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Clinical Analysis of Embryonic Posterior Cerebral Artery

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Abstract: *Objective:* To understand the clinical characteristics of patients with embryonic posterior cerebral artery and its correlation with abnormal vascular development. *Methods:* The clinical data of 396 patients with embryonic posterior cerebral artery confirmed by magnetic resonance angiography (MRA) and computed tomography angiography (CTA) were analyzed. *Results:* Two-hundred patients had clinical manifestations of posterior circulation ischemia, including recurrent dizziness, vertigo, and tinnitus; 45 had headaches, 97 had limb weakness, and 16 patients had syncope or impaired consciousness. Seventy-six patients with circulatory infarction were admitted to the hospital. There were 251 patients with history of hypertension, 74 with diabetes, 113 with hyperlipidemia, 13 with dominant vertebral artery, 10 with intracranial aneurysm, and 19 with absence of A1 segment of the anterior cerebral artery (considering developmental variation). *Conclusion:* Embryonic posterior cerebral artery develops abnormally during the embryonic period, often accompanied by abnormal vascular access. Due to abnormal hemodynamics, the incidence of posterior circulation ischemia, aneurysm, and infarction increases in such patients.

Keywords: Embryonic posterior cerebral artery; Posterior circulation ischemia; Posterior circulation infarction; Intracranial aneurysm

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1. Introduction

The circle of Willis is an important collateral circulation in the brain, mainly composed of the A1 segment of bilateral anterior cerebral artery, anterior communicating artery, P1 segment of bilateral posterior cerebral artery, and posterior communicating artery [1]. The most common type of congenital vascular morphological abnormality of the posterior circulation is the embryonic posterior cerebral artery (fetal-type posterior cerebral artery, FTP). FTP is one of the common vascular variations that causes an incomplete circle of Willis, which is closely related to cerebral ischemia. Its incidence is about 5.22% [2]. Due to the abnormal development of the posterior cerebral artery during the embryonic period, the circle of Willis cannot establish rapid and effective collateral compensation; thus, it is prone to ischemic cerebrovascular events [3]. The clinical data of 396 patients with embryonic posterior cerebral artery were analyzed by magnetic resonance angiography (MRA) and computed tomography angiography (CTA) in our hospital.

2. Materials and methods

2.1. General data and methods

Inpatients who were found with embryonic posterior cerebral artery in our hospital using Siemens 64-slice CT machine or 1.5 T or 3.0 T magnetic resonance imaging (MRI) scanner for cerebrovascular imaging

from January 2016 to December 2019 were included in the study. By observing the occurrence of embryonic posterior cerebral artery and recording the location of embryonic posterior cerebral artery (i.e., left or right or double) in each patient, a total of 396 cases were included, among which 194 were male, aged 18–88 years, with an average age of 64, and 202 were female, aged 35-92 years, with an average age of 65 years.

2.2. Embryonic posterior cerebral artery diagnostic criteria

The posterior communicating artery originates from the internal carotid artery and continues to the ipsilateral posterior cerebral artery. The outer diameter of the posterior communicating artery is normally larger than the ipsilateral posterior cerebral artery P1 diameter. The condition in which P1 segment is absent is known as complete (c)FTP, while the presence of a small P1 segment is known as partial (p)FTP [4,5]. The main clinical manifestations of posterior circulation infarction are dizziness/vertigo, diplopia, dysarthria, dysphagia, fall episodes, ataxia; other symptoms include speech disturbance, impaired consciousness, vomiting, pain over the head and neck, tinnitus, hearing loss, perioral numbness, limb numbness and weakness, *etc*. [6,7]. The MRI manifestations of cerebral infarction include low signal on T1-weighted image (WI), high signal on T2WI and fluid attenuated inversion recovery (FLAIR), as well as new and old cerebral infarction lesions.

2.3. Exclusion criteria

Patients who had head trauma, arteriovenous malformation, heart disease, different degrees of anemia, space-occupying lesions, intracranial infection, poisoning, dizziness caused by non-nervous system diseases, drug use, and mental disorders were all excluded from the study.

2.4. Statistical analysis

The clinical manifestations of all 396 patients were summarized statistically.

3. Results

Embryonic posterior cerebral artery was found in 396 hospitalized patients using CTA, MRA, or DSA. Among the 396 patients, 194 were male and 202 were female. As shown in **Table 1**, 110 (27%) patients had bilateral FTP, 117 (30%) had left FTP, and 169 (43%) had right FTP.

Table 1. Analysis and comparison of clinical symptoms

Type	Number of cases	Dizziness, vertigo, and tinnitus	Syncope/impaired consciousness	Headache	Limb weakness
Bilateral	110	50	4	9	22
Left	117	61	5	8	37
Right	169	89	6	28	38

Among the patients, 200 patients presented with symptoms of posterior circulation ischemia, including recurrent dizziness, vertigo, and tinnitus; 45 had headaches, 97 had limb weakness, and 16 patients had syncope or impaired consciousness. There were 251 patients with history of hypertension, 74 with diabetes, and 113 with hyperlipidemia.

Among the 200 patients who presented with symptoms of posterior circulation ischemia, 22 developed posterior circulation cerebrovascular accidents, accounting for 11%; among the 61 cases of left embryonic posterior cerebral artery, there were 6 cases of vascular accidents, accounting for 9.8%; among the 89 cases

of right embryonic posterior cerebral artery, there were 6 cases of posterior circulation cerebral infarction, accounting for 6.7%; among the 50 cases of bilateral embryonic posterior cerebral artery, 10 patients had posterior circulation cerebral infarction, accounting for 20%.

There were 13 cases of dominant vertebral artery, 10 cases of intracranial aneurysm, and 19 cases of absent A1 segment (considering developmental variation).

With regard to the posterior circulation relationship of embryonic posterior cerebral artery with cerebral infarction, among the 117 cases of left embryonic posterior cerebral artery, 20 cases had posterior circulation cerebral infarction, accounting for 17%; among the 169 cases of right embryonic posterior cerebral artery, 19 had posterior circulation cerebral infarction, accounting for 19%; among the 110 cases of bilateral embryonic posterior cerebral artery, 37 had posterior circulation cerebral infarction, accounting for 33.6%.

4. Discussion

Embryonic posterior cerebral artery refers to a type of vascular structure, in which the origin of the posterior cerebral artery (P1 segment) is abnormal compared with the normal anatomy. Under physiological conditions, the posterior cerebral artery originates from the basilar artery, the embryonic posterior cerebral artery continues to develop downward from the ipsilateral posterior communicating artery, and the internal carotid artery system replaces the basilar artery as the main blood supply to the brain tissue (mainly the base of the temporal lobe, occipital lobe, brainstem, thalamus, geniculate body, *etc*.).

Studies have shown that the incidence of FTP is between 4% and 46% [8]. Among them, the incidence rate of unilateral and bilateral pFTP is 11%~29% and 1%~9%, respectively, while the incidence rate of unilateral and bilateral cFTP is 4%~26% and 2% ~4%, respectively [9]. Embryonic posterior cerebral artery is often defined as the absence or hypoplasia of ipsilateral P1 segment. The reason may be that the development of the vertebral basilar artery is later than that of the internal carotid artery and the thickened posterior communicating artery replaces or partially replaces the blood supply of the P1 segment. The development of the P1 segment of the vertebrobasilar artery origin is thus inhibited.

This study found that the proportion of patients with dizziness in cases of embryonic posterior cerebral artery and the incidence of posterior circulation infarction in those with bilateral embryonic posterior cerebral artery were high. According to several studies, the MRA of most patients with embryonic posterior cerebral artery shows that the diameter of the vertebrobasilar artery is thinner, the diameter of the basilar artery is not significantly different from that of the dominant vertebral artery, and the diameter of the superior cerebellar artery is smaller than that of the posterior inferior cerebral artery. Dizziness, tinnitus, and other manifestations are observed clinically. In patients with bilateral posterior cerebral arteries, dehydration, frequent vomiting, and other factors lead to insufficient blood volume; heart failure and arrhythmia may cause unstable blood pressure or subclavian artery steal blood, which can aggravate the insufficient blood supply of the basilar artery. As a result, the blood flow of the perforating arteries (long circumflex arteries, short circumflex arteries, *etc*.) supplying the brain stem from the basilar artery decreases. Patients with FTP are more likely to have congenital variations, such as dysplasia or absence of the A1 segment of the ipsilateral anterior cerebral artery [10]. It is not conducive to the establishment of other collateral circulation, thus easily causing brainstem ischemia. The activation system of the ascending reticular structure and the functions of the pyramidal tract and brainstem nuclei or cerebellum are inhibited; thus, a spectrum of symptoms of brainstem or cerebellar ischemia may occur, such as limb weakness, ataxia, loss of consciousness, falls, dizziness, tinnitus, nausea and vomiting, *etc*. This is the similar to Li Dongxu *et al*.'s study of 148 patients with abnormal cerebrovascular structures [11].

This study also found that the incidence of aneurysm was higher in patients with embryonic posterior cerebral artery, similar to those with fetal variant of the circle of Willis [12]. The internal carotid artery

system replaces the basilar artery to ensure blood supply to the respective brain tissue in the embryonic posterior cerebral artery population. This shunt phenomenon inevitably leads to a decrease in blood flow to the area of the brain supplied by the internal carotid artery system. In the unilateral embryonic posterior cerebral artery population, the blood flow velocity in the ipsilateral internal carotid artery system (especially the ipsilateral middle cerebral artery and posterior communicating artery) is significantly higher than that of normal vessels under the same conditions; this changes the corresponding intravascular pressure to a certain degree. The abnormal vascular access and hemodynamics in the embryonic posterior cerebral artery population indicate that the blood flow of the posterior cerebral artery originates from the ipsilateral internal carotid artery system. In that case, the corresponding blood vessels need to compensate by increasing the blood flow, thus increasing the intravascular pressure. Under the long-term intravascular load of the ipsilateral internal carotid artery system, the inner wall of the blood vessel is likely to be damaged, thus resulting in atherosclerosis, which leads to local stenosis and consequently the formation of aneurysms.

In conclusion, for patients with recurrent vertigo attacks and loss of consciousness, routine cranial MRA and cranial perfusion examination should be performed to determine if the patient has embryonic posterior cerebral artery and ischemia. For patients with FTP, MRA or CTA of the head should be done regularly for early detection and treatment of aneurysms, so as to prevent the fatal consequences of aneurysm rupture.

Disclosure statement

The authors declare no conflict of interest.

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