Lipids in Health and Disease

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The effect of blood lipids on the comorbidity of multiple large arachnoid granulations

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Abstract

Background Currently, studies on the formation mechanism for the enlargement of arachnoid granulation (AG) are lacking. The impact of dyslipidemia on the formation of multiple large arachnoid granulations (LAGs) was studied in this research.

Methods The study included patients diagnosed with cerebral venous sinus stenosis (CVSS) related to LAG. The number of LAGs was assessed via high-resolution black blood magnetic resonance imaging. The relationships between blood lipids and multiple LAGs were explored to evaluate the effects of dyslipidemia on the formation of multiple LAGs.

Results A total of 163 participants with a diagnosis of LAG were included. The levels of total cholesterol (TC) ($P=0.004$) and low-density lipoprotein cholesterol (LDL-c) ($P=0.01$) in the multiple LAGs group were greater than those in the non-multiple LAGs group. Multivariate logistic regression analysis revealed that TC (odds ratio (OR), 2.19; 95% confidence interval (CI), 1.26–3.80; $P=0.006$) and LDL-c (OR, 2.18; 95% CI, 1.16–4.07; $P=0.02$) were independently associated with multiple LAGs.

Conclusions TC and LDL-c are independently related to multiple LAGs, indicating that dyslipidemia may be a potential cause of CVSS. Therefore, monitoring blood lipids may be necessary for patients with LAGs.

Keywords Arachnoid granulation, Cerebral venous sinus stenosis, Blood lipid, Dyslipidemia, High-resolution black blood magnetic resonance imaging

Introduction

Arachnoid granulation (AG) is one of the pathways for cerebrospinal fluid reflux. Large arachnoid granulations (LAGs) in the venous sinus may cause severe cerebral venous sinus stenosis (CVSS), which may contribute to idiopathic intracranial hypertension (IIH) [1]. However, the mechanism behind the enlargement of AGs remains unclear. Obesity can lead to the development of IIH [2] and is linked to lipid metabolism disorders [3]. During pathological examinations, foam cells were present in LAGs [4]. In clinical practice, patients with LAGs tend to have high blood lipid levels, indicating that obesity and related lipid metabolism abnormalities may affect the number and size of AG.

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The effect of blood lipids on the circulatory system is a hot research topic. Both in vitro and in vivo, lipoproteins are key contributors to the onset and development of atherosclerosis, which is responsible for their association with cardiovascular and cerebrovascular diseases. After passing through the endothelial barrier and depositing at the artery walls, lipoproteins can lead to a complex inflammatory process that initiates atherosclerosis [5–7]. Previous studies have shown that the progression of atherosclerotic plaques is directly correlated with the plasma low-density lipoprotein cholesterol (LDL-c) concentration [8, 9]. The above evidence suggests that blood lipids play an important role in arterial diseases. However, little attention has been given to the effects of blood lipids on the cerebral venous system.

The differences in hemodynamics and structures between veins and arteries make it difficult for veins to develop atherosclerotic processes. However, previous studies have shown that dyslipidemia can also lead to venous endothelial damage [10]. Following coronary artery bypass grafting, dyslipidemia leads to intimal thickening of vein grafts and rapid development of atherosclerosis [11, 12]. Lipid-lowering therapy after bypass surgery can reduce the atherosclerotic burden of vein grafts and improve cardiovascular outcomes [13]. In addition, cholesterol deposits are observed in the wall of the varicose saphenous vein [14], suggesting that blood lipids are related to lesions in the peripheral vein. Hypercholesterolemia has been shown to be associated with the development of cerebral venous sinus thrombosis (CVST) [15], suggesting that dyslipidemia may also play a potential role in cerebral venous diseases. Currently, the interaction between dyslipidemia and the increase in AGs has remained ambiguous. We hypothesized that dyslipidemia may affect the formation of LAG by causing intracranial venous endothelial dysfunction and triggering local inflammatory responses. Therefore, this research analyzed the role between blood lipids and multiple LAGs. Furthermore, the present study used high-resolution black blood magnetic resonance imaging (BB-MRI), which accurately diagnosed and measured the size of AG, to assess the number of LAGs.

Methods

Approval was obtained from the Xuanwu Hospital Institutional Ethics Committee. Patients who were enrolled signed an informed consent. The above process was in line with the Declaration of Helsinki.

The present study retrospectively enrolled eligible patients consecutively who completed high-resolution BB-MRI scanning with a diagnosis of non-thrombotic CVSS in the Neurology Department at Xuanwu Hospital, Capital Medical University. Admissions for the patients took place between January 2019 and December 2021. When the smallest cross-sectional area at the stenotic location is less than 50% of the cross-sectional area of an adjacent nonstenotic segment, this is known as non-thrombotic CVSS [16]. LAG was defined as the margin of the AG extending to the median of the venous sinus or when the largest diameter of the AG was greater than 4 mm [17, 18]. The controls were sex- and age-matched patients who visited the clinic for non-venous-related reasons. In accordance with the standard clinical protocol used at our institution, blood samples for blood routine, biochemistry, and coagulation function were drawn on the first morning of admission. The blood lipids included apolipoprotein A (apo A), apolipoprotein B (apo B), LDL-c, high-density lipoprotein (HDL-c), triglycerides (TG), total cholesterol (TC), non-HDL-c, the LDL-c/HDL-c ratio, the non-HDL-c/HDL-c ratio, the apo B/HDL-c ratio, the TC/HDL-c ratio and the apo B/A ratio. The lipid ratios present better predictive value in the progression of atherosclerosis [19–21]. BB-MRI examinations were performed according to institutional standardized clinical protocols. BB-MRI, a type of T1-weighted high-resolution imaging, is widely used to diagnose cerebral venous diseases [22–24], and it can distinguish between the AG and CVST with high spatial resolution and show the luminal structure directly by suppressing blood signals.

The requirements for inclusion were as follows: (a) diagnosis of non-thrombotic CVSS; (b) high-resolution BB-MRI scanning for imaging assessment; (c) completed blood tests on the first morning after admission; (d) no age or sex restrictions. The requirements for exclusion were as follows: (a) no LAG in the venous sinus; (b) history of cerebral infarction or intracerebral hemorrhage; (c) history of thyroid diseases or other complex endocrine diseases; (d) history of liver, renal dysfunction, or neoplastic diseases; (e) insufficient clinical or radiological data; (f) history of hormone-based medication, including hormone contraceptives; (g) history of intracranial space-occupying lesions.

Assessment

The distribution and number of LAGs were assessed with high-resolution BB-MRI. LAG was defined as the margin of the AG extending to the median of the venous sinus or when the largest diameter of the AG was greater than 4 mm [17, 18]. Representative images of LAGs are shown in Fig. 1. The AG in high-resolution BB-MRI shows ovoid soft tissue with a cerebrospinal fluid signal inside and a relatively sharp border, and it does not show enhancement features similar to a thrombus or tumor [17]. The cross-sectional view of the venous sinus in the axial direction was obtained to assess the AG size. All the LAGs in the cerebral venous sinus were counted, which included the left sigmoid sinus (SS), left transverse sinus

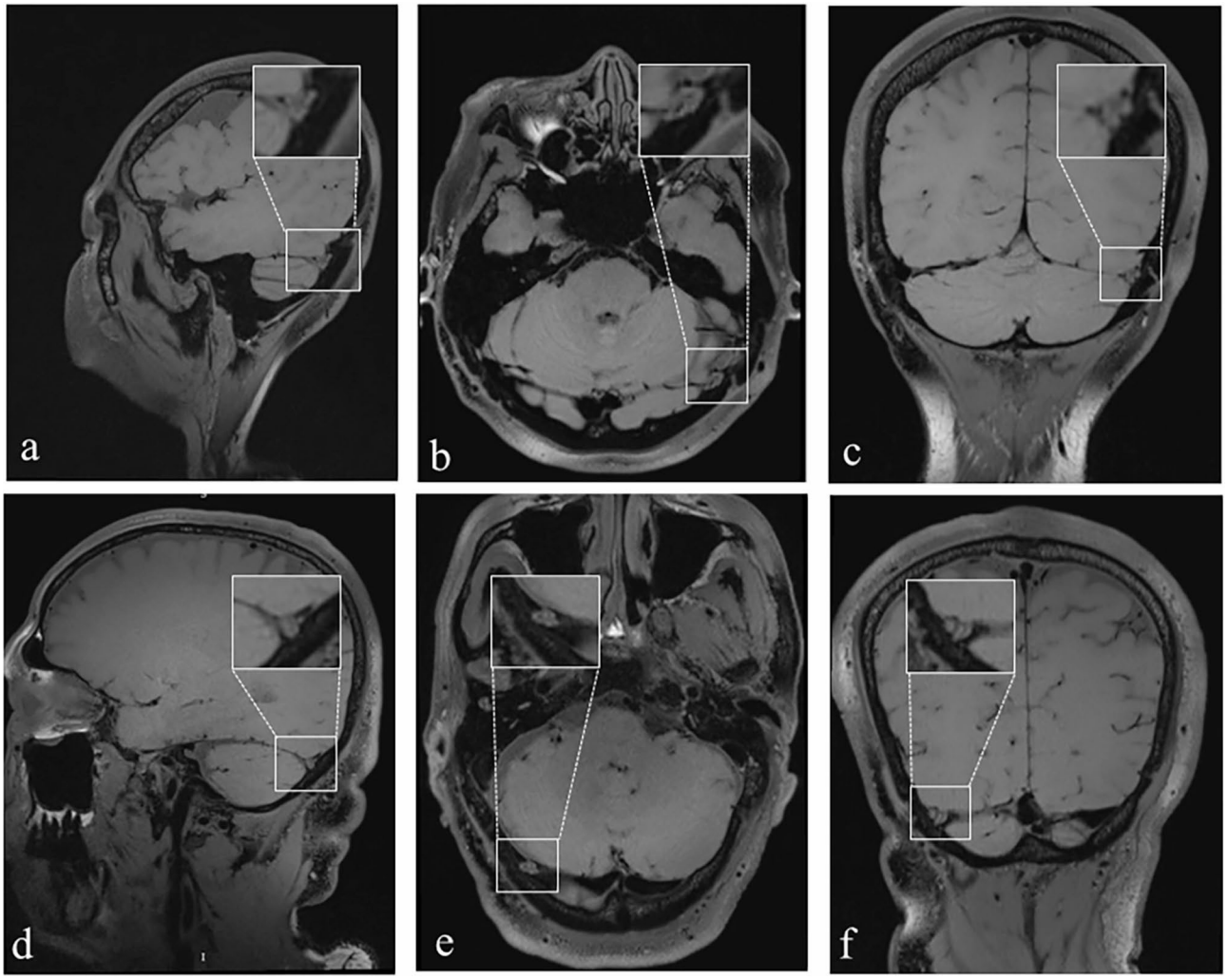

Fig. 1 Sagittal (**a, d**), transverse (**b, e**), and coronal (**c, f**) BB-MRI show LAG in the TS. The square insets in the panels are 2.0× magnifications of the original LAG images. Panel **a-c**: a patient with the LAG at the left TS. In a and c, the marginal of the AG extended to the median of the venous sinus. Panel **d-f**: The LAG is located at the right TS. In d, e, and f, the largest diameter of LAG was greater than 4 mm. Abbreviations: BB-MRI, black blood magnetic resonance imaging; LAG, large arachnoid granulation; TS, transverse sinus

(TS), right SS, right TS, torcular herophili, straight sinus, and superior sagittal sinus. The patients were grouped with the 75th quartile of the total LAG count being the cut-off.

BB-MRI image evaluation

BB-MRI data was obtained via a 3.0T MRI system according to previously reported methods [22, 23]. Two physicians with 6 years (ZA.W.) and 10 years (RM) of clinical experience in cerebral venous diseases independently performed the high-resolution BB-MRI image assessments in a blinded and randomized manner. Any controversies were resolved by a third researcher (XM.J.), who has 13 years of diagnostic experience.

Statistical analysis

Medians (interquartile range (IQR)) and means±standard deviation (SD) represent non-normal distributions and normal distributions. Categorical data are presented as numbers (percentages). Histograms and probability distribution plots are used to assess the normality of the data. Intraclass correlation coefficients (ICCs) were calculated for interobserver reliability when the LAGs were assessed. Reliability was divided into four groups: (a) less than 0.50 (poor); (b) between 0.75 and 0.50 (moderate); (c) between 0.90 and 0.75 (good); (d) greater than 0.90 (excellent). When ICCs were considered excellent, the results from the two readers were averaged to obtain the final number of LAGs. Group comparisons were tested via the t-test and the Mann-Whitney U test, which were used respectively for variables with regular distributions and non-normal distributions. The independent factors

linked to the multiple LAGs were determined via multivariable binary logistic regression. Correlations were evaluated via Pearson or Spearman correlation analysis. A statistically significant two-sided *P*-value was less than 0.05. SPSS Statistics 21.0 was utilized to conduct statistical analysis.

Results

Clinical presentations

The study initially included 237 individuals diagnosed with non-thrombotic CVSS. After the assessments, 74 patients were excluded. Of these, 50 had no LAG in the venous sinus, 5 lacked sufficient clinical or radiological data, 4 had a history of cerebral infarction, 5 had thyroid dysfunction, 8 had liver dysfunction and 2 had intracranial space-occupying lesions. Figure 2 illustrates the process for enrollment of patients. Ultimately, the study included 163 patients with a mean BMI of 25.48±4.21 kg/m2 and an average age of 49.58±15.07 years (14–78 years). Patients were divided into two groups (multiple LAGs and non-multiple LAGs groups) according to the number of LAGs at the cut-off point of 2, with 35 patients in the multiple LAGs group (the number of LAG>2) and 128 patients being in the non-multiple LAGs group (0<the number of LAG≤2). There was no significant difference in demographic characteristics or past medical histories between the two groups (Table 1). Common clinical symptoms included headache (44.8%), tinnitus (38.7%), head noise (38.7%), dizziness (36.8%), visual impairment (29.4%), and dry or puffy eyes (7.4%). Tinnitus was more common in the group with multiple LAGs ($P=0.01$). The remaining symptoms showed no discernible difference between the two groups (Table 1). 166 patients without a diagnosis of venous disease made up the control group. The LAG group had a higher BMI than the control (25.48±4.21 vs. 23.67±3.43 kg/m2, $P<0.001$). Other demographic characteristics showed no notable variations between the two groups (Table 2).

Imaging presentations

Among the 163 patients in the study, the LAG distribution was as follows: 90 (55.2%) patients with LAGs in the left TS; 27 (16.6%) patients with LAGs in the left SS; 41 (25.2%) patients with LAGs in the right TS; 14 (8.6%) patients with LAGs in the right SS; 69 (42.3%) patients with LAGs in the superior sagittal sinus; 17 (10.4%) patients with LAGs in the straight sinus; and 2 (6.7%) patients with LAGs in the torcular herophili. The ICCs

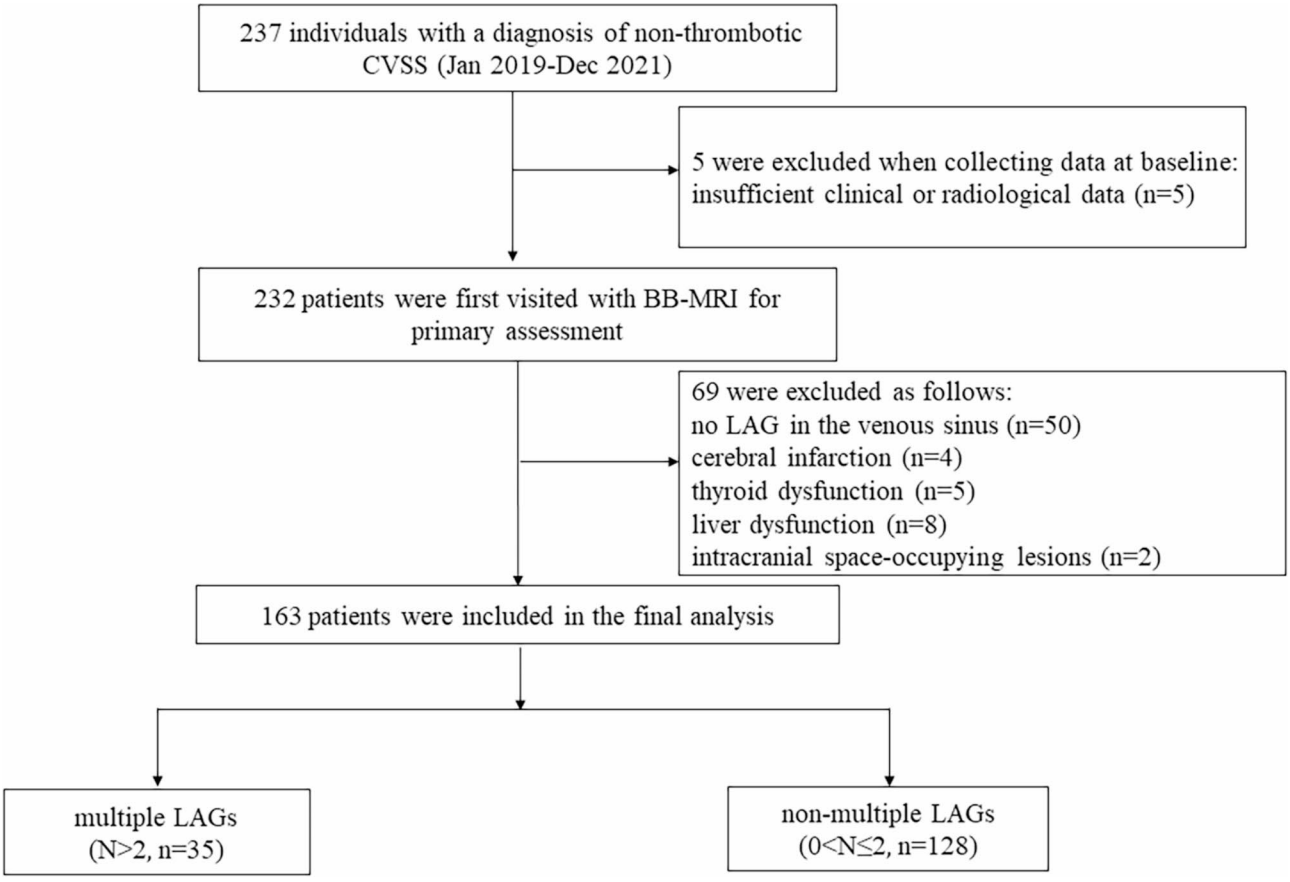

Fig. 2 Patient flow chart

Table 1 Comparison between the multiple LAGs and non-multiple LAGs group

	multiple LAGs (> 2) (*n* = 35)	non-multiple LAGs (≤ 2) (*n* = 128)	*P*
Demographics			
Sex (male)	11 (31.4%)	48 (37.5%)	0.51
Age (years) (Mean ± SD)	52.17 ± 12.16	48.87 ± 15.73	0.25
BMI (kg/m^2) (Mean ± SD)	26.26 ± 4.35	25.26 ± 4.16	0.21
Onset-to-door time (months) (Median, IQR)	17 (5–51)	24 (6–72)	0.19
Blood lipid			
TG (mmol/L)	1.22 ± 0.41	1.36 ± 0.68	0.25
TC (mmol/L)	4.21 ± 0.74	3.80 ± 0.74	0.004
HDL-c (mmol/L)	1.20 ± 0.34	1.09 ± 0.28	0.04
LDL-c (mmol/L)	2.49 ± 0.63	2.19 ± 0.64	0.01
apo A (g/L)	1.21 ± 0.22	1.17 ± 0.22	0.38
apo B (g/L)	0.84 ± 0.17	0.77 ± 0.19	0.053
non-HDL-c (mmol/L)	3.01 ± 0.70	2.71 ± 0.75	0.04
TC/ HDL-c ratio	3.68 ± 0.94	3.69 ± 1.15	0.94
Apo B/A ratio	0.72 ± 0.20	0.68 ± 0.22	0.38
LDL-c/HDL-c ratio	2.20 ± 0.73	2.15 ± 0.87	0.76
non-HDL-c/HDL-c ratio	2.68 ± 0.94	2.69 ± 1.15	0.94
apo B/HDL-c ratio	0.74 ± 0.25	0.76 ± 0.31	0.77
Symptoms and signs			
Headache	15 (42.9%)	58 (45.3%)	0.80
Tinnitus	20 (57.1%)	43 (33.6%)	0.01
Head noise	15 (42.9%)	48 (37.5%)	0.56
Dizziness	11 (31.4%)	49 (38.3%)	0.46
Visual impairment	12 (34.3%)	36 (28.1%)	0.48
Dry or puffy eyes	5 (14.3%)	7 (5.5%)	0.08
Past Medical History			
Hypertention	13 (37.1%)	41 (32.0%)	0.57
Hyperlipidemia	10 (28.6%)	31 (24.2%)	0.60
Diabetes	4 (11.4%)	16 (12.5%)	0.86
Smoking	5 (14.3%)	12 (9.4%)	0.40
Drinking	3 (8.6%)	19 (14.8%)	0.34

Abbreviations: TC, total cholesterol, TG, triglycerides, HDL-c, high-density lipoprotein cholesterol, LDL-c, low-density lipoprotein cholesterol, apo A, apolipoprotein A, apo B, apolipoprotein B

for calculating the number of LAGs was 0.91 (95% confidence interval (CI) 0.88 to 0.94). The median LAGs number was 2 (range, 1–7.5), and IQR was 1–2. The median LAGs number was 3 (range, 3–7.5; IQR, 3–3.5) in the multiple LAGs group and 1.5 (range, 1–2; IQR, 1–2) in the non-multiple LAGs group ($P<0.001$).

Correlation between AG and blood lipid

Compared to the non-multiple LAGs group, the multiple LAGs group showed an increased prevalence of dyslipidemia. The TC concentration was 4.21 ± 0.74 in the multiple LAGs group and 3.80 ± 0.74 in the non-multiple LAGs group ($P=0.004$). The HDL-c concentration was 1.20 ± 0.34 in the multiple LAGs group and 1.09 ± 0.28 in the non-multiple LAGs group ($P=0.04$). The non-HDL-c concentration was 3.01 ± 0.70 in the multiple LAGs group and 2.71 ± 0.75 in the non-multiple LAGs group ($P=0.04$). The LDL-c concentration was 2.49 ± 0.63 in the multiple LAGs group and 2.19 ± 0.64 in the non-multiple LAGs group ($P=0.01$). The units for the above blood lipid indicators are mmol/L. The remaining blood lipid parameters show no discernible difference between the two groups (Table 1). The HDL-c and apo A levels in the LAG group were lower than those in the control group ($P<0.01$). Other blood lipid parameters in the LAG group were greater than those in the control group ($P<0.01$). Specific results were listed in Table 2.

Table 2 Comparison of the demographic and blood lipid between LAG and control group

	LAG group (*n* = 163)	Control group (*n* = 166)	*P*
Demographics			
Sex (male)	59(36.2%)	61(36.7%)	0.99
Age (years) (Mean ± SD)	49.58 ± 15.07	49.52 ± 15.39	0.97
BMI (kg/m^2) (Mean ± SD)	25.48 ± 4.21	23.67 ± 3.43	< 0.001
Blood lipid			
TG (mmol/L)	1.33 ± 0.64	1.12 ± 0.46	0.001
TC (mmol/L)	3.89 ± 0.76	3.69 ± 0.43	0.004
HDL-c (mmol/L)	1.11 ± 0.30	1.23 ± 0.28	< 0.001
LDL-c (mmol/L)	2.25 ± 0.65	2.04 ± 0.34	< 0.001
apo A (g/L)	1.18 ± 0.22	1.25 ± 0.19	0.001
apo B (g/L)	0.78 ± 0.18	0.77 ± 0.18	0.43
non-HDL-c (mmol/L)	2.78 ± 0.75	2.46 ± 0.44	< 0.001
TC/ HDL-c ratio	3.69 ± 1.10	3.11 ± 0.65	< 0.001
apo B/A ratio	0.69 ± 0.21	0.63 ± 0.19	0.007
LDL-c/HDL-c ratio	2.16 ± 0.84	1.74 ± 0.49	< 0.001
non-HDL-c/HDL-c ratio	2.69 ± 1.10	2.11 ± 0.65	< 0.001
apo B/HDL-c ratio	0.69 ± 0.37	0.55 ± 0.24	< 0.001

Abbreviations: TC, total cholesterol, TG, triglycerides, HDL-c, high-density lipoprotein cholesterol, LDL-c, low-density lipoprotein cholesterol, apo A, apolipoprotein A, apo B, apolipoprotein B

The study analyzed correlations between blood lipids and multiple LAGs with multivariate logistic regression models. As shown in Fig. 3, TC ($P=0.006$), LDL-c ($P=0.02$), and non-HDL-c ($P=0.03$) were independently associated with multiple LAGs, whereas, HDL-c was not independently associated with multiple LAGs. Notably, after 23 patients (14%) receiving long-term lipid-lowering medications therapy were excluded, the TC ($P=0.002$), LDL-c ($P=0.005$), HDL-c ($P=0.04$), and non-HDL-c ($P=0.02$) were still independently associated with multiple LAGs. The odds ratios and 95% CIs are displayed in Supplement Fig. 1. Additionally, BMI was linearly correlated with TG ($P<0.001$), HDL-c ($P=0.003$), and apo A ($P=0.01$), but not with TC, LDL-c, or apo B (Fig. 4). According to Supplement Fig. 2, apo A showed a linear correlation with HDL-c ($P<0.001$) and TC ($P=0.03$). Apo B had a linear connection with TC ($P<0.001$), LDL-c

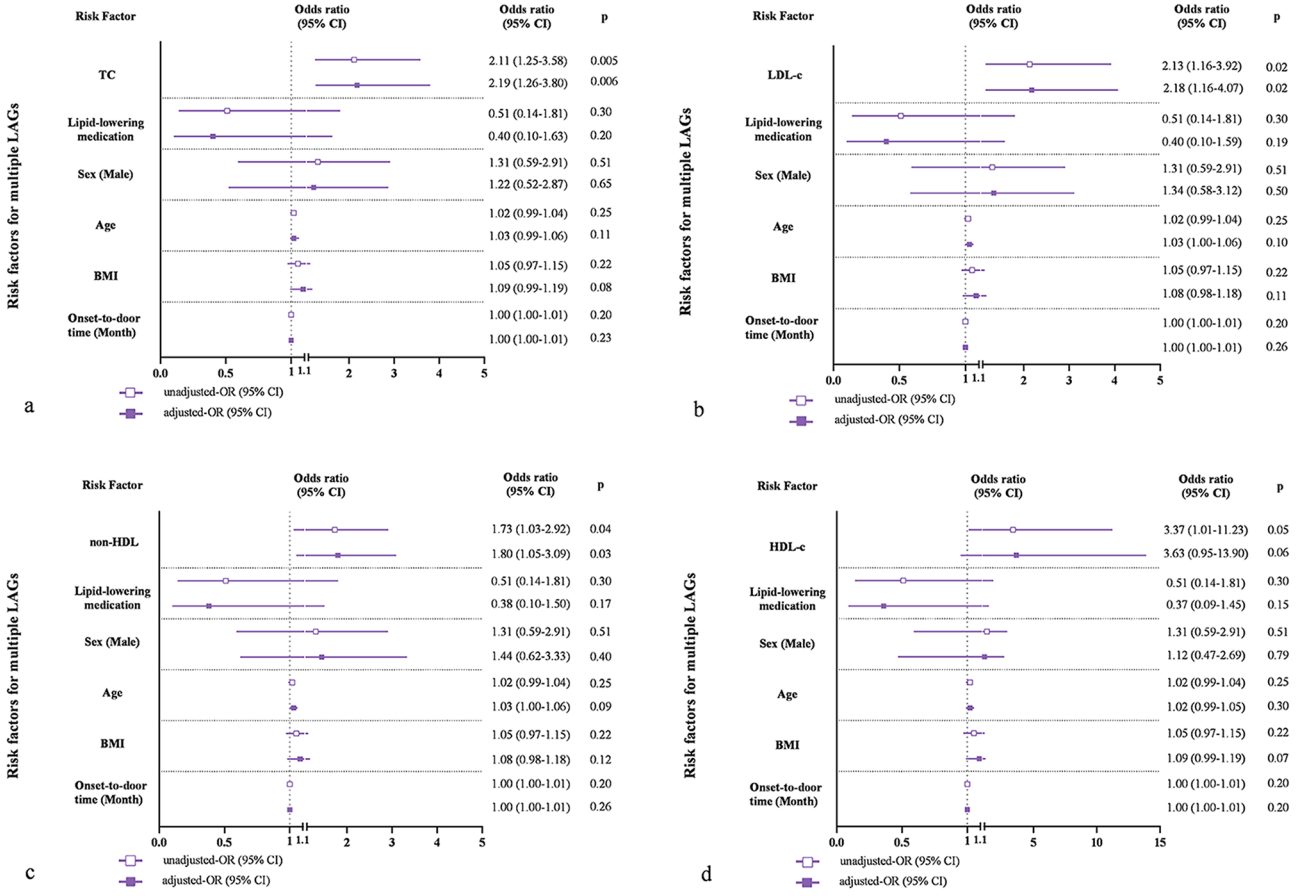

Fig. 3 Forest plot of the correlations between blood lipids and multiple LAGs. Forest plots showing logistic regression results for associations between multiple LAGs and TC (**a**), LDL-c (**b**), non-HDL (**c**), and HDL-c (**d**). The multivariable logistic regression models were adjusted for sex, age, BMI, lipid-lowering medication and onset-to-door time. The data are displayed as ORs and 95% CIs. Abbreviations: TC, total cholesterol; LDL-c, low-density lipoprotein cholesterol; HDL-c, high-density lipoprotein cholesterol; BMI, body mass index; OR, odds ratio; CI, confidence interval; LAG, large arachnoid granulation

($P<0.001$), HDL-c ($P=0.04$), non-HDL-c ($P<0.001$), and TG ($P<0.001$).

Discussion

Blood lipids may be associated with the formation of multiple LAGs, which may result in CVSS [1, 25]. The multiple LAGs group showed higher levels of blood lipids than the non-multiple LAGs group, and the blood lipid indicators are interrelated. Multiple logistic regression indicated that elevated LDL-c, TC, and non-HDL-c had an independent association with multiple LAGs after controlling for potential confounding factors including BMI, age, sex, and onset-to-door time, whereas HDL-c was insignificant after adjustment. These results indicated that blood lipids might be independently associated with the formation of multiple LAGs and that elevated TC and LDL-c played important roles. The levels of blood lipids in the LAG group did not meet the diagnostic criteria for severe dyslipidemia. However, the values of LDL-c, TC, and TG in the LAG group were noticeably higher than those in the control group, which suggested that the LAG patients had an underlying lipid disturbance in the present study.

BMI was not identified as the factor that contributed to the formation of multiple LAGs in the enrolled patients. In this study, BMI was higher in the patients with multiple LAGs. BMI was closely related to CVSS and the corresponding IIH. Patients with higher BMI usually have a greater risk of dyslipidemia [3]. Therefore, it is necessary to control for the confounding effect of BMI. The present study found a linear correlation between BMI and both TG and HDL-c, but no association with TC or LDL-c, consistent with findings from previous studies [3, 26]. However, the present study indicated that TC and LDL-c were the main indicators affecting multiple LAGs. Furthermore, multifactorial logistic regression analysis revealed that LDL-c and TC were independently linked to multiple LAGs even after adjusting for BMI, and BMI was not associated with multiple LAGs. Together, these results suggested that BMI is probably not the primary factor contributing to the development of multiple LAGs,

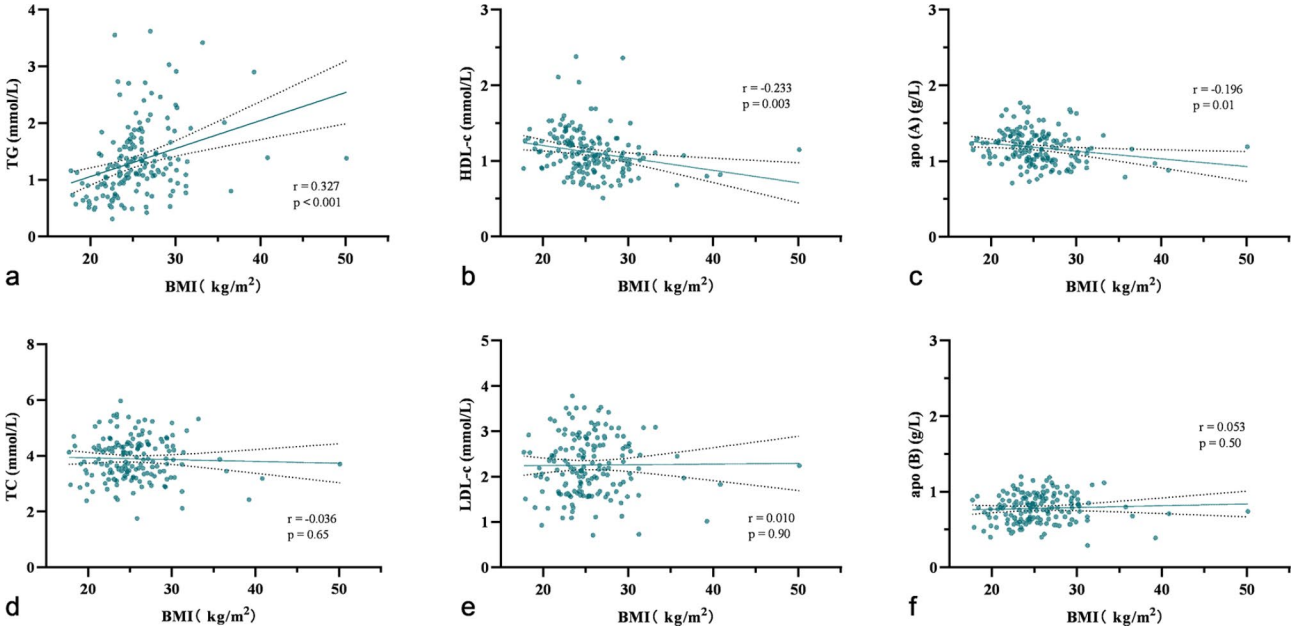

Fig. 4 Scatter plot of the correlation between blood lipids and BMI. BMI was linearly correlated with TG (**a**), HDL-c (**b**), and apo A (**c**), but it was not significantly associated with TC (**d**), LDL-c (**e**), or apo B (**f**). Abbreviations: TC, total cholesterol; LDL-c, low-density lipoprotein cholesterol; HDL-c, high-density lipoprotein cholesterol; apo A, apolipoprotein A, apo B, apolipoprotein B; BMI, body mass index

further supporting the relationship between blood lipids and the formation of multiple LAGs.

Lipid-lowering medications do not substantially impact the relationship between blood lipids and multiple LAGs. Because a small proportion of patients with LAGs were taking lipid-lowering medications for a long term due to hyperlipidemia, lipid-lowering medications were used as a covariate in the multivariable logistic regression analysis to exclude interference. Blood lipids were still significantly associated with multiple LAGs after adjusting for confounding factors. These results were consistent in the logistic regression analyses after the exclusion of individuals on long-term lipid-lowering medications therapy. Therefore, the inclusion of patients taking lipid-lowering drugs did not interfere with the assessment of the correlation between blood lipids and multiple LAGs in the present cohort. Moreover, exclusion may lead to the omission of patients with long-term hyperlipidemia, causing selection bias in the data. Based on the above results, the study did not exclude patients treated with long-term lipid-lowering drugs.

Dyslipidemia may lead to LAGs by inducing inflammation and endothelial dysfunction. Previous studies have found a link between hypercholesterinemia and CVST [15] and its recurrence [27, 28]. Studies on peripheral veins have revealed that dyslipidemia can lead to accelerated atherosclerosis in vein grafts after coronary artery bypass grafting surgery [10]. After pathological examination, intimal thickening and foam cell infiltration have been detected in the vein grafts [29]. Moreover, lipid-lowering therapy alleviates the progression of atherosclerosis in grafts [30]. Pathological examinations of the varicose saphenous vein have also revealed lipid deposition in the vessel wall [14]. These findings suggest that dyslipidemia may contribute to venous damage. Lipid accumulation can accelerate atherosclerosis through oxidative stress and inflammatory processes [31, 32]. Although the structure of veins is vastly different from that of arteries, damage to the endothelium by blood lipids may be consistent. In addition, LAGs exhibit reactive changes, including foam cell-rich infiltrates and notable inflammation [33]. Therefore, blood lipids may cause intracranial venous endothelial dysfunction and trigger local inflammatory reactions, leading to enlarged AG and cerebral venous outflow disturbances. According to the present findings, the screening programs for patients with LAGs should include blood lipid measurements. Future research should clarify the pathological mechanism and assess if lipid-lowering treatment reduces LAG formation risk.

Strengths and limitations

This present study found a correlation between dyslipidemia and LAGs. BB-MRI was used to evaluate the number and distribution of LAGs, which improved the diagnostic accuracy. These results provide new clinical ideas for the clinical treatment of LAGs. Nevertheless, the study still included several limitations. First, a small sample size was used in this retrospective analysis, which was carried out in a single center. The evidence

was inadequate to determine a causal link between multiple LAGs and blood lipids. The external validation by researchers from different centers and prospective, longitudinal studies are necessary to validate the findings. Second, because BMI does not precisely reflect the body fat percentage of an individual, the confounding effect of BMI on blood lipids may be undetermined in patients with LAG. More systematic indicators of body fat content are needed to assess the influence of obesity on LAG formation. Third, owing to image limitation, the rough classification of AGs was not sufficient to describe the differences in the sizes of AGs. In the future, computer-aided methods can be used to quantitatively measure the size of AGs. The prospective studies involving larger sample sizes are necessary to explore a link between blood lipids and multiple LAGs.

Conclusion

Blood lipids may be independently associated with the formation of multiple LAGs in venous sinuses which may result in CVSS, and elevated TC and LDL-c are the major influencing factors for multiple LAGs. Therefore, monitoring blood lipids and reducing TC and LDL-c may be necessary when patients show evidence of LAGs. These findings provided a new window to explore the underlying etiology of LAG, which might be a potential target for treating the non-thrombotic CVSS associated with LAG.

Abbreviations

AG	Arachnoid granulation
LAG	Large arachnoid granulation
CVSS	Cerebral venous sinus stenosis
CVST	Cerebral venous sinus thrombosis
IIH	Idiopathic intracranial hypertension
BB-MRI	Black blood magnetic resonance imaging
LDL-c	Low-density lipoprotein cholesterol
TC	Total cholesterol
TG	Triglyceride
HDL-c	High-density lipoprotein cholesterol
apo A	Apolipoprotein A
apo B	Apolipoprotein B

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12944-024-02341-4.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

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Author contributions

YG and ZW wrote the first draft of the manuscript; YG, DZ, and MG performed the material preparation, data collection, and statistical analysis; ZW, RM, and XJ contributed to imaging assessments; RM and YD wrote sections of the manuscript and contributed to manuscript revision; XJ and RM contributed conception and design of the study; RM takes full responsibility for the data, the analyses and interpretation, and the conduct of the research. All authors read and approved the submitted version.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The study was approved by the Institutional Ethic Committee of Xuanwu Hospital, Capital Medical University, and was conducted according to the guidelines laid down in the Declaration of Helsinki. Prior to any study procedure, written informed consent was obtained from all participants.

Competing interests

The authors declare no competing interests.

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