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De novo formation of remote dural arteriovenous fistula following treated cavernous sinus dural arteriovenous fistula

Prasert Iampreechakul [a,*], Korrapakc Wangtanaphat [a], Songpol Chuntaroj [b], Chonlada Angsusing [b], Yodkhwan Wattanasen [b], Sunisa Hangsapruek [b], Punjama Lertbusayanukul [b], Somkiet Siriwimonmas [c]

[a] Department of Neurosurgery, Neurological Institute of Thailand, Bangkok, Thailand
[b] Department of Neuroradiology, Neurological Institute of Thailand, Bangkok, Thailand
[c] Department of Radiology, Bumrungrad International Hospital, Bangkok, Thailand

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ABSTRACT

Background: The development of new dural arteriovenous fistulas (DAVFs) at another location following endovascular treatment of cavernous sinus DAVFs (CSDAVFs) are extremely rare. Our aim is to review cases of de Novo DAVFs that occurred after treatment of CSDAVFs at our institution and those reported in the literature.
Methods: We reviewed all cases of CSDAVFs evaluated by 2 experienced neuroradiologists. A literature search was performed using the PRISMA (preferred reporting items for systematic reviews and meta-analyses) guidelines focusing on De Novo DAVFs following the endovascular treatment of cerebrovascular malformations. Addition articles were searched through the reference lists of the included articles.
Results: From June 2004 and September 2019., we identified 3 (2.5%) cases of De Novo DAVFs occurred after endovascular treatment or spontaneous obliteration of CSDAVFs from 119 treated CSDAVFs at our institute. Our review yielded 9 articles involving 12 patients with 15 de novo DAVFs, including our 3 patients. The mean age was 55.08 ± 12.9 years (range 43–69), 83.3% were females (n = 10). The new remote DAVFs occurred after endovascular treatment of CSDAVFs in 10 (83.3%) patients. The de novo DAVFs occurred following spontaneous complete regression in 2 (16.7%) patients. All de novo DAVFs developed after complete obliteration of treated CSDAVFs.
Conclusion: Sinus thrombosis and elevated venous pressure may play an important role in the pathogenesis of a de novo DAVF formation. In addition, thrombophilic abnormalities and the use of contraceptives may contribute to sinus thrombosis, leading to the development of the second remote DAVF after treatment of CSDAVFs.

1. Introduction

Cavernous sinus dural arteriovenous fistulas (CSDAVFs), the second most common location of intracranial DAVFs, are abnormal arteriovenous communications between dural arteries and the CS. The etiology of most CSDAVFs remains unclear, but they may be acquired lesions following trauma, venous sinus thrombosis, and previous craniotomy.[1] CSDAVFs commonly appear in middle-aged female and symptoms of the patients usually relate to venous drainage patterns of the CS. Symptoms and signs of CSDAVFs are usually insidious in onset and often mild and less severe than direct carotid-cavernous fistulas, including redness of the eye, proptosis, chemosis, bruit, retro-orbital pain, unilateral headache, diplopia, dilated conjunctival veins, transient sixth-nerve palsy, elevated intraocular pressure, and/or diminished visual acuity.[2] Arterial supplies may arise from dural branches of the cavernous segment of the internal carotid artery (ICA), i.e., the meningohypophyseal trunk and/or inferolateral trunk, and/or the external carotid artery (ECA), i.e., the middle meningeal artery (MMA), accessory meningeal artery, artery of foramen rotundum, and/or ascending pharyngeal artery.[3] Spontaneous regression of CSDAVFs is rare and usually occurs in benign fistulas.[4] Due

* Corresponding author. Department of Neurological Surgery, Neurological Institute of Thailand, 312 Ratchawithi Road, Khwaeng Thung Phaya Thai, Bangkok 10400, Thailand.
E-mail addresses: bangruad@hotmail.com (P. Iampreechakul), win0000000@gmail.com (K. Wangtanaphat), joesongpol@hotmail.co.th (S. Chuntaroj), chonlada.angs.ra@gmail.com (C. Angsusing), slainooo@hotmail.com (Y. Wattanasen), hsunisa@gmail.com (S. Hangsapruek), lpunjama@hotmail.com (P. Lertbusayanukul), somkietsiri@hotmail.com (S. Siriwimonmas).

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to the risk of embolic complications associated with transarterial embolization of CSDAVFs, transvenous approach through the inferior petrosal sinus (IPS) using coils and/or liquid embolic materials is the first-line therapy of the vast majority of CSDAVFs.[5]

The development of new DAVFs at another location following endovascular treatment of CSDAVFs is extremely rare.[6–8] The pathogenesis of the newly developed DAVFs following the treatment of CSDAVFs remains unknown. The authors reviewed patients of de novo DAVFs following the treatment of CSDAVFs at our institution and in the literature. The mechanisms of the development of the second remote DAVF are discussed.

2. Methods

2.1. Patient enrollment and data collection

The authors retrospectively analyzed patients of CSDAVFs treated at our institute between June 2004 and September 2019. We searched for patients who developed de novo DAVF after endovascular treatment or during follow-up. Information was collected from the patient chart records, and imaging studies (including computed tomography (CT) scan, magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and cerebral angiography. Imaging studies were evaluated by 2 experienced neuroradiologists (Y. W. and S. H.).

2.2. Endovascular procedure

All procedures were performed under general anesthesia. Complete cerebral angiography, including bilateral ECAs, ICAs, and vertebral artery (VA), were performed for every patient. Transfemoral transvenous embolization using fibered coils through the IPS was the first-line method in our institution. The microcatheter was navigated into the CS under roadmap guidance. After embolization, all patients obtained follow-up cerebral angiography at 2–6 months. In case of incomplete obliteration of fistula, another angiography was performed at 6 months later. In case of complete obliteration of fistula, the clinical follow-up was performed every 3–6 months for 2 years, and annually thereafter. If the patient developed any symptoms, such as tinnitus, cerebral angiography or magnetic resonance angiography will be carried out.

2.3. Literature review

Using the PRISMA (preferred reporting items for systematic reviews and meta-analyses) guidelines, we reviewed the previously reported case and series which have enough clinical description and clearly demonstrated figures of de novo DAVF following endovascular treatment of CSDAVFs. A literature search was performed using the Ovid MEDLINE, Pubmed, Cochrane Database of Systemic Reviews, and Google Scholar with the following key words of “cavernous sinus dural arteriovenous fistulas”, “de novo development of dural arteriovenous fistulas”, and “endovascular treatment”. Exclusion criteria included non-English-language articles, commentaries, and information from expert opinions. Addition articles were searched through the reference lists of the included articles.

2.4. Data analysis

Patients at our institute and those reported in the literature were reviewed in detail. The collected data in this review included patient demographic data (i.e., patient gender and age), initial location of CSDAVFs, the presence of sinus occlusion prior to the development of de novo DAVF, approach route and treatment method of the fistulas, the interval time between complete treatment and the de novo DAVF identification, de novo location of the fistulas, treatment of the de novo fistulas, and angiographic outcome of the new remote fistulas.

The SPSS 16.0 software package (SPSS Inc, Chicago, Illinois, USA) was used for analysis. Continuous data were expressed as mean (SD), and categorical variables are presented as numbers and corresponding percentages.

3. Results

One hundred and nineteen patients with CSDAVFs treated between June 2004 and September 2019 were reviewed. We identified three patients who developed de novo DAVFs after endovascular or during on follow-up that were not present before treatment (Table 1). The fistulas developed after endovascular treatment of CSDAVFs in 2 patients and following spontaneous regression of the CSDAVF in 1.

3.1. Case illustration 1

A 54-year-old man presented with headache, right proptosis, and double vision for 3 days. There was no history of trauma. MRI of the brain showed abnormal bulging of the left CS with dilated left superior ophthalmic vein (SOV). Cerebral angiography showed the right CSDAVF supplied by ipsilateral and contralateral branches of the ICA and ECA with drainage into the right superior petrosal sinus, IPS, and SOV. In addition, marked narrowing of the right transverse and sigmoid sinuses, particularly at the proximal part of the right transverse sinus, was observed (Fig. 1). Under general anesthesia, transvenous embolization with fibered coils via the right IPS was performed with the result of incomplete obliteration. His symptoms gradually improved after embolization. Follow-up cerebral angiography, obtained 2 months following embolization, demonstrated nearly complete regression of the right CSDAVF. Seven months later, the patient complained of tinnitus in his left ear and cerebral angiography revealed the disappearance of the right CSDAVF and new DAVF at the left sigmoid sinus supplied by bilateral occipital arteries (OAs), left MMA, posterior auricular artery, meningohypophyseal trunk (MHT) of the left ICA and muscular branches of the left VA (Fig. 2A-G). Furthermore, the venous phase of the right ICA injection disclosed venous stasis at the distal end of the right transverse sinus (Fig. 2H). His protein C was low, at 20% (normal range: 70–140%), but other Hypercoagulability tests were within normal ranges. The patient was diagnosed with protein C deficiency and received anticoagulant drug. Follow-up imaging studies, including cerebral angiography and MRA, demonstrated gradual regression of the second fistula until complete resolution during a long-term period (Fig. 2I).

3.2. Case illustration 2

A 48-year-old woman was admitted to the local hospital due to sudden headache following by generalized seizure and loss of consciousness. One week ago, she noticed redness in her left eye. She had used oral contraceptives for a total of 22 years. Hypercoagulability tests, such as lupus anticoagulant, protein C, protein S, antiphospholipid antibodies, antithrombin III, homocysteine levels anti-cardiolipin autoantibodies IgG, and anti-double–stranded deoxyribonucleic antibodies, were within normal ranges. CT scan of the brain shows intracerebral hemorrhage at left rectus gyrus. Cerebral angiography revealed the left CSDAVF fed by ipsilateral and contralateral branches of the ICA and ECA. The shunt drained into the left SOV, IPS, superficial middle cerebral veins, and frontal cortical veins. Another drainage route filled into the right CS through the intercavernous sinus and subsequently drained into the right frontoparietal cortical veins (Fig. 3). The left CSDAVF was successfully treated by transvenous embolization with fibered coils via the left IPS (Fig. 4A and B). At the 1-month follow-up, she had no evidence of residual symptoms. Follow cerebral angiography, obtained 6 months following embolization, confirmed complete obliteration of the left CSDAVF and new left distal transverse sinus DAVF supplied by the left MMA, posterior auricular artery, MHT, and muscular branches of the left VA (Fig. 4C–I). She had no tinnitus and received no additional

Table 1
Summary of patients harboring De Novo DAVF following the treatment of CSDAVFs at our institute.

Patient	Gender/ Age	Initial location	Sinus stenosis or occlusion	Treatment	Approach route	Time interval (months)	De Novo location	Treatment	Angiographic outcome
1. Figs. 1 and 2	M/54	Rt. CS	+	TVE	Rt. IJV-IPS	7	Lt. SS	Anticoagulant drug	CR
2. Figs. 3 and 4	F/48	Lt. CS	–	TVE	Lt. IJV-IPS	6	Lt. TS	None	Remaining
3. Figs. 5 and 6	F/56	Rt. CS	–	None	–	25	Rt. SS	None	Remaining

CR: complete remission; CSDAVF: cavernous sinus dural arteriovenous fistula; F: female; M: male; SS: sigmoid sinus; TS: transverse sinus; TVE: transvenous embolization.

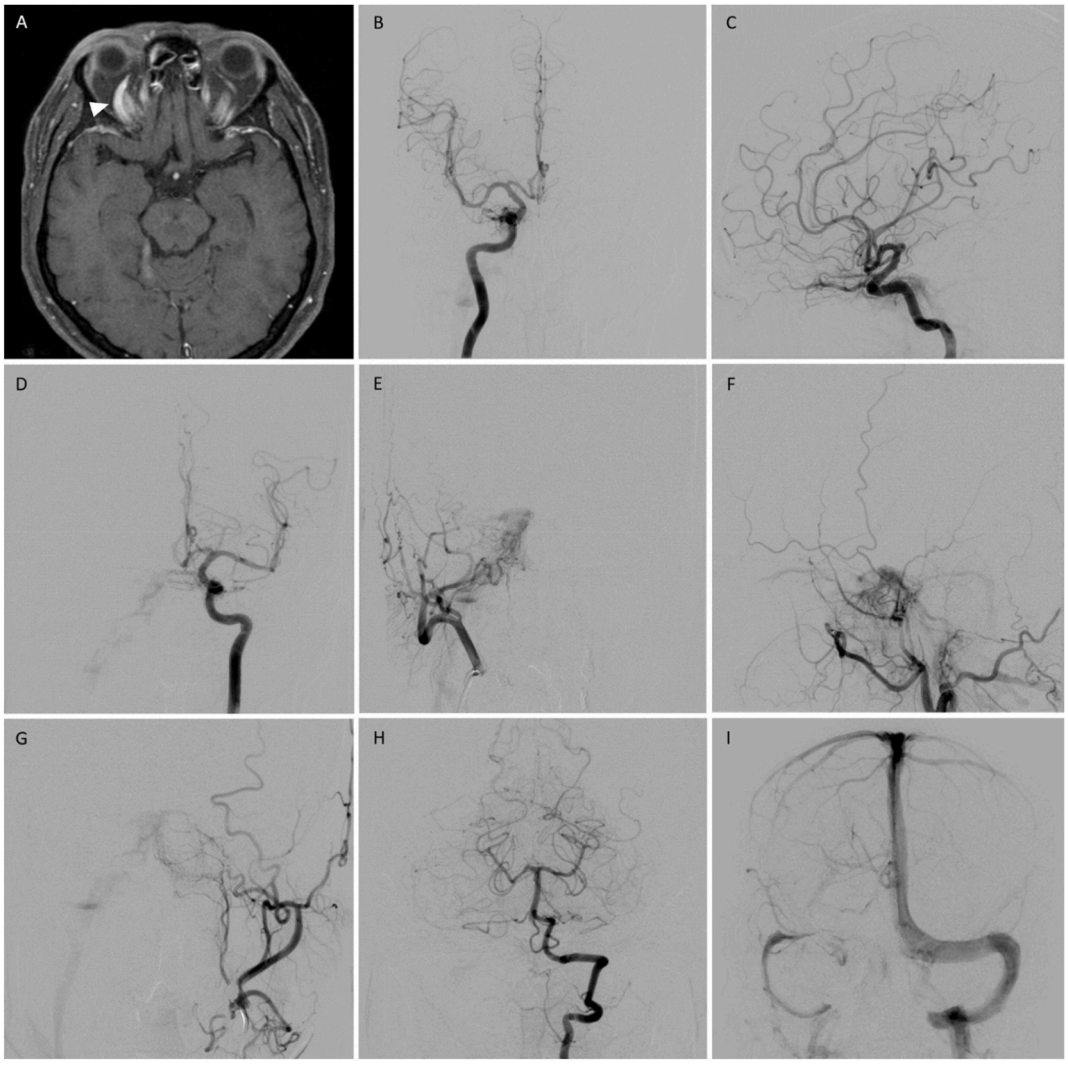

Fig. 1. Case illustration 1. (A) Axial fat suppressed contrast-enhanced T1-weighted magnetic resonance imaging shows an enlarged right superior ophthalmic vein (SOV) (arrowhead). (B) Anteroposterior (AP) and (C) lateral views of the right internal carotid artery (ICA), (D) AP view of the left ICA, (E) AP and (F) lateral views of the right external carotid artery (ECA), and (G) AP view of the left ECA reveal the right cavernous sinus dural arteriovenous fistula supplied by ipsilateral and contralateral branches of the ICA and ECA with drainage into the right superior, inferior petrosal sinuses and SOV. (H) AP view of the left vertebral artery demonstrates no abnormality. (I) AP view of venous phase of the left ICA injection shows markedly narrowing of the right transverse and sigmoid sinuses, particularly at the proximal part of the right transverse sinus.

treatment. Follow-up cerebral angiography, obtained 2 years after detection of the de novo fistula, showed that the fistula remained stable.

3.3. Case illustration 3

A 56-year-old woman suffered from right-sided headache for 2 months. One month later, she developed right red eye and proptosis. She had used contraceptive injections for a total of 25 years. Hypercoagulability studies were within the reference ranges. Cerebral angiography, obtained 1 month following her ocular symptoms, showed the right CSDAVF supplied by the right artery of the foramen Rotundum, MMA, and inferolateral trunk with drainage into the right SOV (Fig. 5A–C). Due to improvement of her symptoms, she refused endovascular treatment. Cerebral angiography, obtained 1 year after initial angiography,

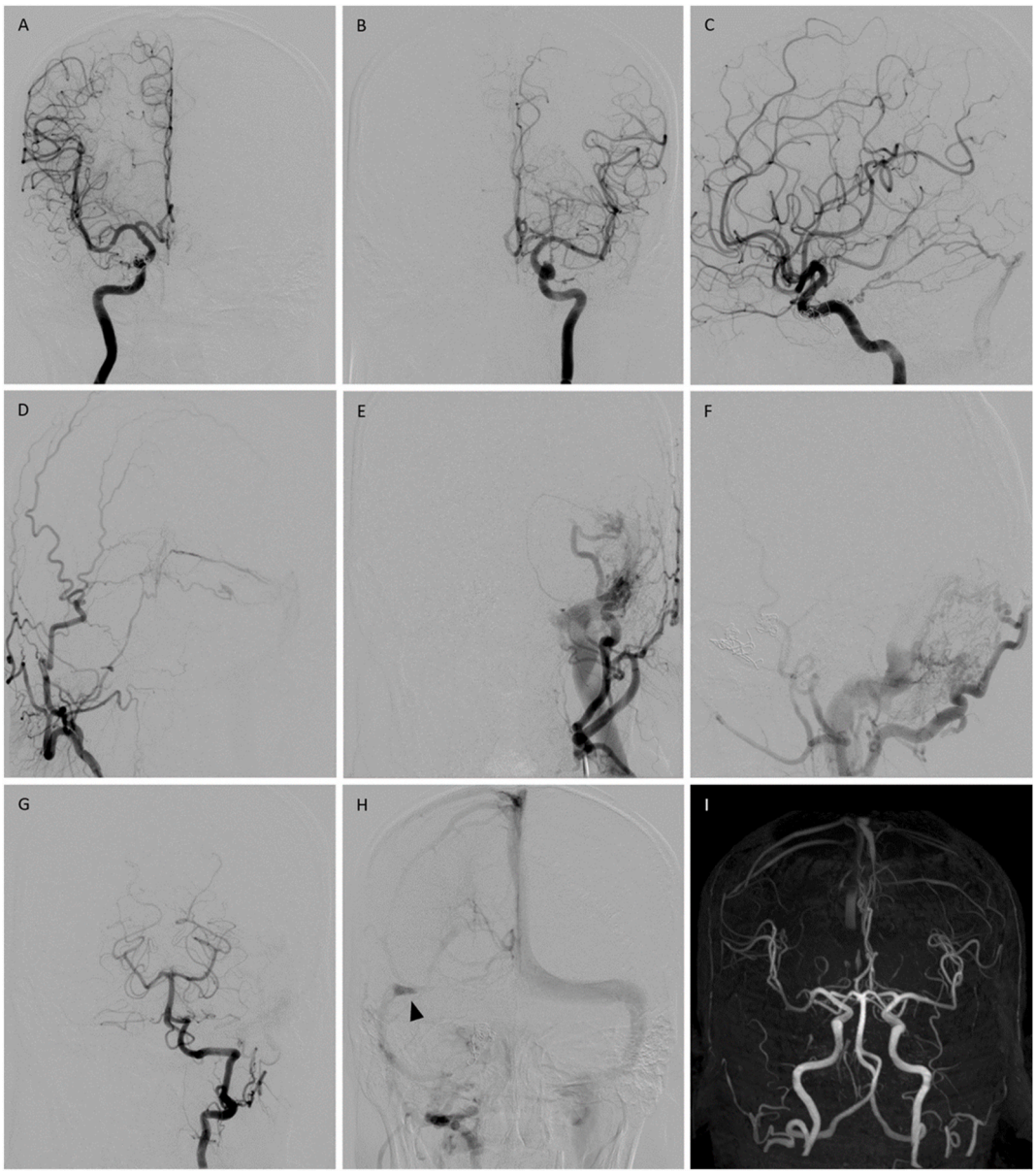

Fig. 2. Case illustration 1. Cerebral angiography obtained 9 months after embolization. (A) Anteroposterior (AP) view of the right internal carotid artery (ICA), (B) AP and (C) lateral views of the left ICA, AP views of the (D) right and (E) left external carotid arteries (ECAs), (F) lateral view of the left ECA, and (G) AP view of the vertebral artery (VA) reveal complete obliteration of the right cavernous sinus dural arteriovenous fistula (DAVF) and de novo left sigmoid sinus DAVF supplied by bilateral occipital arteries, left middle meningeal artery, posterior auricular artery, meningohypophyseal trunk of the left cavernous ICA and muscular branches of the left VA. (H) AP view of late venous phase of the right ICA injection demonstrates venous stasis (arrowhead) at the distal end of the right transverse sinus. (I) AP view of the cranial MRA obtained 11 years after medical treatment confirms no recurrent fistula.

confirmed spontaneous complete obliteration of the fistula (Fig. 5D–F). Twenty-five months later, she noticed tinnitus in her right ear. Follow-up cerebral angiography revealed de novo right sigmoid sinus DAVF supplied by the right MMA, posterior auricular artery, OA, and MHT with antegrade drainage into the right internal jugular vein (Fig. 5G–I). Comparison with initial angiography, there was filling defect at left proximal transverse sinus suspected (Fig. 6). The neurologist was consulted for role of anticoagulation, but he suggested no role of any anticoagulant use for this patient. The patient received no additional treatment because of no cortical venous reflux and was scheduled for follow-up cerebral angiography or MRA annually.

3.4. Literature review

A total of 39 articles were identified. All studies were screened based on their titles and abstracts, and non-relevant studies ($n = 25$) were excluded. The remaining articles ($n = 14$) were then fully read for eligibility. Finally, 9 studies met the eligibility criteria for the final review (Fig. 7).

From the literature review, there were 12 patients, including our 3 patients, with 15 de novo DAVFs due to two new lesions in 3 cases (Tables 1 and 2).[6–13] Ten (83.3%) females and 2 (16.7%) males with a mean age was 55.08 ± 9.51 years, range 43–69 years, were included in this review. The CSDAVFs were right sided in 3 (25%) patients and left sided in 9 (75%). The presence of sinus occlusion occurred before development of de novo DAVFs in 3 (25%) patients.

The new remote DAVFs occurred after endovascular treatment of CSDAVFs in 10 (83.3%) patients. Of those patients, seven (58.3%) CSDAVFs were treated with transvenous embolization, one (8.3%) with transarterial embolization, and 2 (16.7%) with combination of

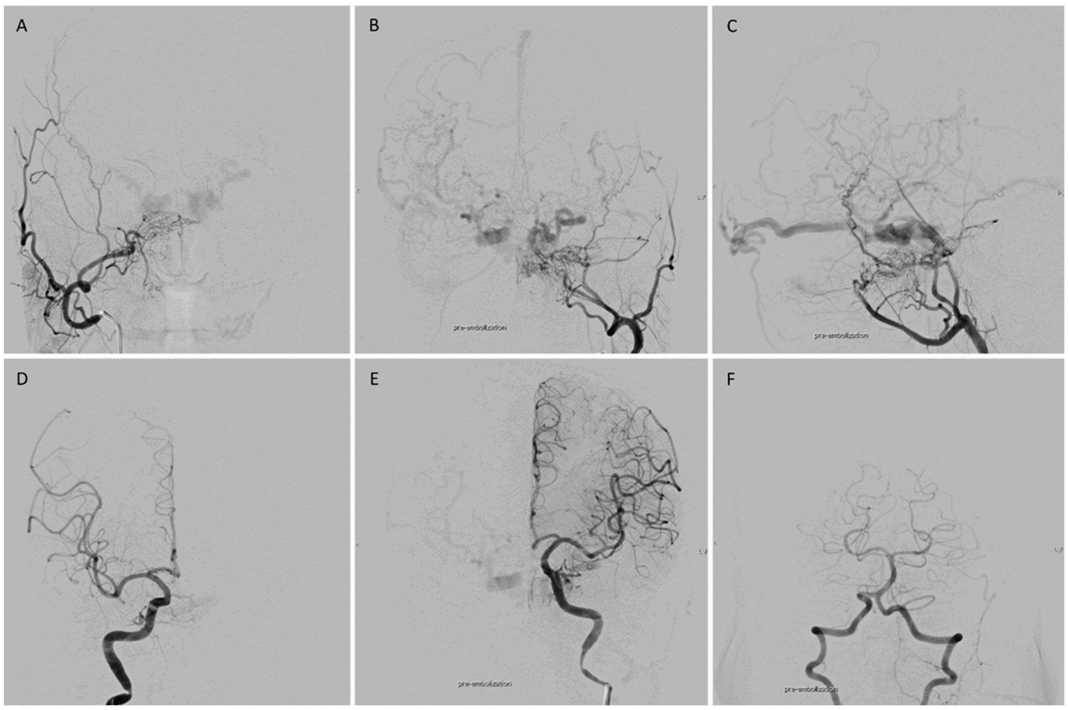

Fig. 3. Case illustration 2. Anteroposterior (AP) views of the (A) right and (B) left external carotid arteries (ECAs), (C) lateral view of the left ECA, and AP views of the (D) right and (E) left internal carotid arteries (ICAs) injections reveal the left cavernous sinus dural arteriovenous fistula fed by ipsilateral and contralateral branches of the ICA and ECA with drainage into the left superior ophthalmic vein and bilateral cortical venous reflux. (F) AP view of the left vertebral artery shows no abnormality.

transarterial and transvenous treatments. The de novo DAVFs occurred following spontaneous complete regression in 2 (16.7%) patients.

All de novo DAVFs developed after complete obliteration of treated CSDAVFs. The mean interval time from complete obliteration of CSDAVFs to the diagnosis of de novo DAVFs was 11.21 ± 9.94 months, range 4–36 months. Most de novo fistulas involved transverse sinus, sigmoid sinus, and/or internal jugular vein on the ipsilateral side of CSDAVFs. Two de novo fistulas occurred on the contralateral side of CSDAVFs. By transvenous approach, seven patients were treated via IPS through internal jugular vein, and two via SOV only.

The de novo DAVFs were treated with endovascular treatment and/or surgery in 6 patients, and anticoagulant therapy in one patient associated with protein C deficiency. Eight de novo DAVFs were left untreated. The Angiographic outcome of these fistulas was complete regression in 10 fistulas, remaining in 2, and no available data in 3.

4. Discussion

Endovascular treatment, especially by transvenous approach, has been considered the most effective method for CSDAVFs.[2] However, this technique is not without any risks. The development of a remote DAVF after endovascular treatment of CSDAVF has been reported infrequently.[6,8,11,12] In our review, we found that de novo DAVFs could develop following spontaneous resolution of CSDAVFs.

4.1. The pathogenesis of the de novo DAVF after treated CSDAVFs

The pathogenesis of multiple cranial and spinal DAVFs remains speculative.[9,15] Based on the theory about the formation of cranial DAVFs proposed by Miyachi et al.,[16] neovascularization and vessel dilatation were induced by local inflammation near the area of the emissary draining veins, and subsequent arteriovenous connection occurred on the arteriole level. Additionally, the maturation of the fistula subsequently developed following the occlusive change of the venous draining system. In our review, de novo fistulas developed in only a few patients having venous sinus occlusion.

The injury to the sinus wall during endovascular procedures by the catheter and/or guidewire may have provoked the development of dural AVF.[12] However, some de novo fistulas occurred in the new location contralateral to the first following endovascular treatment or developed after spontaneous regression of CSDAVFs, as shown in our 2 cases and few cases in the literature.[7,9]

In a rat model, Herman et al.[17] demonstrated the role of sinus thrombosis and elevated sinus pressures in the pathogenesis of DAVFs. Subsequently, the study of angiogenesis in pathogenesis of DAVFs in rat model by Lawton et al.[18] indicated that venous hypertension may induce angiogenesis activity either directly or indirectly by reducing cerebral perfusion and producing ischemia, leading to the formation of new fistula. In our opinion, venous thrombosis and/or hypertension of the involved sinus may play an important role in the development of a second DAVF on the contralateral side or after the resolution of the original CSDAVF. The shunt flow of the original fistula may cause the turbulence and reversal of the normal venous flow, probably resulting in thrombosis and/or venous hypertension away from the original shunt, leading to the formation of the de novo fistula. Venous stasis represents a sign of venous thrombosis in angiography, as shown in our one case.[19]

Interestingly, we found our one case of de novo development of DAVF associated with cerebral venous thrombosis and protein C deficiency in the present study. Protein C is a vitamin-K dependent plasma protein, a natural anticoagulant protein, and has an essential role in the regulation of thrombosis and hemostasis in humans. The etiology of protein C deficiency may be inherited or acquired.[20,21] Interestingly, thrombophilic factors may play a role in venous thrombosis, leading to the formation of cranial DAVFs.[22] A deficiency in protein C or S can cause a hypercoagulable state with increased risk of thrombosis.[23,24] The incidence of thrombophilic abnormalities has a wide range between different ethnic groups. Genetic thrombophilic abnormalities occurred in a higher percentage in Caucasian population. In Thai patients, deficiencies in protein C, protein S, and antithrombin were found in 25.9% of the patients. The most common type of deficiency was protein S

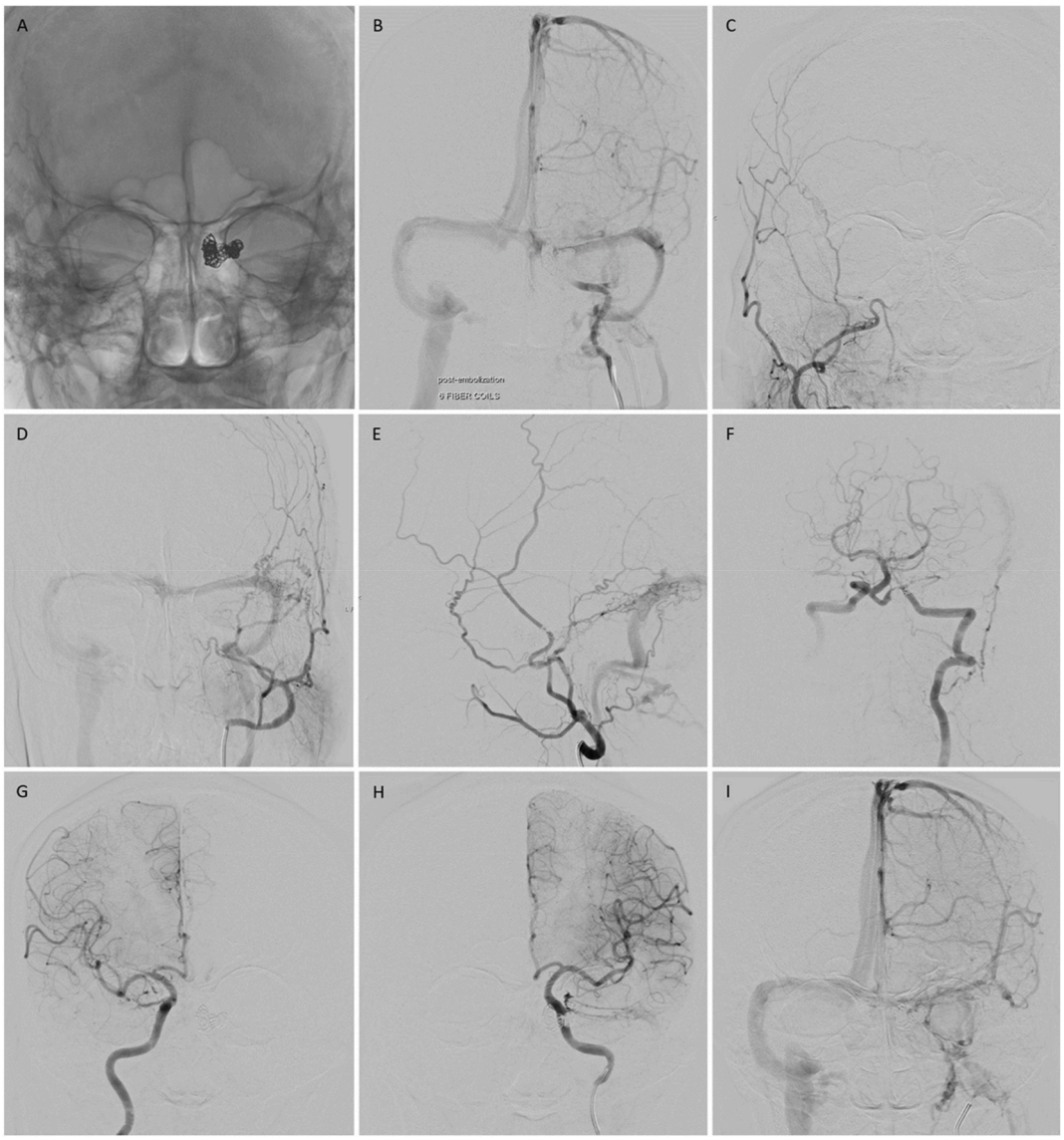

Fig. 4. Case illustration 2. (A) Anteroposterior (AP) view of unsubtracted image demonstrates the coil mass at the left CS. (B) AP view of venous phase of the left internal carotid artery (ICA) injection shows normal sinus drainage after transvenous coil embolization. AP views of the (C) right and (D) left external carotid arteries (ECAs), (E) lateral views of the left ECA, (F) AP view of the left vertebral artery (VA), and AP views of (G) right and (H) left ICAs injections obtained 6 months after embolization reveal complete obliteration of the left cavernous sinus dural arteriovenous fistulas (CSDAVF) and de novo left distal transverse sinus DAVF supplied by the left middle meningeal artery, posterior auricular artery, meningohypophyseal trunk, and muscular branches of the left VA. (I) AP view of venous phase of the left ICA injection obtained 6 months after embolization shows no venous drainage into the left transverse-sigmoid sinuses.

deficiency, followed by protein C and antithrombin deficiencies.[25–27] Therefore, Thai patients harboring multiple cranial DAVFs may be considered for a thrombophilia workup.

Another our two cases had prolonged use of contraceptives. The use of oral contraceptives was the common acquired risk factor for venous thromboembolism, including cerebral venous thrombosis, in the Thai patients.[26] Oral contraceptive use seems to increase the risk of cerebral venous sinus thrombosis in women of reproductive age 7-fold when compared to those not using this method of contraception. Future studies are required to answer whether duration and type of hormonal contraceptive use modifies the risk cerebral venous thrombosis formation.[28,29] The risk of cerebral venous thrombosis is further increased in oral contraceptive-using women with thrombophilic abnormalities.[30] We speculated that long-term use of contraceptives may cause cerebral venous thrombosis, leading to the occurrence of remote de novo DAVFs following treated CSDAVFs.

4.2. Management strategy of the de novo DAVFs

In the present study, we found that the mean interval time from complete obliteration of CSDAVFs to the diagnosis of de novo DAVFs was 11.21 ± 9.94 months, range 4–36 months. Therefore, Clinical and imaging follow-up should be performed at least 1 year after the complete resolution of CSDVAVFs. As mentioned earlier, patients with metachronous occurrence of multiple DAVFs should be tested for thrombophilia. Patients with thrombophilic abnormalities should be considered for treatment with long term anticoagulation therapy.[31]

Most de novo fistulas were benign course, characterized by fistula without cortical venous reflux. Therefore, a conservative approach may be the initial treatment of choice.[32] Patients harboring small de novo fistulas with a minimal flow can safely be observed. Endovascular treatment should be performed in patients harboring aggressive fistulas with cortical venous drainage or patients with intolerable symptoms.

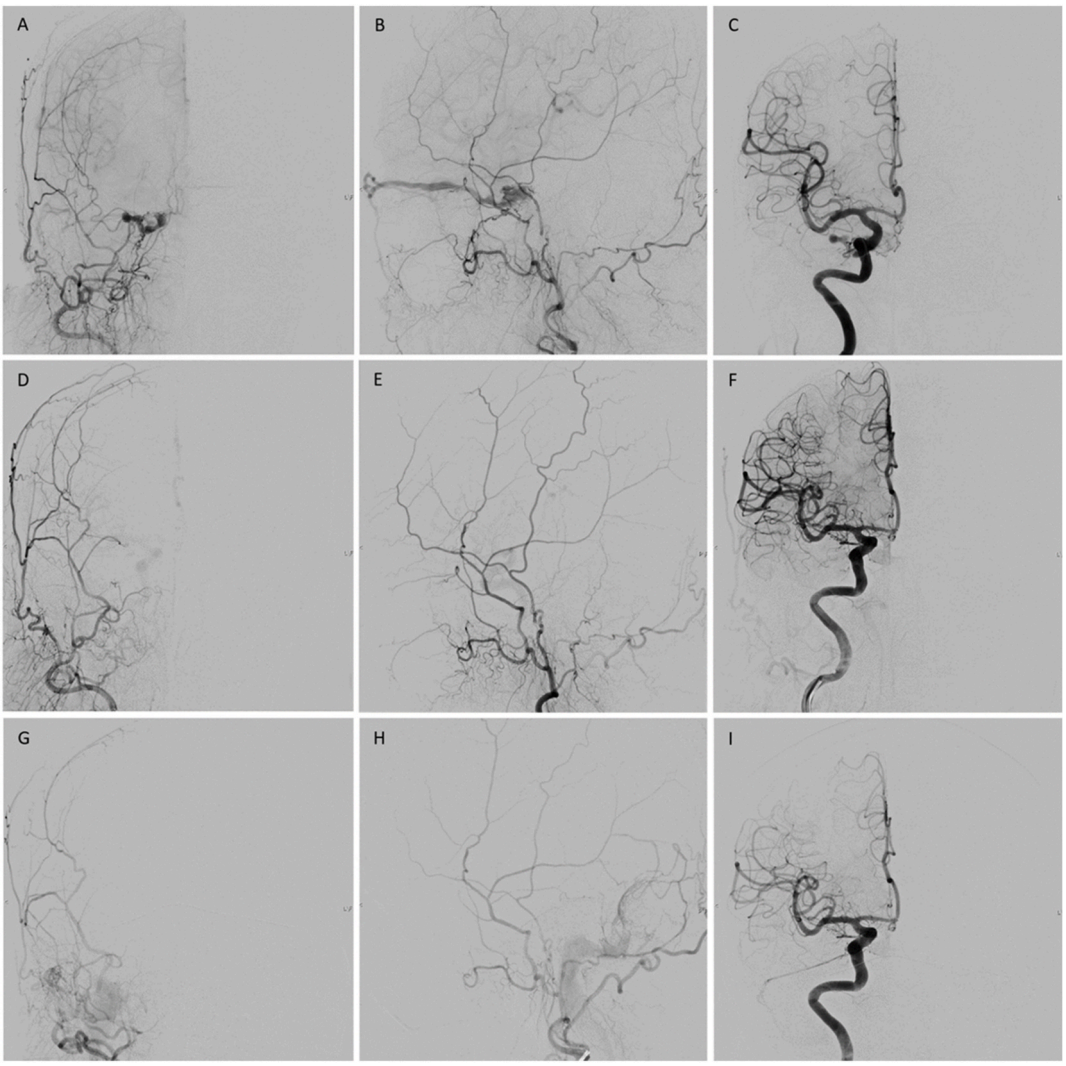

Fig. 5. Case illustration 3. (A) Anteroposterior (AP) and (B) lateral views of the right external carotid artery (ICA), and (C) AP view of the right internal carotid artery (ICA) injections show the right cavernous sinus dural arteriovenous fistula (CSDAVF) supplied by the artery of the foramen Rotundum, middle meningeal artery, and inferolateral trunk with drainage into the right superior ophthalmic vein. (D) AP and (E) lateral views of the right ECA, and (F) AP view of the right ICA injections obtained 1 year after initial angiography reveal spontaneous complete regression of the right CSDAVF. (G) AP and (H) lateral views of the right ECA, and (I) AP view of the right ICA injections obtained 25 months after complete regression of the fistula demonstrate de novo right sigmoid sinus dural arteriovenous fistula supplied by the right middle meningeal artery, posterior auricular artery, occipital artery, and meningohypophyseal trunk with antegrade drainage into the right internal jugular vein.

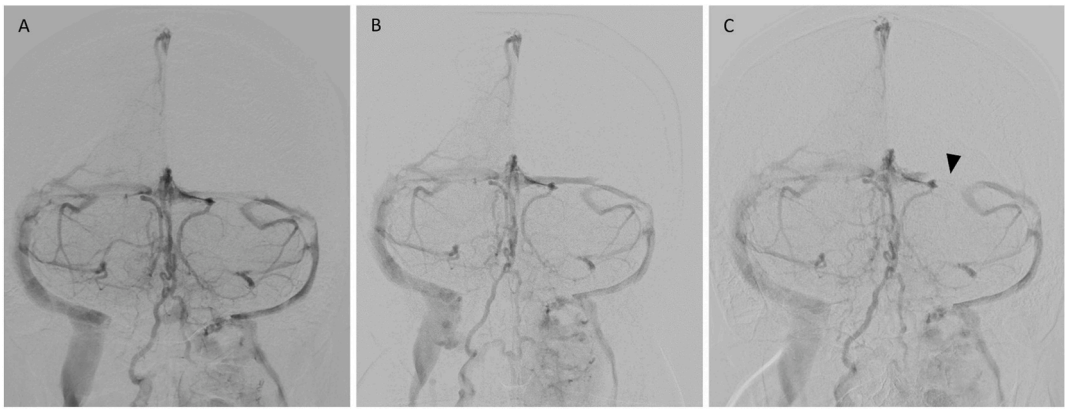

Fig. 6. Case illustration 3. Anteroposterior views of venous phase of the left vertebral artery injection show normal sinus drainage. At (A) initial presentation, (B) 1 year during the disappearance of fistula, and (C) 4 years during occurrence of the de novo fistula. Filling defect (arrowhead) at left proximal transverse sinus is suspected.

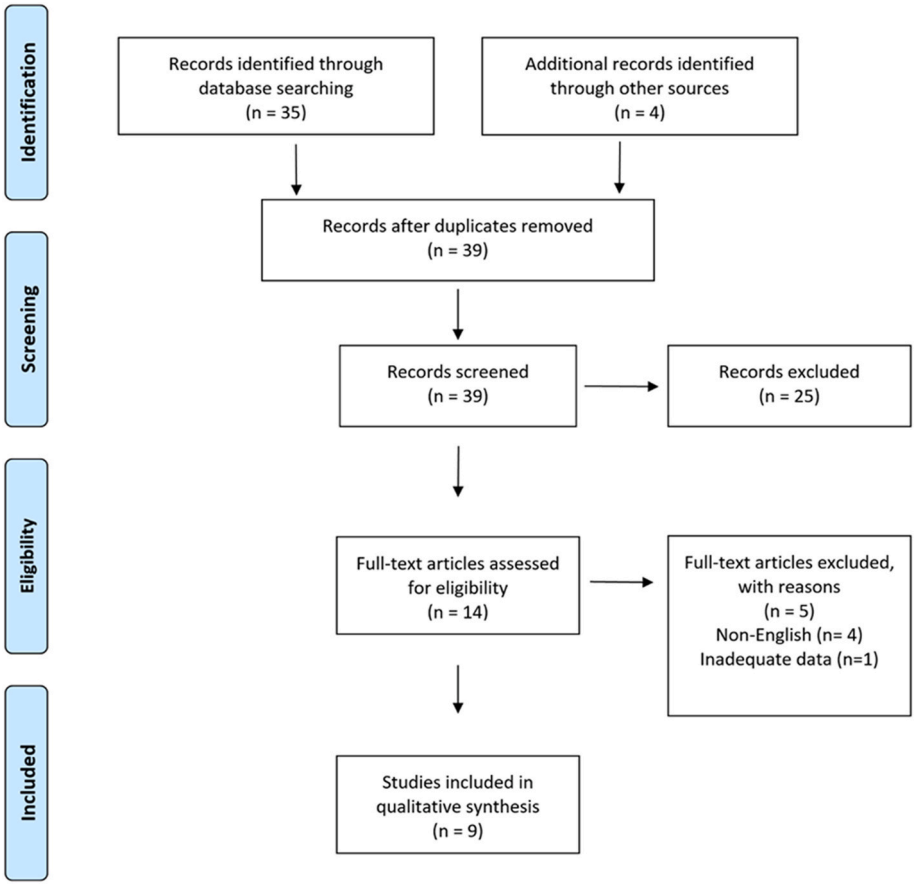

Fig. 7. Flow diagram showing a summary of our search strategy using the PRISMA (preferred reporting items for systematic reviews and meta-analyses) guidelines for relevant studies on de novo dural arteriovenous fistulas following the treatment of cavernous sinus dural arteriovenous fistulas.

Table 2
Summary of reported De Novo DAVFs following the treatment of CSDAVFs in the literature.

Authors	Gender/ Age	Initial location	Sinus stenosis or occlusion	Treatment	Approach route	Time interval (months)	De Novo location	Treatment	Angiographic outcome of new fistula
Kuwayama et al.[9]	F/58	Rt. CS	+	None/SR	–	8	LT. TS-SS	TAE (PVA) Surgery	CR
Nakagawa et al.[10]	F/43	Lt. CS	–	TAE (gelform) TVE (coils)	IMA Lt. SOV, IJV, IPS	4	Lt. SS	Ligation of APA. Partial coiling of Lt.IPS Ligation of Lt. IJV	CR
						5	Lt. TS	None	CR
Yamashita et al.[11]	F/54	Lt. CS	–	TAE (coils) TVE	MMA, IMA Lt. IJV-IPS	11	Lt. SS	TAE (coils)	N/A
Makiuchi et al.[12]	M/43	Lt. CS	–	TVE (coils)	Lt. SOV	6	Lt. SS	None	N/A
Kubota et al.[8]	F/43	Lt. CS	–	TVE (coils)	Lt. IJV-IPS (failed) Lt. SOV	4	Lt. JB	None	N/A
Kiyosue et al.[13]	F/66	Lt. CS	–	TVE	Lt. IJV-IPS (failed) Lt. STV-AV-SOV (coils)	5	Lt. JB	None	CR
Gupta et al.[7]	F/59	Lt. CS	–	TAE	Lt. ICA (sacrificed)	4	Lt. SS	TAE (particles, coils)	CR
Hiu et al.[14]	F/68	Lt. CS	+	TVE (coils)	Lt. IJV-IPS	24	Lt. IPS	TVE (coils)	CR
						36	Lt. SS	TVE (coils)	CR
Makita et al.[6]	F/69	Lt. CS	–	TVE	Lt. IJV-IPS	12	Lt. ACC Lt. TS-SS	None	CR

ACC: anterior condylar confluence; APA: ascending pharyngeal artery; AV: angular vein; CR: complete remission; CS: cavernous sinus; CSDAVF: cavernous sinus dural arteriovenous fistula; DAVF: dural arteriovenous fistula; F: female; IPS: inferior petrosal sinus; IJV: internal jugular vein; JB: jugular bulb; IMA: internal maxillary artery; Lt: left; M: male; MMA: middle meningeal artery; N/A: data not available; PVA: polyvinyl alcohol; Rt: right; SOV: superior ophthalmic vein; SR: spontaneous regression; SS: sigmoid sinus; STV: superficial temporal vein; TAE: transarterial embolization; TS: transverse sinus; TVE: transvenous embolization.

5. Conclusion

Sinus thrombosis and elevated venous pressure may play an important role in the development of a de novo DAVF. Thrombophilic abnormalities and the use of contraceptives may contribute to sinus thrombosis, leading to the development of the second remote DAVF after treatment of CSDAVFs. Clinical and imaging follow-up should be performed at least 1 year after the complete resolution of CSDVAVFs by with or without endovascular treatment.

CRediT authorship contribution statement

Prasert Iampreechakul: Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Korrapakc Wangtanaphat:** Software, Resources. **Songpol Chuntaroj:** Validation, Data curation. **Chonlada Angsusing:** Project administration. **Yodkhwan Wattanasen:** Formal analysis. **Sunisa Hangsapruek:** Formal analysis. **Punjama Lertbusayanukul:** Visualization, Investigation. **Somkiet Siriwimonmas:** Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Abbreviation

CS: cavernous sinus
DAVF: Dural arteriovenous fistula
ECA: external carotid artery
ICA: internal carotid artery
IPS: inferior petrosal sinus
MHT: Meningohypophyseal trunk
MRI: Magnetic resonance imaging
MMA: Middle meningeal artery
OA: Occipital artery
SOV: superior ophthalmic vein
VA: vertebral artery