CASE REPORT

Monotherapy with stenting in subarachnoid hemorrhage (SAH) after middle cerebral artery dissection

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SUMMARY

Isolated middle cerebral artery dissection is a rare clinical entity, with descriptions limited to a few case reports and case series. Symptomatic dissection in the anterior circulation can present as an ischemic stroke in a young population; however, it is rarely associated with subarachnoid hemorrhage. We describe a young patient who presented with acute headache from a subarachnoid hemorrhage that was ultimately determined to be due to a vascular dissection in the middle cerebral artery. The initial angiogram showed vascular irregularities in this area with stenosis. Repeat imaging 4 days after presentation identified a pseudoaneurysm proximal to the stenosis. The patient was successfully treated with a self-expanding nitinol stent and followed up with serial angiography during postoperative recovery in the hospital; additional angiograms were performed approximately 1 and 6 months after treatment. Serial angiograms demonstrated incremental healing of the dissection. The patient was discharged and remains neurologically intact at the 6-month follow-up.

BACKGROUND

Intracranial dissection is rare, and there is a paucity of documentation of dissections in the middle cerebral artery (MCA).^{1–5} Generally, intracranial dissections in the posterior circulation present with hemorrhage whereas anterior circulation intracranial dissections typically present with ischemic symptoms.⁶ Medical, endovascular, and surgical treatment options exist for intracranial dissection.⁴ However, middle cerebral artery (MCA) dissection as a cause of subarachnoid hemorrhage (SAH) is rare and there is no standard of practice treatment options for this entity, so deciding on the best treatment strategy is difficult. We present our experience of monotherapy with a self-expanding stent for SAH caused by a MCA dissection. The case illustrates and provides an option for neurointerventionalists who come across this clinical presentation in their practices.

CASE PRESENTATION

A 44-year-old man experienced an acute onset of headaches while lifting weights followed by syncope. Non-contrast CT scan demonstrated SAH, predominantly located in the right Sylvian fissure (figure 1). The past medical history was significant for chronic kidney stones and Crohn's disease. The patient denied any alcohol, smoking, or illicit drug use. Initial cerebral angiography demonstrated

irregular narrowing in the M1 segment of the right MCA (figure 2).

INVESTIGATIONS

After the initial angiogram, the patient was admitted to the ICU for monitoring; follow-up CT angiography (CTA) and MRI/MRA were performed to identify any evidence of intramural hematoma or changes compared with the angiography. However, non-invasive imaging did not contribute to identifying the causative factor for the SAH.

DIFFERENTIAL DIAGNOSIS

Following the initial angiogram, the possible differential diagnoses included ruptured focal dissection of the MCA or ruptured blister aneurysm with vasospasm.

TREATMENT

The patient was brought back 4 days after the initial angiogram for follow-up angiography which showed further narrowing in the lumen of the M1 MCA (residual lumen ~1 mm) with an irregular dissection flap and a pseudoaneurysm (figure 3). This confirmed the diagnosis of a dissection in the

Figure 1 Non-contrast head CT demonstrating subarachnoid hemorrhage predominantly in the right Sylvian fissure.

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Figure 2 Frontal angiography (A) of the right internal carotid artery showing the stenosis at the M1 segment of the middle cerebral artery (arrow). Three-dimensional angiogram (B) and magnified oblique angiogram (C) showing the irregular stenosis (arrows).

Figure 3 (A) Three-dimensional angiogram reconstruction on day 4 after initial angiography showing worsening of the stenosis (measurement line 1, 0.9 mm) and the presence of a pseudoaneurysm (arrow). Post-stenting magnified oblique angiogram (B) and cone beam CT (C) showing luminal improvement.

MCA. The patient was loaded with aspirin (600 mg) and clopidogrel (300 mg) and underwent successful placement of a 3 mm×30 mm Neuroform EZ stent (Stryker Neurovascular, Fremont, California, USA). The device was placed in the proximal M1 to the proximal M2 MCA and covered the entire vascular irregularity. Immediately after stenting there was improvement in the degree of stenosis in the affected segment of the M1, and a decrease in the prominence of the

pseudoaneurysm was observed angiographically (figure 3). The patient remained neurologically intact immediately after the procedure.

OUTCOME AND FOLLOW-UP

The patient was assessed angiographically again on post-operative days 1 and 7 (figure 4). The rationale for early

Figure 4 Magnified oblique angiograms on postoperative day 1 (A) and day 7 (B) showing further luminal improvement and decreased prominence of the flap.

Figure 5 One month follow-up angiography: frontal (A), magnified oblique (B), and cone beam CT (C) showing almost complete resolution of the luminal stenosis and flap.

follow-up angiograms was to assess the angiographic healing of the dissection given the risk of rebleeding on dual antiplatelet therapy. The patient was discharged 14 days after ictus, neurologically intact. Follow-up angiograms were performed at 1 month and 6 months (figures 5 and 6, respectively).

The postoperative angiograms showed incremental healing of the dissection, with the 6-month angiogram showing complete healing, restoration of the luminal size in the treated vascular segment and no visualization of the flap seen on the initial angiography. The patient remained neurologically intact.

Figure 6 Six-month follow-up angiography: frontal (A), lateral (B), and cone beam CT (C) showing complete resolution of the stenosis and the intimal flap.

DISCUSSION

Isolated dissection in the MCA is rarely seen clinically.³ In the 23 cases of isolated MCA dissection described by Lin *et al*,¹ 12% presented with hemorrhage and 88% presented with cerebral infarction. The majority of the literature on this type of dissection is limited to case reports; Lee *et al*² described two cases of MCA dissection presenting as ischemic strokes in young patients. In a total of 194 patients with isolated territorial infarction, intracranial dissection was diagnosed in 17 patients (anterior cerebral artery 64.3%, MCA 4.2%, posterior cerebral artery 6.7%).⁵ In a study of 25 patients treated for intracranial dissections over a 5-year period, the authors summarized the dissections into two groups: the ischemic group with better clinical outcomes (90-day modified Rankin Scale (mRS) score ≤ 3 , 83.3%) compared with the SAH group (90-day mRS score ≤ 3 , 46.2%).⁷ Recently, flow diversion has been used for an MCA blister aneurysm in the acute SAH period,⁸ but in our case flow diversion was not used due to the significant stenosis and a residual lumen of approximately 1 mm. The currently Food and Drug Administration-approved and commercially available flow diverters may not possess adequate chronic outward force compared with nickel-titanium alloy cerebrovascular stents. The chronic outward force of the implanted stent enabled gradual healing of the dissection flap and finally restored the vessel to its original diameter. Overlapping stents have also been used in dissections causing hemorrhage in the supraclinoid internal carotid

artery and the vertebrobasilar system.⁹ One of the major concerns is the hemorrhagic risks of dual antiplatelet therapy after stenting in the setting of acute SAH, including risks for ventricular drains, ventriculoperitoneal shunts, and the risk of rebleeding of the dissection. Hemorrhagic complications are reported in as many as 8% of patients treated with stent-assisted coiling in acute SAH.¹⁰ In a study comparing blister aneurysms and type IV vertebral dissections, the risk of rebleeding with vertebral artery dissections was 28.6%.¹¹ Other series have demonstrated rates of rebleeding as high as 70% from ruptured vertebrobasilar dissecting aneurysms.^{12–14}

Competing interests None.

Patient consent Obtained.

Ethics approval Ethics approval was obtained from the Institutional Review Board.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- ▶ Intracranial dissections are rare and have poor outcomes compared with extracranial dissections.
- ▶ Isolated MCA dissection is not commonly encountered clinically. When present, it commonly causes cerebral ischemia but can also lead to SAH.
- ▶ MCA dissection is diagnosed in the correct clinical setting of vessel irregularities without any known vascular risk factors and/or radiological signs of dissection such as double lumen, string sign, pearl sign, steno-occlusive disease, or pseudoaneurysm.
- ▶ Intracranial dissections can cause SAH due to thinner media and the lack of external elastic lamina in the intracranial vessels.
- ▶ MCA dissections causing ischemic symptoms can be treated with anticoagulants or antiplatelet agents. MCA dissections causing SAH can be treated with surgery (bypass and trapping of the dissected segment),¹⁵ but can also be successfully treated using endovascular reconstructive means. Following endovascular treatment, frequent angiographic follow-up is recommended to assess healing or progression of the dissection.

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