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Delayed Occlusion of the Anterior Choroidal Artery Following Flow Diverter Stent Deployment for Unruptured Aneurysm: A Case Report and Literature Review

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Abstract

Flow diverter stent has been a promising device for intracranial aneurysm treatment. For treating aneurysms located in the anterior circulation, critical branches may be covered by flow diverter stent. The occlusion incidence of these branches has been reported, and even if branch vessel occlusions occur, associated neurological deficits are extremely rare. We present a 55-year-old woman who had a large saccular aneurysm at the right internal carotid artery (ICA). A developed fetal-type posterior communicating artery (PCOM) originated from the sac. We administered flow diverter stent deployment with coil insertion following surgical anastomosis of the superficial temporal artery to the posterior cerebral artery (STA-PCA) with ligation of the origin of the PCOM. On the seventh morning following the intervention, ischemic complication developed due to anterior choroidal artery occlusion jailed by the flow diverter stent. The occlusion of anterior choroidal artery covered by flow diverter stent is extremely rare. However, if the branch arises from the aneurysm sac, occlusion can transpire and induce serious complication. The most probable cause of occlusion in this case was that the orifice was jailed apart from the stent strut because the branch originated from the sac rather than the neck. Furthermore, the progression rate of intra-aneurysm thrombus formation is also an important factor affecting the side branch occlusion.

Keywords: flow diverter stent, anterior choroidal artery, side branch arising from aneurysm sac

Introduction

Flow diverter stent has been a promising device for treating intracranial aneurysms. Numerous reports provide evidence of the safety, long-term durability, and good clinical results of this treatment for cerebral aneurysms, which have been difficult to cure with direct surgery or classical coil embolization.^{1–4} For treating aneurysms located in the anterior circulation,

critical branches, such as the lenticulostriate artery, anterior choroidal artery, posterior communicating artery (PCOM), and ophthalmic artery, may be covered by flow diverter stents. As the flow diverter reduces blood flow into the aneurysm, thrombosis of the aneurysm and endothelialization of the ostium could occur. However, covered branches could also become occluded due to flow stagnation and thrombosis, which might lead to ischemic complications. The incidence of occlusion has been reported for each branch. Even if branch vessel occlusion occurs, it is seldom associated with neurological deficits due to the development of collateral circulations.^{5–10)} Regarding the anterior choroidal artery, an extremely low occlusion rate was reported.^{11–16)}

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In this study, we described a rare case of delayed anterior choroidal artery occlusion which led to critical neurological deficit following the deployment of a flow diverter stent for a large internal carotid artery aneurysm.

Case Report

A 55-year-old woman presented with headache. On magnetic resonance imaging (MRI), a large saccular aneurysm located at the bifurcation of the right internal carotid artery (ICA) and PCOM was confirmed. Thereafter, the patient underwent cerebral digital subtraction angiography. The maximum aneurysm size and neck size were 20.4 mm and 17.6 mm, respectively. A well-developed fetal-type PCOM originated from the aneurysm sac (Fig. 1A–1C). The proximal portion of the posterior cerebral artery (P1 segment) was absent under vertebral arteriography. Moreover, the anterior communicating artery (ACOM) was not seen under the Matas compression test.

This aneurysm was treated by deployment of a flow diverter stent with coil insertion following surgical anastomosis of the superficial temporal artery to the posterior cerebral artery (STA-PCA anastomosis) with ligation of the origin of the PCOM. It was assumed that the aneurysm would be difficult to be cured only with a flow diverter, since continuous flow of the PCOM originating from the sac would disturb thrombotic occlusion. Therefore, we intended PCOM ligation antecedent to flow diverter deployment. Coil insertion was performed to prevent delayed aneurysm rupture and not for aneurysm occlusion.

Though flow diverter stent can only be used for large aneurysms of the ICA located proximal to PCOM in Japan, conventional stent-assisted coil embolization and trapping with bypass treatment could not be adopted for this aneurysm. Very high recurrence rate was expected after conventional coil embolization with neck bridge stent assistance. Furthermore, inadequate interventional treatment was likely to cause aneurysm enlargement. On the other hand, ICA and PCOM trapping with bypass surgery was considered to have a very high risk of ischemic complication because the anterior choroidal artery originated from the aneurysmal sac. We considered this strategy involving flow diverter stent was the most reasonable with preservation of anterograde flow of the ICA. The patient was informed of these treatment options and the details, as well as off-label use of flow diverter stent, and then, informed consent was obtained.

First, STA-PCA anastomosis was performed with temporal craniotomy. The STA parietal branch was exposed and anastomosed to the proximal P2, and then, the PCOM was ligated with a surgical clip precisely at its orifice. Postprocedural MRI revealed acute infarction in a portion of the territory of the posterior cerebral artery and the territory of the tuberothalamic artery (a perforator of PCOM). The patency of bypass flow was obvious by magnetic resonance angiography. The patient presented with mild left-sided hemiplegia postoperatively but completely recovered after 1 month of rehabilitation.

Endovascular treatment was scheduled 3 months following the initial bypass surgery. Dual antiplatelet therapy (aspirin 100 mg + clopidogrel 75 mg) was started 21 days before the endovascular treatment. We performed platelet function testing the day prior to the procedure. The VerifyNow aspirin and P2Y12 assays were utilized to calculate the aspirin reaction unit (ARU) and P2Y12 reaction unit (PRU). The calculated ARU was 381 and PRU was 202. Because aspirin resistance was defined as ARU >550, while clopidogrel resistance was identified as PRU >230 in our institute, the test results were considered to be within the optimal range.

Endovascular treatment was performed under general anesthesia. An initial intravenous bolus of heparin (5000 U) was administered, and an activated clotting time of 250-300 seconds was maintained by an additional heparin administration throughout the procedure. First, we confirmed the patient's bypass patency (Fig. 1D). After the navigation of guiding systems to the right ICA, an optimal working angle was set (Fig. 1E). Thereafter, the Marksman (Medtronic, Minneapolis, MN, USA) was navigated to the middle cerebral artery over the aneurysm neck. Another guiding system was also navigated to the right ICA. From this guiding system, Headway17 (MicroVention, Aliso Viejo, CA, USA), for the purpose of coil insertion, was navigated into the aneurysm prior to the deployment of flow diverter. Pipeline Flex (diameter, 4.5 mm \times length, 35 mm) (Medtronic, Minneapolis, MN, USA) was deployed from the proximal M1 to ICA C3 segment. High-resolution cone beam computed tomography (CT) was performed by injecting a threefold diluted contrast medium. The obtained reconstruction image of the maximum intensity projection (MIP) demonstrated good stent apposition (Fig. 1F). However, as illustrated by an oblique slice of the image, the anterior choroidal artery originated from the distal aneurysm neck and did not have contact with the flow diverter stent (Fig. 1G).

Subsequently, coils were inserted from the jailed microcatheter. The coil insertion was completed when the volume packing density reached about 10%, since the coil insertion was not intended for aneurysm occlusion but for the prevention of postprocedural delayed aneurysm rupture (Fig. 1H). The coils were



Fig. 1 (A–C) Right-sided internal carotid arteriogram shows a large aneurysm located at the bifurcation of the ICA and a well-developed PCOM. Lateral view, working view, and volume-rendered image of a three-dimensional angiography, respectively. The arrowheads in C indicate the anterior choroidal artery. (D) Right-sided external carotid angiogram illustrates anastomotic flow to the posterior cerebral artery from the STA. (E) Right-sided internal angiogram, lateral working view, shows the large aneurysm. PCOM from the sac is completely ligated. (F, G) MIP image obtained by high-resolution cone beam CT with diluted contrast medium shows good apposition of the flow diverter stent. The origin of the anterior choroidal artery (arrowhead) does not make contact with the stent. The arrows indicate the tip of the microcatheter (Headway 17) for coil insertion. (H) Postprocedural internal carotid arteriogram shows body filling of contrast medium to the aneurysm sac. (I) Axial slice of diffusion-weighted image obtained after intervention shows no apparent ischemic lesions. CT: computed tomography, ICA: internal carotid artery, MIP: maximum intensity projection, PCOM: posterior communicating artery, STA: superficial temporal artery.



Fig. 2 (A) Axial slice of diffusion-weighted image obtained on the seventh morning after intervention shows an acute ischemic lesion in the posterior limb of the internal capsule. (B, C) Right-sided internal carotid arteriogram, lateral view and antero-posterior view, shows complete occlusion of the aneurysm and occlusion of the anterior choroidal artery. (D) Internal carotid arteriogram after intraarterial injection of urokinase, antero-posterior view, shows recanalized anterior choroidal artery (arrowhead). (E) Axial images of CT 2 hours after procedure of intraarterial injection of urokinase show subarachnoid and intra-parenchymal hemorrhage in the frontal and parietal lobes accompanied by ventricular perforation. CT: computed tomography.

carefully inserted so that loops did not occlude the orifice of the anterior choroidal artery. The final angiogram revealed no branch occlusion, including the anterior choroidal artery, nor distal embolism. The patient came out from the anesthesia without new neurological deficits. Postprocedural MRI demonstrated no apparent ischemic lesions (Fig. 1I). General heparinization was continued to prevent delayed stent thrombosis for 24 hours after intervention.

Although postprocedural course was uneventful for 6 days after treatment, on the seventh morning, she suffered from left-sided hemiparesis, hemianopsia, and dysarthria. Emergent MRI revealed high signal lesion on diffusion-weighted imaging in the right posterior limb of the internal capsule (Fig. 2A). We suspected occlusion of the anterior choroidal artery; therefore, we performed an emergent digital subtraction angiography. The right internal carotid arteriogram demonstrated occlusion of the anterior choroidal artery that originated from the aneurysm sac accompanied by complete occlusion of the aneurysm (O'Kelly-Marotta (OKM) grading scale D)¹⁷⁾ (Figs. 2B and 2C). We decided to perform recanalization via intraarterial injection of urokinase. Subsequently, after general heparinization, microcatheter was navigated near the origin of the occluded anterior choroidal artery. Thereafter, 240000 units of urokinase were slowly injected through the microcatheter for about 20 minutes. Recanalization of the anterior choroidal artery was achieved (Fig. 2D). However, after the procedure, her consciousness and neurological symptoms rapidly deteriorated. CT just after the procedure revealed multiple subarachnoid hemorrhages in the frontal and parietal lobes. The size of the hematomas was enlarged and was accompanied by an intracerebral hemorrhage, as exhibited by repeated CT (Fig. 2E). Hematoma removal, external decompression, and external ventricular drainage were needed to relieve the mass effect. Subsequently, ventriculoperitoneal shunt was performed for communicating hydrocephalus 3 months after her hemorrhagic event. Although her consciousness gradually improved, left-sided severe hemiparesis, unilateral spatial neglect, and higher brain dysfunction remained. Final neurological status at the time of transfer was a score of 4 on the modified Rankin Scale.

Discussion

We have experienced a rare case, where an occlusion of the anterior choroidal artery jailed by flow diverter stent occurred, and the branch occlusion led to serious ischemic complication. During the treatment of aneurysms located on Willis circle in the anterior circulation with flow diverter stents, branches are inevitably covered with the stent. Occlusion rate of covered branches was reported in several studies. Table 1 demonstrates a summary of literature discussing branch occlusion after being covered by flow diverters in aneurysm treatment located on the Willis circle in the anterior circulation.^{6–16,18–26)} Branch occlusion is most common in the anterior cerebral artery, followed by the PCOM and the ophthalmic artery. Although the occlusion rate for these branches varied in each report, symptoms associated with branch occlusion were rarely encountered. Only one case was reported where a delayed occlusion of the ophthalmic artery led to permanent monocular blindness,²²⁾ and three transiently symptomatic cases were reported.^{19,21)} Other cases of branch occlusion were clinically silent. As for the anterior choroidal artery, occlusion rate was extremely low. In previous reports, we discovered three cases of delayed anterior choroidal artery occlusion. Two of these occlusion cases were asymptomatic,^{11,14)} and only one case, reported by Raz E et al., was symptomatic.¹⁶) In all the cases, the territory of the anterior choroidal artery was reconstituted by collateral flow, such as the posterior choroidal artery, the medial lenticulostriate artery, and the posterior cerebral artery. Furthermore, in the symptomatic case, ischemic symptom was transient. Cagnazzo et al.⁵⁾ reported a meta-analysis on the patency of the branches of the supra-clinoid ICA. The incidence of ophthalmic artery occlusion and associated symptoms were 5.9% and 0.8%, respectively. The occlusion rate of the PCOM was 20.7%, although all cases were asymptomatic. On the other hand, the anterior choroidal artery was occluded in only 1% of cases, with 1% displaying transient neurological symptoms. When discussing the possibility of side branch occlusion, multiple factors should be considered. Particularly, a balance of flow demand to the eloquent area and collateral circulation, including potential pathways, may be associated with the fate of the patency of the side branch. Since these conditions vary for each branch, it is presumed that the occlusion rate is different. The occlusion rate of branches with abundant collateral flow, such as the anterior cerebral artery, PCOM, and ophthalmic artery, is relatively high, while the occlusion rate of the anterior choroidal artery with scarce collateral flow is commonly low.

However, in the case of our patient, the anterior choroidal artery arising from the aneurysm sac was occluded, which led to serious condition. The probable cause of occlusion was that the orifice was not in contact with the deployed flow diverter stent because the branch originated from the sac rather than the neck. There is limited data on whether branches arising from the sac can be preserved after flow diverter deployment. We infer that the rate of progression of intra-aneurysm thrombus formation following the flow diverter deployment, in addition to potential collateral circulation and flow demand of a branch, is an important factor affecting side branch occlusion. Figure 3 illustrates the schematic images of the assumed course depending on the rate of thrombosis. In case of slow progression of thrombus formation, even if an aneurysm becomes completely thrombosed and finally occluded, a side branch route remains in the aneurysm, and anterograde blood flow is preserved due to flow demand (Fig. 3A). Even in a branch with relatively poor potential collateral network, such as the anterior choroidal artery, the patency of the branch can be preserved through this mechanism. In case of moderate progression of thrombus formation, if there is sufficient time for developing collateral network and, if the perfusion territory of the branch has sufficient collateral network, side branch occlusion may be free from ischemic complication (Fig. 3B). For a branch with abundant potential collateral network, such as ophthalmic artery, delayed branch occlusion is less likely to cause ischemic complication. In case of rapid progression of thrombus formation, side branch occlusion can occur without sufficient development of collateral flow and leads to ischemic complication (Fig. 3C). In our patient, anterior choroidal artery occlusion seemed to occur because complete occlusion of the aneurysm was observed within only 7 days after treatment. Dual antiplatelet therapy was appropriately prescribed, and the platelet functioning test calculated by VerifyNow was within the optimal range. Furthermore, general heparinization was continued for 24

| | Ophthalmic artery | | Posterior communicating artery | | Anterior choroidal artery | | Anterior cerebral artery | |
|--------------------------------------|----------------------|------------------|-----------------------------------|-----------------|------------------------------|------------------|-----------------------------|-----------------|
| | Occlusion rate | Symptom rate | Occlusion rate | Symptom rate | Occlusion rate | Symptom rate | Occlusion rate | Symptom rate |
| Wu X et al. 2019 | 12/109 (11.00) | 0/12 (0.00) | 4/24 (16.67) | 0/4 (0.00) | 0/7 (0.00) | 0/0 (0.00) | 7/11 (63.64) | 0/7 (0.00) |
| Bhogal P et al. 2017 | 7/133 (5.26) | 0/7 (0.00) | 20/47 (42.55) | 0/20 (0.00) | 0/91 (0.00) | 0/0 (0.00) | 2/14 (14.29) | 0/2 (0.00) |
| Rangel- Castilla L et al. 2017 | 8/76 (10.53) | 0/8 (0.00) | 3/28 (10.71) | 0/3 (0.00) | 0/21 (0.00) | 0/0 (0.00) | 2/2 (100.00) | 0/2 (0.00) |
| Gascou G et al. 2015 | 2/34 (5.88) | 0/2 (0.00) | 0/2 (0.00) | 0/0 (0.00) | 0/12 (0.00) | 0/0 (0.00) | 0/5 (0.00) | 0/0 (0.00) |
| Vedantam A et al. 2015 | 2/49 (4.08) | 0/2 (0.00) | 1/14 (7.14) | 0/1 (0.00) | 0/11 (0.00) | 0/0 (0.00) | _ | - |
| Burrows AM et al. 2016 | 8/37 (21.62) | 0/8 (0.00) | - | - | - | - | _ | - |
| Griessenauer CJ et al. 2016 | 2/63 (3.17) | 2*/2 (100.00) | - | - | - | - | _ | - |
| Durst CR et al. 2016 | 0/26 (0.00) | 0/0 (0.00) | _ | - | - | _ | _ | - |
| Rouchard A et al. 2015 | 1/28 (3.57) | 1*/1 (100.00) | _ | - | - | _ | _ | - |
| Chalouhi N et al. 2015 | 6/91 (6.59) | 1/6 (16.67) | _ | - | - | _ | _ | - |
| Puffer R et al. 2012 | 5/20 (25.00) | 0/5 (0.00) | - | - | - | _ | _ | - |
| Daou B et al. 2017 | - | - | 16/30 (53.33) | 0/16 (0.00) | - | - | _ | - |
| de Carvalho et al. 2017 | - | - | 5/18 (27.78) | 0/5 (0.00) | - | - | _ | - |
| Brinjikji W et al. 2014 | - | _ | 3/11 (27.27) | 0/0 (0.00) | - | - | _ | - |
| Fujii T et al. 2019 | - | _ | - | - | 1/21 (4.76) | 0/1 (0.00) | _ | - |
| Bhogal P et al. 2018 | - | _ | - | - | 0/30 (0.00) | 0/0 (0.00) | _ | - |
| Srinivasan VM et al. 2018 | - | - | - | _ | 0/18 (0.00) | 0/0 (0.00) | - | - |
| Raz E et al. 2015 | - | - | - | - | 1/29 (3.45) | 1*/1 (100.00) | _ | - |
| Neki H et al. 2015 | _ | - | _ | - | 0/20 (0.00) | 0/0 (0.00) | _ | - |
| Brinjikji W et al. 2015 | - | _ | - | - | 1/15 (6.67) | 0/1 (0.00) | _ | - |

Table 1Literature summary discussing branch occlusion following flow diverter treatment in Willis circle in theanterior circulation

*Transiently symptomatic case.



Fig. 3 The schematic images of the assumed course depending on the rate of thrombosis for the cases with a side branch arising from the aneurysm sac. (A) In case of slow progression of thrombosis, the side branch can be preserved with a remaining route in the aneurysm sac. (B) In case of moderate progression of thrombosis, occlusion of the side branch may not lead to ischemia of perfusion territory if the collateral network is sufficient. (C) In case of rapid progression of thrombosis without sufficient time for the development of collateral network, occlusion of side branch can lead to ischemia of perfusion territory.

hours after intervention. Therefore, the exact cause of such rapid aneurysm thrombosis was unclear. It may have been affected by coil insertion for the prevention of delayed rupture. Due to the difficulty in prediction of the rate of thrombus formation after treatment, we should confirm that the orifice of the side branch contacted the flow diverter stent without any space, especially

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for branches which have poor collateral network, such as the anterior choroidal artery. If possible, balloon angioplasty should be performed to avoid this. A prolonged systemic heparinization might be effective to slow the progression of thrombus formation. Moreover, if the risk of critical branch occlusion is assumed to be high when considering morphological features between aneurysms, the course of the ICA, and the orifice of the side branch to be protected, it is necessary to consider other treatment options. As for the branches which can be jailed by a flow diverter stent without any space between the origin and the stent strut, the thrombosis of aneurysm after flow diverter deployment itself does not influence the occlusion of jailed branch.

The hemorrhagic complication which subsequently occurred following anterior choroidal artery occlusion worsened her prognosis. After rescue therapy of the intra-arterial injection of urokinase, final angiogram demonstrated slight extravasation in peripheral middle cerebral arteries. The exact cause of this hemorrhagic complication was unclear, but was considered to be associated with the combination effect of dual antiplatelet treatment, intra-arterial injection of urokinase, and general heparinization during rescue therapy.

Conclusion

Occlusion of the anterior choroidal artery covered by flow diverter stent is extremely rare. However, if the branch is arising from the aneurysm sac, occlusion can occur and induce serious complication. The most probable cause of occlusion was that the orifice did not have contact with the deployed flow diverter stent due to the origin of the branch, which was from the sac rather than the neck. Moreover, the balance between the rate of progression of intra-aneurysm thrombus formation after flow diverter deployment and development of collateral circulation could be significant factors affecting the emergence of ischemic complication.

Conflicts of Interest Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study. No source of financial or material support was received.

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