Effect of Fetal-type Posterior cerebral artery on cerebral infarction and collateral circulation

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Abstract

We studied 57 patients with cerebral infarction and Fetal-type Posterior cerebral artery (FTP), confirmed by MRA and CTA. 26 cases (45.61%) of Complete Fetal-type Posterior cerebral artery (cFTP), 21 cases (36.84%) of Partial Fetal-type Posterior cerebral artery (pFTP), 10 cases (17.54%) of unilateral cFTP. When the ipsilateral internal carotid artery occlusion of cFTP occurs, the bidirectional collateral circulation from the posterior circulation to the posterior communication artery to the anterior circulation cannot be established to compensate, simultaneous infarction of the ipsilateral anterior and posterior circulation can be caused. In the presence of pFTP, since the diameter of P1 was less than that of the posterior communicating artery (PcoA), the collateral circulation of posterior circulation-posterior cerebral artery-middle cerebral artery pia meningeal anastomosis could not be established, anterior circulation infarction was caused by ipsilateral internal carotid artery occlusion. In the presence of bilateral cFTP, the anterior circulation-posterior communication artery-basilar artery could not be established to compensate for the collateral circulation of other branches except bilateral posterior cerebral artery, resulting in bilateral cerebellar and brainstem infarction. FTP changes the cerebral artery circulation and collateral circulation pathways in the normal state.

Introduction

The Fetal-type Posterior cerebral artery (FTP) is a common anatomical variation of the circle of Willis. Due to the dysplasia of the posterior cerebral artery in embryo, one or both posterior cerebral arteries directly issue from the ipsilateral internal carotid artery. Because of the anterior segment (P1 segment) of FTP traffic is absence or dysplasia, the circle of Willis lacks the potential collateral circulation pathway. When cerebral artery stenosis, embolus detachment and cerebral hemodynamics change, rapid and effective compensation of collateral circulation cannot be established through the circle of Willis, which is prone to abnormal cerebral infarction events[1, 2]. With the progress of imaging technology, the influence of FTP on cerebrovascular diseases, especially its correlation with posterior circulation cerebral infarction, has been widely studied[3, 4]. However, studies on the effects of different types of FTP on collateral circulation in patients with cerebral infarction are still rare. This study retrospectively analyzed the medical records of 57 patients with cerebral infarction complicated with FTP, evaluated the imaging characteristics of collateral circulation in detail, and studied and analyzed the relationship between cerebral infarction, FTP classification and collateral circulation, so as to provide a reference for the precise development of individual intervention measures, optimal treatment decisions and clinical outcomes.

Methods

Between December 1, 2020 and January 1, 2022, 57 inhabitants of Department of General practice and Neurology, Rocket Force Characteristic Medical Center, whom with cerebral infarction complicated with FTP confirmed by Head and Neck CT angiography (CTA) and magnetic resonance angiography (MRA)
were selected. The patients included 35 males and 22 females, aged 65–78 years, with an average age of 72.06 ± 4.3 years.

Inclusion criteria: (1) brain MRI diffusion-weighted imaging sequence examination confirmed cerebral infarction; (2) Head and neck CTA or MRA examination confirmed the presence of FTP; (3) Complete clinical data and imaging data for analysis; (4) Be informed of the purpose and significance of the study.

FTP determination and typing standards: CTA or MRA of the head and neck showed that FTP originated from the ipsilateral internal carotid artery and continued directly to the posterior segment of the ipsilateral posterior cerebral artery. If P1 segment of the posterior cerebral artery was completely absent, it was complete FTP (cFTP). If P1 segment of the posterior cerebral artery exists and the diameter of P1 segment is less than that of the ipsilateral posterior communication artery, it is called partial FTP (pFTP).

Collateral circulation assessment: The collected MRI, MRA and CTA images were transferred to the workstation for processing, and the maximum density projection and volume reproduction techniques were used to reconstruct and analyze cerebral blood vessels. The stenosis of the internal carotid artery system and the vertebrobasilar artery system and the integrity of the circle of Willis were evaluated. And, the diameter of P1 segment of posterior cerebral artery and posterior communicating artery was compared.

Lateral branch opening of cerebral cortex pia meningeal were detected by CTA or MRA. All imaging data were reviewed by blind method. FTP, cerebral infarction, cerebral artery stenosis and collateral circulation were evaluated by two experienced associate chief physicians of neurology department and one associate chief physician of imaging department, and a consistent conclusion was reached.

**Results**

**FTP classification, cerebral artery lesions and cerebral infarction**

Among 57 patients, 26 (45.61%) were cFTP, 21 (36.84%) were pFTP, and 10 (17.54%) were pFTP on one side of cFTP and the other side of pFTP. Among these patients, there were 29 cases (50.88%) of anterior circulation cerebral infarction, 20 cases (35.09%) of posterior circulation cerebral infarction, and 8 cases (14.04%) of anterior and posterior circulation cerebral infarction (Table 1). When the vascular lumen reduction ratio above 25%, [(Normal blood vessel diameter - Minimum diameter of stenotic site)/ Normal blood vessel diameter]×100%, We see this as vascular stenosis. In addition, carotid artery stenosis was found in 15 cases (26.32%), vertebrobasilar artery stenosis was found in 25 cases (43.86%), and both carotid and vertebrobasilar artery stenosis was found in 17 cases (29.82%) by CTA. (Table 2)
Table 1
The number of patients with cerebral infarction at different sites and their FTP classification

<table>
<thead>
<tr>
<th>Infarction locations</th>
<th>Total number of patients (prevalence %)</th>
<th>Number of cFTP</th>
<th>Number of pFTP</th>
<th>Number of one side cFTP and another pFTP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior circulation infarction</td>
<td>29 (50.88%)</td>
<td>12</td>
<td>11</td>
<td>6</td>
</tr>
<tr>
<td>Posterior circulation infarction</td>
<td>20 (35.09%)</td>
<td>10</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Anterior and posterior circulatory cerebral infarction</td>
<td>8 (14.04%)</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Total number and prevalence of patients</td>
<td>26 (45.61%)</td>
<td>21</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 2
The number and prevalence of patients at different locations

<table>
<thead>
<tr>
<th>Stenosis locations</th>
<th>Total number of patients</th>
<th>Prevalence of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid artery</td>
<td>15</td>
<td>26.32%</td>
</tr>
<tr>
<td>Vertebrobasilar artery</td>
<td>25</td>
<td>43.86%</td>
</tr>
<tr>
<td>both Carotid and Vertebrobasilar artery</td>
<td>17</td>
<td>29.82%</td>
</tr>
</tbody>
</table>

**Effects Of Ftp On Cerebral Infarction And Collateral Circulation**

In the presence of cFTP, the posterior cerebral artery is completely supplied by the ipsilateral internal carotid artery, and the bidirectional collateral circulation from the posterior circulation to the posterior communication artery to the anterior circulation cannot be established to compensate. When the ipsilateral internal carotid artery occlusion of cFTP occurs, the presence of cFTP leads to simultaneous infarction of the ipsilateral anterior and posterior circulation (Fig. 1 and Fig. 2). The collateral circulation of posterior circulation-posterior cerebral artery-middle cerebral artery pia meningeal anastomosis could not be established to compensate (Fig. 2).

In the presence of pFTP, the posterior cerebral artery is mainly supplied by the ipsilateral internal carotid artery and partly by the posterior circulation. Since the diameter of P1 was less than that of the posterior communicating artery, the effective collateral circulation from the posterior circulation-posterior communicating artery-anterior circulation could not be established to compensate, resulting in anterior circulation infarction in ipsilateral internal carotid artery occlusion of pFTP (Figs. 3 and 4). However, in the case of vertebrobasilar artery occlusion, the ipsilateral internal carotid artery - posterior communication artery - P1 segment of posterior cerebral artery - distal basilar artery - bilateral superior
cerebellar artery collateral circulation can be established to compensate for pFTP, resulting in brain stem infarction (Fig. 4).

In the presence of bilateral cFTP, bilateral posterior cerebral arteries are completely supplied by bilateral internal carotid arteries. When the internal carotid artery was occluded, the anterior circulation-posterior communication artery-basilar artery could not be established to compensate for the collateral circulation of other branches except bilateral posterior cerebral artery, resulting in bilateral cerebellar and brainstem infarction (Fig. 5).

**Discussion**

FTP refers to the posterior cerebral artery that originates directly from the internal carotid artery system and is the most common congenital variation of the circle of Willis. It has been reported that FTP is found in 11% ~ 46% of adults, and the incidence of unilateral FTP is 4 ~ 26%, and the incidence of bilateral FTP is L % ~ 9%[1, 2, 5]. As an important component of the circle of Willis, the posterior cerebral artery plays an important role in establishing the primary collateral blood flow compensation of the anterior and posterior circulation. However, the variation of the anatomical structure of FTP changes the conventional potential collateral pathways, resulting in abnormal clinical outcomes [2, 6]. Previous studies have suggested that FTP increases the risk of posterior circulation cerebral infarction, but the results have been inconsistent if the classification of FTP and its association with cerebral artery stenosis and collateral circulation are considered. This research showed that the proportion of anterior circulation cerebral infarction, posterior circulation cerebral infarction and anterior and posterior circulation cerebral infarction in 57 patients were 50.88%, 35.09% and 14.04% respectively, and the proportion of posterior circulation cerebral infarction was significantly lower than that of anterior circulation cerebral infarction. The possible reasons are as follows: First, due to the presence of FTP, the blood perfusion in the internal carotid arterial-posterior communicating artery is increased, resulting in the change of pressure gradient, leading to the damage of vascular wall, and the internal carotid artery is more prone to atherosclerotic lesions under the influence of risk factors[10], Secondly, since FTP originates from the ipthalateral internal carotid artery, FTP increases the blood supply burden of the internal carotid artery system, and it is more prone to watershed cerebral infarction when hemodynamic instability, such as hypoperfusion, occurs (Fig. 1). Thirdly, when severe stenosis or occlusion of internal carotid artery causes changes in hemodynamics, cFTP cannot quickly and effectively establish the compensation of lateral branches of the posterior circulation through the posterior communicating artery of the anterior circulation, and the compensation of lateral branches of pia meningeal anastomosis between the posterior cerebral artery, middle cerebral artery and anterior cerebral artery. Even in the presence of pFTP, it is possible that the ipsilateral internal carotid artery-posterior communication artery-posterior cerebral artery P1 segment - basilar artery collateral circulation of pFTP is established to compensate for severe basilar artery stenosis or occlusion, further aggravating anterior circulation ischemia, thus increasing the risk and severity of anterior circulation cerebral infarction in patients[1, 11–14]. In addition, since FTP completely belongs to the internal carotid artery system, when the internal carotid artery system is completely occluded, hypoperfusion or embolus fall off, simultaneous infarction of the anterior and posterior circulation.
contrary to the normal situation may occur (Figs. 1 and 2). The presence of FTP increases the blood flow of the internal carotid artery, leading to reduced perfusion of the basilar artery. The basilar artery in the long-term state of low blood flow gradually becomes thinner, and the perforating artery of the basilar artery may also be reduced or occluded. It has been reported that the basilar artery diameter of patients with FTP decreased by 18% compared with the normal population. In particular, bilateral FTP patients are more likely to be complicated with basilar artery hypoplasia. Due to the slender basilar artery itself, the proportion of patients prone to artery stenosis increases [11]. The results of this study indicated that the proportion of FTP combined with vertebrobasilar artery stenosis or occlusion was 43.86% (25/57), which was significantly higher than that of carotid artery system stenosis or occlusion 26.32% (15/57).

When the vertebrobasilar artery system is occluded, pFTP can compensate for the blood supply from the anterior circulation to the posterior circulation through the posterior communication artery and P1 segment, and its compensatory capacity depends on the diameter of the posterior communication artery and P1 segment, which can alleviate or avoid the occurrence of cerebral infarction in the posterior circulation in the non-posterior cerebral artery supply area. When pFTP was combined with bilateral vertebral artery V4 segment occlusion, the compensatory route of collateral circulation was as follows: ipsilateral internal carotid artery - posterior communication artery - posterior cerebral artery P1 segment - distal basilar artery - bilateral superior cerebellar artery, which could easily lead to brain stem infarction due to failure to compensate middle and proximal basilar artery (Fig. 4). When cFTP was ipsilateral internal carotid artery with basilar artery occlusion, the bidirectional collateral circulation from posterior circulation to posterior communicating artery to anterior circulation could not be established to compensate (Figs. 1 and 2). When bilateral cFTP was combined with basilar artery occlusion, the anterior circulation-posterior communication artery-basilar artery could not be established to compensate for the collateral circulation of other branches except bilateral posterior cerebral artery, resulting in bilateral cerebellar and brainstem infarction (Fig. 5). In the presence of internal carotid artery occlusion, pFTP compensated blood supply from the posterior circulation to the anterior circulation through P1 segment and the posterior communication artery. Since the diameter of P1 is smaller than that of the posterior communication artery, the effective collateral circulation from the posterior circulation-right posterior communication artery-right anterior circulation cannot be established to compensate, resulting in an increased risk of anterior circulation infarction. The main collateral circulation compensation channels for ipsilateral internal carotid artery occlusion of pFTP include: anterior communicating artery collateral, ipsilateral anterior cerebral artery-middle cerebral artery pia meningeal anastomosis collateral, basilar artery-ipsilateral posterior cerebral artery P1 segment - posterior communicating artery-ipsilateral internal carotid artery C7 segment - ipsilateral middle cerebral artery collateral circulation compensation (Fig. 3). When cFTP was combined with ipsilateral internal carotid artery and middle cerebral artery occlusion, the collateral circulation of ipsilateral posterior cerebral artery-middle cerebral artery pia meningeal anastomosis could not be established to compensate (Fig. 2).

The influence of FTP combined with cerebral artery occlusion on the pathogenesis and collateral circulation of cerebral infarction is relatively complex. The subsequent study will include more cases,
analyze the hemodynamic characteristics related to FTP in depth by combining DSA and TCD, and further study the clinical significance of FTP.

**Conclusions**

FTP changes the compensatory pathway of lateral collateral of anterior and posterior circulations in the normal state, and the possibility of FTP should be considered when simultaneous infarction occurs in anterior and posterior circulations or abnormal embolism events with uncertain embolus source.

**Declarations**

**Disclosures**

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the PLA Rocket Force Characteristic Medical Center (Beijing, 100088, China) with the ethical approval number KY20190342. The Ethics Committee of the PLA Rocket Force Characteristic Medical Center agreed to obtain patient consent through telephone interview. Oral informed consent was achieved from each patient through telephone interview. All experiments were performed in accordance with relevant guidelines and regulations of Declaration of Helsinki.

Consent for publication

Not applicable.

Availability of data and materials

The data and images used in the current study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Fan Bai, Bei Hou, Hui Liu - study design, data acquisition, interpretations of results, manuscript preparation and revisions;

Lei Zhu, Shanshan Guo, Chao Yang - interpretations of results, manuscript preparation and revisions;
Minghao Liu and Suhe Dong - data acquisition, manuscript revision;
Aihua Huang and Jianan Wang - study design, manuscript revision. All authors read and approved the final manuscript.

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References


**Figures**

![Figure 1](image-url)
Male, 70 years old. (a and b) MRI shows new infarcts in the left anterior and posterior circulation. (c) CTA showed subtotal occlusion of the initial segment of the left internal carotid artery. (d) CTA shows bilateral cFTP; arrows show severe basilar artery stenosis; The collateral circulation of posterior circulation-left posterior communication artery-left anterior circulation could not be established to compensate; A collateral circulation from the right anterior circulation to the right posterior communication artery to the posterior circulation cannot be established to compensate.

Figure 2

Male, 68 years old. (a and b) MRI showed new infarcts in the left parietal, temporal, and occipital lobes. (c) CTA showed stenosis at the initial segment of the left common carotid artery, and the arrow showed subtotal occlusion at the initial segment of the left internal carotid artery. (d) CTA showed the left middle cerebral artery stenosis, right before the cerebral artery occlusion, arrow show the right PcoA, the right side of the P1 section of pipe diameter = right PcoA diameter arrow show left cFTP, cannot build cycle - the left PcoA - on the left side of the first cycle and the cycle - the left posterior cerebral artery - the left middle cerebral artery pia mater anastomosis compensatory collateral circulation.
Figure 3

Female, 72 years old. (a) MRI showed new infarcts in the right lateral ventricle, and no infarcts in the blood supply area of the right posterior cerebral artery. (b) CTA showed right internal carotid artery occlusion, A1 segment of right anterior cerebral artery was not developed, and anterior communication artery was developed. (c) CTA shows that arrow is the right pFTP, the diameter of P1 segment of the right posterior cerebral artery is less than the diameter of the right posterior communication artery, the initial segment of the right PcoA is narrow, arrow is the left cFTP, and the effective collateral circulation from the posterior circulation-right PcoA-right anterior circulation cannot be established as compensation. The compensatory pathways for right internal carotid artery occlusion with right pFTP collateral circulation are as follows: Left internal carotid artery – left anterior cerebral artery A1 segment – anterior communication artery – right anterior cerebral artery A2 segment – right middle cerebral artery pia anastomosis collateral – right middle cerebral artery, basilar artery – right posterior cerebral artery P1 segment – right posterior communication artery – right internal carotid artery C7 segment – right middle cerebral artery.

Figure 4

Male, 78 years old. (a) MRI showed a new infarction in the left pons. (b): MRA showed bilateral vertebral artery V4 segment occlusion, basilar artery was not developed; Arrow is the right cFTP, arrow is the left pFTP, and the diameter of P1 segment of the left posterior cerebral artery is less than that of the left posterior communication artery. The compensatory route of collateral circulation for bilateral vertebral artery occlusion is: left internal carotid artery - left PcoA - left posterior cerebral artery P1 segment - distal basilar artery - bilateral superior cerebellar artery, resulting in brain stem infarction.
Figure 5

Female, 70 years old. A: MRI showed new infarcts in bilateral cerebellum and right pons. B: CTA showed bilateral vertebral artery V4 segment occlusion, and basilar artery was not developed. C: CTA showed bilateral vertebral artery V4 segment occlusion, basilar artery was not developed, bilateral cFTP; Unable to establish the anterior circulation- PcoA -basilar artery to compensate for the collateral circulation of other branches except bilateral posterior cerebral arteries, resulting in bilateral cerebellar and brainstem infarction.