Aims: This study aims to evaluate the differences in the characteristics of atherosclerotic plaques in the proximal, curved, and distal segments of the curved basilar artery (BA) through high-resolution magnetic resonance imaging (HR-MRI).

Methods: The imaging and clinical data of 146 patients were retrospectively analyzed. On the basis of three-dimensional (3D) time-of-flight magnetic resonance angiography (3D-TOF-MRA), 51 patients with BA curvature were selected for the study. The BA plaque is divided into three groups: proximal, curved, and distal. Plaques were identified and analyzed according to spin echo acquisition imaging via T1-weighted 3D volumetric isotropic Tse acquisition (TIW-3D-VISTA), and compare the differences in clinical related factors and plaque characteristics between groups. Diffusion-weighted imaging (DWI) and/or T2WI identified brainstem infarction. The patients were divided into symptomatic and asymptomatic groups. The correlation between plaque location and symptoms was identified and analyzed.

Results: Among 51 patients, a total of 376 plaques were detected. Plaques in the proximal and curved segments are more common than those in the distal segments. Proximal plaques are more likely to have intraplaque hemorrhage \( (P=0.002 < 0.05) \). There was no significant difference in the distribution of criminal plaques and non-criminal plaques between each group \( (P=0.36 > 0.05) \).

Conclusion: Plaques in the proximal and curved segments of the BA are more common than those in the distal segments. The proximal plaque is more prone to intraplaque hemorrhage.

Key words: High-resolution MRI, Basilar artery, Plaque
associated with the curvature of the BA\(^1\), but these studies are based on the study of the horizontal position (i.e., on the axial image) of the BA plaque. At present, no researchers have studied the formation and characteristics of the proximal and distal plaques of the curved BA, so this study intends to analyze the longitudinal distribution and characteristics of the BA plaque in patients with BA curvature to evaluate the relationship between the longitudinal distribution and stability of plaques for early clinical intervention and treatment.

### 1. Materials and Methods

#### 1.1 Patient Selection

This study is a retrospective analysis of 146 patients with BA high-resolution magnetic resonance imaging (HR-MRI) in the First Affiliated Hospital of Harbin Medical University from February 2017 to October 2020. The inclusion criteria are those who (1) are aged \(\geq 18\) years; (2) had no evidence of cardiogenic embolism, such as atrial fibrillation; (3) had no vertebral artery occlusion; (4) had no non-atherosclerotic diseases such as aneurysm, arteritis, and arterial dissection; (5) had no regular drug therapy for stable plaque was performed; (6) have BA curvature and (7) have imaging data quality meeting the needs of diagnosis. Patients who do not meet the aforementioned inclusion criteria and those with incomplete imaging data are excluded from this study. Finally, 51 cases met the inclusion criteria and were included in this study. Additionally, patients’ clinically related data, such as age; gender; presence of hypertension, dyslipidemia, and diabetes; and smoking and drinking history, were collected.

This study has been approved by the Ethics Committee of the First Affiliated Hospital of Harbin Medical University, and patient's consent has been obtained for privacy processing of all patients' images and clinical data.

#### 1.2 MRI Program

A 3.0T magnetic resonance scanner (Philips Achieva 3.0T) was used for image acquisition, and a 16-channel standard head quadrature coil was used for scanning. All patients underwent scan three-dimensional (3D-) time-of-flight magnetic resonance angiography (TOF-MRA), BA HR-MRI axial 3D-T1-weighted volume isotropic turbo spin echo acquisition (VISTA), and head diffusion-weighted imaging (DWI)/T2WI sequence. The scanning parameters are (1) 3D-TOF-MRA: repetition time (TR)/echo time (TE) 25/3.45 ms, field of view (FOV) 1.94 \(\times\) 1.94 cm, layer thickness 20.0 mm 0 mm, matrix 276 \(\times\) 275; (2) T1 VISTA sequence: TR/TE 800/18.11 ms, FOV 2.00 \(\times\) 1.81 cm, layer thickness 0.6 mm, matrix 332 \(\times\) 330; (3) DWI: TR/TE 1000/47.48 ms, FOV 2.30 \(\times\) 2.30 cm, layer thickness s4.0 mm, matrix 112 \(\times\) 90; (4) T2WI: TR/TE 11,000/120 ms, FOV 1.87 \(\times\) 1.25 cm, layer thickness 6.0 mm, matrix 216 \(\times\) 116. According to the TOF-MRA image, 3D-T1-VISTA axial images were obtained by segmented scanning and perpendicular to the long axis of the vessel, when the BA is curved. 3D-T1-VISTA axial images were obtained via segmented scanning perpendicular to the long axis of the vessel when the BA is curved.

#### 1.3 Image Analysis

Draw a straight line connecting the apex of BA and the junction of the vertebrobasilar artery, which is called the “normal line”. Select the point farthest from the line in the MRA anteroposterior and side views as the maximum bending point of the BA. If the maximum bending point is not in the same location as that in the anteroposterior and side views, the position that is the farthest point from the standard line shall prevail. If a certain distance (>1 mm) between the inner wall of the BA (the one near the standard line) and the normal line at the level of the maximum bending point exists, then the BA curve is also considered to exist. The maximum bending point layer and the upper and lower five layers of the maximum bending point layer (total of 11 layers) are defined as the curved segment. The proximal end is defined as the proximal segment, and the distal end is defined as the distal segment (Fig. 1A). Because of the difference in the length of the BA, we counted the number of layers, and the plaque detected in each layer of the axial 3D-T1-VISTA sequence is counted as one. If the plaque is a continuous plaque involving more than two segments, the segment in which the thickest patch is located will be denoted as the plaque in that segment, and the patch layer that continuously appears with the plaque in that layer will be denoted as the plaque in that segment, until the discontinuous plaque mainly appears. Punctate or patchy short T1 signal appears in BA plaques, and the signal intensity is >150% of the T1 of the adjacent muscle, indicating intraplaque hemorrhage (IPH) (Fig. 1B)\(^b\). Transfer all imaging data to the assistant workstation; the Philips Intellispace Portal software was used to find the plane of maximum lumen stenosis plane, and the vascular area and lumen areas at this plane were measured. When the measured plane was inclined to the long axis of the vessel, Multiplar reconstruction (MPR) reconstruction was performed to adjust and ensure that the measured plane was perpendicular to the lumen. According to the formula, the wall area...
standard deviation; if it is a non-normal distribution, it is expressed by their interquartile range [M (Q1–Q3)]. The continuous variable comparison of categorical data uses one-way analysis of variance and Least Significant Difference (LSD) is used for correction, and the categorical variable comparison uses chi-squared test. The difference is statistically significant when $P<0.05$.

2. Results

The HR-MRI images of 146 patients were retrospectively analyzed. A total of 51 patients with BA curvature and who met the enrollment conditions were included in the study. There were 36 cases with BA plaque, and BA plaque was detected in 376 slices. A total of 14 patients had discontinuous plaques in more than two areas of the BA.

1.4 Statistical Analysis

Data are analyzed using IBM SPSS 23.0. The count data are expressed as a percentage. If the measurement data conforms to the normal distribution and the variance is uniform, it is expressed as mean ± standard deviation; if it is a non-normal distribution, it is expressed by their interquartile range [M (Q1–Q3)]. The continuous variable comparison of categorical data uses one-way analysis of variance and Least Significant Difference (LSD) is used for correction, and the categorical variable comparison uses chi-squared test. The difference is statistically significant when $P<0.05$.

Fig. 1. Examples of segments, intraplaque hemorrhage (IPH), measurement of quantitative data
A. The arrow shows the normal line; the pentagram shows the point farthest from the line. B. The arrow shows the short T1 signal, which represents intraplaque hemorrhage. C. The thin arrow shows the outline of the lumen area; the coarse arrow shows the outline of the vascular area.

Fig. 2. An “image example of a patient with proximal plaques
A. T1-VISTA shows an eccentric plaque (arrow). B. MRA shows the slice of the plaque (line). C. In DWI imaging, the level of the plaque (2A) finds the brainstem infarction.

and plaque load of the maximum lumen stenosis can be calculated as follows: Wall area = blood vessel area at plaque–lumen area. Plaque load = (1 – lumen area/vascular area) × 100% (Fig. 1C). If within 12W after an acute ischemic stroke (IS) or TIA, T2WI and/or DWI shows infarction lesions in the BA blood supply area (brainstem), then the patient are classified into symptom groups, and BA plaques found in the same layer or adjacent continuous layers of the infarct focus are defined as criminal plaques. BA plaques which not at the same or adjacent continuous layers of the infarcts and found in asymptomatic patients were classified as non-criminal plaques (Fig. 2-4). All objections were resolved through consensus when two radiologists disagreed.
segments (proximal vs. curved and distal, 19.4% vs. 9.8%, 5.9%, respectively), and the difference is significant ($P < 0.002$). However, there were no statistically significant differences in the vessel area, lumen area, and wall areas and plaque load at the maximum lumen stenosis between the groups (all $P > 0.05$). The characteristics of the plaques and vascular wall in each segment of the BA are shown in Table 2.

### Table 1. Clinical baseline characteristics between groups

| Groups                  | Proximal | Curved | Distal | $\chi^2$/F | $P$-value |
|-------------------------|----------|--------|--------|------------|-----------|
| Gender (males/%)        | 15/78.9  | 15/65.2| 9/81.8 | 1.491 *    | 0.474     |
| Age (years)             | 58.6 ± 8.09 | 61.74 ± 10.16 | 56.31 ± 8.98 | 1.397  | 0.257     |
| Hypertension (n/%)      | 15/78.9  | 17/73.9| 8/72.7 | 0.202 *    | 0.904     |
| Diabetes (n/%)          | 7/36.8   | 7/30.4 | 6/54.5 | 1.819 *    | 0.403     |
| Dyslipidemia (n/%)      | 7/36.8   | 15/65.2| 4/36.4 | 4.247 *    | 0.120     |
| History of smoking (n/%)| 7/36.8   | 12/52.2| 3/27.3 | 2.197 *    | 0.333     |
| History of drinking (n/%)| 7/36.8 | 9/39.1 | 5/45.5 | 0.218 *    | 0.897     |

Note: * the value of $\chi^2$

and diabetes; and history of smoking and drinking.

#### 2.1 Plaque Characteristics

A total of 376 BA plaques were detected in the proximal, curved, and distal segments, including 132 (35.1%) with intraplaque hemorrhage and 244 (64.9%) without IPH, respectively, and the plaque was located plaques at the proximal segment are more likely to have IPH than those at the curved and distal segments (proximal vs. curved and distal, 19.4% vs. 9.8%, 5.9%, respectively), and the difference is significant ($P = 0.002$). However, there were no statistically significant differences in the vessel area, lumen area, and wall areas and plaque load at the maximum lumen stenosis between the groups (all $P > 0.05$). The characteristics of the plaques and vascular wall in each segment of the BA are shown in Table 2.
curved and distal plaques, but there were no significant differences in the vessel area, lumen area, wall area, and plaque load at the maximum lumen stenosis between the groups. Previous studies have shown that the mechanical force changes due to the geometry of blood vessels mediate the pathophysiological process of atherosclerosis by regulating the signaling pathways of vascular endothelial cells\(^9\). This study found that the proximal and curved plaques in the curved BA are more common than the distal plaques, which may be closely related to hemodynamics. Ravensbergen \textit{et al.} proved that the local regions of low wall shear stress (WSS) (defined as wall shear stress \(\text{WSS} \leq 1 \text{ pa}\)) tends to occur at the junction of blood vessels and the curved inner wall of BA through a series of autopsy models \(^{14}\). WSS is the friction force caused by tangential endothelial blood flow and changes in hemodynamics. The resulting low WSS area combined with vascular risk factors can activate the subsequent inflammatory cascade and lead to the progression of atherosclerosis\(^{15, 16}\), so the curved segment of the BA is prone to form plaque. Secondly, the geometry of the BA has a great influence on the speed of the section\(^{17}\), which makes the blood vessel wall at the bend more prone to blood retention or damage to the endothelium of the blood vessel wall due to the effect of inertial force, so the curved BA promotes the formation of plaques. After the confluence of the bilateral vertebral arteries, the blood flows directly into the proximal segment of the BA, which causes the blood vessel wall in this area to bear a lot of blood pressure. The blood flow in the proximal segment slows down or oscillates. This change in

### 2.2 BA Plaque and Brainstem Stroke

Among all patients with BA plaques, 18 were in the symptomatic group and 18 were in the asymptomatic group. Among the 376 BA plaques detected, there were 128 criminal and 248 non-criminal plaques. The distribution of criminal and non-criminal plaques among each group is shown in Table 3. The results showed that there was no significant difference between the groups in the distribution of criminal plaques and non-criminal plaques \((P=0.356 > 0.05)\), indicating that the longitudinal distribution of BA plaques may not be an influential factor in symptomatic stroke.

### 3. Discussion

IS is the main reason for reducing life expectancy worldwide. Approximately 15\%–37\% of IS cases due to cerebral atherosclerosis, which are considered to be a vital etiology of IS. Therefore, early detection and intervention of cerebral atherosclerosis may help to reduce the risk of IS\(^{11}\). Recently, imaging technology has developed rapidly; technology of black -blood HR-MRI has become a powerful means to evaluate intracranial artery plaque\(^{12}\). HR-MRI helps to characterize intracranial plaques reliably and non-invasively. Clinical studies support its applicability in IS risk assessment and in vitro studies, and verify its ability to classify plaque features\(^{13}\). Therefore, this study used HR-MRI technology to identify and analyze BA plaque, and to explore the stability of plaques and correlation with clinical symptoms.

Our research results show that in the curved BA, the proximal plaque is more prone to IPH than the curved and distal plaques, but there were no significant differences in the vessel area, lumen area, wall area, and plaque load at the maximum lumen stenosis between the groups. Previous studies have shown that the mechanical force changes due to the geometry of blood vessels mediate the pathophysiological process of atherosclerosis by regulating the signaling pathways of vascular endothelial cells\(^9\). This study found that the proximal and curved plaques in the curved BA are more common than the distal plaques, which may be closely related to hemodynamics. Ravensbergen \textit{et al.} proved that the local regions of low wall shear stress (WSS) (defined as wall shear stress \(\text{WSS} \leq 1 \text{ pa}\)) tends to occur at the junction of blood vessels and the curved inner wall of BA through a series of autopsy models \(^{14}\). WSS is the friction force caused by tangential endothelial blood flow and changes in hemodynamics. The resulting low WSS area combined with vascular risk factors can activate the subsequent inflammatory cascade and lead to the progression of atherosclerosis\(^{15, 16}\), so the curved segment of the BA is prone to form plaque. Secondly, the geometry of the BA has a great influence on the speed of the section\(^{17}\), which makes the blood vessel wall at the bend more prone to blood retention or damage to the endothelium of the blood vessel wall due to the effect of inertial force, so the curved BA promotes the formation of plaques. After the confluence of the bilateral vertebral arteries, the blood flows directly into the proximal segment of the BA, which causes the blood vessel wall in this area to bear a lot of blood pressure. The blood flow in the proximal segment slows down or oscillates. This change in

### Table 2. Characteristics of plaques and vascular wall between segments of the basilar artery

|                | Proximal | Curved | Distal | Total | \(\chi^2/F\)  |
|----------------|----------|--------|--------|-------|--------------|
| IPH (n/\%)     | 166/44.1 | 114/30.3 | 96/25.5 | 376/100 | 12.345\(^a\) |
| Have           | 73/19.4  | 37/9.8 | 22/5.9 | 132/35.1 |
| None           | 93/24.7  | 77/20.5 | 74/19.7 | 244/64.9 |
| Vessel area (mm\(^2\)) | 26.3 ± 16.2 | 20.64 ± 9.55 | 19.69 ± 4.23 | 22.47 ± 11.85 | 1.708 |
| Lumen area (mm\(^2\)) | 7.75 ± 4.29 | 5.77 ± 2.67 | 6.00 ± 2.86 | 6.55 ± 3.45 | 2.00 |
| Wall area (mm\(^2\)) | 18.55 ± 14.57 | 14.86 ± 9.31 | 13.69 ± 4.13 | 15.93 ± 10.80 | 0.976 |
| Plaque load (%) | 0.68 ± 0.15 | 0.68 ± 0.12 | 0.69 ± 0.14 | 0.68 ± 0.13 | 0.054 |

Note: \(a\), the value of \(\chi^2\)

### Table 3. Distribution of criminal and non-criminal plaques

| Group (n/\%) | Proximal | Curved | Distal | Total | \(\chi^2\)  |
|-------------|----------|--------|--------|-------|-------------|
| Criminal plaques | 63/16.8 | 36/9.6 | 29/7.7 | 128/34.0 |
| Non-criminal plaques | 103/27.4 | 78/20.7 | 67/17.8 | 248/66.0 |
| Total       | 166/44.1 | 114/30.3 | 96/25.5 | 376/100.0 | 2.067\(^a\) |

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hemodynamics cascades a series of risk factors, leading to the formation of atherosclerotic plaques and damage to the plaques, which may also be proximal causes of bleeding within the plaque. IPH is considered to be one of the related factors of plaque instability. It can cause arterial embolism that can cause small vessel occlusion, and can cause branch occlusive disease that affects the perforating artery and ultimately leads to the occurrence of symptomatic cerebral infarction occur. Some studies have shown that the formation of BA plaques is related to BA curvature, therefore, patients with BA curvature should receive more clinical attention than patients without BA curvature. This study found that proximal plaques were more prone to happen IPH, which is highly correlated with plaque progression, as the plaque progresses, the fibrous cap covering it becomes thinner and causes active inflammation. Inflammatory factors in the plaque, leading to IS when the plaque is ruptured. Therefore, attention should be paid to the BA proximal plaque when it exists, and clinical intervention should be performed early. When the BA is not or mildly to moderately narrowed, use of drugs to stabilize the plaque prevents the plaques peeling off from the wall of a blood vessel and forming embolus to block the distal artery. If BA has been severely narrowed, appropriate interventional therapies, such as early stents prevention, should be selected after comprehensive evaluation, preventing BA occlusion and blood flow interruption, which may cause serious clinical consequences.

In extracranial carotid artery plaque imaging, it is supported that carotid artery IPH is a risk factor for recurrent stroke and has nothing to do with stenosis. Moreover, studies have shown that BA plaque IPH is in the symptomatic basement. The incidence of arterial stenosis is higher than that of the asymptomatic group. Although the degree of stenosis was not classified in this study, it was found that proximal plaques are prone to IPH. This suggests that proximal plaque may be more unstable and more likely to lead to clinical adverse events. While this study shows that longitudinal distribution of BA plaques may not be an influential factor in symptomatic stroke, but our study found there are 5 symptomatic patients appeared discontinuous plaques, even if plaques are present at two or three areas, the proximal plaques were all criminal plaques in all five patients. This suggests that there may still be a potential unknown relationship between the longitudinal distribution of BA plaques and brainstem stroke, which needs further study.

There are some limitations to this study. First, the sample size is small, and prospective experiments with big data are needed for further verification. Second, no enhanced scan was performed on these patients, so no information on inflammatory changes in the vascular wall was obtained. Third, because of incomplete clinical data in some patients, some baseline characteristics (including BMI, homocysteine, and family history of stroke) were not analyzed; Fourth, there is no direct hemodynamic evidence showing the difference in blood flow of different segments of the BA. Finally, thickening and plaques of the BA wall are frequent incidental findings in the elderly. In the presence of infarction or ischemia, these plaques cannot be separated from the criminal plaque in principle.

In summary, this study found that the proximal plaque is more prone to IPH. Clinical work can focus on the characteristics of the proximal BA plaque, which may provide new clinical ideas for the prevention of brainstem stroke.

Conflicts of Interests
The authors declare no conflicts of interests.

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