

Ischemic Colitis Complicating Major Vascular Surgery

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Ischemic colitis is a well-described complication of major vascular surgery, mostly following open abdominal aortic aneurysm (AAA) repair and endovascular aneurysm repair (EVAR), but also with aortoiliac surgery, aortic dissection, and thoracic aneurysm repair [1,2]. Although Boley and colleagues [3] described ischemia of the colon in 1963 as a reversible process secondary to vascular occlusion, the term was not coined until 1966, when Marston and colleagues [4] described its three stages of evolution (transient ischemia, late ischemic stricture, and gangrene), along with the natural history of the disease. Furthermore, the development of colonic ischemia as a result of major vascular and aortic surgery has only been well described since the 1970s [5–9]. The original reports were scattered and mostly consisted of autopsy studies, depicting not only the high mortality associated with this condition but also, in retrospect, the relative lack of insight as to explanations for its onset and progression. Unfortunately, recent outcomes have not shown much improvement. Luckily, the overall incidence remains low, estimated at between 0.6% and 3.1%, with higher rates attributed to ruptured aneurysms, open repair, and emergent surgery [10]. Following the development of ischemic colitis, mortality has been reported to be as high as 67%, highlighting the need for rapidly identifying the commencement of symptoms and, perhaps more importantly, those patients at risk, in attempt to prevent its onset [10,11]. Further emphasizing the seriousness of the development of this condition, in a study of 222 aortic aneurysm repairs, colonic ischemia was the most common cause of death, even more than multisystem organ failure and myocardial infarction [12]. Thus, the physician tasked with treating patients who have vascular disease needs to be well versed in this condition, not only to recognize its occurrence and course but to be aware of how to manage this highly lethal condition.

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This article reviews the causes, presentation, and diagnostic strategies of colonic ischemia. It also covers the operative management and outcomes for bowel resection and vascular repair. Finally, some of the research regarding alternative options for diagnosing this condition is discussed.

Pathogenesis

Simply stated, ischemic colitis occurs when blood flow is interrupted and supply does not equal colonic demand. Most commonly, this interruption is not associated with occlusion of any of the major abdominal vessels, because the collateral circulation of the colonic blood supply from the superior mesenteric, inferior mesenteric, hypogastric, and meandering mesenteric arteries is extensive. Regardless of the cause, the earliest dysfunction is seen at the mucosal level, furthest away from the vasa recta, creating a secondary disruption of the mucosal barrier that can lead to bacterial translocation and sepsis [13]. Ischemic colitis may occur with many different conditions, including embolism from cardiac disease, low-flow states such as congestive heart failure, sepsis, and vasopressor use. However, major vascular surgery, especially involving the abdominal aorta, is a well-known risk factor [14]. As such, ischemic colitis is recognized as a potential complication of both open AAA repair and EVAR, with its development associated with high mortality not only from the colonic insult but from the subsequent physiologic derangements that follow [15]. When occurring in the context of major vascular repair, the pathogenesis may be unique in that, in many cases, an isolated interruption of inferior mesenteric artery (IMA) blood flow may be the sole underlying cause [8]. Following open repair, many different variables can play major roles in its onset, including larger perioperative fluid shifts, cross-clamping of the aorta, compressive retroperitoneal hematomas, and prolonged hypotension [16]. On the other hand, the exact pathogenesis following endovascular repair has yet to be elicited completely. Although multiple theories exist, the most common one involves embolization of cholesterol plaque following implantation of the stent graft or manipulation of catheters and guidewires within the aneurysm sac [17].

Regardless of the method of repair, presentation of the underlying vascular disease also significantly affects the rate and prevalence of ischemic colitis, with 60% of ruptured AAAs developing colonic ischemia [18]. Other factors that have been shown to have a higher association with the development of colonic ischemia are increased intraoperative blood loss, hypotension, and prior pelvic radiation [12,19]. Because radiation damage is progressive and cumulative over time, it may be a precipitating factor not only in early disease but also with chronic changes such as stricture. Damage occurs directly to the bowel wall, in addition to causing secondary hyaline deposition and fibrosis of the microvasculature at the arteriole level [20]. Thus, prior radiation therapy may decrease the major and collateral

circulation of the colonic blood supply that is at most risk during aortic repair, in addition to creating a primary impairment to the colon [19].

On the contrary, established collateral circulation appears to portend a protective effect, with one study showing that a patent meandering mesenteric artery (arc of Riolan) was not associated with any cases of ischemia [6]. Clearly, however, the most important factor in curtailing its morbid effects is to prevent, or at least minimize, all potential variables that can lead to secondary injury.

Diagnosis

Regrettably, the clinical manifestations of ischemic colitis are inconsistent. Most commonly, patients present with abdominal pain, fever, distension, and diarrhea anywhere from hours to days following surgery [16]. The diarrhea may be bloody or nonbloody, depending on the location, degree, and extent of the colon affected. Abdominal examination may show widely variable findings, ranging from mild localized tenderness to diffuse peritonitis. Rectal examination may be completely normal or may demonstrate blood, ranging from bright red to melena. Laboratory examination is often noteworthy for a leukocytosis and metabolic acidosis, and may also present with significant electrolyte abnormalities.

Although any area of the colon may be affected, the watershed areas of the rectosigmoid (Sudeck's point) and splenic flexure (Griffith's point) are commonly involved because of the often incomplete anastomoses of the marginal artery in these locations. The next most common area afflicted is the cecum, secondary to low blood flow in the terminal branches of the ileocolic artery combined with varying competency of the right colic artery [21]. Longo and colleagues [22] found a much higher rate of right colonic involvement than most other studies did, with 46% of their 47 patients having this portion affected. Unfortunately, as reported in his large study of almost 5000 Veterans Administration patients, regardless of the presentation, the mean time to diagnosis was 5.5 days following aortic surgery (range 1–21 days) [14]. This time was secondary to not only the slow evolution of the disease but also, in other cases, to the lack of acumen of the caretaker. Therefore, identifying patients at the onset of their clinical course entails a high index of suspicion. Complete history and physical examination, focusing on the abdominal examination and evaluation for peritoneal signs, is of utmost importance. Any peritonitis mandates emergent exploratory laparotomy for evaluation for bowel viability. Again, because of the high morbidity and mortality once ischemic colitis following major vascular surgery precipitates, the ultimate goal remains to avoid this condition altogether.

In the absence of that, early diagnosis remains the key, and has been the work of several investigators. The early setting may show few clinical symptoms, leading to a delay in diagnosis and increasing the likelihood of complications, or even death. Efforts have therefore been made to identify

changes on both a clinical and biochemical front. Although some of these methods are still often only investigational or not yet widely used, they represent a potentially important adjunct to the currently available diagnostic tools. One of these markers is plasma D-lactate. In a study of 12 patients who had histologically confirmed ischemic colitis after undergoing either elective or emergent open aortic aneurysm surgery, plasma D-lactate was found to be elevated in patients as early as 2 hours postoperatively, with a peak on days 1 and 2 [13]. Lange and colleagues [23] had similar findings, with plasma lactate exceeding the normal level in all 20 patients who had mesenteric ischemia; however, sensitivity for the entire cohort with acute abdominal disease was much lower, at less than 50%.

Although early serum markers are promising and may prove effective, intraoperative assessment may lead to even earlier manifestations and may provide the surgeon with the opportunity to address the issue before leaving the operating room. In a study of 22 patients sustaining a ruptured aneurysm, laser Doppler flowmetry was used to evaluate the erythrocyte flux to the potentially ischemic area of the colon following repair of the rupture [18]. With an overall ischemic incidence of 41%, the investigators were able to identify 100% of these patients when finding erythrocyte flux to be significantly decreased (defined as < 50 perfusion units). Thus, using this value as the lowest threshold tolerable, the investigators proposed an algorithm for revascularization of the colon at the time of aortic repair when this level was reached. Laser Doppler flowmetry has also been used to monitor serosal blood flow in the sigmoid colon during open repair, to evaluate whether revascularization should be performed [24]. Similarly, colonic mucosal oxygen saturation measured by way of a spectrophotometer probe inserted into the rectum has been evaluated during aortic surgery to detect changes in the colonic blood flow [25]. It has been shown to be a sensitive indicator of decreases in colonic flow in both the endovascular and open settings, and correlates with aortic balloon occlusion during EVAR and aortic cross clamping with open repair. Techniques such as this may provide easily detectable and continuous intraoperative monitoring of the changes in the colon blood flow and may serve as indicators of the potential need for revascularization or resection, or may help identify those patients at risk who require further work-up, should the levels not return to normal. The plethora of information alone regarding investigational and research endeavors into techniques to diagnose ischemic colitis speak to the degree of its importance and the high potential for morbidity and mortality.

Aside from the more investigational methods, endoscopy remains a commonly used method for diagnosis when patients present with clinical symptoms or, for some centers, as a part of routine postoperative screening. Ischemia has a wide extent of changes and characteristics when viewed endoscopically (Table 1). Biopsy is rarely useful and is more likely to demonstrate either nonspecific ischemic or inflammatory changes rather than the ghost cells that are classic for ischemia [16]. Supporters of the routine use of

Table 1
Endoscopic findings of ischemic colitis

Stage	Endoscopic findings
Acute	Hyperemia, edema, friable mucosa, superficial ulcerations, petechial hemorrhage, gangrene ^a
Subacute	Edema, exudate, ulceration
Chronic	Stricture, mass, segmental involvement

^a Irreversible damage characterized by gray, green, or black appearance.

endoscopy suggest that the classic pattern of rectal and distal sigmoid sparing, along with varying degrees of more proximal mucosally based changes, is quite helpful in this select patient population [26]. Others point to the potential risks of the procedure, including perforation, along with its inability to predict accurately the degree of necrosis to the bowel wall, as reasons not to perform it routinely. In a review of seven prospective, nonrandomized series evaluating the role of routine colonoscopy following abdominal aortic surgery, endoscopy was able to diagnose ischemic colitis accurately on appearance, but failed to differentiate mucosal-only injury from full-thickness ischemia reliably [27]. Furthermore, some question the importance of the information gathered by endoscopy, suggesting it rarely adds anything beyond that which could be easily identified on routine clinical examination alone, and does not change the eventual management. In a study of 105 patients over 3 years undergoing routine scheduled endoscopy within 72 hours of aortic surgery, only 12 patients were found to have ischemic colitis (11.4%), of which 7 were symptomatic and carried a diagnosis of ischemic colitis before undergoing the procedure [28]. Findings in this cohort included the typical wide spectrum, ranging from superficial ulcerations and mucosal erythema to mucosal sloughing, muscle death, and even full-thickness necrosis. The investigators suggested that routine endoscopy should only be a part of the care of the postoperative patient for whom postoperative clinical symptoms dictate, or for whom intraoperative assessment of the colonic viability is in question. Other investigators have used more of a selective approach to determine which patients are in need of postoperative endoscopy, and find it useful as a confirmatory test only. Brandt and associates [29] found the most common indications for endoscopy to include bloody stools, hemodynamic instability, sepsis, and acidosis. In this light, the scope is used more to corroborate the clinical examination. Overall, despite these differing views, endoscopy has a definite role in the evaluation of these patients, may provide useful information (especially when findings may suggest a worse insult than initially suspected), and should be a part of the surgeon's armamentarium.

Other radiology tests may provide additional insight into the diagnosis or the degree of insult to the bowel. In general, angiography does not help in patients who have acute ischemic changes, although it may be useful occasionally in the patient who has previously undergone reimplantation of the

IMA, to evaluate for patency or collateral circulation. Much more commonly, angiography is used in conjunction with duplex ultrasonography in the chronic setting, to identify the patency status of the major visceral vessels before a proposed revascularization procedure. Multiplanar CT imaging has been used to diagnose ischemic colitis, with associated changes in the bowel wall more often offering clues to the diagnosis (Fig. 1) [30]. Being able to evaluate the bowel and surrounding tissue aids in diagnosis by indicating findings such as wall thickening, mesenteric fat stranding, mucosal enhancement, intramural air, and dilatation, or even more ominous signs such as portal venous gas.

Open aortic aneurysm repair

The incidence of ischemic colitis following elective open AAA repair is approximately 1% to 7% and is thought to be due to patient factors (ie, advanced age, medical comorbidities) and the physiologic insult from prolonged operative times and blood loss associated with an open operation [12,14,31,32]. Although potentially the most devastating complication, colonic ischemia is often the least common gastrointestinal complication following open aortic surgery. In a study of 120 consecutive patients undergoing open aortic aneurysm repair, 25 patients also developed ileus ($n = 12$), upper gastrointestinal bleeding ($n = 5$), *Clostridium difficile* enterocolitis ($n = 5$), acute cholecystitis ($n = 2$), mechanical obstruction ($n = 2$), and ascites ($n = 2$), with only 1 case of ischemic colitis [33]. These conditions can occur in conjunction with, or independent of, one another; therefore, a thorough evaluation is required because treatment can be very different for each.

Risk factors for the development of ischemic colitis include prolonged aortic cross clamp time and loss of patency of the IMA and hypogastric arteries [28]. In a study of 2824 patients undergoing aortoiliac surgery,

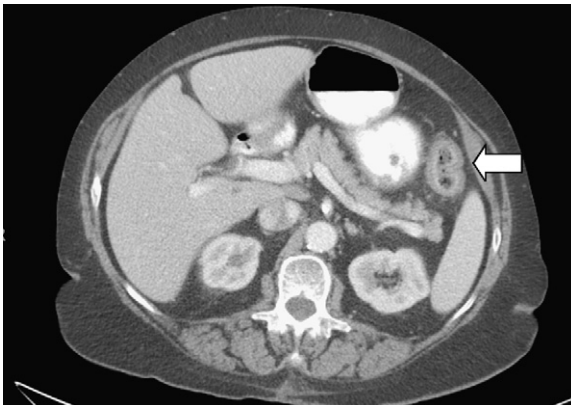


Fig. 1. Ischemic colitis of the splenic flexure.

including 62 patients who developed ischemic colitis, rupture and shock on admission were the primary factors associated with the development of ischemic colitis [32]. Large retrospective series have found these risk factors to be more commonly associated with an open repair, which may be related in part to a paucity of surgeons being comfortable approaching these extremely high-risk patients with an endovascular approach. Other risk factors found to be independently associated with an open approach and the development of ischemic colitis include renal disease, emergency surgery, advanced age, aortobifemoral graft placement, prolonged operative time, and ligation of the hypogastric arteries.

During open repair, surgeons directly evaluate the IMA blood flow when opening the aneurysm sac. Whether or not to reconstruct or reimplant the IMA as a routine component of aortic repair is controversial. Indications for reimplantation include slow oozing from the IMA, borderline perfusion of the sigmoid colon as demonstrated intraoperatively, or decreased stump pressure of the transected pedicle [34]. Yet many investigators have found that routine reimplantation provides no benefit over maintaining stable cardiac output and blood pressure support alone [35,36]. Others have found that IMA reimplantation is helpful under certain conditions. In a study of 151 patients comparing selective ligation with reimplantation of patent IMAs (based on clinical bowel inspection, Doppler signals, and IMA stump pressures), Seeger and colleagues [37] found the rate of colonic ischemia to be 2.7% and 0% ($P < .05$) for selective ligation and reimplantation, respectively, and recommended routine reimplantation for all patent IMAs to limit colonic infarction. Despite the multiple studies evaluating this question, the data remain contradictory, with strong opinions on both sides. Most importantly, surgeons must make every attempt to preserve colonic blood supply and evaluate the viability of the colon at the time of the operation, rather than adhere to the dictum of a “wait and see” approach.

Endovascular aneurysm repair

In 1991, Parodi and colleagues [38] were the first to demonstrate EVAR as a feasible option for the repair of AAA. Since that time, multiple studies have documented the benefit of EVAR in reducing mortality, postoperative complications, and hospital length of stay [39,40]. Relatively small reports of ischemic colitis following EVAR have also demonstrated an incidence of approximately 0% to 6%, depending on the sample size, the aneurysm presentation (ie, elective versus ruptured), and the definition of ischemic colitis (Table 2) [41–46]. Patients presenting with ruptured aneurysms are at a significant risk for ischemic changes, although EVAR appears to be somewhat protective compared with the open approach. In a study of ruptured aneurysms treated with EVAR, the incidence was higher, at 24%, albeit much lower than a similar cohort undergoing open repair (44%) [47]. Although the exact pathophysiology of ischemic colitis following EVAR is still

Table 2
Ischemic colitis following endovascular aortic aneurysm repair

Author	No. patients	No. with ischemic colitis	Early diagnosis (≤ 3 days)	Colectomy	Revascularization	Mortality
Champagne et al 2007 ^a [47]	36	8 (23%)	8 (100)	3 (38%)	0	0%
Nevelsteen et al 2006 [41]	1	1	0	1	0	1 (100%)
Mehta et al 2006 ^a [42]	40	2 (5%)	—	—	—	—
Lee et al 2006 ^b [43]	24	1 (4.2%)	1	0	0	0%
Mehta et al 2005 [44]	175	—	—	1	—	—
Maldonado et al 2004 [50]	311	4 (1.2%)	3 (75%)	3 (75%)	0	2 (50%)
Axelrod et al 2004 [45]	102	0	0	—	—	—
Geraghty et al 2004 [49]	233	4 (1.7%)	3	3	1 ^c	2 (50%)
Verhoeven et al 2002 [46]	17	1 (5.8%)	N/A	0	0	0%
Dadian et al 2001 [48]	278	8 (2.9%)	N/A	2 (25%)	0	3 (38%)

Abbreviation: N/A, not available.

^a All cases of ruptured aneurysms.

^b EVAR with concomitant unilateral iliac artery embolization.

^c Unsuccessful attempt at preserving hypogastric patency at initial surgery (patient lived).

controversial, it is thought to result primarily from embolism of particulate thrombus within the aneurysm sac rather than from a global physiologic insult [48–51]. Occlusion of the hypogastric arteries has led to the development of severe buttock claudication and pelvic ischemia, and this has also been hypothesized to be an additional potential factor in the development of colonic ischemia, by way of occlusion of one of the collateral pathways [49]. In addition, perioperative contributing factors similar to those for open repair, such as hypotension, blood loss, and occlusion of the IMA following graft deployment, are suggested causative sources.

The benefits of EVAR in reducing perioperative complications and hospital length of stay have been clearly demonstrated. The relatively small number of case series reporting the incidence of ischemia in EVAR suggests that EVAR is associated with, on average, a lower risk of developing ischemic colitis than open AAA repair, but the nationwide incidence of ischemia following EVAR compared with open AAA repair is unknown. If continued experience with EVAR demonstrates a lower risk than open AAA repair, this will yield yet another benefit of EVAR in the treatment of AAA. Endovascular repair should not preclude the ability to preserve the potential collateral blood supply by maintaining vascular flow to at least one internal

iliac artery [52]. Unfortunately, the advent of catheter-based technology has also led to the development of known complications though new means that have not yet been described. For example, ischemic colitis has recently been reported following translumbar injection with 8000 units of thrombin for the treatment of an endoleak into the native aneurysm sac from the originating lumbar vessel [53]. This case of ischemic colitis was felt to originate from an embolization of the rectosigmoid arcade through the IMA, leaving the investigators to speculate about the need for IMA outflow occlusion before thrombin injection. Again, as further experience with minimally invasive and catheter-based procedures takes place, one can anticipate increased knowledge into not only the mechanisms of injury but also ways to avoid this complication.

Treatment

Supportive

The mainstay of therapy for ischemic colitis remains supportive therapy, with adequate fluid hydration and blood pressure support. Vasopressor support is controversial because it is also a contributing factor. Low-flow states and sepsis may require improvement in blood pressure; however, in general, because of the vasoconstriction of the splanchnic vessels, vasopressors may add to the ischemic process, worsening the situation. Should pressor support be necessary, beta-adrenergic agonists that also improve cardiac output are preferred, and one should make every attempt to avoid alpha-agonists if possible. Additionally, broad spectrum antibiotics such as fluoroquinolones and flagyl, or monotherapy with Unasyn or Imipenem have been shown to decrease bacterial translocation and morbidity, and should be added empirically [54]. Most of the studies emphasizing the value of nonoperative therapy do have a degree of self-selection, because those patients “healthy” enough to undergo a trial of nonoperative management by definition are likely to be in a more stable condition and have not manifested signs of overt bowel infarction. Yet they are still at high risk for complications and death, with Longo reporting a single death in a study of 16 patients who had ischemic colitis following nonoperative therapy [22]. With rare exceptions, all patients who have evidence of bowel infarction require surgical therapy.

Surgery

Surgical options generally fall into two categories: bowel resection and vascular reconstruction. Typically, when patients require operative intervention, the surgeon must first determine bowel viability. Caution, however, must ensue because often, in cases with less than full-thickness injury and frank necrosis, the serosal appearance of the bowel does not accurately reflect the degree of ischemic changes to the entire bowel wall. Therefore,

surgeons must either rely on the endoscopic appearance and extent of the ischemic changes (which, as stated, is not always accurate) or use adjunctive measures to evaluate the viability of the bowel.

Intraoperatively, several different methods have been described for providing surgeons with additional useful information regarding blood flow. The surgeon must balance the need to avoid leaving behind necrotic bowel with the potential morbidity of overzealous resection leading to short-bowel syndrome. Clinically, unlike mesenteric ischemia of the small bowel, motility does not provide as much help when dealing with the colon. The intraoperative judgment of the well-trained surgeon remains one of the most important factors. Palpation of mesenteric pulses or detecting Doppler signals on the antimesenteric portion of the bowel wall also provides valuable information. Woods lamp evaluation of the bowel wall following administration of fluorescein dye intravenously can also aid in separating perfused from nonperfused bowel. Surgeons have classically mandated a second-look operation in 12 to 48 hours, regardless of the patient's clinical condition, to evaluate the need for further resection and to aid in being less aggressive at resecting potentially viable bowel at the initial surgery [55]. The already elevated mortality of 50% to 67% is even higher, should surgical resection of infarcted bowel be present at the second operation. Longo and colleagues [14] found an 89% mortality for patients meeting this condition. Further highlighting the need for early diagnosis, in 1991 van Vroonhoven and colleagues [56] published a series of 20 patients who had ischemic colitis following ruptured aortic aneurysm. Eighteen patients required a laparotomy for transmural involvement, with all 20 patients dying during the hospitalization. Although the investigators questioned the usefulness of bowel resection in these patients, it may simply suggest that diagnosis delayed past a certain point in the pathologic process portends almost certain mortality.

Vascular repair involves two separate components and is performed in the acute and chronic ischemia settings. First, patency of the IMA supplying the most at-risk portion of the left colon must be assured. Should the ischemic changes be present in other areas of the colon, attention is focused on that particular region's vascular supply. In addition, determining the antegrade flow to the iliac vessels that provide potential pelvic collaterals needs to be a routine part of both the open and endovascular techniques. Although the technical aspects of vascular reconstruction are beyond the scope of this article, options for dealing with the IMA or other major visceral vessel include resection of the base of the IMA along with a small cuff of aortic wall (Carrell patch) and reimplanting it in the aortic graft, patch angioplasty of the stenotic opening, bypass grafting, or endarterectomy of the atherosclerotic plaque [57,58]. Finally, with the development of improved technology, endovascular approaches have also been described in the treatment of segmental ischemic colitis [59]. However, in the absence of further data, the role that this technology plays in this setting remains undefined.

Review of the literature

Outcome

When reviewing the literature on this topic, two points become apparent. First, most published series are retrospective in nature and, in general, contain a small cohort of patients. Second, the degree and extent of colitis have a large impact on overall outcome. In a study of 43 patients over 6 years, segmental colitis was present in most patients (72%) [15]. Those patients in whom the entire colon was affected with ischemic changes had a much worse prognosis, with all requiring surgery and an overall mortality of 75%. Fortunately, ischemic colitis most often affects only the mucosa and most patients are able to be treated successfully using nonoperative means. Surgery is then relegated only to those in the direst of clinical conditions or those presenting with frank peritonitis from infarcted bowel. Those patients who are well enough to be treated with nonoperative means have better outcomes. In a series of 278 patients undergoing EVAR, only 8 developed ischemic colitis [48]. Two patients died immediately, with an additional death in one of the two undergoing surgery, for a mortality of 38% in those with ischemic changes. Yet all four patients who were treated nonoperatively survived. Although results like these are difficult to compare across different series secondary to a large selection bias, they do underscore the wide range of disease severity that occurs in these patients.

Reimplantation of the IMA has also had varied results. In a study of 10 colonic ischemia patients over a 10-year period, reimplantation was deemed necessary in 5 patients because of inadequate colonic perfusion at the time of open aortic repair. Ultimately, transmural (versus limited mucosal) necrosis occurred in 60%, including four of the five patients who were felt to have a technically successful reimplantation [60]. In all six of these cases, intraoperative hypotension was also felt to be a contributing factor. A drawback to this study is the investigators' lack of reporting of the total rate of IMA reimplantation that did not develop ischemic changes over the course of the study; thus, the overall success rate is unknown. However, these results emphasize the need for preventing the factors that may lead to ischemic changes, because in some cases the initial ischemic insult may be nonreversible, even with a technically successful attempt that improves blood flow to the ischemic segment.

A small percentage of patients develop ischemic colitis following surgery, require an initial operative bowel resection, survive, and then go on to require a second operative procedure apart from a "planned take-back." These patients have an even higher mortality, with one study reporting death in seven of eight patients [61]. Indications for the second exploration include sepsis of unknown origin, attempt at further revascularization, and evidence of additional infarcted bowel. Yet the end result remains similar, with a very high mortality rate. Thus, despite an initial stabilization, the

at-risk patient requires continued high-intensity monitoring, with efforts directed at controlling blood pressure, sepsis, and secondary injury.

Chronic changes

Patients sustaining a bout of ischemic colitis who are able to recover fully may have varying degrees of symptoms, ranging from completely asymptomatic, mild constipation, or near-obstructing colonic strictures (Fig. 2). Because of the overall high mortality with fulminate disease and the lack of presentation with milder forms, the true rate of colonic stricture following nonoperative management is unknown. Similarly, the rate of recurrent ischemic colitis has not been well delineated. Longo and colleagues [22] found no rate of relapse at a mean follow-up of 5.3 years in his 47 patients. Yet high-quality, longitudinal studies with adequate follow-up are lacking. Therefore, patients should be counseled on discharge about the potential future ramifications of this disease.

Other patients may develop chronic visceral ischemia, which is manifested by abdominal pain, especially with eating, when supply does not equal demand; this condition may lead to relative bowel ischemia, which may result ultimately in food fear and weight loss. Complete visceral artery revascularization of celiac, superior mesenteric, and inferior mesenteric arteries is often required to alleviate symptoms. Occasionally, however, patients will only be able to have a single IMA revascularization because of technical failures or other considerations. Although data on this procedure are limited, a study of 11 patients over a 6-year period in which isolated IMA repair was performed showed 10 patients with improved or cured symptoms perioperatively and 70% still with patent repairs and symptomatic improvement at longer follow-up [57]. For this procedure to be a success, a well-developed pattern of collateral circulation for the IMA system is required and this should be evaluated before considering this option.

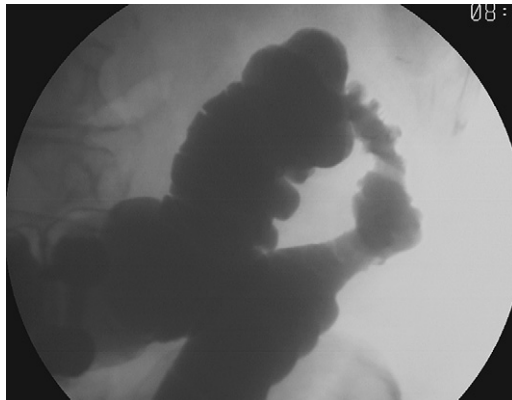


Fig. 2. Barium enema, demonstrating splenic flexure stricture from chronic ischemic colitis.

Finally, concerns regarding infection of the aortic prosthetic graft leading to higher mortality and subsequent complications are present not only in the acute but also in the chronic setting. Infection of the graft has been shown to lead to complications such as need for graft explantation, revascularization including extra-anatomic bypass, aortoduodenal fistula, sepsis, and death following both the open and EVAR settings [62–64]. Yet endovascular graft placement along with prolonged antibiotics has also been described in case reports as a potential treatment for patients who have known infected aneurysms, although recommendation awaits further data collection [65]. Although the comparative rates of graft infection for the endovascular and open settings following ischemic colitis are unknown, theoretic advantages to the endovascular approach include preservation of natural tissue planes secondary to lack of opening up the retroperitoneum during repair. Thus, the graft is never exposed to topical contamination. Should this be borne out in the future, it will provide a further advantage to the endovascular method in this unique setting.

Summary

Ischemic colitis following vascular surgery continues to carry a high morbidity and mortality. Extensive effort has gone into the development of new techniques to diagnose and treat this condition at earlier stages. Physicians need to carry a high degree of suspicion as to the diagnosis because, unfortunately, many patients may have a paucity of symptoms early, leading to a delay in work-up and worse outcomes. Supportive treatment remains the mainstay, including blood pressure, fluid support, and broad-spectrum antibiotics. Although surgical therapy, including bowel resection or vascular reconstruction, remains an option, the development of this condition unfortunately still portends a poor prognosis.

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