Rupture of De Novo Middle Cerebral Artery Aneurysm 8 Years After the Clipping of Ruptured M1 Middle Cerebral Artery Aneurysm

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Patient: Male, 39-year-old
Final Diagnosis: Brain aneurysm • de novo
Symptoms: Sudden onset of headache
Medication: —
Clinical Procedure: —
Specialty: Neurosurgery • Radiology

Objective: Unknown ethiology
Background: Development and rupture of a de novo intracranial aneurysm is rare. Little is known regarding its etiology and the appropriate timing of follow-up angiograms after surgical clipping or coiling.

Case Report: We present a case report of a 39-year-old male smoker with history of hypertension who developed a de novo aneurysm 8 years after surgical clipping of an aneurysm in the middle cerebral artery in the same segment. He presented with neck rigidity and drowsiness. Laboratory analysis did not show blood dyscrasia. Brain computerized tomography showed right temporal lobe hematoma and 4-vessel angiogram demonstrated de novo aneurysm in the same segment of the M1 middle cerebral artery, which was confirmed by intraoperative microsurgical findings. We review the literature on such cases and discuss the pathophysiology, diagnosis, and treatment of this condition. De novo aneurysm, although rare, can develop within days to as long as 10 years after surgical clipping or coiling.

Conclusions: This rare case of de novo aneurysm supports follow-up imaging of patients after initial surgical clipping for up to 10 years.

MeSH Keywords: Angiography • Intracranial Aneurysm • Subarachnoid Hemorrhage

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Background

*De novo* brain aneurysm refers to a newly identified aneurysm arising from vessels previously documented as normal in a follow-up imaging study [1–3]. There is limited information about the frequency, pathophysiology, and adequate follow-up imaging after a successful surgical treatment or coiling of brain aneurysms [4]. It has been reported that the frequency of *de novo* aneurysms is 0.84–1.8% per year [5]. They most commonly appear in the anterior circulation [6,7]. The risk factors involved in the development of these aneurysms are unclear [7,8]. Age, sex, inherited factors, hemodynamic changes, hypertension, and cigarette smoking have been correlated with a higher prevalence of *de novo* aneurysms [5,9]. However, the mechanisms by which these factors contribute to the formation of these condition are unknown. Some authors have recommended performing a follow-up angiography 6 months to 5 years after aneurysm treatment to identify new aneurysms [5,7,10]. Recent advances in image-processing algorithms have enabled automatic segmentation of brain aneurysm on magnetic resonance angiogram (MRA) or computed tomography angiogram (CTA) data sheets [11–13]. We present a case of 39-year-old man who developed an intracerebral hematoma caused by rupture of a *de novo* middle cerebral artery (MCA) M1 aneurysm 8 years after clipping of the same segment.

Case Report

A 39-year-old man with a history of heavy smoking of 4 packs a day for many years was diagnosed with a right MCA aneurysm (M1) (Figure 1A–1D) and was treated with microsurgical clipping. Postoperative imaging control showed no residual aneurysms (Figure 2A–2D). The patient was discharged 10 days after surgery without any neurological deficit. He was followed in our outpatient clinic for a period of 3 months thereafter and then was lost to follow-up.

Figure 1. (A, B) Preoperative angiogram after contrast injection of the right carotid artery, confirming the presence of a sacular aneurysm of the middle cerebral artery in segment M1. (C, D) Preoperative computed tomography angiogram confirming the presence of a right sacular aneurysm of the middle cerebral artery segment M1.
Eight years postoperatively, he was referred again to our clinic after having a spontaneous intracerebral hematoma that was diagnosed in a peripheral hospital. He presented with a 3-day history of headache and vomiting. He stated that he was diagnosed with hypertension 3 years ago, which was controlled with an angiotensin-converting enzyme inhibitor, and that he quit smoking 10 months earlier. On clinical examination, vital signs at presentation at our center showed a blood pressure of 120/75 mmHg and a heart rate of 85 beats/min. There was no clinical evidence of increased intracranial pressure at our center because the patient presented 3 days after the onset of the event; however, the patient appeared drowsy with neck rigidity with no other abnormalities. Laboratory values were: hemoglobin 11.4 g/dL (normal range [nr] 13.8–17.2 g/dL), hematocrit 34% (nr 41–50%), white blood cells 21 200/μL (nr 4500–11 000/μL), platelets 297 000/μL (nr 150 000–350 000/μL), prothrombin time 13.5 s (nr 11–13.5 s), partial thromboplastin time 26 s (nr 30–45 s), international normalized ratio 1 (nr <1.1) without evidence of blood dyscrasia. The brain CT showed a right temporal lobe hematoma that extended from the Sylvian fissure upward and medially (Figure 3A–3D). A 4-vessel angiogram revealed a right MCA multilobular aneurysm measuring 8×6 mm in M1 (Figure 3E, 3F). No additional aneurysms were observed, and the vascular clip from the previous surgery was found in place.

A right 35×40 mm pterional craniotomy was performed. Microsurgical dissection extended from the bifurcation of the internal carotid and M1 segment of the right MCA. The previous aneurysmal clip was found in the proper location without any displacement. The de novo aneurysm was identified on the opposite wall of the vessel. (Figure 4A) The neck of the aneurysm was dissected and a permanent Yasargil aneurysmal clip was inserted, followed by dissection of the aneurysmal sac, which was multiloculated and was cauterized by bipolar forceps (Figure 4B, 4C). The segment of the MCA branch between the old and the de novo aneurysms showed an athermanous aspect (Figure 4D). Cauterization of the sac of the aneurysm, a routine practice in our center, was performed (Figure 4D). After aneurysm clipping, we performed a transcortical evacuation of the large intracerebral hematoma.

Figure 2. (A–D) Postoperative angiogram of the right internal carotid artery 1 week after surgical clipping. A clip was placed on the previous M1 right middle cerebral artery aneurysm (white arrows).
The postoperative course was satisfactory, and a new angiogram was performed in which no aneurysms were observed (Figure 3G, 3H). The patient was discharged at the 10th postoperative day without any neurological deficit.

**Discussion**

*De novo* brain aneurysms have been reported to form several years after the initial normal angiography after surgical clipping or coiling [4,7,14]. The mean time between the first aneurysm and *de novo* brain aneurysm was 10.6 years (range 3–29 years) [15]. However fast-growing *de novo* aneurysms have been reported to develop within 8–47 days after microsurgical clipping [16–18]. Most of the *de novo* aneurysms have been found several months to years after the primary first imaging [19]. Giordano et al. [4] reported that just 11.2% of the *de novo* aneurysms appeared within 5 years and 88.8% were detected after more than 5 years. This author also found that the mean time for rupture of the *de novo* aneurysms was 10 years. In our case, it presented as an acute rupture 8 years after microsurgical clipping. Most *de novo* aneurysms are diagnosed between the fourth and fifth decade [19], although the accumulative incidence decreases with age: 2.2% for age ≤20 years, 0.46% for 21–40 years, 0.19% for 41 to 60 years, and 0.02% for >60 years [7].

Risk factors known to induce aneurysm rupture include sex, age, hypertension, family history of aneurysm, multiplicity, location, and smoking [4,7,9,20]. Estrogen is known to have an inhibitory effect on aneurysm formation; however, collagen content within the cerebral arteries may diminish after menopause, favoring the formation of aneurysms [5]. Brain aneurysm can be associated with some heritable connective tissue diseases, such as neurofibromatosis type 1, Marfan syndrome, and Ehlers-Danlos syndrome, as well as other inherited diseases such as polycystic kidney disease [21], Cowden syndrome [22], and Cheiro-Oral syndrome [23]. In our patient there was no clinical evidence of any of these syndromes. Cigarette smoking of more than 4 packs/day is known to be one of the more significant risk factors for subarachnoid hemorrhage secondary to aneurysmal rupture. Accordingly, smokers have a 3.7±5.7 higher rate of rupture compared with nonsmokers [7,20,24]. In this case report, the patient had 2 significant risk factors: hypertension and a history of heavy smoking. Moreover, an atheroma was also observed within the involved vascular segment.
The presence of the hematoma in the right temporal lobe and the anatomical proximity to the Sylvain fissure were indicative of either a possible slippage of the aneurysmal clip or the formation of a de novo aneurysm.

The pathogenesis of de novo brain aneurysm remains unclear; a few hypotheses have been postulated [25]. Hemodynamic alterations of vessels after treatment may cause the formation of cerebral de novo aneurysm [26]. Defects within the tunica muscularis or lamina elastica interna have been reported as possible triggers for the formation of aneurysms [27]. The incomplete treatment or slippage of the clip after the initial operation may also contribute to the regrowth of the aneurysm. This is known to be the most common cause of recurrent episodes of subarachnoid hemorrhage, which indicates the persistence of an underlying vascular condition [28].

In our case the previous aneurysm was completely clipped; 8 years later, the patient developed the de novo aneurysm in the same area. The presence of an atheromatous plaque observed during surgery in M1 suggests that the pathological vessel wall was the possible cause of the de novo aneurysm. The history of heavy smoking and hypertension probably also favored the formation of the de novo aneurysm.

At first instance, we considered that the patient presented a spontaneous intracerebral hematoma, but the proximity to the Sylvain fissure encouraged us to perform an angiographic study to rule out the possibility of a displaced aneurysmal clip or the presence of a de novo aneurysm. The formation of de novo aneurysms after surgery is rare. A previous study reported a de novo aneurysm 10 years after surgery [7,8].

Many authors have recommended performing a follow-up angiogram 6 months to 5 years after aneurysm treatment to identify de novo aneurysms [5,7,10]. Recent advances in image-processing algorithms have enabled computer-aided segmentation of brain aneurysms and adjoining blood vessels on MRA or CTA data sheets. Segmentation of brain aneurysms is essential to delineate aneurysms and to monitor and detect...
changes on follow-up studies. Many fully automated models have been implemented for different segmentation, analysis, and monitoring of brain aneurysms with the models designed to detect and delineate aneurysm and associated blood vessels on various data sheets [11]. Other models are also available, such as principal component analysis [12]. In our case, 2 aneurysms were treated successfully, of which 1 was a de novo aneurysm occurring in atheromatous ecstatic MCA 8 years after initial clipping. We believe that using a 3-dimensional automated aneurysm segmentation algorithm might be of help in early detection of de novo aneurysm formation compared with regular angiographic study such as CTA or MRA that are typically used for follow-up.

A limitation of this study is the lack of follow-up, including imaging during the period between 3 months after initial clipping and the presentation with de novo aneurysm 8 years later.

Conclusions

The formation of de novo brain aneurysm is rare. Several factors may contribute to its occurrence; history of arterial hypertension and smoking are important risk factors. We recommend follow-up imaging studies for about 10 years after the initial aneurysm.

Conflict of interest

None.

References:

16. Abe T, Saito N, Kunishio K: De novo aneurysm after ten days from the onset of SAH. No Shinkei Geka, 2008; 36: 1109–13

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