

---

## RECANALIZATION OF DURAL VENOUS SINUS THROMBOSIS ASSOCIATED WITH CRANIAL DURAL ARTERIO-VENOUS FISTULA FOLLOWING TREATMENT

Dittapong SONGSAENG,<sup>1</sup> Anchalee CHUROJANA,<sup>1</sup> Pipat CHIEWWIT,<sup>1</sup>  
Orasa CHAWALPARIT,<sup>1</sup> Suthisak SUTHIPONGCHAI<sup>1</sup>

### INTRODUCTION

Pattern of venous drainage from dural arteriovenous fistulae (DAVFs) has been shown to affect natural history and clinical presentation of these lesions. Dural venous sinus thrombosis is usually found associated with aggressive type of DAVF. Recanalization of these dural venous sinuses thrombosis after treatment is rare. We report two cases of DAVF with dural venous sinuses thrombosis and recanalization of dural venous sinuses thrombosis in patient with aggressive cranial dural AVF after complete obliteration of shunts following treatments; Partial surgery and 2 sessions of transarterial embolizations in the first case and multiple sessions of successful transarterial embolizations of dural AVF and affected sinuses in the second case. The patients' symptoms completely disappeared after follow up. Treatment aim of DAVF is certainly to get rid of the shunts. After closure of the shunts, unpredictable satisfactory recanalizing thrombosed dural venous sinuses are apparent. Dural arteriovenous fistula in patients of this single-institution series were diagnosed between 1995 and early 2005, total patients were 74 and treatment by embolizations were instituted in 54 of them.

### BACKGROUND AND PURPOSE

Cranial DAVF can occur with or without dural venous sinus thrombosis. Moreover, this dural venous sinus thrombosis is often associated with an aggressive cranial DAVF, which was dictated by their patterns of venous drainage with features such as cortical venous reflux, galenic drainage and venous congestion. Recanalization of these thrombosed sinuses after treatment were relatively rare, yet, some of those had been earlier mentioned by Ronie, et al.

We retrospectively reviewed 2 unusual cases of recanalizing thrombosed dural venous sinuses after treatment of aggressive cranial DAVFs and discuss their significances and frequencies of such a

presentation.

### MATERIAL & METHODS

Between 1995 and early 2005, 74 patients with intracranial DAVF were diagnosed and 54 of them had been treated in our institution. From our databases, we found only 2 patients had recanalizing thrombosed dural venous sinuses after treatment of aggressive cranial DAVF with dural sinus thrombosis.

**DAVF** = Dural arterio-venous fistulae.

---

<sup>1</sup> Department of Radiology, Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand.  
Presented in part at 8th Congress of the World Federation of Interventional and Therapeutic Neuroradiology, Venice, Italy, October 19-22, 2005.



## CASE REPORTS

### Case 1

A 19-year-old woman presented with progressive seizure after postoperative craniotomy for removal of blood from posttraumatic epidural and subdural hematoma. She was failed for medication to control the seizure. CT scan conducted at our institution suspected of DAVF at skull base and dilated Rt.superior ophthalmic vein, Rt.superficial middle cerebral vein and prominent Rt.cavernous sinus (Fig 1A-C). There was no intracranial hemorrhage. Same day diagnostic angiography was performed and was showed to have cranial DAVF supplied by Lt. middle meningeal artery (MMA), Lt.occipital artery and Lt.superficial temporal artery (Lt.STA) branches of Lt.external carotid artery (ECA) (Fig 2A-D) and from small meningeal branches of Rt.superior cerebellar artery (Rt.SCA), branches of Rt.vertebral artery (Rt.VA) (Fig 2E-F), anterior falx cerebri arteries from bilateral ophthalmic arteries of each internal carotid arteries (ICA) (Fig 2G-H). Complete occlusion of Rt.transverse sinus (TVS), partial occlusion of Lt.transverse sinus and sigmoid sinus (SS) and cortical vein reflux are shown on venous phase angiogram (Fig 2I). Venous drainage was major via Rt.Trolard vein into cavernous sinus, into superior ophthalmic vein and partial occluded Lt.transverse sinus into Lt.jugular vein (Fig 2J-K). Due to almost complete occlusion of bilateral transverse sinuses, transarterial route for embolization was selected. From a transarterial approach, Embolizations were done with 0.5mL 40% mixture of N-butylcyanoacrylate (NBCA, Braun, Melsungen, Suisse) and Lipiodol at feeding Lt.MMA (Fig 2L-M) and with Ivalon (300-500 microns, Cook, U.S.A.) embolization at Lt.STA. Immediate control angiogram of Lt.ECA

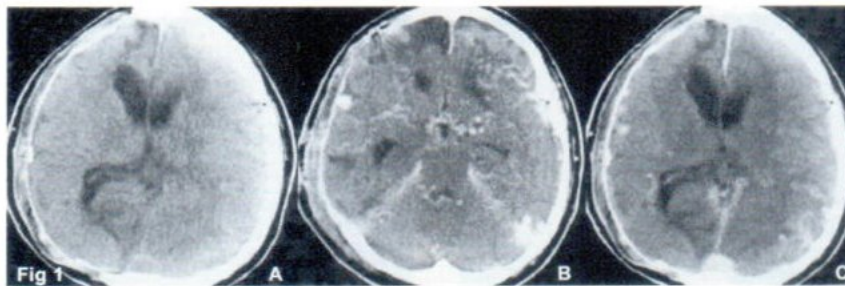
showed significant reduction of feeding arteries and rather slower flow of DAVF at middle 1/3 of SSS with reduction of cortical venous reflux (Fig 2N-O)

Follow up of 2 weeks interval Lt.ECA angiography demonstrated more prominent of a residual osteodural Arteriovenous shunt (AVS) at the same middle 1/3 of SSS fed by Lt.occipital artery which was immediately transarterial embolized by Ivalon (150-300 microns, Cook, U.S.A.) (3A-B). Control Lt.ECA angiography after embolization was obtained and showed complete absent of osteodural AVS (Fig 3C). Rt. and Lt.ICA angiographies showed no significant changed of cerebral cortical venous reflux pattern of ECA (External carotid artery), ICA (Internal carotid artery) and cerebral congestion (3D-G). After the procedure, our management teams had made a decision to refer the case for surgery to disconnect this dural arteriovenous shunt at the middle 1/3 of SSS.

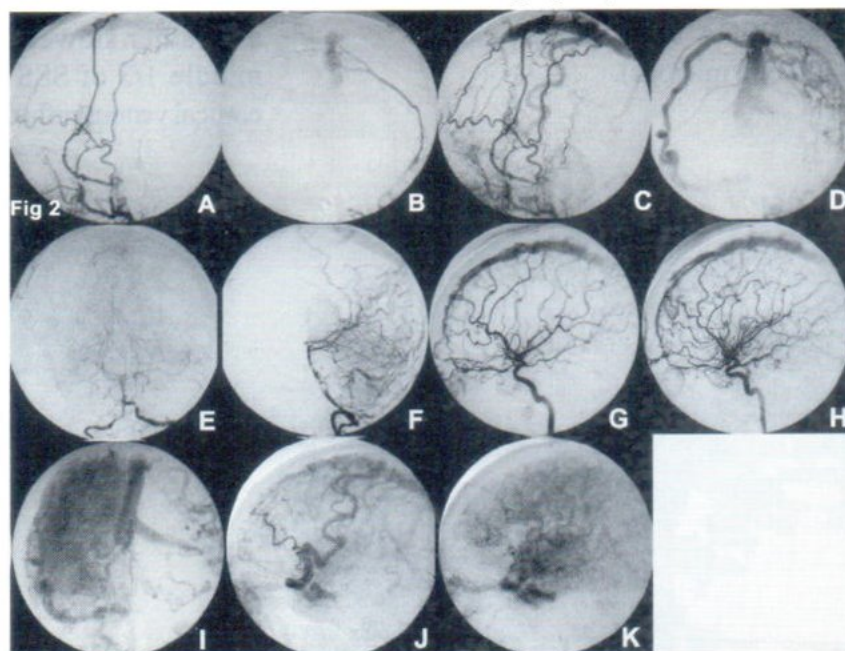
Follow up of 1-month interval showed minimal residual DAVF supplied by posterior meningeal branch of Rt.superior cerebellar artery and artery of falx cerebelli without cortical venous reflux. Partial recanalization of posterior upper 1/3 of SSS was noted (Fig.4A-E)

Follow up of 6 months to 2 years interval Rt.ICA angiographies and MRV of brain showed compatible finding of complete absent of DAVF with further recanalization of posterior 1/3 of SSS (Fig.5A-E).

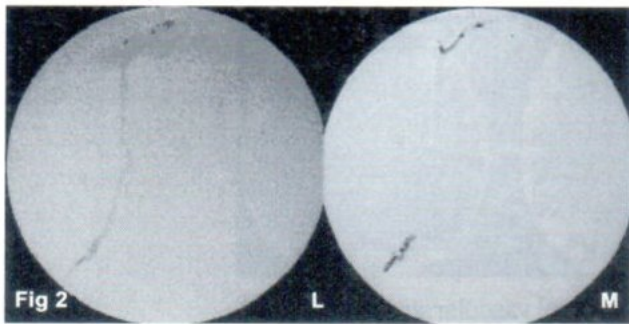




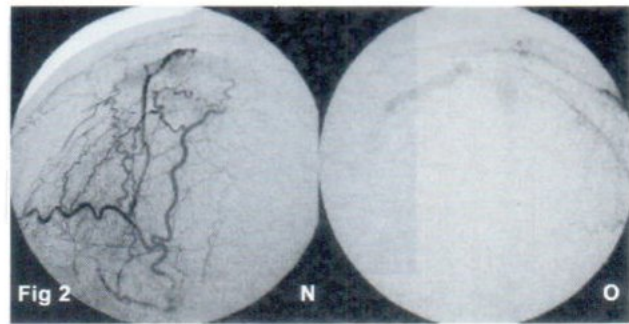
**Fig 1 A-C** Cranial CT scan showed abnormal vascular structure at skull base and dilated Rt. superior ophthalmic vein, Rt.superficial middle cerebral vein and prominent Rt.cavernous sinus without Intracranial hemorrhage (ICH).



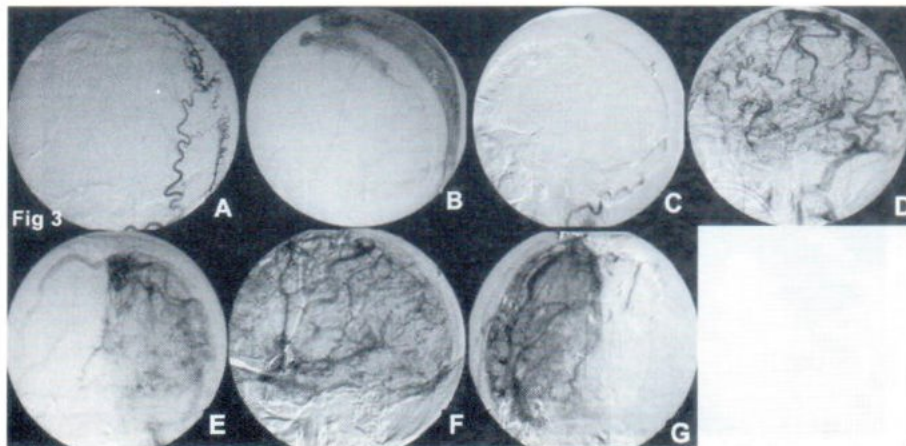
**Fig 2A-K** Lt. external carotid angiography showed early visualization of middle 1/3 part of superior sagittal sinus (SSS) by DAVF supplied by Lt. middle meningeal artery (MMA), Lt. occipital artery and Lt.superficial temporal artery (Lt.STA). Vertebral angiography showed small meningeal branches of Rt. superior cerebellar artery (Rt.SCA). Rt. & Lt. internal carotid angiographies showed other feeding arteries of DAVF; anterior falx cerebri arteries from bilateral ophthalmic arteries of each internal carotid arteries. Complete occlusion of Rt. transverse sinus (TVS), partial occlusion of Lt. transverse sinus and sigmoid sinus (SS) and cortical vein reflux are shown on venous phase angiogram. Venous drainage was major via Rt.Trolard vein into cavernous sinus, into superior ophthalmic vein and partial occluded Lt. transverse sinus into Lt.jugular vein.



**Fig 2 L-M** Lt. Middle meningeal artery (MMA) and Lt. superficial temporal artery (STA) superselective embolization by 40% mixture of NBCA and Lipiodol and Ivalon (300-500 microns).

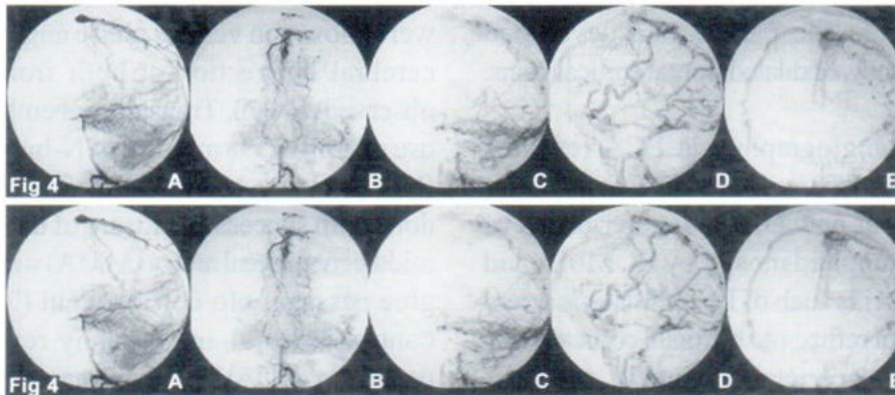


**Fig 2 N-O** A control angiogram of Lt. ECA was obtained after embolizations and showed significant reduction of feeding arteries and rather slower flow of DAVF at middle 1/3 of SSS and reduction of cortical venous reflux.



**Fig 3 A-G** Followed up 2 week-intervals, Lt. ECA angiography demonstrated a residual osteodural Arteriovenous shunt (AVS) at the same middle 1/3 of SSS fed by Lt. occipital artery which was embolized by Ivalon (150-300 microns. (3 A-B). Control Lt. ECA angiography after embolization showed complete absent of osteodural AVS (Fig 3 C) without significant changes of cerebral cortical venous reflux pattern and cerebral congestion (3 D-G).





**Fig 4-5** Follow up of 1-month interval showed minimal residual DAVF supplied by posterior meningeal branch of Rt.superior cerebellar artery and artery of falx cerebelli without cortical venous reflux. Partial recanalization of posterior upper 1/3 of SSS was noted (Fig.4 A-E)

Followed up of 6 months interval, Rt. ICA angiogram and MRV showed compatible finding of complete absent of DAVF with further recanalization of posterior 1/3 of SSS (Fig.5 A-D).

Followed up of 1 & 2 years interval, Rt. ICA angiogram showed no significant changed of findings as compared with study on 6-month interval follow up (5 E).

## Case 2

A 55-year-old man presented with chronic headache and progressive Lt.visual field defect following previous long-timed skull fracture. Cerebral angiogram of bilateral ICA, Vertebral arteries and bilateral External carotid angiography showed DAVF at posterior half of SSS which was thrombosed. These DAVF were supplied especially by bilateral middle meningeal arteries (MMA) and transosseous branches of bilateral Superficial temporal arteries (STA) (Fig 6 A-B). Retrograde drainage into ecstatic cortical veins particularly of Rt.parietooccipital lobe, represented venous congestion was also noted. Venous drainage of normal brain was rerouting from cortical veins into cavernous sinus with draining via dilated superior ophthalmic vein, and from Basal vein of Rosenthal, vein of Galen and vein of Trolard into straight sinus (Fig 6 C-D). Embolization was decided using transarterial route due to complete occlusion of

middle part of SSS. Embolizations with the used of 1.5 mL 25% mixture of N-butylcyanoacrylate (NBCA, Braun, Melsungen, Suisse) and Lipiodol were done at feeding Rt. Middle meningeal artery (MMA) and 3 branches of superficial temporal artery (STA) (Fig 6 E-F). Immediate control bilateral angiography showed nearly total occlusion of the DAVF and better opacification of cerebral parenchymal blush which was empties in venous drainage. Improvement of venous congestion was also noted (Fig 6 G-I).

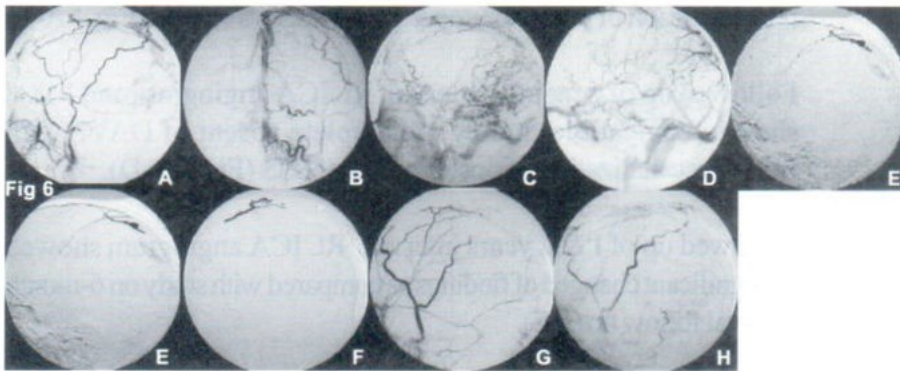
Follow up of 2 months interval cerebral angiography showed complete cure of DAVF with posterior half of SSS thrombosis. However, Redistribution of normal brain drainage from cavernous sinus to superior ophthalmic vein and to Labbe vein into sigmoid sinus was visualized.



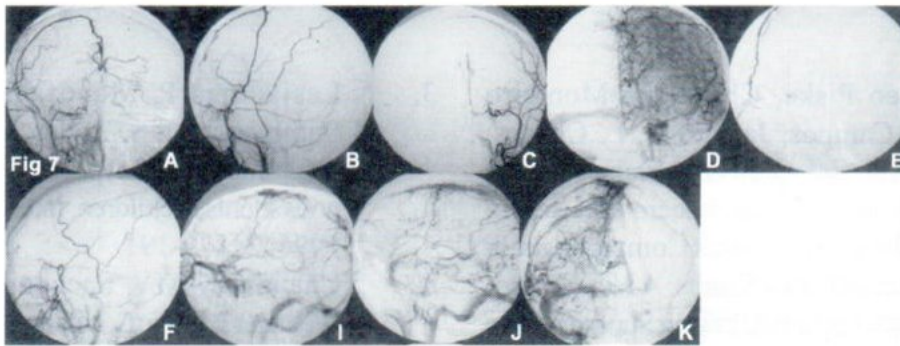
1 year later, the patient had developed seizure. CT brain showed dilated frontal cortical veins.

Cerebral angiography via ECA revealed complete occlusion of previous DAVF at posterior half of SSS yet, but new DAVF at anterior 1/3 of SSS was found, supplied mostly by Rt. MMA and anterior falx cerebri branch of Lt. ophthalmic artery of Lt. ECA. Cortical reflux into bifrontal cortical veins empty into Lt. sphenoparietal vein into Lt. cavernous sinus with rerouted to Lt. superior ophthalmic vein and recanalization of posterior half of SSS (Fig 7 A-D)

were shown on venous phase angiogram. Minimal cerebral congestion at both frontal lobes were observed as well. Transarterial embolization by the use of 1 mL 25% mixture of N-butylcyanoacrylate (NBCA, Braun, Melsungen, Suisse) and Lipiodol was done with successful closure of the shunt fed by Rt. middle meningeal artery (MMA) with no evidence of glue passage into cortical vein (7 E). Immediate control cerebral angiography revealed complete occlusion of DAVF and decreased cerebral congestion (7 F-K). Normal cortical vein could be seen in normal venous drainage right now.



**Fig 6 A-I** Cerebral angiogram and bilateral External carotid angiography showed DAVF at posterior half of SSS with thrombosis of SSS supplied especially by bilateral middle meningeal arteries (MMA) and transosseous branches of bilateral Superficial temporal arteries (STA) (Fig 6 A-B) and retrograde drained into ectatic cortical veins particularly of Rt. parietooccipital lobe and venous congestion, rerouting from cortical veins into cavernous sinus to dilated superior ophthalmic vein. (Fig 6 C-D). Rt. MMA and branches of Rt. STA Glue embolization (Fig 6 E-F). Controlled bilateral ECA angiography showed nearly total occlusion of the DAVF. Both ICA angiographies showed better opacification of cerebral parenchymal blush which were empties in venous drainage. Improvement of venous congestion was noted (Fig 6 G-I).



**Fig. 7 A-K** Cerebral angiography and ECA angiography revealed complete occlusion of previous DAVF at posterior half of SSS yet, but new DAVF at anterior 1/3 of SSS was found, supplied major by Rt. MMA and anterior falx cerebri branch of Lt.ophthalmic artery of Lt. ECA with cortical venous reflux into bifrontal cortical veins empty into Lt.sphenoparietal vein into Lt.cavernous sinus with rerouted to Lt.superior ophthalmic vein and notably of recanalization of posterior half of SSS (Fig 7 A-D). Minimal cerebral congestion at both frontal lobes was observed as well. Glue embolization with successful closure of the shunt fed by Rt. MMA with no evidence of glue passage into cortical vein (7 E). Control cerebral angiography revealed complete occlusion of DAVF and decreased cerebral congestion (7 F-K). Normal cortical vein could be seen in normal venous drainage right now.

## RESULT

Dural venous sinus thrombosis associated with cranial DAVF is found in approximately 15%.<sup>2</sup> Most of these occurred associated with aggressive DAVF. Recanalization of thrombosed dural venous sinus associated in cranial DAVF was infrequently appeared after closure of DAVF. However, 2 of 54 treated patients were found in our institute. The etiology of these unpredictable satisfactory recanalizing thrombosed dural venous sinuses may explain by previous mentioned dural venous sinus compartment in DAVF concept described by Ronie et al<sup>1</sup> which classified dural venous sinuses into 2 types: sinus septation and accessory sinuses. By our observation, we agreed and supported that theory. Ours 2 cases could be

represented for DAVF associated with septated dural venous sinus. Thus, superselective embolization of this abnormal sinus segment, which was believed to be the cause of following DAVF, could allowed us to cure the lesion while preserving the remainder normal venous sinus.

## CONCLUSION

2 cases of recanalization of dural venous sinus thrombosis in aggressive type, acquired cranial dural AVF after treatment were reported. By our observation, this pattern was not well frequently occurred and discussed.



## REFERENCES

1. Ronie Leo Piske, Christiane Monteiro Siqueira Campos, Jacinto B. L. Chaves, Ricardo Abicalaf, Guilherme Dabus, Laecio Leitao Batista, Carlos Baccin and Sergio Santos Lima. Dural Sinus Compartment in Dural Arteriovenous Shunts: A New Angio-architectural Feature Allowing Superselective Transvenous Dural Sinus Occlusion Treatment. *AJNR Am J Neuroradiol* 2005; 26: 1715-1722.
2. L K Tsai, J S Jeng, H M Liu, H J Wang and P K Yip. Intracranial dural arteriovenous fistulas with or without cerebral sinus thrombosis: analysis of 69 patients. *J. Neurol. Neurosurg. Psychiatry* 2004;75;1639-1641
3. Lasjaunias P, Maguifis A, Goulao R, Suthipongchai S, Rodesch R, Alvarez H. Anatomoclinical aspects of dural arteriovenous shunts in children. *Intervent Neuroradiol* 1996; 2: 179-191
4. Chaudhary MY, Sachdev VP, Cho SH, Weitzner I, Puljic S, Huang YP. Dural arteriovenous malformation of the major venous sinuses: an acquired lesion. *AJNR Am J Neuroradiol* 1982; 3: 13-19
5. Garcia-Monaco R, Rodesch G, Terbrugge K, Burrows P, Lasjaunias P. Multifocal dural arteriovenous shunts in children. *Childs Nerv Syst* 1991;7:425-431