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Case Reports

Rapid Growth of an Unruptured Intracranial Aneurysm Over a Short Period: A Case Report

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Abstract

Background: Unruptured intracranial aneurysms (UIAs) generally exhibit slow growth, with rapid enlargement over a short period being uncommon.

Case Description: We report the case of a 69-year-old female in whom a 2-mm rt. middle cerebral artery aneurysm was detected routine brain screening. Follow-up magnetic resonance imaging (MRI) seven months later demonstrated enlargement to 5 mm with subtle hemorrhagic signal changes. Despite the recommendations for early intervention, the patient opted for continued observation. One month later, the patient was referred to our department. Owing to enlargement to 6 mm observed during three-dimensional computed tomography angiography (CTA), surgery was performed. The intraoperative findings revealed further enlargement to approximately 10 mm, and microsurgical clipping was successfully performed.

Conclusion: This case highlights the fact while UIAs rarely exhibit rapid growth over weeks or months, close interval growth and hemorrhagic imaging findings should prompt urgent reconsideration of management strategies.

Keywords: Unruptured Intracranial Aneurysm; Rapid Growth; Case Report; Microsurgical Clipping

Abbreviations

3D-CTA: Three-Dimensional Computed Tomography Angiography; CTA: Computed Tomography Angiography; ISUIA: International Study of Unruptured Intracranial Aneurysms; MEP: Motor Evoked Potential; MRI: Magnetic Resonance Imaging; MRA: Magnetic Resonance Angiography; SUAVe: Small Unruptured Intracranial Aneurysm Verification; UIA: Unruptured Intracranial Aneurysm.

Introduction

Unruptured intracranial aneurysms (UIAs) are increasingly detected with the widespread use of noninvasive neuroimaging modalities such as magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) [1]. Most UIAs are discovered incidentally and remain asymptomatic [1,3]. Additionally, natural history studies have demonstrated that many UIAs, particularly

small aneurysms located in the anterior circulation, have a low risk of rupture and can often be managed conservatively via radiological surveillance [1,3].

The International Study of Unruptured Intracranial Aneurysms (ISUIA) identified aneurysm size and location as major determinants of rupture risk and reported extremely low rupture rates for small (<7 mm) anterior circulation aneurysms [1]. Similarly, the Small Unruptured Intracranial Aneurysm Verification (SUAVe) study demonstrated a low annual rupture rate and minimal growth in aneurysms measuring ≤5 mm, supporting conservative management in selected patients [3]. These findings have strongly influenced current clinical practice guidelines [1,3].

However, accumulating evidence indicates that aneurysm growth during follow-up is a critical marker of instability and is strongly associated with an increased risk of rupture [2,4]. While most UIAs remain stable [1,3], marked and rapid enlargement is uncommon and may reflect aggressive pathological processes such as intramural hemorrhage, inflammation, or adverse hemodynamic stress [5-9].

Herein, we report a rare case of a patient with an unruptured intracranial aneurysm that enlarged rapidly from 2 mm to approximately 10 mm, accompanied by subtle hemorrhagic changes on MRI.

Case Presentation

Routine brain screening in a 69-year-old female with no history of subarachnoid hemorrhage revealed a 2-mm unruptured intracranial aneurysm in the right middle cerebral artery (Figure 1A). As the patient was asymptomatic, conservative management with radiological follow-up was recommended. The patient had no history of hypertension, diabetes mellitus, dyslipidaemia, smoking, or connective tissue disease. She was not receiving antiplatelet or anticoagulant therapy. There was no family history of intracranial aneurysm or subarachnoid hemorrhage, and no antecedent head trauma or intracranial infection.

Seven months later, follow-up MRI and magnetic resonance angiography (MRA) revealed aneurysmal enlargement to 5 mm (Figure 1B). Additionally, focal hypointense signal was observed on *T2*-weighted (STAR) imaging localized to the right Sylvian fissure adjacent to the aneurysm, suggesting minor hemorrhage (Figure 1C). However, the patient reported no history of headaches. Although surgical treatment was recommended at this stage, the patient strongly preferred to continue observation.

One month later, the patient was referred to our institution for further evaluation. Three-dimensional computed tomography angiography (3D-CTA) revealed enlargement of aneurysm (6 mm) (Figure 1D). After additional counselling regarding rupture risk associated with rapid growth and hemorrhagic imaging findings, informed consent was obtained, surgery was performed two weeks later after obtaining consent.

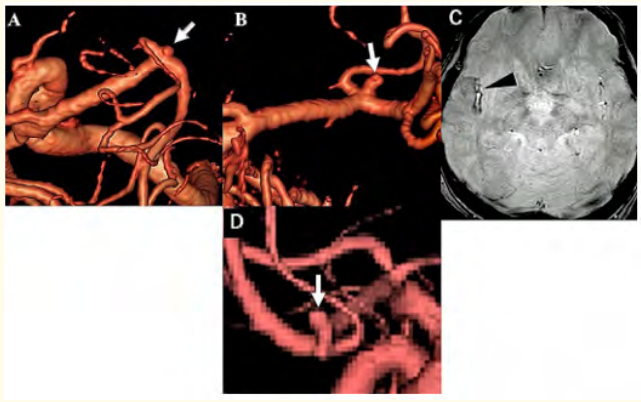

Figure 1: Preoperative neuroimaging findings.

A: Initial MRA demonstrating a 2-mm unruptured intracranial aneurysm.

B: Repeated imaging performed 7 months later shows aneurysm growth to a maximum diameter of 5 mm.

C: MRI (T2 STAR) showed minor hemorrhage (arrow head) in rt. Sylvian fissure.

D: 3D-CTA performed one month later confirming enlargement of aneurysm (6 mm).

MRA, magnetic resonance angiogram; 3D-CTA, three-dimensional computed tomography angiogram; Arrow: aneurysm.

Initial screening was performed using non-contrast three-dimensional time-of-flight MRA; aneurysm size was measured as the maximum dome diameter on source and reconstructed images. Follow-up evaluation included MRI with susceptibility-sensitive sequences and contrast-enhanced 3D-CTA. Although

inter-modality measurement variability exists, the magnitude of enlargement far exceeded expected differences, supporting true biological growth.

The procedure was performed via a right frontotemporal craniotomy using the pterional approach. Motor evoked potential (MEP) was monitored continuously intraoperatively. The Sylvian fissure adhered, likely due to inflammatory spread from a previous hemorrhage. Although significant adhesions were present around the aneurysm, careful dissection exposed them. The aneurysm had enlarged to 10 mm (Figure 2A), and the dome wall appeared red and thin. Neck clipping was performed using a 12 mm straight clip (Figure 2B). No abnormal findings were observed during intraoperative MEP monitoring. Postoperative 3D-CTA confirmed complete neck clipping of the cerebral aneurysm. The postoperative course was excellent, with no new neurological deficits. The patient was discharged on their own on postoperative day 8. Postoperatively, the patient recovered without any neurological deficits, and follow-up imaging confirmed complete occlusion of the aneurysm (Figure 3).

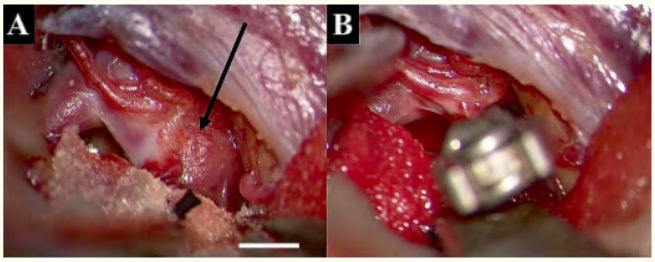

Figure 2: Intraoperative findings.

A: Intraoperative findings showing further aneurysm enlargement to approximately 10 mm with a thin reddish dome wall. Bar: 5 mm.

B: Neck clipping performed using a 12 mm straight clip.

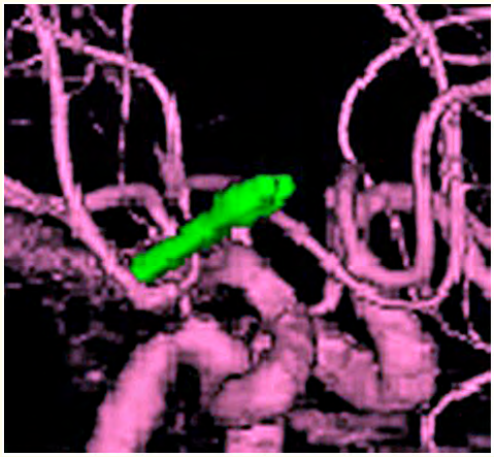

Figure 3: Postoperative neuroimaging.

Postoperative 3D-CTA confirming complete clipping.

3D-CTA, three-dimensional computed tomography angiogram.

Discussion

Unruptured intracranial aneurysms are generally considered lesions with a relatively indolent natural history [1,3]. Large observational studies such as ISUIA and SUAVe have established a basis for conservative management strategies, particularly for small aneurysms detected incidentally [1,3]. In these cohorts, most aneurysms remained morphologically stable over years, and when growth occurred, it typically progressed slowly [1-4].

The present case represents a striking deviation from the expected course. The aneurysm enlarged dramatically from 2 to approximately 10 mm within months, far exceeding the growth rates reported in longitudinal studies [2,4]. Such rapid enlargement strongly suggests an unstable aneurysm phenotype with a high risk of imminent rupture [2,4]. This rapid growth challenges size-only risk stratification and emphasizes the importance of interval change and hemorrhagic signals in clinical decision-making.

Rapid aneurysm growth as a predictor of rupture

Aneurysm growth is consistently identified as among the most powerful predictors of rupture, independent of aneurysm size at initial detection [2,4]. Villablanca., *et al*. demonstrated significantly higher rupture rates among aneurysms exhibiting interval growth compared with stable aneurysms [2]. Backes., *et al*. further

emphasized that aneurysm growth reflects active pathological remodeling of the aneurysm wall, rather than benign geometric enlargement [4]. Notably, the aneurysm in the present case was initially very small, highlighting the limitations of size-based risk stratification alone [1,3].

Possible mechanisms of rapid enlargement

Several mechanisms may explain the aggressive behavior observed in the aneurysm in the present case. Intramural hemorrhage or microbleeds within the aneurysm wall can trigger inflammatory cascades, extracellular matrix degradation, and progressive weakening of the aneurysm wall [5,6]. In the present case, subtle hemorrhagic changes detected on MRI supported the involvement of these mechanisms [6,10].

Inflammation is increasingly recognized as a central driver of aneurysm growth and rupture [7,8]. Experimental and human studies have demonstrated the contributions of macrophage infiltration, cytokine-mediated signaling, and loss of smooth muscle cells to aneurysm wall degeneration [7,8]. Intraoperatively, marked adhesions within the Sylvian fissure suggest a localized inflammatory response, consistent with these findings [5,7]. Although these processes cannot be directly proven in this case, the combination of hemorrhagic imaging findings, Sylvian fissure adhesions, and a thin reddish aneurysm wall is consistent with inflammation-mediated wall destabilization.

Hemodynamic stress may also play a role in sudden aneurysm expansion. Unfavorable wall shear stress patterns at arterial bifurcations can induce focal endothelial injury and aneurysm wall destabilization, particularly in middle cerebral artery aneurysms [9].

Clinical implications

Subtle hemorrhagic imaging findings may represent sentinel bleeding or intramural hemorrhage, which is an intermediate stage between aneurysm stability and frank rupture [6,10]. When such findings accompany rapid aneurysm growth, early intervention should be strongly considered regardless of absolute size [2,4,10].

Conclusion

We report a rare case of an unruptured intracranial aneurysm that exhibited rapid growth from 2 mm to approximately 10 mm. This case highlights the importance of vigilant imaging follow-up and timely surgical intervention in patients with rapidly enlarging UIAs.

Conflict of Interest

The authors declare no competing interests.

Bibliography

1. Wiebers DO., *et al*. "Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment". *Lancet* 362.9378 (2003): 103-110.

2. Villablanca JP., *et al*. "Natural history of asymptomatic unruptured cerebral aneurysms evaluated at CT angiography: growth and rupture incidence and correlation with epidemiologic risk factors". *Radiology* 269.1 (2013): 269:258-265.

3. Sonobe M., *et al*. "Small unruptured intracranial aneurysm verification study: SUAVe study, Japan". *Stroke* 41.9 (2010): 1969-1977.

4. Backes D., *et al*. "Growth of unruptured intracranial aneurysms: a systematic review". *Stroke* 47 (2016): 951-957.

5. Frösen J., *et al*. "Saccular intracranial aneurysm: pathology and mechanisms". *Acta Neuropathologica* 123.6 (2012): 773-786.

6. Kataoka K., *et al*. "Structural fragility and inflammatory response of ruptured cerebral aneurysms". *Stroke* 30.7 (1999): 1396-1401.

7. Chalouhi N., *et al*. "Biology of intracranial aneurysms: role of inflammation". *Journal of Cerebral Blood Flow and Metabolism: Official Journal of the International Society of Cerebral Blood Flow and Metabolism* 32.9 (2012): 1659-1676.

8. Aoki T., *et al*. "The development and the use of experimental animal models to study the underlying mechanisms of cerebral aneurysms". *Journal of Biomedicine and Biotechnology* 2011 (2011): 535921.

9. Meng H., *et al*. "High wall shear stress or low wall shear stress? Complex interactions of hemodynamics with intracranial aneurysm initiation, growth, and rupture". *AJNR. American Journal of Neuroradiology* 35.7 (2014): 125-132.

10. Edjlali M., *et al*. "Intracranial aneurysm wall enhancement and its relationship with aneurysm instability". *Radiology* 273 (2014): 536-542.