Reinterventions for stent restenosis in patients treated for atherosclerotic mesenteric artery disease

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Objective: **Mesenteric artery angioplasty and stenting (MAS) has been plagued by high restenosis and reintervention rates. The purpose of this study was to review the outcomes of patients treated for mesenteric artery in-stent restenosis (MAISR).**

Methods: **The clinical data of 157 patients treated for chronic mesenteric ischemia with MAS of 170 vessels was entered into a prospective database (1998-2010). Fifty-seven patients (36%) developed MAISR after a mean follow-up of 29 months, defined by duplex ultrasound peak systolic velocity >330 cm/s and angiographic stenosis >60%. We reviewed the clinical data, radiologic studies, and outcomes of patients who underwent reintervention for restenosis. End points were mortality and morbidity, patient survival, symptom recurrence, reintervention, and patency rates.**

Results: **There were 30 patients (25 female and five male; mean age, 69 ± 14 years) treated with reintervention for MAISR. Twenty-four patients presented with recurrent symptoms (21 chronic, three acute), and six had asymptomatic preocclusive lesions. Twenty-six patients (87%) underwent redo endovascular revascularization (rER) with stent placement in 17 (13 bare metal and four covered) or percutaneous transluminal angioplasty (PTA) in nine. The other four patients (13%) had open bypass, one for acute ischemia. There was one death (3%) in a patient treated with redo stenting for acute mesenteric ischemia. Seven patients (27%) treated by rER developed complications, including access site problems in four patients, and distal embolization with bowel ischemia, congestive heart failure and stent thrombosis in one each. Symptom improvement was noted in 22 of the 24 symptomatic patients (92%). After a mean follow-up of 29 ± 12 months, 15 patients (50%) developed a second restenosis, and seven (23%) required other reintervention. Rates of symptom recurrence, restenosis, and reinterventions were 0/4, 0/4, and 0/4 for covered stents, 2/9, 3/9, and 2/9 for PTA, 5/13, 8/13, and 5/13 for bare metal stents, and 1/4, 4/4, and 0/4 for open bypass. For all patients, freedom from recurrent symptoms, restenosis, and reinterventions were 70% ± 10%, 60 ± 10%, and 50 ± 10% at 2 years. For patients treated by rER, secondary patency rates were 72 ± 12 at the same interval.**

Conclusions: **Nearly 40% of patients developed mesenteric artery in-stent restenosis, of which half required reintervention because of symptom recurrence or progression to an asymptomatic preocclusive lesion. Mesenteric reinterventions were associated with low mortality (3%), high complication rate (27%), and excellent symptom improvement (92%). (J Vasc Surg 2011;54:1422-9.)**

Over the past decade, mesenteric artery stenting (MAS) has surpassed open bypass as the most frequently utilized method of revascularization to treat chronic mesenteric ischemia (CMI).[1] Several centers have adopted an endovascular-first approach, relegating open revascularization (OR) to patients who failed stenting or have anatomy unsuitable for it.[1] In a recent review of national outcomes, Schemerhorn and associates[1] reported a sevenfold increase in the number of mesenteric interventions since 1988 and a remarkable reduction in mortality from 15% with open bypass to 4% with endovascular treatment. However, several studies have demonstrated that mesenteric stents are less durable when compared with OR, with higher rates of restenosis in the range of 20% to 66%.[2-5] Nevertheless, despite these higher restenoses rates, clinical data on outcomes of mesenteric reinterventions remain scarce.

Treatment is recommended in patients who develop symptoms associated with restenoses, but the type of reintervention is individualized considering physician preference, patient comorbidities, and anatomical characteristics of the lesion. Most often, a secondary endovascular procedure is performed using either primary angioplasty or stent placement. Alternatively, a mesenteric bypass provides a durable option and can be performed with low mortality rates in good-risk patients, particularly in those with fast-recurring lesions, occlusions, or unfavorable anatomy. The aim of this study was to review treatment strategies and

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outcomes of patients treated for mesenteric artery in-stent restenosis (MAISR).

METHODS

The study was approved by the Institutional Review Board of the Mayo Clinic. The clinical data of patients who underwent an index MAS procedure for CMI between 1998 and 2010 were entered into a prospectively maintained database. We identified all patients who had follow-up imaging studies of the treated vessel and were diagnosed and treated for a MAISR. We excluded from the study patients with mesenteric artery vasculitis, median arcuate ligament syndrome, or those who had hybrid retrograde MAS or reinterventions after failed mesenteric bypass or endarterectomy.

Demographics, clinical characteristics, imaging, and operative data were obtained from the medical records. Operative risk was assessed using Society for Vascular Surgery (SVS) clinical scores.[6] Patients were classified into a high- or low-risk category as previously validated by our group. High-risk was defined by SVS class 3 cardiac, pulmonary, renal comorbidity or age >80 years old.[7] MAISR was defined by duplex ultrasound (DUS) peak systolic velocity (PSV) >330 cm/s and/or evidence of in-stent stenosis >60% by computed tomography angiography (CTA), magnetic resonance angiography (MRA), or conventional biplane mesenteric angiography.[8,9]

Mesenteric reinterventions were indicated for recurrent symptoms associated with angiographic evidence of MAISR. Patients with asymptomatic restenoses were followed with repeat DUS. A subset of patients with asymptomatic restenosis was offered a reintervention if the lesion progressed (PSV > 500 cm/s and distal tardus-parvus waveform) and was associated with poor collateral network because of occlusion of the other two mesenteric vessels. The type of reintervention was left at the discretion of the physician performing the procedure. There was no predefined algorithm, but most often, a second endovascular intervention included standard balloon angioplasty (percutaneous transluminal angioplasty [PTA]), cutting-balloon angioplasty (cPTA), or placement of bare metal or covered stent. Mesenteric bypass was indicated in patients who had acute mesenteric ischemia and needed a laparotomy because of peritoneal signs, or in patients who had stent occlusions or long calcified lesions.

The index angiography (first angiography at time of stent placement) and available pre- and postprocedure imaging studies were reviewed by a blinded investigator to determine anatomical characteristics of the index lesion and to correlate these findings with the location of the restenosis. CTA with centerline of flow was analyzed for vessel diameter and length measurements. Target vessel calcification was graded as mild (none or trivial), moderate, or severe (circumferential napkin-ring or >60% eccentric plaque). Conventional completion angiography was reviewed to determine technical success, which was defined by <30% residual stenosis, and presence of other technical imperfections, such as partial stent compressions, missed or tandem lesions. Follow-up consisted of clinical examination and DUS prior to dismissal, every 6 months during the first year, and annually thereafter. CTA or MRA was used in patients who had technically limited DUS or evidence of recurrent stenosis. Medical therapy was clopidogrel for 6 to 8 weeks after an endovascular intervention, followed by aspirin indefinitely; for patients treated by OR, medical therapy was aspirin indefinitely.

Statistical analysis. Data were analyzed using SVS reporting standards. End points were procedure-related mortality and morbidity, and late patient survival, symptom recurrence, reintervention, and patency rates. Time-dependent outcomes were analyzed using Kaplan-Meier estimates, and differences were determined by the log-rank test. The Pearson χ^2 or Fisher exact test was used for analysis of categoric variables. Differences between means were tested with two-sided t test, the Wilcoxon rank-sum test, or the Mann-Whitney test. A value of $P < .05$ was used to determine statistical significance.

RESULTS

Study population

One hundred fifty-seven patients (103 female and 54 male) were treated for CMI with MAS of 170 mesenteric arteries. From this group, 145 patients (92%) had at least one follow-up imaging study of the stented vessel and 57 patients (39%) were diagnosed with MAISR. We included in the study 30 patients (21%) who underwent reinterventions for MAISR.

Clinical characteristics

There were 25 female and five male patients with mean age of 69 ± 14 years. Patients presented at an average of 12 months (range, 1-33) after the index procedure, which included 21 superior mesenteric artery (SMA) and 11 celiac axis (CA) stents, and three SMA angioplasties. Twenty-four patients developed recurrent symptoms (Table I), which were chronic in 21. Three patients had acute presentation, including two patients who had chronic abdominal pain but did not return for follow-up until they developed acute symptoms, and one who had no evidence of in-stent restenosis by ultrasound 6 months prior. Cardiovascular risk factors included hypertension in 24 patients (80%), hyperlipidemia in 20 (67%), and coronary artery disease (CAD), or tobacco use in 19 patients each (63%).

Diagnostic imaging and anatomical characteristics

Index angiography. Angiographic studies performed prior to the first stent placement were analyzed in all patients, including 26 conventional angiographies, 14 CTAs, and six MRAs (Table II, online only). The index lesion was ostial in 17 vessels and nonostial in 15. Five patients had tandem lesions in the SMA, which were not treated. The target lesion length measured 16.2 ± 9.4 mm and the reference vessel diameter was 6.3 ± 1.6 mm. Seven lesions were severely calcified (27%), eight occurred in small vessels (<6 mm), and three were longer than 30 mm (76, 40, and 32 mm).

Table I. Clinical characteristics of 57 patients who developed restenosis

	Restenoses				
	Requiring intervention		*Not requiring intervention*		
Variables	*n = 30*	*%*	*n = 27*	*%*	P *value*
Demographics					
Mean age ± standard deviation (years)	69 ± 14		78 ± 7		.01
Female gender	25	83	18	67	.6
Clinical presentation					
Abdominal pain	24	80	0	0	<.001
Weight loss	16	55	0	0	<.001
Nausea and/or vomiting	10	35	0	0	.004
Food fear	10	35	0	0	.004
Cardiovascular risk factors					
Hypertension	24	80	25	93	.7
Hyperlipidemia	20	67	21	78	.7
Coronary artery disease	19	63	21	78	.6
Tobacco abuse	19	63	15	56	.8
Peripheral artery disease	15	50	14	52	.9
Prior myocardial infarction	14	47	13	48	.9
Chronic obstructive pulmonary disease	9	30	13	48	.3
Cerebrovascular disease	8	27	8	30	.8
Chronic renal insufficiency[a]	7	23	4	15	.5
Society for Vascular Surgery comorbidity score	9.5 ± 6.0		11.5 ± 4.8		
Medical therapy					
Acetylsalicylic acid	24	80	13	48	.2
Clopidogrel	23	77	8	30	.05
Statins	17	57	11	41	.5
Beta-blocker	17	57	13	48	.7
ACE inhibitor	14	47	15	56	.7
Diuretic	13	43	5	18	.1
Warfarin	4	13	1	4	.2

[a]Serum creatinine ≥ 1.5 mg/dL.

Stent length averaged 17.1 ± 6.8 mm. Among 12 patients who had comparative length measurements using centerline of flow, stents were adequate in length in eight patients (longer than the lesion) but short in four (33%). Analysis of the completion angiography showed <30% residual stenosis in 20 patients (66%) and no residual lesion in five (17%). Five patients (17%) had >30% residual stenosis. Other technical imperfections were noted in 15 patients (50%), including missed lesion because of short stent length in 11 (eight proximal and three distal) and partial stent compression in seven (five SMA and two CA).

Ultrasound surveillance. Sixteen patients had a total of 64 DUS studies reviewed (Fig 1). The PSV increased from 307 ± 140 cm/s from the initial study, which was obtained within the first 3 months, to 440 ± 81 cm/s prior to the reintervention. After treatment of the restenosis, PSV averaged 319 ± 151 cm/s, remaining elevated (>330 cm/s) in three patients despite a completion angiography demonstrating no evidence of residual stenosis.

Reintervention angiography. Imaging studies performed prior to the second intervention were analyzed in all patients, including 30 conventional angiographies and 13 CTAs. Of the 13 CTAs, 11 showed high-grade and two demonstrated moderate restenosis. Conventional angiography confirmed high-grade restenosis in all patients, including the six patients with asymptomatic lesion by duplex US. The location of the restenosis (Fig 2, online only) was proximal to the stent in 11 patients (10 ostial and one nonostial), within the stent in nine, and distal to the stent in six. The five tandem lesions noted at the index angiography remained stable. Comparative analysis of the index and the reintervention angiography demonstrated that in 13 patients (43%) the location of the restenosis coincided with a technical imperfection, including 11 missed lesions and four partial stent compressions. These technical imperfections were subtle findings and were associated with residual stenosis >30% in five patients.

Mesenteric reinterventions. Restenoses were treated by redo endovascular revascularization (rER) in 26 patients (87%) and OR in four (13%). Redo endovascular procedures were performed using a femoral approach in 15 patients or branchial approach in 11. Stents were placed in 17 patients (57%), including 13 patients treated with bare metal stents and four treated with covered stents (Fig 3, Icast stents; Atrium Medical Corporation, Hudson, NH). Primary angioplasty was performed using standard balloons in eight patients (27%) and a cutting balloon in one (3%).

Four patients (13%) underwent open surgical bypass (Fig 4), including three patients who had unfavorable lesions because of long (>30 mm) calcified stenosis or stent

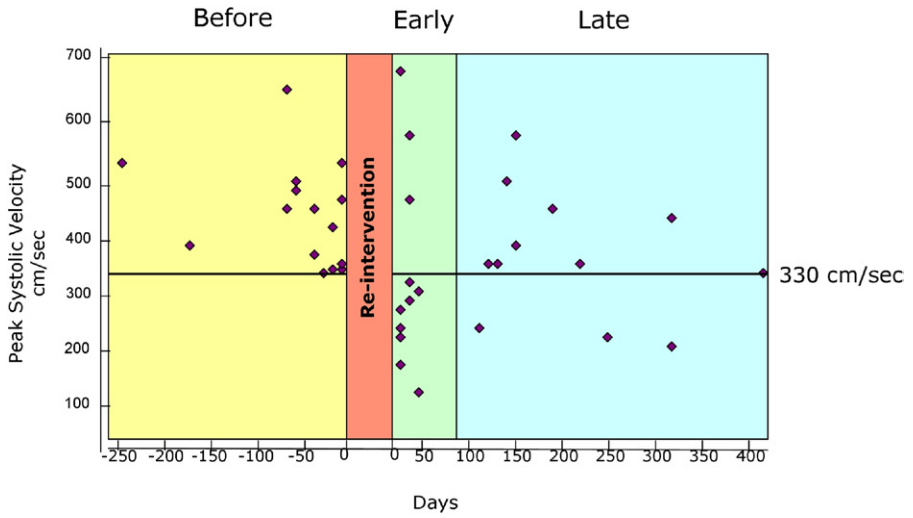

Fig 1. Duplex ultrasound peak systolic velocity (PSV) measurements obtained prior to the first reintervention (*yellow box*) and in the early (*green box*) and late (*blue box*) follow-up after treatment of a mesenteric in-stent restenosis. Note that in a few patients, the PSV remained elevated above our threshold of >330 cm/s.

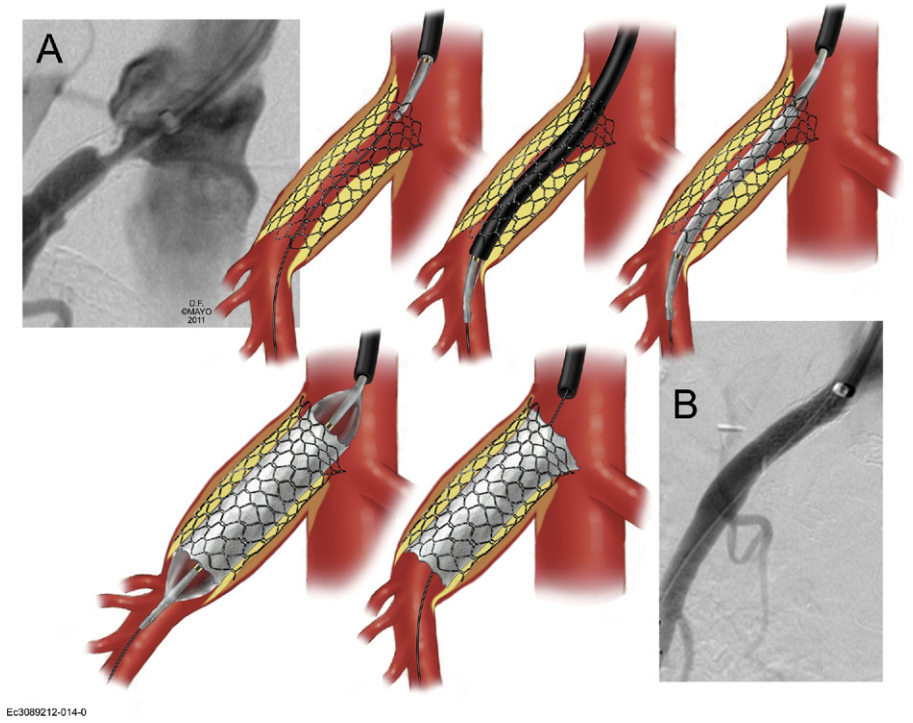

Fig 3. Technique of redo stenting using a covered stent. After diagnostic angiography, which depicted the in-stent stenosis **(A),** the lesion was crossed with a 0.035-inch wire system and a 7F sheath was advanced across the area of stenosis. The covered stent was positioned under protection of the sheath to avoid stent dislodgement. The stent was expanded to profile and a completion angiography confirmed no residual stenosis **(B)**.

occlusion. The other patient was operated emergently because of acute mesenteric ischemia and bowel gangrene. A midline transperitoneal approach was used in all patients. Mesenteric reconstruction was performed using an antegrade supraceliac aortic bypass in three patients (two bifurcated celiac and SMA, and one SMA) or a retrograde iliac artery to SMA bypass in one patient. A polyester graft was used for all reconstructions, including the patient with

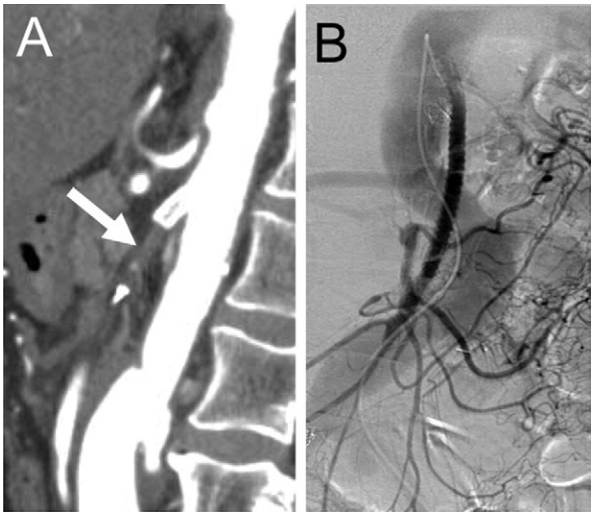

Fig 4. Open reconstruction of a long superior mesenteric artery (SMA) occlusion **(A)** using a supraceliac aorta to SMA bypass **(B)**.

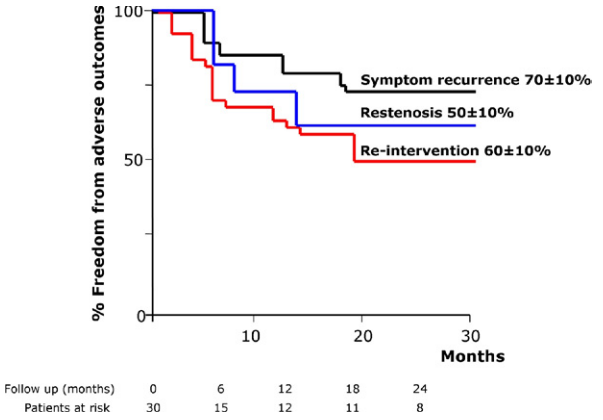

Fig 5. Kaplan-Meier estimates of freedom from symptom recurrence (*black*), reintervention (*blue*), and restenosis (*red*) at 2 years.

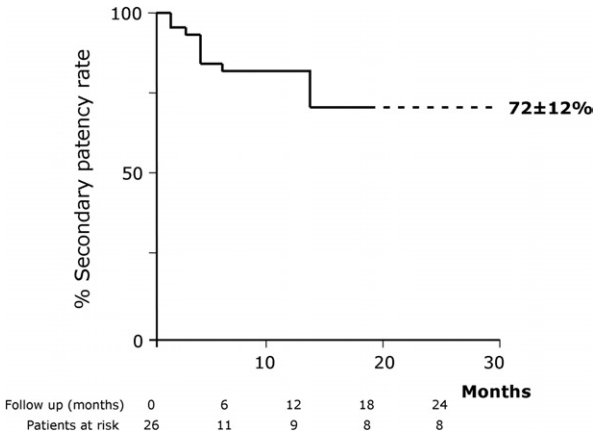

Fig 6. Kaplan-Meier estimates of secondary patency rates at 2 years for 26 patients treated with redo endovascular interventions for mesenteric in-stent restenosis.

acute ischemia who required a Rifampin-soaked graft with omental wrap.

Early outcomes. There was one procedure-related death (3%). The patient was an 80-year-old high-risk female with acute mesenteric ischemia treated by SMA stent placement, which was complicated by distal embolization. Despite emergent exploration and bowel resection, the patient developed multisystem organ failure and died 57 days later. Seven patients (27%) developed complications after endovascular reinterventions, including four puncture site complications (two brachial, two femoral) and three major complications (SVS grade 3). These included the distal embolization described above, stent thrombosis and decompensated congestive heart failure, in one patient each. There were no complications among patients treated by OR.

Rates of complications were 29% (4/14) for high-risk and 19% (3/16) for low-risk patients. Endovascular reinterventions resulted in decreased length of stay in the intensive care unit (0.3 ± 1.4 vs 3.7 ± 5.5 days; $P < .008$) and in the hospital (1.8 ± 2.0 vs 8.2 ± 5.4 days; $P < .007$) compared with open bypass. Hospital stay was significantly less (2.3 ± 2.4 vs 6.0 ± 8.7 days; $P = .001$) among patients who had an elective procedure compared with those who had reinterventions performed emergently.

Late outcomes. Twenty-nine patients were followed for an average of 29 ± 12 months (range, 1-101 months). Symptom improvement was noted in 22 of the 24 symptomatic patients (92%). One patient referred persistent symptoms despite successful rER. Fifteen patients (50%) developed a second restenosis and seven (23%) required additional reinterventions, all indicated for recurrent symptoms. Time for the second reintervention averaged 17 ± 19 months. Five patients were treated by SMA angioplasty, which was combined with celiac stent in one. Two patients underwent supraceliac aorta to common hepatic and SMA bypass.

Rates of symptom recurrence, restenosis and reinterventions were 0% (0/4), 0% (0/4), and 0% (0/4) for covered stents, 22% (2/9), 33% (3/9), and 22% (2/9) for PTA and 38% (5/13), 62% (8/13), and 38% (5/13) for bare metal stents (P = NS). The patient treated by cPTA developed restenosis at 3 months. All four patients treated by OR had PSV >330 cm/s following revascularization, but only one had angiographic confirmation of a significant restenosis. One patient developed atypical abdominal pain, but treatment was not recommended because the symptom was not consistent with mesenteric ischemia. At 2 years, freedom from recurrent symptoms, restenosis, and reinterventions were 70% ± 10%, 60 ± 10%, and 50 ± 10% for all patients (Fig 5). The secondary patency rate of rER was 72% ± 12% at the same interval (Fig 6).

DISCUSSION

Mesenteric artery stenting has surpassed open surgical bypass as the preferred method of treatment for chronic mesenteric ischemia in the United States and at most tertiary care centers. Decisions about the type of revascularization have shifted from patients' clinical risk (eg, endovascular for high-risk and open bypass for low-risk), to where the anatomical characteristics of the lesion are equally or more important. Mesenteric stenting has become the preferred treatment for patients with focal, noncalcified stenosis, whereas open bypass is considered for good-risk patients with mesenteric occlusions or long calcified lesions. However, these changes in treatment paradigm have occurred without a prospective comparison of both treatment methods. A recent systematic review of the best available evidence (level IIa, b) indicates that mesenteric stenting decreases morbidity and hospital stay, has similar mortality rate and clinical efficacy, but results in more restenoses, symptom recurrences, and reinterventions compared with open bypass.[10]

The incidence of mesenteric restenosis has varied widely, from 20% to 66% in prior reports.[2-5,11] Possible explanations for this variability include the lack of reporting standards, the inconsistent use of imaging studies during follow-up, and the absence of an ultrasound criterion validated for in-stent restenosis. Velocity measures used to diagnose native artery stenosis (>200 cm/s for celiac and >275 cm/s for SMA) may overestimate the presence of restenosis after stent placement. Peck and associates[2] found restenosis in 40% of their patients treated by SMA stenting using the native artery criterion, while the Oregon group[8] found a mean PSV of 336 ± 45 cm/s immediately after successful stent placement. In a review of ultrasound surveillance studies after SMA stent placement, Stout and associates[9] reported that a PSV >320 cm/s carries a sensitivity of 100% and a specificity of 50% to diagnose an angiographically proven restenosis >60%. However, this had a positive predictive value of only 23%, with a negative predictive value of 100%. Recognizing these limitations, we have arbitrarily selected a PSV > 330 cm/s to diagnose restenosis by ultrasound and found restenosis in 40% of our patients, which is similar to the rates reported by Fioole[4] (47%) and others.[2-4]

The ability to predict and identify which patients or lesions are more prone to restenosis would be helpful and could optimize treatment selection. Unfortunately, chronic mesenteric ischemia is relatively uncommon and most reports have a small number of patients or incomplete follow-up, which does not allow a meaningful analysis of predictive factors for restenosis. Our prior review of 102 patients treated by mesenteric stents identified higher rates of restenosis among women, and those treated for occlusions, long (>30 mm) or severely calcified lesions and small vessels (<6 mm).[12] Of these factors, occlusions and long lesions were independently associated with restenosis. In this study, we identified one of these features in 13 of our patients (43%) treated for restenosis, including eight patients with vessel diameter <6 mm, seven with severely calcified lesions, and three with lesions >30 mm. In addition, we attempted to describe other anatomical predictors by analyzing the final technical result after the first stent was placed. Interestingly, 43% of our patients developed restenosis in the same location of a technical imperfection, most often a missed lesion proximal or distal to the stent edge, which was considered minor or subtle at the time of the initial procedure. Some of these imperfections may be prevented, such as selection of adequate stent length using preprocedure cross-sectional imaging, deployment of the stent within the entire lesion, and flaring the stent in the aortic origin to avoid residual stenosis. Whereas careful planning of these procedures using length measurements may improve results, some imperfections may not be improved, such as residual stenosis in patients with densely calcified lesions.

There are no guidelines on how to treat mesenteric in-stent restenosis. Most physicians rely on anecdotal experiences and techniques applied for restenoses in other vascular territories. Mesenteric reintervention is indicated for recurrent symptoms. In contrast, prophylactic revascularization of an asymptomatic restenosis generally is not advised, a recommendation that is based on two prospective cohort studies which demonstrated an exceedingly low risk of symptom progression in patients with asymptomatic single-vessel disease, therefore not justifying the risks of revascularization.[13,14] However, an older report by Thomas and associates[14] documented symptom progression in four of their 15 patients with severe three-vessel disease. Although we reserve reinterventions for patients with recurrent symptoms, we intervened in patients with asymptomatic preocclusive lesions associated with poor collateral network between the visceral beds.

A variety of strategies have been used to treat in-stent restenoses. In the renal territory, a prospective comparison of angioplasty and stent placement found no differences in outcome.[15] Others have suggested that placement of a second stent could be deleterious because of excessive stretching of the arterial wall, which could further stimulate development of neointimal hyperplasia.[16] Drug-eluting stents (DES) may prove useful by blocking cellular proliferation and reducing intimal hyperplasia in the treated vessel. The favorable results of DES for native coronary[17] and renal[15] artery stenosis encouraged its use to treat restenosis, but the stent diameters are small (<5 mm) for the mesenteric arteries. While several authors report low incidences of recurrent stenosis after treatment of coronary in-stent restenosis, Stone and associates found no benefit with use of DES to treat renal in-stent restenoses when compared with bare metal stents.[18-20]

Cutting balloon angioplasty has been used for restenosis of coronary bypasses and applied with success for infrainguinal vein graft restenosis. However, its use for in-stent restenosis is anecdotal. Cutting balloons employ microsurgical blades that cut into the lesion, a strategy that reduces recoil and may impact formation of neointimal hyperplasia by inducing a controlled dissection. However, inside a

stent, these blades cut the neointima up to the metal stent cage, which protects the arterial wall. Therefore, its utility for renal and coronary artery in-stent restenoses is limited.[20-22] We used cutting balloon angioplasty in one patient, but this resulted in a recurrent lesion.

A promising alternative is the use of covered stents. In our review of angiographic studies, restenoses affected the stented segment or the proximal and distal edges of the stent, indicating that these lesions are consistent with neointimal hyperplasia and typically do not involve jejunal branches. The use of covered stents may retard the process of neointimal hyperplasia by acting as a physical barrier.[23] Haskal and associates[24] reported a prospective randomized comparison of covered stents and angioplasty alone to treat restenoses of arteriovenous grafts. Covered stents were associated with restenosis and patency rates at 6 months of 28% and 51%, vs 78% and 23% for angioplasty alone. Other authors have reported superior patency rates for covered stents compared with bare metal stents for renal artery restenosis or in the setting of fenestrated renal endografts.[25,26] Recently, Erdoes and associates[27] reviewed 108 patients with chronic mesenteric ischemia treated by covered or bare metal stents. Primary patency at 1 year was 86% for covered stents and 34% for bare metal stents. In addition, of the nine patients who had covered stents used to treat restenosis, only one celiac stent occluded because of mechanical compression by the median arcuate ligament. We used covered stents in only four of our patients (Fig 3), none of whom have developed recurrent stenosis or symptoms at an average follow-up of 18 months. There are some limitations of covered stents using the current platform. These stents are four- to fivefold more expensive than bare metal stents, have the disadvantages of 0.035-inch system, need for larger introducer sheath, and carry a risk of stent dislodgment or inadvertent coverage of side branches. Improvements in covered stent technology and growing evidence on its superior patency rates compared with bare metal stents support the use of covered stents as the primary stent for native artery stenosis, potentially reducing reintervention rates.

This study has several limitations that deserve comment. We were not able to analyze factors that may have affected decision making on indications, choice of intervention, or number of vessels treated, because of the retrospective design and lack of treatment algorithm. We acknowledge that the ultrasound criterion used in this study (PSV > 330 cm/s) was arbitrary and has not been validated for mesenteric stents. The small number of patients treated by a variety of techniques has limited our ability to compare outcomes for different methods of treatment. And finally, our reintervention for silent asymptomatic lesions cannot be a general recommendation. Nonetheless, the strength and novelty of the study are the anatomic correlations and outcomes provided for reinterventions for mesenteric in-stent restenoses.

In summary, mesenteric artery in-stent restenosis occurred in 40% of our patients treated for chronic mesenteric ischemia, of which half developed recurrent symptoms and three presented with acute mesenteric ischemia. This study shows that reinterventions carry an overall mortality of 3%, albeit with no deaths among patients treated electively for chronic symptoms or progressive asymptomatic lesions. Reinterventions effectively relieved symptoms in 92% of the patients. The ideal endovascular strategy cannot be recommended on the basis of this report, but use of covered stents may prove beneficial for most in-stent restenosis. Primary angioplasty or bare metal stents may be preferred for lesions that extent into side branches. Finally, whereas endovascular intervention remains our first option to treat mesenteric in-stent restenosis, open reconstruction should be considered for good-risk patients who develop stent occlusions, fast recurring lesions or have long calcified lesions.

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AUTHOR CONTRIBUTIONS

Conception and design: TT, GO
Analysis and interpretation: TT, GO
Data collection: TT, GO
Writing the article: TT, GO, TM, PG, TB
Critical revision of the article: TT, GO, TM, PG, SM, AD, MK, TB
Final approval of the article: TT, GO, TM, PG, SM, AD, MK, TB
Statistical analysis: TT, GO
Obtained funding: GO
Overall responsibility: GO

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Table II, online only. Anatomical and procedural characteristics of the index procedure in 57 patients who developed restenosis

	Restenoses				
	Requiring intervention		*Not requiring intervention*		
Variables	*n = 30*	*%*	*n = 27*	*%*	P
Vessel treated[a]					
Superior mesenteric artery	24	75	20	74	.8
Celiac axis	11	25	10	37	.9
Inferior mesenteric artery	0	0	2	7	.2
Lesion characteristics[b]					
Length (mm)	16.2 ± 9.4		18.5 ± 8.2		.2
Target vessel diameter (mm)	6.3 ± 1.6		7.2 ± 1.8		.2
Ostial	17	53	29	90	.1
Non-ostial	15	47	3	9	.02
Mild calcification	2	7	7	22	.1
Moderate calcification	5	23	5	16	.9
Severe calcification	7	27	8	25	.7
Stent characteristics					
Length (mm)	17.1 ± 6.8		18 ± 8.3		.3
Diameter (mm)	5.8 ± 0.8		5.9 ± 1.8		.7
Completion angiography					
No residual stenosis	5	17	15	56	.03
Residual stenosis <30%	20	66	6	22	.004
Residual stenosis >30%	5	17	6	22	.7
Technical imperfections	15	50	8	30	.3
Missed lesions	11	37	4	15	.1
Partial stent expansion	7	23	4	15	.7
Tandem lesion	5	17	0	0	.06

[a]Five and 32 lesions we considered in the two groups.
[b]Only 34 CT/CTA were available.

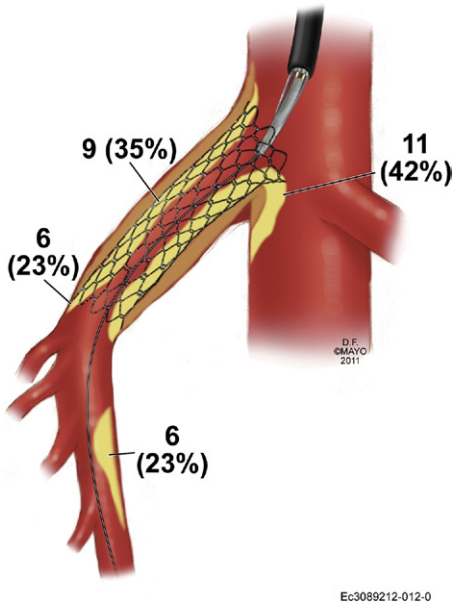

Fig 2, online only. Location of restenosis in 30 patients who underwent interventions for mesenteric in-stent restenoses.