



49th ARGENTINE CONGRESS OF COLOPROCTOLOGY

OFFICIAL REPORT

COLONIC VASCULAR DISORDERS

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Introduction

Vascular Anatomy of the Colon

It is essential to understand the vascular anatomy of the colon to comprehend the pathologies that affect it and ensure the success of colorectal surgery. It is therefore crucial to have a good understanding of the preoperative vascular anatomy of the colon. This will ensure adequate oncological resection with minimal intraoperative complications.¹ In particular, the surgical importance of the vasculature of the right colon has changed with the evolution of the right hemicolectomy technique. The right colic artery (RCA) was considered of little technical relevance. When performing right hemicolectomy, the identification and isolation of the RCA was not regarded as a vital step, irrespective of the approach (medial or lateral).²

The colonic vasculature began to play a central role in colon cancer surgery following Turnbull's description of the "no-touch technique".² This technique employs the principles of a medial-to-lateral approach for lymphovascular isolation before colon mobilization. The foundation for this approach is predicated on observations indicating that neoplastic cells are actively shed into the bloodstream during tumor manipulation, and that vascular ligation before colon mobilization would help minimize this risk. With the advent of laparoscopic colorectal surgery, the medial approach to the right colon pedicle has been re-established as the prevailing standard. Moreover, the surgical importance of the vascular anatomy of the colon was amplified with the introduction of the complete mesocolic excision (CME) technique and D3 lymphadenectomy.³

The colon receives its blood supply from the superior and inferior mesenteric arteries, while the rectum also receives its blood supply through branches of the hypogastric artery (Fig. 1).

Superior Mesenteric Artery

The superior mesenteric artery (SMA) originates from the aorta caudal to the celiac trunk. Its terminal branch is the ileocolic artery (ICA). The vessels supplying the right and transverse colon, the RCA, and the middle colic artery (MCA), arise on its right side. On its left side are the ileal branches. The ICA and MCA are the most important anatomical landmarks during right hemicolectomy because they consistently supply the right colon. A systematic review and meta-analysis of 41 cadaveric dissection and imaging studies found that the ICA had a 99.7% prevalence and was the only artery in all cases.¹ The MCA was present in 96.9% of cases.¹ The prevalence of one, two, or three MCAs was 88.6%, 10.8%, and 0.6%, respectively.

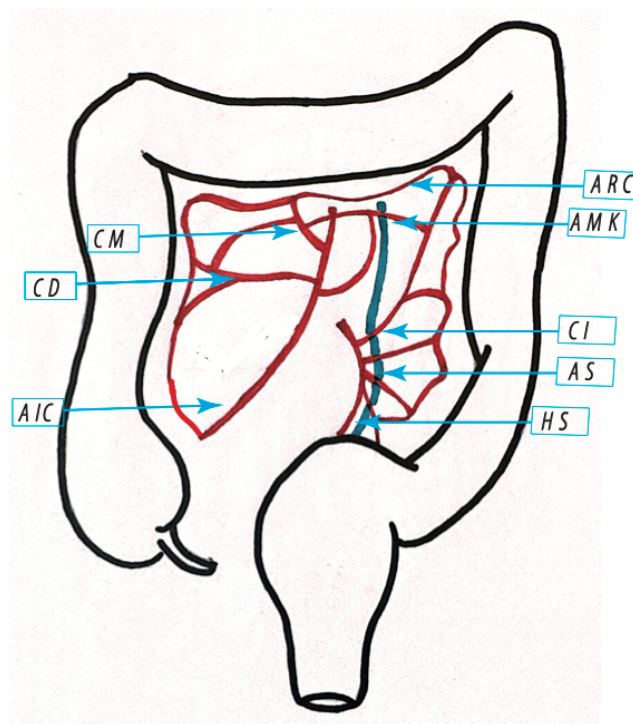


Figure 1. Diagram of the main arteries of the colon and rectum. AMS: superior mesenteric artery; AIC: ileocolic artery; CD: right colic artery; CM: middle colic artery; AMI: inferior mesenteric artery; AR: Arc of Riolan; CI: left colic artery; AS: sigmoid artery; HS: superior haemorroidal artery; AMI: inferior mesenteric artery; AMK: Moskowitz artery; ARC: Riolano arch.⁴

Right Colic Artery

It may be absent in nearly one-quarter of patients. In most cases, RCA is a solitary artery, with only 3.2% of cases exhibiting more than one RCA. During surgery, the high rate of absence of the RCA (27.4%) may be due to the intraoperative difficulty in identifying this artery, which has a small diameter and is therefore not easily isolated during mobilization and division of the mesocolon.¹ This difficulty increases in obese patients because the thicker mesocolon makes it more difficult to identify vascular structures. Other authors report an even lower incidence of RCA, which was absent in 66.6% of tomographic studies.⁵ The absence of the RCA is of great surgical relevance since the MCA could be mistaken for the RCA and clipped or ligated, compromising the blood supply to the transverse colon. Preoperative vascular mapping with 3D reconstruction of the vascular anatomy enables surgeons to anticipate the anatomical variations and perform precise surgical planning. The Yada classification, as described in a report including 22 studies with 1,670 patients, describes the branches of the SMA to the colon (Fig. 2).¹ These variants consider the origin of the RCA (Table 1).⁶

Table 1. Yada classification for the right colic artery (RCA) and reported frequencies.

Type (Yada)	Description	Intraoperative frequency
1	Independent origin from the superior mesenteric artery	55.2%
2	Common trunk of the RCA with the middle colic artery	35.6%
3	Common trunk with the ileocolic artery	9.2%
4	Absence of the right colic artery	10.8%

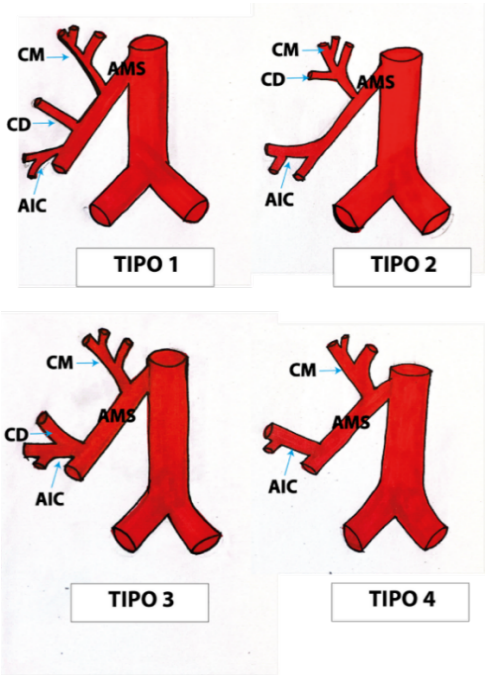


Figure 2. Yada's classification of the different types of origin of the right colic artery (CD). Tipo: Type. AMS: superior mesenteric artery; AIC: ileocolic artery; CM: middle colic artery.

Another possible variation not mentioned in Yada's classification is when the MCA, RCA, and ICA originate from the same common trunk. This variation has a very low prevalence of 0.01%.⁶ Gamo et al.⁷ have proposed a classification of anatomical variants of the SMA (Fig. 3). This classification describes the patterns detailed in Table 2.

Table 2. Gamo classification for right-colon arterial branching patterns and relative frequencies.

Type (Gamo)	Description	Approx. frequency (%)
I	Independent origin of the right colic (RCA), middle colic (MCA) and ileocolic (ICA) from the SMA	40
II.a	Common trunk of MCA and RCA; ICA arises separately	20
II.b	Common trunk of RCA and ICA; MCA arises independently	32
II.c	Common trunk giving rise to the three main branches: MCA, RCA and ICA	0
III	Absence of the right colic artery (RCA)	8
IV	Presence of one or more accessory right colic arteries	0

It is important to consider the variations in the arterial branches and also their relation to the veins. When approaching the right colon for a CME or D3 dissection, the dissection starts at the superior mesenteric vein (SMV) and extends along its ventral surface. Therefore, understanding its relationship with the arterial branches is essential for performing the surgical procedure with proper vascular control, lymphadenectomy, and colon mobilization while preventing venous injuries. The ICA is located anterior to the SMV in 44.5% of cases, posterior to it in 55.1% of cases, and to the right in 0.4% of cases (Fig. 4).^{1,5}

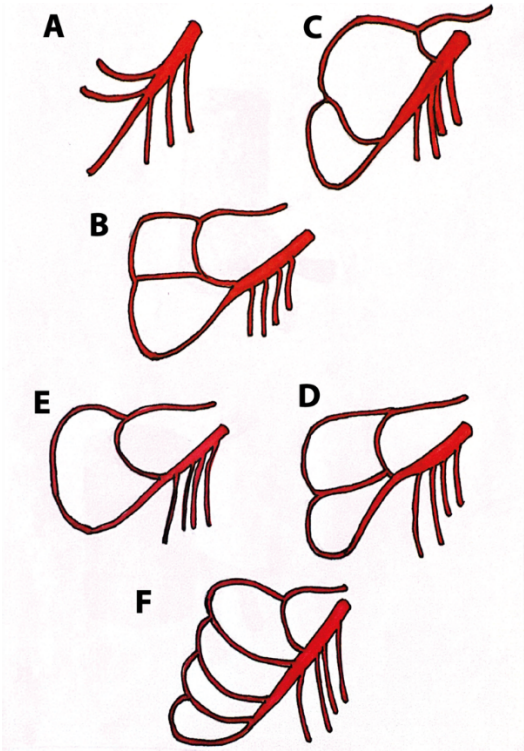


Figure 3. Gamo's classification of anatomical variants of the branches of the superior mesenteric artery (SMA). A. Type I: the right colic artery, the middle colic artery, and the ileocolic artery originate independently from the SMA. B. Type II.a: common trunk of the middle colic artery and right colic artery; the ileocolic artery originates separately. C. Type II.b: common trunk of the right colic artery and ileocolic artery; the middle colic artery originates independently. D. Type II.c: common trunk from which the three main branches (middle colic artery, right colic artery, and ileocolic artery) originate simultaneously. E. Type III: absence of the right colic artery. F. Type IV: presence of one or more accessory right colic arteries.

The CD runs anterior to the VSM in 87.3% of cases, posterior in 11.4%, and to the right in 1.3% (Fig. 5).^{1,5}

We consider the relationship between the MCA and the SMV to be relevant in the context of LND around the MCA. The SMV is a constant vessel that runs laterally to the ICA and, upon joining the splenic vein, forms the portal vein at the pancreatic level.

Depending on the presence or absence of a common trunk, MCA can be classified as Type I (common trunk arising from the SMA), Type II (independent right and left branches arising from the SMA), and Type III (direct branches from the celiac trunk). This classification is shown in Fig. 6.

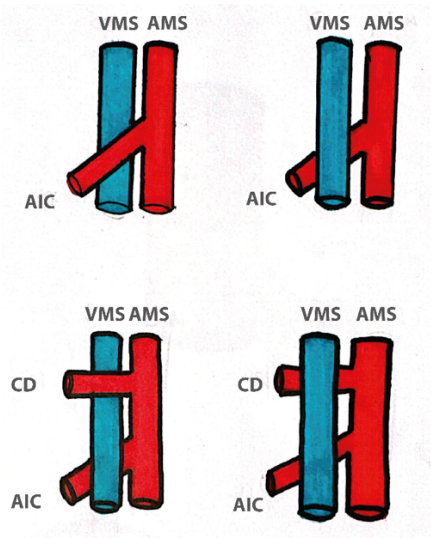


Figure 4. Topographic relationships of the superior mesenteric artery (AMS) with the superior mesenteric vein (VMS)1. AIC: ileocolic artery; CD: right colic artery.

In a study of 505 patients and 44 cadavers, Ogi et al.⁹ evaluated the length of the common trunk of the MCA from the SMA to the bifurcation in Type I. The length, as determined by computed tomography angiography (CT angiography) and cadaver examinations, was 2.6 cm (range: 0.1-9.0) and 2.8 cm (range: 0.5-6.3), respectively. Three types of relationships between the left SMV and MCA are described in Fig. 6. In Type IIA, a common trunk runs along the left border of the SMV (59.7% of cases). In Type IIB, a right branch of the MCA runs along the left border of the SMV (40.0%). In Type C, the MCA is located dorsal to the SMV (0.3%; Table 3). In colon cancer surgery of the hepatic flexure, in cases with Type I disposition, the central ligation encompasses the entire middle colic pedicle. In Type II cases, however, radical resection is completed by ligating the right branch of the CM, allowing sufficient residual flow to the left branch. In Type II cases, for middle or left transverse colon cancer, it is necessary to ligate both branches of the MCA to perform a central vascular ligation, which makes an extended right or left colectomy necessary. If a transverse colectomy is performed, it is essential to assess the perfusion of the ends to be anastomosed before performing the transit reconstruction to ensure adequate perfusion.

There are some accessory MCAs rarely observed, originating from the jejunal artery, pancreatic artery, celiac artery, common hepatic artery, and splenic artery. It has been suggested that the SMV could serve as a reference point for performing adequate and essential LN. The Moskowitz artery, defined as the vessel that directly anastomoses the MCA with the left colic artery (LCA), is present in 2.4% of cases.⁹ It is hypothesized that the Moskowitz artery may develop as collateral circulation due to stenosis or occlusion of the inferior mesenteric artery (IMA). In cases where a Moskowitz artery is identified, calcification surrounding the IMA is more pronounced (Fig. 1).⁹

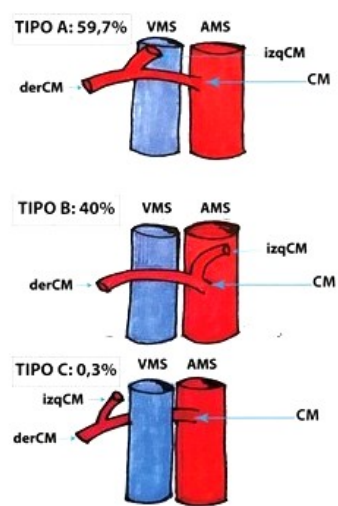


Figure 5. Anatomical relationship between the superior mesenteric vein (VMS) and the middle colic artery (CM). Type A: the common trunk of the CM runs along the left edge of the VMS; Type B: the right branch of the CM originates and runs along the left edge of the VMS. Type C: the CM passes behind the VMS. AMS: superior mesenteric artery. Tipo: type

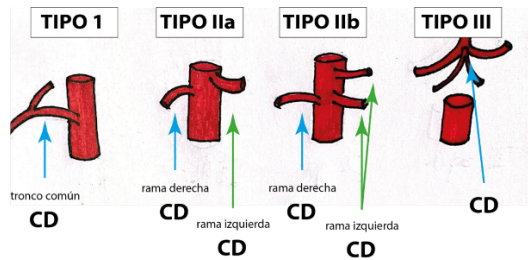


Figure 6. Variables of the middle colic artery (MCA) bifurcation. Type I: a right branch and a left branch originating from a common trunk of the MCA from the superior mesenteric artery (SMA). Type IIa: a right branch and a left branch that originate independently from the SMA. Type IIb is characterized by the presence of one right branch and two left branches of the MCA, with origins from the SMA. Type III: the MCA originates from a vessel other than the SMA, usually the celiac artery. Tipo: type. CD: right colic artery. Tronco común: common trunk, Rama derecha: right branch. Rama izquierda: Left branch.

Table 3. Classification of middle colic artery (MCA) bifurcation patterns observed on CT angiography and cadaveric dissection.

Type of MCA bifurcation, n (%)	CT angiography (n = 505)	Cadaveric (n = 44)
Type I	290 (57.4)	31 (70.5)
Type IIa	211 (41.8)	13 (29.5)
Type IIb	3 (0.6)	0
Type III	1 (0.2)	0

Inferior Mesenteric Artery

The IMA originates on the anterior aspect of the aorta, before its bifurcation. The classification of the IMA bifurcation is determined by the configuration of its initial branch.¹⁰ In the event that the initial branch does not give rise to any vessels prior to the division into ascending and descending branches at the edge of the colon, it is designated as the LCA. In the event that the first branch forms a common trunk with the sigmoid artery (SA) and the LCA, it is designated the colosigmoid artery (CSA). The subsequent branch of the IMA

is known as the superior rectal artery or superior hemorrhoidal artery (SRA). As it progresses towards the pelvis, the SRA divides into a variable number of sigmoid arteries. The most distal sigmoid artery is known as the sigmoid ima artery (SIA). The terminal branches of all the vessels mentioned form the marginal artery near the colonic margin (Figs. 7 and 8).

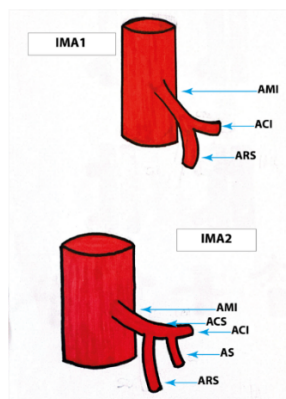


Figure 7. Variants of branching of the left colic artery (ACI) according to its first branch.¹¹ The IMA1 pattern is characterized by a ACI without branching. The IMA2 pattern is characterized by a colosigmoid artery (ACS) as a common trunk for the ACI and the sigmoid artery (AS). ARS: superior rectal artery

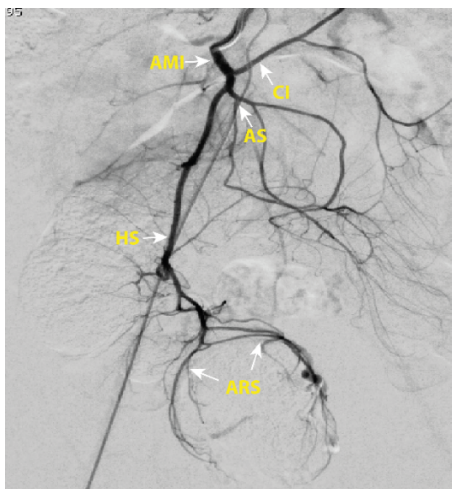


Figure 8. Selective angiography of the inferior mesenteric artery (AMI). The main branches are identified: the left colic artery (CI), the sigmoid artery (AS), the superior hemorrhoidal artery (HS), and the superior rectal arteries (ARS). The IMA1 pattern is shown, which has an unbranched CI.

In 1907, Paul Sudeck identified a critical point in the arterial supply to the colon and rectum through injection experiments. The critical point was identified at the origin of the final sigmoid artery in the SRA.¹² Consequently, the most distal sigmoid artery was designated the "sigmoid ima artery" (Latin 'ima' = "lowest").¹³

The SIA is predominantly located in the pelvic cavity, 1-2 cm below the promontory.¹³ In rare cases, the SIA fails to form the distal end of the marginal artery due to the absence of anastomosis with the next proximal vessel, or the

lack of sigmoid arteries derived from the RASSRA (Fig. 9).¹⁴ This artery supplies the rectosigmoid junction, a critical area in terms of ischemia risk.¹⁴

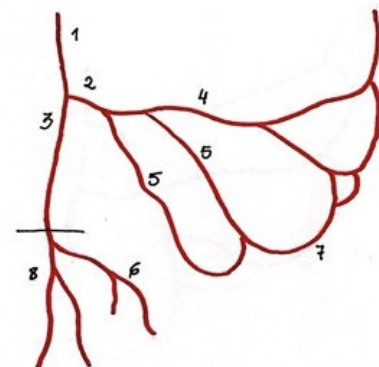


Figure 9. Diagram of colorectal perfusion. An absence of arcade is present at Sudeck's point due to a lack of anastomosis between the inferior sigmoid artery (6) and the rest of the arcade. This constitutes a critical perfusion area. 1: Inferior mesenteric artery; 2: Colosigmoid artery; 3: Superior rectal artery; 4: Left colic artery; 5: Sigmoid arteries; 6: Sudeck's point; 7: Marginal artery; 8: Bifurcation of the superior rectal artery. Diagram adapted from Sunderland (1942).

A comparison of preoperative and postoperative CT scans of patients who underwent surgery for rectal cancer reveals an adequate correlation between the CT scan and the surgical report.¹⁵ Preoperatively, the median length of the IMA trunk is 35 mm (range, 16-80 mm), while the median distance from the origin of the IMA to the aortic bifurcation is 42 mm. Subsequent to the procedure, the median length of the remaining IMA stump is 2 mm following high ligation and 44 mm following low ligation. Following a low ligation, the IMA is displaced laterally with respect to its preoperative position, and the remaining LCA extends upwards towards the remaining descending or sigmoid colon. Conversely, after high ligation, either a small, short stump of the SMA or a nodular-appearing protrusion remains at the point where it exits the abdominal aorta.

The inferior mesenteric vein (IMV) may also present anatomical variants, as demonstrated in Fig. 10. It may terminate in the splenic vein (44.8%), in the SMV (41.6%), or simultaneously confluence with the splenic vein in the SMV (13.6%).⁹

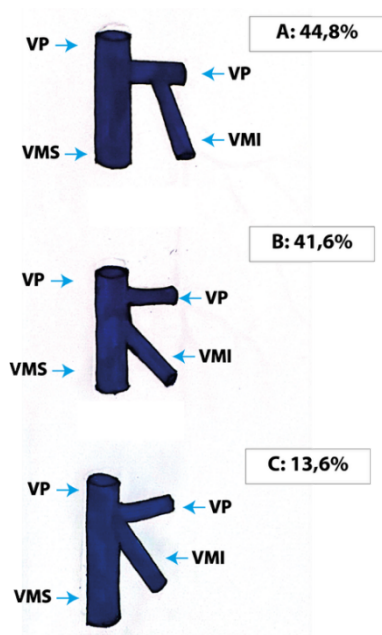


Figure 10. Anatomical variants of the drainage site of the inferior mesenteric vein (VMI). **A.** The VMI drains into the splenic vein, which, in conjunction with the superior mesenteric vein (VMS), constitutes the portal vein (VP). **B.** The VMI drains directly into the VMS. This subsequently forms the VP. **C.** The VMI, VMS and splenic vein converge to form the VP in 13.6% of cases.

Watershed Zones

It is said that watershed zones exist where the colon is supplied by the terminal branches of two main vessels. There are two borderline perfusion areas in the colon. The splenic flexure (Griffith's point) receives blood flow from the SMA and the IMA via Drummond's marginal artery. This artery is present in 95% of people.¹⁶ However, in up to 5% of people, this artery may be absent at the level of the splenic flexure. The rectosigmoid junction (Sudeck's point) receives blood from the SRA and the SMA (Fig. 11). The colorectal junction is a common site for the development of colonic ischemia. This is a clinical problem associated with non-occlusive ischemic colitis, colorectal resections, and repair of abdominal aortic aneurysms.^{14,17} The absence of anastomosis between the sigmoid ima and the last sigmoid branch has already been mentioned. Despite the attention given to these watershed zones, most cases of ischemic colitis do not occur there. Only 3-5% of cases occur at Sudeck's point.^{12,14} An autopsy study found that the areas most commonly affected by the disease were the sigmoid colon (affected in 83% of cases) and the descending colon (affected in 77% of cases), rather than the watershed zones.¹⁸ The splenic flexure, the best-known watershed zone, is affected in approximately 25% of ischemic colitis cases.¹⁸ Less frequently (14%), the splenic flexure is the only area affected.

Recently, Watanabe et al.¹⁹ used indocyanine green (ICG) fluorescent angiography to describe cases of decreased (15%) or absent (5%) blood flow along the rectosigmoid junction. This demonstrated in vivo that some patients have an absence of a permeable vascular arcade at this level. In these cases, the marginal artery ends above the pelvic rim, similar to an abdominal marginal artery.¹⁴

In up to 50% of cases, the marginal artery is underdeveloped in the right colon, which could explain the greater vulnerability of this segment in situations of low blood flow and the greater predisposition of some patients to ischemic

compromise of the right colon,²⁰ which could make the right colon a third critical area.

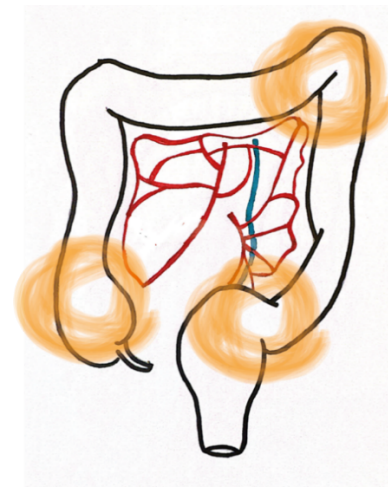


Figure 11. Watershed zones of the colon where it may be vulnerable to states of low systemic flow due to poor development of the marginal artery of Drummond: right colon, splenic flexure (Griffith's point), and rectosigmoid junction (Sudeck's point).

Colonic Perfusion

The anatomical arrangement of the blood vessels ensures adequate blood flow to the colon. This perfusion phenomenon has special characteristics that can be studied in patients.

As with the rest of the body, colon perfusion exhibits a pulsatile pattern related to cardiac contraction. The pulse wave is a fundamental component of cardiovascular physiology because it reflects blood flow and tissue perfusion dynamics. Understanding its phases and characteristics is essential for evaluating tissue perfusion and the patient's overall hemodynamic status. A pulse wave usually consists of an ascending phase, a dicrotic notch, and a descending phase. These phases correspond to different moments in the cardiac cycle (contraction and relaxation). The mechanical properties of the vascular system, arterial compliance, and peripheral vascular resistance determine the morphology of the pulse wave.²¹ Advanced imaging technologies, such as nonlinear ultrasonic localization microscopy, allow for detailed visualization of tissue perfusion and improved understanding of microcirculatory flow.²²

Current ultrasound equipment features Doppler technology, which makes it relatively easy to visualize mural and mesenteric blood flow. However, color Doppler imaging (CDI) predominantly reflects flow in larger-caliber, high-velocity vessels.²³ Signal detection can be difficult in patients with large body mass index, those with weak intrinsic Doppler signals, and those with intestinal loops located deep within the abdomen or pelvis. However, the main limitation of CDI is its inability to demonstrate blood flow at the capillary or perfusion level.

Contrast-enhanced ultrasound (CEUS) is performed by administering microbubble contrast agents, which allow for the acquisition of both subjective and objective parameters that reflect the inflammatory process by visualizing the enhancement of the intestinal wall and mesentery.²³ CEUS can be used to

estimate intestinal wall perfusion and obtain an objective and quantifiable image of intramural microcirculation and inflammatory activity in Crohn's disease (Fig. 12).²⁴ CEUS revealed three distinct patterns of intestinal wall perfusion after microbubble injection: submucosal enhancement, transperietal enhancement toward the lumen (inward), and transperietal enhancement toward the serosa (outward).

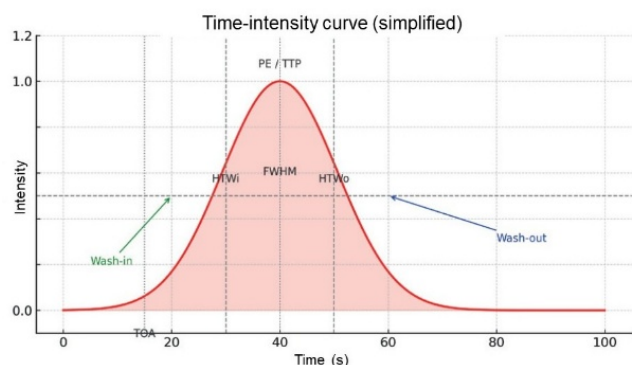


Figure 12. Contrast-enhanced ultrasound (CEUS). Intensity-time curves for determining disease activity. Schematic representation of an intensity-time curve in the intestine. **B.** CEUS analysis provides the following quantitative parameters: fall time (FT), time to peak (TTP), wash-in rate (WiR), and wash-out rate (WoR).²³

Patients with ischemia may experience thinning of the intestinal wall due to loss of tissue, vascularization, and muscle tone.²⁵ Numerous studies have demonstrated that the absence or diminution of intestinal wall enhancement on CEUS is a distinctive manifestation of intestinal ischemia, exhibiting a sensitivity ranging from 85 to 94% and a specificity of 100%.²⁵

CEUS has been used to visualize visceral arteries. In a European study, CEUS was combined with color and spectral Doppler, enabling an unequivocal diagnosis of visceral artery stenosis in patients with abdominal angina.²⁶

Evaluation of Colonic Perfusion by Fluorescence

The implementation of fluorescence-guided surgery has facilitated the observation of phenomena that were previously known but could not be demonstrated during the surgical procedure. The intravenous injection of indocyanine green (ICG) facilitates real-time observation of bowel perfusion (Figs. 13 and 14). The implementation of this technique does not increase surgical time, does not entail associated complications, and has been shown to generate a change in operative behavior in 5-14% of cases.^{27,28}

Furthermore, the use of ICG angiography has been associated with a significant reduction in the rate of anastomotic fistulas (AFs). In a systematic review, series using ICG angiography had half the number of fistulas of series not using it (3.83 vs. 7.58%). In a study of 93 patients, the AF rate was significantly lower in the group evaluated with ICG fluorescence than in the historical control group (3.2 vs. 10.8%, respectively; $p=0.046$).³⁵

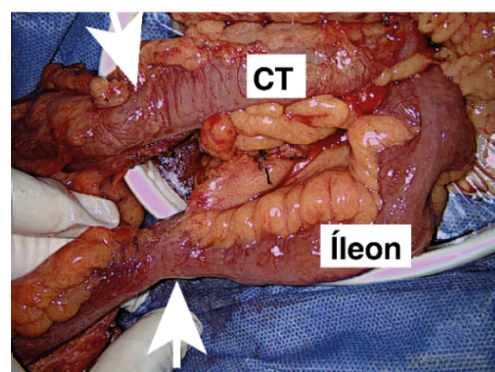


Figure 13. White light identification of perfusion in a colonic and small bowel segment during laparoscopic right colectomy with extracorporeal anastomosis. This assessment is based on the surgeon's subjective evaluation of the color, peristalsis, temperature, and bleeding of the bowel ends. CT: transverse colon. The arrows points to the chosen section site.

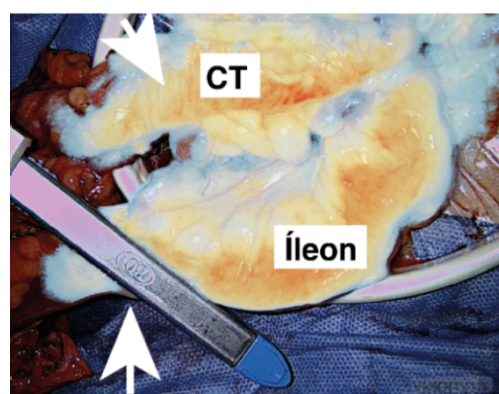


Figure 14. Identification of perfusion in a segment of the colon and small intestine using indocyanine green (ICG) angiography during laparoscopic right colectomy with extracorporeal anastomosis (same case as in the previous figure) at the moment of bowel sectioning with a stapling device. The evaluation was performed with an external camera in color map mode. The perfused intestinal segments are those reached by the ICG and are therefore colored. This indicates that the suture is placed in a perfused area. Note the difference between the ischemic proximal colon (to be resected with ligated mesentery) and the well-perfused distal (transverse) colon. Intensity map mode of the VisionSense platform (Medtronic) was used. CT: transverse colon. The arrows indicate the chosen section site.

In cases of low rectal surgery, the colon that must descend is only perfused by the vascular arcade. According to fluid physics and Poiseuille's law, the longer the conduit, the lower the pressure at its end (Fig. 15). Therefore, the farther the perfusion point is from the aorta, the lower the pressure at that point. In this sense, ligating the IMA increases the distance from the aorta to the left colon, which is supplied by the MCA or the Moskowitz artery, if present. For this reason, fluorescent angiography is more beneficial for left resections.

$$Q = \frac{\pi \cdot r^4 \cdot \Delta P}{8 \cdot \eta \cdot L}$$

Figure 15. Poiseuille's law states that the flow rate (Q) is directly proportional to the pressure difference (ΔP) and the fourth power of the pipe radius (r^4), and inversely proportional to the viscosity of the fluid (η) and the length of the pipe (L).

A systematic review and meta-analysis revealed that the use of ICG angiography in rectal surgery was associated with an 81% reduction in the risk of fistula.³⁰ Specifically, in minimally invasive rectal resections, ICG angiography led to a change in the location of the colonic section in 15% of cases, significantly reducing the incidence of AF by half (Figs. 16).³⁶

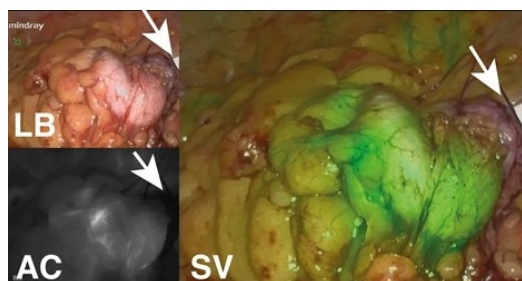


Figure 16. Fluorescent angiography performed during laparoscopic anterior resection. Note the adequate perfusion of the distal end of the colon where the anvil is placed. This is shown by the green color in green overlay (SV) mode and the white color in high contrast (AC) mode. LB: White light mode. The UX5 system (Mindray) was used. The arrows points to the anvil.

A randomized clinical trial involving 850 patients revealed a notable decrease in AF (grades B and C) among those who underwent ICG compared to those who did not (4.7 vs. 8.2%; $p=0.044$).³³ Recent reviews and meta-analyses support this decrease in anastomotic complication rates associated with ICG use. Similar indications and results have been observed with fluorescent angiography in robotic colon and rectal surgery (Figs. 17).³⁷⁻⁴²

High ligation of the SMA has been described as a cause of reduced blood flow in the sigmoid colon, dependent on the marginal artery.³⁹ This could explain cases of colonic ischemia following colorectal resections and abdominal aortic aneurysm repair.⁴⁰⁻⁴²

As in other perfusion studies, such as CEUS, a fluorescence intensity curve can be described at the level of the intestinal wall during ICG angiography (Figs. 18 and 19).^{23,43}

In a recent presentation, the normal fluorescence curve of the colon was described, which has an ascending phase, a peak, an attenuation phase, and a subsequent plateau.⁴⁴

It is important to note that, in contrast to radiographic contrast angiography, in which the contrast agent is completely eliminated after the venous phase, fluorescent angiography results in the retention of ICG in the tissue interstitium. This phenomenon results in a plateau in fluorescent intensity after

venous time, which complicates the evaluation of ICG angiography when dye is reinjected (Fig. 20). The intensity of the plateau signal is directly related to the dosage of ICG administered; a lower dosage yields a lower signal intensity.⁴⁵

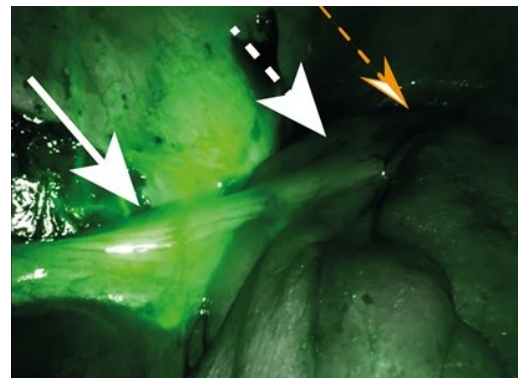


Figure 17. Fluorescent angiography performed during a robotic anterior resection. The Da Vinci Xi platform's firefly mode is used. Note the lack of perfusion at the distal end of the colon with the anvil in place (solid arrow) compared to the proximal colon (dashed arrow). In this case, fluorescent angiography significantly altered the surgical plan, resulting in the resection of an additional segment of the descending colon where adequate perfusion existed for a safe anastomosis. Anvil: orange arrow.

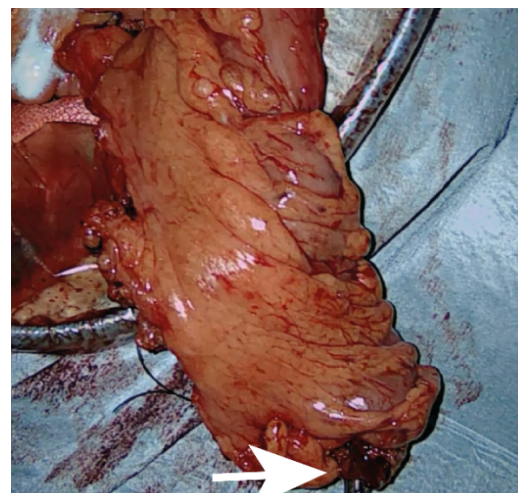


Figure 18. White light identification of perfusion in a left colonic segment after the descent of the splenic flexure and before performing a colorectal anastomosis. The assessment is based on the surgeon's subjective impression of the color, peristalsis, temperature, and bleeding of the tissue ends. The arrow points to the anvil.

There is a widespread consensus in the scientific community regarding the differential and superior perfusion of the small intestine compared to the colon. We have compared the perfusion curve in fluorescent angiograms of the small intestine and the colon. Our analysis revealed no significant differences between them in terms of the ascending slope and maximum intensity obtained.

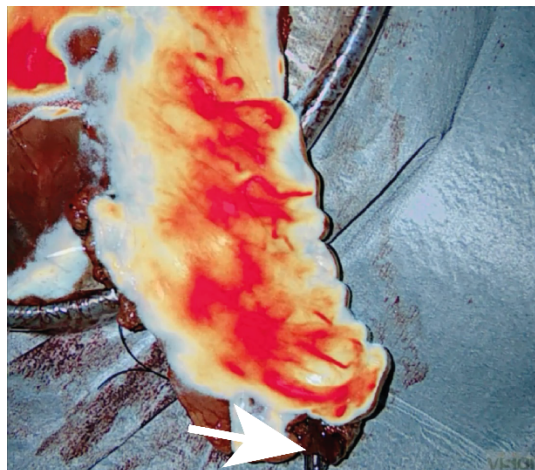


Figure 19. Identification of perfusion in a segment of the left colon using indocyanine green (ICG) angiography, after the descent of the splenic flexure and before the performance of a colorectal anastomosis (same case as the previous figure), following the placement of the anvil. Note the red color (the most intense on the color map) at the distal end of the colon where the anvil is located. This indicates adequate perfusion of the colonic end. The VisionSense system (Medtronic) was utilized. Arrow: anvil.

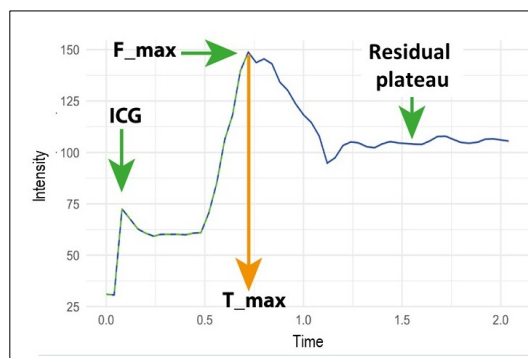


Figure 20. Time-fluorescence intensity curve after indocyanine green (ICG) injection. The main parameters are identified as follows: F_{max} , the maximum fluorescence intensity; T_{max} , the time until maximum intensity is reached; and the residual plateau, which corresponds to the stable fluorescence level after uptake. These values enable an objective evaluation of intraoperative tissue perfusion.

Conversely, by observing these curves, particularly the slope of the ascending phase and the mean time to peak fluorescence, it is possible to determine perfusion patterns that are associated with an increased risk of developing AF. A study of 86 patients undergoing laparoscopic anterior resection analyzed the incidence of anastomotic complications. The authors concluded that slow perfusion was an independent risk factor for anastomotic complications in a logistic regression model, considering the fluorescence emission slope and the time to reach the maximum.⁴³ Quantitative analysis of perfusion patterns with ICG, using the time to reach maximum fluorescence, allows the identification of segments with poor perfusion and the definition of different risk groups for the development of AF.

Conversely, a correlation has been established between these curves and tissue oxygen saturation greater than 60% for T_{max} , $T_{1/2_{max}}$, and slope. The

respective sensitivities are 97, 97, and 71%, and the respective specificities are 90, 54, and 100%.⁴⁶

When evaluating the effect of high vs. low ligation on distal colonic perfusion with ICG, no significant difference in maximum fluorescence (F_{max}) was observed. However, the time to reach maximum fluorescence (T_{max}) was significantly longer, and the emission slope ($Slope_{max}$) was significantly lower in the high ligation group than in the low ligation group. AL has been associated with neoadjuvant chemoradiotherapy and a reduction in F_{max} .⁴⁷

Computed Tomography Angiography

Colonic perfusion patterns, evaluated using CT angiography techniques, provide key information for the diagnosis and therapeutic planning of colorectal cancer. These imaging techniques offer a more comprehensive understanding of vascularization and perfusion dynamics, which are critical for the evaluation of the biological behavior of tumors and predicting surgical outcomes.⁴

Goh et al.⁴⁹ applied a two-dimensional fractal analysis to evaluate the spatial pattern of perfusion in colorectal adenocarcinomas using CT-enhanced dynamic perfusion (CT-Perfusion). Compared with the normal colon, tumors exhibited a significantly higher fractal dimension (1.71 ± 0.07 vs. 1.61 ± 0.07 ; $p \leq 0.001$) and greater fractal abundance (7.82 ± 0.62 vs. 6.89 ± 0.47). The analysis also demonstrated that regions of low perfusion exhibited higher fractal values versus regions of high perfusion, thereby indicating the presence of intratumoral vascular heterogeneity.

Similarly, lacunarity curves exhibited a shift toward higher values in tumors, suggesting a more irregular distribution of flow.

A fractal analysis of CT-Perfusion data reveals different perfusion patterns between cancerous and normal colonic tissue, with higher fractal dimensions in tumors.^{48,49}

Preoperative simulation using 3D computed tomography angiography (3D-CTA) is useful in identifying arteries and determining LND from an oncological standpoint for each patient.⁵⁰ In a retrospective study of 61 patients with splenic angle cancer, Kawashima et al.⁵⁰ were able to identify the vascular anatomy of the splenic angle and determine the extent of LND required (left hemicolectomy or segmental resection) in all patients evaluated using 3D-CTA.

In a study led by Son et al.,⁵¹ vascular parameters such as bifurcation type, vessel, diameter and length, and arterial calcification were evaluated using CT angiography and correlated with the risk of poor sigmoid perfusion after IMA clamping. Atherosclerotic calcification of the main mesenteric arteries was not associated with a reduction in mean arterial pressure in the marginal arcade after IMA clamping. However, cardiovascular risk factors and a short IMA were related to this reduction.

The integration of 3D-CTA with functional assessment of perfusion facilitates safer resection planning, particularly in elderly patients or those with vascular comorbidities.

Relative incidence of vascular disorders in digestive surgery

Vascular disorders of the colon are a rare group of colorectal pathologies that require hospitalization and surgical treatment. When compared to other causes of acute abdomen, their incidence is significantly lower. However, they have a significant clinical impact due to the severity of the condition and the complexity of initial diagnosis. Therefore, it is crucial to be aware of their presence and to be familiar with the necessary diagnostic and therapeutic approaches. The low incidence of the disease contrasts with its high morbidity and mortality rates. While the most common emergency digestive surgeries have mortality rates of less than 5%, surgery for colonic or mesenteric ischemia can exceed 30%. Gastrointestinal vasculitis is an even rarer condition, and if it is not diagnosed early, it can lead to ischemia and intestinal perforation, which can result in fatal outcomes. This discrepancy between rarity and impact demands that colorectal surgeons maintain a high index of clinical suspicion in cases of atypical acute abdomen.

Hospitalization data in the United States corroborate the low incidence of acute mesenteric ischemia compared to other digestive emergencies. Research by the *World Society of Emergency Surgery* (WSES) indicates that mesenteric ischemia accounts for between 0.09 and 0.2% of all emergency surgical admissions, with an overall mortality rate that exceeds 40% in many cases.⁵² This finding is based on a comprehensive analysis of more than three million adult hospital admissions in Maryland, USA. From 2009 to 2013, a total of 2,255 cases of acute mesenteric ischemia were documented. This figure represents only 0.07% of recorded hospital admissions, with an estimated annual rate of 10 per 100,000 inhabitants and an in-hospital mortality rate of around 24%.⁵³ In another population-based study conducted in Malmö, Sweden, the overall incidence of acute mesenteric ischemia was estimated at 2 cases per 100,000 person-years.⁵⁴ A review of the Nationwide Inpatient Sample (NIS) from 2000 to 2012 reveals that the occurrence of hospitalizations due to mesenteric ischemia remained consistent at approximately 1 per 1,000 admissions. These cases exhibited a high mortality rate of nearly 30% in acute forms.⁵⁵

According to estimates, the rate of hospitalizations with a primary or secondary diagnosis of intestinal angiodysplasia in adults with bleeding is 51.3 per 100,000 inhabitants per year. In the same period, the rate of hospitalizations without bleeding is 35.4 per 100,000 inhabitants per year. These data are drawn from a study conducted in the United States between 2001 and 2011.⁵⁶

The incidence of gastrointestinal vasculitis is even lower. A study of 131,367 histological samples from the digestive tract revealed that only 29 cases (0.02%) showed evidence of vasculitis, the majority of which had no initial clinical suspicion.⁵⁷

In our setting, the available published evidence is limited, but it aligns with international literature, indicating that these entities represent a negligible percentage of hospital admissions for digestive diseases. A study from Hospital Fernández (Buenos Aires) on small vessel vasculitis included only 29 patients over 16 years, reflecting the rarity of these diagnoses even in a referral center.⁵⁸ A Chilean national registry of ANCA vasculitis reported an approximate prevalence of 16.5 per 100,000 inhabitants, with variable annual incidences (4.4 to 19.9 per 100,000), although without specifying colonic involvement or the need for surgery.⁵⁹ At the regional level, a Chilean study reported that acute mesenteric ischemia accounts for less than 2% of acute abdomen cases, but with a mortality rate that can exceed 60%. In this context, local hospital case series, although limited in absolute numbers, acquire academic and clinical relevance by demonstrating that these entities, despite their low incidence, constitute one of the most challenging scenarios in emergency colorectal surgery.

A retrospective study was conducted of emergency surgical procedures performed in the Gastroenterological Surgery Division of the Hospital de Clínicas José de San Martín between 2015 and 2024. A review of surgical protocols was conducted, using specialized terminology such as *ischemia*, *infarction*, *necrosis*, *thrombosis*, and *vasculitis* to identify vascular pathologies. A total of 6,669 emergency procedures were examined, with an average of 667 procedures per year (SD: 140). Of these, 583 met the established selection criteria. Twenty-seven cases were excluded due to the absence of vascular pathologies during the review of the surgical protocol, resulting in 556 cases being included in the analysis of vascular pathologies. The annual mean number of vascular disorders diagnosed was 55.3 cases (SD 14.6).

The distribution by location showed 231 cases in the colon (with an annual average of 23.1; SD 4.7), of which 99 were recorded in the right colon (annual average of 9.9; SD 4.0) and 132 in the left colon (annual average of 13.2; SD 4.9). Small bowel involvement was documented in 176 patients (mean per year: 17.6; SD 7.9), while combined small bowel and colon involvement was observed in 146 cases (mean per year: 14.6; SD: 6.5). Finally, three cases of vasculitis were reported as the cause of intestinal ischemia (mean per year: 0.3; SD 0.5). The cases of vasculitis occurred in patients with a history of systemic lupus erythematosus (Fig. 21).

The proportion of vascular disorders of the colon in relation to the total number of emergency surgeries during the period was 0.08% (SD 0.022), reflecting the relatively low frequency of this condition in the context of emergency gastrointestinal surgery. The mortality rate observed for cases of vascular disorders was 33% (18.3 cases per year, SD: 4.87).

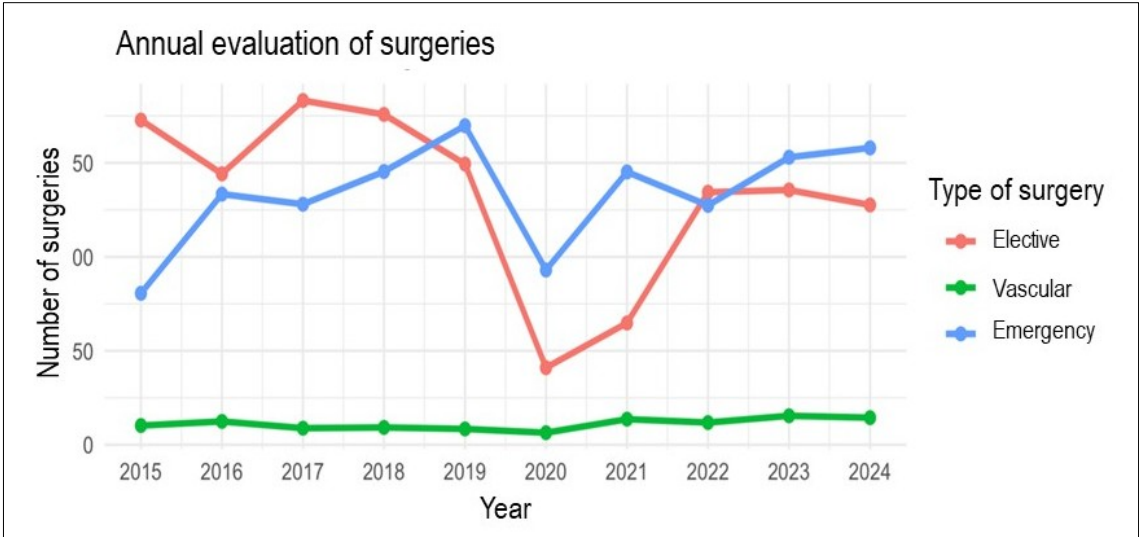


Figure 21. Annual evolution of elective, emergency and vascular surgeries for the period 2015-2024. There is significant variability in the number of elective and emergency procedures throughout the period, with a notable decline in 2020, the year in which the impact of the COVID-19 pandemic was evident. Surgical procedures for vascular disorders remained stable at low levels, representing a small percentage of the total volume of procedur

Intestinal Ischemic Disease

Ischemic bowel disease (ISBODI) is the most prevalent vascular disorder of the gastrointestinal tract. ISBODI has been observed most frequently in the elderly population, especially in patients with atherosclerosis of the mesenteric vessels. This condition arises from an insufficient supply of oxygenated blood to a specific area of the intestinal wall. The term encompasses a heterogeneous group of entities that can be classified as small bowel ischemia or mesenteric ischemia, and large bowel ischemia or ischemic colopathy (IC). In this report, we will focus on the latter presentation.

From a clinical perspective, mesenteric ischemia is classified into two distinct categories: acute mesenteric ischemia (AMI) and chronic mesenteric ischemia (CMI). IC is the most prevalent entity among intestinal disorders associated with ISBODI.

Ischemia is defined as the absence or deficiency of tissue perfusion. This condition can be caused by various situations, allowing it to be classified into three categories: arterial insufficiency, venous insufficiency, and microvascular insufficiency. The causes of arterial insufficiency are described in detail below.⁶⁰⁻⁶²

Arterial insufficiency is the most common cause of intestinal ischemia, classified into two types: occlusive and non-occlusive.⁶⁰ Occlusive arterial insufficiency, also known as peripheral arterial insufficiency, is characterized by an obstruction in arterial blood flow, which can be caused by intraluminal, intramural, or extramural factors (Table 4).^{60,61,53-66}

The most common intraluminal causes of vessel occlusion are thrombi and emboli. The latter generally originate from thrombi in the thoracic aorta or left heart, especially in the context of atrial fibrillation or valvular heart disease. Furthermore, radiological procedures that utilize coils, microspheres, and gels have the potential to cause iatrogenic occlusions.

Intramural causes include atherosclerosis, dissecting aneurysms, radiation damage, amyloidosis, diabetes, and vasculitis (Fig. 22). Certain drugs and toxins that induce arterial vasospasm can lead to occlusion and are also considered causal factors. These include potassium salts, cocaine, and snake and scorpion venoms. In addition to causing vasospasm, cocaine has the capacity to fragment the internal elastic lamina, generate edema in the intima, and promote thrombosis and platelet aggregation.⁶⁶

Extramural causes of this condition include compression of the vessels due to tumors or adhesions, as well as phenomena such as volvulus, torsion, and intussusception (Fig. 23).

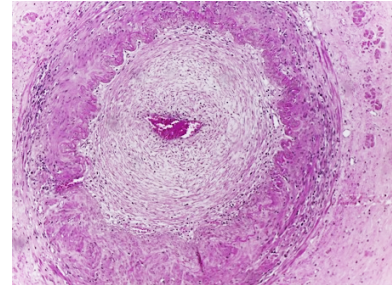


Figure 22. Histopathology. Artery with atherosclerosis. Thickening and fibrosis of the intima are observed, with reduction of the vascular lumen and deposition of fibrotic material, findings characteristic of advanced atherosclerotic disease. Hematoxylin-Eosin, 5



Figure 23. Segment of the small intestine with macroscopic changes of ischemia due to a strangulated hernia in the context of a closed-loop obstruction. Observed characteristics of advanced vascular compromise with transmural necrosis include dilation, parietal edema, and a purplish-black coloration.

The determining factors for prognosis in occlusive disorders are the size of the obstructed vessel and the degree of available collateral circulation. Atheroembolic occlusion of the terminal arterioles in the intestinal submucosa typically results in focal mucosal ulcerations.

Non-occlusive ischemia occurs when there is low blood flow. Reduced intestinal blood flow may be indicative of systemic conditions, including hypotension, heart failure, and various types of shock, such as cardiogenic, hemorrhagic, septic, and traumatic shock (Table 5). Arrhythmias can also alter mesenteric blood flow (Table 5).^{60,61} Using vasoconstrictive drugs such as digitalis, vasopressin, and propranolol can exacerbate ischemia by diminishing vascular diameter. Dehydration is regarded as a contributing factor, as it leads to a reduction in intravascular volume, consequently impairing intestinal perfusion.⁶⁰ Hypoxemia, caused by respiratory failure or decreased oxygen availability, can worsen circulatory failure, exacerbating ischemic damage to the intestinal mucosa.

The duration of arterial insufficiency is a crucial factor in the prognosis of non-occlusive ischemia. The intestinal tract has remarkable tolerance to hypoxia, facilitating the diversion of splanchnic circulation to the central nervous system during severe shock. Although the mucosa is the layer of the intestinal wall most susceptible to hypoxia, it also has the greatest

regenerative capacity. Therefore, complete recovery is possible even after ischemic damage to the mucosa.^{60,63,64}

Table 4. Causes of occlusive arterial ischemia.

Category	Causes
Intraluminal causes	Thrombi, emboli, coils, microspheres, and gels utilized in radiological procedures.
Intramural causes	Atherosclerosis, dissecting aneurysms, radiation damage, amyloidosis, diabetes. Vasculitis: polyarteritis nodosa, granulomatosis with polyangiitis (Wegener's disease), Behçet's disease, rheumatoid arthritis, systemic lupus erythematosus, scleroderma, syphilis. Drugs and toxins: potassium salts, cocaine, snake and scorpion venom.
Extramural causes	Compression of vessels by tumors, adhesions, volvulus, torsion, and intussusception.

Table 5. Causes of non-occlusive ischemia.

Category	Causes
Low flow	Systemic hypotension: Low blood pressure that reduces intestinal blood flow.
	Heart failure: Decreased heart function that affects blood flow.
	Shock (cardiogenic, hemorrhagic, septic, traumatic): Critical conditions affecting blood circulation.
	Arrhythmias: Heart rhythm disturbances that can compromise blood flow.
	Vasoconstrictor drugs: Medications that narrow blood vessels, reducing blood flow.
	Other medications: Digitalis, vasopressin, and propranolol, which can reduce intestinal blood flow.
	Dehydration: Excessive loss of fluids affecting blood perfusion.
Hypoxemia	Respiratory compromise: Breathing difficulty that reduces overall oxygenation.
	Decrease in oxygen available to tissues.

Ischemic Colopathy

Epidemiology

The term ischemic colopathy (IC) is preferred over “ischemic colitis” because some patients do not present a documented inflammatory phase of the disease.⁶⁷ IC is the most common gastrointestinal vascular disease (50-60% of cases) and a frequent cause of lower gastrointestinal bleeding (LGIB).⁶⁸ IC is the etiology in 9-24% of patients hospitalized for LGIB, being one of the most frequent diagnoses along with colon cancer, diverticulosis, and angiodysplasia.⁶⁹ The incidence in the general population varies from 4.5 to 44 cases per 100,000 people per year.^{68,69} However, the incidence of IC is probably underestimated because many patients do not seek medical attention when symptoms are mild. Women appear to be more affected than men, especially among patients under 40 years of age, accounting for 57 to 76% of cases.^{67,69,70} Other report describes a significant preponderance of women, even among those over 69 years of age.⁶⁹ Two clinical contexts are described: spontaneous IC (SIC) and postoperative IC (POIC), which occurs mainly after aortic and colonic surgery.⁷¹ SIC occurs more frequently in women and POIC in men. IC typically occurs after age 60, and patients with SIC are significantly older than patients with POIC (73 vs. 68 years).⁶⁹ Mortality rates range from 4 to 12%, and there is significant variation among different reports due to differences inclusion criteria, case verification methods, comorbidity rates, and surgical procedures.⁶⁷

Recurrence increases over time. The estimated cumulative recurrence rates at 1, 2-3, 4, and 5-6 years are 3%, 5%, 6%, and 10%, respectively.⁷² Hypercoagulable states have been reported as predisposing factors.⁶⁷

Definition

IC is a condition that occurs when blood flow to the colon is reduced to a level insufficient to maintain cellular metabolic function. It is a benign condition that occurs when there is sudden and temporary hypoperfusion of the colon wall.⁷³ It is essential to differentiate this condition from other causes of acute and chronic ischemic injury, such as embolisms and thrombosis.⁷³ In 1963, Boley et al. described 5 patients with a condition they termed “reversible vascular occlusion of the colon”.⁷⁴ In 1966, Marston et al.⁷⁵ described 16 patients with three stages of a spontaneous disease they termed ischemic colitis.⁷⁵ Williams⁷⁶ described his case series of 54 patients with a new clinical entity, ischemic colitis, and distinguished two possible courses: moderate and severe.⁷⁶ In his report, he states: “..., ischemic colitis is an acute colonic process that occurs in both men and women, with an average age of onset of 70 years. The main symptom is mild abdominal pain, accompanied by significant diarrhea, commonly with blood. The diagnosis is made by observing an acute ulcerative process in the mucosa through barium studies and/or endoscopy, or by identifying an exudative process that manifests itself in barium studies as thumbprinting, along with secondary features such as transverse ridges, spasm, or rigidity, and in endoscopy as hemorrhagic bullae. Histological evaluation, either from endoscopic or surgical material, shows the presence of an acute mucosal process with

extensive submucosal edema and/or hemorrhage. There may be an inflammatory component, but identification of a specific microorganism is not essential for diagnosis, as the absence of bacterial colonization is not uncommon. The extent of the process is determined at the initial examination, as progression to other segments of the intestine is unusual”

Pathophysiology

The colon is susceptible to ischemia due to its reduced blood flow compared to other abdominal organs and decreased perfusion when functionally inactive. IC consist of a spectrum of lesions arising from ischemia of the colonic wall due to inadequate regional blood flow.⁷⁷ At one end of the spectrum is colopathy, which is clearly caused by ischemia.

This ischemia may result from occlusion, ligation, or exclusion of the vessels involved (e.g., after repair of an abdominal aortic aneurysm) or from a combination of low flow and vasoconstriction (as occurs in IC that develops in a patient with sepsis receiving high doses of vasopressors). However, the spectrum of ischemic colitis that remains may be associated with a variety of etiologies. In fact, there is little evidence suggesting that ischemia is the direct cause of most cases of "ischemic colitis".^{76, 78}

The initial ischemic injury triggers a cascade of events that exacerbate tissue damage. During periods of ischemia, the metabolism of ATP leads to the accumulation of hypoxanthine and the conversion of xanthine dehydrogenase to xanthine oxidase.⁷⁹ During reperfusion, the presence of xanthine oxidase catalyzes the production of reactive oxygen species, which initiates lipid peroxidation and the release of inflammatory mediators.

The final outcome of the ischemic process is that colonocytes become acidotic and dysfunctional, lose their integrity, and die. Although the etymological root of the word "ischemia" comes from the Greek iskhaimos, meaning "stopping of blood," blood flow does not need to stop completely; it only needs to decrease significantly to cause ischemic damage. Furthermore, ischemia may lead to reperfusion injury, which can result in greater damage compared to reduced blood flow without reperfusion, even after brief periods of ischemia.⁷³ The degree to which colonic blood flow must decrease before ischemia occurs varies depending on the severity of the episode, the degree of preexisting collateral vasculature, and the duration of the low-flow state.⁷³ Mechanical obstruction of the colon's blood supply can reproduce the entire clinical spectrum of ischemic colitis.

CI can manifest as reversible or irreversible damage (Fig. 24). Reversible damage includes colopathy, with rupture of the subepithelial microvasculature, subepithelial hemorrhage or edema, and colitis. Colitis reflects a stage of progression in which the overlying mucosa ulcerates as the edema and subepithelial blood are reabsorbed.⁷³ In irreversible disease, this reabsorption occurs rapidly, usually within 3 days. Ulcerations may persist for several months before resolving, although during this time the patient is usually asymptomatic. The irreversible manifestations of IC include gangrene, fulminant colitis, stenosis formation, and, in rare cases, chronic ischemic colitis (Table 6).^{67, 73} Another uncommon manifestation of irreversibly damaged bowel is recurrent sepsis due to bacterial translocation.⁶⁷

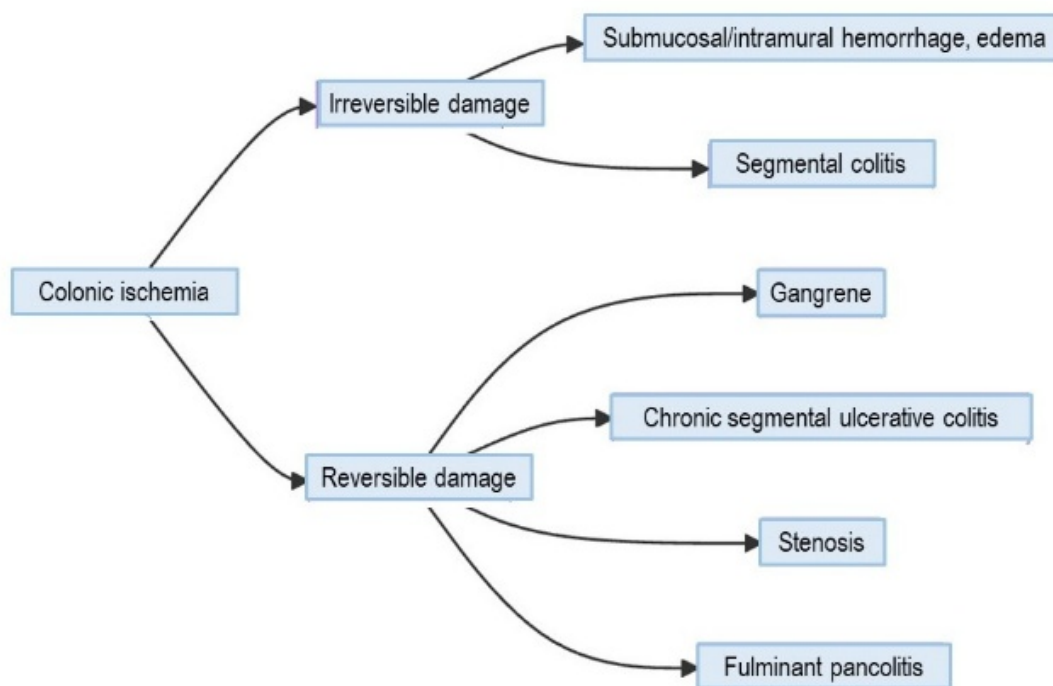


Figure 24. Evolution of ischemic colopathy according to the type of damage

Colonic ischemia is characterized by segmental involvement, with the left colon affected in 80% of cases (splenic flexure: 25%, sigmoid colon: 55%), while the right colon is affected in 10-20% of cases.⁶⁸ On the other hand, total colon involvement is rare, occurring in only 10% of cases. Extensive collateral arterial vascularization provides relative protection to the colon against ischemia. There are three anatomical areas that demonstrate an elevated risk of ischemia in the event of a congenital absence of the marginal vascular arch. These areas have been identified as the splenic flexure at Griffith's point, where the marginal artery of Drummond interconnects the right, middle, and left colic arteries; the right colon, where the arch connects the superior branch of the ileocolic artery to the inferior branch of the right colic artery; and the rectosigmoid junction at Sudeck's point, where the sigmoid artery ima interconnects the caudal sigmoid artery with the superior rectal artery.⁶⁷

Table 6. Clinical course patterns reported in cases of ischemic colitis, and their relative frequencies.

Type	Relative frequency (%)
Reversible colopathy and transient colitis	>50
Transient colitis	10
Chronic ulcerating colitis	20
Stricture	10
Gangrene	15
Fulminant universal pancolitis	<5

Risk Factors

There are recognized risk factors for IC, and their presence should increase the level of suspicion and direct diagnostic efforts to confirm or rule out this condition (Table 7).

In patients with suspected IC, a history of irritable bowel syndrome, constipation, or use of antidiarrheal medications should be investigated. In an epidemiological case-control study comparing 700 patients with IC to a control group, it was found that patients with IC had three times more functional bowel disorders (OR = 2.75) and took more antidiarrheal medications (1 in 3 cases vs. 1 in 9 cases in the control group; OR = 2.29).⁸²

Table 7. Causes of colonic ischemia grouped according to classification: systemic hypoperfusion, small-vessel disease, thrombosis/embolism, and iatrogenic factors.

Systemic hypoperfusion	Small-vessel disease	Thrombosis or embolism	Iatrogenic
Heart failure	Atherosclerosis	Congenital hypercoagulable states	Surgical
Septic shock	Diabetes mellitus	Factor V Leiden mutation	Colectomy with IMA ligation
Haemorrhagic shock	Hypertension	Prothrombin G20210A mutation	ERCP-related mesenteric haematoma
Pancreatitis	Hyperlipidaemia	Protein C or S deficiency	Drugs
Hypovolaemia	Vasculitides (e.g., SLE, PAN,		Alosetron
Diuretics	GPA, RA)	Acquired hypercoagulable states	Antihypertensives, digoxin, cocaine, interferon-
Haemodialysis	Radiation	Antiphospholipid syndrome (aPL, lupus anticoagulant)	ribavirin, vasopressors, NSAIDs, pseudoephedrine,
Endurance exercise (marathon)	Amyloidosis	Disseminated intravascular coagulation	psychotropics,
Major cardiovascular surgery (e.g.,		Paroxysmal nocturnal haemoglobinuria	
CABG, aortic repair)		Oral contraceptives	
Snake venom; anaphylaxis		Cardiac arterial emboli; cholesterol emboli	

SLE: systemic lupus erythematosus; PAN: polyarteritis nodosa; GPA: granulomatosis with polyangiitis; RA: rheumatoid arthritis; CABG: coronary artery bypass graft.

Cardiovascular disease and diabetes mellitus are significantly associated with the onset of IC.⁷⁰ A report comparing 161 patients with IC to 322 control subjects determined that type 2 diabetes, dyslipidemia, occlusive peripheral arterial disease, coronary artery disease, heart failure, digitalis use, and aspirin use were independent risk factors for IC.⁸³

Patients with underlying cardiovascular disease, chronic obstructive pulmonary disease, or those requiring dialysis are at increased risk for POIC.^{84,85}

Further research has identified additional risk factors, including age over 65, female sex, chronic obstructive pulmonary disease, inflammatory bowel disease (IBD), constipation, a history of cancer, and hepatitis C virus infection with cryoglobulinemia.⁸⁶ In a series of 80 patients with IC, lower abdominal pain, with or without LGIB, age over 60 years, hemodialysis, hypertension, type 2 diabetes, hypoalbuminemia, and the use of antidiarrheal medications were present in 40% of cases, compared with 8% of patients in the control group.⁸⁷ In this series, all patients with four risk factors had IC. Another study identified independent risk factors associated with the diagnosis of IC in patients with LGIB. These factors include the occurrence of in-hospital gastrointestinal bleeding, chronic renal failure, and female sex.⁸⁸

The combination of right colon IC and hemodialysis has been the subject of specific studies.⁸⁹ IC has been attributed to repeated episodes of hypotension, causing microvascular vasoconstriction in the right colon. In this context, any suspicion of IC should prompt

a thorough and expeditious investigation to ensure timely diagnosis. According to the study by Flobert et al.,⁸⁹ right colon involvement was identified as the sole independent risk factor associated with severe forms of IC. This risk factor was observed in 82% of patients on renal dialysis and only in 26% of those not on dialysis.⁸⁹ In patients diagnosed with chronic renal failure (CRF) and IC, the likelihood of undergoing surgical intervention and mortality is 8.5 times higher compared to individuals without CRF. Consequently, a more aggressive diagnostic and therapeutic approach is recommended for patients with CRF.

Clinical Diagnosis

The diagnostic strategy varies depending on the clinical presentation of IC, whether it is SCI or POIC. Postoperative symptoms may appear immediately or later. The symptoms exhibited depend on various factors, including the extent of colonic involvement, the depth of mural damage, the severity of onset, the possibility of spontaneous recovery, and variations in vascular anatomy. However, clinical signs and symptoms, as well as laboratory test results, are not specific to IC. Therefore, an urgent CT scan and sigmoidoscopy are necessary to confirm the diagnosis, assess the need for surgery, and determine the prognosis of the disease. No bowel preparation is necessary prior to endoscopy; cleaning the distal colon with a simple enema is sufficient.

The most common symptoms are abdominal pain (typically sudden onset and moderate intensity) and LGIB (infrequently severe, but often accompanied by diarrhea with urgent bowel movements). Abdominal distension is a prevalent occurrence, frequently accompanied by vomiting due to concomitant ileus. The relative frequency of symptoms is as follows: abdominal pain (73%), diarrhea (61%), rectal bleeding (71%), and abdominal pain on palpation (60%).⁷⁰

Systemic inflammatory response syndrome (SIRS) is frequently observed, presenting with symptoms such as fever, tachycardia, and tachypnea. Therefore, it is essential to consider IC in the differential diagnosis of elderly patients presenting with acute-onset incomplete intestinal obstruction with atypical progression, with or without fever and diarrhea. IC can also mimic diverticulitis or intestinal pseudo-obstruction. Therefore, in these cases, the diagnosis of SIC should be considered to avoid delays in diagnosis and treatment.

It has been reported that between 10% and 25% of IC occur in the right colon. This presentation is associated with abdominal pain localized in the right flank, a history of coronary artery disease, or renal failure. Compared with other patterns of IC presentation, involvement of the right colon is associated with a higher frequency of coronary artery disease (39.2 vs. 21.4%) and end-stage renal disease requiring dialysis (20.3 vs. 7.7%).⁹⁰

Fifteen percent of cases manifest as fulminant gangrenous IC, a condition characterized by transmural colonic necrosis, perforation of the colon, and multiple organ failure. The predominant symptoms are peritoneal abdominal pain and shock.

IC patients who present with moderate hematochezia show improved outcomes compared to those who do not present with it or who present with greater bleeding.⁹¹ In a cohort of patients with IC, those who had moderate hematochezia had a lower rate of severe disease (25 vs. 52%, $p=0.001$). They also had a lower frequency of isolated ischemia of the right colon (7 vs. 28%, $p=0.001$), a lower rate of surgical interventions (13 vs. 36%, $p=0.001$), and a shorter hospital stay (12 vs. 17 days, $p<0.001$) compared to the cohort without hematochezia. The cohort with severe hematochezia had a higher frequency of severe disease (73 vs. 18%, $p<0.001$), a higher rate of surgical interventions (55 vs. 6%, $p<0.001$), a higher incidence of non-surgical complications, higher in-hospital mortality (45 vs. 0%, $p<0.001$), and a

longer hospital stay (28 vs. 10 days, $p=0.001$), compared to the cohort with mild hematochezia.

Laboratory Findings

Depending on the patient's preexisting conditions and the stage of ischemia or necrosis of the intestinal wall, laboratory tests may show abnormalities. Therefore, there are no specific laboratory markers for IC. However, elevated inflammatory markers, such as C-reactive protein and neutrophil count, are frequently observed.

The combination of metabolic acidosis, renal failure, and elevated serum lactate suggests the presence of ischemia and intestinal infarction. However, these abnormalities are not specific for the diagnosis of IC and usually manifest only in the severe and advanced stages of the disease. It is important to note that a normal lactate value does not rule out the presence of transmural ischemia of the colon.⁹² Neither leukocytosis nor lactic acidosis has sufficient predictive value for diagnosis. However, elevated arterial lactate levels have been associated with increased mortality.⁹² The mean preoperative plasma lactate level in survivors was 1.5 mmol/L, compared to 3.3 mmol/L in patients who died after surgery ($p < 0.05$). A lactate acidosis threshold of 2.5 mmol/L is established as an unfavorable prognostic factor associated with a significant increase in mortality.⁹³

Diagnostic Imaging

A CT scan is an essential component of the diagnosis and treatment of IC (Figs 25, 26, 27, 28, and 29). It facilitates the confirmation of clinical suspicions, provides a diagnosis of ischemia when it was not initially considered, and identifies associated complications.⁹⁴ Currently, CT scan is regarded as the diagnostic tool of choice for IC, as it enables the establishment of a diagnosis in an appropriate clinical context and the definition of the extent of ischemic damage.⁹⁵

The tomographic appearance of IC can vary. (Table 8) The most prevalent findings include segmental involvement with thickening of the intestinal wall (greater than 3 mm), which may appear "wet," with heterogeneous areas of edema, or "dry," characterized by mild homogeneous thickening.⁹⁴ Concentric thickening is typically greater than 6 mm, with an average of 10 mm, and moderately long segmental involvement (average of 19 cm) with loss of haustra. In 85% of cases, the condition is segmental.⁹⁶

CT scans may reveal narrowing of the colonic lumen, which can sometimes resemble a polypoid mass, a phenomenon referred to as "thumbprinting".⁹⁷ A "target" or "bull's-eye" sign may be evident, indicating hyperdensity of the mucosa and muscle with submucosal edema, irregular and frayed contours, and mesenteric inflammation with fat stranding. The kinetics of mural enhancement can assess the viability of the colon. The presence of the following signs of severity should be noted: intestinal pneumatosis, portal venous gas, abundant free peritoneal fluid, or pneumoperitoneum. The majority of patients exhibit mild to moderate amounts of free fluid in the abdominal cavity.⁹⁶ In 81% of patients with segmental ischemia, no macrovascular abnormality is observed on CT scan.⁹⁴

Table 8. Characteristic tomographic findings of colonic ischemia.

Finding	Description
Colonic wall thickening	Mean ~8 mm; may be heterogeneous (“wet”) or homogeneous (“dry”)
Submucosal edema	Hypodense areas within the bowel wall
Intramural gas	Pneumatosis intestinalis in severe cases
Luminal narrowing	May mimic a polypoid mass (“thumbprinting” sign)
Free peritoneal fluid	Nonspecific; associated with greater severity
Abnormal wall enhancement	Decreased or absent enhancement after contrast administration
Proximal colonic dilation	Associated with segmental ischemic strictures



Figure 25. Axial computed tomography scan, without intravenous contrast and with oral contrast, of a patient with ischemia in the right and transverse colon (arrows). Diffuse thickening of the colonic wall is observed, with segmental distribution, associated with submucosal hypodensity consistent with edema. The findings, in conjunction with the absence of mural enhancement, are indicative of acute colonic ischemia

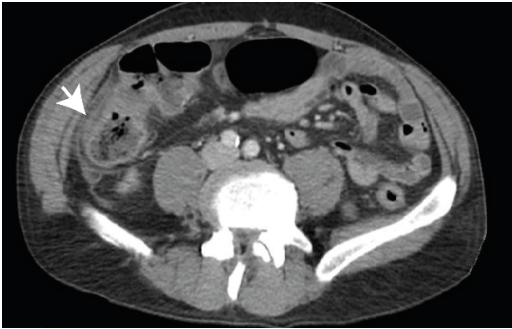


Figure 27. Axial abdominal computed tomography with intravenous contrast. Diffuse thickening of the right colonic wall is observed, with submucosal edema and loss of haustration, findings consistent with ischemic colitis (arrow). Luminal distension and slight striation of the pericolic fat are associated.



Figure 26. Computed tomography of the abdomen with intravenous contrast. Wall thickening and inflammatory changes in the right and transverse colon are observed, consistent with ischemic colitis (arrows). As an associated finding, an atrophic kidney is identified in a patient with chronic renal failure undergoing hemodialysis..

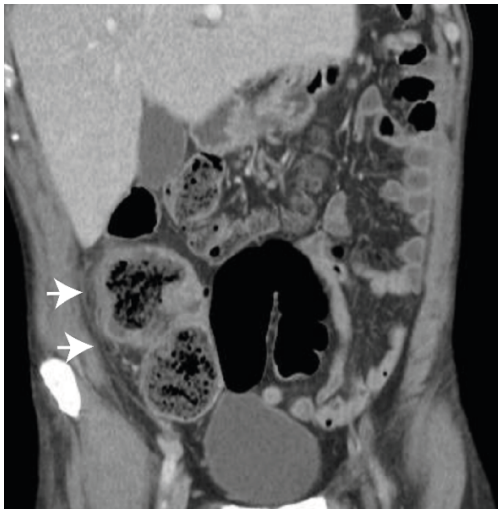


Figure 28. Abdominal computed tomography scan with intravenous contrast, coronal section. Thickening of the wall of the right and transverse colon is observed, with loss of the haustral pattern and mural edema, findings consistent with acute colonic ischemia (arrows). Discrete striation of the adjacent pericolic fat is also apparent.

Table 9. Balhazar tomographic classification of ischemic colitis.

Type	Description
I (acute IC)	Wall thickening with heterogeneous enhancement, low-attenuation areas due to severe edema, mucosal enhancement, irregular contour, loss of haustra, variable pericolic stranding
II (subacute IC)	Mild, concentric and symmetric mural thickening with homogeneous attenuation, well-defined contour, with or without minimal pericolic stranding
III (gangrenous IC)	Circumferential intramural gas consistent with pneumatosis intestinalis

Similarly, nuclear magnetic resonance imaging (MRI) has been proposed as an alternative for evaluating and monitoring IC. It could replace other invasive endoscopic procedures in the detection and classification of ischemic lesions when vascular damage is suspected (Table 10). In cases where iodine allergy or renal dysfunction contraindicate the use of contrast-enhanced CT, MRI may be an alternative for patient follow-up.^{95,96} MRI provides greater structural definition of the colonic wall and can therefore provide information on the evolutionary status of the lesion.

Table 10. MRI-based classification of ischemic colitis according to morphology, clinical interpretation and mean time from CT scan

Type (MRI)	Morphological features	Clinical interpretation	Mean time from CT
I (acute MRI)	Three-layer “sandwich” sign: high signal in the middle layer and low signal in the inner and outer layers	Acute IC	36 h (range 1-54)
II (subacute MRI)	Two-layer sign: high signal in the inner layer and low signal in the outer layer	Subacute IC	420.5 h (range 121-720)
III (subacute MRI)	Inverted two-layer sign: high signal in the outer layer and low signal in the inner layer	Subacute IC	420.5 h (range 384-457)

IC: ischemic colitis.

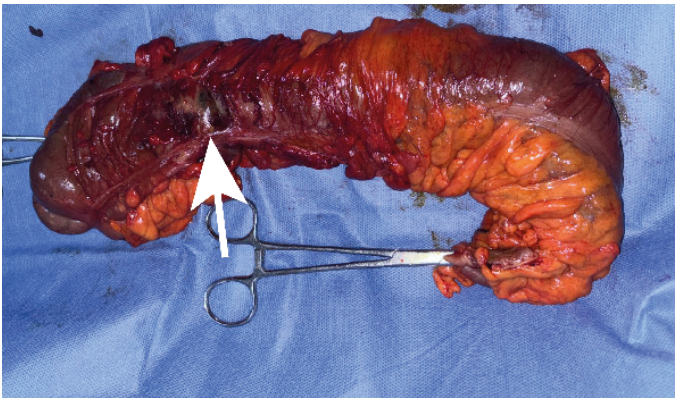


Figure 29. Surgical specimen obtained from a right hemicolectomy due to cecal ischemia. The macroscopic findings, characteristic of advanced colonic ischemia, include diffuse parietal thickening, extensive areas of transmural necrosis, and purplish-black discoloration. Arrow: ischemia with irreversible transmural necrosis.

While CT scan is a valuable diagnostic tool, it is necessary to note that the alterations observed in the image do not allow for reliable prediction of

progression to intestinal infarction. Furthermore, isolated CT findings are not always specific to IC, so the final diagnosis is based on a combination of clinical, laboratory, endoscopic, and histopathological data.⁹⁹ On the other hand, CT can be useful in suggesting the presence of IC in cases of acute abdomen where it was not suspected and in confirming or ruling out other differential diagnoses.

Mesenteric angiography, long considered the gold standard for evaluating vascular pathology of the digestive tract, currently has no place in the diagnosis of IC. Alterations in intestinal blood flow are frequently transient and may have returned to normal by the time symptoms appear. The presence or absence of angiographic abnormalities does not seem to be associated with the prognosis of the disease.⁸⁶ However, angiography may have a role in specific cases. CT angiography is widely regarded as the gold standard for diagnosing acute mesenteric arterial ischemia.¹⁰⁰ Angiography is particularly useful in situations where severe acute arterial occlusion is suspected, as it allows for the potential of endovascular treatment.

Endoscopic diagnosis

Colonoscopy is the most sensitive diagnostic test for identifying IC, and it can be performed without bowel preparation or with only a rapid cleansing. The timing of the procedure is determined by the patient's condition at the time of presentation. The findings will be distributed according to the topography of the IC, which includes patchy involvement, segmental erythema, edema, submucosal hemorrhagic nodules ("thumbprinting" on X-rays), mucosal erosions, and extensive ulcerations with a geographic pattern and confluent circumferential areas of necrosis (Fig. 30). Additionally, the presence of pseudomembranes, ischemic pseudopolyps, and cicatricial strictures can complicate a specific diagnosis. The Favier endoscopic classification is a system used to stage the severity of IC (Table 11).¹⁰¹ This classification guides the diagnosis, prognosis, and timing of surgery, which is indicated in stage 3 or stage 2 with multiple organ failure (MOF).¹⁰²

Table 11. Favier endoscopic classification of ischemic colitis. The extent of intestinal involvement is stratified, ranging from inflammatory changes to transmural necrosis.

Stage	Description
1	Mucosal edema and erythema
2	Mucosal ulcerations without necrosis
3	Colonic necrosis

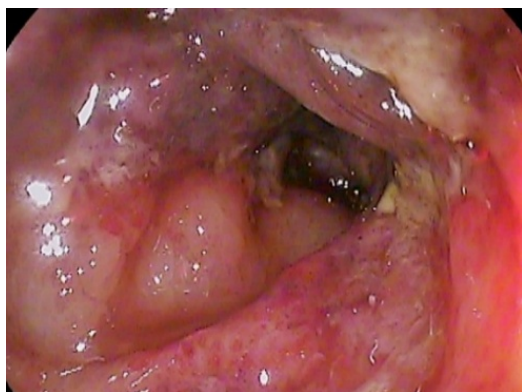


Figure 30. Colonoscopy revealing mucosa with intense erythema, areas of edema, superficial ulcerations, and patchy necrotic plaques. These findings are consistent with ischemic colitis. The presence of loss of the normal vascular pattern and friability of the mucosa corresponds to an advanced stage according to Favier's classification.

Although colonoscopy contributes to diagnosis, its efficacy is limited in severe cases. The indication of surgical intervention is determined by the patient's hemodynamic status. In 62% of cases, the results of a CT scan in 14%, the mucosa (necrosis) and the surgical appearance of the serosa. The decision to perform surgery does not depend on the results of the colonoscopy. Therefore, it is not indicated in severe cases. Zuckerman et al.¹⁰⁴ described a single, typical linear ulcer extending along the left anti-mesenteric colonic wall, known as the "single-stripe sign," as an endoscopic manifestation of mild ischemic colitis.

Pathological Diagnosis

cells are cells that retain their size and shape despite the disappearance of their cellular content.¹⁰⁵ The distribution of lesions is classically segmental and patchy, following the natural distribution of ischemia along the colon. A variety of concomitant nonspecific findings have been documented, including the infiltration of necrotic mucosa and edematous submucosa by hemosiderin-laden lymphocytes and macrophages. Furthermore, mucosal erosions or ulcerations, submucosal edema and hemorrhage, and submucosal microvascular thrombi are observed.¹⁰⁵

While there are similarities between occlusive and non-occlusive IC, there are also key differences. In occlusive disease, ischemia is strictly segmental and uniform within the affected region. In contrast, in non-occlusive cases, the involvement is patchy, with variable severity and, occasionally, greater extent. Segmental ischemic changes can occur in any part of the large or small intestine, but most often affect the colon in the vicinity of the splenic flexure.^{60,61}

In the early stages of acute ischemia, both in endoscopy and in samples obtained during resection, the mucosa is usually fragile, edematous, and

erythematous, showing a tendency to bleed easily when manipulated.⁶⁰⁻⁶²

During the initial phase of acute arterial ischemia, the predominant manifestation is edema. As necrosis progresses, the mucosa will take on a mottled appearance, with reddish and brownish tones.^{60,64}

In its most severe form, acute intestinal ischemia can lead to transmural necrosis and, in some cases, intestinal perforation. (Fig. 31)



Figure 31. Specimen of bowel resection, formalin-fixed, corresponding to the distal ileum and right colon with transmural ischemia. The findings include wall thickening, purplish-black discoloration, and extensive areas of necrosis, which are characteristic of advanced acute vascular compromise.

As the ischemia progresses, there is a gradual loss of the surface epithelium, revealing withered crypts ("ghosts"), dilation and congestion of the mucosal capillaries, hemorrhage, and hyalinization of the lamina propria due to the leakage of plasma proteins from the damaged capillaries, which may contain thrombi.⁶⁰⁻⁶² Residual viable epithelial cells frequently exhibit reactive changes, characterized by the loss of mucin, hyperchromatic nuclei, and mitotic figures. Initially, the inflammation is minimal (Figs. 32 and 33).^{60,64,65} The submucosa, on the other hand, is typically edematous, characterized by congested vessels, with or without concomitant hemorrhage. The muscularis propria may remain viable unless the ischemic damage is almost complete and of relatively acute onset (Fig. 34).^{60,64,65}

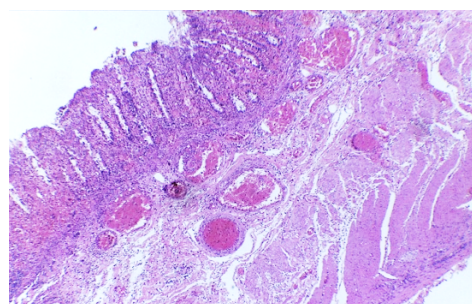


Figure 32. Histopathology of acute colonic ischemia. The superficial epithelium is lost, the lamina propria is hyalinized, and congestive vessels are present. These findings, along with necrosis of the mucosa and submucosa with focal hemorrhage, are characteristic of acute ischemic damage to the colonic wall. (Hematoxylin-Eosin, 15x)

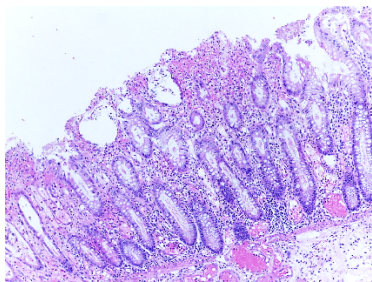


Figure 33. Histopathology of acute colonic ischemia. . The initial ischemic damage to the colonic mucosa is characterized by loss of the superficial epithelium and hyalinization of the lamina propria. (Hematoxylin-Eosin, 15x).

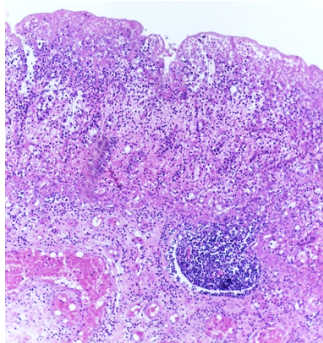


Figure 34. Histopathology of acute colonic ischemia. The morphology corresponds to an advanced stage of ischemic damage, with involvement of the entire thickness of the mucosa and absence of crypts. These findings are characteristic of diffuse mucosal necrosis (Hematoxylin-Eosin, 15x).

In certain cases, a superimposed bacterial infection, such as that caused by *Clostridium*, can induce the formation of gas bubbles within the intestinal wall, a condition known as intestinal pneumatosis (Fig. 35). Furthermore, the release of enterotoxins can result in the formation of a pseudomembranous pattern, analogous to that observed in pseudomembranous colitis (Figs. 36 and 37).^{60,61,64,106}



Figure 35. Colonic pneumatosis. Intraoperative imaging revealed the presence of multiple gas bubbles within the colon wall, visible through the serosa. This finding is indicative of intestinal ischemia

Chronic Ischemia

Chronic ischemia, which typically manifests recurrently, is distinguished by fibrosis. This fibrosis, usually circumferential, affects all layers of the intestinal wall, including the mucosa. At this stage, the presence of hemosiderin deposits can be observed, suggesting the occurrence of previous hemorrhagic episodes. Fibrosis, a pathological condition marked by the formation of fibrous tissue in the colon, can lead to the distortion of the

colonic wall, resulting in narrowing and, consequently, intestinal obstruction^{60,64}(Fig. 38).

In addition to fibrosis, typical morphological findings include:

- Mucosal ulceration with patchy re-epithelialization
- Distortion of crypt architecture and mucosal atrophy
- Paneth cell metaplasia
- Pseudopyloric metaplasia
- Ulcers of varying depth

The aforementioned changes have led to histological overlap with IBD, which can pose a diagnostic challenge.^{60,61,64,65}



Figure 36. Ischemic intestinal segment with dilated lumen and yellow-green exudate (pseudomembranes) on the mucosal surface.

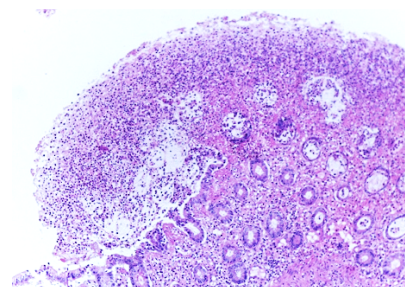


Figure 37. Histopathology of acute colonic ischemia. An inflammatory pseudomembrane is observed on the mucosal surface, with dense inflammatory infiltrate and necrotic debris, a typical finding of the acute phase of ischemic damage (Hematoxylin-Eosin, 7X).

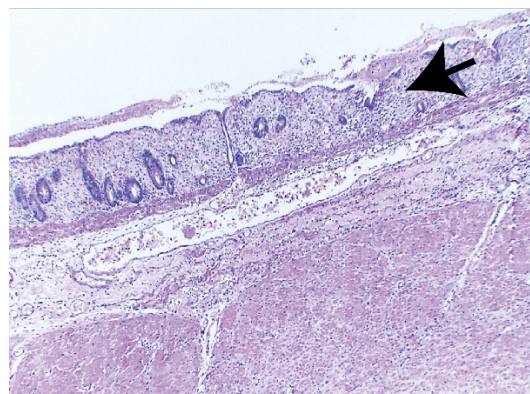


Figure 38. Histopathology of chronic ischemia. The colonic mucosa exhibits signs of atrophy, characterized by the loss of numerous crypts (arrow). (Hematoxylin-eosin, 7X).

Ischemic Colitis with Mass Formation

Ischemic colitis with mass formation is a subtype of colonic ischemia that has been well documented, although its detailed characterization is relatively recent. This type of ischemia is often diagnosed as a suspected neoplastic lesion during a preoperative evaluation when a mass is identified on imaging studies. It is more prevalent in elderly patients and tends to occur in the right colon, particularly in the cecal region. However, cases of rectal involvement have been documented.^{60,63}

Isolated Cecal Necrosis

From an anatomopathological perspective, isolated cecal necrosis is defined as necrosis or infarction confined to the cecum, with varying degrees of severity and extent. In some cases, no obvious vascular abnormalities are identified, highlighting the importance of microvascular factors or transient episodes of hypoperfusion in the genesis of this condition (Fig. 39).^{60,63}



Figure 39. Isolated cecal necrosis. The intraoperative image showing a blackish necrotic plaque in the cecum, located in a congested colonic segment. This finding is characteristic of localized transmural segmental ischemia.

Diagnosis and management of Ischemic Colopathy

Once a diagnosis of IC has been established, the indication for surgery depends on the systemic impact, such as the presence of MOF and the depth of the wall ischemia assessed by sigmoidoscopy. Surgery is indicated in all patients with Favier stage 3 IC and in those with stage 2 IC with MOF. On the other hand, conservative management with monitoring and clinical support is recommended in patients with stage 2 IC without MOF and in stage 1 IC patients. Algorithms have been proposed for the management of patients with suspected IC (Fig. 40).

Medical Management

All patients with suspected IC should be closely monitored if there are no indications for immediate surgery. Management consists of gastrointestinal rest to reduce the colon's oxygen requirements, gastric decompression for associated ileus, fluid and electrolyte resuscitation, and prevention of venous thromboembolism. This treatment is sufficient in most transient cases of IC.¹⁰⁷

The empirical use of antibiotics plays a role as part of supportive care in initial treatment, especially in cases where the severity of the condition is not clearly defined.^{107,108} The rationale for antibiotic use is based on preventing bacterial translocation and possible septic complications. However, it is essential to note that, although their use is common, their specific efficacy in ischemic colitis has not been extensively studied or validated by controlled trials.^{68,109} The decision to prescribe antibiotics should be individualized, considering the severity of the clinical picture and the patient's general condition.^{68,107,108}

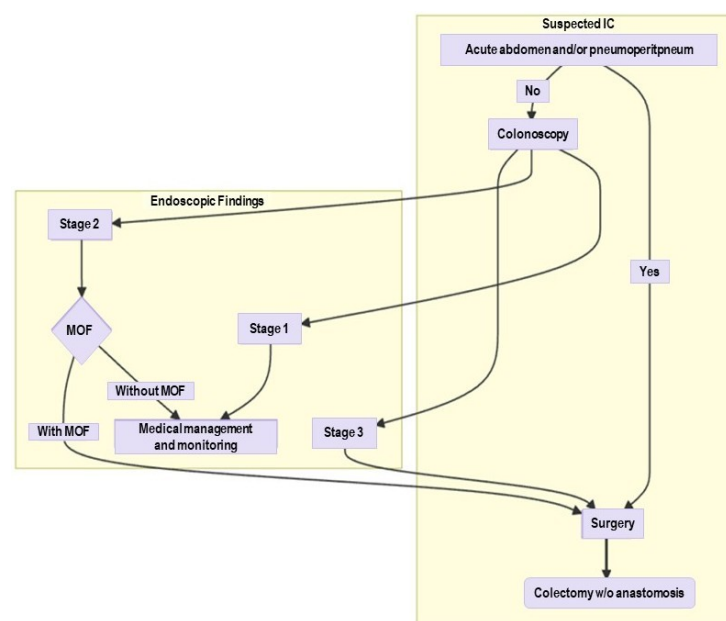


Figure 40. Therapeutic algorithm for ischemic colopathy (IC) The presence of an acute abdomen or pneumoperitoneum directly indicates a colectomy without anastomosis. Absent these findings, sigmoidoscopy facilitates classification according to the Favier classification: Stages 1 and 2 without multiple organ failure (MOF) are managed with medical treatment and monitoring; Stages 2 with MOF and 3 require colectomy without anastomosis.

Systemic corticosteroids are contraindicated because they can potentiate IC and increase the risk of perforation. To ensure optimal colonic perfusion, it is essential to avoid aggravating factors such as vasopressors or digitalis. Furthermore, adequate cardiac output should be restored through fluid resuscitation. In cases where the evolution is prolonged, parenteral nutrition may be necessary.

Indications for Surgery

Approximately 20% of cases require surgical intervention, which involves a colectomy of variable extent based on preoperative and intraoperative findings, without immediate restoration of intestinal continuity.

Evidence of colonic necrosis on CT scan (pneumatosis intestinalis, gas in the portal vein, or free peritoneal gas) is

the classic indication for immediate surgery without the need for endoscopic confirmation. For patients undergoing endoscopy, visualization of irreversible necrosis of the colonic muscle (Favier stage 3) should lead to emergency colectomy, even in the absence of organ failure. Similarly, patients with endoscopic evidence of less severe ischemia (stage 2) should undergo surgery if their clinical condition progresses unfavorably despite medical management. This recommendation is particularly applicable if MOF (acute renal or hepatic failure, shock, sepsis, lactic acidosis) develops and/or endoscopy shows progression to stage 3 ischemia. Finally, rare forms of IC with diarrhea, LGIB, or exudative colitis that persist for more than 14 days may necessitate consideration of colectomy.

Several risk factors associated with the need for surgical intervention following admission and preliminary evaluation were identified. These findings could facilitate the identification of cases in which early intervention could be advantageous, even when symptoms are not sufficiently evident to necessitate surgical treatment. In the study conducted by Paterno et al.,¹¹⁰ it was observed that patients who required deferred surgery during their evolution presented, in the univariate analysis, a series of pathological conditions, such as peripheral vascular disease, atrial fibrillation, hypotension, tachycardia, absence of rectal bleeding, presence of free intraperitoneal fluid on CT scan, admission to the intensive care unit, use of vasopressors, need for mechanical ventilation, and elevated lactate levels at the time of admission.

Emergency Surgery

According to the literature on the subject, the following clinical risk factors have been identified for emergency surgery: male sex, hypertension, abdominal guarding, and absence of LGIB.⁸⁶ Rheumatoid arthritis, chronic renal failure, hemodialysis, and right colonic or pancolonic involvement have also been associated with prolonged hospitalization and an increased rate of colectomy.

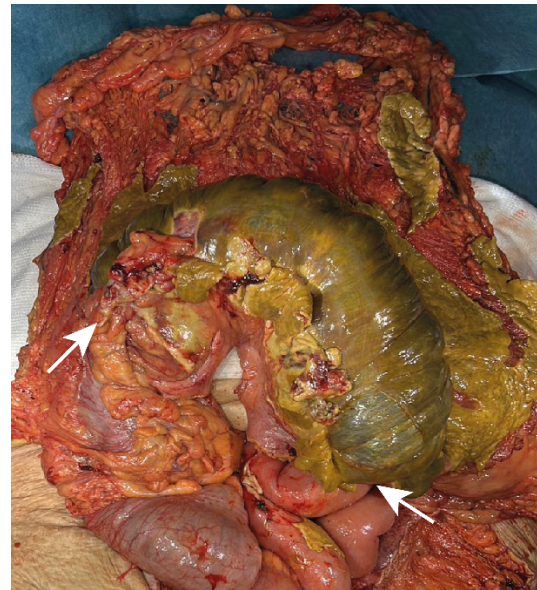


Figure 41. Segment of the colon exhibiting signs of ischemia and transmural necrosis. Surgical resection should include the entire affected segment, from the proximal to the distal limit (arrows), to obtain histological margins free of ischemia. This is crucial as compromised margins are associated with a higher probability of ischemia progression and the need for reoperation.

Due to the challenges associated with accurately determining the safe limits of resection and the severity of the disease, surgical interventions for left-sided ischemia should include both areas of marginal vascularization (splenic flexure and rectosigmoid junction). A Hartmann-type left colectomy, including the splenic flexure and the rectosigmoid junction, is recommended. The rectal stump is closed with staplers, and a left transverse colostomy is performed. Preservation of part or all of the rectum is justified. Abdominoperineal resection is not part of the surgical treatment of IC. Although ischemia of the rectal mucosa is common (50%), it generally evolves favorably on its own, either healing as fibrosis or, in the worst cases, developing a decomposition of the rectal stump. The diagnosis and treatment of this complication pose few problems, although it sometimes requires reoperation. Complications arising from the rectal stump are prevalent in a significant percentage of cases, contributing to high morbidity.

Although ischemia of the rectal mucosa is prevalent (50%), it generally exhibits a favorable spontaneous evolution, manifesting as fibrosis or, in the most severe cases, rectal stump decomposition. The diagnosis and treatment of this complication are relatively straightforward, though it may necessitate additional surgical intervention. Complications arising from the rectal stump are observed in a significant proportion of cases, contributing to elevated morbidity rates.¹¹¹

In instances of IC that is confined to the right colon, a right colectomy accompanied by an ileostomy and the formation of a mucosal fistula in the lower right quadrant is advised.

For cases of extensive or pancolonic conditions, a total colectomy with terminal ileostomy and closure of the rectal stump is proposed. In instances where ischemia of the right colon is accompanied by less pronounced ischemia on the left side, a condition frequently indicative of extensive ischemia resulting from superior mesenteric arterial hypoperfusion, total colectomy is

recommended. In the latter case, the clinical picture is typically characterized by small bowel ischemia, resulting in a distinct therapeutic management approach that involves extensive small bowel resection in addition to colectomy. Routine cholecystectomy has been demonstrated to prevent the development of acute acalculous cholecystitis related to resuscitation and to avoid reoperation in frail patients. However, the evidence available to support this approach is limited. In cases requiring emergency surgery, mortality rates are high, reaching 85% in some series.¹¹²⁻¹¹⁴

The *Ischemic Colitis Mortality Risk Score* was developed based on a study of 177 patients, taking into account specific clinical factors (Table 12). The sum of these factors generates a score ranging from 0 to 5, with mortality estimates of 10.5%, 28.9%, 37.1%, 50.0%, 76.7%, and 100.0% for each increasing level of stratification.¹¹²

Table 12. The *Ischemic Colitis Mortality Risk Score* includes several risk factors associated with an elevated postoperative mortality rate.

High risk factor
Age >75 years
Multiple organ failure
ASA ≥4 (American Society of Anesthesiologists)
Intraoperative blood loss >500 mL
Preoperative lactate >2.5
Acute kidney injury
Preoperative or intraoperative use of catecholamines
Low-output heart failure
Subtotal or total colectomy

Postoperative complications may include anastomotic dehiscence, rectal stump leakage, stoma-related complications, malabsorption syndromes, and short bowel syndrome.¹¹¹ Approximately 16% of patients experience surgical complications, and in many of these cases, ischemic changes are identified at the resection margins.¹¹⁵ Between 20 and 29% of patients require a second laparotomy, either due to clinical deterioration or intraoperative findings during the first procedure.¹¹⁵

Given the potential risks of anastomotic dehiscence in IC, primary anastomosis should be considered only in exceptional cases. A 21% of AL and up to 50% of ostomy or rectal stump complications have been reported.¹¹¹ These complications are associated with the presence of ischemia at the resection margin, as observed in pathological anatomy.

Restoration of intestinal continuity (RC) is a viable option for one-third of patients who survive the acute episode. This procedure should be considered after a thorough assessment of risks and benefits, including the evaluation of anesthetic risk. There is a paucity of data on RC after colectomy for IC.

Longo et al.¹⁷ reported the results of a series of 43 patients. RC was performed in 75% of patients with segmental IC and in only 7.3% of those with pancolonic ischemia. The majority of the latter patients had a fatal outcome.

Huguier et al.¹¹⁶ performed RC in 83% (5/6) of patients. In another study with 60 surviving patients, RC was performed in 24 patients (40%), and the median interval to restoration was 7.9 months.¹¹⁷ We recommend a multidisciplinary discussion between surgeons, anesthesiologists, and cardiologists after the sixth postoperative month to assess the risks of RC.

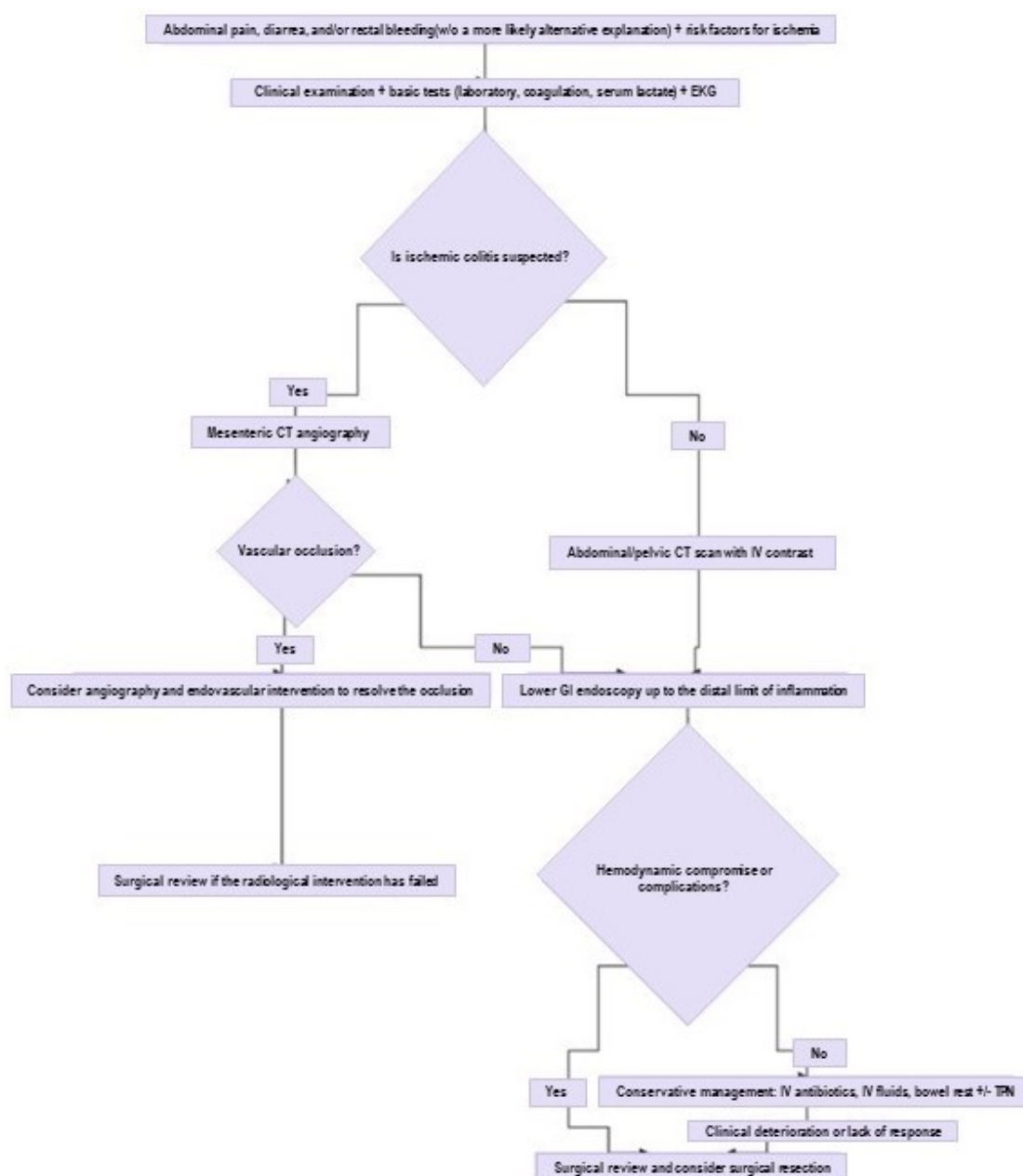


Figure 42. Diagnostic and therapeutic management of ischemic colitis.

Mortality, Morbidity, and Risk of Relapse

IC is associated with significant mortality rates, which vary depending on the severity of the disease, its location, and the therapeutic approach. The overall mortality rates reported range from 25.3 to 54%.^{111,118-120} A review of the literature reveals an overall mortality rate of 22 (6-53) %, ranging from 0 in patients treated conservatively to 75% in those with pancolitis requiring emergency surgery. In severe cases requiring surgical intervention, mortality rates can reach between 65 and 75%.⁷¹⁻¹¹⁹ Conversely, milder cases treated non-operatively have lower mortality rates, ranging from approximately 42 to 45%.¹²¹ Postoperative death related to IC usually occurs within the first 30 days.⁸⁶

According to the classification established by Favier, the estimated mortality rates are as follows: 65.8% for grade 3 with SMOF, 16.6% for grade 3

without MOF, 53.3% for grade 2 with MOF, and 0% for grade 2 without MOF and grade 1.⁸⁶

Patients with left colon involvement generally have a better prognosis than those with right colon and pancolitis involvement, with a lower risk of in-hospital mortality.¹¹¹ IC that occurs after aortic surgery has a poor prognosis, with postoperative mortality ranging from 50 to 60%.^{17,116} Several factors have been identified as being associated with increased mortality. These include a recent history of cardiac or vascular surgery involving aortic clamping (OR=3.99, $p<0.05$), ulcerative or necrotic colonic disease (OR=3.49, $p<0.05$), septic or hypovolemic shock, the presence of mesenteric atheromatosis, and perforated IC.⁸⁶

Late deaths following colectomy are primarily due to cardiovascular disease, underscoring the importance of secondary cardiovascular risk prevention. The presence of mesenteric atheromatosis, an indicator of diffuse vascular

disease, is associated with mortality following IC surgery. It was present in 97% of patients who died.¹¹⁶

The recurrence of CI is uncommon, with a rate of 5.6-13.0%.^{110,122} Active smoking and the presence of an abdominal aortic aneurysm (AAA) are risk factors for recurrence.¹²²

Longstreth et al.⁶⁹ reported a series of 424 episodes of IC in 401 patients, with a mean follow-up period of 2.6 ± 1.9 years. The majority of patients received medical treatment. No cases of chronic colitis were observed, and the rate of ischemic stenosis was 0.3%. The recurrence rate of multiple episodes was 6.7% (recurrence rates of 3%, 5%, 6%, and 10% at 1, 2–3, 4, and 5–6 years, respectively). With a mean follow-up period of 4.5 years, Huguier et al.¹¹⁶ reported a recurrence rate of 8.6% (5/58). With an average follow-up period of 8 years, Leardi et al.¹²⁰ reported no recurrences.

Prognostic Factors

The prognosis for IC is generally favorable, as it is typically a transient condition that resolves without sequelae.^{123,124} However, emergency procedures and total colectomies are significantly associated with higher mortality rates.^{125,126} Several, clinical, laboratory, radiological, and endoscopic factors have been identified as contributing to a more unfavorable prognosis in cases of IC (Table 13).

Table 13. Epidemiological, clinical, laboratory, radiological, and endoscopic factors associated with poorer outcomes in ischemic colitis.

Category	Associated factors
Epidemiology/ history	Male sex; collagen vascular disease; chronic kidney disease, dialysis; chronic constipation; cardiovascular risk factors or prior history; others (cancer, COPD, hyperthyroidism)
Clinical	Absence of lower GI bleeding; peritoneal irritation; multi-organ failure, shock; right colon involvement; pancolonic involvement
Laboratory	Anemia, hyponatremia; hypoproteinemia; leukocytosis; hyperlactatemia >2.5 mM/L
Radiology	Pneumatosis intestinalis, portal venous gas, pneumoperitoneum (CT); loss of arterial flow in the muscularis propria (Doppler US)
Endoscopy	Depth of ischemia (Favier classification); necrosis, perforation; circumferential ulceration

The in-hospital mortality rate (45 vs. 6%; $p=0.003$) and the incidence of non-surgical complications were higher in the cohort with severe hematochezia than in the cohort without hematochezia.⁹¹ En un reporte sobre mortalidad postoperatoria, en el análisis multivariable, la mortalidad hospitalaria fue mayor en pacientes con isquemia del colon derecho (OR 3,8) o isquemia pancolónica (OR 11) en comparación con la isquemia del colon izquierdo, con un menor pH preoperatorio (OR 2,5 por cada disminución de 0,1) y con el antecedente de cirugía cardíaca o aórtica (OR 2,4). In a multivariate analysis of a report on postoperative mortality, the hospital mortality rate was found to be significantly higher in patients with right colon ischemia (OR 3.8) or pancolonic ischemia (OR 11) compared to those with left colon ischemia. A correlation has been demonstrated between lower preoperative pH and higher mortality (OR 2.5 for each 0.1 unit decrease), as well as with a history of cardiac or aortic surgery (OR 2.4).¹¹¹

In addition to the location of the ischemic territory, various patient- and surgery-related characteristics have been associated with postoperative in-hospital mortality in patients with IC. These include elevated preoperative lactate levels, American Society of Anesthesiologists (ASA) classification, emergency surgery, acute renal failure, significant intraoperative bleeding, blood product transfusions (allogeneic red blood cells and fresh frozen plasma), mesenteric arterial occlusion, and low-output heart failure or the need for catecholamine administration.¹²⁷

Long-Term Prognosis: Late Complications

IC can lead to various late complications, but the incidence is low.^{107,128} One of the potential late complications is the development of symptomatic strictures, which can form as a result of the healing process following severe ischemic injury to the wall involving the colonic muscle.⁶⁸ Post-inflammatory strictures may develop after conservative management and occur in up to 10% of cases.¹¹⁵ Since inflammation is usually segmental as a result of its vascular etiology, strictures tend to be relatively extensive. Treatment with endoscopic dilation, stricturoplasty, or segmental resection is reserved for symptomatic strictures (obstruction, liquid stools, bleeding).

Another potential late complication, although not clearly documented, is the formation of pseudotumors. The endoscopic appearance in the colon and rectum can mimic adenocarcinoma, and diagnosis is made by histopathology of the surgical specimen.

Chronic ischemia can lead to persistent symptoms or protein-losing colopathy.⁶⁸ In most cases, the diagnosis is clinical. Alpha-1-antitrypsin can also be measured in stool.^{68,129} In the study by Medina et al.,¹³⁰ none of the patients developed chronic IC during follow-up, suggesting that late complications are uncommon.¹³⁰

Postoperative Ischemic Colopathy

POIC is a specific clinical presentation; for this reason, we will proceed to describe some of its distinctive characteristics.

This serious complication is associated with high morbidity and mortality rates and manifests after various surgical procedures, mainly vascular, involving the aorta and its abdominal branches, affecting those that supply the colon.⁷¹

Early identification in the postoperative period through early colonoscopy can contribute to informed decisions regarding appropriate management, which in turn can increase the chances of survival.¹³¹

IC Following Aortic Surgery

IC is a complication of aortic surgery, with an incidence ranging from 0 to 3% in elective abdominal aortic aneurysm (AAA) surgery, and from 10 to 36% in ruptured abdominal aortic aneurysms (RAAA).¹³² The occurrence of IC after aortic surgery has been associated with an increase in the postoperative mortality rate, reaching up to 60% in certain studies (Fig. 43).¹³²

While there are demographic associations (e.g., age over 68 years, female sex, peripheral vascular disease, renal failure, COPD), the most significant factors in the development of IC after aortic repair appear to be the indication for emergency surgery and hemodynamic patterns during aortic surgery.¹³³ Surgery for RAAA has an OR of 5.9 for developing colonic ischemia compared to elective surgery for AAA.¹³³ Furthermore, lower perioperative cardiac output and the use of α -adrenergic vasoconstrictor agents are significantly associated with the development of this complication.¹³⁴ The likelihood of developing IC increases significantly in the presence of two (48%) or more risk factors, reaching 80% in cases with six (6) or more concurrent factors. The following risk factors have been identified: systolic blood pressure <90 mmHg, hypotension for more than 30 minutes, temperature <35 °C, pH <7.3, >6 units of blood transfusion, and resuscitation with intravenous fluids >5L.¹³⁵



Figure 43. Colonic ischemia with transmural necrosis of the sigmoid colon is observed in a patient in the immediate postoperative period following complex abdominal aortic replacement. The macroscopic findings, which are indicative of ischemic transmural necrosis, include a purplish-black discoloration, loss of serosal brightness, and diffuse vascular compromise.

A study of 157 patients who presented with RAAA revealed that 18% developed postoperative IC.¹³² These patients had more coronary artery disease (38 vs. 17%, $p=0.026$). Nineteen percent of patients underwent colonoscopy due to suspected IC. Of these, 12 exhibited signs of colonic ischemia, necessitating colonic resection.

Twelve patients were excluded from undergoing endoscopy due to the presence of clear clinical and radiological signs of severe IC, indicating surgical treatment. In the study, 92% of patients with IC developed this condition within the first 3 postoperative days. Laboratory findings in the first 7 days revealed differences between patients with and without IC with regard to serum lactate, minimum pH, serum bicarbonate, and platelet count.¹³² Hemoglobin and PaO₂ levels showed no statistically significant differences between the two groups during the study. Conversely, leukocytes exhibited

elevated levels in the IC group starting from the 5th postoperative day, while serum creatinine levels were elevated from the 2nd postoperative day onward. During recovery in the ICU, patients with IC required comparatively higher doses of norepinephrine to maintain a mean arterial pressure above 65 mmHg as opposed to those without IC.¹³²

Reimplantation of the IMA during AAA repair has not been shown to be associated with a decrease in the incidence of IC.¹³⁶ Lee et al.¹³⁶ found that patients with SMA reconstruction had a higher rate of surgical re-intervention (20.0 vs. 7.2%; $p=0.006$), a higher rate of surgical wound complications (17.1 vs. 3.0%; $p=0.001$), and a higher incidence of IC (8.6 vs. 2.4%; $p=0.027$). However, if the SMA is not patent and the mesenteric arterial network is completely dependent on the IMA, the patent IMA should be reimplanted. Although it has been suggested that occlusion of the hypogastric arteries could promote colonic ischemia after aortic replacement, the evidence suggests that pelvic revascularization does not reduce the incidence of IC; in fact, it may increase complications. A statistically significant association was not observed between pelvic revascularization and the onset of postoperative IC.¹³⁷

An ICG test can be performed during aortic repair to identify colonic ischemia, which is associated with a perfusion delay of more than 3 minutes. The mean time to maximum colonic perfusion after ICG injection was 58 seconds in patients without IC.¹³⁸ This technique considers the onset of ischemia at the time of exclusion of the IMA; however, it does not prevent its development as a result of subsequent hypoperfusion due to shock. Consequently, integrating this approach with other monitoring methodologies would be a judicious strategy.

A potential approach for intraoperative monitoring of colonic perfusion in aortic surgery involves the utilization of real-time sigmoid mucosal oximetry. A research study showed that a progressive reduction in the flow of the MC artery to 20% of its baseline value produced a linear decrease in colonic O₂ saturation ($r=0.91$; $p<0.01$). At this perfusion level, colonic O₂ saturation was $54.0\pm4.1\%$. A loss of signal on the transanal pulse oximeter was evident when the baseline MC flow was below 20%.¹³⁹ These findings suggest that transanal pulse oximetry could be a simple tool for continuous monitoring of distal colonic blood perfusion during aortic reconstruction procedures.

Laser Doppler flowmetry is a sensitive method for assessing critical colonic blood flow during AAA repair, identifying a critical microvascular perfusion threshold at approximately 50 perfusion units (PU).¹⁴⁰

The updated recommendations for the management of patients with AAR and suspected colonic ischemia indicate the performance of a flexible sigmoidoscopy (Class IIa recommendation with Level of Evidence B).¹⁴¹ Flexible endoscopy enables the timely confirmation of ischemic involvement of the colon, thereby facilitating early therapeutic decisions.

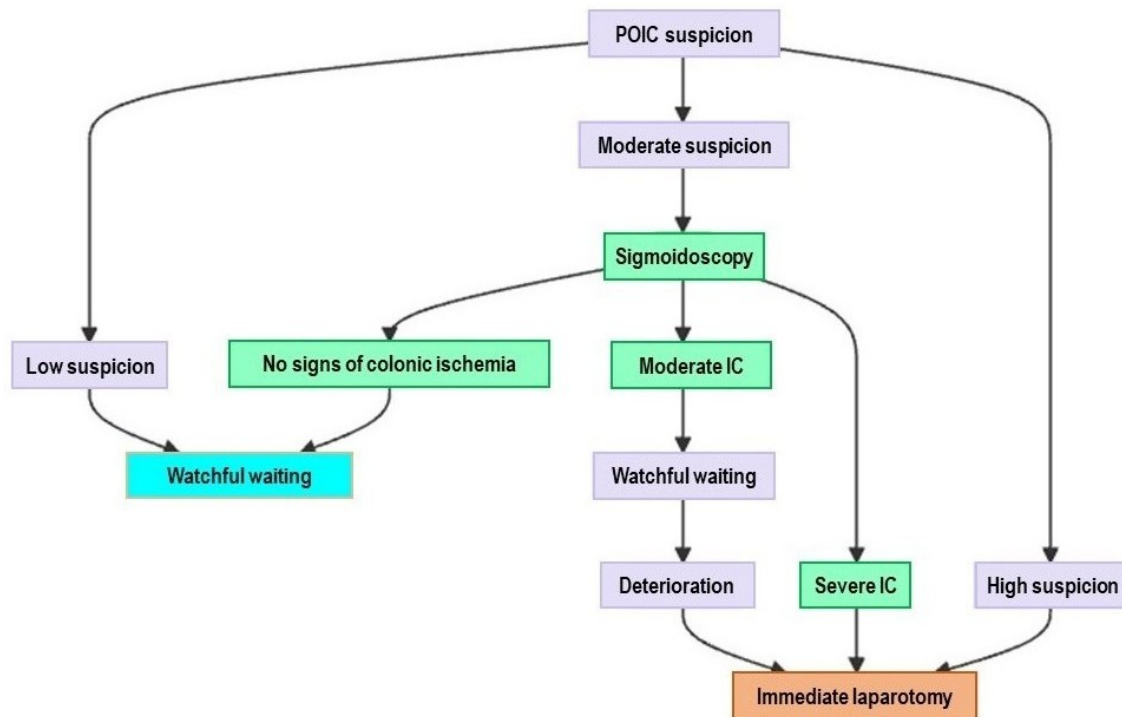


Figure 44. Diagnostic algorithm for suspected colonic ischemia in the postoperative period following repair of a ruptured abdominal aortic aneurysm. Clinical suspicion is based on the presence of signs such as early bowel emptying, diarrhea, bloody stools, sepsis, abdominal pain, sustained hypotension lasting more than one hour, prolonged surgery, extensive blood loss, use of high doses of vasopressors, positive fluid balance, postoperative organ dysfunction, abdominal compartment syndrome, elevated lactate or potassium levels, and low hemoglobin. Depending on the degree of suspicion (low, moderate, or high), there are established diagnostic and therapeutic approaches, including clinical observation, sigmoidoscopy, or immediate laparotomy, according to the severity of the condition. The watchful waiting approach involves ongoing clinical reassessment, monitoring of intra-abdominal pressure, repeat sigmoidoscopy, and additional imaging studies.¹⁴¹ POIC: Postoperative ischemic colopathy; IC: Ischemic colopathy

Postoperative Ischemic Colopathy in Colon Surgery

IC is a potential complication that arises subsequently to colonic or rectal resections, resulting in substantial postoperative morbidity and mortality.¹⁴² This occurrence is primarily attributed to insufficient blood flow to the colon. Its incidence has been documented in up to 2.1% of left colectomies.^{142,143} The risk appears to be elevated in left colon resections compared to right colon resections, particularly after laparoscopic surgery for left colon cancer.^{40,144} After ligation of the IMA, an area of decreased perfusion occurs due to the left colon being perfused solely by the vascular arcade from the MCA, resulting in an area of artificial marginal perfusion. This can lead to ischemia of the descended colonic segment.

There are several perioperative risk factors to consider, including prolonged surgery time, significant blood loss, and the need for blood transfusions during or after surgery.¹¹¹ The risk of acute IC in the proximal anastomotic segment after anterior resection is higher in patients with diabetes mellitus, peripheral vascular disease, and morbid obesity, as well as in those with moderate to severe atherosclerosis, which compromises collateral circulation.¹⁴⁵

Common symptoms of postoperative IC include abdominal pain, diarrhea, and rectal bleeding. In cases of early development of ischemia, anastomotic fistulas may also occur. These symptoms may present immediately or be delayed in the postoperative period. The diagnosis is typically confirmed through an endoscopy, which reveals mucosal inflammation and ischemic changes. CT scans may reveal intestinal edema and other indications of

impaired blood flow.¹⁴⁶ CI after surgery has a high mortality rate, particularly if it is not diagnosed and treated promptly.¹¹¹ In severe cases, reoperation may be necessary.

Prevention involves maintaining adequate perfusion during surgery and close postoperative monitoring with careful management of blood pressure and fluid balance. For patients A study of 1,201 sigmoid and rectal cancer surgeries performed by a single surgeon between 2006 and 2010 reported 10 cases of postoperative colonic ischemia (0.83%).⁴⁰ High ligation of the IMA was routinely performed in all procedures. Intraoperative bleeding was documented in 5 patients (50%), with an average volume of 435 ml. IC occurred, on average, 5 days after surgery (range 2-10 days), and postoperative fever was observed in 8 patients (80%). The mortality rate was 10%. The authors consider postoperative colonic ischemia to be one of the most significant complications of colorectal resection.⁴⁰

High ligation of the IMA in rectal cancer surgery is a widely accepted technique based on oncological principles. The procedure involves ligating and sectioning the IMA at its origin, before the emergence of the LCA. The ascending branch of the left colic artery anastomoses (connects) with the marginal artery of Drummond, while the descending branch also joins the marginal artery in proximity to the sigmoid vascular arcades. The LCA plays a fundamental role in the blood supply to the descending colon and splenic flexure. After high ligation, the blood supply to the proximal segment of the anastomosis (variable length of the descending colon) depends exclusively on the MCA through the marginal artery. From the perspective of ensuring

optimal blood supply, it is recommended to perform the section of the colon proximal to the splenic flexure. However, this option is not feasible due to insufficient colon length for creating a colorectal anastomosis. Consequently, after high ligation of the IMA, the blood supply to the splenic flexure and the proximal segment of the descending colon involved in the anastomosis is compromised.

High ligation of the IMA creates an iatrogenic area of relative ischemia or hypoperfusion. To ensure safe performance of the anastomosis, it is recommended that an ICG perfusion test of the descending colon be performed. Consequently, high ligation of the IMA may play a role in the development of postoperative ischemic colitis. Ligation of the IMA compromises blood flow to the anastomosis, which may increase the leakage rate. It is unclear whether this confers a survival advantage.

To ensure safe performance of the anastomosis, it is recommended that an ICG perfusion test of the descending colon be performed.

Therefore, high ligation of the IMA could contribute to the development of postoperative ischemic colitis.⁴⁰ Ligation of the IMA compromises blood flow to the anastomosis, potentially augmenting the leakage rate. However, the question of whether this results in a survival advantage remains unanswered.

In a study of 590 patients with colorectal cancer, Wang et al.¹⁴⁷ demonstrated that high ligation, compared with low ligation, results in a significantly higher incidence of anastomotic fistula (21/283 vs. 11/307; $p=0.040$) and poorer preservation of urinary function. High ligation was identified as a significant independent factor associated with anastomotic fistula (OR 2.232, 95% CI 1.047-4.758; $p=0.038$).¹⁴⁷ The level of IMA ligation did not demonstrate a substantial impact on either overall survival (OS) or recurrence-free survival. There were no significant differences between high and low ligation in terms of the number of lymph nodes removed or metastatic lymph nodes. The level of IMA ligation does not have a significant impact on long-term outcomes for patients with sigmoid or rectal colon cancer following curative laparoscopic surgery.¹⁴⁸ However, IMA dissection can result in injury to the autonomic nerve plexus, potentially leading to impaired urinary and sexual function. The proximity of the IMA to the nerve fibers of the hypogastric plexus and pelvic nerves means that its surgical manipulation can affect autonomic innervation, increasing the risk of postoperative neurogenic dysfunction.¹⁴⁸

A meta-analysis was conducted to evaluate the impact of high ligation of the mesenteric artery in colorectal cancer surgery. The analysis included 5,917 patients, 3,652 of whom underwent low ligation, while 2,265 underwent high ligation. The AL rate was 9.8% in high ligation vs. 7.0% in low ligation, demonstrating a higher risk of anastomotic dehiscence in high ligation (OR 1.33; $p=0.004$). Furthermore, postoperative morbidity was found to be significantly higher in patients with high ligation (OR 1.39; $p=0.05$).¹⁴⁹ Conversely, no substantial differences were observed between the two groups with respect to postoperative mortality, lymph nodes resected, overall recurrence rate, or 5-year survival.

In cases of benign disease, there is insufficient evidence to recommend the preservation of the IMA as a mandatory technique. The surgeon would determine the approach based on technical considerations to ensure adequate mobility of the descending colon for anastomosis. Cirocchi et al.¹⁵⁰ evaluated, through a meta-analysis, the preservation and ligation of the IMA in colectomy for diverticular disease of the left colon, including 2,190 patients. The

anastomotic leakage rate was higher in the group with IMA ligation (6%) compared to the group with IMA preservation (2.4%), although this difference was not statistically significant (RR: 0.59).¹⁵⁰ Conversely, several studies have demonstrated that high ligation of the IMA enables complete mobilization of the colon, thereby allowing the creation of a tension-free anastomosis. This could potentially contribute to a reduction in the incidence of AL.¹⁵¹ According to the Japanese Society for Colon and Rectal Cancer, the rate of lymph nodes metastasis near the origin of the IMA is 3.6% in pT3/pT4 sigmoid cancer and 5.1% in rectal cancer.¹⁵² Consequently, in such cases, LND at the origin of the IMA is necessary. The greatest potential of high drainage (D3) of the IMA would be for patients with T4, as there is no clear oncological benefit for non-disseminated tumors of lesser depth. In fact, high ligation of the IMA has limited benefit in the context of colorectal cancer, with an increasing incidence of lymph node metastasis as tumor invasion progresses. An analysis of high IMA ligation in sigmoid and rectal colon cancer revealed a 0.8% benefit in terms of overall oncological control. When the data were analyzed according to tumor location, the benefit rate was 1.8% for sigmoid colon cancer and only 0.4% for rectal cancer. Concerning the presence of lymph node metastasis in the origin of the IMA according to T stage, the observed rates were as follows: 0% in pT1, 1.0% in pT2, 2.6% in pT3, and 4.3% in pT4.

Intraoperative ultrasound was used to evaluate the impact of preserving LCA on mesenteric perfusion during rectal cancer surgery, observing that it can improve perfusion in the anastomotic area. The mean flow velocities in the IMA were 47.9 cm/s before clamping and 34.9 cm/s after atraumatic clamping, showing a significant reduction in blood flow velocity ($p < 0.05$). In addition, 13 patients (32.5%) exhibited a *tardus-parvus* pulse pattern subsequent to atraumatic clamping of the IMA, suggesting an alteration in colonic perfusion.¹⁵³ Advanced age and LCA diameter were identified as predictive factors for mesenteric hypoperfusion following IMA ligation.¹⁵³

The technique of lymph node dissection and vessel ligation may vary depending on anatomical variations present in each case, to achieve D3 dissection while preserving optimal vascularization (Fig. 45).^{154,155}

It has been proposed that low ligation with lymphadenectomy at the origin of the IMA should be considered as an alternative to improve vascularization of the descending colon without modifying LND. The IMA can be ligated at a point just below the origin of the LCA, a "low ligation," combined with LND around the origin of the IMA (low ligation with LND).¹⁵² Low ligation with LND is anatomically less invasive and is not oncologically inferior to "high ligation." In one study, 189 patients with sigmoid or rectal cancer who underwent curative surgery were included. Forty-two patients underwent ligation at the origin of the IMA (high ligation), and 147 patients underwent ligation just below the origin of the LCA combined with LND around the origin of the IMA (low ligation with LND). There were no significant differences in complication rates or OS and disease-free survival rates between the two groups.

The risk of ischemia in these cases is also attributed to variable blood supply in the splenic flexure, specifically at Griffiths' point, which is considered a vascular transition zone or "watershed." At this point, the anastomosis between the marginal artery derived from the MCA and the marginal artery originating in the IMA is inconsistent in terms of its presence and caliber. In 5-7% of cases, this anastomosis may be inadequate.¹⁵⁶

Active searching for the Arc of Riolan (Moskowitz) during splenic flexure takedown has been shown to reduce the incidence of postoperative colonic ischemia.⁴ This arterial vessel can be identified preoperatively with a targeted CT scan. Mesenteric angiographic mapping has demonstrated the presence of this vessel in 11.2% of cases.¹⁵⁷

Following the clamping of the IMA, retrograde perfusion is initiated from the rectum to the sigmoid colon. This retrograde perfusion can reach a distance of 40 (range 17-66) cm toward the proximal area from the peritoneal reflection.¹⁵⁸

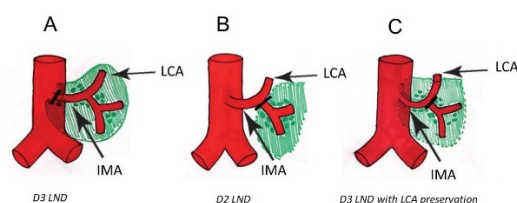


Figure 45. Technical variants for ligation of the inferior mesenteric artery (IMA) and D3 lymph node dissection (LND) in sigmoid colon tumors. **A.** High ligation of the IMA proximal to the left colic artery (LCA), with LND located at its origin (D3). **B.** Low ligation of the IMA distal to the LCA (D2). **C.** Low ligation of the IMA combined with LND located around the origin of the IMA. This allows for a similar number of lymph nodes to be obtained as with D3 with high ligation.

Other factors have been described as potentially interfering with the healing process of an anastomosis. These include emergency surgery and transient hypovolemia.¹⁵⁹ In an experimental model in rats, acute intraoperative loss of 10% of circulating blood volume significantly compromised collagen concentration in ileocolic ($p < 0.02$) and colocolonic ($p < 0.05$) anastomoses on the third postoperative day. However, the loss of blood did not have a significant effect on the early strength of the anastomosis.¹⁵⁹

In another experimental model in rabbits, the partial pressure of tissue oxygen in the colon was significantly lower compared to that in the small intestine (37 ± 18 mmHg vs. 42 ± 18 mmHg; $p < 0.001$). This decrease was further observed after a 10% blood loss ($p < 0.001$).¹⁵⁹

Colonoscopy-Associated Ischemia

Colonoscopies are considered to be relatively safe procedures. The perforation rate is approximately 4 per 10,000 procedures, the major bleeding rate is 8 per 10,000, and the overall mortality rate is estimated to be between 0.007 and 0.07%.⁷³ However, colonoscopy is associated with an increased risk of developing colonic ischemia.

Despite the limited number of reports on this subject, Sadalla et al.⁷³ compiled a comprehensive set of 25 reports on colonoscopy-related colonic ischemia. The causes of colonic ischemia following a colonoscopy were varied and included different pathophysiological mechanisms, such as altered splanchnic circulation, effects of bowel preparation, use of sedatives, insufflation and barotrauma, and mechanical trauma caused by the introduction of the endoscope. In cases where the colon suffers from chronic ischemia, as occurs in peripheral vascular disease, antiphospholipid syndrome, systemic lupus erythematosus, and diabetes, gas insufflation and mechanical compression during colonoscopy can easily cause a critical drop in previously impaired blood flow, leading to ischemic injury.⁷³ Furthermore, laxatives combined with inadequate fluid intake during preparation can lead to dehydration and

potentially precipitate ischemic injury.⁷³ Therefore, bowel preparation should be optimized in at-risk patients.

Phosphate-based preparations should be avoided due to their adverse effects on renal function.¹⁶³ Conversely, high-volume PEG-based regimens have a higher safety profile due to their osmotically balanced formulation and reduced sodium load.¹⁶³ To prevent dehydration induced by osmotic diarrhea, patients should be instructed to drink sufficient water before and after the colonoscopy. Due to reports of bisacodyl-associated colonic ischemia, this medication should be avoided in patients with multiple risk factors.¹⁶⁴

Midazolam, when administered alone or in combination with opioids, has been observed to induce cardiovascular effects such as vasodilation, myocardial contractility depression, and hypotension.⁷³ It is imperative to minimize adverse events related to sedation, including hypotension and hypoxemia, as they could act as precipitating factors in individuals at risk of colonic ischemia. Hypotension is considered a key factor in the development of colonic hypoperfusion. Tang et al.¹⁶⁵ found that in patients undergoing colonoscopies with sedation involving fentanyl and midazolam, low blood pressure before the procedure was a primary risk factor for the development of hypotension during the procedure.

Barotrauma should be considered the main cause of reduced parietal blood flow during colonoscopy.⁷³ Gas insufflation fills the colon up to the cecum, which is the highest point when the patient is in the left lateral decubitus position.

When the pressure in the vessels reaches 30–60 mmHg, it causes a reduction in parietal blood flow. This, in turn, can lead to mucosal damage within approximately 20 minutes. The air pumps incorporated into light sources generate a maximum pressure of 375 mmHg. However, when measured at the tip of the endoscope, this pressure is reduced by 30-40%. In one study, Kozarek et al.¹⁶⁶ demonstrated that, in cadaveric human colon, the air pressures that resulted in serous tears, pneumatosis, and transmural rupture ranged from 52 to 226 mmHg. Rupture of the cecum was observed at an air pressure of 81 mmHg, while sigmoid colon tear required a pressure of 169 mmHg. In the same study, the authors measured intraluminal pressure during routine colonoscopies. Intraluminal pressure measurements ranged from 9 to 57 mmHg when the tip of the endoscope was free, while it reached a maximum of 138 mmHg when the tip was impacted against the intestinal wall. It is important to note that excessive insufflation with air has the potential to cause colon distension and compromise parietal perfusion. In a study on animals, Brandt et al.⁹⁰ demonstrated that CO₂ insufflation minimized the reduction in blood flow and intraluminal pressure compared with air insufflation during colonoscopy. Furthermore, studies have shown that CO₂ insufflation is more effective in reducing pain during and after the procedure when compared to air.¹⁶⁷ With the use of CO₂ insufflation, there is less residual gas present one hour after the colonoscopy. It has been suggested that CO₂ exerts a vasodilatory effect on the colonic microvasculature.¹⁶⁷ The observation of dry, pale mucosa during a colonoscopy could serve as an immediate indicator of ischemia. In such cases, discontinuation of the procedure should always be considered.

In addition, the manipulation of the endoscope can potentially traumatize the vascular pedicles and cause injuries to the mesocolon.⁷³ Tension in the mesocolon can reduce blood flow, impair microcirculation, and activate the

inflammatory cascade, eventually leading to vascular thrombosis. It is imperative to exercise caution when performing straightening maneuvers, manually compressing the abdominal wall, and twisting the endoscope, as these actions can potentially lead to mesocolon injuries.

The symptoms, imaging findings, laboratory results, and treatment of acute colonic ischemia after colonoscopy are consistent with the previously described. These symptoms include the sudden onset of abdominal pain associated with nausea, vomiting, abdominal distension, and bright bloody diarrhea within 24 hours.

Zizzo et al.¹⁶⁸ found that abdominal pain and bloody diarrhea were present in 95% of patients who developed colonic ischemia after colonoscopy. It is estimated that a proportion of cases may present with minimal or subclinical symptoms and therefore remain undetected.

While colonoscopy is the gold standard for diagnosing colonic ischemia optically, the decision to repeat the procedure should be made based on the clinical and radiological presentation to avoid worsening existing conditions. The approach to IC and colonoscopy should aim to prevent its development by considering the risk factors for IC (Table 14).

Ischemia in Intestinal Obstruction

Changes in intraluminal pressure within the colon can influence blood flow, both in terms of total perfusion volume, and distribution in the colonic wall. Repeated increases in luminal pressure can induce prolonged decreases in blood flow, which persist even after pressure returns to normal.⁷⁶ This finding suggests a potential link between IC and obstructive cancer.

Intestinal obstruction can be considered both a cause and a consequence of ischemia. Prolonged obstruction can lead to intestinal distension, which in turn can compromise blood flow and potentially trigger an ischemic episode.¹⁶⁹ Consequently, ischemia can then induce edema and inflammation of the intestinal wall, contributing to the development of obstruction.

Perforation that occurs proximal to an obstruction due to dilation is known as *diastatic perforation*.¹⁵⁶ The most common site of perforation is the cecum, where the diameter of the colon is largest, and according to Laplace's law, its parietal tension is greater with distension.¹⁷⁰

The application of Laplace's law to cylindrical structures (Fig. 46), such as the distended colon, facilitates the estimation of the parietal tension (T) generated in the intestinal wall based on various factors. These factors include the radius of the intestinal lumen (r), the thickness of the colonic wall (h), and the intraluminal pressure (P).

$$T = \frac{P \cdot r}{h}$$

Figure 46. Laplace's law. T: parietal tension, measured in Newtons per meter (N/m) or dynes per centimeter (dyn/cm). P: intraluminal pressure, expressed in millimeters of mercury (mmHg) or pascal (Pa). R: radius of the cylinder, measured in cm or m. h: thickness of the wall, measured in cm or m.

According to the pathophysiological interpretation of Laplace's law, the greater the luminal distension (greater radius [r]) or the greater the intraluminal pressure (P), the tension on the colonic wall increases proportionally. In case the wall thickness (h) remains constant or decreases, as occurs in thin areas such as the cecum, parietal tension can reach critical levels and undergo rupture or ischemia. This principle accounts for the increased susceptibility of the cecum (greater radius and lesser thickness) to perforation during colonic distension (Fig. 47).

Table 14. Proposed pathogenic factors for colonoscopy-related mesenteric ischemia and suggested preventive strategies.

Pathogenic factor	Mechanism of action	Type of ischemia	Prevention
Splanchnic circulation impairment	Chronic mesenteric ischemia (atherosclerosis, smoking); parietal vessel inflammation (connective-tissue disease, systemic lupus erythematosus, antiphospholipid syndrome)	Vascular thrombosis; mild microcirculatory injury	Careful history; recognize specific/nonspecific symptoms; antiplatelet therapy per guidelines; consider pre-colonoscopy tests (serum electrolytes, color-Doppler US).
Bowel preparation	Serum electrolyte imbalance, dehydration; additional risk with stimulant laxatives (e.g., bisacodyl)	Multifactorial	Clear patient information; use high-volume isotonic solutions; split-dose regimens; avoid bisacodyl-containing preparations.
Sedation (midazolam, opioids, propofol)	Vasodilation, depressed myocardial contractility, hypotension	Multifactorial	Minimal-sedation protocols in high-risk patients (verbal response, patent airway, spontaneous ventilation, normal cardiovascular function); consider prophylactic fluids.
Air insufflation / barotrauma	Increased intraluminal pressure with consequent vascular resistance	Non-occlusive mesenteric ischemia	Prefer CO ₂ insufflation; consider water-exchange colonoscopy technique.
Scope manipulation	Mechanical stress on mesocolon, blood-flow reduction, microcirculatory damage and inflammatory cascade activation	Vascular thrombosis	Interrupt procedure if severe pain or endoscopic signs of ischemia; consider pediatric/ultra-slim scopes; reschedule or reconsider indication in complex cases.

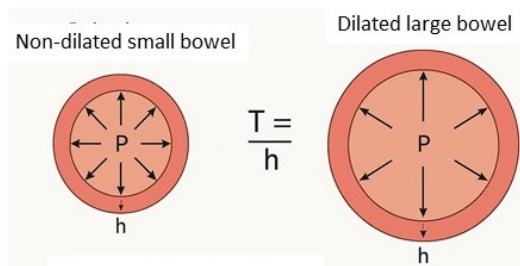


Figure 47. This diagram details the application of Laplace's law to colonic distension. Parietal tension increases directly in proportion to intraluminal pressure (P) and the radius of the intestinal lumen, and decreases inversely with wall thickness (h). In areas with thin walls, such as the cecum, critical tension can cause ischemia or rupture.

Increased intraluminal pressure in the colon can significantly reduce blood flow, promoting the development of ischemic damage to the intestinal wall.¹⁷¹ In equine models, intraluminal distension of the jejunum at a pressure of 25 cm H₂O for 120 minutes, followed by decompression, produced severe morphological alterations in the seromuscular layer, similar to those observed in ischemia-reperfusion injuries. The small intestine was found to be more susceptible to damage to the seromuscular layer than the ascending colon under analogous conditions of ischemia-reperfusion. Experimental studies in porcine models have shown that an increase in intra-abdominal pressure (IAP) significantly reduces blood flow in the mesenteric artery and intestinal mucosa, leading to severe mucosal ischemia.¹⁷¹ With an IAP of 20 mmHg, flow in the mesenteric artery decreased to 73% of baseline, while flow in the intestinal mucosa decreased to 61%. These effects were exacerbated when pressure rose to 40 mmHg, with a significant drop in intramucosal pH, a marker of severe ischemia.¹⁷¹ Ruf et al.¹⁷² demonstrated that a progressive increase in pressure within the intestinal lumen, in both segments of the small intestine and the colon, is associated with a proportional decrease in blood flow. At an intraluminal pressure of 60 mmHg, intestinal blood flow was reduced to 25% of normal levels. This decrease was particularly evident in the intestinal mucosa, indicating its increased vulnerability to ischemic damage from sustained increases in intraluminal pressure.

The colonic wall is considered to be at risk of ischemia when the diameter of the cecum exceeds 12 cm and that of the transverse colon exceeds 6 cm.¹⁷⁰ Significant cecal distension is defined as that which exceeds 10 cm, indicating that the risk may begin to increase even before reaching the 12 cm threshold.¹⁷³ The rate of distension has been shown to influence the risk of ischemia; slow or chronic dilations have been found to pose the lowest risk.¹⁷⁴ It is important to note that the risk of perforation is not solely dependent on the absolute size of the cecum, but rather that the duration of cecal distension may be a more determining factor than the diameter reached in predicting the risk of perforation.¹⁷³

Abdominal pain, especially when accompanied by signs of peritoneal irritation, is a significant clinical manifestation, indicative of intestinal ischemia in the presence of occlusion.^{175,176} Leukocytosis and acidosis, have been associated with the prediction of transmural intestinal necrosis in the context of acute mesenteric ischemia.¹⁷⁷ These parameters, in conjunction with lactic acidosis, serve as crucial indicators of potential ischemic complications, necessitating prompt surgical intervention in cases of

intestinal obstruction.¹⁷⁸ Conversely, the presence of free intraperitoneal fluid and combined portal vein and SMV thrombosis, as observed on CT scan, are recognized as independent predictors of transmural intestinal necrosis.¹⁷⁷

Surgery is typically indicated in cases of complete mechanical obstruction of the colon unresponsive to conservative management (figs. 48, 49, and 50). Some specific indications include acute obstruction due to colorectal cancer, cecal volvulus, sigmoid volvulus that does not resolve with endoscopic devolvulation, intestinal perforation, or signs of peritonitis and intussusception.¹⁷⁹

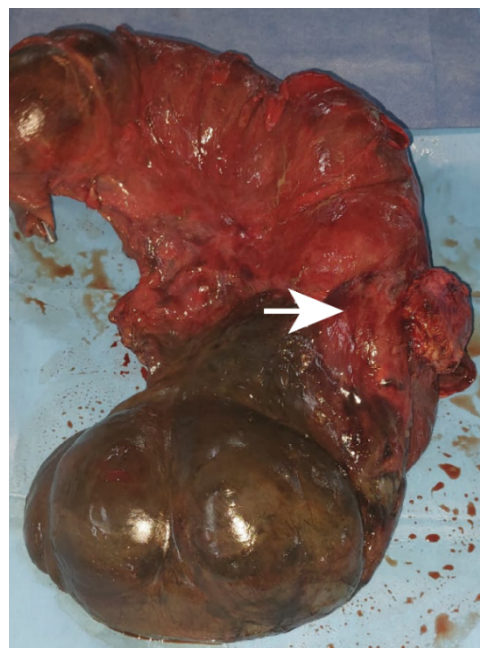


Figure 48. Colonic ischemia with gangrene distal to a colon tumor (arrow).



Figure 49. Abdominal computed tomography of a patient with complete colon obstruction and cecal ischemia due to distension. A dilated cecum with an anteroposterior diameter greater than 12 cm is observed, a radiological finding that indicates a high risk of perforation and ischemic necrosis.

Venous Insufficiency

Venous insufficiency is a less common cause of bowel ischemia than arterial insufficiency, accounting for approximately 5 to 15% of all cases.^{60,64} The progression of damage in venous ischemia is faster than in arterial ischemia, despite presenting less acute symptoms. In the case of venous ischemia, early

thickening of the intestinal wall is observed, while in arterial ischemia, thinning of the wall occurs. External venous compression, thrombosis, or intramural alterations are causes of venous insufficiency.⁶⁰ Volvulus, entrapment by adhesions, intussusception, or surgical obstruction cause external venous compression.^{60,64,65} Mesenteric venous thrombosis has been linked to hypercoagulable states, including those caused by factor V Leiden mutation, protein C deficiency, protein S deficiency, antithrombin III deficiency, the G20210A mutation in the prothrombin gene, and antiphospholipid antibodies.^{60,65}



Figure 50. Cecal wall has undergone necrosis (arrow) as a result of colonic distension in the context of a complete colonic obstruction. Transmurular ischemic compromise is observed, with blackish discoloration and loss of tissue vitality, findings that explain the high risk of perforation.

Acquired hypercoagulable states include dehydration, use of oral contraceptives, paraneoplastic states, and hematological disorders such as polycythemia vera, essential, nocturnal hemoglobinuria, and the JAK2 V617F mutation. There are other related conditions that should be considered, including portal hypertension, intra-abdominal inflammation (e.g., diverticulitis, abscesses, pancreatitis), malignant tumors, sclerotherapy, splenectomy, and the postoperative period.^{60,63} Intramural alterations are a cause of venous obstruction and include conditions such as portal pylephlebitis, mesenteric phlebitis, Behçet's disease, Buerger's disease, systemic lupus erythematosus, rheumatoid arthritis, Wegener's granulomatosis, idiopathic myointimal hyperplasia of the

mesenteric veins, enterocolic phlebitis (lymphocytic, necrotizing, granulomatous), and mesenteric phleboesclerosis (Table 15).^{60,61,63}

Pathological Anatomy

In patients with venous obstruction, mainly in cases of **mesenteric venous thrombosis**, arterial flow to the affected intestinal segment is generally not obstructed. As a result, the tissue progressively distends until drainage occurs through uncompromised collateral veins or until tissue resistance exceeds arterial pressure. At this point, arterial flow ceases, and the affected segment of the intestine progresses to necrosis, similar to arterial insufficiency. As a result, the initial stages of venous insufficiency are marked by significant vascular congestion, hemorrhage, and edema, leading to tissue necrosis.^{60,61-64} Segmental edema of the intestine presents as a swollen and hemorrhagic mucosa, and the circular or semilunar folds are frequently noticeably thickened.⁶⁰ The histopathology is comparable to that observed in cases of arterial ischemia. However, transmural congestion and hemorrhage tend to be more pronounced.⁶⁰ It is crucial to carefully examine for the presence of venous thrombi, phlebitis, or other forms of venopathy. Mesenteric venous thrombosis is characterized by the presence of thrombi in the veins, causing intense transmural vascular congestion, hemorrhage in the affected segments, mucosal congestion, and even ischemic necrosis.^{60,61,180} It is important to note that the presence of thrombosed veins does not always indicate that the thrombus is the primary cause of the damage, as it may manifest as a secondary phenomenon. The organization of the thrombus suggests a long-standing process, which supports the diagnosis of mesenteric venous thrombosis.^{60,61,65} The **portal pylephlebitis** manifests through symptoms such as high fever and jaundice, which are secondary to diverticulitis or appendicitis. The following pathological findings have been associated with this condition: the presence of suppurative thrombophlebitis in the portal venous system and the formation of liver abscesses.^{60,63}

Table 15. Rare venous conditions that can mimic or produce intestinal ischemia, with clinical, etiologic and key pathological features.

Entity	Key clinical features	Etiology	Key pathological features
Portal pylephlebitis	High fever and jaundice after perforated diverticulitis or appendicitis; may lead to liver abscesses	Gram-negative gut bacteria or anaerobes	Suppurative thrombophlebitis of the portal venous system; hepatic abscesses
Idiopathic myointimal hyperplasia of mesenteric veins (IMHMV)	Left-sided abdominal pain, diarrhea/constipation, rectal bleeding in young/middle-aged men; friable mucosa with ulcers and cobblestoning on endoscopy	Possibly secondary to AV fistula	Myointimal hyperplasia of mesenteric veins; mild ischemic changes with thick-walled mucosal vessels; left colon only
Enterocolic phlebitis	Abdominal pain, diarrhea and hematochezia, or right lower-quadrant mass in middle-aged/elderly patients	Unknown	Lymphocytic phlebitis with T-cell infiltrate, venous outflow obstruction, vascular thickening and hemorrhage; may show necrotizing or granulomatous phlebitis; mainly terminal ileum and cecum
Mesenteric phlebosclerosis	Chronic abdominal pain and diarrhea; dark, edematous colonic mucosa	Unknown	Sclerosis and calcification of mesenteric veins; chronic ischemic mucosal changes

Idiopathic myointimal hyperplasia of the mesenteric veins (IMHMV) is a condition that typically manifests in young or middle-aged men and is characterized by left-sided abdominal pain, diarrhea or constipation, and rectal bleeding. The endoscopy revealed friable, ulcerated mucosa with a cobblestone pattern. The pathological diagnosis of IMHMV requires a biopsy, with a thorough examination of the mesenteric vessels. The histological feature of IMHMV is the hypertrophy of the intima layer of small to medium-sized mesenteric veins, without surrounding venulitis or involvement of the arteries. Immunohistochemical staining for smooth muscle actin may highlight the concentric proliferation of smooth muscle cells in the intima and media of the mesenteric veins. Additional findings have been documented, including mucosal vessels with parietal thickening and hyalinization and dilated and ectatic submucosal vessels.^{63,65}

Enterocolic phlebitis is a rare condition that primarily affects middle-aged or older adults. The symptoms include abdominal pain, diarrhea, and hematochezia. In some cases, an abdominal mass in the lower right quadrant may be present. The etiology of this condition is not yet fully understood. Histopathological examination reveals lymphocytic phlebitis, marked by dense T-cell infiltration, venous obstruction, and parietal thickening of the veins, often accompanied by focal hemorrhage. Necrotizing variants have been described, with neutrophilic infiltrate and fibrinoid necrosis, as well as granulomatous forms exhibiting multinucleated giant cells and destruction of the venous wall. The most frequently affected locations include the terminal ileum and the cecum.^{60,63,65}

Mesenteric phlebosclerosis is a chronic disorder whose etiology remains to be elucidated. The clinical manifestations include persistent abdominal pain and diarrhea. A thorough endoscopic evaluation may reveal the presence of a darkened and edematous colonic mucosa. Histopathological analysis reveals the presence of sclerosis and calcification of the mesenteric veins, accompanied by chronic ischemic changes in the colonic mucosa.^{60,63}

Microvascular Insufficiency

Various pathological processes have the potential to affect small-caliber blood vessels, arterioles, capillaries, and venules located in the submucosa and intestinal mucosa.⁶⁰⁻⁶² From a clinical standpoint, the condition may be asymptomatic or manifest as abdominal pain, chronic low-volume bleeding, and anemia.^{60-62,64}

The infiltration of glycation products in patients with diabetes mellitus, the deposition of amyloid proteins in the walls of small submucosal vessels in patients with amyloidosis, and progressive vascular fibrosis in patients with chronic radiation damage can compromise the vascular supply to the mucosa chronically. If exacerbated by hypotension, these conditions can trigger an acute episode.^{60,61,64}

Intestinal angioedema is characterized by one or more episodes of severe, colicky abdominal pain of initially unexplained etiology, which may last for several days.⁶⁴ Radiological studies show segmental edema of the small or large intestine, with no evidence of arterial involvement. Although it can manifest locally in the gastrointestinal tract, angioedema often also involves the oral mucosa, the skin, and the upper respiratory tract.^{60,61,63,64}

Hereditary forms are typically associated with mutations in the C1 esterase inhibitor gene, resulting in low levels of activity. The etiologies of acquired forms are diverse, with the use of angiotensin-converting enzyme (ACE) inhibitors being a common cause. Both variants have been found to be associated with elevated bradykinin levels. This, in turn, increases capillary permeability and promotes the extravasation of intravascular fluid into the extracellular space. The diagnosis is established through the correlation of clinical and radiological findings, supplemented by a quantitative and qualitative assessment of C1 esterase inhibitor.^{60,61,63-65,180}

The most common etiologies of microvascular insufficiency, in conjunction with their morphological characteristics, are outlined in Table 16.¹⁸¹

Table 16. Causes of microvascular insufficiency and corresponding histopathology.

Pathological condition	Features	Predominant location
Disseminated intravascular coagulation-associated thrombosis	Capillary lesions with thrombi	Mucosa
Thrombotic thrombocytopenic purpura	Capillary lesions with thrombi	Mucosa
Sickle cell disease	Erythrocyte obstruction of capillaries	Mucosa
Chronic radiation injury	Dilated (ectatic) capillaries	Mucosa
Leukocytoclastic vasculitis (IgA vasculitis/Henoch-Schönlein)	Predominantly leukocytic vessel inflammation	Submucosa (not always in mucosa)
Diabetes-associated angiopathy	Vascular changes due to diabetes	Submucosa
Amyloidosis-associated angiopathy	Amyloid deposition in vessel walls	Submucosa
Athero-microemboli	Atherosclerotic microemboli occluding capillaries	Submucosa (more numerous than in mucosa)

In cases where the mucosal capillaries are obstructed, as occurs in disseminated intravascular coagulation, thrombotic thrombocytopenic purpura, and sickle cell disease, foci of mucosal ischemia, lamina propria hemorrhage, and superficial erosions may be observed. The severity of the obstruction determines the development of significant areas of mucosal necrosis.^{60,63}

Conversely, obstruction of the submucosal arterioles by atheroemboli can be extensive. However, due to the presence of well-developed collateral circulation in the intestinal mucosa, histological signs of tissue damage are often not evident. Nonetheless, significant compromise to blood flow in the submucosal vessels may result in localized ischemia, manifesting as well-

defined superficial ulcers with a flat base consisting of granulation tissue overlying the muscularis mucosae.

From a histological perspective, atheroemboli are identified as optically empty spaces with sharp edges and an elongated, cigar-shaped morphology. These characteristics are consistent with cholesterol crystals that are dissolved during histological processing. These are found in the obstructed lumens of small submucosal arterioles, usually surrounded by a discrete inflammatory infiltration. The most common cause of atheromicroembolism is instrumental manipulation of the aorta in patients with severe atherosclerosis.^{60,63,181}

Lower Gastrointestinal Bleeding

Historically, LGIB was considered less serious than upper gastrointestinal bleeding (UGIB). However, recent data indicate that patients with LGIB often require longer hospital stays and face a higher mortality or rebleeding risk.¹⁸² The divergent trends between LGIB and UGIB are predicted to become more pronounced, particularly in patients over 80 years of age. This is due to the significant influence of cardiovascular prevention medication and intestinal vascular aging in this demographic. The use of proton pump inhibitors (PPIs) has been associated with an increased risk of LGIB, especially bleeding in the small bowel. This association is particularly significant in patients receiving concomitant treatment with aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs), suggesting a synergistic effect in altering distal intestinal mucosal integrity.¹⁸³

It is recommended that patients presenting LGIB be stratified as unstable or stable. Those with a shock index greater than 1 should be considered unstable (Fig. 51).¹⁸⁴ In cases of stable bleeding, additional classification into major or minor bleeding is suggested, using risk assessment tools such as the Oakland score.¹⁸⁵

$$SI = \frac{HR}{SBP}$$

Figure 51. The shock index (SI) is defined as the ratio between heart rate (HR) and systolic blood pressure (SBP). Its standard value ranges from 0.5 to 0.7. $SI \geq 1$ is indicative of hemodynamic instability and the potential for hypovolemic shock. A $SI > 1.3$ is associated with an increased risk of mortality and the need for urgent intervention.

Oakland Criteria (Table 17):¹⁸⁵

- Acute LGIB: passing red or dark blood through the anus, brown stools, blood mixed with stools, passing clots through the anus, melena without hematemesis.
- Clinically significant bleeding: Hemorrhage associated with systolic blood pressure <100 mmHg, heart rate ≥ 100 bpm, and the need for at least one unit of red blood cells.
- Shock: Heart rate ≥ 100 bpm and systolic blood pressure <100 mmHg.
- Persistent bleeding: Requirement for transfusion of ≥ 2 units of red blood cells and/or decrease in hematocrit $\geq 20\%$.
- Re-bleeding (additional bleeding): New transfusion requirements and/or a decrease in hematocrit of $\geq 20\%$ after 24 hours of clinical stability.
- Rehospitalization for a new episode of LGIB: Additional episodes of LGIB resulting in unscheduled hospitalization within 28 days of discharge.
- In-hospital mortality rate: Mortality from any cause within 28 days of hospital admission for LGIB.

Table 17. Oakland score for LGIB. Patients scoring ≤ 8 , with no other indication for admission, are suitable for safe discharge from the emergency department with outpatient follow-up.

Predictor	Score component value
Age (years)	
<40	0
40–69	1
≥ 70	2
Sex	
Female	0
Male	1
Previous LGIB admission	
No	0
Yes	1
Digital rectal exam (blood)	
No	0
Yes	1
Heart rate (bpm)	
<70	0
70–89	1
90–109	2
≥ 110	3
Systolic blood pressure (mmHg)	
<90	5
90–119	4
120–129	3
130–159	2
≥ 160	0
Hemoglobin (g/L)	
<70	22
70–89	17
90–109	13
110–129	8
130–159	4
≥ 160	0

The Oakland score is a risk assessment tool derived from a UK national audit on LGIB, designed specifically. This classification system enables the categorization of stable bleeding episodes as either major or minor. This is the first scoring system developed specifically for LGIB.¹⁸⁵

This score encompasses seven variables that are systematically assessed during the preliminary evaluation of the patient: age, sex, history of hospitalization for LGIB, findings during digital rectal examination, heart rate, systolic blood pressure, and hemoglobin concentration.

The score is derived by summing the individual values of each component. Patients with a score of ≤ 8 have a 95% chance of being safely discharged from the emergency department and are therefore classified as having minor bleeding. In the absence of other clinical criteria that justify hospitalization, these patients can be discharged with outpatient follow-up.

Patients who score >8 on the Oakland scale are classified as having major bleeding and will likely benefit from hospitalization for evaluation and management.¹⁸⁵

LGIB is a frequent cause of emergency hospitalization. Although endoscopy, radiology, and surgery play a role in its management, the use, timing, and availability of these diagnostic and therapeutic tools are not clearly defined. We hereby propose the following algorithm for the investigation and treatment of severe LGIB (Fig. 52). A recent analysis identified bleeding from colonic diverticula as the most prevalent cause (26% of cases). Forty-eight percent of patients had a benign course, requiring no intervention.¹⁸⁵

Only 29% of cases underwent endoscopic evaluation during hospitalization, and 26% underwent computed tomography. Endoscopic hemostatic treatment was applied in only 2% of cases (8.4% of patients who underwent in-hospital endoscopy), while mesenteric embolization or surgery was employed in less than 1%. The overall mortality rate was 3.4%, primarily attributable to preexisting comorbidities. It is important to note that these cases could have been managed without hospitalization. These findings underscore the necessity for a thorough review of the inappropriate use of red blood cell transfusions in patients with LGIB.

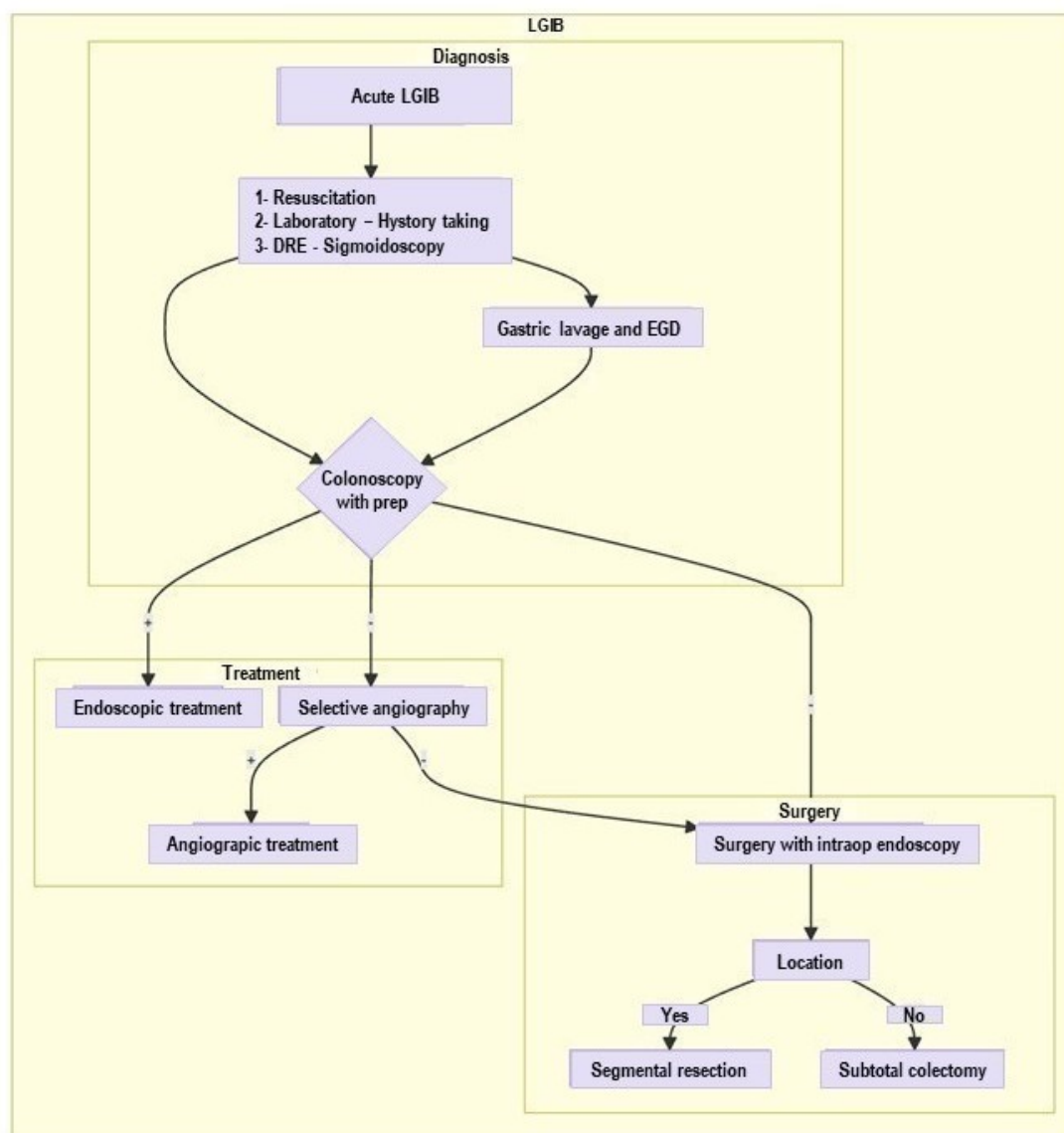


Figure 52. Diagnostic and therapeutic algorithm for acute severe lower gastrointestinal bleeding (LGIB). The diagram illustrates the sequence of initial evaluation and management, beginning with hemodynamic resuscitation), laboratory tests, and anamnesis, followed by digital rectal examination (DRE) and sigmoidoscopy. An esophagogastroduodenoscopy (EGD) can rule out bleeding originating from the upper gastrointestinal tract. Colonoscopy with bowel preparation (prep) guides endoscopic treatment or the need for selective angiography, with possible angiographic treatment or surgery with intraoperative (intraop) endoscopy. Depending on the location of the bleeding, segmental resection or subtotal colectomy is indicated

Angiodysplasias

Angiodysplasia (angiectasia) is the second most common cause of bleeding in the lower gastrointestinal tract in adult patients, with diverticular disease being the first.⁷⁴ A study published by DeBenedet et al.¹⁸⁶ found that of 1,125 patients who underwent esophagogastroduodenoscopy, video capsule endoscopy (VCE), and colonoscopy, 114 were diagnosed with angiectasias, of which 50% were located in the duodenum, 37% in the jejunum, 32% in the stomach, and 44% in the colon. Risk factors for the development of angiectasias and bleeding in the digestive tract include age over 60 years, chronic kidney disease, ischemic heart disease, aortic stenosis, left ventricular assist devices, liver cirrhosis, pulmonary thromboembolism, and the use of antiplatelet agents (impaired platelet adhesion/aggregation).¹⁸⁷

Angiodysplasia is characterized by the presence of a cluster of abnormally dilated blood vessels in the submucosa and mucosa of the lower gastrointestinal tract. The prevalence of angiodysplasia in the general population is 1 to 4%, and its diagnosis increases with age, especially after the age of 60.^{63,102} The prevalence of this condition increases in patients with end-stage renal disease, von Willebrand disease, and aortic stenosis. Heyde syndrome has been described, which links atheromatous aortic stenosis with chronic gastrointestinal bleeding.¹⁸⁸ The concurrence of aortic stenosis and intestinal angiodysplasia ranges from 0 to 41%.¹⁸⁹ The associated bleeding is typically chronic and recurrent, though in 10 to 15% of affected patients, it can manifest as massive bleeding.⁶⁰

While these lesions are typically easily identified during colonoscopies, VCE, and angiography procedures, their microscopic detection in resection specimens can be challenging without the use of specialized injection techniques.^{60,190,191} As demonstrated in the vascular injection studies on resected colons conducted by Boley et al.,⁷⁴ angiodysplasia develops as a result of intermittent partial obstruction of the small veins that drain the colonic

mucosa and submucosa, located at the level of the muscularis propria. It is hypothesized that this obstruction is the result of muscle contraction and increased tension in the intestinal wall, particularly in the region of the colon with the largest diameter (the cecum). Over time, this leads to the dilation and tortuosity of the submucosal veins, as well as the venules and capillaries that drain them. Capillary dilation can result in the loss of capillary sphincter function, which can, in turn, lead to arteriovenous fistulas and secondary alterations in the structure of the feeding arteries.^{61,62} Roskell et al.¹⁹² suggest that the development of angiodysplasias is related to abnormalities in the basal membrane of the vessels.¹⁹² The pathogenesis of angiodysplasia could involve increased expression of angiogenic factors, such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF). These factors can cause alterations in the composition and structure of the extracellular matrix of the bowel wall (Fig. 53).¹⁹³

In cases of chronic kidney disease, bleeding due to angiodysplasia is more prevalent, and its incidence rises in proportion to the duration and severity of renal impairment. The most consistent pathophysiological mechanism appears to be the platelet dysfunction described in these patients.¹⁹⁴ On the other hand, in liver cirrhosis, angiodysplasia is one of the vascular alterations observed in the context of hypertensive enterocolopathy.¹⁹⁵

Angiodysplasias are frequently challenging to diagnose in pathological specimens. In fresh resection samples, only a small focus with prominent blood vessels may be observed, although even these subtle signs may be absent. In formalin-fixed specimens, the lesions are generally not visible on the mucosal surface.⁶⁰⁻⁶² The injection of dyes such as India ink at the time of colectomy facilitates the identification of dilated submucosal vessels (Figs. 54 and 55).

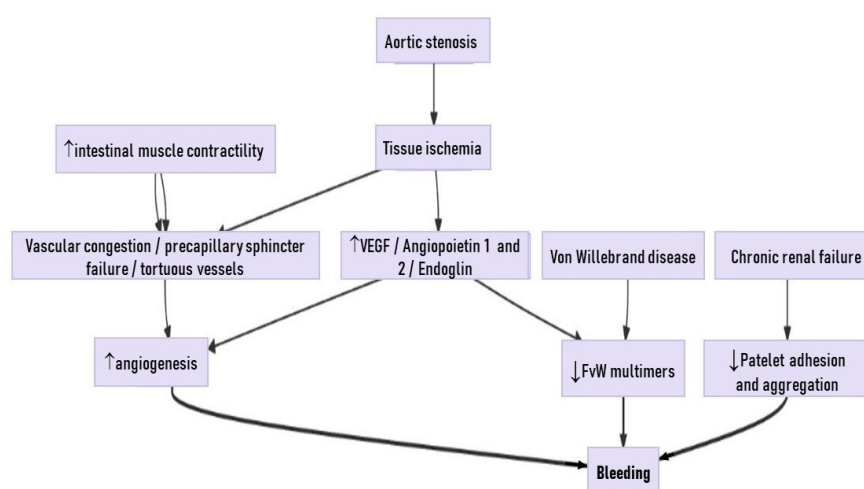


Figure 53. Pathophysiological mechanisms involved in bleeding due to intestinal angiodysplasia. Increased intestinal muscle contractility leads to vascular congestion and dysfunction of the precapillary sphincters, thereby promoting angiogenesis. Tissue ischemia and aortic stenosis contribute to the expression of VEGF, angiopoietins, and endoglin. The degradation of von Willebrand factor (vWF) multimers, whether due to von Willebrand disease or chronic renal failure, decreases platelet adhesion and aggregation, facilitating bleeding.¹⁹⁶

For fixed specimens, it is recommended to section the intestinal wall at the site of suspicious mucosal abnormalities. This procedure will help to highlight the lesion. In the event of a vascular lesion being detected, histological examination reveals the presence of dilated, tortuous, thin-walled submucosal veins, accompanied by capillaries and arterioles with arteriovenous anastomoses. The involvement is predominantly located in the submucosa, although in some cases, it may extend to the mucosa.^{60,63}

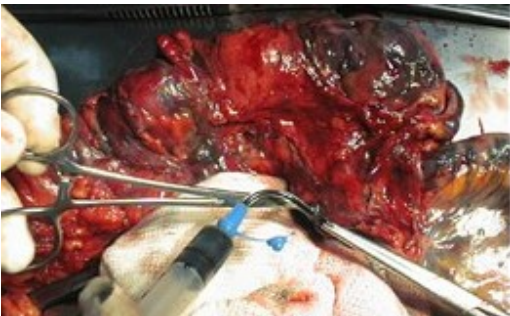


Figure 54. Intraoperative injection of Indian ink into the ileocolic pedicle

In cases of extensive mucosal involvement, clusters of dilated capillaries may be observed, resulting in alterations to the mucosa's architecture. This change displaces the glands and separates the crypts from each other, facilitating more precise identification of the pathology.^{60,61} The diagnosis of angiodysplasia is

typically made through endoscopy, which is indicated to evaluate gastrointestinal bleeding (Fig. 56).

There are several endoscopic options available for evaluating angiodysplasia, including esophagogastroduodenoscopy, colonoscopy, VCE, and enteroscopy. Given the potential for angiodysplasia to occur at any point in the gastrointestinal tract, a combination of endoscopic techniques may be necessary.

Angiodysplasias are characterized by the presence of small (2 to 10 mm), flat, cherry-red lesions with a fern-like pattern of arborizing ectatic blood vessels radiating from a central vessel. Lesions are often multiple. The distinctive appearance may be more apparent in the colon, with 80% of cases occurring proximally to the splenic flexure. Lesions in the small intestine are often smaller than those seen in other parts of the gastrointestinal tract. The fern-like pattern is of particular significance, as other erythematous mucosal lesions or normal blood vessels may be misinterpreted as angiodysplasias (Fig. 56).

The sensitivity of colonoscopy for endoscopic detection of angiodysplasia is unknown because most patients do not undergo angiography, which is the gold standard for diagnosis. However, it is estimated to exceed 80%.¹⁹⁷ Angiodysplasia can be challenging to visualize during colonoscopy in patients who have not undergone optimal bowel preparation or when lesions are located behind a fold. Furthermore, a transient decrease in mucosal blood flow, induced by sedation or insufflation, may render angiodysplasia less visible (Table 18).

Table 18. Stratification of small-bowel angiodysplasias by endoscopic features, clinical manifestations, bleeding causality, and risk of hemorrhagic recurrence (adapted from García-Compeán et al.¹⁹⁶)

Type	Endoscopic characteristics	Causality of bleeding	Clinical manifestations	Probability of recurrence
1	Punctate or patchy lesions with non-pulsatile active bleeding	Certain	Overt bleeding; high frequency of hemodynamic instability	Very high without hemostasis
2	No active bleeding; stigmata of hemorrhage (ulcer, adherent clot, digested blood residues)	High	Frequently overt bleeding; lower frequency of hemodynamic instability than type 1	Highly likely
3	Bright red spots; typical endoscopic images	Moderate or mild	Overt or occult bleeding; low/absent hemodynamic instability; IDA common	Moderate; IDA often dependent on iron therapy or transfusion
4	Pale red spots	Low or none	Usually occult bleeding; chronic IDA; other sources of bleeding should be excluded	Low once other sources are excluded

IDA: iron-deficiency anemia.

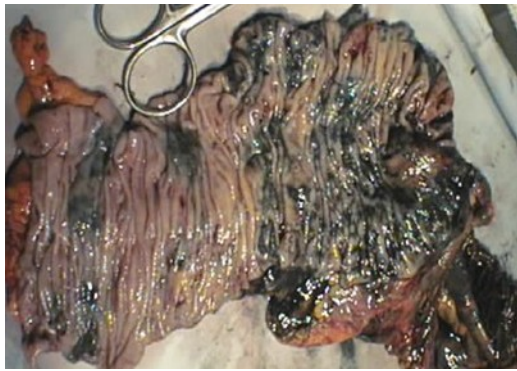


Figure 55. Multiple angiodysplasias in the right colon of a patient with severe gastrointestinal bleeding.



Figure 56. The endoscopic capsule demonstrates the presence of a colonic angiodyplasia. The lesion manifests as a flat, well-defined vascular area in the mucosa, a characteristic finding in acquired arteriovenous malformations in the colon (arrow). These lesions are a frequent cause of recurrent lower gastrointestinal bleeding, particularly in elderly patients or those with cardiovascular and renal comorbidities

Mesenteric angiography can be used to identify angiodyplasias with a sensitivity ranging from 58 to 86%. The efficacy of this diagnostic method depends on the presence of active bleeding during the study. In less than 20% of cases, contrast extravasation can be observed, which is a direct sign of ongoing bleeding.¹⁹⁹ However, even in the absence of active bleeding, characteristic angiographic findings are present, including intense opacification of dilated and tortuous intramural veins with slow emptying, and the presence of vascular tufts corresponding to ectatic submucosal venules and capillaries. In advanced stages, arteriovenous communication secondary to precapillary sphincter dysfunction may be visualized, reflecting the progressive nature of these vascular lesions.

Treatment

Angiodyplasia detected incidentally during screening colonoscopies should not be treated, unless there is a history of LGIB or iron deficiency anemia (IDA), as the risk of bleeding is considered to be potentially low and the lesions tend to be multiple.²⁰⁰ The management of non-bleeding angiodyplasias detected in patients with LGIB is not defined, because there is no way to determine whether they were the cause of occult or overt bleeding. In contrast, actively bleeding lesions require treatment.

Despite thorough endoscopic evaluation, between 10 and 40% of patients may have an undiscovered cause of gastrointestinal

bleeding.²⁰¹ Angiodyplasia is one of the most probable etiologies, particularly in cases involving active occult bleeding, multiple lesions, or hemorrhagic diathesis. In this context, a gradual approach to treatment is reasonable for these patients. It is recommended to treat angiodyplasia found during upper endoscopy or colonoscopy in patients with occult bleeding or IDA, even when the lesions are not actively bleeding at the time of the study.²⁰² If anemia persists despite these measures, other diagnostic and therapeutic options may be considered, such as VCE followed by enteroscopy for treatment.

A variety of endoscopic treatments exist for the treatment of angiodyplasia. The approach selected is contingent upon the location and mode of access to the lesion, the endoscopist's expertise, and the availability of equipment. It is imperative to exercise caution when performing endoscopic therapy in the right colon, given its thin walls. Approximately one-third of patients with angiodyplasia experience rebleeding following endoscopic therapy, with a mean recurrence time of approximately 22 months.²⁰¹

Argon plasma coagulation (APC) is recommended for the management of bleeding related to angiodyplasia.²⁰³ APC involves the use of ionized argon gas to apply a high-frequency electrical current to the affected tissue, resulting in coagulation and hemostasis.^{202, 203} The injection of submucosal saline solution prior to APC treatment has been shown to help protect the intestinal wall and reduce the risk of deep lesions. In a prospective study involving 100 patients, APC demonstrated efficacy in controlling bleeding and stabilizing hemoglobin levels in 85% of cases after a 20-month follow-up period. The probability of remaining free of rebleeding at one year and two years was 98 and 90%, respectively.²⁰³ Another study demonstrated that APC controlled bleeding in 85% of patients with colonic angiodyplasia.²⁰²

Monopolar electrocautery has been utilized as a therapeutic modality for the obliteration of angiodyplasias; however, it exhibits a high rate of hemorrhagic recurrence, estimated at approximately 50% of cases.²⁰⁴ Consequently, APC is currently preferred, as it offers enhanced hemostatic efficacy, a reduced recurrence rate, and an improved safety profile.²⁰⁵

Mechanical hemostatic methods such as endoscopic clips or band ligation have been described for the treatment of localized lesions.²⁰⁶ These methods are particularly beneficial for patients taking anticoagulants and/or antiplatelet agents, as well as those with coagulation disorders.

Angiography is a valuable diagnostic tool that can identify the site of active bleeding, enabling the precise delivery of embolization agents or vasopressin infusions. It is typically used for patients with life-threatening bleeding who are not surgical candidates, or for the localization of lesions before surgical resection.

Selective embolization by angiography has been shown to achieve hemostatic effectiveness of 80 to 90%.²⁰⁶ The procedure involves selectively catheterizing the vessel supplying the bleeding lesion, followed by the injection of embolizing agents. The most commonly used agents include biodegradable sponges and microspheres (microcoils). This technique is associated with complications in 5 to 9% of cases, of which approximately 2% are serious, including hematomas, intestinal infarction, arterial dissection, thrombosis, and pseudoaneurysms.²⁰⁷ Its use is more common in

patients with small bowel angiodysplasias who present with active bleeding, due to the endoscopic inaccessibility of these lesions.¹⁹⁶

Surgery may be considered in patients with bleeding from a clearly identified site who have a high transfusion requirement or severe hemorrhage and who have not responded to and/or are not candidates for endoscopic or angiographic therapies. Preoperative or intraoperative enteroscopy or angiography may be useful in locating lesions. Aortic valve surgery may reduce bleeding in patients with angiodysplasia and aortic stenosis.

Right hemicolectomy for angiodysplasia is considered a second-line therapy. It is indicated in cases where repeated endoscopic ablation has failed, when endoscopic therapies are not available, or in situations of life-threatening hemorrhage.

Surgical resection is associated with a high mortality rate, ranging from 10 to 50%, particularly among elderly patients who often have multiple comorbidities, such as coronary artery disease, coagulopathies, and renal or pulmonary dysfunction. In a study by Meyer et al.,²⁰⁸ right hemicolectomy allowed 63% of patients to remain free of intestinal bleeding after a mean follow-up of 3.6 years, while 37% experienced some degree of rebleeding.

Furthermore, a downward trend has been observed in the necessity of blood transfusions following surgical resection, as well as in patients treated exclusively with electrocoagulation or even in those who did not undergo a specific intervention.

Hormone Therapy

A variety of pharmacological agents have been assessed for their effectiveness in managing gastrointestinal bleeding associated with angiodysplasias.²⁰⁹

Estrogen, administered with or without progesterone, has been used to manage small bowel bleeding in patients with hereditary hemorrhagic

telangiectasia (Osler-Weber-Rendu syndrome), end-stage renal disease, and von Willebrand disease.

Treatment with octreotide has been shown to effectively reduce the need for transfusions in patients with gastrointestinal bleeding secondary to angiodysplasia. A clinical study revealed that 61% of patients treated with octreotide experienced a 50% reduction in transfusion requirements, compared with 19% in the standard treatment group. Similarly, compared to the control group, octreotide reduced the mean number of units transfused by 10.2.²¹⁰ This change was associated with a lower frequency of bleeding episodes and a reduction in the use of health services among patients treated with octreotide.²¹⁰ In addition to its hemostatic effect, treatment with octreotide offered additional benefits, including a reduced need for endoscopic procedures, reduced fatigue, and improved physical and mental health as reported by patients.

Angiogenesis Inhibitors

Angiogenesis inhibitors, such as thalidomide and bevacizumab, are used to treat angiodysplasias.

Thalidomide, which acts by suppressing the plasma expression of vascular endothelial growth factor (VEGF), has an unfavorable safety profile. In a four-month randomized clinical trial, its efficacy was compared to oral iron treatment in patients with angiodysplasias. The results demonstrated that 71% of patients treated with thalidomide exhibited a reduction of more than 50% in the frequency of bleeding episodes, in contrast to only 4% of those treated with oral iron. Seventy-one percent of patients treated with thalidomide reported adverse effects, including fatigue, constipation, and dizziness.

Arteriovenous Malformation

Arteriovenous malformations (AVMs) of the colon and angiodysplasia are not the same from a histopathological and embryological point of view, although both are classified as vascular anomalies of the digestive tract and can cause LGIB. AVMs are a type of vascular anomaly that is present at birth and originates during embryonic or fetal life. While there are some similarities between AVM and angiodysplasia, the two conditions differ in one key way. AVM is characterized by abnormal and direct communication between arteries and veins. This defect causes direct arterial blood flow into the venous system, bypassing the capillary bed, resulting in elevated blood pressure in the veins.^{60,74} From a clinical perspective, arteriovenous malformations (AVMs) can manifest with bleeding at any age and are most frequently observed in the

gastrointestinal tract, particularly in the sigmoid colon and rectum. In the majority of cases, these malformations are located in the subserosa.^{60,74} AVMs are characterized by a tangled network of dilated and tortuous arteries and veins, along with vessels exhibiting an intermediate morphology. A distinguishing characteristic of these malformations is the "arterialization" of veins, which occurs in response to elevated arterial pressure and leads to the thickening of venous walls due to medial hyperplasia.^{60,74} Although the morphological identification of these malformations is possible using histological techniques, angiographic studies offer superior visualizations of the vascular architecture and permit detailed evaluations of abnormal vessel communication.^{60,61,74} Table 19 describes the differences between AVMs and angiodysplasias.

Table 19. Differences between arteriovenous malformation and angiodysplasia of the colon.

Characteristics	Angiodysplasia	Arteriovenous malformation
Etiology	Acquired, associated with aging and intermittent hypoxia of the mucosa	Congenital (although some may be acquired)
Age of onset	Generally in people over 60 years of age	Can occur at any age, even in young people
Common location	Right colon (cecum and ascending colon)	Any part of the gastrointestinal tract
Anatomy	Submucosal capillary and venous ectasia, without true direct artery-vein connection	Abnormal direct communication between arteries and veins, without an interposed capillary bed
Size and morphology	Small, flat, multiple lesions	Larger, single or multiple lesions with higher flow
Symptoms	Chronic or recurrent bleeding, iron deficiency anemia	Can cause massive acute bleeding
Diagnosis	Endoscopy, angiography, capsule endoscopy	Angiography, CT/CT angiography, vascular MRI
Treatment	Argon plasma coagulation, octreotide, selective embolization	Embolization, resective surgery, more complex endovascular management

Vasculitis

Vasculitis is defined as inflammation of the vascular wall, with or without fibrinoid necrosis, which can affect arteries, veins, capillaries, or venules.^{60,63} This process can be localized or systemic, and its clinical expression depends on the caliber of the affected vessel and the pathogenic mechanisms involved. The Chapel Hill nomenclature (2012) is the most widely accepted classification system, integrating vessel size, immunological mechanisms, and possible etiologies.²¹¹ It recognizes the following forms of vasculitis: large vessel vasculitis (Takayasu arteritis, giant cell arteritis), medium vessel

vasculitis (polyarteritis nodosa, Kawasaki disease), and small vessel vasculitis. The latter include ANCA-associated vasculitis—granulomatosis with polyangiitis (GPA), eosinophilic granulomatosis with polyangiitis (EGPA), and microscopic polyangiitis (MPA)—as well as those mediated by immune complexes (Henoch-Schönlein purpura, cryoglobulinemic vasculitis, systemic lupus erythematosus, among others).^{60,63,212-214} Single-organ forms are also described, secondary to systemic diseases or associated with infectious agents and drugs (Tabla 20).

Table 20. Chapel Hill Consensus Conference (2012) classification of vasculitides.

Group	Subtype	Main entities
A. Large-vessel vasculitis	—	Takayasu arteritis; Giant cell arteritis
B. Medium-vessel vasculitis	—	Polyarteritis nodosa (PAN); Kawasaki disease
C. Small-vessel vasculitis	I. ANCA-associated	Microscopic polyangiitis; Eosinophilic granulomatosis with polyangiitis (Churg-Strauss); Granulomatosis with polyangiitis (Wegener)
	II. Immune-complex mediated	Anti-GBM disease (Goodpasture); Cryoglobulinemic vasculitis; IgA vasculitis (Henoch-Schönlein); Hypocomplementemic urticarial vasculitis (anti-C1q)
D. Variable-vessel vasculitis	—	Behçet disease; Cogan syndrome
E. Single-organ vasculitis	—	Cutaneous leukocytoclastic vasculitis; Cutaneous arteritis; Primary CNS vasculitis; Isolated aortitis; Isolated PAN
F. Vasculitis associated with systemic diseases	—	Lupus vasculitis; Rheumatoid vasculitis; Sarcoid vasculitis; Vasculitis in mixed connective-tissue disease
G. Vasculitis with probable etiology	—	HCV-related cryoglobulinemic vasculitis; HBV-associated vasculitis; Syphilis-related vasculitis; Immune-complex drug-induced vasculitis; Drug-induced ANCA vasculitis; Cancer-associated vasculitis

ANCA: antineutrophil cytoplasmic antibody.

Gastrointestinal Involvement

Gastrointestinal (GI) involvement due to vasculitis is uncommon but clinically relevant. In a series of 130,³⁶⁷ digestive biopsies, only 0.02% showed vasculitic lesions, most of which were diagnosed retrospectively.²¹⁵ The clinical presentation includes acute abdominal pain, gastrointestinal bleeding, intestinal ischemia and infarction, perforation, and peritonitis. These symptoms can mimic common conditions such as appendicitis, cholecystitis, or IBD, making initial diagnosis difficult.²¹⁶ Systemic vasculitis has the potential to affect any segment of the digestive tract, from the esophagus to the pancreas. The manifestations of this condition range from mild laboratory abnormalities to fulminant intestinal perforation, which makes diagnosis clinically challenging.²¹⁶ Among medium-vessel vasculitides, polyarteritis nodosa (PAN) in particular presents with GI involvement in 14 to 65% of cases, with postprandial pain (intestinal angina) being the most common GI manifestation. In severe forms, transmural ischemia can progress to necrosis and perforation, with an unfavorable prognosis.²¹⁷

GI involvement in patients with systemic PAN (sPAN) was identified as one of the independent predictors of mortality.²¹⁷ Colonic ischemia may be the initial manifestation of PAN, with histological findings of fibrinoid necrosis in medium-sized arteries. One documented case describes ischemic colitis that led to surgical resection and subsequent diagnosis of PAN.²¹⁸ Another publication reports the case of a young female patient with abdominal pain, diarrhea, and rectal bleeding. A colonoscopy revealed circumferential ulcers from the sigmoid colon to the splenic flexure.²¹⁹ CT angiography showed parietal thickening and hypoperfusion associated with aneurysms in the inferior mesenteric artery. Consequently, she underwent colectomy and the pathological anatomy confirmed the diagnosis of PAN. With regard to small vessel vasculitis, ANCA-associated vasculitis can be categorized into three distinct entities:

- Eosinophilic granulomatosis with polyangiitis (EGPA), a condition marked by severe asthma and peripheral eosinophilia, has been observed to involve the GI tract in approximately 40% of cases. This involvement typically presents as abdominal pain and a risk of perforation.²²⁰
- Granulomatosis with polyangiitis (GPA), primarily affects the airway and kidneys, with GI involvement being rare.²²¹

- Microscopic polyangiitis (MPA), a distinct form of vasculitis, is characterized by p-ANCA/MPO-mediated inflammation without granulomas or eosinophilia, and is primarily manifested by abdominal pain.²²² MPA can mimic PAN, but is distinguished by the absence of immune complexes and granulomas.

Immune complex-mediated vasculitis includes Henoch-Schönlein purpura (IgA vasculitis), a condition common in children, with GI involvement in up to 85% of cases and characterized by abdominal pain, bleeding, and intussusception.²²³ It also includes mixed cryoglobulinemic vasculitis, which is associated with chronic hepatitis C virus infection in approximately 90% of cases.²²⁴ GI, although uncommon (2-6%), includes both the small and large intestine and can present with abdominal pain, diarrhea, ischemia, and perforation. In some severe cases, it leads to visceral failure and even death.²²⁵ In systemic lupus erythematosus (SLE), vasculitis of the intestinal tract typically presents as acute abdominal pain and may exhibit inflammation of small vessels with fibrinoid necrosis and immune complex deposition on histology. While this is a rare complication, its progression can be severe, with

a risk of mesenteric ischemia, transmural necrosis, or intestinal perforation (Fig. 57).²²⁶

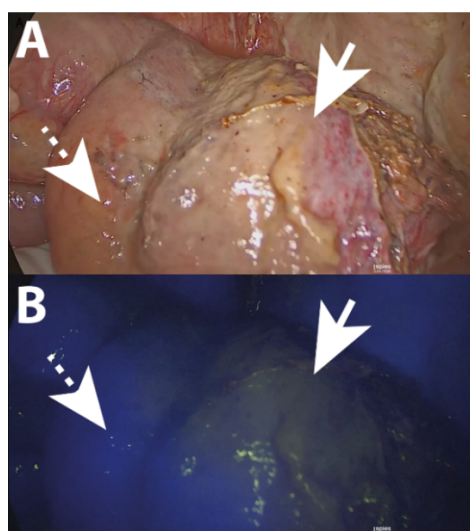


Figure 57. Patient with a history of systemic lupus erythematosus and acute abdomen. During surgical exploration, non-occlusive colonic ischemia with patchy necrosis of the intestinal wall was evident. **A.** White light image showing areas of transmural necrosis covered by fibrin (solid arrow), alternating with segments of viable mucosa (dashed arrows). **B.** Indocyanine green angiography showing perfused areas (dashed arrows) and ischemic areas (solid arrow), consistent with heterogeneous perfusion secondary to microvascular compromise.

Mixed connective tissue disease, although uncommon, can seriously compromise the digestive system. Cases of malabsorption have been reported, as well as perforations of the small intestine and colon secondary to vasculitis.²²⁷

There are also less common forms of GI involvement in the context of vasculitis. Single-organ vasculitis (SOV) is a condition in which inflammation affects one organ of the GI tract. It can occur in any part of the digestive system, from the esophagus to the colon. In some cases, SOV is discovered incidentally during procedures such

as resection or biopsy, which are performed for other reasons.²²⁸

While many of these cases represent localized expressions of an incipient systemic disease, in the absence of associated clinical or serological evidence, they should be considered as primary vasculitis limited to the affected organ.

Conversely, drug-induced vasculitis generally manifests in the skin as leukocytoclastic vasculitis. Involvement of the digestive tract is rare, and when it occurs, it is typically part of a broader systemic picture.

In such cases, diagnosis necessitates establishing a temporal correlation between exposure to the suspected drug and the presence of histological confirmation of small vessel vasculitis.

Surgical Relevance

For colorectal surgeons, it is important to consider vasculitis in the differential diagnosis of acute abdomen. From a surgical perspective, these vasculitides can present as abdominal emergencies, with a high risk of colonic ischemia, perforation, and peritonitis. Colonic involvement by vasculitis can mimic common conditions (such as ischemic colitis not associated with vasculitis), making it crucial to maintain a high level of suspicion when there are unusual endoscopic or surgical findings, especially in conjunction with concomitant systemic data. In certain cases, such as mesenteric vasculitis due to PAN, early laparoscopy may be adequate to assess intestinal viability and guide medical management, thereby avoiding unnecessary resections. The definitive diagnosis is histological, emphasizing the importance of clinical-pathological correlation and intraoperative suspicion when atypical ischemic lesions are found in the absence of obvious atherosclerotic disease (Fig. 58). Tables 21 and 22 highlight some key points relevant to the surgeon.

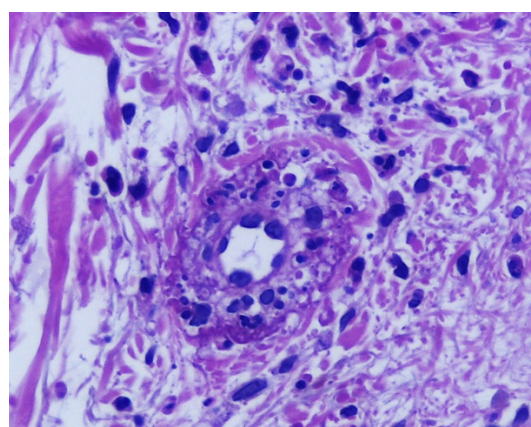


Figure 58. Leukocytoclastic vasculitis. Fragmented neutrophil nuclear debris ("nuclear dust") is observed in the wall of small blood vessels. This histological pattern is associated with Schönlein-Henoch purpura (IgA vasculitis), hypersensitivity vasculitis (drugs, infections), mixed cryoglobulinemia, systemic autoimmune diseases (lupus, rheumatoid arthritis, Sjögren's syndrome), and ANCA-associated vasculitis (such as granulomatosis with polyangiitis or microscopic polyangiitis).

Table 21. Key surgical considerations in gastrointestinal vasculitis.

Aspect	Practical recommendation	Clinical relevance
1. High index of suspicion	Consider vasculitis as a cause of acute abdomen with ischemic findings, especially when overt atherosclerosis is absent	Prevents diagnostic delay and reduces severe complications
2. Urgent surgical indications	Ischemia, perforation, or GI bleeding secondary to vasculitis require prompt assessment and immediate intervention	These complications are potentially fatal if not addressed in a timely manner
3. Value of histopathology	Always submit surgical specimens (colon, small bowel, gallbladder) in atypical ischemic presentations	Clinicopathologic correlation confirms vasculitis and guides immunosuppressive/antiviral therapy
4. Prognostic implications	In systemic polyarteritis nodosa , GI involvement markedly increases mortality	Early recognition enables integrated strategies: surgery plus immunosuppressive management

Table 22. Clinical summary with a surgical approach.

Vasculitis	Mechanism	Colonic presentation	Diagnosis	Surgical implications
PAN	Fibrinoid necrosis of medium-sized arteries	Ischemic colitis, ulceration, perforation	Resection histology	High urgency; resection in severe cases; consider laparoscopy
LES	Vasculitis of small mesenteric vessels	Colonic ischemia, bleeding	Endoscopic biopsy	Urgent evaluation; resection if perforation is present
Other vasculitis	Nonspecific vascular involvement	Pain, hemorrhage, signs of ischemia	Histology + clinical context	Depends on compromise and surgical findings

Conclusions

A comprehensive understanding of the vascular anatomy of the colon and individual anatomical variations is a fundamental component of both colorectal surgical practice and the management of intestinal ischemic pathologies. Vascular disorders of the colon, although rare, are conditions with high morbidity and mortality that require early diagnosis, proper interpretation of perfusion studies, and timely and accurate surgical management.

The integration of advanced diagnostic methods, such as three-dimensional CT angiography, contrast-enhanced ultrasound, and ICG fluorescent angiography, has enabled more precise characterization of intestinal perfusion and facilitates more confident decision-making during surgery. Fluorescence-guided surgery has proven to be an effective tool for the

objective assessment of colonic perfusion and the prevention of anastomotic complications.

From the colorectal surgeon's perspective, the approach to vascular disorders of the colon requires a combination of anatomical knowledge, clinical judgment, and technical skill. The identification of critical perfusion areas, the assessment of ischemic risk, and the correct selection of medical or surgical treatment must be guided by the underlying pathophysiology and current scientific evidence.

Finally, a comprehensive understanding of these disorders, combined with technological advances and interdisciplinary collaboration between surgeons, gastroenterologists, radiologists, and pathologists, will enable continued progress toward earlier diagnosis, safer surgery, and better clinical outcomes for patients with colonic vascular compromise.

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