

Why Does Large Venous Pouch Thrombosis after Cerebral Arteriovenous Shunts Embolization?

Large venous pouch thrombosis following endovascular embolization is a serious and devastating complication.^[1-3] It can cause venous infarction, mass effect or thrombus growth leading to a particularly poor prognosis with high morbidity and mortality rates.^[2,3] We presented two cases of severe venous pouch thrombosis developed after AVS embolization and the mechanism of venous thrombus formation and growth was discussed. The patients consented to the procedure and these cases were approved by the ethics committee of our hospital.

CASE 1

An 18-year-old man presenting with 3-month history of chemosis of the right eye underwent Magnetic Resonance Imaging (MRI) scan, which showed an orbital AVS. Cerebral angiography revealed an intraorbital AVS with a large ophthalmic vein outflow [Figure 1]. The patient underwent coil embolization of the AVS from the arterial side successfully resulting in a reduction of the shunt flow. One month later, the patient developed proptosis of his right eye, blurred vision and pulsatile tinnitus and the residual AVS was embolized completely with Onyx. Two months later, the patient presented with proptosis of his right eye and blurred vision, and MRI scanning demonstrated thrombosis of the venous pouch. At 3-year follow-up, right proptosis and blurred vision were

present. MR imaging was obtained and demonstrated a massive thrombosis of the ophthalmic vein.

Case 2

A 54-year-old man who had chronic headaches underwent MRI scan examination suggested cerebral AVS. Cerebral angiography revealed a dural arteriovenous shunt with a large venous pouch fed by the right middle meningeal and left posterior auricular arteries [Figure 2]. The patient underwent complete embolization of the AVS from the right middle meningeal and left posterior auricular arteries using Onyx. On the second day, the patient developed bilateral oculomotor nerve palsy with bilateral ataxia is named Claude's syndrome. MR imaging was obtained and demonstrated a thrombosis of precentral vein and the venous pouch, brain edema. Six months after embolization, the symptoms were still persistent.

DISCUSSION

In this report, 2 cases with massive thrombotic change of drainers after embolization were introduced. Such complications are well-known but expansive thrombosed varix like as these cases may be very rare. In the two cases presented by Gonzalez *et al.*,^[1] venous thrombus after treatment of fistula resulted in significant complications. In series on

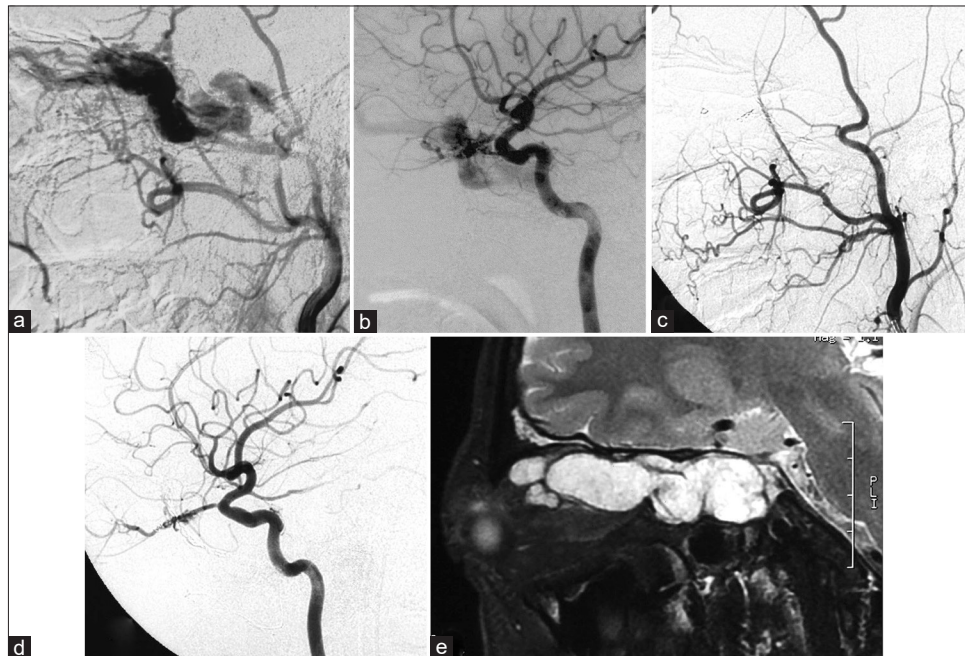


Figure 1: An 18-year-old man underwent embolization of a cavernous sinus. The external (a) and internal (b) carotid artery angiograms showing a dural AVF with multiple arterial feeders from the right middle meningeal artery, as well as a smaller contribution from the right ophthalmic artery. 3 years after the second treatment, the external (c) and internal (d) carotid artery angiograms demonstrating a residual dural fistula with a smaller contribution from the right ophthalmic artery. MRI (e) revealed prominence of the right superior ophthalmic vein

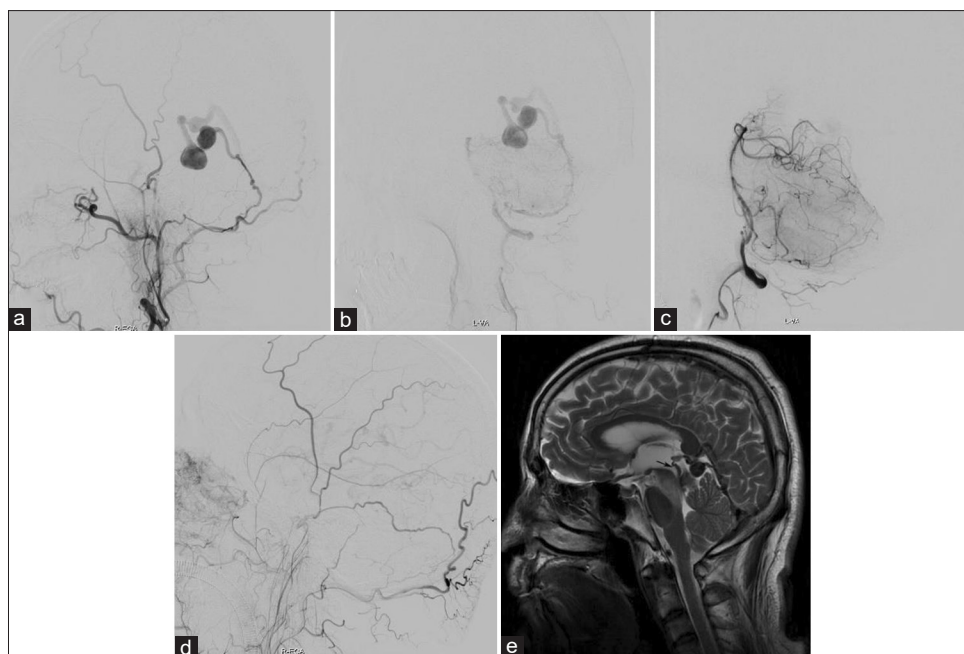


Figure 2: A 54-year-old man who had chronic headaches. The right external carotid artery (a) and the left vertebral artery (b) angiograms showing a dural AVS of the tentorial with large venous pouch fed by the right middle meningeal and left posterior auricular arteries. The right external carotid artery (c) and the left vertebral artery (d) angiograms after Onyx embolization revealing complete obliteration of the fistula. (e), sagittal MRI scan, T2-weighted, demonstrating precentral vein (arrow) and venous pouch thrombosis and mesencephalic edema

30 patients with dural AVS treated with Onyx embolization and strict anticoagulation, Cognard *et al.*^[4] reported one case of extensive thrombus of the draining vein with cerebellar bleeding following successful obliteration of the shunt. In another large series that included 121 dural AVS treated with transarterial glue embolization, Kim *et al.*^[5] noted some degree of venous thrombus in the postoperative period in 5 patients. One of these patients recovered spontaneously, while the other patients were treated with anticoagulant or steroid therapy. As the authors indicate, the use of anticoagulation may be one way to reduce the risk of this complication. As this complication is rare, it remains unclear which patients should be anticoagulated.

Venous thrombus forming under lower shear stress on the surface of a largely intact endothelium are fibrin-rich (so called “red clots” because they also contain red blood cells) and are treated with anticoagulant drugs.^[6-9] Rivaroxaban was shown to be superior to the low-molecular-weight heparin enoxaparin in reducing venous thrombosis embolization in four clinical trials involving total knee and hip replacement.^[10] Reduced hemodynamics or vascular wall pathology allows the accumulation of procoagulant proteases, such as thrombin, that may induce thrombosis. One may propose that the first step in venous thrombosis is activation of the endothelium and expression of the adhesion receptors P-selectin and E-selectin as well as von Willebrand factor (vWF). The activated endothelium then captures circulating leukocytes, tissue factor (TF) positive monocyte-derived microvesicles (MVs), and platelets. Finally, induction of TF expressed by the bound leukocytes together with TF on MVs triggers thrombosis.

CONCLUSIONS

Changes in blood flow in the blood itself and in the endothelium all increase the risk of venous thrombosis embolization. Leukocytes, platelets and MVs in the initiation and propagation of the thrombus and suggest that inhibition of the binding of leukocytes and MVs to the activated endothelium may represent a new therapeutic strategy to reduce the risk of venous thrombosis embolization.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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REFERENCES

1. Gonzalez LF, Chalouhi N, Jabbour P, Teufack S, Albuquerque FC, Spetzler RF. Rapid and progressive venous Thrombus after occlusion of high-flow arteriovenous fistula. *World Neurosurg* 2013;80:e359-65.
2. Jiang C, Lv X, Wu Z. Clinical-pathology review: Supratentorial cerebral arteriovenous fistula. *Eur J Radiol Extra* 2009;72:e97-102.
3. Lv X, Jiang C, Li Y, Yang X, Wu Z. Clinical outcomes of endovascular treatment for intracranial pial arteriovenous fistulas. *World Neurosurg* 2010; 73:385-90.
4. Cognard C, Januel AC, Silva NA Jr, Tall P. Endovascular treatment of intracranial dural arteriovenous fistulas with cortical venous drainage: New management using Onyx. *AJNR Am J Neuroradiol* 2008;29:235-41.
5. Kim DJ, Willinsky RA, Krings T, Agid R, Terbrugge K. Intracranial dural arteriovenous shunts: Transarterial glue embolization—experience in 115 consecutive patients. *Radiology* 2011;258:554-61.
6. Wakefield TW, Myers DD, Henke PK. Mechanisms of venous thrombosis and resolution. *Arterioscler Thromb Vasc Biol* 2008;28:387-91.
7. Undas A, Ariens RA. Fibrin clot structure and function: A role in the pathophysiology of arterial and venous thromboembolic diseases. *Arterioscler Thromb Vasc Biol* 2011;31:e88-99.
8. Wolberg AS. Plasma and cellular contributions to fibrin network formation, structure, and stability. *Haemophilia* 2010;16(Suppl 3):7-12.
9. Mackman N. Role of tissue factor in hemostasis, thrombosis, and vascular development. *Arterioscler Thromb Vasc Biol* 2004;24:1015-22.
10. Turpie AG, Lassen MR, Eriksson BI, Gent M, Berkowitz SD, Misselwitz F, *et al.* Rivaroxaban for the prevention of venous thromboembolism after hip or knee arthroplasty. Pooled analysis of four studies. *Thromb Haemost* 2011;105:444-53.

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