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Case Report

Transvenous embolization of a ruptured thalamic arteriovenous malformation supplied by the tuberothalamic artery

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ABSTRACT

Background: Basal ganglia and thalamic arteriovenous malformations (AVM) represent 10% of all AVM. They are associated with a high rate of morbidity and mortality due to their high hemorrhagic presentation and eloquence. Radiosurgery has been the first line treatment, whereas surgical removal and endovascular therapy are possible in selected cases. Deep AVM with small niduses and a single draining vein can achieve cure with embolization.

Case Description: A 10-year-old boy with sudden headache and vomiting underwent a brain computed tomography scan that showed a right thalamic hematoma. Cerebral angiography revealed a small ruptured right anteromedial thalamic AVM with a single feeder arising from the tuberothalamic artery and a single drainage to the superior thalamic vein. Transvenous approach using precipitating hydrophobic injectable liquid 25%* achieved a complete obliteration of the lesion in a single-session. He was discharged home without neurological sequelae and maintained clinically intact at follow-up.

Conclusion: Transvenous embolization of deep-located AVM as a primary treatment is curative in selected cases, with complication rates comparable to other therapeutic strategies.

Keywords: Deep arteriovenous malformation, Transvenous embolization, Tuberothalamic artery

INTRODUCTION

Basal ganglia and thalamic arteriovenous malformations (AVMs) represent 3–13% of all AVMs in the surgical series and 23–44% in the radiosurgical series. These lesions have a poor natural history and higher bleeding rates when compared to superficial AVMs. Previous studies reported good clinical and radiological results in small and deep-located AVMs treated with a variety of approaches.[9,17]

Basal ganglia and thalamic AVMs pose a high morbidity and mortality rates after bleeding due to their location. Surgical resection is feasible in a small group of patients.[14,15] Endovascular treatment is curative in selected cases, but is it has been typically used as a preoperative adjuvant therapy before microsurgery or radiosurgery.[6,9,10] Stereotactic radiosurgery (SRS) has been

used as an alternative treatment for deep-located AVMs such as basal ganglia and thalamus with high obliteration rates and good clinical outcomes.[2,6,7] Few publications have reported curative results for this subgroup of AVMs using the transvenous approach. This technique seems to be safe and potentially curative for some deep-located AVMs.[3,11,13]

We report a challenging case of a ruptured thalamic AVM supplied by the tuberothalamic artery (TTA), also called premamillary artery, arising from the right posterior communicating artery (PComA) which was treated in a single-session with endovascular embolization through the transvenous approach.

CLINICAL CASE

A previously healthy 10-year-old boy without relevant past medical history suddenly suffered headache and vomiting. Brain computed tomography (CT) scan showed a bleeding in the right thalamus associated with intraventricular hemorrhage and hydrocephalus. Contrast-enhanced brain CT scan was performed but was not conclusive to detect a vascular pathology [Figure 1]. Clinically, he was alert, without motor or sensitive impairment. Digital subtraction angiography showed a ruptured right thalamic AVM with a maximal diameter of 3.9 mm, with a single feeder arising from the right TTA, an intranidal aneurysm and a single draining vein to the right superior thalamic vein and internal cerebral vein [Figure 2].

Endovascular treatment was decided. Under general anesthesia, the right femoral artery was approached and a Chaperon 6F guide catheter (Microvention Terumo, Tustin, CA, USA) was advanced over a 0.035-inch guidewire into the right internal carotid artery. Then, a Magic 1.2F microcatheter (Balt, Montmorency, France) over a 0.007-inch Hybrid microguidewire (Balt) were navigated into the right PcomA. A superselective injection showed the nidus fed by a single-feeder arising from the TTA of the PComA. Under road-mapping, the TTA was catheterized without complications. A new superselective angiogram showed the AVM nidus, an intranidal aneurysm, the blush of the right thalamus and a single draining vein to the superior thalamic vein. Due to the thalamic blush and the high risk of infarction, the transvenous approach was decided. Thereafter, the right femoral vein was approached and a 6F Envoy (Codman, USA) guide catheter was navigated over a 0.035-inch guidewire into the right internal jugular vein. Under road-mapping, the detachable tip microcatheter Sonic 1.5F (Balt) over an Hybrid 008 microguidewire (Balt) was navigated through the transverse sinus, straight sinus, right internal cerebral vein, and right superior thalamic vein to the AVM nidus, and the microcatheter tip was placed as close as possible to the AVM nidus. Once in the correct position, precipitating hydrophobic injectable liquid (PHIL 25%; Microvention, Tustin, CA, USA) was injected manually during a period of 10 min and a total obliteration of the nidus was achieved [Figure 3]. Immediate control angiogram showed the absence of the nidus and under fluoroscopy the cast of the embolic agent was in accordance with the nidus.

Immediate brain CT scan was done and no bleeding was detected [Figure 4]. After the procedure, the patient was awaken and without neurological impairments. He was discharged home on the 2nd postoperative day. One-month follow-up showed a clinically intact patient with Modified Rankin Score of 0. Six-month angiographic follow-up revealed no nidus.

DISCUSSION

We demonstrated the endovascular management of a challenging deep-located AVM supplied by the TTA which had an *en-passage* configuration, making the trans-arterial route risky. In this particular case, the transvenous approach

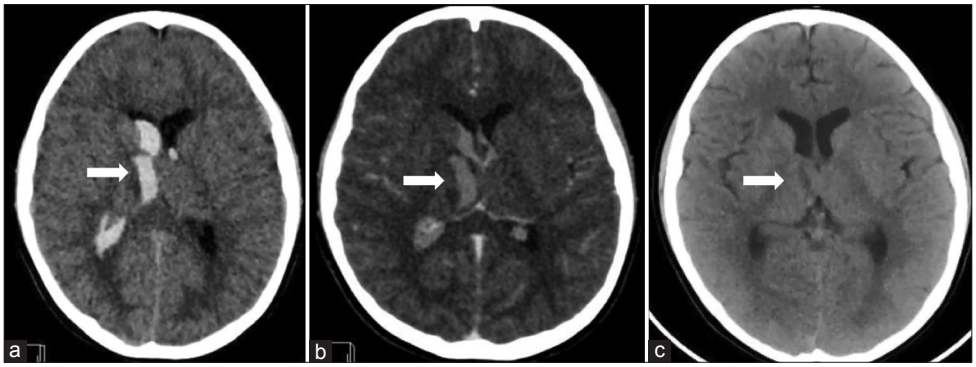

Figure 1: (a) Non-contrast brain computed tomography (CT) scan at admission showed a bleeding in the right anteromedial thalamus (arrow) and intraventricular hemorrhage. (b) Contrast-enhanced CT scan showed no evidence of abnormal enhancement in the right thalamus (arrow). (c) Before embolization, non-contrast CT scan showed encephalomalacia in the anteromedial portion of the right thalamus (arrow) with slight ventricular enlargement.

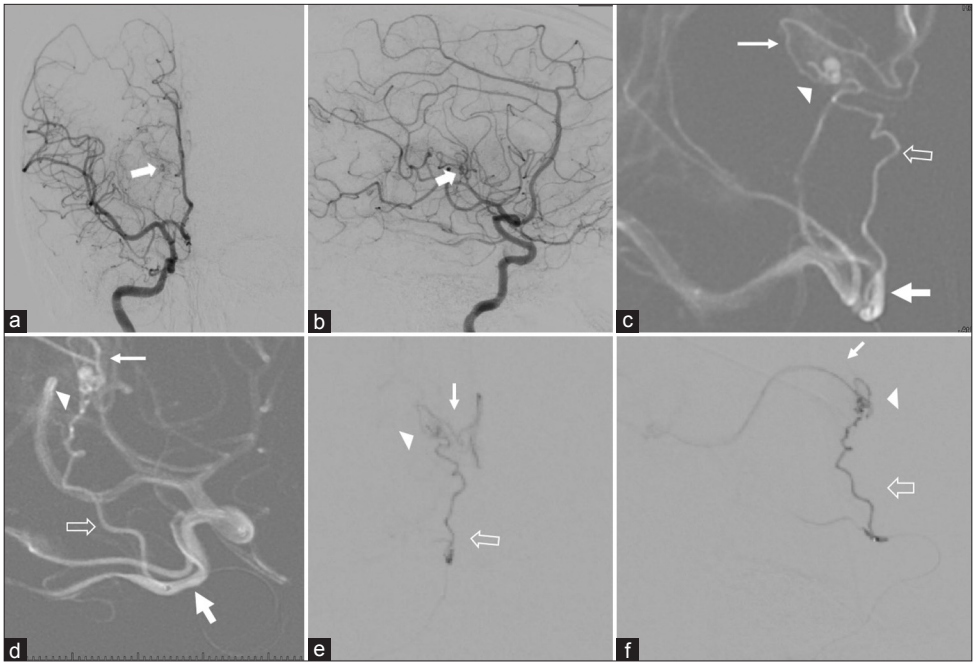

Figure 2: (a and b) Right internal carotid artery angiography in frontal and lateral views showing a thalamic micro-arteriovenous malformations (AVM) nidus (arrow). Road-mapping of the posterior communicating artery (large arrow) shows the feeding artery arising from the right tuberothalamic artery (empty arrow), the AVM nidus with an intranidal aneurysm (arrowhead) and the draining vein that is the superior thalamic vein (small arrow) in frontal (c) and lateral views (d). Superselective angiography of the right tuberothalamic artery (empty arrow), showing the AVM nidus (arrow head) and the superior thalamic vein (arrow) in frontal (e) and lateral views (f).

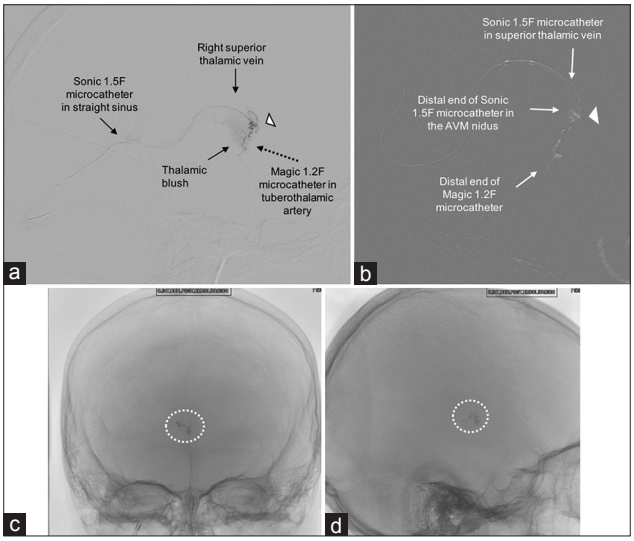

Figure 3: Ruptured right anteromedial thalamic arteriovenous malformations (AVM) supplied by the tuberothalamic artery, the AVM nidus with intranidal aneurysms (arrow head) and the right superior thalamic vein. The venous and arterial microcatheters are indicated, as well as their distal ends. (a) Superselective angiography performed through the right superior thalamic vein in lateral view. (b) Road-mapping in lateral view. (c and d) Fluoroscopic view showing the cast of precipitating hydrophobic injectable liquid 25% (dotted circle) in frontal and lateral views.

was selected to completely obliterate the nidus in a single-session with the embolic agent PHIL.

Basal ganglia, thalamic, and insular AVMs are lesions that involve highly eloquent structures. This group of AVMs present a more aggressive natural history with higher rates of bleeding, morbidity, and mortality when compared to other locations.[11] Deep-located AVMs occur rarely in the neurosurgical practice, corresponding between 4.3% and 11% of all AVMs.[5] Previous studies stated that deep location and deep venous drainage are associated to a more aggressive clinical course with an annual bleeding rate between 10% and 34% and a mortality rate as high as 62.5%.[5,11] These lesions are supplied mainly by perforators, especially from the P1 segment of the posterior cerebral artery, but can also be fed by meningeal branches, reported in 4% of the patients. Usually, thalamic AVMs pose a deep venous drainage, mainly to the vein of Galen or the internal cerebral vein.[16]

The TTA is a very rare feeder in cases of AVMs. The number and diameter of the perforating arteries are relatively constant and depend on the size of the PComA. Between 4 and 12 branches (average of 7) with diameters between 0.1 and 0.6 mm arise from the PComA. The largest branch of this group of perforators is known as the TTA, thalamotuberal artery, premamillary artery, polar artery, or anterior thalamus

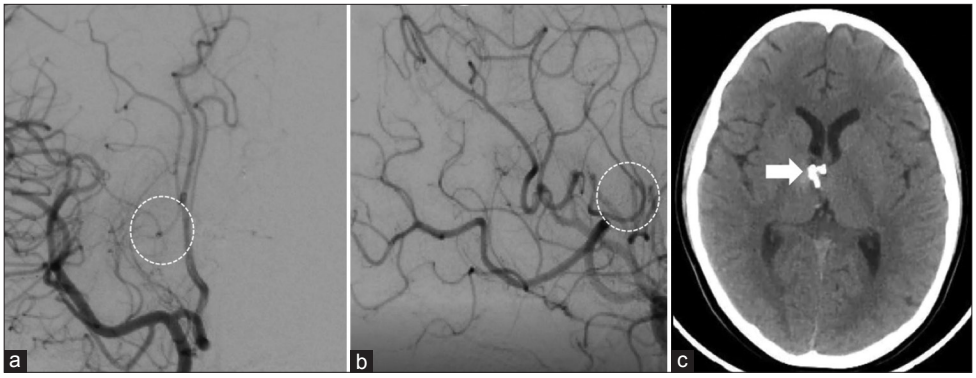

Figure 4: Six-month angiographic follow-up. (a) Right internal carotid artery (ICA) in frontal view demonstrates the absence of the arteriovenous malformations nidus (dotted circle). (b) Right ICA in lateral view shows a complete obliteration of the nidus (dotted circle). (c) Non-enhanced brain computed tomography shows the embolization material in the anteromedial part of the right thalamus (arrow).

perforating artery.[4] Commonly, the TTA arises from the caudal part of the PComA, which is close to the posterior cerebral artery, or on the border between the middle third and the caudal third of PComA,[4] as shown in our case. This artery supplies the ventral section of the thalamus, which includes the reticular nucleus, the anteroventral nucleus, the rostral section of the ventrolateral nucleus, the ventral pole of the medial dorsal nucleus, the mamillothalamic tract, the ventral tonsilofugal tract, the ventral section of the internal medullary lamina, and anterior thalamic nucleus.[4] Infarction of the TTA causes loss of recent memory, especially when the lesion is on the left side, or can manifest as mild or transient hemiparesis due to compression of the internal capsule.[1,8]

Multimodal treatment of AVMs has become an acceptable treatment because a single approach sometimes is not sufficient to obliterate challenging deep-located or high-grade AVMs with acceptable morbidity rates.[14] Several treatment modalities have been used, such as SRS which is often the first line treatment in these type of AVMs, but with obliteration rates lower than superficial AVMs.[2,6,7] The two markers of a successful treatment of an AVM are radiographic obliteration of the lesion and maintain the preoperative clinical status.[9] The surgical management of these AVMs is complex, due to limitation of surgical exposure, the comprise of eloquent neutral structures and the irrigation by perforating arteries.[11,14,15] Ruptured basal ganglia and thalamic AVMs may be considered for surgical removal if a surgical corridor is caused by the hematoma.[12,15]

Most of the indications proposed in the literature suggest that the transvenous approach is a salvage or last-resort therapy. There are strong indications for this approach, which includes a small and compact nidus (<3 cm in maximum diameter), deep AVMs, with high surgical risk, and ruptured AVMs. Lesions with a single draining vein are more favorable for the transvenous approach, and penetration of the embolic agent into the nidus is easier to achieve. Other indications for the transvenous approach are unsafe arterial access, which includes not having defined arterial pedicles, thin perforating arteries, and feeding arteries *en-passage*. Inaccessible nidus identified with persisting drainage vein, incomplete microsurgery, and failed radiosurgery are also candidates.[3] In our case, an *en-passage* single feeding artery and a single draining vein made the transvenous approach feasible.

In our case, we did not select the trans-arterial approach because the superselective angiography of the right TTA showed the blush of the thalamus, which configures this feeder as an *en-passage* artery. Therefore, all these characteristics made the transvenous approach the best alternative, with a total embolization of the AVM without any neurological sequelae.

CONCLUSION

Basal ganglia and thalamic AVMs are associated to high rates of morbidity and mortality mainly due to bleeding, and a high rate of complications due to treatment in a highly eloquent area. Transvenous embolization may be a curative technique in selected cases, with complications comparable to other therapeutic strategies.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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