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Carotid Plaque-related Factor Analysis: A Cross-Sectional Study Based on a Large Sample

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Abstract: *Objective:* Aimed to conduct a cross-sectional study to analyze factors associated with carotid atherosclerotic plaque formation through a retrospective review of medical examination data. *Methods:* Data from medical examinations at Gongli Hospital of Shanghai Pudong New Area from July 2013 to October 2021 were collected. The analysis focused on carotid plaques, using both univariate and multivariate logistic regression. Subgroup analyses were conducted based on age, sex, BMI, and blood pressure measurements to identify risk factors for carotid plaques in different populations. *Results:* A total of 20,570 medical examination cases were included in the study. Among these cases, 7,435 showed carotid plaques, resulting in a detection rate of 36.1%. There were significant differences in the examined indicators between the groups with and without plaques. Both univariate logistic and multivariate regression analyses were conducted to identify independent factors related to carotid plaque formation. The results revealed the following factors that were positively correlated with plaque formation: advanced age, elevated systolic pressure, increased white blood cell count, increased basophil count, and elevated fasting blood sugar level ($P < 0.05$, odds ratio (OR) > 1). Additionally, female sex was found to be negatively correlated with carotid plaque formation ($P < 0.05$, OR < 1). *Conclusions:* Carotid plaques are influenced by several factors, including sex, age, systolic pressure, white blood cell count, basophil count, and fasting blood sugar levels. However, additional research is needed to determine the direct impact of these factors on the formation of carotid plaques.

Keywords: carotid plaques; stroke; risk factors; univariate logistic analyses; atherosclerosis

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1. Introduction

Stroke is a major disease that endangers both physical and mental health[1]. In China, there are approximately

2.4 million new cases annually, most of which result in varying degrees of disability. According to statistics, stroke is the leading cause of death among residents[2]. Active prevention and treatment have been implemented; however, the overall incidence of stroke remains stable, and the incidence of ischaemic stroke continues to increase[3]. The carotid artery is one of the arteries most frequently affected by systemic atherosclerosis[4]; therefore, its observation can reflect the state of systemic atherosclerosis and help predict the risk of cardiovascular events[5].

There are various risk factors for carotid atherosclerosis. For instance, Wang and colleagues identified age, albumin, fasting blood glucose, HbA1c, uric acid, hypertension, and diabetes as risk factors for carotid atherosclerosis through univariate analysis[6]. Jiao analyzed data from health check-ups and found that in addition to traditional cardiovascular risk factors, non-traditional pathogenic factors (e.g., lymphocyte percentage, platelet count) may be associated with carotid plaque progression, compared with those without plaque progression[7]. While previous studies have investigated the potential influences on carotid plaque formation, the majority have been limited by relatively small sample sizes, which may restrict the generalizability of their findings. Based on this, we aimed to analyze the risk factors and mechanisms of carotid atherosclerosis based on a large-sample cross-sectional study.

The findings of this study are expected to offer valuable insights into the etiology of carotid plaque formation, allowing healthcare providers to better identify high-risk individuals and implement targeted interventions. Moreover, the large sample size and robust statistical analysis employed in this research will enhance our understanding of the relative importance of different risk factors, enabling more accurate risk stratification and personalized preventive strategies.

Ultimately, this study's contributions will strengthen the evidence base for carotid atherosclerosis management, ultimately helping to reduce the burden of cardiovascular disease and improve patient outcomes.

2. Materials and methods

2.1. Participants

This study is a cross-sectional study. The study selected 20,570 individuals who underwent medical and arterial ultrasound examinations at the participants were divided into plaque and non-plaque groups based on the presence or absence of carotid atherosclerotic plaques. The inclusion criteria were an identifiable unique ID and having undergone a carotid ultrasound examination. The exclusion criteria were untraceable identity information, duplicate medical examination data, age less than 18 years, and not having undergone a carotid ultrasound examination. Based on these criteria, 20,570 cases were included, of which 7,435 (36.1%) and 13,135 (63.9%) were in the plaque and non-plaque groups, respectively. There were 11,047 male individuals (53.7%) and 9,523 female individuals (46.2%), with an average age of 55.01 years (range: 20–99 years).

2.2. Research methods

2.2.1. General measurements

Age, sex, height (m), weight (kg), systolic blood pressure (mmHg), and diastolic blood pressure (mmHg) were recorded. The body mass index (BMI) was calculated as weight (kg)/height^2 (m^2).

2.2.2. Laboratory tests

Urine pH, nitrite, urobilinogen, specific gravity, leukocyte esterase, bilirubin, glucose, protein, ketone, occult blood, liver function-alanine aminotransferase, biochemical kidney function-urea, kidney function-creatinine, fasting blood glucose, neutrophil count, neutrophil percentage, basophils, mean red blood cell volume, mean platelet volume, lymphocyte percentage, white blood cell count, haematocrit, haemoglobin content, haemoglobin concentration, red blood cell count, platelet distribution width, platelet count, haemoglobin values, and those of 28 other indicators were obtained.

2.2.3. Carotid ultrasound examination

The Philips EPIQ5 ultrasound scanner was used to examine the internal and external carotid arteries. Based on the 2008 American Society of Echocardiography consensus on carotid plaque definition, carotid local intima-media thickness (IMT) > 50% of the surrounding vessel IMT or IMT > 1.5 mm were the diagnostic criteria for plaques[8]. The participants were divided into plaque and non-plaque groups based on the presence or absence of carotid plaques, respectively.

2.3. Statistical analyses

The study data included demographic characteristics, laboratory test results, and carotid ultrasound examination results. Normally distributed continuous variables were described using means and standard deviations, and t-tests were used for comparisons. For non-normally distributed data, the median and the 25th and 75th percentiles were used for descriptions. Nonparametric tests were compared using the Mann-Whitney U test. Count data were described using frequency and rate, and comparisons between groups were made using the chi-square or corrected chi-square test. Means between the two groups were compared using the t-test. Logistic univariate regression analysis was used to determine the presence of carotid plaques as the dependent variable and each extracted indicator as an independent variable. The results are expressed as odds ratios (OR) and 95% confidence intervals (CI). Statistical significance was set at $P < 0.05$. Considering the sample distribution, the Pearson or Spearman method was used to analyze the correlation between the samples. Significant variables from the univariate analysis were included in the multivariate logistic regression analysis to further assess their independence. Central standardization was used for specific indicators that conformed to the geometric distribution. Combined with the results of the correlation and multivariate logistic regression analyses, nomograms were used to further analyze the comprehensive effects of these indicators. Finally, a subgroup analysis based on age, sex, BMI, and two blood pressure measurements was conducted to investigate risk indicators for carotid plaques in different populations. R (version 4.3.0) was used for data analysis.

3. Results

3.1. Comparison of clinical information

We continuously enrolled 236,475 patients from the department of cardiovascular surgery from between July 2013 and October 2021. Of these 78,288 medical examination data without traceable ID number 15,743 duplicate medical examination data (multiple medical examinations for the same ID number), 5,763 Had biochemical glucose fasting blood sugar data miss, 1,939 ad basophil count data miss 114,172 people did not have a carotid ultrasound who did not meet the eligibility criteria were excluded. Therefore, 20,570

patients were included, the participants were divided into plaque (7,435 cases, 36.1%) and non-plaque groups (13,135 cases, 63.9%). Comparisons were made between the groups regarding age, sex, systolic pressure, diastolic pressure, BMI, and urinalysis data; significant differences were observed between the two groups ($P < 0.05$). The plaque group included older participants and a higher proportion of male individuals than did the non-plaque group. However, both groups showed no significant differences in weight, liver function, haemoglobin, or mean platelet volume ($P > 0.05$).

3.2. Carotid atherosclerosis-related risk factors

3.2.1. Univariate regression analysis of carotid artery plaque-related factors

General clinical data and laboratory serology test results of the plaque and non-plaque groups were subjected to univariate logistic regression analysis. The results showed significant differences in several variables ($P < 0.05$), including sex, age, height, BMI, systolic pressure, diastolic pressure, urine pH, nitrite, specific gravity, glucose, protein, ketone, occult blood, liver function-alanine aminotransferase, biochemical kidney function-urea, kidney function-creatinine, fasting blood glucose, neutrophils, neutrophil percentage, basophils, mean red blood cell volume, white blood cell count, haematocrit, haemoglobin content, haemoglobin concentration, red blood cell count, platelet distribution width, and platelet count (**Table 1**).

Table 1. Univariate regression analysis of carotid artery plaques

Factor	*OR*	95% *CI*	*P*-value
Age	1.19	(1.18–1.19)	< 0.001
Height	0.97	(0.97–0.97)	< 0.001
Weight	1	(1–1)	0.43
BMI	1.06	(1.06–1.07)	< 0.001
SBP	1.05	(1.05–1.06)	< 0.001
DBP	1.02	(1.02–1.02)	< 0.001
U_pH	0.89	(0.86–0.93)	< 0.001
U_SG	0.86	(0.83–0.88)	< 0.001
ALT	1	(1–1)	0.01
Urea	1.36	(1.33–1.38)	< 0.001
Crea	1.02	(1.02–1.02)	< 0.001
Neut	1.18	(1.15–1.21)	< 0.001
Neut%	1.01	(1.01–1.01)	< 0.001
MCV	1.09	(1.08–1.09)	< 0.001
MPV	1.01	(0.99–1.03)	0.42
LY%	1.02	(0.98–1.06)	0.44
WBC	1.12	(1.11–1.14)	< 0.001
HCT	1.01	(1.01–1.01)	< 0.001
MCH	1.19	(1.17–1.21)	< 0.001
MCHC	0.99	(0.99–1)	< 0.001
RBC	0.65	(0.61–0.69)	< 0.001
PDW	1.03	(1.01–1.04)	< 0.001
PLT	1	(1–1)	< 0.001
HGB	1	(1–1)	0.59

Factor	*OR*	95% *CI*	*P*-value
FBS	1.56	(1.52–1.6)	< 0.001
Baso	1.27	(1.24–1.31)	< 0.001
Sex	0.66	(0.62–0.7)	< 0.001
U_NIT	2.32	(1.88–2.88)	< 0.001
U_URO	1.03	(0.87–1.23)	0.74
U_LEU	1.06	(0.98–1.14)	0.15
U_BIL	1.14	(0.91–1.42)	0.25
Unglue	2.77	(2.37–3.24)	< 0.001
U_PRO	1.96	(1.75–2.19)	< 0.001
U_ket	0.82	(0.69–0.97)	0.02
U_BLD	1.14	(1.07–1.21)	< 0.001

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; U_pH, urine pH; U_SG, urine specific gravity; ALT, alanine transaminase; Crea, creatinine; Neut, neutrophils; MCV, mean corpuscular volume; MPV, mean platelet volume; LY%, percentage lymphocytes; WBC, white blood cell; HCT, haematocrit; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; RBC, red blood cell; PDW, platelet distribution width; PLT, platelet; HGB, haemoglobin; FBS, fasting blood sugar; Baso, basophils; U_NIT, urine nitrite; U_URO, urine urobilinogen; U_LEU, urine leukocytes; U_BIL, urine bilirubin; U_Glu, urine glucose; U_PRO, urine protein; U_ket, urine ketone; U_BLD; urine blood; *OR*, odds ratio; *CI*, confidence interval.

3.2.2. Multivariate correlation analysis

According to the multivariate correlation matrix diagram, there was a significant positive correlation between haemoglobin content and red blood cell count, weight and BMI, systolic and diastolic blood pressure, white blood cell count, neutrophil count, and mean corpuscular volume, with different levels of predictive function inclusion (**Figure 1A**).

After excluding these factors, logistic multivariate regression analysis showed that advanced age (OR = 1.18, 95% CI: 1.18–1.19, $P < 0.001$), elevated systolic blood pressure (OR = 1.03, 95% CI: 1.03–1.04, $P < 0.001$), increased white blood cell count (OR = 1.15, 95% CI: 1.06–1.26, $P = 0.002$), increased basophil count (OR = 1.23, 95% CI: 1.17–1.3, $P < 0.001$), and elevated fasting blood glucose level (OR = 1.21, 95% CI: 1.17–1.26, $P < 0.001$) were independent risk factors for the formation of carotid plaques. Only female sex was negatively correlated with carotid plaque formation ($P < 0.05$; multivariate analysis results were consistent with univariate analysis results and OR < 1) (**Table 2**).

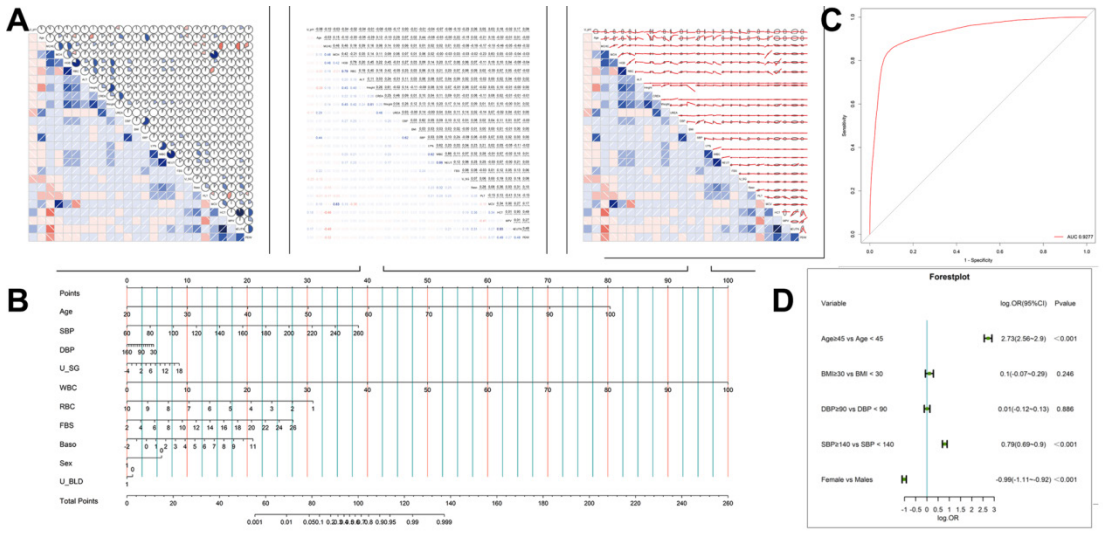

Figure 1. Under the curve value of the nominate. D, Forest map of subgroup analysis.

Table 2. Results of multivariate logistic regression analysis of carotid artery plaque-related factors

Factor	*OR*	95% *CI*	*P*
Age	1.18	(1.18–1.19)	< 0.001
SBP	1.03	(1.03–1.04)	< 0.001
FBS	1.21	(1.17–1.26)	< 0.001
Baso	1.23	(1.17–1.3)	< 0.001
Sex	0.46	(0.4–0.53)	< 0.001
WBC	1.15	(1.06–1.26)	0.002

SBP, systolic blood pressure; FBS, fasting blood sugar; Baso, basophils; WBC, white blood cell OR, odds ratio; CI, confidence interval

3.3. Significance of nomogram and area under the curve

An R 4.3.2 based risk-scoring nomogram model for carotid atherosclerotic plaques was constructed based on the magnitude of the regression coefficients for individual risk factors for carotid atherosclerotic plaques in the logistic prediction model (**Figure 1B**). The nomogram showed that age, white blood cell count, systolic blood pressure, basophil count, and fasting blood glucose levels had relatively high scores in the overall predictive function and were highly correlated with carotid plaque formation. The sum of the indicators in the nomogram was included in the overall evaluation function, which exhibited a high level of correlation. The area under the curve value was 0.928 (**Figure 1C**).

3.4. Subgroup analysis forest plot

The results of the subgroup analysis of the forest plot showed that advanced age (≥ 45 years) and elevated systolic blood pressure (≥140 mmHg) were significantly positively correlated with the formation of carotid artery plaques. Female sex was significantly negatively correlated with the formation of carotid artery plaques (**Figure 1D**).

4. Discussion

Carotid atherosclerotic plaques are a major cause of ischemic stroke. Their clinical presentation is

closely associated with the nature and state of the plaques; thus, unstable plaques can easily cause serious cardiovascular and cerebrovascular events. Therefore, detecting, diagnosing, treating, and assessing plaque stability early in patients with carotid atherosclerosis is crucial.

By analyzing the risk screening data of carotid artery plaques in a population undergoing health examinations, we found that advanced age, elevated systolic blood pressure, increased white blood cell and basophil counts, and elevated fasting blood glucose levels were positively correlated with the formation of carotid artery plaques. However, female sex was negatively correlated with carotid artery plaque formation.

Analysis of the risk screening data for carotid artery plaques in the population undergoing health examinations in Shanghai showed that advanced age is positively correlated with the formation of carotid artery plaques. Ageing is a significant factor affecting the formation of carotid artery plaques, consistent with previous studies showing that plaque detection rates increase continuously with age[9]. The Journal of the American College of Cardiology published data from early atherosclerosis-related research showing an approximately 40% prevalence of arterial plaques in asymptomatic people aged 40–50 years. The average age of the participants in this study was 55.01 years, the average age of those in whom carotid artery plaques were detected was 67 years, and the average age of those without carotid artery plaques was 48 years. The incidence of carotid artery plaque increases with age; however, the underlying mechanisms are complex and diverse. First, with increasing age, physiological and pathological changes that occur in the vascular wall are closely associated with the formation of carotid artery plaques[10]. The vascular wall gradually loses its elasticity owing to the gradual reduction and degeneration of its structural components, such as collagen and elastic fibres in the vascular wall[11]. His loss of vascular elasticity reduces the buffering capacity of blood vessels for blood flow, increases the impact of blood flow on the vascular wall, and damages the vascular intima, providing conditions for lipid deposition and plaque formation.

White blood cells are essential in the formation of carotid artery plaques. Neutrophils are the primary driving factors in the vascular and systemic inflammatory processes associated with atherosclerosis. They form and develop plaques through phagocytosis, production of reactive oxygen species, release of metalloproteinases, and production of extracellular traps[12]. In the early stages of plaque development, macrophages facilitate the release of pro-inflammatory cytokines (IL-6, IL-17, TNF-α, IFN-γ), which further attract more macrophages and T and B lymphocytes towards the plaque [13]. This process triggers the development of a proinflammatory environment that accelerates plaque progression.

Additionally, interactions between platelets and white blood cells and those between platelets and endothelial cells promote the progression of atherosclerotic plaques. These interactions lead to the synthesis and release of pro-atherosclerotic cytokines, stimulating the proliferation and migration of vascular smooth muscle cells and further promoting plaque formation. Therefore, to prevent and treat carotid atherosclerotic plaques, in addition to focusing on traditional risk factors, such as age, blood pressure, and blood glucose, the role of white blood cells and related cytokines should be considered in evaluating the risks more comprehensively and formulating effective intervention measures[14]. In addition to age, sex is a significant factor in large populations. According to 2019 data from the World Health Organization, over 18 million people died from cardiovascular and cerebrovascular diseases, including 9.6 million men and 8.9 million women[15]. Notably, more men than women died from atherosclerotic cardiovascular and cerebrovascular diseases, at an average age of 40–60 years, approximately 7–10 years earlier than that in women[16]. This is consistent with previous study results, which showed that the incidence rate of carotid artery disease was

higher in men than in women and increased with age[17]. This may be associated with the protective effects of oestrogen on cardiovascular endothelial function and lipid homeostasis. This protective effect counteracts the development of atherosclerotic plaques[18]. Additionally, unhealthy lifestyle factors in the male population, such as excessive smoking, are risk factors for the occurrence of vulnerable carotid plaques[19].

5. Conclusions

In conclusion, advanced age, elevated systolic blood pressure, increased white blood cell count, increased basophil count, and elevated fasting blood glucose levels positively correlated with the formation of carotid artery plaques. However, female sex is a negatively correlated factor. Further research should be conducted to analyze possible causal relationships. Moreover, the impact of unhealthy lifestyles and early detection and management of risk factors, such as hypertension, hyperlipidaemia, and diabetes, should also be considered. This can slow the progression of atherosclerosis. Therefore, investigating how these factors interact to promote carotid artery plaque formation and their impact on plaque stability can provide insights. Moreover, the effects of different factors on different types of carotid artery plaques (vulnerable and non-vulnerable) can be explored. By understanding the causal relationships between these factors and carotid artery plaque formation, early intervention and treatment can provide a scientific basis for formulating more effective prevention and treatment strategies to reduce the risk of carotid artery plaque formation.

This paper has the following limitations, we explored the existence of certain associations between carotid plaques such as age, gender, blood pressure, WBC and basophils, but this study was a cross-sectional study and the causal relationship between the factors could not be determined. Future prospective studies are needed to further understand the etiological relationship between these factors and carotid plaque formation.

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Disclosure statement

The author declares no conflict of interest.

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