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## HANDLING OF AORTIC AND PERIPHERAL ARTERIAL PATHOLOGIES

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# Chronic mesenteric ischemia: when and how to intervene on patients with celiac/SMA stenosis

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## ABSTRACT

BACKGROUND: Studies that compared open surgical mesenteric artery repair (OSMAR) with percutaneous mesenteric artery stenting (PMAS) in patients with chronic mesenteric ischemia (CMI) are based on merely older studies in which only a minority of patients received PMAS. This does not reflect the current PMAS-first choice treatment paradigm. This article focused on the present opinions and changes in outcomes of OSMAR for CMI in the era of preferred use of PMAS.
METHODS: Patients who received OSMAR for CMI from 1997 until 2014 in a tertiary referral centre for chronic mesenteric ischemia were included in this report. Patients were divided into two groups, the historical OSMAR preferred group and present PMAS preferred group.
RESULTS: Patient characteristics, SVS comorbidity severity score, clinical presentation and number of diseased mesenteric arteries were not significantly changed after the widespread introduction of PMAS. In the present PMAS first era there were trends of less open surgical mesenteric artery multivessel repair, less antegrade situated bypasses, decreased clinical success but improved survival after OSMAR.
CONCLUSIONS: Elective OSMAR should only be used in patients with substantial physiologic reserve and who have unfavourable mesenteric lesions, failed PMAS or multiple recurrences of in-stent stenosis/occlusion. PMAS in CMI patients is evolved from "bridge to surgery" to nowadays first choice treatment and "bridge to repeated PMAS" in almost all patients with CMI.

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**Key words:** Mesenteric ischemia - Vascular diseases - Vascular surgical procedures - Vascular patency.

Atherosclerosis is the most common diseases of the vascular tree and the major cause of death in the western world. Atherosclerotic disease occurs in all human arteries and ultimately it can lead to stenosis, occlusions and aneurysms. Among the more uncommon clinical manifestations of atherosclerosis is symptomatic mesenteric diseases. Synonyms of the word "mesenteric" are "splanchnic", "visceral" and "intestinal". Mesenteric is derived from the new Latin word mesenterium and indicates "*any peritoneal membrane that enfolds an internal vertebrate organ and attaches it to the body wall*". Splanchnic is derived from the new latin word *splanchnicus*, which indicates "*pertaining to, or supplying the organs in the cavities of the body*". Visceral is derived from medieval Latin and means "*pertaining to, or affecting the organs in the cavities of*

*the body*". Intestinal is of new Latin origin and means "*pertaining to, being in, or affecting the lower part of the alimentary canal, extending from the pylorus to the anus*". Although the word splanchnic is clear, the synonym mesenteric is preferred to indicate the celiac artery (CA), the superior (SMA) and inferior mesenteric arteries (IMA), and ischemia in that region, since it is used by far most frequent in the literature.

Chronic mesenteric ischemia (CMI) is defined by abdominal symptoms due to inadequate blood supply to the gastrointestinal tract, most frequently caused by atherosclerosis.[1] The majority of patients suffer from abdominal pain and weight loss. Without revascularization of the intestine, CMI can ultimately progress to acute-on-chronic mesenteric ischemia with a high mortality rate up to 90%.[2, 3] Open surgical mesenteric artery repair (OSMAR) has been the gold standard of treatment since 1958 and is associated with excellent long-term patency and symptom relief.[4-6] As an alternative, the use of percutaneous mesenteric artery stenting (PMAS) has increased greatly during the past decades.[7] Advantages include less in-hospital morbidity, shorter hospital stay and availability in patients with high surgical risk. In contrast, primary patency and symptom recurrence rates are traditionally inferior to OSMAR. Furthermore, PMAS can be challenging in heavily calfied osteal lesions. Studies that compared OSMAR and PMAS cohorts showed that patients who received OSMAR had less extensive comorbidity, but more severe mesenteric artery atherosclerosis when compared to patients who received PMAS.[8, 9] Nevertheless indications for treatment with PMAS have broadened in recent years, and now also include patients who would have been candidates for OSMAR in the past. Consequently, the fast majority of patients with CMI now receive PMAS.[8]

One recently published [10] and two shortly published reviews [11, 12] collectively discuss nearby all relevant topics of acute and chronic mesenteric ischemia. In these careful edited papers outcome of OSMAR and PMAS are based on merely older studies in which only a minority of patients received PMAS. This does not reflect the current PMAS-first choice treatment paradigm. Because of this changed daily practice traditional results of OSMAR may not be applicable nowadays. Therefore the present study focused on changes in outcomes of open surgical mesenteric artery repair for CMI in the era of preferred use of PMAS.

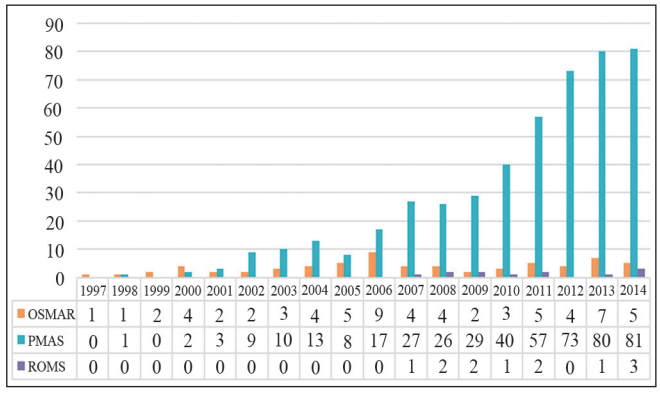
## Materials and methods

### *Patient inclusion*

Medisch Spectrum Twente is a tertiary referral center for mesenteric ischemia in the Netherlands. Starting in 1997 all admitted patients diagnosed with mesenteric ischemia have been prospectively enrolled in our database. Patients who received OSMAR for CMI from 1997 until 2014 were included in this report. Excluded were patients who received OSMAR for acute mesenteric ischemia, acute-on-chronic mesenteric ischemia, celiac artery compression syndrome, or previous mesenteric artery revascularisations. Patients who received OSMAR were divided into two groups based on date of intervention. OSMAR became the preferred treatment for CMI in our centre since 2006, therefore 01/01/2006 was used as a dividing line between the historical OSMAR preferred group and present PMAS preferred group. Annual interventions with OSMAR and PMAS for the treatment of CMI in our clinic is shown in Figure 1. According to institutional regulations, review board individual patient approval was not required for this retrospective study. Therefore no patient informed consent was obtained. Patient data were analyzed anonymously.

### *Diagnosis*

Each patient referred to our center for evaluation of CMI received a standard diagnostic workup as described previously.[13] Complete screening consisted of thorough interviews by a gastroenterologist and a vascular surgeon, mesenteric artery duplex ultrasound, 24-hour $CO_2$




| | 1997 | 1998 | 1999 | 2000 | 2001 | 2002 | 2003 | 2004 | 2005 | 2006 | 2007 | 2008 | 2009 | 2010 | 2011 | 2012 | 2013 | 2014 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| OSMAR | 1 | 1 | 2 | 4 | 2 | 2 | 3 | 4 | 5 | 9 | 4 | 4 | 2 | 3 | 5 | 4 | 7 | 5 |
| PMAS | 0 | 1 | 0 | 2 | 3 | 9 | 10 | 13 | 8 | 17 | 27 | 26 | 29 | 40 | 57 | 73 | 80 | 81 |
| ROMS | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 2 | 1 | 2 | 0 | 1 | 3 |

Figure 1.—Overview of number and type of interventions for chronic mesenteric ischemia in a tertiary referral hospital.

tonometry and/or gastric exercise tonometry, laboratory studies, and multidisciplinary assessment of diagnostic evidence. When suspicion of CMI was confirmed, mesenteric digital subtraction angiography or computed tomography angiography was performed resulting in the final diagnosis and treatment advice.

*Treatment*

In general, treatment preference was based on life expectancy, vascular anatomy, nutritional status and surgical risk. OSMAR was preferably performed using autologous antegrade revascularization techniques. Retrograde bypass was performed when the supraceliac anatomy was considered unsuitable for aortic clamping, or the hemodynamic risk of supraceliac clamping was considered too challenging in the context of patient comorbidities.

*Supportive care and follow-up*

All patients received proton pump inhibition. Postoperatively all patients were placed on a strict stepwise progressive oral refeeding protocol including daily clinical and hematological assessments to reduce the ischemia reperfusion sequels. Patients were heparinized until oral anticoagulant or antiplatelet therapy was effective. Patients received at that moment state of the art medical treatment for secondary prevention of atherosclerosis. Patients underwent a standardized follow-up schedule, consisting of outpatient visits and duplex ultrasound at 3, 12, 24, and 48 months, followed by once every 2 years.

*Data gathering and outcomes*

For this report the prospective patient database was supplemented with data from hospital records and referring physician or patient correspondence. Data registered were demographics, presenting symptoms, cardiovascular risk factors according to Society for Vascular Surgery (SVS) reporting standards, SVS comorbidity severity score, previous interventions for mesenteric ischemia, the number of significant stenosed or occluded mesenteric arteries, and treatment details. A vessel lumen diameter reduction of >70% was considered significant.

Primary outcomes were postoperative mortality and complications. Grade 3a or greater complications according to the Dindo-Clavien Classification were recorded. Secondary outcomes were patency rates according to SVS reporting standards and clinical success rates. Clinical success was defined as anatomical success and improvement or continued absence of symptoms. Clinical failure was defined as persistence or recurrence of symptoms caused by residual, recurrent, or additional vessel stenoses or occlusions. Ischemia- or therapy-related death was also recorded as clinical failure.

The Netherlands municipal personal records database was accessed for confirmation of survival. The in-hospital/30-day (IH/30D) follow-up period was defined as the first 30 postoperative days or until discharge when postoperative admittance duration was >30 days. The late follow-up period started at 30 days, or at discharge when postoperative admittance duration was >30 days.

*Statistical analysis*

Continuous variables were expressed as mean with standard deviation (SD) when normally distributed or as median with interquartile range (IQR) when not normally distributed. Categorical variables were expressed as counts with percentages. Continuous variables were compared using independent sample t-tests when normally distributed or Mann-Whitney U tests when not normally distributed. Categorical variables were compared using Pearson's $\chi^2$ or Fisher's exact tests as appropriate. Survival, patency and clinical success rates were analyzed by Kaplan-Meier plots, using log-rank tests to determine significance. P-values less than 0.05 were considered significant and values between 0.05 and 0.20 were considered a trend. Univariate analysis of survival, patency and clinical success rates was performed using Cox regression. Data were analyzed using SPSS, version 22 (IBM Corp. Armonk, NY, USA).

## Results

*Patient characteristics*

In total 64 patients with OSMAR were included; 23 in the historical group and 41 in the present group (Fig-

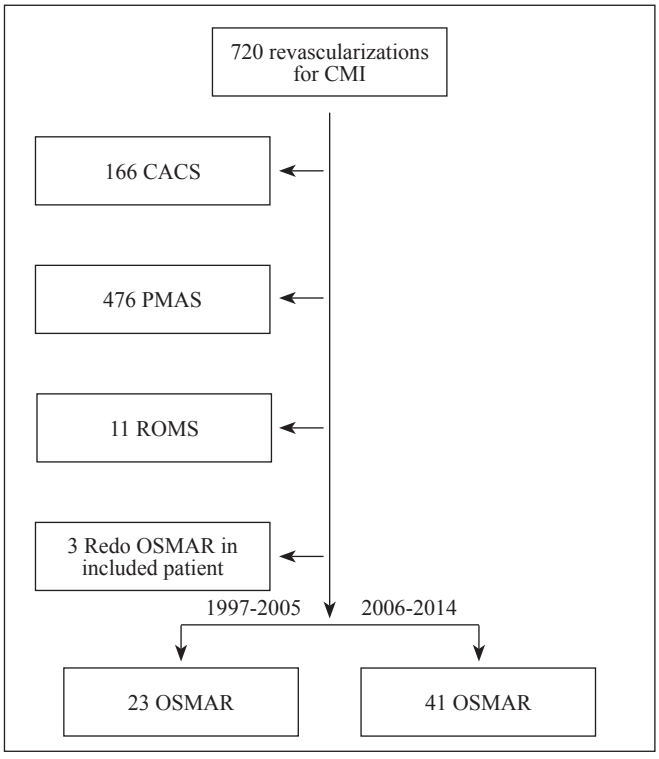



Figure 2.—Flowchart of patient treated for CMI between 1997 and 2014.

ure 2). Patient and disease characteristics are shown in Table I. In the historical group, 21 patients received OSMAR for newly diagnosed CMI and 2 patients with previous PMAS for CMI. In the present group 20 patients received OSMAR for newly diagnosed CMI and 21 patients with previous PMAS for CMI (8.7% *vs.* 51.2%; P=0.001). Both groups had similar gender, age and body mass index (BMI). The present group had higher incidences of hypertension (21.7% *vs.* 51.2%; P=0.021) and hyperlipidemia (26.1% *vs.* 53.7%; P=0.033). Both groups had similar incidences of other comorbidities and median SVS. comorbidity severity score. Both groups had similar clinical presentation except for a trend towards greater incidence of adapted eating pattern in the present group (56.5% *vs.* 79.4%; P=0.064). Both groups had equal numbers of diseased vessels per patient.

### *Treatment*

A summary of treatment characteristics is shown in Table II. Multivessel repairs were performed in 73.9% in the historical group and in 56.1% in the present group (P=0.158), 78.9% of bypasses in the historical group and 61.1% in the present group were antegrade (P=0.180). More single SMA bypasses were performed in the present group (P=0.056) and a significant but clinical not relevant decrease in blood loss in the present group was noted (P=0.039). Technical success was achieved in all patients. Simultaneous procedures in the historical group were nephrectomy for renovascular hypertension in one patient and aortoiliac endarterectomy for claudication in another patient. Simultaneous procedures in the present group were aortobi-iliac bypass for claudication in one patient, combined right sided iliofemoral bypass and left iliac endarterectomy for claudication in one, cicatricial hernia correction in three and liver segment resection for a carcinoid tumor metastasis found incidentally in another one. There were no intraoperative deaths.

TABLE I.—*Patient characteristics of 64 patients treated with OSMAR for CMI.*

| Variable | Historical group (N.=23) | Present group (N.=41) | P value |
|---|---|---|---|
| Male sex (N., %) | 3 (13.0%) | 10 (24.4%) | 0.227 |
| Age, years (mean, SD) | 53.2 (±11.1) | 54.2 (±10.9) | 0.719 |
| BMI (mean, SD) | 21.0 (±3.4) | 21.8 (±3.7) | 0.405 |
| Risk factors (SVS) | | | |
| Diabetes mellitus (N., %) | 1 (4.3%) | 4 (9.8%) | 0.646 |
| Currently smoking (N., %) | 18 (78.3%) | 26 (63.4%) | 0.219 |
| Hypertension (N., %) | 5 (21.7%) | 21 (51.2%) | 0.021 |
| Hyperlipidemia (N., %) | 6 (26.1%) | 22 (53.7%) | 0.033 |
| Coronary artery disease (N., %) | 4 (17.4%) | 8 (19.5%) | 1.000 |
| Cerebrovascular disease (N., %) | 1 (4.3%) | 3 (7.3%) | 1.000 |
| Renal disease (N., %) | 2 (8.7%) | 2 (4.9%) | 0.614 |
| Pulmonary disease (N., %) | 4 (17.4%) | 9 (22.0%) | 0.755 |
| Peripheral artery disease (N., %) | 7 (30.4%) | 15 (36.6%) | 0.619 |
| SVS comorbidity score (median, IQR) | 1 (0-5) | 2 (0-5.5) | 0.317 |
| Previous treatment for CMI (N., %) | | | |
| PTA | 1 (4.3%) | 1 (2.4%) | 1.000 |
| PMAS | 1 (4.3%) | 15 (36.6%) | 0.004 |
| ROMS | 0 (0.0%) | 2 (4.9%) | 0.532 |
| OSMAR | 0 (0.0%) | 4 (9.8%) | 0.288 |
| Any | 2 (8.7%) | 21 (51.2%) | 0.001 |
| Symptoms among symptomatic patients (N., %) | | | |
| Weight loss | 22 (95.7%) | 30 (88.2%) | 0.638 |
| Postprandial pain | 22 (95.7%) | 28 (82.4%) | 0.233 |
| Adapted eating pattern | 12 (56.5%) | 27 (79.4%) | 0.064 |
| Diarrhea | 11 (47.8%) | 20 (58.8%) | 0.413 |
| Nausea | 8 (34.8%) | 13 (38.2%) | 0.791 |
| Vomiting | 7 (30.4%) | 7 (20.6%) | 0.397 |

TABLE II.—*Operative treatment characteristics of 64 patients treated with OSMAR for CMI.*

| Variable | Historical group (N.=23) | Present group (N.=41) | P value |
|---|---|---|---|
| Bypass (N., % of total cases) | | | |
| CA and SMA bypass | 13 (56.5%) | 21 (51.3%) | 0.683 |
| Single CA bypass | 3 (13.0%) | 3 (7.3%) | 0.451 |
| Single SMA bypass | 2 (8.7%) | 12 (29.3%) | 0.056 |
| IMA patch angioplasty to SMA bypass | 1 (4.3%) | 0 (0.0%) | 0.359 |
| Any bypass | 19 (82.6%) | 36 (87.8%) | 0.711 |
| Bypass configuration (N., % of all bypasses) | | | |
| Antegrade | 15 (78.9%) | 22 (61.1%) | 0.180 |
| Retrograde | 4 (21.1%) | 14 (38.9%) | 0.180 |
| Bypass material (N., % of all bypasses) | | | |
| Autologous vein | 13 (68.4%) | 32 (88.9%) | 0.071 |
| Autologous artery | 5 (26.3%) | 3 (8.3%) | 0.124 |
| Synthetic (6-mm Dacron) | 1 (5.3%) | 0 (0.0%) | 0.359 |
| Combined artery and vein | 0 (0.0%) | 1 (2.8%) | 1.000 |
| Endarterectomy (N., % of total cases) | 4 (17.4%) | 4 (9.8%) | 0.443 |
| SMA reimplantation (N., % of all cases) | 0 (0.0%) | 1 (2.4%) | 1.000 |
| Vessels treated (N., % of all cases) | | | |
| CA | 20 (87.0%) | 27 (65.9%) | 0.067 |
| SMA | 19 (82.6%) | 37 (90.2%) | 0.443 |
| IMA | 1 (4.3%) | 1 (2.4%) | 1.000 |
| 1 vessel treated | 6 (26.1%) | 18 (43.9%) | 0.216 |
| 2 vessels treated | 17 (73.9%) | 22 (53.7%) | 0.216 |
| 3 vessels treated | 0 (0.0%) | 1 (2.4%) | 0.216 |
| Intraoperative blood loss, mL (median, IQR) | 600 (400-1500) | 400 (300-700) | 0.039 |
| Intervention duration, min (mean, SD) | 309 (±83) | 296 (±87) | 0.595 |

TABLE III.—*Complications of 64 patients treated with OSMAR for CMI.*

| Variables | Historical Group (N.=23) | Present Group (N.=41) | P value |
|---|---|---|---|
| Early follow-up morbidity, Dindo-Clavien grade >2 (N., %) | | | |
| Procedure related | | | |
| Bleeding | 2 (8.7%) | 5 (12.2%) | 1.000 |
| Infection | 0 (0.0%) | 3 (7.3%) | 0.547 |
| Occlusion/stenosis | 1 (4.3%) | 7 (17.1%) | 0.241 |
| Other | 4 (17.4%) | 4 (9.8%) | 0.443 |
| Any | 6 (26.1%) | 12 (29.3%) | 0.786 |
| Systemic | | | |
| Multiorgan failure | 2 (8.7%) | 1 (2.4%) | 0.291 |
| Pulmonary | 2 (8.7%) | 3 (7.3%) | 1.000 |
| Cardiac | 1 (4.3%) | 0 (0.0%) | 0.359 |
| Cerebrovascular | 0 (0.0%) | 0 (0.0%) | NA |
| Renal | 0 (0.0%) | 0 (0.0%) | NA |
| Any | 4 (17.4%) | 4 (9.8%) | 0.443 |
| Early follow-up highest Dindo-Clavien grade | | | 0.675 |
| 3a | 1 (4.3%) | 2 (4.9%) | |
| 3b | 3 (13.0%) | 6 (14.6%) | |
| 4a | 1 (4.3%) | 3 (7.3%) | |
| 4b | 1 (4.3%) | 0 (0.0%) | |
| 5 | 3 (13.0%) | 2 (4.9%) | |
| Late follow-up morbidity, Dindo-Clavien grade >2 (N., %) | | | |
| Procedure-related | | | |
| Bleeding | 0 (0.0%) | 0 (0.0%) | NA |
| Infection | 0 (0.0%) | 1 (2.4%) | 1.000 |
| Occlusion/stenosis | 3 (13.0%) | 6 (14.6%) | 1.000 |
| Other | 3 (13.0%) | 3 (7.3%) | 0.658 |
| Any | 5 (21.7%) | 10 (24.4%) | 0.810 |

### *Mortality and complications*

Median hospital admission duration was 13 (IQR 10-17) days in the historical group and 16 (IQR 11-31) days in the present group (P=0.139). Median intensive care unit admission was 2 (IQR 1-5) days in the historical group and 1 (IQR 1-2) day in the present group (P=0.059). Three patients (13.0%) died during the IH/30D period in the historical group. Causes were small bowel necrosis despite a patent bypass, small bowel necrosis caused by SMA dissection following endarterectomy, and multi-organ failure. Two patients (4.9%; P=0.341) died during the IH/30D period in the present group. One patient died of multi-organ failure following partial resection of necrotic small bowel caused by SMA dissection during antegrade 2-vessel bypass. One patient died of hypovolemic shock caused by a retrograde SMA bypass anastomosis rupture after hospital discharge. The five patients who died during the IH/30D period had a median BMI of 18.0 (IQR 15.9-22.2) compared to 21.5 (IQR 19.7-23.0) among those who survived (P=0.070). Median SVS comorbidity severity score was 4 (IQR 0-9) compared to 2 (IQR 0-5) among those who survived (P=0.515).

Median follow-up was 92 (IQR 16-123) months in the historical group and 20 (IQR 11-49) months in the present group. Five patients died during follow-up in the historical group. The reasons were congestive heart failure, diabetic foot complicated by sepsis, end-stage chronic obstructive pulmonary disease, stroke and chol-

angiocarcinoma. Three patients died during follow-up in the present group, the cause of death was unknown but in all these three patients no symptoms consistent with CMI were recorded. For both groups the IH/30D and late complications were demonstrated in Table III.

Kaplan Meier estimates of survival are shown in figure 3, there was a trend of improved survival in the present group (P=0.134). One-, three- and five-year survival in the historical and present group was 87.0% *versus* 95.1%, 78.3% *versus* 92.3% and 73.9% *versus* 88.5% respectively. Factors associated with decreased survival were duration of intensive care unit admittance (P=0.001), age (P=0.007), cardiac disease (P=0.041), conservative treatment of celiac artery disease (P=0.054), carotid disease (P=0.057) and intraoperative blood loss (P=0.117).

### *Clinical success and patency rates*

There was a trend of decreased clinical success in the present group (P= 0.174). One-, three, and five-year clinical success rates in the historical and the present group were 82.6% *versus* 72.1%, 82.6% *versus* 60.1% and 70.7% *versus* 53.4% respectively Factors associated with decreased clinical success duration were intensive care admittance duration (P=0.017), carotid disease (P=0.070), cardiac disease (P=0.092) and intraoperative blood loss (P=0.093).

Clinical failure occurred in seven patients (30.4%) in the historical group. In addition to the three in-hospital deaths, four patients experienced a symptomatic recurrence. Of these four patients, one had CA endarterectomy restenosis, one had combined CA endarterectomy reocclusion and SMA endarterectomy restenosis, one had occlusion of both limbs of an antegrade 2-vessel bypass and one had occlusion of a single vessel CA bypass. Treatment was PMAS of the CA, 2-vessel antegrade bypass, PMAS of the native CA combined with PTA of the native SMA, and conservative respectively.

Clinical failure occurred in 14 patients (34.1%) in the present group. In addition to the two IH/30D deaths, 11 patients had a symptomatic recurrence and one patient received preventive PMAS of the IMA after asymptomatic SMA limb occlusion of a 2-vessel antegrade bypass. Of the 11 patients who experienced a symptomatic recurrence, three had stenosis or occlusion of a single vessel SMA bypass. The other eight had a two vessel antegrade or retrograde bypass. Of these eight patients, four had occlusion of both bypass limbs, two had stenosis or occlusion of the SMA limb, one had occlusion of the CA limb, and one had bypass origin stenosis. One patient who experienced a symptomatic recurrence was treated conservatively and 10 patients received a reintervention. Reinterventions were PMAS or PTA of the bypass and/or native vessels in five patients, surgical bypass revision in three, and redo retrograde SMA bypass in two patients. One patient who was treated with PMAS of the native SMA also underwent relaparotomy and subsequent drainage of a necrotic gallbladder.

One patient with asymptomatic occlusion of a single vessel CA bypass in the classic group and one patient with asymptomatic double limb occlusion of a 2-vessel antegrade bypass in the modern group were treated conservatively. One-, 3-, and 5-year primary patency of SMA reconstructions in the historical and present group was 94.7% *versus* 71.8%, 88.4% *versus* 53.8% and 88.4% *versus* 47.9% respectively (P=0.024). No factors were associated with decreased primary patency. 1-, 3-, and 5-year secondary patency in the historical and present group was 94.7% *versus* 83.1%, 88.4% *versus* 69.8% and 88.4% *versus* 62.5% respectively (P=0.155).

One-, 3-, and 5-year primary patency of CA reconstructions in the historical and present group was 89.4% *versus* 81.2%, 89.4% *versus* 60.9% and 75.0% versus 60.9% respectively (P>0.222). Factors associated with decreased primary patency were retrograde bypass (P=0.050), hyperlipidemia (P=0.054), previous intervention for CMI (P=0.075), carotid disease (P=0.094) and diabetes mellitus (P=0.141). One-, 3-, and 5-year secondary patency in the historical and present group was 89.4% *versus* 88.7%, 89.4% *versus* 75.1%, and 82.5% *versus* 75.1% respectively (P>0.569).

## Discussion

The present study demonstrated that patient characteristics, SVS comorbidity severity score, clinical presentation and number of diseased mesenteric arteries were not significantly changed after the widespread introduction of PMAS in a cohort of patients with CMI treated with OSMAR. In the present PMAS first era there were trends of less open surgical mesenteric artery multivessel repair, less antegrade situated bypasses, decreased clinical success but improved survival after OSMAR.

The most presumable explanation is that with the rise of PMAS, patients with more extensive atherosclerosis of the mesenteric arteries and in a better physical condition now undergo OSMAR. Patient characteristics indicating that advanced generalized atherosclerotic disease was associated with decreased in hospital survival after OSMAR. Morbidity and mortality increased substantially in patients undergoing OSMAR with significant weight loss. BMI below 19.5 kg/m$^2$ was associated with two to tenfold increased major morbidity and mortality.[4, 14] Also in the present study the five patients who died shortly after OSMAR had a significant lower BMI compared to those who survived, 18.0 *versus* 21.5 kg/m$^2$.

One or two vessel repair in case of multi-vessel CMI is still a point of debate. An evidence summary report, although albeit with a high risk of confounding, supported for both PMAS and OSMAR the statement that long term relief of symptoms can be achieved best by repair of more than one splanchnic artery.[8]

The level of evidence for convincing advice which material to use in OSMAR is low and local preferences rules the opinion. On the one hand, a couple of reports state the use of polyester grafts.[9, 16] On the other hand, because of observed superb patencies there is support to justify, like in PAD, an autologous vein bypass of the CA and/or the SMA as the preferred technique.[6, 17]

The main priorities in revascularization of CMI are improving quality of life and prevention of bowel infarction. Secondary weight gain is a bonus. PMAS is now the primary treatment choice.[7] But when analysing available literature there is a clear selection bias, which should be taken in to account in coming to a conclusion. The forthcoming review by Blauw *et al.*[12] underlines that PMAS has lower mortality and morbidity, length of stay and recovery time compared to OSMAR, but the counterpart is that more recurrence of symptoms, restenosis and re-interventions are seen after PMAS and there is a higher recurrence of symptoms. A retrospective analysis of our own data on PMAS [18] between November 2004 and November 2012 showed that PMAS primary patency was 77% at 1 year and 45% after 5 years. But primary assisted, respectively 90% and 69.8%, and secondary patency, respectively 98.3% and 93.6%, were excellent and comparable to those published by centres of excellence after OSMAR. In our experience ostial mesenteric artery occlusions does not exclude successful PMAS.

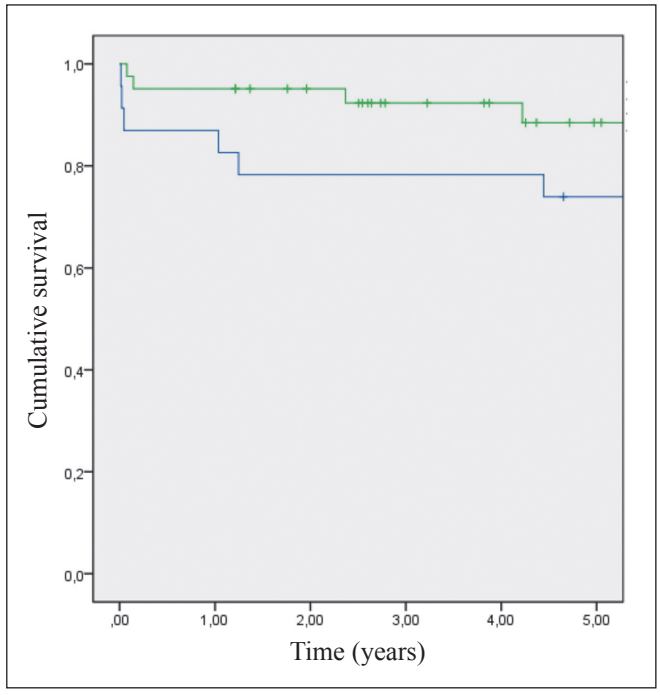



Figure 3.—Kaplan Meier estimates of overall survival after OSMAR for the 23 patients with CMI in the historical and 41 patients in the present cohort. Blue line historical group, green line present group (P=0.134).

Although literature is scares, covered stents were associated with less restenosis, a lower clinical symptom recurrence rate, and fewer re-interventions when compared to bare metal stents.[19] A Dutch Randomised Controlled Trial comparing bare metal with covered stents for PMAS in patients with CMI is including patients yet, first result are awaited in 2018.

All patients need life-long anticoagulation to prevent atherosclerosis. There was a clear shift in postoperative thromboprophylaxis from lifelong single antiplatelet therapy to 3-6 months of double antiplatelet therapy followed by lifelong single therapy (P<0.001).

As mentioned in the introduction several excellent reviews were recently or will be published soon.[10-12] We assume that this reviews gain strengths if two points has been added. Firstly, single-vessel CMI can only be diagnosed when a validated functional test indicates splanchnic ischemia. Without such a function test the diagnosis of single vessel CMI is more or less a coin flip. With gastric tonometry in the workup of single vessel CMI a durable relief of symptoms is achievable in 84% of patients.[20] Secondly, the influ-

ence of respiration and collateral flow on the normal values of Duplex ultrasonography is not accentuate.[21] Consequently the degree of stenosis of the CA or the SMA could be easily over or underscored with Duplex ultrasound.

## Conclusions

The present study supports that the historical results of OSMAR cannot be extrapolated to the current CMI patient population. There are no randomized controlled trials comparing an endovascular first *versus* an open surgery first approach and level I evidence is lacking. There is widespread support that long-term primary anatomical patency were better after OSMAR, with significantly more and mainly endovascular reinterventions in the PMAS group, secondary patency was not significantly different between PMAS and OSMAR. Nowadays, elective OSMAR should only be used in patients with substantial physiologic reserve and who have unfavorable mesenteric lesions, failed PMAS or ROMS or multiple recurrences of in-stent stenosis/occlusion.[11, 12, 22, 23] The type of OSMAR should be tailored to the anatomy and the patient's clinical risk assessment. Planning OSMAR involves selection of the type of incision (transperitoneal *vs.* retroperitoneal), conduit (vein *vs.* prosthetic), graft configuration (antegrade *vs.* retrograde), source of inflow (aortic *vs.* iliac) and the number of vessels to be reconstructed (single *vs.* multiple). PMAS in CMI patients is evolved from "bridge to surgery" to nowadays first choice treatment and "bridge to repeated PMAS" in almost all patients with chronic mesenteric ischemia.

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*Conflicts of interest.*—The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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