Vascular Health and Risk Management

Dovepress
Taylor & Francis Group

Open Access Full Text Article

CASE REPORT

"Double Trouble": Ultra-Early Formation and Rupture of a De Novo Terminal Basilar Artery Aneurysm After Aneurysmal Subarachnoid Hemorrhage

Ying Wang[1], Fenghua Chen[1], Hua Chen[2], Longbo Zhang[1,3]

[1]Departments of Neurosurgery and National Clinical Research Center for Geriatric Diseases (Xiangya Hospital), Central South University, Changsha, Hunan, People's Republic of China; [2]Department of Neurosurgery, Changde Hospital, Xiangya School of Medicine, Central South University (The First People's Hospital of Changde City), Changde, Hunan, People's Republic of China; [3]Department of Neurosurgery, Xiangya Hospital, Central South University, Jiangxi (National Regional Center for Neurological Diseases), Nanchang, Jiangxi, People's Republic of China

Correspondence: Hua Chen, Department of Neurosurgery, Changde Hospital, Xiangya School of Medicine, Central South University (The First People's Hospital of Changde City), 818 Renmin Street, Wuling District, Changde, Hunan, 415000, People's Republic of China, Email 936483206@qq.com; Longbo Zhang, Departments of Neurosurgery and National Clinical Research Center for Geriatric Diseases (Xiangya Hospital), Central South University, Changsha, Hunan, People's Republic of China, Email zhanglb@csu.edu.cn

Introduction: The formation of de novo intracranial aneurysms (DNIAs) often occurs months or years after the initial identification of an intracranial aneurysm. Consensus on surveillance after surgery remains elusive.

Case Presentation: A 53-year-old woman presented with a recurrent subarachnoid hemorrhage (SAH) caused by a rapidly expanding de novo basilar tip aneurysm, which developed and ruptured within 6 days of a previous SAH due to the rupture of a posterior communicating artery aneurysm. Both SAH incidents were successfully treated with microsurgical clipping. A computed tomography angiography at a 1-year follow-up did not detect any DNIAs.

Conclusion: DNIAs can form within days after primary surgery. Therefore, conducting early postoperative angiography is crucial to detect and manage DNIAs and prevent SAH, especially in high-risk cases. In addition, since DNIAs are more prone to rupture, early clinical intervention is recommended.

Keywords: de novo intracranial aneurysm, subarachnoid hemorrhage, case report

Introduction

Intracranial aneurysm (IA) stands as the primary cause of life-threatening subarachnoid hemorrhage (SAH). Despite the well-established treatments of microsurgical clipping and endovascular therapy for IAs, patients remain susceptible to the insidious development of de novo intracranial aneurysms (DNIAs) which are at a high risk of rupture. Due to the low incidence and unpredictable onset,[1,2] DNIAs are prone to being overlooked or missed. DNIAs often manifest months or even years after the initial identification of the primary IA.[3] On rare occasions, DNIAs can emerge within weeks of the initial evaluation, but they are rarely detected, as current follow-up imaging is typically scheduled 3 to 6 months after treatment. Ultra-early DNIA formation is primarily driven by the interplay of hemodynamic shifts, pre-existing vascular wall vulnerability, and a robust inflammatory cascade that disrupts the arterial matrix. In addition to the latent onset and uncertain timing, DNIAs are particularly prone to rupture compared to normal IAs, leading to high mortality and morbidity rates. However, there are difficulties in distinguishing true DNIA from initially occult lesions. Herein, we present a case involving a rapidly expanding DNIA at the bifurcation of the terminal basilar artery, which developed and ruptured within 6 days following the clipping of an anatomically distinct aneurysm. To the best of our knowledge, this case represents the earliest detection of a DNIA.

https://doi.org/10.2147/VHRM.S582055
Received: 17 November 2025
Accepted: 3 April 2026
Published: 23 April 2026

Case Presentation

A 53-year-old woman presented to our emergency department 1 hour after the sudden onset of severe headache and somnolence (Glasgow Coma Scale: 13). An initial computed tomography (CT) scan revealed a supratentorial subarachnoid hemorrhage (SAH) (Figure 1A and Supplementary Figure 1A–C). Further CT angiography (CTA) was performed to determine the origin of the hemorrhage, and a 3.0×1.8 mm aneurysm of the right posterior communicating artery (PComA) was identified (Figure 1B). The patient's past medical history was significant for a more than 20-year history of pyoderma gangrenosum, and an 8×10 cm^2 deep ulcer of the left leg with surrounding erythema and induration. The patient had been on prednisolone 5 mg/day for more than 20 years for her pyoderma gangrenosum.

Following the initial evaluation of the patient, we proceeded with microsurgical intervention. During the procedure, blood clots and a laceration were identified in the dome of the PComA, indicating its likely role in precipitating the subarachnoid hemorrhage (SAH). The aneurysm was meticulously dissected and clipped with preservation of the PComA. Intraoperative indocyanine green angiography revealed patency with excellent flow through. We examined the adjacent Willis artery, including the terminal basilar artery, and found no suspicious aneurysms. Subsequently, the patient was transferred to the ICU for Triple-H therapy (induced hypertension, hypervolemia, and hemodilution). Perioperative blood pressure was meticulously managed to ensure stable cerebral perfusion pressure. A postoperative CTA performed 4 days after surgery confirmed complete clipping of the PComA aneurysm. However, to our surprise, a de novo 3.0×1.6 mm aneurysm was discovered at the terminus of the BA, which had not been present on the prior CTA (Figure 1C). Unexpectedly, the patient became unconscious 2 days later (Glasgow Coma Scale: 9), and emergent CT identified a new SAH (Figure 1D and Supplementary Figure 1D–F). Subsequent CTA revealed significant enlargement of the previous BA tip aneurysm (3.8×2.1 mm vs 3.0×1.6 mm) (Figure 1E). The basilar DNIA of the basilar artery was considered responsible for the patient's new infratentorial SAH. After discussing treatment options with the patient's family, a second microsurgery was performed, and the ruptured aneurysm was successfully clipped. The patient recovered well with an uneventful postoperative course and was discharged 2 weeks later after spending 7 days in the ICU. At discharge, she was alert, oriented, and able to follow commands but had residual mild right hemiplegia. After spending 2 months in a rehabilitation center, the patient presented to our department again with hydrocephalus. A ventriculoperitoneal shunt with a programmable valve was implanted. The patient's condition progressively improved, and at a 1-year follow-up, no DNIAs were detected by CTA (Figure 1F). The entire management procedures of the case were illustrated in Figure 2.

Discussion

Intracranial aneurysms (IAs) were once considered to be a once-in-a-lifetime event. Nevertheless, IA patients who survive a previous SAH are at risk of a "second hit" due to DNIAs or IAs that were not detected during initial angiography or visible during the first operation. The first description of a new aneurysm developing on a previously angiographically normal middle cerebral artery (MCA) was by Graf and Hamby in 1964.[4] Although numerous cases have since been reported, DNIAs are still rare, with a frequency ranging from 0.3% to 4.13% per patient year.[1,2] Epidemiological studies have identified several risk factors associated with the development of DNIAs, including hypertension, previous SAH, alcohol consumption, cigarette smoking, female sex, age under 40 years old, family history of aneurysms, multiple aneurysms, and the internal carotid artery as the initial site.[1,5] Apart from being female and having experienced a previous SAH, our patient did not exhibit any of these other risk factors. A history of pyoderma gangrenosum suggests leukocytoclastic vasculitis, which compromises the integrity of the vessel wall and could potentially contribute to the formation of DNIAs.[6,7]

The formation of DNIAs often occurs 7.9 years after the initial identification of an intracranial aneurysm.[8] Most DNIAs occurred after 5 years of follow-up (88.8% vs 11.2%).[2,8] In rare instances, DNIAs have been identified within weeks or months of the initial evaluation.[9] Here, we report a case of a rapidly expanding DNIA at the bifurcation of BA that developed 4 days after the clipping of an anatomically distinct aneurysm, which, to our knowledge, is the earliest detection of a DNIA. The rapid enlargement of the DNIA over 2 days and its subsequent rupture may have been due to a combination of high wall shear stress (WSS) and positive WSS gradient (WSSG) caused by massive

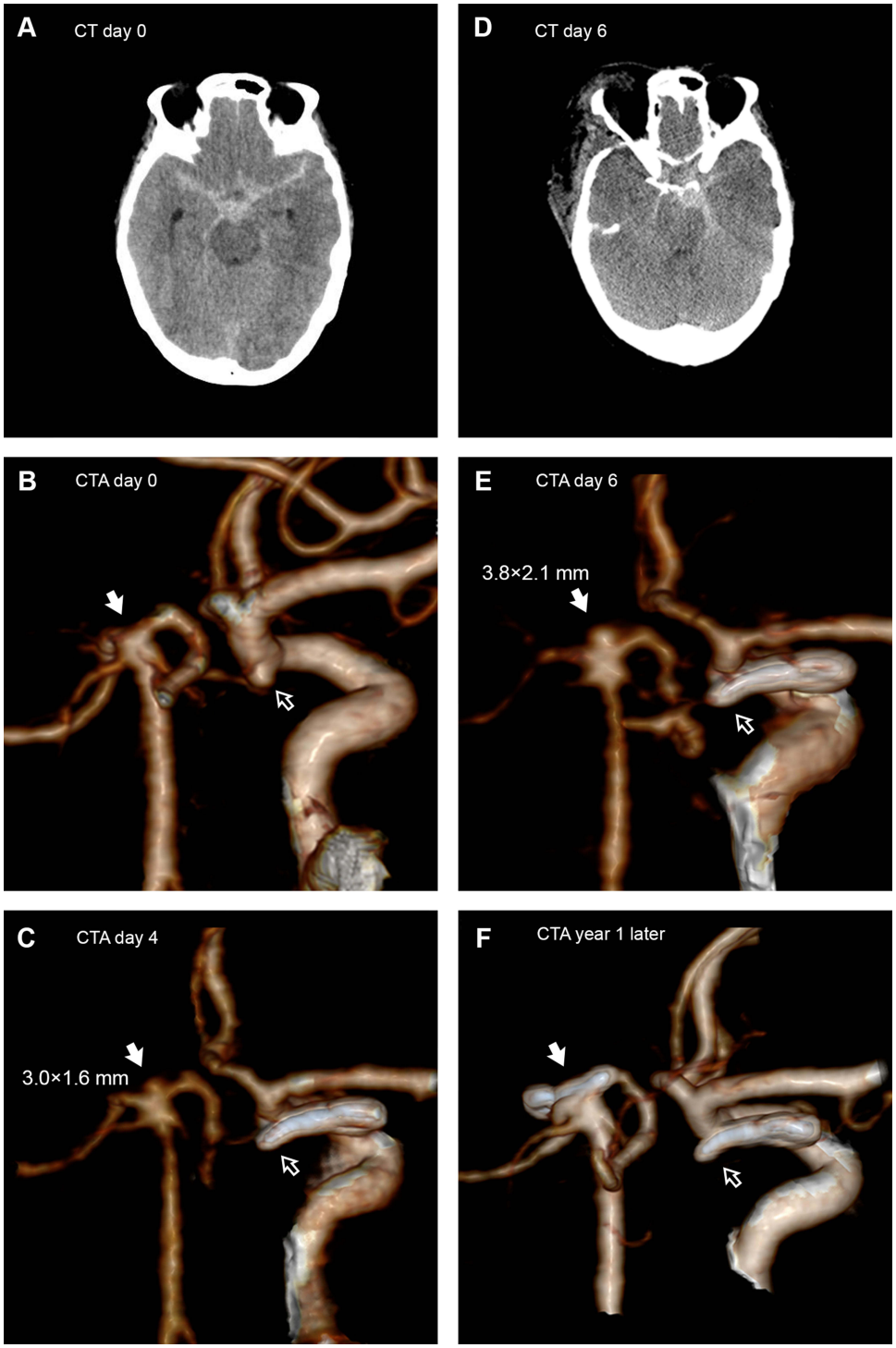

Figure 1 Rapid development and rupture of a de novo terminal basilar artery aneurysm. (**A**) The initial CT scan revealed a supratentorial SAH. (**B**) Subsequent CTA (day 0) identified a 3.0×1.8 mm intracranial aneurysm on the right PComA, presented here in a three-dimensional reconstruction image (empty arrow). The anatomy of the basilar trunk and tip is normal, with no indication of an aneurysm (filled arrow). (**C**) CTA postoperative 4 days confirmed that the PComA aneurysm was completely clipped with the PComA preserved. Preservation of the clip is shown in the three-dimensional reconstruction image (empty arrow). However, a new 3.0×1.6 mm aneurysm was found at the terminal basilar artery (filled arrow). (**D**) A new infratentorial SAH was shown by CT postoperative 6 days later. (**E**) Enlargement of the terminal basilar artery aneurysm (3.8×2.1 mm) was disclosed by postoperative CTA 6 days (filled arrow), and the aneurysm had ruptured, as evidenced by the location of SAH shown on CT (**D**). (**F**) CTA follow-up performed 1 year after the second clipping indicated that both aneurysms were completely occluded, and no DNIAs were detected.

https://doi.org/10.2147/VHRM.S582055

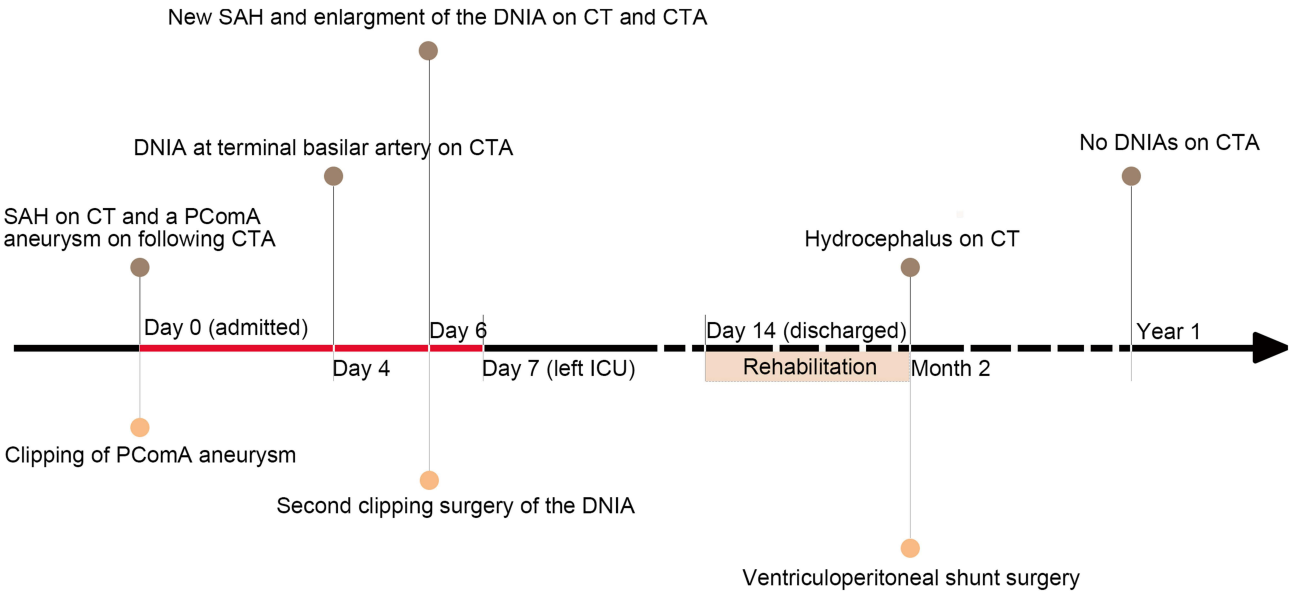

Figure 2 Timeline of management procedures. The illustration of the entire management of the case.

SAH-induced vasospasm or operative traction. Of note, the ultra-early timeline warrants consideration of arterial dissection, as dissections can resemble saccular aneurysms, particularly when intraluminal thrombus obscures the parent vessel's structural defects. Dissecting aneurysms typically occur along non-branching vessel segments. In this case, the absence of an intramural hematoma on CTA was crucial in distinguishing a primary hemodynamic remodeling from a dissection.

DNIAs are more prone to rupture,[3] and Kemp et al reported a 5-year hemorrhage risk of 14.5%, even with small (< 10 mm) DNIAs,[10] which is higher than the expected SAH risk of small, unruptured aneurysms reported in the 4060 patients assessed in the ISUIA (International Study of Unruptured Intracranial Aneurysms) trial. This trial revealed 5-year cumulative rupture rates for patients who did not have a history of subarachnoid hemorrhage with aneurysms less than 7 mm or 7–12 mm were 2.5% and 14.5%.[11] In our case, the rupture of DNIA and recurrent SAH may be prevented if microsurgical intervention is undertaken at the time of DNIA identification. Previous studies have shown that periodic follow-up with angiography significantly increases the identification of DNIAs compared to irregular imaging surveillance (4.9% vs 0.86%), with a detection peak between 0 and 2 years.[1] Meanwhile, DNIAs require careful and timely management due to their potential for rapid growth and rupture. Treatment options typically include microsurgical clipping or endovascular therapy, with the choice depending on the aneurysm's location, size, morphology, and the patient's clinical condition. Microsurgical clipping offers durable results and is often preferred for complex aneurysms, especially when associated with hematoma evacuation, while endovascular therapy provides a minimally invasive option for anatomically suitable cases. In our case, the DNIA developed well beyond the typical window of detection reported in the literature, prompting the decision to perform microsurgical clipping alongside detailed vascular exploration.

The "Triple-H" therapy was initially used to prevent cerebral vasospasm following SAH. However, with the widespread adoption of cerebral blood flow monitoring, the efficacy of "Triple-H" therapy in combating cerebral vasospasm has been questioned.[12] This therapy may lead to the rupture of unsecured IAs and other complications such as renal dysfunction and exacerbation of cerebral edema. While the goal of "Triple-H" therapy is to increase cerebral blood flow, the accompanying hypertension may increase the risk of developing DNIAs. Chagoya et al reported a case of rapid enlargement and rupture of an incidental pericallosal artery aneurysm after 7 days of "Triple-H" therapy in a patient with aneurysmal SAH.[13] For patients at high risk of DNIAs, caution should be exercised when considering "Triple-H" therapy.

Limitations

We acknowledge that this case report has several inherent limitations, including the difficulty in completely excluding an initially occult aneurysm despite negative baseline CTA. The absence of advanced hemodynamic or vessel wall imaging limits our understanding of the proposed mechanisms of DNIA formation and rupture. The hypothesis requires confirmation through larger clinical studies and laboratory research. The recommendation for early postoperative angiography is based on observational inferences, rather than prospective evidence.

Conclusions

In conclusion, early imaging follow-up, postoperative angiography, and early clinical intervention are recommended for high-risk patients to prevent further subarachnoid hemorrhage (SAH); however, these implications should be interpreted with caution. As this is based on a single case report, further prospective studies are needed to validate these findings and guide broader clinical practice.

Data Sharing Statement

Data that support the study were included in the article and available from the corresponding author upon reasonable request.

Ethics and Patient Consent

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Xiangya Hospital of Central South University (202103613). No additional ethical approval was required to publish these case details, as they are part of the study that was approved by the committee mentioned above. Written informed consent for publication was obtained from the patient.

Acknowledgments

The authors thank the patient presented in this research.

Funding

There is no funding to report.

Disclosure

The authors declare no conflicts of interest in this work.

References

1. Wang JY, Smith R, Ye X, et al. Serial imaging surveillance for patients with a history of intracranial aneurysm: risk of de novo aneurysm formation. *Neurosurgery*. 2015;77(1):32–42; discussion 42–33. doi:10.1227/NEU.0000000000000730
2. Giordan E, Lanzino G, Rangel-Castilla L, Murad MH, Brinjikji W. Risk of de novo aneurysm formation in patients with a prior diagnosis of ruptured or unruptured aneurysm: systematic review and meta-analysis. *J Neurosurg*. 2018;131(1):14–24. doi:10.3171/2018.1.JNS172450
3. van der Schaaf IC, Velthuis BK, Wermer MJ, et al. New detected aneurysms on follow-up screening in patients with previously clipped intracranial aneurysms: comparison with DSA or CTA at the time of SAH. *Stroke*. 2005;36(8):1753–1758. doi:10.1161/01.STR.0000173160.21182.3b
4. Graf CJ, Hamby WB. Report of a case of cerebral aneurysm in an adult developing apparently de novo. *J Neurol Neurosurg*. 1964;27:153–156. doi:10.1136/jnnp.27.2.153
5. Hu S, Yu N, Li Y, et al. A meta-analysis of risk factors for the formation of de novo intracranial aneurysms. *Neurosurgery*. 2019;85(4):454–465. doi:10.1093/neuros/nyy332
6. Khatibi K, Heit JJ, Telischak NA, Elbers JM, Do HM. Cerebral vascular findings in PAPA syndrome: cerebral arterial vasculopathy or vasculitis and a posterior cerebral artery dissecting aneurysm. *BMJ Case Rep*. 2015;2015:bcr2015011753. doi:10.1136/bcr-2015-011753
7. Alghamdi M. Autoinflammatory disease-associated vasculitis/vasculopathy. *Curr Rheumatol Rep*. 2018;20(12):87. doi:10.1007/s11926-018-0788-3
8. Vourla E, Filis A, Cornelius JF, et al. Natural history of de novo aneurysm formation in patients with treated aneurysmatic subarachnoid hemorrhage: a ten-year follow-up. *World Neurosurg*. 2019;122:e291–e295. doi:10.1016/j.wneu.2018.10.022
9. Serrone JC, Tackla RD, Gozal YM, et al. Aneurysm growth and de novo aneurysms during aneurysm surveillance. *J Neurosurg*. 2016;125(6):1374–1382. doi:10.3171/2015.12.JNS151552
10. Kemp WJ, Fulkerson DH, Payner TD, et al. Risk of hemorrhage from de novo cerebral aneurysms. *J Neurosurg*. 2013;118(1):58–62. doi:10.3171/2012.9.JNS111512

11. Wiebers DO, Whisnant JP, Huston J, et al. Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. *Lancet*. 2003;362(9378):103–110. doi:10.1016/s0140-6736(03)13860-3
12. Daou BJ, Koduri S, Thompson BG, Chaudhary N, Pandey AS. Clinical and experimental aspects of aneurysmal subarachnoid hemorrhage. *CNS Neurosci Ther*. 2019;25(10):1096–1112. doi:10.1111/cns.13222
13. Chagoya G, Salehani A, Tabibian BE, et al. Rapid evolution and rupture of an incidental aneurysm during hyperdynamic therapy for cerebral vasospasm. *World Neurosurg*. 2021;145:205–209. doi:10.1016/j.wneu.2020.09.078