Intracranial Dural Arteriovenous Fistulas With and Without Pial Artery Supply: Analysis of Treatment Outcomes

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BACKGROUND AND OBJECTIVES: The prevalence of pial arterial supply (PAS) to intracranial dural arteriovenous fistulas (DAVFs) and its implications for the management of these fistulas have been limited to relatively small cohort studies and remain somewhat controversial. We conducted a retrospective study to characterize PAS in DAVFs and explore its implications for treatment.

METHODS: Consecutive patients evaluated over a 21-year period were retrospectively reviewed. Angiograms were examined to characterize the angioarchitecture of DAVFs and identify the presence of PAS. PAS was classified into 2 types: dilated preexisting dural branches and pure pial supply. Baseline characteristics, treatment approaches, and treatment and follow-up outcomes were compared between the DAVF cohorts with and without PAS. To minimize patient selection bias, the 2 cohorts were matched in a 1:1 ratio using propensity score matching.

RESULTS: In this cohort, 259 out of 1101 patients (23.5%) exhibited an additional PAS. Multivariate analysis identified 7 independent predictors of PAS: younger age ($P < .001$), longer disease duration ($P = .021$), multiple DAVFs ($P < .001$), tentorial DAVFs ($P < .001$), transverse–sigmoid sinus DAVFs ($P < .001$), and the presence of venous ectasia ($P = .019$) and congestion ($P < .001$). Complication rates were higher in the PAS group, particularly for postoperative hemorrhage ($P < .001$) and ischemia-related complications ($P < .001$), which remained significant even after propensity score matching ($P = .013$ and $P = .001$).

CONCLUSION: The findings suggest that embolization of PAS before DAVF closure may significantly increase the risk of both intracranial hemorrhagic and ischemic complications. Therefore, routine embolization of PAS before DAVF closure is not supported by these results, particularly given the exceptionally low incidence of presumed hemorrhagic complications arising from an unobliterated "pure" pial supply before DAVF obliteration.

KEY WORDS: Dural arteriovenous fistula, Pial arterial supply, Treatment, Outcomes, Complication

The presence of pial arterial supplies (PAS) in dural arteriovenous fistulas (DAVFs) is an increasingly recognized phenomenon. Recent studies indicate that 11.3% to 23.8% of DAVFs have PAS, which is associated with factors such as younger patient age, tentorial location of the DAVF, and a more aggressive fistula.[1,2] Furthermore, patients with DAVFs supplied by pial arteries may be at a higher risk of post-treatment complications, including ischemic strokes and intracranial hemorrhage (ICH), compared with those without pial artery involvement.[3-8] There is ongoing controversy regarding whether embolization of the PAS should be performed in cases of DAVFs with PAS.[1-4,6-8] In addition, descriptions of the characteristics of the PAS in DAVFs, including their frequency, locations, and morphological features, are limited to small sample cohort studies.[1,2]

To gain a deeper understanding of the clinical and angiographic variables associated with the PAS, as well as the treatment outcomes and prognosis, we retrospectively reviewed our institutional case series

ABBREVIATIONS: DAVF, dural arteriovenous fistula; **DREAM INI,** dural arteriovenous fistula research and management in China; **ICH,** intracranial hemorrhage; **PAS,** pial artery supply/supplies.

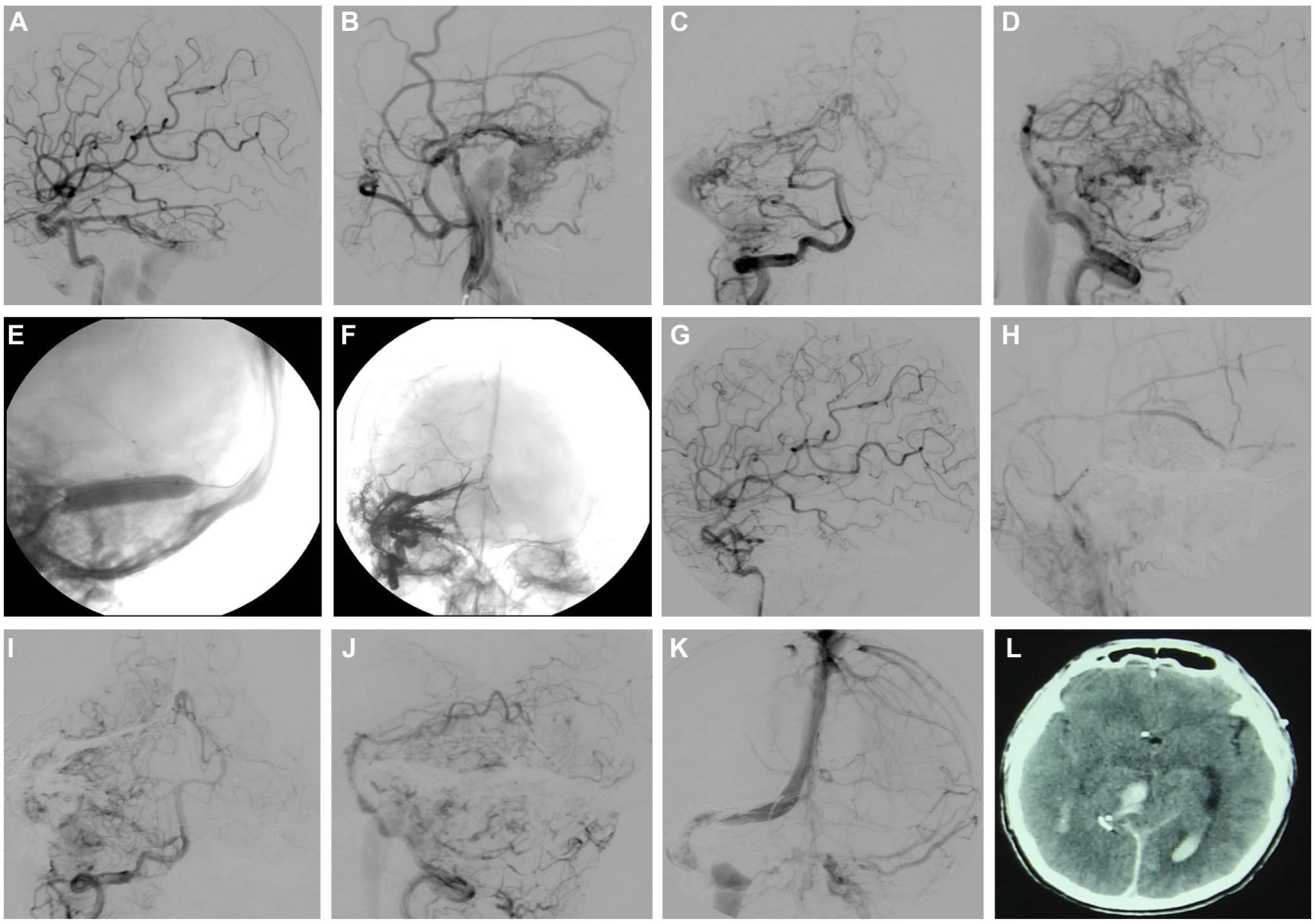

FIGURE 1. **A-D**, *Digital subtraction angiography of the internal carotid, external carotid, and vertebral arteries revealed a Borden type I DAVF supplied by multiple pure pial and dural arteries. These included the meningohypophyseal trunk, pure pial branches of the middle cerebral artery, the occipital artery, the middle meningeal artery, and branches from the posterior circulation, such as the posterior cerebral artery, superior cerebellar artery, and anterior inferior cerebellar artery. The patient had a history of venous sinus stent placement, with the fistula precisely located at the site of the previously implanted stent. In addition, flow-related aneurysms were observed in the posterior circulation pial supply.* **E-H**, *The fistula was completely occluded using Onyx via the arterial route through the middle meningeal artery, with balloon protection of the venous sinus. No embolization was performed on any pial arterial feeders.* **I** *and* **J**, *Immediate postprocedural vertebral artery angiography after complete embolization revealed no abnormalities.* **K**, *In the late venous phase, the transverse–sigmoid sinus on the side of the DAVF remained patent.* **L**, *The patient remained conscious and responsive postoperatively. However, upon returning to the ward, he suddenly suffered respiratory arrest. An urgent computed tomography scan revealed a right temporal lobe hematoma accompanied by intraventricular hemorrhage. DAVF, dural arteriovenous fistula.*

of DAVFs treated over the past 21 years using the Dural Arteriovenous Fistula Research and Management in China (DREAM-INI) database. We also compared DAVFs with and without PAS and conducted a subgroup analysis of DAVFs with PAS.

METHODS

Standard Protocol Approvals, Registrations, and Patient Consents

Patient data were obtained from the DREAM-INI database, which is based on a retrospective, single-center observational study conducted between January 2001 and December 2022.[9] All admitted patients were included in the study. Data on DAVFs were collected after approval from the institutional review board (Xuanwu Hospital, No. 2017010). The institutional review board waived the requirement for informed consent because of the retrospective design of the study.

Data Acquisition and Analysis

Aggressive symptoms comprised ICH and nonhemorrhagic neurological deficits. Additional angioarchitectural details of DAVFs included their location, the presence of venous dilatation, venous ectasia, venous congestion, and draining vein stenosis/occlusion. Venous dilatation and venous varix/ectasia were defined according to established criteria by Cognard et al.[10] Venous congestion was defined as the presence of a

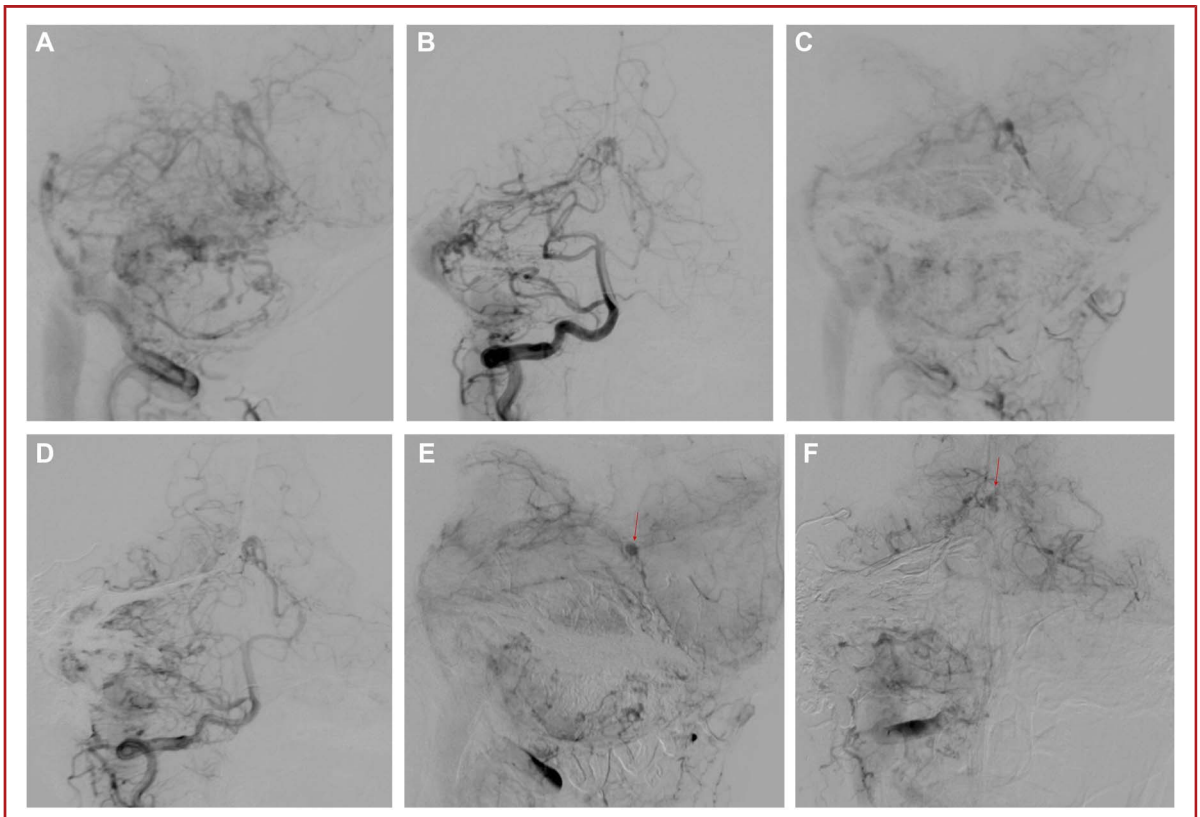

FIGURE 2. **A** *and* **B**, *Preoperative vertebral artery angiography,* **C** *and* **D**, *immediate postoperative vertebral artery angiography, and* **E** *and* **F**, *immediate follow-up angiography after ICH. Compared with the previous images, an abnormally dilated structure arising from a pial arterial feeder (red arrows) was observed, suggesting a potential association with the ICH. ICH, intracranial hemorrhage.*

pseudophlebitic pattern.[11] Tentorial DAVFs were classified into 6 subtypes based on Lawton's classification.[12] The presence of a PAS to a DAVF was assessed using conventional angiography. In cases where the angioarchitecture of the DAVF, including the suspected PAS, was not clearly visualized on conventional angiography, superselective angiography was performed. However, because of the retrospective nature of the study, superselective angiography was not available for every case. In such instances, the PAS was inferred based on conventional angiographic findings. A PAS was defined as blood flow from a pial artery to DAVFs.

PAS were classified into 2 categories based on morphological and anatomic characteristics. The first category was defined by linear, dilated vessels that represented "physiological" preexisting dural branches of pial arteries.[1,13] This type of supply was categorized as dilated dural branches of pial arteries. The second category consisted of tortuous, ramified vessels located outside the established physiological connections between pial arteries and the dura mater. This type was designated as a "pure" PAS.[1,2,8]

The common PAS in DAVFs includes dural and/or pure pial branches of the anterior cerebral artery, middle cerebral artery, posterior cerebral artery, superior cerebellar artery, anterior inferior cerebellar artery, and posterior inferior cerebellar artery.[1-3,8,13]

Treatment

Over 20 years ago, we encountered several cases of patients with DAVF who experienced catastrophic hemorrhagic complications after endovascular treatment via the transarterial approach. At the time, we were uncertain about the underlying cause, but we noticed a common feature among these patients: the presence of PAS. Upon further analysis, we observed that after achieving complete occlusion of the fistula through the dural feeders, the pial supply exhibited abnormal dilation, which appeared to be closely associated with hemorrhage (Figures 1 and 2) (Video 1). This correlation was later corroborated by several studies.[4,6,7] From that point onward, we implemented a treatment strategy to ensure the complete occlusion of PAS before achieving definitive DAVF cure (Figures 3 and 4). For DAVFs with PAS, we prioritize achieving complete occlusion of PAS, after a thorough evaluation of the embolization challenges and potential complications. Glubran was the preferred agent for embolizing PAS, as its ability to be diluted allowed for deeper penetration than Onyx. This was particularly advantageous, given that PAS are generally thin and tortuous. Routine angiographic follow-up is typically scheduled 6 to 12 months after achieving angiographic DAVF cure.

Statistical Analyses

All statistical analyses were conducted using R (version 4.2.3). Baseline characteristics, treatment approaches, and treatment and follow-up outcomes were compared between the DAVF cohorts with and without PAS. Continuous variables were compared using Student's *t*-test or Wilcoxon rank-sum tests, while categorical variables were

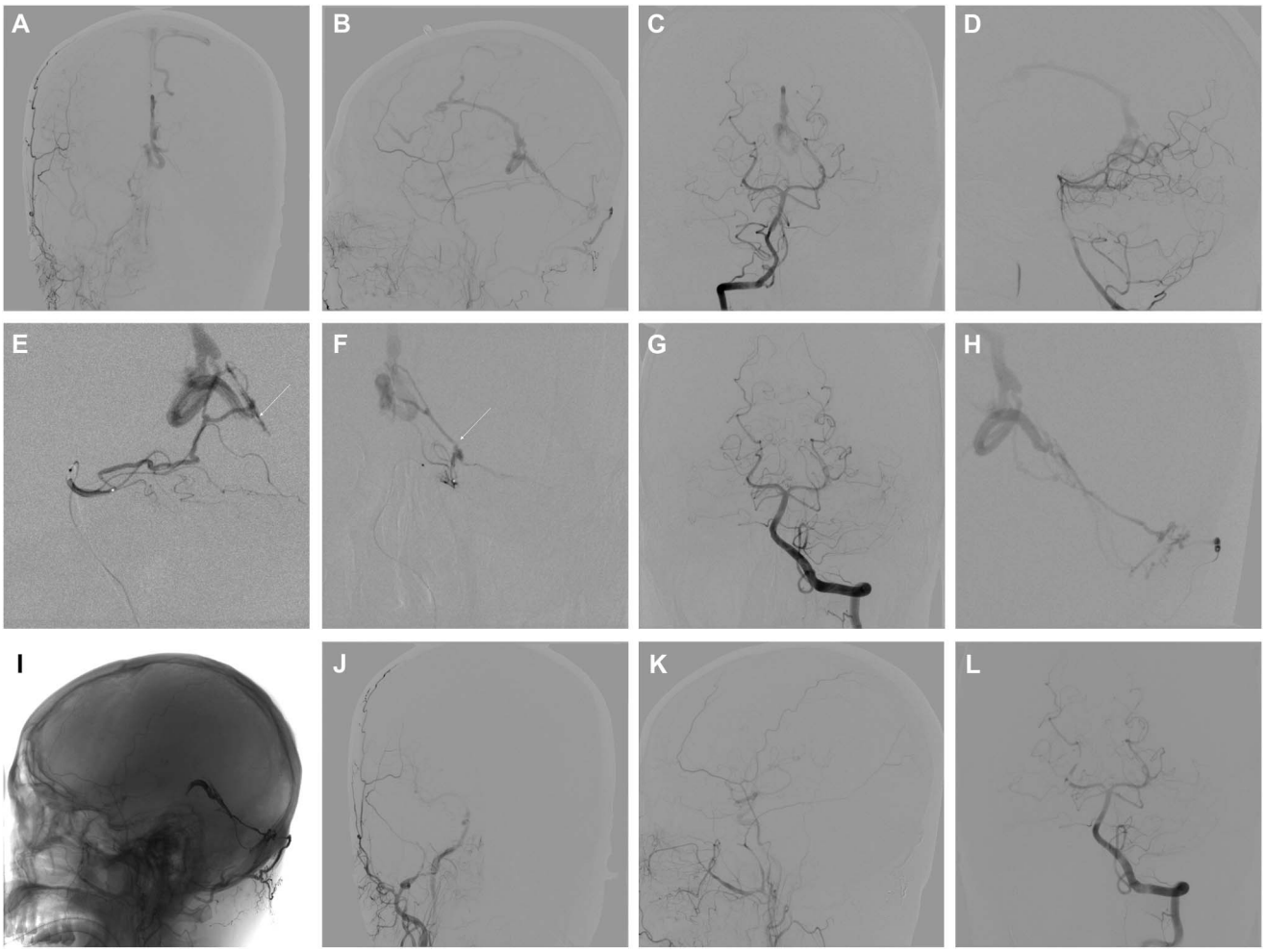

FIGURE 3. **A-D**, *Anteroposterior and lateral views of external carotid and vertebral artery angiography revealed a Borden type III DAVF in the Galenic region, with the absence of straight sinus opacification.* **E** *and* **F**, *Superselective angiography of the left PCA revealed a linear, dilated feeding artery, considered to be a dural branch arising from the pial artery (white arrows).* **G**, *After advancing the microcatheter tip as close to the fistulous point as possible, Glubran was administered for embolization, achieving complete occlusion of the feeder from the PCA.* **H**, *Superselective angiography of the occipital artery.* **I**, *Cast of the embolic agent after complete occlusion of DAVF via the occipital artery using Onyx.* **J-L**, *Immediate postprocedural angiography demonstrated complete occlusion of the DAVF, and the patient reported no significant discomfort after the procedure. DAVF, dural arteriovenous fistula; PCA, posterior cerebral artery.*

compared using Pearson's χ^2 or Fisher's exact tests, as appropriate. Multiple comparisons were not further conducted. To reduce patient selection bias, the 2 cohorts were matched in a 1:1 ratio without replacement, using a caliper of 0.2. Propensity scores were calculated based on age, sex, history of previous treatment, DAVF location, venous dilation, venous ectasia, venous congestion, draining vein stenosis or occlusion, and treatment modalities. Univariate analysis was conducted to assess covariates predictive of DAVFs with PAS. Factors identified as predictive in univariate analysis ($P < .1$) were included in multivariate logistic regression models for DAVFs with PAS. Statistical significance was set at $P < .05$, and all tests were two-tailed. Missing data were not imputed.

RESULTS

Patient and Fistula Characteristics

A total of 1101 patients with intracranial DAVFs were admitted to our hospital, as recorded in the DREAM-INI database. PAS was identified in 259 patients (23.5%). Table 1 provides a comparison of baseline characteristics between DAVF patients with and without a PAS. Compared with DAVF patients without a PAS, those with a PAS were younger ($P < .001$, 95% CI = 0.95-0.97), had a longer disease duration ($P = .002$, 95% CI = 1.01-1.02), and were more likely to have lesions located at the

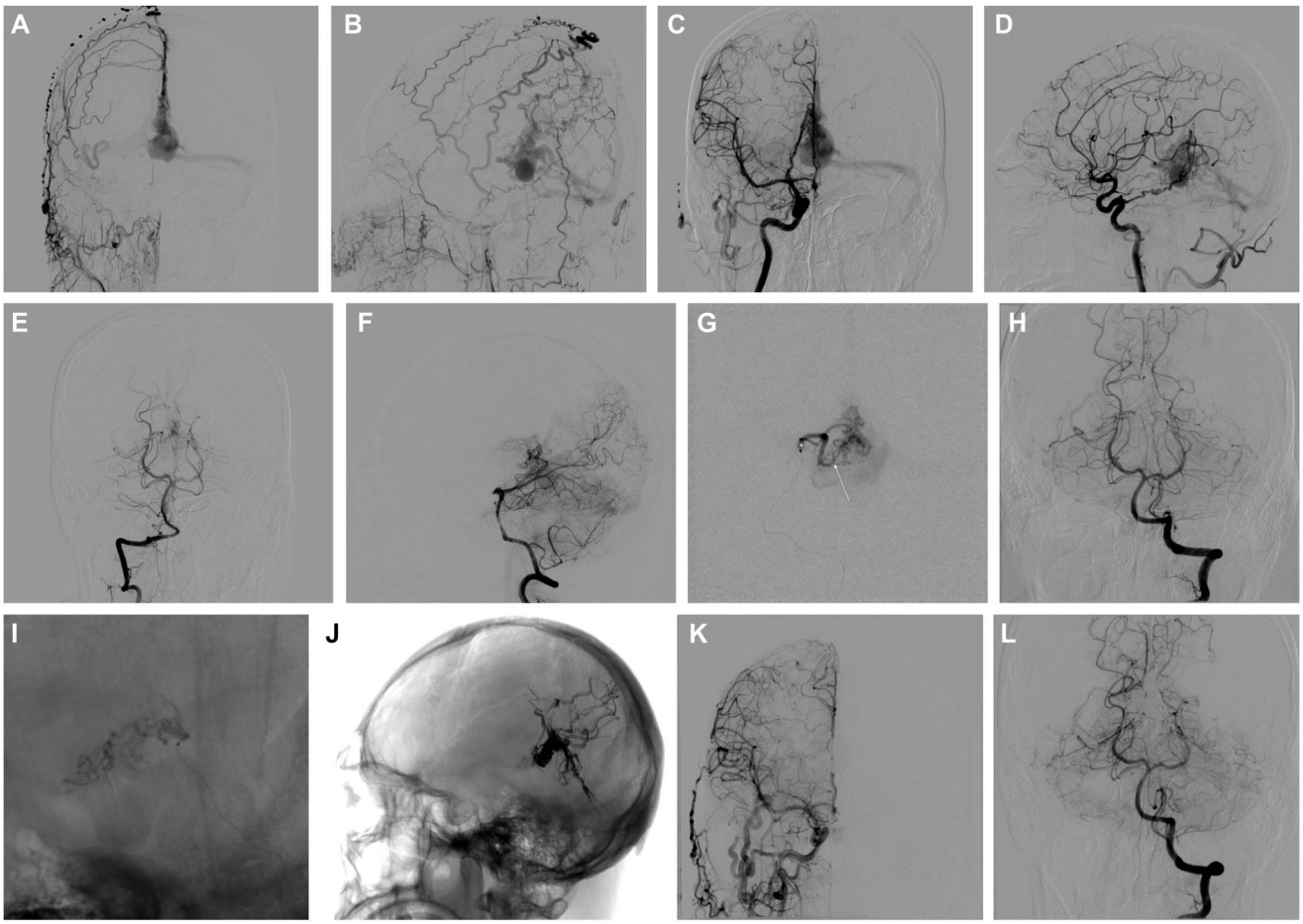

FIGURE 4. **A-F**, *Anteroposterior and lateral angiograms of the external carotid, internal carotid, and vertebral arteries revealed a high-grade midline tentorial DAVF, with venous drainage into an abnormally dilated, varix-like structure suggestive of a high-risk lesion.* **G**, *Superselective angiography of the right PCA revealed a tortuous, ramified feeder, suggestive of a purely pial arterial supply (white arrow).* **H**, *To minimize the risk of cerebral infarction-related complications, the microcatheter tip was advanced as close as possible to the fistulous point. Glubran was then administered for embolization, resulting in complete occlusion of the pial feeder arising from the PCA.* **I** *and* **J**, *Subsequently, Onyx embolization via the occipital artery resulted in complete occlusion of the fistula and the proximal portion of the draining vein.* **K** *and* **L**, *Immediate postprocedural angiography confirmed complete obliteration of the DAVF, with no evidence of cerebral ischemia or hemorrhagic complications. DAVF, dural arteriovenous fistula; PCA, posterior cerebral artery.*

tentorium ($P < .001$, 95% CI = 2.30-4.28) or in multiple locations ($P < .001$, 95% CI = 2.13-4.66). A higher proportion of these patients presented with high-grade DAVF (Borden II) ($P < .001$, 95% CI = 2.29-4.54) and exhibited aggressive symptoms ($P < .001$, 95% CI = 1.49-2.61). On angiography, venous ectasia ($P < .001$, 95% CI = 1.74-3.08), venous congestion ($P < .001$, 95% CI = 5.12-9.56), and draining vein stenosis or occlusion ($P < .001$, 95% CI = 2.31-4.12) were more frequently observed in this group. The multivariate analysis identified 7 independent predictors of a PAS: younger age ($P < .001$; odds ratio [OR]: 0.97), longer disease duration ($P = .021$; OR: 1.01), tentorial ($P < .001$; OR: 8.56), transverse–sigmoid sinus ($P < .001$; OR: 2.58), and multiple locations ($P < .001$; OR: 5.20), venous ectasia ($P = .019$; OR: 1.53), and venous congestion on angiograms ($P < .001$; OR: 5.30).

Treatment Outcomes and Prognosis in DAVF With and Without a PAS

Table 2 provides a comparison of treatment characteristics and outcomes between DAVF patients with and without a PAS. There was a notable difference in the treatment modalities for fistulas with PAS compared with those without PAS ($P < .001$). In this cohort, fistulas with PAS were more likely to be treated endovascularly compared with those without PAS. Patients with

TABLE 1. Comparison of Patients With and Without Pial Arterial Supply in 1101 Cases of Intracranial Dural Arteriovenous Fistulas

Variable, n (% of patients)	With pial arterial supply (n = 259)	Without pial arterial supply (n = 842)	*P* value Univariate analysis	Multivariate analysis	
				OR (95% CI)	*P* value
Age (mean ± SD), y	46.0 ± 12.3	52.4 ± 12.4	**<0.001**	0.97 (0.95-0.98)	**<.001**
Female	80 (30.9%)	295 (35.0%)	0.218		
Disease duration (mo), median, IQR	4 (1-22)	3 (1-8)	**0.002**	1.01 (1.01-1.02)	**.021**
Previous treatment	46 (17.8)	82 (9.7)	**<0.001**	1.26 (0.78-2.03)	.352
Location					
Transverse–sigmoid	67 (25.9%)	173 (20.6%)	0.070	2.58 (1.58-4.22)	**<.001**
Tentorial	97 (37.5%)	135 (16.0%)	**<0.001**	8.56 (5.33-13.76)	**<.001**
Cavernous sinus	0 (0.0%)	195 (23.2%)	**<0.001**		
Foramen magnum	2 (0.8%)	52 (6.2%)	**<0.001**		
Superior sagittal sinus/convexity	28 (10.8%)	81 (9.6%)	0.575		
Jugular foramen/hypoglossal canal	4 (1.5%)	59 (7.0%)	**0.002**		
Anterior cranial fossa	6 (2.3%)	49 (5.8%)	**0.024**		
Sylvian/middle cranial fossa	1 (0.4%)	26 (3.1%)	**0.026**		
Multiple	54 (20.8%)	65 (7.7%)	**<0.001**	5.20 (2.98-9.07)	**<.001**
Other	0 (0.0%)	7 (0.8%)	0.209		
Borden classification					
I	41 (15.8%)	326 (38.7%)	**<0.001**		
II	76 (29.3%)	96 (11.4)	**<0.001**		
III	142 (54.8%)	420 (49.9%)	0.164		
Aggressive symptoms	139 (53.7%)	312 (37.1%)	**<0.001**	1.19 (0.84-1.70)	.326
Intracranial hemorrhage	47 (18.2%)	129 (15.3%)	0.278		
Venous dilation	207 (79.9%)	645 (76.6%)	0.264		
Venous ectasia	128 (49.4%)	250 (29.7%)	**<0.001**	1.53 (1.07-2.19)	**.019**
Venous congestion	138 (53.3%)	118 (14.0%)	**<0.001**	5.30 (3.48-8.07)	**<.001**
Draining vein stenosis/occlusion	129 (49.8%)	205 (24.4%)	**<0.001**	1.28 (0.86-1.90)	.228

OR, odds ratio.
Values in bold indicate statistical significance.
For cases of multiple dural arteriovenous fistulas, classification was based on the lesion with the highest grade.

DAVFs involving PAS demonstrated lower immediate complete occlusion rates ($P < .001$, 95% CI = 0.17-0.35), higher incidences of both permanent ($P < .001$, 95% CI = 1.94-6.73) and temporary complications ($P = .004$, 95% CI = 1.22-3.08), a higher risk of postoperative ICH ($P < .001$, 95% CI = 3.23-14.98) and cerebral infarction ($P < .001$, 95% CI = 3.81-19.93), and higher DAVF-related/treatment-related mortality ($P < .001$, 95% CI = 2.05-10.04) compared with those without PAS. Special attention is given to the treatment complications of postoperative ICH and cerebral infarction in DAVFs with PAS. Among the 1101 patients diagnosed with DAVFs, 21 patients with PAS showed complications of ICH and cerebral infarction, as observed on computed tomography or MRI scans, respectively. In comparison, 10 and 8 patients without a PAS presented with ICH and infarction complications, respectively. In the DAVF with PAS group, cerebral infarction complications were directly attributed to the

TABLE 2. Treatment Characteristics and Outcomes in Patients With and Without Pial Arterial Supply Among 1101 Cases of Intracranial Dural Arteriovenous Fistulas

Variable, n (% of patients)	With pial arterial supply (n = 259)	Without pial arterial supply (n = 842)	*P* value
Treatment modality			**<.001**
Conservative treatment	1 (0.4)	47 (5.6)	
Embolization alone	235 (90.7)	687 (81.6)	
Surgery alone	7 (2.7)	89 (10.6)	
Embolization + surgery	13 (5.0)	19 (2.3)	
Embolization + radiosurgery	3 (1.2)	0 (0.0)	
Immediate obliteration rate[a]	179/258 (69.4)	718/795 (90.3)	**<.001**
Permanent complications[a]	22/258 (8.6)	20/795 (2.5)	**<.001**
Temporary complications[a]	32/258 (12.4)	54/795 (6.8)	**.004**
Postoperative intracranial hemorrhage[a]	21/258 (8.1)	10/795 (1.3)	**<.001**
Postoperative intracranial infarction[a]	21/258 (8.1)	8/795 (1.0)	**<.001**
Death related to fistulae/treatment[b]	15/216 (6.9)	11/680 (1.6)	**<.001**

[a]Based on 1053 patients who received treatment.
[b]Based on 896 patients who received clinical follow-up after treatment.
Values in bold indicate statistical significance.

embolization of the PAS in 17 cases (17/21, 81.0%) (Figure 5). The remaining cases occurred after the embolization of other dural branches or after embolization of venous pathways. ICH complications were directly attributed to pial artery rupture during embolization in 12 (12/21, 57.1%) cases (Figure 6). The remaining cases occurred after the embolization of other dural branches (Figures 1 and 2) or transvenous embolization. These cases may have resulted from PAS rupture because of previous occlusion of the venous side or complications related to venous pathways.

Table 3 provides a comparison of treatment characteristics and outcomes between DAVF patients with and without a PAS after propensity scores match. Patients with DAVFs involving PAS still demonstrated lower immediate complete occlusion rates (P = .001, 95% CI = 0.30-0.78), higher incidences of permanent (P = .012, 95% CI = 1.24-8.17) complications, and a higher risk of postoperative ICH (P = .013, 95% CI = 1.24-8.17) and cerebral infarction (P = .001, 95% CI = 2.08-40.02) compared with those without PAS.

Classification of PAS

Table 4 summarizes the characteristics of each PAS. The mean age of patients with a pure pial supply was 46.4 years, while those with both a dilated dural branch and a pure pial supply were younger, with a mean age of 42.7 years. The patients in both groups were younger than the average age of 48.2 years observed in those with a dilated dural branch of a pial artery alone. DAVFs with both dilated dural branches and pure pial supplies exhibit a higher proportion of venous congestion on angiograms compared with the other 2 groups (P < .001, 95% CI = 0.28-0.81).

Treatment Outcomes in DAVFs With PAS Using Different Strategies

Table 5 presents a comparison of the treatment characteristics and outcomes of 258 DAVFs with PAS, categorized by treatment strategy. In this subgroup analysis, the group undergoing pial artery embolization exhibited a higher rate of permanent complications (P = .036, 95% CI = 1.14-10.61). The rates of postoperative ICH (9.9% vs 5.6%, P = .211) and ischemic complications (11.9% vs 2.8%, P = .016, 95% CI = 1.35-16.36) were higher in the embolization group.

Specifically, embolization via a pial artery was performed in 28 patients primarily exhibiting a dilated preexisting dural branch and in 123 patients with predominantly pure PAS. A further subgroup analysis was conducted comparing embolization of pure PAS (n = 123) with embolization of a dilated preexisting dural branch (n = 28) (data not shown in the table). There was no statistically significant difference in the incidence of postoperative ICH between the 2 groups (14/123, 11.4% vs 1/28, 3.6%, P = .370), despite a higher overall complication rate in the pure PAS embolization group. However, a statistically significant difference was observed in the occurrence of ischemic complications (18/123, 14.6% vs 0/28, 0.0%, P = .026) between the groups.

In the cohort of 110 DAVF patients with pure PAS, 108 underwent endovascular treatment, while 2 patients underwent direct surgical ligation of the draining vein. Among the 108 patients with

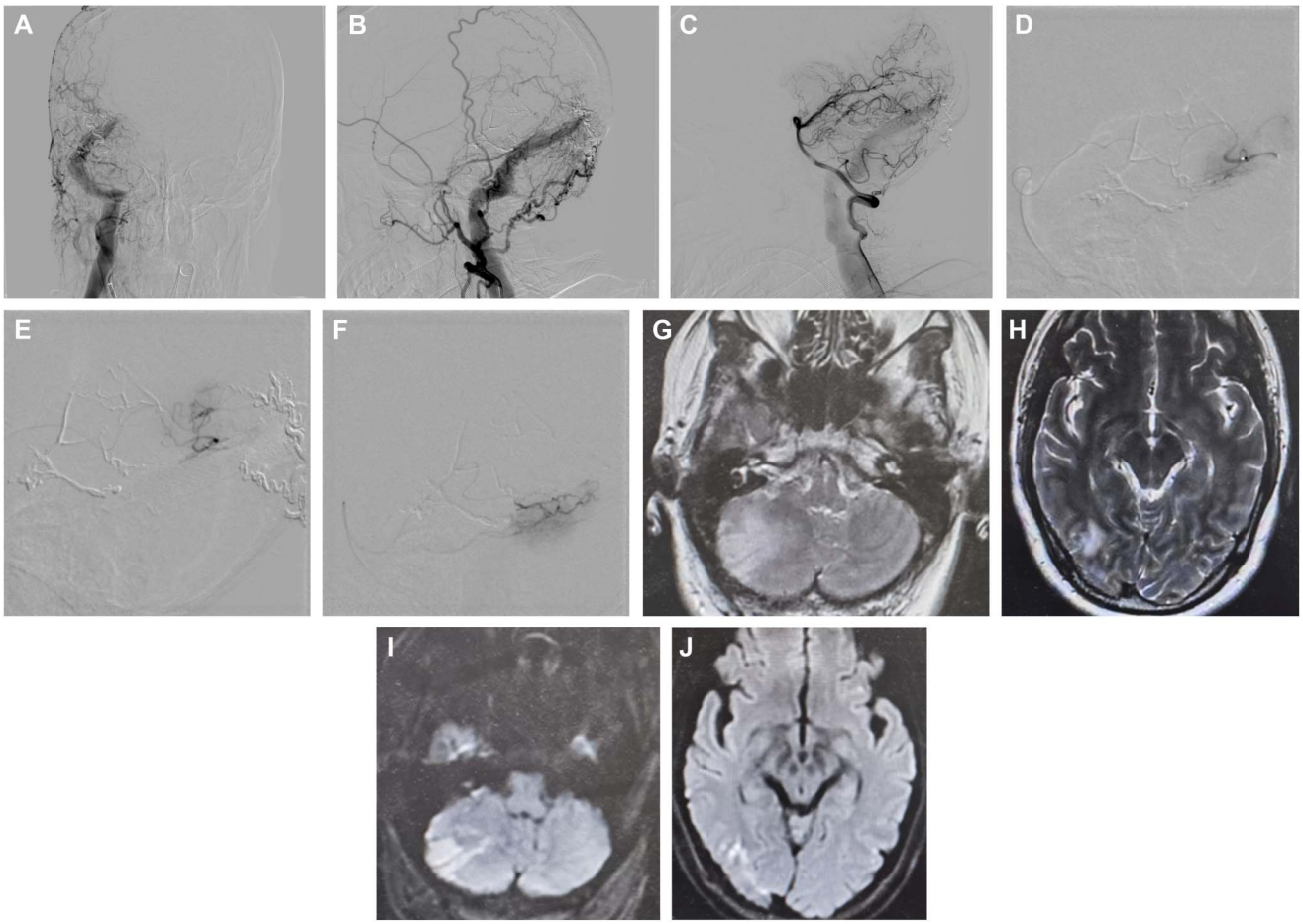

FIGURE 5. **A-C**, *This is a Borden type I dural arteriovenous fistula in the right transverse–sigmoid sinus region, likely secondary to previous venous sinus stenting. The patient had previously undergone endovascular treatment at other institution. The fistulous network was relatively diffuse, with arterial feeders arising from multiple dural branches of the external carotid artery and several pial branches from the posterior circulation.* **D-F**, *Superselective angiography of the posterior cerebral artery and superior cerebellar artery revealed multiple tortuous pure pial and dural arterial feeders. During embolization, the microcatheter was advanced as close to the fistulous point as possible, and Glubran was used for embolization.* **G-J**, *However, postoperative MRI revealed new infarcts in the right cerebellum and occipital lobe (***G** *and* **H,** *T2-weighted images;* **I** *and* **J,** *diffusion-weighted imaging).*

DAVF who underwent endovascular treatment, 70 received embolization of the pure PAS. A further subgroup analysis was performed comparing patients who underwent embolization of the pure PAS (n = 70) with those who did not undergo pure PAS embolization (n = 38) (data not shown in the table). No statistically significant difference was found in the incidence of postoperative ICH between the 2 groups (3/70, 4.3% vs 3/38, 7.9%, P = .732). However, a statistically significant difference was observed in the occurrence of ischemic complications (10/70, 14.3% vs 0/38, 0.0%, P = .014).

DISCUSSION

In this study, an additional PAS was observed in 23.5% of patients with DAVFs, accounting for 259 out of 1101 cases. Patients with DAVFs in the PAS group were younger on average, had a longer disease duration, and were more likely to have undergone previous endovascular treatment compared with those in the non-PAS group. DAVFs with PAS were more likely to have a higher Borden grade, making them more prone to presenting with aggressive symptoms. In terms of location, tentorial, transverse–sigmoid sinus, and multiple fistulas were more commonly observed in the PAS group. Complication rates were higher in the PAS group, particularly for postoperative hemorrhage and ischemia-related complications, which remained a significant concern even after propensity score matching. In the PAS group, 57.1% of postoperative hemorrhagic complications and 81.0% of postoperative ischemic complications were attributed to intraoperative embolization of the pial artery. A subgroup analysis within the PAS group comparing patients with and without pial artery embolization revealed that those who underwent pial artery embolization had higher rates of postoperative hemorrhagic and ischemic complications, with ischemia-related

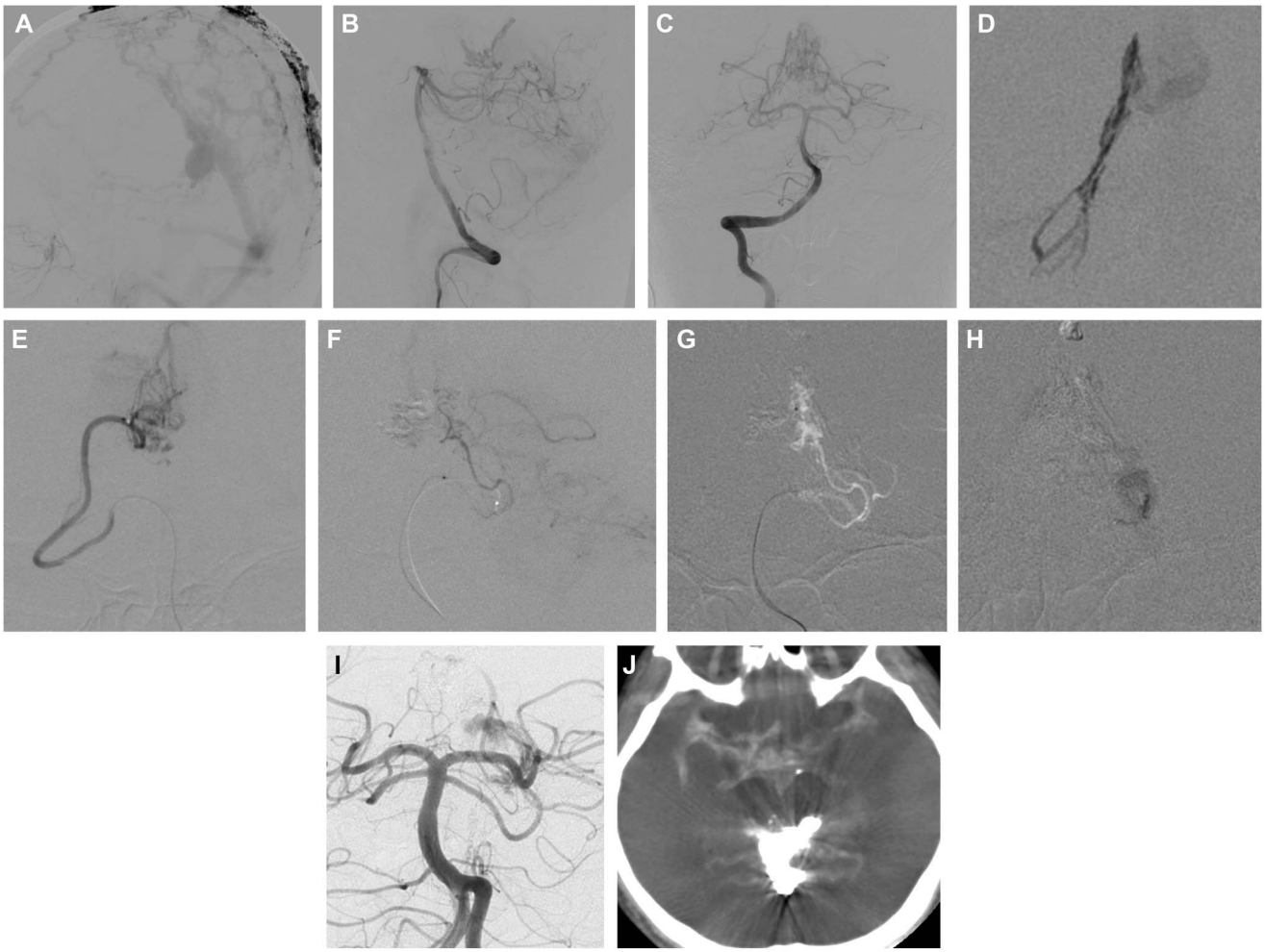

FIGURE 6. **A-C**, *This is a case of a high-grade dural arteriovenous fistula in the Galenic region, with arterial supply from multiple dural branches of the external carotid artery, as well as both dural and pure pial arterial feeders arising from the PCA.* **D**, *Superselective angiography of the right PCA revealed a linear dural arterial feeder.* **E** *and* **F**, *In addition, tortuous and ramified pure pial arterial feeders arising from the posterior cerebral arteries were identified bilaterally.* **G-I**, *Arterial rupture was suspected during embolization via the left pial arterial feeder.* **J**, *Postoperative DynaCT revealed the presence of subarachnoid hemorrhage. PCA, posterior cerebral artery.*

complications showing a statistically significant difference. In the PAS group, among hemorrhagic and ischemic complications, only 1 instance of intracranial infarction and 5 instances of ICH were not associated with pial artery embolization. These findings are significant, as they indicate that PAS is a relatively common phenomenon, accounting for over one-fifth of all DAVFs. Pial artery embolization in DAVFs with PAS carries a higher risk of complications, which seems to surpass the potential risks of not embolizing the PAS before DAVF occlusion and is generally unnecessary.

Intracranial Hemorrhagic Complications Associated With PAS

In the endovascular treatment of DAVFs with PAS, ICH complications related to PAS can be broadly divided into 2 categories. The first involves rupture and bleeding of the pial arteries during the embolization of the PAS (Figure 6). The second occurs when PAS is left untreated after the complete occlusion of dural arterial feeders. In such cases, fragile pial arteries may rupture because of increased pressure caused by restricted venous drainage after arterial embolization (Figures 1 and 2) (Video 1). This mechanism resembles that observed in arteriovenous malformation embolization, where feeding arteries may rupture if the draining vein is completely occluded first.[8,14,15] However, the second cause remains largely speculative and is primarily supported by evidence from case series and case reports, with an incidence likely to be very low.[4-7] This has led to controversy regarding whether pial artery embolization should be performed in DAVFs with PAS before complete obliteration of the DAVF. Recent studies have shown that even without embolization of PAS, the risk of postoperative hemorrhagic

TABLE 3. Matched Comparison of Patients With and Without Pial Arterial Supply in 438 Cases of Intracranial Dural Arteriovenous Fistulas

Variable, n (% of patients)	With pial arterial supply (n = 219)	Without pial arterial supply (n = 219)	*P* value
Age (mean ± SD), y	46.9 ± 12.2	47.8 ± 12.0	.437
Female	67 (30.6)	62 (28.3)	.600
Disease duration (mo), median, IQR	4 (1-18)	3 (1-10)	.071
Previous treatment	37 (16.9)	34 (15.5)	.697
Location			.061
Transverse–sigmoid	62 (28.3)	56 (25.6)	
Tentorial	82 (37.4)	86 (39.3)	
Cavernous sinus	0 (0.0)	5 (2.3)	
Foramen magnum	2 (0.9)	1 (0.5)	
Superior sagittal sinus/convexity	28 (12.8)	16 (7.3)	
Jugular foramen/hypoglossal canal	4 (1.8)	5 (2.3)	
Anterior cranial fossa	6 (2.7)	6 (2.7)	
Sylvian/middle cranial fossa	1 (0.5)	6 (2.7)	
Multiple	34 (15.5)	35 (16.0)	
Other	0 (0.0)	3 (1.4)	
Borden classification			.066
I	35 (16.0)	36 (16.4)	
II	59 (26.9)	39 (17.8)	
III	144 (65.8)	125 (57.1)	
Aggressive symptoms	117 (53.4)	114 (52.1)	.774
Intracranial hemorrhage	42 (19.2)	38 (17.4)	.621
Venous dilation	177 (80.8)	182 (83.1)	.534
Venous ectasia	106 (48.4)	109 (49.8)	.774
Venous congestion	99 (45.2)	96 (43.8)	.773
Draining vein stenosis/occlusion	98 (44.8)	96 (43.8)	.847
Treatment modality			.216
Conservative treatment	1 (0.5)	3 (1.4)	
Embolization alone	196 (89.5)	193 (88.1)	
Surgery alone	7 (3.2)	13 (5.9)	
Embolization + surgery	12 (5.5)	10 (4.6)	
Embolization + radiosurgery	3 (1.4)	0 (0.0)	
Immediate obliteration rate[a]	159/218 (72.9)	185/216 (85.7)	**.001**
Permanent complications[a]	18/218 (8.3)	6/216 (2.7)	**.012**
Temporary complications[a]	26/218 (11.9)	14/216 (6.5)	.050
Postoperative intracranial hemorrhage[a]	18/218 (8.3)	6/216 (2.8)	**.013**

TABLE 3. Continued.

Variable, n (% of patients)	With pial arterial supply (n = 219)	Without pial arterial supply (n = 219)	*P* value
Postoperative intracranial infarction[a]	17/218 (7.8)	2/216 (0.9)	**.001**
Death related to fistulae/treatment[b]	12/188 (6.4)	8/198 (4.0)	.299

[a]Based on 434 patients who received treatment.
[b]Based on 386 patients who received clinical follow-up after treatment.
Values in bold indicate statistical significance.

complications does not increase.[1,2,16] In this cohort, a subgroup analysis of DAVFs with PAS showed a higher incidence of postoperative ICH in patients who underwent PAS embolization compared with those who did not; however, the difference did not reach statistical significance.

Sato et al[6] reported a case of a tentorial DAVF complicated by severe ICH after Onyx embolization. Emergency craniotomy revealed persistent arterial bleeding from a glomus-like vascular structure near the proximal portion of the embolized draining vein, which was supplied by a pial artery originating from the posterior cerebral artery. The nidus-like structure from the PAS is not observed in every case of DAVF with PAS. This nidus-like structure within the PAS may potentially be the underlying cause of hemorrhagic complications when the PAS is not embolized before complete DAVF closure. Embolization of the PAS before complete DAVF closure may be required only in cases of DAVF with pure PAS that exhibit a nidus-like structure.

Ischemic Stroke Complications Associated With PAS

In the endovascular treatment of DAVFs with PAS, ischemic stroke complications related to PAS can also be categorized into 2 types. The first involves embolization of the pial arteries, which can lead to infarction in the brain parenchyma supplied by these arteries (Figure 5). The second occurs during transarterial embolization of DAVFs with PAS, where embolic agents may migrate retrogradely (from the dural arteries to the pial arteries), potentially obstructing blood flow to the associated brain parenchyma and causing ischemia.[3] DAVFs with PAS exhibited a higher rate of postoperative ischemic complications compared with those without PAS. Furthermore, in this cohort, embolization of the PAS itself understandably significantly increased the risk of ischemic stroke complications compared with leaving these feeders untreated.

Limitations

There are several limitations to the findings reported in this study. First, the single-center nature of our cases somewhat limits the generalizability of these findings to patients managed at academic centers. Among the 258 treated DAVF cases with PAS, 151 (58.5%) underwent embolization of the PAS, which undoubtedly contributed to an increased incidence of ICH and ischemic complications. Furthermore, the immediate complete cure rate after the final treatment of DAVFs with PAS was relatively low. This outcome was not only influenced by the complex angioarchitecture but also likely associated with the impact of multiple embolizations of the PAS on the treatment process. Second, because superselective angiography was not performed in all patients in this retrospective study, low-flow or small additional PAS may have gone undetected, and the specific classification may have been misjudged. Third, the presumed hemorrhagic complications resulting from a "pure" pial supply not being obliterated before the DAVF is closed were not definitively observed in our cases, although a few instances were suspected to be due to rupture of an unobliterated "pure" pial supply before fistula obliteration (Figures 1 and 2). Moreover, in this study, only 2 cases in the pure pial artery group underwent direct surgical treatment, with no observed complications. However, the small sample size limits the feasibility of meaningful statistical analysis or comprehensive clinical evaluation. Nonetheless, existing literature discusses surgical treatment for DAVFs with PAS.[6] Staged embolization or a combined approach involving surgical disconnection and embolization may provide a safer alternative to complete single-session Onyx embolization, reducing the risk of hemorrhagic complications. At a minimum, major pial feeding arteries—and ideally, the entire pial network—should be obliterated before attempting curative embolization. However, these recommendations remain largely theoretical, as they are based on individual case reports. Despite this potential risk, the relatively low likelihood of hemorrhage is far outweighed by the significantly higher risks of both hemorrhage and ischemia associated with PAS embolization.

CONCLUSION

In this retrospective study, a PAS was identified in 259 out of 1101 patients with DAVFs. The findings suggest that embolization of PAS before DAVF closure may significantly increase the risk of both intracranial hemorrhagic and ischemic complications. Therefore, routine embolization of PAS before DAVF closure is not supported by these results, especially considering the exceptionally low incidence of presumed hemorrhagic complications arising from an unobliterated "pure" pial supply before DAVF obliteration.

TABLE 4. Characteristics of 259 Dural Arteriovenous Fistulas With Pial Arterial Supplies in Different Classifications

Variable, n (% of patients)	Dilated dural branch (n = 79)	Pure pial supply (n = 110)	Dilated dural and pure pial supply (n = 70)	*P* value
Age (mean ± SD), y	48.2 ± 12.8	46.4 ± 11.6	42.7 ± 12.3	**.023**
Female	19 (24.1)	36 (32.7)	25 (35.7)	.263
Disease duration (mo), median, IQR	3 (1-12)	3 (1-18)	10 (3-24)	**.001**
Feeding arteries				
ACA	6 (7.8)	18 (16.4)	15 (21.4)	.055
PCA	33 (41.8)	50 (45.5)	54 (77.1)	**<.001**
SCA	26 (32.9)	21 (19.1)	35 (50.0)	**<.001**
AICA	11 (13.9)	10 (9.1)	21 (30.0)	**<.001**
PICA	12 (15.2)	0 (0.0)	22 (31.4)	**<.001**
MCA	0 (0.0)	24 (21.8)	34 (48.6)	**<.001**
Location				**.004**
Transverse–sigmoid	16 (20.3)	26 (23.6)	25 (35.7)	
Tentorial	35 (44.3)	43 (39.1)	19 (27.1)	
Foramen magnum	2 (2.53)	0 (0.0)	0 (0.0)	
Superior sagittal sinus/convexity	11 (13.9)	13 (11.8)	4 (5.7)	
Jugular foramen/hypoglossal canal	3 (3.8)	0 (0.0)	1 (1.43)	
Anterior cranial fossa	1 (1.3)	5 (4.6)	0 (0.0)	
Sylvian/middle cranial fossa	1 (1.3)	0 (0.0)	0 (0.0)	
Multiple	10 (12.7)	23 (20.9)	21 (30.0)	
Borden classification				**<.001**
I	14 (17.7)	14 (12.7)	13 (18.6)	
II	14 (17.7)	29 (26.4)	33 (47.1)	
III	51 (64.6)	67 (60.9)	24 (34.3)	
Aggressive symptoms	42 (53.2)	57 (51.8)	40 (57.1)	.779
Intracranial hemorrhage	16 (20.3)	24 (21.8)	7 (10.0)	.113
Venous dilation	62 (78.5)	90 (81.8)	55 (78.6)	.807
Venous ectasia	34 (43.0)	60 (54.6)	34 (48.6)	.292
Venous congestion	32 (40.5)	56 (50.9)	50 (71.4)	**<.001**
Draining vein stenosis/occlusion	37 (46.8)	51 (46.4)	41 (58.6)	.229

ACA, anterior cerebral artery; AICA, anterior inferior cerebellar artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery.
Values in bold indicate statistical significance.
For cases of multiple dural arteriovenous fistulas, classification was based on the lesion with the highest grade.

TABLE 5. Treatment Characteristics and Outcomes of 258 Dural Arteriovenous Fistulas With Pial Arterial Supply by Treatment Strategy Classification

Variable, n (% of patients)	Embolized pial arterial supply group (n = 151)	Nonembolized pial arterial supply group (n = 107)	*P* value
Immediate obliteration rate	104 (68.9)	75 (70.1)	.834
Permanent complications	18 (11.9)	4 (3.7)	**.036**
Temporary complications	20 (13.3)	12 (11.2)	.626
Postoperative intracranial hemorrhage	15 (9.9)	6 (5.6)	.211
Postoperative intracranial infarction	18 (11.9)	3 (2.8)	**.016**
Death related to fistulae/treatment	8 (5.3)	7 (6.5)	.674

Values in bold indicate statistical significance.

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VIDEO. A case, corresponding to Figures 1 and 2, of presumed hemorrhagic complications resulting from an unobliterated 'pure' pial arterial supply before DAVF obliteration.[17-19]

COMMENTS

Intracranial dural arteriovenous fistulas (DAVF) are formed by abnormal connections between arteries and veins without involving the

brain parenchyma. Arterial supply to DAVFs is typically from meningeal arteries. Less commonly, pial arteries can contribute to DAVFs. The presence of pial arterial supply (PAS) has been shown to be associated with higher rates of hemorrhagic presentation. Treatment strategies include surgical ligation of the fistula and transarterial/transvenous embolization. However, the necessity of embolizing the PAS prior to the fistula remains debated. Some suggest that embolizing the fistula without addressing the PAS may predispose the patient to a higher risk of rupture of the pial arteries, while others argue that the risks of embolizing the PAS outweigh any potential benefits. These risks include vessel perforation and strokes from inadvertent infiltration of embolic agents through anastomotic connections.[1-4] This study attempts to address 2 questions: 1. differences in presentation/outcomes of DAVF with PAS and those without, and 2. whether embolizing the PAS prior to DAVF increases postoperative complications. To answer the first question: the authors successfully demonstrate that the PAS group has higher overall rates of postoperative hemorrhage and infarction compared to those without PAS. Regarding the second question, the authors argue that embolization of PAS prior to the fistula is not recommended and is associated with higher postoperative intracranial hemorrhage. However, the subgroup analysis of the PAS embolized group failed to demonstrate statistical significance in postoperative hemorrhagic rate compared to the PAS nonembolized group ($P = .211$). This is one of the largest studies of intracranial DAVF with PAS to date and demonstrates a higher risk of complications within this group. However, the data do not support the authors' claims that embolizing PAS prior to DAVF is associated with higher postoperative hemorrhage. Careful interpretation of the data is recommended to avoid overreaching conclusions.

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