JNS

CLINICAL ARTICLE

Dissecting aneurysm in poor-grade subarachnoid hemorrhage

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OBJECTIVE Intracranial dissecting aneurysms (DAs) are rare and challenging lesions, often associated with high rates of rebleeding and poor clinical outcomes. There is limited evidence regarding optimal treatment strategies, timing, and outcomes, especially in the context of poor-grade subarachnoid hemorrhage (pSAH). The authors aimed to describe the

ABBREVIATIONS aOR = adjusted OR; aSAH = acute subarachnoid hemorrhage; DA = dissecting aneurysm; EVD = external ventricular drainage; FD = flow diverter; ICA = internal carotid artery; ICH = intracerebral hemorrhage; IVH = intraventricular hemorrhage; mRS = modified Rankin Scale; pSAH = poor-grade subarachnoid hemorrhage; SA = saccular aneurysm; SAH = subarachnoid hemorrhage; WFNS = World Federation of Neurosurgical Societies.

SUBMITTED March 30, 2025. **ACCEPTED** June 25, 2025.
INCLUDE WHEN CITING Published online November 7, 2025; DOI: 10.3171/2025.6.JNS25834.

clinical features and treatment outcomes of patients with DAs included in a national multicentric registry of pSAH and to identify independent outcome predictors within this subpopulation.

METHODS The authors conducted a retrospective analysis of prospectively collected data from the multicenter Poor-Grade Aneurysmal Subarachnoid Hemorrhage (POGASH) registry, including consecutive patients admitted between January 1, 2015, and June 30, 2024. Poor grade was defined as a pretreatment World Federation of Neurosurgical Societies grade IV–V. Outcomes were assessed using the modified Rankin Scale. DAs were classified according to the Mizutani classification.

RESULTS Of the 693 consecutive pSAH patients included in the registry, data from 60 patients with DA were analyzed. Among the 54 treated patients, 88.9% underwent endovascular treatment (vessel occlusion [48%], flow diversion [26%], and coiling [26%]), while 11.1% were treated surgically. The median (IQR) time to treatment was 6 (4–9) hours from symptom onset. Rebleeding occurred in 23.3% of patients, significantly more frequently than in the overall cohort ($p < 0.048$). Rebleeding independently predicted in-hospital mortality (adjusted OR 7.4; 95% CI 1.5–35.1; $p = 0.011$) and long-term disability (adjusted OR 0.08; 95% CI 0.007–0.98; $p = 0.04$). Internal carotid artery blister aneurysms were independently associated with rebleeding (adjusted OR 8; 95% CI 1.2–50; $p = 0.027$).

CONCLUSIONS In the context of pSAH, DAs are characterized by distinct clinicoradiological features and carry a significant risk of ultra-early rebleeding, which strongly influences clinical outcome. These findings suggest a potential benefit of ultra-early or immediate treatment in this patient population, pending further validation.

https://thejns.org/doi/abs/10.3171/2025.6.JNS25834

KEYWORDS dissecting aneurysm; rebleeding; poor-grade subarachnoid hemorrhage; cerebral aneurysm; interventional neurosurgery; endovascular neurosurgery; vascular disorders

Intracranial dissecting aneurysms (DAs) are a rare and challenging subset of ruptured intracranial aneurysms, resulting from a sudden and often extensive disruption of the internal elastic lamina and media.[1] Blood blister aneurysms—Mizutani type IV—share a similar pathogenesis: they lack a well-defined neck and have fragile, thin walls, suggesting damage due to shear forces or wall stress.[2] Considering the distinctive nature of DAs, both surgical and endovascular approaches primarily focus on either repairing the injured vessel or occluding it.[2–7] Endovascular reconstructive techniques, particularly flow diverters (FDs), are commonly employed to reconstruct the parent vessel.[8–11] However, the requirement for antiplatelet therapy poses significant management challenges in patients with subarachnoid hemorrhage (SAH), especially in those with poor-grade presentations (pSAH), where additional interventions such as external ventricular drainage (EVD), decompressive craniectomy, or hematoma evacuation are frequently required. Moreover, both pSAH and DAs have been repeatedly linked to a heightened rebleeding risk.[12–14] Although current guidelines emphasize early aneurysm treatment to mitigate rebleeding, they lack specific recommendations on optimal treatment modality and timing for DAs.[15]

Data on DAs in the pSAH context are particularly scarce.[12] Therefore, the objective of this study was to evaluate the clinical and radiological characteristics, treatment timings, strategies, and outcomes of ruptured DAs in a large, multicenter national registry of poor-grade acute subarachnoid hemorrhage (aSAH) patients.

Methods

Standard Protocol Approvals, Registrations, and Patient Consent

The study was approved by the local ethics committees and included in the POGASH (Poor-Grade Aneurysmal Subarachnoid Hemorrhage) registry.

Study Population and Variable Description

POGASH (NCT04945603) is an ongoing prospective multicentric registry pooling anonymized data from prospectively collected and maintained institutional databases at nine academic institutions/tertiary referral centers. All poor-grade aSAH patients included in the present analysis were consecutively admitted from January 1, 2015, to June 30, 2024, to provide at least 6 months of follow-up for patients surviving the acute phase of hospitalization. Patients were included irrespective of whether they received treatment or abstention was chosen. American Heart Association guidelines for the management of aSAH were applied.[12]

Inclusion criteria were as follows: patients admitted to the emergency department because of aSAH considered poor grade (IV–V) according to the World Federation of Neurosurgical Societies (WFNS) classification. Exclusion criteria were as follows: patients < 18 years old and aSAH due to trauma or vascular malformations other than cerebral aneurysms.

The studied variables were grouped according to the different phases of the clinical course of the disease: early brain injury, intensive care unit, hospitalization, follow-up. Poor grade was defined according to the WFNS classification[16] considering the worst pretreatment clinical grade, i.e., at nadir.[17] Global cerebral edema was defined as extensive sulcal effacement and basal cisterns and bilateral gray-white matter junction disruption at the centrum semiovale.[18] Loss of consciousness was defined on the basis of > 60 minutes in duration.[19] Baseline CT was evaluated in a core laboratory imaging setting in the promoting center by two experienced neuroradiologists who considered presence/absence or grading of acute hydrocephalus, acute vasospasm, modified Fisher grade,[20] acute subdural hematoma, intraventricular hemorrhage (IVH), intracerebral hemorrhage (ICH), and global cerebral edema. DAs were defined as aneurysms arising from the arterial trunk

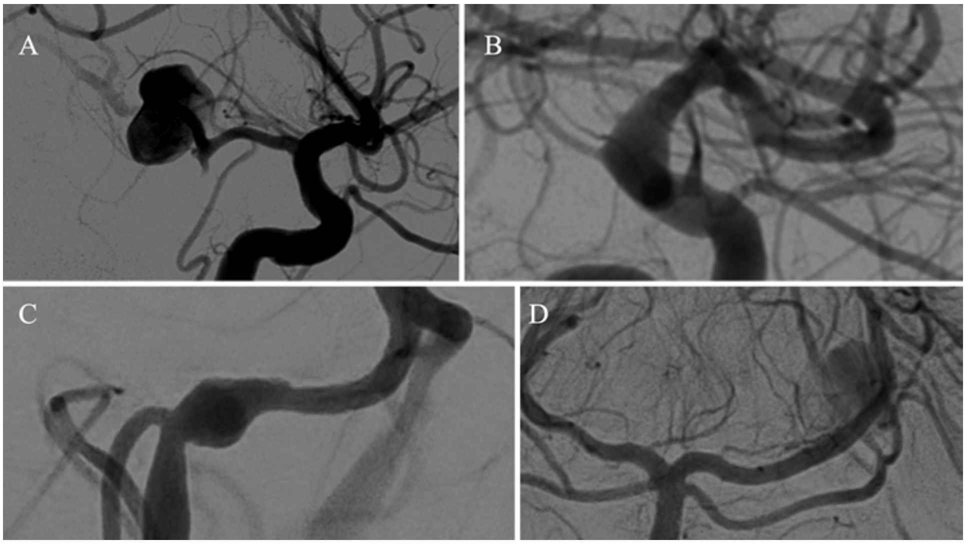

FIG. 1. Examples of ruptured DA according to the Mizutani classification.[21] **A and D:** Type I DA of the posterior cerebral artery with delayed contrast filling of the pseudoaneurysm sac. **B:** Type IV ("blood blister–like") DA arising from the superior wall of the internal carotid artery. **C:** Type I DA involving the post–posterior inferior communicating artery cisternal segment of the vertebral artery.

unrelated to branching zones, with irregular morphology or saccular type unrelated to branching zones (Mizutani types I and IV) (Fig. 1).[21] Time-related variables (time to EVD placement and time to aneurysm treatment) were derived considering SAH onset as the time of ambulance call. A rebleeding episode was defined if at least one of the following conditions was met: 1) new clinical deterioration with signs of increased hemorrhage on consecutive CT scans, or 2) clinical deterioration with fresh blood in the EVD (direct evidence of rebleeding)

Delayed cerebral infarction was defined according to a previously reported definition.[22] The clinical outcome was scored according to the modified Rankin Scale (mRS), as assessed by qualified personnel in each center at discharge and at last follow-up.

Statistical Analysis

Quantitative variables are reported as mean ± SD and categorical variables are expressed as number (%). First, a descriptive analysis of the global population was performed in order to detect clinical and radiological features associated with DAs. Bivariate comparisons between subgroups defined according to presence/absence of DAs were performed using the chi-square test, or Fisher exact test when appropriate, for categorical variables or the Mann-Whitney U-test for quantitative variables. Then, we aimed to ascertain independent predictors of rebleeding, in-hospital mortality, and long-term disability in the population harboring DAs. To do so, bivariate comparisons between subgroups defined according to the 3 prespecified outcomes (rebleeding, mortality, long-term disability) were performed, as previously stated. Variables emerging from bivariate comparison ($p < 0.05$) were then entered into a binary logistic regression model (one for each of the 3 predetermined outcomes). The results of multivariable modeling were expressed as p values, ORs, and 95% CIs after adjustment for covariates and admitting center, to account for potential center-specific treatment biases. The Hosmer-Lemeshow test was used to assess the goodness-of-fit of each model. All statistical analyses were conducted using IBM SPSS version 30.0 and replicated in R statistical environment version 4.1.3.

Results

DA Population Characteristics

A total of 693 consecutive poor-grade aSAH patients were prospectively included in the registry between January 1, 2015, and June 30, 2024. Among them, 60 patients (8.65%) harbored DAs. The results of bivariate comparisons between the DA population and the one affected by saccular aneurysms (SAs) are reported in Table 1. DA patients were more frequently admitted with WFNS IV–V ($p < 0.045$) and more likely to have IVH ($p = 0.002$) and increased IVH volume ($p = 0.010$). Conversely, ICH occurred more frequently in the SA group ($p = 0.008$).

DAs were more commonly located in the posterior circulation (63.3%, $p < 0.001$) and were associated with higher rebleeding rates ($p = 0.048$), despite a comparable ultra-early treatment approach between groups (median [IQR] time to treatment 6 [4–9] hours, $p = 0.323$). DAs were also more frequently treated endovascularly ($p < 0.001$) and had a higher overall complication rate ($p = 0.002$). Six of the 60 DA patients (10%) did not undergo any treatment due to either absent or minimal intracranial contrast enhancement on CT angiography and/or diffuse acute hypodensities on baseline CT, consistent with early brain ischemia or fatal rebleeding.

No significant differences were observed between the DA and SA groups in terms of in-hospital mortality, long-term disability, or follow-up duration (all $p > 0.05$).

DA Treatment Characteristics

Deconstructive treatment (vessel occlusion) was per-

TABLE 1. Bivariate comparison of factors associated with the presence of DA in the studied population (n = 693 patients)

	DA	SA/Nondissecting Aneurysm	p Value
Baseline & demographic			
Age, yrs	57 (51–66)	61 (51–69)	
Male/female sex	33/27 (55/45)	411/222 (64.9/35.1)	0.083
Hypertension	25/31 (44.6/55.4)	291/267 (52.2/47.8)	0.176
Smoking	9/44 (17/83)	160/380 (29.6/70.4)	0.017
Early brain injury phase			
WFNS grade V/IV	46/14 (76.7/23.3)	403/216 (65.1/34.9)	0.045
GHV, ml	89 (54–126)	89 (59–126)	0.956
ICHV, ml	14 (3–28)	1 (0.5–38)	0.061
IVHV, ml	6 (2–47)	2 (0.5–25)	0.010
SAHV, ml	66 (38–126)	82 (45–132)	0.253
Cardiac arrest	7/52 (11.9/88.1)	44/538 (7.6/92.4)	0.307
Loss of consciousness	48/11 (81.4/18.6)	443/153 (74.3/25.7)	0.151
GCE (SEBES 3/4)	23/31 (42.6/57.4)	193/331 (36.8/63.2)	0.245
Modified Fisher grade 4/3	50/4 (92.6/7.4)	424/78 (84.5/15.5)	0.260
IVH	46/8 (85.2/14.8)	341/178 (65.7/34.3)	0.002
ICH	14/40 (25.9/74.1)	227/292 (43.7/56.3)	0.008
aSDH	5/48 (9.4/90.6)	72/439 (14.1/85.9)	0.239
Hydrocephalus	45/11 (80.4/19.6)	375/137 (73.2/26.8)	0.161
Acute spasm	9/51 (15/85)	63/522 (10.8/89.2)	0.213
Time to EVD, hrs	4 (3–6)	4 (3–7)	0.249
Time to treatment, hrs	6 (4–9)	5 (3–10)	0.323
Aneurysm location (posterior circulation)	38/22 (63.3/36.7)	70/538 (11.5/88.5)	<0.001
Size (largest diameter), mm	7 (4–14)	7 (5–10)	0.898
Surgical/endovascular treatment	6/48 (13.8/77.6)	177/371 (31.7/66.4)	<0.001
Complications	24/36 (40/60)	155/478 (24.5/75.5)	0.002
Rebleeding	14/46 (23.3/76.7)	87/528 (14.1/85.9)	0.048
Location			
PCom	2/58 (3.3/96.7)	57/576 (9/91)	0.095
ACA	2/58 (3.3/96.7)	40/591 (6.3/93.7)	0.569
ACoA	3/57 (5/95)	205/429 (32.2/67.8)	<0.001
MCA	6/53 (10.2/89.8)	143/486 (22.7/77.3)	0.014
ICA	8/52 (13.3/86.7)	78/553 (12.4/87.6)	0.478
AChA	2/58 (3.3/96.7)	3/630 (0.5/99.5)	0.062
BA	2/58 (3.3/96.7)	25/608 (3.9/96.1)	>0.99
PCA	7/53 (11.7/88.3)	6/627 (0.9/99.1)	<0.001
PICA	12/48 (20/80)	10/622 (1.6/98.4)	<0.001
SCA	0/60 (0/100)	1/631 (0.2/99.8)	>0.99
VA	8/52 (13.3/86.7)	4/629 (0.6/99.4)	<0.001
NeuroICU			
Ventilation length, days	9 (3–19)	9 (3–17)	0.327
Tracheostomy	25/33 (43.1/56.9)	268/336 (44.4/55.6)	0.483
EVD	50/9 (84.7/15.3)	434/176 (71.1/28.9)	0.019
Craniectomy	11/46 (19.3/80.7)	150/461 (24.5/75.5)	0.238
Stunned myocardium	5/52 (8.8/91.2)	33/568 (5.5/94.5)	0.364
Cerebral infarction	13/45 (22.4/77.6)	139/4565 (23.4/76.6)	0.510
Delayed brain injury phase & outcomes			
In-hospital mortality	26/34 (40/60)	201/403 (33.3/66.7)	0.182

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TABLE 1. Bivariate comparison of factors associated with the presence of DA in the studied population (n = 693 patients)

	DA	SA/Nondissecting Aneurysm	p Value
Delayed brain injury phase & outcomes (*continued*)			
FU length, mos	10 (1–42)	7 (1–30)	0.436
mRS score at discharge	5 (4–6)	5 (3–6)	0.158
mRS at FU	3 (1–6)	4 (1–6)	0.804
mRS score 0–2 vs 3–6 at long-term FU	19/41 (31.7/68.3)	175/458 (27.6/72.4)	0.300

ACA = anterior cerebral artery; AChA = anterior choroidal artery; ACoA = anterior communicating artery; aSDH = acute subdural hematoma; BA = basilar artery; FU = follow-up; GCE = global cerebral edema; GHV = global intracranial hemorrhage volume; ICHV = intracranial hemorrhage volume; IVHV = intraventricular hemorrhage volume; MCA = middle cerebral artery; PCA = posterior communicating artery; PCom = posterior communicating artery; PICA = posterior inferior communicating artery; SAHV = subarachnoid hemorrhage volume; SCA = superior cerebellar artery; SEBES = Subarachnoid Hemorrhage Early Brain Edema Score; VA = vertebral artery.
Values are shown as number (%) or median (IQR).

formed in 22/54 cases (40.7%). In 16/22 patients (72.7%), treatment involved the posterior circulation with vertebral artery and posterior communicating artery as the most frequent locations (5 cases each [22.7%]). Platinum coils were more frequently used (16/22 [72.7%]) followed by *n*-butyl cyanoacrylate (NBCA) in the remaining 6 (27.3%). The reconstructive technique (flow diversion) was performed in 12/54 (22.2%) and coiling in 12/54 (22.2%). Stent-assisted coiling was performed in 2 cases (3.7%). Six cases (11.1%) were surgically treated (aneurysm trapping with or without bypass).

In FD cases, 9/12 (75%) received aspirin (500 mg IV) plus heparin (most frequently 5000 IU) and 3/12 (25%) received tirofiban plus heparin. Survivors continued on aspirin plus clopidogrel.

Detailed treatment characteristics, complication rates, rebleeding rates, and unadjusted ORs are reported in Table 2.

Overall, 20 patients (37%) experienced treatment-related complications, including 11 ischemic and 9 hemorrhagic events. Complications occurred intraprocedurally in 4 patients (20%), periprocedurally in 15 (75%), and in the delayed phase (> 30 days) in 1 case (5%). Complication rates did not differ significantly between treatment modalities (Table 2). Three patients (23%) who received double antiplatelet therapy experienced self-limiting hemorrhages along the EVD tract.

Posttreatment rebleeding occurred in 6 of 54 cases (11.1%): 3 cases in the coiling group (25%), 2 in the flow diversion group (16.6%) (Fig. 2), and 1 in the vessel occlusion group (4.5%) (Fig. 3).

Among the 22 treated with vessel occlusion, 7 (32%) experienced complications, all ischemic, with mass effect in 3 of 7 (43%). In the FD group, 4 of 12 patients (33.3%) had complications, including 3 ischemic events and 1 rebleeding.

There were no significant differences in complication rates, rebleeding, or clinical outcomes across treatment strategies. Among survivors, complete long-term occlusion was achieved in 27 of 30 endovascularly treated patients. Retreatment was necessary in 2 of 8 patients (25%) after coiling and 1 of 10 patients (10%) after stent placement. All surgically treated patients achieved stable occlusion.

Clinical Outcomes and Results of Multivariable Analyses

Rebleeding occurred in 14 of 60 patients (23.3%): in 8 cases (57%) before treatment and in the remaining 6/14 (43%) after treatment. The median (IQR) time to EVD placement was 4 (3–6) hours. Rebleeding occurred in 5 patients before EVD placement (35.7%), in 8 after EVD placement (57.1%), and in 1 patient (7.1%) 1 month after treatment due to aneurysm recurrence. Supplemental Tables 1–3 report the results of bivariate comparisons for variables associated with rebleeding, in-hospital mortality, and long-term independence. Among early baseline and early brain injury–related (first 96 hours) variables, only internal carotid artery (ICA) blister aneurysm emerged as an independent predictor of rebleeding (adjusted OR [aOR] 8, 95% CI 1.2–50; p = 0.027) (Table 3). A total of 24 patients (40%) patients died during hospitalization. Shorter time to EVD placement and higher rebleeding rates were significantly associated with mortality ($p < 0.05$). A trend toward higher mortality was observed in WFNS grade V patients, although this was not statistically significant. Patients who died also had greater ICH and IVH volumes, although differences were not significant. In multivariable analysis, rebleeding emerged as the only independent predictor of in-hospital mortality (aOR 7.4, 95% CI 1.5–35.1; p = 0.011).

At last follow-up (median [IQR] 24 [6–51] months), 19 patients (31.7%) achieved functional independence. WFNS grade V (aOR 0.09, 95% CI 0.02–0.5; p = 0.005) and rebleeding (aOR 0.09, 95% CI 0.009–0.98; p = 0.04) were independent predictors of long-term disability.

Discussion

Most recently available studies, systematic reviews, and meta-analyses on DA have largely focused on endovascular techniques, especially stenting and flow diversion,

TABLE 2. Treatment-related complications and outcomes of patients with DA

	Complications			Rebleeding*			In-Hospital Mortality			mRS Score 0–2 at FU		
	No. (%)†	OR (95% CI)	p Value	No. (%)	OR (95% CI)	p Value	No. (%)	OR (95% CI)	p Value	No. (%)	OR (95% CI)	p Value
Coiling (12/54 [22.2]‡)	1/3/1 (42)	1.3 (0.35–5.1)	0.65	3 (25)	5 (0.87–28)	0.072	4 (33.3)	0.65 (0.17–2.5)	0.5	4 (33.3)	1.2 (0.27–5.6)	0.77
Flow diversion (12/54 [22.2]‡)	1/4/0 (42)	1.7 (0.47–6.3)	0.4	2 (16.7)	2.2 (0.35–14)	0.4	4 (33.3)	0.77 (0.2–3)	0.7	5 (41.7)	1.8 (0.36–8.9)	0.48
Vessel occlusion (22/54 [40.7]‡)	2/7/0 (41)	1.09 (0.36–3.3)	0.87	1 (4.5)	0.31 (0.03–2.9)	0.3	10 (45.5)	1.3 (0.45–3.9)	0.62	7 (31.8)	0.73 (0.19–2.7)	0.64
Surgery (6/54 [11.1]‡)	0/1/0 (12.5)	0.27 (0.03–2.5)	0.25	0	NA		1 (12.5)	NA		1 (16.7)	0.44 (0.04–5.4)	0.52

NA = not assessed.
* Numbers are reported relative to posttreatment (either intra- or postprocedural) rebleeding episodes. There are no significant differences between groups (ANOVA $p > 0.05$) in terms of treatment-related time metrics (time to EVD and time to treatment), clinical severity (WFNS grades), and volume of IVHV.
† No. of intra-/peri-/postprocedural complications (% of complications) are shown.
‡ No. (%) is shown.

while providing limited data on the initial clinical severity of SAH at presentation.[14,23–25] This is a significant gap, as clinical status at presentation influences rebleeding risk, the choice of treatment, and clinical outcome, particularly in pSAH, where the need for multiple invasive procedures is more frequent. In our study, DAs in the context of pSAH showed notably high rates of ultra-early rebleeding (23.3%) and in-hospital mortality (41.4%), considerably higher than rates previously reported for lower grade or unselected aSAH populations.[13,15,26]

In a 2018 meta-analysis by Cagnazzo et al.,[8] which included 181 DA cases from 20 studies (104 of them classified as blister aneurysms), only 28 (15.4%) were in WFNS grade IV–V. In line with our findings, DA was more common in males, although male predominance was observed in the registry. The median (IQR) time to treatment was 4 (3–9.6) days, and the posttreatment rebleeding rate was 4%. Similarly, Mokin et al.[27] reported a cohort of 49 blister aneurysms, 14 of which were poor grade, with a 19% mortality rate and a 2.3% posttreatment rebleeding rate. Ten Brinck et al.[28] more recently evaluated flow diversion in ruptured aneurysms (44 patients, including 20 DA/blister and 6 poor-grade aneurysms), with a rebleeding rate of 11%. However, treatment occurred at a median of 15 days after onset, and limited data were available for pSAH patients. In contrast, our study reports a 23.3% overall rebleeding rate, higher than that of SAs, including an 11.1% (6/54) rate of posttreatment rebleeding despite ultra-early intervention (median 6 hours from onset). This supports recent findings suggesting that rebleeding risk peaks within the first few hours following rupture.[12,29–31] Importantly, rebleeding in our cohort was a major driver of mortality and poor functional outcome, significantly higher than in previous studies, and likely exacerbated by the pSAH clinical context.

Among all cases of DA, blood blister aneurysms of the ICA emerged as an independent predictor of rebleeding, with a 25% posttreatment rebleeding rate in those treated with flow diversion. This aligns with previous studies highlighting the aggressive behavior of these lesions.[32,33] For example, Egashira et al.[32] reported a 14% pretreatment rebleeding rate in ICA blisters treated with a mean delay of 3.4 ± 6.8 days. Our findings support the hypothesis that the combination of poor clinical grade and DA-specific fragility may synergistically increase the risk of rebleeding. This has direct implications for treatment timing, reinforcing the need for immediate intervention where feasible.

Additionally, our study provides new clinicoradiological data that distinguishes DA from SA in the pSAH population. The higher prevalence of WFNS grade V in the DA group was largely attributed to a greater incidence and volume of IVH, both associated with increased mortality.[34] The predominance of distal posterior circulation aneurysms (e.g., P2–P3 of the posterior cerebral artery and posterior inferior cerebral artery) may partly explain this pattern. Furthermore, rebleeding was significantly more common in DAs then in SAs (23.3% vs 14.1%), emphasizing their more aggressive clinical profile.

Treatment Modality and Outcome

Contrary to recent literature favoring reconstructive techniques, our cohort more frequently underwent decon-

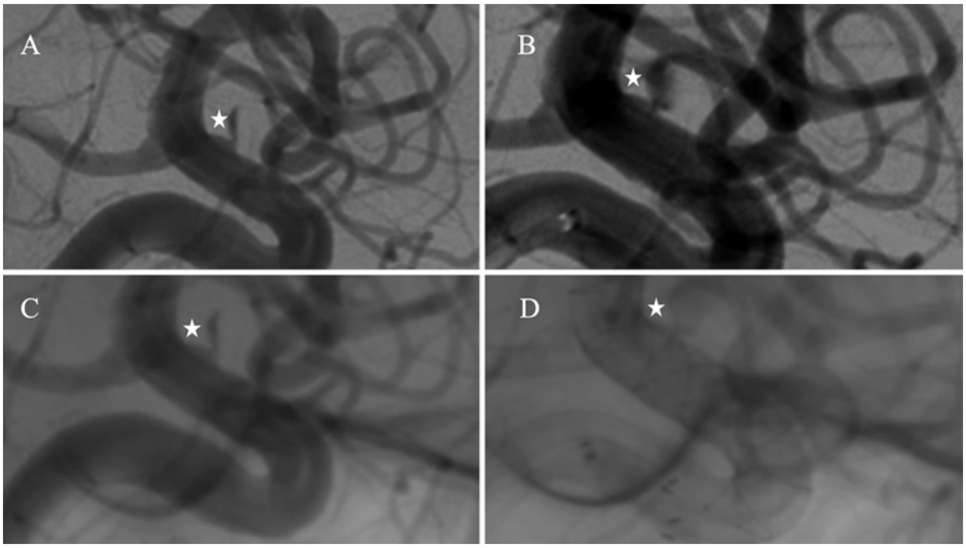

FIG. 2. Persistent intraoperative bleeding after flow diversion in ruptured ICA blister aneurysm. **A and C:** Active contrast extravasation (rebleeding, *star*) during the first DSA run (subtracted in panel A and unsubtracted in panel C). **B and D:** Persistent extravasation (*star*) after flow diversion (subtracted view panel B and unsubtracted panel D).

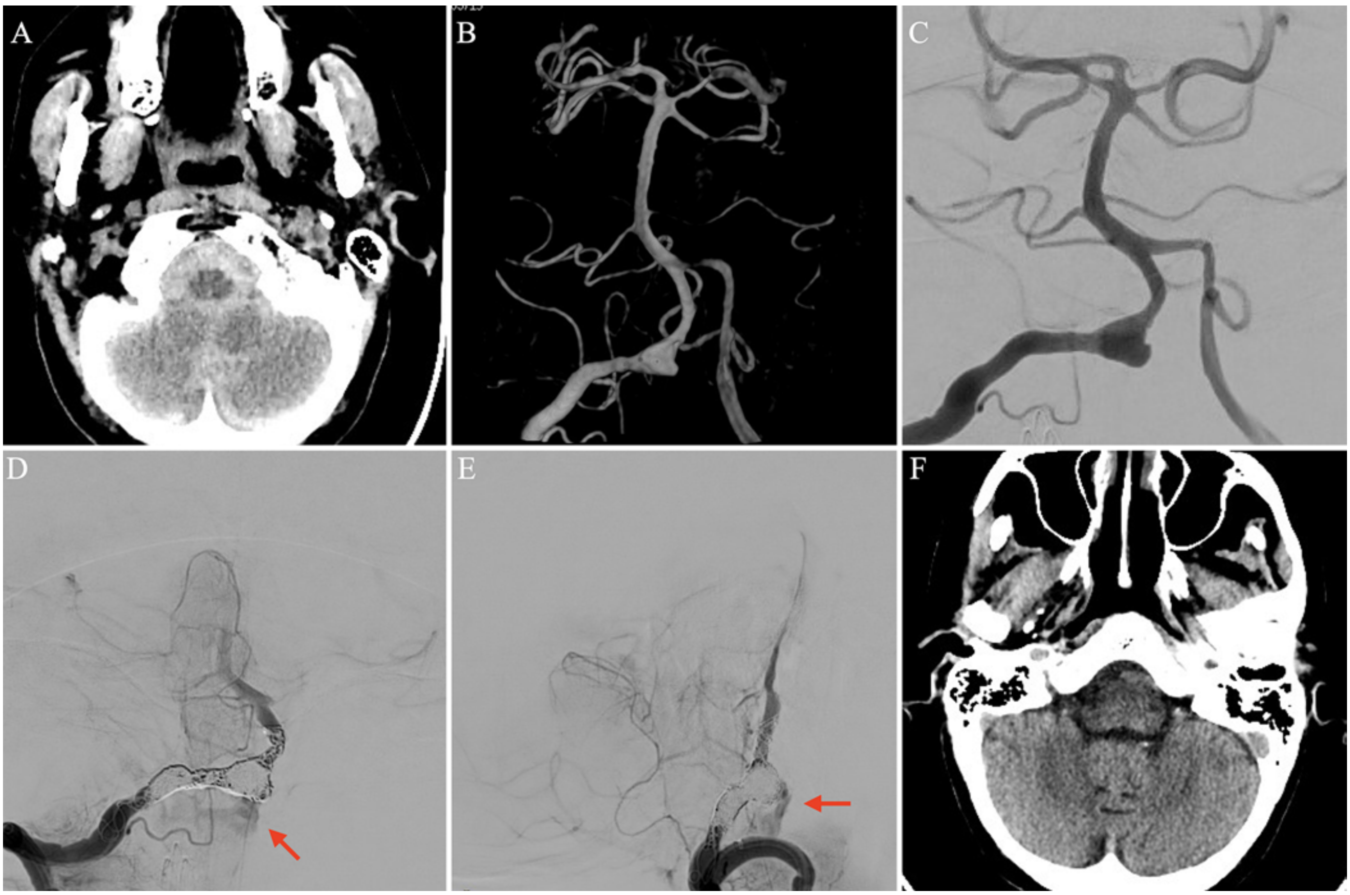

FIG. 3. A 54-year-old man presented with a sudden loss of consciousness (Glasgow Coma Scale score 4). Axial CT image (**A**) shows diffuse SAH in the posterior cranial fossa cisterns, related to DA of the V4 segment of the right vertebral artery, as demonstrated in the 3D reconstruction (**B**) and frontal (**C**) angiogram. Frontal (**D**) and lateral (**E**) angiograms demonstrated self-limited rebleeding (*red arrows*) of the aneurysm despite the occlusion of both the aneurysm and the parent vessel with platinum coils. The 3-month follow-up axial CT image (**F**) shows reabsorption of the SAH. Figure is available in color online only.

TABLE 3. Results of the multivariable analysis for independent predictors of aneurysmal rebleeding, in-hospital mortality, and long-term functional independence

	Rebleeding		In-Hospital Mortality		Long-Term Functional Independence*	
	aOR (95% CI)	p Value	aOR (95% CI)	p Value	aOR (95% CI)	p Value
Clinicoradiological features						
WFNS	NS		NS		0.09 (0.02–0.5)	**0.005**
ICA blister	8 (1.2–50)	**0.027**	NS		NS	
IVH	0.46 (0.07–5)	0.414	NS		NS	
Rebleeding	NI		7.4 (1.5–35.1)	**0.011**	0.09 (0.009–0.98)	**0.04**
Treatment-related variables						
Vessel occlusion	0.111 (0.013–0.971)	**0.047**	0.48 (0.07–3.4)	0.47	0.73 (0.19–2.7)	0.64
Coiling	2.2 (0.27–17.2)	0.46	0.13 (0.01–1.3)	0.082	1.2 (0.27–5.6)	0.77
Flow diversion	1.8 (0.22–15.2)	0.56	0.09 (0.008–1.2)	0.067	1.8 (0.36–8.9)	0.48
Surgery	NA		NA		0.44 (0.04–5.4)	0.52

NI = not included; NS = nonsignificant.
Boldface type indicates statistical significance ($p < 0.05$).
* Defined as mRS score 0–2.

structive treatment. Reasons include prevalence of posterior circulation and distal location (Fig. 1), high IVH burden, anticipated rebleeding risk under dual antiplatelet therapy,[26] and feasibility of safe parent artery occlusion.

Reconstructive techniques such as flow diversion and stent-assisted coiling (25% and 6.3%, respectively, in our cohort) were more common in anterior circulation aneurysms but showed higher than expected posttreatment rebleeding rates (16.7% in flow diversion cases), in contrast with previously reported rates of 3%–5%.[8,11] While deconstructive techniques may offer better protection against rebleeding, they were associated with increased long-term disability and in-hospital mortality, consistent with other reports,[35] though without statistical significance.

The overall complications rate was high, approaching 40% and including 33.3% of FD and 41% of deconstructive approaches. This may reflect the hypercoagulable state of acute aSAH–elevated D-dimer levels, both linked to early brain injury in pSAH,[36,37] and may partly explain the increased thromboembolic complication rate seen in flow diversion and stenting.

Notably, recent systematic reviews have reported much lower complication rates for FD (7%–18%).[8,10] In the meta-analysis by Cagnazzo et al.,[8] flow diversion in SAs had a higher complication rate (23%) than in DA (13%). In our study, however, DA cases had significantly higher complication rates than saccular ones, possibly because only 15.4% of patients in prior studies had poor-grade aneurysm. The heterogeneity in FD types and antiplatelet regimens in our registry reflects real-world practices and underscores the multicentric nature of this study.[38]

Cangrelor is a promising antiplatelet agent due to its rapid onset of action, potent and reversible platelet inhibition, and short half-life (3–6 minutes), with platelet function typically recovering within 1 hour of discontinuation. These pharmacodynamic features make it particularly well suited for the pSAH population, where the likelihood of requiring subsequent surgical interventions (e.g., EVD placement or decompressive craniectomy) is high.[39] Additionally, emerging data support the use of ticagrelor or prasugrel as maintenance monotherapy, which may offer a safer and more individualized long-term antiplatelet strategy. Finally, the development of surface-modified FDs represents a promising technological advance, with preliminary evidence suggesting a reduced risk of ischemic complications, especially in high-risk clinical scenarios.[40]

While deconstructive techniques were more effective in preventing rebleeding, they carried substantial morbidity. Labeyrie et al.[41] reported only 5% morbidity in unruptured fusiform and giant ICA aneurysms treated with parent vessel occlusion. In our ruptured cohort, 32% experienced ischemic complications after parent vessel occlusion, with mass effect in 43% of those cases. This may indicate a more abrupt pathophysiology in ruptured DAs, limiting the development of an adequate collateral flow. The current literature remains inconclusive on the superiority of specific treatment techniques,[2–4,7,23] highlighting the complexity of managing DAs in pSAH patients.

Surgical trapping with or without bypass was reserved for selected cases on the basis of technical feasibility, local expertise, and when endovascular options were unsuitable. Outcomes were generally favorable, with effective rebleeding prevention and acceptable complication rates. Interestingly, 3 of 6 surgical cases involved posterior inferior communicating artery aneurysms, while only 1 case involved clipping and wrapping of an ICA blister aneurysm.[42,43]

These findings pave the way for more questions. In the poor-grade aSAH context, are we facing more severe forms of vessel wall disruption (i.e., larger tears with an increased bleeding volume), which makes the aneurysm more prone to an increased rebleeding rate? Future studies incorporating volumetric bleeding assessment and intravascular imaging may provide valuable insight into the structural behavior of these aneurysms.

It is worth noting that growing evidence suggests a progressive improvement in the rate of functional independence among treated poor-grade patients over the recent decades,[23,44] including those with WFNS grade V aneurysm. Our findings are consistent with the recent literature, showing approximately 30% of patients achieving long-term functional independence (mRS score 0–2), with a 23% rate observed in the WFNS V subgroup. Notably, no significant differences in clinical outcomes were found between patients with DA and those with SA.

Strengths and Limitations

To date, this is the largest prospective series examining ruptured DAs in the pSAH population. The multicenter design provides a real-word overview of current ultra-early management strategies and exposes variability in practice, highlighting the urgent need for standardized treatment protocols.

However, several limitations should be acknowledged. The relatively small sample size may have led to the overestimation of certain outcomes. Additionally, despite adjusting for center in multivariable models, center-specific biases and treatment preferences cannot be entirely excluded.

Conclusions

In the poor-grade SAH population, DAs and blister aneurysms exhibit distinct clinical characteristics and challenging treatment profiles, with high rebleeding rates, significant complications, and worse clinical outcomes. Based on our findings, the prevention of rebleeding should be considered a clinical priority. If validated by additional multicenter prospective studies, our results indicate that rebleeding may represent a significant and potentially modifiable predictor of outcome. In settings where ultra-early treatment is already standard practice, immediate intervention for poor-grade patients with DAs may be justified, regardless of the time of the day, to minimize the risk of rebleeding. Notably, a meaningful proportion of pSAH patients can achieve long-term functional independence. Therefore, the presence of a DA should not be regarded a self-fulfilling prophecy of poor outcome. Our findings could help inform treatment timing and strategic decisions in managing this fragile subgroup of pSAH patients.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Supplemental Information

Online-Only Content

Supplemental material is available with the online version of the article.

Supplemental Tables 1–3. https://thejns.org/doi/suppl/10.3171/2025.6.JNS25834.

Data Availability

All data can be accessed upon reasonable request to the principal investigator, Pietro Panni, MD.

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