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CLINICAL ARTICLE

Assessing the rate, natural history, and treatment trends of intracranial aneurysms in patients with intracranial dural arteriovenous fistulas: a Consortium for Dural Arteriovenous Fistula Outcomes Research (CONDOR) investigation

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OBJECTIVE There is a reported elevated risk of cerebral aneurysms in patients with intracranial dural arteriovenous fistulas (dAVFs). However, the natural history, rate of spontaneous regression, and ideal treatment regimen are not well characterized. In this study, the authors aimed to describe the characteristics of patients with dAVFs and intracranial aneurysms and propose a classification system.

METHODS The Consortium for Dural Arteriovenous Fistula Outcomes Research (CONDOR) database from 12 centers was retrospectively reviewed. Analysis was performed to compare dAVF patients with (dAVF+ cohort) and without (dAVF-only cohort) concomitant aneurysm. Aneurysms were categorized based on location as a dAVF flow-related aneurysm (FRA) or a dAVF non–flow-related aneurysm (NFRA), with further classification as extra- or intradural. Patients

ABBREVIATIONS AVM = arteriovenous malformation; CONDOR = Consortium for Dural Arteriovenous Fistula Outcomes Research; dAVF = dural arteriovenous fistula; ECA = external carotid artery; FRA = flow-related aneurysm; ICA = internal carotid artery; NFRA = non–flow-related aneurysm.

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with traumatic pseudoaneurysms or aneurysms with associated arteriovenous malformations were excluded from the analysis. Patient demographics, dAVF anatomical information, aneurysm information, and follow-up data were collected.

RESULTS Of the 1077 patients, 1043 were eligible for inclusion, comprising 978 (93.8%) and 65 (6.2%) in the dAVF-only and dAVF+ cohorts, respectively. There were 96 aneurysms in the dAVF+ cohort; 10 patients (1%) harbored 12 FRAs, and 55 patients (5.3%) harbored 84 NFRAs. Dural AVF+ patients had higher rates of smoking (59.3% vs 35.2%, $p < 0.001$) and illicit drug use (5.8% vs 1.5%, $p = 0.02$). Sixteen dAVF+ patients (24.6%) presented with aneurysm rupture, which represented 16.7% of the total aneurysms. One patient (1.5%) had aneurysm rupture during follow-up. Patients with dAVF+ were more likely to have a dAVF located in nonconventional locations, less likely to have arterial supply to the dAVF from external carotid artery branches, and more likely to have supply from pial branches. Rates of cortical venous drainage and Borden type distributions were comparable between cohorts. A minority (12.5%) of aneurysms were FRAs. The majority of the aneurysms underwent treatment via either endovascular (36.5%) or microsurgical (15.6%) technique. A small proportion of aneurysms managed conservatively either with or without dAVF treatment spontaneously regressed (6.2%).

CONCLUSIONS Patients with dAVF have a similar risk of harboring a concomitant intracranial aneurysm unrelated to the dAVF (5.3%) compared with the general population (approximately 2%–5%) and a rare risk (0.9%) of harboring an FRA. Only 50% of FRAs are intradural. Dural AVF+ patients have differences in dAVF angioarchitecture. A subset of dAVF+ patients harbor FRAs that may regress after dAVF treatment.

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KEYWORDS dural arteriovenous fistula; feeding artery aneurysm; vascular disorders

Aneurysms associated with brain arteriovenous malformations (AVMs)[1,2] and moyamoya disease[3] represent a distinct pathology compared with saccular aneurysms, including higher rupture risk and spontaneous resolution after AVM treatment.[4] Multiple case reports and small series have collectively reported an elevated risk of associated aneurysm in patients with intracranial dural arteriovenous fistula (dAVF) ranging from 13% to 21%.[5,6] A limitation in these studies is the lack of a control group of dAVF patients without aneurysms. Furthermore, little is known regarding their natural history, rate of spontaneous regression or dAVF treatment-related regression, and ideal management for this population.

The Consortium for Dural Arteriovenous Fistula Outcomes Research (CONDOR) has collected information for more than 1000 patients with intracranial dAVF. Using this database, we aimed to define 1) the prevalence of aneurysms in dAVF patients; 2) subtypes of aneurysms in dAVF patients; 3) differences in baseline characteristics between dAVF patients with and without aneurysms; and 4) the treatment trends of intracranial aneurysms.

Methods

The infrastructure for CONDOR is described elsewhere in detail.[39] Briefly, collaborating institutions obtained individual institutional review board approval to conduct a retrospective analysis of all dAVFs treated at each institution. Details were collected from the clinical record and imaging databases, de-identified, and shared via a third-party host institution. Discrepancies in coded patient information were addressed directly with each contributing center. Twelve centers contributed a total of 1077 patients with dAVF presenting between 1990 and 2017. Patients were excluded if the status of a coexisting aneurysm was missing or unknown.

Patients were dichotomized into two cohorts based on the presence of aneurysms: 1) dAVF patients with coexisting aneurysms (dAVF+ cohort); and 2) dAVF patients without coexisting aneurysms (dAVF-only cohort). Statistical analysis was performed by an independent statistician, using the Mann-Whitney U-test for continuous variables and Fisher's exact test for categorical variables. Continuous variables are presented as the mean ± SD. No adjustments were made for multiple comparisons. Aneurysms were then further categorized as either intra- or extradural, and as either a dAVF flow-related aneurysm (FRA), defined as an aneurysm located on an arterial pedicle to the dAVF, or a completely dAVF non–flow-related aneurysm (NFRA). Aneurysms were also categorized (separately) based on their location in relation to the dAVF (i.e., ipsilateral or contralateral). Patients with bilateral aneurysms, midline aneurysm(s), or midline dAVFs were assigned to the "other" group. Predictors of associated aneurysms were identified using a multivariable logistic regression model with forward selection algorithm ($p < 0.05$ to enter), on a per-patient basis (i.e., each patient was enrolled in the model once, irrespective of the number of aneurysms). Any potential bias associated with missing covariate information was accounted for using multiple imputation by chained equations.

Results

Of the 1077 patients maintained in the CONDOR data set, 1043 were eligible for inclusion in this study based on the known presence or absence of coexisting intracranial aneurysms. The dAVF-only and dAVF+ cohorts comprised 978 (93.8%) and 65 (6.2%) patients, respectively. The dAVF+ cohort had a total of 96 aneurysms (mean 1.48 aneurysm/patient). The total mean clinical and radiographic follow-ups for the dAVF+ cohort were 3 ± 3.29 and 2.38 ± 3.01 years, respectively.

Table 1 compares patient demographics between dAVF-only and dAVF+ cohorts. The two cohorts were comparable, with the exception of higher rates of illicit drug use ($p = 0.02$) and smoking ($p < 0.001$) in the dAVF+ cohort. Table 2 shows all the aneurysms and the specific locations, categorized into FRA or NFRA, and intra- or extradural. Fifty-five patients (5.3%) had 84 NFRAs, most

TABLE 1. Demographics for patients with dAVF-only versus those with dAVF and aneurysm

	dAVF Only (n = 978)	dAVF+ (n = 65)	p Value
Age	59.5 ± 14.5	60.8 ± 11.3	0.596
Female	428/978 (43.8)	35/65 (53.8)	0.123
CAD	73/960 (7.6)	6/63 (9.5)	0.623
DM	121/962 (12.6)	10/64 (15.6)	0.442
HTN	392/962 (40.7)	33/64 (51.6)	0.115
Cancer	119/961 (12.4)	11/63 (17.5)	0.241
Illicit drugs	12/823 (1.5)	3/52 (5.8)	**0.020**
Smoking	275/781 (35.2)	32/54 (59.3)	**<0.001**

CAD = coronary artery disease; DM = diabetes mellitus; HTN = hypertension. Values represent the number of patients/total number of patients with data (%) or mean ± SD unless stated otherwise. Boldface type indicates statistical significance; p values do not adjust for multiple comparisons.

TABLE 2. Locations of all 96 aneurysms and the 16 ruptured aneurysms

	Location	Total	Ruptured
Unrelated (n = 84)			
Extradural (n = 13)	ICA (cavernous segment)	10	0
	ICA (cervical, petrous, lacerum segments)	2	0
	ICA (clinoidal segment)	1	0
Intradural (n = 71)	ICA (ophthalmic segment & "supraclinoid")	14	1
	M1/M2/MCA bifurcation	11	0
	A1/ACoA	11	5
	ICA (communicating segment)	10	3
	V4/VB junction	6	2
	ICA, "paraclinoid"	5	0
	Anterior choroidal artery	4	1
	Midbasilar	2	1
	ICA (terminus)	2	0
	A3, A4, & "pericallosal"	2	0
	ICA ("cave")	2	0
	PICA	1	1
	Basilar tip	1	0
	Posterior choroidal artery (FRA for tumor)	1*	0
FRA (n = 12)			
Extradural (n = 6)	Ophthalmic artery	3	0
	ICA (cavernous segment)	1	0
	Inferolateral trunk	1	0
	Posterior meningeal artery	1	0
Intradural (n = 6)	PICA	2	1
	AICA	1	1
	ACoA	1	0
	SCA	1	0
	PCA	1	0

ACoA = anterior communicating artery; AICA = anterior inferior cerebellar artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; PICA = posterior inferior cerebellar artery; SCA = superior cerebellar artery; VB = vertebrobasilar.
* Coded as "multiple aneurysms" but counted as 1 for purposes of table.

of which (84.5%) were intradural. There were no ruptured extradural aneurysms. Ten patients (1%) had 12 FRAs, half of which were intradural and half were extradural. The ICA was the most common location for aneurysms, with 33 (34.4%) being either "paraclinoid" (i.e., including the cavernous and clinoid segments, and carotid cave aneurysms) or supraclinoid (i.e., ophthalmic segment), 11 (11.5%) in the anterior communicating artery and A_1 segment, and 11 in the middle cerebral artery bifurcation and M_1 segment.

Table 3 compares the presentations and dAVF angioarchitecture between dAVF-only and dAVF+ cohorts, and then between FRA and NFRA/dAVF-only cohorts. Patients in the dAVF-only cohort were more likely to present with dAVF symptoms ($p < 0.001$). Patients in the dAVF-only cohort also had a higher rate of dAVF rupture, although this did not achieve statistical significance. Patients in the dAVF+ cohort were more likely to have dAVFs located along the convexity or with superior sagittal sinus drainage ($p = 0.002$). Conversely, patients in the dAVF-only cohort were more likely to have dAVFs in the region of the cavernous sinus (i.e., cavernous-carotid fistula, $p = 0.038$). The most common dAVF location for both cohorts was the transverse-sigmoid junction. Pial arterial supply to the dAVF was more common in the dAVF+ cohort, whereas conventional dAVF supply from the external carotid artery (ECA) and dural branches of the internal carotid artery (ICA) was more commonly detected in the dAVF-only cohort. Similarly, conventional venous sinus drainage (i.e., to the transverse-sigmoid sinus junction) was more common in the dAVF cohort. Rates of cortical venous drainage and Borden type distributions were similar between the two cohorts, although the dAVF-only cohort had a higher rate of venous ectasia ($p = 0.048$). These relationships were all very similar when comparing FRA with NFRA/dAVF-only groups. Table 4 demonstrates the independent predictors of coexisting aneurysms in dAVF patients. Current smoking status, convexity location, and pial arterial supply were independent predictors of the presence of aneurysms, while venous ectasia was an independent predictor of the absence of aneurysms. Table 4 displays the details for the nonimputed multivariate model as well (complete-case analysis).

Table 5 and Fig. 1 show details of the dAVF+ cohort. Multiple aneurysms were seen in 24.6% of the dAVF+ patients. Most (57.3%) aneurysms were small (< 7 mm), and only 1 (1.0%) was giant (≥ 25 mm). Most aneurysms (78.1%) were saccular. The majority of patients (88%) were diagnosed with an aneurysm and dAVF simultaneously. Seven patients (11%) were diagnosed with an aneurysm first and subsequently developed de novo dAVFs during the follow-up period. Of these 7 patients, 5 had adequate angiography (i.e., all vessels imaged) at the time of aneurysm diagnosis, suggesting that the dAVFs developed de

TABLE 3. Presentation and angioarchitecture details for patients with dAVF only versus those with dAVF and aneurysm

	No. of Patients/Total No. of Patients w/ Data (%)			No. of Patients/Total No. of Patients w/ Data (%)		
	dAVF Only (n = 978)	dAVF+ (n = 65)	p Value	FRA (n = 10)	dAVF & Unrelated (n = 1033)	p Value
Symptomatic on presentation from dAVF	811/977 (83.0)	23/65 (35.4)	**<0.001**	5/10 (50)	829/1032 (80.3)	**0.035**
Ruptured dAVF at presentation	242/974 (24.8)	12/65 (18.5)	0.297	4/10 (40)	250/1029 (24.3)	0.27
Location*						
Transverse-sigmoid junction	355/970 (36.6)	19/64 (29.7)	0.286	3/10 (30)	373/1024 (36.4)	>0.99
Convexity/SSS	95/970 (9.8)	15/64 (23.4)	**0.002**	2/10 (20)	108/1024 (10.5)	0.289
Tentorial	146/970 (15.1)	10/64 (15.6)	0.858	2/10 (20)	154/1024 (15.0)	0.653
Cavernous sinus	115/970 (11.9)	2/64 (3.1)††	**0.038**	1/10 (10)	108/1024 (10.5)	>0.99
Petrosal	33/970 (3.4)	5/64 (7.8)	0.08	1/10 (10)	37/1024 (3.6)	0.313
Anterior cranial fossa	51/970 (5.3)	7/64 (10.9)	0.083	1/10 (10)	57/1024 (5.6)	0.44
Arterial supply to dAVF†						
Occipital artery	553/957 (57.8)	22/62 (35.5)	**0.001**	4/9 (44.4)	571/1010 (56.5)	0.514
Other ECA‡	364/958 (38.0)	13/63 (20.6)	**0.007**	3/10 (10)	374/1011 (37.0)	0.753
Small ICA§	366/959 (38.2)	17/63 (27.0)	0.082	2/10 (20)	381/1012 (37.6)	0.336
Pial artery¶	188/957 (19.6)	20/64 (31.2)	**0.036**	7/10 (70)	201/1011 (19.9)	**0.001**
Venous drainage of dAVF**						
Transverse-sigmoid junction	415/957 (43.4)	18/65 (27.7)	**0.014**	2 (20)	431/1012 (42.6)	0.204
CVD	627/967 (64.8)	42/65 (64.6)	>0.99	7/10 (70)	662/1023 (64.7)	>0.99
Venous ectasia	321/927 (34.6)	13/60 (21.7)	**0.048**	2/10 (20)	332/977 (34.0)	0.509
Borden type			0.411			0.349
I	327/966 (33.9)	23/65 (35.4)		3/10 (30)	347/1021 (34)	
II	167/966 (17.3)	4/65 (6.2)		0	171 (16.7)	
III	472/966 (48.9)	38/65 (58.5)		7/10 (70)	503 (49.3)	

SSS = superior sagittal sinus.
Boldface type indicates statistical significance; p values do not adjust for multiple comparisons.
* Only the 3 most common locations and those with near significance ($p < 0.1$) are included in the table. Eleven locations were analyzed in total.
† Nine arterial pedicles analyzed in total.
‡ Includes any branches of the ECA other than middle meningeal, occipital, and ascending pharyngeal arteries.
§ Includes inferolateral trunk, meningohypophyseal trunk, small meningeal branches, and marginal tentorial branch.
¶ Includes branches from the anterior, middle, and posterior cerebral arteries, and the superior, anterior inferior, and posterior inferior cerebellar arteries.
** Only categories with statistical significance ($p < 0.05$) are included in table. Six venous sinuses were analyzed in total.
†† Both patients with associated aneurysm were indirect fistulas.

novo. For these 5 patients, the average time to dAVF diagnosis was 34.3 ± 27 months. No patient developed de novo aneurysms. A significant proportion (40%) of patients in the dAVF+ cohort had aneurysm(s) ipsilateral to the side of the dAVF.

Most patients presented with an unruptured aneurysm (80%), and 17% had a ruptured aneurysm at presentation, while 1% experienced rupture during follow-up (rupture status was missing in 2% of patients). Only 1 AICA and 1 PICA (2/16 = 12.5%) in the ruptured aneurysm cohort were FRAs, similar to the representation of FRAs within the unruptured aneurysm cohort (10/78 = 12.8%).

Aneurysms were managed conservatively in 44.8%, treated with endovascular modalities in 36.5%, surgically in 15.6%, and with both surgery and endovascular techniques in 1.0% of the cases. Of the treated aneurysms (51), 68.6% remained stable and 13.7% had recurrence or re-treatments at the time of final follow-up. Of the untreated aneurysms (43), most (44.2%) had no follow-up or were stable during follow-up (37.2%). One patient (1.5%) had a fusiform basilar trunk aneurysm that ruptured during follow-up. Six (6.3%) of the aneurysms (in 4 patients) resolved spontaneously; 4 of these were FRAs that resolved after dAVF treatment, 1 was an aneurysm associated with a brain tumor feeding artery and resolved after removal of the tumor, and 1 was a small (2 mm) NFRA aneurysm that also resolved after dAVF treatment. Figure 2 illustrates a case example of an FRA that resolved after dAVF treatment.

Discussion

It is now well recognized that patients with intracranial dAVFs can also harbor a coexisting underlying intracranial aneurysm. Cagnazzo et al. recently published a systematic review of all intracranial aneurysms identified in patients with dAVF,[7] pooling data from 26 studies (3 case series[5,8,9] and 23 case reports[6,10–31]), which included 43 patients with 62 aneurysms, with 23 (37.1%) being FRAs.

TABLE 4. Univariate and multivariate predictors of associated aneurysms in dAVF patients using forward stepwise logistic regression starting with the set of best univariate predictors

	Multiple-Imputation Analysis				Complete-Case Analysis			
	Univariate		Multivariate Forward Selection		Univariate		Multivariate Forward Selection	
Variable	OR	p Value	OR	p Value	OR	p Value	OR	p Value
Smoking		**0.002**		0.003		**0.001**		**0.002**
Past (vs never)	1.91	0.087	2.05	0.064	2.18	**0.018**	2.20	**0.018**
Current (vs never)	3.43	**0.002**	3.20	**0.004**	3.79	**<0.001**	3.47	**0.001**
Illicit drugs		0.095		0.321	—	0.078		0.393
Nonstimulants (vs no)	2.51	0.178			4.51	**0.024**		
Stimulants (vs no)	1.23	0.763			0.00	>0.99		
Anterior cranial fossa location	2.22	0.060		0.072	2.21	0.062		0.222
Convexity/SSS location	2.78	**0.001**	2.68	**0.002**	2.82	**0.001**	2.99	**0.001**
Cavernous location	0.24	0.048		0.185	0.24	**0.049**		0.202
Petrosal location	2.33	0.090		0.102	2.41	0.078		0.183
Pial artery feeders	1.83	**0.033**	1.97	**0.021**	1.86	**0.028**		0.264
Venous ectasia	0.54	0.057	0.47	**0.022**	0.52	**0.043**		0.051
Cox-Snell R^2			3.1%				2.8%	

Boldface type indicates statistical significance.

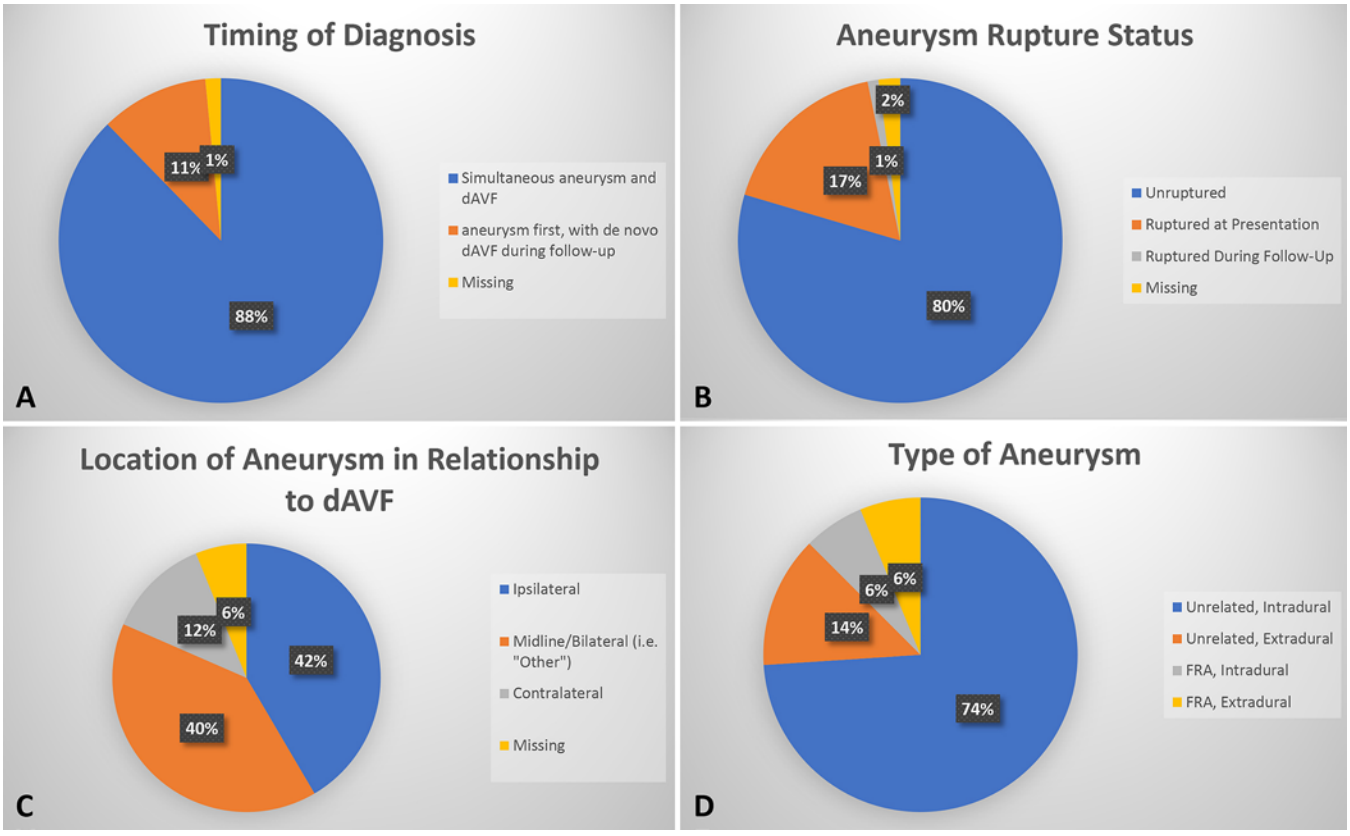

FIG. 1. Characteristics of aneurysms found in patients with dAVFs. **A:** Order of diagnoses for the aneurysm and dAVF (for 65 patients in dAVF+ cohort). Note that there were no cases of de novo aneurysm development. **B:** Rupture status of aneurysms (includes all 96 aneurysms). **C:** Location of aneurysm(s) in relationship to the location of the dAVF (for 65 patients). **D:** Categorization of aneurysms (for all 96 aneurysms). Figure is available in color online only.

TABLE 5. Overview of patients with a dAVF and aneurysm

Category	Value
No. of patients (%; n = 65)	
No. of aneurysms	
1	48 (73.8)
2	9 (13.8)
3	3 (4.6)
4	1 (1.5)
5	2 (3.1)
6	1 (1.5)
Multiple	1 (1.5)
No. of aneurysms (%; n = 96)	
Maximum aneurysm dimension, mm	
0–3.9	33 (34.4)
4–6.9	22 (22.9)
7–12.9	25 (26.0)
13–24.9	5 (5.2)
≥25	1 (1.0)
Missing	10 (10.4)
Morphology	
Saccular	75 (78.1)
Fusiform &/or dissecting	14 (14.6)
Missing	7 (7.3)
Treatment	
None	43 (44.8)
Endovascular	35 (36.5)
Surgery	15 (15.6)
Both	1 (1.0)
Missing	2 (2.1)
Follow-up	
Treated	
Stable	35 (36.5)
No follow-up	9 (9.4)
Recurrence/progression of residual/re-treatment	7 (7.3)
Untreated	
No follow-up	19 (19.8)
Stable	16 (16.7)
Progression/rupture	2 (2.1)
Spontaneous regression	6 (6.2)
Missing	2 (2.1)

The included patients derived from an international cohort, including patients from the United States, Japan, China, Italy, Germany, Canada, and beyond. Interestingly, only 1 patient was from a CONDOR contributing center.[20] However, the included patient reported was not an adult and thus was not included in the current CONDOR database. Therefore, we present an entirely new large database that is distinct from this recent review, representing a different perspective that includes a "control group" with dAVF patients without concomitant aneurysms for comparisons of features and characteristics.

Importantly, the risk of patients with intracranial dAVFs harboring a concomitant NFRA intracranial aneurysm appears similar (5.3%) when compared with the general population (quoted rates range from 1% to 5%[32,33]). A small subset of dAVF+ (1%) do harbor FRAs, half of which are intradural. Overall, these rates sum to 6.3%, which is lower than the previously reported rate of 13%[8] or 21%[5] in dAVF patients. Dural AVF patients with and without concomitant aneurysms shared similar risk factors.[34,35] The prevalence of aneurysm in dAVF patients appears to be quite different from that of associated aneurysm in AVM patients, which has been estimated to be 20.2%.[36] This likely relates to higher rates of intracranial arterial feeders in AVMs compared with dAVFs. The proportion of dAVF+ patients presenting with aneurysmal hemorrhage is different from that of aneurysm rupture in patients with AVM; 17% of dAVF+ presented with aneurysm rupture, 18.5% of dAVF+ presented with fistula rupture, and 24.8% of the dAVF-only cohort presented with fistula rupture, while 64% of patients with AVM-associated aneurysms presented with rupture (compared with 50% without aneurysm), 49% of which (approximately 32% total) were related to the aneurysm.[36] Thus, while the presence of aneurysm in dAVF patients does increase the overall risk that the patient will present with hemorrhage of any kind, the overall rate of hemorrhage at presentation is much lower than that of AVM-associated aneurysms.

Contrary to prior reports, angioarchitectural features of the dAVF+ cohort were distinctly different from those of the dAVF cohort. The transverse-sigmoid junction was the most common dAVF location for both cohorts. However, there was a higher proportion of fistulas located along the convexity or near the superior sagittal sinus and a lower proportion of CCFs in the dAVF+ cohort. Convexity/superior sagittal sinus location emerged as an independent predictor of coexisting aneurysms. While typical arterial contributors to dAVF (ECA and dural ICA branches) were more commonly identified in the dAVF-only cohort, pial intracranial feeders were more common in the dAVF+ cohort. There did not seem to be differences in venous drainage between the two cohorts, except that the dAVF-only cohort more commonly had transverse-sigmoid junction venous drainage (consistent with this cohort having a more typical fistula location/appearance). Finally, while rates of CVD and Borden grade distributions were similar between the two groups, it is interesting to note that dAVF+ patients less often had venous ectasia, which held true after adjusting for other covariates. This warrants further histological and microbiological analyses, as there may be different underlying endothelial dysfunction in the arterial and venous systems that contributes to preferential aneurysm formation.

FRAs

Only 12 (12.5%) of 96 aneurysms in the study were FRAs, which was substantially lower than the rate of 71.4% reported in AVM-associated aneurysms.[36] The FRAs in this study can be categorized into those that are located on 1) meningeal feeding arteries (e.g., the inferolateral trunk, posterior meningeal arteries), 2) pial feeding arteries that otherwise supply parenchyma (such as the ophthalmic segment of the ICA when the ophthalmic ar-

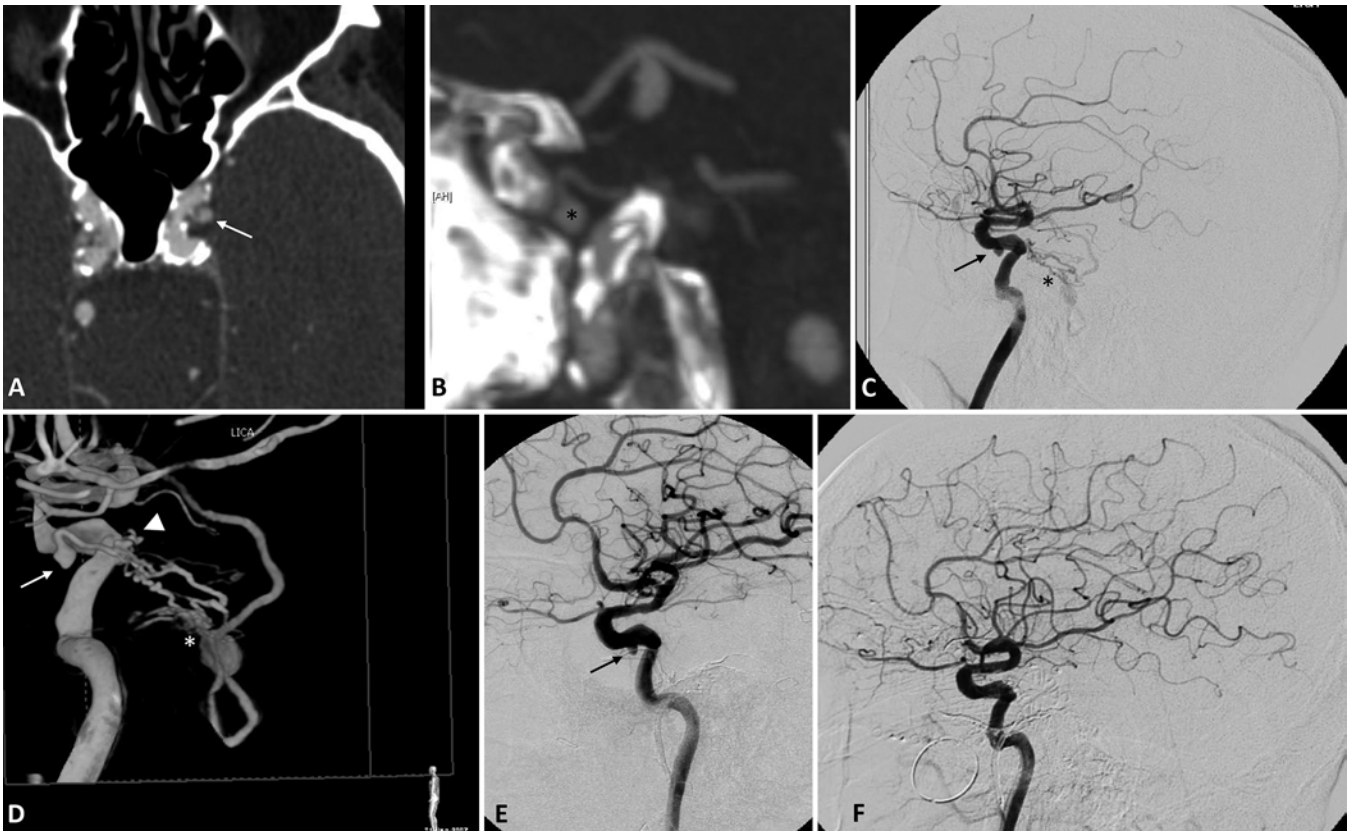

FIG. 2. Example of spontaneous regression of FRA in a treated ruptured Borden type III tentorial dAVF. **A and B:** Initial axial (A) and sagittal (B) CT angiograms showing an aneurysm (*arrow* in A, *asterisk* in B) on a branch vessel of left cavernous. **C:** Cerebral angiography, lateral view, left ICA injection, confirming aneurysm (*arrow*) as well as tentorial dAVF (*asterisk*). **D:** Three-dimensional reconstructed angiogram showing meningohypophyseal trunk (*arrowhead*) and inferolateral trunk arterial feeders to tentorial dAVF (*asterisk*), the inferolateral trunk harboring an FRA (*arrow*). **E:** Posttreatment intraoperative angiogram (which included 3 sequential stages of transarterial Onyx embolization to the middle meningeal artery, meningohypophyseal trunk, and occipital arteries, with a final craniotomy for clip ligation), showing resolution of previous dAVF but persistent sluggish filling of the FRA. **F:** Follow-up angiography at 9 months confirms obliteration of dAVF with subsequent regression of the FRA.

tery supplies the dAVF), or 3) other intracranial branches not supplying dura or brain (such as the intraorbital ophthalmic artery). Accordingly, FRAs can be grouped into intra- and extradural locations, with 50% in each category in this series. When triaging FRAs, one must acknowledge the less aggressive natural history associated with extradural aneurysms, as they do not carry a risk of subarachnoid hemorrhage. Importantly, FRAs had a similar rate of rupture at presentation compared to NFRAs (2 [16.7%] of 12 FRAs were ruptured, 15 [17.9%] of 84 NFRAs were ruptured or ruptured during follow-up).

Regression

Similar to distal FRAs in the AVM literature,[37] we found that 4 FRAs spontaneously regressed after treatment of the dAVF, 1 after treatment of a brain tumor (this aneurysm was associated with arterial supply to tumor, not dAVF), and 1 resolved spontaneously. This likely stems from a similar mechanism; after the underlying pathology (i.e., arteriovenous shunt) is eliminated, previously prominent arterial feeders likely have a reduction in blood flow, promoting stasis and eventually thrombosis within the aneurysm.[38] Figure 2 shows serial angiography for an example of an FRA that spontaneously regressed 9 months after dAVF treatment.

Limitations

This study, despite representing the largest of its type, has limitations. The retrospective design implies that there may be potential confounding bias. For example, it is not clear why venous ectasia seems to predict dAVF over dAVF+, even in multivariable modeling. This may be due to an intimate relationship between venous ectasia and another variable. Second, the data were collected by multiple people at multiple centers. The architects of CONDOR did include a validation process for all chart reviewers prior to data collection to decrease the potential spectrum of variability, but this remains a concern. Third, follow-up varied significantly from patient to patient and from institution to institution. While the medical records of all patients with dAVF and aneurysm were investigated more thoroughly after identification, this analysis was performed retrospectively and not in a prospective longitudinal fashion with specific attention to aneurysm or dAVF development.

Finally, while we suppose that most patients here had simultaneous diagnosis of dAVF and aneurysm (based on retrospective chart analysis and available angiographic data), we are limited in the imaging performed before presentation, so the precise timing of dAVF and/or aneurysm development is not totally clear. Future studies might aim to follow a group of patients with either intracranial aneurysm or dAVF (not both) prospectively, to delineate the precise rates and timing of de novo lesion development.

Conclusions

The prevalence of intracranial aneurysms in dAVF patients is 6.2%, most of which are unrelated to the dAVF. A minority of concomitant aneurysms are located on arteries supplying the dAVF (defined as FRAs). Aneurysms in dAVF patients seem to rupture at lower rates than those associated with AVMs. Compared with dAVF patients without aneurysm, those with dAVFs with aneurysms harbor unique angioarchitecture features. Spontaneous regression of aneurysms has been observed with dAVF treatment.

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Appendix

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Supplemental Information

Companion Papers

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