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Case Report

Successful embolization of ventricular arteriovenous malformation supplied by the choroidal artery: A case report and literature review

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ABSTRACT

Background: Ventricular arteriovenous malformations (AVMs) are localized in the ventricles and are mainly fed by the anterior choroidal artery (AChoA) and posterior choroidal artery (PChoA). Surgical resection of ventricular AVMs is difficult as the lesions are localized deep in the brain. Therefore, endovascular treatment is expected to treat ventricular AVMs. However, embolization from the AChoA and PChoA carries the risk of ischemic complications. Even though there are some major reports on embolization strategies from the choroidal arteries, embolization of these arteries remains technically challenging. In this article, we report two successful cases of ventricular AVM embolization using AChoA and PChoA.

Case Description: Case 1: A 34-year-old male presented with intraventricular hemorrhage (IVH). Subsequently, ventricular AVM embolization in the anterior horn was performed using n-butyl-2-cyanoacrylate (NBCA) through the AChoA and medial PChoA, and complete obliteration was observed without neurological deterioration. Case 2: A 71-year-old female presented with IVH. Subsequently, ventricular AVM embolization in the lateral ventricle was performed through the AChoA and lateral PChoA with Onyx and NBCA, and partial obliteration was observed without complications. Furthermore, Gamma Knife surgery for residual lesions resulted in complete obliteration.

Conclusion: Embolization through the choroidal arteries for ventricular AVMs is an effective curative or adjunctive treatment.

Keywords: Arteriovenous malformation, Cerebral ventricular hemorrhage, Embolization

INTRODUCTION

Ventricular arteriovenous malformations (AVMs) are rare and account for 5–19% of all cerebral AVMs.[19] They cause intraventricular hemorrhage (IVH) and often have severe outcomes.[16,22] These AVMs are mainly fed by the anterior choroidal artery (AChoA) and posterior choroidal artery (PChoA). This type of AVM is difficult to treat surgically due to their deep localization. Therefore, endovascular treatment is expected to contribute to treat ventricular AVMs. Recent advancements in endovascular devices and interventional techniques have enabled the embolization of AVM from the choroidal arteries.[4,14] However, the AChoA and PChoA are frequently recruited to supply AVM; this involves important eloquent paraventricular structures,

such as the thalamus, splenium of the corpus callosum, and parietal and temporal lobes.[4,14] Consequently, the embolization of feeders arising from AChoA and PChoA carries the risk of ischemic complications. Although there are a few major reports on embolization strategies from the choroidal arteries, embolization of these arteries remains technically challenging and has a risk of complications.[12,27]

Here, we report two cases of ventricular AVM which was successfully treated by endovascular embolization through the AChoA and PChoA. We also describe the features of ventricular AVM and the possibility of embolization through the choroidal arteries based on the anatomical features, including a literature review.

CASE DESCRIPTION

Case 1

A 37-year-old male patient with a preceding headache was admitted to our hospital. His consciousness level was a Glasgow Coma Scale (GCS) 13 (E3V4M6), without motor and sensory deficits. Computed tomography (CT) scan of the brain showed IVH and acute obstructive hydrocephalus [Figure 1a]. In addition, cerebral angiography showed 1.2 cm-sized AVM nidus, fed by the left AChoA, medial striate artery, medial posterior choroidal artery (MPChoA), and lateral posterior choroidal artery (LPChoA), which drain into the internal cerebral vein (ICV) through the superior choroidal vein (Spetzler-Martin grade Ⅱ) [Figures 1b-e]. A volume rendering image showed an intranidal aneurysm to be the cause of the rupture [Figures 1f and g]. Maximum intensity projection images, which were reconstructed from three-dimensional rotational angiography (3D-RA), showed an AVM located in the caudate body and an exposed intranidal aneurysm on the left anterior horn ventricular wall [Figures 1h-j]. Therefore, we planned curative embolization because the AVM was at a deep location with deep venous drainage. Embolization was performed under general anesthesia on the 4th day, followed by external ventricular drainage (EVD) for hydrocephalus. A Magic 1.2FM flow-guided microcatheter (Balt, Montmorency, France) was navigated to the left MPChoA and advanced distally near the nidus [Figures 2a and b]. Selective angiography showed a proper feeder and an AVM nidus draining into the ICV without reflux to the normal territory [Figure 2c]. In addition, embolization was performed with 0.1 mL of 25% n-butyl-2-cyanoacrylate (NBCA) [Figure 2d]. The NBCA injected from the MPChoA even penetrated the nidus region supplied by the LPChoA, and the AVM was not visualized from the PChoA, resulting in partial occlusion

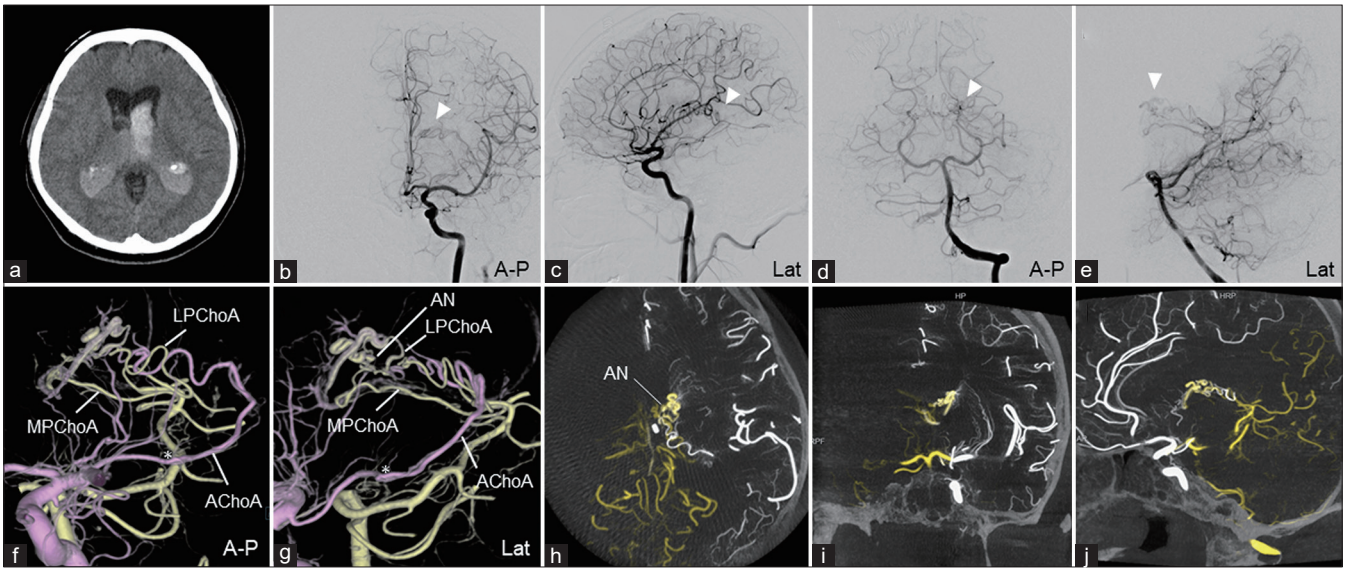

Figure 1: Preoperative clinical imaging of Case 1 (a) brain computed tomography shows intraventricular hemorrhage and acute hydrocephalus. (b and c) The left ICA angiography shows ventricular AVM fed by the left AChoA and the medial striate artery. (d and e) The left VA angiography shows AVM fed by the left LPChoA and MPChoA. The AVM drains into the internal cerebral vein. White arrowheads show the nidus of the AVM. (f and g) 3D-volume rendering (VR) fusion images (f: A-P, g: lateral view) reconstructed from 3D-RA of ICA (pink) and VA (yellow). An intranidal aneurysm was revealed (AN). The asterisk indicates the choroidal point of the AChoA. (h-j) The maximum intensity projection fusion images (h: axial, I coronal, and j: sagittal view) reconstructed from 3D-RA of ICA (white) and VA (yellow). The AVM is located in the body of the caudate. The intranidal aneurysm is within the ventricle. 3D: Three-dimensional, 3D-RA: Three-dimensional rotational angiography, AChoA: Anterior choroidal artery, A-P: Anterior-posterior view, AVM: Arteriovenous malformation, ICA: Internal cerebral artery, Lat: Lateral view, LPChoA: Lateral posterior choroidal artery, MPChoA: Medial posterior choroidal artery, VA: Vertebral artery.

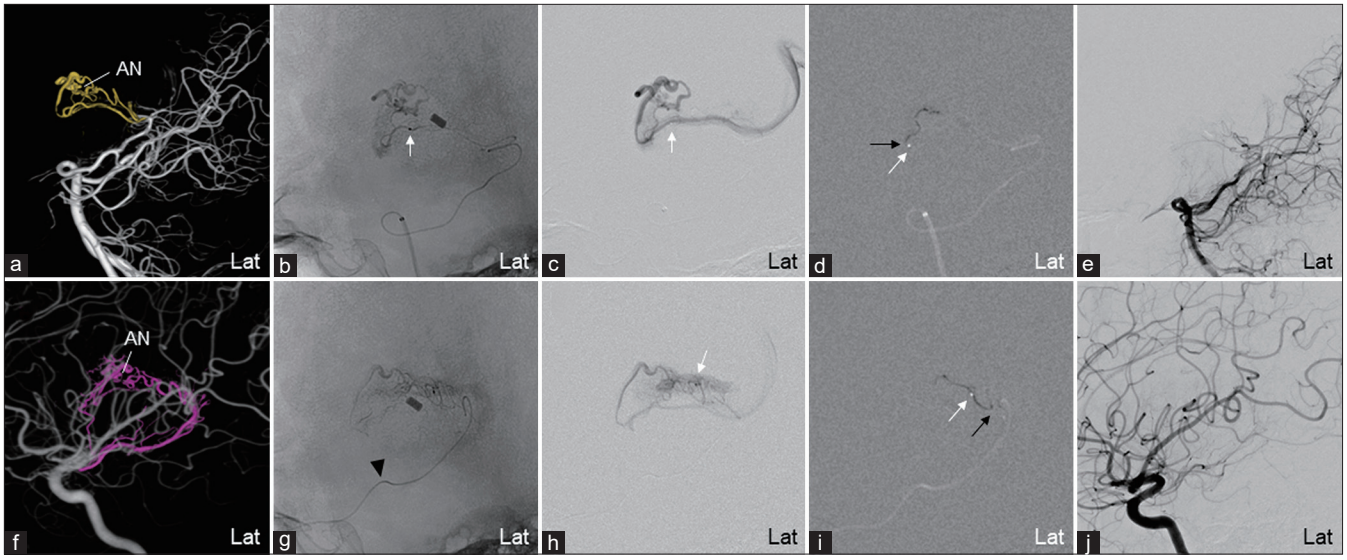

Figure 2: Embolization of the AVM through the MPChoA and AChoA (Case 1) (a) VR image reconstructed from the 3D-RA of the VA. The AVM is fed by the MPChoA and the LPChoA from the left PCA P2 segment. (b, c) Fluoroscopic and DSA of the selective microcatheter injection. The microcatheter was advanced to the MPChoA plexal segment. (d) Image of the NBCA glue casting injected from the MPChoA. (e) The VA angiography shows the disappearance of AVM from the MPChoA and LPChoA after embolization. (f) VR image reconstructed from the 3D-RA of the ICA. The AVM is fed by the AChoA (pink) and the medial striate artery from the ICA. (g and h) Fluoroscopic and DSA of the microcatheter injection. The microcatheter was advanced to the AChoA plexal segment. Black arrowheads show the choroidal point of the AChoA. (i) Image of the NBCA glue casting injected from the AChoA with slight reflux. (j) ICA angiography shows the disappearance of the AVM after embolization. White and black arrows show the microcatheter's tip and the reflux of the embolic agent, respectively. There is an aneurysm in the nidus (AN). 3D-RA: Three-dimensional rotational angiography, 3D-VR: Three-dimensional volume rendering, AchoA: Anterior choroidal artery, AVM: Arteriovenous malformation, DSA: Digital subtraction image, ICA: Internal carotid artery, Lat: Lateral view, LPChoA: Lateral posterior choroidal artery, MPChoA: Medial posterior choroidal artery, NBCA: n-butyl-2-cyanoacrylate, PCA: Posterior cerebral artery, PChoA: Posterior choroidal artery, VA: vertebral artery.

[Figure 2e]. Subsequently, a Magic 1.2FM was advanced to the left AChoA distally beyond the choroidal point. After microcatheter injection confirmed that there was no reflux to the normal territory, embolization was performed with 0.08 mL of 20% NBCA [Figures 2f-i], with slight glue reflux. Angiography revealed complete obliteration of the AVM after the second injection [Figure 2j], and no neurological deficits were observed after embolization. Postoperative magnetic resonance imaging (MRI) showed no acute cerebral infarction. Postoperative follow-up cerebral angiography 25 days after embolization revealed complete obliteration. On day 29 postadmission, he was transferred to a rehabilitation hospital with a modified Rankin Scale (mRS) 2.

Case 2

A 71-year-old comatose female patient was transported to the former hospital. Her consciousness level was a GCS score of 6 (E1V1M4). A brain CT scan showed IVH and acute obstructive hydrocephalus [Figure 3a], and CT angiography showed a brain AVM. On the day of admission, EVD was performed for the acute hydrocephalus. She was transferred to our hospital for treatment of the brain AVM. Cerebral angiography showed an AVM located in the trigone of the right lateral ventricle, which was fed by AChoA and LPChoA and was draining into the inferior ventricular vein to the basal vein of the rosenthal, tentorial sinus, and transverse-sigmoid sinus junction (Spetzler-Martin grade II) [Figures 3b-j]. Therefore, we planned adjunctive embolization before Gamma Knife surgery (GKS) since the AVM was deep and had deep venous drainage. First, embolization was performed under general anesthesia with monitoring of the transcranial motor-evoked potential (MEP) and somatosensory evoked potential (SEP). Next, a Marathon flow-guided microcatheter (eV3 Inc., Plymouth, MN, USA) was advanced to the plexal segment of the right AChoA and distally beyond the choroidal point [Figures 4a and b]. In total, 0.19 mL of Onyx18 (eV3 Inc.) was injected, and it penetrated the nidus without excessive Onyx reflux [Figures 4c and d]. After that, the microcatheter was removed smoothly. Subsequently, a Magic 1.2FM microcatheter was advanced to the right LPChoA through the posterior communicating artery and distally closer to the nidus [Figures 4e and f]. Next, 4 mL of 25% NBCA was injected from the LPChoA for residual AVM [Figures 4g and h]. NBCA partly penetrated the draining side beyond the nidus. Angiography showed that partial obliteration resulted in a significant flow reduction [Figure 4i]. The intraoperative

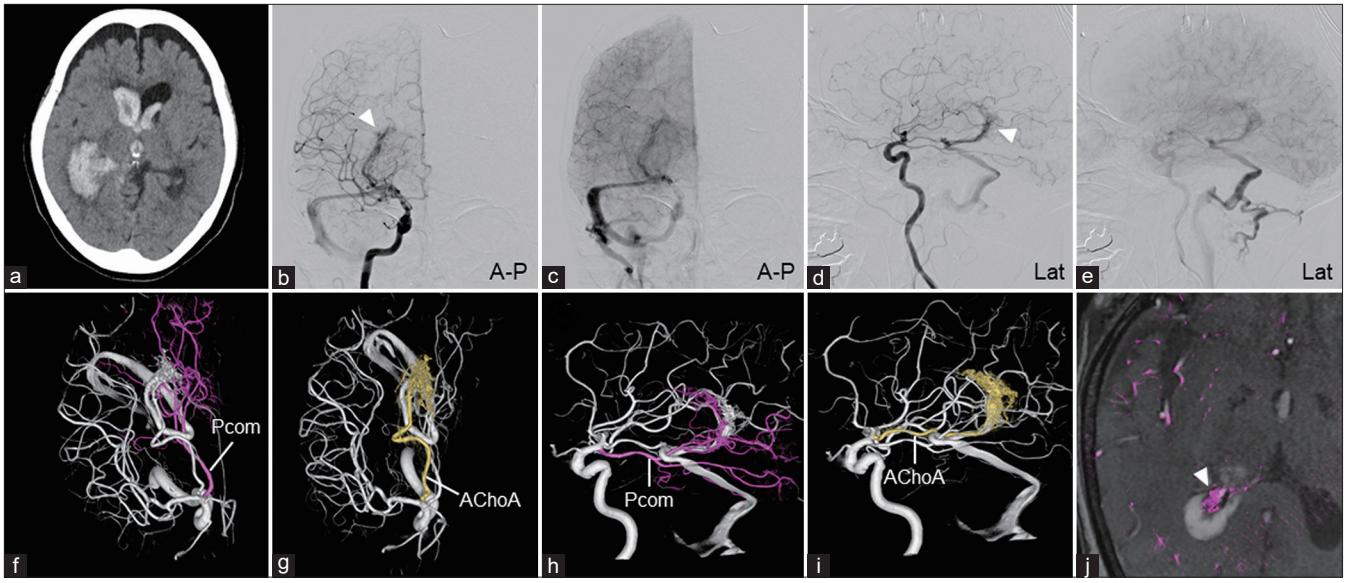

Figure 3: Preoperative clinical imaging of Case 2 (a) brain computed tomography shows intraventricular hemorrhage and acute hydrocephalus. (b-e) Right ICA angiography shows the AVM fed by AChoA and LPChoA, draining into the inferior ventricular vein to the basal vein of the rosenthal, tentorial sinus, and transverse-sigmoid sinus junction. (f-i) 3D-VR images reconstructed from the ICA (f and g: Cranial, h and i: Lateral view) shows the AVM fed by LPChoA originating from the Pcom (pink) and AChoA (yellow). (j) Fusion image of 3D-RA of ICA and time-of-flight magnetic resonance angiography shows the AVM located within the trigone of the right ventricle. White arrowheads show the nidus of the AVM. 3D-RA: Three-dimensional rotational angiography, 3D-VR: Three-dimensional volume rendering, A-P: Anterior-posterior view, AChoA: Anterior choroidal artery, AVM: Arteriovenous malformation, ICA: Internal carotid artery, Lat: Lateral view, LPChoA: Lateral posterior choroidal artery, Pcom: Posterior communicating artery.

MEP and SEP monitoring during embolization remained unchanged. In addition, no new neurological deterioration was observed after embolization, and postoperative MRI showed no acute cerebral infarction. Furthermore, on postoperative day 16, a GKS was performed for the residual nidus. Finally, she was discharged with a mRS 1. The AVM disappeared on a follow-up angiography 3 years after GKS without latency period rebleeding [Figure 4j].

DISCUSSION

We performed successful embolization from the AChoA and PChoA for two ruptured ventricular AVMs without ischemic complications. Although ventricular AVMs within the ventricles are not eloquent locations, they are high-risk AVMs for treatment because they frequently possess deep drainers and require surgical removal through the eloquent cortex.[9] Embolization should also be performed through eloquent feeders, which include choroidal arteries. Conventionally, surgery and radiotherapy have been attempted for ventricular AVMs due to the difficulty in advancing the microcatheter into the proper feeder and the risk of ischemic complications to eloquent structures. However, safe resection of the ventricular AVMs is challenging because they are fed by the AChoA and PChoA and are located deep in the narrow surgical field.[3,4,15,19,25] Since the obliteration of the deep arterial feeding pedicles tends to be the final step in the surgical resection of deep-seated AVMs supplied by the choroidal arteries, presurgical embolization of these feeders could prove beneficial.[4] Therefore, preoperative adjunctive embolization of the ventricular AVM could be expected. Although good obliteration rates (77% at 5 years) have been reported in GKS for ventricular AVM, the rebleeding rate during latency periods is also higher than that for AVMs at other sites.[1] However, the obliteration rate is lower with GKS for ventricular AVM due to the relationship between the ventricles and surrounding cerebral spinal fluid, which prevents temperature increase.[17] Therefore, treatment with GKS alone may not be compelling.

There are high expectations for endovascular treatment in contributing to adjunctive or curative embolization. The main feeders of ventricular AVMs are AChoA, MPChoA, and LPChoA.[4,15] Recently, safe and effective embolization techniques have been reported for ventricular AVMs with advances in endovascular devices, such as flow-related catheters, embolic materials, including Onyx, and imaging analysis.[13,14,22] Although the effectiveness of curative or adjunctive embolization from AChoA has been reported, the risks of embolization are mainly ischemic complications to the eloquent structures and cerebral hemorrhage at the time of catheter extraction.[4,12] In addition, the perforating branches of the AChoA originate from the cisternal segment,

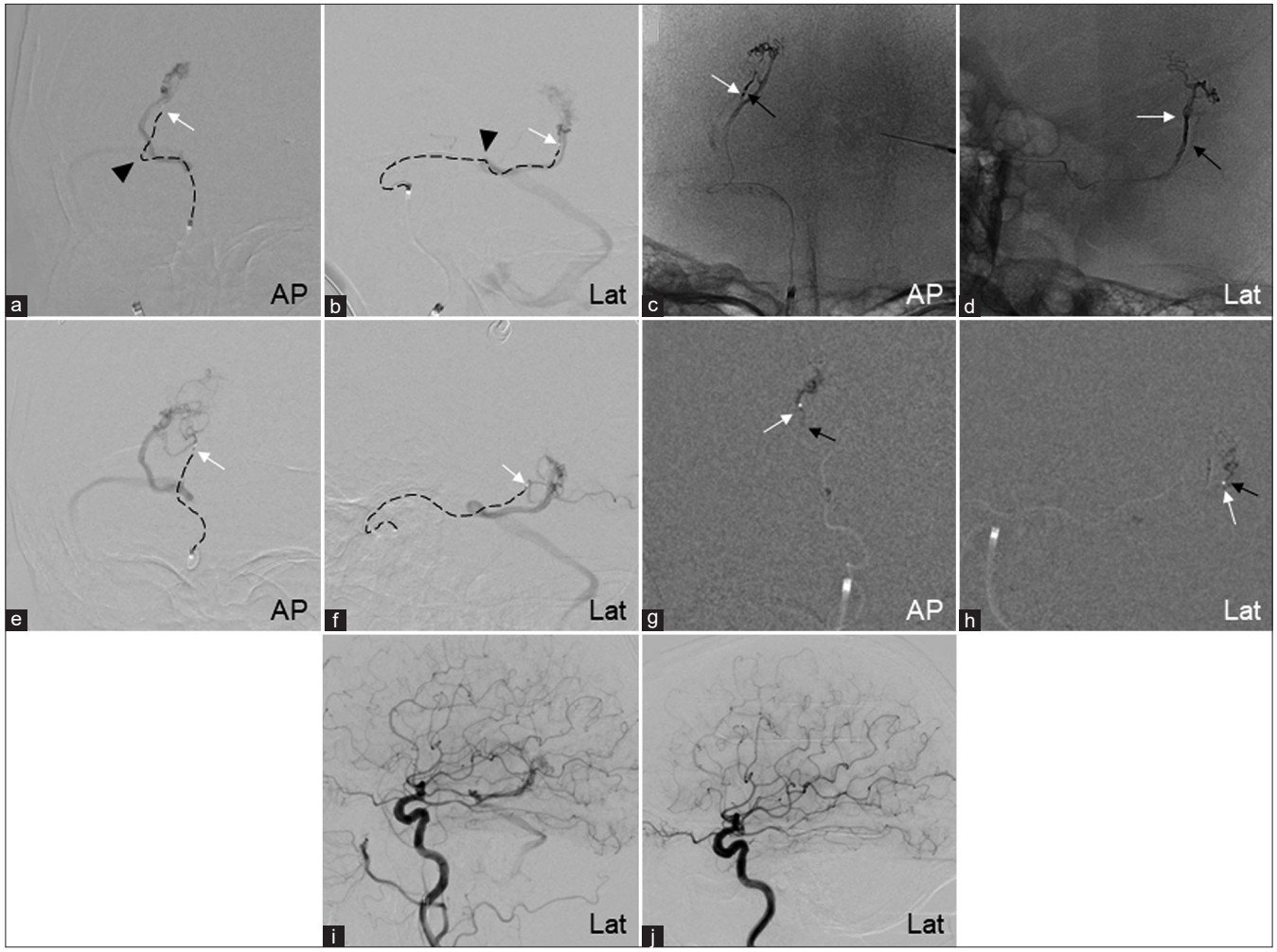

Figure 4: Embolization of AVM through the right AChoA and LPChoA (Case 2) (a and b) Selective angiography of the AChoA. The microcatheter (dotted line) is advanced to the AChoA plexal segment. Black arrowheads show the choroidal point of the AChoA. Selective angiography shows AVM draining into the transverse-sigmoid sinus junction through inferior ventricular vein, basal vein of the rosenthal, and tentorial sinus. (c and d) Fluoroscopic images of the Onyx cast injected from the AChoA. (e and f) The microcatheter (dotted line) is advanced to the right posterior cerebral artery through the Pcom. Selective angiography shows the AVM fed by the LPChoA. (g and h) Fluoroscopic images of the NBCA cast injected from the LPChoA. (i) ICA angiography after embolization shows residual lesion with flow reduction. (j) The AVM disappeared on a follow-up angiography 3 years after gamma knife surgery (GKS). White and black arrows show the microcatheter's tip and the reflux of the embolic agent, respectively. 3D-VR: Three-dimensional volume rendering, A-P: Anterior-posterior view, AChoA: Anterior choroidal artery, AVM: Arteriovenous malformation, ICA: Internal carotid artery, Lat: Lateral view, LPChoA: Lateral posterior choroidal artery, NBCA: n-butyl-2-cyanoacrylate, PChoA: Posterior choroidal artery, Pcom: Posterior communicating artery.

which is proximal to the plexal point. Specifically, the plexal point is the entry of the AChoA into the lateral ventricle at the choroidal fissure, resulting in a steep downward shape on lateral angiograms. Therefore, the catheter tip should be placed beyond the plexal point to avoid severe ischemic complications during AVM embolization through the AChoA.[3,4,14] However, anatomical variations in the capsulothalamic artery's branching position arise from the AChoA, so the vessel's occlusion results in hemiparesis, unilateral sensory disturbance, and homonymous hemianopia, should be considered.[6] In a cadaver study, 38% of the capsulothalamic arteries originated from the first portion of the plexal segment.[6] In another study, 24% of the arterial branch arising from the plexal segment of the AChoA supplies the lateral geniculate body.[7] These anatomical variations may be associated with unexpected ischemic complications during embolization.[4] In our cases, safe embolization through the AChoA plexal segment was possible by advancing the catheter to the proper feeder closer to the nidus.

Furthermore, it is important to understand the anatomical and angiographic features of PChoA for the safe embolization

of ventricular AVMs.[4,8] PChoA can be categorized into MPChoA and LPChoA. Specifically, MPChoA arises mainly from the posterior cerebral artery (PCA) distal P1 or proximal P2 segments, supplying small branches to the midbrain, tegmental, thalamus, and pineal gland as cisternal segments. It penetrates the velum interpositum of the third ventricle roof and runs as a plexal segment to the foramen of Monro. In addition, the MPChoA, which runs dorsally to the pineal body, marking numeral 3 on lateral angiograms, is considered the plexal point of the MPChoA.[7,21] Furthermore, the plexal segment of the MPChoA has branches to the thalamus and the choroid plexus.[7] Therefore, embolization is not necessarily safe even when the microcatheter advances beyond the cisternal segment. In our cases, safe embolization was achieved by guiding the microcatheter to the position where the plexal segment of the MPChoA ran into the foramen of Monro.

The LPChoA originates mainly from the P2 and P3 segments of the PCA and courses through the ambient cistern toward the choroidal fissure, between the thalamus and crus, the fornix, through the dorsal horn of the lateral ventricle to the body, and toward the foramen of Monro.[7,24] LPChoA branches into the choroid plexus and trigone of the lateral ventricle. In addition, the cisternal segment of the LPChoA produces the branches supplying the thalamus, fornix, and splenium of the corpus callosum.[18,24,29] However, embolization from the LPChoA remains challenging since an established angiographic safety point has not been reported.[4] The plexal segment of the LPChoA also has branches to the lateral ventricle wall, thalamus, fornix, and choroid plexus.[7,24]

From a literature review of AVMs with embolization through the PChoA, Ezura *et al*. reported the first safe embolization case of thalamic AVM through the PChoA as adjunctive embolization followed by GKS in 1992;[5] however, only a few reports of AVMs embolized through PChoA have been reported.[2,5,9,14,26,27] The location of AVMs embolized from PChoA included the ventricle, splenium, thalamus, and temporal lobe.[4,14,26] In addition, Onyx and NBCA were the main embolic agents.[4,14,27] Although few descriptions of the detailed microcatheter position during PChoA embolization exist, advancing the microcatheter as distally as possible and positioning the microcatheter wedge with minimum embolic agent reflux could avoid the risk of irretrievable complications.[9,14] However, excessive glue penetration should be avoided since almost all AVMs supplied from PChoAs are accompanied by deep drainers, which can lead to unexpected drainer occlusion.[9,11] To the best of our knowledge, ischemic complications of PChoA embolization have been reported in only one case of splenium and pulvinar infarction when embolizing from the LPChoA with Onyx.[4] As the largest series, Hou *et al*. reported AVM embolizations from PChoA in 24 patients.[9] Notably, they obtained relatively good outcomes, with good obliteration by embolization alone in half of the cases.[9]

We successfully embolized the ventricular AVMs from the AChoA and PChoA without complications by guiding the microcatheter to a more distal proper feeder. However, the safety of embolization may not be ensured by anatomical knowledge alone, even when embolization is performed beyond the angiographic safety point. Consequently, anatomical variations should be considered as unexpected complications can also occur. In addition, similar to the two cases reported here, the anastomosis of the AChoA and PChoA as complementary or that between the PChoA and the anterior cerebral artery should be noted,[10,20,23,28,29] leading to an unexpected occlusion through anastomosis due to excessive embolization. Furthermore, for safe embolization, it is important to sufficiently navigate the microcatheter distally and confirm the absence of reflux to the normal territory by microcatheter injection before the glue injection; however, a steal phenomenon of blood flow due to AVM may obscure eloquent normal vessels, even with selective injection. Therefore, minimizing the embolic material reflux is critical to avoiding unexpected feeder occlusion, and the possibility of retrograde thrombosis of choroidal arteries that lead to ischemia should be considered.[4]

CONCLUSION

In this article, we reported the effectiveness of curative and adjunctive embolization of the choroidal arteries for ventricular AVMs. The microcatheter should be advanced as distally as possible to safely embolize the choroidal arteries while avoiding ischemic complications and the anatomical variation of the perforator branching position.

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Declaration of patient consent

The Institutional Review Board (IRB) permission obtained for the study.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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