Long-Term Results of Endovascular Treatment of Atherosclerotic Stenoses or Occlusions of the Coeliac and Superior Mesenteric Artery in Patients With Mesenteric Ischaemia

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WHAT THIS PAPER ADDS

Excellent long-term secondary patencies are described after percutaneous mesenteric artery stenting in a well defined cohort of patients with acute and chronic mesenteric ischaemia. This study underscores the evolving role for endovascular treatment in mesenteric ischaemia, from "bridge to surgery" to first choice treatment and "bridge to repeat endovascular treatment" in most patients with acute and chronic mesenteric ischaemia.

Introduction: Over the past decade, primary percutaneous mesenteric artery stenting (PMAS) has become an alternative to open revascularisation for treatment of mesenteric ischaemia. Institutes have presented favourable short-term outcomes after PMAS, but there is a lack of data on long-term stent patency.

Methods: One hundred and forty-one patients treated by PMAS for acute and chronic mesenteric ischaemia over an 8 year period were studied. Anatomical success was assessed by duplex ultrasound and/or CT angiography. A stenosis $\geq 70\%$ was considered to be a failure.

Results: Eighty-six coeliac arteries (CA) and 99 superior mesenteric arteries (SMA) were treated with PMAS in 141 patients. Nine CAs (10%) and 30 SMAs (30%) were occluded at the time of treatment. Median follow-up was 32 months (IQR 20–46). The overall primary patency rate at 12 and 60 months was 77.0% and 45.0%. The overall primary assisted patency rate was 90.3% and 69.8%. Overall secondary patency was 98.3% and 93.6%.

Conclusion: This study shows excellent long-term secondary patencies after PMAS, comparable with published data on long-term patencies after open surgical revascularisation.

INTRODUCTION

Acute and chronic mesenteric ischaemia are uncommon diseases. Chronic mesenteric ischaemia (CMI) is defined as longstanding abdominal symptoms caused by inadequate blood supply to the intestine.[1] Symptoms are non-specific and may include abdominal pain and weight loss. Acute (AMI) and acute on chronic mesenteric ischaemia (A-CMI) can progress to intestinal infarction in hours-days, with a high mortality rate.[2,3]

Open surgical revascularisation (OSR) was the gold standard for many years,[4–8] but percutaneous mesenteric artery stenting (PMAS) has become a valuable, less invasive alternative. The use of PMAS has been shown to lead to favourable short-term outcomes.[9–17]

Van Petersen et al.[18] compared the literature concerning the clinical and anatomical outcome after OSR and PMAS in case of CMI. They concluded that the short-term results were comparable, but there was a lack of robust data on mid- and long-term outcome after PMAS.

The main objective of the present study was to determine the long-term patency of PMAS of the CA and the SMA in a well defined cohort of patients with AMI and CMI.

METHODS

This study was performed with patients from a prospective database, including all patients with mesenteric ischaemia who were referred to Medisch Spectrum Twente, Enschede,

The Netherlands, a nationwide referral centre for mesenteric ischaemia. For the current study all consecutive patients who underwent PMAS for acute (including acute on chronic) and chronic mesenteric ischaemia in the period between November 2004 and November 2012 were included. According to institutional regulations, additional IRB approval was required for this retrospective analysis. Patient data were analysed anonymously.

All patients were analysed and treated according to a strict protocol, which included a structured medical history, assessment of vessel anatomy, and ischaemia function test as published previously.[19]

Patients with coeliac artery compression syndrome were excluded. Patients with previous revascularisation of the mesenteric arteries (PMAS or OSR) or patients who had had retrograde operative mesenteric stenting (ROMS) were also excluded. Data on cohort patients with ROMS have been published previously.[20] Patients lost to follow-up or those with inadequate documentation of vessel patency were also excluded from analysis. During the study period, no drug eluting stents or covered stents were used for PMAS.

Follow-up was performed at 3 months and yearly thereafter, or whenever recurring abdominal complaints suggested re-stenosis. To assess vessel patency, Doppler ultrasound of the mesenteric arteries was used as standard,[21] occasionally followed by CT angiography.

Procedural details

A vessel-first approach and a two-vessel revascularisation were adapted, in which the PMAS preceded laparotomy. For an acute abdominal condition, such as gastrointestinal perforation, laparotomy preceded PMAS and retrograde revascularisation of the SMA was then the preferred option.

All PMAS procedures were performed by dedicated interventional radiologists. Under local anaesthesia, a sheath was introduced into the femoral or brachial artery, preferably under ultrasound guidance. Then a pigtail catheter was advanced into the abdominal aorta, just above the level of the coeliac artery, and antero-posterior and lateral angiography was obtained to identify the origin of the mesenteric vessels. This was performed with an injection of 25 mL at 15 mL/s (Visipaque 320).

The standard technique for selective mesenteric vessel angiography was obtained with a curved or angled Mach 1 (LIMA) guiding catheter (Boston Scientific Corporation, USA) after steady positioning at the desired vessel ostium. Before intended treatment of stenotic lesions 5.000 IU of heparin was administered intra-arterially to reduce thromboembolic events during the procedure. In most cases, the lesion could be passed by a 0.035 inch hydrophilic coated angled tip Terumo wire (Terumo Medical Corporation, USA). In a severe stenosis or occlusion a 0.014 (Journey, Boston Scientific, USA) or 0.018 inch (V-18, Boston Scientific, USA) guide wire was preferred. After obtaining stable wire position, the guiding catheter or sheath was placed into the ostium and the diagnostic catheter was advanced through the lesion, and whenever possible, the guiding catheter or sheath was also advanced through the lesion. If the guiding sheath could not pass the lesion, a pre-dilating 4 mm PTA was performed. Then, the guiding catheter was advanced over the balloon, while deflating it.

It is highly recommended to forward the guiding catheter through the lesion before deploying a balloon expandable stent, to avoid losing the stent when passing tight lesions.

Another angiogram was performed to prove adequate intraluminal post-stenotic position. The 0.035 inch hydrophilic coated wire was then, when necessary, replaced by a 0.014 or 0.018 inch wire depending on the type of stent used.

A crucial step in the procedure is to mark the ostium of the mesenteric vessel by selective lateral angiography. With ostial lesions, the stent should be placed 2—3 mm into the aorta to prevent ostial intima hyperplasia. The stent should not extend further into the aorta to prevent problematic re-intervention in the future.

Depending on the location of the lesion and the lesion's characteristics (calcified/non-calcified), different types of stents can be used to treat the stenosis. Ostial stenoses, which are often very calcified, were usually treated with balloon expandable stents. More distal stenoses, that are often long and curved, are better treated by self expanding stents. Self expandable stents keep the natural curved anatomy of the mesenteric vessels intact and are less affected by movements of respiration.

The diameter and length of the stents were determined by CT angiography or DSA by measuring the length of the stenosis to be covered. The stent diameter was tailored to the arterial diameter immediately distal to the target lesion. There was no oversizing, with stent size always according to the vessel's diameter. In most patients a 6 mm stent was used. When determining the stent length, the extent of the stent beyond the proximal and distal end of the lesion was taken into account.

After stent placement, selective angiography was performed to check the stent placement. The procedure was completed by antero-posterior and lateral angiography by a pigtail catheter to ensure restored vessel patency and outflow.

Since 2013, puncture sites in femoral arteries have been closed using MYNX closure devices (AccessClosure Inc, USA.), while brachial puncture sites are always manually compressed.

All patients with a pre-procedural indication for warfarin or NOAC therapy continued this therapy with an additional daily dose of 100 mg carbasalate calcium for 6 months. All other patients received double antiplatelet therapy: including lifelong treatment with a daily dose of 100 mg of carbasalate calcium, combined with a daily dose of 75 mg clopidogrel for 6 months.

Outcome measures

Demographics were collected from medical records. Primary, primary assisted, and secondary patencies are reported, as previously defined by the Society for Vascular
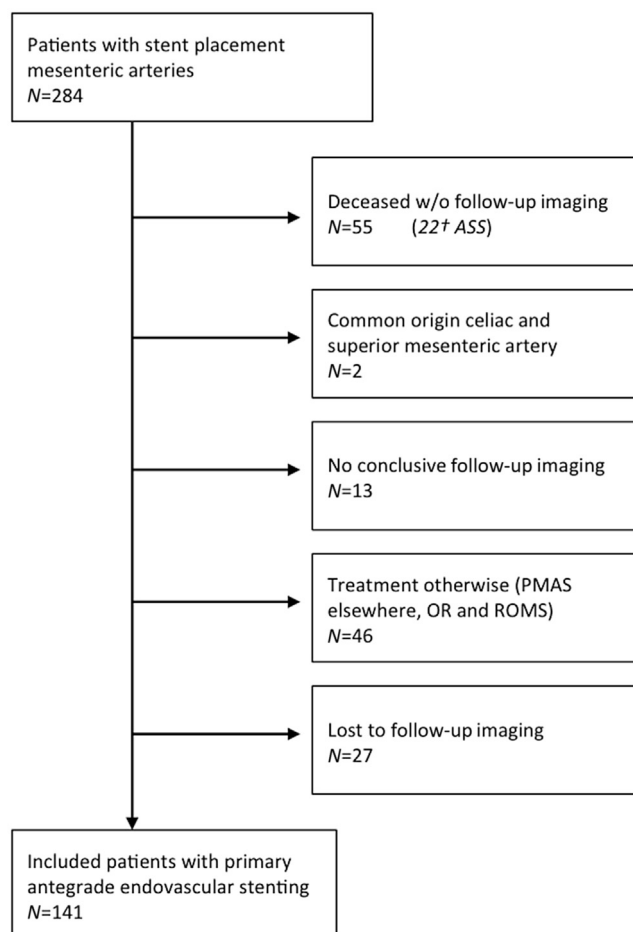
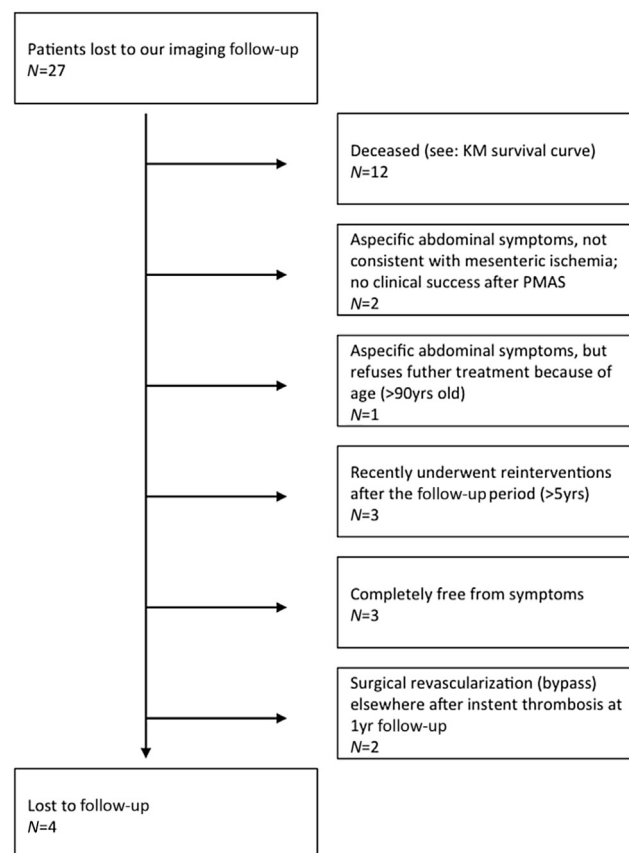**Figure 1.** Flowchart: from all patients with mesenteric artery stent placement to inclusion cohort.

Figure 2. Flowchart: patients "lost to follow-up imaging" analysis.

Surgery.[22] In short, primary patency is defined as uninterrupted patency following the revascularisation procedure being evaluated. Primary assisted patency is defined as patency following a re-intervention in the re-stenotic, but non-occluded tract. Secondary patency is defined as patency after recanalisation of an occluded, previously revascularised, tract.

Patency is defined as an open vessel on CTA or duplex ultrasound (B-mode) or velocities (PSV/EDV) consistent with vessel narrowing of <70%.

The degree of stenosis for CT scanning or DSA was calculated as previously described:[23] the diameter of the narrowest part of the stenosis divided by the diameter of the normal vessel distal to the stenosis and expressed as a percentage of the normal vessel.

Blood flow measurement by Doppler ultrasound was performed as previously described,[21,24] using a measurement angle of less than 60°. Significant stenosis was characterised by high velocity jets within the vessel and overall increased flow velocity. Cutoff values for the CA were peak systolic velocity (PSV) 200 m/s and end-diastolic velocity (EDV) 55 m/s; and for the SMA, PSV and EDV of 275 and 45 m/s, respectively.

The outcome of PMAS for each treated vessel was recorded separately.

Statistics

Continuous variables are expressed as mean with standard deviation (SD) or median with interquartile range (IQR); categorical variables as counts with corresponding percentages.

Time to loss of patency was analysed by Kaplan–Meier survival curves and the log-rank test was used to compare the patency curves between the CA and SMA. Data were analysed using SPSS, version 20 (SPSS Inc., Chicago, IL, USA).

RESULTS

During the 8 year study period, 284 patients underwent PMAS of the CA and/or the SMA (Fig. 1). In 46 patients a previous revascularisation of the mesenteric arteries was performed elsewhere or a primary OSR had been performed, and consequently they were excluded from this study.

Of the remaining 238 patients, the procedure was performed for acute mesenteric ischaemia in 21.0% (50 patients) and chronic mesenteric ischaemia in 79.0% (188 patients). Of these 238 patients, 27 were also excluded because imaging that could be evaluated of the PMAS during follow-up was not available and four patients remained completely lost to follow-up (Fig. 2). Of these 27 patients, four were treated for acute mesenteric ischaemia. After discharge 12 patients died in this group without appropriate follow-up.
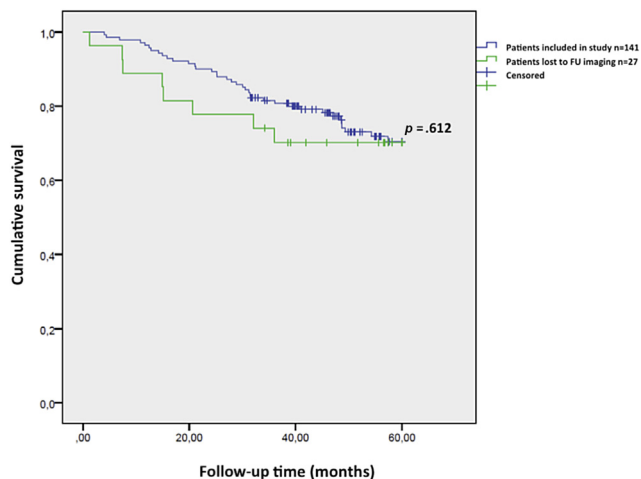
Figure 3. Comparison of cumulative survival between included cohort and cohort lost to follow-up imaging.

The cumulative survival of this cohort did not differ compared with the included cohort over a 5 year follow-up period (Fig. 3, $p = 0.612$). The overall in hospital mortality after PMAS was 11.3% (27 patients, Fig. 4). In the CMI cohort the in hospital mortality was 3.2% (6/188). In the AMI cohort 21 of 50 patients (42%) died from advanced or ongoing bowel infarction or irreversible ischaemia-reperfusion sequel and leading to multi-organ failure and death. Thirty-one patients in the AMI group (62%) underwent laparotomy and 28 patients (56%) needed bowel resection. In 19 cases PMAS preceded laparotomy. Of the remaining 12 cases, six patients underwent laparotomy and bowel resection elsewhere prior to referral to hospital for revascularisation.

At the study hospital, two patients were suspected of gastrointestinal perforation and consequently laparotomy was performed including resection. One patient had suspected bowel strangulation and also went to the operating room first; this turned out to be bowel ischaemia, but the patient still underwent a resection. The remaining three patients underwent a laparotomy prior to PMAS, of which one patient had a bowel resection. The reason is not clearly documented.

Of the 27 in hospital deaths, causes of death were identified in two patients, with proven stent patency, as multiple organ failure (MOF) (1) and ongoing bowel ischaemia (1). Of the remaining 25 patients, 10 had a CTA in the first few days after PMAS, with all of these showing stent patency. All these CT scans showed ongoing bowel ischaemia or MOF. Fifteen patients had no imaging of the mesenteric arteries after PMAS, but well documented medical records supporting the diagnosis of MOF/ongoing bowel infarction (9), pulmonary failure (4), and finally complicated peripheral artery disease (2).
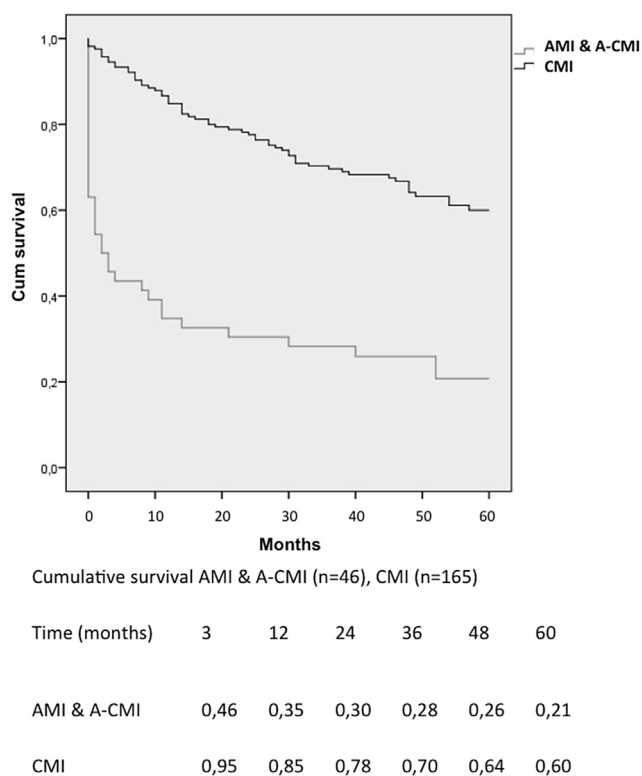
After discharge and before the first follow-up visit, another 28 patients died (Fig. 4). These included another six patients with AMI and 22 with CMI. Two patients had undergone autopsy and neither had intestinal ischaemia. One patient died from pancreatic necrosis with a patent stent. The other patient died from liver necrosis, but unfortunately this report did not support or deny stent patency. The causes of death of the remaining 26 patients were well documented and included cardiac failure (7), cerebrovascular disease (2), renal failure (2), malignancies (5), infections (6), and various causes (3). One case was a patient with abdominal pain, who had ischaemic colitis and later died from irreversible shock.

Cumulative survival rates for the acute and chronic mesenteric ischaemia cohorts are shown in Fig. 5. The cumulative survival for the AMI group ($n = 46$) after 3, 12, and 60 months is 46%, 35%, and 21%. The cumulative survival for the CMI group ($n = 165$) after 3, 12, and 60 months is 95%, 85%, and 60% ($p < 0.001$).

Thirteen patients had inconclusive follow-up imaging, mostly because of poor visualisation of the mesenteric arteries on ultrasound caused by overlying bowel gas, and they were excluded according to protocol for further
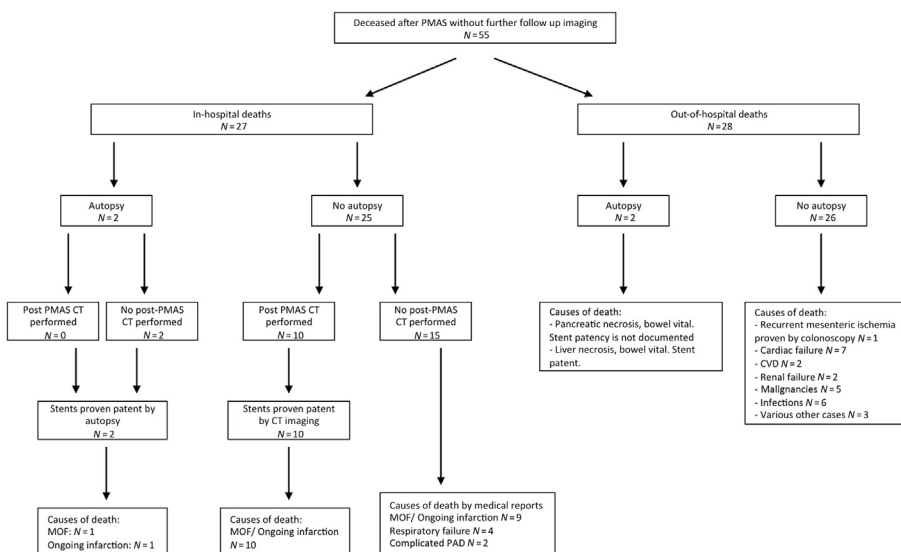
Figure 4. Imaging, autopsy, and medical report diagnoses of in hospital and out of hospital deaths.

Figure 5. Cumulative patient survival after PMAS in AMI vs. CMI cohort.

Cumulative survival AMI & A-CMI (n=46), CMI (n=165)

Time (months)	3	12	24	36	48	60
AMI & A-CMI	0,46	0,35	0,30	0,28	0,26	0,21
CMI	0,95	0,85	0,78	0,70	0,64	0,60

Table 1. Baseline demographics and characteristics ($n = 141$).

Variable	
Mean age in years (range)	64 (20−89)
Female, *n* (%)	92 (65)
N vessel disease, *n* (%)	
1	24 (17)
2	65 (46)
3	52 (37)
History of smoking, *n* (%)[a]	
Current smoker	63 (46)
Quit smoking prior to procedure	47 (34)
Non smoker	28 (20)
Hypertension, *n* (%)	90 (64)
Hyperlipidemia, *n* (%)[b]	68 (49)
Diabetes mellitus, *n* (%)	27 (19)
CAD, *n* (%)	51 (36)
PAD, *n* (%)	50 (36)
CVD, *n* (%)	26 (18)
Renal failure, *n* (%)	28 (20)

CAD = coronary artery disease; CVD = cerebrovascular disease; PAD = peripheral artery disease.
[a] $n = 138$ patients.
[b] $n = 139$ patients.

patency analysis. These 13 patients were all without symptoms at the time of follow-up examination, and therefore no additional analysis was performed to assess stent patency.

In two patients a common origin of the coeliac and the superior mesenteric arteries was found, making it impossible to perform a comparative analysis between the CA and the SMA; they were also excluded.

This left 141 patients who underwent primary PMAS, with follow-up data, included in this study. Of these, the procedure was performed for acute mesenteric ischaemia in 9.2% (13 patients) and chronic mesenteric ischaemia in 90.8% (128 patients).

The demographics of the included patients are summarised in Table 1. The median follow-up period was 32 months (IQR 20−46).

In these 141 patients a total of 185 vessels were treated; 86 revascularisations of the CA and 99 of the SMA. All procedures included stenting, with or without prior PTA in the same session. Among these revascularised vessels, nine (10%) CAs and 30 (30%) SMAs were occluded prior to PMAS.

Access was achieved through the left brachial artery in 66% and the femoral artery in 34% of cases.

The overall (CA and SMA combined) patency rates are shown in Fig. 6. The primary patency rate at 12, 24, 36, 48, and 60 months was 77%, 61%, 56%, 51%, and 45%. The primary assisted patency rate at 12, 24, 36, 48, and 60 months was 90%, 84%, 79%, 75%, and 70%. Finally, secondary patency at 12, 24, 36, 48, and 60 months was 98%, 97%, 95%, 94%, and 94%.
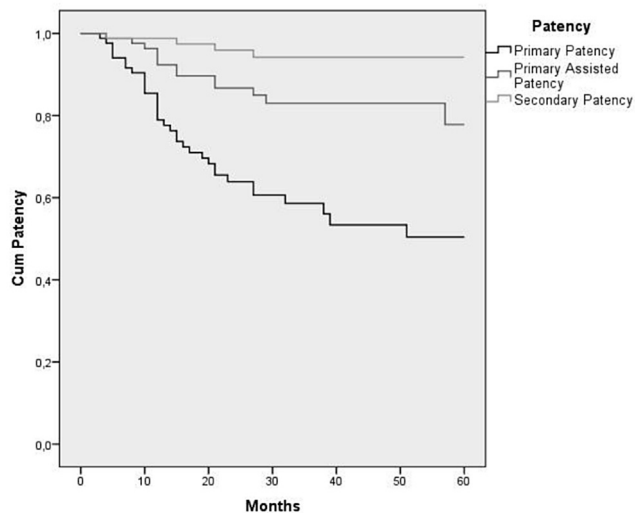
The patency rates of the CA and the SMA are shown in Figs. 7 and 8. For the CA the patency rates at 12 and 60 months were primary patency 79% and 50%, primary assisted patency 92% and 78%, and secondary patency 99% and 94%. For the SMA the 12 and 60 month results showed a primary patency of 75% and 39%, primary assisted patency 87% and 62%, and secondary patency 98% and 93%. Comparative analyses between the CA and SMA with the log-rank test showed no significant differences in

Vessels at risk (n=185)

Time (months)	12	24	36	48	60
PP	133	83	51	31	19
PAP	153	105	68	36	25
SP	166	121	85	41	28
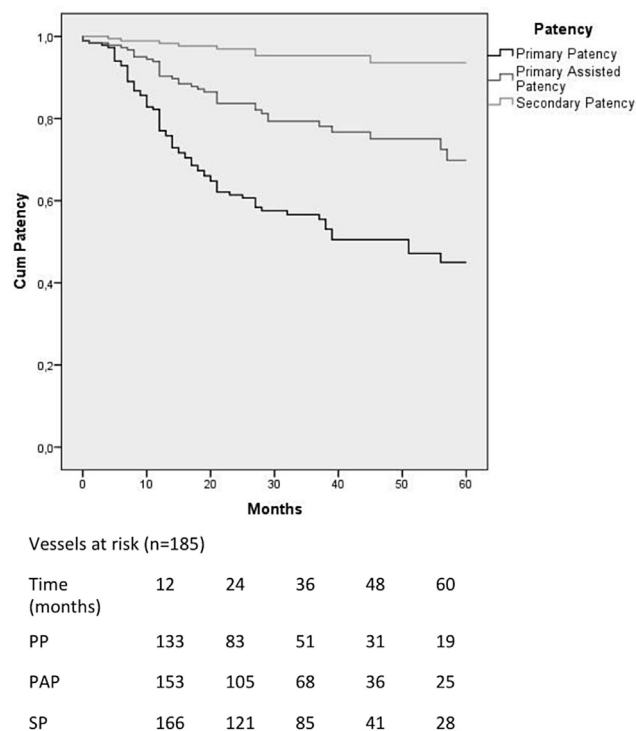
Figure 6. Overall cumulative patency rates after PMAS.

Vessels at risk (n=86)

Time (months)	12	24	36	48	60
PP	61	40	26	18	12
PAP	70	51	36	20	14
SP	74	57	42	22	16

Figure 7. Cumulative patency rates of the CA after PMAS.

primary ($p = 0.311$) primary assisted ($p = 0.101$), and secondary patency ($p = 0.871$).

DISCUSSION

This study demonstrated a modest primary (45%), but excellent 5 year secondary patency (94%) in a large cohort
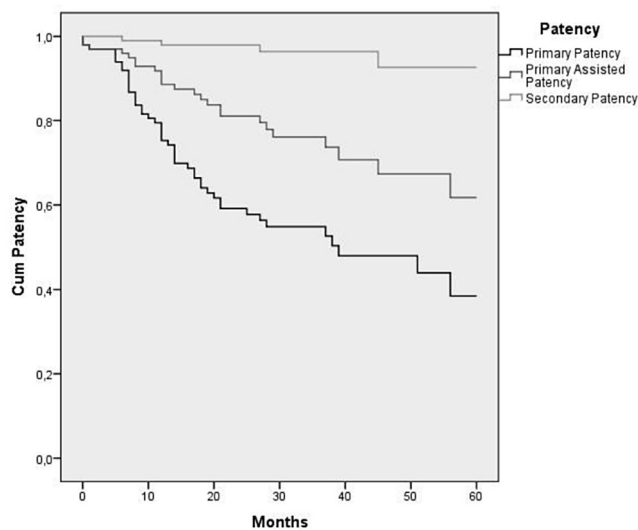
Vessels at risk (n=99)

Time (months)	12	24	36	48	60
PP	75	43	25	13	6
PAP	86	54	31	16	10
SP	91	64	42	19	11

**Figure 8.** Cumulative patency rates of the SMA after PMAS.

of patients with acute or chronic mesenteric ischaemia treated with primary PMAS. All patients with primary patency failure underwent percutaneous endovascular re-intervention to achieve primary assisted or secondary patency. The secondary patency figures are comparable with those published by centres of excellence after OSR.[8,25] Another important finding is that ostial occlusion does not exclude successful PMAS. In the present cohort, 22% of revascularised vessels were occluded before initial treatment. This study also underlined the rationale for close monitoring after PMAS to minimise the re-occlusion rate.

The present patency rates are in line with the meta-analysis by van Petersen et al.,[18] which demonstrated that PMAS had lower morbidity, but a decreased primary patency compared with OSR after 1–2 years of follow-up. After this meta-analysis, several institutes published experiences and results after PMAS, both in an acute or chronic setting,[8–17] and largely confirmed these observations. In most series the follow-up was restricted to 1–2 years, but, recently, Oderich[8] and AbuRahma[13] reported 5 year follow-up. Oderich et al.[8] compared OSR versus percutaneous endovascular revascularisation (PER) in CMI patients in 265 and 105 vessels, respectively. After a median follow-up of 30 months (range 4–174), they reported 5 year primary patency rates of 88% and 41%, and secondary patency rates 97% and 88% for OSR and PER, respectively. The mortality was similar, but OSR patients had more morbidity and longer hospitalisation than PER. Both treatments effectively improved symptoms, but restenosis, recurrent symptoms, and re-interventions were more likely in PER patients. Of note, the study only included CMI patients and the PER group consisted of PMAS (76) and/or balloon angioplasty alone (29).

AbuRahma et al.[13] reported 105 treated vessels after PMAS (54 SMA and 51 CA), with a mean follow-up of 31 months (range 1–124). The reported overall primary patency rates at 12 and 60 months were 69% and 19%, respectively. Assisted primary patency rates for the whole series were 80% and 34%, respectively. This study did not report secondary patencies and patients with vessel occlusions were excluded from treatment.

A technical point of PMAS for consideration is oversizing. The present authors always use a stent size according to the vessel diameter. Oversizing is much more painful, moreover oversizing causes more micro-injury of the vessel wall with consequently more inflammation and possibly making intimal hyperplasia more likely. The present authors always try to achieve two vessel patency in accordance with a previously published meta-analysis by van Petersen et al. in 2010.[18] This report states that long-term relief of symptoms can be achieved best by repair of more than one mesenteric artery, based on almost all reviewed studies. So the authors' policy is: "one is good, but two is better".

The high in hospital mortality of the present study was mainly associated with inclusion of patients with acute mesenteric ischaemia. Most of these patients had advanced or ongoing bowel infarction, or died from post-intervention complications. The latter mainly consisted of MOF. Because
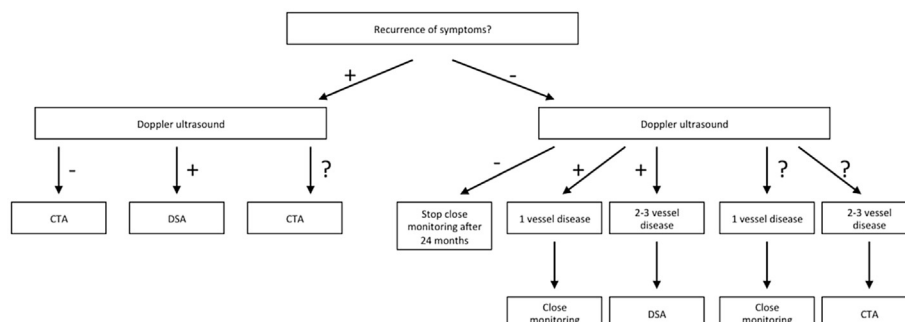
Figure 9. Flowchart: management of clinical surveillance and imaging findings in patients after PMAS.

this study adapted a vessel-first approach, the PMAS preceded laparotomy in most cases. In an institution with 24/7 availability of interventionalists, PMAS will be the fastest way to obtain revascularisation and bowel perfusion. When the mesentery has not been revascularised, it is not possible to distinguish between reversible and irreversible severe ischaemic bowel. Resecting all severe ischaemic bowel might result in increased survival, but this will definitely result in more short bowel syndromes with a lower quality of life in the initial surviving patients and increased mid-term mortality.

The present data show that 50 patients were diagnosed with AMI, of whom 28 (56%) needed bowel resection because of proven transmural necrosis. An in hospital AMI mortality rate of 42% (21/50) is reported and this rate is in the lower range of reported mortality figures (mean 70%, range 27–100%).[3] For transmural necrosis, mortality rates are even higher and up to approximately 90%. This does emphasise the importance of early diagnosis and treatment.[26]

Figure 5 demonstrates an initial 50% cumulative survival decrease of the AMI group compared with the CMI group at 3 months, thereafter the difference in survival between both groups remains stable. Those surviving the initial sequelae of AMI including revascularisation had excellent long-term secondary patency without recurrence of life threatening mesenteric ischaemia.

In the opinion of the present authors, advising patients to "return to my office if you have any symptoms again" is not useful because patients with chronic mesenteric ischaemia are well known for making adjustments to their lifestyle. At 12, 24, 36, 48 and 60 months, the stent occlusion rate (primary assisted patency rate) is 10%, 16%, 21%, 25% and 30%, respectively. Close imaging and clinical surveillance policy allow for faster identification of patients with recurrent symptoms (and adjusted lifestyles) caused by in-stent thrombosis, possibly enhancing their quality of life. Moreover, the excellent primary assisted patency rates in the present study imply that close surveillance might also prevent in-stent thrombotic occlusions and, consequently, episodes of AMI. Increased mortality is not seen among these patients with stent occlusion and this also supports "always two vessel approach if feasible".

During the study period, the policy after endovascular treatment was to do follow-up clinical assessment and DUS after 3 months, and then yearly. Nevertheless, the current data show approximately 40% primary patency loss within the first 24 months and approximately 15% cumulative loss in the following 3 years. These data might support a follow-up protocol in which patients are followed closely at 3, 6, 12, 18, and 24 months. In cases where no in-stent thrombosis has occurred, patients can be discharged, but should return if symptoms recur (Fig. 9).

There are limitations to the present study. At 5 year follow-up there were fewer cases left to be at risk of events, leading to larger margins of error.

Although all patients were prospectively analysed for mesenteric ischaemia, this analysis was performed retrospectively. Some data are missing, despite contacting referring vascular surgeons, gastroenterologists, and primary care physicians, which may have influenced the observed patency rates. For instance, one patient was reported as dying because of recurrent ischaemia, proven by colonoscopy. Unfortunately, no imaging was performed to assess the stent patency in this case. Furthermore, 21 patients died during primary admission because of intestinal infarction. In 10 of these advanced intestinal infarction was demonstrated immediately after PMAS. Stent patency was not assessed in these patients, and therefore early in-stent stenosis or occlusion might have been missed.

Finally, 13 patients were excluded because follow-up PMAS imaging was not assessable and four patients remained completely lost to follow-up, despite all efforts.

CONCLUSION

High anatomical success rates were obtained for percutaneous mesenteric artery stenting in this large cohort of AMI and CMI patients, including stenotic and occluded CA and SMA's. The 5 year secondary patency of >90% is comparable with that reported by open surgery series. This study underscores the evolving role for endovascular treatment in CMI patients, from "bridge to surgery" to first choice treatment and "bridge to repeat PMAS" in most patients with acute and chronic mesenteric ischaemia.

CONFLICT OF INTEREST

None.

FUNDING

None.

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