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Cerebral Microbleeds and Antiplatelet Therapy in Mongolian and Han Patients with Ischemic Cerebrovascular Disease

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Objective: To analyze the differences in cerebral microbleeds (CMBs) and their correlation with intracerebral hemorrhage (ICH) in Mongolian and Han Chinese patients with ischemic cerebrovascular disease.

Methods: A total of 160 patients with ischemic cerebrovascular disease who took aspirin or clopidogrel for over one year were retrospectively analyzed, including 80 Mongolian and 80 han patients. The incidence, number, and distribution of CMBs were compared between groups. Logistic regression was used to identify risk factors for the occurrence of cerebral hemorrhage.

Results: The detection rate of CMBs was significantly lower in Mongolian patients compared to Han patients (P = 0.040). Mongolian patients had a higher distribution of CMBs in the deep or infratentorial regions (66.6% vs 58.1%), while Han patients had a higher lobar distribution (P = 0.007). Prolonged antiplatelet therapy (over 3 years) was a risk factor for CMB development in both groups and was also linked to an increased risk of ICH. Patients with a higher number of CMBs had a greater likelihood of experiencing ICH.

Conclusion: Mongolian patients had a lower likelihood of developing CMBs than Han patients, but with a higher deep or infratentorial distribution. The presence of CMBs, especially with long-term antiplatelet therapy, is a significant predictor of ICH. No significant difference in ICH risk was found between ethnic groups. Close monitoring of patients with CMBs during prolonged antiplatelet therapy is crucial to reduce hemorrhagic events.

Keywords: cerebral microbleeds, antiplatelet drugs, intracerebral hemorrhage, white matter hyperintensities

Introduction

Cerebral microbleeds (CMBs) are a special subtype of cerebral small vessel disease (CSVD), which is a subclinical injury to the brain parenchyma caused by leakage hemorrhage of small blood vessels in the brain due to a variety of factors, and ferritin-containing deposition around the diseased cerebral small blood vessels is the main pathological feature is the presence of ferritin deposits around the small cerebral vessels.[1] Since cerebral microhemorrhage may be caused by erythrocyte leakage from small arteries and capillaries, cerebral microhemorrhage not only predicts the onset of progression of cerebral small-vessel lesions, but it is also an important risk factor for the development of hemorrhagic outcomes after cerebral ischemia. Studies have shown that blood leakage to form cerebral microhemorrhage foci after cerebral microhemorrhage in patients with acute cerebral infarction can cause clot-occupying effects, which can compress the surrounding neural tissues, resulting in aphasia, hemiparesis, cognitive impairment in Parkinson's disease, and confusion,[2] which will not only increase the cognitive decline of patients and increase the risk of dementia,[3,4] but also the accumulation of the number of CMBs is also the recurrence of cerebral hemorrhage (ICH).[4,5] With the development of imaging equipment and the wide application of susceptibility weighted imaging (SWI) in the central nervous system, researchers have found that the detection rate of CMBs is not low in different disease populations. The

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detection rate of CMBs in different disease populations is not low. Therefore, it is important to actively search for the pathogenic factors of CMBs in order to sustain the treatment of patients.

Antiplatelet drugs, the most common of which are aspirin and clopidogrel, are widely used to treat patients with atrial fibrillation or thromboembolic disease, but there is a risk of major bleeding or even non-material clearance. Although intracerebral hemorrhage is uncommon, the risk of death increases dramatically once intracerebral hemorrhage occurs.[6] Previous studies have shown that the use of anticoagulant drugs will increase the risk of intracerebral hemorrhage (ICH) in patients with CMBs, and there are some hidden dangers in the prescription of anticoagulant drugs in patients with CMBs.[5,7,8] However, whether the use of anticoagulants will promote the prevalence of CMBs remains controversial. Some meta-analyses have shown that the use of anticoagulants is associated with the prevalence of CMBs.[9–11] However, there is also a study[12] found that conventional oral antiplatelet therapy has nothing to do with the development of CMBs, regardless of whether the patient has cerebrovascular disease, under the premise of good control of hypertension, the use of antiplatelet drugs is reasonable. At present, there is still no expert consensus on the safety of platelet drug therapy, especially in reducing the risk of recurrence in patients with ischemic stroke and its potential risk of causing cmb and cerebral hemorrhage. This is still a clinical problem and requires more experimental data to verify.

At present, most of the studies on CMBs in China are aimed at the Han population, while there are few studies on the Mongolian population. Mongolians belong to China ‘s ethnic minorities. They live in grasslands for a long time. The Mongolian diet is mainly composed of milk and meat, and the drinking of alcohol is also more prominent. Previous studies have also shown that the prevalence of cardiovascular disease in the Mongolian population is higher than that in the Han population.[13–15] These ethnic-specific factors, including dietary habits and genetic predispositions, may contribute to the distinct patterns of cerebrovascular disease observed in Mongolian and Han patients. Understanding these differences is essential for developing tailored prevention and treatment strategies for CMBs in different ethnic groups. However, there is no more detailed study on whether ethnic differences, life differences, dietary structure differences, and ethnic genetic factors have an impact on the CMBs after oral administration of aspirin or clopidogrel. The purpose of this study is to analyze whether ethnic differences will increase the risk of cerebral hemorrhage events in patients with ischemic cerebrovascular disease after taking aspirin or clopidogrel, and to provide reference for analyzing the reasons behind ethnic differences and clinical prevention of CMBs in patients with ischemic cerebrovascular disease.

Material and Methods

Study Subjects

This study is a retrospective study. The sample size was calculated based on the following assumptions: a two-sided α level of 0.05, a power of 80% (β = 0.20), and an estimated difference in the proportion of cerebral microbleeds (CMBs) between Mongolian and Han patients. Based on preliminary studies, we assumed the proportion of CMBs in Han patients would be approximately 60% and in Mongolian patients 40%. Using these assumptions, the required sample size was calculated to be 73 patients per group, for a total of 146 patients. To account for potential dropouts or incomplete data, we enrolled 80 patients from each group, resulting in a total of 160 patients. This sample size is sufficient to detect a statistically significant difference between the two populations. The subjects of the study were 160 patients with ischemic cerebrovascular disease who were treated with antiplatelet drugs in the Affiliated Hospital of Inner Mongolia Medical University from January 2020 to December 2021. The subjects included 80 Mongolian patients and 80 han patients. Inclusion criteria: (1) Patients have been diagnosed as ischemic stroke or transient ischemic attack, the diagnostic criteria refer to the Chinese Guidelines for the Diagnosis and Treatment of Acute Ischemic Stroke.[16,17] The severity of the disease was classified according to the Clinical Neurological Deficit Score of Stroke Patients in China.[18] (2) Patients were treated with oral aspirin (100 mg/day) or clopidogrel (75 mg/day) for more than 1 year. (3) The patient was a native resident of Inner Mongolia, Han or Mongolian (three or more generations), with complete basic information. Exclusion criteria: (1) Receiving anticoagulant or thrombolytic therapy. (2) Receive aspirin and clopidogrel combination therapy. (3) Cerebral amyloid angiopathy. (4) Severe heart, liver, lung, kidney dysfunction. (5) Patients with craniocerebral trauma, malignant tumor, hematological diseases, Parkinson ‘s disease and other major diseases. (5) There are motion artifacts in MRI examination, and MRI examination cannot be completed for any reason. (6) Incomplete clinical

data. We included a total of 45 patients with intracerebral hemorrhage (ICH) in the study. Additionally, 62 patients had received antiplatelet therapy for more than 3 years. Specifically, 38% of Mongolian patients and 41% of Han patients had been on antiplatelet therapy for over 3 years, highlighting no statistically significant difference in therapy duration between the two ethnic groups. This study was in line with the principles of the "Helsinki Declaration" and approved by the Ethics Committee of the Affiliated Hospital of Inner Mongolia Medical University.

MRI Examination

A Siemens Skyra 3.0 T magnetic resonance imaging system (Siemens Healthcare, Germany) and associated laboratory equipment were used. All patients underwent the following examinations: T1-weighted imaging (T1WI) (repetition time, 1660 ms; echo time, 10 ms; field of view, 23×23 cm; matrix, 240x320; slice thickness, 5.5 mm), T2-weighted imaging (T2WI) (repetition time, 5500 ms; echo time, 117 ms; field of view, 23×23 cm; matrix, 320x320; slice thickness 5.5 mm), diffusion-weighted imaging (DWI) (b=1000; repetition time, 3530 ms; echo time, 81 ms; field of view, 23×23 cm; matrix, 160x160; slice thickness, 5.5 mm), and SWI (repetition time, 27 ms; echo time, 20 ms; field of view, 22×22 cm; matrix, 220x256; slice thickness, 1.5 mm).

MRI images were independently evaluated by two experienced imaging physicians, provided that the analysts were blinded to the basic information and clinical history of the patients in this study, and recorded the number of CMBs occurring at each site in patients with CMBs. In case of dispute regarding the results of the analysis, the decision was made by consultation between the two physicians. The diagnostic criteria for CMBs[19] widely used in clinical practice were as follows: (1) hyposignal or signal-deficient foci with a round or ovoid morphology on SWI images; (2) clear margins of the lesion; (3) a diameter of the lesion between 2 and 5 mm, with a maximum of no more than 10 mm; and (4) no edematous signals around the lesion, and at least 1/2 of it was surrounded by brain parenchyma. Some imaging manifestations similar to CMBs need to be excluded: (1) tubular or linear low signal representing vascularization or resorption after massive hemorrhage; (2) cavernous hemangiomas; (3) flow-void signals of soft meningeal vessels with the same signal intensity as that of CMBs, and its cross-sectional morphology is also circular, which can be differentiated by its continuity with other dimensions; and (4) calcification or iron deposits are low-signal in areas such as bilateral basal ganglia, choroid plexus, and pineal body, which can be differentiated from CMBs by the phase images, the specific sites, and the morphological features. In addition, CMBs were assessed using a visual scoring system, including the microbleed anatomical rating scale (MARS),[20] and the brain observer microbleed scale (BOMBS),[21] which strictly categorized CMBs according to their anatomical location into lobar CMBs (cortical and subcortical white matter), and deep or infratentorial CMBs (basal ganglia, thalamus, internal capsule, external capsule, corpus callosum, periventricular white matter, brainstem, and cerebellum), as shown in Figure 1. CMBs are graded using a scoring system into 4 classes: None (0), Mild (1–2), Moderate (3–10), and Severe (>10).[22] On T2WI images, the severity of WMHs was rated as Grade 0 (absence), Grade 1 (punctate foci), Grade 2 (initial confluence of foci) and Grade 3 (large confluence of foci) according to the Fazekas scale.[23] (Figure 2).

Other Clinical Information Collection

Other clinical information was collected according to vascular risk factors of Orken et al.[24] The collected data mainly included the patient 's age, gender, any history of cerebral hemorrhage after oral antiplatelet therapy, whether they had hypertension, diabetes, hyperlipidemia, smoking history, drinking history, whether leukoaraiosis (WMHs) was found in MRI examination, the specific dose of aspirin or clopidogrel, and the time of medication.

The definition of ICH refers to non-traumatic cerebrovascular rupture, resulting in substantial accumulation of blood in the brain. Referring to the diagnostic criteria of the Chinese Guidelines for the Diagnosis and Treatment of Cerebral Hemorrhage (2019),[25] MRI showed hemorrhagic lesions suggesting ICH.

Statistical Methods

The data were analyzed by SPSS 25.0 statistical software (SPSS Inc., Chicago, United States). The mean ± standard deviation $(\overline{X}\pm \text{SD})$ was used to describe the measurement data, and the number of cases and percentage were used to represent the count data. The K-S fitting goodness of fit method was used to test the normality. The *t*-test was used to

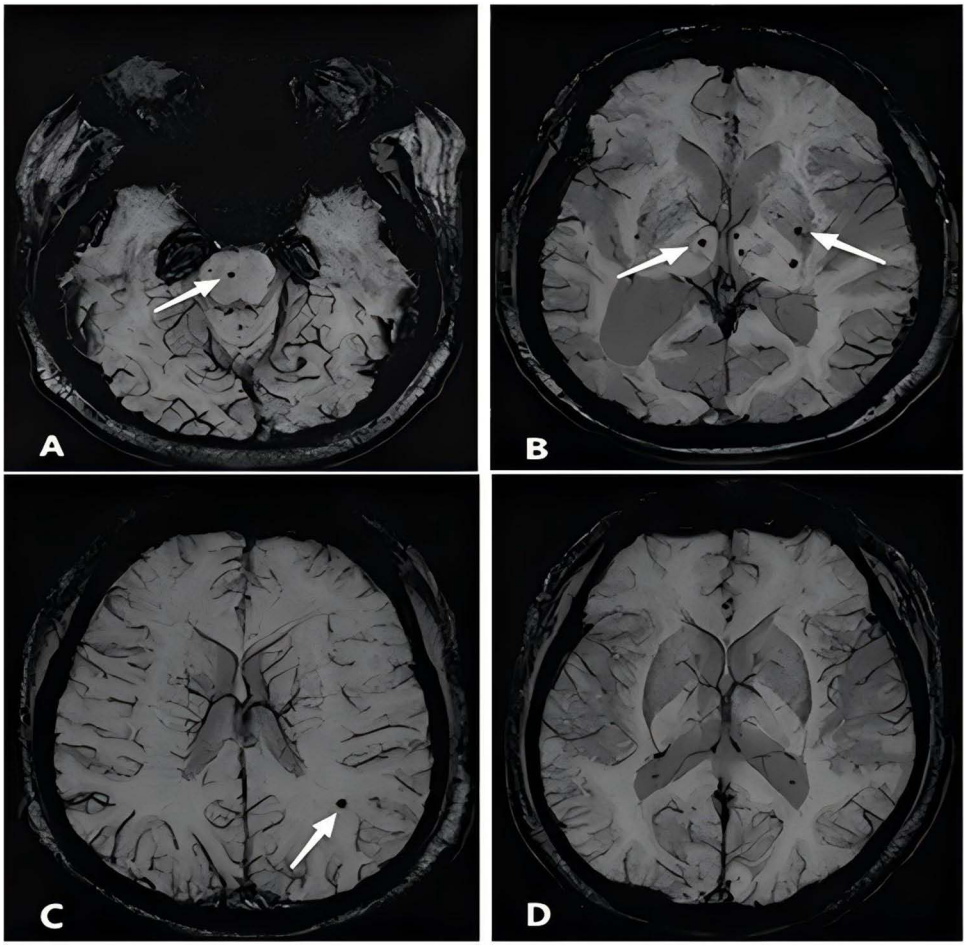

Figure 1 SWI minimal signal projection map (minIP). (**A**) pontine CMBs; (**B**) multiple CMBs in bilateral basal ganglia region; (**C**) left parietal subcortical white matter CMBs; (**D**) no CMBs. White arrows indicate the locations of typical cerebral microbleed (CMB) lesions, which appear as hypointense, round, or oval spots.

compare the measurement data that obeyed the normal distribution between groups, and the chi-square test was used to compare the count data between groups. Multivariate logistic regression was used to analyze the influencing factors of CMBs and ICH. $P < 0.05$ was considered statistically significant.

Results

Baseline Characteristics of Study Participants

160 patients with ischemic cerebrovascular disease were included in this study, including 80 patients of Mongolian ethnicity (mean age 64.99 ± 10.74) and 80 patients of Han ethnicity (mean age 65.21 ± 8.75). No statistically significant differences were found between the two groups in terms of age, sex ratio, hypertension, diabetes mellitus, hyperlipidaemia, history of smoking, history of alcohol consumption, history of WMHs, BMI≥24, severity of stroke, medication compliance, types of antiplatelet drug use and antiplatelet drug treatment time between the two groups ($P > 0.05$) (See Table 1).

Detection Rate of CMBs in Mongolian and Han Chinese Patients

The detection rate of CMBs in Mongolian patients treated with antiplatelet drugs was 42.5%, and the detection rate of CMBs in Han patients was 58.8%. Chi-square test results showed that there was a statistically significant difference in the detection rate of CMBs between the two groups ($P < 0.05$), and the detection rate of Mongolian patients was lower than that of Han patients. There was no significant difference in the severity of CMBs between the two groups. From the perspective of the distribution of CMBs, there was a statistically significant difference in the distribution of CMBs between Mongolian

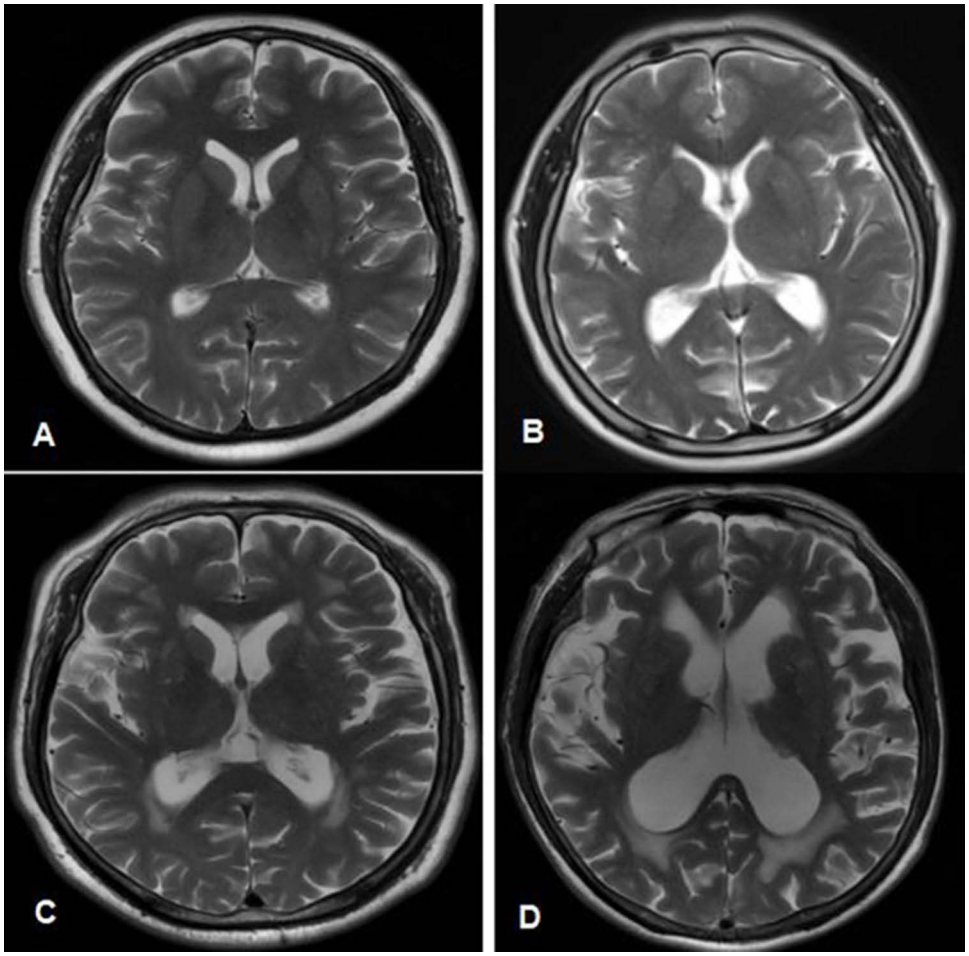

Figure 2 T2-weighted images. (**A**): level 0 (absent); (**B**): level 1 (punctate lesions); (**C**): level 2 (initial confluence of lesions); (**D**): level 3 (substantial confluence of lesions).

and Han patients ($P < 0.05$). The CMBs of Mongolian patients in the deep/infratentorial area were more than those of Han nationality, while the CMBs in the brain lobe area were less than those of Han nationality. (See Table 2).

Analysis of the Influencing Factors of CMBs

Multivariate Logistic regression analysis showed that there was a correlation between ethnicity and long duration of antiplatelet therapy and CMBs (Nationality, OR = 6.30, 95% CI: 1.32–30.18; more than 3 years of drug treatment, OR = 4.58, 95% CI: 1.76–11.89). After adjusting for age, gender, smoking history, hypertension, diabetes and WMHs, multivariate logistic regression analysis showed that there was a correlation between Han nationality and long duration of antiplatelet therapy and CMBs (Nationality, OR = 5.60, 95% CI: 1.06–12.29; more than 3 years of drug treatment OR = 5.10, 95% CI: 1.39–18.65), the difference was still statistically significant.(See Table 3).

Analysis of the Influencing Factors of ICH

Multivariate logistic regression analysis showed that there was a correlation between the presence of CMBs, the prolongation of antiplatelet therapy time and cerebral hemorrhage (CMBs, OR = 5.73,95% CI: 1.65–19.86; the duration of drug treatment was more than 3 years: OR = 4.20; 95% CI: 1.30–13.62). After adjusting for age, gender, hypertension and diabetes, the results showed that the correlation between the presence of CMBs and long-term antiplatelet therapy for cerebral hemorrhage was still statistically significant (CMBs, OR = 4.45,95% CI: 1.09–18.19; the duration of drug treatment was more than 3 years, OR = 4.13,95% CI: 1.06–16.17). There was no significant difference in the incidence of cerebral hemorrhage between different ethnic groups in both cases. (See Table 4). Our study demonstrated that the

Table 1 Baseline Characteristics of Mongolian and Han Chinese Patients

Characteristic	Mongolian (N = 80)	Han Chinese (N = 80)	t/χ^2	*P*
Age, y, $\overline{X}\pm$ SD	64.99 ± 10.74	65.21 ± 8.75	0.145	0.885
Gender, male, n (%)	45 (56.3)	50 (62.5)	0.648	0.421
Hypertension, n (%)	54 (67.5)	44 (55.0)	2.633	0.105
Diabetes, n (%)	19 (23.8)	24 (30.0)	0.795	0.373
Hyperlipidemia, n (%)	38 (47.5)	30 (37.5)	1.637	0.201
Smoking, n (%)	25 (31.3)	33 (41.3)	1.731	0.188
Alcohol addiction, n (%)	28 (35.0)	20 (25.0)	1.905	0.168
BMI≥24, n (%)	19(23.75)	15(18.75)	0.598	0.440
Severity of stroke				
Minor, n (%)	17(21.25)	14(17.50)		
Moderate, n (%)	45(56.25)	50(62.50)		
Severe, n (%)	18(22.50)	16(20.00)	0.671	0.715
WMHs, n (%)	65(81.25)	68(85.00)	0.204	0.615
Treatment duration, n (%)				
≤ 3 years	47(58.75)	45(56.25)		
> 3 years	33(41.25)	35(43.75)	0.102	0.704
Medication compliance				
Regular	63(78.75)	65(81.25)		
Intermittent	17(21.25)	15(18.75)	0.156	0.693
Types of drug use				
Aspirin	47(58.75)	44(55.00)		
Clopidogrel	33(41.25)	36(45.00)	0.043	0.836

Abbrevation: SD, standard deviation.

Table 2 Detection Rate of CMBs in Mongolian and Han Chinese Patients

Nationality	Mongolian (N = 80)	Han Chinese (N = 80)	χ^2	*P*
CMBs (+), n (%)	34 (42.5)	47 (58.8)	4.226	0.040
Severity of CMBs				
Mild, n (%)	6 (17.6)	10 (21.3)		
Moderate, n (%)	16 (47.1)	21 (44.7)		
Severe, n (%)	12 (35.3)	16 (34.0)	0.165	0.915
Distribution of CMBs				
Deep/infratentorial, n (%)	23 (67.6)	27 (57.4)		
Lobar, n (%)	11 (32.4)	20 (42.6)	7.159	0.007

Table 3 Logistic Regression Analysis of CMBs in Patients with Different Nationalities, Antiplatelet Drug Treatment Time and CMBs

Variable	Model	OR	95% CI	*P*
Nationality	Model 1	6.30	1.32–30.18	0.021
	Model 2	5.60	1.06~12.29	0.041
Treatment duration (> 3 years)	Model 1	4.58	1.76~11.89	0.002
	Model 2	5.10	1.39~18.65	0.014

number of CMBs significantly impacts the risk of intracerebral hemorrhage (ICH), with a higher CMB count being associated with an increased likelihood of ICH. This suggests a potential dose-response relationship between CMB burden and hemorrhagic events.

Table 4 Logistic Regression Analysis of the Relationship Between CMBs, Treatment Time and Cerebral Hemorrhage in Patients with Different Nationalities

Variable	Model	OR	95% CI	*P*
Nationality	Model 1	5.10	0.21–8.78	0.875
	Model 2	3.22	0.91–11.31	0.676
CMBs	Model 1	5.73	1.65–19.86	0.006
	Model 2	4.45	1.09–18.19	0.038
Treatment duration (> 3 years)	Model 1	4.20	1.30–13.62	0.017
	Model 2	4.13	1.06–16.17	0.042

Discussion

The results of this study showed that the detection rate of CMBs in Han patients after antiplatelet therapy was higher than that in Mongolian patients. At the same time, there were differences in the anatomical distribution of CMBs. Different races and the time of drug use were predictors of CMBs, but there was no statistical difference in the possibility of cerebral hemorrhage in patients with different nationalities. The time of drug use and CMBs ultimately affected the occurrence of cerebral hemorrhage in patients.

In the current study, few studies have discussed the different conditions of cerebrovascular diseases in different ethnic groups. In our study, the detection rate of CMBs in Chinese Han patients after antiplatelet therapy was higher than that in Mongolian patients. This result may be due to racial differences, vascular anatomical variations, various clinical risk factors and genetic differences, or differences in target organ damage caused by different blood pressure changes. In previous studies on vascular diseases, it was found that the prevalence of hypertension was different between Mongolian and Han nationalities. Different environmental exposures, race-specific genetic susceptibility, and gene-environment interactions may be the reasons for the different prevalence of hypertension. Some studies[13,14,26] have also found that this may be related to the genetic characteristics between the two nationalities. Hypertension is the first risk factor for cerebrovascular hemorrhage. Studies have shown that hypertension can destroy the structure of vascular endothelial cells[27] and eventually lead to organic lesions of blood vessels.[28] The Mongolians have lived on the prairie since ancient times, but they still retain many unique folk customs in the Mongolians. For example, Mongolian people are mainly meat and dairy products, and they consume more animal fat, which easily leads to higher blood lipids.[29] At the same time, Mongolians like to drink high alcohol.[30] The intermediate metabolite acetaldehyde of alcohol in the body has strong lipid peroxidation and toxic effects, which can damage the membrane system of vascular endothelial cells and promote arteriosclerosis. However, this study found that the Han nationality was more likely to have CMBs, which may be because all the samples in this study were from the same place. The two populations studied lived in a similar environment, and the difference in living habits gradually narrowed.

We found that there was no significant difference in the severity of CMBs between Mongolian and Han patients, but there was a difference in the anatomical distribution of CMBs, which may reflect potential microvascular lesions. Relevant studies have shown that the location of CMBs lesions in deep brain tissue displayed by SWI in patients with cerebral infarction is related to the location of primary ICH.[13] Studies[31,32] have shown that arterial lesions associated with hypertension mainly affect the perforating arteries and their branches that supply the basal ganglia and deep white matter, resulting in lipid hyaline and fibrovitreous changes, which are the main causes of CMBs in the deep and infratentorial areas of the cerebral hemisphere. Cerebral amyloid angiopathy is an important cause of CMBs in strict lobar locations. Our study showed that Mongolian patients with ischemic cerebrovascular disease treated with antiplatelet drugs had significantly more CMBs in the deep or infratentorial area than Han patients. It is speculated that the reason may be related to the high fat and high protein diet of Mongolian patients and the influence of BMI[4] on blood pressure. A study[33] has shown that systolic blood pressure, diastolic blood pressure and age are independent risk factors for CMBs in patients with hypertension. In addition, there is a certain correlation between the number of CMBs and the grade of hypertension. The cerebral cortex and basal ganglia are

the preferred sites for the occurrence of CMBs, indicating that there is a special relationship between CMBs and symptomatic ICH, and there is a similar mechanism in hypertensive ICH. There are many conduction fibers and functional units in the basal ganglia region, and CMBs may impair specific cellular functions. Werring et al[34] proposed that cognitive and executive dysfunction is related to CMBs in the frontal and basal ganglia regions, which may be caused by damage to subfrontal cortical fibers. CMBs may be a potential microvascular marker for cognitive impairment. In addition, cerebral amyloid angiopathy is a vascular injury caused by progressive deposition of amyloid β-protein (Aβ) in small arteries and capillary walls in cortex and pia mater,[35] which is the main cause of CMBs and ICH in cerebral lobes. A study[36] showed that the frequency of APOEε4 allele in Han ischemic stroke patients was significantly higher than that in healthy people. In our study, the CMBs in the cortex and subcortical white matter of Mongolian patients with ischemic cerebrovascular disease were less than those of Han patients. The results of this study suggest that there are differences in genes between Mongolian and Han patients with ischemic cerebrovascular disease.

In the Mongolian and Han ischemic cerebrovascular disease population, the use of antiplatelet drugs is related, and the risk of CMB and macrobleeding is further increased in patients with long-term (more than 3 years) antiplatelet therapy.[4,12,37] Naka et al[38] retrospectively analyzed the antiplatelet therapy of 412 patients with cerebral hemorrhage and 1502 patients with ischemic stroke, and analyzed the effect of specific antiplatelet drugs on CMBs, including aspirin, clopidogrel, cilostazol and ticlopidine. The results showed that there was a significant correlation between the use of antiplatelet drugs and the presence of CMBs in patients with cerebral hemorrhage, but no such correlation was found in patients with ischemic stroke. A recent study[39] in China, including 2654 patients with ischemic cerebrovascular disease, found that there was no significant correlation between aspirin treatment and duration of medication and the presence and severity of CMBs. Therefore, although our study suggests that the use of antiplatelet drugs has predictive significance for CMBs, this conclusion is still controversial. Patients who use antiplatelet drugs for long-term treatment should be cautious.

This study has several limitations. First of all, the retrospective nature of the study may limit the generalizability of the results, as it relies on previously collected data and may not fully capture all variables relevant to the current patient population. In addition, the collection of retrospective SWI images cannot determine the occurrence time of CMBs, and CMBs that occurred before the use of antiplatelet drugs cannot be ruled out. Secondly, potential sample bias, particularly related to the selection of participants from a single geographic region, could affect the representativeness of the findings. Finally, this study did not include data on the presence of CMBs before the initiation of antiplatelet therapy, which limits our ability to definitively confirm whether antiplatelet therapy contributes to the development or prevalence of CMBs. However, the results of this study require further refinement and we look forward to more large-sample prospective trials on the benefits and risks of antiplatelet therapy in patients with CMBs to confirm our findings.

Conclusion

This study found that the use of antiplatelet drugs in patients with ischemic cerebrovascular disease is a risk factor for CMBs in patients. At the same time, the detection rate of CMBs in Mongolian patients is lower than that in Han patients, and the difference in the distribution of CMBs between the two is statistically significant. The distribution of CMBs in Mongolian patients is more than that in Han nationality in the deep/infratentorial region, while the CMBs in the lobe region are less than those in Han nationality. In addition, the duration of drug use and the presence of CMBs were associated with an increased risk of ICH, but there was no statistically significant difference in the risk of ICH between Mongolian patients receiving platelet drug therapy and Han patients. The results of this study need further detailed research, and we look forward to more large sample prospective trials on the benefits and risks of antiplatelet therapy in patients with CMBs to confirm our conclusions.

Data Sharing Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of the Affiliated Hospital of Inner Mongolia Medical University. Written informed consent was obtained from all participants.

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Disclosure

All of the authors had no any personal, financial, commercial, or academic conflicts of interest separately.

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