

Chronic Mesenteric Ischemia – Surgical and Interventional Options in Therapy

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Key Words

Chronic mesenteric ischemia · Aorto-mesenteric bypass · Angioplasty

Summary

Chronic mesenteric ischemia (CMI) is an unusual but important cause of abdominal pain accounting for 5–8% of all intestinal ischemic events. Most commonly it is caused by atherosclerotic occlusions or severe stenosis. Symptoms occur when at least two of the three main splanchnic vessels are affected. The course is frequently asymptomatic, resulting from an extensive collateralization. Intestinal angina combined with weight loss and sitophobia are typical clinical features. Therapeutic options are conventional surgical therapy with thrombendarterectomy or bypass as well as percutaneous angioplasty with or without stenting. In symptomatic disease immediate therapy is essential because the natural course is life-threatening. Invasive treatment of asymptomatic but significant CMI has to be seriously considered as it developed into symptomatic disease with a high possibility.

Schlüsselwörter

Chronische mesenteriale Ischämie · Aorto-viszeraler Bypass · Angioplastie

Zusammenfassung

Die chronische mesenteriale Ischämie (CMI) stellt eine seltene, aber wichtige Ursache abdomineller Beschwerden dar. Häufigste Ursachen sind atherosklerotisch bedingte höchstgradige Stenosen oder Verschlüsse der Viszeralgefäße. Symptome entstehen bei Befall von 2 der 3 Viszeralgefäße. Aufgrund der ausgezeichneten Kollateralisationsmöglichkeiten wird die Erstdiagnose häufig als Zufallsbefund und/oder fortgeschrittener Verschlussprozess gestellt. Typische Symptome sind postprandialer krampfartiger Abdominalschmerz, Gewichtsverlust und die Angst vor Nahrungsaufnahme: Die operative Therapie mit Thrombendarterektomie oder aorto-viszeralem Bypass sowie die interventionelle Therapie mittels perkutaner Angioplastie mit oder ohne Stent stellen die invasiven therapeutischen Optionen dar. Aufgrund des lebensgefährlichen Verlaufs ist die zeitnahe Behandlung der symptomatischen CMI essentiell. Die invasive Therapie der asymptomatischen CMI sollte aufgrund der Tatsache, dass die Erkrankung häufig symptomatisch wird, ernsthaft erwogen werden.

Introduction

A variety of gastrointestinal and systemic diseases can result in chronic abdominal pain. Chronic mesenteric ischemia (CMI) is a rare but important condition with significant clinical consequences. Typically patients show postprandial ab-

dominal pain, weight loss, and sitophobia. Quite often the appearance of this disorder is confusing with no or unspecific complaints. The diagnosis of CMI is important as CMI has the potential to worsen into acute intestinal angina with bowel infarction.

Etiology

CMI is a fairly uncommon cause for ischemic intestinal events, accounting for 5–8% of the cases. Even if atherosclerosis is a male-weighted disease in patients above 65 years of age, CMI occurs more likely in elderly women with typical cardiovascular risk factors; most of that patients are free of symptoms [1]. 10–20% of all acute mesenteric ischemias are caused by progression of atherosclerotic disease of the superior mesenteric artery (SMA) [2, 3].

Although CMI can result from a variety of conditions, the most common cause is atherosclerotic occlusion or severe stenosis of the splanchnic vessels being responsible for more than 90% of all instances.

Among all intestinal ischemic events, the celiac artery (CA) and SMA are more often affected as the inferior mesenteric artery [4].

End stage renal disease and diabetes can cause diffuse atherosclerosis of the splanchnic arteries with a lack of collateralization, leading to symptoms with only insignificant narrowing. The majority of patients show the classical pattern of occlusion of the proximal segments of the visceral arteries [5]

As 5–15% of CMI are caused by primary and secondary thrombosis of the mesenteric venous system, this condition should also be considered as a cause of CLI [4].

Options in Treatment

Medical Treatment

Medical treatment has its role in mesenteric venous thrombosis where anticoagulation (warfarin, heparine) antiplatelet, or antispasmodic agents reduced mortality and recurrence of acute venous thrombotic events [6]. Sometimes life-long medication with warfarin is indicated [7]. In all other modalities causing CMI, medical therapy does not play a role in causal treatment [8].

Natural History and Indication for Invasive Treatment

The natural course of CMI dictates its therapy. Asymptomatic high-grade arterial stenosis of at least 2 of the 3 splanchnic vessels can be progressive with consequences in malnutrition and unpredictable conversion into acute intestinal infarction. In a study with a long-term follow-up with significant CMI, 86% passed over from a asymptomatic into a symptomatic status with abdominal pain and malnutrition, with some developing bowel infarction and death. Furthermore, acute intestinal ischemia may be the first presentation of CMI in 15–50% of cases [9]. Therefore, definitive invasive therapy should be strongly considered in asymptomatic patients with significant CMI.

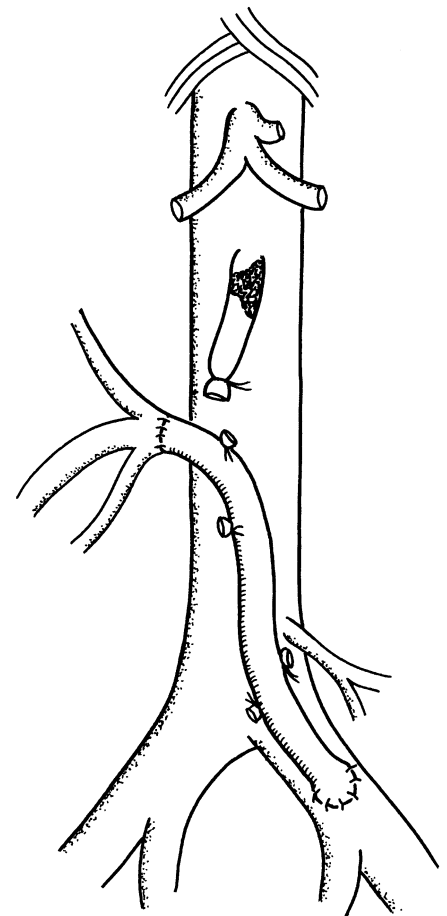


Fig. 1. Autologous iliaco-mesenteric bypass using greater saphenous vein in retrograde position originating from the common iliac artery.

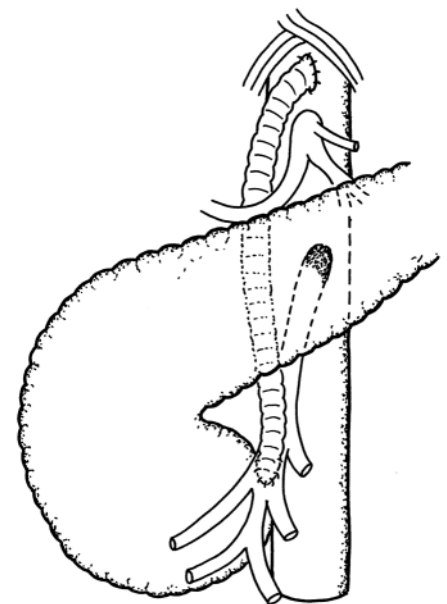


Fig. 2. Alloplastic aorto-mesenteric bypass in antegrade position originating from the suprarenal aorta using a 6 mm Dacrongraft.

As symptomatic CMI patients with weight loss, sitophobia, and abdominal angina are urgently threatened by severe symptoms or progression of the disease, invasive therapy should be started without delay.

Table 1. Published data

Reference	n	Postprandial pain, %	Weight loss, %	Therapy	Female, %	Mean age, years	30-day morbidity, %	30-day mortality, %	Follow-up, years	Survival rate, %	Primary patency, %
Rapp et al., 1986 [11]	67	83	79	TEA/Bypass	76	59	21	7.5	4.4	73.7	93
Moawad et al., 1997 [12]	24	75	58	Bypass	75	58		4.2	4	71	78
Maspes et al., 1998 [13]	23	100	100	PTA	78	72		0	2.3	95	88
Kasirajan et al., 2001 [14]	28			PTA ± stent	75	72	18	10.7	3	81	93
Cho et al., 2002 [15]	25	100	84	TEA/Bypass	60	64	60	0	5.3	88	59
Jimenez et al., 2002 [16]	47	85		Bypass	70	62	66	10.6	5	74	74
Lau et al., 2002 [17]	14	92	50	TEA	86	67	50	0	3	77	92.8

PTA = Percutaneous transluminal angioplasty; TEA = transaortic endarterectomy.

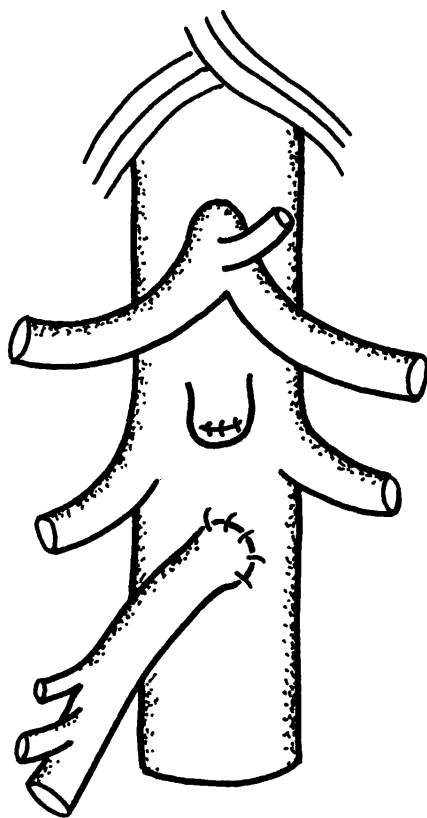


Fig. 3. Transposition of the superior mesenteric artery.

There are no randomized controlled trials in the treatment of CMI. The data in the literature are of case series from different centers with different criteria for patient selection and based on personal experience and preference [10].

Surgical Therapy

Surgical options are local endarterectomy (LEA), transaortic endarterectomy (TEA) (fig. 1, 6), and aorto-visceral bypass (AVBP) (fig. 1, 2) (table 1). TEA has high initial success rate (93%) and a primary patency of $85 \pm 10\%$ after 1 year and $77 \pm 11\%$ after 3 years [17]. Other studies showed unsatisfactory results of TEA with high failure rates due to re-thrombosis and recurrent symptoms [11, 18].

The follow-up results investigated in a group of 25 patients and 41 vessels receiving either AVBP ($n = 24$), LEA ($n = 7$) or TEA ($n = 10$). The overall long-term patency was 52% after 5 and 46% after 10 years with better patency for AVBP and TEA compared to LEA [1]. The reason that LEA might leave with a significant part of the unlaying plaque, i.e. the paraaortic plaque, untreated might be the reason for the worse patency [20]. In cases of concomitant critical limb ischemia (CLI) and occlusive disease of the renal artery, TEA can be effectively used to treat both conditions simultaneously with minimal mortality [20].

AVBP can be achieved as antegrade reconstruction (fig. 2) from the thoracic or supraceliac aorta or as retrograde reconstruction (fig. 1) containing inflow from the infrarenal aorta or common iliac artery [21, 22].

Antegrade bypass (5-year graft patency 78%, 5-year survival rate 71%) was superior to retrograde bypassing, showing a better long-term symptom-free survival and acceptable operative morbidity in a study including 24 patients [12] Another study following up 47 patients after antegrade alloplastic bypass showed a hospital mortality of 11%. Primary, primary-assisted and secondary patency rates after 5 years using

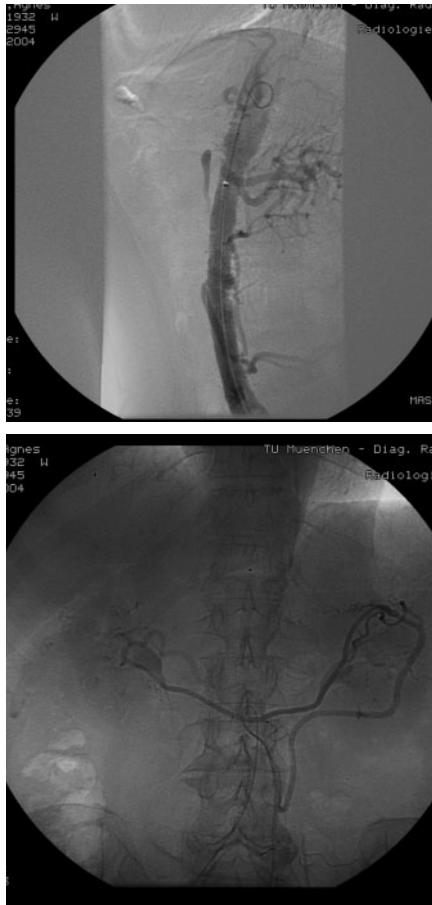


Fig. 4. Symptomatic orificial high-grade stenosis of the SMA with collateralization through the inferior mesenteric artery in a 73-year old female with postprandial pain, weight loss and sitophobia.



Fig. 5. Percutaneous angioplasty of an orificial stenosis of the superior mesenteric artery.

Kaplan-Meier life-table analysis in this study were 69, 94 and 100%, respectively, with a 5-year survival rate of 74% [16].

The question, if alloplastic or autologous bypass has to be preferred is still matter of discussion. In state of concomitant bowel infarction or peritonitis an alloplastic bypass is no option, and autologous grafts should be used due to the life-threatening situation of graft infection. Autologous grafts are more likely to kink, with the risk of early stenosis or occlusion, especially when used as retrograde bypass from the infrarenal aorta or common iliac artery [20].

A surgical option not mentioned yet is the transposition of the SMA (fig. 3). Due to the heavily atherosclerotic disease of the aortic wall, this option might be difficult as satisfying cross-clamping of the aorta might be hardly achieved.

Endovascular Therapy

Even if surgical therapy in CMI represents the gold standard, many studies suggest variability, with perioperative complications occurring in the range of 19–54% and mortality rates ranging between 0 and 17% [14, 23, 24].

Due to its potential for decreased morbidity and mortality minimal invasive therapy (fig. 4, 5), therefore, has become a more acceptable approach for stenotic or occlusive lesions.

The ideal morphology for endovascular treatment is a short stenosis (less than 2 cm) that is located near the ostia of the main splanchnic branches as demonstrated by initial technical success rates of 95% in ostial lesions and 78% in nonostial lesions [25, 26].

Regarding patency of the endovascular therapy, a study following up 23 consecutive patients showed overall clinical success after 27 months in 88%. Two of 23 patients required repeat angioplasty to maintain the initial result [13].

In a later study, primary stenting orificial stenosis of the main splanchnic vessels was analyzed in 25 consecutive patients with CLI. Technical success was achieved in 96%. Major complications occurred in 12%. Stent patency at 6 months was 91%, and clinical benefit after 11 months was 92%, suggesting that primary stenting of CA and SMA is superior in outcome to primary percutaneous transluminal angioplasty (PTA) without stenting, at least in a short-term period [27].

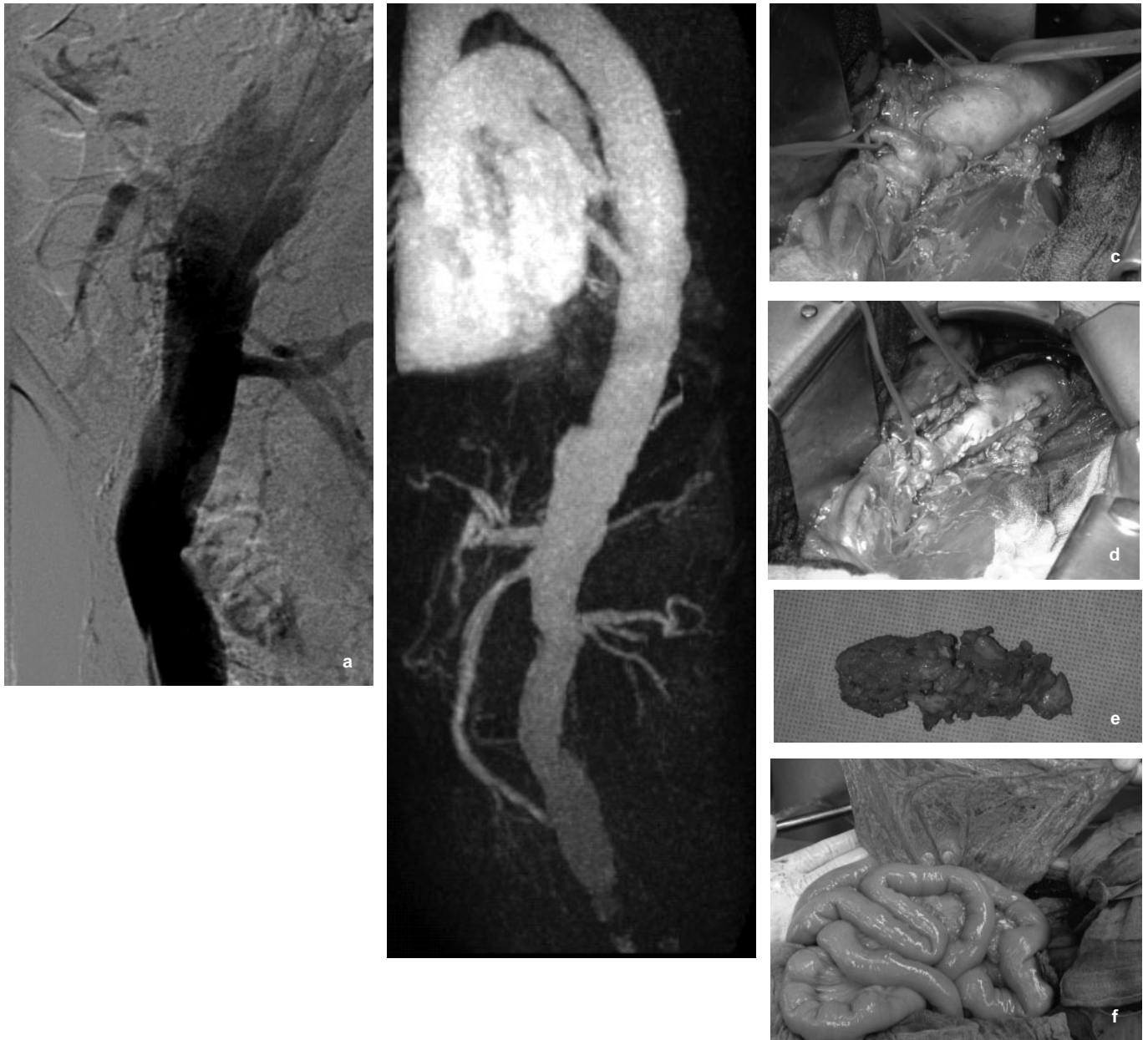


Fig. 6. Symptomatic CMI in a patient with a high grade stenosis of the aortic wall and the superior mesenteric artery. **a** Preoperative angiography, **b** postoperative MR-angiography. **c** Intraoperative situs from a left-sided retroperitoneal access showing the aorta, left renal artery and superior mesenteric artery. **d** Same situs after transaortic local thrombendarterectomy and **e** the plaque material being removed. **f** Small bowel after revascularisation showing good perfusion and hyperemia after restoring the blood supply.

Comparing Studies

A retrospective comparison of endovascular therapy with open surgery was performed, with 28 patients receiving endovascular therapy and 85 patients receiving open surgery [14]. As the authors found no differences in the 2 groups for complications and mortality rates after a 3-year follow up, recurrent symptoms of CMI were more common in the endovascular group compared to the surgically managed group (34 vs. 13%; $p = 0.001$) [8]. Another study [19] compared the surgical

therapy in 33 patients with endovascular therapy in 14 patients using low-profile systems combined with a protection device. Compared to open surgery, stent patients had a lower perioperative major morbidity (30 vs. 0%; $p < 0.01$). Stent patients had 7 times more likely restenosis ($p < 0.01$), 4 times more likely recurrent symptoms ($p < 0.01$), and had to undergo 15 times more likely re-intervention ($p < 0.01$). There was 1 death after 13 months in the stent group due to mesenteric infarction, and 1 patient was successfully converted to open surgery after his second restenosis, having regained 20 pounds

before operation to be in better operative conditions. The authors concluded that endovascular therapy is recommended for patients with severe nutritional depletion or high surgical risk if close follow-up is provided. Open surgery for restenosis should be used if nutritional status and surgical risk can be improved.

In case of stenting of the main splanchnic vessels, a dual platelet aggregation inhibition with clopidogrel (75 mg/day) and aspirin (325 mg/day) is recommended for at least 4 weeks to minimize the risk of early restenosis caused by neointimal hyperplasia [19].

Conclusion

Based on acceptable morbidity and mortality rates and excellent short-term and long-term success rates, surgical therapy represents the actual gold standard in the treatment of CMI. Initial studies confirm promising results for endovascular therapy in the treatment of single- or short-segment vascular disease. Long-term studies comparing endovascular and surgical therapy regarding clinical outcome, vessel patency, morbidity and mortality are awaited. Nutrically depleted high-risk patients seem to profit from endovascular therapy as bridging procedure.

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