Original Article

Effect of dominant vertebral artery angle on basilar artery curvature and plaque

Yangchen Li[1], Runcai Guo[2], Xuebing Zhang[2], Yang Yang[1], Yuchun Yan[1], Xinyu Yuan[1], Ce Wang[2], Sheng Xie[2]

[1]Department of Radiology, Children's Hospital, Capital Institute of Pediatrics, Beijing, China; [2]Department of Radiology, China-Japan Friendship Hospital, Beijing, China

Contributions: (I) Conception and design: S Xie, Y Li; (II) Administrative support: S Xie, Y Yang; (III) Provision of study materials or patients: R Guo, X Zhang; (IV) Collection and assembly of data: Y Li, C Wang; (V) Data analysis and interpretation: Y Li, X Yuan, Y Yan; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Sheng Xie, MD. Department of Radiology, China-Japan Friendship Hospital, No. 2 East Yinghua Road, Chaoyang District, Beijing 100029, China. Email: xs_mri@126.com.

Background: Quantification of vertebral arteries can provide insights into basilar curvature and plaque. Therefore, this retrospective study aimed at identifying the dominant vertebral artery (VA) causing basilar artery (BA) curvature and to further quantify the effect of dominant VA angle on BA curvature and BA plaque using high-resolution magnetic resonance imaging (HRMRI) and 3-dimensional time-of-flight magnetic resonance angiography (3D-TOF-MRA).

Methods: This retrospective analysis included 521 participants who underwent HRMRI in the China-Japan Friendship Hospital from November 2015 to October 2021 for neurological symptoms or signs. The VA angle more related to BA curvature was defined as the dominant VA angle. Multivariable linear regression analysis was used to evaluate the relationship between the dominant VA angle and mid-BA angle, while multivariable logistics regression was used to evaluate the influence of the dominant VA angle and clinical risk factors on BA plaque.

Results: In total, 259 participants were included in this study (mean age 53.71±13.12 years; 146 males). The balanced-type participants had a significantly lower probability of BA plaques (P<0.001). The Chi-squared test showed that the BA curvature direction was significantly associated with the side with larger VA diameter (P<0.001) and larger VA angle (P<0.001). As a result, the VA angle on the side with the larger diameter or the larger VA angle when the diameters were similar was considered to be the dominant VA angle. The dominant VA angle was independently correlated with the mid-BA angle (P<0.001). In addition, the dominant VA angle was also an independent risk factor for BA plaque. Additionally, 80° was the cutoff value of the dominant VA angle, and when the dominant VA angle was greater than 80°, the risk of BA plaque increased about 18-fold [odds ratio, 18.951; 95% confidence interval (CI): 4.545–79.026; P<0.001].

Conclusions: The dominant VA angle was independently associated with BA plaque, and a dominant VA angle greater than 80° may be a marker for a high risk of posterior circulation atherosclerosis.

Keywords: Dominant vertebral artery; high-resolution magnetic resonance imaging (HRMRI); basilar artery plaque

Submitted Jan 16, 2023. Accepted for publication Jun 26, 2023. Published online Jul 10, 2023.
doi: 10.21037/qims-23-74
View this article at: https://dx.doi.org/10.21037/qims-23-74

Introduction

Strokes of the posterior circulation supplied by the vertebrobasilar arteries account for about 20–25% of all ischemic stokes and remain a significant cause of patient disability and mortality (1,2). Local branch occlusion is a relatively more important mechanism in posterior circulation diseases and is most frequently associated with the basilar artery (BA), which accounts for up to 64% of these occlusions (3). The presence of atherosclerotic plaque in the BA makes small and short perforated vessels prone to occlusion. A growing body of evidence suggests that BA plaque is associated with symptomatic pontine infarction (4,5), while BA plaque assessed using high-resolution magnetic resonance imaging (HRMRI) is associated with progressive motor deficits in patients with acute unilateral pontine infarction (6).

Vascular geometric characteristics may influence local hemodynamic forces and play an important role in the development of atherosclerotic plaques (7). Recent studies have used vascular geometrical risk factors to explain the presence of atherosclerotic plaques. For example, Ravensbergen *et al.* employed autopsy and a series of junction models to prove that vertebrobasilar geometry affects hemodynamics and that atherosclerotic plaques frequently occur in regions with low wall shear stress and/or complex flow patterns (8,9); The curvature of the BA is related to the presence of plaque, its location, and mean wall thickness (10-13). The vertebrobasilar system is the only site in the human body where a third artery is formed via the merging of 2 arteries with greater degree of geometric variation than the anterior circulation (10). The curvature of the BA is largely caused by a vertebral artery (VA) with asymmetrical blood flow and an internal diameter (14). Therefore, it is crucial to better understand the relationship between the VA and BA curvature and plaque before the occurrence of ischemic events. However, few studies have directly explored which side of the VA angle is the dominant angle causing BA curvature or have quantified the effect of the VA on BA curvature and plaque. The aim of this study was thus to propose a new classification method for vertebrobasilar configuration, to find the dominant angle causing BA curvature, and to further explore the effect of the dominant VA angle in on mid-BA angle and BA plaque. We present this article in accordance with the STROBE reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-23-74/rc).

Methods

Study population

This retrospective analysis included 521 participants who had undergone HRMRI in the China-Japan Friendship Hospital from November 2015 to October 2021 for neurological symptoms or signs such as headache, dizziness, giddiness, vertigo, or stroke. Some patients in this study overlap with those of previously published studies (399/521). However, the content of this study does not overlap with previous work and will not affect the results of this study. The exclusion criteria were: (I) clinical and imaging signs suggesting nonatherosclerotic vascular disease (e.g., dissection, moyamoya disease, or vasculitis); (II) unilateral or bilateral internal carotid artery severe stenosis or occlusion; (III) unilateral or bilateral VA stenosis or not seen on magnetic resonance angiography (MRA) or HRMRI; (IV) a minimum diameter of the BA greater than 4.5 mm (15); (V) vertebrobasilar artery fenestration; (VI) subclavian steal syndrome detected by Doppler ultrasound; (VII) poor imaging quality, and (VIII) incomplete clinical data. The patients' demographic and clinical information were collected from the medical records. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and approved by the China-Japan Friendship Hospital Institutional Review Board (No. 2021-BZR-24). Individual consent for this retrospective analysis was waived.

HRMRI protocol

The HRMRI sequence was then performed by using a 3-dimensional (3D) volumetric isotropic turbo spin-echo acquisition (VISTA; Philips Healthcare, Best, the Netherlands) optimized for flow suppression and intracranial vessel wall delineation. A standard magnetic resonance imaging (MRI) protocol was used that included pre- and post-contrast HRMRI imaging and 3D time-of-flight MRA (3D-TOF-MRA) sequences. All HRMRI examinations were performed using a 3-Tesla MRI scanner (Ingenia; Philips Healthcare) with a 15-channel phased-array head coil. The images were acquired in a traversal plane to cover the major intracranial arteries identified on 3D-TOF-MRA. The image scanning parameters were as follows: repetition time/echo time =800 ms/21 ms, field of view =180×180×105 mm^3, matrix =300×300×350, and number of excitations =2. The acquisition voxel volume

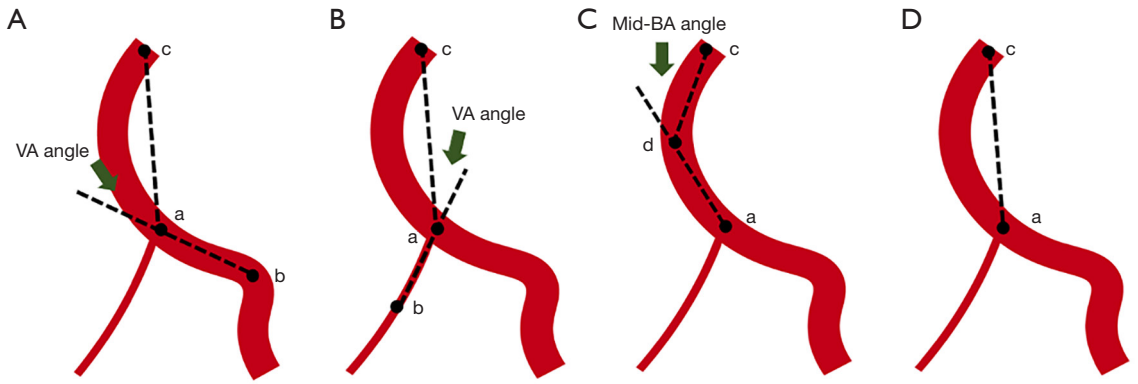

Figure 1 Schematic diagram of the angle and the basilar artery curvature direction measurement. (A,B) The measurement of the VA angle: point a is the vertebrobasilar junction, point b is the closest and most curved point of the VA from BA, and point c is the top of the BA. The imaginary lines were drawn from point a to point b and point c. The acute angle between these 2 lines was considered the VA angle. (C) The measurement of the mid-BA angle: point d is the most curved point of the BA. The imaginary lines were drawn from point d to point a and point c. The acute angle between these 2 lines was considered the mid-BA angle. (D) The measurement of the BA curvature direction: a line was drawn between point c and point a as a reference to decide the side of the BA curvature. This picture shows the right deviation of the BA. VA, vertebral artery; BA, basilar artery.

was 0.5×0.6×0.5 mm^3, and the reconstruction voxel volume was 0.5×0.5×0.5 mm^3. The short axial cross sections were constructed automatically with a 0.5-mm slice thickness.

Image analysis

All imaging analyses were blinded to clinical information. Specifically, assessors of plaque were blinded to 3D-TOF-MRA. The methods of measurement are described below.

The diameter measurement

This included vertebral arteries on both sides. In the anteroposterior view of 3D-TOF-MRA, the whole diameter was measured transverse to the long axis of the vessel. The diameter of each vessel was calculated as the average of the measurements made at 3 consecutive points, 3-mm apart, starting from the vertebrobasilar junction (both VA and the BA) (14). The images were independently measured on a digital picture archiving and communication system workstation by 2 radiologists (3 and 5 years of experience, respectively), and the average was taken as the result.

The angle measurement

This included the angle between bilateral VA and the mid-BA angle (*Figure 1*). All the angles were measured in the anteroposterior view of 3D-TOF-MRA. Imaginary lines were drawn from the vertebrobasilar junction (point a) to the closest and most curved point of VA from BA (point b) and the top of the BA (point c) (if the VA is straight, then b is at random in the VA). The acute angle between these 2 lines was considered to be the VA angle (*Figure 1A*,*1B*). Another imaginary line was drawn from the most curved point of the BA (point d) to the vertebrobasilar junction (point a) and the top of the BA (point c). The acute angle between these 2 lines was considered to be the mid-BA angle (*Figure 1C*). The images were independently measured on a digital picture archiving and communication system workstation by 2 radiologists (3 and 5 years of experience, respectively), and the average was taken as the result.

Measurement of basilar artery curvature direction

A line was drawn between the top of the BA (c point) and vertebrobasilar junction (a point) for reference to decide the side of basilar artery curvature (*Figure 1D*). If the basilar artery ran in an S-shape over the standard line, measurements were taken at the closest bend to the vertebrobasilar junction. The curvature of the artery was classified as right-sided, left-sided, or straight.

Configuration definition

For the first time, we defined the vertebrobasilar configuration as a balanced type if both vertebral arteries had similar diameters and if the angle difference between the vertebral arteries and the basilar artery was fewer than

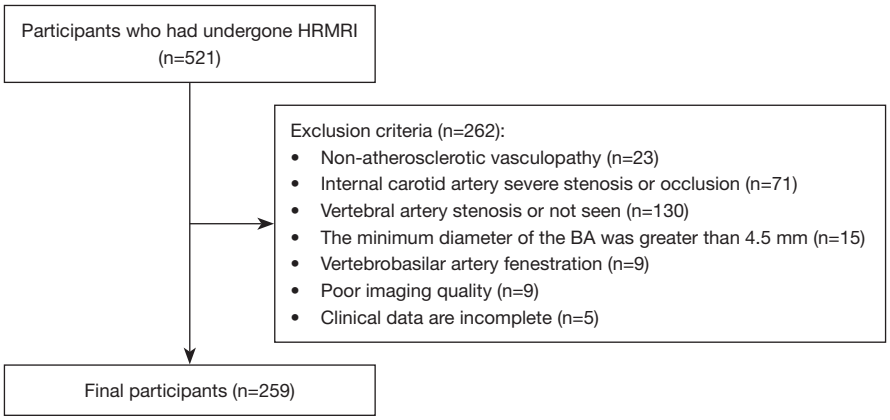

Figure 2 Flowchart of patient exclusion from the study. HRMRI, high-resolution magnetic resonance imaging; BA, basilar artery.

10 degrees. Otherwise, it was defined as dominant type.

Plaque identification

Atherosclerotic plaque on magnetic resonance (MR) images was defined as eccentric wall thickening with or without luminal stenosis identified on both the reconstructed pre-contrast and the reconstructed postcontrast images (16). All HRMRI images were independently interpreted by 2 experienced neuroradiologists (10 and 12 years of experience, respectively) for determining the presence of atherosclerotic lesions. Any disagreement between the 2 observers was resolved by a third senior neuroradiologist (20 years of experience).

Statistical analysis

Statistical analyses were performed using SPSS 25.0 (IBM Corp., Armonk, NY, USA). Categorical variables are presented as frequencies, and continuous variables are presented as means ± standard deviations. The *t*-test was used to compare quantitative variables, and the chi-squared test or continuity correction was used for qualitative variables. Multivariable linear regression analysis was used to evaluate the relationship between the dominant VA angle and mid-BA angle. Multivariable logistics regression analysis was used to analyze the effect of clinical risk factors and dominant VA angle on BA plaque. P values of <0.05 were considered to indicate statistical significance.

Interreader agreement for the measurement of diameter and angle were estimated using intraclass correlation coefficient (ICC) before reader consensus to settle disagreements. Interreader agreement for plaque identification was estimated by using k coefficients before reader consensus to settle disagreements. Reliability values less than 0.4 were characterized as poor, those 0.4–0.75 as fair to good, and those greater than 0.75 as excellent (17). A P value <0.05 was considered indicative of a significant difference.

Results

Patient characteristics

In total, 259 participants were included in this study (*Figure 2*). Among them, 23 nonatherosclerotic participants were excluded based on the clinical and imaging characteristics of other vascular diseases mentioned in the literature (18). Additionally, some studies have suggested that unilateral or bilateral internal carotid artery occlusion may lead to collateral blood supply to the anterior circulation through the posterior communication artery (19,20). Therefore, 71 participants with serious abnormalities of anterior circulation that may affect posterior circulation were excluded. The mean age of 259 participants was 53.71±13.12 years (146 men; *Table 1*), 75 patients (mean age 61.93±9.29 years) had BA plaque, and 184 patients (mean age 50.36±12.99 years) had no BA plaque. Participants with BA plaque (n=75) were older than those without BA plaque (n=184) at baseline (61.93±9.29 *vs.* 50.36±12.99 years; P<0.001). The group with BA plaque had a higher proportion of males (with plaque: 53/75, 70.7%; without plaque: 93/184, 50.5%) as well as individuals with hyperlipidemia (with plaque: 56/75, 74.7%; without plaque: 112/184, 60.9%), hypertension (with plaque: 58/75, 77.3%;

Table 1 Characteristics of participants with and without BA plaque

Characteristics	All participants (n=259)	BA plaque (n=75)	Without BA plaque (n=184)	P value
Age (years)	53.71±13.12	61.93±9.29	50.36±12.99	<0.001
Male	146 (56.4)	53 (70.7)	93 (50.5)	0.003
Risk factors				
Hyperlipidemia	168 (64.9)	56 (74.7)	112 (60.9)	0.035
Hypertension	145 (56.0)	58 (77.3)	87 (47.3)	<0.001
Diabetes	80 (30.9)	37 (49.3)	43 (23.4)	<0.001
Homocysteine	35 (13.5)	12 (16.0)	23 (12.5)	0.455
Smoking	90 (34.7)	31 (41.3)	59 (32.1)	0.155
Alcohol drinking	51 (19.7)	21 (28.0)	30 (16.3)	0.032
Obesity	23 (8.9)	8 (10.7)	15 (8.2)	0.519
Vertebrobasilar configuration				<0.001
Balanced type	30 (11.6)	0 (0.0)	30 (16.3)	
Dominant type	229 (88.4)	75 (100.0)	154 (83.7)	
Dominant VA angle (°)	–	57.92	42.99	<0.001

Data are shown as mean ± standard deviation or number (frequency). BA, basilar artery; VA, vertebral artery.

without plaque: 87/184, 47.3%), diabetes (with plaque: 37/75, 49.3%; without plaque: 43/184, 23.4%), and alcohol drinking (with plaque: 21/75, 28.0%; without plaque: 30/184, 16.3%). There were no significant differences between the 2 groups in homocysteine level, smoking status, or obesity (all P>0.05). The comparison of clinical risk factors between the 2 groups is shown in *Table 1*.

Configuration comparison

The balanced type was present in 30 of 259 participants (11.6%) (*Figure 3*). Compared with dominant-type participants, the balanced-type participants had a significantly lower probability of BA plaques (P<0.001). The balanced type was found only in participants without BA plaques (*Figure 4*).

Dominant VA angle

Of the 229 participants who were dominant type, there were 189 cases of bilateral VA diameter asymmetry and 40 cases of symmetrical VA diameter with angle asymmetry. *Table 2* summarizes the results of the chi-squared test on the directional relationship between the bilateral VA and BA curvature. The findings indicated that the BA curvature side was significantly associated with the side with a larger VA diameter (P<0.001) and larger VA angle (P<0.001), with most participants (106/189, 56.1%; 29/40, 72.5%) having an opposite directional relationship between the VA and BA curvature. Therefore, we considered the VA angle as more related to BA curvature as the dominant VA angle; that is, the VA angle on the side with larger diameter or the larger VA angle when the diameter was similar.

Table 3 shows the results of multivariable linear regression analyses of parameters associated with mid-BA angle. Only the dominant VA angle was independently associated with mid-BA angle (β=0.409; 95% CI: 0.245–0.455; P<0.001).

Effects of dominant VA angle on BA plaque

Table 4 shows the results of the multivariable logistics regression analyses for the effects of clinical risk factors and dominant VA angle on BA plaque. Older age (odds ratio, 1.073; 95% CI: 1.029–1.118; P=0.001), male sex (odds ratio, 3.733; 95% CI: 1.537–9.063; P=0.004), hypertension (odds ratio, 2.448; 95% CI: 1.060–5.655; P=0.036), diabetes (odds ratio, 2.336; 95% CI: 1.101–4.959; P=0.027), and the dominant VA angle (P<0.001) were independently associated with BA plaque. Other clinical risk factors, such as hyperlipidemia, homocysteine, smoking, alcohol

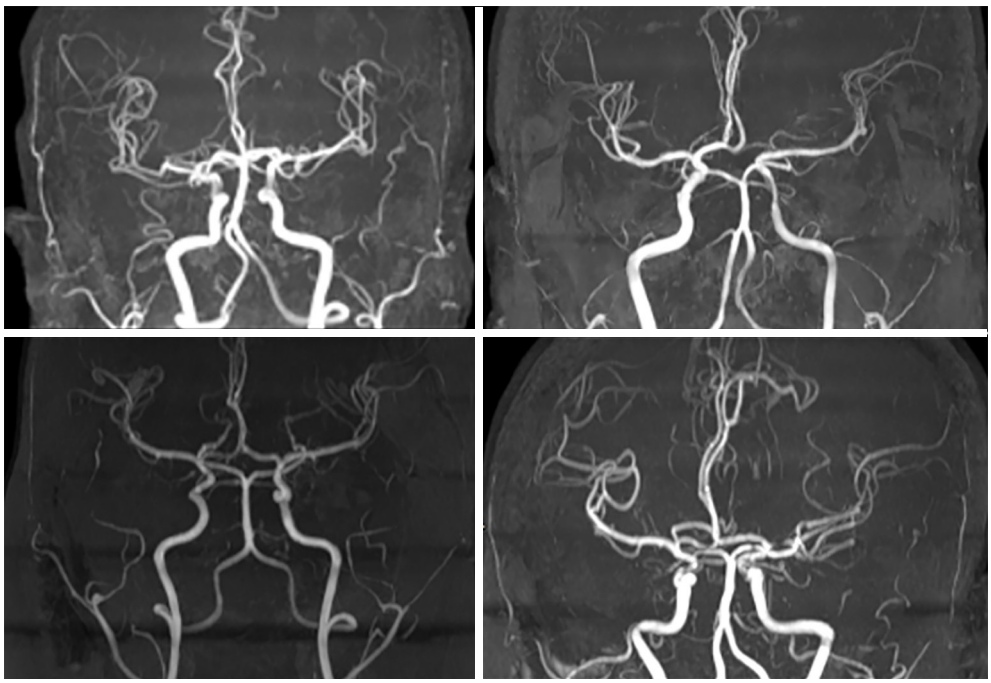

Figure 3 Example images from 4 participants with the balanced-type vertebrobasilar configuration.

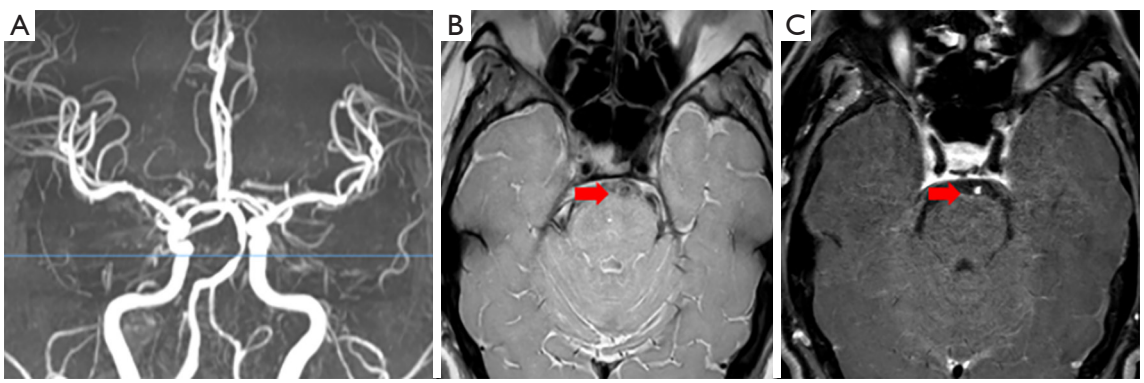

Figure 4 A 54-year-old man with basilar artery plaque. (A) 3D TOF-MRA showing the vertebrobasilar configuration as the dominant type. (B,C) The PDWI and CE-T1WI, respectively, indicating an eccentric plaque in the basilar artery (arrows). 3D-TOF-MRA, 3-dimensional time-of-flight magnetic resonance angiography; PDWI, proton density-weighted imaging; CE-T1WI, contrast-enhanced T1-weighted imaging.

drinking, and obesity were not significantly associated with BA plaque (all P>0.05). The degrees of the dominant VA angle varied from 0.06° to 118.66° (median 85.18°; IQR, 68.34°–108.02°). Taking 0° as the starting point and every 20 degrees as a group, the dominant VA angle of all participants was divided into 5 groups: 0°–20.00°, 20.01°–40.00°, 40.01°–60.00°, 60.01°–80.00°, and >80.00°. The results showed that 80° was the cutoff value of the dominant VA angle, and when the dominant VA angle was greater than 80°, the risk of BA plaque increased about 18-fold (odds ratio, 18.951; 95% CI: 4.545–79.026; P<0.001).

MRI measurement reproducibility

Interreader agreement for plaque identification (k=0.961) and the measurements of the diameter (vertebral arteries on

Table 2 Chi-squared test on the directional relationship between the vertebral artery and basilar artery curvature

Vertebral artery angle	Basilar artery curvature side			Total
	Right	Left	Straight	
The VA angle on the side with the larger diameter, n (%)				
Right	19 (18.6)	24 (61.5)	23 (47.9)	66
Left	83 (81.4)	15 (38.5)	25 (52.1)	123
The larger VA angle, n (%)				
Right	1 (4.3)	7 (63.6)	4 (66.7)	12
Left	22 (95.7)	4 (36.4)	2 (33.3)	28

VA, vertebral artery.

Table 3 Multivariable linear regression analyses of parameters associated with mid-BA angle

Parameter	β (95% CI)	P value
Dominant VA angle	0.409 (0.245–0.455)	<0.001
Age (years)	0.118 (–0.028–0.411)	0.087
Male sex	0.053 (–3.738–8.215)	0.461
Hyperlipidemia	–0.031 (–6.976–4.252)	0.633
Hypertension	–0.043 (–7.464–3.857)	0.531
Diabetes	–0.080 (–9.478–2.234)	0.224
Homocysteine	0.032 (–5.839–9.819)	0.617
Smoking	0.255 (–6.170–8.000)	0.799
Alcohol drinking	–0.069 (–7.891–7.358)	0.945
Obesity	1.499 (–2.169–15.935)	0.135

BA, basilar artery; β, regression coefficient; VA, vertebral artery.

Table 4 Results of multivariable logistics regression analyses of the effects of clinical risk factors and dominant VA angle on BA plaque

Parameter	OR (95% CI)	P value
Age (years)	1.073 (1.029–1.118)	0.001
Male sex	3.733 (1.537–9.063)	0.004
Hyperlipidemia	0.904 (0.406–2.013)	0.805
Hypertension	2.448 (1.060–5.655)	0.036
Diabetes	2.336 (1.101–4.959)	0.027
Homocysteine	1.189 (0.404–3.502)	0.753
Smoking	0.975 (0.366–2.601)	0.96
Alcohol drinking	1.177 (0.426–3.252)	0.753
Obesity	1.465 (0.459–4.676)	0.519
Dominant VA angle (°)		<0.001
0–20.00 (n=31)	–	–
20.01–40.00 (n=47)	1.194 (0.394–3.621)	0.754
40.01–60.00 (n=57)	1.135 (0.383–3.365)	0.819
60.01–80.00 (n=29)	2.463 (0.702–8.638)	0.159
>80.00 (n=28)	18.951 (4.545–79.026)	<0.001

VA, vertebral artery; BA, basilar artery; OR, odds ratio; CI, confidence interval.

both sides) and angle (bilateral VA and the mid-BA angle) were excellent (ICC 0.895–0.976).

Discussion

We propose a new and simple classification method for vertebrobasilar configuration and a new definition of "dominant VA angle". Our related study found that the dominant VA angle was independently related to mid-BA angle and BA plaque, with 80° being the cutoff value for the dominant VA angle: when the dominant VA angle was greater than 80°, the risk of BA plaque increased about 18-fold.

Several previous studies investigated the vertebrobasilar configuration to discuss the relationship between different configurations and BA plaque. Yu *et al.* enrolled 84 patients and divided them by vertebrobasilar artery geometry into 4 basic geometric configurations via visual classification; the results indicated that the geometrical configuration of vertebrobasilar artery had a significant effect on the distribution of BA plaque (21). Zheng *et al.* further found that the presence of BA plaque was associated with the lambda, walking, and no confluence geometry; ≥3 bends

in the VAs; and a large diameter difference between the bilateral VAs (22). We used a new classification approach for vertebrobasilar configuration that was different from previously used configurations, namely the balanced and dominant type. The advantages of this dichotomy are its simplicity and widespread availability. Moreover, measurement assessments are more reliable and immediately translatable than is visual classification and can provide a foundation for further research. Interestingly, among the 259 participants in this study, BA plaque only occurred in the dominant type, which may suggest that the balanced type of this configuration has high specificity for the presence of BA plaque. However, these findings need to be further verified in a larger sample with a more rigorous study design.

Previous studies have shown that the presence of VA dominance may contribute to the growth of BA length and that bending length growth may depend on VA dominance mainly in the contralateral direction (23). Our findings suggest that the BA curvature side is significantly associated with the side with the larger VA diameter and larger VA angle, and most participants had an opposite directional relationship between the VA and BA curvature. We therefore concluded that the VA angle is more related to BA curvature as the dominant VA angle; that is, the VA angle on the side with larger diameter or the larger VA angle when the diameter is similar.

The relationship between VA dominance and BA curvature has been extensively examined. In their study, Hong *et al.* found that the difference in the diameters of the right and left VA was the only independent predictor for moderate-to-severe BA curvature (14). These results may be expected for a few reasons: first, the adventitia of intracranial vessels lacks an external elastic lamina and rather contains only a few small elastic fibers (24), with the increase of collagen fibers replacing the muscle fibers of the media (25). Second, nonequivalent blood flow from the VAs may contribute significantly to changes in the BA (23). The smaller caliber size of the nondominant VA may result in a lower mean flow and irregular wall shear stress, which increases the risk of BA curvature (26,27). Therefore, the vector of BA flow merging from unequal VAs makes the BA flow curve to the side of the weaker VA. The results of the present study also support this conclusion, as the BA curvature was associated with asymmetric VA in dominant-type participants based on the classification of vertebrobasilar configuration. When flow in the smaller side of VA was reduced, the slipstreams from the dominant artery passed along the wall of the BA ipsilateral to the side of the flow-reduced VA more vigorously. Generally, when the diameters of the VA on both sides are similar, the VA on the side with a greater angle will exert a greater transverse force on the BA, making the BA bend laterally. Moreover, multiple regression analysis found that the dominant VA angle, which was mainly related to BA curvature, was independently related to the mid-BA angle. This finding has the consequence of rendering the influence of an asymmetric VA on BA curvature quantifiable, and lays a foundation for further research on the influence of asymmetric VAs on BAs.

Our findings indicate that among the clinical risk factors, older age, male sex, hypertension, and diabetes mellitus likely contributed to the risk of BA plaque. In a previous study, posterior circulation atherosclerosis was more often associated with hypertension and diabetes (3). Our data are partially consistent with these findings and agree more fully with a series of related studies showing that diabetes mellitus (28-30) or hypertension (30) is more often present in those patients with posterior than in those with anterior circulation stroke. However, our data conflict to a degree with the conclusion of another study, in which posterior circulation intracranial atherosclerosis was more closely associated with older age and female gender (31). The gender difference in results may be explained by ethnic differences as well as the variability in posterior circulation vessel selection and stenosis degree.

The risk factors of posterior circulation atherosclerosis plaque include both systemic risk factors and focal vessel geometry (32,33). Previous studies have shown that VA asymmetry is associated with BA curvature (14,23) and that BA curvature is an independent risk factor for BA plaque (34). However, very few studies have further established the direct relationship between the dominant VA and BA plaque. Our study used HRMRI technology, and results showed that the dominant VA angle is an independent risk factor for BA plaques. We set 0° as the starting point, with each 20° set being a group and thus divided all participants into 5 groups. Further analysis found that 80° was the cutoff value of the dominant VA angle, and when the dominant VA angle was greater than 80°, the risk of BA plaque increased about 18-fold. However, this result should be interpreted with caution. Because of the limited sample size, participants above 80° were not further subdivided, and the range of odds ratio values was too wide. This result may suggest that the dominant VA may increase the risk of BA plaque by

increasing BA curvature. VA dominance and its resulting BA curvature may cause a turbulent blood flow which plays an important role in the pathogenesis of atherosclerosis (35). Since the dominant VA takes time to cause BA curvature, a dominant VA angle greater than 80° may be a better clinical warning before ischemic events occur. Further clinical studies that to treat the dominant VA angle as a risk factor for atherosclerosis similar to hypertension and diabetes are warranted and may provide a reference for the hierarchical management by stroke neurologists. Based on its linking of vascular risk factors and BA atherosclerotic plaque, the results of our study further support the suggestion that vascular risk factors coupled with dominant VA angle greatly increases the risk of BA plaque.

Our study had some limitations. First, the sample size of balanced-type participants was limited. Future studies with larger samples are needed to fully explore the role of the balanced type in posterior circulation atherosclerosis. Second, the choice of blood vessels in our study was narrow, as only the influence of the section of the VA anterior to the BA was discussed. Moreover, we only examined the influence of the VA on the BA. Future studies are expected to include more segments of the posterior circulation vessels. Finally, a single-center, retrospective study design was used, and thus a comprehensive multicenter follow-up prospective study is needed to confirm the sensitivity and specificity of the vertebrobasilar configuration, and to verify the cutoff value of the dominant VA angle as a predictor of potential posterior circulation atherosclerosis.

Conclusions

This study examined the indices of the vertebrobasilar system's morphological and dominant VA angle. Dominant VA angle is an independent risk factor for BA plaque, and its effect may be influenced by BA curvature, with 80° being the cutoff value for a dominant VA angle. When the dominant VA angle is greater than 80°, the risk of BA plaque increases about 18-fold. Dominant VA angle can provide insight into the likelihood of future events occurring in patients with clinical atherosclerotic risk factors, enabling the identification of individuals who harbor an occult burden of vulnerable features and who might benefit from preventive therapeutic interventions.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-23-74/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-23-74/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and was approved by the China-Japan Friendship Institutional Review Board (No. 2021-BZR-24). Individual consent for this retrospective analysis was waived.

References

1. Feigin VL, Brainin M, Norrving B, Martins S, Sacco RL, Hacke W, Fisher M, Pandian J, Lindsay P. World Stroke Organization (WSO): Global Stroke Fact Sheet 2022. Int J Stroke 2022;17:18-29.
2. Merwick Á, Werring D. Posterior circulation ischaemic stroke. BMJ 2014;348:g3175.
3. Kim JS, Nah HW, Park SM, Kim SK, Cho KH, Lee J, Lee YS, Kim J, Ha SW, Kim EG, Kim DE, Kang DW, Kwon SU, Yu KH, Lee BC. Risk factors and stroke mechanisms in atherosclerotic stroke: intracranial compared with extracranial and anterior compared with posterior circulation disease. Stroke 2012;43:3313-8.
4. Klein IF, Lavallée PC, Mazighi M, Schouman-Claeys E, Labreuche J, Amarenco P. Basilar artery atherosclerotic plaques in paramedian and lacunar pontine infarctions: a

high-resolution MRI study. Stroke 2010;41:1405-9.
5. Yu J, Li ML, Xu YY, Wu SW, Lou M, Mu XT, Feng F, Gao S, Xu WH. Plaque distribution of low-grade basilar artery atherosclerosis and its clinical relevance. BMC Neurol 2017;17:8.
6. Lim SH, Choi H, Kim HT, Kim J, Heo SH, Chang DI, Lee JY, Lee YJ, Kim JY, Kim HY, Kim YS. Basilar plaque on high-resolution MRI predicts progressive motor deficits after pontine infarction. Atherosclerosis 2015;240:278-83.
7. Brown AJ, Teng Z, Evans PC, Gillard JH, Samady H, Bennett MR. Role of biomechanical forces in the natural history of coronary atherosclerosis. Nat Rev Cardiol 2016;13:210-20.
8. Ravensbergen J, Krijger JK, Hillen B, Hoogstraten HW. The influence of the angle of confluence on the flow in a vertebro-basilar junction model. J Biomech 1996;29:281-99.
9. Ravensbergen J, Ravensbergen JW, Krijger JK, Hillen B, Hoogstraten HW. Localizing role of hemodynamics in atherosclerosis in several human vertebrobasilar junction geometries. Arterioscler Thromb Vasc Biol 1998;18:708-16.
10. Kim BJ, Lee KM, Kim HY, Kim YS, Koh SH, Heo SH, Chang DI. Basilar Artery Plaque and Pontine Infarction Location and Vascular Geometry. J Stroke 2018;20:92-8.
11. Kim BJ, Kim HY, Jho W, Kim YS, Koh SH, Heo SH, Chang DI, Lee YJ. Asymptomatic Basilar Artery Plaque Distribution and Vascular Geometry. J Atheroscler Thromb 2019;26:1007-14.
12. Deng S, Zheng J, Wu Y, Yang D, Chen H, Sun B, Xue Y, Zhao X. Geometrical characteristics associated with atherosclerotic disease in the basilar artery: a magnetic resonance vessel wall imaging study. Quant Imaging Med Surg 2021;11:2711-20.
13. Sun J, Liu G, Zhang D, Wu Z, Liu J, Wang W. The Longitudinal Distribution and Stability of Curved Basilar Artery Plaque: A Study Based on HR-MRI. J Atheroscler Thromb 2021;28:1333-9.
14. Hong JM, Chung CS, Bang OY, Yong SW, Joo IS, Huh K. Vertebral artery dominance contributes to basilar artery curvature and peri-vertebrobasilar junctional infarcts. J Neurol Neurosurg Psychiatry 2009;80:1087-92.
15. Lou M, Caplan LR. Vertebrobasilar dilatative arteriopathy (dolichoectasia). Ann N Y Acad Sci 2010;1184:121-33.
16. Qiao Y, Zeiler SR, Mirbagheri S, Leigh R, Urrutia V, Wityk R, Wasserman BA. Intracranial plaque enhancement in patients with cerebrovascular events on high-spatial-resolution MR images. Radiology 2014;271:534-42.
17. Senn S. Review of Fleiss, statistical methods for rates and proportions. Res Synth Methods 2011;2:221-2.
18. Choi YJ, Jung SC, Lee DH. Vessel Wall Imaging of the Intracranial and Cervical Carotid Arteries. J Stroke 2015;17:238-55.
19. Rots ML, de Borst GJ, van der Toorn A, Moll FL, Pennekamp CWA, Dijkhuizen RM, Bleys RLAW. Effect of bilateral carotid occlusion on cerebral hemodynamics and perivascular innervation: An experimental rat model. J Comp Neurol 2019;527:2263-72.
20. van Everdingen KJ, Visser GH, Klijn CJ, Kappelle LJ, van der Grond J. Role of collateral flow on cerebral hemodynamics in patients with unilateral internal carotid artery occlusion. Ann Neurol 1998;44:167-76.
21. Yu J, Zhang S, Li ML, Ma Y, Dong YR, Lou M, Feng F, Gao S, Wu SW, Xu WH. Relationship between the geometry patterns of vertebrobasilar artery and atherosclerosis. BMC Neurol 2018;18:83.
22. Zheng J, Sun B, Lin R, Teng Y, Zhao X, Xue Y. Association between the vertebrobasilar artery geometry and basilar artery plaques determined by high-resolution magnetic resonance imaging. BMC Neurosci 2021;22:20.
23. Nishikata M, Hirashima Y, Tomita T, Futatsuya R, Horie Y, Endo S. Measurement of basilar artery bending and elongation by magnetic resonance cerebral angiography: relationship to age, sex and vertebral artery dominance. Arch Gerontol Geriatr 2004;38:251-9.
24. Portanova A, Hakakian N, Mikulis DJ, Virmani R, Abdalla WM, Wasserman BA. Intracranial vasa vasorum: insights and implications for imaging. Radiology 2013;267:667-79.
25. Qureshi AI, Caplan LR. Intracranial atherosclerosis. Lancet 2014;383:984-98.
26. Katsanos AH, Kosmidou M, Kyritsis AP, Giannopoulos S. Is vertebral artery hypoplasia a predisposing factor for posterior circulation cerebral ischemic events? A comprehensive review. Eur Neurol 2013;70:78-83.
27. Caplan LR. Arterial occlusions: does size matter? J Neurol Neurosurg Psychiatry 2007;78:916.
28. Subramanian G, Silva J, Silver FL, Fang J, Kapral MK, Oczkowski W, Gould L, O'Donnell MJ; . Risk factors for posterior compared to anterior ischemic stroke: an observational study of the Registry of the Canadian Stroke Network. Neuroepidemiology 2009;33:12-6.
29. Nagao T, Sadoshima S, Ibayashi S, Takeya Y, Fujishima M. Increase in extracranial atherosclerotic carotid lesions in patients with brain ischemia in Japan. An angiographic study. Stroke 1994;25:766-70.
30. Miyamoto N, Tanaka Y, Ueno Y, Tanaka R, Hattori N, Urabe T. Comparison of clinical backgrounds with

anterior versus posterior circulation infarcts. J Stroke Cerebrovasc Dis 2010;19:393-7.
31. Turan TN, Makki AA, Tsappidi S, Cotsonis G, Lynn MJ, Cloft HJ, Chimowitz MI; . Risk factors associated with severity and location of intracranial arterial stenosis. Stroke 2010;41:1636-40.
32. Xu Z, Li M, Lyu J, Hou Z, He J, Mo D, Gao F, Liu X, Sui B, Shen M, Pan Y, Wang Y, Lou X, Miao Z, Luo B, Ma N. Different risk factors in identical features of intracranial atherosclerosis plaques in the posterior and anterior circulation in high-resolution MRI. Ther Adv Neurol Disord 2020;13:1756286420909991.
33. Malek AM, Alper SL, Izumo S. Hemodynamic shear stress and its role in atherosclerosis. JAMA 1999;282:2035-42.
34. Kim BJ, Kim SM, Kang DW, Kwon SU, Suh DC, Kim JS. Vascular tortuosity may be related to intracranial artery atherosclerosis. Int J Stroke 2015;10:1081-6.
35. Wake-Buck AK, Gatenby JC, Gore JC. Hemodynamic characteristics of the vertebrobasilar system analyzed using MRI-based models. PLoS One 2012;7:e51346.

Cite this article as: Li Y, Guo R, Zhang X, Yang Y, Yan Y, Yuan X, Wang C, Xie S. Effect of dominant vertebral artery angle on basilar artery curvature and plaque. Quant Imaging Med Surg 2023;13(9):5748-5758. doi: 10.21037/qims-23-74

