Acute Infarction in the Artery of Percheron Distribution during Cerebral Angiography: A Case Report and Literature Review

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ABSTRACT

Improvements in techniques, contrast agents, and catheter design have significantly decreased angiography-related neurological deficits and complications. This article reports a case involving an angiographic total obliteration arteriovenous malformation (AVM) in a patient with an acute infarction in the artery of Percheron (AOP) distribution following angiography. Furthermore, imaging of an AOP acute infarction in cerebral angiography is presented.

CASE REPORT

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The patient, a 48-year-old woman with a history of a 2-cm left occipital arteriovenous malformation (AVM; Figures 1 and 2), underwent Cyberknife radiosurgery in 2008 and angiographic total obliteration in 2011 (Figure 3). Unfortunately, she experienced a post-obliteration hemorrhage in 2014 and developed a left occipital hematoma (5.02x3.5x3.06 cm³) (Figure 4). Thus, additional cerebral angiography was arranged. Before angiography was performed, the patient was completely conscious (Glasgow Coma Scale (GCS): E4M6V5). Acute conscious disturbance (GCS: E2M5V3) with bradycardia was observed when a catheter was advanced into the left vertebral artery (VA), and we attempted to perform digital subtraction angiography (DSA) in the posterior circulation (Figure 5). DSA did not reveal any AVM nidus, feeder or early venous enhancement but showed contrast accumulation in a small branch of the right posterior cerebral artery (PCA), the artery of Percheron (AOP). The AOP is one of four blood supply variants of the thalamus and midbrain (Figure 6). The enhancement of the AOP did not disappear even as the venous phase nearly finished (Figure 7). In addition to acute neurological deterioration, acute AOP infarction was suspected; thus, the

procedure was immediately terminated. An emergent non-contrast brain computed tomography (CT) scan was arranged to rule out new intracerebral hemorrhage (ICH). The CT scan showed no focal lesions; therefore, the patient was transferred to the intensive care unit for close monitoring.

At arrival, her GCS was E1M1V4-5, and brainstem signs (pupils: 2 mm/2 mm; light reflex: -/-; corneal reflex: left, - and right, mild +; cough reflex: poor) with weakness in all four limbs (muscle power: right >3; left: 2 under pain stimulation; Babinski sign: +/+) were observed. Because of the suspicion of AOP infarction, brain magnetic resonance imaging (MRI) with contrast was performed, revealing a bilateral thalamic infarction, which was consistent with the clinical diagnosis of an AOP infarction (Figure 8). The patient received hydration with supportive care, her brainstem reflex gradually recovered within 24 hours, and her level of consciousness also improved (GCS: E3–4M5–6VT).

She was subsequently discharged and demonstrated independence in simple daily living activities 5 weeks after this event. An elective craniotomy for hematoma evacuation was performed after four months for the increasing mass effect from the slowly growing hematoma and perifocal edema

(Figure 9). The evacuated hematoma and obliterated AVM nidus were sent to pathology for examination. The pathologist reported that the microcirculation could be detected under a microscope even with the confirmed obliteration of the AVM nidus by DSA (Figure 10).

DISCUSSION

Etiology & Demographics:
AOP infarctions and angiography-induced neurological complications

The AOP is the only artery that arises from the unilateral PCA and supplies the bilateral thalamus. Occlusion of the AOP causes 4 to 35% of thalamic stroke cases [1] and 0.1% to 0.6% of all ischemic stroke events [2,3]. AOP infarction may occur at any age; however, it usually occurs after 30 years of age, with a peak incidence between 60 and 70 years of age. There is a slight male predominance with a 3:2 M:F ratio [1,2,3,4,5,6]. Independent risk factors for AOP infarcts include hypertension, diabetes mellitus, smoking, history of small artery disease and cardioembolism events. Percheron has thoroughly described four blood supply variants of the thalamus and midbrain (Figure 6). The most common variant is type I in which the thalamus is supplied by the bilateral PCA through many perforators. Type IIa indicates that the perforators arise from the unilateral PCA. Type IIb, the AOP, is a rare variant of the posterior thalamoperforating artery that arises from the unilateral mesencephalic or the 1st segment of the posterior cerebral artery (P1) and supplies the bilateral thalamic nucleus, including the dorsomedial nucleus, internal medullary lamina, and intralaminar nuclei [5]. Thus, AOP occlusion may cause bilateral thalamic infarction. For the type III variant, the perforating arteries arise from a single bridging artery that connects the bilateral P1 segments [5,6]. Acute AOP infarctions are rarely diagnosed by angiography, and no report has addressed cerebral angiography-induced AOP infarction.

According to Willinsky RA et al., the risk of neurologic complications from cerebral angiography is approximately 1.3% [7]. The transient and reversible complication rates are 0.7 and 0.2%, respectively. The risk factors for neurologic complications include 55 years of age or older, a history of cerebral vascular disease and a fluoroscopy time of 10 minutes or longer (Table 1).

Clinical & Imaging findings:
In AOP infarct cases, the bilateral paramedian anterior thalamus with or without the midbrain will be influenced. Mental status deterioration, motor deficits and ocular movement disorders are the major clinical findings in neurological examination. Aphasia, dysarthria, behavior changes, and cerebellar signs are difficult to check during the procedure but may be observed after patients regain consciousness.

Except for intra-operative monitoring, exercising caution in the procedure should be emphasized because the most common cause of angiography-induced neurological complications is thromboembolism from catheters or guide wires [7]. The thromboembolism may be identified by intracerebral angiography transcranial Doppler, which shows blood clots and microbubbles during the procedure phases [8]. Post-angiography hyperintense lesions identified via diffusion-weighted imaging (DWI) are used to evaluate the risk of thromboembolism-related neurological complications after angiography. The risk of hyperintense lesions on DWI after angiography ranges from 9 to 26%; however, most lesions are silent [9,10]. The neurologic complication rate may increase when dealing with vascular lesions and vasculature variants, particularly a variant that lacks normal, multiple blood supplies. The prolonged arterial phase of the AOP indicates an acute infarction (Figure 8).

In our case, the contrast agent continued to accumulate in the AOP even after the venous phase was completed; thus, the entire AOP was enhanced. Neurological deterioration began simultaneously. With respect to the acute bilateral thalamic stroke, a hyperintense signal in the MRI diffusion-weighted image and a decreased apparent diffusion coefficient (ADC) value were identified (Figure 6).

Differential Diagnoses:
With the exception of AOP occlusion, vascular etiologies that can result in bilateral acute thalamic infarction, including deep cerebral venous thrombosis and basilar occlusion syndrome, should be considered (Table 2).

Increased intracranial pressure, seizure, and mental status deterioration can be observed in patients who suffer from occlusion of the deep cerebral venous system. Internal cerebral veins, the vein of Galen or straight sinus thrombosis can be detected by magnetic resonance venography or CT venography. With extension of the congestive infarct area, a hyperintense T2 signal may be visualized within the bilateral thalami and basal ganglia. Hemorrhagic transformation may occur in these congestive infarction areas.

Occlusion of basilar artery may affect not only the PCA, superior cerebellar arteries (SCA) and pontine perforators but also the thalamoperforating arteries, which arise from the top of basilar artery bifurcations and supply the bilateral thalami. Thromboembolic diseases that cause basilar artery occlusion or basilar artery dissection may also result in bilateral thalami infarcts. Cerebral angiography, magnetic resonance angiography (MRA), or computed tomography angiography (CTA) may demonstrate filling defects in the basilar artery. Hyperintense T2 signal may be observed not only in the bilateral thalami but also in the brainstem, SCA, or PCA territories.

Studies have rarely reported an increased risk of AOP infarction in angiography. However, if this rare variant is incidentally identified on posterior circulation angiography, it is advisable to carefully operate the guidewire and shorten fluoroscopy times. Other potential confounding factors may exist. Additional studies are required to understand the underlying pathophysiology, the hemodynamic changes after AVM obliteration and the effect of radiation to prevent this morbidity in the future.

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Treatment & Prognosis:

Among patients with bilateral thalamic infarcts with midbrain involvement, only 25% will have a favorable outcome [1]. Acute AOP infarction is managed as an acute stroke. Similar to ischemic stroke management, hydration with supportive care is sufficient. If the infarct area involves the midbrain or respiratory distress occurs, ventilator support and ICU management will be needed. DSA-assisted recanalization and thrombolysis may be attempted in select candidates. Several reports have indicated a dramatic improvement after thrombolysis [6]. However, in this case, thrombolysis is contraindicated because of AVM rebleeding. Conservative treatments, including hydration and blood pressure control, were initiated in this patient.

The necessity of angiography in angiographic obliteration AVM

The risk of bleeding after angiographic obliteration is extremely rare. According to a 2005 case study, the rebleeding rate was approximately 0.3%. The cumulative risk over 10 years is 2.2% [11]. The authors have often used cerebral angiography to confirm AVM obliteration. Cerebral angiography is useful for diagnosing intracranial vascular lesions; however, it is not useful for demonstrating slow and diffuse blood flow. In histological examinations, microcirculation could be detected in some obliterated vessels (Figure 10). This histological finding was also noted in a previous study [11]. Therefore, angiography is limited for confirming total AVM obliteration. This microcirculation phenomenon also suggests that radiation causes different severity levels of arterial stenosis, and this effect may be observed in all irradiated areas.

MRI with contrast may play a role in AVM follow-up. Persistent enhancement of the irradiated nidus constituted the major risk factor for post-obliteration bleeding, and these findings reached statistical significance [11]. In this rebleeding case, angiography may not be necessary if no recurrent nidus is evident on CTA or MRA.

TEACHING POINT

Neuro-radiologists should emphasize the need to exercise caution when dealing with a rare vasculature variant, particularly a variant that lacks normal, multiple blood supplies. The prolonged arterial phase due to contrast agent accumulation is an important sign of acute infarction. Furthermore, cerebral angiography may not be necessary in cases that involve post-angiographic obliteration rebleeding if no recurrent nidus is detected by CTA or MRA.

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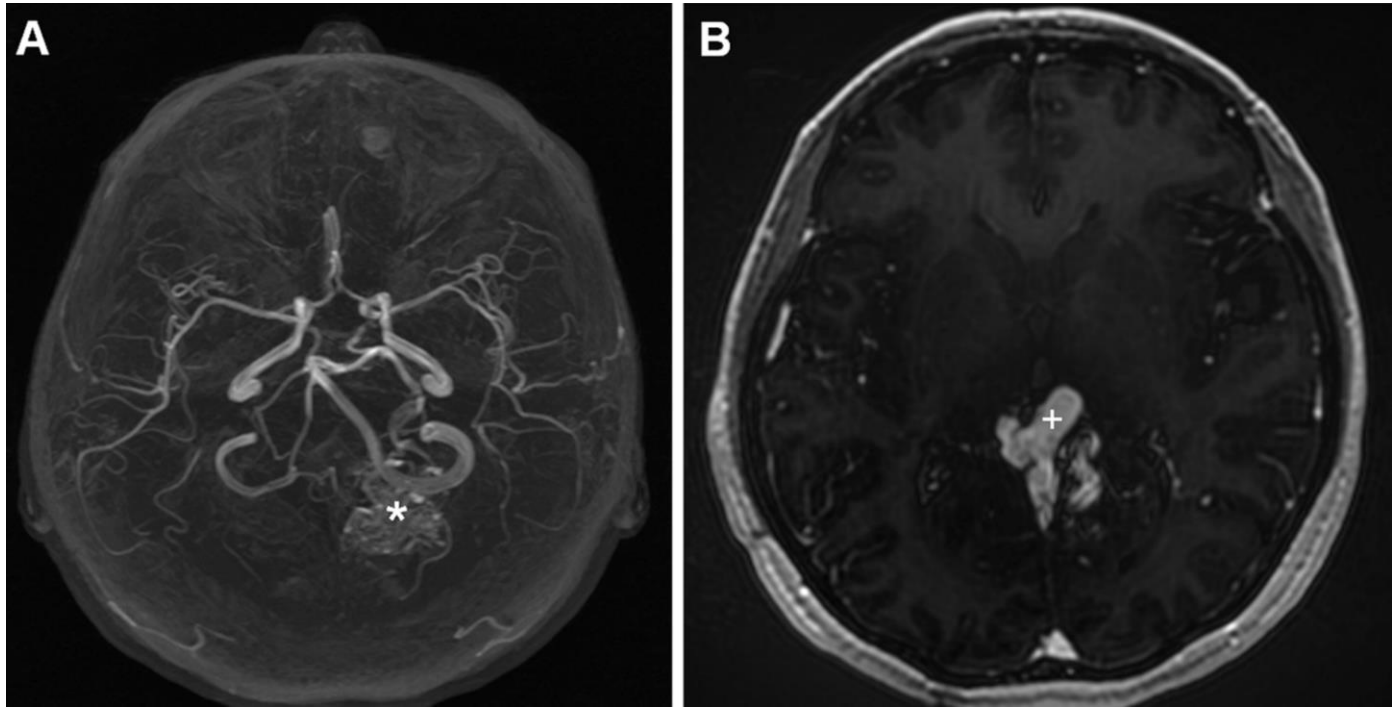www.RadiologyCases.com

FIGURES

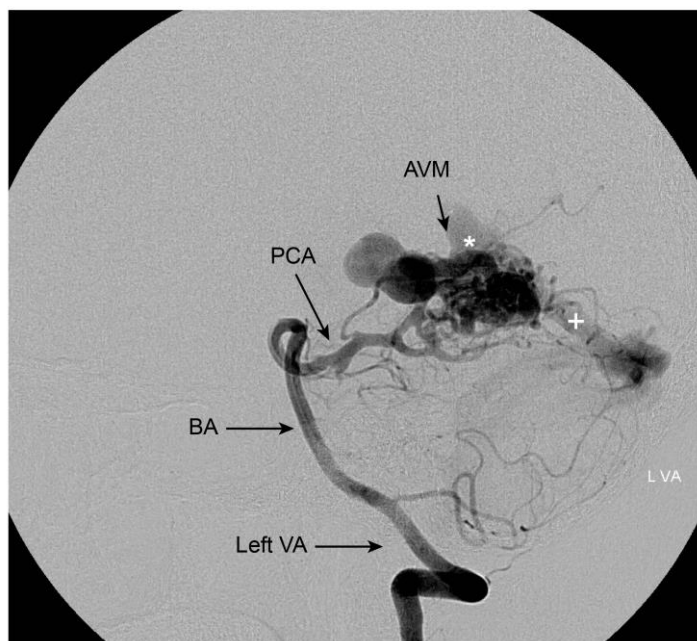**Figure 1:** A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: (A) MRA showed a 2.5-cm left medial occipital lobe AVM* and (B) an engorged drainage vein+ at the pineal region can be observed in T1 sequence with contrast.
*: Left occipital AVM
+: Engorged drainage vein at the pineal region
Technique:
(A)   GE Signa EXCITE 1.5T, axial MRA time-of-flight (TOF) sequence with contrast, TR 30 ms, TE 6.3 ms.
(B)   GE Signa EXCITE 1.5T, axial MRI T1 sequence with contrast, TR 11.59 ms, TE 5.116 ms.

Figure 2 (left): A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: Angiography (lateral view) indicated that the feeding artery of the AVM originated from the enlarged left posterior cerebral artery (PCA), and early opacification of the draining veins was mainly observed through the vein of Galen and into the straight sinus.
*: Vein of Galen; +: Straight sinus
Technique: Digital subtraction angiography of the left VA via a right transfemoral approach using a 5F H1 catheter.
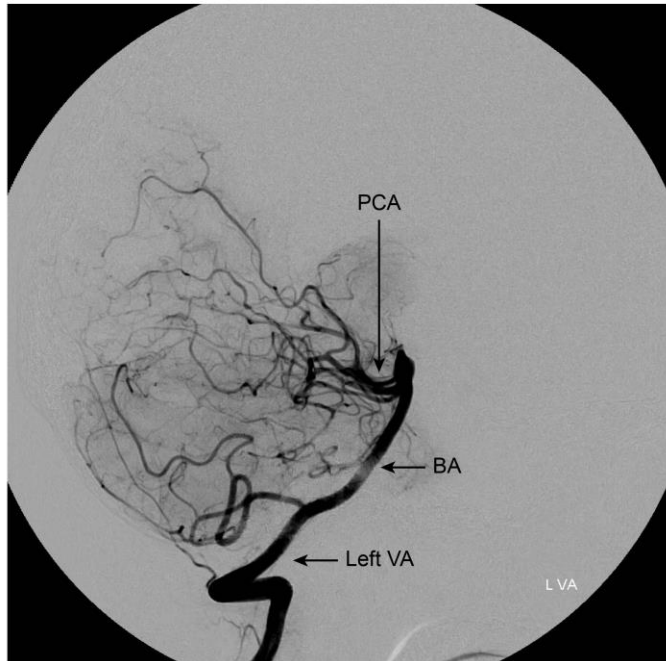
Figure 3: A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution. Angiographic total obliteration of left occipital AVM was demonstrated 3 years later after Cyberknife radiosurgery in 2008.
Findings: Angiography indicated the absence of a definite residual AVM and a patent major dural sinus.
Technique: Digital subtraction angiography of the left VA via a right transfemoral approach using a 5F H1 catheter.

Figure 4: A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: Axial CT imaging indicated approximately 25 ml (4.6x3.3x3 cm3) of heterogeneous parenchymal hemorrhage* in the left occipital area with adjacent perifocal edema.
Technique: CT: GE LightSpeed VCT, mAs 280, kVp 120, 0.7 mm slice thickness, no contrast.
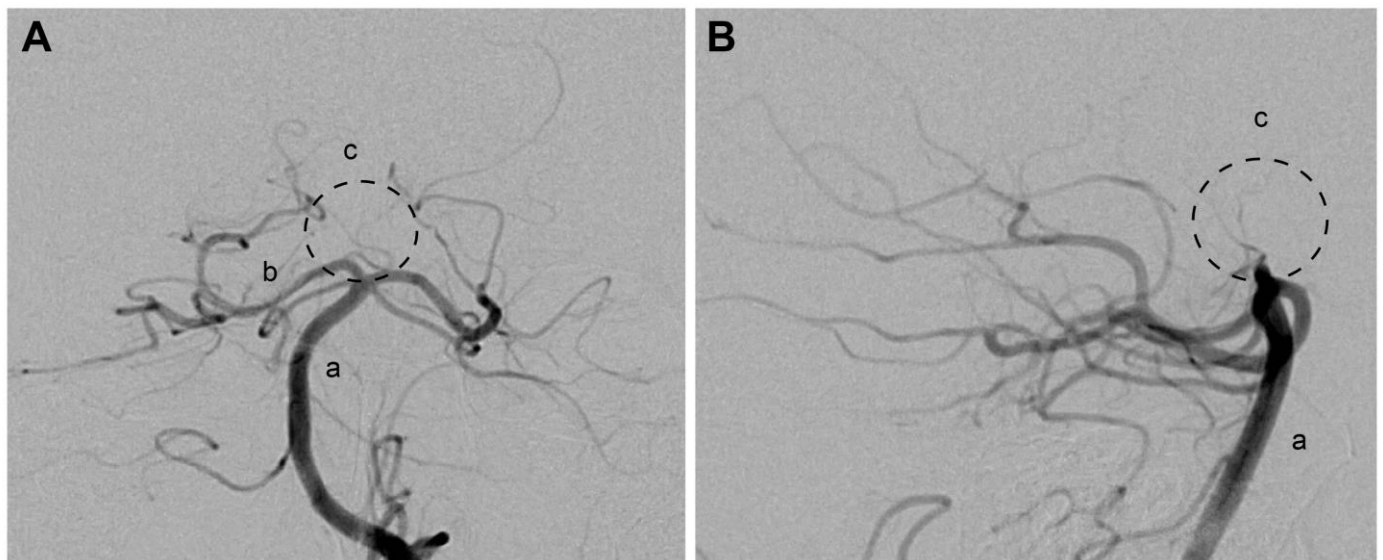*: Left occipital intracerebral hematoma

Figure 5: A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: Cerebral angiography through the left vertebral artery showed no evidence of residual AVM or abnormal filling defects in the arterial phase. However, the AOP can be observed in the arterial phase.
(A)   A-P view.
(B)   Lateral view.
a. Basilar artery, b. Right PCA, c. AOP.
Technique: Digital subtraction angiography of the left VA via a right transfemoral approach using a 5F H1 catheter.
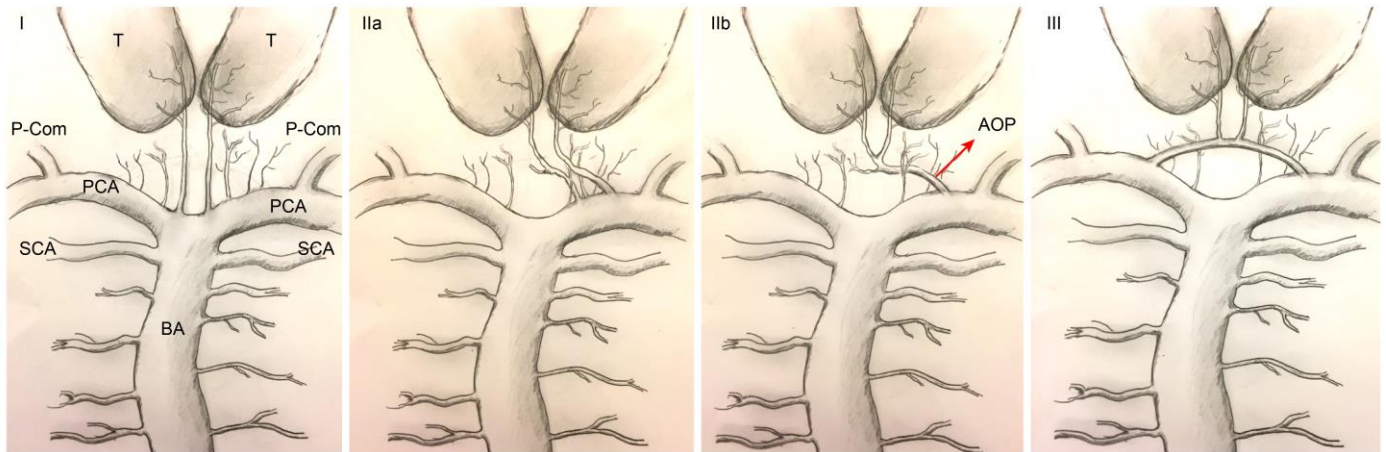
Figure 6: Four blood supply variants of the thalamus and midbrain.
Type I, the thalamus is supplied by the bilateral PCA through many perforators.
Type IIa, the perforators arise from the unilateral PCA.
Type IIb, the artery of Percheron arises from the unilateral mesencephalic or the P1 artery and supplies the bilateral thalamic nucleus.
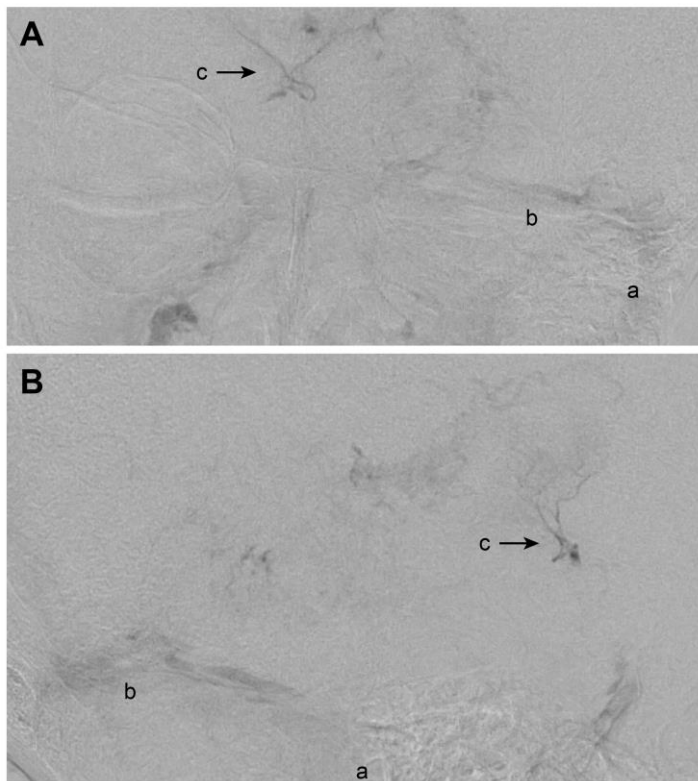Type III, the perforating arteries arise from a single bridging artery that connects bilateral P1 segments.

Figure 7 (left): A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: Cerebral angiography through the left vertebral artery showed the AOP in the venous phase. A prolonged arterial phase of the AOP was observed even though the venous phase was nearly completed. This finding indicated an acute AOP infarction.
(A)   A-P view: The contrast agent accumulated in the artery of Percheron distribution and its two branches to the bilateral thalamus.
(B)   Lateral view: The enhancement of the sigmoid and transverse sinus suggests the venous phase of angiography; however, the artery of Percheron continued to be enhanced.
a. Sigmoid sinus, b. Transverse sinus, c. AOP.
Technique: Digital subtraction angiography of the left VA via the right transfemoral approach using a 5F H1 catheter.
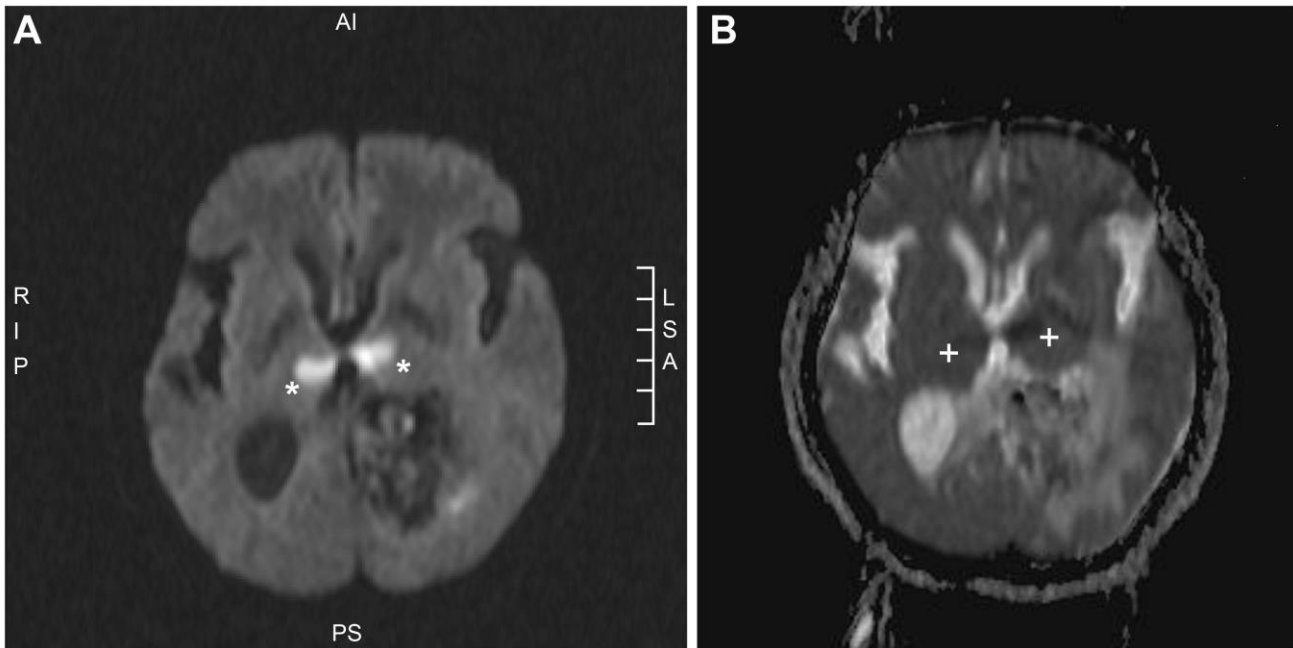
Figure 8: A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: (A) Focal diffusion-weighted imaging (DWI) hyperintensities* and (B) a decreased+ apparent diffusion coefficient (ADC) value at the bilateral thalami, which is consistent with acute infarction.
(A) Diffusion-weighted imaging
*: Bilateral thalamus hyperintensity lesions in DWI
Technique: Siemens sonata 1.494T, axial DWI sequence without contrast, TR 4000 ms, TE 122 ms.
(B) Apparent diffusion coefficient
+: Bilateral thalamus hypointensity lesions in ADC
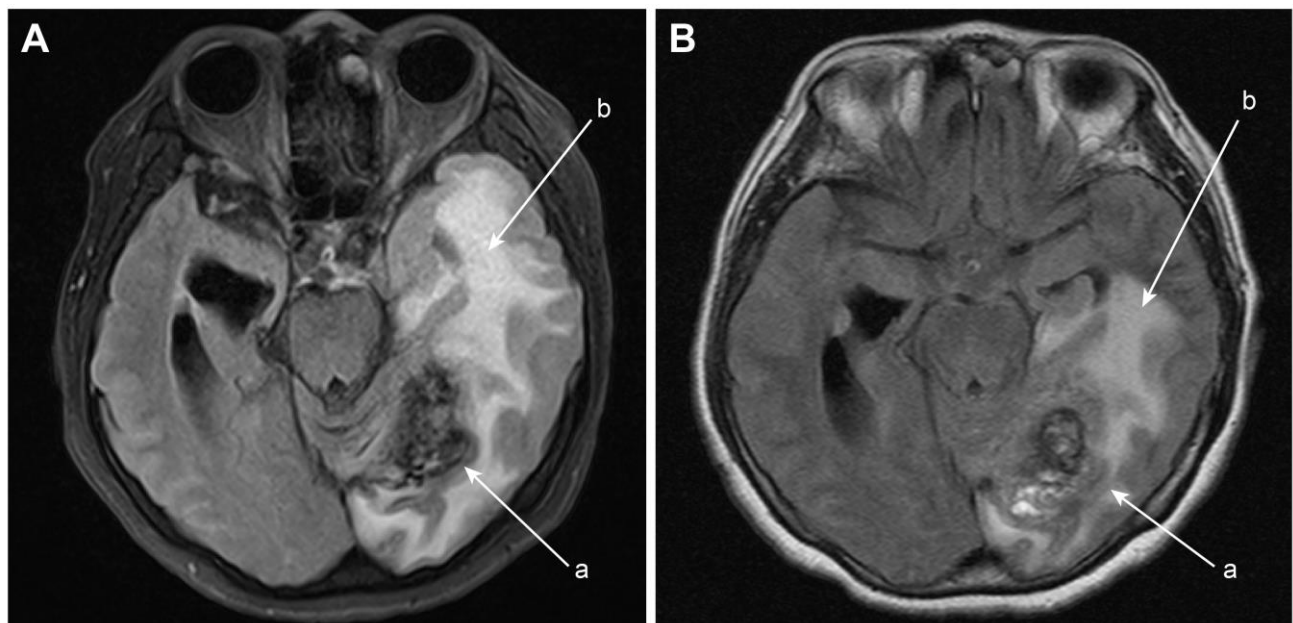Technique: Siemens sonata 1.494T, axial ADC sequence without contrast, TR 4000 ms, TE 122 ms.

Figure 9: A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: The two T2 fluid attenuation inversion recovery (FLAIR) images indicated a heterogeneous intensity hematoma in the occipital region (a) and a hyperintense perifocal edema in the left temporal region (b). The sizes of the hematoma and perifocal edema were increased after 6 months. The mass effect increased and caused a deformity in the brainstem.
A: MRI T2 FLAIR image in Oct. 2014.
Technique: Siemens Trio TIM 3.0T, fluid attenuation inversion recovery (FLAIR) sequence without contrast, TR 1e+004 ms TE 93 ms.
B: MRI T2 FLAIR image in Apr. 2014.
Technique: Siemens sonata 1.494T, axial T2 FLAIR sequence without contrast, TR 9000 ms, TE 99 ms.
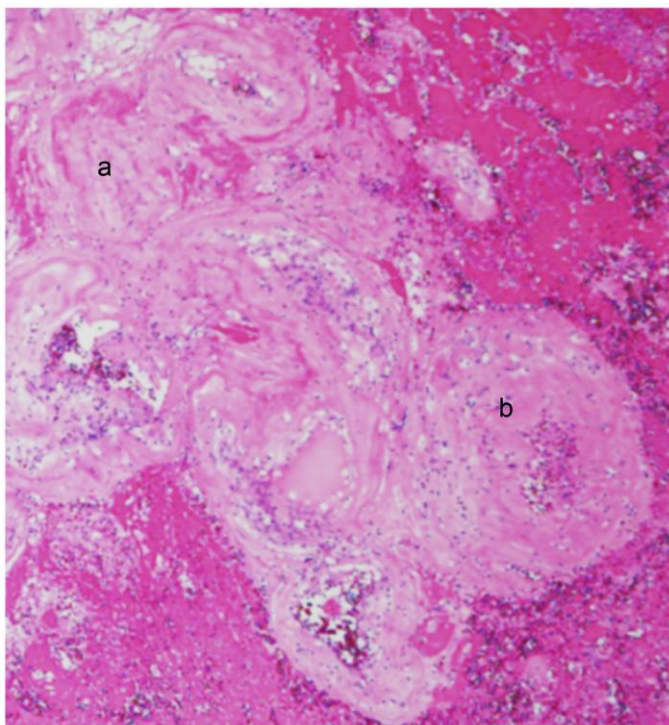
Figure 10 (left): A 48-year-old female diagnosed with an acute infarction in the artery of Percheron distribution.
Findings: The AVM histology specimen from this patient showed different severity levels of stenosis. A completely obliterated artery (a) and red cells in the partially obliterated artery lumen (b) were identified. Microcirculation was difficult to detect using angiography.

Etiology	Angiography-induced thromboemboli caused the occlusion of the artery of Percheron.
Incidence	• Occlusion of the AOP causes 4 to 35% of thalamic stroke cases and 0.1% to 0.6% of all ischemic stroke events. • The risk of neurologic complications from cerebral angiography is approximately 1.3%.
Gender ratio	M:F = 3:2
Age predilection	30s-70s
Risk factor	• Independent risk factors for AOP infarcts: hypertension, diabetes mellitus, smoking, history of small artery disease and cardioembolism events. • The risks associated with neurologic complications from cerebral angiography: age 55 years or older, a history of cerebral vascular disease and fluoroscopy times of 10 minutes or longer.
Treatment	• Studies have suggested that AOP acute infarction should be treated with endovascular thrombolytic therapy in the acute stage; however, in most cases, similar to the management of ischemic stroke, hydration with supportive care is sufficient. • If the infarct area involves the midbrain or respiratory distress occurs, ventilator support and ICU management will be needed.
Prognosis	• The prognosis of an AOP infarct is relatively good. Approximately 67% of patients with bilateral paramedian thalamic infarcts without midbrain involvement will recover well. • In patients with bilateral thalamic infarcts with midbrain involvement, only 25% of patients will have a favorable outcome.
Imaging findings	• A prolonged arterial phase of the AOP was observed even though the venous phase was nearly completed. This finding indicated an acute AOP infarction. • With respect to acute bilateral thalamic stroke, a hyperintense signal in the MRI diffusion-weighted image and a decreased apparent diffusion coefficient (ADC) value can be identified.

Table 1: Summary table of angiography-induced acute infarction in the artery of Percheron distribution.

Diagnosis	MRI, MRA and MRV	CT, CTA and CTV	DSA
Artery of Percheron occlusion	• No AOP signal can be identified in the previously existing AOP.	• No contrast enhancement can be identified in the previously existing AOP. • Bilateral thalamic hypodensity can be identified in subacute or chronic stages.	• No contrast enhancement can be identified in the previously existing AOP. • Contrast continues to accumulate in the AOP even after the arterial phase is completed.
Cerebral venous thrombosis	• Lack of normal venous signal; at times, T1 isointensity or a hypointense thrombus may be identified in a vein. • As the signal return depends on the time frame of imaging, a clot in the lumen may be hyperintense.	• Occluded or decreased venous flow is identified in the CTV.	• Occluded or decreased venous flow is identified in the venous phase.
Basilar occlusion syndrome	• A lack of normal basilar artery flow signal or in some cases, T1 isointensity or a hypointense thrombus may be identified in the basilar artery. • As the signal return depends on the time frame of imaging, a clot in the lumen may be hyperintense.	• No contrast enhancement or decreased blood flow can be identified in the top basilar artery or the bilateral PCA.	• No contrast enhancement or decreased blood flow can be identified in the top of the basilar artery or the bilateral PCA.

Table 2: Differential diagnosis table for bilateral thalamic infarction.

ABBREVIATIONS

ADC = Apparent diffusion coefficient
AOP = Artery of Percheron
AVM = Arteriovenous malformation
CT = Computed tomography
CTA = Computed tomography angiography
DSA = Digital subtraction angiography
DWI = Diffusion-weighted imaging
ICH = Intracerebral hemorrhage
MRA = Magnetic resonance angiography
MRI = Magnetic resonance imaging
P1 = 1st segment of posterior cerebral artery
PCA = Posterior cerebral artery
P-Com = Posterior communicating artery
SCA = Superior cerebellar artery

KEYWORDS

Artery of Percheron; Thalamic infarction; Cerebral angiography; Arteriovenous malformation; Post-angiographic obliteration rebleeding

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