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Review Article

Surgical management and outcomes of chronic mesenteric ischemia

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ABSTRACT

Chronic mesenteric ischemia (CMI) is a vascular condition resulting from atherosclerotic blockage of the mesenteric arteries, which reduces blood flow to the intestines. Though rare, CMI is critical to diagnose due to the risk of progression to acute mesenteric ischemia, which can be life-threatening. This review discusses the pathophysiology, clinical presentation, and diagnostic challenges of CMI, emphasizing the need for timely revascularization to improve patient outcomes. Both endovascular and open-surgical techniques are examined, with a focus on patient selection and procedural outcomes. Endovascular interventions, preferred for their lower morbidity, have shown promising results despite a higher risk of restenosis. Comprehensive management, including aggressive medical therapy and lifestyle modifications, remains essential to address the underlying atherosclerosis. Given the complexity of CMI, a multidisciplinary approach is recommended to ensure accurate diagnosis and optimal treatment. This review emphasizes the significance of early diagnosis and customized treatments to improve the quality of life and survival rates in patients with CMI.

Keywords: Chronic mesenteric ischemia, Atherosclerosis, Revascularization, Endovascular therapy, Surgical outcomes

INTRODUCTION

Chronic mesenteric ischemia (CMI) is the most prevalent vascular disorder affecting the mesenteric arteries, yet it remains frequently underdiagnosed in clinical settings.[1] CMI occurs due to the inadequate intestinal blood flow following meals, resulting in an imbalance between oxygen supply and metabolite demand. This hemodynamic disruption is due to atherosclerotic occlusion at the origins of the mesenteric vessels, including the celiac artery (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA), which is responsible for 35–75% of cases.[2,3] Furthermore, other authors have observed that atherosclerotic narrowing of the mesenteric arteries accounts for over 95% of mesenteric arterial stenosis (MAS) cases.[4-6] Non-atherosclerotic causes, such as

vasculitis, fibromuscular dysplasia, segmental arterial mediolysis, and median arcuate ligament syndrome, also contribute to MAS.[2]

Diagnosing CMI clinically is difficult due to the variability of symptoms and the potential lack of correlation between diagnostic tests and clinical findings.[3,7] With the progress in endovascular techniques, open surgical revascularization procedures have been supplanted by less invasive percutaneous stenting.[1]

Symptomatic CMI is rare in clinical settings, representing less than 2% of all atheromatous revascularization procedures. The prevalence of MAS is reported to increase with age, affecting 6% of individuals at age 40, 14% at age 60, and ranging from 18% to 67% in those over 75 years.[5] CMI is observed twice as frequently in women as in men. Angiographic studies have shown asymptomatic MAS in 40% of patients with an abdominal aortic aneurysm, 29% with aortoiliac stenosis, and 25% with lower extremity peripheral arterial disease.[4] The natural progression of symptomatic CMI indicates that between 20% and 50% of patients may develop life-threatening acute mesenteric ischemia.[1]

Comparing the prevalence rates of various vascular diseases, CMI is significantly less common. Peripheral artery disease (PAD) has a prevalence of 3% to 10%, coronary artery disease (CAD) is at 4.5%, and cardiovascular disease (CVD) has a prevalence of 2.4%.[5,8] In contrast, CMI has a prevalence of only 0.03% (30 per 100,000 individuals), highlighting its rarity. This low prevalence is due to the extensive collateral circulation in the gastrointestinal (GI) tract, a remnant of the embryonal network, which provides remarkable flexibility to prevent ischemia caused by stenoses in most cases. This adaptive mechanism plays a vital role in ensuring sufficient blood supply to the gastrointestinal tract, which helps explain the lower incidence of CMI compared to other vascular conditions.[5,9]

METHODS

This study is based on a comprehensive literature search conducted on 19 August 2024, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed surgical management and outcomes of chronic mesenteric ischemia. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

The pathophysiology of CMI is primarily centered on significant stenosis of the mesenteric arteries, usually due to atherosclerotic disease. Traditionally, CMI is thought to occur when at least two mesenteric arteries are stenosed; however, other conditions such as fibromuscular dysplasia, Takayasu's arteritis, and median arcuate ligament syndrome (MALS) also play a role in its development. Additionally, nonocclusive mesenteric ischemia (NOMI) represents a distinct condition that often arises without visible vascular stenosis. While most patients with mesenteric artery stenosis are asymptomatic, some may progress to acute mesenteric ischemia, underscoring the complex and variable nature of this disease.[1]

Clinical signs and symptoms

CMI is defined by ischemic symptoms that persist for at least three months, resulting from insufficient blood supply to the gastrointestinal tract.[5,7] CMI is significantly more common in women, accounting for 70% of cases (Table 1). Include the typical symptoms, ischemic colitis symptoms, and other symptoms of CMI.[1]

Table 1: Initial clinical presentation of patients with CMI.[1]

Typical CMI symptoms	Ischemic colitis symptoms	Other symptoms
Post-prandial abdominal pain (lasting 1–2 hours)	Abdominal pain and cramping	Nausea, vomiting
Fear of eating	Diarrhea, nausea	Sitophobia (aversion to food)
Adapted eating pattern	Gastrointestinal bleeding	Abdominal pain
Weight loss (>20 pounds)	Hematochezia	Right upper quadrant discomfort or pain
Ischemic gastropathy		Weight loss
		Ischemic colitis

Diagnosing of CMI

The diagnosis of CMI hinges on three critical components: medical history, assessment of mesenteric artery stenosis, and evidence of mesenteric ischemia.[5]

Medical history

There are multiple questions to be asked for patients during diagnosis (Table 2).

A recently developed model also highlighted the predictive value of the duration of symptoms and the presence of concomitant cardiovascular disease.[10] However, relying solely on medical history proved to be a poor predictor of CMI.[10-12]

Table 2: Key questions to ask for diagnosis CMI.[5]

S. no.	Key questions to ask
1	Does the pain occur after eating?
2	Is there an absence of pain when not eating?
3	Is there weight loss (and if so, is it due to fear of eating)?
4	Has the patient's eating pattern changed (e.g., more frequent, smaller, and lower-fat meals)?
5	Does the patient experience unexplained diarrhea?

Assessment of stenoses

When evaluating vessel anatomy, it is important to consider the degree of arterial stenosis, the type of stenosis such as atherosclerosis, soft plaques, or external compression, and the presence and type of collateral vessels.[13] The likelihood of CMI significantly depends on which artery is stenosed and to what degree. Since flow resistance in vessels increases with the fourth power of the diameter, the actual reduction in blood flow exceeds 75%.[14] Most stenoses occur at the origin of the arteries, making it crucial to evaluate the remaining artery for atherosclerotic changes when planning treatment. Several techniques are available to assess these parameters, though ideally, information on flow characteristics and pressure changes in the artery would be valuable. However, current methods to assess these are not widely accessible. The main diagnostic tools for vascular assessment include the following.

Duplex ultrasonography is an established screening tool for patients suspected of having MAS. It is patient-friendly, non-invasive, and quite reliable in experienced hands, with an accuracy of over 80%. However, its accuracy is highly operator-dependent, and the reported accuracy figures may not be achievable in centers with less experience or exposure. Duplex ultrasonography is also useful for follow-up in patients who have undergone endovascular treatment. It is important to note that normal systolic and diastolic flow velocities in patients with stents are higher than in normal vessels. If flow velocity in the treated vessel increases over time, physicians should be aware that a critical stenosis may be developing, and any recurrence of pain should prompt further analysis and treatment of the stenosis.[5]

CT angiography (CTA) has become the standard for diagnosing mesenteric vasculature issues. It combines high anatomical resolution with the capability for three-dimensional reconstructions and provides essential information on non-vascular structures as well. Additionally, CTA is investigator-independent and can be performed in most locations even in sick patients. The primary drawback is the high radiation exposure associated with CT angiography. State-of-the-art CTA should include an arterial phase with a slice thickness of no more than 1 mm and venous phase slices of no more than 3 mm.[5]

MR angiography (MRA) offers the potential advantage of no radiation exposure. However, there are limited validation studies comparing MRA to DSA or CTA.[15,16] In one small study, CTA was found to be superior to MRA.[17] With further advancements in MRA technology, both in software and hardware, MRA may regain some ground as a diagnostic tool for MAS that has currently been lost to CTA. One potential advantage of MRA is its ability to measure flow velocity and volume. Some small studies have suggested that reduced blood flow in the portal and superior mesenteric veins could be used as an indirect measure of mesenteric hypoperfusion. However, commonly used stents are often not compatible with standard MR angiography imaging due to the creation of various artifacts and loss of signal, making it impossible to assess stented arterial segments.[5] Currently, MRA serves as a secondary option after CTA, particularly when CTA is contraindicated, such as in cases of renal failure.

Digital subtraction angiography (DSA) was long considered the gold standard for assessing mesenteric vasculature. For diagnostic purposes, it has been replaced by CTA and is now primarily used for treating MAS via the endovascular route. In our experience, DSA still has some value in the preoperative evaluation of patients with MALS, as it can show the length of the compressing ligament and demonstrate the influence of respiration on the stenosis, guiding the retroperitoneal endoscopic approach.[18]

Ischemia functional testing

Two functional tests have been evaluated for patients with CMI, partial pressure of carbon dioxide (PCO_2) tonometry and visible light spectroscopy (VLS).[5] PCO_2 tonometry can be used with provocative tests such as eating or exercise, but it is labor-intensive and not widely available. VLS, on the other hand, is more widely available and can be used during endoscopy, though it has limitations including poor specificity, uncertainty about reproducibility, and a short measurement time that complicates its use with provocative tests. While small studies on serological markers, particularly i-FABP, show promise, there is a lack of large validation studies.[19] In cases where otherwise unexplained ulcers are present in the stomach, duodenum, or right colon, these can be considered indicative of ischemia, making functional ischemia testing unnecessary in such scenarios.

Management and outcomes

Conservative treatment: medical therapy

Atherosclerosis is the most prevalent cause of CMI. Management of atherosclerosis progression primarily involves aggressive medical therapy to address hypertension, hyperlipidemia, and diabetes, and to implement antiplatelet therapy, along with smoking cessation and lifestyle modifications. Current guidelines recommend that patients with symptomatic CMI undergo

imaging studies and be evaluated for potential revascularization.[3,20]

Revascularization

Successful revascularization in patients with occlusive or stenotic CMI can relieve symptoms, improve quality of life (QoL), restore normal weight, and enhance survival by preventing bowel infarction associated with acute-on-chronic CMI. The primary challenge is identifying the ideal candidates who will benefit most from the procedure. Revascularization is typically recommended for patients with critical stenoses (≥70%) in multiple mesenteric vessels who exhibit classic CMI symptoms. For those with single-vessel disease and atypical symptoms, a thorough investigation into alternative causes is required. In cases of suspected CMI with single-vessel disease and/or atypical symptoms, a multidisciplinary team should reach a consensus diagnosis of occlusive/stenotic CMI before initiating revascularization therapy. A definitive diagnosis of CMI is confirmed retrospectively when successful revascularization results in symptom relief.[1]

Catheter-based endovascular therapy

Catheter-based endovascular revascularization techniques are preferred over open surgery due to their lower procedural morbidity. Stenoses located at the aorto-ostial site are common causes of CMI, and the technical considerations for stent placement are like those for renal artery interventions (Figure 1).[1] Stent placement offers better long-term patency compared to balloon angioplasty alone.[9,21]

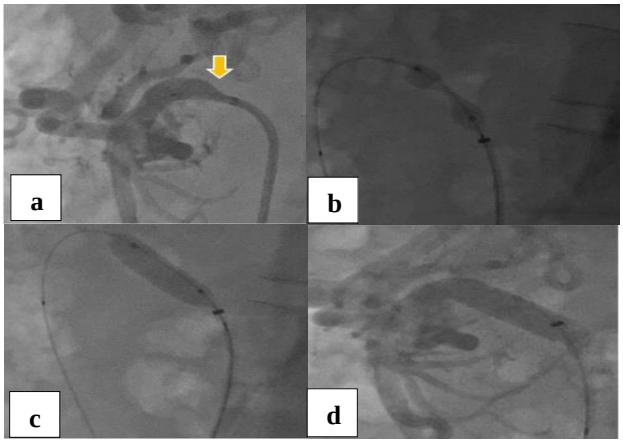

Figure 1: Significant celiac artery stenosis in a patient with chronic mesenteric ischemia and celiac artery stent procedure: (a) selective celiac artery angiography via the femoral artery, lateral view shows 80% stenosis of the ostial celiac artery (arrow), (b) balloon angioplasty, (c) balloon expandable bare metal stent placement, and (d) final angiography.[1]

Due to the rarity of this disease and diagnostic challenges, there are no clinical trials directly comparing surgical versus catheter-based revascularization strategies for CMI. Data from the Mayo Clinic on 229 consecutive CMI patients showed that surgical patients experienced greater morbidity and longer hospitalization compared to those undergoing endovascular procedures. Although endovascular therapy had a higher restenosis rate, there was no difference in secondary patency between surgical and stented patients.[9] A meta-analysis of 100 observational studies (22 comparatives, 78 non-comparatives; 18,726 patients) compared open surgery with catheter-based endovascular therapy. Open surgery was linked to a higher risk of in-hospital complications (relative risk 2.2) and a non-significant increase in 30-day mortality. However, surgical revascularization demonstrated a lower risk of 3-year recurrence rates (relative risk 0.47) and comparable 3-year survival rates.[22]

A propensity-matched analysis from the National Inpatient Sample database found that major adverse cardiovascular and cerebrovascular events, as well as composite in-hospital complications, occurred significantly less frequently after endovascular therapy compared to open surgery (8.6% versus 15.9%; $p<0.001$ and 15.3% versus 20.3%; $p<0.006$). Endovascular therapy was also associated with lower median hospital costs ($20,807.00 versus $31,137.00; $p<0.001$) and shorter hospital stays (5 versus 10 days; $p<0.001$) compared to surgery.[23]

Procedural complications of endovascular therapy typically relate to vascular access and may include hematomas, pseudoaneurysms, dissection, abrupt occlusion, and retroperitoneal bleeding. In a Mayo Clinic series of 156 patients, serious complications occurred in 7%, including branch perforation, distal embolization, vessel dissection, and stent embolization.[24] Restenosis has been a significant issue with endovascular intervention.[1]

Surgical revascularization

Historically, revascularization for MAS has been conducted through open surgery, either via endarterectomy or bypass grafting. Patients with CMI often present with systemic atherosclerosis and malnutrition, which increase their risk for surgical complications. As a result, the perioperative mortality rate for these procedures ranges from 3.5% to 15%.[25]

Over the past twenty years, there has been a substantial increase in the number of endovascular procedures conducted for CMI in the USA.[23]

Clinical outcomes after treatment

The European Society for Vascular Surgery (ESVS) guidelines recommend regular follow-up after therapy for chronic mesenteric ischemia (CMI) to monitor for symptomatic restenosis. While routine imaging may identify restenosis, the benefits of treating asymptomatic restenosis are not well established. Post-revascularization, antiplatelet therapy is advised, with dual antiplatelet therapy considered for 3–12 months.[26] In-stent stenosis is

observed in 28–36% of patients treated endovascularly within 2 years, compared to 0–25% after surgical revascularization.[20,27,28]

Independent predictors of restenosis include endovascular revascularization, a history of previous mesenteric intervention, female gender, and a small (<6 mm) SMA diameter.[26] Severe mesenteric calcification, occlusions, longer lesions, and small vessel diameters are associated with increased risks of distal embolization, restenosis, and reinterventions following endovascular revascularization.[28] Surgical revascularization offers a superior long-term patency rate compared to endovascular approaches, with a cumulative odds ratio of 3.57 (95% CI 1.82–6.87, p=0.0002).[29]

Immediate symptom relief is reported in 90–98% of patients undergoing surgical revascularization, and this high rate of relief is maintained after 5 years (89–92%). For endovascular revascularization, immediate symptom relief is achieved in 87–95% of patients, with symptom relief rates of 61–88% at 3 years and 51% at 5 years.[27] A retrospective analysis of prospectively collected data (10,920 endovascular vs. 4,555 surgical patients) showed that endovascular revascularization is associated with significantly lower in-hospital mortality (2.4%), shorter hospital stays by about 10 days, and reduced hospitalization costs, saving approximately $25,000 compared to surgical revascularization.[30]

Future directions

Future research on CMI should focus on optimizing diagnostic and therapeutic strategies, particularly considering emerging technologies and treatment modalities. Investigating advancements in imaging techniques, such as high-resolution MRI and novel biomarkers, could enhance early detection and more accurate assessment of CMI. Additionally, exploring personalized treatment approaches that tailor revascularization strategies based on patient-specific factors may improve outcomes. Future studies should also evaluate the long-term efficacy of endovascular versus surgical interventions, including the impact of new devices and techniques on restenosis rates and overall patient survival. Addressing these areas will be crucial for advancing the management of CMI and improving patient quality of life.

CONCLUSION

Effective management of CMI requires a multidisciplinary approach, integrating aggressive medical therapy with timely revascularization. Endovascular techniques are preferred for their lower morbidity; though surgical revascularization offers superior long-term patency. Early diagnosis and appropriate treatment are crucial to improving patient outcomes and preventing progression to acute ischemia.

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