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BRIEF COMMUNICATION

A clinical study of artery of Percheron infarction

Yi-Kuan Chiang[a,b], Yu-Hsiang Ling[a,b], Feng-Chi Chang[b,c], Jong-Ling Fuh[a,b,d,*]

[a]Department of Neurology, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan, ROC; [b]School of Medicine, National Yang Ming Chiao Tung University College of Medicine, Taipei, Taiwan, ROC; [c]Department of Radiology, Taipei Veterans General Hospital, Taipei, Taiwan, ROC; [d]Brain Research Center, National Yang Ming Chiao Tung University, Taipei, Taiwan, ROC

Abstract

Artery of Percheron (AOP) infarction, a rare cause of acute altered mental status (AMS), is characterized by bilateral paramedian thalamic infarction. The aim of this study was to review the clinical manifestation, radiological patterns, treatment, and prognosis of patients with AOP infarction. This retrospective case series included patients with AOP infarction from 2009 to 2020 from a medical center in Taiwan. We defined AOP infarction as acute bilateral paramedian thalamic infarction from magnetic resonance imaging, and patients were further categorized by their additional AOP territorial involvements. We determined outcomes with the modified Rankin Scale at discharge. Among the 10 included patients, AMS was the most common presentation (90%). We identified two patients with bilateral vertebral artery (VA), five with unilateral posterior cerebral artery (PCA), and one with bilateral PCA occlusion. Atherosclerosis was the most common presumed etiology (60%). Two and eight patients had favorable and unfavorable prognoses, respectively. PCA occlusion, rather than VA and BA occlusion, was common in angiography. Residual symptoms often resulted in significant disability at discharge. Basilar tip syndrome may share indistinguishable thalamic infarct patterns with AOP infarction but could be differentiated by angiography and other infarcted territories.

Keywords: Angiography; Cerebral infarction; Magnetic resonance imaging; Prognosis; Thalamus

1. INTRODUCTION

Infarction of the artery of Percheron (AOP), a rare anatomical variant of the thalamoperforating artery, accounts for approximately 4–35% of thalamic stroke cases and 0.1–0.6% of ischemic stroke events.[1,2] In most cases, the paramedian part of thalamus is supplied by the thalamoperforating arteries of the thalamus, which usually originate from the ipsilateral posterior cerebral artery (PCA). However, perforating branches may arise from the AOP, a single thalamoperforating artery from the unilateral P1 segment of the PCA irrigating the bilateral paramedian thalamus, with variable contribution to the anterior thalamus and midbrain.[3] Therefore, AOP occlusion is mostly characterized by bilateral paramedian thalamic infarctions.

The aim of this study is to summarize the findings of clinical manifestation, radiological patterns, management, and prognosis of AOP infarction in clinical settings.

2. METHODS

We retrospectively reviewed patients with acute bilateral thalamic infarction from January 2009 to August 2020 in Taipei Veterans General Hospital, a tertiary medical center in Taiwan. We defined acute infarction as diffusion-weighted imaging sequence hyperintensity on magnetic resonance imaging (MRI) with corresponding decrease in the apparent diffusion coefficient.[3]

We defined the inclusion criteria for AOP infarction as the presence of acute bilateral paramedian thalamic infarction with or without involvement of anterior thalamus and rostral midbrain, which is consistent with past studies.[3–5] The thalamus was divided by both its long and short axes into four quarters, considering it imperfectly oval. We then assigned the medial-anterior quarter as the paramedian part and the lateral-anterior quarter as the anterior part (Fig. 1). We excluded patients with (1) no acute infarction in the bilateral paramedian thalamus, (2) thalamic infarction extending beyond the bilateral paramedian and anterior portion, (3) preexisting or active noncerebrovascular brain parenchymal diseases that might interfere with radiological studies of acute stroke, and (4) an overt diagnosis other than AOP infarction.

We recorded the patients' demographic characteristics and clinical presentations. We reviewed the patients' diagnostic findings, including ischemic territories in brain imaging and stenotic patterns on magnetic resonance angiography (MRA). We defined arterial stenosis as >50% stenosis of the large arteries on MRA. Following the classification system from past research,[3] we categorized the brain MRIs of all patients into four distinct infarct patterns: (1) bilateral paramedian thalamus with rostral midbrain, (2) bilateral paramedian thalamus without midbrain, (3) bilateral paramedian and anterior thalamus with rostral midbrain, and (4) bilateral paramedian and anterior thalamus without midbrain. The presumed stroke etiologies were in accordance with the trial of ORG 10172 in acute stroke treatment (TOAST) classification system, which denotes five subtypes of ischemic stroke: (1) atherosclerosis, (2) small vessel occlusion, (3) cardioembolism, (4) stroke of other determined etiologies, and (5) stroke of other undetermined etiologies.[6]

We collected data regarding treatment and prognosis, which included stroke onset to diagnosis time, summary of treatment

*Address correspondence. Dr. Jong-Ling Fuh, Department of Neurology, Neurological Institute, Taipei Veterans General Hospital, 201, Section 2, Shi-Pai Road, Taipei 112, Taiwan, ROC. E-mail address: jlfuh@vghtpe.gov.tw (J.-L. Fuh).

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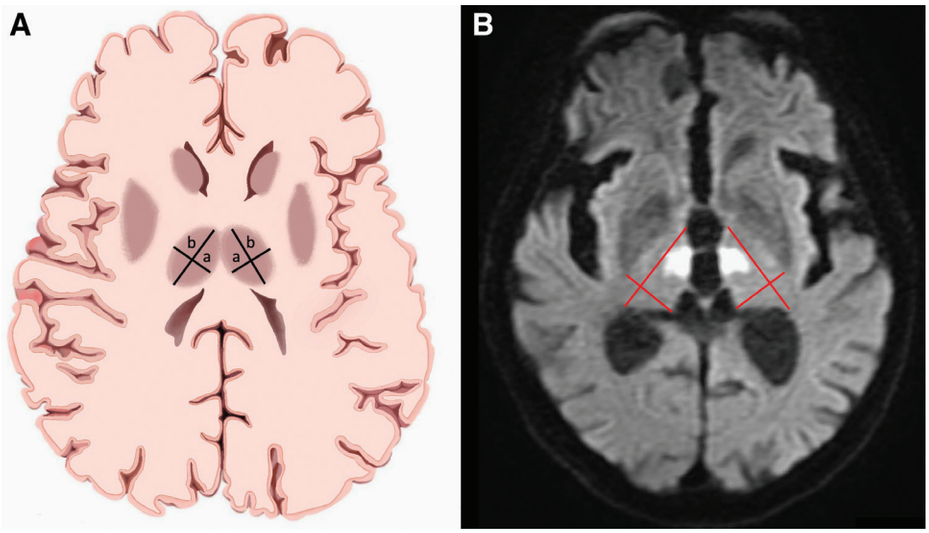

Fig. 1 Illustration of our method for determining the paramedian (zone a) and anterior (zone b) parts of the thalamus (A) and its radiological application in case 2 (B). In (B), the diffusion-weighted imaging sequence from magnetic resonance imaging is used, and red lines that indicate two axes were inserted by computer software afterward.

approach, and graded general prognosis. The stroke onset to diagnosis time was defined as the time interval between the first neurological symptom onset/last time normal and to acquisition of the first brain MRI adequate for AOP infarction diagnosis. We adopted binominal prognosis grading, which included both favorable and unfavorable prognoses, based on a review of medical charts and stroke databases. We defined a favorable prognosis as a modified Rankin Scale (mRS) ≤2 and an unfavorable prognosis as an mRS ≥3 at discharge.[7,8]

3. RESULTS

During the study period, we identified 20 patients with bilateral thalamic infarction. We excluded 10 patients because one had no available MRI, four no bilateral paramedian thalamic infarction, two thalamic infarctions beyond the paramedian and anterior part of the thalamus, two presumed diagnosis of basilar tip syndrome (BTS), and one preexisting medulloblastoma with previous stenting and embolization of multiple posterior circulation aneurysms. Finally, 10 patients (six males and four females, onset age 42–100 years, median 82.5 years) were enrolled in the study (Table 1). Hypertension (50%) was the most common comorbidity. For clinical presentation, altered mental status (AMS) was most common in nine patients.

After reviewing MRA, bilateral occlusion in the vertebral artery (VA) was seen in two patients, and unilateral VA dissection was seen in one. For PCA, five patients had unilateral occlusion, and one had bilateral occlusion. No patient had basilar artery (BA) occlusion, but BA intramural hematoma and flap were seen in one patient each. Among the four AOP infarct patterns, bilateral paramedian thalamus with rostral midbrain, bilateral paramedian thalamus without midbrain, and bilateral paramedian and anterior thalamus without midbrain were the three most common patterns.

The duration from stroke onset to diagnosis ranged from 4.9 to 552.0 hours (median 39.70 hours), after excluding one patient (case 8) without available records.

All patients received antiplatelet therapy. No patient died due to direct results or sequelae of cerebral infarction. The most common presumed etiology was atherosclerosis (60%). "Unfavorable prognosis" and "favorable prognosis" were obtained for eight and two patients, respectively.

Table 1

Summary of demographics, clinical presentations and imaging findings in all study subjects

Variables	n or %
Age, y, median (25–75th percentile)	82.5 (52.8–88.3)
Sex (male/female)	6/4
Common comorbidities	
Hypertension	50% (5/10)
Diabetes mellitus	40% (4/10)
Clinical presentation	
AMS	90% (9/10)
Psychosis	40% (4/10)
Loss of PLR	30% (3/10)
VGP	20% (2/10)
MRI infarct patterns	
P + M	30% (3/10)
P	30% (3/10)
P + A + M	10% (1/10)
P + A	30% (3/10)
MRA arterial stenosis	
VA (N/U/B)	8/0/2
PCA (N/U/B)	4/5/1
BA (N/O)	10/0
Median time to diagnosis in hours (25–75th percentile)[a]	39.70 (18.85–156.00)
Presumed etiology (1/2/3/4/5)[b]	6/0/0/0/4
Prognosis on discharge (favorable/unfavorable)	2/8

A = anterior thalamus; AMS = altered mental status; B = bilateral arterial occlusion; BA = basilar artery; M = midbrain; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging; N = no occlusion; O = occlusion; P = paramedian thalamus; PCA = posterior cerebral artery; perc. = percentile; PLR = pupillary light reflex; U = unilateral arterial occlusion; VA = vertebral artery; VGP = vertical gaze palsy.

[a]Data missing for one patient (case 8).

[b]Five presumed etiologies are represented by numbers: 1, atherosclerosis; 2, small vessel occlusion; 3, cardioembolism; 4, other determined etiologies; and 5, other undetermined etiologies.

4. DISCUSSION

Our study found that AMS was the most common clinical presentation (90%) and was present in almost all cases. Although atherosclerosis was the main presumed etiology (60%) for AOP stroke, atherosclerotic changes in VA (20%)

and BA (0%) were markedly rarer than in the P1 segment of the PCA (60%). Therefore, infarcts were mostly restricted to the thalamus.

The age distribution in our study was slightly higher than that the few existing case series of AOP infarction.[3,5,9] Seven of the nine patients (78%) with complete records had a confirmed diagnosis of AOP infarction more than 24 hours after stroke onset. The low mortality (0%) was similar to results from the few published case series of AOP infarction. However, 80% of patients had an unfavorable prognosis (mRS ≥3) at discharge after the acute stroke in our study. This indicates that residual symptoms of AOP infarction often persist and affect daily life activity despite medical therapy and rehabilitation.

We retrospectively examined the excluded patients with bilateral thalamic infarction and found that the presumed stroke mechanism could be cardioembolism, cerebral venous thrombosis, BTS, and atherosclerosis of other main trunks. This indicates that various stroke mechanisms can lead to bilateral thalamic infarction, highlighting the importance of recognizing territories irrigated by the AOP. However, we also noticed two patients with bilateral paramedian thalamic infarction who were eventually excluded after a comprehensive review. The final diagnosis was BTS due to compatible findings on MRA and the involvement of other territories typically irrigated by the BA. Past studies have suggested that BTS due to distal BA occlusion often involves the cerebellum, pons, or occipital lobe in addition to the bilateral thalamus.[10,11] Therefore, it is important not to solely rely on the infarct pattern in the thalamus but also in other posterior circulation territories.

This study has limitations. We adopted the "four-quarter method" to objectively define the paramedian and anterior part of the thalamus. This method might not perfectly identify AOP ischemic areas in all patients due to the highly variable vascular supply of the thalamus and different observers' selection of MRI slices. However, it was still supported by general compatibility with the "interpeduncular profundus territory" and "tuberothalamic territory" in a widely accepted research-use computed tomography template proposed by Graff-Radford et al[12] and the fact that the infarct territories of all 37 patients in the largest case series for AOP stroke to date, as recognized in the superimposed graph of every MRI ischemic area, were restricted to these two zones.[3] Finally, cases with unilateral thalamic infarction, which were excluded from our study, may occasionally occur in AOP infarction. This may lead to potential missing cases during patient enrollment.

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