Association Between Configuration of Basilar Artery and Vessel Wall Features: A Prospective High-resolution MR Imagine Study

Ziqi Xu (zyuxuziqi@zju.edu.cn)
Zhejiang University
https://orcid.org/0000-0002-5516-4817

Mingyao Li
Beijing Tiantan Hospital

Zhikai Hou
Beijing Tiantan Hospital

Jinhao Lyu
Chinese PLA General Hospital

Na Zhang
Paul C.Lauterber Research Center for Biomedical Imaging, Shenzhen Institutes of Advanced Technology, Chinese Academy of Sciences

Xin Lou
Chinese PLA General Hospital

Zhongrong Miao
Beijing Tiantan Hospital

Ning Ma
Beijing Tiantan Hospital

Research article

Keywords: vertebrobasilar artery, atherosclerotic disease, high resolution MRI, collateral circulation, configuration

Posted Date: July 12th, 2019

DOI: https://doi.org/10.21203/rs.2.11252/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License

Version of Record: A version of this preprint was published on December 26th, 2019. See the published version at https://doi.org/10.1186/s12880-019-0388-3.
Abstract

Background We aimed to investigate the relationship between distal and proximal anatomical configurations of basilar artery (BA) and vessel wall features on high resolution magnetic resonance imaging (HRMRI). Methods From September 2014 to January 2017, patients with suspected symptomatic intracranial arterial stenosis underwent HRMRI. Patients with severe BA stenosis were enrolled. Configurations of BA were divided complete and incomplete groups based on with or without bilateral vertebral arteries and posterior cerebral arteries. Culprit vessel wall features on HRMRI included enhancement grade, intraplaque hemorrhage, remodeling patterns, and plaque distribution. Culprit vessel wall features were compared between complete and incomplete groups. Results Among the 298 consecutively enrolled patients, 34 consecutive patients had severe BA stenosis. Seventeen patients had complete configuration and 14 patients with incomplete configuration. There were no statistics difference in vessel wall features between complete and incomplete groups configuration groups. The proximal configuration of BA was associated with intraplaque hemorrhage\(p=0.002\) and the distal configuration of BA correlated with strong enhancement of BA plaque\(p=0.041\). Conclusions The complete and incomplete groups configuration of BA did not associate with vessel wall features. The proximal configuration of BA was related with intraplaque hemorrhage and the distal configuration of BA was associated with strong plaque enhancement. These findings are continuously needed to confirmed in future studies.

Background

Basilar artery (BA) atherosclerotic occlusive disease is the most common reason of posterior circulation strokes, and it can cause disastrous outcome and patients with it had a high risk of recurrent stroke [1-2]. The complete configuration of BA including bilateral vertebral arteries (VA), branches of cerebellar artery and bilateral posterior cerebral arteries (PCA). The VA size is various individually and 6-26% of cases with equal size in angiographic studies [3]. The previous studies found that VA hypoplasia was predisposing factor for posterior circulation stroke [4-5]. Dominant VA often caused BA curvature and development of peri-vertebrobasilar junction infarcts [4-5]. There were also studies showed that fetal type PCA (fPCA) may predispose to ischemic events in the posterior circulation [6-7].

The grade of intracranial artery stenosis and vulnerability of plaques are indications being considered to guide the clinical management [8]. The study had demonstrated that symptomatic vertebrobasilar artery stenosis was associated with a greatly increased risk of recurrent stroke [9]. The studies had proved that vulnerability of intracranial artery plaque and hypoperfusion of blood flow at the distal stenotic site were highly associated with stroke events and recurrence[10-11]. Anterior circulation acute ischemic stroke study had proved the associations between collateral circulation and thrombus characteristics, that patients with higher collateral scores had lower thrombus burden and more previous thrombi [12-13].

However, the relation between vessel configuration and intracranial vessel wall features is not well investigated. Configurations of BA play a role in posterior circulation hemodynamics, and may influence
the vessel wall features of BA. To test this hypothesis, we investigate the demographics, variants of VA and PCA, vessel wall features of BA. We compared the relationship between different configurations of BA and vessel wall features of BA on HRMRI.

**Methods**

The study was a prospective and registry study, had been approved by ethics committee of our hospital. Written informed consent was obtained from the patients or their legally relatives.

**Enrollment of Patients**

Patients suspected to symptomatic intracranial atherosclerotic stenosis (ICAS) at admission were enrolled. All patients received thorough evaluations to determine the cause of ischemic events including transit ischemic attack (TIA) or ischemic stroke, including carotid duplex, transcranial Doppler, echocardiography, electrocardiogram, computer tomography (CT), magnetic resonance imaging (MRI), CT angiography (CTA), magnetic resonance angiography (MRA) and digital subtract angiography (DSA).

Patients were enrolled in this study according to the following criteria: 1) age $\geq 18$ years; 2) ischemic stroke or TIA in the target territories posterior circulation within 90 days; 3) basilar artery stenosis $\geq 70\%$, and without coexistent $\geq 50\%$ ipsilateral extracranial vertebral artery stenosis; 4) Without potential sources of cardioaortic embolism based on the modified Trial of ORG 10 172 in Acute Stroke Treatment (TOAST) classification [14], 5) one or more atherosclerotic risk factors; 6) all the patients received DSA examination. Risk factors were recorded including hypertension, dyslipidemia, diabetes, smoking and obesity.

Patients with the following conditions were excluded: 1) nonatherosclerotic vasculopathy such as vasculitis and arterial dissection, diagnosed by comprehensive laboratory examinations (such as erythrocyte sedimentation rate or C-reactive protein elevations, antinuclear antibody, or antiphospholipid antibody positivity), vascular imaging, and clinical evaluation. 2) contraindication to MR examination, medical instability precluding MR examination.

**HRMRI acquisition and analysis**

All HRMRI studies were performed on a 3T GE DISCOVERY MR 750 (GE Healthcare, Waukesha, WI, USA) or a 3T Siemens Trio MR scanner (Siemens Healthcare, Ehrlangen, Germany). The details were presented in study protocol (see the supplemental etable 1). Image reconstruction were conducted by Reformate tool in AW 4.5 workstation (GE Healthcare) and 3D multiple planer reconstruction tool in Siemens workstation. MR images were then processed for all identified plaques using commercially available software (Vessel Mass; Leiden University Medical Center, Leiden, The Netherlands).

A culprit plaque was defined as the single lesion at the supplying artery for the infarct zone or the most severe stenotic lesion when multiple plaques were present at the supplying artery [15-16].
Arterial remodeling index (RI) was calculated as the ratio of outer wall area (OWA) at the site of maximal lumen narrowing to that at the reference site (RI=OWA lesion/OWA reference) [17]. The reference site was selected based on the Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial method [18]. There are three remodeling categories as previously described, following RI ≥ 1.05 as positive remodeling, 0.95 <RI< 1.05 as intermediate remodeling, RI ≤ 0.95 as negative remodeling. Plaque distributions were dichotomized into diffuse and non-diffuse patterns at culprit lesion. The anatomical location of the plaque was recorded as ventral, dorsal, left, and right quadrants [19]. Plaques spreading across four quadrants were defined as diffuse and that involving ≤3 quadrants were defined as non-diffuse. Intraplaque hemorrhage (IPH) was defined as a signal intensity greater than 150% of T1 signal of adjacent muscle [20]. As to plaque enhancement, non-enhancement was defined as similar to or less than that of normal intracranial arterial walls nearby, while enhancement meant signal intensity was greater than non-enhancement, and less than or greater than that of the pituitary infundibulum [15].

We adopted the same principal when interpreting HRMRI vessel wall imaging over arterial remodeling and vessel wall features as we have published before with small intrao-bserver and inter-observer variability [10,17,21]. The intra- and inter-observer variability of the two scanners and vessel wall features of HRMRI were good to excellent (weighted k =0.82, 95% CI: 0.46, 1.00 and 0.83, 95% CI: 0.41-1.00, respectively).

**Definition of configurations of BA**

Configurations of BA were divided as complete and incomplete configuration. (see figure 1) The patients with normal bilateral vertebral arteries and posterior cerebral arteries were categorized as complete configuration. If the patients had one VA dysplasia and (or) fPCA were defined as incomplete configuration on DSA and(or) CTA, MRA. The presence of posterior communicating arteries was also recorded. The presence of hypoplastic VA was defined as with a diameter <2 mm, ended in the posterior inferior cerebellar artery, or lumen diameter more than 50% difference [4-5]. The fPCA was defined as the blood flow of posterior cerebral artery from internal carotid artery, and without P1 segment of PCA or dysplasia of P1 segment of PCA [6-7]. Two neurologists (Z.Q.X. and N.M) reviewed the DSA images independently and discrepancies were resolved by consensus.

**Statistical analysis**

Continuous variables were presented as means ± SD or median with interquartile range. Categorical variables were presented as percentages. All baseline characteristics, plaque enhancement, intraplaque hemorrhage, arterial remodeling patterns and plaque distribution were compared with χ2 test for categorical variables and 1-way analysis of variance or the Kruskal-Wallis test for continuous variables between complete and incomplete configurations group. The analyses were performed using SPSS 23.0 statistical software (IBM, Chicago, IL, USA). A two-tailed P value less than 0.05 was considered statistically significant.

**Results**
Baseline characteristics

From September 2014 to January 2017, among 298 consecutively enrolled patients, 34 patients were included in our study. Among them, 6 patients were TIA and 28 were stroke. The mean time from events to HRMRI examination was 37.75 ± 25.84 days. 64.2% of plaques had enhancement, 20.6% had intraplaque hemorrhage, 50.0% had positive remodeling, and 97.1% had diffuse distribution. The demographic data and risk factors between the complete and incomplete configuration group were no statistical different. (The detailed result see table 1).

The association between artery configuration and BA vessel wall features

Among 34 patients with culprit severe BA stenosis, 20 patients (58.8%) had complete configuration and 14 patients (41.2%) had incomplete configuration. There were no statistics difference in vessel wall features between complete and incomplete configuration groups. (The detailed result see table 1).

Among them, 8 patients (23.5%) had fPCA including 2 patients (5.9%) with bilateral fPCA. Eleven patients (32.4%) had hypoplastic VA including 3 patients with VA (8.8%) ending in PICA and 7 patients (20.6%) with VA occluded. The proximal configuration of BA was associated with intraplaque hemorrhage(p=0.002) and the distal configuration of BA correlated with strong enhancement of BA plaque(p=0.041). (The detailed result see table 2 and 3).

Discussion

When the BA is involved by atherosclerotic lesion, whether the configuration of cerebral vascular is associated with the vessel features remain uncertain. So, we try to explore the effects of variant configuration on culprit plaque characteristics of BA. To our knowledge, this is the first study to explore the correlation of posterior circulation artery configuration with culprit plaque features in BA by 3D HRMRI.

The present study found that there were no statistics differences in vessel wall features between complete and incomplete configuration of BA. The present study showed the variations of configuration BA tree had no relationship with vessel wall features of BA. The fPCA and VA lumen size different are common variations of posterior circulation. The fPCA is a common variant of circle of Willis and can accompany with hypoplastic BA [22]. In our study, 23.5% patients with fPCA, consistent with previous report [22]. Lochner et al found that fPCA accompanied by hypoplastic BA may predispose to ischemic events in the posterior circulation [23]. The study found that unequal VA may cause the BA curvature and development of peri-vertebrobasilar junctional infarcts [5]. Ravensbergen et al showed that the geometry of vertebrobasilar junction correlated with occurrence of atherosclerotic plaque at the apex of vertebrobasilar junction and lateral wall of BA [24]. In our study, 10 patients (29.4%) with nondominant VA occlusion, and among them 3 patients’ VA (8.8%) ending in posterior inferior cerebellar artery. Overall, although variants of VA and PCA were associated with ischemic stroke, the variations of configuration BA
tree without relationship with vessel wall features of BA. The results may suggest that the variant of artery configuration could not trigger the formation of plaques.

Our study showed proximal configuration tree of BA was associated with strong plaque enhancement. In the present study, 64.2% of plaques had enhancement. A previous study had found that BA plaque enhancement and composition correlated with stroke events [25]. Plaque enhancement reflects the extent of inflammation of vessel wall. The incomplete configuration of proximal VA may cause difference in longer outcome, but this hypothesis needs to be confirmed or refuted in further studies.

Our study also found the proximal configuration tree of BA was associated with strong plaque enhancement and the distal configuration tree of BA was closed related with intraplaque hemorrhage. In the present study, 64.2% with plaque enhancement and 20.6% with intraplaque hemorrhage on HRMRI. Plaque enhancement and intraplaque hemorrhage on HRMRI are markers of plaque destabilization and progression strongly associated with stroke events [25], which are also associated with endothelial dysfunction and neovascularization of the artery wall [26-27]. The relationship of incomplete distal configuration of BA and intraplaque hemorrhage is hard to explain in the present understanding. The incomplete distal configuration of BA indicated different hemodynamics and blood flow reserve on the top of BA. The fPCA indicated that most or all the blood flow of PCA was from ipsilateral internal carotid artery. When complete BA tree, the most common distribution pattern of blood flow within the top of the BA is parallel [28]. The fPCA will change this the flow pattern causing the changing of regional wall shear force. The study showed that blood flow shear stress act on wall causing endothelial injury and plaque instability, presenting with plaque enhancement [29]. Poor collaterals circulation and high grade of stenosis produced the high-speed blood flow around the plaque causing erosion of fibrous cap or endothelium injury in the middle cerebral artery and carotid artery, and causing plaque enhancements [30-31]. The underlying mechanism of plaque enhancement correlated with distal incomplete configuration of BA is need to be studied further.

The previous study found that unequal VA may cause the BA curvature and development of peri-vertebrobasilar junctional infarcts [5]. Dominant VA flow acted on the contralateral wall of BA caused the tortuous of BA geometry which strongly affected velocity and wall shear stress distribution [32], and finally, trigger the formation of BA plaque and affect intraplaque hemorrhage. The studies showed that the geometry of vertebrobasilar artery correlated with occurrence of atherosclerotic plaque and plaque distribution [32]. Asymmetric VAs causing the BA bending is a chronic process, and not only influenced by shear stress but also vascular risk factors [32-33]. The underlie mechanism of intraplaque hemorrhage correlated with incomplete proximal configuration of BA is also need to further study.

This study has some limitations. First, the number of subjects is little small and come from the single stroke center, selection bias should be concerned. Second, our study population was exclusively high degree of stenosis. Therefore, caution should be taken when generalizing the findings to other degree of stenosis.
In summary, we found that the complete configuration of BA was not associated with vessel features of BA, the proximal configuration of BA was related with intraplaque hemorrhage and the distal configuration of BA was associated with strong plaque enhancement. These findings are continuously needed to confirmed or refuted in future studies.

Abbreviations

BA: Basilar artery; PCA: posterior cerebral arteries; VA: vertebral arteries; fPCA: fetal type PCA; HRMRI: high resolution magnetic resonance imaging; ICAS: intracranial atherosclerotic stenosis; TIA: transit ischemic attack; WASID: Warfarin-Aspirin Symptomatic Intracranial Disease trial; CT: computer tomography; MRI: magnetic resonance imaging; CTA: CT angiography; MRA: magnetic resonance angiography; DSA: digital subtract angiography; RI: Arterial remodeling index; IPH: Intraplaque hemorrhage.

Declarations

Acknowledgements

None.

Funding

This work was supported by the National Natural Science Foundation of China (Contract grant number: 81671126 and 81730048 to X.L., 81471390 to N.M., 81371290 to Z.R.M.), Beijing High-level Personnel Funds (Contract grant number: 2013-2-19 to Z.R.M.), and National Key R&D Program of China (Contract grant number: 2016YFC0100100 to X.L.), Medical and Health Science and Technology of Zhejiang Province (Contract grant number: 2015KYA080 to Z.Q.X).

Author's contributions

All authors have reviewed and approved the submitted manuscript for publication. ZX contributed to writing the manuscript and interpretation of the data. ZKH, XL and MYL contributed to acquisition the data. NZ and JHL contributed to acquisition of HRMRI data. ZRM and NM contributed to the critical revision of the manuscript for intellectual content.

Ethics approval and consent to participate

Current study was approved by the first affiliated hospital Zhejiang University and Beijing Tiantan Hospital. Written informed consent was obtained from the patients or their legally relatives.

Consent for publication

All enrolled patients had been signed an informed consent form regarding publication of the study in all formats hard and electronics including personal data and images irrespective of time and language.
Competing interests

The authors declare that they have no competing interests.

References

1. Savitz SI, Caplan LR. Vertebrobasilar disease. N Engl J Med. 2005,352:2618-26.
2. Gulli G, Marquardt L, Rothwell PM, Markus HS. Stroke risk after posterior circulation stroke/transient ischemic attack and its relationship to site of vertebrobasilar stenosis: pooled data analysis from prospective studies. Stroke. 2013,44:598-604.
3. Jeng JS, Yip PK. Evaluation of vertebral artery hypoplasia and asymmetry by color-coded duplex ultrasonography. Ultrasound Med Biol. 2004,30:605-9.
4. Perren F, Poglia D, Landis T, Sztajzel R. Vertebral artery hypoplasia: a predisposing factor for posterior circulation stroke? Neurology. 2007,68:65-7.
5. 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.
6. Lochner P, Golaszewski S, Caleri F, et al. Posterior circulation ischemia in patients with fetal-type circle of Willis and hypoplastic vertebrobasilar system. Neurol Sci. 2011,32:1143-6.
7. Arjal RK, Zhu T, Zhou Y. The study of fetal-type posterior cerebral circulation on multislice CT angiography and its influence on cerebral ischemic strokes. Clin Imaging. 2014,38:221-5.
8. Holmstedt CA, Turan TN, Chimowitz MI. Atherosclerotic intracranial arterial stenosis: risk factors, diagnosis, and treatment. Lancet Neurol. 2013,12:1106-14.
9. Abuzinadah AR, Alanazy MH, Almekhlafi MA, et al. Stroke recurrence rates among patients with symptomatic intracranial vertebrobasilar stenoses: systematic review and meta-analysis. J Neurointerv Surg. 2016,8:112-26.
10. Lou X, Ma N, Ma L, Jiang WJ. Contrast-enhanced 3T high-resolution MR imaging in symptomatic atherosclerotic basilar artery stenosis. AJNR Am J Neuroradiol. 2013,34:513-7.
11. Amin-Hanjani S, Pandey DK, Rose-Finnell L, et al; Vertebrobasilar Flow Evaluation and Risk of Transient Ischemic Attack and Stroke Study Group: Effect of Hemodynamics on Stroke Risk in Symptomatic Atherosclerotic Vertebrobasilar Occlusive Disease. JAMA Neurol. 2016,73:178-85.
12. Alves HC, Treurniet KM, Dutra BG, et al; MR CLEAN trial investigators. Associations Between Collateral Status and Thrombus Characteristics and Their Impact in Anterior Circulation Stroke. Stroke. 2018,49:391-6.
13. Tan IY, Demchuk AM, Hopyan J, et al. CT angiography clot burden score and collateral score: correlation with clinical and radiologic outcomes in acute middle cerebral artery infarct. AJNR Am J Neuroradiol. 2009,30:525-31.
14. Ay H, Furie KL, Singhal A, Smith WS, Sorensen AG, Koroshetz WJ. An evidence-based causative classification system for acute ischemic stroke. Ann Neurol. 2005,58:688-97.

15. 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.

16. Qiao Y, Anwar Z, Intrapiromkul J, et al. Patterns and implications of intracranial arterial remodeling in stroke patients. Stroke. 2016,47:434-40.

17. Ma N, Jiang WJ, Lou X, et al. Arterial remodeling of advanced basilar atherosclerosis: a 3-tesla MRI study. Neurology. 2010,75:253-8.

18. Chimowitz MI, Kokkinos J, Strong J, et al. The warfarin-aspirin symptomatic intracranial disease study. Neurology. 1995,45:1488-93.

19. Chen Z, Liu AF, Chen H, et al. Evaluation of basilar artery atherosclerotic plaque distribution by 3D MR vessel wall imaging. J Magn Reson Imaging. 2016,44:1592-9.

20. Yu JH, Kwak HS, Chung GH, Hwang SB, Park MS, Park SH. Association of intraplaque hemorrhage and acute infarction in patients with basilar artery plaque. Stroke. 2015,46:2768-72.

21. Ma N, Xu Z, Lyu J, et al. Association of Perforator Stroke After Basilar Artery Stenting With Negative Remodeling. Stroke.2019;50(3):745-9.

22. van Raamt AF, Mali WP, van Laar PJ, van der Graaf Y. The fetal variant of the circle of Willis and its influence on the cerebral collateral circulation. Cerebrovasc Dis. 2006,22:217-24.

23. Lochner P, Golaszewski S, Caleri F, et al. Posterior circulation ischemia in patients with fetal-type circle of Willis and hypoplastic vertebrobasilar system. Neurol Sci. 2011,32:1143-6.

24. 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.

25. Bodle JD, Feldmann E, Swartz RH, Rumboldt Z, Brown T, Turan TN. High-resolution magnetic resonance imaging: an emerging tool for evaluating intracranial arterial disease. Stroke. 2013,44:287-92.

26. Millon A, Boussel L, Brevet M, et al. Clinical and histological significance of gadolinium enhancement in carotid atherosclerotic plaque. Stroke. 2012,43:3023-8.

27. Xu WH, Li ML, Gao S, et al. Middle cerebral artery intraplaque hemorrhage: prevalence and clinical relevance. Ann Neurol. 2012,71:195-8.

28. Smith AS, Bellon JR. Parallel and spiral flow patterns of vertebral artery contributions to the basilar artery. AJNR Am J Neuroradiol. 16(1995,16:1587-91.

29. Dolan JM, Kolega J, Meng H. High wall shear stress and spatial gradients in vascular pathology: a review. Ann Biomed Eng. 2013,41:1411-27.

30. Suh DC, Park ST, Oh TS, et al. High shear stress at the surface of enhancing plaque in the systolic phase is related to the symptom presentation of severe M1 stenosis. Korean J Radiol. 2011,12:515-8.
31. Tuenter A, Selwaness M, Arias Lorza A, et al. High shear stress relates to intraplaque haemorrhage in asymptomatic carotid plaques. Atherosclerosis. 2016, 251:348-54.

32. Han HC. Twisted blood vessels: symptoms, etiology and biomechanical mechanisms. J Vasc Res. 2012, 49:185-97.

33. Kim BJ, Lee KM, Kim HY, et al. Basilar Artery Plaque and Pontine Infarction Location and Vascular Geometry. J Stroke. 2018, 20:92-8.

Tables

**Table 1** Demographic and clinical characteristics between complete and incomplete configuration of basilar artery patients.
| Variable                              | All patients (n=34) | Complete configuration of BA (n=20) | Incomplete configuration of BA (n=14) | P value |
|--------------------------------------|---------------------|-------------------------------------|--------------------------------------|---------|
| **Age-years (SD)**                   | 58.77±9.39          | 59.18 ± 8.13                        | 58.35 ± 10.74                       | 0.803   |
| **Body mass index-Kg/m²(SD)**        | 26.60 ±2.83         | 26.24 ± 2.05                        | 26.96 ± 3.27                        | 0.463   |
| **LDL-mmol/L(SD)**                   | 1.91±0.71           | 1.77 ± 0.76                         | 2.05 ± 0.65                         | 0.263   |
| **Male-No. (%)**                     | 28(82.4)            | 16(94.1)                            | 12(70.6)                            | 0.072   |
| **Hypertension-No. (%)**             | 28(82.4)            | 12(70.6)                            | 16(94.1)                            | 0.072   |
| **diabetes-No. (%)**                 | 14(41.2)            | 7(41.2)                             | 7(41.2)                             | 1.000   |
| **dyslipidemia-No. (%)**             | 13(38.2)            | 3(17.6)                             | 10(58.8)                            | 0.013   |
| **smoking-No. (%)**                  | 25(73.5)            | 12(70.6)                            | 13(76.5)                            | 0.697   |
| **Coronary artery disease-No. (%)**  | 8(23.5)             | 5(29.4)                             | 3(17.5)                             | 0.419   |
| **Qualifying events-No. (%)**        |                     |                                     |                                      |         |
| TIA                                 | 6(17.6)             | 5(29.4)                             | 1(5.9)                              | 0.072   |
| infarction                          | 28(82.4)            | 12(70.6)                            | 16(94.1)                            | 0.072   |
| **Time from event to HRMRI-days (IQR)** | 33(16-52)            | 33(13-52)                           | 33(23-46)                           | 0.462   |
| **Enhancement grade*-No. (%)**       |                     |                                     |                                      |         |
| None                                | 10(35.7)            | 7(70.0)                             | 3(30.0)                             | 0.453   |
| Mild to moderate                    | 8(28.6)             | 5(62.5)                             | 3(37.5)                             | 0.903   |
| Strong                              | 10(35.7)            | 5(50.0)                             | 5(50.0)                             | 0.387   |
| **Intraplaque hemorrhage-No. (%)**   | 7(20.6)             | 2(28.6)                             | 5(71.4)                             | 0.068   |
| **Arterial remodeling-No. (%)**      |                     |                                     |                                      |         |
| Negative                            | 12(35.3)            | 11(64.7)                            | 6(35.3)                             | 0.486   |
| Positive                            | 17(50.0)            | 9(52.9)                             | 8(47.1)                             |         |
| **Distribution patterns-No. (%)**    |                     |                                     |                                      |         |
| Non-diffuse                         | 1(2.9)              | 0(0)                                | 1(100.0)                            | 0.225   |
| Diffuse                             | 33(97.1)            | 20(60.6)                            | 13(39.4)                            |         |

* Data from 28 patients

Table 2 Comparison of plaque features between complete and incomplete proximal configuration of basilar artery patients.
| Characteristics | Complete proximal configuration (n = 25) | Incomplete proximal configuration (n = 9) | P value |
|-----------------|----------------------------------------|----------------------------------------|---------|
| Enhancement grade* - No. (%) |                                        |                                        |         |
| None            | 8 (80.0%)                              | 2 (20.0%)                              | 0.454   |
| Mild to moderate| 5 (62.5%)                              | 3 (33.3%)                              | 0.508   |
| Strong          | 7 (70.0%)                              | 3 (30.0%)                              | 0.901   |
| Intraplaque hemorrhage | 2 (28.6%)                      | 5 (71.4%)                              | 0.002   |
| Remodeling patterns - No. (%) |                                        |                                        |         |
| Negative        | 8 (66.7%)                              | 4 (33.3%)                              | 0.503   |
| Positive        | 13 (76.5%)                             | 4 (23.5%)                              | 0.697   |
| Distribution patterns - No. (%) |                                        |                                        |         |
| non-diffuse     | 0 (0%)                                 | 1 (100%)                               | 0.274   |
| diffuse         | 25 (75.8%)                             | 8 (24.2%)                              | 0.091   |

*Data from 28 patients

Table 3 Comparison of plaque features between complete and incomplete distal configuration of basilar artery patients.

| Characteristics | Complete distal configuration (n = 26) | Incomplete distal configuration (n = 8) | P value |
|-----------------|----------------------------------------|----------------------------------------|---------|
| Enhancement grade* - No. (%) |                                        |                                        |         |
| None            | 9 (90.0%)                              | 1 (10.0%)                              | 0.272   |
| Mild to moderate| 7 (87.5%)                              | 1 (12.5%)                              | 0.466   |
| Strong          | 6 (60.0%)                              | 4 (40.0%)                              | 0.074   |
| Intraplaque hemorrhage | 5 (71.4%)                      | 2 (28.6%)                              | 0.724   |
| Remodeling patterns - No. (%) |                                        |                                        |         |
| Negative        | 10 (83.3%)                             | 2 (16.7%)                              | 0.486   |
| Positive        | 11 (64.7%)                             | 6 (35.3%)                              | 0.106   |
| Distribution patterns - No. (%) |                                        |                                        |         |
| Non-diffuse     | 0 (0%)                                 | 1 (100%)                               | 0.067   |
| diffuse         | 26 (78.8%)                             | 7 (21.2%)                              |         |

*Data from 28 patients
Figures

Figure 1

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.
• supplement1.docx