Neuroform Intracranial Stenting for Aneurysms Using Simple and Multi-stent Technique Is Associated With Low Risk of Magnetic Resonance Diffusion-Weighted Imaging Lesions

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BACKGROUND: Detection of procedural thromboembolism by diffusion-weighted magnetic resonance imaging (MR-DWI+) can help identify and mitigate endovascular risk factors. Data remain scant on procedural MR-DWI+ following the use of the Neuroform open-cell design stent in aneurysm embolization.

OBJECTIVE: We sought to evaluate the incidence of MR-DWI+ in Neuroform simple and multi-stent construct stenting for intracranial aneurysms in an attempt to delineate baseline risk and identify possible associated procedural factors.

METHODS: Seventy-six patients receiving 97 Neuroform stents in the treatment of intracranial aneurysm were identified from a prospective database and eligible for inclusion in the study. Diffusion-weighted magnetic resonance imaging (MR-DWI) was obtained in all patients within 48 hours of the procedure and reviewed for the presence of MR-DWI+ with patient records reviewed for analysis of factors predisposing to these lesions.

RESULTS: Patients were treated with single-stent Neuroform constructs in 57 cases (73%) and multi-stent Neuroform constructs in 21 cases (27%). Y-stent technique was used in 16 cases. MR-DWI+ was identified in 7 of 78 cases (9.0%), with MR-DWI+ in 0 of 10 subarachnoid hemorrhage cases. No MR-DWI lesions led to a permanent neurological deficit at discharge. There was no MR-DWI+ in patients treated with Y-stenting or multi-stent Neuroform constructs. The only factor associated with ipsilateral MR-DWI+ was target aneurysm location on an arterial sidewall over bifurcation ($P = .01$).

CONCLUSION: The Neuroform stent carries a very low risk of MR-DWI+ compared with its closed-cell design counterpart. Subarachnoid hemorrhage and deployment of multiple stents in the same anatomical region in configurations such as the Y-stent construct did not increase the risk of acute procedural thromboembolism.

KEY WORDS: Aneurysm, Diffusion-weighted imaging, Intracranial stent, Thromboembolism, Y-stent

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Intracranial stenting has expanded the spectrum of aneurysms that are amenable to endovascular therapy.[1] By virtue of its complexity and the need for additional catheter manipulations, stent-mediated coiling may presumably be prone to greater risk of thromboembolic complications. Recent data by Piotin et al[2] showed stent-mediated coiling to be associated with a 7.4% risk of permanent neurological procedure-related complications, nearly twice that of procedures performed without a stent.

ABBREVIATIONS: DWI, diffusion-weighted imaging; **MR-DWI,** diffusion-weighted magnetic resonance imaging; **MR-DWI+,** magnetic resonance diffusion-weighted imaging hyperintense lesion; **SAH,** subarachnoid hemorrhage

For more than a decade, intracranial stent-mediated coiling has been readily available through the open-cell design Neuroform intracranial stent.[3] In contrast to the newer, lower profile, and more maneuverable closed-cell design Enterprise stent,[4] the Neuroform stent represents a higher-profile device that requires a larger microcatheter (0.027 in vs 0.021 in) for

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delivery. The Neuroform is prone to fish-scaling around tight bends and provides a less uniform surface for scaffolding the coil mass in comparison with the Enterprise stent.[5,6] Although the Neuroform stent was initially described as difficult to maneuver, requiring exchange technique for intracranial positioning and deployment within the tortuous cerebral circulation, modifications in technique and delivery systems have mitigated some of these limitations. Recent reports have highlighted technical improvements to the stent delivery system that have simplified its clinical use, although it still requires a larger profile microcatheter.[7]

Recent reports have highlighted an increased risk of procedural diffusion-weighted magnetic resonance imaging (MR-DWI) lesions associated with poor stent apposition with the closed-cell design Enterprise stent.[8] In light of these data, our clinical practice has favored use of the Neuroform stent as an alternative for lesions around tortuous bends where the Enterprise stent would be susceptible to incomplete stent apposition and Crescent sign formation. The Neuroform has also been relied upon for the near majority of cases using Y-stent technique[9] and telescopic stenting without coiling for shallow aneurysms because of its open-cell design. The goal of the current study was to determine the thromboembolic procedural risk of Neuroform stent-mediated aneurysm embolization evaluated by occurrence of MR-DWI lesions with a focus on the role of complex, multi-stent constructs such as Y-stent and dual-telescopic stent constructs in perforator-rich territories such as the basilar artery bifurcation.

PATIENTS AND METHODS

All patients undergoing neuroendovascular procedures at the medical center for treatment were obtained from a prospectively maintained database. Any patient receiving a Neuroform stent for treatment of an intracranial aneurysm was identified from this database and eligible for inclusion in the study. Beginning in January 2009, all patients undergoing embolization of an intracranial aneurysm have been imaged on post-operative day 1 to 2 with MR-DWI for detection of acute embolic lesions. All patients treated after that time are herein included in the study with the exception of 1 patient who was unable to undergo MRI because of an implanted spinal stimulator. Any patient who received MR-DWI within 48 hours of the procedure before 2009 as part of a trial assessment of MRI utility was also included.

Patient clinical and radiographic records were analyzed. Demographic data collected included patient age, sex, and patient presentation. Procedural reports and imaging were analyzed for aneurysm location, aneurysm size, and embolization technique. All MR-DWI sequences were reviewed by neuroradiologists with confirmation of findings by the authors of the study. Foci of slow diffusion detected on MR-DWI were considered eligible lesions for the study if they were greater than 2 mm in diameter, with hyperintense signal on diffusion-weighted images and hypointense signal on the corresponding apparent diffusion coefficient map.

Per the standards of our clinical practice, all patients undergoing elective treatment of aneurysms were started on dual-antiplatelet therapy with aspirin and clopidogrel beginning at least 3 days before the procedure. Antiplatelet therapy was held in patients presenting with subarachnoid hemorrhage until the time of the aneurysm embolization procedure, at which point they received aspirin 325 mg and a bolus of 300 or 600 mg clopidogrel by nasogastric tube. GPIIb/IIIa inhibitors were administered in 3 cases (eptifibatide in 2 cases, abciximab in 1 case) of acute subarachnoid hemorrhage (SAH) where patients were unable to be adequately loaded with dual-antiplatelet therapy before treatment. In all cases, the GPIIb/IIIa inhibitors were administered after the framing coil and the majority of the subsequent filler coils had been deployed to minimize the risk of rebleeding. Eptifibatide was administered in a fourth case of a patient who, upon waking from anesthesia, reported a transient subjective upper extremity hemiparesis. The patient was re-sedated, and diagnostic angiography demonstrated no evidence of in-stent thrombosis or other thromboembolic branch occlusions. The patient was started on eptifibatide as a precaution. After the procedure, patients were continued on scheduled antiplatelet therapy dosing.

All patients were anticoagulated during the procedure with an intravenous heparin drip with a goal activated clotting time of greater than 240 seconds. One patient required procedural anticoagulation with bivalirudin 85 mg in lieu of heparin because of a previous episode of heparin-induced thrombocytopenia antibody reaction; activated clotting time parameters for this patient were congruent with the remainder of the population.

The current study was approved by the Institutional Review Board of Tufts Medical Center. Statistical analysis was performed using JMP (version 8.0, SAS, Cary, North Carolina) with differences in values evaluated with 1-way analysis of variance and likelihood ratios. Statistics are reported to 2 significant figures and significance was assumed for $P < .05$.

RESULTS

Seventy-six patients treated for intracranial aneurysms with Neuroform open-cell design stents over the study period were eligible for inclusion. The study population consisted of 11 men and 65 women with a mean age of 56 years (range, 29-86 years). Ninety-seven Neuroform stents were deployed over a total of 78 procedures. One patient with concurrent aneurysms of the paraclinoid segment of the internal carotid artery and basilar artery bifurcation underwent stent-mediated coiling of the aneurysms on 2 separate occasions. Another patient underwent stent-mediated embolization of an aneurysm on consecutive days after the initial stenting procedure failed to induce sufficient flow diversion from the aneurysm.

Presenting signs and symptoms leading to aneurysm treatment were incidental finding in 33 cases, transient ischemic attack/stroke in 13 cases, headache in 14 cases, SAH in 10 cases, re-treatment of a previously coiled aneurysm in 5 cases, re-treatment of a previously clipped aneurysm in 1 case, mass effect in 1 case, and tonic-clonic seizure in 1 case. The locations of the treated aneurysms are listed in Table 1. Aneurysms were located at large-vessel bifurcations (basilar artery, middle cerebral artery, or internal carotid artery) in 30 cases (38%). The mean aneurysm size was 6.4 mm (range, 1-31 mm).

Patients were treated with single-stent Neuroform constructs in 57 cases (73%). Stent-supported coiling technique was used in 49 cases (63%), whereas 7 aneurysms were embolized with a Neuroform stent alone (9.0%). One case (1.3%) of an aneurysm at the basilar bifurcation was embolized by using a transcirculation approach via the posterior communicating artery with a Neuroform

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TABLE 1. Aneurysm Locations

Internal carotid artery	
Petrous segment	2
Cavernous segment	10
Paraclinoid segment	7
Ophthalmic segment	19
Posterior communicating segment	5
Bifurcation	6
Anterior cerebral artery	
A1 segment	1
Middle cerebral artery	
M1 segment	1
Bifurcation	2
Basilar artery circulation	
Mid-basilar artery	2
Basilar bifurcation	22
Posterior cerebral artery	
P2 posterior cerebral artery	1
Total	78

stent extending from the right P1 artery to the left P1 artery in addition to coil insertion.

Patients were treated with multi-stent Neuroform constructs in 21 cases (27%). Dual Neuroform stents were deployed in Y-fashion in 16 cases (21%; Figure 1), 1 of which was performed without coiling. Telescopic stent-in-stent constructs without coiling were used in 4 cases (5.1%). Waffle-cone technique was used in 1 case (1.3%), with a Neuroform stent deployed within the basilar artery and an Enterprise stent extending from the basilar artery to the aneurysm dome. Multi-stent constructs were deployed in 17 of 30 procedures (57%) treating bifurcation aneurysms.

A total of 12 of 78 (15%) procedures used stent embolization alone without coil deployment for aneurysm treatment. These aneurysms were smaller than the cohort of aneurysms that were treated with stent-coiling (1.9 ± 0.8 mm vs 7.2 ± 5.3 mm, respectively; $P = .001$).

MR-DWI was reviewed for detection of acute ischemic events. Hyperintense lesions indicative of ischemia that were ipsilateral to the territory of the stent-containing artery were identified following 7 of 78 cases (9.0%; Table 2; Figure 2) in the total population. None of these lesions resulted in a permanent neurological deficit, although 1 patient did experience post-operative confusion, aphasia, and increased urine output that completely resolved by the time of discharge. Ipsilateral magnetic resonance diffusion-weighted imaging hyperintense lesions (MR-DWI+) were identified in 6 of 7 (86%) cases. There was a single case of a patient with MR-DWI+ contralateral to the side of the target aneurysm and deployed Neuroform stent. In this case, a transcirculation approach was used to gain access to an internal carotid artery bifurcation aneurysm by threading the microcatheter through the anterior communicating artery. The diffusion abnormality in this case was located in the caudate nucleus contralateral to the aneurysm, and was included in the analysis because it was felt that the MR-DWI+ could be attributed to the procedure technique.

Of the 4 cases where intraoperative GPIIb/IIIA inhibitors were administered, all patients were MR-DWI−, including the case where eptifibatide was administered as a precaution for a transient subjective upper-extremity hemiparesis.

All aneurysms with MR-DWI+ were treated with stent-coiling. There were no acute ischemic events detected in patients treated with Y-Stent or telescopic Neuroform constructs despite the added complexity associated with these approaches, and, similarly, none were detected in patients treated with Neuroform stenting alone (without use of coils). The rate of DWI lesions was 10.3% in the unruptured cohort (7/68) and 0% in the SAH cohort (0/10). Of the 7 patients with DWI lesions, none experienced a permanent neurological deficit.

Statistical analysis was restricted to MR-DWI+ lesions located ipsilateral to the deployed Neuroform stent and target aneurysm in order to evaluate the isolated contribution of the implant; the contralateral MR-DWI+ was excluded because its etiology is of a procedural nature in the setting of codominant A1 flow. Intraprocedural response to heparin anticoagulation therapy was available for 75 of 77 (97%) procedures; none of the cases without data on heparin therapy were found to harbor MR-DWI+.

Analysis of the total population and unruptured cohorts (Table 3) demonstrated no association between the presence of MR-DWI+ and age ($P = .37$ and $P = .42$, respectively), sex ($P = .19$ and $P = .18$, respectively), aneurysm size ($P = .25$ and $P = .31$, respectively), intraprocedural response to heparin (Table 3), guide catheter size ($P = .26$ and $P = .34$, respectively), number of guide catheters used ($P = .40$ and $P = .46$, respectively), stent deployment technique ($P = .22$ and $P = .20$, respectively), or jailing vs sequential aneurysm embolization technique ($P = .69$ and $P = .90$, respectively). Target aneurysm location on an arterial sidewall over large-artery bifurcations was significantly associated with the presence of ipsilateral MR-DWI lesions in both the total population and the unruptured cohort ($P = .01$ and $P = .01$, respectively).

DISCUSSION

Recently published data evaluating the rate of MR-DWI hyperintensities following aneurysm coiling without stent assistance have found an incidence ranging from 24% to 55%[10-13] in unruptured cohorts and ranging from 44 to 70%[10-12] in ruptured cohorts. Although many studies have evaluated the rate of MR-DWI lesions in entire neurointerventional populations or have focused on coiling alone, relatively fewer studies have strictly evaluated the effect of Neuroform stenting on acute thromboembolism. Altay et al[12] included data on 14 patients receiving a Neuroform stent and reported an MR-DWI hyperintensity incidence rate of 30%. These authors noted that use of balloon assistance was associated with a lower rate of MR-DWI lesions.

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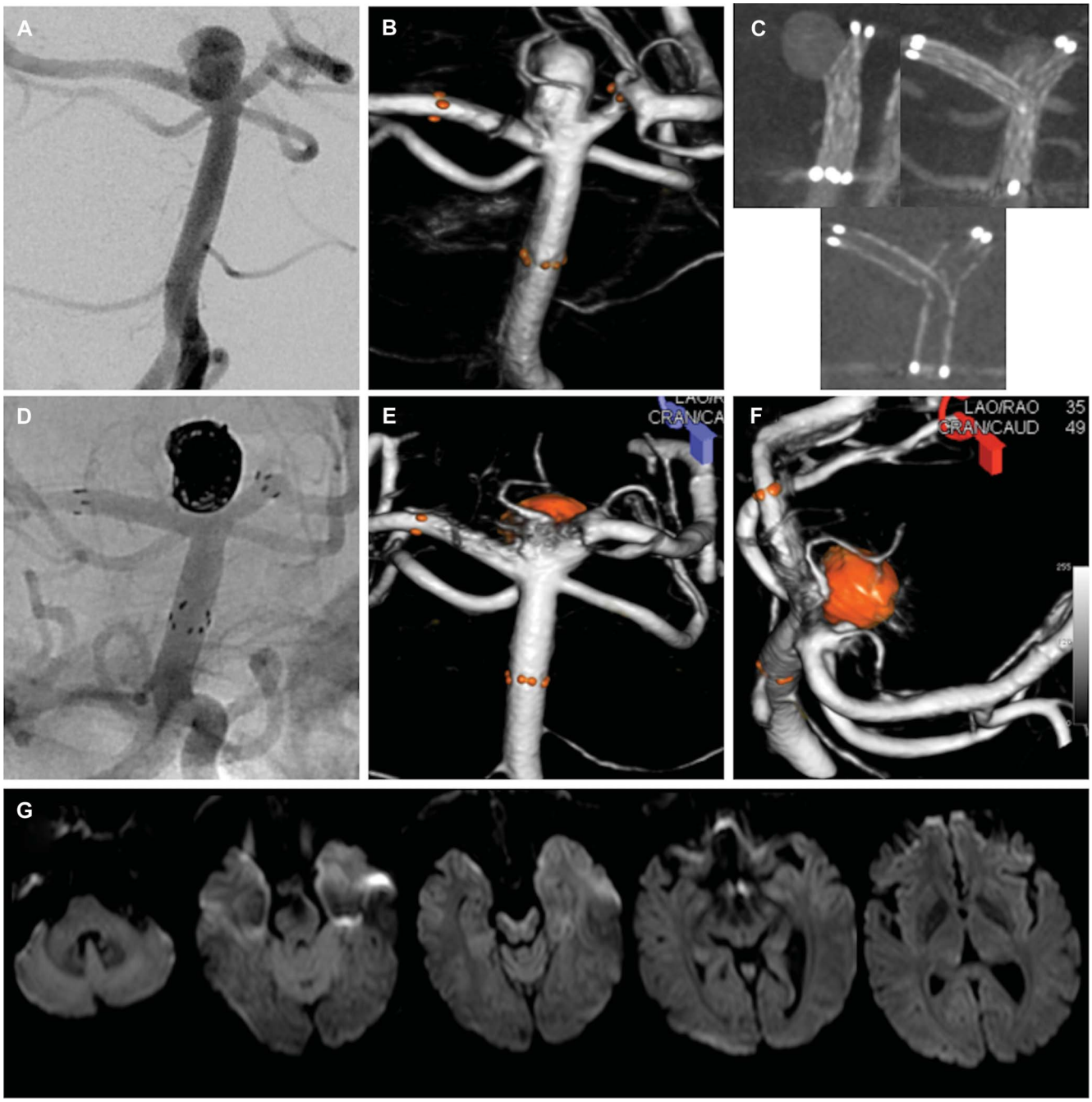


FIGURE 1. *A 59-year-old woman was found to harbor an incidentally found 7.8-mm aneurysm of the basilar artery bifurcation visualized on AP-view digital-subtraction angiography (**A**). Because of a wide-neck measuring 3.5 mm, stent-coil embolization was planned with the use of the Y-stent technique. Stenting was performed initially with appropriate deployment of the 2 stents in Y-fashion visualized through 3-D reconstructions (**B**) and Dyna-CT cross-sectional imaging (**C**) confirming adequate coverage of the aneurysm neck and interstrut interaction. **D**, coiling of the target aneurysm was performed after stenting, with 3-D reconstructions (**E**, **F**) demonstrating preservation of arterial flow around the aneurysm and delineation of the perforating arteries emanating off of the basilar apex. **G**, postoperative diffusion-weighted MRI revealed no hyperintense lesions and no evidence of acute procedural thromboembolism. AP, anterior-posterior.*

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TABLE 2. Summary of Patients With MR-DWI Hyperintense Lesions Following Neuroform Stent-Coiling[a]

Age/ Gender	Aneurysm Size/ Location	Presentation	Treatment Technique	Number of Lesions	Lesion Size, mm	Clinical Course
68/F	6.0-mm cavernous ICA	Incidental	Balloon test occlusion followed by stent-coil	1 ipsilateral	5	Postoperative course complicated by increased urine output, confusion, and aphasia. All symptoms resolved by discharge
54/F	5.2-mm paraclinoid ICA	Incidental	Stent-coil	3 ipsilateral	3; 4; 5	MR-DWI+; asymptomatic
57/F	17-mm cavernous ICA	Mass effect	Stent-coil	3 ipsilateral	6; 5; 4	MR-DWI+; asymptomatic
46/F	6.0-mm cavernous ICA	Re-treatment of previously coiled, ruptured aneurysm	Stent-coil	1 ipsilateral	5	MR-DWI+; asymptomatic
74/F	12-mm posterior communicating artery	Incidental	Stent-coil	3 ipsilateral	4; 4; 3	MR-DWI+; asymptomatic
56/F	6.1-mm ophthalmic ICA	Headache	Stent-coil	1 ipsilateral	4	MR-DWI+; asymptomatic
63/M	7.5-mm ICA bifurcation	Incidental	Stent-coil	1 contralateral[b]	8	MR-DWI+; asymptomatic

[a]MR-DWI, diffusion-weighted magnetic resonance imaging; MR-DWI+, magnetic resonance diffusion-weighted imaging hyperintense lesion; ICA, internal carotid artery.
[b]See text for detailed description of MR-DWI+.

The observed 9.0% rate of MR-DWI+ in the current study of 78 cases confirms our hypothesis that the Neuroform stent is safe perioperatively for intracranial aneurysm embolization. The single case of a contralateral MR-DWI+ could be explained by 2 possibilities: the formation of microemboli on the delivery microcatheter and the effect of flow disruption through the anterior circulation. However, because of the transcirculation technique used and the location of the lesion, the diffusion abnormality can most likely not be attributed to the Neuroform stent itself.

The safety of the Neuroform is further confirmed by the finding that of the patients with MR-DWI+, none experienced a permanent neurological deficit. In 7 of 78 patients, there was a total of 13 hyperintense lesions on MR-DWI, which is fewer than the total of 60 hyperintense lesions that we observed in 12 of 18 patients with a Crescent sign following Enterprise stent-coiling.[8] With a lower average number of MR-DWI+ per patient (1.9 lesions per patient) and relatively consistent lesion size (Table 2, with the exception of the contralateral MR-DWI+) in this series vs our previous Enterprise study with a higher average MR-DWI+ per patient (5 lesions per patient) and relatively discrepant lesion size (range, 2-7 mm), these results indicate that the Neuroform stent and delivery system may be less thrombogenic than the Enterprise stent and delivery system.

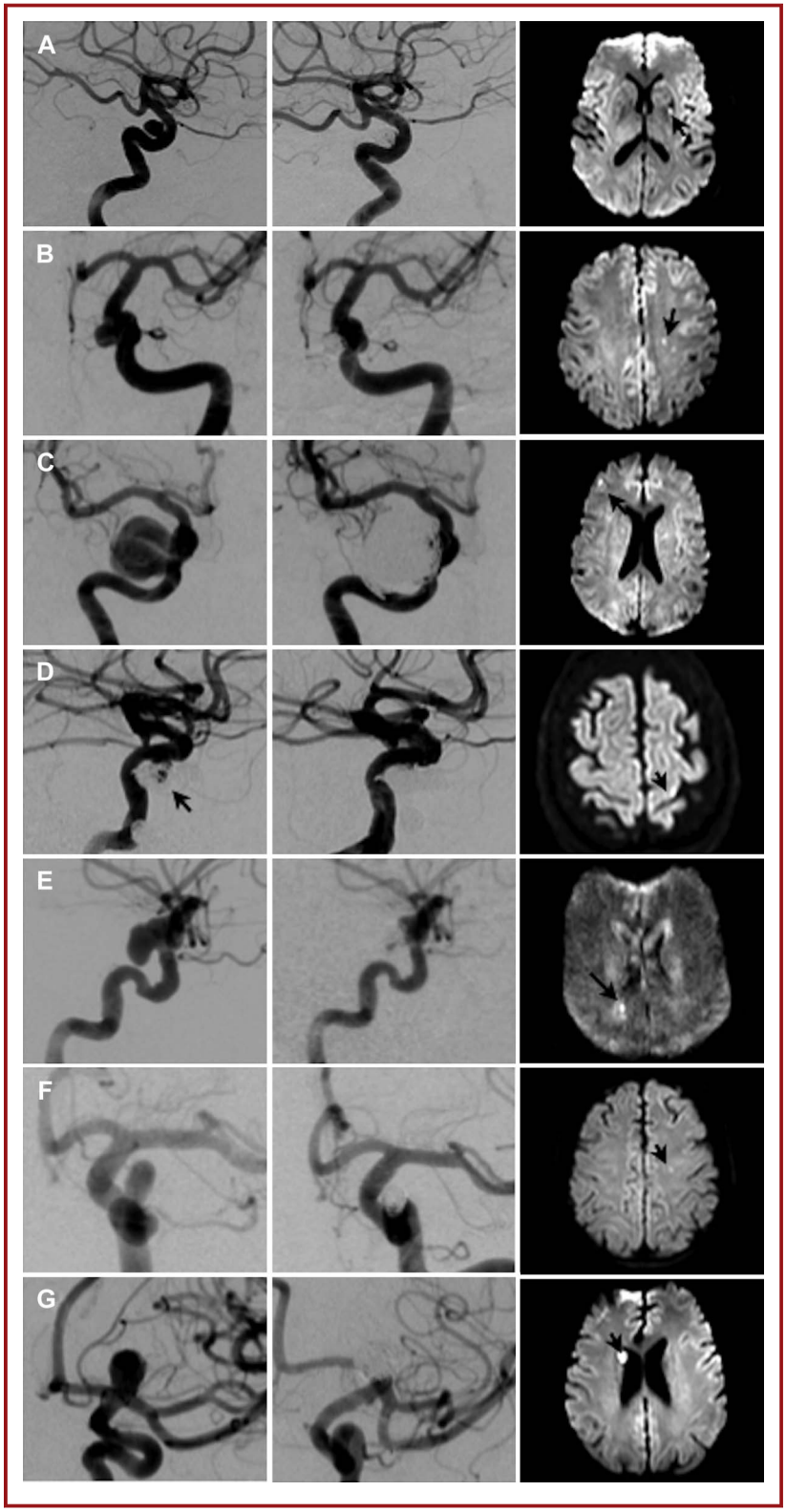
Perhaps the most compelling finding of the current study is the 0% rate of MR-DWI+ following procedures involving deployment of multiple Neuroform stents, including a cohort of 16 consecutive Y-stent cases. We originally hypothesized that Y-stent and telescopic stent constructs would predispose to acute thrombus formation because of stent-on-stent interactions (Figure 1) and the inherent difficulties in navigating catheters and guide wires through deployed stent struts, and the need for additional manipulation or the use of a tri-axial technique. These results demonstrate that the versatility of the Neuroform stent to be deployed in various configurations such as the Y-stent does not increase the procedure risk of thromboembolic complications in the current series.

The finding that aneurysms in sidewall locations are more susceptible to procedural thromboembolism could be explained by the difference in hemodynamic forces within the aneurysm and stent struts after deployment. The inflow angle of sidewall aneurysms may produce greater shear stress on the stent struts and predispose to thrombus formation.

Furthermore, the absence of detected lesions in the SAH cohort (0%) in comparison with the unruptured cohort (10.3%) provides evidence for the possible safe deployment of the Neuroform stent in the presence of acute SAH. The cohort of patients with acute SAH is relatively small (n = 10), and thus a definitive association cannot be asserted. In their systematic review of the literature, Bodily et al[14] identified a 6% rate of clinically significant thromboembolic events in patients undergoing stent-assisted coiling for ruptured aneurysms, whereas Amenta et al[15] identified a 7.7% rate of intraoperative stent thrombosis in cases of ruptured aneurysms. Although these studies evaluated clinically evident thromboembolic events rather than through the use of MR-DWI, they demonstrate that there is a risk of thromboembolism for patients undergoing stent-mediated coiling for ruptured aneurysms.

The need for antiplatelet therapy acutely after stent deployment can complicate the management of SAH patients, including the need for ventricular drainage and related procedures. However, in

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FIGURE 2. *Preembolization digital-subtraction angiography (**Left**), postembolization digital-subtraction angiography (**Center**), and MR-DWI (**Right**) of the 6 patients with acute thromboembolic events. **A**, a 68-year-old woman incidentally found to harbor a 6.0-mm left-sided cavernous ICA aneurysm demonstrated a 5-mm hyperintense lesion (black arrow) on MR-DWI in her left putamen. **B**, a 54-year-old woman incidentally found to harbor a 5.2-mm left-sided paraclinoid ICA aneurysm demonstrated multiple hyperintense lesions (black arrow) on MR-DWI in her left cerebral white matter. **C**, a 57-year-old woman who presented with mass effect from a 17-mm right-sided cavernous ICA aneurysm demonstrated multiple hyperintense lesions on MR-DWI, one of which was located in the right frontal lobe (black arrow). **D**, a 46-year-old woman requiring re-treatment of a 6.0-mm previously coiled, ruptured left-sided cavernous ICA aneurysm with residual filling (black arrow) demonstrated a hyperintense lesion (black arrow) in the left pre-central gyrus. **E**, a 74-year-old woman incidentally found to harbor a 12-mm aneurysm at the right posterior communicating artery demonstrated multiple hyperintense lesions on MR-DWI, one of which was located in the right cerebral white matter (black arrow). **F**, a 56-year-old woman who presented with headache harbored a 6.1-mm left-sided ophthalmic ICA aneurysm and demonstrated a single 4-mm hyperintense lesion on MR-DWI located in the left centrum semiovale. **G**, a 63-year-old man incidentally found to harbor a 7.5-mm left ICA bifurcation aneurysm demonstrated a hyperintense lesion (black arrow) on MR-DWI in his right caudate nucleus. MR-DWI, diffusion-weighted magnetic resonance imaging; ICA, internal carotid artery.*

properly selected patients, the benefit of stenting to enable aneurysm treatment, or as a rescue for coil herniation, may outweigh the risks. Because acute bleeding may affect the reactivity of the cerebral vasculature and make it more susceptible to manipulation during endovascular procedures, further work remains to be done to determine the safety of stent deployment in patients with SAH.

There were no patients in the current study that experienced acute stent thrombosis, especially interesting considering the inability to adequately inhibit platelet function preoperatively in the SAH subset. We hypothesize that the practice of dual-antiplatelet therapy with aspirin and clopidogrel significantly contributes to the decreased risk of thromboembolic complications observed in the current study.

TABLE 3. Univariate Analysis for Prediction of Ipsilateral Procedural Embolism Detected by MR-DWI[a,b]

		MR-DWI− (n = 71)			
	MR-DWI+ (n = 6)	Total Population (n = 71)	*P*	Unruptured Cohort (n = 61)	*P*
Age, y	59.8 ± 10.0	55.4 ± 11.6	.37	55.6 ± 12.1	.42
Sex (M:F)	0:6	10:61	.19	9:52	.18
Aneurysm size, mm	8.8 ± 4.7	6.2 ± 5.3	.25	6.4 ± 5.5	.31
Bifurcation:sidewall	0:6	29:42	.01	25:36	.01
Intraprocedural anticoagulation					
Mean ACT, s	239 ± 16	231 ± 20	.37	234 ± 18	.54
ACT fluctuation, s	60 ± 14	69 ± 31	.54	70 ± 30	.45
Trough ACT, s	207 ± 14	196 ± 19	.22	199 ± 16	.25
Peak ACT, s	267 ± 23	265 ± 33	.88	269 ± 31	.88
Heparin dose/BMI	170 ± 88	159 ± 61	.70	165 ± 63	.85
Guide catheter Fr size (5:6:7:8)	2:3:1:0	45:24:1:1	.26	36:23:1:1	.34
Number of guide catheters (1:2)	4:2	58:13	.40	49:12	.46
Procedure technique			.22		.20
Stent-coiling	6	43		36	
Stent only	0	7		6	
Waffle cone	0	1		1	
Telescopic stenting	0	4		2	
Y-stenting	0	16		16	
Embolization technique[c] (jailing:sequential)	4:2	25:18	.69	23:13	.90

[a]MR-DWI, diffusion-weighted magnetic resonance imaging; MR-DWI+, magnetic resonance diffusion-weighted imaging hyperintense lesion; MR-DWI−, magnetic resonance diffusion-weighted imaging without hyperintense lesion; ACT, activated clotting time; BMI, body mass index.
[b]Values are reported as mean ± standard deviation.
[c]This analysis was restricted to the 49 patients (42 in the unruptured cohort) that underwent simple stent-mediated coiling.

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In a previous study, we described the increased risk for thromboembolism that incomplete stent apposition confers upon intracranial stents.[8] Although the closed-cell design of Enterprise stent makes it susceptible to gross incomplete apposition, close examination of the Neuroform shows that it too is susceptible to a subtle form of incomplete stent apposition through the fish-scaling effect of the open-cell struts.[6] Even with this finding, the potential fish-scaling effect of the Neuroform does not appear to increase the risk of perioperative thromboembolic complications. This could possibly be the result of the self-expanding struts of the Neuroform being pushed out of the central portion of the vessel where the high flow velocities and high shear rates are expected in comparison with near the wall.

The rate of MR-DWI findings presented in this study is lower than might have been expected from such a cohort of patients. A contemporaneous cohort of patients receiving the closed-cell design Enterprise stent at our institution[16] harbored a 38% rate of MR-DWI+ following stent-assisted coiling procedures complicated by incomplete stent apposition. Because this cohort was treated by the same neuroendovascular team with identical procedural technique, equipment, and anticoagulation protocols as the patients in this study, the likelihood of chance difference in the data of the current study is rather low. With such similar protocols for patient treatment, the discordant rates of thromboembolism and source of these events might be attributed to the guide catheters/microcatheters and implant devices themselves. Paradoxically, we hypothesized that the Neuroform would have a higher rate of MR-DWI+ because the intravessel profile of its 0.027-in microcatheter is larger than that of the 0.021-in Enterprise microcatheter. Yet, the Neuroform stent appears to confer a lower risk of thromboembolism than other currently available devices, even in complex and Y-stent procedures.

Limitations

Limitations of the current study include the absence of platelet function assays, which would have provided an objective evaluation of preprocedural aggregation status and identified patients more prone to thromboembolic events. Formal platelet aggregometry testing for neurointerventional procedures was begun at our institution in October 2011, and thus is not available for the entire population. Although the standard dual-antiplatelet therapy regimen could not be formally evaluated, we observed a low MR-DWI+ rate of 9.0% in the current study, with each patient harboring no more than 3 lesions. Another limitation of the study is the long time period over which patients were treated, where new techniques and improved technology may have affected the procedures. Finally, preprocedural MR-DWI was not available for control in the study population.

Gao et al[17] reported a 5.6% rate of procedural thromboembolism associated with Neuroform stent-coiling of aneurysms in 232 patients. In their report on 235 patients treated with Neuroform stents for intracranial aneurysms, Lessne et al[18] reported a similar combined rate of 5.4% for clinical and imaging evident lesions within 90 days of the procedure that climbed to 20% in the cohort of patients with SAH. However, these studies were limited by a reliance on clinical events. Using MR-DWI in a cohort of 66 consecutive cases of diagnostic cerebral angiography, Bendszus et al[19] noted a rate of MR-DWI+ in 26% of cases. Patients undergoing diagnostic cerebral angiography are often not anticoagulated with dual-antiplatelet therapy as aggressively as those undergoing interventional stent-mediated coiling, and this may contribute to the lower rate of MR-DWI+ in the current study.

We chose to evaluate radiographic thromboembolic events in this study for 2 main reasons. The first is that the majority of these cases were unruptured aneurysms, and for a study with such a high proportion of elective cases to evaluate clinical ischemic events (strokes and transient ischemic attacks), the number of patients required for analysis would need to be significantly larger. The second is that, by counting and measuring each focus of slow diffusion on MR-DWI, this study was able to provide a more quantitative method of detecting ischemic events. This analysis will aid in determining the safety of any given procedure by analyzing events on an all-comer basis, rather than the much more rare clinically detectable events.

There has been extensive analysis of the relevance of subclinical ischemic events detected by MR-DWI in both the cardiac surgery and carotid artery stenting literature. With the exception of a study by Barber et al,[20] the literature evaluating the occurrence of these events following cardiac surgery has found that ischemic lesions on postoperative MR-DWI are not associated with neurocognitive decline seen commonly in many patients.[21,22] Meanwhile, studies evaluating the significance of ischemic events on MR-DWI following carotid artery stenting have indicated that the small diffusion abnormalities observed on postprocedure imaging are associated with neurocognitive decline.[23-25] Until sufficient studies of this nature have been completed in the neuroendovascular procedure with regard to the effect of stent implantation within the cerebral circulation on neurocognitive decline, careful monitoring of these lesions is warranted in order to reduce any potential procedure-related risks.

The results of the current study obtained using surveillance MR-DWI on all patients demonstrate that the Neuroform stent carries a very low risk of procedural thromboembolism (9.0%). The deployment of multiple stents in the same anatomical region in configurations such as the Y-stent and telescopic stents is also a safe maneuver. Although enthusiasm for flow-diverter stents continues to build, the Neuroform stent remains a safe method for stent-assisted coiling and stent-induced flow diversion for treatment of intracranial aneurysms.

CONCLUSION

We sought to evaluate the procedural risk of Neuroform stent deployment for treatment of intracranial aneurysms as defined by the presence of acute ischemic thromboembolism detected by MR-DWI. Over the course of 78 procedures featuring Neuroform

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stent deployment, we observed a 9.0% rate of MR-DWI+, with a rate of 0% in cases where stents were deployed in complex constructs. As such, the Neuroform stent carries a low risk of MR-DWI+. Subarachnoid hemorrhage and deployment of multiple stents in the same anatomical region in configurations such as the Y-stent did not increase the risk of acute procedural thromboembolism in this series.

Disclosures

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COMMENT

The authors report low incidence (9%) of MR-DWI+ lesions after the treatment of intracranial aneurysms with Neuroform stenting.

Seventy-six patients were treated in 78 procedures. A single-stent was used in 57 cases (73%), multi-stent Neuroform constructs were used in 21 cases (27%) with Y configuration in 16 cases (mostly Basilar Tip location), and in 4 cases telescopic stent-in-stent. Ten cases were treated in acute settings of subarachnoid hemorrhage. All patients were treated under full anticoagulative therapy and double-antiplatelet therapy. Location on an arterial sidewall was associated with the presence of MR-DWI lesions, but none were observed in Y stenting or acute phase cases.

The findings of this study are important because there are accumulating data showing the low thromboembolic risk of open-cell design stent vs the closed-cell design.

Nonetheless, the fact that no lesion was found in cases of multiple stenting use (Y stenting) is compelling and should be balanced by the absence of MCA and ACA localization. The anatomy of the basilar tip and the relatively straightforward access to this localization certainly account for the extremely low complication rate.

In our experience, the use of the Y-stenting technique has dramatically broadened the possibility of endovascular treatment of complex wide-neck aneurysms in the MCA and ACA locations. But the navigation with the large (0.027 in) microcatheters required for the Neuroform stent remains

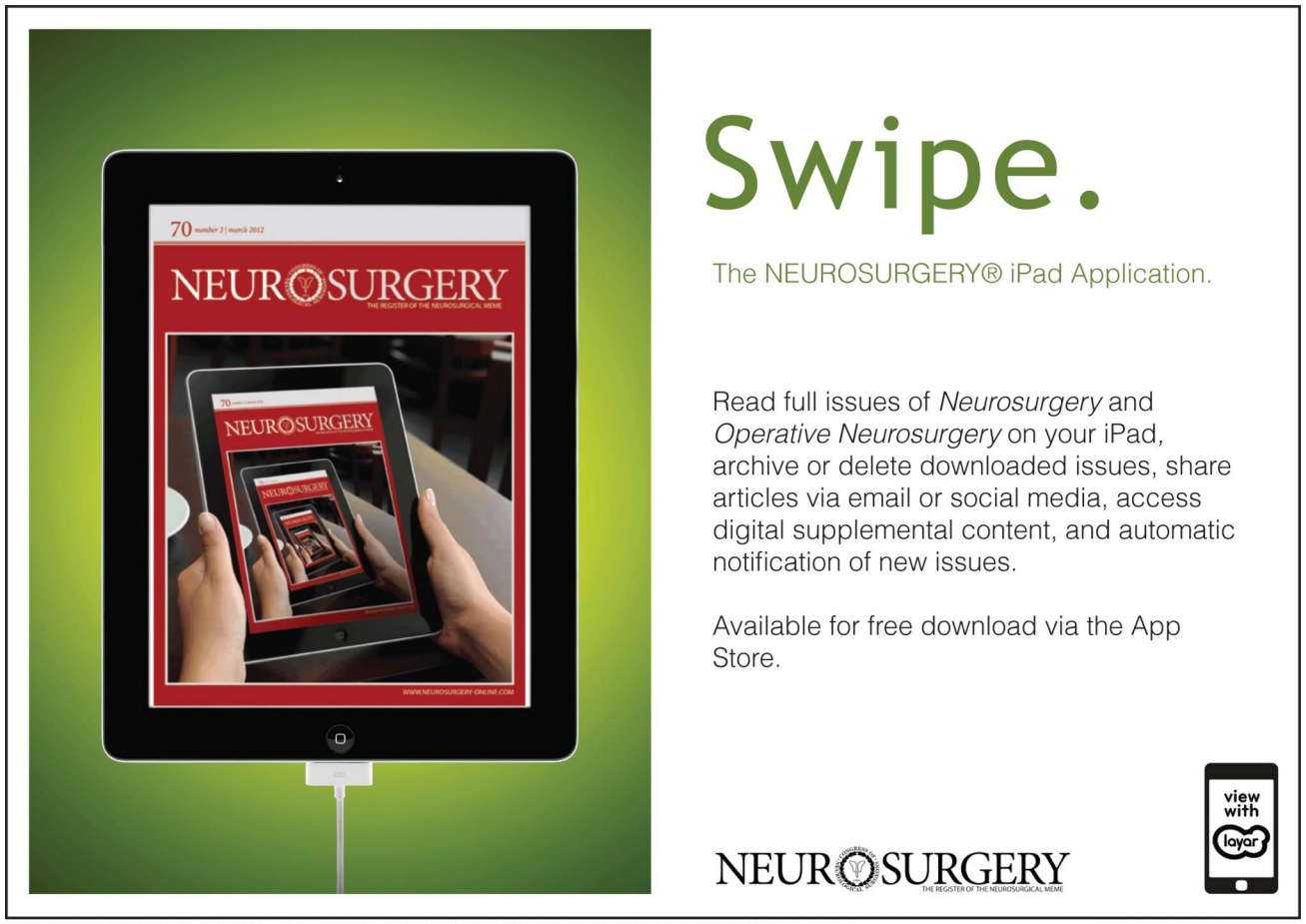
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cumbersome, sometimes impossible, and accounts for ischemic or even more feared hemorrhagic complication. It is noteworthy that the most significant ischemic lesion reported in this article is contralateral during a procedure where the microcatheter was navigated through the anterior communicating complex.

As prompted for a long time, the use of a lower-profile and more maneuverable catheter (0.014-0.021) for open-cell design stent navigation would certainly be extremely beneficial for the safety in the bifurcation locations.

The other ischemic complications have been found for lateral type aneurysms, where the deployment of the open-cell stent type is known to be better than the closed-cell design but still suboptimal, we consider in those locations that other technologies such as braided stent type (Flow diverters) are more suitable.

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