

# Evaluation of Clinical Features and Stroke Etiology in Patients with Bilateral Middle Cerebellar Peduncle Infarction

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## Keywords

Middle cerebellar peduncle · Anterior inferior cerebellar artery · Bilateral infarctions · Hypoperfusion · Large artery atherosclerosis · Bilateral middle cerebellar peduncle infarction

## Abstract

**Objective:** The aim of this study was to characterize clinical features, etiologies, and mechanisms of strokes due to bilateral middle cerebellar peduncle infarction (BMCPI). **Methods:** Cases diagnosed as BMCPI in our hospital were retrieved, and a literature review was performed. Data on clinical features and brain MRI were obtained. Extracranial and intracranial segments of the vertebrobasilar artery were assessed by using digital subtraction angiography, magnetic resonance angiography, or computed tomography angiography. **Results:** Thirteen cases (11 men and 2 women) of BMCPI were identified. A high-intensity signal of diffusion-weighted imaging sequence involving the bilateral middle cerebellar peduncle was observed in all patients. Most patients experienced vertigo, dysarthria, ataxia, and hearing disorders. Eleven of these cases were classified as large ar-

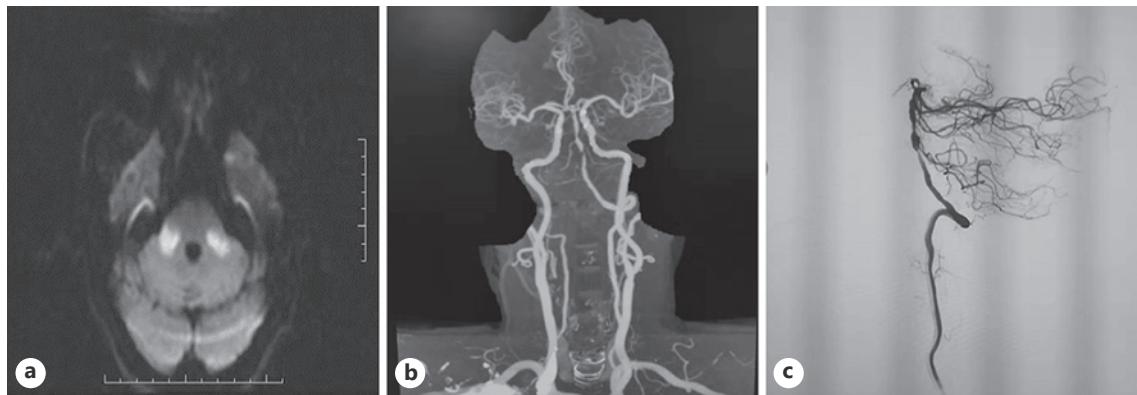
tery atherosclerosis, one as traumatic vertebral artery (VA) dissection, and one as giant cell arteritis. **Conclusion:** BMCPI is a rare cerebrovascular disease characterized by vertigo, ataxia, and dysarthria, which may also be accompanied by a hearing deficit or clinical signs of brainstem damage. BMCPI may be associated with hypoperfusion secondary to occlusive disease of the bilateral VA or proximal basilar artery.

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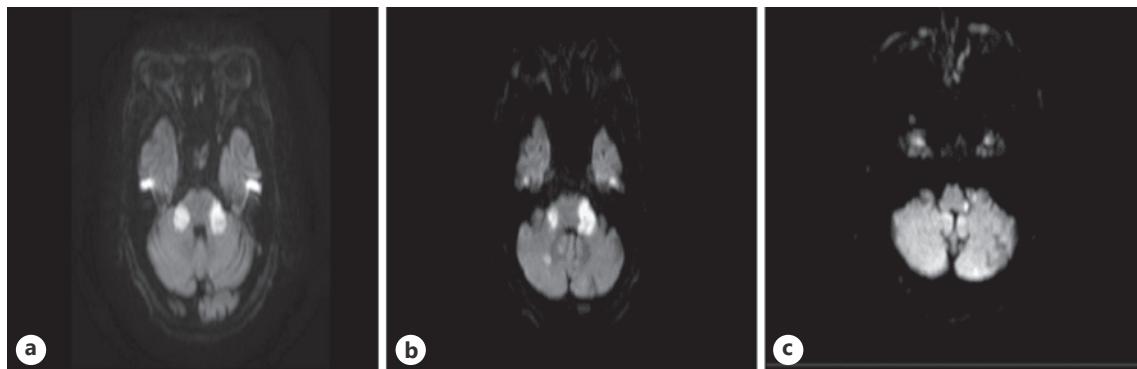
## Introduction

The middle cerebellar peduncles (MCPs) are paired structures connecting the cerebellum to the pons and are located in the watershed area between the anterior inferior cerebellar artery (AICA) and superior cerebellar artery [1]. MCPs are mainly supplied by the AICA but also partly receive irrigation from the superior cerebellar artery, which anastomoses with the AICA. As a result, MCPs are recognized as the radiological hallmark of AICA territory infar-

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**Fig. 1.** Diffusion-weighted magnetic resonance imaging showing BMCPI (a); CTA showing hypoplastic RVA and severe stenosis of the intracranial LVA (b); DSA: lateral view of LVA injection confirmed the stenosis of the intracranial LVA (c). BMCPI, bilateral middle cerebellar peduncle infarction; CTA, computed tomography angiography; RVA, right vertebral artery; LVA, left vertebral artery; DSA, digital subtraction angiography.



**Fig. 2.** DWI revealed the high-intensity signal involving the BMCPs (a, b), bilateral inferior lateral pons (more prominent on the left side) (b), dorsal lateral medullary oblongata (c), and cerebellar hemisphere (b). DWI, diffusion-weighted imaging; BMCPs, bilateral middle cerebellar peduncles.

tion [2]. However, AICA infarction is infrequent due to the abundant collateral circulation and is estimated to occur in only 0.9% of patients with acute stroke [3]. Bilateral middle cerebellar peduncle infarction (BMCPI) is more uncommon and is reported to be associated with vertigo, dysarthria, hearing loss, and ataxia [4–11]. Extensive profiling of BMCPI clinical features, etiologies, and mechanisms of strokes has not been reported. Therefore, in this study, we aimed to investigate and summarize BMCPI aspects utilizing our case series and a literature review.

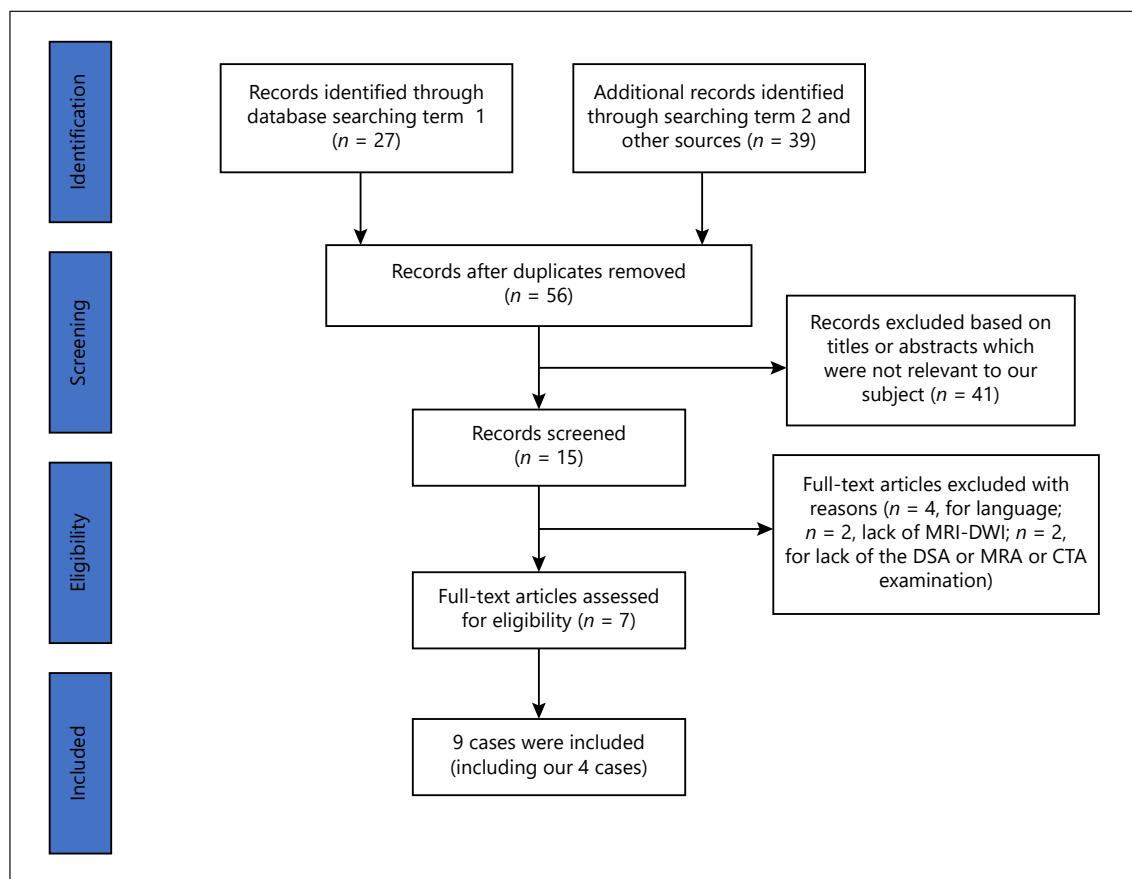
## Methods

### Representative Case Presentation

Case 1: a 68-year-old man with a 20-year history of hypertension was admitted to our neurology ward for a 12-day history of vertigo, slurred speech, and unsteady gait. He also had a smoking

and alcohol consumption history of >20 years. Neurological examination upon admission revealed direction-changing horizontal nystagmus and upbeat nystagmus in up gaze without gaze paresis. We also found hypoesthesia of the left side and face and prominent dysarthria and ataxia in all 4 limbs and the trunk. The patient's NIH Stroke Scale score was 4. Diffusion-weighted imaging (DWI) revealed a high-intensity signal involving the BMCPs, pons, and bilateral cerebellar hemispheres (Fig. 1a). Computed tomography angiography (CTA) and digital subtraction angiography (DSA) showed hypoplasticity of the right vertebral artery (VA) and severe stenosis of the intracranial segment of the left VA (Fig. 1b, c). Transthoracic echocardiography revealed left ventricular hypertrophy, but 24-h Holter monitoring revealed no significant arrhythmias. After determining that the stroke mechanism was likely attributed to hypoperfusion secondary to large artery atherosclerosis, the patient underwent left VA angioplasty.

Case 2: a 74-year-old woman with a long-standing history of hypertension and diabetes mellitus was admitted to the hospital after a sudden onset of vertigo, slurred speech, and difficulty in walking. Neurological examination revealed bilateral horizontal



**Fig. 3.** The selection process. DWI, diffusion-weighted imaging; CTA, computed tomography angiography; MRA, magnetic resonance angiography; DSA, digital subtraction angiography.

nystagmus, dysarthria, ataxia in all 4 limbs and the trunk, left facial weakness, and Horner sign on the left side. DWI showed an acute infarction involving the BMCP, bilateral inferior lateral pontine (more prominent on the left side), left dorsal lateral medulla oblongata, and cerebellar hemisphere (Fig. 2). Severe, multiple stenoses of the left VA, right VA, and BA were found on magnetic resonance angiography (MRA). Holter monitoring for 24 h were normal. The patient refused to undergo a DSA examination for further evaluation and resolution. She was then treated with oral dual antiplatelet (acetylsalicylic acid and clopidogrel) therapy.

#### Literature Search

The authors reviewed the literature for cases of BMCPI, searching the PubMed databases using the following terms: “[bilateral] AND [middle cerebellar peduncle] AND [infarction]” or “[bilateral] AND [anterior inferior cerebellar artery] AND [infarction].” A hand-search by reviewing the reference sections of the retrieved articles was also performed. Non-English language articles were excluded.

#### Literature Review Results

We identified 66 articles published between February 1983 and June 2019 upon initial electronic literature search and manual re-

trieval. After eliminating duplicates, 56 articles remained, yet 42 more were excluded based on the titles or abstracts. Four articles were also excluded because they were not in English. DWI result was absent in 2 cases, and detailed MRA, CTA, or DSA assessment for VA and BA were absent in 2 cases. In total, we identified 13 cases, 9 published and 4 of our own. The selection process is shown in Figure 3.

We performed a detailed assessment of these 13 patients with BMCPI. Basic clinical data, risk factors, etiology/stroke mechanisms, and radiological findings are summarized in Table 1. Our cohort included 11 men and 2 women. The age of onset ranged from 47 to 74 years, and the mean age was 62 years. Multiple patients had known vascular risk factors, including 11 patients with hypertension, 4 with diabetes mellitus, 4 with a smoking history, and 4 with a history of significant alcohol consumption. Vertigo, dysarthria, and ataxia were the primary clinical manifestations in most patients. Hearing deficits were reported in 6 patients (No. 3, 4, 6, 7, 8, and 9). Peripheral facial paralysis occurred in 2 patients (No. 2 and 13). Hemiparesesthesia was found in 3 patients (No. 2, 6, and 13) and 1 patient (No. 13) presented with a Horner sign.

DWI revealed characteristic lesions involving the BMCPs in all patients. Six cases had infarction limited to the BMCPs. The re-

**Table 1.** Basic clinical data, risk factors, etiology, stroke mechanism, and radiological findings associated with BMCPI

No.	Age, years/ sex	Risk factors	Clinical symptoms or signs	Lesion location (DWI)	Vascular abnormality	Etiology; mechanism	Treatment (acute period)	Reference
1	55/M	HTN, HI, and DM	Vertigo	BMCP (limited)	Occlusion of distal RVA and the stenosis of distal LVA; occlusion of the BA in close to the origin of the AICA	LAA; hypoperfusion <sup>1</sup>	Dual antiplatelets <sup>2</sup>	John et al. [4]
2	51/M	HTN and smoking	Dizziness, numbness of the left side of the face, ataxia, dysarthria; nystagmus, and left peripheral facial paralysis	BMCP, the left flocculus, and left anterolateral pons	Extracranial occlusion of the LVA at the C1 level and severe stenosis at the origin of the LVA; occlusion of the RVA at the origin	LAA; hypoperfusion	Therapy with Aggrastat	Kalla et al. [5]
3	63/M	HTN, smoking, and hyperuricemia	Vertigo, auditory distortion, nystagmus, dysarthria, and ataxia	BMCP	Occlusion of the RVA (C1-foramen magnum) and hypoplastic of LVA	LAA; hypoperfusion	Ticlopidine (oral)	Kataoka et al. [6]
4	59/M	Unknown	Dizziness, dysarthria, nystagmus, ataxia, and bilateral hearing disturbance	BMCP	Complete occlusions in the LVAs and RVAs at the C1-C2 level	VAD; hypoperfusion	–	Akiyama et al. [7]
5	72/M	HTN, smoking, and DR	Dysarthria, nystagmus, and ataxia	BMCP and cerebellar hemispheres	Bilateral vertebral artery occlusions at the origin	GCA; hypoperfusion	Intravenous methylprednisolone, clopidogrel, and aspirin	Healy et al. [8]
6	50/M	HTN, HI, smoking and DR	Vertigo, slurred speech, right side numbness and weakness, unsteady gait, right hearing loss, right ataxia, and right hemihypesthesia; left eye abduction dysfunction and diplopia	BMCP, lateral pons, right thalamus, bilateral cerebellar hemispheres, and vermis	Occlusion of the intracranial LVA and severe stenosis of the intracranial RVA and BA	LAA; hypoperfusion	Angioplasty stent placed	Dong et al. [9]
7	61/M	HTN, HI, smoking, DR, and AF	Vertigo, nystagmus, slurred speech, dysarthria, dysphagia, bilateral hearing loss, and somnolence	BMCP and bilateral inferior lateral pontine	Invisible of the RVA (intracranial segment)	LAA; hypoperfusion	Angioplasty stent placed	Dong et al. [9]
8	68/M	HTN, HI, and DM	Vertigo and severe ataxia, dysarthria, somnolence, hearing loss, and nystagmus	BMCP	Severe stenosis of the V5 segment of the RVA and moderate to severe stenosis of the LVA	LAA; hypoperfusion	Oral dual antiplatelet	Dong et al. [9]
9	69/F	Unknown	Dysarthria, ataxia, nystagmus, and hearing loss	BMCP and bilateral flocculus	Severe stenosis of BA proximal to the origin of AICA arteries	LAA; hypoperfusion	Angioplasty stent placed	Kattan et al. [10]
10	47/M	HTN, DM, and CAD	Vertigo, dysarthria, nystagmus, and ataxia	BMCP	Severe stenosis of the proximal BA at the origin and multiple stenosis of LA and VA	LAA; hypoperfusion	Oral dual antiplatelet	Present 1

**Table 1** (continued)

No.	Age, years/ sex	Risk factors	Clinical symptoms or signs	Lesion location (DWI)	Vascular abnormality	Etiology; mechanism	Treatment (acute period)	Reference
11	74/F	HTN and DM	Vertigo, ataxia, dysarthria, nystagmus, left peripheral facial palsy, and Horner sign of left side	BMCP, bilateral inferior lateral pontine, left dorsal lateral medulla oblongata, and cerebellar hemisphere	Severe stenosis of LVA, RVA, and BA	LAA; hypoperfusion	Oral dual antiplatelet	Present 2
12	64/F	HTN, DM, CAD, and hyperuricemia	Vertigo, ataxia, and nystagmus	BMCP	Severe stenosis of LVA and RVA (intracranial segment)	LAA; hypoperfusion	Oral dual antiplatelet	Present 3
13	68/M	HTN, DR, and smoking	Vertigo, ataxia, dysarthria, nystagmus, and numbness of the left side of the face	BMCP, pontine, and cerebellar hemisphere	Hypoplastic of RVA and severe stenosis of the intracranial LVA	LAA; hypoperfusion	Angioplasty stent placed	Present 4

BMCP, bilateral middle cerebellar peduncle infarction; DWI, diffusion-weighted imaging; HTN, hypertension; HL, hyperlipidemia; DM, diabetes mellitus; DR, drinkers; BMCP, bilateral middle cerebellar peduncle; LVA, left vertebral artery; RVA, right vertebral artery; BA, basilar artery; AICA, anterior inferior cerebellar artery; GCA, giant cell arteritis. <sup>1</sup>Thrombus or plaques in situ involve the ostia of the AICA. <sup>2</sup>Acetylsalicylic acid and clopidogrel.

maining 7 patients had lesions in the pons, flocculus, cerebellar hemisphere, and medulla oblongata, in addition to the BMCP lesions. Based on the results of MRA, CTA, and DSA, we found stenosis or occlusion always involving 2 or more vessels, including bilateral VA or BA. We found hypoperfusion as the primary stroke mechanism of patients with BMCP. Regarding etiology, 11 were classified as large artery atherosclerosis, one as traumatic VA dissection, and one as giant cell arteritis. Four patients underwent VA angioplasty and others were treated with antiplatelet or anticoagulation.

## Discussion

### *The Anatomy of the MCPs*

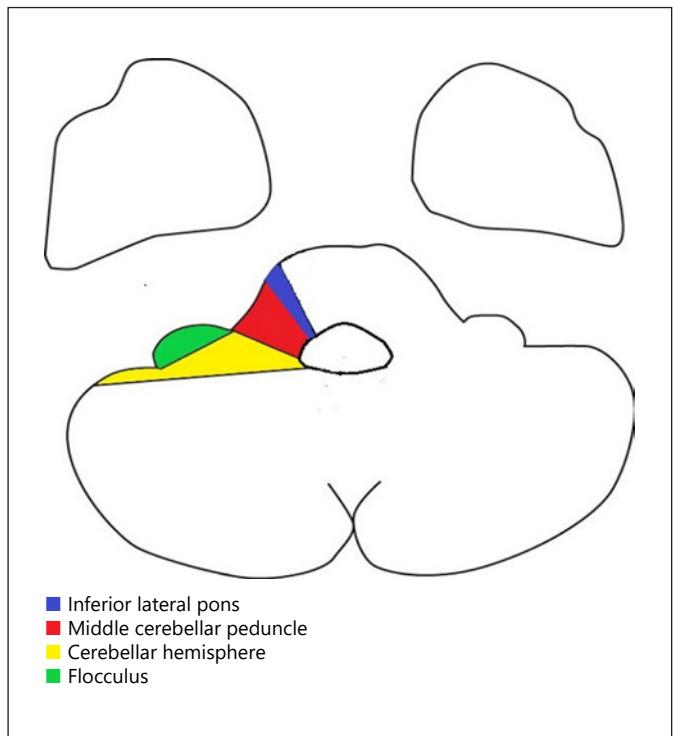
The MCPs are paired structures connecting the cerebellum to the pons. The primary component of the MCP is the transversely coursing corticopontocerebellar fiber, which originates in the contralateral pontine nuclei. As a result, the paired MCPs are associated with the coordination and planning of motor tasks by transmitting interactive information between the cerebellar and prefrontal cortex.

### *The Supply Region of the AICA*

The MCPs are regarded as a watershed area that is largely supplied by the AICA and partially by the SCA. The AICA is well known as 1 of 3 vessels supplying the cerebellum and usually arises from the lower third of the basilar artery [1]. Although the AICA has a variable origin, course, and the supply territory [1], it most commonly supplies the lateral pons, MCP, anterior inferior cerebellum, and inner ear. As the inner ear is not well visualized on routine MRI, prior studies have shown that the radiological hallmark of AICA supply territory can be divided into 4 identifiable regions: MCP, inferior lateral pons, anterior cerebellar hemisphere, and flocculus (Fig. 4) [1]. The AICA also supplies the superior part of the lateral region of the medulla in some rare instances [1]. In our study, dorsal lateral medulla oblongata involvement was found in 1 patient (No. 13), which may be explained by the AICA's variable supply territory.

### *Clinical Manifestation and Relevant Topical Diagnosis*

Unilateral AICA infarct is rare and is associated with following neurological signs and symptoms such as vertigo, facial weakness, hearing disorder, facial sensory loss, gait ataxia, limb ataxia, and Horner's syndrome [2, 3, 12]. A previous study demonstrated that 98% of pa-



**Fig. 4.** Radiological hallmark of AICA supply territory can be divided into 4 identifiable regions, including MCP, inferior lateral pons, anterior cerebellar hemisphere, and flocculus. AICA, anterior inferior cerebellar artery; MCP, middle cerebellar peduncle.

tients with AICA infarction had acute prolonged vertigo and vestibular dysfunction of peripheral, central, or combined origin [13]. Similarly, we found that vertigo accompanied by nystagmus was also the most common manifestations of BMCPI. Unfortunately, most of the cases included in this study lacked objective vestibular function testing. When it comes to the topical diagnosis, vertigo and vestibular dysfunction can be attributed to the areas involving vestibular-associated anatomical structures, such as the vestibular nucleus in the lateral pons, the MCPs, flocculus, or cerebellar hemisphere. Slurred speech/dysarthria, gait ataxia, and limb ataxia may also be related to the bilateral corticopontocerebellar fibers in the MCPs. The occurrence of brainstem signs such as facial weakness/peripheral facial paralysis and facial/limb sensory disorders indicates a lateral pontine lesion in the territory supplied by the AICA. The inferior lateral pons consists of important structures, including the facial nucleus, spinothalamic tract, spinal trigeminal tract, and its nucleus, vestibular nuclei, the trapezoid body, and other structures. Interestingly, a Horner sign

from dorsal medulla oblongata appeared in the BMCPI patient (No. 13). As mentioned above, the involvement of dorsolateral medulla oblongata is likely to be attributed to variations in the blood supply region of the AICA. In addition, Horner's sign can also be explained by the involvement of the pupillodilatator fibres from the hypothalamus in the lateral pars of the pons [1]. Therefore, Horner sign is an extremely rare concomitant brainstem sign of BMCPI. Meanwhile, we found hearing deficits were reported in 6 BMCPI patients. Hearing disorders in the presence of vertigo and vestibular dysfunction have been recognized as an important sign for the diagnosis of AICA territory infarction [13, 14]. Additionally, we need to note that suddenly isolated hearing disorder may be a prodrome of AICA infarction [13–15]. Clinicians should be cognizant that audiovestibular loss may serve as an early window to identify posterior circulation ischemic stroke, even if other brainstem signs are absent and MRI does not demonstrate acute infarction. In this case, a timely evaluation of the vertebrobasilar artery is essential.

#### *The Stroke Etiology and Mechanism of BMCPI*

A study of AICA infarcts by Amarenco et al. [12], showed that isolated unilateral AICA infarcts are indicative of small artery atherosclerotic disease and more widespread infarctions are due to BA occlusive disease. In our study of BMCPI, we found stenosis or occlusion always involved 2 or more large vessels, including bilateral VA or proximal BA. As shown in Table 1, bilateral VA disease was present in all patients and can occur in both the intracranial and extracranial segments or alterations, such as one in a unilateral intracranial segment and another in an extracranial segment (as in case No. 2). Interestingly, we noticed that 1 patient (No. 5) showed bilateral VA occlusions at its origin, due to giant cell arteritis. Except for bilateral VA disease, occlusion or stenosis of the BA at its origin or close to the origin of the AICA is also related to the occurrence of BMCPI (as in case No. 1). We suppose that occlusion of both VA or proximal BA may lead to hypoperfusion in the watershed area between the AICA and the superior cerebellar arteries, resulting in infarction in the BMCPs. Similar to the bilateral cerebral peduncular infarction [16], a hypoperfusion mechanism was also found in patients with BMCPI. Interestingly, Au et al. [17] reported a typical BMCPI patient who was diagnosed as cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy.

## Clinical Differential Diagnosis and Experience

Varying etiologies can be found with the involvement of BMCPs, such as Fragile X-associated tremor ataxia syndrome (FXTAS), multiple system atrophy, Wilson's disease, adrenoleukodystrophy, Wallerian degeneration of the BMCPs secondary to pontine infarction, extrapontine myelinolysis, hypoglycemic coma, and other causes [18, 19]. However, in the context of restricted diffusion visualized on DWI, Wallerian degeneration, extra pontine myelinolysis, and hypoglycemic coma should be the major radiological differential diagnoses of BMCPI [18, 19]. If patients with vertigo and trunk and limb ataxia are suspected to be having an acute ischemic stroke, caudal paramedian midbrain infarction should also be considered when MRI examination is unavailable [20]. Hearing deficits and facial weakness may help distinguish BMCPI and caudal paramedian midbrain infarction. Early suspicion of BMCPI can help clinicians evaluate the risk of disease progression and deterioration because BMCPI indicates a high likelihood of significant stenosis and occlusion changes in bilateral VA or proximal BA. Prompt vascular assessment and intensive therapy and care may be critical for these patients.

## Conclusion

BMCPI is a rare cerebrovascular disease characterized by vertigo, ataxia, and dysarthria and is associated with hypoperfusion. This is often secondary to occlusive disease of bilateral vertebral arteries or proximal basilar arteries. Hearing deficits and brainstem signs may also be present.

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## Statement of Ethics

This study was approved by the Institutional Review Board of the affiliated hospital of the Fifth Affiliated Hospital of Zhengzhou University. All procedures performed in studies were in accordance with the ethical standards of the Institutional Review Board of the affiliated hospital of the Fifth Affiliated Hospital of Zhengzhou University and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The written informed consent was obtained from the patient from our center for the publication of this study.

## Conflict of Interest Statement

We declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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## Author Contributions

Chenguang Zhou analyzed the data and drafted the manuscript for intellectual content. Chenguang Zhou and Haixia Fan designed and conceptualized the study, analyzed the data, and drafted the manuscript for intellectual content. Hao Chen, Haoyue Wang, Zuopeng Li, Ning Xu, Ruonan Sun, Yinghui Zhu, and Yuanhong He played a major role in the acquisition of data from the review.

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