10 - 340 Mesenteric Vascular Insufficiency

340 Mesenteric Vascular Insufficiency

maintains the tract patency and reduces the likelihood of recurrent infection and inflammation. Once the inflammation is reduced (4–6 weeks), the exact relationship of the fistula tract to the anal sphincters can be ascertained. Two general approaches are com mon: sphincter-cutting procedures and sphincter-sparing proce dures. A simple fistulotomy or fistulectomy is a sphincter-cutting procedure and is usually performed for intersphincteric and low (less than one-third of the muscle) transsphincteric fistulas. In a systematic review, sphincter-cutting procedures were associated with a 94% success rate and a 13% rate of mild fecal incontinence. Sphincter-sparing surgery is usually performed for a higher trans sphincteric fistula. These procedures include an anorectal advance ment flap, ligation of the internal fistula tract (LIFT procedure), or insertion of fibrin or cyanoacrylate glue. Sphincter-preserving procedures are associated with up to 78% success rates with no incidence of fecal incontinence.

Fistulizing disease of the anus is common in Crohn’s disease, and the use of mesenchymal stem cell therapy may improve heal ing rates of fistula associated with Crohn’s disease. The ADMIRE study examined the use of allogeneic expanded adipose-derived mesenchymal stem cells in the treatment of complex perianal fistula in Crohn’s disease. The study included 212 patients randomized to stem cell therapy or placebo. Fistula remission rates at 52 weeks were significantly higher with the use of stem cell therapy over pla cebo (59 vs 42%, respectively). Further studies are currently being performed to elucidate the benefits of stem cell therapy in perianal fistula. As with any stem cell therapy, the cost can be prohibitive to its standard use. PART 10 Disorders of the Gastrointestinal System ■ ■ANAL FISSURE Incidence and Epidemiology Anal fissures occur at all ages but are more common in the third through the fifth decades. The preva lence is equal in males and females. It is associated with constipation, diarrhea, infectious etiologies, perianal trauma, and Crohn’s disease. Anatomy and Pathophysiology Trauma to the anal canal occurs following defecation. This injury occurs in the anterior or, more com monly, posterior anal canal. Irritation caused by the trauma to the anal canal results in an increased resting pressure of the internal sphincter. The blood supply to the sphincter and anal mucosa enters laterally. Therefore, increased anal sphincter tone results in a relative ischemia in the region of the fissure and leads to poor healing of the anal injury. A fissure that is not in the posterior or anterior position should raise suspicion for other causes, including tuberculosis, syphilis, Crohn’s disease, and malignancy. Presentation and Evaluation A fissure can be easily diagnosed on history alone. The classic complaint is pain, which is strongly asso ciated with defecation and is relentless. The bright red bleeding that can be associated with a fissure is less extensive than that associated with

hemorrhoids and is usually only noted on wiping. On examina tion, most fissures are located in either the posterior or anterior posi tion. A lateral fissure is worrisome because it may have a less benign nature, and systemic disorders should be ruled out. A chronic fissure is indicated by the presence of a hypertrophied anal papilla at the proxi mal end of the fissure and a sentinel pile or skin tag at the distal end. Often the circular fibers of the hypertrophied internal sphincter are visible within the base of the fissure. TREATMENT Anal Fissure The management of the acute fissure is conservative. Stool softeners for those with constipation, increased dietary fiber, topical anes thetics, glucocorticoids, and sitz baths are prescribed and will heal 60–90% of fissures. Chronic fissures are those present for >6 weeks. These can be treated with modalities aimed at decreasing the anal canal resting pressure including nifedipine ointment applied three

times a day and botulinum toxin type A, up to 20 units, injected into the internal sphincter on each side of the fissure. Both treatments are associated with a fissure healing rate of >80%. Surgical manage ment includes anal dilatation and lateral internal sphincterotomy. Usually, one-third of the internal sphincter muscle is divided; it is easily identified because it is hypertrophied. Recurrence rates from medical therapy are higher, but this is offset by a risk of inconti nence following sphincterotomy. Lateral internal sphincterotomy may lead to incontinence more commonly in women. Acknowledgment The author thanks Cory Sandore for providing some illustrations for this chapter. ■ ■FURTHER READING Bharucha AE et al: Surgical interventions and the use of device-aided therapy for the treatment of fecal incontinence and defecatory disor ders. Clin Gastroenterol Hepatol 15:1844, 2017. Daniels L et al: Randomized clinical trial of observation versus antibi otic treatment for a first episode of CT-proven uncomplicated acute diverticulitis (DIABOLO trial). BJS 104:52, 2017. Guttenplan M: The evaluation and office management of hemor rhoids for the gastroenterologist. Curr Gastroenterol Rep 19:30, 2017. Hall J et al: The American Society of Colon and Rectal Surgeons Clinical Practice Guidelines for the treatment of left-sided colonic diverticulitis. Dis Colon Rectum 63:728, 2020. Panes J et al: Long-term efficacy and safety of stem cell therapy (Cx601) for complex perianal fistulas in patients with Crohn’s disease. Gastroenterology 154:1334, 2018. Prichard D, Bharucha AE: Management of pelvic floor disorders: Biofeedback and more. Curr Treat Options Gastroenterol 12:456, 2014. Salfity HV et al: Minimally invasive incision and drainage technique in the treatment of simple subcutaneous abscess in adults. Am Surg 83:699, 2017. Strate LL, Morris AM: Epidemiology, pathophysiology, and treat ment of diverticulitis. Gastroetenterology 156:1282, 2019. Sugrue J et al: Sphincter-sparing anal fistula repair: Are we getting better? Dis Colon Rectum 60:1071, 2017. Tursi A: Dietary pattern and colonic diverticulosis. Curr Opin Clin Nutr Metab Care 20:409, 2017. Daniel Willie-Permor, Mahmoud Malas

Mesenteric Vascular

Insufficiency INTESTINAL ISCHEMIA ■ ■INCIDENCE AND EPIDEMIOLOGY Intestinal ischemia occurs when splanchnic perfusion fails to meet the metabolic demands of the intestines, resulting in ischemic tissue injury. Mesenteric ischemia affects 2–3 people per 100,000 with an increasing incidence in the aging population. Mortality with acute presentation remains high (between 50 and 80%), and early diagnosis and prompt intervention are crucial in improving clinical outcomes. Intestinal isch emia is further classified into chronic mesenteric ischemia (CMI) and acute mesenteric ischemia (AMI). CMI is secondary to multiple major visceral arterio-occlusive disease with involvement of the superior mesenteric artery (SMA) most worrisome. AMI is most associated with

(1) arterio-occlusive mesenteric ischemia, (2) nonocclusive mesenteric ischemia, and (3) mesenteric venous thrombosis. CMI is the failure to achieve normal postprandial hyperemic intes tinal blood flow. This occurs due to an imbalance between supply and demand of oxygen metabolites to the intestinal tract similar to cardiac angina. CMI occurs due to significant atherosclerotic disease leading to the narrowing of the SMA and/or celiac artery origins. AMI is the occurrence of an abrupt cessation of mesenteric blood flow, usually embolic or thrombotic in nature. Approximately 50% of AMI is due to embolus to the mid to distal SMA. The embolus etiology includes atrial fibrillation, recent myocardial infarction, soft atherosclerotic plaque, infective endocarditis, valvular heart disease, and recent cardiac or vascular catheterization. Approximately 25–30% of the cases are characterized by an acute-on-chronic thrombosis in patients with preexisting mesenteric atherosclerosis. Thrombotic occlusion most commonly occurs at areas of severe atherosclerotic narrowing at the SMA and the celiac artery. Nonocclusive mesenteric ischemia represents 20% of the cases and is secondary to intestinal ischemia when subjected to acute hemody namic instability. Patients above the age of 50, especially those with coexisting conditions like myocardial infarction, congestive heart failure, aortic insufficiency, and renal or liver disease, who are also undergoing cardiovascular surgery, face the highest risk. Hypovolemia, shock, and use of vasoconstrictive agents (e.g., digoxin, α-adrenergic agonists, cocaine) can precipitate ischemia in these patients. It is the most prevalent gastrointestinal disease complicating cardiovascular surgery. The incidence of ischemic colitis following elective aortic repair is 5–9%, and the incidence triples in patients following emergent repair. Mesenteric venous thrombosis accounts for <10% of cases and is generally precipitated by a hypercoagulable state due to an underlying inherited disorder such as factor V Leiden, prothrombin mutation, protein S deficiency, protein C deficiency, antithrombin deficiency, and antiphospholipid syndrome. It may also occur as a result of acquired thrombophilia in malignancies, hematologic disorders, and use of oral contraceptives. ■ ■ANATOMY AND PATHOPHYSIOLOGY The blood supply to the intestines is supplied by the celiac artery, SMA, and inferior mesenteric artery (IMA) (Fig. 340-1). Extensive collat eralization occurs between major mesenteric trunks and branches of the mesenteric arcades. Collateral vessels within the small bowel are numerous and meet within the duodenum and the bed of the pancreas. Collateral vessels within the colon meet at the splenic flexure and descending/sigmoid colon. These areas, which are inherently at risk for decreased blood flow, are known as Griffiths’ point and Sudeck’s point, respectively, and are the most common locations for colonic ischemia (Fig. 340-1, shaded areas). The splanchnic circulation can receive up to 30% of the cardiac output. Protective responses to prevent intestinal ischemia include abundant collateralization, autoregulation of blood flow, and the ability to increase oxygen extraction from the blood. Occlusive ischemia is a result of disruption of blood flow by an embolus or progressive thrombosis in a major artery supplying the intestine. In >75% of cases, emboli originate from the heart and pref erentially lodge in the SMA just distal to the origin of the middle colic artery. Progressive stenosis of typically two of the three major vessels supplying the intestine is required for the development of chronic intestinal angina. Involvement of the SMA is most worrisome. Non occlusive ischemia is disproportionate mesenteric vasoconstriction (arteriolar vasospasm) in response to a severe physiologic stress such as shock. If left untreated, early mucosal stress ulceration will progress to full-thickness injury. ■ ■PRESENTATION, EVALUATION,

AND MANAGEMENT Patients with CMI typically present with insidious onset of symptoms and classically present with recurrent episodes of acute dull, crampy, postprandial epigastric pain, which has also been referred to as “intesti nal angina.” Patients also describe fear of eating

resulting in weight loss.

Left phrenic a. Aorta Right phrenic a. Splenic a. Griffiths’ point Celiac trunk Pancreaticoduodenal a. Arc of Riolan SMA IMA Marginal a. IIA Sudeck’s point Hemorrhoidal aa. Superior Middle Inferior CHAPTER 340 FIGURE 340-1 Blood supply to the intestines includes the celiac artery, superior mesenteric artery (SMA), inferior mesenteric artery (IMA), and branches of the internal iliac artery (IIA). Griffiths’ and Sudeck’s points, indicated by shaded areas, are watershed areas within the colonic blood supply and common locations for ischemia. Mesenteric Vascular Insufficiency Chronic diarrhea may also be noted. Duration of symptoms is typically 6–12 months. Physical examination will often reveal a malnourished patient with other manifestations of atherosclerosis. Prompt diagnostic investigation is necessary to rule out gastrointestinal malignancies and other possible causes, and may include accelerated evaluation via esophagogastroduodenoscopy, colonoscopy, abdominal computed tomography (CT) scan, and abdominal ultrasound examination. Duplex ultrasound has gained popularity as a screening tool for evaluation of the mesenteric vessels due to high sensitivity and speci ficity. Mesenteric duplex scan demonstrating a high peak velocity of flow in the SMA is associated with an ~80% positive predictive value of mesenteric ischemia. More significantly, a negative duplex scan vir tually precludes the diagnosis of mesenteric ischemia. It is important to perform the test while the patient is fasting because the presence of increased bowel gas prevents adequate visualization of flow distur bances within the vessels or the lack of a vasodilation response to feed ing during the test. Thin-sliced CT angiography is the gold standard diagnostic tool in assessing the degree of atherosclerotic disease of the aortic and visceral vessels as well as evaluating the bowels. Venous phase can also help diagnose mesenteric vein thrombosis. The management of CMI includes aggressive medical therapy of atherosclerotic disease including cessation of smoking and antiplatelet and lipid-lowering medications. A full cardiac and vascular evaluation should be performed before intervention on CMI. Treatment, involving either endovascular, open surgical, or hybrid revascularization, should be individualized based on the patient’s comorbidities and anatomy. Endovascular revascularization involves targeted vessel treatment with visceral stents with the SMA anatomy being the key determinant. The revascularization of the celiac axis and IMA represents secondary focal points, offering potential therapeutic benefits. This approach becomes particularly relevant when the SMA is deemed unsuitable for intervention or when technical outcomes are deemed suboptimal. Open revascularization involves antegrade bypass from the supraceliac aorta or retrograde bypass typically from

the common or external iliac arteries with a synthetic or autogenous graft to the targeted vessels, usually the SMA and/or celiac artery. In patients with suitable lesions requiring revascularization, an endovas cular approach is recommended as the first-line therapy. It is especially favorable for short segment stenosis with minimal to moderate calci fication or thrombus. Angioplasty with endovascular stenting in the treatment of CMI is associated with an 80% long-term success rate. Open revascularization should be considered in patients with lesions not amenable to endovascular treatment, such as severe calcification, longer lesions, small vessel diameter, or failed endovascular interven tions, or in a specific subset of younger, healthier patients, in whom the potential long-term advantages may outweigh the heightened perioperative risks. Retrograde open mesenteric stenting (ROMS) is a hybrid approach, combining aspects of both traditional open surgical bypass and percutaneous endovascular therapy. ROMS is primarily indicated for treating mesenteric ischemia, both acute and chronic, particularly in cases where conventional

endovascular or open surgical approaches are not feasible or have been unsuccessful. This includes scenarios where there is significant stenosis or occlusion in the mesen teric arteries that cannot be adequately addressed through less invasive percutaneous methods from the aorta or in situations where immediate direct visualization and potential resection of the bowel are necessary due to the presence of necrosis or perforation. The technique involves a laparotomy that allows for direct bowel assessment and the exposure and stenting of the SMA using a retrograde approach through the midsegment of the SMA, thus allowing for immediate revascularia tion, assessment of bowel viability, and, if necessary, bowel resection. ROMS also offers the advantage of faster operative times compared to traditional bypass and avoids the placement of prosthetic material in potentially contaminated peritoneal cavities, which is a significant concern in the setting of bowel necrosis and peritonitis.

PART 10 Disorders of the Gastrointestinal System Acute intestinal ischemia remains one of the most challenging diagnoses. The mortality rate of AMI is >50%. The most significant indicator of survival is the timeliness of diagnosis and treatment. An overview of diagnosis and management of each form of intestinal isch emia is given in Table 340-1. AMI resulting from arterial embolus or thrombosis presentation is nonspecific and requires a high index of suspicion for the diag nosis. The most common complaint, occurring in 95% of cases, is severe, acute, nonremitting abdominal pain that is strikingly out of proportion to the physical findings. The reason behind the pain being TABLE 340-1 Overview of the Management of Acute Intestinal Ischemia KEY TO EARLY

DIAGNOSIS TREATMENT OF UNDERLYING CAUSE TREATMENT OF SPECIFIC LESION CONDITION Arterio-occlusive mesenteric ischemia 1. Arterial embolus Computed tomography angiography (CTA) Early laparotomy Anticoagulation Cardioversion Thrombectomy Broad-spectrum antibiotics 2. Arterial thrombosis Duplex ultrasound CTA Anticoagulation Broad-spectrum antibiotics Resuscitation Mesenteric venous thrombosis Venous thrombosis CTA with venous phase Anticoagulation Resuscitation Nonocclusive mesenteric ischemia Vasospasm: Hypoperfusion: CT Resuscitation Support cardiac output Avoid vasoconstrictors Broad-spectrum antibiotics Source: Modified from GB Bulkley, in JL Cameron (ed): Current Surgical Therapy, 2nd ed. Toronto, BC Decker, 1986.

disproportionate to the clinical findings is that ischemia initiates from the mucosal layer and progresses toward the serosal layer. This may be associated with nausea (44%), vomiting (35%), diarrhea (35%), and blood per rectum (16%). Later findings will demonstrate peritonitis and cardiovascular collapse. Specific clinical features can help differen tiate the underlying etiology, whether embolic or thrombotic. Patients with embolic ischemia are typically older adults with underlying con ditions that predispose to embolism such as atrial fibrillation, prior embolic event, or recent infective endocarditis. Thrombotic ischemia typically presents as an acute occlusion in patients with underlying atherosclerotic disease who may have been previously diagnosed with CMI. AMI is a surgical emergency requiring emergent admission to a monitored bed or intensive care unit for resuscitation with fluids and broad-spectrum antibiotics in addition to further evaluation. If the diagnosis of intestinal ischemia is being considered, consultation with a surgical service is necessary. Often the decision to operate is made on a high index of suspicion from the history and physical exam despite normal laboratory findings. In patients with suspected AMI, a CT angiography with 1-mm or thinner cuts should be used to detect mesenteric arterial occlusive disease most likely from embolic or thrombotic etiology and is the gold standard. Additional diagnostic modalities that may be useful in diagnosis, but should not delay surgi cal therapy,

include electrocardiogram (ECG) and echocardiogram. Patients with AMI should be given a heparin bolus immediately and started on a therapeutic heparin drip. Correction of electrolyte abnor malities and empiric broad-spectrum antibiotic therapy should also be initiated instantly. If CT angiography verifies acute embolic occlusion of the SMA, sur gical exploration should not be delayed. The goal of operative explora tion is to resect compromised bowel, restore blood supply, and preserve all viable bowel. The entire length of the small and large bowel begin ning at the ligament of Treitz should be evaluated. The SMA artery should be localized, typically at the mesocolon of the transverse colon. A transverse arteriotomy of the SMA should be made with removal of embolus with an embolectomy Fogarty catheter passed in a retrograde and antegrade manner to restore blood flow. In the case of SMA occlu sion where the embolus usually lies just proximal to the origin of the middle colic artery, the proximal jejunum is often spared while the remainder of the small bowel up to the transverse colon may become ischemic. Nonviable bowel should be resected. Questionable bowel TREATMENT OF SYSTEMIC CONSEQUENCE Laparotomy Embolectomy Assess viability and resect nonviable bowel Anticoagulation Resuscitation Broad-spectrum antibiotics Emergent surgical intervention Assessment of bowel Endovascular approach: thrombolysis, angioplasty, and stenting Endarterectomy/thrombectomy or vascular bypass Assess viability and resect nonviable bowel Anticoagulation Resuscitation Broad-spectrum antibiotics Emergent surgical intervention Assessment of bowel Anticoagulation Hypercoagulable workup Anticoagulation Resuscitation Broad-spectrum antibiotics Support cardiac output Avoid vasoconstrictors Vasospasm Intraarterial vasodilators Hypoperfusion Assess viability and resect dead bowel Resuscitation Broad-spectrum antibiotics Support cardiac output Avoid vasoconstrictors

---

Revision #1
Created 2026-01-06 16:34:35 UTC by Omar Ayman
Updated 2026-01-06 16:34:35 UTC by Omar Ayman