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Case Report

Transvenous retrograde embolization of ruptured brain arteriovenous malformations: A case report and review of the literature ☆

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ABSTRACT

Hemorrhagic stroke due to ruptured brain arteriovenous malformations (AVMs) is a common cause in young stroke patients. When the ruptured AVMs are in deep location, the choice of endovascular intervention with the arterial approach to AVM embolization is routine but in many cases, it is not feasible due to the inability to access because of the small and tortuous arterial branch, however, the intravenous approach also results in high complete obliteration rates but also carries a higher risk of stroke than the intra-arterial route. We describe a 36-year-old female patient diagnosed with intracranial and intraventricular hemorrhage who underwent complete transvenous embolization of the ruptured AVMs, and achieved near-complete clinical recovery after 1 month with the modified Rankin scale 1.

Case report

A 36-year-old female patient with absolutely no history. One day before, the patient suddenly had a severe headache, accompanied by numbness in the right half of the body. On admission, her Glasgow 15 points, blood pressure 120/70 mmHg, heart rate 78 beats/min, respiratory rate 18 breaths/min, SpO2 99% with room air conditions, her pupils are 2 mm equal and reactive to light. Basic coagulation and complete blood count were normal. The patient underwent a non-contrast-CT scan of the brain, showing brain bleeding in the left thalamus size 10 × 17 mm with cerebral edema around the hematoma, bilateral intraventricular hemorrhage, and the fourth ventricle (Fig. 1). On CTA, there is an arteriovenous malformation corresponding to the location of the hematoma, nidus size 3 × 3.5 mm, the pedicle from the left posterior cerebral artery, the draining vein draining into the deep vein, no pseudoaneurysm was seen (Fig. 2). Spetzler-Martin score 3 points.

A multidisciplinary team including neurosurgery, stroke physician, and interventional radiology with indications for embolization due to the inability to operate in the deep brain

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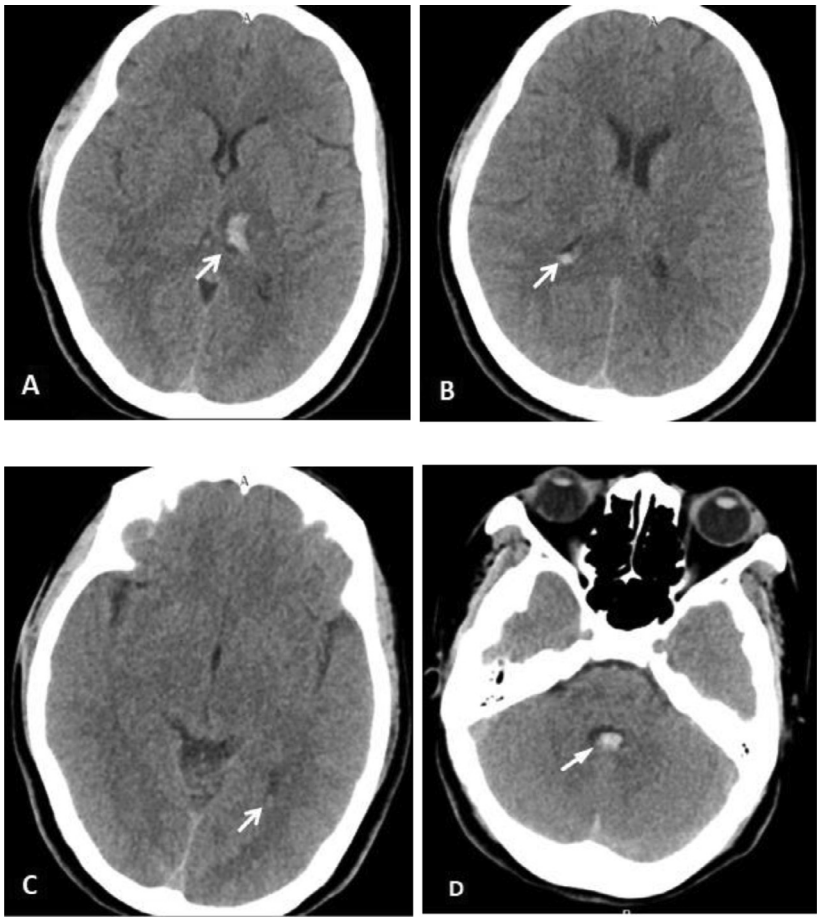

Fig. 1 – A non-contrast CT scan of brain showing left thalamus hematoma (arrow in figure 1-A), right ventricular occipital horn hematoma (arrow in figure 1-B), horn occipital left ventricle (arrow in figure 1-C), and fourth ventricle (arrow in figure 1-D).

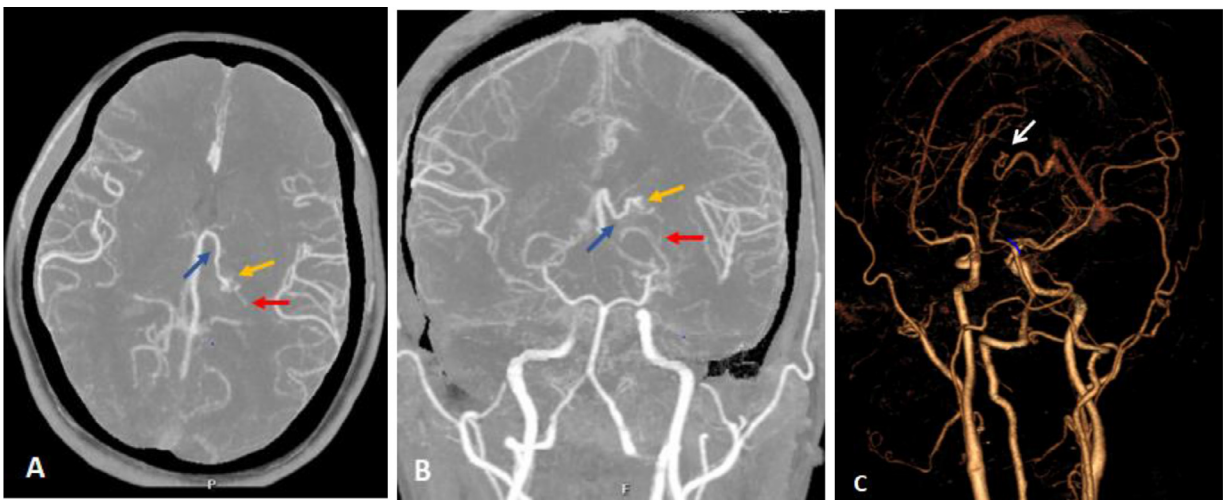

Fig. 2 – (A and B) CTA scan showing left thalamus arteriovenous malformation (AVM), pedicle from the thalamic branch of the left posterior cerebral artery (red arrow), nidus (yellow arrow)), drainage to the left internal cerebral vein (blue arrow), (C) 3D rendering showing a small AVM (white arrow). (Color version of the figure is available online.)

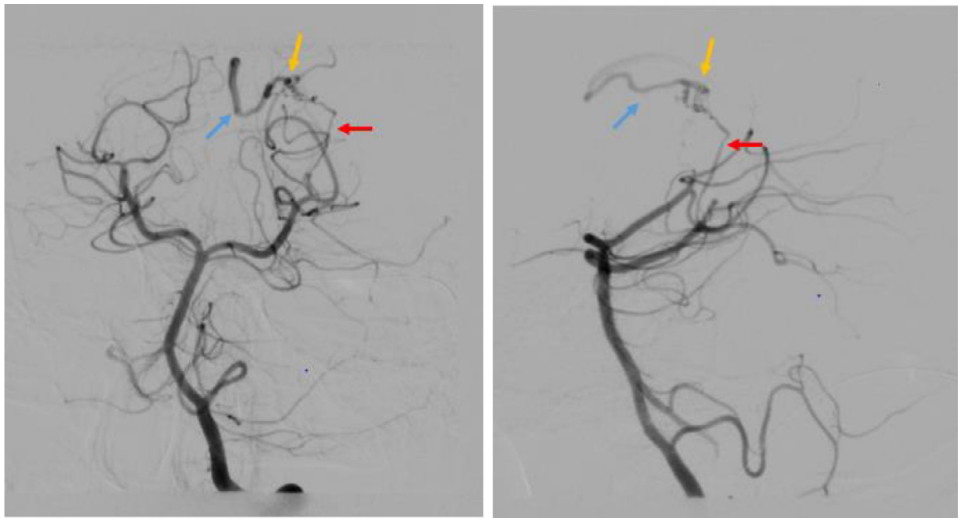

Fig. 3 – On DSA, the arteriovenous malformation (AVM) has a peduncle feeding from the left posterior cerebral artery (red arrow), nidus (yellow arrow), draining to the left internal cerebral vein (blue arrow). (Color version of the figure is available online.)

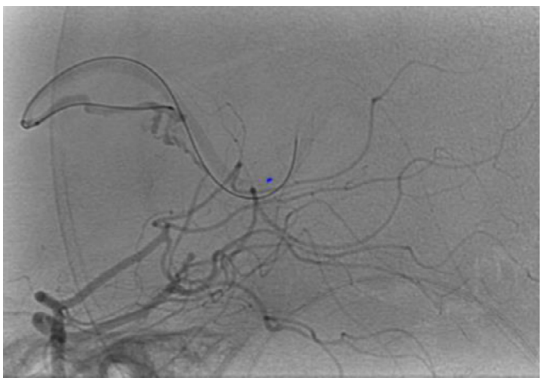

Fig. 4 – Microcatheter with intravenous access through a vein draining into the arteriovenous malformation (AVM).

bleeding site. The patient underwent bilateral carotid angiography and vertebral artery angiography: the left thalamic arteriovenous malformation was seen, with the blood supply thalamic branch from the posterior cerebral artery and the choroidal vessel drained by the internal cerebral vein (Fig. 3). Without arterial access, we performed right femoral vein puncture, 6F sheath placement, using SONIC 1.2F microcatheter, assisted Asahi Chikai 0.008 access to the left hippocampal vein of the cerebral vein in (Fig. 4). Inject 0.5 ml Onyx within 10 minutes, while the pump has ruptured malformation causing bleeding, proceed to pump more to completely block the malformation. Re-angiogram to check for complete occlusion of the arteriovenous malformation (AVM) malformation, no exit point (Fig. 5). CT scan of the brain without contrast injection 1 day (Fig. 6) and 5 days (Fig. 7) showed a left thalamic hematoma and blood in the lateral ventricle, blood in the fourth ventricle is relatively dissolved. The patient was discharged on the 10th day with a clinical recovery on the modified Rankin scale 1.

Discussion

Hemorrhagic stroke due to ruptured AVMs is a common cause in young stroke patients. The annual risk of recurrent bleeding in patients with ruptured AVM is 4.8% [1], within the first year is 6%-15.8% [2]. Therefore, ruptured AVMs should be treated. Treatment modalities for AVM include surgery, radiosurgery, endovascular intervention, and conservative treatment. The treatment strategy depends on the location, size, vascular features of the malformation, and clinical symptoms [3,4]. Surgery is difficult to perform when the AVM is deep location, inaccessible, or in a function area. Radiosurgery is not indicated in case of ruptured AVMs. The patient in our report was young, had a Spetzler-Martin score of 3, had bleeding in the left thalamus, drained into the deep vein, although there was bleeding of the ventricular system, no ventricular dilation, Glasgow score 15 points. Therefore, we choose endovascular intervention to treat the patient. Most interventional radiologists usually choose the transarterial intervention. However, in some cases the feeding artery is very small, the tortuous path makes it impossible for the microcatheter to reach the nidus, the interventionist can access it through the vein, as in our case, and complete occlusion of the nidus was obtained.

Currently, indications for interventional treatment of AVMs through transvenous routes include AVMs located in deep locations with a high risk of surgical complications, small nidus size (less than 3cm in greatest diameter), unfavorable arterial access, a single draining vein, and ruptured AVMs [5,6]. In particular, the prognostic ability must completely occlude the malformation according to an “all-or-nothing” technique, otherwise, the risk of bleeding is very high due to active obstruction of the draining vein.

Endovascular intervention for the treatment of AVMs through the transvenous route has difficulties due to anatomical changes and the tortuous path structure of the venous sys-

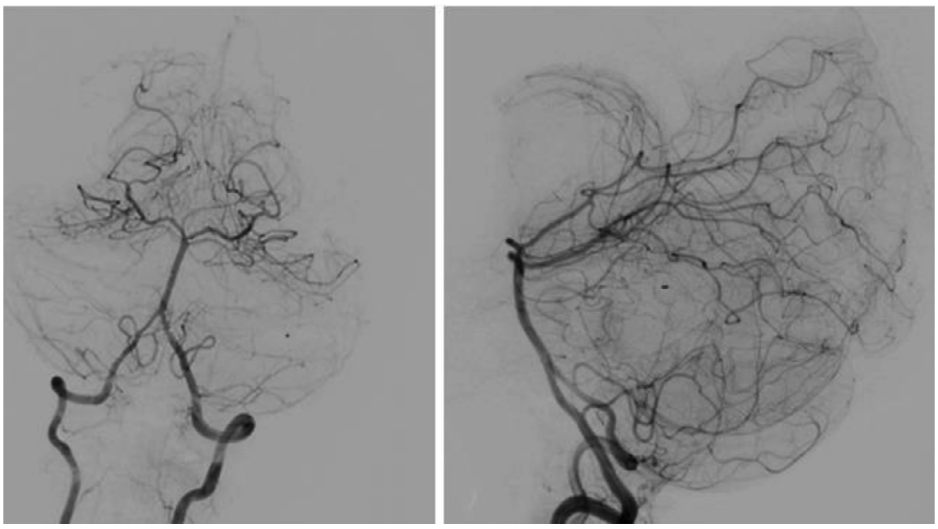

Fig. 5 – Re-scan after the Onyx pump showed complete occlusion of the arteriovenous malformation (AVM) drive, no exit point.

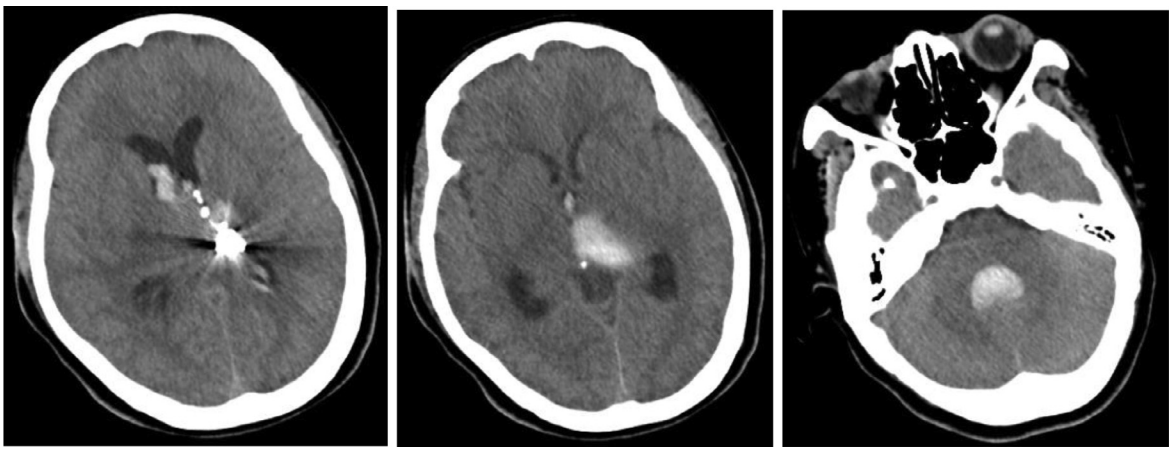

Fig. 6 – CT scan of the brain 1 day after the intervention showed a slight increase in blood count in the left thalamus and fourth ventricle.

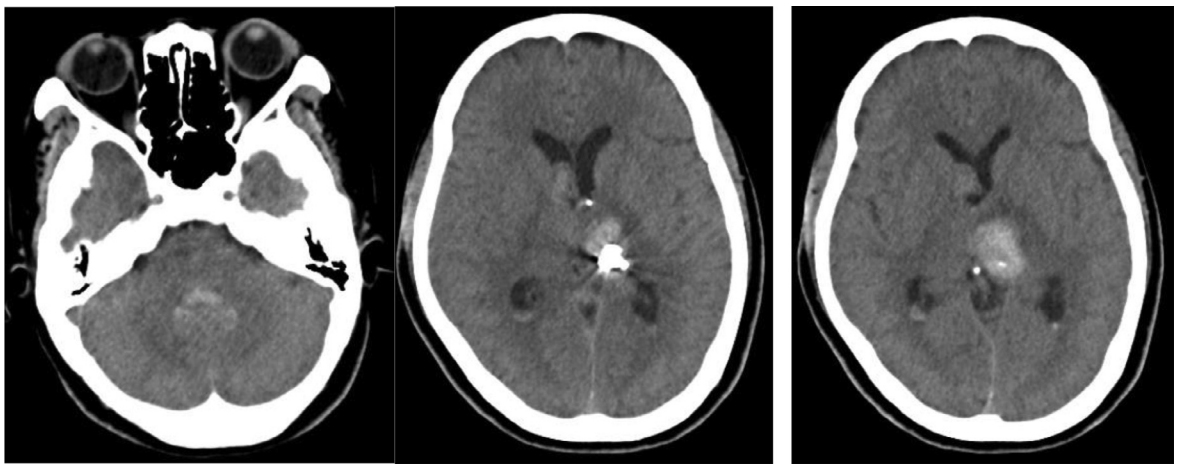

Fig. 7 – CT scan of the brain after 5 days of intervention showed decreased blood volume in the left thalamus and blood in the lateral and fourth ventricles was almost completely dissolved.

tem, and at the same time has the potential for certain complications. Intravascular insertion of instruments such as microcatheters and microguidewires can cause perforation of cortical veins due to their thin, fragile walls. The risk of rupture of the AVM occurs due to the increased pressure in the nidus when embolic agents are injected into the draining vein before it overflows and obstructs the nidus. To reduce this risk, which can cause systemic hypotension, accessible arterial embolization, and trans-arterial assisted balloon occlusion. If embolic material regurgitates into the cortical vein, partial or complete occlusion of the draining vein causes parenchymal congestion and delayed cerebral bleeding. If they enter the bloodstream to the lungs or other organs, they can cause infarction in the corresponding organ of the blocked vessel. In our case, the AVM rupture occurred when venous drainage was obstructed while Onyx had not been filled into the malformation. We noted that the patient had mild irritation despite general anesthesia. Immediately, we continued to inject more reflux into the shape to completely block the malformation in about 5 minutes. Checked on DSA, the AVM no longer shows up.

In recent years there have been individual case reports [7,8] as well as series of cases [5,9–11] showing that this technique gives promising results, high rate of embolism, less technical complications. However, the number of cases is still small, and the effectiveness and safety are still unknown. Therefore, a randomized trial comparing the two transarterial and transvenous approaches is needed. There is currently a TATAM trial comparing transvenous embolization with standard transarterial embolization (NCT03691870) slated to end in 2023 [12].

Conclusion

We report a case with the successful intervention of a small ruptured AVM occlusion deep in the thalamus via transvenous access. This is a new approach in the treatment of small-sized, deep-lying, bleeding AVMs when surgery will have a high risk of complications and there is no indication for radiosurgery. We hope to have more reports on the effectiveness and safety of this technique in the future.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Author contributions

Anh Tuan Tran and Duy Ton Mai contributed equally to this article as co-first authors. All authors read and approved the final manuscript.

Patient consent

Written informed consent was obtained from the patient for publication of the case details.

Data availability statement

All data generated or analyzed during this study are included in this article [and/or] its supplementary material files. Further inquiries can be directed to the corresponding author (Dr Duy Ton Mai; tonresident@gmail.com).

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