OPEN

Artery of Percheron Occlusion in China
A Case Report and Chinese Literature Review

Lina Cai, MD, Qi Wang, MD,* Bin Cui, MD,† and Peifu Wang, PhD**

Introduction: The artery of Percheron (AOP) is a rare anatomical variant in which bilateral paramedian thalami are supplied by a single vascular branch arising from the P1 segment of the posterior cerebral artery. We present a case of AOP occlusion presenting as loss of consciousness and summarize the literature in Chinese to find the clinical characteristics.

Case Report: An 83-year-old woman was found unconscious for 1 day at home and was sent to the hospital the next day. Cerebral magnetic resonance imaging on day 1 of the patient showed a recent bilateral paramedian thalamic infarction. Simultaneously, magnetic resonance angiography found evident artery stenosis of the right P1 segment of the posterior cerebral artery, suggesting that the patient was diagnosed with AOP occlusion. Since the patient has missed the best time for thrombolytic therapy, anticoagulant therapy was given immediately; as the patient was then found to have pulmonary infections, antibiotic therapy was also initiated. The neurological status of this patient improved very slow. In about 2 weeks, the patient becomes more conscious but still could not speak or move.

Conclusion: Our report suggests that unusual mood disorder and language disorder of aged patients might indicate the AOP occlusion, and cerebral imaging of magnetic resonance imaging (better with magnetic resonance angiography) should be performed to establish the diagnosis of AOP occlusion. The fast and accurate diagnosis of stroke because of AOP occlusion could best benefit the patients.

Key Words: case report, artery of percheron, paramedian thalamic infarction, midbrain infarction, stroke

(*The Neurologist* 2022;27:214–217)

The artery of Percheron (also known as AOP) was firstly described by Percheron in 1973, which indicated an anatomic variant of the paramedian arteries arising from segment P1 of the posterior cerebral artery.[1] The critical feature of AOP is that the bilateral paramedian thalami are supplied by a single vascular branch arising from the P1 segment of the posterior cerebral artery. The exact prevalence of AOP remains unknown.[2] However, autopsy studies revealed the occurrence of AOP could be up to 11.7% in investigated patients.[3,4]

From the Departments of *Neurology; and †Medical Imaging, Aerospace Center Hospital, Beijing, China.

The authors declare no conflict of interest.

Correspondence to: Peifu Wang, PhD, Aerospace Center Hospital, No. 15 Yuquan Road, Beijing 100049, China. E-mail: pfuwang@163.com.

Supplemental Digital Content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's website, www.theneurologist.org.

ISSN: 2331-2637/22/2704-0214
DOI: 10.1097/NRL.0000000000000381

The bilateral paramedian thalamic infarction because of the occlusion of the AOP is a rare type of ischemic stroke; studies found that AOP territory related infarction may approximately account for 0.29% of all ischemic strokes,[5] 0.51% of first clinical strokes,[6] and 2.33% of thalamic infarction.[7] Because of the rarity and various patterns of AOP occlusion,[8] as well as the computed tomography (CT) could be identified as normal at first, the early diagnosis could be challenging for physicians.[2] Therefore, magnetic resonance imaging (MRI) is currently the most informative tool for early diagnosis of AOP occlusion, as well as case reports and summaries could help inform physicians of this specific type of stroke.

In this study, we reported 1 case from our hospital and summarized the clinical features and management of life-threatening AOP occlusion based on a review of Chinese literature to provide helpful information for clinical physicians.

CASE REPORT

We received this 83-year-old woman on March 14, 2020. She was found unconscious for 1 day at home and was sent to our hospital the next day. During the inquiry about the patient's status before the stroke, the patients' family mentioned the patient had hypertension and diabetes and was in poor physical health for years, and the patient showed some mood disorder and language disorder before the stroke. The patient used to be optimistic but showed to be a bit apathetic before the stroke; meanwhile, the family had found that the patient sometimes could not speak clearly (more severe than usual). However, these signs did not make aware the family of the stroke until the patient lost consciousness. When the patient was sent to our hospital, she could only react to calling by open her eyes but could not answer or move; her limbs could lift a little to nociceptive stimuli and showed a positive Babinski's sign; although her eyes showed no obvious mydriasis or myosis, they could not perform the horizontal or vertical move. Other clinical examinations indicated the patient had atrial fibrillation, atheroma plaques, and pleural effusion; no other organ dysfunctions were found. Cerebral MRI on day 1 of the patient showed a recent bilateral paramedian thalamic infarction (Figs. 1A–F), simultaneous magnetic resonance angiography (MRA) found evident artery stenosis of the right P1 segment of the posterior cerebral artery (Figs. 1G–I), suggesting that the patient had AOP occlusion. The stroke was ascribed to embolism from atheroma plaques of the vertebrobasilar system arteries seen on the transcranial Doppler. Considering that we could not exclude the possibility of a cardioembolism origin (because of the atrial fibrillation), and the patient has already missed the best time for thrombolytic therapy, anticoagulant therapy was initiated at the emergency department to prevent possible stroke in the future. The neurological status of this patient improved very slow, in about 2 weeks (the patient was found pulmonary infections within 2 days, additional antibiotic therapy was initiated at the emergency department, so the patient did not transfer to the neurology department, and we attended all the consultations for the patient) the patient becomes more conscious but still could not speak or move.

The patient was discharged home because of the requirement of her family; no better neurological status was improved then. At follow-up 1 month later, we were told the patient passed away, and we informed the family of their consent to include the patient in this manuscript (patient information has been anonymized).

Literature Review

For the literature review, we searched CNKI (the largest literature database for the Chinese language) using the Chinese indexing terms for

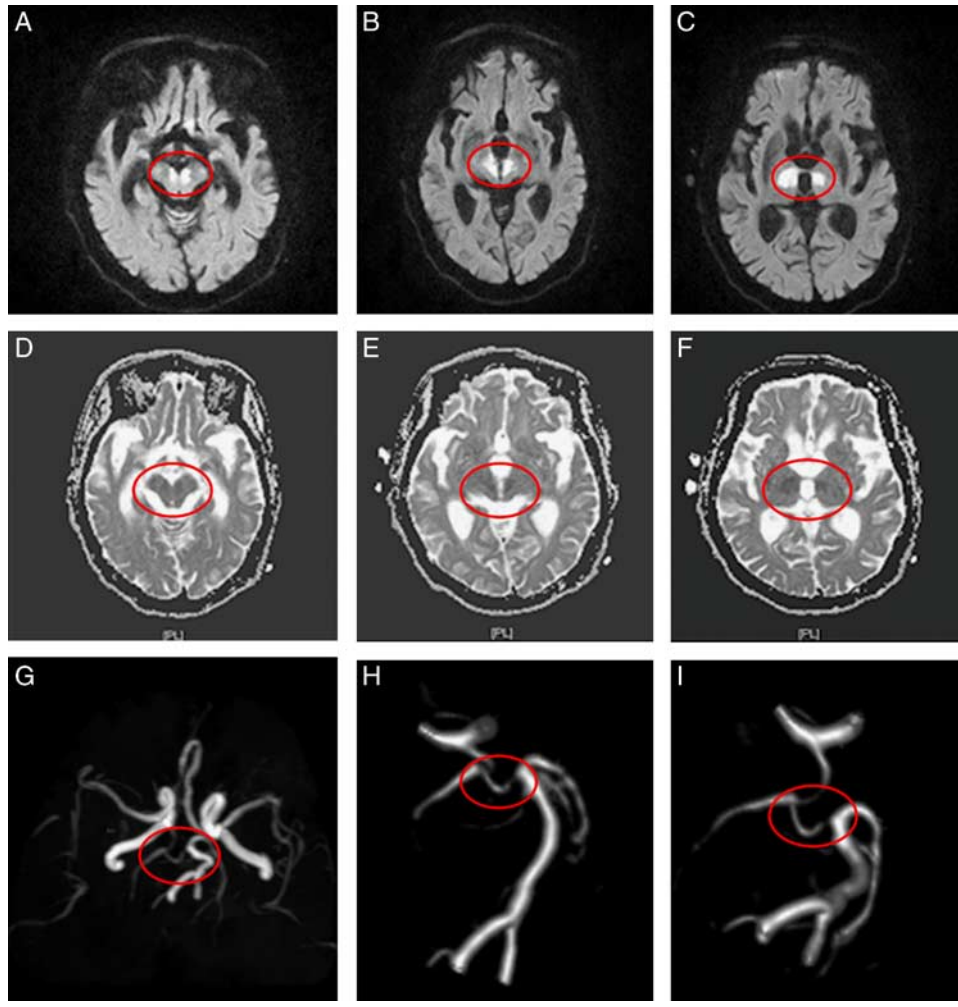

FIGURE 1. Cerebral images of the patient [magnetic resonance imaging (MRI and magnetic resonance angiography (MRA)]. (A–C) MRI, diffusion-weighted imaging sequence showing high-intensity foci in the paramedian thalamic areas (the red circles), the midbrain "V" mark could be observed in panel (B). (D–F) MRI, apparent diffusion coefficient imaging sequence showing low intensity foci in the same areas (the red circles). (G–I) MRA, different angles of the view of the right P1 segment of the posterior cerebral artery (the red circles).

"thalamic," "paramedian," "Percheron," and "stroke." We excluded the kinds of literature that (1) the patient has thalamic or paramedian stroke history, (2) the patient has other pathogeny induced thalamic or paramedian lesion, (3) no detailed clinical data, (4) no cerebral imaging that proves to be AOP occlusion. The quantitative parameters were described as median and percentage; descriptive analyses were performed by using the R software package.[9]

There are 64 patients (1 of our patients and 63 patients from the literature review, Supplemental Digital Content 1, http://links.lww.com/NRL/A61) included in this study. The characteristics of patients with AOP occlusion are shown in Table 1. There were 40 men and 24 women, with a median age of 61.5 years. The co-morbidities of the patients were as follows: atrial fibrillation in 18.8%, coronary heart disease in 12.5%, diabetes in 14.1%, dyslipidemia in 9.4%, hypertension in 65.6%, myocardial infarction in 3.1%. The clinical signs were dominated by neurological signs such as coma (39.1%), confusion (31.3%), and drowsiness (43.8%); neuropsychological signs such as language disorder (18.8%), memory deficit (6.3%), arm/leg plegia (37.5%), and Babinski's sign (46.9%) were common; importantly, ophthalmological signs could be found in half of the patients, including blurred vision (12.5%), mydriasis/myosis (35.9%), and ocular motility disorder (53.1%). All patients underwent cerebral imaging, including CT (34.4%), DSA (15.6%), MRI (96.9%), and MRA (71.9%); some patients could conduct more than 1 examination. Origin of stroke was predominantly attributed to large artery atherosclerosis in 40% and cardioembolism in 28.1%; only 10.9% of origin of stroke remained unknown. Of all the patients after therapy, only 26.6% patients have no sequelae; the most common sequelae were ocular motility disorder (32.8%) and memory deficit (32.8%), the second common sequelae were language disorder (23.4%) and mood disorder (20.3%), the less common sequelae were arm/leg plegia (6.3%).

DISCUSSION

Our analysis of 64 patients with AOP occlusion in China provides a detailed picture of this condition and the outcome. Although patients with AOP occlusion could resolve rapidly from the coma, other abnormalities such as ocular motility disorder, memory deficit, language disorder, mood disorder, and motor impairments might persist in the long term. In our review, the most common mechanism of infarction is large artery atherosclerosis and cardioembolism; only 10.9% remains unknown.

The AOP occlusion is a rare type of stroke in clinical patients, and males and females are equally affected. Many patients with AOP occlusion have cardiovascular risk factors or

TABLE 1. Case Summary of the Chinese Literatures

Summary of AOP Infarction Cases in China		
Demographics		
Number of patients	64	
Age, median and range (y)	61.5 (24-86)	
Male/female (%)	62.5/37.5	
Co-morbidities (%)		Some patients could have more than one co-morbidity
Atrial fibrillation	18.8	
Coronary heart disease	12.5	
Diabetes	14.1	
Dyslipidemia	9.4	
Hypertension	65.6	
Myocardial infarction	3.1	
Clinical signs (%)		Some patients could have more than one clinical sign
Coma	39.1	
Confusion	31.3	
Drowsiness/lethargy	43.8	
Language disorder	18.8	
Memory deficit	6.3	
Paresis	37.5	
Babinski's sign	46.9	
Blurred vision	12.5	
Mydriasis/myosis	35.9	
Ocular motility disorder	53.1	
Cerebral imaging (%)		The presented numbers indicate the ratio of patients conducted this examination and the ratio of positive cases in the examination, respectively
CT	34.4/22.7	
DSA	15.6/40.0	
MRI	96.9/100	
DWI midbrain "V" sign	96.9/20.9	
MRA	71.9/65.2	
Mechanism of stroke (%)		Mechanism of stroke was as described by the authors of the case reports
Cardioembolism	28.1	
Large artery atherosclerosis	40.0	
Small vessel occlusion	14.1	
Other determined etiology	4.7	
Undetermined etiology	10.9	
Prognosis (%)		The prognosis was as described by the authors of the case reports
Arm/leg plegia	6.3	
Language disorder	23.4	
Memory deficit	32.8	
Mood disorder	20.3	
Ocular motility disorder	32.8	
Without sequelae	26.6	

AOP indicates artery of Percheron; CT, computed tomography; DSA, digital subtraction angiography; DWI, diffusion-weighted imaging; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.

embolic heart disease for years. The most common clinical sign is conscious impairment, while it could range from drowsiness to deep coma. Ophthalmological signs are another primary abnormality, which could aid the diagnosis of the AOP occlusion.

The diagnosis of AOP occlusion is primarily dependent on cerebral imaging findings. The most common cerebral imaging including CT and MRI; however, because of CT findings may be normal,[10] MRI findings are more dependable for the diagnosis of AOP occlusion. In our review, only 34.4% of patients conducted CT, and only 22.7% of them were diagnosed to be AOP occlusion, while 96.9% of patients conducted MRI and 100% of them were successfully diagnosed to be AOP occlusion (Table 1). Although CT findings may be easier to obtain the first time and MRI may be normal in some cases,[11] our findings also support that MRI is the better way of diagnosis of AOP occlusion.

The midbrain "V" sign along the pial surface of the midbrain in the interpeduncular fossa is considered as a diagnostic sign for AOP occlusion,[8] which is derived from a high-intensity signal on axial fluid-attenuated inversion recovery and diffusion-weighted imaging. This sign has been reported in 67% of patients (37 patients in total) with AOP occlusion[8]; however, in our review, we found the midbrain "V" sign only presented in 20.9% of patients who conducted MRI (Table 1). Together with the previous studies, it is recognized that the midbrain "V" sign on diffusion-weighted imaging could help clinicians to diagnose the AOP occlusion, while patients with no midbrain "V" sign need more careful examinations to make an accurate diagnosis. In this case, 3D MRA is recommended to be coupled with MRI to rule out basilar artery occlusion. As shown in Figures 1G–I, it is clear that the right P1 segment of the posterior cerebral artery is stenosis, and according to the classification of AOP occlusion types,[8] our patient belongs to the "bilateral paramedian thalamic with midbrain" type.

In previous studies, the cause of the AOP occlusion mainly was identified as embolic heart disease,[5,12] while large artery atherosclerosis and small-artery occlusion were exceedingly rare.[8,13] In our review, we found that the primary cause of the AOP occlusion was large artery atherosclerosis (40%, Table 1). This inconsistency in results may be because of the increasing high sugar/fat diet in Chinese people, which could lead to a higher rate of large artery atherosclerosis,[14] and finally, a higher rate of stroke.[15] A recent study has found the up growing trend of obesity in China that could relate to high sugar/fat diet,[16] which could help to explain why large artery atherosclerosis has been the primary cause of AOP occlusion in the literature reviewed (literature covered the recent 10 years).

Thalamus could affect 5 functional aspects, such as (1) nociception, (2) receiving inputs from all major sensory domains, (3) language and motor function, (4) higher cognitive functions, and (5) motivation and mood.[17] As a result of AOP occlusion, the patients usually have sequelae related to these aspects. In our review, only about 1/4 patients have no sequelae; the other 3/4 patients have different kinds of dysfunctions in language, motor, cognitive, and mood, similar to the previous study.[18] Although stroke of the thalamus because of AOP occlusion is usually not fatal,[17] patients with other co-morbidities may need more attention. As in this case, our patient had been in hypertension and bad health condition for years, and she was diagnosed to have atrial fibrillation, atheroma plaques, and pleural effusion; more importantly, the family did not realize that the mood and language disorder could be an early sign of stroke, and ascribed the unconsciousness as a bad health condition (the patient was sent to hospital 1 day later), which all eventually resulted in the miss of the best time for thrombolytic therapy, the stroke might thus aggravate her condition and threaten her life.

Ischemic stroke because of AOP occlusion is a rare type of stroke and could be life-threatening, especially when the treatment is delayed. The typical clinical signs could be similar to other ischemic stroke types; however, clinicians need to be aware of the combination of consciousness impairment, neuropsychological and ophthalmological disorder, respiratory and cardiovascular dysfunctions. Cerebral imaging of MRI (better with MRA) should be performed to establish the diagnosis of AOP occlusion, as CT could be normal initially.

There are limitations to our study. First, our study focused on introducing Chinese literature to a broader audience, which limited the number of cases collected. Second, the kinds of literature included in our study are primarily from the recent decade;

literature from earlier times usually did not have images or detailed clinical data. Thus the result may only reflect the current conditions. Finally, although limitations exist, our study also provided detailed information on the condition of AOP occlusion in China and could be helpful for further global studies.

ACKNOWLEDGMENTS

The authors thank Dr Ge from the University of Chinese Academy of Sciences for his help and comments during the preparation of this manuscript.

REFERENCES

1. Percheron G. The anatomy of the arterial supply of the human thalamus and its use for the interpretation of the thalamic vascular pathology. *Z Neurol*. 1973;205:1–13.
2. Krampla W, Schmidbauer B, Hruby W. Ischaemic stroke of the artery of Percheron (2007: 10b). *Eur Radiol*. 2008;18:192–194.
3. Kocaeli H, Yılmazlar S, Kuytu T, et al. The artery of Percheron revisited: a cadaveric anatomical study. *Acta Neurochir (Wien)*. 2013;155:533–539.
4. Uz A. Variations in the origin of the thalamoperforating arteries. *J Clin Neurosci*. 2007;14:134–137.
5. Kumral E, Evyapan D, Balkır K, et al. Bilateral thalamic infarction: clinical, etiological and MRI correlates. *Acta Neurol Scand*. 2001;103: 35–42.
6. Carrera E, Michel P, Bogousslavsky J. Anteromedian, central, and posterolateral infarcts of the thalamus: three vairant types. *Stroke*. 2004;35:2826–2831.
7. Pezzini A, Del Zotto E, Archetti S, et al. Thalamic infarcts in young adults: relationship between clinical-topographic features and pathogenesis. *Eur Neurol*. 2002;47:30–36.
8. Lazzaro NA, Wright B, Castillo M, et al. Artery of percheron infarction: imaging patterns and clinical spectrum. *AJNR Am J Neuroradiol*. 2010;31:1283–1289.
9. R Core Team. *R: A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing; 2020.
10. Lamboley J, Le Moigne F, Have L, et al. Artery of Percheron occlusion: value of MRI. A review of six cases. *J Radiol*. 2011;92: 1113–1121.
11. Cassourret G, Prunet B, Sbardella F, et al. Ischemic stroke of the artery of Percheron with normal initial MRI: a case report. *Case Rep Med*. 2010;2010:425734.
12. Gerber O, Gudesblatt M. Bilateral paramedian thalamic infarctions: a CT study. *Neuroradiology*. 1986;28:128–131.
13. Zappella N, Merceron S, Nifle C, et al. Artery of Percheron infarction as an unusual cause of coma: three cases and literature review. *Neurocrit Care*. 2014;20:494–501.
14. Kesse-Guyot E, Vergnaud A-C, Fezeu L, et al. Associations between dietary patterns and arterial stiffness, carotid artery intima-media thickness and atherosclerosis. *Eur J Cardiovasc Prev Rehabil*. 2010; 17:718–724.
15. O'Donnell MJ, Xavier D, Liu L, et al. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case-control study. *Lancet*. 2010;376: 112–123.
16. Pan X-F, Wang L, Pan A. Epidemiology and determinants of obesity in China. *Lancet Diabetes Endocrinol*. 2021;9:373–392.
17. Schmahmann JD. Vascular syndromes of the thalamus. *Stroke*. 2003;34:2264–2278.
18. Arauz A, Patiño-Rodríguez HM, Vargas-González JC, et al. Clinical spectrum of artery of Percheron infarct: clinical–radiological correlations. *J Stroke Cerebrovasc Dis*. 2014;23:1083–1088.