Anatomical and Ophthalmic Clinical Outcomes of Endovascular Treatment for Dural Arteriovenous Fistulas (DAVFs), 13 years' Experience in a Single Institute.

Uprimporn Suthiwong, Raveewan Choontanom, Patcharaporn Chandraparnik, Sombat Tanaboonyawat

Department of Ophthalmology, Phramongkutklao Hospital, Thailand

Background: Dural arteriovenous fistulas (DAVFs) are abnormal communications between the dural arteries and venous sinuses or cortical veins. Cerebral angiography remains the diagnostic and therapeutic gold standard. Variation in time to endovascular embolization may affect ocular outcomes. This study aimed to evaluate the relationship between treatment timing and clinical outcomes in DAVF patients.

Methods: This retrospective cohort included 102 eyes from 85 patients with angiographically confirmed DAVFs who underwent endovascular embolization between January 2009 and August 2022. Patients without ocular symptoms or with incomplete follow-up were excluded. Main outcomes—best-corrected visual acuity (BCVA), intraocular pressure (IOP), and proptosis—were assessed at baseline, day 1, month 1, and month 3 post-treatment.

Results: Cognard types IIa (32.4%) and IIa + IIb (31.4%) were most frequent. Mean IOP improved significantly on day 1 in the < 4-week (P = 0.014) and 4-8-week (P = 0.043) groups compared with > 8 weeks. At 1 month, IOP changes were not significant in any group. At 3 months, mean IOP again improved significantly in the < 4-week (P = 0.001) and 4-8-week (P = 0.009) groups versus > 8 weeks. BCVA and proptosis showed no significant differences among duration groups.

Conclusion: Endovascular embolization yields excellent anatomical and functional outcomes in DAVFs. Early intervention within 8 weeks produces faster and greater IOP improvement, underscoring the importance of prompt diagnosis and treatment to prevent irreversible ocular damage. These findings support timely referral pathways and prospective studies to determine the optimal treatment window.

Conflicts of Interest: None

Keywords: Dural arteriovenous fistulas, Cerebral angiography, Cerebral embolization

EyeSEA 2025;20(2):40-48

Introduction

Dural arteriovenous fistulas (DAVFs) are abnormal communications between the dural arteries and dural venous sinuses or cortical veins. DAVFs increase the risk of intracranial hypertension and hemorrhage.[1] The incidence rate of DAVFs was previously reported to be 0.15-0.29 cases per 100,000 person-years.[2-4] DAVFs can be caused by many etiologies such as traumatic head injury (closed head injury, skull base fracture, penetrating head trauma), cavernous sinus thrombosis, iatrogenically after craniotomies or endoscopic transsphenoidal sinus surgery, musculoskeletal diseases (Ehler's Danlos syndrome, Pseudoxanthoma elasticum, Osteogenesis imperfecta, Fibromuscular dysplasia) and spontaneously without identifiable causes. Thirty percent of DAVFs occur spontaneously and are most commonly found in post-menopausal elderly women.[5,6]

The clinical manifestations and duration of symptoms vary depending on the pathological anatomy of the fistula and venous drainage pattern. Abnormal anterior drainage will result in signs and symptoms of orbital congestion, such as eye pain, swelling, diplopia, pulsatile

Correspondence to:
Uprimporn Suthiwong, *Department of Ophthalmology, Phramongkutklao Hospital, Thailand, Email: eurngus@gmail.com*
Received : July 28th, 2025
Accepted : November 11th, 2025
Published : December 15th, 2025

tinnitus, proptosis, conjunctival injection, dilated corkscrew vessels, decreased visual acuity, increased intraocular pressure, and central retinal vein occlusion (CRVO). Posterior drainage is associated with neurological abnormalities or intracranial hemorrhage.

Patients with signs and symptoms that are compatible with DAVFs require further imaging for diagnosis. Cerebral catheter angiography is the gold standard in diagnosis and also utilized in treatment of DAVFs. However, there is significant variation in the timing of embolization across hospitals, attributable to differences in clinical presentation, resource availability, and treatment strategies. Timely intervention is clinically crucial, as previous studies have shown that hemorrhagic DAVFs with cortical venous drainage carry a high risk of early rebleeding—up to 30-35% within hours to days after the initial event—leading to irreversible venous congestion, neurological deterioration, or vision-threatening complications if treatment is delayed.[7-9] Therefore, understanding how the interval to endovascular therapy influences anatomical closure and ophthalmic recovery is essential to guide optimal management and improve patient outcomes.

The main objective of this study was to analyze the relationship between different times to treatment with embolization and ophthalmic outcomes including IOP, proptosis, and BCVA. Other objectives were to describe ocular characteristics, angiographic findings, complications after embolization, and survival analysis after treatment of patients in Phramongkutklao Hospital with 3-month to 13-year follow-up.

Materials and Methods

This retrospective cohort study included 85 consecutive patients with 102 eyes who presented to the Ophthalmology Department, Phramongkutklao Hospital, with ocular manifestations and were diagnosed with dural arteriovenous fistulas (DAVFs) confirmed by cerebral angiography between January 1, 2009 and August 31, 2022. All medical records and imaging studies were reviewed retrospectively. Patients were identified from the hospital's neurointerventional database. Inclusion criteria were: (1) patients with angiographically confirmed DAVFs, (2) presence of ocular symptoms (e.g., proptosis, chemosis, diplopia, elevated intraocular pressure), and (3) patients who underwent endovascular embolization as the primary treatment. Exclusion criteria were: (1) patients managed conservatively or surgically without embolization, (2) patients without ocular manifestations, and (3) incomplete clinical or angiographic follow-up data. All patients underwent endovascular embolization performed by experienced neurointerventional radiologists. The embolization techniques included transarterial or transvenous approaches depending on the angioarchitecture and venous drainage pattern of each case. Embolic materials used included coils, Onyx (ethylene-vinyl alcohol copolymer), balloons or Gelfoam, either alone or in combination, according to operator preference and lesion accessibility. Post-procedural angiography was performed immediately to assess treatment completeness. Anatomical outcomes were categorized based on follow-up cerebral angiography at 3-6 months as:

- Complete closure – no residual arteriovenous shunt
- Incomplete closure – residual but reduced shunting
- No change – persistent shunt without improvement

Main clinical outcomes included intraocular pressure (IOP), proptosis (measured by Hertel exophthalmometer), and best-corrected visual acuity (BCVA). Other ocular signs—eye pain, diplopia, red eye, dilated corkscrew vessels, and extraocular movement (EOM) limitation—were also recorded. Clinical assessments were performed at baseline and post-treatment at day 1, 1 month, and 3 months. The duration from first ophthalmologic presentation to embolization was categorized into three groups:

- < 4 weeks
- 4-8 weeks
- > 8 weeks

Descriptive statistics (mean, standard deviation, median, and percentage) were used for baseline characteristics. The Chi-squared test was used to compare anatomical outcomes among different treatment-delay groups. Changes in IOP, BCVA, and proptosis before and after treatment

were analyzed using the Wilcoxon signed-rank test. Kaplan–Meier survival analysis was used to estimate overall survival. A P value < 0.05 was considered statistically significant.

This study was approved by the Institutional Review Board of the Royal Thai Army Medical Department (IRBRTA), approval code R139h/64, on September 27, 2021.

Results

Patient characteristics (Table 1)

Eighty-five patients with 102 symptomatic eyes underwent ophthalmic examination, angiographic evaluation and endovascular embolization for treatment of DAVFs. The mean age was 53.74 ± 14.95 years (range 20-88 years). Forty-one patients (48.24%) were male and forty-four (51.8%) were female. Nearly all were Thai (98.8%), and one patient (1.2%) was Cambodian. Thirty-eight patients (44.7%) had systemic disease, most commonly hypertension (63.16%), hyperlipidemia (44.74%), and diabetes mellitus (13.16%). One patient had Ehlers–Danlos syndrome type 4 and another had cerebral venous sinus thrombosis. Thirty-six patients (42.35%) had a history of ocular or head trauma, occurring on average 2 years before symptom onset. Five patients (5.88%) had prior ocular or head surgery. Details are summarized in Table 1.

Clinical Manifestations (Table 2)

The median baseline BCVA was 0.2 logMAR (range 0-3) and mean IOP was 17.71 ± 6.05 mmHg. Red eye (76.47%), bulging eye (49.41%), and decreased vision (43.53%) were the most common presenting symptoms. The median symptom duration was 2 months (range 0.03-48). Abnormal fundus findings were present in 22.55% of eyes. Table 2 summarizes presenting symptoms and signs.

Cerebral Angiography Findings

All patients underwent diagnostic cerebral angiography and were classified according to the Cognard's system: type I (9.8%), IIa (32.4%), IIb (8.8%), IIa+IIb (31.4%), III (5.9%), and IV (11.8%). No cases of type V were identified.

Treatment characteristics

Patients were grouped by duration from initial presentation to embolization: < 4 weeks (50 eyes, 49%), 4-8 weeks (27 eyes, 26.5%), and > 8 weeks (25 eyes, 24.5%). Coils were the most commonly used embolic material (57.84% alone, 72.55% combined with other agents). Other embolic agents were used alone or in combination with each other composed of balloon (11.8%), balloon combined with others (12.8%), EVOH (6.9%), EVOH combined with others (10.8%), Gelfoam (5.9%), and Gelfoam combined with others (21.6%).

Anatomical outcomes

Complete angiographic closure after embolization was achieved in 80 eyes (78.4%), while 21 eyes (21.6%) had residual shunting. Multiple treatment sessions were required in 9.8%. No significant difference in complete closure was observed among the three treatment-duration groups (P = 0.071).

Table 1: Patient characteristics

Characteristics	Incidence n = 85(%)
Age (years): Mean ± SD	53.74 ± 14.95
Sex	
Male	41 (48.24)
Female	44 (51.76)
Nationality	
Thai	84 (98.82)
Other: Cambodian	1 (1.18)
Smoking	
Yes	7 (8.24)
No	78 (91.76)

Table 1: Patient characteristics (Cont.)

Characteristics	Incidence n = 85(%)
Alcohol drinking	
Yes	16 (18.82)
No	69 (81.18)
Systemic disease	
Yes	38 (44.71)
Hypertension	24 (63.16)
Hyperlipidemia	17 (44.74)
Diabetes mellitus	5 (13.16)
Old CVA	3 (7.89)
Gout	2 (5.26)
Other diseases	12 (31.58)
No	47 (55.29)
History of traumatic head/ocular injury	36 (42.35)
Duration (year): median (min, max)	2 (0.08, 50)
History of traumatic head/ocular surgery	5 (5.88)
Craniectomy with clot removal	2 (2.35)
Repair orbital wall surgery	3 (3.53)

Table 2: Clinical manifestations

Clinical manifestations	n = 102 eyes (%)
BCVA	
Median (min, max)	0.2 (0, 3)
IOP	
Mean ± SD	17.71 ± 6.05
Symptoms at presentation	
Eye pain	37 (43.53)
Red eye	65 (76.47)
Decreased VA	37 (43.53)
Bulging eye	42 (49.41)
Diplopia	33 (38.82)
Tinnitus	29 (34.12)
Others	56 (65.88)
Duration of symptoms	
Median: Month (min, max)	2 (0.03, 48)
Signs at presentation	
Eyelid swelling	17 (16.67)
Dilated corkscrew vessels	67 (65.69)
Blood in Schlemm's canal	10 (9.8)
Relative afferent pupillary defect	11 (10.78)
Proptosis	38 (37.35)
Auditory bruit	31 (30.39)
Limited EOMs	47 (46.08)
Abnormal fundus (disc edema, tortuous vessels, and optic pallor)	23 (22.55)

Clinical outcomes

The main clinical outcomes were BCVA, IOP, and proptosis. At baseline, median BCVA was 0.20 logMAR, mean IOP 17.71 ± 6.05 mmHg, and proptosis was observed in 38 eyes (37.3%).

BCVA

Median BCVA improved significantly at 1 month (0.10 logMAR, $P < 0.001$) and 3 months (0.10 logMAR, $P < 0.001$) after treatment. No significant BCVA differences were found among treatment-duration groups ($P > 0.05$ for all time points).

Intraocular Pressure (IOP)

When comparing across treatment durations, mean IOP improved significantly in the < 4-week group ($P = 0.014$) and 4–8-week group ($P = 0.043$) versus > 8 weeks at day 1; no significant difference was found at 1 month ($P > 0.05$); and improvement reappeared at 3 months ($P = 0.001$ and $P = 0.009$, respectively).

Proptosis

Proptosis decreased from 38 eyes (37.3%) at baseline to 33 (32.4%) at day 1, 15 (14.7%) at 1 month, and 8 (8.1%) at 3 months ($P < 0.001$ vs baseline). No significant difference was found among duration groups ($P > 0.05$).

Other Clinical Outcomes and Complications

Post-embolization complications occurred in 26 eyes (25.5%), including hemorrhagic neurologic events (30.8%): ruptured DAVFs (7.7%), intracerebral/intraventricular hemorrhage (15.4%), and cavernous sinus thrombosis (7.7%). Other complications included CNS infection (7.7%), CN III palsy (3.9%), CN VI palsy (53.9%), CRVO (11.5%), and dizziness (7.7%).

Survival Analysis

Three patients died following embolization—two within the first year from ruptured DAVFs with septic shock and one during the second year from intraventricular hemorrhage. Kaplan–Meier survival analysis (Figure 2) demonstrated 1-year and 2-year survival rates of 97.6% and 96.3%, respectively.

Clinical Significance

The mean IOP reduction represents not only statistical significance but also a clinically meaningful improvement that may reduce the risk of glaucomatous optic nerve damage. Early intervention (< 8 weeks) yielded the most rapid and sustained IOP decrease, underscoring the importance of timely diagnosis and treatment.

Discussion

DAVFs are intracranial lesions consisting of abnormal communication between meningeal arteries and dural venous sinuses or leptomeningeal veins. DAVFs can occur spontaneously or be acquired. The most common cause of DAVFs is previous trauma. Previous studies reported an incidence of DAVFs in patients with traumatic brain injury of 0.2% and in those with skull base fractures of 4%.[10,11] In addition, underlying musculoskeletal diseases have been associated with DAVFs development. The most common presentation occurs in post-menopausal elderly women, suggesting a hormonal influence, whereas men are less frequently affected (10-27%).[12,13] However, in the present study, the prevalence was nearly equal between males and females.

Clinical manifestations of DAVFs vary according to the angioarchitecture and venous drainage pattern. Patients with anterior drainage usually present with ocular symptoms such as decreased visual acuity, elevated intraocular pressure (IOP), proptosis, and limited extraocular movement, whereas posterior drainage more often leads to neurological dysfunction and carries a higher risk of intracranial hemorrhage. In this study, the most common presenting symptoms were red eye, proptosis, decreased vision, and ocular pain, with a median symptom duration of two months before hospital presentation. Reduced visual acuity may result from ischemic optic neuropathy, chorioretinal dysfunction, or secondary glaucoma due to venous hypertension. If left untreated, chronic venous congestion may lead to central retinal vein occlusion, optic atrophy, or permanent visual loss.

Endovascular embolization remains the treatment of choice for DAVFs. Several embolic materials, including coils, Gelfoam, balloons, and ethylene-vinyl alcohol copolymer (Onyx), can be used either alone or in combination. Previous studies demonstrated high rates of anatomical cure and visual improvement after embolization, with complete occlusion rates ranging from 78% to 92%.[14,15] In our study, complete angiographic closure was achieved in 80 eyes (78.4%), and residual shunting persisted in 21 eyes (20.6%).

Regarding clinical outcomes, this study demonstrated significant improvement in BCVA, IOP, and proptosis at one and three months post-treatment ($P < 0.001$ for all). Notably, IOP reduction was most rapid and significant in patients treated within 8 weeks of symptom onset, suggesting that early intervention prevents irreversible trabecular and venous outflow damage. This emphasizes the importance of early recognition of ocular signs and prompt referral for angiographic evaluation and embolization.

Our findings are consistent with previous reports showing high success rates after embolization. Lee et al. reported complete closure in 92% of cases, while Preechawat et al. found anatomical cure in 78% of Thai patients with improved vision.[14,15] However, few prior studies have focused on the effect of treatment timing. The present study highlights that early treatment (<8 weeks) was associated with faster and greater improvement in IOP, suggesting that delayed treatment may allow chronic venous congestion to cause irreversible ocular damage.

The overall complication rate after embolization was 25.5%. Among these, 38.46% were classified as serious, including ruptured DAVFs, cavernous sinus thrombosis, central nervous system infection, and intracerebral or intraventricular hemorrhage. Despite these complications, survival remained high at 97.6% at one year and 96.3% at two years.

This study has several limitations. The retrospective, single-center design may introduce information and selection biases. Inclusion was limited to patients who underwent embolization and completed follow-up, which may introduce survivorship bias, as untreated or lost-to-follow-up patients might have had poorer outcomes. However, this criterion was necessary to ensure a homogeneous cohort for evaluating treatment efficacy. In addition, baseline IOP before initiation of antiglaucoma medications was not available in all cases, which could confound IOP comparisons. Variability in lesion angioarchitecture and operator technique may also have influenced outcomes.

Future prospective multicenter studies with standardized ophthalmic evaluations, larger sample sizes, and longer follow-up periods are recommended to validate these findings. Subgroup analysis by Cognard classification and embolization technique would further clarify which subtypes of DAVFs benefit most from early intervention.

In conclusion, endovascular embolization is an effective treatment for dural arteriovenous fistulas, resulting in significant anatomical and functional improvement. Among the main clinical outcomes, intraocular pressure (IOP), best-corrected visual acuity (BCVA), and proptosis all showed significant improvement after treatment, with the most rapid reduction in IOP observed on the first day and sustained improvement at three months. Patients who received treatment within eight weeks of symptom onset demonstrated faster and greater improvement in IOP compared with those treated later, suggesting that early intervention may prevent irreversible venous congestion and secondary glaucomatous damage. Although anatomical cure rates did not differ significantly among treatment-delay groups, the early-treated group achieved better functional recovery. These findings support the role of timely endovascular management in improving both ocular and anatomical outcomes in DAVF patients.

Table 3: Main clinical outcomes in each duration to treatment

Main clinical outcome	Duration from presentation to treatment		
	Within 4 weeks (n = 50)	4-8 weeks (n = 27)	> 8 weeks (n = 25)
At day 1 after endovascular treatment			
BCVA: Median (min, max)	0.2 (0, 1)	0.3 (0, 0.7)	0.1 (0, 0.8)
IOP: mean ± SD	13.84 ± 3.88	14.04 ± 3.94	16.12 ± 3.23
Proptosis	18 (36)	9 (33.33)	6 (24)
At 1 months after endovascular treatment			
BCVA: median (min, max)	0.1 (0, 0.6)	0.1 (0, 1.8)	0.2 (0, 0.4)
IOP: mean ± SD	13.5 ± 3.45	14.22 ± 3.92	14.8 ± 2.97
Proptosis	9 (18)	4 (14.81)	2 (8)
At 3 months after endovascular treatment			
BCVA: median (min, max)	0.1 (0, 0.4)	0.05(0, 0.7)	0.1 (0, 0.3)
IOP: mean ± SD	12.66 ± 2.33	12.75 ± 2.49	14.68 ± 2.48
Proptosis	5 (10)	1 (4.17)	2 (8)

Note: Proptosis cases in Table 2 represent baseline findings before treatment, whereas Table 3 shows the number of eyes with persistent proptosis at each follow-up time.

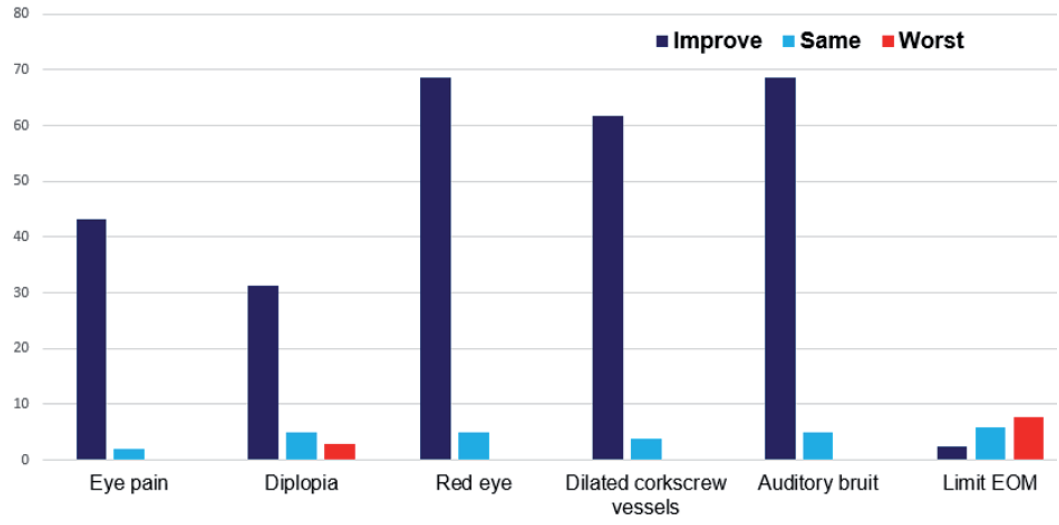

Figure 1: Other clinical outcomes

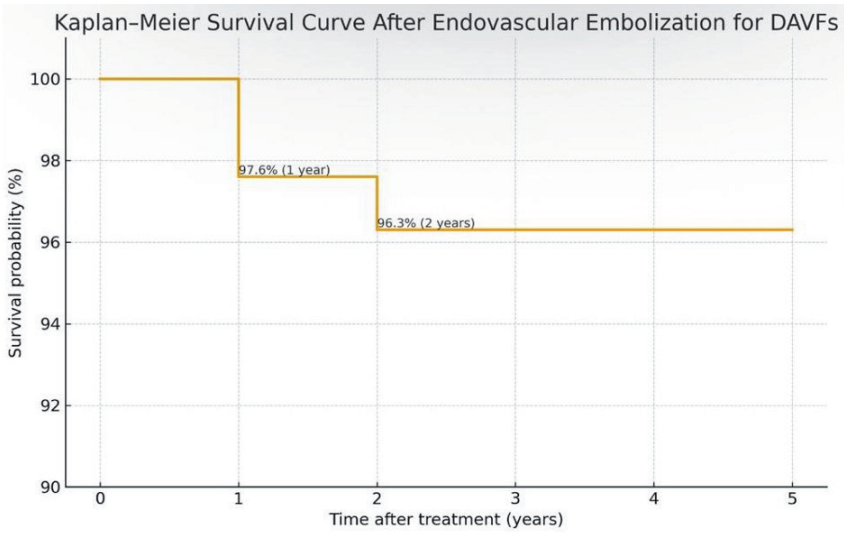

Figure 2: Kaplan–Meier survival analysis

Acknowledgements

Thank you, Komalpreet Tur (BS), Wayne State University School of Medicine for your kindness in English language editing

Conflicts of Interest:

The authors have no conflict

References

1. Lasjunias P, Chiu M, Brugge KT, Tolia A, Hurth M, Berenstein M. Neurological manifestations of intracranial dural arterio-venous malformation. J Neurosurg. 1986;4:724-30.
2. Brown RD, Wiebers DO, Torner JC, O'Fallon WM. Incidence and prevalence of intracranial vascular malformations in Olmsted County, Minnesota, 1965 to 1992. Neurology. 1996;46:949-952.
3. Al-Shahi R, Bhattacharya JJ, Currie DG, Papanastassiou V, Ritchie V, Roberts RC, et al. Scottish Intracranial Vascular Malformation Study Collaborators. Prospective, population-based detection of intracranial vascular malformations in adults: the Scottish Intracranial Vascular Malformation Study (SIVMS). Stroke. 2003;34:1163-1169.
4. Kuwayama N. Epidemiologic survey of dural arteriovenous fistulas in Japan: clinical frequency and present status of treatment. Acta Neurochir Suppl. 2016;123:185–188.
5. Ellis JA, Goldstein H, Connolly ES, Meyers PM. Carotid-cavernous fistulas. Neurosurgical Focus. 2012;32(5):1-11.
6. Keizer R. Carotid-cavernous and orbital arteriovenous fistulas: ocular features, diagnostic and hemodynamic considerations in relation to visual impairment and morbidity. Orbit. 2003;22(2):121-142.
7. Duffau H, Lopes M, Janosevic V, Sichez JP, Faillot T, Capelle L, et al. Early rebleeding from intracranial dural arteriovenous fistulas: report of 20 cases and review of the literature. J Neurosurg. 1999;90(1):78-84.
8. Sorteberg W, Bakke SJ, Sorteberg A. Intracranial hemorrhage from dural arteriovenous fistulas: Rebleed prior to treatment occurred in 35% of the patients at median 7.5 h. Neurosurg Pract. 2020;1(4):373-381.
9. Durnford AJ, Kirkman MA, Santarius T, Higgins JN, Mankad K, Renowden SA, et al. Timing of treatment and risk of rebleeding in dural arteriovenous fistulas presenting with intracranial hemorrhage. Stroke. 2022;53(12):3704-3712.

10. Liang W, Xiaofeng Y, Weiguo L, Wusi Q, Gang S, Xuesheng Z. Traumatic Carotid Cavernous Fistula Accompanying Basilar Skull Fracture: a study on the incidence of Traumatic Carotid Cavernous Fistula in the Patients with Basilar Skull Fracture and the Prognostic Analysis About Traumatic Carotid Cavernous Fistula. J Trauma. 2007;63(5):1014-1020.
11. Helmke K, Kruger O, Laas R. The direct carotid cavernous fistula: A clinical, pathoanatomical, and physical study. Acta Neurochirurgica. 1994;127(1-2):1-5.
12. Meyers PM, Halbach VV, Dowd CF, Lempert TE, Malek AM, Phatouros CC, et al. Dural carotid cavernous fistula: definitive endovascular management and long-term follow-up. Am J Ophthalmol. 2002;134:85-92.
13. Cheng KM, Chan CM, Cheung YL. Transvenous embolization of dural carotid-cavernous fistulas by multiple venous routes: a series of 27 cases. Acta Neurochir (Wien). 2003; 145:17-29.
14. Holland L, Ranzcr KM, Harrizon JD, Brauchli D, Wong Y, Sullivan TJ. Endovascular treatment of carotid-cavernous sinus fistulas: ophthalmic and visual outcomes. Orbit. 2018;28(4):290-299.
15. Preechawat P, Narmkerd P, Jiarakongmun P, Poonyathalang A, Pongpech SMD. Dural carotid carvenous sinus fistula: Ocular characteristics, Endovascular management and Clinical outcome. J Med Assoc Thai. 2008;91(6):852-857.