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Case Report

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Ruptured Proximal Anterior Cerebral Artery Aneurysm Treated with Flow Diverter

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ABSTRACT

AIM: To discuss the use of flow modulation in treating ruptured aneurysms of the proximal segment of the anterior cerebral artery (A1 aneurysms). A1 aneurysms are rare, constituting approximately 1% of all intracranial aneurysms.

CASE REPORT: We report a left A1 aneurysm with a wide neck and small sac (3 × 1.8 mm). In order to treat the lesion, a flow diverter (4 × 12–18 mm, FRED, Microvention) was placed from M1 to the proximal end of the paraophthalmic internal carotid artery, without directly covering the neck of the aneurysm. No procedural bleeding occurred. During stent deployment, abciximab was infused. A day after the procedure, double antiplatelet therapy was initiated for 1 month, followed by single antiplatelet therapy for another 3 months.

Due to the aneurysm morphology, we opted for a competitive flow diversion, covering the parent artery origin and leaving the A1A neck uncovered. A decreased flow into the aneurysmal parent artery gradually promoted aneurysm sac thrombosis. Both digital subtraction angiography at a 12-month follow-up and computed tomography angiography 24-month follow-up confirmed the regular patency of the stent and resolution of the aneurysm. In addition, the competitive modulation of flow in the ipsilateral anterior cerebral artery results in the narrowing of the vessel.

CONCLUSION: A1 aneurysm endovascular treatment is often challenging. Coiling or assisted coiling is the most frequently employed. Although flow diverter stent (FDS) is a consolidated technique for treating ruptured intracranial blister-like and dissecting aneurysms, its role in treating intracranial saccular ruptured aneurysms has to be elucidated. However, more number of case studies is needed to confirm the efficacy and safety of an FDS in treating ruptured A1 aneurysms.

KEYWORDS: Endovascular, Flow diverter, A1 Aneurysm, Ruptured aneurysm

ABBREVIATIONS: A1As: Anterior cerebral artery aneurysms, **ACA:** Anterior cerebral artery, **ASA:** Acetylsalicylic acid, **CT:** Computed tomography, **CTA:** CT angiography; **DSA:** Digital subtraction angiography, **EVD:** External ventricular drain, **FDS:** Flow diverter stent, **GCS:** Glasgow coma score, **ICA:** Internal carotid artery, **MCA:** Middle cerebral artery, **MLA:** Medial lenticulostriate arteries, **mRs:** modified Rankin scale, **SAH:** Subarachnoid hemorrhage, **WFNS:** World Federation of Neurosurgical Society

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INTRODUCTION

Unlike the anterior communicating artery aneurysms (13,14), proximal anterior cerebral artery aneurysms (A1As) are rare vascular malformations, representing approximately 1% of all intracranial aneurysms (15). The incidence of A1As is between 0.9% (15), and 1.7% (10), as reported in the largest published series. The Dashti series reported that A1As represent 0.5% of all intracranial aneurysms and 2% of all the anterior cerebral artery (ACA) aneurysms (5). A1As are more unstable as compared to other intracranial aneurysms (5). Due to the morphological and structural characteristics of A1As, both microsurgical and endovascular treatments become challenging. In the literature, different types of treatment have been proposed, although a common opinion regarding the treatment of A1As among authors is still lacking.

In this study, we present a complex case of tiny ruptured A1A in the internal carotid artery bifurcation zone treated with a flow diverter stent (FDS) released from the middle cerebral artery (MCA) to the paraophthalmic internal cerebral artery (ICA).

CASE REPORT

History and Examination

A 60-year-old woman was admitted to our hospital due to a sudden and rapidly worsening headache associated with nausea and vomiting. An initial cerebral computed tomography (CT) scan revealed a Fisher scale grade 3 subarachnoid hemorrhage (SAH) (Figure 1). Subsequent CT angiography (CTA) highlighted the presence of a tiny millimetric A1A (Figure 1) with a wide neck. Two other small aneurysms in the posterior communicating artery (2.5 mm maximum diameter) and in the left ICA in the cavernous segment (5 mm maximum diameter) were discovered. Successive digital subtraction angiography (DSA) confirmed the presence of the ruptured left A1A directed upward and backward, with a 3-mm neck, 1.8-mm maximum length, and an irregular morphology inclusive of a baby aneurysm in its cranial portion (Figure 2). After a multidisciplinary assessment, we opted for an endovascular treatment. Due to a moderate World Federation of Neurosurgical Societies grade (II) and Fisher scale grade 3, the risk of hydrocephalus was estimated to be moderate. No external ventricular drain (EVD) was placed before the procedure.

Endovascular Procedure

Under general anesthesia, a guide catheter from the right femoral access through a triaxial system (7F Envoy, Codman Neurovascular, Raynham, MA, USA) was placed in the proximal ICA and an intracranial support catheter (Navien 5F, ev3, Irvine, CA) was placed in the cavernous segment of the ICA. A microcatheter (Headway 27, 0.027 inch, Microvention

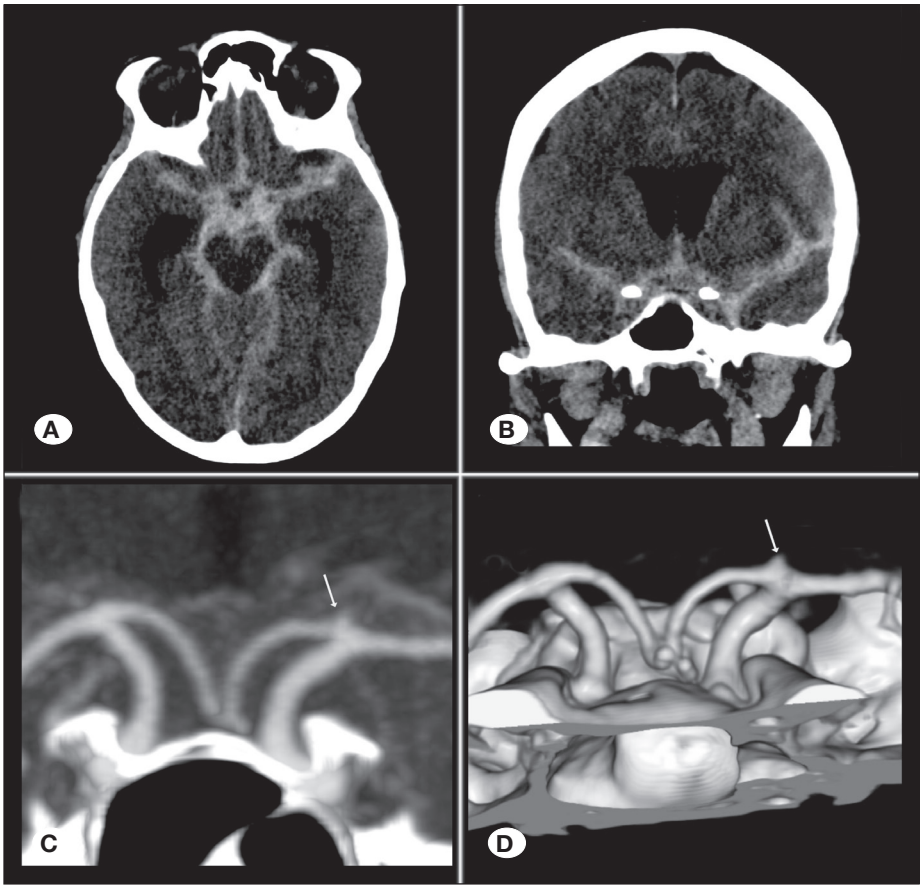

Figure 1: Initial non-enhanced CT, axial **(A)**, and coronal **(B)** views, showing the presence of a diffuse subarachnoid hemorrhage in the basal cisterns and bilateral Sylvian fissures, Fisher scale III. A subsequent CT angiogram, MIP view **(C)**, and 3D VR **(D)**, showing a tiny and irregular ruptured A1 aneurysm (white arrows).

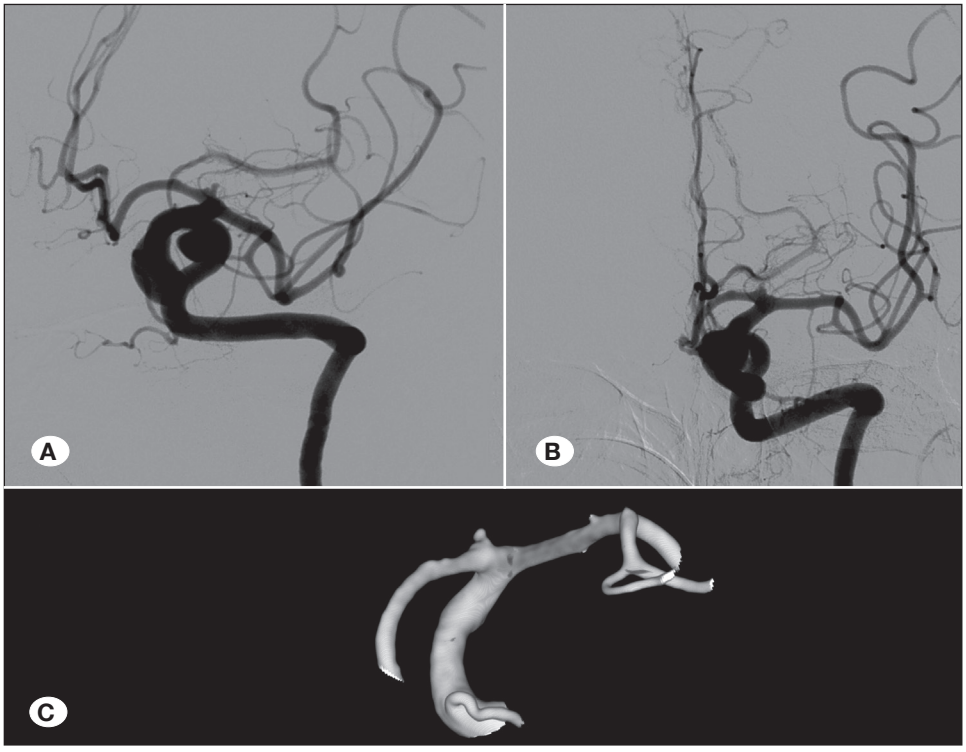

Figure 2: Pretreatment DSA showing the presence of a tiny A1 aneurysm **(A,B)**. **C)** A 3D acquisition highlighting the irregular morphology of the A1 aneurysm.

Terumo, USA) and a microguidewire Synchro 0.014" (Stryker Neurovascular, Fremont, CA), an FDS, were placed (4 × 12–18 mm, FRED®, Microvention) from M1 to the proximal end of the paraophthalmic ICA (Figure 3).

During stent deployment, 8 mg of abciximab (ReoPro) was infused intravenously, with a subsequent infusion of 4.5 ml abciximab in 250 ml of saline solution in 12 hours (17 ml/h). No further intraprocedural bleeding occurred, as confirmed by a post-procedural CT.

Postoperative Course and Follow-up

A double antiplatelet therapy with 75 mg clopidogrel and 100 mg acetylsalicylic acid (ASA) was initiated a day after the procedure and maintained for 1 month. A single antiplatelet therapy with 100 mg ASA was maintained for another 3 months. Due to the emergence of hydrocephalus on day 3, an EVD was placed. On day 20, the patient developed vasospasm of the right MCA and right carotid siphon. Distal superselective infusion of 2.5 mg and 1 mg of nimodipine in the right and left ICAs, respectively, resulted in a complete resolution of vasospasm. On day 33, a ventriculoperitoneal shunt was placed because of a persistent hydrocephalus. Discharge modified Rankin score (mRs) was 0.

DSA at a 12-month follow-up and CTA at a 24-month follow-up (Figure 3 and Figure 4) confirmed the regular patency of the FDS and resolution of the aneurysm.

DISCUSSION

A1As represent a small percentage of all intracranial aneurysms. Surgical clipping is the first choice for the treatment of A1As (5); however, it is often challenging due to the unfavorable location of A1As. In particular, A1As are often projected upward or backward, probably due to the origin of perforating arteries that usually arise from the superior and/or posterior wall of the ACA (5,7). Thus, A1As are often behind the surgical field, making the exposure and/or dissection of the aneurysm and perforating vessels difficult (7). Commonly, the neck or dome of the aneurysm is adherent to the perforating arteries, making the surgical treatment even more difficult (7). The perforating arteries of the precommunicating ACA segment (A1) are divided into two subgroups (5). The first one includes the medial lenticulostriate arteries (MLAs), which originate from the proximal half of the A1 trunk. The septum pellucidum, anterior commissure, pillars of the fornix, anterior limb of the internal capsule are supplied by the MLAs (5). Also, the pallidum, striatum, posterior paraolfactory gyrus, and anterior hypothalamus are perfused by the MLAs (5). In addition, a smaller group of perforating arteries sprouting distally from the MLA vascularize the optic nerves and tracts and chiasm (5). The second subgroup corresponds to the recurrent artery of Heubner, which arises from the first portion of the postcommunicating ACA segment (A2). It perfuses important white matter structures, such as the striatum and anterior limb of the internal capsule, and other structures, such as the olfactory region, anterior hypothalamus, and frontobasal cortex (5).

In our case, due to the A1 anatomy- and A1A morphology-related difficulties, the surgical approach was deemed not adequate for the patient. Moreover, a high risk of brain damage during vessel manipulation and an elevated risk of perforator occlusion reinforced the idea of selecting

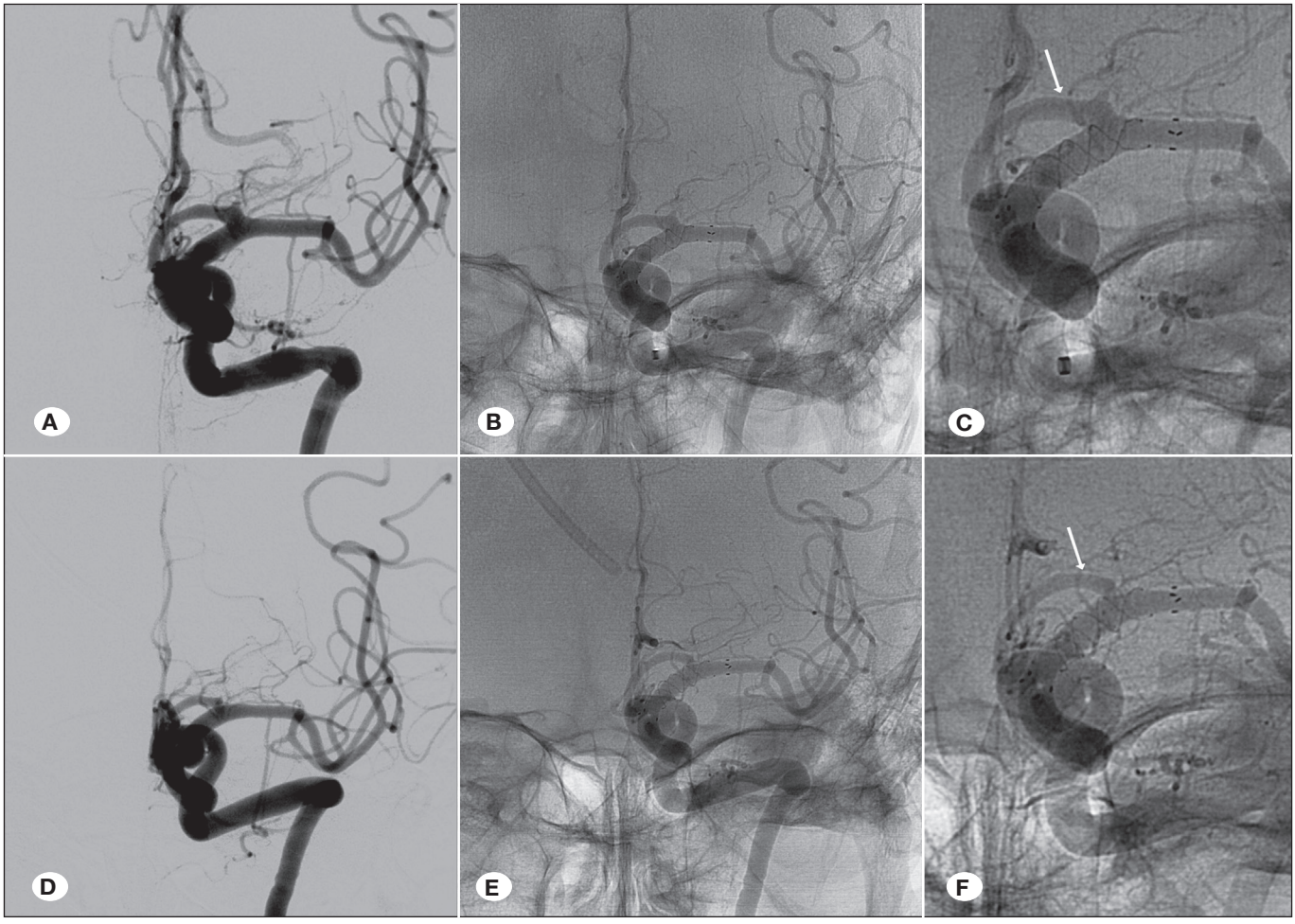

Figure 3: Working projection, subtracted DSA **(A)**, and unsubtracted DSA **(B)**, showing the placement of a flow diverter stent (4 × 12–18 mm, FRED, Microvention) from M1 to the proximal end of the paraophthalmic ICA with regular A1 patency (white arrows). Working projection enlargement showing the correct release of the FDS **(C)**. Twelve-month follow-up: subtracted DSA **(D)**, and unsubtracted DSA **(E)** showing left A1 patency and complete exclusion of the aneurysm. Enlarged follow-up DSA image, **(F)** showing the narrowing of the A1 lumen due to flow diversion (white arrows).

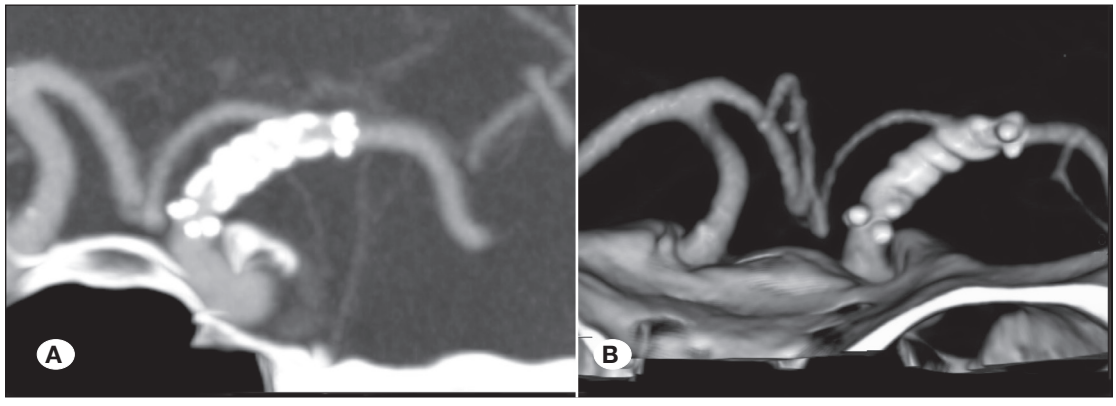

Figure 4: A CT angiogram at 24-month follow-up, MIP view **(A)**, and 3D VR **(B)**, showing correct FDS positioning and complete exclusion of the aneurysm. The CT angiogram also showed left A1 patency and narrowing.

endovascular embolization as the first treatment (3). Endovascular treatment is challenging, and coiling or assisted coiling are the most frequently employed techniques (6). Size and morphology evaluation highlighted that performing a simple coiling or releasing other endosaccular devices was impossible. Moreover, due to the anatomical site of the aneurysm, stent-assisted or balloon-assisted coiling was considered technically unfavorable. As a last choice, FDS was released. Nowadays, FDS is commonly used to treat ruptured aneurysms, especially ruptured blood-blister ICA aneurysms (11,12) and dissecting aneurysms (4). FDS placement is also reported for the treatment of unruptured A1As (8,9). FDS release in ruptured aneurysms has two possible major complications: 1) thromboembolic events related to perforator infarcts and intrastent thrombosis and 2) hemorrhagic events, since complete occlusion of the aneurysm only occurs within weeks after the procedure (3); therefore, there is a risk of rerupture or rebleeding during this post-procedural period (1).

We released the FDS from MCA to ICA to preserve the anterograde filling of MCA. Indeed, the unpredictable fate of a covered MCA origin may delay the filling of the covered MCA territory and cause ischemic stroke due to hypoperfusion (8). We opted for a competitive flow diversion and covered the origin of the parent artery proximal to the aneurysm, leaving the A1A neck uncovered. A progressive deconstruction by redirecting flow into the normal artery aimed to reduce flow in the diseased aneurysmal parent artery. This eventually promoted aneurysm sac thrombosis. As suggested by Mahmoud et al. (9), the A1A healing process could be explained by two mechanisms: 1) proximal ACA occlusion and reperfusion by the anterior communicating artery and 2) hemodynamic modification, even the slowing of the flow across ACA may alter the intra-aneurysmal environment and evolve toward aneurysmal thrombosis. As recently described by Cagnazzo et al. (2), the flow modulation across ACA, covered by an FDS, often results in the narrowing of the ACA (52%) and in the occlusion of the ACA origin (31%). In our case, although the neck of the aneurysm was not directly covered, the competitive modulation of the flow in the ipsilateral ACA resulted in the narrowing of the vessel (Figure 3), allowing us to achieve the correct aneurysm exclusion.

The preferred management by the authors is surgical clipping (5-7,14), followed by simple coiling and balloon- or stent-assisted coiling (6,10). FDS release for A1A treatment is less reported and detailed in the literature. Lin et al. proposed FDS placement from ICA to ACA, covering the MCA origin for the treatment of five A1As; however, there was an underlying risk of MCA stroke (8). Mahmoud et al. recently described FDS release from MCA to ICA for the treatment of four unruptured and one ruptured A1As (9); the latter was successfully treated by placing two FDS.

CONCLUSION

FDS embodies an important support in the treatment of ruptured aneurysms if other techniques, such as surgical and endovascular techniques, are not feasible. To the best of our knowledge, only few cases reported in the literature described the use of a competitive modulation by FDS for acutely ruptured A1As, which ultimately resulted in a successful aneurysm occlusion. The authors believe that this approach can be potentially applied to similar ruptured A1A aneurysms.

Although FDS is a consolidated technique for treating ruptured intracranial blister-like and dissecting aneurysms, it is still a “no man’s land” technique for treating intracranial saccular ruptured aneurysms. Studies with more number of cases are needed to validate the efficacy and safety of this endovascular procedure.

AUTHORSHIP CONTRIBUTION

Study conception and design: GV, AG

Data collection: GV

Analysis and interpretation of results: GV, EA, AG

Draft manuscript preparation: GV, EA

All authors (AG, GV, EA, SM, AVT, FC, FAV, FB, DL) reviewed the results and approved the final version of the manuscript.

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