in the CHD group, and the difference in smoking prevalence between the two groups was statistically significant (P<0.001). The non-CHD group had 137 (44.77%) patients with a hypertension duration of more than 10 years, while the CHD group had 68 (55.74%) such cases, showing a significant difference (P=0.040). The proportion of diabetic patients in the CHD group was significantly higher than in the non-CHD group (P=0.004). No significant differences were observed in other general characteristics between the two groups (all P>0.05).

Comparison of clinical information

In the non-CHD group, 58 (18.95%) patients had abnormal TC levels, compared to 45 (36.89%) patients in the CHD group (P<0.001) (**Table 2**). The proportion of patients with abnormal LDL-C and Lp-a levels was significantly higher in the CHD group than in the non-CHD group (both P<0.05). No significant differences were found in TG and HDL-C levels between the two groups (both P>0.05).

Univariate logistic regression analysis

Univariate Logistic regression showed that older age was significantly associated with an increased risk of secondary CHD (**Table 3**). Additionally, smoking, a hypertension duration of more than 10 years, and diabetes were all associated with a higher risk of CHD. Abnormal TC, LDL-C, and Lp-a levels were also found to be significantly associated with an increased risk of CHD (**Table 3**).

Multivariate logistic regression analysis

Multivariate logistic regression identified old age, smoking, hypertension duration >10 years, diabetes, and abnormal TC as independent risk factors for CHD in hypertensive patients (**Table**

Table 1. Comparison of general information

Variables	Total (n=428)	Non-CHD group (n=306)	CHD group (n=122)	Statistic	*P*
Age, Mean ± SD	70.17±3.85	69.77±3.60	71.16±4.29	t=-3.15	0.002
Gender, n (%)				χ^2=2.00	0.158
Female	103 (24.07)	68 (22.22)	35 (28.69)		
Male	325 (75.93)	238 (77.78)	87 (71.31)		
BMI, n (%)				χ^2=1.00	0.316
<24 kg/m^2	306 (71.50)	223 (72.88)	83 (68.03)		
≥24 kg/m^2	122 (28.50)	83 (27.12)	39 (31.97)		
Smoking, n (%)				χ^2=12.97	<0.001
No	287 (67.06)	221 (72.22)	66 (54.10)		
Yes	141 (32.94)	85 (27.78)	56 (45.90)		
Drinking, n (%)				χ^2=0.26	0.607
No	292 (68.22)	211 (68.95)	81 (66.39)		
Yes	136 (31.78)	95 (31.05)	41 (33.61)		
Lack of exercise, n (%)				χ^2=0.51	0.476
No	257 (60.05)	187 (61.11)	70 (57.38)		
Yes	171 (39.95)	119 (38.89)	52 (42.62)		
Family history of CHD, n (%)				χ^2=0.15	0.700
No	376 (87.85)	270 (88.24)	106 (86.89)		
Yes	52 (12.15)	36 (11.76)	16 (13.11)		
Duration of hypertension, n (%)				χ^2=4.20	0.040
<10 years	223 (52.10)	169 (55.23)	54 (44.26)		
≥10 years	205 (47.90)	137 (44.77)	68 (55.74)		
Diabetes, n (%)				χ^2=8.14	0.004
No	318 (74.30)	239 (78.10)	79 (64.75)		
Yes	110 (25.70)	67 (21.90)	43 (35.25)		
Hyperuricemia, n (%)				χ^2=0.36	0.546
No	348 (81.31)	251 (82.03)	97 (79.51)		
Yes	80 (18.69)	55 (17.97)	25 (20.49)		

Abbreviations: CHD, coronary heart disease; SD, standard deviation; BMI, body mass index.

Table 2. Comparison of clinical information

Variables	Total (n=428)	Non-CHD group (n=306)	CHD group (n=122)	Statistic	*P*
TC, n (%)				χ^2=15.35	<0.001
Normal	325 (75.93)	248 (81.05)	77 (63.11)		
Abnormal	103 (24.07)	58 (18.95)	45 (36.89)		
TG, n (%)				χ^2=0.11	0.737
Normal	251 (58.64)	181 (59.15)	70 (57.38)		
Abnormal	177 (41.36)	125 (40.85)	52 (42.62)		
HDL-C, n (%)				χ^2=2.86	0.091
Normal	382 (89.25)	278 (90.85)	104 (85.25)		
Abnormal	46 (10.75)	28 (9.15)	18 (14.75)		
LDL-C, n (%)				χ^2=9.06	0.003
Normal	272 (63.55)	208 (67.97)	64 (52.46)		
Abnormal	156 (36.45)	98 (32.03)	58 (47.54)		
Lp-a, n (%)				χ^2=7.28	0.007
Normal	316 (73.83)	237 (77.45)	79 (64.75)		
Abnormal	112 (26.17)	69 (22.55)	43 (35.25)		

Abbreviations: CHD, coronary heart disease; TC, total cholesterol; TG, triglycerides; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; Lp-a, Lipoprotein(a).

Table 3. Univariate Logistic regression analysis

Variables	β	S.E	Z	*P*	OR (95% CI)
Age	0.09	0.03	3.31	<0.001	1.10 (1.04-1.16)
Gender					
Female					1.00 (Reference)
Male	-0.34	0.24	-1.41	0.159	0.71 (0.44-1.14)
BMI					
<24 kg/m^2					1.00 (Reference)
≥24 kg/m^2	0.23	0.23	1.00	0.317	1.26 (0.80-1.99)
Smoking					
No					1.00 (Reference)
Yes	0.79	0.22	3.56	<0.001	2.21 (1.43-3.41)
Drinking					
No					1.00 (Reference)
Yes	0.12	0.23	0.51	0.608	1.12 (0.72-1.76)
Lack of exercise					
No					1.00 (Reference)
Yes	0.15	0.22	0.71	0.477	1.17 (0.76-1.79)
Family history of CHD					
No					1.00 (Reference)
Yes	0.12	0.32	0.39	0.700	1.13 (0.60-2.13)
Duration of hypertension					
<10 years					1.00 (Reference)
≥10 years	0.44	0.22	2.04	0.041	1.55 (1.02-2.37)
Diabetes					
No					1.00 (Reference)
Yes	0.66	0.23	2.83	0.005	1.94 (1.23-3.07)
Hyperuricemia					
No					1.00 (Reference)
Yes	0.16	0.27	0.60	0.547	1.18 (0.69-1.99)
TC					
Normal					1.00 (Reference)
Abnormal	0.92	0.24	3.85	<0.001	2.50 (1.57-3.98)
TG					
Normal					1.00 (Reference)
Abnormal	0.07	0.22	0.34	0.737	1.08 (0.70-1.65)
HDL-C					
Normal					1.00 (Reference)
Abnormal	0.54	0.32	1.67	0.094	1.72 (0.91-3.24)
LDL-C					
Normal					1.00 (Reference)
Abnormal	0.65	0.22	2.99	0.003	1.92 (1.25-2.95)
Lp-a					
Normal					1.00 (Reference)
Abnormal	0.63	0.23	2.68	0.007	1.87 (1.18-2.96)

Abbreviations: SE, Standard Error; OR, Odds Ratio; CI, Confidence Interval; BMI, body mass index; TC, total cholesterol; TG, triglycerides; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; Lp-a, Lipoprotein(a).

4). Furthermore, there was a trend toward a statistically significant association between abnormal LDL-C (P=0.051) and Lp-a (P=0.077) levels and an increased risk of CHD.

Table 4. Multivariate Logistic regression analysis

Variables	β	S.E	Z	*P*	OR (95% CI)
Age	0.09	0.03	2.90	0.004	1.09 (1.03-1.15)
Smoking					
No					1.00 (Reference)
Yes	0.68	0.24	2.85	0.004	1.97 (1.24-3.13)
Duration of hypertension					
<10 years					1.00 (Reference)
≥10 years	0.51	0.23	2.20	0.028	1.67 (1.06-2.63)
Diabetes					
No					1.00 (Reference)
Yes	0.54	0.25	2.14	0.033	1.72 (1.05-2.81)
TC					
Normal					1.00 (Reference)
Abnormal	0.74	0.25	2.92	0.003	2.11 (1.28-3.47)
LDL-C					
Normal					1.00 (Reference)
Abnormal	0.46	0.24	1.95	0.051	1.59 (1.00-2.52)
Lp-a					
Normal					1.00 (Reference)
Abnormal	0.44	0.25	1.77	0.077	1.56 (0.95-2.55)

Abbreviations: SE, Standard Error; OR, Odds Ratio; CI, Confidence Interval; TC, total cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; Lp-a, Lipoprotein(a).

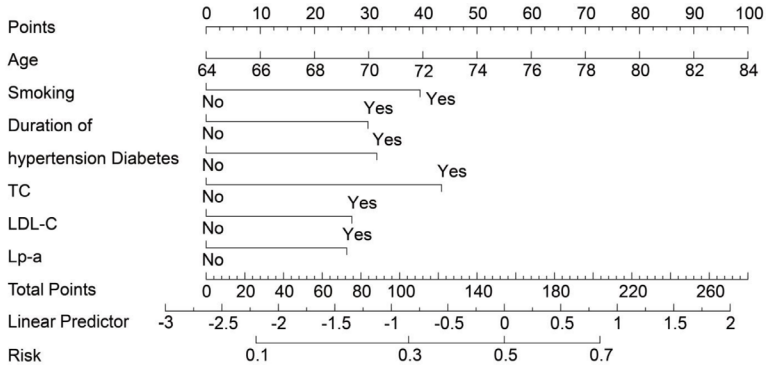

Figure 1. Nomogram prediction model. Abbreviations: TC, total cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; Lp-a, Lipoprotein(a).

Construction of a clinical prediction model

Based on the logistic regression results, age, smoking, hypertension duration, diabetes, TC, LDL-C, and Lp-a were included in the prediction model. **Figure 1** presents the prediction model in the form of a nomogram. For each risk factor, a score corresponding to the factor's contribution to the risk of CHD was assigned, and the total score was used to estimate the probability of CHD.

Evaluation of clinical prediction models

The receiver operating characteristic (ROC) curve results showed that the area under the curve (AUC) of the model was 0.71 (95% CI: 0.65-0.76), indicating good discriminatory ability (**Figure 2A**). The calibration curve and Hosmer-Lemeshow test (P=0.907) further confirmed the model's good calibration (**Figure 2B**). The accuracy, sensitivity, specificity, PPV and NPV of the nomogram model were 0.69 (95% CI: 0.64-0.73), 0.72 (95% CI: 0.67-0.77), 0.60 (95% CI: 0.51-0.69), 0.82 (95% CI: 0.77-0.86), and 0.46 (95% CI: 0.38-0.54), respectively (**Table 5**).

Clinical application value analysis

Decision curve analysis (DCA) was used to evaluate the clinical utility of the predictive model. The DCA results showed that a positive net

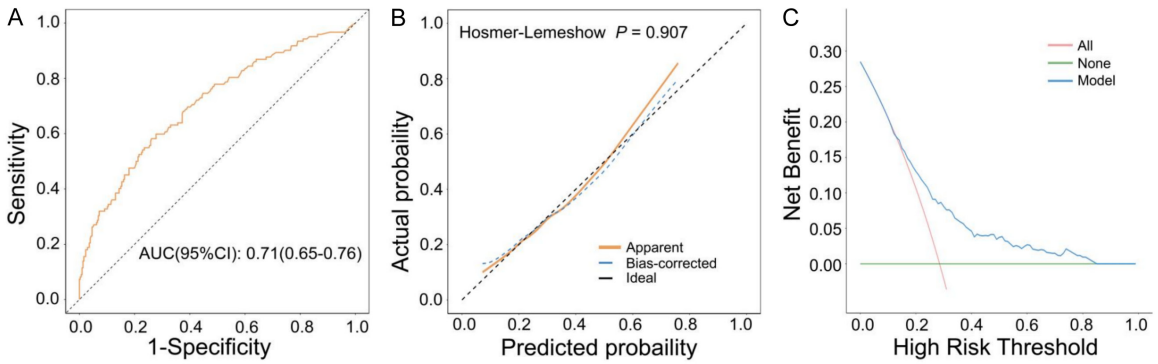

Figure 2. ROC curve, calibration curve and DCA of the nomogram model. A. ROC curve; B. Calibration curve; C. DCA. Abbreviations: ROC, Receiver Operating Characteristic; DCA, Decision Curve Analysis.

Table 5. Confusion matrix analysis

AUC (95% CI)	Accuracy (95% CI)	Sensitivity (95% CI)	Specificity (95% CI)	PPV (95% CI)	NPV (95% CI)	Cut off
0.71 (0.65-0.76)	0.69 (0.64-0.73)	0.72 (0.67-0.77)	0.60 (0.51-0.69)	0.82 (0.77-0.86)	0.46 (0.38-0.54)	0.316

Abbreviations: AUC, Area Under the Curve; CI, Confidence Interval; PPV, Positive Predictive Value; NPV, Negative Predictive Value.

benefit could be achieved by applying the model in clinical decision-making for patients with a risk probability between 0.15 and 0.80 (**Figure 2C**).

Discussion

The objective of this study was to evaluate the potential value of lipid levels in predicting the risk of CHD in an elderly hypertensive population, with the aim of providing a scientific basis for clinical prevention and treatment. The findings of this study demonstrate that age is a significant independent risk factor for CHD in elderly hypertensive individuals. As early as the previous century, Jousilahti et al. reported a strong association between age and CHD risk in a large cohort study [16]. With aging, significant changes occur in vascular structure and function, including endothelial dysfunction, increased vascular stiffness, and thickening of the arterial walls [17]. These alterations reduce the elasticity of blood vessels and increase fluctuations in blood pressure, both of which contribute to atherosclerosis. In elderly individuals, lipid deposits in the arteries form atherosclerotic plaques that narrow blood vessels and heighten the risk of myocardial ischemia [18]. Moreover, older individuals often suffer from multiple chronic conditions, such as hypertension, diabetes, and chronic kidney disease. These comorbidities, along with their treatments, can further exacerbate cardiovascular risks, creating a complex pathophysiological network that increases the risk of CHD [19]. In conclusion, advanced age is a critical risk factor for CHD in elderly hypertensive patients due to age-related vascular changes and the presence of multiple comorbidities.

This study also identified smoking as a significant risk factor for CHD. Smoking is known to be a major contributor to atherosclerosis, primarily by inducing endothelial dysfunction [20]. Endothelial cells, which protect blood vessels, are responsible for maintaining their vasodilation, anticoagulant, and anti-inflammatory functions. Smoking impairs these functions by reducing nitric oxide (NO) synthesis and activity, which in turn increases oxidative stress and contributes to the formation of atherosclerotic plaques [21]. In addition, smoking may interact synergistically with hypertension, placing further strain on blood vessels, which accelerates their stiffness and plaque formation. Thus, smoking significantly elevates the risk of CHD in elderly hypertensive patients through endothelial dysfunction and its synergistic effects with hypertension. However, further studies are needed to fully elucidate the underlying mechanisms involved.

The study also found that a longer duration of hypertension and the presence of diabetes were associated with an increased risk of CHD. Chronic hypertension results in altered shear

stress on vascular endothelial cells, leading to endothelial dysfunction, increased lipid permeability, and the release of inflammatory factors [22]. These changes promote the development of atherosclerosis. Additionally, prolonged vascular wall tension induces smooth muscle cell proliferation and vascular remodeling, resulting in wall thickening and reduced compliance, both of which further accelerate atherosclerosis and increase the risk of CHD [23]. Therefore, for hypertensive patients, especially those with long-standing hypertension, enhanced blood pressure control is essential to reduce the risk of CHD. In diabetic patients, elevated blood glucose levels and free fatty acids trigger inflammation in the vascular walls, impairing normal endothelial function and promoting the secretion of inflammatory mediators by immune cells [24]. This inflammatory state may also lead to vascular calcification, further accelerating atherosclerosis [25]. Studies indicate that more than half of individuals with type 2 diabetes will develop cardiovascular disease, and their risk of fatal CHD events is significantly higher than in non-diabetic individuals [26]. Therefore, careful monitoring of cardiovascular health in diabetic patients is crucial. In summary, both prolonged hypertension and diabetes significantly contribute to CHD risk through distinct pathological mechanisms, underscoring the importance of aggressive management of these conditions.

The results of this study suggest that abnormalities in certain lipid parameters, such as TC, LDL-C, and Lp-a, may be associated with an increased risk of CHD in hypertensive patients. Cholesterol is a lipid molecule essential for cell membrane formation, hormone synthesis, and other physiological functions in the body [27]. Dyslipidemia can lead to vascular endothelial dysfunction, a key event in the early stages of atherosclerosis. Endothelial dysfunction impairs the vasodilatory capacity of blood vessels and increases the risk of thrombosis [28]. Elevated TC levels can amplify the inflammatory response in the vascular endothelium, damaging it and accelerating the progression of atherosclerosis [29]. LDL-C, the primary lipid component of atherosclerotic plaques, contributes to cholesterol deposition in the vascular walls, leading to plaque formation and an increased risk of vessel narrowing and blockage, which, in turn, heightens the risk of CHD [30]. LDL-C, deposited in damaged endothelium, is prone to oxidation, resulting in oxidized LDL-C, which is subsequently phagocytosed by macrophages, forming foam cells and contributing to plaque development [31]. These plaques can obstruct blood flow, increasing the risk of myocardial ischemia and infarction. Furthermore, Lp(a) is a unique lipoprotein that can accumulate in damaged endothelium, binding to fibrinogen and the extracellular matrix, thereby promoting the formation and growth of atherosclerotic plaques [32]. Lp(a) also facilitates platelet aggregation and activates clotting factors, raising the risk of thrombosis, particularly following plaque rupture [33]. In summary, dyslipidemia is a critical risk factor for CHD in elderly hypertensive patients, emphasizing the importance of lipid management in clinical practice.

This study has some limitations. First, the use of a single sample source may limit the generalizability and accuracy of the model. Second, as a retrospective study, it may be subject to information bias and confounding factors. Moreover, factors such as genetic predisposition and lifestyle, which could influence CHD risk, were not considered. Future research should aim to expand the sample size and conduct multi-center, prospective studies to validate and refine the model. Additionally, exploring more biomarkers and genetic factors associated with CHD incidence could help develop a more comprehensive and accurate prediction system, thereby enhancing the clinical management of elderly hypertensive patients and the prevention and control of cardiovascular diseases.

In, conclusion, this study retrospectively analyzed the relationship between blood lipid levels and the risk of CHD in elderly hypertensive patients. The findings reveal that advanced age, smoking, a hypertension duration greater than 10 years, and abnormalities in TC, LDL-C, and Lp(a) are associated with an increased risk of CHD in this population. These results underscore the importance of lipid management in the prevention and treatment of CHD in elderly hypertensive patients and provide new strategies and treatment targets for clinical practice.

Disclosure of conflict of interest

None.

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