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Endovascular Repair of Peripheral and Visceral Aneurysms With the Cardiatis Multilayer Flow Modulator: One-Year Results From the Italian Multicenter Registry

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◆CLINICAL INVESTIGATION ◆

Endovascular Repair of Peripheral and Visceral Aneurysms With the Cardiatis Multilayer Flow Modulator: One-Year Results From the Italian Multicenter Registry

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Purpose: To assess the efficacy of the Cardiatis Multilayer Flow Modulator (CMFM), a bare cobalt alloy self-expanding stent, in the treatment of peripheral and visceral artery aneurysms.
Methods: In this multicenter (n=22), prospective, voluntary registry, 54 patients (47 men; mean age 68 years, range 19–87) who underwent CMFM deployment for peripheral (n=35) or visceral aneurysms (n=19) in a variety of target arteries were enrolled between June 2009 and June 2010. Among the 54 lesions, 44 had a total of 53 side branches. The main effectiveness endpoint was stent and side branch patency with no aneurysm rupture or reperfusion at 1, 6, and 12 months after stent implantation. Outcome measures were complete aneurysm thrombosis and sac shrinkage. The safety endpoint was freedom of complications (death, aneurysm rupture, endoleak, need for reintervention, stent foreshortening, stent occlusion, and access-site sequelae). Aneurysms were categorized as saccular (type I) or fusiform (type II) without a side branch or with branch(es) in the sac (subtype A), neck (subtype X), or both (subtype AX). Kaplan-Meier estimates were calculated for primary and secondary endpoints. Sac shrinkage was correlated to aneurysm morphology subtypes and presence/absence of mural thrombus.
Results: Technical success was achieved in all patients. Mortality at 1 year was 5.5% (n=3), including 1 perioperative death. Six patients were lost to follow-up. There was no aneurysm rupture. Six (11.1%) stents occluded over the 1-year period; 3 asymptomatic patients were not treated, 2 symptomatic patients had successful stent dilation to restore patency, and 1 symptomatic patient required bypass (the only side branch lost). Cumulative primary and secondary patency estimates were 86.9% and 90.7% at 1 year. The cumulative side branch patency was 96.1% and the freedom from all complications was 83.0% at 1 year. Complete aneurysm thrombosis was recorded in 42 (93.3%) of 45 patients at 1 year. Percent diameter reduction was 15.5%, 3.8%, and 11.0% at 1, 6, and 12 months ($p<0.05$), respectively. Presence of mural thrombus did not influence the time course of shrinkage ($p>0.05$), while complex lesion anatomy (presence of side branches) delayed shrinkage ($p<0.05$).
Conclusion: Results at 1 year show that CMFM can be safely used in the treatment of PAA and VAA, with good results in terms of freedom from rupture, patency of the stents and side branches, complete aneurysm thrombosis, shrinkage, and acceptable freedom from morbidity and mortality.

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Key words: visceral artery aneurysm, peripheral artery aneurysm, aneurysm classification, sac shrinkage, flow modulator, stent, aneurysm diameter, aneurysm rupture, aneurysm thrombosis, complications, mural thrombus

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Peripheral (PAA) and visceral artery aneurysms (VAA), though less common than aortic aneurysms, are clinically important due to their high incidence of rupture and threat to end organs or life. Surgery has always been considered the treatment of choice for these lesions, but in the past few years, endovascular techniques using covered stents and embolization coils have increased the treatment options available to comorbid patients not suitable for open repair.[1–7] However, endovascular techniques were, until recently, unsuitable for aneurysms with side branches from the sac or neck: covered stents would block the side branches and coils would occlude the target artery.

Recently, a new endovascular device was introduced for peripheral and visceral aneurysm exclusion; its unique multilayer wire construction laminated flow through the lumen such that organized thrombus formed in the aneurysm sac but the lumen and any branch vessels remained patent. The Cardiatis Multilayer Flow Modulator (Cardiatis, Isnes, Belgium) was approved for the European market in 2009, at which time an independent, voluntary, multicenter registry was begun in Italy to evaluate the device. Early results were promising,[8] and we now present the 1-year outcomes with this new concept in stent-based aneurysm exclusion.

METHODS

Registry Design

The Italian Registry of Peripheral and Visceral Aneurysm Exclusion Procedures is a nonrandomized, multicenter, voluntary registry of patients implanted with the Cardiatis Multilayer Flow Modulator (CMFM). Since June 2009, Italian physicians (Appendix) in 22 clinical centers have submitted data regarding patients undergoing CMFM implantation for storage in a dedicated central database and subsequent periodic analysis. The registry was endorsed by the Italian Society of Vascular and Interventional Radiology (no administrative, analytical, or financial involvement).

Patients were enrolled in the registry if they were stable, >18 years old, had a life expectancy >1 year, and had a true or false peripheral or visceral aneurysm with anatomical characteristics suitable for treatment with the CMFM. Patients were not enrolled in the registry if they were unstable, pregnant or breastfeeding, or had (1) a ruptured true/false aneurysm or contained rupture on computed tomography (CT), (2) a target vessel diameter not suitable for treatment with the CMFM, (3) a contraindication to antiplatelet therapy, (4) reported adverse reaction to contrast medium, or (5) an allergy to cobalt. Informed consent for the procedure and data collection was obtained from the patients prior to enrollment.

Clinical and physical examination data were recorded in a case report form and collected at patient enrollment, device implant, discharge, and scheduled/unplanned follow-up visits. An external, independent observer (clinical radiologist) reviewed imaging studies for consistency and to adjudicate complications.

Device Design

The CMFM is a bare, self-expanding, braided wire tube of metallic cobalt alloy wire constructed in multiple interconnecting layers (Fig. 1). The concept of the 3-dimensional layers is to modulate blood flow to significantly reduce turbulence inside the aneurysm (up to 90% reduction in flow velocity), leading to formation of an organized thrombus; flow to any collateral branch is not compromised. The mechanism of flow modulation is based on the configuration of the aneurysm and

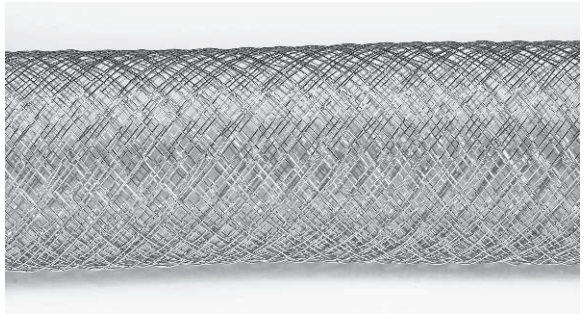

Figure 1 ◆ Close-up view of the multiple layers of cobalt alloy wires comprising the Cardiatis Multilayer Flow Modulator.

presence or absence of collaterals. In an aneurysm without collaterals, thrombus organizes into stable layers that do not put pressure on the sac wall/neck, protecting against rupture. If branches are present, the flow is redirected to the collaterals owing to the Venturi effect. The sac progressively retracts over time. Though closure may take several months, the sac is no longer subject to pressure. Flow through the branch ostia is laminar, without turbulence.

At the time this registry was begun, the CMFM for peripheral/visceral aneurysms was available in lengths of 40 to 120 mm and diameters ranging between 5 and 12 mm, compatible with 6-F to 12-F sheaths.

Patient Population

Between June 2009 and June 2010, 54 patients (47 men; mean age 68 years, range 19–87) with peripheral (n=35) or visceral (n=19) aneurysms underwent CMFM deployment in 22 clinics. The majority of the aneurysms (44, 81.5%) had side branches (n=53); mean aneurysm diameter was 32.5 mm (range 9–90). The aneurysms were classified according to a new morphological scheme (Fig. 2) based on sac configuration and presence/absence of branches, a classification better suited to the analysis of CMFM data (Table). According to this scheme, 8 (14.8%) lesions were type I, 3 (5.5%) were type I A, 3 (5.5%) type II, 18 (33.4%) type II A, 14 (26.9%) type II X, and 8 (14.8%) type II AX (Table 1). The 6-month results in the 19 VAA patients were included in a previous publication.[8]

Patient Evaluation and Procedure

Preoperative diagnostic work-up consisted of contrast-enhanced CT scans reconstructed from 0.625 to 2.5-mm axial slices adequate for lesion evaluation. Aneurysm diameters were evaluated as the shortest transverse diameter of the artery on the axial image by the same independent external observer. Duplex ultrasound was performed initially in 2 popliteal aneurysms, but CT scans were requested before the procedure to evaluate lesion and target artery diameter. Physicians proposed intervention based upon well established criteria.[9–20]

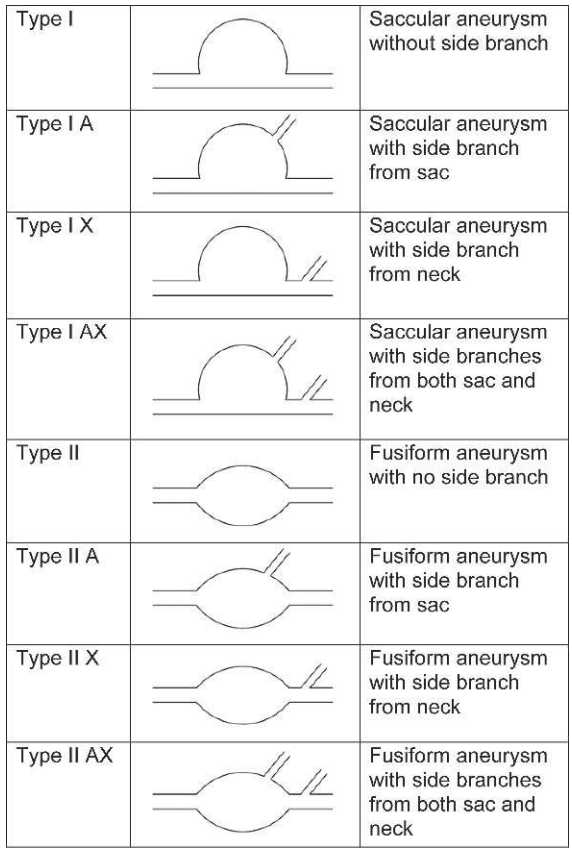

Figure 2 ◆ Aneurysm classification according to sac configuration and side branch pattern.

The procedures were performed on patients under conscious sedation with local anesthetic in a dedicated endovascular suite using a standard treatment protocol as previously described.[8] All patients received systemic anticoagulation with heparin (2500 units). After the procedure, dual antiplatelet therapy [aspirin 100 mg/d and either clopidogrel (75 mg/d) or ticlopidine (500 mg/d) according to physician preference] was prescribed for 1 month and then aspirin indefinitely (100 mg/d). Imaging follow-up with CT scans was scheduled at 1, 6, and 12 months.

Endpoints and Definitions

The main effectiveness endpoints were stent and side branch patency and freedom from aneurysm rupture or reperfusion at 1, 6, and 12 months after stent implantation. Outcome measures to evaluate efficacy were complete

TABLE 1
Demographic and Lesion Characteristics for the 54 Study Patients

Age, y		68±13.9 (19–87)	
Men		47 (87.0%)	
Comorbidities			
High blood pressure		21 (38.8%)	
Dyslipidemia		1 (1.8%)	
Renal insufficiency		2 (3.6%)	
Smoking, active and history		18 (33.3%)	
Diabetes mellitus		5 (9.2%)	
COPD		1 (1.8%)	
Obesity		2 (3.6%)	
Marfan syndrome		1 (1.8%)	
History of vascular surgery/EVAR		7 (12.8%)	
History of CAD/CABG/stenting		3 (5.5%)	
Aneurysms			
Maximal diameter, mm		32.5±18.9 (9–90)	
With side branches		44 (81.5%)	
Side branches at risk		53	
Locations	**Diameter, mm***	**Branches**	**Types†**
Subclavian artery (n=1)	40	0	I
Celiac trunk (n=3)	38 (34–50)	6	II A, II X, II AX
Splenic artery (n=5)	20.8 (20–35)	1	4 I, 1 I A
Hepatic artery (n=5)	60.6 (43–90)	6	1 I A, 3 II A, 1 II X
Gastroduodenal artery (n=1)	9	0	I
Superior mesenteric artery (n=2)	54, 70	5	2 II AX
Renal artery (n=3)	11.7 (10–14)	3	I, II X, II AX
Iliac artery (n=22, 2 in EVAR)	36.5 (23–65)	22	10 II A, 10 II X, 2 II AX
Aortobi-iliac bypass (n=3)	25 (17–30)	1	2 II, 1 II A
Femorofemoral bypass (n=1)	18	0	I
Popliteal artery (n=6)	23.2 (19–26)	8	I A, II, 2 II A, II X, II AX
Femoropopliteal bypass (n=2)	18, 23	1	II A, II AX

Continuous data are presented as the means ± standard deviation (range); categorical data are given as the counts (percentage).
CABG: coronary artery bypass graft, CAD: coronary artery disease, COPD: chronic obstructive pulmonary disease, EVAR: endovascular aneurysm repair.
* Mean (range) for n≥3.
† See Figure 2 for description.

aneurysm thrombosis and shrinkage of the lesions at the same time points. Stent and side branch patency was defined as absence of ≥50% lumen diameter stenosis. Aneurysm reperfusion was defined as loss of complete sac thrombosis without sac shrinkage.

The primary safety endpoint was freedom from complications, including minor events (e.g., access-site hematoma, pseudoaneurysm, skin infection) serious adverse events (death, aneurysm rupture, and any event resulting in disability, requiring percutaneous or surgical intervention, or prolonging hospitalization); endoleak; and stent occlusion or foreshortening. Only type I endoleak (incomplete sealing) and type III endoleaks (inadequate overlap of multiple devices) were relevant to the CMFM; types II and IV were not applicable to this device.

Technical success was defined as successful deployment of the stent within the target artery with stent and side branch patency at the end of the procedure. Clinical success was defined as exclusion of the aneurysm with preservation of collateral branch patency at each follow-up after CMFM implantation.

Statistical Analysis

The prospectively collected, anonymized data were analyzed retrospectively by an independent statistician using the intention-

to-treat principle. Descriptive statistics were calculated; continuous data are presented as the means ± standard deviation (range), and categorical data are given as the counts (percentage). Morbidity and mortality were determined at 30 days and cumulatively to 1 year. Lesion dimensions were evaluated over 3 intervals (0–1, 1–6, and 6–12 months) and cumulatively. Aneurysm shrinkage at each interval and cumulative lesion diameter reduction were analyzed per lesion using a 2-tailed paired *t* test. Differences in sac shrinkage according to aneurysm morphology subtypes and presence/absence of mural thrombus were evaluated using an analysis of variance. Kaplan-Meier estimates were generated for stent patency (primary and secondary), side branch patency, and freedom from all complications. $P<0.05$ was considered the threshold of statistical significance. Analyses were performed using OpenEpi, Open Source Epidemiologic Statistics for Public Health, version 2.3.1 (available at: *http://www.openepi.com/Menu/OpenEpiMenu.htm)*.

RESULTS

Technical success was achieved in all patients with no access-related complications. One (1.8%) patient died from pulmonary embolism at 24 hours and another 2 patients died from preexisting liver cancer within 1 year (5.5% 1-year mortality). One patient was lost to follow-up at 1 month and another 5 did not appear for the 12-month visit, leaving 45 patients at the 12-month analysis point.

Stent Patency

Six (11.1%) stents had occluded over the 1-year observation period. One common iliac artery (CIA) stent was occluded at 24 hours in a thrombophilic patient who underwent surgical aortoiliac bypass. One superior mesenteric artery (SMA) stent partially occluded at 48 hours owing to poor runoff (the hepatic artery was occluded at its origin); angiography confirmed that the device was patent from the origin of the vessel until below the takeoff of 2 collateral branches, one which fed the hepatic artery retrograde and the other supporting the bowel's vascular bed (Fig. 3). The patient had no symptoms of hepatic infarction or bowel ischemia. A splenic artery stent occluded after 2 weeks, likely due to poor compliance with the dual antiplatelet therapy reported by the patient, who did not experience splenic infarction or a hematologic or infectious complication. Angiography confirmed antegrade flow into the side branches.

At 2 months, 2 patients treated for pseudoaneurysms at the distal anastomosis of femoropopliteal bypass grafts experienced claudication. Duplex scans showed bypass and stent obstruction, probably due to poor

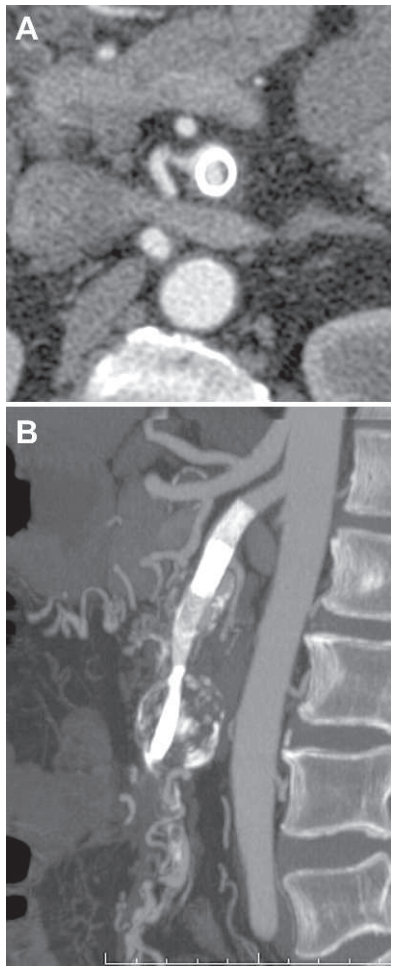

Figure 3 ◆ (**A**) An axial CT scan and (**B**) MIP reconstruction show partial obstruction of a CMFM deployed in a SMA aneurysm; the stent remained patent in front of the takeoff of 2 collateral branches, one going retrograde to the hepatic artery, which was occluded at its origin, and the other supporting the bowel's vascular bed.

runoff (only 1 vessel to the foot); both underwent thrombolysis and subsequent stent dilation with complete recovery. At 6 months, primary and secondary stent patency rates were 90.4% (47/52) and 94.2% (49/52), respectively.

At 1 year, a hepatic artery stent in a patient who spontaneously stopped antiplatelet therapy was occluded along its length, so flow was not antegrade but retrograde from collaterals. The aneurysm was not reperfused, and the side branch was patent (Fig. 4). Cumulative primary and secondary patency estimates by Kaplan-Meier analysis were 86.9% and 90.7% at 1 year (Table 2A,B).

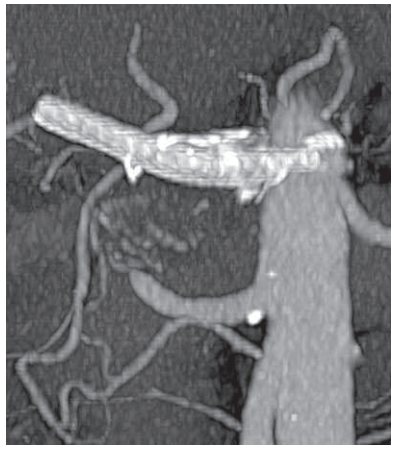

Figure 4 ◆ MIP reconstruction shows an obstructed CMFM in a hepatic artery with side branch patency.

Side Branch Patency

Forty-four of 54 patients had lesions with side branches, for a total of 53 collateral branches covered by the CMFM. At 1 month, branch patency was 98.1% (51/52 available branches) owing to the occluded CIA stent patient who underwent aortoiliac bypass that sacrificed the internal iliac artery. At 6 and 12 months, side branch patency was 98.1% (51/52 side branches in 42 patients) and 97.9% (48/49 side branches in 37 patients), respectively (Fig. 5). The cumulative side branch patency estimate by Kaplan-Meier analysis was 96.1% at 1 year (Table 2C).

Complications

The Kaplan-Meier estimate for freedom from all complications was 83% at 1 year (Table 2D). In addition to the 3 deaths and 6 stent occlusions noted above, 2 (3.8%) cases of stent foreshortening led to type III endoleak at 3 months: both patients (popliteal aneurysm and internal iliac artery aneurysm) underwent deployment of another CMFM with recovery of stent continuity.

Aneurysm Thrombosis

Complete aneurysm thrombosis was recorded in 45 (86.5%) of 52 patients at 1 month and 49 (94.2%) of 52 patients at 6 months. At the 6-month imaging follow-up, a SMA aneurysm (type II AX) was still patent but with increased mural thrombus and a renal artery aneurysm (type I A) and a CIA aneurysm (type II A) without mural thrombus had shrunk without complete thrombosis. At 1 year, the above 3 aneurysms were still not completely thrombosed, so complete aneurysm thrombosis was recorded in 42 (93.3%) of 45 patients.

Aneurysm Diameter Changes

Diameter variations at 1, 6, and 12 months were compared in 41 of the 45 patients who had adequate CT scans at all time points, representing 6 type I aneurysms, 2 type I A, 2 type II, 14 type II A, 10 type II X, and 7 type II AX lesions. The mean diameter of these 41 lesions at baseline was 34.12 ± 15.52 mm (range 9–80), which was reduced to 29.93 ± 14.86 mm (range 6–70) at 1 month ($p < 0.001$), 29.10 ± 14.96 mm (range 6–70) at 6 months ($p = 0.004$), and 26.88 ± 15.39 mm (range 6–63) at 1 year ($p < 0.001$). There was no aneurysm growth.

The percent shrinkage was 15.5% in 28 (68.3%) of the 41 aneurysms at 1 month, 3.1% in 6 (14.6%) lesions at 6 months, and 11.0% in 21 (51.2%) lesions at 12 months (Fig. 6A). Five (12.2%) of the 41 lesions did not change. The difference between shrinkage at the 3 time points was statistically significant ($p = 0.002$); however, the difference between shrinkage at

TABLE 2
Kaplan-Meier Estimates for Study Endpoints

Time, mo	At Risk	Events	Censored	Failure Rate	Interval Patency	Cumulative Patency, %	Standard Error, %
A. Primary Stent Patency							
1	54	4	1	0.074	0.926	92.6	3.43
2	49	0	0	0.000	1.000	92.6	3.43
3	49	2	0	0.041	0,959	88.8	4.24
4	47	0	0	0.000	1.000	88.8	4.33
5	47	0	0	0.000	1.000	88.8	4.33
6	47	0	0	0.000	1.000	88.8	4.33
7	47	0	0	0.000	1.000	88.8	4.33
8	47	0	0	0.000	1.000	88.8	4.33
9	47	0	0	0.000	1.000	88.8	4.33
10	47	0	0	0.000	1.000	88.8	4.33
11	47	0	0	0.000	1.000	88.8	4.33
12	47	1	7	0.023	0.977	86.9	4.60
13	39	0	0	0.000	1.000	86.9	5.05
B. Secondary Stent Patency							
1	54	4	1	0.074	0.926	92.6	3.43
2	49	0	0	0.000	1.000	92.6	3.60
3	49	0	0	0.000	1.000	92.6	3.60
4	49	0	0	0.000	1.000	92.6	3.60
5	49	0	0	0.000	1.000	92.6	3.60
6	49	0	0	0.000	1.000	92.6	3.60
7	49	0	0	0.000	1.000	92.6	3.60
8	49	0	0	0.000	1.000	92.6	3.60
9	49	0	0	0.000	1.000	92.6	3.60
10	49	0	0	0.000	1.000	92.6	3.60
11	49	0	0	0.000	1.000	92.6	3.60
12	49	1	7	0.022	0.978	90.7	3.98
13	41	0	0	0.000	1.000	90.7	4.35
C. Side Branch Patency							
1	53	0	1	0.000	1.000	100.0	0.00
2	52	1	0	0.019	0.980	98.1	2.60
3	51	0	0	0.000	1.000	98.1	2.65
4	51	0	0	0.000	1.000	98.1	2.65
5	51	0	0	0.000	1.000	98.1	2.65
6	51	0	0	0.000	1.000	98.1	2.65
7	51	0	0	0.000	1.000	98.1	2.65
8	51	0	0	0.000	1.000	98.1	2.65
9	51	0	0	0.000	1.000	98.1	2.65
10	51	0	0	0.000	1.000	98.1	2.65
11	51	0	2	0.000	1.000	98.1	2.65
12	49	1	7	0.020	0.979	96.1	2.89
13	41	0	0	0.000	1.000	96.1	2.89
D. Complications							
1	54	4	1	0.074	0.926	92.6	3.43
2	49	2	0	0.041	0.959	88.8	4.24
3	47	2	0	0.043	0.957	85.0	4.80
4	45	0	0	0.000	1.000	85.0	4.90
5	45	0	0	0.000	1.000	85.0	4.90
6	45	0	0	0.000	1.000	85.0	4.90
7	45	0	0	0.000	1.000	85.0	4.90
8	45	0	0	0.000	1.000	85.0	4.90
9	45	0	0	0.000	1.000	85.0	4.90
10	45	0	0	0.000	1.000	85.0	4.90
11	45	0	0	0.000	1.000	85.0	4.90
12	45	1	7	0.024	0.976	83.0	5.10
13	37	0	0	0.000	1.000	83.0	5.63

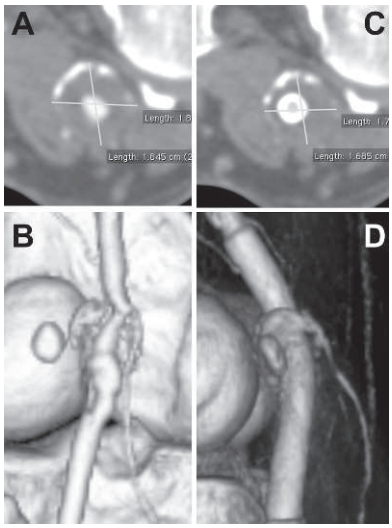

Figure 5 ◆ (**A**) An axial CT and (**B**) 3D reconstruction show a type II A popliteal aneurysm (1.9×1.8 cm) with mural thrombus and side branches from the sac. (**C,D**) Images at the same levels at 1 year show shrinkage of the treated lesion (1.7×1.7 cm), a patent CMFM, and patency of side branches arising from the sac.

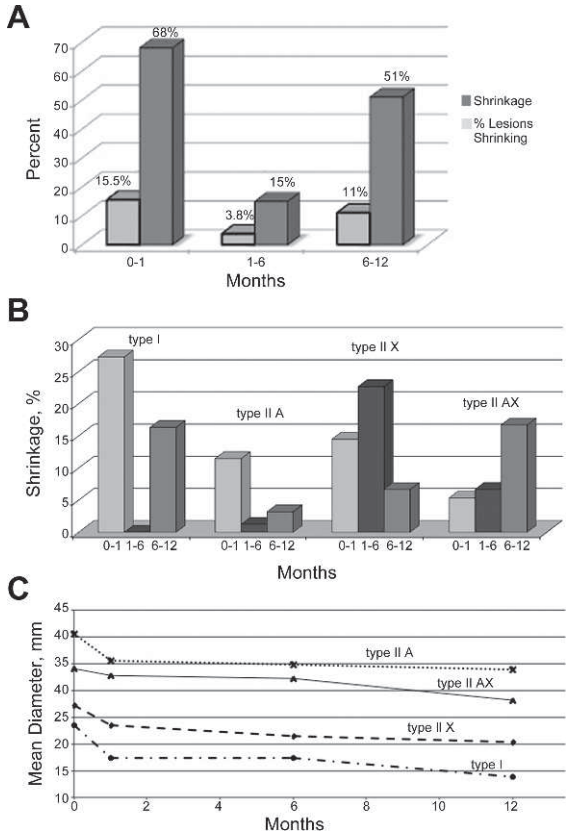

Figure 6 ◆ (**A**) Shrinkage rate at intervals for all lesion types and (**B**) percent shrinkage and (**C**) mean diameter according to the morphology classification. Cumulative reduction was statistically significant for all lesion types and varied by interval for each lesion type (see text).

the 0–1 interval vs. the 6–12 interval was not statistically significant (p=0.2).

Subgroup Analysis

Using the proposed aneurysm classification, the cumulative diameter reduction for aneurysm types with an incidence >10% in our series [37 (90.2%) of 41 lesions] was statistically significant for all lesion types at 12 months (Fig. 6B,C): 6 type I (p=0.02), 14 type II A (p<0.001), 10 type II X (p=0.019), and 7 type II AX (p=0.036).

For type I lesions (Fig. 7), diameter reduction was statistically significant only during the 0–1 month interval (p=0.03), while type II A lesions shrunk significantly between 0–1 month (p<0.001) and 6–12 months (p=0.013). Type II X aneurysm shrinkage was slow during the first 6 months, but it became statistically significant between 6 and 12 months (p=0.018). Type II AX lesions showed slow shrinkage during all time periods, and only the cumulative reduction was significant (p=0.036, as reported above).

Comparison of cumulative shrinkage at 12 months showed a significant difference between type I saccular lesions and all type II fusiform aneurysms (p=0.003), while differences among the 3 type II morphological subtypes (A, X, and AX) were not significant.

Changes in aneurysm size according to the presence (31 of 41 lesions) or absence of mural thrombus were compared (Fig. 8). In lesions with mural thrombus, shrinkage was statistically significant during the first month (p<0.001), between 6 and 12 months (p<0.001), and at 12 months (p<0.001), while in lesions without mural thrombus, shrinkage was statistically significant just during the first month (p=0.014) and at 12 months (p=0.007).

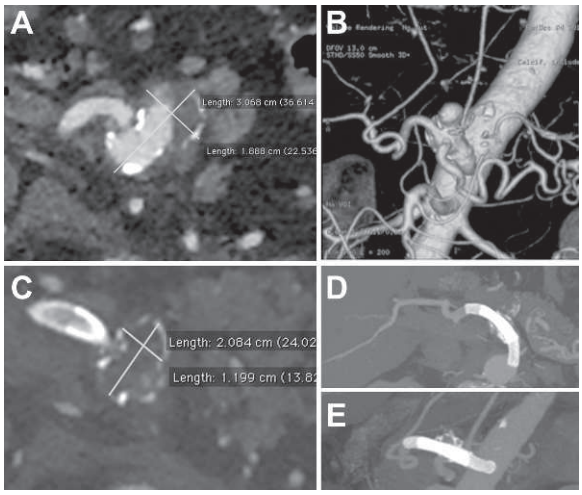

Figure 7 ◆ (**A,B**) CT images show a type I A celiac trunk aneurysm (3.06×1.88 cm) with mural thrombus and 3 side branches (hepatic, splenic, and left gastric) arising from the sac. (**C–E**) At 1-year follow-up, the treated lesion has shrunk (2.08×1.19 cm) while the CMFM and side branches remain patent.

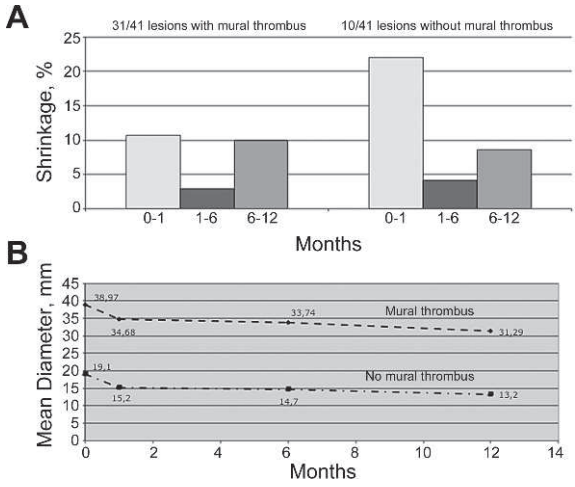

Figure 8 ◆ (**A**) Percent shrinkage and (**B**) mean diameter according to presence/absence of mural thrombus over time.

The presence of mural thrombus did not influence the time course of shrinkage (84% power).

DISCUSSION

The natural history of peripheral and visceral aneurysm is enlargement and rupture. These lesions are usually asymptomatic, and a significant number are discovered only when the patient becomes symptomatic (pain, distal embolization in PAA).

There is no consensus on the guidelines for PAA/VAA treatment, although rapid growth, symptoms, and identification in women of childbearing age seem to support a therapeutic approach. Open repair (ligation, aneurysmectomy, venous or synthetic grafting) was considered the gold standard. However, in the last decade, endovascular therapy (embolization and stent-graft placement) has offered a reliable option thanks to its low invasiveness, morbidity, and mortality, particularly in comorbid patients considered at high risk, even though some concerns still exist regarding flow restoration to the aneurysm after failed percutaneous therapy.[21]

The basic techniques for percutaneous aneurysm treatment are exclusion of the lesion from the circulation by packing the sac with coils or using covered stents as mechanical barriers. Unfortunately, these techniques have some limitations: coil deployment is not possible in wide-necked or complex lesions, and even when possible, this method causes occlusion not only of the aneurysm, but also of the target artery and parent vessel. On the other hand, stent-grafts are difficult to deploy in tortuous vessels. They also obstruct side branches and disrupt vital collaterals, even though they preserve patency of the target artery.

The 1-year analysis of registry data on the CMFM indicates that this is a safe and effective alternative for endovascular treatment of PAA and VAA in patients unsuitable for open repair, particularly those lesions in which it is better to maintain patency of both the target artery and side branches. The CMFM is not a mechanical barrier as is a conventional stent-graft, but a sort of "dynamic" barrier, a bare structure acting as a flow modulator reducing up to 90% of the flow velocity within the aneurysm while improving laminar flow in the main artery and side branches. These effects lead to local pressure and stress reduction on the aneurysm wall, flow stasis, and formation of an organized thrombus. The device minimizes the chance of lesion rupture while maintaining perfusion of side branches that are overstented. Due to

these characteristics, CMFMs cannot be used to treat ruptured aneurysm because thrombosis does not occur immediately as with stent-graft coverage.

In the literature, successful treatment of PAA/VAA with the CMFM has been reported in a variety of arteries (2 renal artery aneurysms,[22,23] 3 hepatic artery lesions,[24,25] a celiac trunk aneurysm,[26] and our 6-month registry results in 19 VAAs[8]). We have now expanded our analysis to evaluate the CMFM in PAA lesions as well and reported 1-year results for the aggregate group. In the registry's 54 cases, technical success was universal, with only a single perioperative death not related to the device. The relatively few complications at 1 year support the safety of the device in these lesions, while the high rate of aneurysm thrombosis speaks to the efficacy of the device in reducing the risk of rupture. Primary (87%) and secondary (91%) stent patency at 12 months was higher than reported for stent-graft treatment of peripheral aneurysms[27–29] and similar to rates obtained in VAA using conventional endovascular exclusion.[2,30–32] CMFM obstruction occurred in vessels with poor runoff or patients who reported poor compliance with the therapeutic protocol, the latter suggesting that dual antiplatelet therapy is mandatory in all these patients. Adequate distal runoff must be guaranteed with the CMFM as for any other device.

As a technical note, the 2 cases of CMFM foreshortening with consequent type III endoleak due to inadequate overlapping of dual stents taught us that the overlap should be at least of 3 cm to avoid this complication.

The excellent side branch patency confirmed that the CMFM can be safely deployed over collateral branches; even in the event of stent obstruction, side branches arising from the treated artery segment remain patent.

In our series we had no evidence of aneurysm growth or delayed aneurysm rupture; conversely, aneurysm diameter reduction was observed at all observations points and was statistically significant per interval and cumulatively. Aneurysms tended to decrease in dimension particularly during the first month and in the 6–12 month interval, while the 1–6 month interval appeared to be a "quiescent" or "remodeling" period before subsequent reduction in size.

Analysis of shrinkage according to sac morphology showed that anatomical characteristics can influence shrinkage over time: saccular aneurysms shrink mainly from the first month, while fusiform aneurysms with side branches tend to reduce later and more gradually. In particular, among fusiform aneurysms, those with branches from the sac shrink faster than those with branches from the neck, while lesions with side branches from both the sac and neck tend to reduce their dimension slowly and, in most of cases, not before 1 year. In sum, complex aneurysms take longer to reduce than simple lesions. All these data suggest that our proposed aneurysm morphology classification is not arbitrary but reflects clinical evidence of different shrinkage timing after CMFM deployment. While aneurysm morphology can influence shrinkage over time, our data showed that mural thrombus has no influence on dimension reduction.

Limitations

This was a voluntary registry enrolling patients with a heterogeneous array of lesions. The voluntary participation of clinical centers notably reduced the number of patients involved. In the time frame that we collected data from 54 patients, more than 100 patients underwent CMFM implantation in Italy. Thus, significant selection bias may have occurred in the decision to enroll patients at each of the clinical sites.

As to the diversity of treated lesions, we do not consider this aspect a real limit because the device was designed to allow treatment of peripheral and visceral aneurysms with or without side branches regardless of where the lesions were located. Due to the design limitations of the CMFM (it is not a mechanical barrier), only ruptured aneurysms were contraindicated.

Conclusion

Although further follow-up is needed to evaluate the durability of the CMFM over the long term, 1-year data from this registry show

efficacy of the CMFM in the treatment of PAA and VAA in terms of freedom from aneurysm rupture/reperfusion, patency of the stents and side branches, complete aneurysm thrombosis, and acceptable freedom from morbidity and mortality. Sac shrinkage during follow-up and absence of aneurysm growth or rupture demonstrate the effectiveness of this stent design.

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APPENDIX

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