MULTIPLE DURAL ARTERIOVENOUS MALFORMATIONS

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ABSTRACT

Four cases of multiple dural arteriovenous malformations (DAVMs) including base of skull are reported. Two of them have a DAVMs involving the cavernous sinus and separate AVMs in base of skull.The third patient has the symptomatic DAVMs involving straight sinus and the last patient has DAVMs involving torcular and both also have the second DAVMs involving superior sagittal sinus. All of the second DAVMs are incidentally detected by cerebral angiography. Reviewing of previous reports of this occurrence and the vascular anomalies that can association with dural AVMs are discussed.

INTRODUCTION

Dural arteriovenous malformations (DAVMs) account for 10-15 % for intracranial malformations. These lesions may occur within any dural structures but usually occur in the transverse sinus, sigmoid sinus and cavernous sinus. 35% of DAVMs are located in the posterior fossa. The DAVMs are generally considered as acquired lesions which may evolve from organization and revascularization of a previously thrombosed sinus.[2,4,5,9] Recent studies both angiographically and histologically[7,8] considered the site of fistula located within the sinus wall. The natural history of dural AVMs is highly variable. The most common clinical presentation are bruits, headache, intracranial hemorrhage, however, these depend mainly on the location of shunt, direction and route of the venous drainage of dural arteriovenous malformations.[2,3,14] Venous drainage is usually through the dural sinus and / or other dural and leptomeningeal venous channels. Retrograde leptomeningeal venous drainage is oftenly developed tortuous, variceal and frankly aneurysmal which associated risk of aggressive behavior such as intracranial hemorrhage or neurodeficits.[6,8] Spontaneous regression or thrombosis is not uncommon,[11,12,13] however, it should not be occurred in patients with high flow lesions, cortical venous drainage, or in children.[10]

As previously mentioned, DAVMs are relatively rare conditions and report of patients in case of multiple dural AVMs are very rare. We report four cases of dural AVMs associated with separate another dural AVMs. Two of them have a dural AVMs involving cavernous sinus and another separate arteriovenous malformations in right jugular bulb in one and left mastoid region in the other ones. The third patient has a symptomatic dural AVMs in posterior fossa and separate incidentally detected dural AVMs involving SSS. The fourth patient has a symptomatic dural AVMs of the torcular and the second dural AVMs draining into the SSS. Those are well demonstrated by diagnostic angiography.

(SSS = Superior Sagittal Sinus)

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CASE REPORTS

CASE 1

Clinical course: A 55-year-old woman had a history of a red eye for two years. She experienced a severe bifrontal headache. The pain was across the eyes and sometimes extended down to the maxillary sinus region. Approximately two years ago she suddenly started having the redness of the left eye. Approximately three months ago she started having a double vision and proptosis of the left eye. She had been managed conservatively for five months without any improvement. She was referred for evaluation and possible endovascular treatment.

Examination: The neuro-opthalomological examination revealed mild to moderate engorgement of the conjunctival vessels, slight proptosis, asymmetrical intraocular pressure and slight engorged retinal veins on the left eye. No detectable bruit. The extraocular muscles revealed minimal limitation of abduction in the left eye.

The angiographic study was performed and demonstrated a rapid dural arteriovenous shunting at the posteromedial aspect of the left cavernous sinus. Arterial blood supply was mainly by clival branches from neuromeningeal trunk of left ascending pharyngeal artery as well as small dural branches of C5 segment of both internal carotid arteries. Venous drainage was mainly into the left cavernous sinus appeared in aneurysmal fashion approximately 10 mm. in diameter. Further retrograde venous drainage was into a large posterior fossa vein which drain further into multiple cerebellar cortical veins as well as superiorly through anterior pontomesencephalic vein into basal vein of Rosenthal. Venous drainage anteriorly from left cavernous sinus into the left opthalmic veins was also documented. Another arteriovenous malformation was detected at anterior aspect of the right jugular bulb which mainly fed by superior and middle pharyngeal branches of pharyngeal trunk of ascending pharyngeal arteries bilaterally. The venous drainage of this dural malformation was via right jugular vein. The patient was treated endovascular route with GDC coils packing in the aneurysmal dilatation of the left cavernous sinus. The result is impressive.

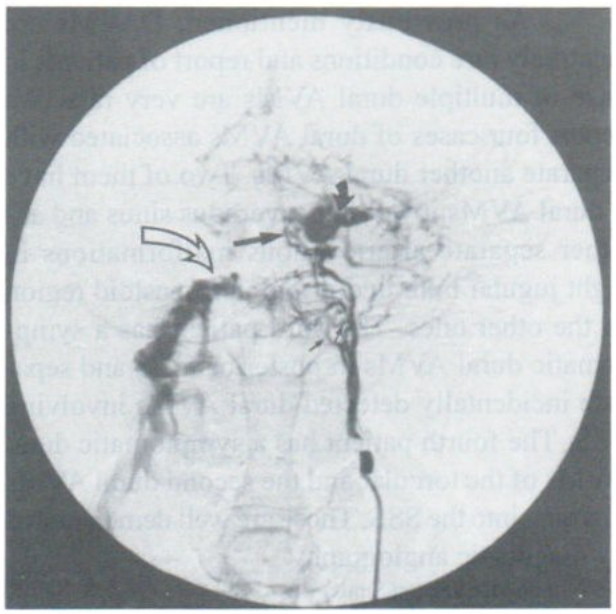

Fig. 1 A

Fig. 1 Case 1. A. Anterosuperior (AP) view of left ascending pharyngeal artery demonstrates left dural carotid-cavernous sinus malformations (curve arrow ➲) and fed by clival branches (arrow ➧) from neuromeningeal trunk. The separate DAVMs in right jugular fossa (curve open arrow ➲) is fed by left pharyngeal branches (small arrows ➧) of ascending pharyngeal artery pass through anastomosis to contralateral sided branches to the AVM and draining to the right jugular vein.

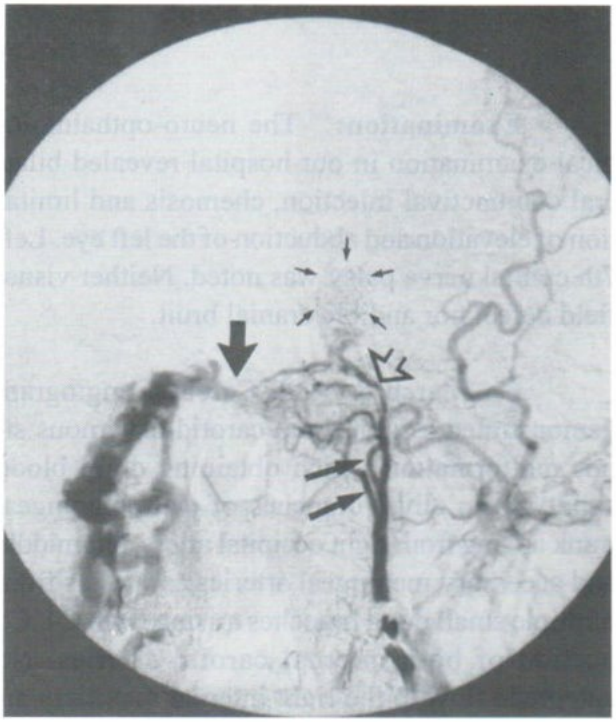

Fig. 1 B. AP view Control angiography of left ascending pharyngeal artery (open arrow ⇧) immediately after embolization of left dural AVMs with GDC coils (small arrows ▲) reveals no longer presence of abnormal venous drainage. The right jugular fossa AVM (large arrow ⬆) is fed by superior pharyngeal and middle pharyngeal (arrows ⬆) branches.

Fig. 1 B

Fig. 1 C

Fig. 1 D

Fig. 1 C and D. AP and Lateral views during right ascending pharyngeal artery injection show a silent AVM at right jugular fossa (open arrow⇧) fed by pharyngeal branch (small arrows▲) and neuromeningeal branch of left ascending pharyngeal artery (curve arrow ➲). The venous drainage is into the right jugular vein (large arrow ⬆).

CASE 2

Clinical course: A 77-year-old woman who first noticed a tinnitus on the right sided of her head in September 1994, approximately nine months after a history of facial trauma. The symptoms then progressed into redness of her left eye, double vision and eventually proptosis of the eye. She underwent cerebral angiogram on October 1994. This study was interpreted as bilateral carotid-cavernous sinus fistula with a small associated parasellar arteriovenous malformation. She was underwent two endovascular treatment attempts which included right external carotid artery embolization with polyvinyl alcohol (PVA) particles on December 30, 1994 and left external carotid artery embolization with PVA particles on February 1, 1995. The patient had some relief of her ocular symptoms such as her double vision, proptosis of left eye. She was referred on March 2, 1995 for evaluation and possible further endovascular treatment.

About the past history of trauma on December 1993, she was falling on her face while walking. She reported no blood loss or any surgical treatment needed.

Examination: The neuro-opthalmological examination in our hospital revealed bilateral conjunctival injection, chemosis and limitation of elevation and abduction of the left eye. Left 7th cranial nerve palsy was noted. Neither visual field defect nor audible cranial bruit.

On March 8, 1995 Cerebral angiogram demonstrated a right dural carotid-cavernous sinus malformation which obtaining dural blood supply from clival branches of neuromeningeal trunk arising from right occipital artery, left middle and accessory meningeal arteries as well as from multiple small dural branches arising from C4, C5 portion of both internal carotid arteries. No antegrade flow to the right internal maxillary artery due to status post previous embolization. The right internal maxillary artery was reconstituted via infraorbital artery and buccal branches of facial artery. Early venous drainage was demonstrated at right cavernous sinus with retrograde filling to the right opthalmic vein and simultaneously antegrade to right inferior petrosal sinus. Another separate arteriovenous malformation was demonstrated at the base of skull in the inferior aspect of left mastoid temporal bone.

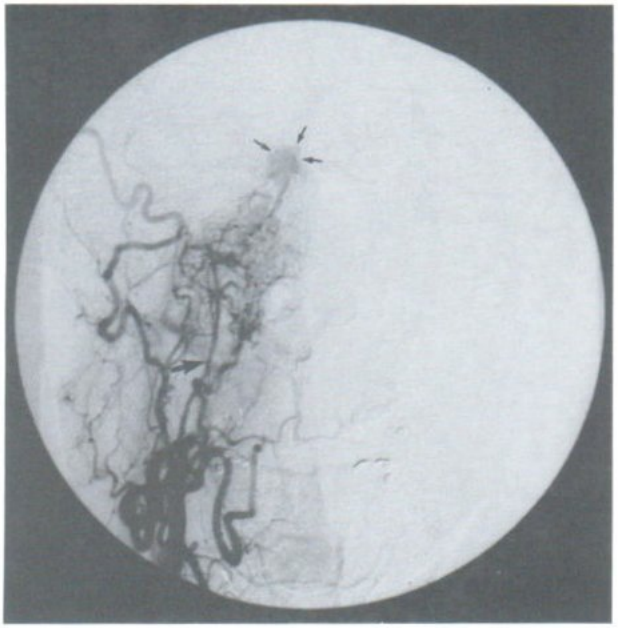

Fig. 2 A

Fig. 2 B

Fig. 2 Case 2, A and B. AP and Lateral view during right external carotid arteriography reveal right dural carotid-cavernous sinus malformations fed by clival branches of neuromeningeal trunk (arrow⬆) from occipital artery. No demonstrable of right internal maxillary artery due to previous embolization, however, filling of infraorbital artery from facial artery via buccal branches (open arrows ⇧) is documented. The venous drainage is into cavernous sinus and inferior petrosal sinus (small arrows ↑) as orderly.

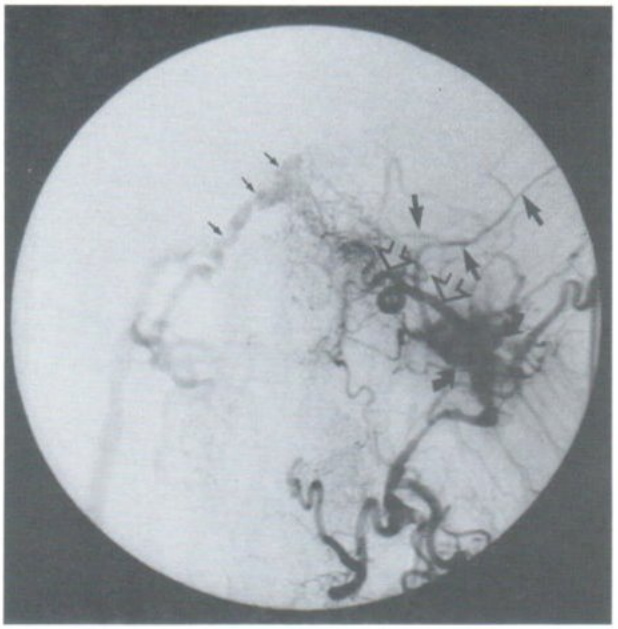

Fig. 2 C

Fig. 2 C. AP view of left external arteriography shows right dural carotid-cavernous malformations fed by left middle meningeal artery (arrows ⬆) and distal internal maxillary artery (open arrows ⇧). The venous drain age is into right cavernous sinus (small arrows ↑). The separate AVM opacified at left occipital area (curved arrows ↄ).

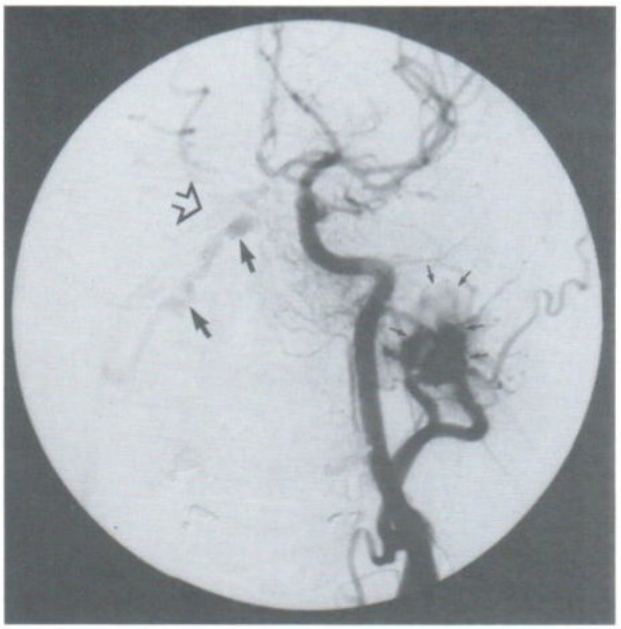

Fig. 2 D

Fig.2 D. AP view of left common carotid arteriography after platinum fibered coils (open arrow ⇧) embolization of anterosuperior compartment of right cavernous sinus demonstrates residual AVMs draining into posteroinferior compartment of cavernous sinus and to inferior petrosal sinus (arrows ⬆). Opacification of separate AVM at basal skull of left occipital bone is noted (small arrows ↑).

CASE 3

Clinical course: A 63-year-old right handed man had a symptoms of headache with nausea and vomiting on November 1995. He was subsequently worked up and disclosed of acute subarachnoid hemorrhage and intraventricular hemorrhage on Computerized tomography scan. The cerebral angiography was underwent from other hospital and demonstrated a posterior fossa dural arteriovenous malformation along the inferior surface of straight sinus which mainly supplied by right occipital artery via transmastoid and transcalvarial branches as well as C5 tentorial branches from bilateral internal carotid arteries. The right and left middle meningeal artery were recruited into the dural AVM via right occipital, squamosal and left parieto-occipital, temporo-occipital and sqaumosal branches respectively. The right ascending pharyngeal artery was involving in this particular case as well. Early venous drainage was well defined in superior vermian vein with further drainage into inferior vermian vein as well as superiorly through posterior mesencephalic vein into the vein of Galen and straight sinus, and anteriorly through lateral mesencephalic, anterior

pontomesencephalic vein and basal vein of Rosenthal into superficial middle cerebral vein.

Another dural AVM was incidentally detected by right internal carotid arteriography and demonstrated dural AVM in superior sagittal sinus (SSS) supplying by right anterior falx artery arising from right anterior ethmoid artery of right opthalmic artery. Early venous drainage was into SSS.

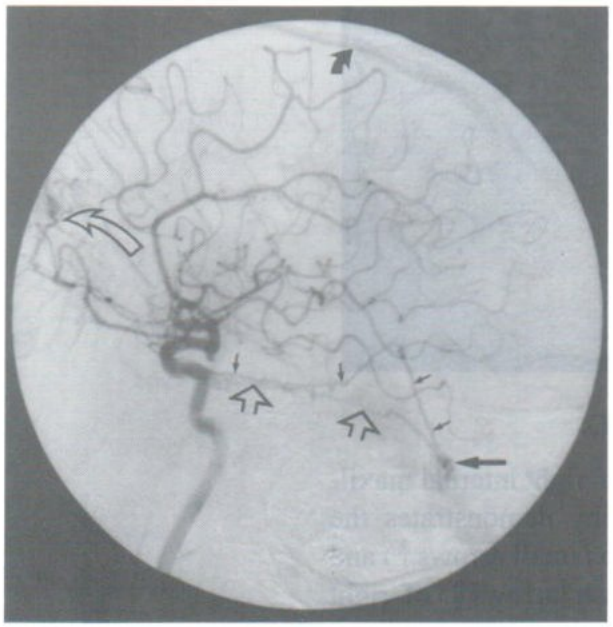

Fig. 3 A

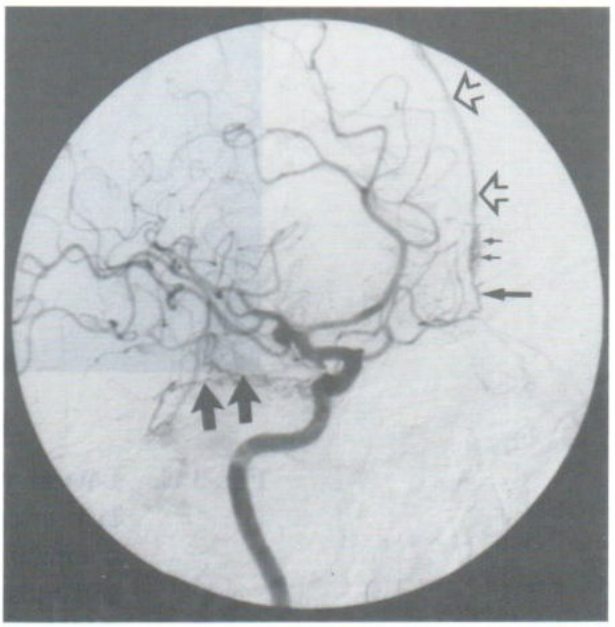

Fig. 3 B

Fig. 3 Case 3, A. Lateral view of right internal carotid arteriography demonstrates multiple dural AVMs, one is fed C5 branches of internal carotid artery as marginal tentorial artery (small arrows ↟) and basal tentorial artery (open arrows ⇧) direct fistulas (arrow ⬆) into the posterior fossa veins and the other is in anterior cranial fossa (curved open arrow ⤺) draining into superior sagittal sinus (curved arrow ↱).

Fig. 3 B. Oblique view of right internal carotid arteriography clearly demonstrates silent dural AVMs (small arrows ↟) supplied by anterior falx artery (arrow ⬆) and draining into the superior sagittal sinus (open arrows ⇧). The tentorial branches from C5 portion of internal carotid artery are noted (large arrows ⬆).

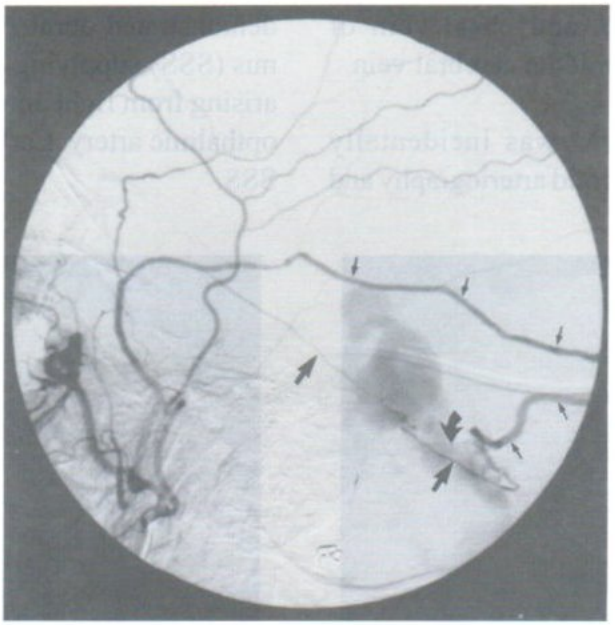

Fig. 3 C

Fig. 3 C. Lateral view of right internal maxillary arteriography demonstrates the occipital branch (small arrows ↑) and squamosal branch (arrows ⬆) of right middle meningeal artery supplying the dural AVMs and draining into the superior vermian vein (curved arrow ↷).

CASE 4

Clinical course: A 27-year-old right handed man who had a car accident in July 1989 with resultant severe closed head injury and coma of four to five weeks. The patient was stable with residual cognitive and gait difficulties. Over the last several months there was a decline in both cognitive and physical functioning. A follow up MRI showed an arteriovenous malformations. The cerebral angiogram demonstrated the multiple dural AVMs, the extensive one involving the torcular and peritorcular regions which supplying by bilateral occipital arteries via transmastoid branches, posterior meningeal branches of right vertebral artery, dural branches of posterior cerebral arteries, the tentorial branches from right internal carotid artery and petrosal branches of bilateral middle meningeal arteries. The venous drainage was into the torcula, left transverse sinus, left sigmoid sinus in orderly fashion. There was severe venous hypertension seen after injection of internal carotid arteries and vertebral arteries as shown by the prominence of cortical veins without filling of the deep venous system. The second dural AVMs was a small arteriovenous shunting at SSS which fed by superficial temporal artery and drained into SSS directly.

Fig. 4 A

Fig. 4 B

Fig. 4 Case 4, A. AP view of right occipital arteriography shows extensive supply of right occipital artery via transmastoid branches (small arrows ↑) and draining into torcular (arrow ⬆).

Fig. 4 B. Lateral view of left external carotid arteriography demonstrates multiple dural AVMs, symptomatic one (open arrow ⇧) is fed by squamosal branches (arrow ⬆) of middle meningeal artery. The second dural AVMs involving SSS curved open arrow ⤴) is fed by superficial temporal artery (arrowhead ▲).

DISCUSSION

Anomalous communication between dural arteries and venous system occurring in the absence of significant trauma have been described by a number of authors as dural arteriovenous malformations. Approximately 10-15% of intracranial arteriovenous malformations were in dural origin [1]. Mostly located in the cavernous sinus and transverse-sigmoid sinuses. In large series of Aminoff, et al. (1973) the lesion located in the former area in 12% and the latter in 62% as well as Lasjuanias, et al. (1986) reported 191 cases with cavernous lesion in 19% and lateral sinus in 67%. The etiology of dural arteriovenous malformations (DAVMs) is not exactly known. The convincing evidences that have recently been presented and suggested at least some of them are acquired in origin resulting from the dural venous sinus thrombosis.[2,4,5,9] We reported four cases of multiple dural AVMs. Two of them had dural AVMs occurring in cavernous sinus and associated with separate AVM in the basal skull. Both of them presented to us as classical cases of dural AVMs of carotid-cavernous sinus region that occurred in elderly woman with neuro-opthalmological signs and symptoms of slow-flow low-pressure shunting such as conjunctival hyperemia, proptosis, and double vision.[10,15-17,19-21,23-25] According to the angiographically classification proposed by Barrow, et al. (1985), which classified[1] carotid-cavernous sinus fistula into 4 categories and the most

common type is type D which obtaining meningeal supply from both external and internal carotid arteries.[16] Our patients, both of them were in type D as described in CCFs of Barrow & classification. The first patient had received conservative treatment for five months before comimg to hospital. It was probably reasonable to conservative treatment at that time because of mild symptoms and possibilities of spontaneous regression. The incidence of spontaneous regression or cure was varied from series to series such as 3 of 20 cases (15%) in Vinuela & series, 5 of 14 cases (36%) of Barrow & series, and 3 of 37 cases (8%) in Debrun & series [15-17]. Although, there was no clinical signs and symptoms of hazardous CCFs, we decided to treat this patient immediately due to her angiographic features associated with risk of morbidity and mortality. Those features were aneurysmal dilatation of left cavernous sinus and venous drainage into cortical veins of posterior fossa which can produce aggressive neurological deficits. Multimodalities in treatment dural CCFs have been reported.[15-20,22,24-27] Surgical approaches in treatment dural CCFs were arterial feeders ligation, placement of thrombogenic material into the sinus to promote thrombosis and closure.[18,19,22] Recently, transarterial embolization have been described for alleviation symptoms or even cure lesions.[15-17,20,27] Manual compression therapy have been reported as a safe technique in selected cases with cure rate 17% in direct CCFs and 30% in indirect CCFs.[24] More recently transfemoral transvenous embolization has been used as primary method for cure dural CCFs and other dural AVMs.[25,26] We chose GDC coils as embolic material in this patient due to its opacity, thrombogenicity, accurate disposition in proper site and no movement of coils during detachable. The results both angiographically and clinically are impressive to us.

Considering vascular anomalies that associated with Dural AVMs, it is somewhat rare and was reported in the literatures as multiple DAVMs,[2,9,27-31] cerebral AVM,[32,38,39] Rendu Osler Weber disease,[10] arterial aneurysm.[10] In term of multiple DAVMs it is surprising that there are only seven reports with nine cases of multiple DAVMs documented.[2,9,27-31] In our study, we present four cases of multiple DAVMs which occurred in different locations such as cavernous sinus, straight sinus, torcular, SSS, and base of skull which varying from case to case. Basically, they can occur anywhere along the dura mater. The pathophysiology, natural history and treatment of which are similar to the usual DAVMs. It is considered to be an acquired lesion originating from recanalized blood clot clarifying by histological sections[9] and some cases do have a spontaneous regression [28]. The association of a dural AVM and a brain AVM was first reported by Tamaki in 1971. In that patient, Tamaki described AVM involving the scalp as well as dura, retina, cerebrum and posterior fossa.[32] Then, two additional reports were followed by Willinsky[36] and Schlacter.[37] The Dural AVM was also noted in patient with Rendu Osler Weber disease.[10] The Rendu Osler Weber disease is charactered by a triad of mucocutaneous and visceral telangiectasia, recurrent epitaxis and familial history.[33] The central nervous system involvement in this disease is common causing by pulmonary arteriovenous fistula (cerebral hypoxemia, septic emboli and brain abscess), vascular malformations of the brain, spinal cord and porto-systemic encephalopathy respectively.[34,35] Another disease that having separate AVMs involved central nervous system is retinocephalic vascular malformations (Wyburn-Mason sydrome). The Wyburn-Mason sydrome composed of arteriovenous malformations of one or both sides of midbrain with ipsilateral or bilateral arteriovenous malformation of the retina and cutaneous nevi. Brain AVMs in this disease usually follow the optic tracts and optic nerves.[38,39] As previously mentioned, Tamaki reported one case of AVM involving scalp, dura, retina, cerebrum and posterior fossa which possibility of unilateral retinocephalic disease is considered. Dural AVM also associated with arterial aneurysm.[10] This study presents four additional cases of multiple AVMs,

CCFs = Carotid-Cavernous Fistulas

two cases of those which has a dural AVM associated with a second dural AVMs occupying in the base of skull, one is in the anterior aspect of right jugular foramen and the other is in the base of skull of left occipitomastoid bone. These cause no symptoms with incidentally found during cerebral angiographic procedures. We have never seen dural AVM associated with separate AVMs in the base of skull areas.The other two cases have the second silent DAVMs in the superior sagittal sinus. All cases are unrelated to any known disease processes or familial preponderance. We believe that multiple DAVMs found in the same patients without associated vascular disease are rare and very interesting.

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