Online June 5, 2020
Journal of Neuroendovascular Therapy 2020; 14: 381–389 DOI: 10.5797/jnet.cr.2020-0004

Case Report

Five Cases of High-grade Arteriovenous Malformation Treated by Presurgical Embolization through the Anterior Choroidal Artery

Miyahito Kugai,[1] Takehiro Suyama,[2] Masahiko Kitano,[1] Yoshiko Tominaga,[1] and Shinsuke Tominaga[1]

Objective: In cases of cerebral arteriovenous malformation (AVM) in which perforators are involved as feeder, hemostasis is difficult during surgical removal and postoperative hemorrhage may develop. If possible, presurgical embolization should be performed. However, when the anterior choroidal artery (AChA) is the feeder, the risk of embolization is particularly high, and there are few reports describing this situation. Authors report the treatment results of five cases of AVM in which a single operator performed presurgical embolization through the AChA and describe the technique with a review of the literature.

Case Presentations: Of the five total cases (three men and two women; average age was 43.4 years [28–68 years]), one case presented with hemorrhage, two with epilepsy, the other ones with headache and trigeminal neuralgia, respectively. The lesions were located in the frontal lobe in one case and in the temporal lobe in four cases. On the Spetzler-Martin (SM) grading scale, four cases were grade III and one was grade IV. The eloquent area was involved within the nidus in four cases. Multimodal treatment was planned because all cases were high-grade AVM. Authors thought that performing presurgical embolization through the AChA would reduce the overall risk of treatment and performed the presurgical embolization. The embolization was possible in all cases, and the AVM was not angiographycally visible through the AChA in three cases. The blood flow through the AChA was reduced in two cases. All cases were awake immediately after embolization and no case had neurological symptom after embolization. CT or MRI after embolization revealed asymptomatic infarction in two cases. The AVM was removed safely without difficulty including hemostasis.

Conclusion: In this series, there were no morbidity and embolization was performed relatively safely. Embolization through the AChA was suggested to be an effective treatment, but careful consideration is required in each individual case.

Keywords ▶ anterior choroidal artery, arteriovenous malformation, presurgical embolization

[1]*Department of Neurosurgery, Tominaga Hospital, Osaka, Osaka, Japan*
[2]*Department of Neurosurgery, Kansai Medical University Medical Center, Moriguchi, Osaka, Japan*

Received: January 9, 2020; Accepted: April 23, 2020
Corresponding author: Miyahito Kugai. Department of Neurosurgery, Tominaga Hospital, 1-4-48, Minatomachi, Naniwa-ku, Osaka, Osaka 556-0017, Japan
Email: md002042@yahoo.co.jp

Introduction

In cases of cerebral arteriovenous malformation (AVM) in which perforators are involved as feeder, hemostasis is difficult during surgical removal and postoperative hemorrhage may develop. If possible, presurgical embolization should be performed. However, when the anterior choroidal artery (AChA) is a feeder, the risk of embolization is particularly high and there are few reports on this situation. In this study, authors present the results of the treatment in five cases of AVM in which a single operator performed presurgical embolization through the AChA.

Table 1 Summary of cases with AVM embolized through AChA

Case no.	Age (years)	Sex	Location	SM grade	Microcatheter	Position of the microcatheter	Embolic agent	Angiographic occlusion through AChA	Complications
1	43	F	Temporal	III	PROWLER SELECT plus	Cisternal segment	5% NBCA	Flow obliteration	Asymptomatic Infarction
2	34	F	Temporal	III	Marathon	Plexal segment	5% NBCA Coils	Flow reduction	No
3	28	M	Frontal	III	Marathon	Plexal segment	5% NBCA	Flow obliteration	No
4	68	M	Temporal	IV	SL-10	Plexal point	20% NBCA	Flow obliteration	Asymptomatic Infarction
5	44	M	Temporal	III	Marathon	Cisternal segment	5% NBCA	Flow reduction	No

AVM: arteriovenous malformation; AChA: anterior choroidal artery; NBCA: N-butyl cyanoacrylate; SM: Spetzler-Martin

Authors report the technical strategies with a review of the literature.

Case Presentation

All cases had high-grade AVM and multimodal treatments were planned. Authors thought that performing presurgical embolization through the AChA would reduce the overall risk of treatment and performed the presurgical embolization for all cases (**Table 1**).

Case 1: A 43-year-old female. Detailed studies for headache revealed AVM in the right temporal lobe. Spetzler-Martin (SM) grading scale of the AVM was III. The first session of the embolization was conducted through the middle cerebral artery (MCA), and the second session was through the AChA (**Fig. 1**). CT after embolization revealed an infarction in the right caudate nucleus (**Fig. 1F**), but there was no neurological symptom. Surgical removal of the AVM was performed the following day. She was discharged with a modified Rankin Scale (mRS) score of 0.

Case 2: A 34-year-old female presented with epilepsy and an AVM of the left temporal lobe was found. The SM grade was III. The localization of the nidus involved the visual area. The first session of the embolization was performed through the MCA, and the second session was through the MCA, posterior cerebral artery (PCA), and AChA (**Fig. 2**). CT after embolization revealed no abnormalities. Surgical removal was conducted the following day. She was discharged with a mRS score of 1 (epilepsy).

Case 3: A 28-year-old male presented with hemorrhage and an AVM was found in the right frontal lobe. The SM grade was III. The nidus involved the motor area. The presence of an intranidal aneurysm was suspected. The first and second sessions of embolization were performed through the MCA, and the third session was through the AChA (**Fig. 3**). Neither CT nor MRI after embolization revealed an infarction, and there was no deterioration of neurological symptoms. Three days after embolization, surgical removal was conducted. He was discharged with a mRS score of 3.

Case 4: A 68-year-old male presented with the right trigeminal neuralgia and the temporal lobe AVM was found. The SM grade was IV. The nidus involved the visual area. The sixth session of embolization was performed through the AChA (**Fig. 4**). CT and MRI after the embolization revealed an infarction in the right lateral geniculate nucleus. However, there was no neurological symptom (**Fig. 4F**). Three days after embolization, surgical removal was conducted. He was discharged with a mRS score of 1 (cerebellar infarction developed on the fifth session of embolization, inducing mild cerebellar ataxia, but it improved during subsequent follow-up).

Case 5: A 44-year-old male presented with epilepsy and the left temporal lobe AVM was found. The SM grade was III. The nidus involved the visual area. The first and second sessions of embolization were performed through the MCA, and the third session was through the AChA (**Fig. 5**). CT and MRI after embolization revealed no infarction. Two days after embolization, surgical removal was conducted. He was discharged with a mRS score of 0.

In all cases, embolization was performed under general anesthesia. A 4-6-Fr. FUBUKI Dilator Kit (Asahi Intecc, Aichi, Japan) was inserted as a guiding sheath through the right femoral artery. As microcatheters, a PROWLER SELECT Plus (STR) (Johnson & Johnson,

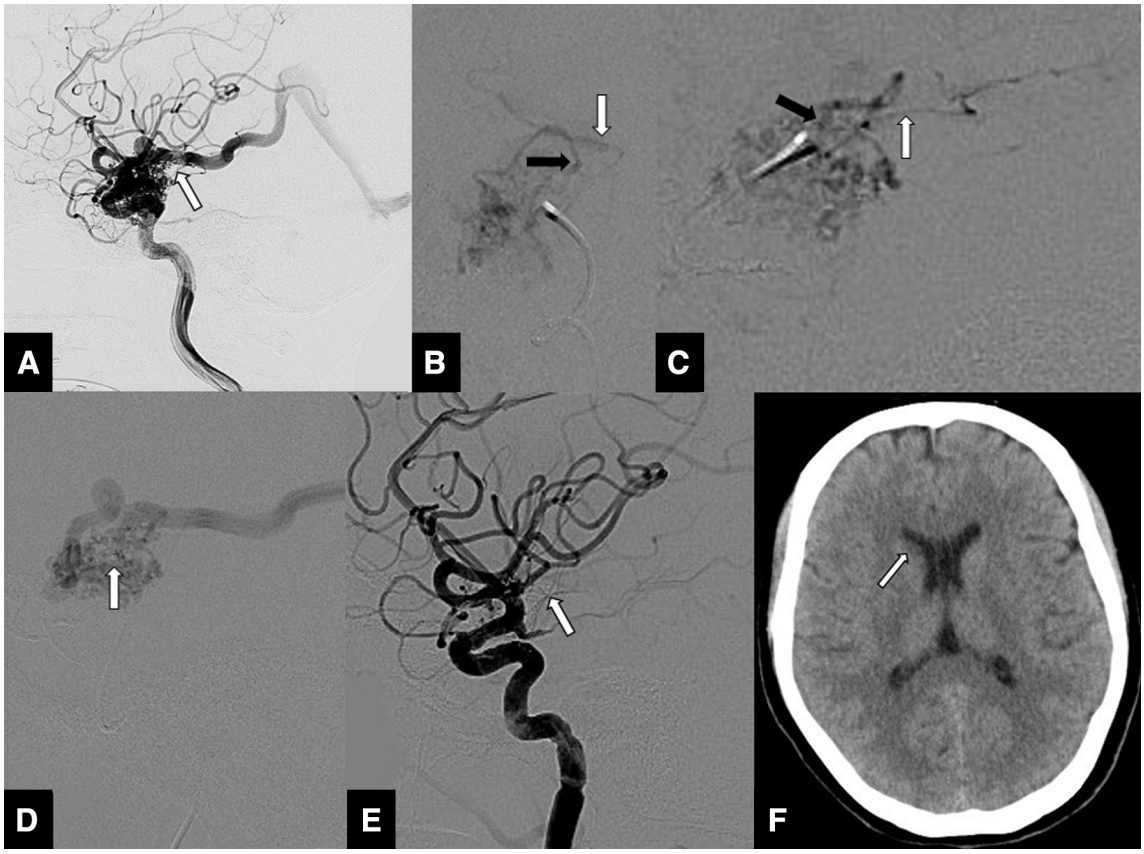

Fig. 1 (**A**) Lateral view on right internal carotid angiography before embolization. AChA (white arrow). (**B** and **C**) Microcatheter angiography before embolization (right AChA). Frontal and lateral views. AChA (white arrow). Feeder (black arrows). (**D**) A microcatheter was unable to be guided beyond the cisternal segment. Microcatheter tip was positioned at a short distance from the orifice of the AChA. Normal perfusion of the AChA was unable to be confirmed, but the nidus was visualized. Microcatheter tip (white arrow). (**E**) Lateral view on right internal carotid angiography after embolization. The nidus was not visualized through the right AChA. AChA (white arrow). (**F**) CT after embolization. A small infarction was detected in the right caudate nucleus (white arrow). AChA: Anterior choroidal artery

Miami, FL, USA) was used in Case 1, a Marathon Flow Directed Micro Catheter (Medtronic, Minneapolis, MN, USA) (Marathon) in Cases 2, 3, and 5, and an Excelsior SL-10 (90 degrees) (Stryker, Kalamazoo, MI, USA) (SL-10) in Case 4. Concerning microguidewires, CHIKAI 10, CHIKAI 008, and CHIKAI X 010 (Asahi Intecc) were used. Embolization was performed in the cisternal segment (proximal to the plexal point), at a short distance from the orifice of the AChA in Case 1 (**Figs. 1D and 6A**) and at an area distal of the feeder branching from the cisternal segment in Case 5 (**Figs. 5E and 6E**). In Cases 2 and 3, embolization was conducted in the plexal segment (distal to the plexal point) (**Figs. 2B, 3C, 6B, and 6C**). In Case 4, it was performed at the plexal point (**Figs. 4D and 6D**). In Cases 1, 3, and 5, the AVM was embolized with 5% N-butyl-2-cyanoacrylate (NBCA, Histoacryl; B. Braun, Melsungen, Germany). In Case 2, the AVM was unable to be completely embolized with 5% NBCA, and the proximal side of a feeder in the plexal segment was embolized with coils (**Fig. 2C**). A feeder from the cisternal segment remained (**Figs. 2D and 2E**). In Case 4, the AVM was embolized with 20% NBCA. Embolization was possible in all cases. In Cases 1, 3, and 4, no AVM was visualized on angiography through the AChA (**Figs. 1E, 3D, and 4E**). In Cases 2 and 5, angiographically decreased blood flow was achieved through the AChA (**Figs. 2E and 5F**). All cases were awake immediately after embolization, and no case had neurological symptom after embolization. CT or MRI after embolization revealed asymptomatic cerebral infarction in two cases (Cases 1 and 4) (**Figs. 1F and 4F**), but they did not cause any morbidity. During the surgical removal of the AVM, there was no difficulty of hemostasis related to AChA, for the following reasons: the detachment and hemostasis of a feeder involving the AChA, which is difficult to secure at the deepest area of the surgical field, was unnecessary; and sufficient decompression of the nidus

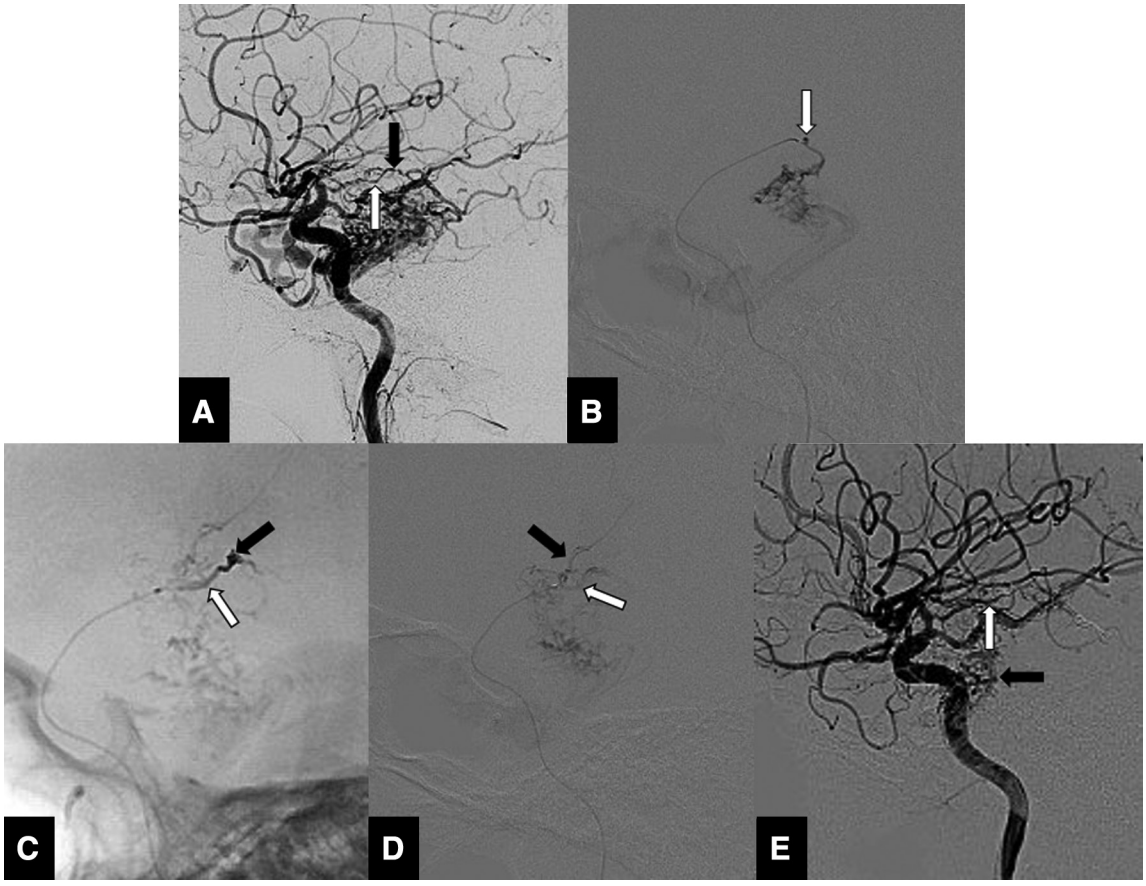

Fig. 2 (**A**) Lateral view on left internal carotid angiography before embolization. Plexal point (white arrow). Position of the microcatheter tip (black arrow). (**B**) Microcatheter angiography before embolization (left AChA). A microcatheter was guided to the plexal segment beyond the plexal point. A nidus was visualized through the left AChA. Microcatheter tip (white arrow). (**C**) Microcatheter angiography after embolization (left AChA). Plexal point (white arrow). The plexal segment was embolized with coils (black arrow). (**D**) Microcatheter angiography after embolization (left AChA). A feeder from the plexal segment disappeared, but a feeder from the cisternal segment remained. Plexal point (white arrow). Capsulothalamic artery (black arrow). (**E**) Lateral view on left internal carotid angiography after embolization. Visualization of the nidus through the left AChA remained, but decreased blood flow was achieved (black arrow). Plexal point (white arrow). AChA: Anterior choroidal artery

was achieved by embolization, facilitating floor treatment, which is highly difficult on nidus dissection. Thus, the AVM was safely removed. There was no hemorrhage related to the "normal perfusion pressure breakthrough (NPPB)" phenomenon, and the course of all cases was favorable.

Discussion

Recently, AVM embolization has been increasingly used due to advances in devices for endovascular therapy, and its role is highly important.[1] Presurgical embolization has reduced the difficulty of hemostasis and NPPB phenomenon. In addition, regarding surgical removal, advances in intraoperative angiography, image-guided neurosurgery, and intraoperative electrophysiological monitoring have improved the results of treatment.[2] The purpose of embolization is to facilitate surgical removal through decompression of a nidus, prevent the NPPB phenomenon, and facilitate the detachment and hemostasis of a feeder from the deep area of the surgical field, where it is difficult to secure the feeder. For multimodal treatment of high-grade AVM, embolization is highly useful.[3] However, according to previous studies, the incidence of complications related to AVM embolization is approximately 10%,[4,5] of which ischemic complications is 6%,[6] and the mortality/morbidity is 6.6%.[7] When a feeder involving a perforator is not embolized, hemostasis may be difficult during surgical removal or postoperative hemorrhage may develop.[8] If possible, presurgical embolization should be performed. In addition, during surgical removal, the perforator is located in the deepest area of the surgical field; it is difficult to

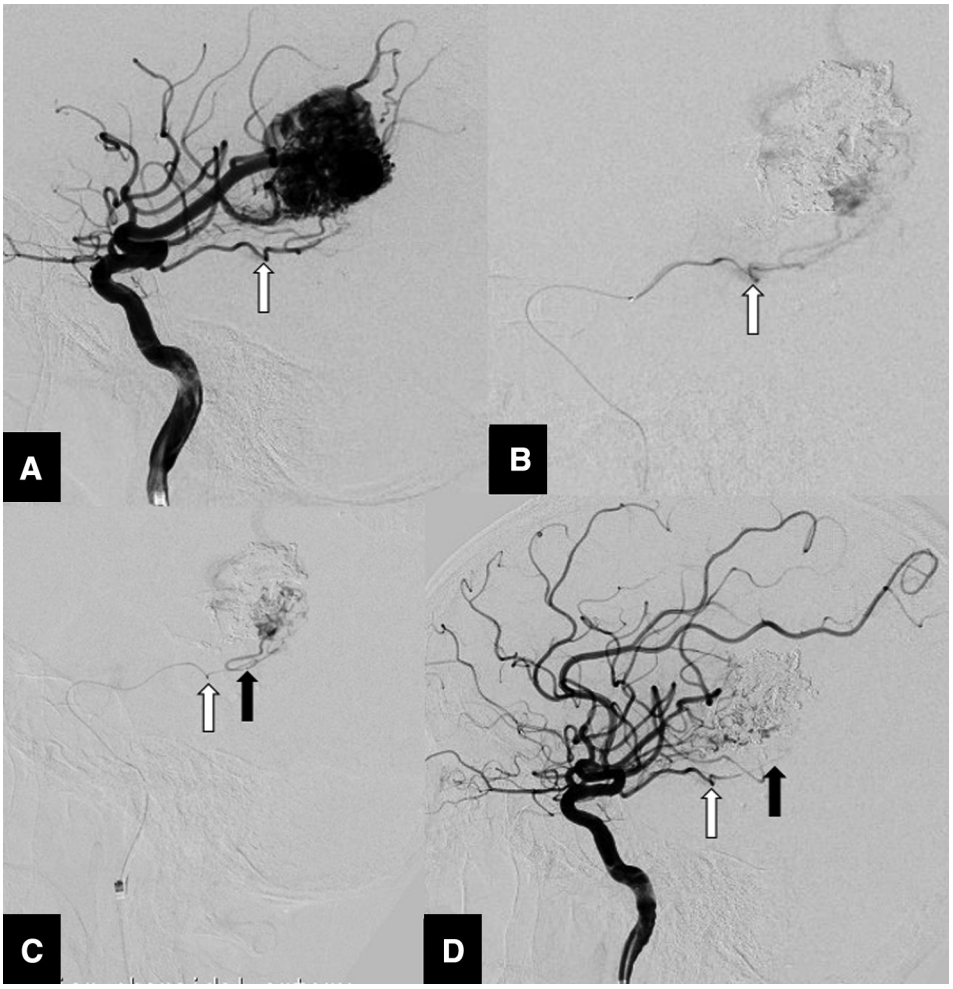

Fig. 3 (**A**) Lateral view on right internal carotid angiography before embolization. Plexal point (white arrow). (**B** and **C**) Microcatheter angiography before embolization (right AChA). A microcatheter was guided to the plexal segment beyond the plexal point. A nidus was visualized through the right AChA. Plexal point (white arrows). Microcatheter tip (black arrow). (**D**) Lateral view on right internal carotid angiography after embolization. The nidus was not visualized through the right AChA (black arrow). Plexal point (white arrow). AChA: Anterior choroidal artery

treat a feeder involving the AChA in many cases.[9] In many cases of high-grade AVM of the temporal lobe, it is difficult to reach the lateral ventricle through the cortex to secure the AChA. Considering this, presurgical embolization was performed through the AChA in Cases 1, 2, 4, and 5.

Embolization of AVM with a feeder involving the AChA is a high-risk procedure. To our knowledge, four reports have been published.[10–13] AChA syndrome (Abbie or Monakow syndrome) is a particularly serious complication.[14] To perform embolization through the AChA safely, cautious anatomical and radiological consideration is necessary.[15,16] Normally, the AChA branches as a single artery, then into two to four vessels. A perforator that passes through the anterior perforated substance branches from the cisternal segment; therefore, if embolization is performed in the plexal segment beyond the plexal point, the procedure may be relatively safe. However, there is a variation in which the capsulothalamic artery branches from the first part of the plexal segment.[17] Embolization in the cisternal segment does not always induce infarction, but even embolization beyond the plexal point may cause infarction of the lateral geniculate nucleus.[18] Elkordy et al.[12] reported eight cases who had undergone embolization through the AChA. According to their article, permanent hemiplegia was noted in one of four cases in whom embolization was performed in the cisternal segment, and asymptomatic cerebral infarction involving the lateral geniculate nucleus in one of four

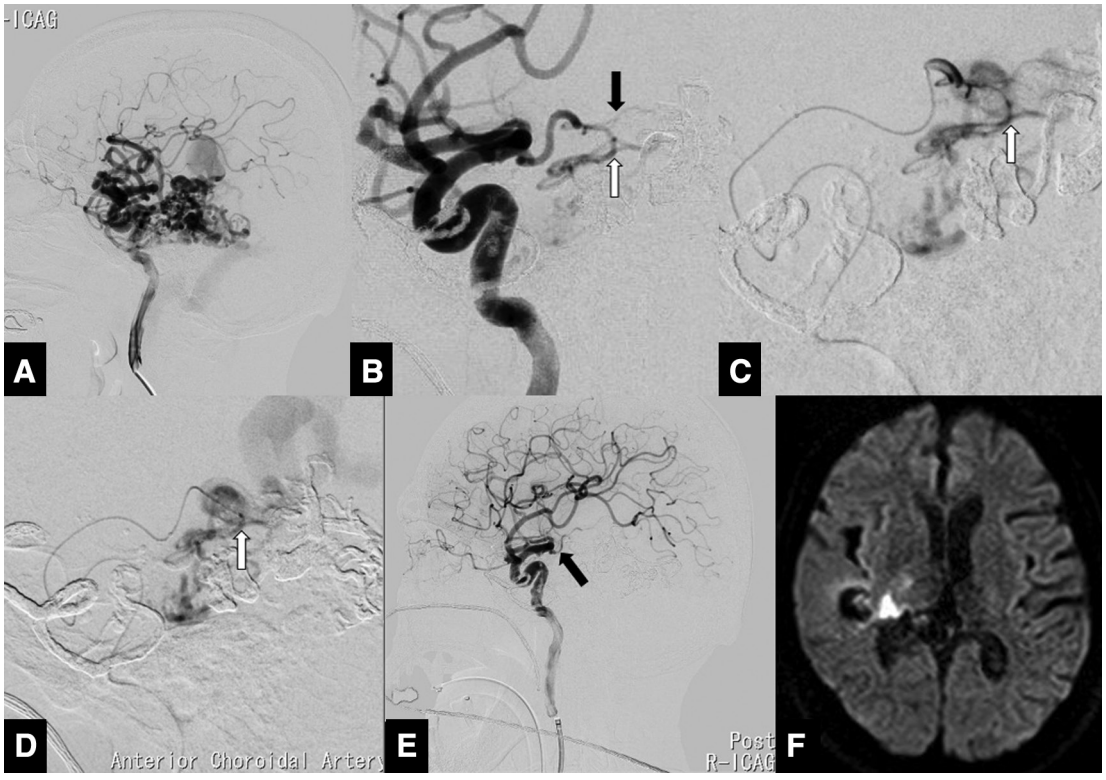

Fig. 4 (**A** and **B**) Lateral views on right internal carotid angiography before embolization. Capsulothalamic artery (black arrow). Plexal point (white arrow). (**C** and **D**) Microcatheter angiography before embolization (right AChA). A microcatheter was unable to be guided beyond the plexal point (microcatheter tip was positioned at the plexal point). A nidus was visualized through the right AChA. Plexal point (white arrow). (**E**) Lateral views on right internal carotid angiography after embolization. The nidus was not visualized through the right AChA. AChA (black arrow). (**F**) MRI after embolization. An infarction was detected in the right lateral geniculate nucleus. AChA: Anterior choroidal artery

cases in whom embolization was performed in the plexal segment. In addition, another study found that embolization reduced the blood flow through AChA, leading to retrograde occlusion of this artery.[11] Concerning embolization in the plexal segment, Lv et al.[13] were able to perform this procedure beyond the plexal point in all cases. According to Elkordy et al.,[12] embolization beyond the plexal point was possible in four of eight cases. Authors also consider it difficult to guide a microcatheter to a sufficiently distal area beyond the plexal point in some cases. In Case 4, the pushability of the Marathon was weak, making it difficult to guide it to the plexal segment. Even after switching this microcatheter to an SL-10, it was impossible to guide it beyond the plexal point (**Figs. 4D** and **6D**). Embolization was conducted at the plexal point, inducing asymptomatic infarction of the right lateral geniculate nucleus (**Fig. 4F**). The territory of AChA was affected, suggesting a complication related to NBCA infusion. On the other hand, in Cases 2 and 3, it was possible to guide a microcatheter to an area distal to the plexal point (**Figs. 2B, 3C, 6B,** and **6C**); therefore, the risk of embolization was considered to be low. Thus, it is important to recognize that embolization can be performed safely only at an area sufficiently distal to the plexal point as a rule. The plexal point should be identified through the assessment of microcatheter angiography findings of the AChA, and the allowable extent of occlusion must be determined. A microcatheter should be guided to a distal area at least beyond the plexal point. Concerning embolization in the cisternal segment, Hodes et al.[10] performed embolization at the orifice due to difficulty in selective catheterization, and reported the onset of AChA syndrome after embolization. Authors also conducted cisternal-segment embolization of a feeder branching from the origin of the AChA at the orifice in Case 1 (**Figs. 1B** and **1C**). Initially, a Marathon and SL-10 were used, but their pushability was weak and it was difficult to guide them. Even when adopting a PROWLER SELECT Plus, it was impossible to guide the microcatheter to the cisternal segment at a sufficient distance from the orifice (**Figs. 1D** and **6A**). It was difficult to confirm whether the vessel in which

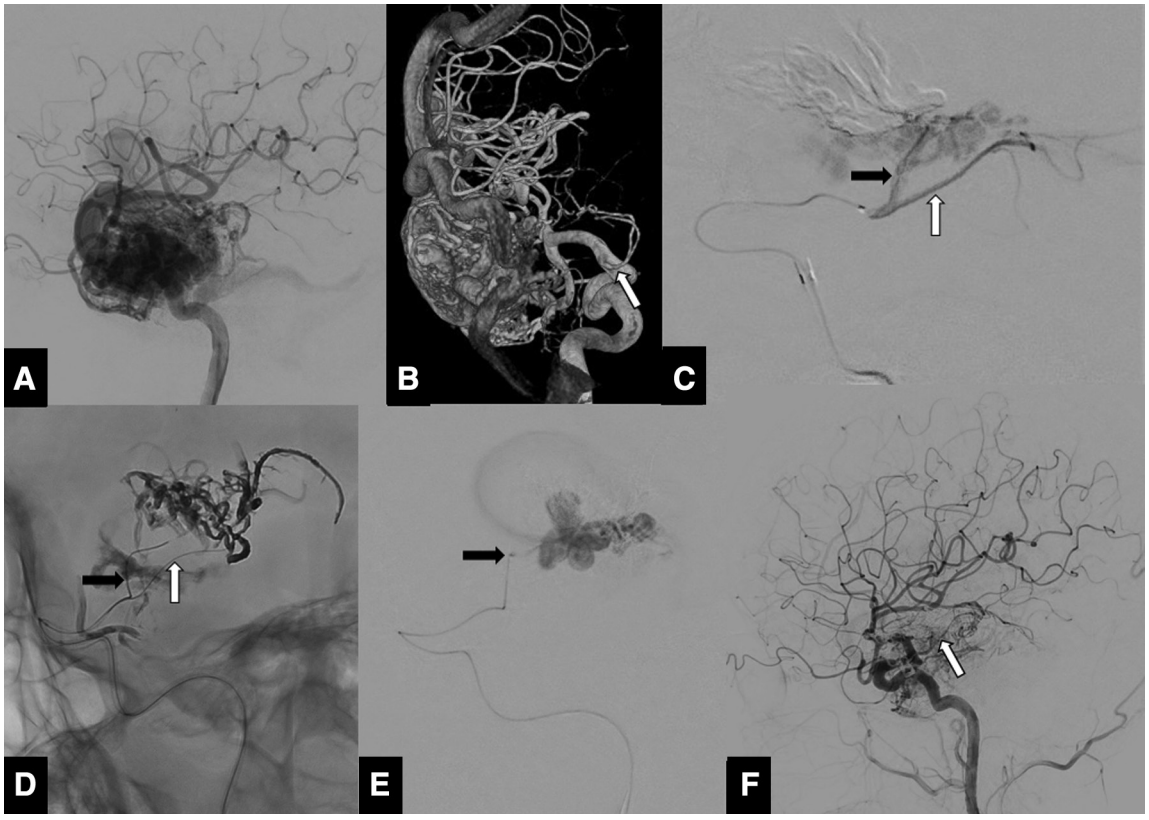

Fig. 5 (**A**) Lateral view on left internal carotid angiography before embolization. (**B**) 3D-DSA before embolization. A nidus was visualized through the left AChA (white arrow). The origin of left AChA was tortuous. (**C**) Microcatheter angiography before embolization (left AChA). A feeder (black arrow) had branched from the cisternal segment of the left AChA (white arrow). (**D**) The left AChA (white arrow) was straightened by a CHIKAI X 010 initially advanced, and it was possible to guide a microcatheter into a feeder (black arrow) by the second guidewire (the buddy wire technique). (**E**) Microcatheter angiography before embolization (left AChA). The nidus was visualized through a feeder branching from the left AChA. Microcatheter tip (black arrow). (**F**) Lateral view on left internal carotid angiography after embolization. Visualization of the nidus through the left AChA remained, but decreased blood flow was achieved (white arrows). AChA: Anterior choroidal artery

the microcatheter was placed branched from the AChA or directly from the internal carotid artery. However, as normal perfusion of the AChA was not visualized on angiography, authors considered that the microcatheter was placed in the correct feeder and infused NBCA (**Fig. 1D**). After embolization, asymptomatic minor infarction of the right caudate nucleus was observed (**Fig. 1F**). As this was not localized to the territory of AChA, an ischemic complication related to balloon use for guiding a microcatheter may have occurred, excluding the possibility of complication related to NBCA infusion. One previous study found no symptoms in 60%–74% of cases even when occluding the AChA at its origin,[19] but a collateral pathway should not be expected from the lateral posterior choroidal artery. It is important to recognize that such embolization has a relatively high risk and the indication for such embolization are limited. Furthermore, in some cases, such as Case 5, selective catheterization is difficult due to tortuosity of the origin of AChA and a feeder branching from the cisternal segment of this artery at a sharp angle (**Figs. 5B** and **5C**). In this case, the AChA was straightened by a CHIKAI X 010 initially advanced, and it was possible to guide a microcatheter into a feeder by the second guidewire using the buddy wire technique (**Figs. 5D, 5E**, and **6E**). Embolization of a feeder branching from the cisternal segment may be more risky than that from the plexal segment. Elkordy et al.[12] reported a similar case in whom infarction of the posterior limb of the internal capsule developed, inducing permanent hemiplegia. As its cause, they reviewed misinterpretation of a proper feeder on microcatheter angiography, but a microcatheter was unable to be guided to a distal area of the feeder; this may also have been a cause. Embolization of a feeder branching from the cisternal segment, as observed in Case 5, may be safely performed if a microcatheter can be guided to an area adjacent to the nidus. In Cases 1 and 5, cisternal-segment embolization was performed, considering the risks of

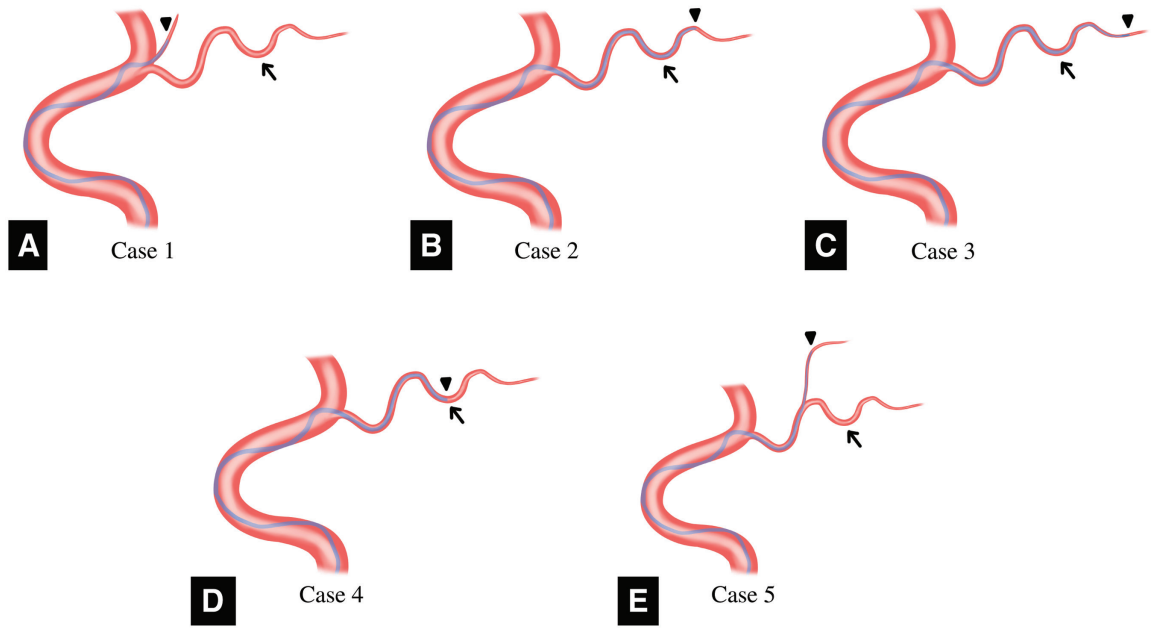

Fig. 6 The position of the microcatheter in all cases treated by presurgical embolization through the AChA is presented. Scheme of lateral views on angiography. The blue line indicates the position of the microcatheter. The arrows indicate the plexal point. The arrow heads indicate the position of the microcatheter tip. (**A**) Case 1. A microcatheter was unable to be guided beyond the cisternal segment. Microcatheter tip was positioned at a short distance from the orifice of the AChA. (**B**) Case 2. A microcatheter was guided to the plexal segment beyond the plexal point. (**C**) Case 3. A microcatheter was guided to the plexal segment beyond the plexal point. (**D**) Case 4. A microcatheter was unable to be guided beyond the plexal point (microcatheter tip was positioned at the plexal point). (**E**) Case 5. A microcatheter was guided to a feeder branching from the cisternal segment. AChA: Anterior choroidal artery
*The color version of this figure is available online, visit the website (https://www.jstage.jst.go.jp/browse/jnet/).

embolization-free surgery was higher than of NBCA infusion at the site. Reflux was minimized, differing from Onyx Liquid Embolic System (Medtronic, Minneapolis, MN, USA) (Onyx), and this may have played a role to reduce the serious complications.

Concerning the incidence of complications, one study reported no significant difference between Onyx and NBCA.[20] Authors only used NBCA as an embolic agent for perforators. If the use of Onyx in small-diameter blood vessels, such as the AChA, makes removal of microcatheter difficult through refluxed Onyx cast, the risk of ischemic or hemorrhagic complications may increase. For this reason, authors have not used Onyx for embolization of perforators. For AVM embolization, authors used 5% NBCA sufficiently heated. Heated ultra-low-concentration NBCA facilitated embolization of a nidus, and the rate of infusion was able to be controlled in the absence of difficulties in removal of microcatheter; this embolic agent can be relatively safely used. In Case 4, it was impossible to guide a microcatheter beyond the plexal point (microcatheter tip was positioned at the plexal point) (**Figs. 4D** and **6D**), and embolization with 20% NBCA was performed, prioritizing feeder occlusion. The concentration of NBCA can be changed on an individual-case basis. Infusion was completed when NBCA returned to the microcatheter tip and the microcatheter was removed. As Onyx reflux is more marked than NBCA reflux, a more proximal side of AChA is occluded. Therefore, it is important to minimize reflux using NBCA (not Onyx) after evaluating the allowable extent of occlusion.

The purpose of embolization through the AChA is not to completely occlude a nidus. Indeed, the AVM was not visualized on angiography through the AChA in three cases (**Figs. 1E, 3D,** and **4E**), whereas embolization was incomplete in two (**Figs. 2E** and **5F**). However, if the technical difficulty of surgical removal had been reduced, the purpose of presurgical embolization may have been achieved. Even if embolization is incomplete, the prevention of complications should be prioritized. Furthermore, embolized NBCA cast was able to be confirmed as a mark during surgical removal, which was useful for maintaining orientation. To secure a feeder involving the AChA in case of embolization-free surgery, the AChA must be traced from cisternal segment to plexal segment while extensively dissecting the brain and cranial nerves. Unless presurgical embolization is conducted, the extent

of craniotomy may be extended, the operative time may be prolonged, and the volume of intraoperative blood loss may increase. In addition, if detachment and hemostasis of the AChA are insufficient in case of embolization-free surgery, the risk of postoperative hemorrhage may increase. However, if the AChA is secured in the cisternal segment, the risk of AChA syndrome may develop. Therefore, when the risk of presurgical-embolization-free surgery/gamma knife radiosurgery exceeds that of embolization through the AChA, the latter may be considered as a treatment option. If ruptured intranidal or feeder aneurysms are present, as demonstrated in Case 3, in which hemorrhage developed, embolization through the AChA may be a good option.

Conclusion

In this series, there was no morbidity and embolization was able to be relatively safely performed. This study suggested the efficacy of embolization through the AChA, but whether this procedure should be indicated must be carefully considered on an individual-case basis, recognizing that its risk is high.

Disclosure Statement

We declare no conflict of interest.

References

1) Crowley RW, Ducruet AF, McDougall CG, et al: Endovascular advances for brain arteriovenous malformations. *Neurosurgery* 2014; 74 Suppl 1: S74–82.
2) Kurita H, Kikkawa Y, Ikeda T, et al: Current treatment strategies and neurosurgical practice for cerebral AVMs. *Jpn J Neurosurg* 2016; 25: 33–41. (in Japanese)
3) Ishii A, Miyamoto S: Multimodal therapy for cerebral arteriovenous malformation: the role of embolization. *Jpn J Neurosurg* 2015; 24: 180–188. (in Japanese)
4) Hartmann A, Stapf C, Hofmeister C, et al: Determinants of neurological outcome after surgery for brain arteriovenous malformation. *Stroke* 2000; 31: 2361–2364.
5) Kondo R, Matsumoto Y, Endo H, et al: Endovascular embolization of cerebral arteriovenous malformations: results of the Japanese Registry of Neuroendovascular Therapy (JR-NET) 1 and 2. *Neurol Med Chir* 2014; 54 Suppl 2: 54–62.
6) Baharvahdat H, Blanc R, Termechi R, et al: Hemorrhagic complications after endovascular treatment of cerebral arteriovenous malformations. *AJNR Am J Neuroradiol* 2014; 35: 978–983.
7) van Beijnum J, van der Worp HB, Buis DR, et al: Treatment of brain arteriovenous malformations: a systematic review and meta-analysis. *JAMA* 2011; 306: 2011–2019.
8) Torné R, Rodríguez-Hernández A, Lawton MT: Intraoperative arteriovenous malformation rupture: causes, management techniques, outcomes, and the effect of neurosurgeon experience. *Neurosurg Focus* 2014; 37: E12.
9) Du R, Keyoung HM, Dowd CF, et al: The effects of diffuseness and deep perforating artery supply on outcomes after microsurgical resection of brain arteriovenous malformations. *Neurosurgery* 2007; 60: 638–646; discussion 646–648.
10) Hodes JE, Aymard A, Casasco A, et al: Embolization of arteriovenous malformations of the temporal lobe via the anterior choroidal artery. *AJNR Am J Neuroradiol* 1991; 12: 775–780.
11) Dowd CF, Halbach VV, Barnwell SL, et al: Particulate embolization of the anterior choroidal artery in the treatment of cerebral arteriovenous malformations. *AJNR Am J Neuroradiol* 1991; 12: 1055–1061.
12) Elkordy A, Endo H, Sato K, et al: Embolization of the choroidal artery in the treatment of cerebral arteriovenous malformations. *J Neurosurg* 2017; 126: 1114–1122.
13) Lv X, Hu X, Li W, et al: Curative and adjunctive AVM Onyx embolization of AVMs through the choroidal arteries. *Interv Neuroradiol* 2017; 23: 392–398.
14) Friedman JA, Pichelmann MA, Piepgras DG, et al: Ischemic complications of surgery for anterior choroidal artery aneurysms. *J Neurosurg* 2001; 94: 565–572.
15) Takahashi S, Suga T, Kawata Y, et al: Anterior choroidal artery: angiographic analysis of variations and anomalies. *AJNR Am J Neuroradiol* 1990; 11: 719–729.
16) Morandi X, Brassier G, Darnault P, et al: Microsurgical anatomy of the anterior choroidal artery. *Surg Radiol Anat* 1996; 18: 275–280.
17) Fernández-Miranda JC, de Oliveira E, Rubino PA, et al: Microvascular anatomy of the medial temporal region: part 1: its application to arteriovenous malformation surgery. *Neurosurgery* 2010; 67 (3 Suppl Operative): ons237–276.
18) Fujii K, Lenkey C, Rhoton AL: Microsurgical anatomy of the choroidal arteries: lateral and third ventricles. *J Neurosurg* 1980; 52: 165–188.
19) Rand RW, Brown WJ, Stern WE: Surgical occlusion of anterior choroidal arteries in parkinsonism; clinical and neuropathologic findings. *Neurology* 1956; 6: 390–401.
20) Crowley RW, Ducruet AF, Kalani MY, et al: Neurological morbidity and mortality associated with the endovascular treatment of cerebral arteriovenous malformations before and during the Onyx era. *J Neurosurg* 2015; 122: 1492–1497.